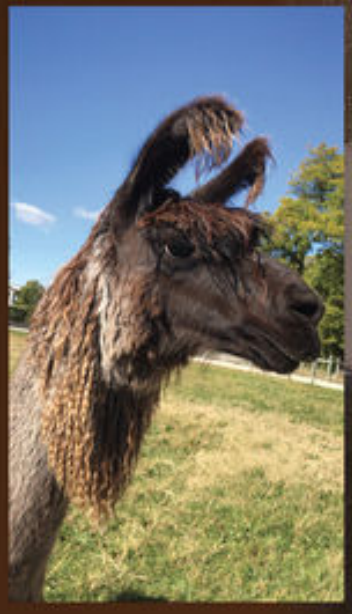
FOURTH EDITION

MEDICINE AND SURGERY OF Camelids



EDITED BY

ANDREW J. NIEHAUS

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Medicine and Surgery of Camelids

Fourth Edition

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I would like to dedicate this book to my family, especially my loving wife, Laurie, and children, Zach and Hannah, who have tolerated me during the countless hours I have spent writing, reading, and editing this text. They have forgiven me for taking time from them to pursue this endeavor. Also, my parents and extended family have always been supportive of me and encouraged me to try new things. Finally, the farm animal team at The Ohio State University, my work-family, have also tolerated my absence from the clinic as I finished this project.

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Preface

I am honored to have been asked to edit the fourth edition of *Fowler's Medicine and Surgery of Camelids*. The previous editions of this text, originally published in 1989 as *Medicine and surgery of South American camelids: llama, alpaca, vicuña, guanaco*, and most recently published in 2010 with contributions from Dr. Walter Bravo, have been seminal works on veterinary medicine, surgery, husbandry, and general management of camelids and these foundational references have influenced most other works published on the subject. To say "I have big shoes to fill" would be an understatement. When Dr Fowler first authored this text, the camelid industry was an emerging industry in North America and was largely considered an exotic farm animal species. At that time, most of the veterinary practice on these animals was based largely on anecdotal evidence and extrapolation from other domestic species.

Dr LaRue Johnson in his 1989 *Veterinary Clinics of North America* on "Llama Medicine," challenged the next generation of camelid scientists to fill the void in the veterinary literature on the subject. Over the past three decades since Drs Fowler and Johnson published their initial works, the camelid industry in North America grew to peak around the turn of the century. Though their commercial population has since declined, the demand for veterinary care and scientific information persists. Today, camelids can hardly be considered an exotic farm animal species in North America as they once were, and much has been learned about these creatures. We have learned that there are many similarities between them and their ruminant counterparts, but we have also learned of significant differences precluding us from making broad generalizations and highlighting the need for camelid-specific veterinary references. Today, PubMed alone indexes over 3600 articles (MeSH Major Topic) published since Dr Johnson's call in 1989. There will always be more to discover but we are answering that call. Though this edition still retains much of the information published in previous editions, I hope the reader of the fourth edition finds that much of the anecdotal evidence in previous editions has been supported or replaced with peer-reviewed, evidence-based, scientific information.

Murray Fowler is a legacy in the fields of camelid and exotic animal medicine. The breadth of his veterinary knowledge was unparalleled, and I cannot think of a veterinarian who has had as great an impact on the profession than he. Though my personal interaction with Murray Fowler was not extensive prior to his passing in 2014, I am grateful to have known him for a short time. He was always very supportive and encouraging of me in my early career, and he treated me as if I was a long-time acquaintance and a respected colleague in the field. He was also willing to take time and go out of his way to offer me advice full of wisdom gained from his illustrious career, or share a related story or antidote, of which he had many.

Though all of my interactions with Murray were related to the field of camelid veterinary medicine, and I realize that many reading this probably know him best for his work with llamas and alpacas, his legacy is perhaps greatest in the field of Zoo and Wildlife medicine. He is recognized as the father of zoological medicine. He was the founder of the American College of Zoological Medicine and the first veterinarian who began teaching zoological medicine in a university setting. He has been described by close colleagues as "a veterinarian that was not afraid of taking on anything," and his accomplishments and achievements run throughout the profession. Learning about his career and accomplishments in so many facets of veterinary medicine is truly humbling.

As I said at the beginning, I am honored to have been given this opportunity, and I realize that I am filling some really big shoes. I certainly could not have done it without the continuous, loving support of my wife, Laurie, and children, Zach and Hannah. Their patience and support with me as I spent countless hours writing, reviewing, and editing we're paramount in the success of this work. I also thank my parents and sister for the love and encouragement with all of my personal and professional endeavors and for instilling in me a work ethic and teaching me that I can accomplish anything.

After I agreed to lead this new edition, one of my earliest realizations was that, unlike Drs. Fowler and Bravo, I would

not be able to do this alone, and I would make it a multi-authored work. I want to thank all of those who contributed to this fourth edition. First, I would like to thank the authors of the chapters. Your expertise in your various subject areas was an extremely valuable contribution. Many others contributed indirectly to the text, many of whom probably didn't even know that their help and support enabled me and my team to accomplish this enormous task. I thank all of the faculty, residents, interns, and technicians of the Farm Animal service at the Ohio State University (some of whom are co-authors in the book), who assumed many of my clinical responsibilities during this time.

Additionally, there are many others who have had great influence on my professional career and without them, I would not be in this position today. I have been blessed with a plethora of incredible mentors, advisors, and friends who helped lay my foundation in large animal veterinary medicine and surgery. To name a few, Drs Dave Anderson, Jeff Lakritz, Bruce Hull, Mike Rings, Clare Whitehead, Pam Walker and Chris Cebra, have had an influence by their mentorship, coaching, teaching, and supporting me in all of my endeavors. If it were not for their guidance and coaching, I am certain that I would not have developed my professional interest or knowledge that has positioned me where I am today.

We've done much of the final editing for this new edition during a global pandemic, and there have been many pitfalls, challenges, and unexpected hurdles. During the production, at least three of the authors have lost loved ones. Some endured days without utilities due to unprecedentedly severe and peculiar weather patterns leading to failing power grids. *Many* of us in academia had to completely restructure our veterinary courses as we learned how to deliver our educational content online, and others had to learn to adjust their clinical practice to a "new normal." We had to juggle efforts on this text with our daily lives, clinical duties, and our responsibilities to our "day jobs." It has not been an easy task, and although many obstacles seemed insurmountable, we persevered.

It is my desire that this edition builds positively upon the previous editions of this text and serves as a useful reference for veterinarians, veterinary students, and camelid enthusiasts. Though we have made our best effort at improving accuracy and the evidence-based medicine included here, we are still learning and undoubtedly there is more yet to discover about these unique animals. I would like to once again give credit to Dr Fowler for establishing the framework for this book and thank all of the many others who contributed substantially to these pages.

"Challenges may come, but you can overcome them and reach the potential destined for you ... Keep on the upward trail" – Murray Fowler, 1999.

Andrew J. Niehaus

About the Companion Website

This book is accompanied by a companion website:

www.wiley.com/go/niehaus/camelids

The website includes:

- Video clips
- PowerPoint of figures from the book, per chapter

1

General Biology and Evolution

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The domesticated camelids of the world have had a significant impact on civilizations throughout recorded history. Populations of camels, referred to as Old World Camelids (OWCs), are indigenous to Africa and Asia, while South American camelids (SACs), also called New World Camelids (NWCs), have evolved in the countries along the western mountains of South America. OWCs include the one-humped (dromedary) camel, the two-humped (Bactrian) camel and, more recently, the wild Bactrian camel of Mongolia. NWCs include alpacas, llamas, guanacos, and vicuñas. Alpacas and llamas exist only as domestic species, while guanacos and vicuñas are undomesticated. The vast majority of camels alive today are domesticated dromedary camels in Africa and the Middle East and domestic Bactrian camels in Asia. Wild Bactrian camels are considered critically endangered with an estimated population of about 1000 individuals inhabiting a few select geographic areas in Asia [1, 2].

Taxonomy

Though the classification of OWCs has remained consistent, the classification of NWCs has changed frequently over time, and controversy continues regarding their classification. Carl Linnaeus placed the llama, alpaca, and Old World camels in a single genus, *Camelus*, in 1758. The scientific name for the dromedary camel is *Camelus dromedarius* (Figure 1.1) and that for the Bactrian camel is *Camelus bactrianus* (Figure 1.2). The more recently classified wild Bactrian camel of Mongolia is named *C. bactrianus ferus* (Figure 1.3) [3–6]. More recent taxonomic classifications place SACs into the genera *Vicugna* and *Lama*. The most widely accepted nomenclature for SACs is the following: alpacas are classified as *Vicugna pacos*, vicuñas are classified as *Vicugna vicugna*, llamas are classified as *Lama glama*, and guanacos are classified as *Lama glama guanicoe*. (Table 1.1) More detailed information on taxonomic history is found in earlier editions of this text.

General Biology and Genetics

The karyotype of all camelids is 2n = 74. All SACs have produced fertile hybrids. Dromedary and Bactrian camels also produce fertile crosses. Though less common, it is possible to produce camel – SAC hybrids, however, most experimentation with this hybridization has resulted in non-viable offspring. Llama – camel crosses are known as camas [7], and alpaca – llama hybrids are known as wari or wakayu [8]. In Israel, a cross between an alpaca male and a dromedary female by artificial insemination produced a stillborn full-term fetus. One report of a guanaco inseminated with camel semen produced a viable calf [9].

Significant size differences exist among camelid species, though they maintain similar anatomic relationships. Domesticated camelids tend to have a larger body size than their wild ancestors. Though there is overlap, vicuñas tend to have the smallest body size, followed by alpacas, guanacos, and llamas are the largest [10]. Environmental pressures influenced different adaptations of camels from the SACs. The camels completed Pleistocene evolution in a semidesert environment in southern Asia, the Middle East, and North Africa and developed sophisticated adaptations for dealing with heat and dehydration. The SACs became adapted to South American habitats, especially the high-altitude lands of the Andes.

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Figure 1.1 Dromedary camel cow nursing her calf (*Source:* Photo courtesy of Dr. Sharon Deem at the Saint Louis Zoo Institute for Conservation Medicine).

Figure 1.2 Bactrian camel.

Camelids are often compared to true ruminants, though distinct differences in gastrointestinal (GI) anatomy and physiology place them in separate phylogenic suborders. Geologic evidence suggests that camelids and ruminants began to diverge near the beginning of the tertiary period from primordial, monogastric species. Camelids have a complex, three-compartmented stomach. Similar to ruminants, camelids consume fibrous forage and have developed similar foregut fermentation systems by parallel evolution (Chapters 3 and 12). Camelids regurgitate and rechew ingested forage, as do ruminants, however, camelids have more efficient mechanisms than ruminants for extracting protein and energy from poor quality forages [11]. More detail on the differences between camelids and ruminants are found in earlier editions of this text [12].

South American Camelids

The guanaco (Figure 1.4) has the broadest distribution, both historically and currently, of the four SACs. Four geographic variants of guanaco have been described, ranging from sea level in Tierra del Fuego at the southernmost tip of South America to 4600 m in the Andes [8, 10, 13]. The northernmost populations exist at latitude 8° south in Peru (Figure 1.5). Guanacos live in both migratory and sedentary groups [16]. Captive-born guanacos may be tamed and handled similarly to llamas; the Incas used them as pack animals [8]. All guanaco subspecies share uniform coloration: a dark brown upper body, neck, and limbs with whitish fiber on the underside of the neck and belly and a grayish to black face [8]. However, their size varies based on geographic distribution with the smallest being from the low- and mid-latitudes and the largest animals in the higher latitudes such as Tierra del Fuego [10].

Vicuña distribution is limited to the puna (Quechua for highland) life zone of the Andes (elevation 4200–4800 m) [14]. The vicuña (Figures 1.6 and 1.7) is the smallest of the SACs and has the finest fiber coat [10]. It has a cinnamon-colored coat, white underparts, a pale cinnamon face, and a bib of white hair on the chest. There are two geographic subspecies of vicuña: the Peruvian, with long white hairs on the bib, and the Argentinian, with shorter hairs on the bib. In Incan civilizations, the vicuña was considered the property of the kings, and only royalty were allowed to wear garments made from their fiber.

(a)

(b)

Figure 1.3 (a) Wild Bactrian camel from the Gobi Desert in Mongolia. (b) Multiple wild Bactrian camels are pictured here. The wild Bactrian camel is considered to be critically endangered today and is only found in a narrow region in southern Mongolia. *Source:* Used by permission from George B. Schaller, Wildlife Conservation Society.

Table 1.1 Taxonomic classification of common Artiodactyla[a].

Common Name	Kingdom	Phylum	Class	Order	Suborder	Family	Genius	Species	Subspecies
Dromedary Camel	Animalia	Chordata	Mammalia	Artiodactyla	Tylopoda	Camelidae	*Camelus*	*dromedarius*	
Bactrian Camel								*bactrianus*	
Wild (Mongolian) Bactrian Camel									*ferus*
Alpaca							*Vicugna*	*pacos*	
Vicuña (Argentine)								*vicugna*	*vicugna*
Vicuña (Peruvian)									*mensalis*
Llama							*Lama*	*glama*	
Guanaco									*guanicoe*
Cattle[b]					Ruminantia[c]	Bovidae	*Bos*	*taurus*[b]	
Sheep							*Ovis*	*aries*	[d]
Goat[e]							*Capra*	*aegagrus*	*hircus*[e]
Deer						Cervidae	*Odocoileus*	*virginianus*	
Pig					Suiformes	Suidae	*Sus*	*domesticus*	
Hippopotamus[f]						Hippopotamidae	*Hippopotamus*	*amphibius*	

[a] Does not include all the Artiodactylids but includes the most common to highlight the general relationship between camelids and other Artiodactylids.
[b] Common American/European cattle. Indian ("humped") cattle are classified *Bos indicus* or *Bos taurus indicus*.
[c] Members of the suborder "Ruminantia" are often referred to as "true ruminants" however members of the Tylopoda (camelids) are also foregut fermenters and therefore undergo a process termed "rumination" and therefore camelids can be referred to as "ruminants."
[d] Multiple subspecies classified.
[e] Domestic Goat. Some sources classify this as simply *C. hircus*. Multiple species (subspecies) classified.
[f] Though hippopotamus is classified as a suiformes, it has a three-chambered gastric compartment, different than Suidae.

Figure 1.4 Guanaco (*Source:* Photo courtesy of Dr. Benji Alcantar at Wildlife Safari).

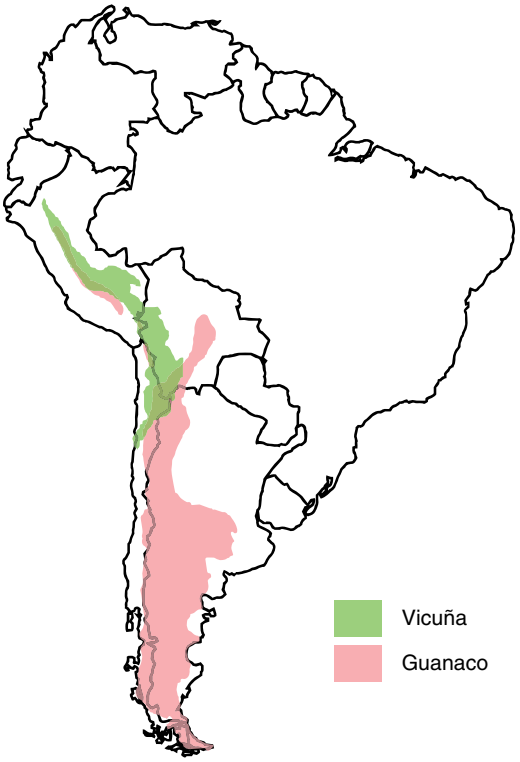

Figure 1.5 Geographic distribution of undomesticated SAC's [14, 15].

Figure 1.6 Peruvian vicuña. Note the long bib hair on their ventral chest (*Source:* Photo courtesy of Dr. Gianmarco Rojas Moreno at the Parque Zoologico Huachipa – Perú).

Figure 1.7 Argentine vicuña. Argentine vicuña's have short bib hair.

Figure 1.8 Heavily fibered huacaya alpaca.

The two breeds of alpaca; huacaya (Figures 1.8 and 1.9) and suri (Figure 1.10), have both become popular in the United States. Alpacas are separated on the basis of fiber coat characteristics. In Peru, 90% are of the huacaya breed. Huacaya fiber is shorter than that of the suri breed and is crimped and spongy, giving it the appearance of Corriedale sheep wool [17, 18]. The coat of suri alpacas consists of long fibers with no crimp that hang down alongside the body in ringlets or dreadlocks. Alpaca coloration varies from white to black with intermediate shades and combinations. The alpaca is the primary SAC fiber producer of the Andean highland.

Similarly to alpacas, the Spanish conquest in the sixteenth century led to a rapid decline in the number and the geographic distribution of llamas in South America. While smaller groups of llamas presently live in Ecuador and Columbia, the largest number of llamas are found between 11° south latitude and 21° south latitude in higher elevations [19]. Two breeds are recognized in Peru: the woolier varieties are called "ch'aku" in Quechua (Figure 1.11), and those individuals with less fiber on the neck and body are called "q'ara" (wooless) (Figure 1.12) [17].

Tables 1.2 and 1.3 outline general similarities and differences between South American camelids.

Figure 1.9 Huacaya alpaca female.

Figure 1.10 Suri alpaca.

Camels

Dromedary camels (Figure 1.1) are especially adapted to life in hot, arid areas of the world, notably the Middle East, North Africa, and India, with a large feral population in Australia. Dromedaries have been used since ancient times for transport of people and goods, warfare, food, fiber, and companionship. With the advent of modern transportation and the establishment of paved highways, camels have become somewhat obsolete. However, some countries still treasure some of the traditional uses of dromedaries and have fostered camel racing as a sport.

The Bactrian camel is adapted to the cooler, arid climates of Mongolia, southern areas of the former Soviet Union, China, and south-central Asia. It is a beast of burden for carrying goods throughout its native lands and was the cargo carrier of goods along the Silk Road from inner China to the Mediterranean. Bactrian camels have a heavy fiber coat to cope with the cooler, arid climate and provide the bulk of the fiber used in the manufacturing of camel hair garments.

Figure 1.11 Wooly-necked llama. In Peru these animals are called "ch'aku" in Quechua.

Table 1.4 outlines characteristics of camels.

Evolution

Camelid

Camelid evolution began in North America over 40 million years ago during the Eocene epoch [18]. Geologic and paleontologic time scales are estimates and subject to revision.

Figure 1.12 Llama.

Figure 1.13 provides a diagram of the relationships between the various artiodactylid families since the Eocene epoch. Webb published a definitive work on the evolution of Pleistocene camelids based on paleontologic evidence [20]. He suggested three major tribes, separated as early as the Eocene epoch. This family tree is questionable, with insufficient fossil records to trace lines accurately (postulates indicated by broken lines) (Figure 1.14) [20, 21]. Two tribes, Camelopini (Camelops) and Camelini (Camelus), evolved in North America only west of the Mississippi River. The tribe Lamini (Lama) was also found in Florida [21, 22].

The Pleistocene epoch was characterized by a series of periods of extreme cold and glaciation in northern North America and Europe [23]. The last glacial retreat occurred about 10 000 years ago, marking the beginning of the recent epoch. It was during the Pleistocene epoch that the Camelidae flourished [24–26]. Many genera in the family Camelidae became extinct, for unknown reasons, before the recent epoch.

Camel

Asia and Alaska are now separated by the 90-km (56-mile)-wide Bering Strait. However, during the height of one of the early Pleistocene glacial periods, the sea level was lowered sufficiently to expose a wide land bridge [27]. Plant and animal species moved back and forth across this bridge; the camel line of Camelidae migrated from North America into Asia to continue the evolutionary process, dying out in North America.

Once in Asia, Camelus radiated through eastern Europe (Romania and southern Russia), the Middle East, and North Africa as far west as the Atlantic and as far south as Tanzania. It is likely that the dromedary evolved from the Bactrian camel, though the hump(s) may be an acquired characteristic of domestication. Wild camels had become virtually extinct in North Africa before historic times (3000 BCE). Only *C. bactrianus ferus* now exists in the wild state in one small area in the Trans-Altai Gobi Desert on the border of Mongolia and China, with a limited population of roughly 1000 animals (Figure 1.15) [4, 5, 28].

South American Camelids

The first SACs migrated to South America at the beginning of the Pleistocene epoch (approximately three million years ago) when an open land connection between North and South America developed [8, 29]. The Isthmus of Panama was formed by volcanic eruptions from the ocean floor, forming a series of islands that ultimately became interconnected (the Caribbean land bridge). The major

Table 1.2 Characteristics of South American camelids.

Characteristic	Vicuña	Alpaca	Guanaco	Llama
Types/breeds/ subspecies/races	**Peruvian** – Apron or bib on frontal chest	**Huacaya** – Fibers with light crimp. Fleece at right angles to body.	May be as many as four subspecies or races	South American breeds: Heavy neck fiber (chaku, lanuda, tapada)
	Argentine – No chest bib	**Suri** – Fiber lacks crimp. Fleece hangs in ringlets.		Short neck fiber (ccara, pelada). No North American breed standards. Alpaca and Llama Show Association show classification: Light, medium, and heavy wool divisions.
Conformation				
Topline	Straight with rounded rump	Straight with rounded rump	Straight or slightly rounded from withers to tail	Straight from withers to tail
Head and neck carriage				
Alert	Vertical	Vertical	Vertical	Vertical
Resting	Vertical	15° from vertical	Vertical	Vertical
Ears	Short, curved on both borders	Short, spear-shaped, curved on both borders. Huacaya – sharp tip. Suri – rounded tip.	Medium length, curved on both borders	Long, inner border straight or curved inward (banana ears)
Face	Short	Short	Medium	Long
Pastern	Sloped	Almost vertical	Sloped	Sloped
Fiber				
Diameter in microns (avg.)	10–30 (13–14)	Huacaya – 16–40 (22) Suri – 16–35 (23)	18–24	Undercoat – 16–40 (26) Guard hair – 40–150 (70)
Quality	Finest of all SACs. Staple short.	Excellent. Staple long.	Inner coat is excellent; juvenile pelts are used for fur garments. Staple short.	Variable; inner coat may make excellent garments. Staple may be long or short.
Coverage	Uniform over body, head, and upper legs. No topknot.	Topknot present. Fiber extends below the knee and hock.	No topknot. Uniform over body, head, and upper legs.	Fiber usually doesn't extend much below the knee or hock or onto the face.
Guard hair	The bib and lower body fiber are guard hairs.	Highest-quality animals have no guard hair.	Concentrated on lower parts of body and limbs.	Numerous.
Color	Color pattern similar to that of guanaco, but the basic body color is a yellowish light brown. The white in front of the rear limbs may extend to the top of the back. Argentine subspecies has long white guard hairs on the chest, but this is extended into a bib (8–14 in./ 20–35 cm) in Peruvian subspecies.	22 solid colors recognized, ranging from white to black. Multicolored fleeces also produced.	Basic body color light to dark reddish brown (cinnamon) above, whitish hair below (countershading). White extends up behind the foreleg and in front of the rear leg, around the perineum, inside of legs, and up the bottom of the neck. Front and outside of the upper limbs are body color or gray. Head, face, and ears are dark gray to black, with the darkest color over top of the head and bridge of the nose.	Numerous solid colors from white to black. Multicolors (pinto, appaloosa) also seen.
Teeth	Incisors long, narrow (sides parallel) and continue to grow throughout life. Enamel present only on labial side of the incisor teeth.	Incisors elongated and continue to grow into adulthood. Enamel present only on labial side of incisors.	Incisors broad, spatulate-shaped and do not continue to grow. Enamel surrounds the crown of the incisors.	Incisors like those of guanaco.
Feeding strategy	Grazer/browser.	Prefers to graze succulent forage in marshes and moist places but also utilizes drier grasses and shrubs.	Uses broad range of habitat for grazing and browsing.	Browser by preference but grazes grasses, even dry, harsh species.

Table 1.3 Weights and sizes of South American camelids.

Characteristic	Vicuña	Alpaca	Guanaco	Llama
Weight (lb/kg)				
Adult	99–121/45–55	121–200/55–90	220–265/100–120	250–550/113–250
Birth	9–13/4–6	13–20/6–9	18–33/8–15	18–40/8–18
Height (in/cm)				
Adult, at withers	34–38/86–96	30–38/76–96	43–45/110–115	40–47/102–119 Some imports 38 in.

Table 1.4 Characteristics of camels.

Characteristic	Dromedary *Camelus dromedarius*	Bactrian *Camelus bactrianus*	Wild Bactrian *C. bactrianus ferus*
Breeds/types	50 different breeds recognized Draft type: heavy body, stocky legs Riding type: Slim body, long legs Racing type: Similar to riding	Geographical differences	Single type
Weight (kg)	300–650[a]	450–700	450–690
Weight of newborn (kg)	26–45	35–54	?
Height at shoulder (cm)	180–210	180–195	180–200
Body length (cm)	120–200	120–200	140–156
Shape	1 firm, upright hump	2 large humps, may be flopped over	2 small, conical humps
Color	Cream to tan to dark brown	Cream to tan to dark brown	Cream to gray–brown
Unique anatomy	Male has a soft palate diverticulum (dulaa) which may protrude from mouth	No dulaa, ears 15 cm	No dulaa, prominent toenail, small foot and flat sole for rapid gait to escape predation. Able to drink salty water, face narrow, ears 10 cm
Fiber/hair	Diameter 20–50 μ	Diameter 10–40 μ, long staple, primary source for camel hair garments.	Short fiber
Special adaptations		Adapted to cooler, arid environments.	Adapted to the deserts of northern China and Mongolia.
Running speed (kph)	21.6–40.3	15–20	40

[a] Males, castrated as juveniles, may grow taller and larger than 1134 kg (2500 lb).

earliest SAC genus appearing in South America was Hemiauchenia (Tanupoloma), which radiated throughout the flatter regions east of the Andes. During the middle Pleistocene, the genera Palaeolama, Lama, and Vicugna developed from the long-limbed, flatland-adapted Hemiauchenia. These genera had shorter limbs, which more easily adapted them to the mountainous Andes.

Various species of Palaeolama migrated back to North America. Fossils associated with North American Hemiauchenia have been found along the Gulf Coast and Florida (Figure 1.16) [30].

Domestication

Camels

The approximate time of domestication of a few animals is listed in Table 1.5. The precise time and location of domestication of the Bactrian camel is unknown, but it is thought to have occurred sometime prior to 2500 BCE. On the border of Turkmenistan and Iran on the east side of the Caspian Sea [4]. The name Bactrian is derived from a place called Baktria, located on the Oxus River in northern Afghanistan.

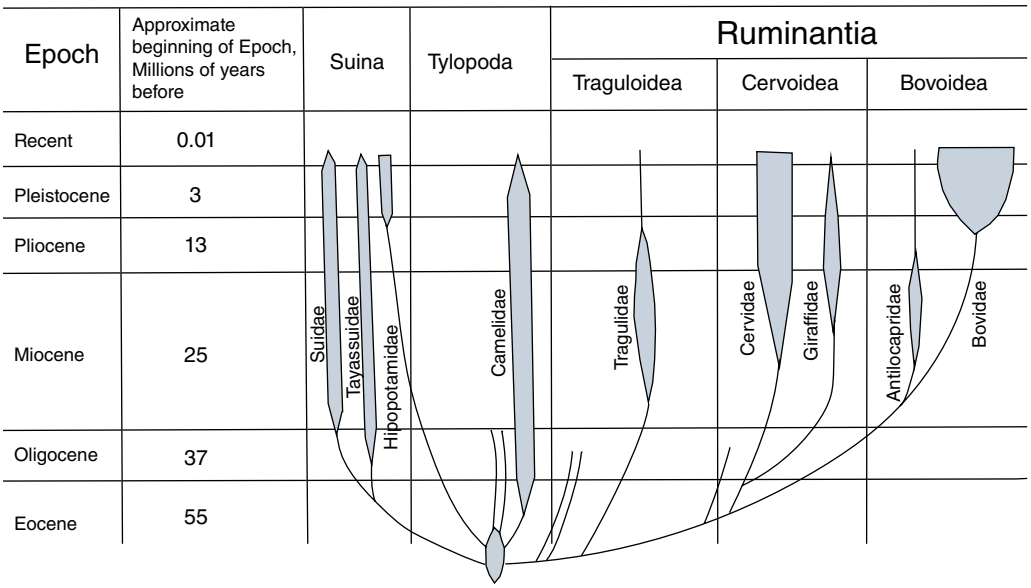

Figure 1.13 Suggested evolution of recent artiodactylids. *Source:* Adapted from Romer [24] and Simpson [29].

Figure 1.14 Skeletons of various prehistoric camelids removed from the La Brea tar pits in Los Angeles, California. Scientists have discovered an incomplete fossil record.

Strangely, this is not the place of origin of the domestic two-humped camel, nor is the species found in this area today.

Domestic Bactrian camels had spread north into southern Russia by 1700–1200 BCE. and were in western Siberia by the tenth century BCE. Bactrian camels were used in China as early as 300 BCE. as the original Silk Route camels but were later replaced by crossbreeds of the dromedary and Bactrian camels [4].

Domestication of the dromedary occurred prior to 3000 BCE. in the Arabian peninsula. The term "dromedary" is derived from dromos (Greek for road) and thus is only directly applicable to the riding or racing dromedary camel. However, dromedary is the name used throughout the world for this species, which existed in historic times only as a domesticated animal. Dromedaries were first associated with nomadic Semitic cultures and did not become important until the rise of the Arabian culture [4].

Dromedaries were reintroduced into North Africa in the third century BCE. More were brought into Egypt during the Roman period, after the third century CE., but became

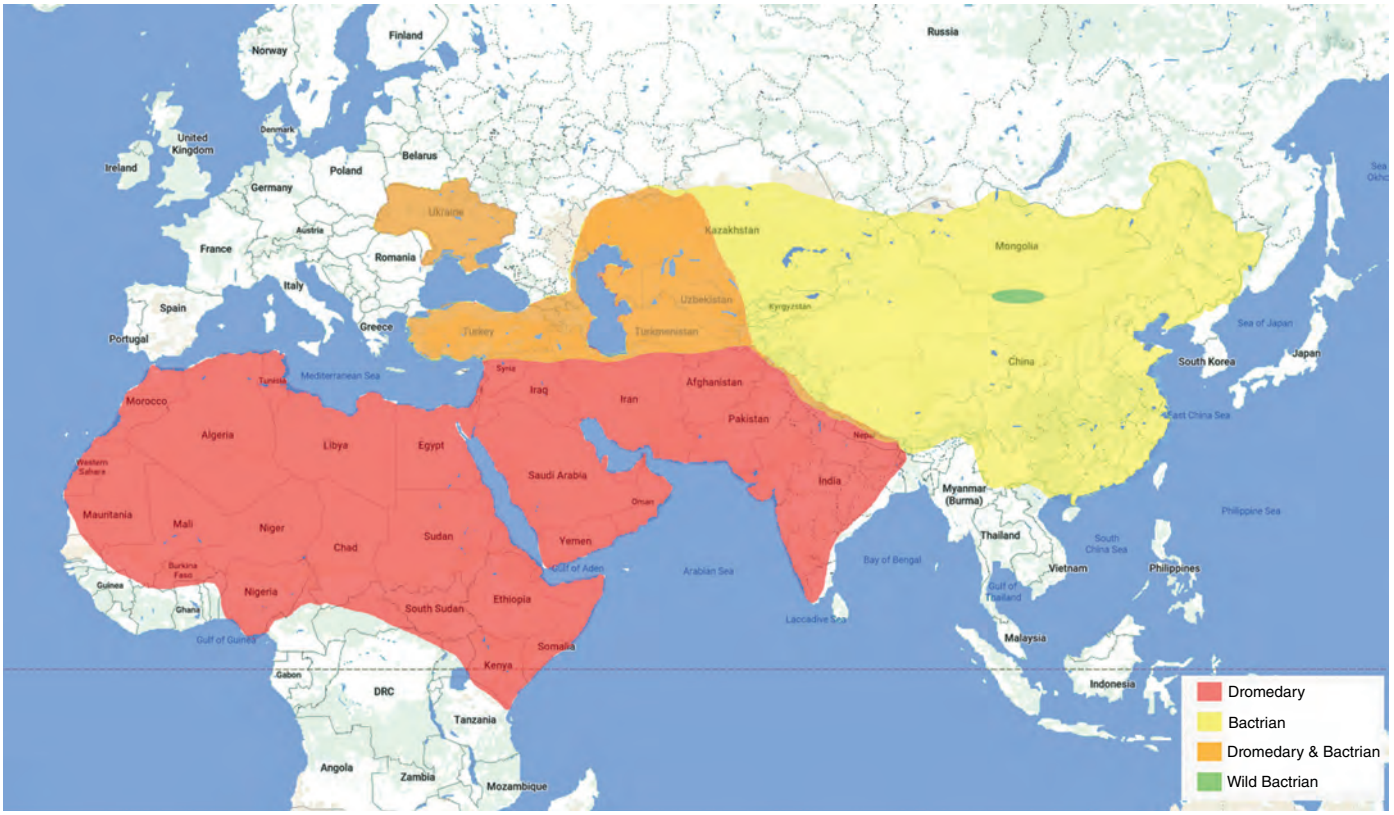

Figure 1.15 Native distribution of camels in Africa and Asia.

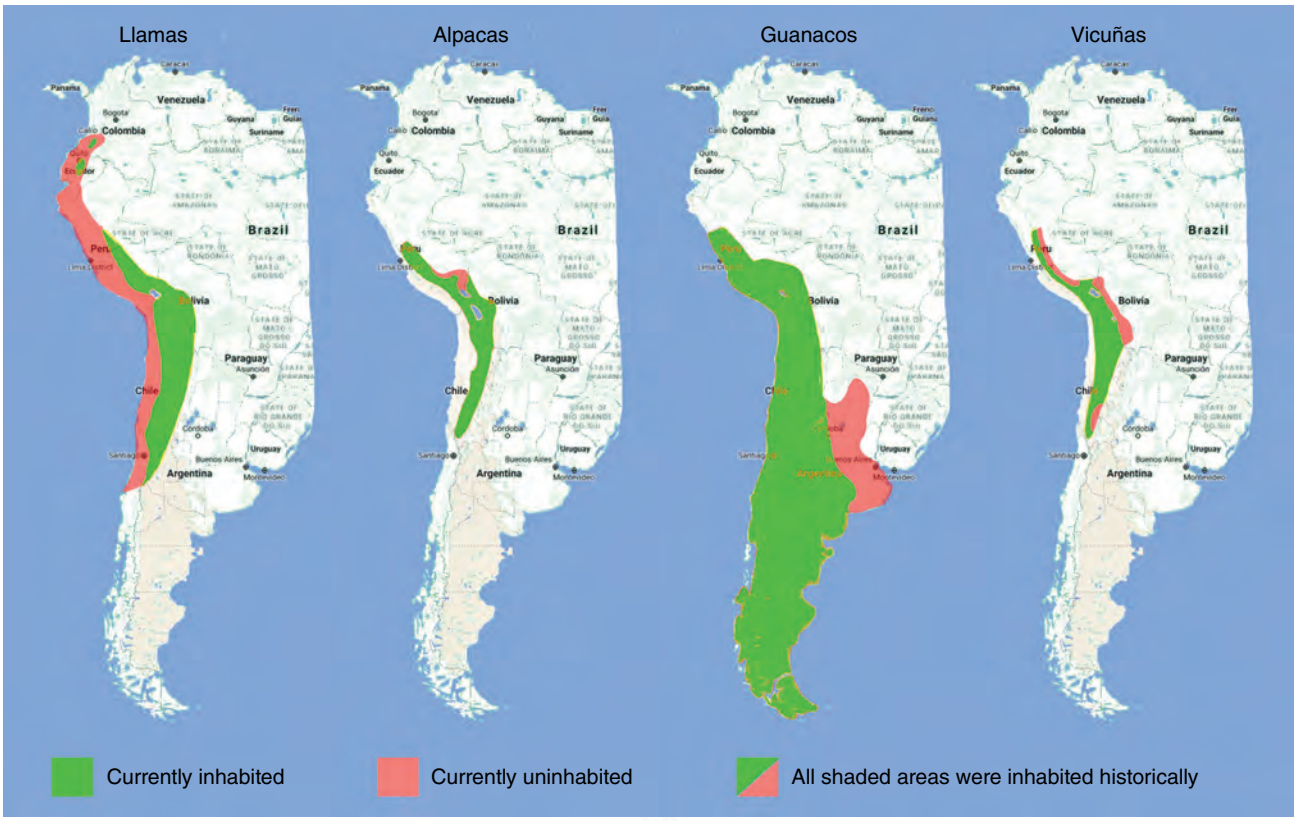

Figure 1.16 Historic and current distribution of SAC in South America.

Table 1.5 Approximate number of years that certain animals have been domesticated.

Reindeer	14 000	Llama and alpaca	6000–7000
Dog	12 000–15 000	Horse	5500
Goat	11 500	Dromedary camel	5000
Sheep	11 000	Bactrian camel	4500
Cattle	9000	Asian elephant	4000
Pig	9000	Cat	3000–4000

important domestic animals only with the Moslem conquests of Egypt in the seventh to eleventh centuries CE.

South American Camelids – Llamas and Alpacas

The cradle of llama domestication is the Andean puna (elevation 4000–4900 m), probably around Lake Titicaca, at approximately 5000–4000 BCE. Alpaca domestication probably occurred elsewhere, perhaps near Telarmachay. Alpaca-type incisors have been found in middens at Telarmachay, dated at 4000–3500 BCE. [8, 31, 32]. Once domesticated, llama and alpaca herding economies spread beyond the limits of the puna and became important in the economy of the Andean people from sea level to high mountain elevations [32, 33].

The Inca empire was dependent upon the llama and alpaca for food, fuel, clothing, transport of goods (the wheel was not introduced to South America before the conquest), and religious ceremonies. All SACs were the property of the government, and production of domestic species was rigidly controlled. The maximum numbers and broadest distribution of SACs developed under Inca rule. After the Spanish invasion of 1532, and the introduction of European breeds of livestock, numbers and distribution of SACs declined. Invaders did not recognize the unique adaptations of these species for this environment and considered them an unimportant component of the life of this region. However, llamas and alpacas survived because they are essential to Andean culture. Only in the last several decades have these animals been recognized as a valuable resource and efforts made to research their unique physiology and adaptation to hostile environments. They are the most reliable source of food, fiber, and fuel in the high, cool Andean environment. SAC ownership is the primary source of wealth for indigenous people [8, 32]. Currently, llamas and alpacas are raised by traditional pastoralists.

Additional historical information is found in earlier editions of this text.

In the nineteenth century, SACs were exported to other countries from South America as zoo animals. Peru enacted legislation in 1843 prohibiting the export of live alpacas. Approximately 90 years ago, all of the Andean countries banded together to prevent exploitation of SACs by other countries. No legal exportations occurred from then until the 1980s, when the ban was lifted from alpacas and llamas [17, 34].

North American llamas have expanded from the small population imported from South America prior to 1930 and a few animals that had been imported from other countries. Current numbers of North American llamas are estimated to be between 100 000 and 120 000, and alpacas are thought to number more than 150 000 [35]. Sporadic importation of llamas and alpacas from Chile into the United States began in 1984 after the U.S. government periodically recognized Chile as free of foot-and-mouth disease. Importations have also originated in Bolivia and Peru, but these must meet special quarantine protocols for animals deriving from countries where foot-and-mouth disease is endemic. A few animals have been imported by England, New Zealand, Canada, and Australia.

Uses of Camelids

Camels are an important part of the culture of the nomadic peoples of Asia and the Middle East, supplying food (meat and milk), fuel (the fecal pellets), fiber (clothing, ropes), leather, transportation (packing, riding; Figure 1.17), and racing. Special breeds of dromedaries were developed for riding and became important in the mobilization of military expeditions. Camels are also used for draft purposes, pulling plows and wagons (Figure 1.18). Camels were used heavily in Australia prior to the development of modern vehicles for packing and pulling heavy and awkward items into the Outback. The feral camels of Australia are descendants of escapees. Camels also were used in the United States as part of a military experiment in the hot, desert environment of the Southwest. The Camel Corps was soon disbanded for various reasons.

Camel racing has become a popular sport in Kuwait, Saudi Arabia, Qatar, Oman, and the United Arab Emirates (Figure 1.19). Adult female camels over seven years of age are generally used for major racing. Male camels are less easily managed. Younger camels may participate in short races but the major races are 4–10 km.

The camels are ridden by lightweight jockeys (formerly by children) sitting behind the hump. They are now sometimes ridden by mechanical robot jockeys. The camels move out at a gallop and may reach speeds up to 40.32 kph (25.05 mph) for the first 2 km. Then, they settle down to a fast pace at an average speed of 32.4 kph (20.1 mph).

Camels may change gaits (pace to gallop, and vice versa) during the course of the race, much like the flying change of leads in a galloping horse. The camel gallop is slightly faster than the pace and is often used in the dash to the finish line. Wagering (betting) is not allowed in Muslim cultures, but prizes are awarded to the race winners.

Figure 1.17 Dromedary camel as a riding camel for tourists.

Figure 1.18 Dromedary camels as draft animals.

Figure 1.19 Racing dromedary camels in the United Arab Emirates.

Figure 1.20 Showing alpacas.

Figure 1.21 Packing llamas on trek (*Source:* Photo courtesy of Barb Baker).

Llamas have a long history of interaction with Andean people, supplying meat, leather, and fiber (garments, blankets, rope, costales [burlap bags]), transporting goods, and serving as sacrificial animals [17]. In North America, llamas fill numerous niches, including, but not limited to, breeding, showing (Figure 1.20), parades, companion animals, packing (Figure 1.21), driving, and fiber (cottage industry). Well-trained, quiet llamas are ideal for taking to schools and convalescent hospitals and for interacting with emotionally and physically disadvantaged children and adults. Another important niche is the use of llamas to guard sheep and goats from predation by coyotes (Figure 1.22).

Alpacas are the premier fiber-producing animals in the Altiplano region of the Andes. They are also harvested for meat in South America, and the leather is used to make ropes. The pelts of crias are used to make fine rugs and wall hangings; they are sewn together in intricate patterns and designs. In North America, alpacas serve as companion animals and are used for breeding and showing.

Figure 1.22 Llamas can be used as guard animals to protect smaller, more vulnerable species from predation.

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2

South American Camelid Behavior and the CAMELIDynamics Approach to Handling

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This chapter focuses on behavior and handling in llamas and alpacas. Chapter 23 discusses behavior and handling for camels. Veterinarians as well as camelid owners often have the assumption that patient restraint precedes any work with the patient. The fallacy with this mentality is that for animals that run as a primary defense, the scariest part of the procedure may be the restraint itself. In many situations, if the restraint is skipped, the handler will be pleasantly surprised at how cooperative many animals become. Animals known as the most difficult to treat are typically those fearful of restraint. These commonly become tractable if you do not begin with holding them still. It may seem illogical that you can skip the restraint and simply do the procedure but in fact with some handling skills and an appropriate facility you can often do exactly that.

Running is a camelid's first line of defense, and when that option is taken away, the animal will resort to avoidance behaviors such as rearing, kicking, spitting, biting, and lying down. If the animal is permitted to remain in control of his balance, given the option to move around within a small area, and kept calm, most medical procedures can be accomplished. If the working area is too large, the handler has no choice but to hold the animal still, which will lead to fear-driven behaviors. Much of what the author suggests depends on working in an area NO LARGER than a 12 × 12 (ideally smaller) box stall. With painful, lengthy procedures, it may be more practical and humane to use a sedative.

Owners often base their opinions of veterinary care not only on health outcomes but also on how the veterinarian and veterinary staff handle their animals. The thing owners remember and admire about their veterinarian is most often how well they handle the animal. The approach to handling will have an impact on the owner and the animal's behavior long after interaction with the animal.

Animals often sense when the veterinarian or owner is in a hurry, upset, angry or impatient. An animal that is anxious will become unreasonable when it is not offered an opportunity for him to process his situation. Restraining an animal by pure physical means can be difficult, dangerous, and increases stress in the animal. Offering time for the animal to process its situation can make a remarkable difference in the animal's attitude.

> I make no assertion that it is possible to make an animal's life totally anxiety or pain free. Into each life a little rain must fall. It is the sum total of the experience an animal has with humans that forms his overall impression. It is often said that animals know when you are trying to help them. I believe that to be true.
>
> – Marty McGee Bennett

Camelid Behavior

When working with any animal, being able to read and predict their behavior is vitally important. Llamas and alpacas are different and less familiar to people than dogs, cats, horses, and other barnyard animals. South American camelids also spit, which, while not injurious, is daunting for people who do not have familiarity with these animals, carrying a psychological punch that is out of all proportion to its actual impact. Many of the techniques and principles discussed in this chapter can be applied to many farm animal species, however, the focus in this chapter will be on South American Camelids; see Chapter 23 for a discussion about Camel behavior and handling.

Table 2.1 is a list of behavioral indicators that can help a veterinarian, owner, or handler assess the state of agitation and fear possessed by their camelid patients. Being familiar

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Companion website: www.wiley.com/go/niehaus/camelids

Table 2.1 Behaviors typical of South American camelids when calm and fearful.

Calm camelids	Fearful camelids
• are quiet and still • will blink regularly (A slow deliberate blink indicates an animal that was tense and is beginning to relax.) • will stand in balance on all four feet • will breathe at a normal rate (10–30 respirations per minute) • the animal's weight is evenly distributed, and his feet are square under his body (Look carefully at the animal's feet. An animal is in balance when ALL pressure is removed from the lead or around the neck and the animal does not move away.) • will eat or ruminate • will hold its tail lightly against the body • will swallow on a regular basis • carries the neck slightly forward of the shoulders • moves its ears regularly and holds them loosely • will watch the handler with interest • will walk (not run) inside the confines of the holding pen	• will lean or pull against the handler, lead rope, catch rope or the side of the chute indicating he is out of balance • the animal's weight is not evenly distributed (head up, neck angled back, back dropping down in the rear) • will lay down and get back up repeatedly • will seek to escape by leaping forward, pulling back, rearing or may try to crawl under or through an opening • will stomp their feet and/or kick. • will hold tail over the back, clamp it to the body or arch it up and over the back • will stare fixedly • will spit • will refuse food or consume food rapidly w/o chewing • will throw head around (Sometimes this is in a distinctive pattern called "orbiting" in which the camelid looks straight up and then whirls the head around in a circle. This is common in high-strung animals. You may observe this behavior in the pasture when there is no human interaction. These animals also commonly pace along a fence line and orbit at the same time.)

with this list can allow one to determine that an animal is becoming more agitated and frightened or that a frightened animal is calming down. These indicators are more useful than the precise meaning of ear position or body posture. It is useful to keep track of these indicators and work toward making the patient become calmer (Figure 2.1a and b).

The behaviors of humans in proximity will directly affect the behavior of the patient. It is necessary to overstate the importance of modeling calm behavior as you work with animals. If the handler is holding their breath, out of balance, or agitated, the animal notices, and this will cause the animal to be more anxious. Standing in a relaxed way, breathing normally (if not deeply), and standing with shoulders canted away from the animal communicates a relaxed, non-threatening disposition (Figure 2.2).

Most humans are heavy handed. It is typical for a handler to put 3–5 pounds of unconscious pressure on a lead or on the animal as he or she works. This weight is uncomfortable and frightening to the animal. The handler should be responsible for the weight of their own arms and they should think about helping the animal to remain in balance instead of holding it and forcing it to rely on the human for balance (Figures 2.3 and 2.4).

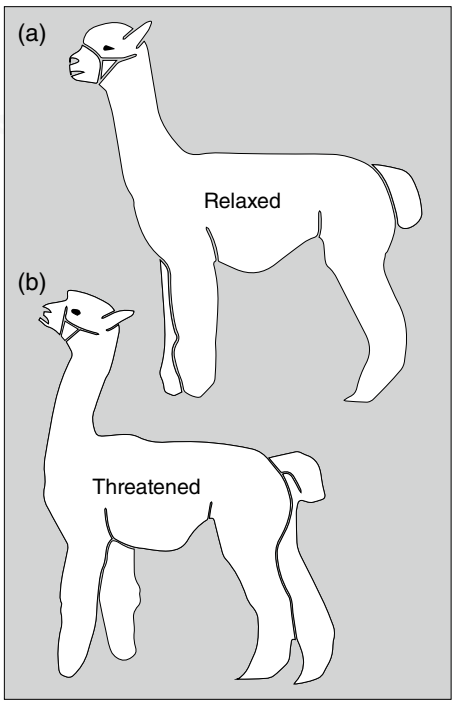

Figure 2.1 (a) Illustration of the stance of a relaxed alpaca. (b) Illustration depicting the stance of a threatened or frightened alpaca.

Containment Vs. Restraint

Frequently owners and veterinarians are skeptical that it is possible for camelids to accept many common procedures without some form of restraint; consequently, they do not give the animal a chance to try (a necessary requirement prior to acceptance). Most camelids will accept routine procedures without restraint if they are given the opportunity [1].

Figure 2.2 This handler is standing in a pen with an alpaca in a non-threatening manner. She is outside arm's length standing with her shoulders canted away from the animal. Her shoulders are relaxed.

Figure 2.3 Handler is holding steadily as the llama pulls. This technique will cause the animal to feel frightened because he cannot get away.

Figure 2.4 Neutral Rope. There is no tension in the lead rope and the llama remains in balance. This technique allows the animal to feel in control of his own body and therefore remain calm.

Restraint is the act of holding the animal against his will, creating a loss of freedom for the patient. Restraint in camelid patients usually involves tying the animal up or forcing the animal against an immovable object, eliminating the animal's ability to move. Unlike horses, camelids are often small enough to physically restrain with just the strength of an individual handler, making this form of restraint tempting. However, due to their anatomy, physical restraint on a per body weight basis is more difficult and can be dangerous for the animal and handler.

Camelids have a long neck which makes their center of mass forward on their body. Camelids carry approximately 67% of their body weight over the front legs.[2] (L. Johnson, personal communication 2020). The animals are light in the back and more muscular in the front. The long neck also makes restraining by only immobilizing the head dangerous and difficult because the long neck offers the opportunity for significant body movement even if the animal is held securely by the head. These animals are incredibly agile, and a long neck offers advantages for escape and evasion. Handlers are easily injured by an animal throwing his head around.

Llamas and alpacas are naturally head shy. These animals know instinctively how much leverage is afforded when their head is controlled and will do their best to avoid having their heads handled or restrained (Figure 2.5). However, the animals do not dislike having their heads touched; they respond well to massage and other touching techniques provided they are not restrained and feel safe with their handler. Their fear is nested in restraint and unfortunately, many humans tend to use the neck and head to hold the animals against their will.

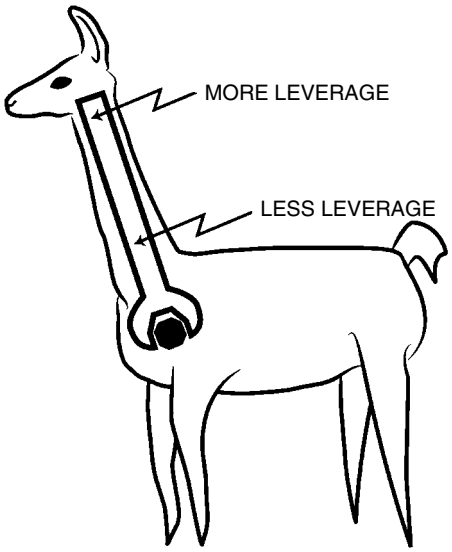

Figure 2.5 Drawing illustrating how the long neck of South American camelids allows for a great deal of leverage to be generated when a handler controls an animal by the head. Because the animals are wary of humans, they are wary of this control of the head.

It is possible to balance the head instead of restraining it for examination and treatment purposes. This technique takes practice but is a skill worth cultivating for veterinarians, owners, and alpaca handlers. By learning balancing techniques and the use of containment, a handler can achieve procedures more quickly and safely. A catch pen and containment go a long way toward making human proximity and touching less frightening.

Restraining camelids by the ears or the tail is unpleasant and results in an animal that becomes protective of these areas making him difficult later when it comes to medical handling, haltering and routine procedures like toenail trimming.

As an example, injections often begin when the animal is tied up, pushed against a wall, tied in a chute, or held still by a team of humans. The restraint is applied before the procedure has begun, and the animal becomes excited and fights the restraint prior to the procedure. The handlers react by using more restraint, and the animal fights harder. All this fighting occurs **before** the needle touches the skin, so the injection **is not** the issue. The restraint provokes the fight. From the animal's perspective, most herd management procedures pale in comparison to the ordeal of being physically subdued by humans.

Containment involves enclosing or keeping the animal within certain limits. Camelids respond more positively to containment rather than restraint. Containment is a limitation of space within which the animal is allowed full freedom to move. Camelids are less likely to panic and struggle when contained compared to when they are restrained.

The principles of containment are effective for highly strung and difficult animals. Even animals that fight violently when restrained will be able to handle the freedom provided by containment for routine procedures. The key to using containment is a good setup.[3]

Tips to Low Stress Handling of Llamas and Alpacas

1) **Safety in numbers:** Camelids need other camelids to feel safe. Even if you have only one patient, it is unwise to work with one animal alone. Pack a catch pen with five to six other animals to take up room; this slows their movement and provides a feeling of safety. For every pen, there is an ideal number of animals. Too few animals, and it doesn't work as well (you can make the pen smaller or add more animals), too many animals and they will become agitated. Ideally, there should be

Figure 2.6 This photo shows a nicely packed pen. The animals have room to move but not enough to move too quickly. Handling South American camelids in packed pens makes handling without restraint easier because the animals feel safer in groups.

about 20–25% free space in a stall (Figure 2.6). If the animals are smaller, more will be required; if the animals are larger or in full fleece fewer animals will be required. This technique works with both llamas and alpacas. The animals kick less and move less, which makes handling easier. If one animal spits or is very volatile, work with that animal first and release him from the pen. This is a great arrangement for physical exams, giving injections, or administering oral dewormer. Even if you work in a chute or mini-catch pen (described below), the presence of other animals can make the animals feel much safer and less likely to panic. When working with mothers, keep the baby with her if possible. She will stay calmer if she can be with her baby.

2) **Think like a camelid:** Let the animal have a chance to think. Wait a bit when trying to drive an animal into a holding pen, or loading an animal onto a trailer, usually, it only takes one animal that will move or load; the others are likely to follow. Camelids are very resistant to being split up. If you are letting each animal go after a procedure, the last one will panic when left alone, and it will be difficult to control if the rest of the herd has wandered back to the field. It is better to keep a few or even the whole herd nearby in a holding pen until the whole group is finished. Do not create anxiety in the process of getting the animals to the catch pen or other container. Laneways leading to a small paddock adjacent to the handling area keeps things calm. Even untrained animals can be herded into a trailer if kept calm and done as a group (remember they are worked more easily as a group) (Figure 2.7).

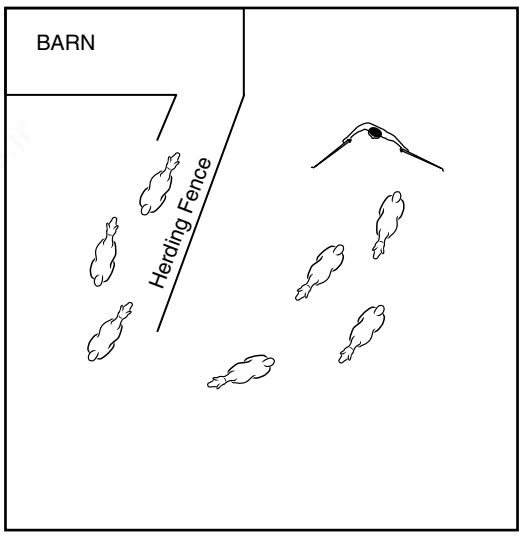

Figure 2.7 Drawing illustrating a setup for corralling animals calmly into a handling area. Light weight or temporary fence is adequate for herding provided the animals have a place to go and it is not being used to trap animals.

3) **Halter fit is important:** If you are using a halter, it is vital that it fits properly. There is NOTHING more important than this! The dynamics of halter fit are discussed in detail later. It is IMPERATIVE that the halter fits well up on the nasal bone and is adjusted so that it will not slip down. Halters used for restraint that slip rostrally ending up on cartilage will compromise the airway causing panic and potentially respiratory distress. An experience like this can result in an extraordinarily difficult animal to handle for the rest of its life.

4) **Consider the handling facilities:** The author suggests beginning with a catch pen and altering the situation from there. Camelids are wiry but they do not require facilities as robust as cows and horses. Lightweight panels are sufficient, but they should be attached to a barn wall or a post driven into the ground if they are to be safe for handling.
   Your handling area should be well-lit and located in a well-ventilated area. Animals do not like to enter blind alleys or find themselves in dark corners.
   First impressions are especially important. Animals will be much more cooperative and think better of your handling area if they can experience it first without a procedure. If you ask the owner to feed the animals in the catch pen or handling area a few times before you arrive, the animals will be more relaxed and more cooperative.
5) **Avoid the spit without muzzling:** Covering the mouth, nose, or head is a common handling technique to protect the handler from spit, however, there are negative implications to this technique. Confined animals act out in part because they feel closed in without an escape route. Under these circumstances, restricting the airway is counterproductive. The cartilage at the end of the nose is very delicate and can be compressed easily even by a light towel or rag. Camelids are semi-obligate nasal breathers, and if the nose is partially occluded, this may compromise airflow and cause an animal to panic. This may even backfire, as the chance of aspiration of stomach contents is increased when the mouth is covered. Coveralls, safety glasses, directing the head in a different direction, and the occasional shower after the procedure may be necessary in the case of a spitting camelid.
6) **Avoid blindfolding:** Some veterinarians have described having some success with covering the eyes, and there is a mask sold for this purpose. The author has found masking or blindfolding to be undesirable and unnecessary. The act of blindfolding an animal often creates more drama than the procedure itself, and it can detrimentally affect future haltering or handling. Often techniques work the first time because the animal is surprised and does not know how to react. The second or third time the animal will recognize the antecedent and will develop behaviors that are more problematic than the original behavior that prompted use of the blindfold.

Options for Containment

1) **A secure catch pen.** Square is best and size can range from 8 × 8 to 10 × 10 ft; a larger area is too big. Stacking bales of hay two feet high inside a 12 × 12 box stall will transform it into a suitable working area for camelids (Figure 2.8).
2) **A mini- or half-catch pen.** This is a space roughly half the size of a catch pen (4 × 8 to 5 × 10 ft). It can be

Figure 2.8 An ideal catch pen. This pen is 8 × 8 ft. square. If the area is much larger, the animals will have too much freedom and will be more difficult to control.

Figure 2.9 "Mini" or "half" catch pen. If the animals are small or you don't have many, this is a great solution for making the pen more crowded.

constructed of plywood or, if you already have a catch pen, by moving one panel closer to the other and securing it with baling twine or rope; heavy duty zip ties are also effective. You can experiment with the idea by piling bales of hay inside your catch pen until only a mini-catch pen remains. A mini-catch pen is a useful tool for handling untrained crias and weanlings. Because of their smaller size, a standard catch pen is too large for these animals. A mini-catch pen can take the place of a standard catch pen for herd management tasks such as injections and oral deworming (Figure 2.9).

3) **A livestock trailer.** This is the author's favorite place to work. Most trailers have a top on them, and this discourages rearing, which is one of the more problematic forms of evasion. Longer livestock trailers are more useful if they have a divider. Animals that arrive at a vet clinic in a trailer should be treated in the trailer if possible. On the farm, the trailer can be parked adjacent to or in the barn. Animals that are not halter trained can be herded into the trailer and treated there.
4) **Camelidynamics Handling Facility (**CHF**).** This is the Cadillac of handling systems and combines a variety of handling areas in which the animal can be moved from one area to another depending on the task and the animal. Incorporating your trailer into a CHF offers all the options described thus far into one facility and provides more value when it comes to the investment of a trailer (Figure 2.10).
5) **A livestock chute.** A chute can be a useful piece of equipment for many situations; however, the author finds it more useful to view a chute as an intermediate step rather than a permanent solution for handling. Animals can "graduate" from the chute to the mini-catch pen and finally to a catch pen. Chemical sedation should be considered if the animal must be completely immobilized or if the procedure is longer than a few minutes. Even though they are typically called restraint chutes, many models can be modified successfully to serve as containers rather than actual restraint devices. The author has worked effectively in chutes that had solid sides and chutes that had a single rail at chest height. The author advises against the kind of chute that has a single chin support in the front designed to be used with a halter and to immobilize the head. To be safe, these head-restraint devices must be used with belly and back bands turning them into "camelid straight-jackets." Without total restraint of the body, the risk of neck, head and leg injuries is great.

Chutes with upright shoulder supports can be humane, safe, and useful for certain procedures that involve the head and neck. Shoulder supports limit the movement in the front half of the body without restraining the head (Figure 2.11).

To be effective for containment rather than restraint, a chute *must* have a rear barrier. The animal is restricted to the chute by virtue of the rear barrier in

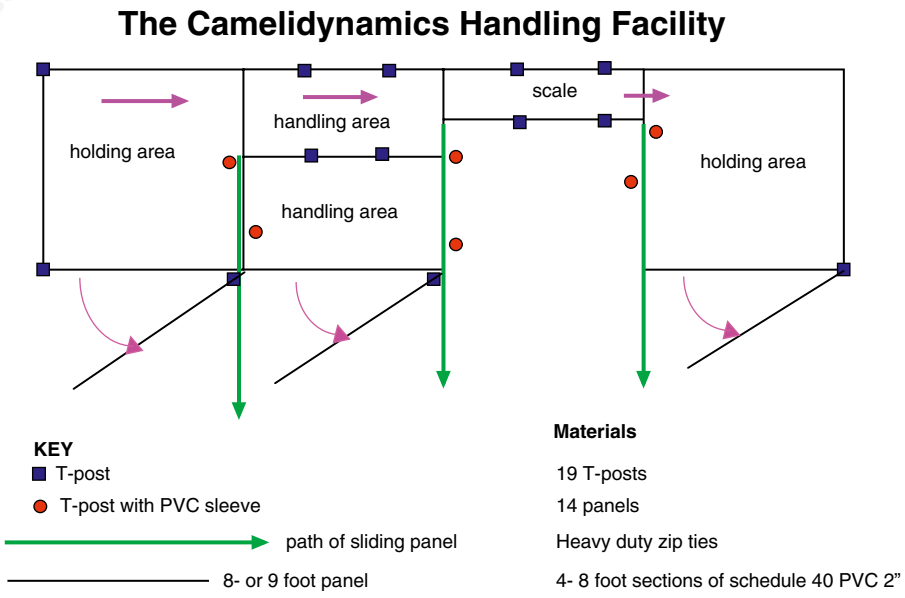

Figure 2.10 Schematic of the Camelidynamics handling facility. This system combines a variety of handling areas depending on the task at hand. The animals can be moved from one size area to another by sliding panels or opening gates.

Figure 2.11 Llama contained in a chute. This chute has solid sides and a bar at the rear of the animal to prevent it from backing up and therefore does not require tying the animal by the head.

the back and the gate or shoulder supports in the front; therefore, the animal does not need to be tied. Camelids are much less likely to panic if restricted to the chute from the back than if tied by the head. In addition, these chutes can be used to handle animals who are not halter trained. The worst introduction to a halter is to have it put on the first time to be restrained in a chute. Adding a rear barrier is a modification that the author considers essential for all chutes. When using a rear barrier, it is not necessary to tie the llama or alpaca in the chute. A handler can stand at the front of the chute, offer food, and prevent the animal from turning with signals on the lead. When working alone, the animal can be tied with enough slack in the rope so it can back up and feel the rear barrier. In this way, the rear barrier stops the animal before he runs out of rope and panics.

A rear barrier is the key to using any type of chute for containment rather than restraint.

The Midline Catch, bracelet and Handler Helper

Working with camelids is often a team effort. It is important that both team members are working together and using the principles of balance instead of restraint. Working

in a small container as mentioned before is crucial, particularly if your assistant is not experienced with camelids. If the animal is not haltered, you can still use the head for balance by using a technique the author calls "the bracelet" [4]. The first order of business is to approach and catch the animal without causing a fight before the medical issue is even attempted. The author finds when animals are confined in a small area you do not need to trap the animal in a corner to get the animal under control. She uses an approach technique she calls the "midline catch" [4]. She approaches from behind the eye and moves the animal using her body position so that the animal is parallel to a panel and moving around the pen in a counterclockwise direction. Most animals are accustomed to being approached from their left side. She then approaches from behind the animal and lays her right hand on the midline of the neck about halfway between the head and back. Figures 2.12–2.14 are the midline catch and bracelet [4]. She does NOT put her hand on the off side of the neck but uses the V formed by her thumb and fingers on the midline of the neck. She uses her hand and body position to keep the animal moving in the counterclockwise direction and will allow the animal to walk along for a step or two as she walks with him before using her left hand on the front of the neck just behind the jaw but NOT on the head. Next, she slides her right hand up behind the ears and finally sliding her left hand up on the jaw until the webbing between her thumb and fingers is seated just behind the bottom lip. Her hands are now in the bracelet. Using the bracelet, she has full control over the animal's head as if she had a halter on him. The author can balance and steer the animal as the veterinarian works (Figures 2.12–2.14).

The handler helper is a useful tool for procedures that take several minutes such as trimming toenails or complex procedures involving multiple steps [4]. The handler helper is a short length of nylon with a hook and double-ring system that allows the handler to gain leverage over the head without having to continually hold the animals or use a halter. The handler helper can also be left on temporarily while an animal is in a clinical setting so that people who are not adept at catching camelids can use it to easily catch the animal. Camelids are often frightened by the close proximity of humans and even more so when multiple people are around; a handler helper can allow the animal to distance from them and then it can be used to easily recatch the animal when needed.

To place the handler helper, the handler allows the animal to rest its head in their left hand. The handler can then take the handler helper out of their pocket (a pocket on the right side works best), slip it over the camelid's head, and snap it around the top of the neck. It is quite safe to let go of this tool as it cannot tighten and is not long enough to be dangerous. It is perfectly safe to let the animal go within the catch pen to offer a break (Figures 2.15 and 2.16).

Figure 2.12 Demonstration of catching an alpaca with a "midline catch." The first step is placing the right hand on the midline of the neck at the base of the skull. The animal will likely move forward into the escape route inside the pen.

Figure 2.13 Demonstration of control of an alpaca with a "midline catch." The second step of the technique involves placing the flat palm of the left hand in front of the neck. The animal is free to move. The handler stays with the animal as he walks. Once the animal stops, the hand moves into the bracelet position making it possible to balance the head and neck in line with the body.

(a)

(b)

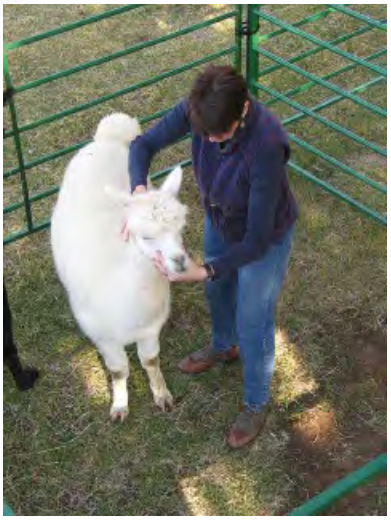

Figure 2.14 Demonstration of head control with the "bracelet" technique. The webbing between the thumb and forefinger is the active part of both hands. This is a balancing technique not a holding technique; the animal is allowed to move.

The Neck Wrap – A Thunder Shirt for Camelids

A neck wrap can be used to decrease anxiety, for training, and for routine medical procedures. Veterinarians can even use a neck wrap for drawing blood from the jugular vein if the area for the vena puncture is left uncovered.

To effectively apply a neck wrap, an elastic wrap or a long stretchy ace bandage is wrapped around the neck beginning at the base of the neck, wrapped in a crisscross fashion, ending at a point just below the ears, and fastened. Begin at the base of the neck by rolling the two ends of the neck wrap toward each other and crossing as you go, meeting halfway. You will need to work in a small space and

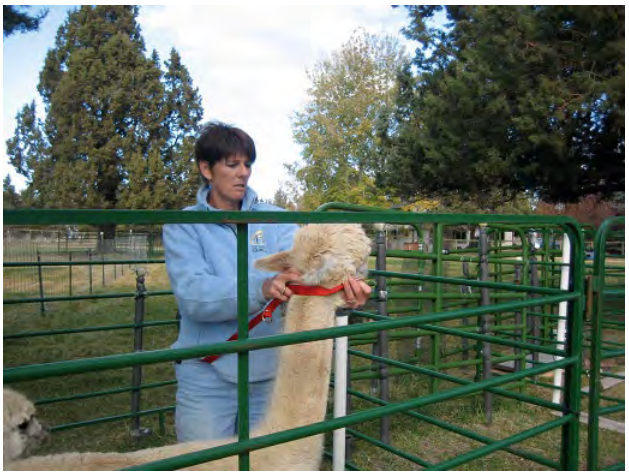

Figure 2.15 Demonstration of application of the handler helper to an alpaca to aid in controlling the animal. The tool makes it easy to balance the animal while offering some distance from the handler. You can also leave the handler helper on while the animal is in a pen making it easier to re-catch the animal later.

Figure 2.16 Alpaca with the handler helper in place.

allow the animal to move as you put the wrap on. Ideally, enlist the help of a second person to help steady the animal as you work. Finish by attaching the wrap to itself. Buckles that avoid use of Velcro are recommended to secure the wrap, as the sound created by the latter can be frightening to animals. The number of wraps is less important than making sure that the snug wrap is felt the length of the neck. Following the procedure, the wrap is removed by unhooking it and unwinding it from the neck. Do be careful to keep the ends in your hands as you unwrap it so that it does not dangle around the legs and frighten the animal.

Keeping an animal in balance is key to helping them remain calm, and handlers often use the head as our point of connection to balance the animal. The length and

Figure 2.17 Application of a neck wrap to an alpaca to help keep the animal calm.

flexibility of the neck allows the animal to move its body freely even when restrained by the head. It is theorized that a snug neck wrap creates a heightened physical connection between the head and the rest of the body, or it might act as a distraction to the animal. Sometimes, the neck wrap does not have any effect on the animal, but in the author's experience, working with many difficult animals, she has experienced amazing transformations using the neck wrap. To be most effective, the neck wrap should be put on before working with the animal. The neck wrap should be applied inside a confined area (Figures 2.17 and 2.18).

Trimming Toenails

Trimming toenails should be done routinely, and may be performed by the owner, shearer, or veterinarian. Properly trimmed toenails are important for the health of the animal and esthetics. Likely, a major reason that this procedure is challenging is that the animals are not taught to accept having their legs handled *before* attempts are made to pick up their feet. In other words, we must not ask an animal to pick up his feet before we teach him to pick up his feet.

Camelids frequently exhibit adverse behavior during toenail trimming. The reason for this is unknown but it may be because they use their legs as their primary means of defense (running from dangerous situations), and they are instinctively protecting their major defense mechanism. It is also important to note that male camelids frequently bite the legs of other males during fighting, and females to encourage them to cush for breeding. In addition to these factors, there is an inherent problem in how humans handle their feet and legs, inciting fear in these animals.

Figure 2.18 Alpaca with neck wrap in place.

The actual trimming of toenails does not require much time, or any specialized tools and it does not have to hurt. It does not require any particular skill or strength to snip off the extra growth of a toenail, particularly when animals have been standing on damp or wet grass. "Nibbling" rather than cutting big chunks is recommended to avoid quicking the animal, which is painful. If small "nibbles" are removed at a time, any mistake will be a small one. The quick grows along with the toenails so you cannot assume that you can take big hunks off even if the animal has exceptionally long toenails.

Holding the leg and foot long enough to trim toenails is commonly the most challenging part of the toenail trimming procedure. Llamas and alpacas learn quickly how effective it is to simply lay down and hide their feet very tightly under their body or to fight so hard that it becomes impossible to get near the legs or feet.

Veterinarians do not have the luxury of multiple sessions with the animal to teach him to accept toenail trimming and most owners with a herd of animals will not take the time to completely desensitize all of their animals. Fortunately, there are things you can do that will help make the process easier for you when you trim and avoid making the process traumatic for the animal thereby making the next round of trimming harder.

Sedation is highly recommended for difficult animals. Camelids hate restraint, and a fight over toenails will not be quickly forgotten and may affect how the animal feels about other handling tasks. Escape and evasion techniques learned in the process of having its toenails trimmed can be employed to thwart haltering and other handling chores at a later date. The author has spoken with many veterinarians who were able to successfully wean an animal off sedation. What does not work is hoping that repeated horrible experiences will somehow result in cooperation.

Success with toenails is best achieved by using the principles of balance and a no-restraint approach to catching (see the midline catch, handler helper, and containment above). Animals do not feel safe if they are not in balance, falling down is the WORST outcome. Most toenail fights are a result of humans making the animal feel vulnerable and out of balance. More detail on these techniques follows. The author's personal favorite for trimming toenails is to work inside a trailer, but any small sturdy space will work better than tying. The most crucial element of the setup is to create a way of limiting movement without tying the animal tightly or using physical restraint. Trailers are small, sturdy and have a top, which limits the animals' inclination to jump. A neck wrap can be helpful, but success is mostly about BALANCE! A camelid's legs are their escape equipment and as such are crucial to their survival. Working in a way that makes a camelid feel as if they might fall is going to cause panic and the animal will do everything he can to escape. To help the animal feel safe and help him maintain control of his own balance always work so that the animal is standing next to a panel and NEVER between two humans. If you have a helper, his job is to act as a "balance spotter" – that means to help the animal keep his own balance and NOT to be dependent on the human. The handler at the head will make small corrections using the head/neck to help the animal keep his own balance. The toenail trimmer will keep the foot under the body in a natural position and will pay attention to signals that the animal needs a break and allow it to have its foot back. A camelid in balance is standing so that its head is lined up with its neck, its neck is in line with its body, and the body is held naturally over the feet, similarly to the way a judge wants to see an animal stand in the show ring. If your helper is not actively helping the animal keep its balance, chances are he will be inadvertently causing the animal to be off balance. A light support using the bracelet described previously is a great technique and can be combined with the handler helper at the top of the neck, combined with a flat hand under the jaw as a support, to offer more distance and the animal will not feel as threatened. Another way to help ensure you are not disrupting an animal's balance is to use a holster strapped to your lower leg (Figure 2.19).

Figure 2.19 A holster on the lower leg, can be useful when trimming feet to allow retrieval of the trimers while maintaining the animal's balance.

With a holster on your leg, you can use one hand on the top line to shift the animal's balance as you lift the leg with the other hand. In this way, you can help the animal feel safe and in balance as you work with its feet. The animal's weight should remain on the leg until you ask the animal to lift the foot. Another balancing technique for toenail trimming is to lift the foot off the ground from a point higher on the body closer to the center of mass (above the knee in the front and above the hock in the back). This affords the handler less leverage, making the animal feel safer and more balanced.

Another technique in training a camelid to allow a handler to work with its feet is allowing it to put its foot back down immediately after the animal lifts his foot. Though this sounds counterintuitive, this process should be repeated 5–10 times (up and down, up and down, up and down . . .). This teaches the animal to expect that if he wants his foot back, he will get it back. If, during the procedure, the animal wants to have his leg back, GIVE IT TO HIM! The biggest mistake to make is to hold on to a leg for even a split second as the animal struggles. The animal should be permitted to have its foot back, you can simply pick it up again (Figures 2.20–2.27).

Once the foot is off the ground, the animal is stable and in balance, you can transfer your hands from higher up on the body, down the leg in order to hold the foot. If you are in balance and the animal is in balance, the method by which one uses to get their hand into trimming position is personal preference. The author's approach is as follows: To trim the left front foot, she stands facing the rear with her left hand on the top line and her right hand above the knee. With her left hand she adjusts the animal's weight to the right foot and asks the animal to lift the left foot with her right hand. Once the animal is in balance and is not leaning on her, she

Figure 2.20 Preparing to pick up the left front foot. The handler is using her left hand on the midline to keep the weight on the foot until asking for the animal to shift his weight and allow the foot to be lifted. The handler contacts the leg above the knee with the right hand which is less threatening.

Figure 2.21 The handler has now moved the left hand from the midline to the canon bone to support the foot for trimming.

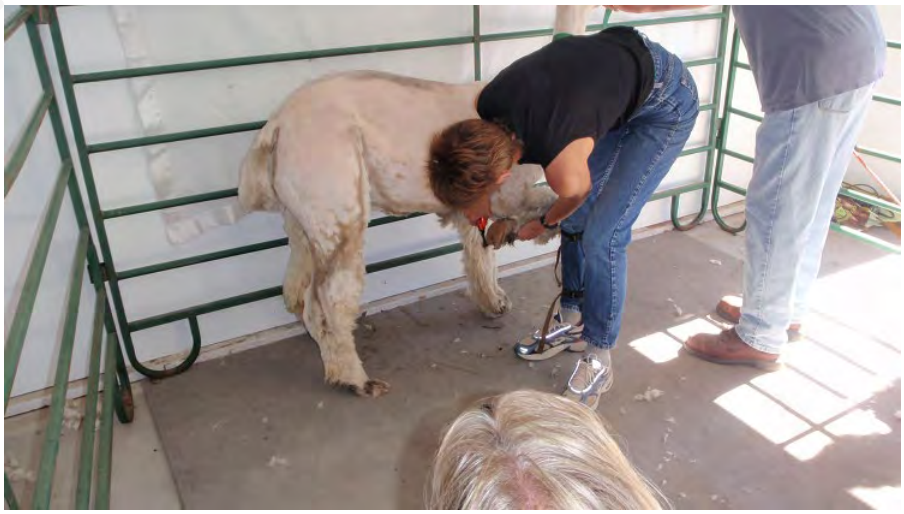

Figure 2.22 On the opposite side, the hands are switched so that the handler can use the dominant hand for the trimmers. A second handler is balancing this alpaca and the animal is next to a wall which makes trimming feel safer for the animal.

Figure 2.23 The handler is managing the animal's balance using the right hand on the topline keeping the weight on the leg until the right hand is in place just in front and above the hock.

takes her left hand off the top line and uses it to support the cannon bone. Her right hand is now free, and she uses it to get her nippers out of the holster on her lower right leg and trims the toenails with her right hand. Trimming the left rear is essentially the same except her right hand is originally placed on the front of the rear leg above the hock. Since she is right-handed when trimming the right front and right rear, she follows the same process (switching right and left in the above directions) until she gets the foot in her hand, then swaps hands so that she is operating the trimmers with her stronger right hand. Trimming with the foot as close to the ground as possible; lifting the foot only a few inches still gives you room to work but feels much safer for the animal. Keep the leg under the body for balance; do not hold the foot out to the side (note the trade-off here is less visualization for increased balance of the animal). A common pitfall is that many people will squeeze the nippers with one hand and simultaneously squeeze the foot with the supporting hand; it is important to avoid doing this because the animal will react to having his foot squeezed and struggle to get his foot away from the handler.

Severely overgrown toenails, particularly those that curl out to the side, can be trimmed while the animal is standing on his feet. Many animals are not as concerned with the trimming as they are with having their legs handled. You can slide the nippers under the toenail and "nibble" the long bits away. Most animals are unconcerned about this procedure if their legs are not touched (Figure 2.28).

The balancing technique described earlier, and a hand placed on the animal's top line can be used to bring the weight onto the foot being trimmed. If the animal is bearing weight on the foot, it cannot lift it. Trimming toenails this way is a compromise but is preferred to making the animal averse to the trimming process with a fight. The author has used a rotary (Dremmel) tool on toenails, both with the foot on the ground, and with the foot elevated. It can be an effective way to trim without the risk of quicking the toenail. Box 2.1 lists some tips that will facilitate toenail trimming.

Figure 2.24 The handler uses the left hand on the back to ask the llama to shift his weight to the right at the same time asking this llama to lift his left foot.

Figure 2.25 The handler now moves the left hand off the back and uses it to support the foot freeing up the right hand for trimming.

Figure 2.26 Trimming the toenail is best done by trimming small pieces of the nail. Care should be taken to avoid quicking the nail. Longer nails will have to be trimmed incrementally. Ideally, the nails will be even with the pad of the foot.

Handling for Veterinary Procedures

Medical procedures can be distressing and dangerous for the owner, the animal, and the veterinarian. The techniques described below make things easier for all parties. The author does not advocate any technique that sacrifices the safety of handlers or veterinarians.

(a)

(b)

Figure 2.27 Trimming nails inside a trailer is much easier with difficult animals and much less threatening than using a chute.

Figure 2.28 Photo demonstrating trimming of an alpaca's toenails while it is standing on the ground.

No-Restraint Injections

When you put something sharp into an animal, it is likely that it is going to move. With this assumption, most people restrain the animal before they inject. The fallacy with this approach is the restraint causes the animal to fight before the needle touches the skin.

Given a camelid's long neck, it is exceedingly difficult to eliminate bodily movement by tying the head, even when the animal is inside a chute. Complete and total restraint takes a lot of time and can be dangerous to do before an injection is given. Containing the animal instead of restraining it and learning to stand in a way that produces predictable movement eliminates the need for restraint (Box 2.2).

The easiest way to give injections is in a catch pen or in a trailer. If you are doing herd health, pack the pen as described earlier (the author prefers to do herd health work in groups of 8–10). Put as many animals as will comfortably fit inside the catch pen. The animals will appear crowded; leaving approximately 20% of empty space in the pen. Eight

Box 2.1 Tips for trimming camelids toenails

- Use a holster that fits on the lower leg. This allows you to retrieve your nippers without moving very much. A lot of movement attempting to get nippers out of a pocket will likely take the animal out of balance.
- Trim very conservatively at first – we all quick an animal now and again but you don't want it to be the first time the animal is trimmed.
- Allow the llama or alpaca to put his foot down a few times during the trimming process if he needs to. The more practice he gets picking up his foot and getting it back the better off you both are.
- Trim toenails after a rain. The toenails are much easier when they are moist. If you live in a dry climate, wet down a small area and keep the animals in this area for a few minutes before trimming.
- Don't be reluctant to use a sedative for really difficult animals if you need to. Wrestling to trim toenails makes the process more traumatic and only teaches the animal to resist more.

Box 2.2 Advantages of the No-restraint Method of Administering Injections

- There is less movement
- The movement is less violent, less erratic and more predictable
- The animal stays calm
- The muscles are not tight
- There is less likelihood of abscess
- It is faster and easier
- One can work alone, saving time, labor and makes the patient feel safer

to ten alpacas or 5–6 llamas in a 9 × 9-ft pen is ideal. For situations where there are not enough animals to fill the pen, the pen can be made smaller with bales of hay, or the panels adjusted to make a smaller area.

Crowding the animals slows them down and they feel safer in a group. If you are giving injections to one animal, it is still useful to pack the pen with others; the patient will be much more cooperative with company and the other animals will help facilitate the injection process. The animals in a group pen are less reactive and do not kick or lie down as much. If there is an animal that is known to spit readily, the author recommends treating that animal first and then allowing it to leave.

There are several options for injection sites (Figure 2.29). Subcutaneous injections can be given just ahead of the shoulder blade about halfway down the body by reaching over the body to inject on the opposite side. In this way, the animal moves toward the person giving the injection when the needle enters the skin, and it is easier to keep the needle in the animal without the need for restraint. It is convenient to give intramuscular (IM) injections in the triceps muscle using this method as well. The triceps muscle is a large muscle and accommodating for IM injections. This muscle mass will accommodate up to 5 cc of medication for alpacas and slightly more for llamas (personal communication with Dr. David Anderson). Injections in the rear end are difficult to do without restraint and are difficult to inspect later or treat if there is a problem with the injection site.

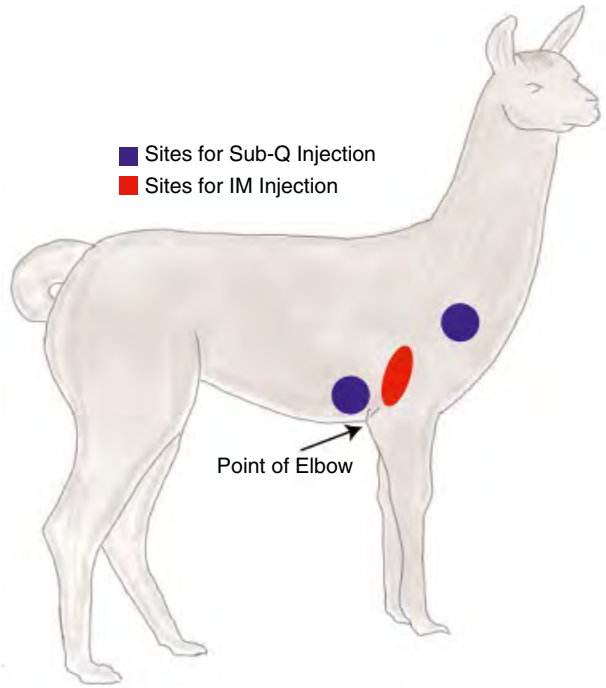

Figure 2.29 Illustration of proper location for giving a subcutaneous and intramuscular injection.

Techniques for No Restraint Injections

Working Alone

Decide which animal is to receive the injection; fixate on that animal, and do not change target animals throughout this process (Figures 2.30–2.33). Hold the syringe in your dominant hand. Approach the animal from behind its line of sight when the animal is parallel to a panel. Make sure to stay behind its line of sight, position yourself on the side of the animal toward the center of the pen with the animal tracking around the outside next to the panels. The handler puts their left hand firmly on the opposite side of the animal just over the top line in the area where the neck and shoulder come together; this is the injection site. Note the injection site is on the side opposite from where the person is standing. Most importantly, reach forward and stay behind the animal's line of sight. At the same time, keep your hip as close to the animal's hip as possible. The animal changes direction by pivoting around the shoulders and using this technique, the animal can be prevented from changing direction. The animal has a forward escape route and will use it. It is not possible to give injections without restraint unless you can prevent the animal from changing direction. Working in a packed pen helps with this immensely. Though it may seem daunting to work in a group of animals within a pen, in the author's experience the animals are much less fearful, which means they kick, rear, and spit less.

Working with an Assistant

If the person giving the injection is having trouble managing the animal's direction by using their body position and hip, they can have a helper use the bracelet technique or the handler helper to assist with this by steering the animal into the forward escape route (Figures 2.34 and 2.35) It is especially important to impress upon the helper that his or her job is only to steer and NOT to attempt to slow the animal down or hold it still. If your helper puts pressure on the handler helper or attempts to hold or stop the animal, the animal will begin to fight and move erratically, often rearing, making the process of injecting much more difficult, resulting in the need for restraint.

For subcutaneous injections, use the fiber to tent the skin and inject into the subcutaneous space as the animal takes a few steps. It is not necessary to visualize the tent; it can be felt. Push the needle under the skin, being careful not to insert the needle completely through the skin tent and depress the plunger. In the case of a shorn animal, it is easy to pick up a big fold of skin and the skin tent can be easily visualized. For IM injection, the large triceps muscle just dorsal to the elbow can be palpated on the opposite side of

Figure 2.30 Photo illustrating working in a packed pen. One handler is balancing and/or steering the animal using the bracelet, second hander administers a subQ injection. The animal is never restrained but can move.

Figure 2.31 The handler is giving an injection solo without restraint using a panel and a knee to keep the animal contained but not restrained.

the animal, and the needle can be directed into this large muscle mass.

For IM injections, lean over the animal and use the triceps muscle (Figure 2.36). In the case of an IM shot, the needle goes straight into the muscle and there is no need to tent the skin. The motion should be smooth and quick; there is no need to tap on the skin or use an abrupt movement. If the animal walks, simply walk with it, and the person should use their body position and hip to keep the animal tracking around the pen as you maintain your position behind the line of site.

Giving injections on the opposite side of the animal is critical to the success of this technique. The animal will naturally move away from the needle. By using this technique, the animal will move toward the person giving the injection while moving away from the needle. This makes it easy to stay with the animal and keep the needle under the skin as the injection is administered. For a right-handed person, an approach from the animal's left, with the injection given on the right is preferred by the author, but this technique can be used from either side. Box 2.3 lists tips for giving intramuscular and subcutaneous injections.

Injections for Babies and Weanlings

Even handlers who embrace the idea of no-restraint injections for adults, may still be tempted to pick up babies (if assistance is available) or sit on them (if no assistance is

Figure 2.32 This photo illustrates keeping the alpaca near and parallel to the panel. The presence of other animals both slows the progress of the animal being injected and adds to the sense of security.

Figure 2.33 Injections can be given while multiple animals are contained in a round pen.

available). These techniques only teach resistance and fear, and eventually they stop working. At three or four months of age, a flailing young camelid can cause injury to itself and the humans trying to restrain it.

Crias can stand on their own four feet in balance and accept injections like adults. The main advantage of the no-restraint technique is that it does not require any level of training for the animal. Halters, lead ropes, tying, and standing still are not requirements. If your herd management protocol calls for injections in the first few days of life, you can give the injections just as the author has described. Always keep a new baby with its mother; a catch pen is great for this. As crias get older, you can work them with the herd.

Drawing Blood

Blood can be taken from the neck, ear, tail, or front leg. In the author's experience, the easiest location to draw blood without restraint is from the jugular vein. Always work inside a catch pen or a trailer. To assist the person drawing blood, stand on the left side of the animal, with the person drawing blood in the front and a bit to the right. Standing on the left side of the animal, the author prefers to put one hand on the handler helper (high and snug on the neck) and the other hand under the jaw. When a halter is used, there is a tendency to rely on it to hold rather than balance the head. Balance the animal and turn the head to the left, which helps the person drawing the blood to find the vein.

Figure 2.34 In this photo, a helper is balancing the animal for an IM injection.

Figure 2.35 This photo illustrates using a handler helper, along with a balancing hand under the jaw, as the alpaca receives a subQ injection. Notice that the handler injecting the medication is using the wool to lift the skin away from the body, distracting the animal from the entry of the needle.

Ask the person drawing blood to let the handler know when he or she is going to push the needle in. When the animal reacts, try to rebalance it but do not attempt to hold it down or still. It is important for both parties to remember to breathe.

If the animal rears up, let him, and do not try to hold him down; this only turns a startle reaction into a panic

Figure 2.36 An intramuscular injection into the triceps muscle is done easily by reaching over the body when the needle enters. If the animal moves, he will move toward the person and away from the needle making it easier to stay with the animal without using restraint.

reaction (Figure 2.37). The skin of the neck is very thick and once the needle is in, the person drawing blood can let go of the needle; it will not fall out. The person drawing blood can wait until the animal is steadied. Once the animal is standing quietly again, the person drawing blood can continue, adjust the needle placement if necessary and draw the blood. If this approach is undesirable, a chute can be used as described earlier. Shoulder neck supports and a rear barrier make drawing blood easier. Make sure that the butt board is adjusted so that the animal has minimal front-to-back movement. The animal should be up against the

Box 2.3 Tips for Giving Injections

- Use an appropriate needle for the job. Thicker medications require a larger needle.
- For IM injections, once the needle is in the muscle, push the medication slowly. It is more comfortable for the animal and is less likely to tear the muscle fibers.
- Changing needles between drawing up the drug and injecting ensures that a sharp needle is puncturing the skin and that there is no irritating drug on the outside of the needle which may prompt a premature response from the animal (especially important if injecting irritating medication). (Personal communication with Dr. LaRue Johnson).
- If multiple injections are to be given, always give a stinging one last.
- Breathe, and breathe evenly! There is a tendency to hold one's breath just prior to putting the needle into the skin. From the animal's point of view, this is a signal to run.
- Make a practice of beginning a procedure with the least amount of containment. You can always move to the chute if you need to. You may find that you are able to offer more freedom each time. If your time is limited, and you are concerned that this method will take too long, work with a few animals in this way during each herd management session.
- Remember in all cases the panel is your friend. The more you can maneuver the animal so that he is standing parallel to a panel the better the process proceeds.

Figure 2.37 This animal is held forward in the shoulder supports by virtue of a rear barrier and does not need to be tied by the head. The jugular vein is much easier to use when the animal is not tied by the head.

shoulder supports, and do not tie the animal in the chute. To assist, stand on the left side of the chute to steady and turn the head to the left following the same procedure as outlined above. Food can be offered as encouragement and reward to make the animal feel less anxious.

Dr. David Anderson has developed the "Buckeye Blood Draw" (Figure 2.38). This is a method that allows a single person to draw blood from the jugular with no restraint.

Rectal Palpation and Transrectal Ultrasound

These procedures do not require restraint, but they do require containment. A trailer, mini-catch pen or a chute as described is more than enough in most cases. The author finds it useful to walk the animal into the chute and restrict them to the chute from the back with the butt board, even though it will have to be removed to do the exam. There is normally a bit of preparation required for a rectal exam and the animal will be more likely to stand quietly for preparation with a rear barrier in place. After the animal has been prepped, remove the butt board. Once the rectal exam begins, it is unlikely the animal will attempt to back up!

The practice of tying a female's head high for rectal exams is not recommended. When the head is tied high, the back drops. This posture causes the animal to tighten the whole body including the anal sphincter, making the procedure less comfortable (Figure 2.39).

Also, holding the tail directly over the back to get it out of the way or as a means of restraint hurts; moving the tail gently to the side is more comfortable. The tail is another location that offers a potential attacker considerable leverage and is, therefore, high on the list of places for the llama or alpaca to protect. Therefore, a rectal exam is both uncomfortable and threatening. The underside of a

Figure 2.38 This is a brilliant technique from a handling perspective. It allows for one person to draw blood; once the head is down the leverage belongs to the handler. Getting the head down is best done by incrementally moving the head down not by pushing steadily.

Figure 2.39 This photo illustrates performing a rectal ultrasound using balancing techniques and panels for containment.

camelid's tail is bare skin. The skin-to-skin contact that occurs when a person slides their hand under the tail can be shocking. It is preferable to slide the wool from the top of the tail head to act as a buffer between a person's skin and that of the patient.

Relaxing the tail before the exam helps both the animal and the examiner (Figure 2.40). One should stand just to one side facing the front of the animal and slide his or her hand under the tail close to the body. The other hand should rest on the top of the hips. The animal's tail may be

Figure 2.40 Llamas and alpacas often do not object to being touched even around the tail when they are not restrained. Working with an animal's tail decreases tension in the hind quarters and assists with reactivity to ultrasounds, helps to decrease kicking and makes toenail trimming easier.

clamped firmly against the body; this is normal but is a sign that the whole body is tense (this animal is likely holding her breath). The tail can be relaxed by rotating in a circle from the base. The tail should be pushed toward the body and rotated around the tail-head. The tail will begin relaxing. Next, gentle traction should be placed on the tail. The person doing this technique should inhale as traction is applied and exhale as tension is released. Usually, the animal will take a deep breath during this procedure. The tail can also be relaxed by gently moving the small tailbones back and forth from the tail head to the end of the tail. The tail work can be done as preparation for a rectal exam or it can be done while the animal is standing freely in a catch pen with or without a helper. The tail should always be worked when the animal is standing in balance. It is important to remember to work in line with the animal's body; never twist the tail or hold it over the back.

Other tips to help a camelid relax during these invasive procedures include the use of warm lubricant and the offering of food. Warm lubricant is preferred over cold lubricant as warm lubricant is more physiologic and helps to relax the anal sphincter. One might think that animals would not be willing to eat during these invasive procedures, but the author has witnessed many camelids willingly eat during rectal examinations. Eating makes the animal feel more relaxed and diverts their attention from the procedure.

Transabdominal Ultrasound Exams

Transabdominal exams are usually less tolerated than rectal exams for most animals. The author recommends making initial contact on the top of the animal's back and then sliding the hand down from the back, eventually arriving under the abdomen. Initially, touching the ventral abdomen often will startle the animal. It is worth the time to warm up lubricant in a bucket of hot water. Cold lubricant will encourage an animal to lie down. A firm hand pressing down on the top of the hips will also help. In any case, it seldom helps to try to prevent an animal from lying down by tying the head high. Offering grain at top-line level will lower the head a bit and may discourage an animal from lying down.

Physical Exams

Balancing techniques are useful for physical exams. Performing physical exams in a catch pen rather than a chute keeps the animal calm and gives the examiner more accurate readings for resting temperature, respiratory rate, and heart rate. When making contact with the animal, initiate contact with the dorsum of the back, allowing the animal to move rather than holding him still. If food does not interfere with the exam, it can be offered and will make the animal more relaxed during the examination.

Procedures Involving the Head

Difficulties encountered when doing procedures on or around the head are usually caused by fear and anxiety of the patient. Frequently, these can be attributed to an animal that is unaccustomed to having the head handled in a safe manner. An improperly fitting halter, or a handler that pulls on the head instead of balancing the animal can lead to fear in the patient. By adjusting the way these animals are handled, many techniques of the head can be made easier, safer, and less time consuming.

Oral Medication

Administering oral medications to camelids can be challenging. The medication is often foul-tasting, making the animal averse to having it in their mouth. The anatomy of the camelid is problematic, with animals having a small head at the end of a long neck. It is easy for the animals to successfully resist human attempts at control and it is difficult to hold the head still and introduce the dosing syringe at the same time. Camelids are also skilled at spitting out medication after it is deposited into their mouth.

The author prefers to work alone in a smaller area rather than working in a larger area with a helper. The exception is having an assistant whose sole job is to depress the plunger of the dosing syringe. A trailer is perfect. Two people often work against each other with the animal caught in between. Administering oral or ocular medications is the only time the author puts her arm around the animal's neck, but even with her arm around the neck, the intent is still to keep the animal in a natural state of balance with the weight naturally over its own feet and not to restrain. She does not hold from the head but instead uses her arm like a cradle as a place to steady the head as if making a small catch pen for the animal's head. A crowded catch pen, mini-catch pen, or trailer makes administering oral medication MUCH easier. Consider putting a neck wrap on the animal as it can be a major help when giving oral medication.

A 3–4-in. curved metal applicator (Figure 2.41) and a larger dosing syringe is better than a smaller one because it is easier to get the syringe in the mouth and depress the plunger if the end of the plunger is not protruding way out of the syringe barrel as it would be if the capacity of the syringe is being approached (for example, administering 8 cc with a 10 cc syringe).

The syringe is placed in an apron or another easy-to-reach location. In a catch pen, the animal is moved so that it is parallel to one side of the catch pen for support and balance. The neck is encircled with the dominant hand and the jaw is cradled with the same hand. The thumb can be raised and placed against the cheek to give you more control of the head (Figure 2.42). The elbow is kept up high to provide as much leverage as possible. The dosing syringe

Figure 2.41 An olive-tip dosing syringe.

Figure 2.42 (a) The handler is using the right arm with the elbow high up on the neck to contain but not restrain the head to administer an oral paste. (b) The handler is using a halter to help guide and contain this llamas head for oral deworming.

can be positioned with the non-dominant hand so that the thumb can be used to depress the plunger. Or, you can have an assistant depress the plunger. The tip of the dosing syringe should be slid into the corner of the mouth with the bend in the syringe pointing toward the cheek (away from the center of the mouth). The aim is to deliver the medicine into the cheek pouch, NOT into the throat. The medicine will flow between the teeth and into the mouth slowly, and it will be much less likely to end up being aspirated or spit out. When dosing a llama, given its taller size, it is easier to use a halter to balance the head to insert the dosing syringe (Figure 2.43).

The animal should be permitted to settle down after being caught and again after the syringe is put into the mouth. Attempting to administer medication into the mouth of an animal that is flailing, or pushing it too fast, will usually result in failure, and the animal will be more averse to the process in the future. Once the animal is calm, the nose should be tilted up a bit and the medication slowly administered into the mouth. Do not hurry. Keep the animal's nose tipped up until you see that the animal has finished swallowing. The consistency of the medication is important. The author prefers to have a solution mixed with the consistency of a thin milkshake; paste medications can be thinned with water to make it easier to be delivered via a preferred syringe. It is quite easy for an alpaca or llama to spit out an entire bolus of paste if it is too thick.

Eye Medication

Similar head support techniques as outlined above for administering oral medications can be used for administering eye medication. The head is cradled as previously described and gently rotated (forehead toward, jaw away)

Figure 2.43 Using the halter to balance a llama's head to administer oral medication.

into the chest of the holder. This positioning causes the eye to roll downward because of the internal balancing mechanism, which makes the animal want to keep the eye horizontal. The eye will rotate downward below the lower eyelid exposing the sclera. The medication can be applied onto the sclera. Since the pupil is below the lower lid, the animal does not see the medication coming and does not get frightened. The upper eyelid can be rolled up to make more room for the medication.

Trimming Fighting Teeth and Incisors

South American camelids frequently require trimming of teeth as a routine husbandry practice. Overgrown incisors may require trimming to allow for proper prehension of forages. Mature males have prominent upper and lower canine teeth and upper third incisors which curve caudally in the mouth. These teeth are known as the "fighting teeth," and are formidable weapons. It is recommended for these teeth to be trimmed to increase safety for human handlers and other male camelids, if pastured together. The technique for trimming fighting teeth and incisors is detailed in Chapter 12.

Chemical sedation is recommended for trimming fighting teeth. If there is some mild pain associated with the procedure, sedation with help as an analgesic and the person doing the trimming can do a better job if the animal is still. Sawing fighting teeth off at the gum line is not easily done on a conscious animal, and the teeth tend to break before sawing is complete. Sedation allows a more thorough job, affords the opportunity to apply topical pain relief, and allows the person trimming to make certain that the cut edge is smooth.

The process of trimming incisors with a motorized tooth saw and a speculum made for camelids is often so quick, that this procedure can be done with a light sedation or no sedation, depending on the temperament of the animal and the skill of the handler. In any case, working with the head and mouth before the procedure, understanding balance and using containment rather than restraint is recommended. If you elect to use OB (obstetrical) wire to shorten the incisors, a sedative for the same reasons mentioned above should be used to allow for a more thorough and careful job.

Microchipping

Microchipping is a method of identification and a popular practice in camelids. The preferred location is the top line or shoulder for two reasons: it is easy to insert the microchip under this loose skin, and it is easy to read once inserted. If the chip is to be inserted in the body, the same handling principles discussed for injections apply. If the microchip is to be inserted in the ear, it is recommended to prepare the animal by working with ears first and to use a neck wrap if available. Working ears can be done with a catch rope or handler helper at the top of the neck or with the animal haltered. Camelids tend to be ear sensitive, influenced by experiences within the herd. By slowly moving the hand from the head to the base of the ear and gradually to the tip, camelids will tend to accept gentle stroking (Figure 2.44).

Figure 2.44 Working with the ears helps facilitate easier haltering as well as medical treatment of the ears.

Earring

Earring is a technique that can be used to eliminate movement (Figure 2.45). Earring can be done humanely. If it is done correctly, it does not cause pain, but suggests to the animal that if it does move, it might be painful. The author compares it to someone holding my earrings firmly in each hand. It would not hurt but she can promise you she is not going to move around for fear that the movement might cause pain. The process involves squeezing upwardly and *not* twisting the base of the ear. The process of earring should not be painful. Earring preoccupies the animal and makes him reluctant to move. Hurting an animal by twisting the ears causes a fight; it does not prevent one.

Earring should not be used as a primary method for controlling an animal, but as a last resort. Times when this technique may be useful include placing a microchip in the ear. The animal must be still until the microchip has been inserted and the tool removed from the ear. If the animal tosses his head at the wrong moment, the tool used to insert the microchip can rip the skin of the ear. It is not possible to follow the movement of the ear and allow freedom of the head for this procedure.

The animal can be prepared for the earring by stroking the ear from the base to the tip with the bottom edge of the hand. If the animal is nervous, strokes should be faster at first and then slow down. A neck wrap is an extremely helpful tool for this procedure. A mini-catch pen or trailer is ideal. Grasp the base of the right ear and squeeze up firmly. Make sure that the nose is pointed forward, the head is in line over the neck, that the neck is in a comfortable angle and in line with the body, and that the body is in balance over the feet. In other words, make sure your animal is in balance.

Figure 2.45 Earring humanely requires that the ear be squeezed but never twisted.

Sedation

Many medical procedures are uncomfortable but not painful. Furthermore, many techniques are performed quickly. Sedation should be considered for medical procedures that are painful, and those that are long. Animals that are particularly highly strung or agitated may benefit from sedation for procedures for which other animals may not require sedation. Animals that are not able to stand quietly in a chute without restraint are candidates for sedation. It is more preferable to sedate a patient than risk injury to helpers, the animal, or yourself. By successfully performing a procedure in a sedated, but conscious animal, the animal may learn that the procedure itself is not bad and sedation may not be necessary in the future. For details about sedation protocols, see Chapter 6.

Halter Fit

Improper halter fit creates a multitude of behavioral problems. Box 2.4 lists some common behavioral problems that can be related to poor halter fit. A camelid's small head,

Box 2.4 Behavioral Problems Related to Improperly Fitting Halters

- Animal misbehaves when on a lead rope
- Animal kicks when being worked with
- Animal spits and kushes inappropriately when being worked with
- Animal is averse to routine, non-painful procedures such as shearing, trimming toenails, giving injections, doing ultrasound exams, or any other medical procedure

coupled with the leverage provided by their long neck, make proper halter fit trickier and more important. Improperly fitted halters can interfere with the animal's ability to breathe, eat, and ruminate.[5]

Llamas and alpacas are semi-obligate nasal breathers. They do not naturally breathe through the mouth unless there is no alternative. Camelids are either grazing or ruminating a significant percentage of the time; mouth breathing increases the likelihood of aspiration in these animals. Camelids have a SHORT nasal bone and soft cartilage for most of the length of the nose; the nasal bone on most adult camelids extends only about one inch rostral to the eyes. Downward and rostral pressure on the halter is likely to cause the halter to slip over the soft nasal cartilage where it will cause the cartilage to collapse and compromise the airway. Any indication that the halter can slip forward, off the nasal bone, and onto the soft cartilage where it may compromise airflow is likely to induce panic.

A tight noseband that encircles the mouth will limit the animal's ability to chew freely, a necessary requirement for eating and proper rumination. Since they are either grazing or ruminating a sizable percentage of the time, the animals are also chewing a sizable percentage of the time. Animals not able to do this may be prone to other issues such as malnutrition and weight loss.

Many llama and alpaca halters are not proportioned so that the halter can fit safely and comfortably. The noseband is often not big enough to permit it to slide all the way back on the sturdiest part of the nasal bone, which means it can compress the soft nasal cartilage. Even if the noseband is large enough, often there is not enough take up provided by the crown piece to keep the noseband in the proper place, allowing it to slip rostrally (Figures 2.46 and 2.47).

With the crown piece snug, there should still be slack in the noseband! Fitting the noseband means taking up extra slack, it does not mean tightening it around the nose. One or two fingers should fit inside the noseband of a fitted halter. A noseband that compresses the skin covering the bottom mandible or impedes the animal's ability to chew is uncomfortably tight.

Figure 2.46 The camelid skull clearly shows there is not much bone forward of the eye for a halter to rest on.

Figure 2.47 When the noseband is not on bone, it can easily compress the cartilage and compromise the airway.

The author has designed a halter that is proportioned to solve these problems. It may be better to work without a halter than to work with an ill-fitting one. If the noseband can be pulled forward more than 3/4″, it would be advisable to tighten the crown piece. It is better to have the crown piece temporarily a bit snug than to risk the noseband sliding off the nasal bone. Box 2.5 outlines the steps involved to fit a halter on a llama or alpaca.

Llamas and alpacas have small heads and young camelids have even smaller heads; be precise! (Figures 2.48–2.51). You must pay attention every time you put a halter on! Most haltering mishaps happen when we are just putting a halter on for "a few minutes" to do something quickly.

As a rule, take the halter off whenever you can and, if possible, do not ask an animal to wear a halter overnight.

Box 2.5 Step-by-Step Fitting of a Halter

1) Start with a halter that is properly proportioned and has the option to adjust the size of the noseband. Open up the noseband to one of the two largest holes.
2) Put the halter on.
3) Tighten the crown piece as much as you can.
4) Put your fingers on each side of the noseband and tug forward. If you can pull the noseband to the very edge or off the nasal bone, you must tighten up the crown piece or get a smaller halter.
5) Check to make sure that the halter throat piece is not pushing against the trachea or esophagus. If it is, this indicates the halter is too big.
6) After the crown piece adjustment, you **may** tighten the noseband. The noseband should never more than just gently touch all the way around the nose without pressing or restricting the animal's ability to move his mouth and jaw. Provided the crown piece holds the halter in place well up on the nasal bone, the adjustment of the noseband is less crucial for purposes of handling.
7) Check the crown piece about 10 minutes after you put the halter on or just before attaching a lead rope and leaving the catch pen. Nylon stretches by approximately 33%. As the nylon stretches and the fiber compresses, you could end up with a dangerous amount of slack in the crown piece.

Figure 2.48 This halter fits properly; the nose band is adequately sized to fit well up on the nose band and still provides room for the animal to open its mouth.

It is more comfortable for them to go without a halter. This is particularly important for animals that are otherwise sick. Llamas and alpacas housed at a veterinary hospital should not be left wearing a halter. A device called the handler helper can be left on safely and comfortably in situations when a novice must catch an animal easily (Figure 2.52).

Figure 2.49 This alpaca is wearing a halter that is too far forward on the nose. In addition, there is not enough room in the nose band to allow it to sit further back and it is restricting movement of the jaw.

Figure 2.50 This llama is wearing a halter that looks like it fits but, in fact, it is sitting just at the edge of the nose bone and the crown piece is not snug enough to keep it from slipping forward and off the bone. Additionally, the nose band is too small to allow for chewing.

Understanding Male Behavior in Camelids

Camelids are generally quiet, but males in the midst of a disagreement are rambunctious, aggressive, and violent. Neck wrestling and leg biting are common male interactions. Breeding male camelids present challenges and

Figure 2.51 This halter fits. You can see that there is slack in the nose band and the halter fits well up on the nose bone.

Figure 2.52 A handler helper is a great way to facilitate catching by people who are not trained to catch properly.

require more thoughtful and deliberate management. Breeding males are territorial and highly sexual (Figures 2.53 and 2.54).

To successfully shape the behavior of males, it is helpful to understand their behavior in relation to other camelids and understand their behavior in relation to humans. To convince males *not* to engage in natural behaviors is a losing proposition. A better approach is to make fighting or other problematic behaviors unnecessary. Anyone working with intact males should pay close attention and anticipate

Figure 2.53 Male llamas fighting.

Figure 2.54 Male alpacas fighting.

behavior. Doing this will allow prevention of unwanted behavior, which is more effective and safer than attempting to correct misbehavior once it has occurred.

Trying to force males that live together to play nicely all the time is impossible, but their environment can be controlled, which can diffuse many situations. Good facilities will allow for handling breeding males easily and safely. An intact male on a lead rope can learn to be respectful and cooperative even when females are around.

It is not always about dominance! A common misconception of many camelid owners and veterinarians is that undesirable male behavior is because of their desire to express dominance over another. The dominance model is overused to explain behavior between camelids as well as behavior between camelids and their human caretakers. The primary problem with the dominance explanation of behavior stems from the improper adaptation of the model from wolves and dogs. The term dominance means different things to different people; professionals and academics commonly disagree on the meaning of the word though most agree that the lay public frequently misuse it. Additionally, applying an across-the-board dominance hierarchy to all camelids, in all situations, oversimplifies very fluid, context-specific behaviors that may or may not have to do with a pecking order.[6] It also assumes that aggression is the result of natural camelid behavior when, in fact, it may be caused by human mismanagement. Dominance, according to most experts, should *not* be used to indicate a temperamental attribute, motivation, territoriality, or aggressive acts (even offensive ones).

Dominance is not absolute. "Every individual assumes the subordinate role at some point with some individual in some context unless the individual is *pathological*."[6] In terms of camels, natural feeding behavior seems to be devoid of dominance, and there does not seem to be rigid rules about who goes first or who initiates a grazing shift. With readily available forage, hierarchical relationships appear to be very laissez-faire. Schulte and Klingel observed camels moving from the boma (overnight corral) to the feeding grounds. They could not recognize a true leader, as the camels seemed to share the lead position, and the changes in front position were friendly and the relieved animal was not observed attempting to regain its former position.[7] In the camel population, males can drive and chase females in a sexual context but have absolutely no privileges when it comes to other things like food and salt. Schulte and Klingel concluded that camels do not routinely express dominance rather than in sexual situations and theorize that it is due to the evolution of the species in an environment where there was no need to monopolize resources.[7] Truly dominant individuals rarely engage in aggressive encounters. "Although aggression at times is used to establish dominance, agonistic encounters, particularly between familiar individuals are normally resolved with non-injurious ritualistic behavior."[6]

If there is limited food offered to a group of camelids, there will be fighting, but it is important to realize that this is about food, not about social rank. We humans are starting this fight and we can prevent it by managing feeding time differently. Based on the environment that shaped their behavior, camelids, particularly males, should not

have to compete for food. Once fighting is understood as competition for food and not an unavoidable issue of dominance, unnecessary fighting can be eliminated. Feeding males well away from each other defuses battles before they begin. Three feet per animal is often quoted as a rule of thumb but the greater the distance the better.

Remember the same rule of thumb, "distance = happy camelids," applies to all resources. Do not make your males fight over anything! Make sure that there is shade, water, minerals, and salt available to all members of a male group. Think of managing males as reverse musical chairs: there is always one more chair than players, so there is no need to defend or compete for limited resources.

Pen Size and Shape

Pen size can be manipulated, and it is natural to use the number of animals as a gauge for pen size. However, this rule of thumb does not take into consideration all necessary factors to determine how many males can safely be housed in a pen. Distance from coveted resources is key. The camel literature would suggest that there is a distance that young bachelor males must maintain from females. According to Schulte and Klingel, "The bull was able to chase bachelors of age up to 5 years which were kept in the vicinity. Whenever they came too close to the herd, they were attacked and chased up to 50 meters or further away. In no case was there any resistance."[7] There does seem to be a minimum size pen that will work for boys regardless of the numbers. Three intact males may need as much room as 10 females if they are going to get along. Subordinate animals must be able to get far enough away to communicate that they are giving up any claim to the coveted resource – in most cases females. Males will chase other males very vigorously to protect territory. In a pen that is too small, no matter what they do, subordinates cannot provide adequate distance and are always in trouble.

In addition to pen size, pen shape, the contour of the land, presence or absence of buildings, and the location of females in relation to the shape of the pen are influencing factors. If you have males that are not getting along you might think of offering more space or tinkering with feeding locations in relation to the females. For example, move temporary shelters or add temporary fencing to create a baffle that creates an impediment to chasing. Ideally, the females are completely out of sight. If the females can be seen but only from a specific part of the male pasture it can lead to fights, as this spot will be the prime piece of real estate on the premises. If you are in the process of setting up your farm, choose a pasture for your males that has hills or areas that provide visual cover for junior males and one that is either completely hidden or completely in view of the females.

Temperament is probably both genetic and environmental. The author's own experience would indicate that the genetic component is more important. Hyper-aggressive males that cannot live in a group without risk to all members of the group are a given and are likely born that way. Ironically, these males are not often particularly good breeders, they are easily distracted during copulation and are often more interested in what other males are doing than in breeding. Camelids that rely on aggression for every situation are almost always the same ones that have difficulty interacting with people. These males are not confusing humans with herd mates, they simply meet every encounter with aggression regardless of who is on the receiving end.

It is extremely important to re-shape the behavior as early as possible. The single biggest problem when dealing with males is the human practice of cornering, grabbing and holding camelids to catch them. The practice (fortunately or unfortunately) works most of the time in that most animals will eventually submit and allow themselves to be caught, haltered, and managed to some degree or another. BUT the ones that will not submit to the corner-grab-hold approach, are exceedingly difficult to deal with and the more they are handled physically the more adept and comfortable they become interacting physically with humans. These males are management problems, and since they may be passing their temperament along to future generations, they should be used for breeding with caution. Castration helps with this problem, but the tendency to resist physically instead of submitting persists, and these animals are not suitable for new owners.

Observing and understanding behavior is necessary for proper management. However, returning like behavior (spitting back, wrestling, kicking or other dominance approaches) to misbehaving animals is easily misinterpreted by the recipient and may be dangerous to the human particularly when it comes to breeding animals. Communication between animals is incredibly nuanced and relies at least in part on having the proper anatomy.

To manage males successfully, you must manage their environment and work in a way that does not frighten them into behaving aggressively. Good animal management – laneways, catch pens, good fences and handling skills are the same things that also make managing males easy. With intact males, these things are essential. Cornering an adult male camelid and trying to wrestle him to a standstill will scare him so he may respond in kind and it will have nothing to do with dominance and everything to do with self-defense.

Berserk Male Syndrome or Novice Handler Syndrome?

Berserk male syndrome (BMS) is a phrase that has been around for a long time. Llama Breeder Paul Taylor originally used the term BMS in an article in late 1980 or early 1981. Paul said, "It seems to be the end result of a series of confusing and negative interactions with humans, beginning with the breakdown of the normal standoffishness that herd raised llamas show in their relationship to humans. A male llama that has been bottle fed or constantly petted and fondled as a baby will show no hesitation about initiating contact with humans, as in the mild case of the pushy llama who runs up to be petted or bumps with his chest against people in the pasture with him. Such a llama is apt to be pushed or slapped to keep him away. This conflict can escalate over a period of time, possibly with changing owners or eventually the use a whip or club to keep the animal at bay. The final result seems to be a tangled combination of normal llama behavior for dominance assertion, breeding and defense."[8]

The term "Berserk male syndrome" is outdated and does not reflect the true cause of the adverse behavior, the human. While this behavior is more prevalent in intact males, the behavior is not restricted to males.[9] It is also important to note that this is not a condition that happens exclusively in camelids; similar aggressive tendencies toward humans are observed in all domestic species (cattle, cats, dogs, and horses). Dr. LaRue Johnson proposed the term **A**berrant **B**ehavior **S**yndrome for BMS, and today the phrase BMS or ABS is used to describe llamas and alpacas that are anything from innocently engaging in problematic behavior for which they have been reinforced to those animals that are completely out of control and dangerously aggressive, which can become confusing.

In order to decide how to prevent aggressive behavior, it is important to be aware of what causes it. It is also important to be able to recognize the early warning signs of what could become problem behaviors and take action at that time.

The Novice Handler Syndrome

The cause of inappropriate aggression in most animals is multifactorial. Camelids are born with different personalities and tendencies. Humans have different behavioral styles, levels of confidence and experience. Environmental factors such as multiple owners/handlers, age of weaning and herd composition also play a part in the behavior of a llama or an alpaca. The number one cause of behavior issues in camelids is due to inappropriate management and interaction by people. "Novice Handler Syndrome" is a more accurate and descriptive term describing these inappropriate behavior tendencies than BMS or ABS. One especially important facet of the "**N**ovice **H**andler **S**yndrome" is the tendency of the human to misinterpret what animals do and why they do it or . . . how behavior works and why animals repeat some behaviors and not others.

A common scenario that leads to this inappropriate behavior is a novice owner who has a small herd and their first llama or alpaca cria, and it is a boy. His instincts tell him to play, wrestle, bite, and bump. In this typical scenario, there is not a lot of room to run and not much to do, and the other animals in his environment do not want to play, in fact they rebuff him when he tries to mount or chest butt. This young animal is seriously bored. The high point of his day is when the owner comes out and sits with him. He starts investigating the owner by interacting with her. From the owner's point of view, everything that this baby does is cute, and she encourages this interaction. As time goes by the owner continues to interact with him and he becomes increasingly comfortable with being close to this human companion. Often, he picks at her clothes, puts his nose in her face and rubs against her. This common scenario, though cute when the cria is young, can become dangerous when he is an adult weight, as these animals will try to chest bump, rearing up, and wrapping the front legs around human caretakers. This is a typical example of instinctive adult behavior that was shaped and encouraged by the novice human caretaker.

Young camelids are going to explore their environment and check out what behaviors net them something they want, what behaviors are neutral and not worth repeating and which behaviors are frightening or result in discomfort. In a herd environment, where humans are occasional visitors to the pasture, behavior is shaped almost entirely by conspecifics. When humans are new to camelids consumed by their interest in them and spend hours in the barn, they have a much greater influence on the behavior of young alpacas and llamas. Young llama or alpaca babies that rub, lean, stand closely, walk right up and put their nose in your face or crotch and a fail to yield space when you move toward them have been encouraged to do these behaviors by receiving attention when they do them. These behaviors are NOT indications of love or friendship they are simply behaviors that were tried on humans just as they were tried on other herd mates and reinforced by someone who had no knowledge of where the behavior would likely lead. These trial behaviors can become dangerous to human caretakers and because of that, dangerous for the wellbeing of the animal. It is in the animal's best interest to guide him in a different direction.

The FIRST and best solution is do not encourage behavior that you do not want. Remember that behavior that seems harmless in a 20-pound baby is not going to magically go away when the animal is full-grown. Teach animals that they do not touch you and be respectful of them by handling them in a catch pen and using balancing techniques, not physical force. Mutual nose greetings are the only allowed physical contact. When you have neonates around, watch them from outside the pasture for the first few days. Do not encourage them to approach you and, if they do approach you, participate in a quick nose to nose greeting and walk away. If you have a young animal that is overly interested in humans try closing all animals, including the youngster, away from you when you clean the pastures. Do not do ANYTHING that encourages these youngsters to follow, sniff or play with you. You are going to be too interesting, particularly if the baby has no other babies to play with. The adult females in the herd, as well as the mother, are going to ignore this young baby or chase him away, so if you interact with him, you will be the most interesting item around.

Personal space is important to maintain; however, it is not necessary to push inquisitive crias away. A foot and a half of distance is a good rule of thumb. A human need only stop an animal from entering this space. And it is okay for you to reach out to pet and touch the animal if it is okay with him. Reach out away from your body and you can make contact without wrapping yourself around the animal. It is fine for us to touch our animals, but it is NOT fine for them to touch us.

Your interactions with animals must be respectful and should not be physical. It is not okay to wrestle and physically control the animals and not expect that they might do the same with you. Screaming and chest butting are natural behaviors that camelids engage in with each other – when humans yell and push it is remarkably similar. By becoming physical with your animals, these behaviors are encouraged. It is much better to put your hand up and show the young camelid what you want him to do which is to stop and notice your personal space. Use your hand to indicate the edges of your personal space. Do not rely on verbal cues, use your hands to show young animals to stay a polite distance from you, you will probably never have to do anything more (Figures 2.55 and 2.56).

Animals that have already been taught to rub and/or push will have to be re-educated. One should figure out what this animal is gaining by following a person too closely or jumping on him or her. In most cases he is alleviating boredom. Most of the time, these young animals only choose to express these behaviors when they are deprived of age-appropriate playmates (more important for males to have male playmates). If simply putting your hand out does not work, you may have to alter the environment so that the animal cannot engage in the behavior and then work on replacing it with something that works even better for the animal to get what he wants. Training a new behavior is a bigger subject than can be addressed here. My preference for training behaviors is to use food and a clicker. I begin by teaching the animal to target on my hand (that is to put his nose on my palm) or a plastic training aid and

Figure 2.55 This photo demonstrates a respectful and safe interaction between a cria and people.

Figure 2.56 This photo demonstrates an appropriate interaction in the field.

Figure 2.57 This camel was previously aggressive when fed. The author taught the animal to target and then nailed the target on the back wall of the stall. At feeding time, the camel was cued to touch the target and was safely away from the feed bowl allowing caretakers to feed safely.

then I can put my hand/training aid out to the side away from my body. The animal targets on my hand or the training aid and is reinforced. It is not possible for him to be targeting on my outstretched hand and be too close to me at the same time (Figures 2.57 and 2.58).

Another suggestion for preventing the development of unwanted behavior in young camelids is to keep children away until the cria understands how to behave appropriately. Children are small and less intimidating to approach. Children are not disciplined enough to interact with impressionable young animals appropriately. When children are around, animals that do not understand how to behave should be haltered (Figure 2.59). Gelding an animal will help, however, it will not solve the problem – it simply uncomplicates it. The pros and cons of early age gelding are discussed in Chapter 16.

It is recommended to treat females no differently; treat them respectfully and insist on respectful behavior in return. Crias do best if they have other peers to play with. With other babies around, in many cases, the whole problem becomes a non-issue. If you are new to camelids, it would be preferable to arrange for single babies to board at another breeder's farm during the first few months of a youngster's life. This may represent a bit of inconvenience but will save potential problems from developing later.

Camelids that become dangerously aggressive and will not respond to retraining are especially problematic. Once an animal learns that aggression works to make him/her feel more secure, it is exceedingly difficult to convince him to NEVER use these behaviors again. Training techniques that rely on force or reciprocal aggression usually escalate the behavior. When treated this way, most aggressive camelids will become selectively obedient to those humans

Figure 2.58 By using a pouch to secure the food, a clicker and food reinforcement can be used even when working with camelids in groups.

Figure 2.59 Children frequently interact inappropriately with young crias. While this may be cute, it is teaching this animal that it is acceptable to engage in physical contact with humans.

that have "dominated" them and of whom they are afraid. It is also likely that these camelids will eventually turn on the owners and so they cannot be trusted. Often euthanasia is the only safe and most humane option for these animals.

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3

Feeding and Nutrition

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Though many of the camelid species (Old and New World) have been domesticated for thousands of years it seems inconceivable that there is such a paucity of widely published, peer-reviewed studies addressing specific nutritional information to scientifically support a camelid feeding program. As Dr. Fowler wrote in the third edition of this text: "Camelid owners and managers develop strong views on what constitutes appropriate feeding, and newcomers to the industry are bombarded with; this is the way you have to feed." Resultant from the lack of camelid nutrition research, owners rely on internet information and mutually shared information that may or may not be based on valid science. As Dr. Fowler stated, there are no absolutes in feeding practices for camelids. It is not what you feed but how you feed it! It does help to understand, however, why a feed may be appropriate or not.

In North America, the South American camelids (SAC) industry has advocated that pasture or hay are the "best" forage feeding programs and avoid the more economic feeding of ensiled grass (haylage). This perspective is extrapolated from the sheep and goat industry out of a fear of listeria contamination of ensiled feeds and risk of listeria infections. In England and other European countries, grass silage is very successfully fed to SAC (W. Barnett, personal communication). There is no reason why the SAC digestive system could not utilize grass or alfalfa silage or even corn silage as a reasonable feed source. As with other ruminant animals, proper harvesting, ensiling, and storage practices are necessary to minimize the risk of silage-based disease concerns. Another camelid feeding myth is the recommendation that alfalfa should not be fed to SAC. Concerns voiced include alfalfa being too high in protein, causes obesity, increases fiber micron size, induces bloat, among others. The author observed alfalfa hay being placed in areas of the Moroccan desert to entice dromedary camels to be managed as well as supplement their limited forage resources. Yes, if alfalfa is fed inappropriately, it may have higher protein or result in selective feeding of the leaves leading to obesity issues, but that is a feeding management issue and not an issue with alfalfa as a forage feed.

Veterinarians may be unable to clarify the issues without understanding basic information available on camelid nutrition, hence the purpose of this chapter. This chapter will address feeding practices of camelids from the perspective of the "nutritional triad," which addresses animal factors (e.g. physiologic state, body condition, health status), feed composition, feeding management and their interactions. Much of the information presented addresses SAC and this information can be extrapolated to Old World camels (OWC). Specific OWC data will be highlighted. The term "camelids" will be used inclusively to address both SAC and OWC throughout this chapter.

Anatomy and Nutritional Adaptations

Camelid digestive tract anatomy and physiology is described in Chapter 12 and published reviews are available [1–4]. Morphologic descriptions of SAC and OWC forestomach are similar with only observable differences in comparative lengths of small and large intestines [3]. One should have some appreciation of the anatomy in understanding approaches to nutritional management and identifying potential nutritional issues. Camelids belong to the Suborder Tylopoda within the Order Artiodactyla. Camelids are misrepresented as "pseudo-ruminants" due to differences in foregut anatomy with species from the Suborder Ruminantia. The term "ruminant" refers to

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Companion website: www.wiley.com/go/niehaus/camelids

the ability of a species to regurgitate consumed food and remasticate ("chewers of the cud"), which camelids can accomplish equally as well as ruminant species. Throughout this chapter the term "ruminant" will refer to taxonomic ruminant species as the argument just made would indicate camelids can effectively accomplish rumination. Camelids in comparative feeding trials are superior to ruminant species in fermentative digestion of lower quality forages suggesting a greater rumination ability [5–12].

Camelid digestive tract function is considered a process of parallel evolution with ruminants in the camelid's adaptation to their native environment (see Chapter 1). To fully appreciate feeding management practices for camelids in any environment, one needs to understand their evolutionary, physiologic and metabolic adaptations to their native environment.

Physiologic Adaptations

Llamas and alpacas are concentrated in the Altiplano, which is an area of broad, high-altitude valleys and plateaus at elevations over 3800 m (12 500 ft). The climate of this area consists of a long, dry season and a short, wet season, with 75% of the rainfall occurring from December through March. The dry season is from May through October. The growing season (wet) is characterized by low temperatures (more than 300 nights of frost per year) and intense solar irradiation. Daytime temperatures reach a maximum of 18.3 °C (65 °F); nighttime temperatures fall to a low of −12 °C (10 °F) (Figures 3.1 and 3.2).

In South America, SAC are not fed concentrates or nutritional (e.g. mineral) supplements and are rarely fed cured hay. They feed on the native grasses and forbs. As plants grow, in relation to solar radiation and rainfall, wet season forages are of high quality and abundant, on a par with quality grass and legume forages agronomically grown in North America (refer to Forage Quality discussion). In contrast, dry season forage is reproductively mature, highly lignified and limited in available quantity. Recent research and educational symposia in Peru with alpaca managers have prompted many large SAC operations to start considering supplying free-choice mineral supplements and even some commercial concentrate products to improve animal productive efficiency.

With forage variation in availability and quality between wet and dry seasons, SAC have adapted to the "feast and famine" forage cycle by depositing layers of fat in the subcutaneous, muscular, and retroperitoneal tissue during the wet season and efficiently mobilizing these reserves during lean times. To accommodate the low availability and quality of dry season forages, SAC have reduced the rate of passage of plant cell wall material through compartment-1 (C1) allowing for prolonged microbial fermentation resulting in an observed greater extent of cell wall degradation. In conjunction with the slower rate of fiber passage, SAC have a more rapid rate of liquid passage in C1 that would increase microbial flow to the small intestine and maintain log-phase growth of microbial populations. A consequence of prolonged dietary fiber retention in C1 is reduced feed intake. San Martin documented a 36% reduction in feed intake in llamas and alpacas compared to sheep when fed a low-quality forage in contrast to a 26% intake reduction between SAC and sheep for higher quality forage [11].

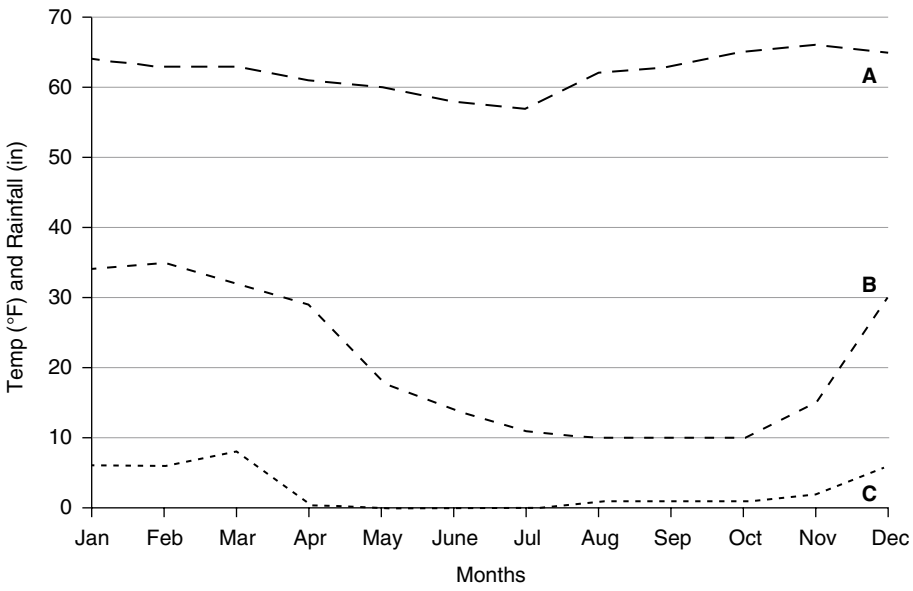

Figure 3.1 Average ambient maximum (A) and minimum (B) temperatures (°F) and rainfall in inches (C) at a station in the Altiplano region Peru.

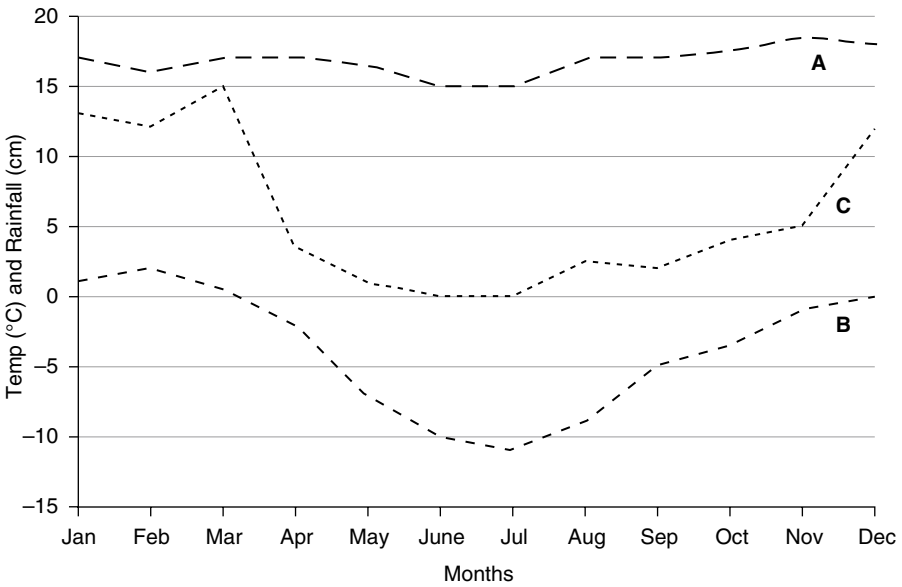

Figure 3.2 Average ambient maximum (A) and minimum (B) temperatures (°C) and rainfall in centimeters (C) at a station in the Altiplano region Peru.

In most ruminant animals, ruminal rate of passage is increased with consumption of low-quality forages resulting in greater passage of dietary forage in feces and reduced digestibility. Anatomic differences between ruminants and camelids relative to orifice size between C1 and C2 being much smaller in camelids compared to the reticulo-rumen orifice may account for observed differences in intake capacity as related to the passage of larger particles [3]. Alpacas were found to have less forestomach volume than sheep on a metabolic body weight (MBW) basis, thus this selective ability to retain larger particles would account for the observed lower intake capacity of SAC and most likely OWC compared to ruminants ([1, 11]. This adaptation to food followed by seasonally deplorable feeding conditions is usually completely forgotten by North American managers. Since there is hardly ever a lean time in North American feeding systems, animals often become obese.

Camels are highly adapted to the consumption of dry, harsh forage. Dromedary camels foraging in their native environment prefer to browse during the wet season and graze during the dry season. Camels do not exhibit the feast and famine syndrome, as described for SAC, but do store fat in their humps. The well-nourished camel has a firm upright hump, whereas an emaciated camel's hump is smaller and may flop to the side, particularly in Bactrian camels. If Bactrian camels are overfed when maintained in captivity, the humps may also bend over to the side because of the excessive weight. There may also be a genetic component to the hump's fibrous infrastructure that is not strong enough to cope with extra weight.

Metabolic Adaptations

There are two unique metabolic adaptions in SAC compared to ruminant animals that have significant impact on feeding practices and nutritional diagnostics. In contrast to all ruminant animals, camelids maintain a blood glucose concentration (5.7–8.9 mmol/L [103–160 mg/dL]) equivalent to non-ruminant herbivores, omnivores, or carnivores; nearly twice that of ruminant species [13]. Ruminants rely on ruminal propionate production from microbial fermentation of sugars and starches predominately as the primary substrate for hepatic gluconeogenesis. Given the low dietary content of sugars and starches in non-supplemented forage diets of SAC, one questions how they metabolically maintain such high blood glucose concentrations.

Cebra and colleagues have documented a reduced ability of SAC to secrete insulin and reduced tissue insulin sensitivity [14–16]. This metabolic adaptation occurs over the first year of life as llama crias showed a robust insulin response to glucose loading [17]. This modification in insulin-glucose dynamics facilitates the SAC's ability to rapidly mobilize body lipid reserves as insulin blocks action of hormone sensitive lipase in adipocytes, thus promoting lipolysis releasing free fatty acids to be oxidized for energy. Tissue insulin insensitivity contributes to the observed higher blood glucose concentration. Consuming a high forage diet results in predominate C1 fermentation end products of acetate and butyrate. It is assumed SAC tissues oxidize these short chain fatty acids as their primary metabolic fuels. These findings with insulin would suggest SAC would not metabolically manage high starch diets very well in contrast to traditional ruminant feed supplements to promote production.

Van Saun, proposed another pathway to account for the observed SAC blood glucose concentration [18]. Blood urea nitrogen (BUN) concentration in SAC as reported in the literature is greater (>7 mmol/L [>20 mg/dL]) than the concentration of most ruminant animals [13, 19]. Elevated BUN concentration may be interpreted as prerenal azotemia, which would not be expected across a population. More likely causes may include feeding excess dietary protein, altered urea metabolism, higher rate of protein turnover or some combination [18]. Described physiologic adaptations of the SAC digestive tract would indicate inherent promotion of microbial growth even in the face of consuming low-quality forages. Mixed rumen microbes contain between 45 and 60% crude protein (CP), which is of high quality and digestibility [20]. Increased flow of microbes to the small intestine would deliver large quantities of amino acids for absorption. These amino acids could then be used as substrate for hepatic gluconeogenesis in lieu of propionate as in ruminants. Utilization of amino acids for gluconeogenesis results in excess ammonia from initial deamination. Ammonia being a cellular toxin would enter the hepatic urea cycle for detoxification into urea. The generated urea would account for the greater blood urea concentration observed in SAC.

In ruminants and camelids BUN is recycled via saliva to the foregut. Llamas have been shown to be very efficient urea recyclers and have lower urea turnover and renal excretion rates compared to other ruminants [21, 22]. Recycling of urea to the foregut fermentation vat facilitates dietary fiber fermentation as cellulolytic bacteria solely utilize ammonia as their nitrogen source to generate microbial protein [23]. Llamas have greater forestomach urease activity allowing for greater urea utilization [21, 22]. Providing an ammonia source to the forestomach allows the microbes to ferment low-quality forages that typically contain low nitrogen content. Collectively, these two metabolic adaptations would allow the SAC to survive under the harsh conditions of the prolonged dry period. Though these metabolic adaptations facilitate survival on low-quality forages. They also predispose SAC to energy-related disease conditions, most notably hepatic lipidosis [24]. Unlike ruminants, SACs are not ketogenic with negative energy balance and respond to such nutritional deficits more like cats where hepatic lipidosis occurs in both genders and in any physiologic state. This observation further supports the hypothesized role of amino acids in the unique SAC metabolism. Similarly, OWC have higher blood glucose concentration compared to ruminants and generally higher BUN concentration compared to cattle [25, 26]. These findings would suggest OWC have similar physiologic and metabolic adaptations of SAC.

Camelid Digestive Process

The camelid digestive process is similar to a moderately-sized browsing or grazing ruminant species with modifications resultant from their unique metabolic adaptations. The digestive process has been reviewed and described for SAC [4, 27–30] and OWC [31].

Prehension

All camelids have incisor teeth that are firmly fixed in the mandible, like dentition of sheep and goats but in contrast to the loose attachment of bovine teeth. These teeth, pressing against the upper dental pad, shear vegetation. These animals are able to graze short plants and cut them close to the ground, but their general behavior is to move about a feeding area, picking a mouthful here and there, not mowing any one spot clean unless they are tethered.

The lips of camelids are unique. The upper lip is split by a labial cleft. Each side of the lip can be manipulated independently to investigate a potential food item and draw it to the teeth. SACs rarely consume foreign bodies because of this fastidious checking of feed with the lips, but camels may (see Chapter 12).

The tongue does not participate in prehension. Because the tongue rarely protrudes from the mouth, camelids do not, as a rule, lick themselves, their young, or salt blocks. Some may lick at a salt block long enough to obtain some supplement, but usually camelids chew at the block rather than lick it.

Mastication

Initial chewing is cursory, sufficient only to mix feed with saliva to form a bolus to be swallowed. The amount of time required by a camelid to complete feed ingestion varies with the quality and availability of the forage, but in most cases, it occupies a third of the daylight time. Camelids are not active in the dark.

Mastication is also a part of the rumination cycle. Koford described the rumination cycle in the vicuña [32]. While resting, the vicuña brings up a bolus and begins chewing, with the mandible scribing a horizontal figure-eight arc. A deer or a bovine masticates with a unilateral elliptic movement of the jaw. The vicuña retains the cud in the mouth for 15 seconds, during which time it chews 25–35 times. That bolus is then swallowed, and the process repeated. Other camelids chew with a similar pattern.

Saliva

The gross and microscopic anatomy of the salivary glands are similar to those of other artiodactylids [28, 33]. Salivary secretion is necessary to moisten and lubricate feed so that it can be swallowed. Ortiz et al. characterized alpaca parotid saliva composition and flow rate [34]. Prefeeding parotid saliva flow was 140 mL/hour, had a pH of 8.6, and electrolyte composition of 121 mEq/L HCO_3, 33.5 mEq/L HPO_4, 164.8 mEq/L Na, and 13.7 mEq/L K. While feeding, the flow was 202 mL/hour, pH was 8.59, composition was 127.8 mEq/L HCO_3, and other ion concentrations were similar to prefeeding levels. At postfeeding, the flow remained higher than during prefeeding, at 159 mL/hour, and the pH stayed at 8.58. Other parameters returned to prefeeding levels. Alpaca salivary flow has been reported to be similar or greater than sheep, which would result in greater buffering capacity for alpacas given their smaller sized forestomach compared to sheep.

Forestomach Development

Development of the forestomach is less well understood in camelids compared to ruminants. In contrast to ruminant forestomach anatomy, there are no papillae extending from C1 or C2 surface to facilitate volatile fatty acids (VFA) absorption. Progressive rumen development in ruminants is described relative to papillae growth stimulated by fermentation of sugars and starches to produce butyric acid [35], and without papillae, a parallel assessment is unable to be made in camelids. It is possible that the observed higher death loss of weaned crias (i.e. tuis) born late in the calving season is a result of inadequate C1 development to accommodate the available lower quality forages of the dry season.

During milk feeding, the ruminant forestomach has a muscular reticular (e.g. esophageal) groove that when stimulated will direct consumed milk directly to the omasum and abomasum for digestion. Camelids have a similar structure that directs suckled milk directly to C2 and into C3. Vallenas found the response to chemical stimulation was more like that of a bovine than of an ovine [36, 37]. There was good to fair response with 10% solutions of sodium sulfate, sodium chloride, and sodium bicarbonate, but no reaction to a copper sulfate solution [37].

Physical size changes of the three forestomach compartments reach adult comparisons by approximately eight weeks of age. Significant microbial activity occurs around 12 weeks of age. Ruminants show a decline in blood glucose concentration in concert with decreased forestomach pH and VFA production indicating significant rumen fermentation capacity. Elevations in blood β-hydroxybutyrate concentrations have been suggested as an indicator or rumen activity and ability to be weaned in calves [38]. With the difference in forestomach anatomy and physiology, a similar marker is not likely in camelids. Samaniego indicated there was a decline in blood glucose concentration in association with production of VFA and drop in C1 and C2 pH at approximately 16 weeks in growing SAC [39].

Forestomach Fermentation

The fundamental concept behind modeling processes of rumen fermentation is the competition between the rate of passage of material versus the feed's inherent rate of digestion [40]. Fermentation reactions must be facilitated by removal of end products, namely VFA and ammonia and continuous influx of substrate. Production of fermentation acids induces a drop in pH that must be buffered to prevent slowing of microbial growth or alteration of VFA proportional composition. Though there are many potential fermentation products generated in the forestomach, the quantifiably predominant end products are acetate, propionate, and butyrate equivalent to that observed in all ruminants [41, 42]. Llamas fed at maintenance had proportional concentrations of acetate, propionate, and butyrate of 68, 19.7, and 12%, respectively [43]. Waste gases from fermentation include methane, carbon dioxide, and hydrogen sulfide predominately.

The camelid forestomach structurally composed of compartments C1, C2 and C3 is uniquely adapted to facilitate microbial growth even when consuming low-quality forages. A unique feature of camelid C1 and C2 are the presence of ventral saccules covered by a glandular mucosa and nonkeratinized squamous epithelium covering the dorsal lining. These saccules secrete significant amounts of bicarbonate to complement salivary buffering action as well as facilitating absorption of VFAs [4, 33, 44]. A glandular epithelium is also present in the upper two-thirds where fermentation of smaller particles can continue. Absorption rate of generated VFAs is significantly greater compared to omasal absorption in sheep and goats [44]. The distal aspect of C3 has typical gastric epithelium possessing fundic glands responsible for initial proteolytic digestion.

Forestomach contents in camelids are more homogeneous and relatively dry compared to ruminants. In ruminants, the rumen contents are stratified with a gas layer dorsally, a solid layer of ingesta (i.e. fibrous mat) in the middle, and a more liquid and small particulate matter layer ventrally. This causes some difficulty when attempting to aspirate ingesta to transfaunate another animal. Bloat is relatively rare in camelids, especially relative to alfalfa

consumption compared to ruminants, and the homogeneity of the ingesta may be a factor in avoiding frothy bloat. Homogeneity of camelid forestomach contents may be a function of different motility pattern. In camelids at rest, three to four contractions of C1 per minute are average [45, 46]. If the animal has eaten recently, the rate is faster. The contraction wave of C1 is from caudad to craniad, in contrast to the craniad-caudad wave of the ruminant. The increased frequency and exerted pressure of C1 contractions leads to better mixing of contents and potential for greater microbial association with feedstuffs.

Many studies have investigated camelids' superior increased efficiency in extracting energy and other nutrients from coarse forages [6, 10, 12, 47]. Some early studies indicated that alpacas were 50% more efficient than sheep in digesting dietary fiber [10]. Later and more nutritionally detailed studies demonstrated greater efficiency, but to a lesser degree, in experiments involving poor quality forage, and no difference in those involving high-quality forage [7]. Liu et al. reported no difference in digestibility between sorghum-sudan grass and alfalfa fed to alpacas and sheep; however, this study fed additional dietary supplements beyond the forage, which included 22% ground corn [48]. In this study, alpacas performed similar to sheep with either forage, but diets exceeded protein requirements and both diets included quality forages based on total dietary fiber content. Dietary ingredient interactions have not been addressed in SAC feeding research and need to be further elucidated relative to digestive performance. Recent comparative studies indicate that forage CP and neutral detergent fiber (NDF), a metric of forage maturity and quality, are key factors in defining whether camelids have a digestive advantage over ruminants. Use of *in situ* methods to determine C1 feed digestibility indicated a digestibility advantage in SAC compared to ruminants even when retention time was equal [9, 49]. It is often assumed that the observed longer retention time was underpinning digestibility advantage for SAC; however, many of the previously described differences in forestomach physiology between ruminants and SAC collectively account for potential differences in feed digestibility capacity.

Forestomach Microbiology

The microbial flora that comprises enteric fermentation systems across herbivorous species are essentially the same diversity of species; however, relative microbial species population proportions will vary based on predominate feedstuffs consumed [23, 40]. More than 300 bacterial species along with fungi, yeast, viruses and bacteriophages have been identified in rumen environments, with bacteria playing the predominate role in feed fermentation. Populations of bacteria perform a limited range of anaerobic fermentation reactions on selected substrates generating specific end products (Table 3.1). Other

Table 3.1 Characterization of microbial populations and associated metabolic requirements found in an anaerobic fermentation system.[a]

Class of organism	Primary substrate	Specific requirements	Primary End products	pH Tolerance
Fiber Fermenting Bacteria	Cellulose, Hemicellulose, Pectins	Ammonia Iso-acids Cofactors	Acetate Succinate Formate, CO_2	Neutral 6.2–6.8
General Purpose Bacteria	Cellulose Starch	Ammonia Amino Acids	Propionate, Succinate, Butyrate Ammonia	Acid 5.5–6.6
Nonstructural CHO Bacteria	Starch Sugars	Amino Acids Ammonia	Propionate Lactate Butyrate Ammonia	Acid 5.0–6.6
Secondary Feeders	Succinate, Lactate Fermentation Endproducts	Amino Acids	Ammonia Iso-acids Propionate	Neutral 6.2–6.8
Protozoa	Sugars, Starch Bacteria	Amino Acids	Acetate Propionate Ammonia	Neutral 6.2–6.8
Methane Producing Bacteria	CO_2, H_2 Formate	Coenzyme M Ammonia	Methane	Neutral 6.2–6.8

[a] Adapted from Chase, L.E. and C.J. Sniffen, Cornell University.

bacterial populations may utilize end products of fermentation from other species to generate final VFA products as well as generate necessary metabolic precursors for other bacterial species. The microbial fauna of anaerobic fermentation systems collectively contributes to feed digestibility and animal performance. The primary challenge to anaerobic fermentation systems is the potential for excess production of lactic acid from sugars and starches lowering environmental pH and adversely affecting cellulolytic activity. This occurs predominately due to increased growth of *Streptococcus bovis* induced through the provision of readily fermentable carbohydrates in the diet [23].

There is little difference in the diversity of microbial species in the forestomach of camelids compared to other ruminants [50, 51]. Increased acetogenic bacterial populations has been indicated in llamas, which would indicate greater fiber fermentation activity [52]. Similar VFA profile and changes in VFA profile with different forages between ruminants and SAC would support the concept of similar forestomach fauna and fermentation activity. Alpacas and sheep fed diets containing 70% sorghum-sudan grass or alfalfa had similar VFA molar ratios that varied with forage [53]. In this study, there was a slight advantage in alpacas relative to acetate production consistent with the suggestion of greater acetogenic bacteria in SAC compared to ruminants. The ability to successfully transfaunate camelids, as assessed by improved intake, with rumen contents from cattle, sheep, or goats would be further evidence of minimal forestomach microbiome differences between camelids and ruminants [54].

Intestinal Function

Intestinal digestion and absorption in the camelid are apparently comparable to the process in ruminants. Camelids lack a gallbladder, so bile may flow continuously. The cecum and large colon do not function as primary fermentative chambers. The large intestine forms a spiral colon that diminishes in diameter by two-thirds within the coils. Fecal pellets are formed approximately halfway through the spiral. The diminution of size contributes to the formation of impactions in llamas. The transit time of particles through the gastrointestinal system (GI) varies with particle size but averages approximately four days [55].

Ratio of small-to-large intestinal length was 1.26 and 2.0 in alpacas and dromedary camels, respectively [3]. Both ratios are lower than typically observed in ruminants. The large intestinal length is accounted for in the greater number of gyri of the spiral colon in camelids (5.5 vs. 3 or 4) compared to ruminants [3]. This lower ratio is suggestive of greater ability for water conservation by the large intestine consistent with the water regulation abilities observed in camelids compared to other ruminants. It is also noted that water is absorbed in the proximal portion of C3 of camelids compared to ruminants adding to their ability to conserve water from digesta.

Nutrient Requirements of Camelids

Prior to the publication of the National Research Council's (NRC) report on nutrient requirements for small ruminants (sheep, goats, cervids and SAC) in 2007, there was limited controlled research published in the literature defining dietary nutrient composition for llamas and alpacas outside of South America [56]. The situation is similar or even worse for OWC. Of the published literature, most studies only addressed either llamas, alpacas, or dromedary camels and were limited to maintenance requirements. The nutrient requirements for SAC defined for different physiologic states in the NRC (2007) report were derived on extrapolated models using sheep and goat production requirement data and defined maintenance requirements for energy and protein for SAC [18]. Lopez and Raggi, published llama and alpaca dietary feeding recommendations for differing physiologic states, again using extrapolations from goat data [57]. A similar descriptive method for determining OWC nutrient requirements has been generated [58]. Summarized recommendations for dietary nutrient content in support of different physiologic states for SAC and OWC are provided. Appendix tables provide a range of conversion factors (Table 3A.1) and (Table 3A.2) commonly used in nutrition.

Water

The drinking behavior of SACs is to suck in water with the mouth slightly opened. Camelids have learned to drink from all types of waterers and containers but are slow to become accustomed to automatic waterers, especially if it is necessary to press a lever to cause the water to flow. Running streams and ponds are natural for them. They are fastidious and may refuse to drink polluted water. This is interesting because packing llamas on the trail are inclined to urinate and defecate in a stream while they are drinking. Trail llamas will surprise the novice in that they frequently do not drink during the day, even with ample access to water.

Requirement

Like all animals, camelids require adequate amounts of good quality water to sustain life, reproduce, work, and produce fiber and milk. Water is lost through urine, milk, perspiration, and evaporation from the respiratory tract. Situations resulting in an increased production of any of these fluids will increase water needs. Though OWC are legendary in their ability to subsist on little or no water for long periods, they nevertheless need an adequate balance.

Only limited studies have been conducted on the water requirements of SACs [59]. It has been determined that the water content of the body of the llama is approximately 67%, compared with the goat at 60%. This varies with age and body fat content. In this study, it was found that goats and llamas have similar water requirements. In an indoor experiment in a neutral environment, the water turnover rate (water intake by drinking plus food and oxidative water) was 62.1 and 50 mL/$BW_{kg}^{0.82}$ in 24 hours in the llama and goat, respectively [59]. Consumed water amount was 42.5 and 40 mL/$BW_{kg}^{0.82}$ in the llama and goat, respectively. With a 40% reduction in food intake, water consumption of the llama decreased by 18%. When water was restricted, the llama was able to maintain food consumption. In this study, where llamas were on pasture, water turnover rates were double those of the indoor experiments (122.2 mL/$BW_{kg}^{0.82}$) [59]. This water intake is consistent with observed water turnover in llamas during the winter (116 mL/$BW_{kg}^{0.82}$) [60]. Free-ranging alpacas were found to have a higher water turnover (152 mL/$BW_{kg}^{0.82}$) [61]. Water turner was higher in spring (245 mL/$BW_{kg}^{0.82}$) and summer (260 mL/$BW_{kg}^{0.82}$) compared to winter [60]. Lactating llamas in pastoral systems had a water turnover of 396 mL/$BW_{kg}^{0.82}$ [60]. Lactating goat recommendations suggest adding 1.43 L water for each kilogram of milk produced. Required water in support of tissue gain has been evaluated in various small ruminant species. Suckling and weaned animals require 8–13 mL/g BW gain and 7–8 mL/g BW gain, respectively [56]. Incorporating these suggested water requirements for gain with maintenance, suckling animals gaining between 100 and 400 g/day have an estimated water requirement of 255–316 mL/kg $BW^{0.75}$, whereas weaned animals gaining 200–400 g/day would have a water requirement of 244–348 mL/kg $BW^{0.75}$ [56]. These gains for suckling neonates would be consistent with expected growth in SAC, but weaned animals would not have this high rate of gain. A simplified approach to water requirements is to relate directly to caloric requirements (1 mL of water per 1 kcal of required energy). This later value is useful in calculating water requirements for sick animals.

Basic water requirements for camelids based on these studies are indicated in Table 3.2. This correlates quite closely with water requirements of other ungulate species. Water requirements can be met by free water consumption, moisture in feed, and water produced by the oxidative processes associated with energy metabolism. Camelids on lush pasture may obtain a majority of the water requirement from feed. Dry feed necessitates greater water intake. Salt and other mineral content of forage may also affect the water requirement. Camelids will not break through ice to obtain water thus a waterer must be heated in cold climates. Lactation places a significant demand on water consumption. Clean water should be provided as unlimited free-choice consumption for SACs. Intake will be adjusted to body needs. If water is restricted below the requirement, feed consumption will decrease, lactation will slow down or cease, and, in the extreme, hyperthermia may result.

Elevated ambient temperatures result in extra demands for water in SACs because they lack the ability of OWC to withstand elevation of body temperature. OWC, particularly the dromedary, have evolved special physiologic mechanisms to deal with water deprivation. The camel can control the water content of feces and concentration of urine, which reduces excretion of vital fluids during heat stress. Contrary to popular belief, the camel does not store water any more than any other species, but because of its ability to conserve body fluids, it need not drink water for considerable periods.

The dromedary is able to tolerate extreme dehydration and has been known to survive the loss of body water equal to 40% of its body weight. In contrast, a human losing 12% of the body's weight in water would be close to death. The small oval erythrocyte of the camel continues to circulate despite increased blood viscosity. Even after severe dehydration, the camel is able to drink sufficient water at one session to make up the deficit. Immediate intake of this amount of water would cause severe osmotic problems in humans or other animals of temperate climates. In the camel, water is absorbed from the stomach and intestines slowly, allowing establishment of equilibrium. The erythrocytes are able to avoid osmotic problems by swelling to 240% of their initial volume without rupturing. SACs may share this ability with OWC. In other species, erythrocytes can swell only to 150%.

Camels can also endure a wide range of diurnal fluctuation of body temperature, from 36.5 to 42 °C. The body acts as a heat sink during the hot times of the day, thus

Table 3.2 Water requirements for llamas and alpacas.

Body weight		Body weight ($kg^{0.82}$)	Water turnover rate		Water requirements[c]	
			Controlled environment[a]	Pasture[b]	Moderate environment	Pasture-based intake
kg	lb		L or qt	L or qt	mL/kg/24 hours	mL/kg/24 hours
10	22	6.61	NA	1.0[d]	NA	NA
20	44	11.66	NA	2.0[d]	NA	NA
40	88	20.59	2.57	0.88	44	129
50	110	24.73	3.09	1.05	42	124
75	165	34.48	4.31	1.47	39	115
100	220	43.65	5.46	1.86	37	109
125	275	52.42	6.55	2.23	36	105
150	330	60.87	7.61	2.59	35	101
175	385	69.07	8.63	2.94	34	99
200	440	77.06	9.63	3.28	33	96
225	496	84.88	10.61	3.61	32	94
250	550	92.54	11.57	3.91	31	93

[a] Equation: 42.5 mL/$kg^{0.82}$ /24 hours consumed water; Actual consumption may be 1.5–2.5 times this figure, depending on weather, activity, and forage consumption.
[b] Equation: 125 mL/$kg^{0.82}$/24 hours.
[c] Expected intake for each respective environment is approximately 2 × the turnover rate.
[d] Based on milk consumption at 10% of body weight. Not calculated (NA).
Source: Rubsamen and Engelhardt [59].

conserving vital water that would otherwise be lost through evaporative cooling. The excess heat is dissipated by conduction and radiation during the cool desert nights.

Assessment

The water requirement defines the quantity of water to be provided and consumed daily by the animal depending upon physiologic status and environmental modifiers. A second consideration to address is the quality of water. Water being the universal solvent may contain a range of contaminants that may reduce acceptability to consumption or induce potential infectious or toxicologic disease conditions.

Initially, organoleptic water qualities such as color, odor, taste, and visual contaminants can provide some perspective on quality. Water should be transparent and essentially tasteless. Foul smells, especially "rotten egg" smell due to bacterial production of hydrogen sulfide gas are of concern. Always be cautious of directly examining water due to the potential of it being electrified.

Objective measures of water quality can include pH (suggested range of 6–9), total dissolved solids (TDS; Table 3.3), and mineral content. Elevated water iron (>0.3 mg/L) or manganese (>0.5 mg/L) can cause palatability issues. Any mineral dissolved in water is considered highly available and contributes to dietary mineral intake.

Table 3.3 Total dissolved solids or substances (TDS) as a measure of water quality for livestock.

TDS mg/L or ppm	Interpretation
<1000	Relatively low salinity; suitable for all classes of livestock
1000–2999	Satisfactory for all classes of livestock; may produce transient diarrhea in animals not accustomed to them but should not affect health or performance
3000–4999	Temporary water refusal and diarrhea may be seen when animals are introduced to them; may reduce productivity in lactating animals
5000–6999	Likely to reduce productivity in lactating animals; may reduce growth rates; may result in water refusal and diarrhea; avoid in lactating and pregnant animals; avoid, if possible
7000–10 000	Unfit for poultry or swine; considerable risk in all other species; animals may subsist on them in low-stress situations; avoid unless extreme circumstances
>10 000	High saline water; not recommended for use under any conditions; dangerous, avoid at all costs

Source: Modified from Pfost DL, et al.: Water quality for livestock drinking, Bull EQ 381, University of Missouri-Columbia Extension, 2001; and Bagley CV, et al.: Analysis of water quality for livestock, Bull AH-Beef-28, Utah State University Extension, 1997.

Excess dissolved mineral may induce interactions resulting in altered availability of other minerals. High soluble sulfates (>500 mg/L) may interfere with dietary copper or selenium availability. Defining guidelines for livestock water quality is not well documented and often, human guidelines are used. This may seem appropriate; however, with the unique fermentation system and its potential impact on mineral metabolism, human guidelines may not be appropriate relative to livestock health and performance. For example, soluble sulfates in water for human consumption can be as high as 2000 mg/L but this is too high for a ruminant animal. Bacterial contamination of water is another example, expected coliform count for potable water is zero whereas, livestock may tolerate much higher bacterial contamination without obvious disease. Livestock-based water quality guidelines (Table 3.4) have been generated by various agricultural university extension services [62–64].

Water may also be contaminated with a range of compounds such as nitrates, insecticides, herbicides, medications, and organic compounds. Ground water may become contaminated from various mining and industrial activities. Identifying such potential contaminants requires sophisticated testing methods at high expense. More challenging is attempting to determine the potential contaminants for testing. Many veterinary diagnostic laboratories or agricultural testing services will provide water-testing packages for the more common potential contaminants. Prior to collecting a sample for analysis, be sure to contact the laboratory for the specific procedure and containment vessel needed for the appropriate analyses.

Table 3.4 Water quality guidelines for livestock based on dissolved mineral content.

Mineral	Safe upper limit mg/L (ppm)[a]	Comment
Aluminum (Al)	5.0	Potential toxicant, Interfering agent
Arsenic (As)	0.2	Toxicant
Boron (B)	5.0	
Cadmium (Cd)	0.05	Toxicant
Chloride (Cl)	<300	High values of concern when not balanced with sodium
Chromium (Cr)	1.0	Toxicant
Cobalt (Co)	1.0	
Copper (Cu)	0.5	Potential toxicant
Iron (Fe)	0.3–2.0	Aesthetic issues, Interfering agent
Fluoride (F)	2.0	Toxicant
Lead (Pb)	0.05	Toxicant
Manganese (Mn)	<0.1	Aesthetic issues, Interfering agent
Mercury (Hg)	0.01	Toxicant
Nickel (Ni)	1.0	Toxicant
Nitrite (NO_2)	<33	Toxicant
Nitrite-N (NO_2-N)	<10	Toxicant
Selenium (Se)	0.05–0.1	Potential for intoxication
Sulfate (SO_4^{-2})	<300	Aesthetic issues, Interfering agent, Potential toxicant
Vanadium (V)	0.1	Toxicant
Zinc (Zn)	25.0	Potential toxicant, Interfering agent

[a] Milligrams per liter = mg/L; ppm = parts per million; both are equivalent measures.

Source: Modified from Pfost DL, et al.: Water quality for livestock drinking, *Bull EQ 381*, University of Missouri-Columbia Extension, 2001; and Bagley CV, et al.: Analysis of water quality for livestock, *Bull AH-Beef-28*, Utah State University Extension, 1997.

Energy

Beyond water, dietary energy is the most significant essential nutrient for animal health, production and well-being. Daily energy requirement is defined by the number of calories (or joules) needed to support metabolism of a given physiologic state. A calorie is defined as the energy necessary to raise 1 g of water by 1 °C at a pressure of 1 atm. One calorie is equivalent to 4.184 joules. A calorie is a small quantity; thus, animal requirements are described in mega- (× 1 000 000; Mcal) or kilocalories (× 1000; kcal). Energy can be derived from chemical bond energy stored in carbohydrate, lipid, or protein molecules. Lipids are the most concentrated form of dietary energy compared to carbohydrates and proteins.

The animal's energy requirement can be defined at different levels of digestion, namely digestible (DE), metabolizable (ME), or net (NE) energy requirements. Total energy within a feed is defined as gross energy; however, this is not descriptive enough relative to predicting animal performance. Historically, DE has been the metric for defining animal energy requirements based on feed digestibility studies. Metabolizable energy is a more precise estimate of true energy needs as it accounts for additional feed energy losses in urine and fermentation gases (predominately methane). Net energy is the absolute amount of energy required to support a specific biologic process and differs from ME by metabolic losses or what is termed the heat increment. Quantifying metabolic losses is mechanistically challenging requiring sophisticated calorimetric measurements, thus ME is commonly used to define animal energy requirements. Another measure of energy need is total digestible nutrients (TDN), which fits somewhere between

DE and ME values for feeds. Feed energy values are not directly measured but predicted using various regression models. Typical terms used in these models may include some combination of CP, acid detergent fiber (ADF), and NDF. Most regression models are based on data for cattle, which may not be accurate for camelids especially relative to low-quality forages. MBW is defined as body weight (BW) in kilograms raised to the 0.75 power, which accounts for differences in surface area to BW. Metabolic BW is used to standardize defined energy requirements for a species.

Maintenance Energy Requirement

A limited number of studies has specifically measured maintenance energy requirements of llamas [65, 66], alpacas [67], or dromedary camels [68–70]. Maintenance llama ME requirement was characterized as 61.2 and 84.5 kcal/kg $BW^{0.75}$; however, calculated fasting ME requirement was 59.3 and 52 kcal/kg $BW^{0.75}$, respectively. The similarity in fasting energy and discrepancy in maintenance requirement between these two studies might suggest underlying confounding issues of dietary adaptation, type of diet, metabolic adaption, level of intake, or any combination. Van Saun proposed an average of the two studies for a maintenance energy requirement of 72.85 kcal/kg $BW^{0.75}$, which was adopted by the NRC (2007) report [18, 56]. This averaged value is similar to the adjusted maintenance energy requirement of 74 kcal/kg $BW^{0.75}$ based on zero energy storage suggested by Carmean et al. [65]. Flores reported a maintenance ME requirement of 71 kcal/kg $BW^{0.75}$ for alpacas, suggesting a similar energy model for llamas and alpacas [67]. Another study using alpacas in addressing energy intake effects on fiber production suggested a maintenance ME requirement of 66 kcal/kg $BW^{0.75}$, somewhat between the original llama study and averaged model [71]. A second New Zealand study again addressing dietary energy intake on fiber production indicated a dietary ME supply of 70.5 kcal/kg $BW^{0.75}$ resulted in BW loss (−69.1 g/day) whereas supplying 105 kcal/kg $BW^{0.75}$ resulted in a daily gain of 70.3 g/day [72]. Neither of these two studies was designed to test ME requirement was confounded by the crossover design, intake level, and differences in dietary composition (hay vs. hay and grain). Until further research is reported and impacts of dietary composition, intake, and adaptation on energy requirements are elucidated the averaged energy model of 72.85 kcal/kg $BW^{0.75}$ seems appropriate for determining absolute ME requirements for llamas and alpacas and was used to determine values in the requirement tables [56].

Maintenance energy requirement for dromedary camels determined by colorimetric and energy balance studies has been reported [68–70]. Reported maintenance ME models were 75 kcal/kg $BW^{0.75}$ [70, 73], 89.3 kcal/kg $BW^{0.75}$ [68], and 72.6 kcal/kg $BW^{0.75}$ [69]. These outcomes are reasonably consistent and similar in magnitude to SAC energy models. Most camel studies were performed using similar methodology, but effects of dietary composition and nutritional plane will account for some variability. If these models are averaged like the SAC energy models, a suggested maintenance ME model would be 78 kcal/kg $BW^{0.75}$, just slightly higher than the 75 kcal/kg $BW^{0.75}$ often used for OWC. This higher value was used to generate daily ME requirements for OWC in the dietary requirement tables, which is consistent with recent energy recommendations [74].

A maintenance requirement is assuming a net BW change of zero and minimal activity. Environment factors, foraging activity, and terrain will result in greater energy expenditure due to muscular activity or altered metabolic efficiencies. Data quantifying additional energy needs for foraging activity have not been reported for camelids. Generic increases in maintenance energy of 25, 50, 75, and 125% have been previously suggested for camelids based on older goat recommendations [18]. These graded increases account for greater distances traveled, more rugged terrain covered, and forage quality and availability during grazing. One study using double-labeled water determined total daily energy expenditure (ME) in male alpacas grazing in bofedal pastures of 3.4 Mcal/d, which is 2.5 times the predicted maintenance ME requirement [61]. Though field maintenance requirement data must be interpreted with caution, these data would suggest a greater energy need than recommended for traditional pastoral grazing systems.

Energy Supporting Other Physiologic States

Few studies have been performed on camelids to address requirements for physiologic stages of growth, pregnancy, and lactation. Two studies have provided suggested dietary nutrient content recommendations for SAC in different physiologic states of growth, pregnancy, and lactation [18, 57]. A factorial approach to modeling requirements within each physiologic state was provided in the NRC (2007) based on the models of Van Saun, 2006 [18, 56].

Pregnancy energy requirement will increase in concert with the exponential fetal growth curve where 70% of growth occurs in the last three months of gestation [75]. Gestational energy supports not only fetal growth, but placental growth and metabolism. Gestational energy requirement is typically a function of birth weight. Fowler, recommended a 90% increase to maintenance energy needs by adding 93 kcal DE/kg $BW^{0.75}$ (76.26 kcal ME/kg $BW^{0.75}$) to address late pregnancy needs in SACs [76]. A model accounting for pregnancy energy for the last three months, inclusive, was extrapolated from sheep data by proportionally decreasing the multiplying coefficient to account for lower SAC maintenance requirement. Prediction equations of 138.8 or 160.9 kcal ME/kg $BW^{0.75}$ estimate total energy needs for the last three months of gestation, depending on the maintenance energy value used [18].

The latter model is not different from the original model proposed by Fowler [76]. A second modeling process using sheep data generated equations based on expected birth weight (8, 10, or 12% of maternal BW) for gestation months 8, 9, and 10 was generated from sheep data adjusting for gestation day and birth weight [18, 56]. Using these models, estimated energy requirement at gestation months 8, 9, and 10 averaged 1.27, 1.55 and 1.92 times maintenance, respectively, which was consistent with observations in other species. Wardeh suggested an increase of 1.2, 1.2 and 1.5 times maintenance for 9th, 10th, and 11th and greater months of gestation for OWC [58].

Lactation energy requirement is a function of milk production volume and its energy composition; a function of lactose, fat, and protein content. This energy amount is added to maintenance to determine the total daily energy requirement. Milk production abilities of OWC is well documented given their use in dairy production systems with production records like that of dairy cattle. Milk yield and composition data for SAC are limited and essentially no full lactation information. Both SAC (946.2 kcal ME/kg) and OWC (1200 kcal ME/kg) lactation models use a singular milk energy composition value to predict additional lactation energy needs [18, 58]. Milk energy differences are due to greater fat in OWC compared to SAC milk. Recent studies with llamas monitoring milk yield and composition over longer portions of the lactation have indicated a higher milk energy value (1296 ± 7.4 kcal ME/kg of milk) due to greater fat content, consistent with OWC data [77–79]. Alpaca milk composition and energy content are not significantly different from those of llamas [77, 78, 80]. Lactation curves have been generated for OWC [81] and SAC [82] using Wood's gamma function. Using data from two isotope studies characterizing llama milk production, averaged milk yield at 3–4, 10–11, and 18–19 weeks of lactation was determined to be 64.2, 54.5, and 48.9 g/kg $BW^{0.75}$, respectively [83, 84]. Llama lactation curves were similar in shape to cattle with a rapid rise to peak milk in contrast to a slower rise to peak in OWC curves. Parity and most likely nutrition influenced OWC lactation curve shape [81].

Energy to support growth will be based on the rate of gain (g/day) and composition of gain over-and-above maintenance requirements. Data are lacking on changes in tissue composition over the period of growth in SAC; however, early growth is primarily protein accretion and post-pubertal growth has greater fat accretion. Recommended daily energy and protein requirements for SAC (Table 3.5) and OWC (Table 3.6) in different physiologic states are provided.

Table 3.5 Nutrient requirements for South American camelids.[a]

Body weight		Dry matter intake		Metabolizable energy (ME)		TDN	Crude protein	Ca	P	Vitamin A		Vitamin E
kg	lb	kg/d	% BW	Mcal/d	MJ/d	kg/d	g/d	g/d	g/d	RE/d	IU/d	IU/d
Maintenance												
40	88	0.60–0.72	1.51–1.79	1.16	4.85	0.32	55.7	1.2	1.0	1256	4145	212
60	132	0.82–0.97	1.37–1.62	1.57	6.57	0.44	75.5	1.8	1.6	1884	6274	318
80	176	1.02–1.20	1.27–1.50	1.95	8.16	0.54	93.6	2.4	2.1	2512	8365	424
100	220	1.21–1.42	1.21–1.42	2.30	9.62	0.64	110.7	3.0	2.6	3140	10 456	530
120	275	1.38–1.63	1.15–1.36	2.64	11.05	0.73	126.9	3.6	3.1	3768	12 547	636
140	308	1.55–1.83	1.11–1.31	2.97	12.43	0.82	142.5	4.2	3.6	4396	14 639	742
160	352	1.71–2.02	1.07–1.27	3.28	13.72	0.91	157.5	4.8	4.2	5024	16 730	848
Pregnancyb, 8–9th month												
60	132	1.03–1.14	1.72–1.90	1.93	8.08	0.53	96	4.2	2.5	2730	9091	336
80	176	1.28–1.42	1.60–1.77	2.44	10.21	0.67	119	5.6	3.3	3640	12 121	448
100	220	1.52–1.68	1.52–1.62	2.92	12.22	0.81	141	7	4.1	4550	15 152	560
120	275	1.74–1.92	1.45–1.54	3.39	14.18	0.94	161	8.4	4.9	5460	18 182	672
140	308	1.95–2.16	1.40–1.54	3.85	16.11	1.06	181	9.8	5.7	6370	21 212	784
160	352	2.16–2.38	1.35–1.49	4.29	17.95	1.19	200	11.2	6.6	7280	24 242	896
Pregnancyb, 9–10th month												
60	132	1.08–1.25	1.80–2.08	2.32	9.71	0.64	117	4.2	2.5	2730	9091	336
80	176	1.34–1.55	1.67–1.94	2.96	12.38	0.82	146	5.6	3.3	3640	12 121	448

Table 3.5 (Continued)

Body weight		Dry matter intake		Metabolizable energy (ME)		TDN	Crude protein	Ca	P	Vitamin A		Vitamin E
kg	lb	kg/d	% BW	Mcal/d	MJ/d	kg/d	g/d	g/d	g/d	RE/d	IU/d	IU/d
100	220	1.58–1.83	1.58–1.83	3.58	14.98	0.99	172	7	4.1	4550	15 152	560
120	275	1.81–2.10	1.51–1.75	4.18	17.49	1.16	197	8.4	4.9	5460	18 182	672
140	308	2.04–2.36	1.45–1.69	4.77	19.96	1.32	221	9.8	5.7	6370	21 212	784
160	352	2.25–2.61	1.41–1.63	5.34	22.34	1.48	245	11.2	6.6	7280	24 242	896
Pregnancy[b], 10th month to parturition												
60	132	1.08–1.29	1.80–2.16	2.88	12.05	0.80	145	4.2	2.5	2730	9091	336
80	176	1.34–1.60	1.67–2.01	3.66	15.31	1.01	180	5.6	3.3	3640	12 121	448
100	220	1.58–1.90	1.58–1.90	4.42	18.49	1.22	213	7	4.1	4550	15 152	560
120	275	1.81–2.18	1.51–1.81	5.17	21.63	1.43	244	8.4	4.9	5460	18 182	672
140	308	2.04–2.44	1.45–1.74	5.90	24.69	1.63	274	9.8	5.7	6370	21 212	784
160	352	2.25–2.70	1.41–1.69	6.62	27.70	1.83	303	11.2	6.6	7280	24 242	896
Lactation[c]												
60	132	1.03 – 1.40	1.72–2.34	2.22	9.29	0.61	113.5	3.3	2.5	3210	10 689	280
80	176	1.28–1.74	1.60–2.17	2.60	10.88	0.72	131.6	3.9	3.0	4280	14 252	448
100	220	152–2.37	1.52–2.37	3.60	15.06	1.0	185.7	6.0	4.3	5350	17 816	560
120	275	1.74–2.72	1.45–2.27	3.94	16.48	1.09	201.9	6.6	4.8	6420	21 379	672
140	308	1.95–3.05	1.40–2.18	4.91	20.54	1.36	255.5	8.7	6.2	7490	24 942	784
160	352	2.16–3.37	1.35–2.11	5.22	21.84	1.45	270.5	9.3	6.8	8560	28 505	896
Growth – Alpacas												
BW kg	ADG g/d	kg/d	% BW	Mcal/d	MJ/d	kg/d	g/d	g/d	g/d	RE/d	IU/d	IU/d
10	150	0.42–0.48	4.22–4.78	0.89	3.72	0.25	62	1.5	0.8	1000	3330	100
	100	0.39–0.42	3.94–4.22	0.73	3.05	0.20	48	1.5	0.8	1000	3330	100
20	150	0.71–0.80	3.55–4.02	1.52	6.36	0.42	76	2.9	1.5	2000	6660	200
	100	0.61–0.71	3.07–3.55	1.24	5.19	0.34	62	2.9	1.5	2000	6660	200
	90	0.59–0.68	2.93–3.40	1.19	4.98	0.33	59	2.9	1.5	2000	6660	200
30	125	0.83–0.94	2.78–3.12	1.62	6.78	0.45	80	4.4	2.3	3000	9990	300
	90	0.71–0.83	2.35–2.78	1.43	5.98	0.40	70	4.4	2.3	3000	9990	300
40	100	0.84–1.03	2.11–2.58	1.71	7.15	0.47	84	5.8	3.0	4000	13 320	400
	70	0.76–0.95	1.91–2.39	1.55	6.49	0.43	76	5.8	3.0	4000	13 320	400
50	80	0.85–1.03	1.69–2.07	1.81	7.57	0.50	89	3.0	2.1	5000	16 650	500
	40	0.75–0.94	1.50–1.88	1.59	6.65	0.44	77	3.0	2.1	5000	16 650	500
60	50	0.86–1.08	1.44–1.80	1.85	7.74	0.51	90	3.6	2.5	6000	19 980	600
	20	0.86–0.97	1.44–1.62	1.68	7.03	0.46	81	3.6	2.5	6000	19 980	600
70	20	0.97–1.02	1.38–1.45	1.87	7.82	0.52	90	4.2	2.9	7000	23 310	700
	10	0.92–0.97	1.31–1.38	1.82	7.61	0.50	88	4.2	2.9	7000	23 310	700
Growth – Llamas												
20	450	0.76–0.85	3.78–4.26	2.18	9.12	0.60	161	2.9	1.5	2000	6660	200
	350	0.71–0.80	3.55–4.02	1.85	7.74	0.51	133	2.9	1.5	2000	6660	200
	250	0.66–0.71	3.31–3.55	1.52	6.36	0.42	104	2.9	1.5	2000	6660	200

(*Continued*)

Table 3.5 (Continued)

Body weight		Dry matter intake		Metabolizable energy (ME)		TDN	Crude protein	Ca	P	Vitamin A		Vitamin E
kg	lb	kg/d	% BW	Mcal/d	MJ/d	kg/d	g/d	g/d	g/d	RE/d	IU/d	IU/d
30	400	1.03–1.15	3.42–3.85	3.14	13.14	0.87	158	4.4	2.3	3000	9990	300
	325	0.96–1.09	3.20–3.63	2.73	11.42	0.75	137	4.4	2.3	3000	9990	300
	250	0.90–1.03	2.99–3.42	2.31	9.67	0.64	116	4.4	2.3	3000	9990	300
40	300	1.19–1.30	2.98–3.26	2.81	11.76	0.78	141	5.8	3.0	4000	13 320	400
	150	0.99–1.15	2.47–2.86	1.99	8.33	0.55	98	5.8	3.0	4000	13 320	400
50	250	1.32–1.41	2.63–2.82	2.75	11.51	0.76	137	7.3	3.8	5000	16 650	500
	125	1.13–1.22	2.26–2.44	2.06	8.62	0.57	101	7.3	3.8	5000	16 650	500
60	225	1.40–1.55	2.34–2.59	2.81	11.76	0.78	139	8.7	4.5	6000	19 980	600
	100	1.25–1.40	2.08–2.34	2.12	8.87	0.59	104	8.7	4.5	6000	19 980	600
70	200	1.57–1.74	2.25–2.49	2.87	12.01	0.79	142	10.2	5.3	7000	23 310	700
	90	1.21–1.50	1.73–2.14	2.26	9.46	0.63	110	10.2	5.3	7000	23 310	700
80	175	1.55–1.74	1.94–2.17	2.91	12.18	0.81	143	4.8	3.4	8000	26 640	800
	80	1.34–1.47	1.67–1.84	2.39	10.00	0.66	116	4.8	3.4	8000	26 640	800
90	150	1.61–1.75	1.79–195	2.96	12.38	0.82	145	5.4	3.8	9000	29 970	900
	75	1.31–1.61	1.46–1.79	2.54	10.63	0.70	124	5.4	3.8	9000	29 970	900
100	125	1.58–1.83	1.58–1.83	2.99	12.51	0.83	146	6.0	4.2	10 000	33 300	1000
	75	1.42–1.68	1.42–1.68	2.72	11.38	0.75	132	6.0	4.2	10 000	33 300	1000
110	100	1.63–1.77	1.48–1.61	3.03	12.68	0.84	147	6.6	4.6	11 000	36 630	1100
	65	1.53–1.63	1.39–1.48	2.83	11.84	0.78	137	6.6	4.6	11 000	36 630	1100
120	90	1.56–1.81	1.30–1.51	3.14	13.14	0.87	152	7.2	5.0	12 000	39 960	1200
	55	1.45–1.52	1.21–1.27	2.94	12.30	0.81	143	7.2	5.0	12 000	39 960	1200
130	90	1.62–1.73	1.24–1.33	3.30	13.81	0.91	160	7.8	5.5	13 000	43 290	1300
	50	1.46–1.62	1.13–1.24	3.08	12.89	0.85	149	7.8	5.5	13 000	43 290	1300
140	75	1.63–1.83	1.16–1.31	3.38	14.14	0.93	164	8.4	5.9	14 000	46 620	1400
	30	1.55–1.71	1.10–1.22	3.13	13.10	0.87	151	8.4	5.9	14 000	46 620	1400

[a] Abbreviations: TDN = total digestible nutrients; Ca = calcium; P = phosphorus; RE = retinol equivalents; IU = international units; ADG = average daily gain.
[b] Assumes cria birth weight is 10% of maternal mature weight.
[c] Assumes 0.5, 1.0 and 1.5 kg/d milk production for body weights 60 and 80 kg, 100 and 120 kg, and 140 and 160 kg, respectively.
Source: Modified from NRC [56], Van Saun [90].

Table 3.6 Nutrient requirements for dromedary camels.[a]

Body weight		Metabolic weight	DM intake		Metabolizable energy (ME)		Digestible protein	Crude protein	Ca	P	Vitamin A
kg	lbs	$W_{kg}^{0.75}$	kg/d	% BW	Mcal/d	MJ/d	g/d	g/d	g/d	g/d	kIU/d
Maintenance[b]											
200	440	53.2	2.50	1.48	4.15	17.36	121.8	191.9	8	7	9
250	550	62.9	2.96	1.18	4.90	20.52	144.0	226.9	10	9	11
300	660	72.1	3.39	1.11	5.62	23.52	165.1	260.1	12	10	13
350	772	80.9	3.80	1.08	6.31	26.41	185.3	292.0	14	11	15

Table 3.6 (Continued)

Body weight		Metabolic weight	DM intake		Metabolizable energy (ME)		Digestible protein	Crude protein	Ca	P	Vitamin A
kg	lbs	$W_{kg}^{0.75}$	kg/d	% BW	Mcal/d	MJ/d	g/d	g/d	g/d	g/d	kIU/d
400	882	89.4	4.20	1.05	6.98	29.19	204.8	322.8	17	13	17
450	992	97.7	4.59	1.02	7.62	31.89	223.7	352.6	18	14	19
500	1102	105.7	4.97	0.99	8.25	34.51	242.1	381.6	20	15	21
550	1216	112.6	5.34	0.97	8.86	37.06	260.1	409.8	21	16	23
600	1323	121.2	5.70	0.95	9.46	39.56	277.6	437.5	22	17	26
Pregnancy, 9th and 10th months[c]											
300	660	72.1	4.29	1.43	6.75	28.23	175.4	276.4	16	14	25
350	772	80.9	4.81	1.37	7.57	31.69	196.9	310.3	21	16	27
400	882	89.4	5.32	1.33	8.37	35.03	217.7	343.0	23	18	30
450	992	97.7	5.80	1.29	9.15	38.26	237.8	374.7	26	20	34
500	1102	105.7	6.29	1.26	9.90	41.41	257.3	405.5	29	22	38
550	1216	112.6	6.75	1.23	10.63	44.48	276.4	435.5	31	24	42
600	1323	121.5	7.20	1.20	11.35	47.48	295.0	464.9	34	26	46
Pregnancy, 11th month[c]											
300	660	72.1	5.36	1.79	8.43	35.29	219.3	345.5	26	20	34
350	772	80.9	6.10	1.74	9.47	39.61	246.2	387.9	29	22	38
400	882	89.4	6.64	1.66	10.46	43.78	272.1	428.8	31	24	42
450	992	97.7	7.26	1.61	11.43	47.83	297.2	468.3	34	26	46
500	1102	105.7	7.86	1.57	12.37	51.76	321.7	506.9	36	28	50
550	1216	112.6	8.44	1.53	13.29	55.60	345.5	544.4	39	30	53
600	1323	121.5	9	1.50	14.18	59.35	368.8	581.1	42	32	57
Lactation[d]											
300	660	72.1	6.55	2.2	14.40	60.25	470	740.6	26	20	13
350	772	80.9	7.00	2.0	15.43	64.56	493	776.9	28	21	15
400	882	89.4	7.56	1.9	16.64	69.62	516	813.1	31	23	17
450	992	97.7	7.90	1.8	17.38	72.72	539	849.4	32	24	19
500	1102	105.7	8.33	1.76	18.32	76.65	560	882.4	34	25	21
550	1216	113.6	8.74	1.59	19.23	80.46	582	917.1	35	26	23
600	1323	121.2	9.15	1.5	20.15	84.31	602	948.6	36	27	26
Growth											
kg	lbs	Gain, g/d	kg/d	% BW	Mcal/d	MJ/d	g/d	g/d	g/d	g/d	IU/d
100	220	750	2.96	2.96%	8.29	34.69	304	479.0	20	11	6000
		500	2.75	2.75%	6.62	27.7	249	392.4	15	9	6000
		250	2.25	2.25%	4.96	20.75	195	307.3	10	7	6000
150	330	750	3.93	2.62%	10.23	42.8	353	556.3	21	13	9000
		500	3.46	2.31%	8.38	35.06	298	469.6	16	10	9000
		250	2.90	1.93%	6.38	26.69	244	384.5	12	9	8000
200	440	750	4.64	2.32%	12.08	50.54	394	620.9	21	15	13000
		500	4.12	2.06%	9.90	41.42	340	535.8	16	12	12000

(*Continued*)

Table 3.6 (Continued)

Body weight		Metabolic weight	DM intake		Metabolizable energy (ME)		Digestible protein	Crude protein	Ca	P	Vitamin A
kg	lbs	$W_{kg}^{0.75}$	kg/d	% BW	Mcal/d	MJ/d	g/d	g/d	g/d	g/d	kIU/d
		250	3.50	1.75%	7.71	32.26	285	449.1	11	9	11 000
250	550	750	5.78	2.31%	13.86	57.99	427	672.9	22	17	14 000
		500	5.19	2.08%	11.42	47.78	373	587.8	16	14	13 000
		250	4.49	1.80%	8.98	37.57	318	501.1	12	10	12 000
300	660	750	6.50	2.17%	15.59	65.23	454	715.4	23	18	15 000
		500	5.86	1.95%	12.89	53.93	400	630.3	19	14	14 000
		250	5.10	1.70%	10.19	42.63	345	543.6	15	11	13 000
350	770	750	6.85	1.96%	17.82	74.56	474	746.9	25	18	17 000
		500	6.50	1.86%	14.32	59.91	420	661.8	20	16	16 000
		250	5.68	1.62%	11.37	47.57	365	575.2	16	14	15 000
400	880	750	8.61	2.15%	18.94	79.24	488	769.0	26	21	18 000
		500	7.15	1.79%	15.73	65.81	433	682.3	21	18	17 000
		250	6.26	1.57%	12.51	52.34	378	595.7	16	15	15 000

[a] Modified from: Wardeh [58] and Al Jassim [74].
[b] Energy requirements may be increased by 25–40%, respectively, when camels work two to four hours/d.
[c] Pregnancy requirements are increased by 20 and 50% for 9-10th month and 11th month, respectively.
[d] Assuming 5 kg/d milk production.

Protein

Animals truly do not have a requirement for protein but for the building blocks, i.e. amino acids, that comprise proteins. There are over 200 chemical compounds fitting the definition of an amino acid; however, only 21 amino acids are biologically important in protein metabolism across all species. Amino acids are identified by the length and composition of the carbon chain beyond the amino ($-NH_2$) and carboxyl (-COOH) groups characterizing them into polar basic, polar acidic, polar neutral, and non-polar neutral. Biologically, amino acids are classified as essential, meaning they are required in the diet, and non-essential. Amino acids are unique from other nutritional compounds resultant of their nitrogen content. Biologically important proteins contain on average 16% nitrogen; hence, protein analysis (i.e. CP) is based solely on nitrogen content and multiplied by a factor of 6.25 (100/16 = 6.25) to determine protein content of feeds. Protein cannot be determined directly due to the vast diversity in proteinaceous compounds.

Protein nutrition of camelids is further complicated, similar to ruminants, in that dietary protein can provide nitrogen sources to forestomach microbial populations, termed degradable protein, or pass unmodified to C3 where it can be digested, or not, and pass into the duodenum for further digestion and absorption of amino acids [20]. Forestomach microbial populations can utilize nitrogen in the forms of ammonia, amino acids, or peptides (see Table 3.1). Ruminant nutrition terminology to describe dietary protein fractions relative to meeting microbial or host animal needs can be applied to camelids. CP is the metric used to describe dietary protein content or animal requirement; however, this measure is not useful in partitioning nitrogen compounds in support of forestomach microbial populations and the host animal. CP can be partitioned into various components chemically to describe potential utilization (Figure 3.3). From this partitioning, dietary protein can be classified as degradable (DIP) or undegradable (UIP) intake protein, depending upon the proportion available to support forestomach microbial growth. Percent degradable or undegradable for any protein source will total 100% of dietary CP. Protein that is soluble in rumen or C1 fluids is considered 100% degradable whereas, insoluble protein may or may not be degradable depending upon rate of passage through the fermentation system. Protein associated with the ADF residue is considered unavailable to microbes or animal. This protein fraction is termed bound or heat-damaged protein. There are few data characterizing dietary protein fractions relative to C1 degradability in camelids. It would seem reasonable to use laboratory derived feed protein degradability values for cattle in addressing camelid diets realizing

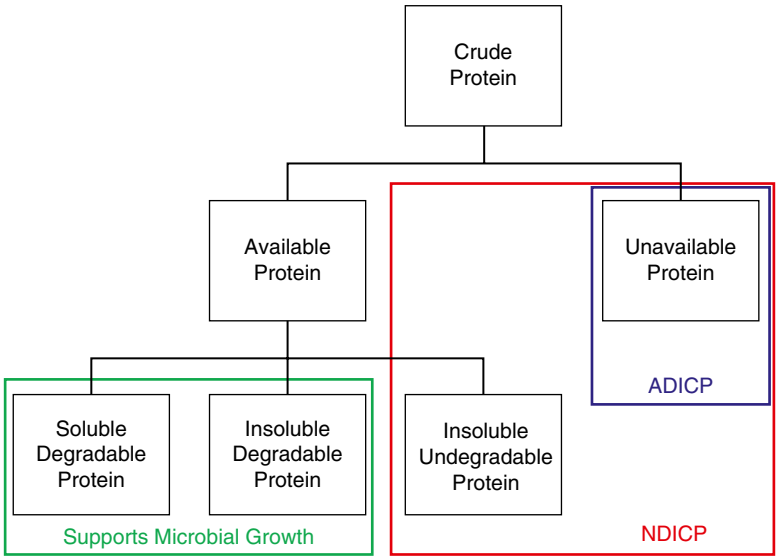

Figure 3.3 Fractionation of dietary crude protein into chemical components addressing potential degradability in the rumen or C1/2 or available for enzymatic digestion. Abbreviations: ADICP = acid detergent insoluble crude protein (bound or unavailable protein); NDICP = neutral detergent insoluble crude protein (a measure of bypass or escape protein).

that degradability may range from 5 to 10% higher in some circumstances due to the longer C1 retention time observed in camelids. At present, there are no established recommendations for dietary CP partitioning in camelid diets.

Maintenance Protein Requirement

Only one study has defined a CP requirement for alpacas [85]. This thesis project from San Marcos University in Lima, Peru defined a digestible protein requirement of 2.38 g/kg $BW^{0.75}$ and using conversion factors for protein digestibility and availability would result in a CP requirement of 3.5 g/kg $BW^{0.75}$. Other recent studies have measured alpaca and llama CP requirements with variable outcomes and attributed some study differences to metabolic efficiency at higher altitudes [86, 87]. Using a Lucas test analysis of these study results, reported nitrogen retention intake was regressed onto nitrogen intake and the intercept value of 0.57 g N/kg $BW^{0.75}$ considered the maintenance requirement. Converting this value to CP results in a suggested maintenance of 3.6 g/kg $BW^{0.75}$, essentially equivalent to the original work.

Only one study has estimated protein requirement of OWC [68]. Using a factorial approach, protein requirement to maintain BW was 349.1 mg digestible N/kg $BW^{0.75}$, which was reported at 2.23 g digestible protein/kg $BW^{0.75}$. Using the same protein digestibility and available coefficients for SAC data, a camel maintenance CP requirement is defined as 3.28 g/kg $BW^{0.75}$. A slightly higher value, 3.97 g CP/kg $BW^{0.75}$ was suggested for defining OWC maintenance CP requirement accounting for an allowance to address dietary protein quality variability [58].

Protein to Support Other Physiologic States

Limited studies have defined specific protein requirements in support of other states beyond maintenance. Gestational protein requirements are assessed based on protein deposition into the fetus and subsequent birth weight. Protein loss in fetal fluids and deposited in the placenta account for the low efficiency of utilization of amino acids [88]. In SAC, Hospinal suggested a 68% increase in the maintenance protein requirement (5.88 g CP/kg $BW^{0.75}$) to support the last three months of gestation [89]. Using a defined protein-to-energy ratio (48 g CP/Mcal ME) and the energy models predicting gestational requirements, additional CP for the 8th, 9th, and 10th months of gestation were 0.95, 1.94, and 3.23 g CP/kg $BW^{0.75}$, respectively [18]. A similar approach was used for OWC; however, the cattle ratio of protein-to-energy (38 g CP/Mcal ME) was suggested [58].

Lactation protein requirements are based on the needed amino acids to support milk true protein production by the mammary gland. Milk can contain a proportion of urea nitrogen that does account for amino acid needs. Recent studies have described milk protein content over a lactation in SAC [77–80]. Using cattle lactation efficiency coefficient, a CP requirement of 75.4 g/kg milk is suggested [90]. A digestible protein requirement of 55 g/kg milk was suggested for OWC [58]. Assuming similar digestibility and biologic values for conversion, this would be equivalent to 83.3 g CP/kg milk.

Growth protein requirements will change with changes in deposited tissue composition. Early growth contains more protein while post-pubertal growth has increasing fat content. A growth CP requirement of 0.284 g/g gain was based on data from goats [18]. A measured value of 0.3 g CP/g gain was determined in growing llama crias [84]. No data estimating OWC growth is available and recommendations are based on a cattle model [58]. In this model, the slope was 0.218 g digestible protein per gram of gain. This value would be similar to the relationship for SAC on a CP basis. The SAC model for protein needs during growth was used to generate growth protein requirements of OWC.

Macrominerals

Minerals are accounted for in the ash component of feed analysis. The NRC (2007) publication did not provide SAC-specific mineral recommendations due to a lack of controlled feeding trials. Data for sheep were used to generate the suggested dietary calcium (Ca) and phosphorus (P) amounts shown in requirement tables in the publication [56]. In generating a factorial model system for SAC nutrient requirements, Van Saun, calculated mineral requirements on a BW and metabolic BW basis for beef cattle, sheep, and goats [18]. These calculations were similar across the three species and thus, extrapolated for application to SAC assuming similar mineral metabolism and adjusted for intake difference. A recent study from Peru completed a controlled feeding trial manipulating dietary P in growing alpacas and impact on puberty and reproduction [91]. In calculating dietary P intake relative to alpaca performance, study results would be consistent with suggested dietary P models proposed. Similarly, there are no published studies defining mineral requirements of OWC; however, suggested amounts of Ca and P were provided by Wardeh [58]. Concerns relative to macrominerals are more often related to dietary imbalances between Ca and P resulting in metabolic bone disease of some form [92].

The previous edition of this text provided a detailed description of micromineral functions and disease conditions common across species [93]. Though there are limited published case reports of micromineral disease conditions in camelids, the underlying assumption is for similar metabolic and biologic macromineral roles and deficiency or toxicity disease conditions. The only notable difference is a suggested greater requirement for sodium and chloride for OWC [58]. The reader is directed to the third edition of this text [93] for the detailed description of mineral functions and disease or comprehensive mineral texts [94–97]. Macromineral functions and disease conditions are summarized (Table 3.7). Suggested dietary

Table 3.7 Summarized macromineral functions and disease conditions.

		Disease condition		
Mineral	**Major functions**	**Deficiency**	**Toxicity**	**Interrelationships**
Calcium (Ca)	Bone and teeth formation; Blood coagulation; Muscle contraction; Milk production; Nerve conduction; Cell permeability	Rickets (young) or Osteomalacia (adults); Lactational eclampsia; Muscle weakness; Decreased bone cortex density	Soft tissue mineralization with hypercalcemia, cardiac blockade	Highly regulated with Parathormone, Calcitonin and Vitamin D; Excess P, Mg, and fat decrease absorption
Phosphorus (P)	Bone and teeth formation; Blood buffer; Acid–base balance; High-energy bonds; Cellular metabolism	Rickets (young) or Osteomalacia (adults); Pica; Poor growth, reproduction	Excess P may cause urinary calculi, nutritional secondary hyperparathyroidism	Vitamin D required for renal reabsorption and bone deposition; Excess dietary Ca, Mg, Al reduce availability;
Magnesium (Mg)	Enzyme activator; Nerve conduction; ATP activation; Bone formation	Hyperexcitability; Tetanic convulsions; Grass tetany		Availability decreased by K, Ca, Fat
Potassium (K)	Osmotic pressure; Acid–base balance; Major intracellular cation	Hypokalemia; Muscle weakness; Lethargic; Diarrhea; Pica	Cardiac toxicity at higher intakes	Excess reduces Mg absorption;
Sodium (Na)	Major extracellular cation; Osmotic pressure; Cell permeability; Ion potentials; Acid–base balance	Reduced growth; Pica; Polydipsia/Polyuria	Salt toxicity >8%; Water availability dependent; Nervous condition with staggering gait, blindness, hypertension	
Chlorine (Cl)	Major extracellular anion; Osmotic pressure; Acid–base balance; Hydrochloric acid secretion	Reduced growth; Pica	Metabolic acidosis	Typically follows Na in extracellular fluids
Sulfur (S)	Sulfur-containing amino acids; Biotin; Thiamin; Hoof and fleece growth	Reduced growth rate; poor wool growth	Non thiamine-responsive Polioencephalomalacia;	Interferes with Cu availability in conjunction with Mo

Source: from Van Saun [157].

Table 3.8 Recommended macromineral requirements for South American camelids.

Mineral	Averaged requirement	Extrapolated requirement		
		Daily Intake[a]	% DM of Diet[b]	Feeding Group[c]
Calcium	30 mg/kg BW	1.8–4.8 g/d	0.2–0.24	M
	145 mg/kg BW		0.53–0.73	G (1–12 mo)
	60 mg/kg BW		0.30–0.48	G (12–36 mo)
	0.40 g/kg Fetus	2.4–6.4 g/d	0.45–0.56	P
	3.0 g/kg Milk	2.3–7.5 g/d	0.45–0.62	L
Phosphorus	26 mg/kg BW	1.6–4.2 g/d	0.17–0.21	M
	75 mg/kg BW		0.27–0.38	G (1–15 mo)
	42 mg/kg BW		0.21–0.28	G (15–36 mo)
	0.15 g/kg Fetus	0.9–2.4 g/d	0.28–0.33	P
	1.7 g/kg Milk	1.3–4.25 g/d	0.32–0.45	L
Magnesium	19.4 mg/kg BW	1.2–3.1 g/d	0.13–0.16	M, G
	27.3 mg/kg BW	1.6–4.4 g/d	0.18–0.22	P, L
Potassium	92.6 mg/kg BW	5.6–14.8 g/d	0.6–0.7	M, G, P
	121 mg/kg BW	7.3–19.4 g/d	0.8–0.96	L
Sodium	10.5 mg/kg BW	0.6–1.7 g/d	0.07–0.08	M, G, P
	17.5 mg/kg BW	1.1–2.8 g/d	0.12–0.14	L
Sulfur	28.5 mg/kg BW	1.7–4.6 g/d	0.19–0.23	M, G, P, L

[a] Estimated daily requirement based on a range of adult body weights from 60 to 160 kg. Calcium and phosphorus pregnancy and lactation intake requirements based on a range of 6–16 kg fetal weight and 0.75– 2.5 kg milk production, respectively. Values are in addition to maintenance for total requirement.
[b] Dietary concentration on dry matter (DM) basis for total requirement. Nutrient density calculations based on an assumed range of DM intake between 1.25 and 1.7% of body weight (maintenance and pregnancy) and 2.0 and 2.75% of body weight (lactation).
[c] Physiologic states of maintenance (M), growth (G), lactation (L), and pregnancy (P) for which the requirement is defined.
Source: Modified from Van Saun [18].

macromineral content, based on SAC requirement models (Table 3.8) [18].

Microminerals

The microminerals, cobalt (Co), copper (Cu), iodine (I), iron (Fe), manganese (Mn), selenium (Se), and zinc (Zn) are required in milligrams per day, yet there is more interest in defining micromineral status and disease risk compared to macrominerals. Micromineral nutrition is much more geographically based compared to macromineral nutrition due to differences in soil conditions and weather within and between regions, states, or countries affecting forage micromineral content [98]. Additionally, there are numerous interactions among microminerals and forestomach alterations resulting in greater potential risk for disease conditions compared to homeostatic regulated macrominerals. It is assumed that documented dietary and forestomach mineral interactions (i.e. copper-molybdenum-sulfur) in ruminants are similar in camelids [96, 97]. Again, the reader is referred to the third edition of this text or published texts for detailed information on micromineral nutrition and disease conditions, which are summarized in Table 3.9 [93, 95–97].

The NRC (2007) report did not provide any SAC-specific recommendations for micromineral requirements, except for Se [56]. The suggested Se requirement for llamas and alpacas was 0.74 mg/day based on a single study having this as an average intake of supplemental Se [99]. In this study, it was suggested that 1 mg Se/day was recommended for adult pregnant SAC based on adequate blood Se concentrations in females and their crias. This seems consistent with the recommendation of 2–4 mg Se/day for OWC [100]. The NRC report suggested using sheep Cu recommendations for SAC based on their known sensitivity to Cu toxicosis. There are no published dietary recommendations for

Table 3.9 Summarized micromineral functions and disease conditions.

		Disease conditions		
Micromineral	**Major functions**	**Deficiency**	**Toxicity**	**Interrelationships**
Cobalt (Co)	Component of vitamin B_{12} (ruminants and camelids only)	Megaloblastic anemia; Enzootic marasmus (ketosis), reduced growth, body weight	Toxicity unlikely	Co needs to be supplied to rumen microbes; High Fe, Mn reduce availability
Copper (Cu)	Hemoglobin synthesis; Bone formation; Pigmentation; Myelin formation; Ox-Reduction reactions; Immune function	Achromotrichia; Neonatal ataxia (Swayback); Developmental orthopedic disease; Reproductive failure; Stillbirth, neonatal deaths; Hypochromic-Microcytic Anemia; Immune failure	Toxicity a problem in camelids, results in hepatic necrosis	High Fe, Zn, Se reduce availability; Excess Mo and S inhibit utilization and storage in ruminants;
Iodine (I)	Thyroxine formation; Basal metabolic rate	Goiter, Stillbirths, Infertility; Malformations – hairless neonates; Immune failure	Toxic intake results in goiter, fetal malformations	High intakes reduce uptake efficiency; Selenium deficiency induces hypothyroidism
Iron (Fe)	Cellular respiration, Oxygen transport, antioxidant (catalase)	Hypochromic-Microcytic anemia; easily fatigued	Excessive amounts are pro-oxidants	High Ca may decrease absorption; Cu status influences metabolism;
Manganese (Mn)	Bone formation; Activator of enzymes with CHO, Lipid, Protein metabolism	Impaired fertility; Abnormal bone formation; Poor growth; Impaired Immune function	Toxicity unlikely	Excess Ca and P decrease availability;
Selenium (Se)	Antioxidant – cellular Iodine function; Immune function	Nutritional myopathy (aka white muscle disease); Acute death; Retained placenta; Impaired immune function	Acute toxicity from parenteral products – respiratory collapse; Alkali disease	Sulfate, Fe, Acid soil conditions interferes with See availability;
Zinc (Zn)	Component of >90 enzymes influencing all aspects of metabolism; Immune function	Anorexia; Parakeratosis; Poor skin and hair formation; Male infertility; Impaired immune function	Toxicity unlikely	High Ca, phytates or oxalates bind Zn; High Zn interferes with Cu metabolism

Source: from Van Saun [157].

microminerals in either SAC or OWC, except the extrapolated models for SAC (Table 3.10) [18].

In contrast to macrominerals, there is a large body of published studies and case reports addressing micromineral status evaluation and disease conditions in SAC and OWC. Most SAC reports address Cu toxicosis [101–104] or deficiency [105–107]. There are limited reports on Fe [108, 109] or Zn [110, 111] deficiency for SAC. In comparison, there are many published micromineral studies for OWC, especially relative to Cu and Se deficiencies [100, 112–115].

Vitamins

Vitamins are organic compounds required from dietary or enteric microbial sources that provide a range of functions as cofactors for most metabolic reactions or gene-regulating hormones. Vitamins, like minerals, are categorized into two groups, water- or fat-soluble vitamins. The water-soluble vitamin group is composed of the B-complex compounds and vitamin C. Camelids, like non-primate species, do not have a documented requirement for vitamin C due to sufficient denovo synthesis. The fat-soluble vitamins include vitamins A, D, E, and K. As with the B-vitamins, vitamin K is synthesized by enteric bacteria and not considered a vitamin to be supplemented in the diet. A specific fat-soluble vitamin represents a collection of compounds that have similar biologic function; hence, measurement of international units (IU) relative to a standard compound. For the fat-soluble vitamins A, D, and E 1 IU of vitamin activity is defined as 0.3 μg retinol, 0.025 μg cholecalciferol, and 1 mg *dl*-α-tocopheryl acetate, respectively. Vitamin A activity is currently defined in retinol equivalents (REs), though the conversion efficiency of various vitamin A precursors is species dependent. For sheep and goats, 1 RE is equivalent to 1 μg all-trans retinol; 5 μg all-trans β-carotene; or 7.6 μg other provitamin A carotenoids [56].

Table 3.10 Recommended micromineral requirements for South American camelids.

		Extrapolated requirement		
Nutrient	**Averaged requirement[a]**	**Intake, mg/day[b]**	**Diet, mg/kg[c]**	**Group[d]**
Cobalt	1.76 µg/kg BW	0.11–0.28	0.12–0.15	M, G, P, L
Copper	0.12 mg/kg BW	7.2–19.2	8–12	M, G
	0.15–0.18 mg/kg BW	9–27.2	9–12	P, L
Iodine	8.8 µg/kg BW	0.5–1.4	0.55–0.65	M, G, P
	15.7 µg/kg BW	0.9–2.5	0.65–0.75	L
Iron	0.6 mg/kg BW	36–96	35–40	M, G, P, L
Manganese	0.33 mg/kg	19.8–52.8	22–25	M, G, L
	0.52 mg/kg	31.2–83.2	28–30	P
Selenium	6.5–6.8 µg/kg BW	0.4–1.07	0.42–0.45	M, G
	7–7.5 µg/kg BW	0.44–1.2	0.46–0.5	P, L
Zinc	0.56 mg/kg BW	33.6–89.6	45	M, G
	0.8–1.3 mg/kg BW	60–160	55–60	P, L

[a] Extrapolated from nutrient requirements for beef cattle (National Research Council: *Nutrient requirements of beef cattle*, ed. 8, Washington, DC, 2016, National Academy Press), sheep and goats (National Research Council: *Nutrient requirements of small ruminants: sheep, goats, cervids and New World camelids,* Washington, DC, 2007, National Academic Press).
[b] Estimated daily requirement based on a range of adult body weights from 60 to 160 kg.
[c] Dietary concentration (mg/kg, ppm) on dry matter (DM) basis. Nutrient density calculations based on an assumed range of DM intake between 1.25 and 1.7% of body weight.
[d] Physiologic states of maintenance (M), growth (G), lactation (L), and pregnancy (P) for which the requirement is defined.
Source: Modified from Van Saun [90].

B-Complex Vitamins

Vitamins in the B-complex category are usually produced in the GI tract of ruminants and presumably in that of camelids as well. Little attention has been paid to these vitamins except when requirements are not met. Thiamin (B_1) and cyanocobalamin (B_{12}) may both be in this category. Thiamin is often administered therapeutically in diseases that affect the central nervous system (CNS). Thiamin deficiency, usually a result of diet-induced forestomach degradation, is implicated in polioencephalomalacia cases in cattle and sheep. Production of vitamin B_{12} is dependent upon dietary availability of Co as in ruminants (see Chapter 19).

In situations where animals are anorexic or have altered forestomach fermentation, it can be hypothesized that microbial B-vitamin synthesis may be reduced, thus the animal may benefit from supplementation. Typically, supplementation is administered parenterally from commercial B-vitamin preparations or individually as thiamin or cyanocobalamin. Various commercial oral probiotic supplements are available and often used in off-feed or perceived forestomach conditions. These products contain a range of B-vitamins in varying amounts; however, they also contain live lactobacillus species bacteria. Lactobacillus bacteria generate lactic acid as their end product of fermentation, which is not a desirable compound to have in the forestomach. Lactobacilli bacteria are best positioned to facilitate a more stable microbiome in the intestine rather than the forestomach. No determination of B-vitamin requirements has been completed for camelids.

Vitamin A

The primary biologically active forms of vitamin A are retinol and retinoic acid. Vitamin A is necessary for normal growth, vision, and reproduction. Keratinization of the skin and mucous membranes, failure of regeneration of rhodopsin in the retina, and interference with osteoblast and osteoclast activity occur during hypovitaminosis A. Carotenes, found in plant material, must be converted in the small intestine to retinol for vitamin A activity. Species vary in the efficiency of that conversion. Of the carotene pigments, β-carotene is the most significant relative to providing vitamin A activity. Camelids grazing green forage will consume large quantities of carotenes that will be more than adequate in meeting their vitamin A needs. Cured and stored forage will have much reduced vitamin A

activity given the sensitivity of carotenoids to destruction by oxidation and UV light. In ruminants, the polyunsaturated fatty acid chain of retinol is degraded in the ruminant, thus making ruminants susceptible to vitamin A deficiency. The degree of forestomach degradation of vitamin A in camelids has not been determined.

One study has described vitamin A intake and associated blood retinol concentration in llamas [116]. Calculated vitamin A intake was much less than recommendations; however, serum retinol concentration (78 μg/dL) was similar to healthy dromedary camels (69.1 μg/dL) [117]. In the OWC survey, hospitalized camels had lower serum retinol concentrations (59.2 μg/dL) [117] Alpacas grazing pasture with sheep had higher serum retinol concentrations (77.9 μg/dL versus 39.5 μg/dL) [118, 119]. Another alpaca study found a mean serum retinol concentration of 70.3 μg/dL in a total of 923 samples [120] These comparisons might suggest camelids have greater capacity to convert carotenoids to retinol compared to ruminants, or less forestomach degradation activity.

No studies have performed controlled feeding trials to determine vitamin A requirement. Previously, Johnson [121] had suggested a dietary vitamin A requirement of 6600–11 000 IU/kg dietary dry matter, whereas using extrapolations from beef and sheep requirements, Van Saun [18] suggested a lower requirement of 3000–3600 IU/kg dry matter. NRC suggested using the current sheep vitamin A requirements [56]. Requirements for maintenance were defined as 31.4 RE/kg BW (104.7 IU/kg BW) with increasing amounts to support late pregnancy (45.5 RE/kg BW, 151.5 IU/kg BW) and lactation (53.5 RE/kg BW, 178.2 IU/kg BW). Growing animals had the highest vitamin A requirement of 100 RE/kg BW (333 IU/kg BW).

Vitamin D

Vitamin D is the generic term for closely related steroids that function with Ca and P in bone metabolism. Vitamin D occurs in two forms. Vitamin D_2 (ergocalciferol, D_2), is produced by irradiation of ergosterol in plant material. There is little or no D_2 in pasture plants, but sun-cured hay is a reasonable source of D_2. Vitamin D_3 (cholecalciferol, D_3) is found only in animal tissues (fish oils, liver). It is produced by ultraviolet irradiation of 7-dehydrocholesterol (derived from cholesterol) in the skin of animals when exposed to sunlight.

Vitamins D_2 and D_3 are the precursors to the steroid hormonal biologic activity of vitamin D. Each form of vitamin D follows its own metabolic pathway, but with similar chemical reactions. The first step is the conversion, in the liver, to the 25-hydroxy derivative (25-OHD). Under the action of parathormone, 25-OHD is converted to 1,25-dihydroxy cholecalciferol (the active form of vitamin D) in the kidney. Renal or hepatic disease may lead to hypovitaminosis D. The activated form of vitamin D enters target cells and is transported to the nucleus where gene expression is upregulated to generate specific proteins influencing Ca homeostasis. Primary target tissues for vitamin D are the intestine to increase Ca and P absorption efficiency, bone to facilitate osteoclastic activity releasing Ca and P, and kidney to modulate tubular Ca reabsorption and P excretion [122].

Classically, vitamin D is intimately associated with Ca homeostasis; however, this is not the case for all species. Hypophosphatemic rickets is well documented in SAC. The underlying etiology implicated is vitamin D deficiency [123]. In experimental supplementation of vitamin D to llamas and alpacas, it was observed that serum P was seasonally cyclic with vitamin D. Furthermore, it was found that serum P, but not Ca, concentration increased with vitamin D supplementation [124]. Further studies addressing potential vitamin D toxicosis also showed a greater response in serum P concentrations compared to Ca. This seemingly opposite biologic effect of vitamin D in SAC may be an evolutionary adaption to the inherently low P content of low-quality forages. Serum P is recycled to the forestomach via saliva to support microbial growth. This would result in a net loss of P from the body without a more efficient absorption process. A similar biologic adaptation might be considered for OWC given the low-quality, phosphorus deficient forage typically consumed.

The recommended vitamin D requirement for small ruminant species is 5.6 IU/kg BW with additional amounts added for late pregnancy (213 IU/day), lactation (760 IU/kg milk), and growth (54 IU/d for every 50 g daily gain) [56]. In vitamin D feeding trials of llamas and alpacas, serum vitamin D (25-OHD) concentration did not change until higher concentrations were provided resulting in a recommendation of 30 IU/kg BW [124]. Reported serum 25-OHD concentrations in dromedary camels varied by season and were of similar magnitude as what is reported for [125]. It is uncertain as to whether OWC require the higher dietary vitamin D supplementation.

Vitamin E

Compounds having biologic antioxidant activity associated with vitamin E include four isomers (α, β, δ, and γ) each of tocopherol and tocotrienols. The RRR-α-tocopherol isomer is the most potent and biologically available form. Most supplements are racemic mixtures and esterified for stability. Vitamin E serves as a membrane-based biologic antioxidant to help maintain cellular integrity. Its primary action is to prevent formation of lipid hydroperoxide and subsequent lipid peroxidation of the cell membrane. Vitamin E and Se have much the same ultimate action;

however, Se, as a component of glutathione peroxidase (GSH-Px), converts hydrogen peroxide and lipoperoxides that are already formed to less harmful alcohols and water in the cell cytosol. Thus, vitamin E prevents formation of free radicals while Se eliminates free radicals. The two compounds function synergistically across the cell entirety.

Serum α-tocopherol concentration is similar across ruminant species and reflective of dietary status. Llamas consuming alfalfa hay had serum α-tocopherol concentration of 1.28 μg/mL [116], which is consistent with observations in other ruminant species consuming dry hay. When consuming fresh pasture typical serum, concentrations approach 3 μg/mL of α-tocopherol. Healthy dromedary camels were reported to have a serum vitamin E concentration of 1.06 μg/mL compared to hospitalized (0.76 μg/mL) camels [117]. No controlled feeding trials have been performed relative to vitamin E in camelids. Van Saun had suggested an extrapolated requirement of 0.25–0.5 IU/kg BW for SAC, based on previous NRC requirements for beef cattle and sheep [18]. Vitamin E requirements were greatly increased in the more recent NRC publications for production animals. Recommended vitamin E requirements for all small ruminants, including SAC, were 5.3, 5.6, and 10 IU/kg BW for maintenance, reproduction, and growth, respectively [56].

Feeding Behavior and Intake Capacity

One area of SAC nutrition that has been intensively studied is that of diet selectivity [11, 126–136]. These studies have characterized preferred forages for consumption for llamas and alpacas under South American pastoral management systems. Studies of diet selectivity have been performed by direct observation of selective feeding, use of esophageal fistula, or microhistologic analysis of fecal material. Lip anatomy and the narrow muzzle of SAC facilitate their ability to select feed resources.

Feeding Niches

The vicuña is one of two species of wild camelids. It inhabits the high Andes at elevations of 3700–4800 m. This is a harsh environment of semiarid grassland and barren pampas, with cold temperatures and sparse vegetation. The vicuña is a grazer of forbs and grasses.

The guanaco, also a wild species, has the broadest geographic and altitudinal distribution, ranging from sea level to 4250 m. The guanaco is both a grazer and a browser, inhabiting desert grassland, savanna, and shrub land; it may even be seen in forests. It is found in one of the driest deserts of the world, the Atacama in Chile, and also in the wet archipelago of Tierra del Fuego, where rain falls year-round [130, 131, 137–139].

The llama is found at moderate elevations of 2300–4000 m. This distribution pattern may be more cultural than biologic since the llama has shown ready adaptability to diverse altitudinal and geographic habitats in locations outside South America. The llama is both a grazer and a browser, preferring to pasture on dry tablelands and slopes, feeding on tall, coarse bunchgrasses, which are collectively called "ichu" (Stipa, Festuca, Calamagrostis) and dominated by fescue (*F. doli chophylla*) [128, 129]. Llamas also browse available shrubs and trees [127]. Llamas are better adapted to more arid environments. Many excellent studies have been conducted to determine the nutrient composition of alpaca and llama forages at different seasons of the year. Llamas prefer to graze tall grasses in native grassland and consume fewer leaves compared to sheep and alpacas. These studies support the concept that, to have evolved and become domesticated in this harsh environment, llamas and alpacas must be hardy, resilient, and efficient in extracting the maximum amount of nourishment from forages.

The alpaca is found at elevations of 4400–4800 m and is a grazer, preferring the bottomland vegetation of meadows and marshes collectively called "bofedales" (Figure 3.4) [132–134, 136]. The nutrient composition of Peruvian plants varies greatly seasonally, by altitude, and according to soil type. In dry seasons, the vegetation is sparse and of poor nutrient quality. However, proximate analysis of Peruvian plants (Table 3.11) shows that, at least during part of the year, the nutrient quality may be quite high. Even though camelids have become evolutionarily adapted to survival in their harsh environment, it is important to recognize that they may grow better and be more fertile if given optimal nutrition, at least for a major portion of the year.

A select list of plant species eaten by dromedary camels is found in Table 3.12. As the table shows, there is considerable variation in the nutrient composition of the plants, but this is true of plants available to livestock throughout the world. Selective characteristics of llamas resemble that of OWC that prefer sparse vegetation and dry grasses often avoided by other ruminants [140].

Dry Matter Intake

The cornerstone to any nutritional program and formulating nutrient balanced diets to meet animal needs is dry matter intake (DMI). Characterizing feed intake in llamas and alpacas has been a challenge. Early information suggested that maintenance feed intake was lower compared with other species based on observations documenting a

Figure 3.4 Alpacas grazing on bofedales. The distant slopes are used by llamas.

Table 3.11 Composition of Peruvian llama and alpaca pasture plants.

South America feed–pasturage	Stage of development	Dry matter (%)	Protein (%)	Digestible energy (Mcal/kg)	Crude fiber (%)	Calcium (%)	Phosphorus (%)	Ca : P Ratio
Muhlenbergia, aerial part, fresh	Early	100.0	6.8	2.60	31.2	0.37	0.13	2.8
Muhlenbergia fastigiata	Mature	100.0	5.8	2.86	27.5	0.41	0.13	3.2
Reedgrass, aerial part, fresh	Early	100.0	6.2	2.59	33.8	0.38	0.11	3.5
Calamagrostis antonia	Mature	100.0	4.6	2.34	32.0	0.64	0.07	9.1
Fescue, aerial part, fresh	Midbloom	100.0	8.3	2.12	41.3	0.09		
Festuca dolichophylla	Mature	100.0	2.5	1.21	44.3	0.20	0.04	5.0
Spikesedge, aerial part, fresh	As fed	21.7	3.4	0.59	7.4	0.07	0.04	1.8
Eleocharis geniculata	Dry	100.0	15.5	2.73	34.1	0.32	0.19	1.7
Plantain, aerial part, fresh	Dry	100.0	14.7	3.05	35.4	0.16	0.26	0.6
Plantago sp.								
Crimson clover, aerial part, fresh	As fed	17.6	3.0	0.49	4.9	0.24	0.05	4.8
Trifolium incarnatium	Dry	100.0	17.0	2.81	27.7	1.38	0.29	4.8
Ladysmantle, aerial part, fresh								
Alchemilla pinnata	Dry	100.0	10.9	2.79	28.9	1.90	0.24	7.9

Source: McDowell LR, et al.: Latin American Tables of Feed Composition, University of Florida, Gainesville, 1974.

Table 3.12 Chemical composition of selected plants eaten by dromedary camels.

Plant	Part eaten	Dry matter %	Crude protein %	Crude fiber %	Ash %
Acacia nilotica	Leaves, stems	48	17.50	33.70	5.70
Acacia tortilis	Browse	72.62	9.28	38.33	8
Anachis hypogaea	Mature peanut foliage without nuts	100	DP 10.5	20.3	–
Aristida kelleri	Aerial part		4.90	27	34.90
Artemesia campestris	Browse	31	17.30	24.80	8.80
Capparis decidua	Browse		4.90	31.60	13.50
Cassia spp.	Browse	39	10.80	16.70	5.70
Chenopodium album	Leaves	95.60	21.20	8.30	32
Chloris gayana	Rhodesgrass hay	91	10.1	46.7	9.6
Cyprus esculentus	Aerial part		6.50	21.90	25.40
Ephedra aeta	Browse	95.20	10.40	45	7.50
Hordeum glaucum	Aerial part	94.1	3.70	36	9
Lolium spp.	Aerial part	91	9	3.90	11.30
Prosopis africana	Leaves	54	14.20	34	8.60
Phoenix dactylifera	Fruit	80	3	3.60	4
Opuntia vulgaris	Fruit	15	9	42.90	3.20
Tribulus terrestris	Aerial part	310	17.20	45.10	17.70
Salsola kali	Aerial part	21	15	23.70	20

Source: Wardeh ME, et al.: The nutritive value of preferred plant species by camels. Int. Conf. Camel Production and Improvement, Tobruk, Libya, pp. 126- 145, 1990.

longer retention time for forage particles in C1 [11, 55]. The prolonged retention time provides for greater fiber digestibility by the fermentative microflora in C1. This is an adaptive mechanism facilitating animal survival in the more hostile environment with only sparse, low-quality forage available. In their natural environment, camelids consume this type of diet. From another perspective, high-quality forages like alfalfa may result in excessive fat accumulation when fed to camelids. Granted, there is much individual variation, social behaviors as well as feed ingredient-based issues that control feed intake. Very little documentation of feed intake has occurred with camelids leaving a large hole in our ability to properly manage a feeding program. This situation means that other monitors need to be used to evaluate the adequacy and appropriateness of a feeding program.

The NRC (2007) shows expected DMI for llamas and alpacas to range from 1.0 to 1.5% of body weight (BW) [56]. Summarized data from South America suggested higher intakes rates of 2.0 and 1.8% of BW for alpacas and llamas, respectively [30]. Data from Chile suggested slightly lower DMI expectations for llamas (1.5% BW) and alpacas (1.7% BW) [57]. If models to predict amounts of nutrients required are correct, then these differences in suggested DMI will result in variable expectations for required dietary nutrient density. This may explain differences in dietary energy and protein concentrations between North American and South American feeding guidelines.

Studies directly measuring daily DMI in llamas and alpacas are needed to address these observed differences. Unfortunately, estimating DMI for animals managed on pasture is extremely difficult. Nine published studies were identified that had sufficient individual intake data and adequately characterized feed composition to evaluate expected DMI and dietary factors that might control intake [47, 48, 65, 86, 87, 141–144]. In experimental diets, forage CP and NDF ranged from 2.2 to 20.5% and 45.2 to 74.6% dry matter, respectively. Across these studies with 23 different forage comparisons DMI (3.27 vs 1.59 lb./d) and intake per kg MBW (46.4 vs 35.2 g/kg MBW) were higher for llamas compared to alpacas. Observed intakes within species were highly variable given the wide range of forage quality fed. Adjusted mean DMI (% BW) for llamas (1.47 ± 0.06, range: 0.75–1.71) and alpacas (1.30 ± 0.07,

range: 0.37–2.31) were not different [145]. These intake amounts are intermediate between those recommended by NRC (2007) [56] and Lopez and Raggi [57].

Intake potential in llamas and alpacas was suggested to be related to dietary CP content with depressed intake resulting from low-protein diets [30]. In ruminants, microbial fermentation of fiber is the rate limiting step of intake and dietary NDF content is highly associated with feed intake regulation [141, 146]. From these studies, relationships between intake and dietary NDF and CP content were investigated. Modeling DMI and NDF intake across species did not provide robust predictive models. Models accounting for dietary CP, NDF content and their interaction were developed independently for alpacas and llamas [145]. These models suggest dietary NDF and CP content control DMI capacity of camelids as in other ruminants, but to a lesser capacity (0.8–0.9% BW) resulting from a documented slower rate of passage for dietary fiber. Llamas seem more sensitive to dietary NDF as an intake limiter compared to alpacas given the stronger relationship observed between NDF, CP and DMI capacity. It must also be remembered that late pregnant animals and young growing animals will have a lower NDF intake capacity. Based on other ruminant data, NDF intake of 0.55–0.75% BW might be appropriate for late pregnant and growing llamas and alpacas. Predicted intake potential (% of body weight) can then be determined from forage NDF content and estimated NDF intake capacity (Table 3.13) in evaluating whether a diet can be reasonably consumed.

Table 3.13 Calculated forage intake as a percent of body weight (% BW) based on forage NDF and camelid NDF intake capacity. For example, a forage containing 38% NDF and an NDF intake capacity of 1.0% BW could be consumed at a rate of 2.63% BW. As forage NDF content increases or camelid NDF capacity decreases intake potential for the forage decreases.

Forage quality	Forage NDF %	NDF intake (% of BW)			
		1.0	0.9	0.8	0.6
Excellent	38	2.63	2.37	2.11	1.58
	42	2.38	2.14	1.90	1.43
	44	2.27	2.04	1.74	1.36
	46	2.17	1.96	1.82	1.30
	50	2.0	1.80	1.60	1.20
	54	1.85	1.67	1.48	1.11
	58	1.72	1.55	1.38	1.03
	62	1.61	1.45	1.29	0.97
Poor	66	1.52	1.36	1.21	0.91

Forages and Feed Supplements

The two predominate families of agronomic forages used in feeding domesticated herbivores are either grasses (Family Gramineae or Poaceae) or legumes (Family Leguminosae) [147]. Grasses can be further characterized by their initial carbohydrate synthesized, three (C_3) or four (C_4) carbon sugars. Tropical grasses are C_4 grasses while temperate grasses are C_3, though subdivided into cool or warm season based on preferred temperature for growth. Tropical grasses initiate growth between 10° and 15 °C (50° and 59 °F) and reach maximal growth at 30–35 °C (86–95 °F) in comparison to temperate grasses (0–20 °C; 32–68 °F). Tropical grasses are generally less digestible compared to temperate grasses, often the result of lower protein and higher NDF content [40].

Legumes can have perennial, biennial and annual growth patterns. In contrast to monocotyledon grasses, legumes are dicotyledon plants. Structurally, legumes differ from grasses. Legume plant tissue is organized into distinct stems with leaves in contrast to grasses where structural cell wall components are found within leaves. Legume leaf digestibility is not adversely affected with plant maturity as are grasses. Legumes often have a deep tap root allowing for drought tolerance. Legume growth initiates slightly later than cool season grasses and continues into the summer. Symbiotic *Rhizobium* bacteria associated with legume root nodules allow capture of atmospheric nitrogen as ammonia to facilitate legume protein synthesis resulting in higher plant protein content compared to grasses. The following are brief descriptions of common grass and legume forages species.

Grasses

Grasses are the most dominant and by far the most economically important plant family in the world (600+ genera, 9000–12 000 species). All camelids depend upon grass, either as a standing crop or as cured hay, for the bulk of their nutritional needs [148]. These highly adaptable animals can utilize grass wherever it is found. A comparison of grasses cut at the same stage of maturity is found in Table 3.14. The relationship between composition and maturity is illustrated in Figure 3.5. Compositional analysis of common grasses used for pasture or hay for llamas and alpacas are provided (Table 3.15).

BAHIAGRASS, Paspalum Notatum. Bahiagrass is a deep-rooted, warm season perennial native to South America. It was introduced into North America in 1913. It is principally adapted to the coastal area of the southern United States, as are coastal bermudagrass and dallisgrass. However, bahiagrass tolerates a broader range of soil conditions than other southern-adapted grasses. Bahiagrass

Table 3.14 Comparison of the nutrient composition of grasses cut at the same growth stage (all values on dry matter [DM] basis).

Grass	Crude protein %	Crude fiber %	TDN %[a]
Orchardgrass	9.3	31.4	53
Bromegrass	10.9	29.4	53
Timothy	7.9	30.3	63
Reed canarygrass	13.2	26.4	65
Kentucky bluegrass	12.3	27.6	56
Tall fescue	14.5	24.6	67
Perennial ryegrass	10.4	23.2	68

Note: Growth stage of grasses determined by percent of dry matter.
[a]TDN = total digestible nutrients.

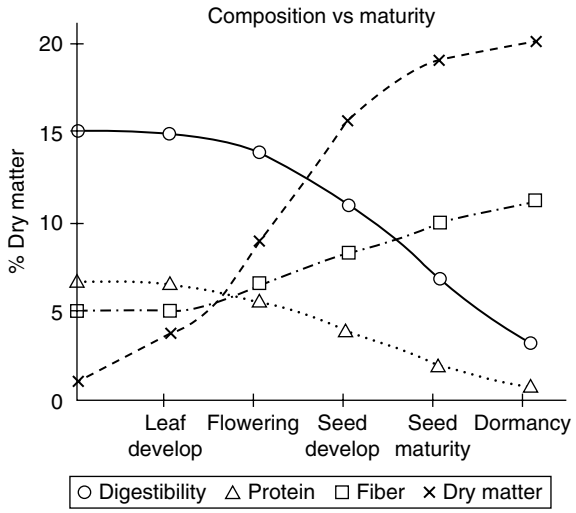

Figure 3.5 A diagram illustrating the change in plant composition with the stage of maturity of grass. Legumes have a similar change, though they have different values for the parameters.

seed head stems may grow to a height of 60 cm (24 in.), but more commonly, the leaves spread out near the ground.

COASTAL BERMUDAGRASS, *C*ynodon Dactylon (WIREGRASS, DEVILGRASS, VINEGRASS, DOGTOOTHGRASS). Bermudagrass was introduced from the Old World and is a hardy perennial that forms heavy sod by means of extensive creeping rhizomes and stolons. It is popular as lawn sod in areas of heavy human traffic, though it becomes dormant and brown during the winter. Coastal bermudagrass (a variety) is a popular pasture forage in the southern and southeastern United States and is harvested for hay.

Table 3.15 Composition of selected grasses used for pasture and hay in feeding llamas and alpacas.[a,b]

	DM	NDF	TDN	CP	NFC	Fat	Ash	Ca	P	Mg	K	Na	S
	%	% DM	% DM	% DM	% DM	% DM	% DM	% DM	% DM	% DM	% DM	% DM	% DM
Bahiagrass 30% Dry Matter	30	68	54	8.9	11.0	2.1	10	0.46	0.22	0.25	1.45	—	—
Bahiagrass Hay	90	72	51	8.2	7.2	1.6	11	0.5	0.22	0.19	—	—	—
Barley Straw	91	72.5	40	4.4	13.7	1.9	7.5	0.3	0.07	0.23	2.37	0.14	0.17
Bermudagrass Late Vegetative	91	76.6	49	7.8	4.9	2.7	8	0.26	0.18	0.13	1.3	0.08	0.21
Brome Hay Late bloom	91	68	55	10	10.7	2.3	9	—	—	—	—	—	—
Brome Hay Mature	92	70.5	53	6	14.3	2	7.2	0.26	0.22	0.12	1.85	0.01	—
Brome Hay Mid Bloom	88	57.7	56	14.4	14.8	2.2	10.9	0.29	0.28	0.1	1.99	0.01	—
Brome Hay Pre-bloom	88	55	60	16	16.4	2.6	10	0.32	0.37	0.09	2.32	0.02	0.2
Fescue Meadow Hay	88	65	56	9.1	15.5	2.4	8	0.37	0.29	0.5	1.84	—	—
Fescue, Alta Hay	89	70	55	10.2	7.6	2.2	10	0.39	0.24	0.23	2.38	—	—
Fescue, K31 Hay	91	62.2	61	15	8.3	5.5	9	0.51	0.37	0.27	2.3	—	0.18
Fescue, K31 Hay, Full bloom	91	67	58	12.9	6.8	5.3	8	0.43	0.32	0.17	2.3	—	0.26

(*Continued*)

Table 3.15 (Continued)

	DM	NDF	TDN	CP	NFC	Fat	Ash	Ca	P	Mg	K	Na	S
	%	% DM	% DM	% DM	% DM	% DM	% DM	% DM	% DM	% DM	% DM	% DM	% DM
Fescue, K31 Mature	91	70	44	10.8	7.7	4.7	6.8	0.41	0.3	0.16	1.96	0.02	—
Grass Pasture Fall	24	67	53	22	−2.7	3.7	10	—	—	—	—	—	—
Grass Pasture Spring	23	47.9	74	21.3	16.4	4	10.4	0.55	0.45	0.32	3.16	—	0.2
Grass Pasture Summer	25	55	67	15	17.3	3.7	9	—	—	—	—	—	—
Meadow Fall	20	52	51.9	13.4	21.6	3	10	0.26	0.15	—	—	—	—
Meadow grass Hay	90.7	65.9	62	6.6	17.2	2.8	7.5	0.22	0.13	0.15	1.71	0.389	0.15
Meadow Hay	90	67.6	60	13.4	5.0	3	11	0.26	0.15	—	—	—	—
Meadow Spring	15	53	44.8	20.3	13.7	3	10	0.26	0.15	—	—	—	—
Mix Pasture Spring	21	41.5	79	26	19.1	3.2	10.25	—	—	—	—	—	—
Mix Pasture Summer	22	46.5	67	19.5	21.4	3.2	9.4	—	—	—	—	—	—
Napiergrass Fresh 30 day DM	20	70	55	8.7	9.3	3	9	0.6	0.41	0.26	1.31	0.01	0.1
Napiergrass Fresh 60 day DM	23	75	53	7.8	10.2	1	6	0.6	0.41	0.26	1.31	0.01	0.1
Oat Hay	91	63	53	9.5	17.2	2.4	7.9	0.32	0.25	0.29	1.49	0.18	0.23
Oat Hay	91.9	51	68	7.8	30.0	3.8	7.38	0.16	0.18	0.12	1.55	0.346	0.15
Oat Straw	92.2	74.4	45	4.4	11.2	2.2	7.8	0.23	0.06	0.17	2.53	0.42	0.22
Orchardgrass Hay, Early bloom	89	59.6	65	12.8	16.2	2.9	8.5	0.27	0.34	0.11	2.91	0.01	0.26
Orchardgrass Hay, Late bloom	90.6	65	54	8.4	13.1	3.4	10.1	0.26	0.3	0.11	2.67	0.01	—
Pangolagrass Fresh	21	70	55	9.1	11.0	2.3	7.6	0.38	0.22	0.18	1.43	—	—
Prairie Hay	91	72.7	48	5.3	11.0	3	8	0.35	0.14	0.26	1	—	—
Red Top Fresh	29	64	63	11.6	12.5	3.9	8	0.62	0.37	0.25	2.35	0.05	0.16
Reed Canarygrass Hay	89	64	55	10.3	12.6	3.1	10	0.36	0.24	0.22	2.91	0.02	0
Ryegrass Hay	88	41	64	8.6	38.2	2.2	10	—	—	—	—	—	—
Ryegrass Hay mature	92	68	58	7	14.0	2.5	8.5	0.28	0.17	0.08	1.7	0.005	0.12
Sorghum Sudan Hay	91	66	56.1	11.3	11.3	1.8	9.6	0.51	0.31	0.37	2.08	0.02	0.06
Sorghum-Sudan Pasture	18	55	65	16.8	15.3	3.9	9	0.49	0.44	0.35	2.14	—	0.11
Timothy Hay Early bloom	89	61.4	59	10.8	19.3	2.8	5.7	0.51	0.29	0.13	2.41	0.01	0.13
Timothy Hay Full bloom	89	64.2	56	8.1	19.6	2.9	5.2	0.43	0.2	0.09	1.99	0.07	0.14
Timothy Hay Late Vegetative	89	55	62	14	20.0	3	8	0.45	0.4	0.11	3.05	0.07	0.13
Timothy Hay Mid bloom	89	63.7	57	9.7	16.9	2.7	7	0.48	0.23	0.13	1.82	0.01	0.13
Timothy Hay Seed stage	89	72	47	6	14.0	2	6	—	—	—	—	—	—
Wheat Straw	89	78.9	41	3.5	7.9	2	7.7	0.17	0.05	0.12	1.41	0.14	0.19
Wheatgrass crest., hay	92	65	53	9	14.7	2.3	9	0.26	0.15	—	—	—	—

[a] Data from feed analysis tables in Nutrient Requirements of Beef Cattle, 7th rev. ed., 2000.
[b] Abbreviations: DM = dry matter; NDF = neutral detergent fiber, TDN = total digestible nutrients, CP = crude protein, NFC = nonfiber carbohydrates (determined by calculation), Ca = calcium, P = phosphorus, Mg = magnesium, K = potassium, Na = sodium, S = sulfur.

KENTUCKY BLUEGRASS, Poa pratensis. Kentucky bluegrass is sometimes thought to be native to the limestone soils of the northeastern United States but was actually introduced from Europe. It is a cool season, long-lived perennial grass growing 30–75 cm (14–30 in.) in height. As the most popular lawn grass in the Unites States, it is also grown extensively as a pasture grass in the northeast and north central states.

SMOOTH BROME, Bromus inermis. Smooth brome was introduced from Europe. It is a cool season, sod-forming perennial adapted to temperate climates from the middle of the United States northward to Canada and Alaska. With irrigation, it is planted throughout the United States. Smooth brome is frequently planted with a legume.

TALL FESCUE, Festuca arundinacea. Tall fescue has become the most widely grown pasture grass in the United States (approximately 14.16 million hectares [35 million acres]). Tall fescue grows to a height of 60–200 cm (24–79 in.) and is a deep-rooted, perennial grass that forms a dense sod through spreading short, underground rhizomes. Tall fescue is capable of withstanding environmental extremes, including drought; flooding; extremes of heat and cold; soils varying in texture, moisture, salinity, and pH; and heavy traffic. Cool, moist conditions are optimal for growth, so surges occur in the spring and fall.

OATS, *A*VENA SATIVA. Oats is an annual harvested as grain or as hay in the Pacific Coast states but is rarely used as a pasture grass. The stage of maturity when cut has a profound influence on the nutritive value of the hay. The best quality hay is obtained if cut when the flowers are in the boot, but the greatest yield of TDN results from cutting when seed heads are in the soft-dough stage. If allowed to go past maturity, the food value of oats rapidly declines to almost nothing as the oats become straw.

ORCHARDGRASS, DACTYLIS GLOMERATUS. Orchardgrass is a perennial, cool season grass introduced into the United States from Europe more than 200 years ago. Cultivars are grown in all states except Hawaii. It may grow to heights of 60–200 cm (2–6.5 ft). Orchardgrass has become one of the most popular pasture grasses for llama and alpaca managers because of its adaptability to many areas of the United States and its palatability.

RYEGRASSES. Various species of ryegrass have become naturalized in most temperate and subtropical climates of the world. Perennial ryegrass (*Lolium perenne*) is grown as a pasture plant and harvested as hay from millions of hectares; it is the second most popular pasture grass in the United States. Perennial ryegrass is a short-lived perennial that grows to a height of 25–50 cm (10–20 in.). Annual ryegrass (*Lolium multiflorum*) may grow to a height of 1 m (3 ft). Both species are native to Europe.

SUDANGRASS, Sorghum Sudanense. Sudangrass and other sorghums were introduced from Africa in the early part of this century. The sorghums are extremely important as forage for animals (pasture, green chop, silage, hay, grain) and as a seed crop for human consumption. Sudangrass is a highly productive annual.

TIMOTHY, *P*hleum pratense. Timothy is a perennial bunchgrass native to Eurasia but now distributed throughout temperate and subarctic climates of the world. It is a cool-season grass and doesn't grow well under drought conditions. It is grown primarily as a hay crop in the northeast, north central, and northwestern United States. Timothy is the hay of choice of horse owners throughout the country. It grows to a height of 80–110 cm (30–43 in.). When planted for pasture, it is usually mixed with other grasses and legumes.

CRESTED WHEATGRASS, *AGROPYRON CRISTATUM*. Crested wheatgrass is a native of Eurasia. Crested wheatgrass is a winter-hardy, drought-resistant perennial bunchgrass, primarily used as pasture forage. It has become a popular grass for seeding rangelands in the northern great plains and the dry northern intermountain area.

Legumes

ALFALFA, *M*edicago Sativa (LUCERNE). Alfalfa is an herbaceous perennial legume. It has become the most important forage crop in the United States ("queen of the forages") and is grown in every state. Alfalfa probably originated near Iran, but similar species are found throughout central Asia and Siberia. It has been introduced all over the world by invading armies, explorers, and missionaries. Alfalfa was described as an excellent forage for horses and other animals by Pliny the Elder as early as 490 BCE. More than 12 million hectares (49 million acres) are planted to alfalfa in the United States. Alfalfa is also the most important source of nectar for bees in the United States.

A mature alfalfa plant may have from 5 to 25 stems growing to a height of 60–90 cm (15–36 in.). Alfalfa is most commonly harvested for hay because ruminants that graze alfalfa pasture tend to bloat. Camelids are generally resistant to bloat due to their forestomach motility.

As with other legumes, alfalfa is able to convert atmospheric N to nitrate by a symbiotic relationship with *Rhizobium meliloti*, an efficient N-fixing bacterium. Colonies of Rhizobium are found in nodules on the roots (Figures 3.6 and 3.7). Seed must be inoculated with the bacteria before sowing to initiate the process.

Alfalfa is harvested one to 10 times a season, depending on the climate and availability of irrigation. First-cut hay is leafier and has fewer stems than later cuttings. The first cutting is the premier dairy cattle feed but is not the first choice for feeding camelids.

Figure 3.6 Alfalfa root nodules containing colonies of nitrogen-fixing bacteria (*Rhizobium* spp.).

Figure 3.7 Alfalfa root nodule containing colony of nitrogen-fixing bacteria (*Rhizobium* spp.).

A close relative of alfalfa, burclover (*Marchantia polymorpha*) has a procumbent growth, so it is rarely harvested for hay. It is, however, an important component of open ranges in California and other regions of the west. It is of value only during the wet season and before the curled pods (with spines) mature and become entangled in fiber.

Red Clover, Trifolium Pratense. Red clover is the most widely grown true clover, planted extensively in the Pacific Northwest, California, Idaho, Nevada, Utah, and the entire eastern half of the United States east of the Mississippi River. Red clover is a perennial legume and shares many characteristics with alfalfa. Cultivars are adapted to either single or multiple cuttings per year. Red clover is a common component of mixed pastures.

White Clover, Trifolium Repens (Ladino Clover). White clover is closely related to red clover and has a slightly broader distribution than red clover in the western and northwestern United States. White clover is a common component of irrigated mixed pastures. Alsike clover, *Trifolium hybridum*, is an intermediate between red and white clover.

Miscellaneous Legumes. Numerous species of legumes are adapted to specific regions of the United States. There are more than 250 species of the genus Trifolium worldwide, including strawberry clover (*Trifolium fragiferum*), crimson clover (*Trifolium incarnatum*), and subterranean clover (*Trifolium subterraneum*). Additional legumes used for forage in the United States include birdsfoot trefoil (*Lotus corniculatus*), lespedezas (*Lespedeza cuneata or L. striata*), and hairy vetch (*Vicia villosa*). A comparison of the composition of selected legumes cut at 1/10 bloom stage is found in Table 3.16. Composition of common legume plants used for pasture or hay for llamas and alpacas are provided (Table 3.17).

Forbs, Browse and Grass-like Plants

A multitude of non-agronomic plants are present in a wide range of environments. Any nonwoody, broadleaf vegetation not classified as grasses, grass-like or legumes are

Table 3.16 Nutrient composition of legumes cut at 1/10 bloom (all values on dry matter basis).

Legume	Productivity (lb DM/acre)	TDN (%)	Crude protein (%)	Crude fiber (%)	Fat (%)	Calcium (%)	Phosphorus (%)	Potassium (%)
Alfalfa	4632	57.4	17.9	33.9	1.54	1.41	0.28	1.42
Red clover	4008	62.9	15.9	26.2	1.54	1.36	0.22	1.36
White clover	2044	77.4	21.8	17.4	1.72	1.55	0.28	1.96

Note: 1/10 bloom based on 100% of dry matter.

Table 3.17 Nutrient composition of legumes forages for llamas and alpacas.[a,b]

	DM	NDF	TDN	CP	NFC	Fat	Ash	Ca	P	Mg	K	Na	S
	%	% DM	% DM	% DM	% DM	% DM	% DM	% DM	% DM	% DM	% DM	% DM	% DM
Alfalfa Hay Early Vegetative	91	33	66	30	23.0	4	10	1.5	0.33	0.21	2.51	0.12	0.54
Alfalfa Hay Early Vegetative	91	36	67	23.4	27.4	3.2	10	1.5	0.33	0.21	2.51	0.12	0.54
Alfalfa Hay Late Vegetative	91	37	63	27	23.2	3.8	9	1.5	0.33	0.21	2.51	0.12	0.54
Alfalfa Hay Late Vegetative	91	39	64	21.7	26.3	3	10	1.5	0.33	0.21	2.51	0.12	0.54
Alfalfa Hay Early Bloom	91	39.3	60	25	23.6	2.9	9.2	1.41	0.22	0.34	2.51	0.12	0.3
Alfalfa Hay Early Bloom	91	42	62	19.9	26.0	2.9	9.2	1.63	0.22	0.21	2.51	0.12	0.54
Alfalfa Hay Mid Bloom	91	47.1	58	22	19.8	2.6	8.5	1.37	0.22	0.35	1.56	0.12	0.28
Alfalfa Hay Mid Bloom	91	49	60	17	23.0	2.39	8.57	1.39	0.24	0.35	1.56	0.12	0.28
Alfalfa Hay Full Bloom	91	48.8	55	17	23.0	3.4	7.8	1.19	0.24	0.27	1.56	0.07	0.27
Alfalfa hay mature	89.3	50.7	59	14.9	21.9	3	9.5	1.06	0.21	0.15	1.31	0.026	0.11
Alfalfa Hay Full Bloom	91	51	56	13	25.2	1.8	9	1.19	0.24	0.27	1.56	0.07	0.3
Alfalfa Hay Late Bloom	91	53	52	17	20.5	1.5	8	1.19	0.24	0.27	1.56	0.07	0.3
Alfalfa Hay Late Bloom	91	55	53	12	23.4	1.6	8	1.19	0.24	0.27	1.56	0.07	0.3
Alfalfa Hay Mature	91	58	50	14	19.7	1.3	7	1.18	0.21	0.22	2.07	0.08	0.25
Alfalfa Hay Weathered	89	58	48	10	24.0	0	8	2.29	0.23	0.27	2.42	0.06	0
Alfalfa Hay Seeded	91	70	45	12	10.0	1	7	1.18	0.21	0.22	2.07	0.08	0.25
Alfalfa Meal dehydrated 15%CP	90	55.4	59	17.3	15.0	2.4	9.9	1.38	0.25	0.29	2.46	0.08	0.21
Birdsfoot Trefoil, Hay	91	47.5	59	15.9	27.1	2.1	7.4	1.7	0.23	0.51	1.92	0.07	0.25
Clover Ladino Hay	89	36	60	22.4	29.5	2.7	9.4	1.45	0.33	0.47	2.44	0.13	0.21
Clover Red Hay	88	46.9	55	15	27.8	2.8	7.5	1.38	0.24	0.38	1.81	0.18	0.16
Leg Pasture Spring	20	33	79	28	26.3	2.7	10	1.71	0.3	0.36	2.27	0.21	0.36
Leg Pasture Summer	23.2	38	66	22.2	26.7	2.9	10.2	1.71	0.3	0.36	2.27	0.21	0.36
Vetch Hay	89	48	57	20.8	21.2	3	7	1.36	0.34	0.27	2.12	0.52	0.15

[a] Data from feed analysis tables in Nutrient Requirements of Beef Cattle, 7th rev. ed., 2000.
[b] Abbreviations: DM = dry matter; NDF = neutral detergent fiber, TDN = total digestible nutrients, CP = crude protein, NFC = nonfiber carbohydrates (determined by calculation), Ca = calcium, P = phosphorus, Mg = magnesium, K = potassium, Na = sodium, S = sulfur.

collectively defined as forbs. Examples would include ornamental plants and weeds. Both ornamental plants and weeds have potential to contain one or more toxic principles. Azalea and rhododendrons are especially toxic to SAC. Be aware of potential plant toxicities in grazing areas. These plants can provide highly digestible nutrients as was demonstrated by analysis of blackberry (Rubrus) leaves [149].

Browse collectively includes woody plants such as trees, bushes, and shrubs. Bark may be consumed by some herbivores during winter; however, buds, leaves and twigs are more often selectively consumed from browse. Forbs and browse can comprise a significant proportion of the camelid diet in most countries other than North America and Europe. Llamas and alpacas will selectively consume forbs and browse during the dry season in South America [89, 126, 128, 134, 150].

Grass-like plants include the sedges (Family Cyperaceae) and rushes (Family Juncaceae) plant groups. These plants are often confused with grasses by general appearance; however, there are distinct structural differences in stem, leaves and flowers. These plants grow in colder and wetter conditions compared to grasses. These plants are not important forages in North America but are selectively consumed by SAC [89, 126, 128, 134, 150].

Commercial Feed Supplements

A variety of commercial products are available to supplement forage in feeding SAC. These products are more typically used outside of South America in SAC feeding programs. The intent of these products is to provide additional nutrients above which would be provided by forage.

Typically, these products provide additional energy sources from starch or fermentable fiber ingredients and protein. Vitamins, primarily fat-soluble sources, and minerals are provided as most conserved forage is depleted of fat-soluble vitamins and variable in mineral content. Additional feed additives such as specific B-vitamins, yeast products, flavorings, pellet binders, antioxidants, among other items that provide a proprietary formulated product.

Commercial llama and alpaca products can be formulated as complete feeds, meaning the product is balanced for the nutrient requirements of the animal for the intended feeding rate and minimal additional forage is provided. These products are not as common as "complete supplements," which are also termed "grain," "pellets" or "concentrate". Complete supplements are formulated products to be fed at a defined feeding rate (i.e. lb./100 lb. body weight or lb./day) and complement a forage feeding program. These products may come as a pellet of varying size, a crumble or mixed ingredient meal with varying molasses content. Molasses helps to bind the fine materials to prevent sorting, which SAC are highly efficient. Molasses content over 8% is typically termed a "sweet feed" and will stick together when squeezed in your hand. Ingredients may be whole grains or seeds or processed to smaller particle size or altered by cooking, heating or extraction.

Many llama and alpaca specific supplement products were initially formulated like similar products for sheep, goats, or cattle. As SAC digestive physiology has become better understood, the use of cereal grains such as corn, oats, barley, wheat to provide readily fermented starch for energy have been reduced due to the SAC unique glucose metabolism and reduced insulin secretion. These products have been mostly replaced with fermentable fiber sources such as beet or citrus pulp, wheat middlings, wheat bran, soyhulls, rice bran or similar sources. Fermentable fiber sources provide short-chain fatty acids used as tissue energy substrates in lieu of glucose and do not negatively affect C1 pH. Protein sources like soybean meal, canola meal, other plant proteins and protein byproducts can be included as protein content of cereal grains and fiber byproducts is not sufficient. Most commercial supplement products range from 10 to 18% CP. Camelids, like other ruminants, can utilize urea as a non-protein nitrogen source for their microbial populations. Urea feeding guidelines for ruminants can be applied to camelids.

Complete supplements will also be fortified with fat-soluble vitamins, typically A, D and E, and minerals (both macro- and micro-). There is tremendous variation in the nutritional profile of vitamins and minerals across products. One reason for the variation is difference in expected feeding rate. Products are formulated to deliver the desired nutrient amounts based on an intended feeding rate as indicated on the feed tag. If a product is not fed according to the label directions some nutrients may be insufficient or excessive relative to requirements. Another reason for nutrient variation is the intended forage species (e.g. grass vs. legume) and quality to be fed. Most SAC products have moderate to high calcium as grass is the preferred forage fed. Trace minerals and vitamins vary due to minimal information defining SAC requirements. Generally, products will contain higher zinc (>150 ppm) and minimize copper (<35 ppm) based on clinical perceptions of zinc-associated skin issues and risk of copper toxicity. Selenium content will depend on geographic region where the product is produced and intended for use and the selenium status of the region.

Nutrient content of commercial products can be found on the product's feed tag, though this information is very limited. More detailed product information may be obtained from the company's website. Representative samples of a product can be sent to commercial feed laboratories for nutrient analysis. Interpretation and use of feed tag information is provided in the feed evaluation section (see Feed Label Interpretation).

Mineral Supplements

Mineral nutrition is geographically dependent as forage mineral content is influenced by soil type, pH, moisture and organic matter content, annual precipitation, soil mineral content and forage species. A large regional survey of forage samples showed agronomic grasses and legumes were mostly low in zinc, copper, and selenium as well as having significant amounts of iron, molybdenum, and sulfur that can act as inhibitors of many trace minerals [151]. Large multi-year feed analysis databases provided by commercial laboratories (Dairy One: https://dairyone.com/services/forage-laboratory-services/feed-composition-library) show wide forage mineral content variation within and between geographic regions.

At an absolute minimum, salt needs to be provided to camelids as forages are low in sodium content (<0.01% DM) unless they are produced in areas near saltwater. Contrary to popular belief, camelids, like other species, do not have specific appetites for all the essential minerals or vitamins. Feeding minerals as a cafeteria of individual sources will not result in the camelid consuming a balanced mineral supplement. Camelids have a specific appetite for salt, or more appropriately, sodium. Thus, salt is used as a vehicle to facilitate intake of other mineral ingredients and a limiter of intake. Various mineral elements will need to be balanced relative to expected intake of sodium unless some palatable ingredient such as soybean meal, distiller's grains or similar ingredient is included in the mineral supplement formulation.

Mineral supplements are manufactured in three primary forms, loose granular, blocks or lick tubs. Camelids do not typically lick but obtain their feed substances by chewing. Camelids will chew mineral blocks, but providing loose granular mineral sources is preferred. Salt products are often described as "white salt" or "red salt." White salt is essentially sodium chloride or traditional salt. Some white salt products may also contain sodium or potassium iodate, thus are iodized salts. Red salt is sodium chloride with various trace mineral sources added and red in color from ferric oxide being added. Individual trace mineral sources used most often are inorganic forms of the mineral. Mineral source availability is dependent upon the mineral form. Mineral sulfates and chlorides are most available and oxides least, though this is not consistent across all minerals. Most important here is copper oxide is an unavailable form of copper to ruminant animals as a mineral source. Various organic mineral sources are being utilized in many products with the intent of providing a more available mineral source. Trace mineral supplements will be formulated to provide trace minerals based on defined intake for a given product, thus accounting for much of the diversity in product composition. Mineral supplements may also contain macrominerals and vitamins.

Since salt is the driving factor for intake, only one salt source should be available if one wants to use a free-choice feeding approach for trace-mineral supplementation. Average expected daily intake for a high salt (>90%) mineral product in camelids is between 0.5 and 0.75 oz/day (14–21 g/day). Many products have far lower salt content and suggest intake rates between 1 and 4 oz/day. How these expected intakes are determined is uncertain and personal experience suggests these intakes are not achieved with camelids. Studies documenting mineral intake in cattle show individual variability though the overall average intake may be within reason. One can determine average intake by determining mineral provided and consumed over a 30-day period divided by the number of animal-days (i.e. number of animals × number of days). Knowing the average mineral supplement intake is important in evaluating adequacy of mineral supplement in providing individual trace minerals (see Feed Label Interpretation).

Feeding Management

Camelids are highly adaptable to a range of feeding systems depending upon feed resources available. Camelid feeding systems will depend upon intended management objectives achievable with the available geographic forage resources and financial resources for animal facilities and supplement products, if available.

Extensive Management

Extensive management systems are based on use of native pastures within minimal additional inputs. This is the traditional South American practice of managing llamas and alpacas in the Altiplano regions (Figure 3.8). Supplementation with a concentrate or mineral product is

Figure 3.8 Traditional pastoral management of alpacas in the Altiplano region of Peru.

not used, though the practice is gaining attention with Peruvian producers. In an extensive management system, pasture forage quality is determined by environmental conditions, forage maturity and animal stocking density. The Andean grassland environment experiences a large percent of annual rainfall between December and March with dry to drought conditions the remaining part of the year. Pasture forage growth and quality follows the precipitation cycle resulting in long periods of low quality and quantity of forage that would not meet animal energy, protein or both requirements [152].

Animal life cycle management needs to be aligned with plant growth in the rainy season. Calving and rebreeding must occur at the beginning of the rainy season to ensure a reasonable match between requirements and forage quality. Animals not aligned with the rainy season, or climate change altering plant growth, results in impaired dam reproduction and cria survival. Critical periods of the SAC life cycle, in relation to forage quality and quantity, are weaning and late pregnancy, which occur during the dry season. These critical dietary imbalance periods may result in lower birth weights and reduced colostrum quality and quantity due to inadequate late pregnancy nutrition accounting for observed high cria losses compared to intensive management systems [153]. These nutritional issues may also account for the high embryonic mortality observed in alpacas [154]. Similarly, crias may experience impaired growth rate following weaning and this may relate to greater disease susceptibility.

Strategies to minimize these negative impacts on animal health and reproduction would include reducing stocking density, use of cultivated pastures, selective grazing, use of supplements, or some combination of these [155]. Economic factors minimize the potential for reducing stocking density as a result of higher animal losses experienced. Cultivated forages using ryegrass and white clover could improve forage quality over a longer period and be targeted for grazing by those animals having higher nutrient requirements. Pellets consisting of harvested oat and legume forages are being investigated as possible dietary supplements in the Peruvian puno region. Adoption of new technologies to augment traditional extensive systems will depend upon education of, and economic return to, the producer.

Intensive Management

A more intensive feeding management approach is more commonly practiced outside South America. These programs are high input where supplement feeding (see Feed Supplements) is provided in addition to forage in some managed animal housing and feeding facilities. Animal housing needs are predicated on the greater range in ambient temperature and weather conditions experienced by managed SAC outside of South America [156]. Forage feeding may consist of pasture for part of the year followed by harvested forage in the form of hay, though some regions of the world will feed ensiled forage. In this system, animals will be provided diets targeted to various feeding groups, typically based requirements in support of differing physiologic states (Table 3.18) [157]. Using an intensive feeding management approach, where animals are not exposed to cyclic feeding depravation periods, SAC reproduction can occur year-round with no defined breeding or calving season and cria growth and health is generally optimized. However, there are potential pitfalls in an intensive feeding approach if not managed appropriately.

Usage of pasture will depend upon local climatic conditions, plant varieties, seasonal availability and grazing strategies [158, 159]. A primary limitation to pasture usage is the potential carrying capacity of the pasture, which dictates number of animals (i.e. stocking density), length of exposure and percent of nutrient requirements that can be obtained [160]. Stocking density is defined as the number of animal units per acre (or hectare) of grazing land. An animal unit is defined as a 1000 lb. nonlactating beef cow with other animals rated proportionally (Table 3.16) [160]. Fertilized temperate cool season grass pasture would typically be capable of supporting 1–1.2 animal unit months per acre. This would translate into 4–5 alpacas or 3–4 llamas per month per acre. Summer, rangeland or unimproved pastures would have a lesser carrying capacity. In many SAC situations, the pasture carrying capacity is overwhelmed by excess animal numbers or duration of grazing resulting in greater parasitic issues as well as malnutrition and weight loss if not supplemented.

Hay is the predominate forage source for many intensive feeding systems. Many SAC owners do not raise their own forage and purchase hay from single or multiple sources. Quality of hay forage can vary tremendously depending upon stage of maturity at harvest and plant species composition. Plants grow and mature in relation to ambient temperature, light and their interaction, soil nutrient content and precipitation [161]. Soil nutrient content can be manipulated via fertilization. Environmental conditions have the greatest impact on forage quality within a geographic region resulting in variation within (first, second, third or more cuttings) and between years even from the same field. Mature grass forage hay has a nutritional composition consistent with late dry season South American forages and could be the source of nutritional disease if fed to productive animals. Laboratory analysis is the definitive method of evaluating hay quality (see Forage Evaluation section); however, many owners are reluctant to spend

Table 3.18 Suggested dietary composition for SAC feeding groups.

Group	Physiologic state	Feeding plan	Dietary guidelines[a]
Nursing Dams with crias	Lactation	Highest nutrient requirements, feed best-quality forages, with energy/protein supplements	60–70% TDN, 12–14% Crude protein, 0.9% BW NDF, 0.45–0.62% Ca, 0.32–0.45% P,[b]
Weanlings up to 1.5 years	Growth	Highest nutrient requirements, feed best-quality forages, with energy/protein supplements	55–65% TDN, 14–16% Crude protein, 0.6–0.8% BW NDF, 0.53–0.73% Ca, 0.27–0.38% P,[b]
Males >1 year	Maintenance	Low requirements unless working, then adjust accordingly, low-to-moderate quality forage	55–60% TDN, 8–10% Crude protein, 0.8–1.0% BW NDF, 0.3–0.48% Ca, 0.21–0.28% P,[b]
Pregnant females 1–8 months	Maintenance	Low requirements, but ensure no loss of body condition, adequate protein, minerals, and vitamins	50–55% TDN, 8–10% Crude protein, 0.8–0.9% BW NDF, 0.2–0.24% Ca, 0.12–0.2% P
Pregnant females 9–11 months	Pregnancy	Moderate to high forage quality with supplement for additional mineral and vitamin needs	55–70% TDN, 10–14% Crude protein, 0.6–0.8% BW NDF, 0.45–0.56% Ca, 0.28–0.33% P,[b,c]
Breeding females	Maintenance	Low to moderate; ensure do not become fat or lose condition	50–55% TDN, 8–10% Crude protein, 0.8–1% NDF, 0.2–0.24% Ca, 0.12–0.2% P
Obese females	Sub-Maintenance	Low; low-quality forages with mineral/vitamin supplement unless pregnant	45–53% TDN, 8–9% Crude protein, 0.8–1% BW NDF, 0.2–0.24% Ca, 0.12–0.2% P

[a] Ensure adequate available water and free choice salt. White salt should be used when trace minerals are included in a supplement. Otherwise, trace mineral salt should be available.
[b] These feeding groups require higher amounts of trace minerals and vitamins preferably delivered by a supplement (refer to text for details).
[c] Dietary energy and crude protein (CP) content may need to be increased further in late pregnancy if dry matter intake (DMI) drops below 1.5% of body weight.

money on forage testing as they are constantly procuring hay. Hay should be purchased based on some level of nutrient content testing or testing performed to document quality. Keeping forage test records can identify desired hay producers and sellers.

Pellet, grain and mineral supplements are often fed to complement the forage program in an intensive feeding system. These products can provide additional energy, protein, minerals and vitamins not furnished by the forage thus providing a balanced diet for all physiologic feeding groups. Unfortunately, most commercial SAC feed supplements are purchased based on recommendations rather than matching a product's nutrient content to the forage program. Supplements may also not be fed according to the product's label feeding directions leading to inadequate or excessive nutrient supplementation and potential disease issues.

Dietary Evaluation

Undertaking a nutrition evaluation can be daunting and one needs to understand what is being asked. Is the interest in determining the nutrient composition of the diet or nutritional status of the animal? In nutritional circles, it is often stated that there are four diets on the farm: (i) formulated; (ii) prepared and delivered; (iii) animal consumed; and (iv) animal digested. Ideally, the first three should be reasonably equivalent and they can be evaluated by nutrient compositional analysis. The fourth diet would require animal nutritional status analysis, which would include body weight and condition score determinations, blood nutrient and metabolic parameter assays and fecal and urinary nutrient composition. Given individual variation and associated costs of testing, evaluation of the digested diet is not routinely practical. Individual animals or groups may be tested for various minerals, fat-soluble vitamins, liver function, protein and energy status parameters as part of either a nutritional evaluation or disease diagnostics.

The first step in dietary evaluation is accurately defining the animal group being fed to determine requirements. Animal descriptors would include body weight, physiologic state, rate of gain, milk production and composition, month of pregnancy, fleece weight and activity level. For an animal group, averages of these metrics are used. Milk production and composition information is typically not available and would need to be estimated [82] The NRC

(2007) provides tables to estimate daily requirements for llamas and alpacas [56].

The next step is to define the dietary components, their nutrient composition and amounts offered. In camelid diets, the primary dietary component is forage. To determine nutrient composition of forage, a representative sample needs to be collected. Harvested hay is best sampled using a bale-coring device, as hay flakes or grab samples should not be sent for analysis (Figure 3.9). The National Forage Testing Association (NFTA, www.foragetesting.org) provides a review of various commercially available hay probes and their use (NFTA.org/hay probes) [162]. The recommended method of collecting a representative hay sample is to randomly probe at least 20 bales mixing all samples in a dry, clean, air-tight plastic bag (NFTA.org/hay sampling) [163]. Up to 35 core samples should be taken from larger lots of hay or hay that may be highly variable in composition. Approximately ½ lb. (225 g) total sample should be collected and submitted to an accredited laboratory. States with Cooperative Agricultural Extension offices are likely to have a list of laboratories that perform analysis of forages and soils. The NFTA website provides a listing of accredited laboratories [164].

Sampling pasture is more challenging. Ideally, one should observe the animals grazing behaviors and mimic what the animals were eating. Alternatively, one can use a "ring" to toss randomly around the pasture and clip the upper half of the standing forage. Pasture samples need to be frozen immediately, given their higher water content, to accurately determine moisture to define dry matter nutrient content. State Cooperative Extension offices may have an agronomy educator that can help collect pasture samples or feed company representatives can provide such service.

Evaluation of commercial concentrate feeds (grains, meals, pellets) and mineral supplements can be accomplished through feed label interpretation (see Feed Label Interpretation) or submission of a representative sample to a commercial laboratory just as for forage. It is recommended that a representative sample of any new commercial concentrate feed be collected (1/2 lb. [225 g]) identified by name, sample date and frozen. Should there be any untoward situation identified this sample can be submitted for analysis.

Figure 3.9 Obtaining a core sample of hay for analysis of nutrient composition.

The final step in dietary evaluation is the most challenging; determining amounts consumed. DMI is the cornerstone of any nutrition evaluation but is the most challenging metric to quantify. Most SAC are fed in groups so an averaged intake could be determined by weighing amounts provided and refused and calculating the difference averaged over the number of animals in the group. Challenges to this approach are feeding dietary components separately (hay and pellets) and the mixture of animals within a group. Estimating the number of grains consumed is straightforward as grains are typically totally consumed so the average intake would be the amount provided divided by the number of animals. The amount of hay consumed needs to account for not only the amount provided and left but what may be wasted on the ground. Depending upon the hay feeding system, hay wastage may range from 10 to 25% of what is provided. Use of forage NDF content can be used to estimate potential intake (see DMI section; Table 3.13) [145]. Once dietary composition and intake have been determined, calculated daily nutrient intake can be calculated and compared to determined requirements. There are no commercially available software programs for SAC dietary analysis, but this can be accomplished using a spreadsheet program. Recommendations may then be made for appropriate supplementation, change or not in the diet or improvement in forage quality.

Forage Assessment

Forages may be grown on the farm or ranch, but most often, they are purchased. The purchaser must be able to judge the quality of the forage or rely on the integrity of the supplier. The quality of hay is determined by the stage of maturity when it is cut, method of curing and conditions of storage [161]. Usually, the owner/manager evaluates the forage, but veterinarians should be able to determine the quality of forages and recommend a change or supplementation as appropriate.

Forage can be assessed based on qualitative organoleptic characteristics or quantitative chemical composition analysis. Organoleptic properties include color; odor (e.g. sweet, musty); texture (e.g. soft and pliable, coarse and rigid); presence of seed heads; leaf-to-stem ratio and presence of foreign material. Foreign material may include

weeds (particularly poisonous plants), dirt, sticks, unpalatable grasses, grass awns, or wire and nails. A desired color for hay is green compared to yellow or brown, both of which indicate oxidation or heating. Color, however, does not ensure good or poor quality. Presence of mold is always a concern though lack of obvious mold does not confirm the absence of mold spores. One should be cautious of smelling hay due to potential mold spore contamination and respiratory health issues.

Hay maturity is best assessed by the presence of seed heads in grass and the leaf-to-stem ratio in legume forages. As the plant matures, it is gaining cell-wall components resistant to digestion resulting in a dilution of CP, energy and mineral plant content. Ideally, grasses should be harvested at the boot to early head stage to optimize amount of forage digestible organic matter. If the grass is allowed to continue to mature, yield (tons/acre [tonne/hectare]) will increase but digestibility and intake capacity will be compromised. In evaluating legumes (e.g. alfalfa, birdsfoot trefoil, clover) maturity is assessed by observing the ratio of leaves to stem. Lush early growth legumes will have high leaf content and thin, pliable stems. As the plant matures, the proportion of leaf decreases as the stem lengthens and thickens. Digestibility of legume leaves does not change dramatically with maturity in contrast to stems that become highly lignified and indigestible with maturity. Take a legume stem and attempt to wrap it around your finger. If the stem is very pliable and easily wraps around your finger this is an immature, lush, digestible forage. If the stem does not wrap and breaks like a pencil, then the plant is highly lignified and mature. Legumes flower as they mature. Alfalfa should be harvested between 10 and 25% bloom to optimize yield of digestible organic matter. Clover should be harvested between 25 and 50% bloom.

Quantitative analysis of forage involves chemical methodologies to determine specific nutrient composition. Feed testing has evolved tremendously in response to the needs for feeding cattle efficiently and economically. Many parameters measured for dairy cattle nutrition can be extrapolated for use in camelid nutrition. Fractionation methods for protein and carbohydrates have been the focus of feed analysis improvements in feeding ruminant animals. Historically, CP has been the metric for measuring protein content of feeds; however, this measure defines nothing of how the feed protein can support forestomach microbial growth or the animal. To this end, additional protein parameters such as soluble protein, unavailable or bound protein and rumen degradable protein (RDP) fractions are determined. Though RDP is measured in reference to dairy cattle, this parameter is important for camelids as C1 degradable protein is essential for efficient fiber fermentation. Given the inherent differences in rate of passage and degradability, one could assume that the cattle RDP is a minimum value for camelids.

Carbohydrate fractionation methods have also been expanded. The original crude fiber measure has been replaced by acid (ADF) and neutral (NDF) detergent fiber determinations. The calculated nonfiber carbohydrates (NFC) content is further evaluated for sugar and starch content, termed nonstructural carbohydrates (NSC). Degradability of NDF at specific time points has been used to better characterize the quality of dietary fiber and its potential degradability. Lignin content as a proportion of NDF is another proxy for maturity and NDF degradability.

The gold standard of feed analysis is use of wet chemistry methods, though application of near infrared (NIR) spectroscopy has gained validity. Many accredited feed analysis laboratories will provide a blended methodology testing service. NIR is less expensive and time consuming, though minerals should only be quantified using wet chemistry. Today's feed analysis report is quite extensive and to the novice, quite overwhelming to interpret all the values. The following is a systematic overview of key nutritional parameters in evaluating forage. Remember that evaluation of forages should be performed on a dry matter (DM) and not as fed (AF) basis.

1) ***Dry matter content***: Hay should have >85% DM or <15% moisture. As moisture increases to 20% or greater, the risk for mold growth increases exponentially. As molds grow and metabolize plant sugars they generate heat, which can cause heat damage to forage protein or become hot enough to have the hay bale spontaneously combust. To allow for higher moisture content in harvested forage, there is a need for fermentation to generate lactic acid dropping pH, thus preventing mold growth and heating.
2) ***Crude protein (CP) content:*** CP is somewhat of a misnomer as protein cannot be directly measured. Nitrogen (N) is measured and then adjusted to CP (N% × 6.25) assuming N comprises 16% of protein molecules. Higher CP typically indicates a more immature grass or legume, though fertilization can increase protein in more mature forage. CP value of legumes does not change dramatically from immaturity (22–24% DM) to maturity (13–15% DM) forage. In contrast, immature grasses may contain high CP, especially pasture (20–35% DM) but mature grasses will be much lower in CP content (4–8% DM). What is important relative to CP is the availability of dietary protein for the forestomach microbial populations in support of their growth and production of microbial protein.
   The fiber fermenting bacterial populations have a critical need for ammonia (degraded amino acids from

protein) in support of their N needs [23, 165]. Soluble protein or ammonia as a percent of CP can be measured in feeds to indicate the amount of readily available N in support of C1 microbial fiber fermentation. Soluble protein and ammonia as a percent of CP less than 20 and 5%, respectively, indicates a potential lack of readily available N to support fiber fermentation. Another measure, RDP, is the estimated degradability of dietary protein. Both soluble and insoluble dietary protein sources can be degraded in the rumen; however, insoluble source degradability is sensitive to retention time in the fermentation vat [165]. Camelids have a slower rate of passage in their forestomach compared to ruminants, thus estimated RDP values most likely underestimate degradability in camelid feeding. A desired RDP for a forage fed to camelids would be >65% to ensure adequate microbial N availability.

Another feed protein metric to evaluate is the amount of bound or unavailable CP. The amount of N in the acid detergent residue is termed acid detergent insoluble crude protein (ADICP). This value is often represented on a DM or percent of total CP basis. This fraction is a result of heating inducing a molecular bonding between sugar and amino acids in the feed. The ADIN fraction should be <10% of feed CP. Higher values (>12% CP) result in reduced dietary protein digestibility and forestomach degradability.

3) Fiber content: Total plant cell wall is evaluated by NDF content with increasing values taking account of advancing plant maturity. A subset of NDF is ADF that measures the more slowly fermentable or indigestible fiber component (i.e. cellulose). Both ADF and NDF will increase with advancing plant maturity. The difference between ADF and NDF is an estimation of hemicellulose plant content. Grasses (15–25%) contain more hemicellulose than legumes (5–8%). The ratio between ADF and NDF can indicate the type of plant, which can be useful in appreciating the predominate plant species in a mixed composition forage. The ADF-to-NDF ratio is approximately 0.6 and 0.8 for grasses and legumes, respectively. Calculated values between these two extremes suggests a mixed grass-legume forage. Immature grasses will range between 40 and 50% NDF while mature grasses will have >60% NDF. Immature and mature legumes will contain <35% and >45% NDF, respectively.

   Like CP, rumen degradability of NDF (dNDF) can be measured (*in vitro*) or estimated (regression equations) at different time points mimicking perceived retention time. Time periods of 24, 30, or 48 hours are most often used for predicting NDF degradability in dairy cattle. Retention times of 30 and 48 hours may be most appropriate for camelids, though what is more important in interpretation is the comparison between forages at a given time point. Higher values indicate a less mature forage though the range in dNDF varies between grasses and legumes.

   Another method of evaluating NDF digestibility is calculating the ratio of lignin-to-NDF. Lignin is the cementing compound of the plant cell wall that increases its rigidity but also reduces hemicellulose and cellulose digestibility. Grasses and legumes on average have a lignin-to-NDF ratio of 8 and 17%, respectively. Calculated ratios above these values indicate a less degradable forage, while lower values indicate greater dNDF. Mixed species forages are more challenging to interpret with this ratio and performing an *in vitro* degradability would be suggested.

4) *Mineral content*: Forage mineral content should be evaluated on an absolute amount as well as various ratios between minerals. If there is a total ash determination provided, review this value to see if it exceeds 10% DM. High ash is associated with soil contaminated samples or high silica western forages. For macrominerals, look first at calcium (Ca) and phosphorus (P) and their ratio (Ca:P). Maintenance requirements are 0.25 and 0.13% DM for Ca and P, respectively. Growth, pregnancy and lactation will require higher amounts, though most forages will not exceed 0.22% P without fertilization. The Ca:P ratio should be 1.5 : 1 or greater in any forage. Legumes will have more Ca than grasses and will have a Ca:P ratio >4-8:1 or greater. The worst-case scenario is to have a less than 1 : 1 Ca:P ratio. Any forage with an inverse Ca:P ratio should not be fed without additional Ca supplementation. It would be best not to purchase such a forage given the potential nutritional issues.

   Forage magnesium (Mg) less than 0.12% DM is low and additional Mg will need to be in a mineral or grain supplement. Forage potassium (K) is typically in excess of requirement and can potentially interfere with Mg absorption. A dietary ratio of 4 : 1 for K:Mg is desirable. Sodium (Na) content in forages is quite low (< 0.05% DM) unless the forage was grown near saltwater. Forages with high sodium will reduce the animal's need to consume additional salt or salt-based mineral products.

   For trace minerals, iron (Fe) should be <200 ppm DM and values exceeding 1000 ppm DM indicate soil contamination of the sample. Grasses (40–200 ppm DM) have higher manganese (Mn) content compared to legumes (20–40 ppm DM). Copper (Cu) content is variable with higher values (>8 ppm DM) found in forages east of the Mississippi River. Western forages are lower in Cu (<7 ppm DM) with the potential for

deficiency [151]. Extremely high forage Cu (>20 ppm DM) can be seen in some forages, typically those from dairy farms that spread copper sulfate footbath contents on their fields. Copper availability is influenced by Fe, sulfur (S) and molybdenum (Mo). High forage Fe (>300 ppm DM) or S (>0.25% DM) will reduce Cu availability. A ratio of Cu:Mo is used to gauge Cu availability. The desired Cu:Mo ratio is between 6:1 and 10:1. Forage Cu:Mo ratio < 4:1 are at risk for inducing Cu deficiency disease conditions [96].

Feed Label Interpretation

The American Association of Feed Control Officials (AAFCO) publishes a model bill every year that outlines suggested feed labeling guidelines as well as officially accepted feed ingredient definitions [166]. Individual state feed control officials can use the AAFCO guidelines or modify as desired. An animal feed label is comprised of many components that provide a range of information related to animal species to be fed the product and purpose; any medications and associated withdrawal times; feeding directions; guaranteed nutrient analysis; ingredient listing and manufacturer's address. Assessment of a product's nutritional value is accomplished in reviewing the guaranteed analysis and ingredient listing.

Guaranteed Analysis

The guaranteed analysis provides a limited nutritional breakdown of the product. The nutrients presented are defined by AAFCO guidelines for a given species. There are no specific labeling guidelines for camelids, though given their sensitivity to Cu it would seem appropriate that sheep and goat product guidelines (includes minimum and maximum for Cu) are used. Product manufacturers can provide more nutrients on their guaranteed analysis than required for a species. The guaranteed analysis presents the product nutrient composition on an as-fed basis. To convert these numbers to a DM basis divide each number by a value between 0.88 and 0.90 as most commercial pellet or grain products will range from 88 to 90% DM. Notice that the values provided are not absolutes, but maximums, minimums or both. This allows for some flexibility for the manufacturer in ingredient inclusion without having to use multiple labels. Feed regulatory officials will routinely analyze products to determine label compliance.

For ruminant products, CP minimum and maximum of nonprotein nitrogen (NPN) equivalent of CP must be provided. If an NPN value is provided, look for urea in the feed ingredient listing. Camelids can utilize NPN like ruminants but there are no validated guidelines for feeding. Crude fat content is supplied as a minimum. Fat has the potential to reduce fiber digestion and should be limited to <5% DM of total diet. A suggested limit for a concentrate product is 8–10% crude fat. The only currently legal measure of fiber on feed labels is crude fiber (CF). Some manufacturers will provide ADF or NDF maximums in addition to CF. Crude fiber is a poor determinant of total dietary fiber for ruminant herbivores and underestimates fiber content. One can estimate the nitrogen free extract (NFE) of the feed by calculation. Most commercial concentrate products contain approximately 9–11% DM ash. To calculate NFE, subtract the DM values of CP, crude fat, CF and ash from 100. The NFE content represents the readily digestible carbohydrates, namely sugars and starch, though this will also include some lignin and hemicellulose due to the inefficient fiber analysis of CF. The ingredient listing will be used to further interpret the calculated NFE value.

Calcium, P and salt are the primary macrominerals provided in the guaranteed analysis. A minimum and maximum is provided for Ca and salt where only a minimum is provided for P. This indicates the product P content could be far higher depending upon ingredients used. Of the trace minerals, Cu and selenium (Se) are required for sheep and goat products though zinc (Zn) is often included. For minerals where a minimum and maximum are provided, use the average value as the typical mineral content. Given Se supplementation is regulated by the Food and Drug Association (FDA) in the U.S., manufacturers often lower the Se content below what could be included as a precautionary effort to avoid violation. Often, these products will have higher suggested feeding rates to provide the allowable supplemental Se. There are no FDA regulations addressing Se supplementation in camelids. The NRC (2007) [56] suggests a daily Se intake of 0.74 mg/day for llamas and alpacas based on one single study [99]. The allowable rate of Se supplementation in sheep and goats is 0.7 mg/day, thus appropriately formulated sheep and goat products may have the minimal amount of supplemental Se required in Se-deficient regions [167].

To calculate mineral contribution from either grain or mineral supplements one needs to be proficient in unit interconversions as minerals may be presented as a percent or parts per million (ppm). To convert a percent value to ppm, the decimal is moved 4 places to the right. For example, 0.009% Se is equivalent to 90 ppm Se. The reverse process, moving the decimal 4 places to the left, will convert ppm to percent (120 ppm Se equals 0.012% Se). To calculate daily mineral delivery from the supplement, multiply the intended daily intake by the concentration making sure of the units. A ppm is equivalent to mg/kg or μg/g and, if daily intake is in pounds, the appropriate conversion factor will need to be applied. Pounds can be converted to kg by dividing by 2.2 lb./kg or grams by multiplying by 454 g/lb. Calculated nutrient intakes can then be compared to suggested daily requirements.

Ingredient Listing

The FDA requires non-pet animal foods to provide the ingredient listing of a commercial product to be in descending order based on as-fed and not dry weight [168]. Pet foods are also required to have this descending listing [166]. The first three listed ingredients essentially comprise the bulk of the product. If cereal grains such as corn, barley, wheat, or oats are the first two or three ingredients and the calculated NFE is >45% then the product contains significant starch content. High starch intake in camelids may predispose to forestomach acidosis [169]. The AAFCO (2019) publication provides allowable ingredient names to be included on product ingredient listings. Some ingredients are identified by specific names (e.g. corn grain, barley spouts, sodium selenite) or collective terms (e.g. plant protein products, fiber products, grain products) [166]. Collective terms allow for ingredient flexibility without needing to alter the feed label; however, they may also lead to greater variation in nutrient content, especially for P. Plant byproducts or fiber products may include ingredients such as distiller's grain, wheat middlings or wheat bran, all of which are high in P. The guaranteed minimum P content may be much lower than the actual content, which could be an issue with Ca and P metabolism and a risk for urinary calculi. Mineral sources may be listed as inorganic (e.g. oxides, chlorides, sulfates) or organic forms (e.g. chelates, proteinates). Other minor ingredients may be pellet binders, antioxidants, preservatives, or stabilizers that help maintain product quality.

Nutrition Related Diseases

Reviews of SAC nutritional diseases have been published [170, 171]. Camelids, both SAC and OWC, are potentially susceptible to the full range of ruminant animal nutritional diseases. The following are the most common camelid nutritional diseases.

Protein-Energy Malnutrition

There are enough new, inexperienced camelid owners that veterinarians may see a starved animal (Figures 3.10 and 3.11). A thick fiber coat masks a loss of condition and, unless the owner can and does routinely feel the backbone, there will be little to indicate weight loss. Many large operations regularly monitor the weights of animals. Owners should be encouraged to weigh their animals regularly, either on a purchased farm scale (Figure 3.12) or at a public weigh station. Another method of assessment is to determine body condition score (BCS). Body condition scoring is used in all species to subjectively assess subcutaneous fat deposits. The process is best performed by observing and palpating selected regions of the body (See Figure 4.3) to assign an overall score from 1 (emaciated) to 5 (morbidly obese) with half scores (Figure 3.13) [172].

Figure 3.10 A thin llama, whose condition is obscured by the fiber coat.

Figure 3.11 The vertebral column of the previous thin llama showing atrophy of the longissimus dorsi muscles.

Having found a thin animal, it is necessary to determine if weight loss is caused by a nutritional problem, chronic dental disease or chronic infectious or parasitic disease [156]. In contrast to chronic disease causes where intake is most often reduced, animals experiencing malnutrition will maintain a good appetite. An evaluation of feeding practices may help in making a diagnosis. Forage testing to identify the NDF and CP content is needed as feeding of mature grass forage is most often the underlying nutritional issue [156]. Appropriate remedial measures should be instituted. Use transfaunation to inoculate C1 for

Figure 3.12 Mechanical scale that can be used for monitoring body weight.

	Score	Animal Description	1 Frontal Profile	2 Rear Profile	3 Spinous to Transverse Process	4 Paralumbar Fossa
Emaciated	**1.0**	No visible or palpable fat or muscle between skin and bones. Ribs, dorsal spinous and transverse processes, and pelvic bones are individually prominent. Extreme loss of muscle mass.	Prominent "V" Keel	Acutely Inverted "V"	Deep depression	Gaunt, tucked-in fossa
Poor	**1.5**					
Thin	**2.0**	Slight cover over bony structure. Ribs, spinous processes still visible and easily palpated as sharp. Less muscle mass loss.	Gradual Flattening of Sternum	Gradual Filling of "V"	Obvious depression	Prominent shelf
Borderline	**2.5**					
Moderate	**3.0**	Overall smooth appearance. Slight fat cover over ribs and other bony processes. Ribs and spinous processes can be palpated with slight pressure. No muscle mass loss present.	Moderate fat	Moderate fat	Smooth concave curve	Slight shelf
High Moderate	**3.5**				Smooth slope	
Excess	**4.0**	Fleshy appearance with visible coverage of fat. Moderate to firm pressure necessary to palpate bony structures under skin.			Nearly flat	No shelf
Fat	**4.5**					Edge barely discernible
Grossly Obese	**5.0**	Excessive fat cover over entire body with smooth, rounded appearance. Bony prominences cannot be palpated, even with firm pressure. Bulging fat pads visible around tailhead.	Sternum Bulging in fat	Inguinal Area Bulging in fat	Rounded	Buried in fat

Figure 3.13 Body condition scoring chart.

camelids not eating. Offer a variety of forage resources to entice intake. At necropsy, a starved animal will lack body fat, and serous atrophy of fat around the heart and kidneys may be seen. Bone marrow will be watery rather than gelatinous. Fatty infiltration of the liver is often observed. Prevention is based on routine feed analysis to evaluate dietary supply relative to requirements and routine monitoring of BCS [173].

Obesity – Hepatic Lipidosis

Probably a more frequent problem than underfeeding, especially among new, overly solicitous owners, is overfeeding. This easily occurs if concentrates are used routinely without regard to actual requirements. Overfeeding is not only costly, but obese animals are more likely to be infertile, develop musculoskeletal problems, experience metabolic derangements and be susceptible to hyperthermia compared to camelids of normal weight (Figures 3.14–3.16) [174]. Higher BCS animals are also more prone to hepatic lipidosis should they go off feed due to heat stress or consuming low-quality forage during late pregnancy or lactation, though excess body condition is not always necessary for hepatic lipidosis [24, 175]. Serum nonesterified fatty acids (NEFA; >0.8 mEq/L) and ß-hydroxybutyrate (BHB; >5 mg/dL) concentrations can be used to assess the potential for hepatic lipidosis [174, 175].

There is no question that some animals become obese more readily than others. Prevention is based on reducing caloric intake or increasing caloric requirement. The latter is less practical unless one increases animal activity. Supplements should be restricted or removed with only mineral supplements. Obese animals should be fed more mature forages but be cautious about CP content and potentially compromising NDF digestibility. In group-feeding situations, be sure that all animals can access feed equally and obese animals are not able to consume extra feed from others. Similar to malnutrition, body weight or BCS should be monitored routinely and diet adjusted to fit individual needs [172].

Figure 3.14 Obese female llama.

Figure 3.15 Roll of fat on the flank/abdomen of an obese llama.

Figure 3.16 Accumulation of retroperitoneal fat from necropsy of an obese llama.

Forestomach Acidosis

A common dietary issue in ruminants is ruminal acidosis resultant from excess consumption of rapidly fermentable NSC (i.e. sugars, starch, fructosans) leading to marked decline in rumen pH. This process has been documented in SAC and found to have a similar pathogenesis [169]. Camelid susceptibility to acidosis has been debated where some have suggested they are less inclined to C1 acidosis due to their inherent greater buffering capacity and VFA absorption efficiency. However, greater efficiency of camelid fermentation and longer C1 retention may contribute to greater susceptibility. In documented cases, based on observed C1 pH measures, consumption of cereal-based grains between 150 g and 1 kg resulted in clinical acidosis presentation. Clinical situations in group feeding suggest underlying issues of social hierarchy and feeding management as contributing factors. Affected animals will present as dehydrated, lethargic and

depressed with metabolic acidosis and electrolyte disturbances reflective of the pH alteration. Treatment with fluid therapy and alkalizing agents generally results in favorable recovery.

Prevention is focused on minimizing intake of high starch feed ingredients and controlling feed accessibility. In group-feeding situations where a concentrate supplement is provided, sufficient linear feed bunk space should be provided so all animals can consume the supplement at the same time. Supplement products should be stored in an animal-safe storage facility to prevent accidental over consumption. Evaluate supplement feeds relative to their starch content either by submitting to a laboratory for sugar and starch analysis or by reviewing the feed label. Concentrate supplements with corn, oats, barley, or wheat as the first three ingredients combined with a CF content <12% are the higher risk products for initiating acidosis with improper feeding management.

Hypophosphatemic Rickets

This disease condition was first identified and characterized by Dr. Fowler [176, 177]. Though phosphorus deficiency was hypothesized as the underlying issue due to the observed hypophosphatemia, further research showed the critical role of vitamin D [123]. Growing crias between three and six months of age and, more commonly born between September and February in the Northern Hemisphere, were most susceptible [178]. A similar syndrome was recognized in the Southern Hemisphere in Australia and New Zealand [179]. There has not been any documentation of this disease condition in South America suggesting the possibility that their natural habitat may provide greater UV light to support sufficient *denovo* vitamin D synthesis. Clinically affected animals presented with shifting leg lameness, hunch-backed stance, unwillingness to move around, reduced growth rate and joint enlargements ultimately leading to angular limb deformities (Figures 3.17–3.19) [176, 177, 179]. A similar process is seen in OWC (Figure 3.20). Diagnosis is based on radiographic evidence of altered growth plates consistent with rachitic changes (Figures 3.21 and 3.22) coupled with hypophosphatemia (< 3 mg/dL [< 0.97 mmol/L) and low serum 25-hydroxyvitamin D_3 (< 25 nmol/L [< 10 ng/mL]) [123].

Treatment and prevention are based on therapeutic or preventive supplementation of vitamin D parenterally (1000–1500 IU/kg BW) or orally (33 000 IU every two weeks or 100 000 IU once monthly) [123, 124]. The suggested daily requirement for vitamin D in camelids (30 IU/kg BW) is much higher than other species potentially due to lower vitamin D bioavailability [56, 124]. Care must be taken in vitamin D supplementation, as toxicity inducing dystrophic soft-tissue calcification is a risk from supplementation exceeding recommendations [180, 181].

Figure 3.17 An alpaca cria with rickets, standing humpbacked and reluctant to walk.

Figure 3.18 An alpaca cria with rickets, spending most of her time recumbent and reluctant to play with other pen mates.

Figure 3.19 Rachitic rosary at the costochondral junction in rickets. Typical enlargement of joints associated with rickets.

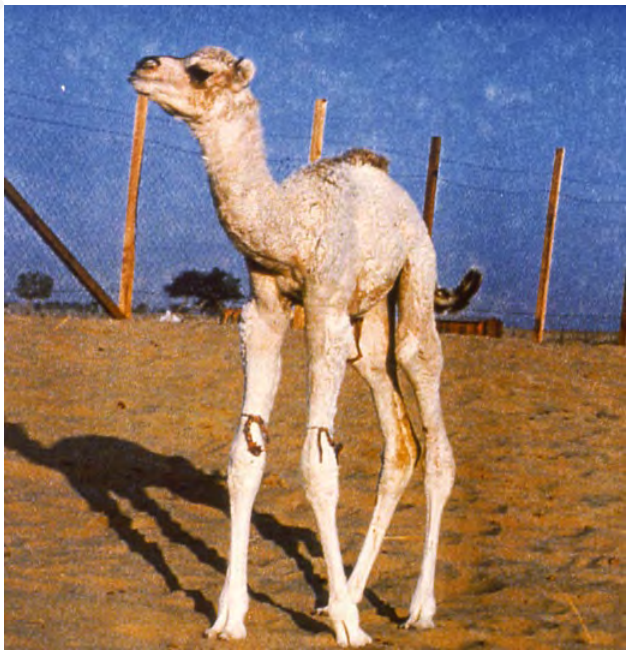

Figure 3.20 Camel calf with rickets.

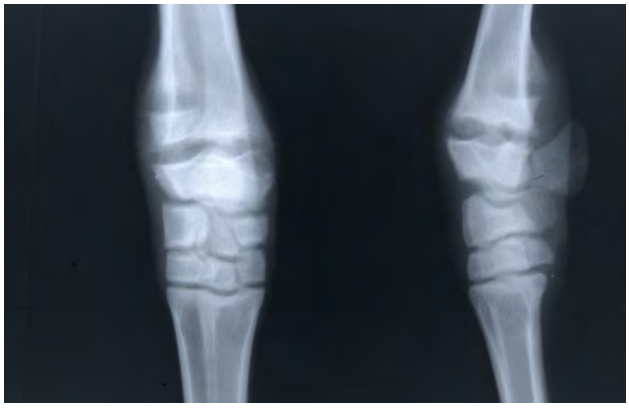

Figure 3.21 Radiograph of the carpus of a rachitic alpaca shows wide physis, metaphyseal flaring, and thinning of the cortices.

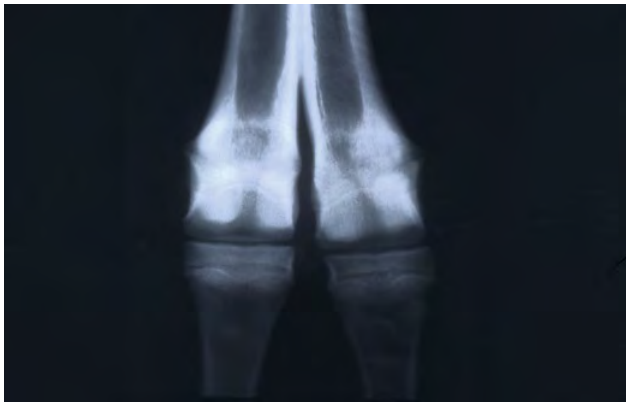

Figure 3.22 Radiograph of the fetlock of a camel calf with rickets. Note the widened physis and flaring.

Selenium (Se) Deficiency/Toxicity

The role of Se as a biologic antioxidant was recognized when its essentiality to glutathione peroxidase was discovered [182]. Historically, Se was more recognized for its toxicity potential, though ruminant animals are less susceptible compared to nonruminant species due to reduction of dietary inorganic Se in the rumen reducing biologic availability [96]. Selenium deficiency is often associated with vitamin E status with nutritional myodegeneration recognized as a deficiency disease process of either nutrient. There are no published reports of Se deficiency in SAC; however, there are reports in dromedary camels [113, 114]. Selenium metabolism in OWC has been investigated relative to maternal-fetal associations, supplementation and disease prevention [100, 183–187].

Selenium deficiency is more often recognized in young growing animals but can be seen in older animals. Clinical presentation will depend upon muscles affected. Clinical signs include acute death with myocardial degeneration, recumbency and inability to stand, weakness, and stiffness [100]. Muscle fiber (i.e. skeletal or myocardial) degeneration due to oxidative damage results in fibrous tissue replacement and calcification. Diagnosis is based on documenting Se deficiency via serum or whole blood concentrations or reduced glutathione-peroxidase (GSH-Px) activity. Camelids have a different distribution of Se in blood compared to ruminants, which may be due to differences in distribution of GSH-Px in erythrocytes and serum. Camelid serum and whole blood concentrations are very similar in magnitude in contrast to cattle where whole blood Se concentrations are 10–50% greater than serum concentrations [188, 189]. Serum Se concentrations <35 ng/mL and 0.8–3.7 ng/mL were associated with affected camel calves and three-year old camels, respectively [100]. Selenium can be supplemented parenterally or orally via diet or slow-release boluses [100, 186, 190]. Suggested daily Se intake in dromedary camels is between 2 and 4 mg [100], which is consistent with the extrapolated Se requirement (6.5–6.8 μg/kg BW) for SAC [90].

Selenium is well-known relative to its potential toxicity in non-ruminant animals [96]. Ruminant animals are considered less sensitive to Se toxicity due to the impact of the rumen environment to reduce dietary inorganic Se availability. Though there are no published reports of Se toxicity in camelids, experimental toxicosis suggests OWC are more sensitive than ruminants with toxicity signs initiated at 8 mg/d (0.05 mg/kg BW) and become severe at 16 mg/d (0.10 mg/kg BW) [100]. This higher rate would suggest 6 mg Se/d would be a potential toxic dose for SAC assuming an equivalent metabolic sensitivity.

Copper Deficiency/Toxicity

Copper serves multiple biologic functions in the body and as a metal cation it has potential for inducing oxidative damage. Like selenium or any other micromineral, dietary copper in

excess or deficient relative to requirement will result in clinical toxicity or deficiency disease, respectively. The sensitivity of sheep to copper toxicosis due to reduced biliary excretion capacity is well documented [96]. Ruminant animals, including camelids, are more susceptible to copper deficiency, compared to nonruminants, due to unique dietary nutrient-rumen interactions reducing copper availability [191].

Copper deficiency in llamas presenting with anemia and poor condition was documented by low serum Cu concentrations [105]. Anemia due to Cu deficiency is a common clinical manifestation due to Cu's role in iron metabolism. Other reports suggested a Cu deficiency role in hind-limb ataxia and posterior paresis similar to "swayback" disease of sheep; however, serum Cu concentrations were not considered deficient and affected animals did not respond to Cu supplementation [106, 107]. A 6-month-old llama presenting with ascending paralysis was documented to have deficient liver and kidney Cu concentrations [192]. Copper deficiency has been reported in OWC presenting with similar posterior paresis [193]. Issues of high dietary Mo and S, interfering with Cu availability in OWC, have also been recognized [193].

Published reports and anecdotal observations from clinical cases support that SAC are sensitive to Cu toxicity, but not as sensitive as sheep [101–104]. In all cases either serum (2.2–5.7 μg/mL; reference: 0.4–0.5 μg/mL) or liver (640–1700 μg/g dry weight) Cu concentrations exceeded laboratory reference values, consistent with toxicity [101–103]. Hemolysis and icterus, commonly observed in Cu-toxic sheep, was not associated with SAC Cu toxicity cases. Necropsy findings consistently showed moderate-to-severe hepatic necrosis. Kidney and liver tissue should be evaluated for Cu toxicosis for the best diagnostic outcomes. In these case reports, dietary Cu ranged from 33 to 36.6 mg/kg DM and the dietary Cu:Mo ratio exceeded 12 : 1 [101–103]. A dietary Cu:Mo ratio between 6- and 10-1 is recommended. Forage Cu content should also be evaluated beyond any supplements as it is the greatest proportion of the SAC diet. Clinical cases of Cu toxicity in alpacas were found to be due to excessive forage Cu (22–26 mg/kg DM) (Van Saun unpublished data). Forages with these excessively high Cu concentrations are unusual but may be the result of forage harvested following discharge of used copper sulfate footbath contents onto fields on dairy farms. No published reports documenting Cu toxicosis in OWC were identified; however, it would be assumed there is a similarity in response across camelids.

Zinc-Responsive Dermatitis

Camelids have lower serum zinc (Zn) concentrations compared to other ruminant species [119, 193–195]. A Zn-responsive skin inflammatory condition has been well-recognized in SAC [110, 111]. Classically, Zn deficiency presents with parakeratosis, a hyperkeratotic process where the nuclei are retained within keratinocytes [96]. Skin lesions are observed more commonly in colored fleece animals on hairless regions of the body (e.g. axilla, inguinal region, medial thighs and ventral abdomen) [111]. Lesions are described as a skin thickening with tightly adhering crusts. Supplementation with pharmacologic doses of Zn over a 2- to 3-month period is recommended for remission. Ensuring sufficient vitamin E status may also be beneficial. Diagnosis may result more from response to therapy rather than serum Zn concentrations as these are not always diagnostic. Clauss et al., did report low serum Zn concentrations in llamas (0.22 μg/mL) and alpacas (0.17 μg/mL) with clinical signs and affected animals responded to Zn supplementation [110]. Forage Zn concentration is mostly deficient to marginal [151].

Treatment or prevention of this condition is potentially achieved with Zn supplementation. The challenge is understanding the recommended amount of supplementation and source. Forms of Zn used typically are zinc oxide, zinc sulfate or zinc methionine. Confusion comes in whether supplementation recommendations are for the compound or the amount of delivered Zn as these sources vary in their Zn content. One gram of zinc oxide (78% Zn) or Zn sulfate monohydrate (36.3% Zn) would provide 780 and 36.3 mg elemental Zn, respectively [56]. If a recommendation is for 1 g/d or 2–4 g/d, these are relative to the entire compound. Use of Zn methionine is even more confusing as there are multiple forms of this Zn-amino acid complex. In the U.S., the company ZinPro (Eden Prairie, MN) produces Zn methionine in various concentrations, ZnPro® 40, 120, and 180 as well as Zn-Pro 100 in other countries (https://www.zinpro.com/products/original-line/zinpro). The number associated with the product is the amount of elemental Zn concentration in g/kg compound. To convert to a % Zn divide each of these values by 10; therefore, the products contain in ascending order 4, 10, 12, and 18% Zn. A typical therapeutic dosing suggested is 2–4 g/d, which would represent the total product as the absolute requirement for Zn is between 48 and 78 mg/d for a 60-kg pregnant or lactating alpaca [90]. Thus, 2–4 g of these different products would deliver 80–160 mg (Zinpro-40), 200–400 mg (Zinpro-100), 240–480 mg (Zinpro-120) and 360–720 mg (Zinpro-180) Zn. Excessive Zn intake can inhibit Cu availability as well as other divalent cations and become a prooxidant [96].

Plant Poisonings

Several poisonous plants have been identified as toxic to camelids (Chapter 22). In connection with feeding, it is important to recognize that processed feeds present a special hazard for all animals. Given a choice, a camelid may not select and eat a toxic plant. However, when included in a cube or a pellet, poisonous plants may be undetectable. Unfortunately, cubes and pellets may be made using poor quality forage or hay containing poisonous plants. The reputation of the supplier is the only safeguard available.

Table 3.A.1 Conversion of conventional units to Système International units for blood, plasma, serum. *Additional conversion factors allow for more options on conversion of units.

Analyte	Conventional units	Multiply by		SI units
		Conventional to SI units	SI units to conventional	
All enzymes	U/L	0.0167	60	μKat/L
Albumin	g/dL (g%)	10	0.1	g/L
Copper	μg/dL (μg%)	0.1574	6.35	μmol/L
Iron	μg/dL (μg%)	0.1791	5.58	μmol/L
Lead	μg/dL (μg%)	0.04826	20.72	μmol/L
Magnesium	mg/dL (mg%)	0.4114	2.43	mmol/L
Manganese	ng/mL	18.2	0.055	nmol/L
Zinc	μg/dL (μg%)	0.1530	6.54	μmol/L
Calcium	mg/dL (mg%)	0.2495	4.01	mmol/L
Phosphate	mg/dL (mg%)	0.3229	3.10	mmol/L
Energy	calorie	4.184	0.239	Joule (J)

*Note: Additional conversion factors:
1 ppm (part per million) = 1 mg/kg = 1 μg/g = 0.454 mg/lb. = 0.91 g/ton
1 ppm = 0.0001% = 0.002 lb./ton
0.1 ppm = 0.000011% = 0.0002 lb./ton = 0.09 g/ton = 0.0454 mg/lb.
1 mg/g = 100 ppm
0.001 ppm = 1 ppb (part per billion)
1 fl oz. = 29.6 mL
1 oz. = 28.35 g
1 kg = 2.2 lb.
1 pt. = 473.2 mL = 1 lb. (approx.)
1 qt = 946.4 mL = 1 l (approx.)
1 microgram (mcg or μg) = 0.000001 g
1 nanogram (ng) = 0.000,000001 g
1 picogram (pg) = 0.000,000,000001 g.

Table 3.A.2 Glossary of terms used in feeding and nutrition.

Term	Definition or Descriptor
Joule	The work done when the point of application of a force of one newton is displaced through a distance of one meter in the direction of the force.
One joule	0.239 cal
Kilojoules	1000 joules
Megajoules	One million joules
Energy density	The amount of energy in a feed (Kcal/kg or MJ/kg).
Calorie	The amount of heat required to raise the temperature of 1 g of water from 14.5 °C to 15.5 °C at standard pressure. The standard calorie used for expressing the chemical energy in feeds.
One small calorie	4.184 joules
Kcal	1000 small calories
Mcal	One million small calories
Dry matter	The nutrient composition of a feed minus the moisture.
As fed	The nutrient composition of a feed as consumed as hay, pasture, or a concentrate. Includes moisture.
Metabolic weight	Body weight in kilograms raised to the 0.75 power $\left(W_{kg}^{0.75}\right)$.
Basal metabolic rate	The energy used by an animal to maintain body temperature in a thermoneutral environment (Kcal d = C × $W_{kg}^{0.75}$), C = A constant for a group of animals.
Gross energy	The amount of heat that is released when a substance is completely oxidized in a bomb calorimeter containing 25–30 atm of oxygen.
Digestible energy	Gross energy minus fecal energy.
Metabolizable energy	Food intake gross energy, minus fecal energy, minus energy in the gaseous products of digestion, minus urinary energy.
Net energy	Metabolizable energy minus losses from digestive fermentation and nutrient metabolism.
Parts per million (ppm)	mg/kg
Concentrate	A feed used with another feed to improve the nutritive balance of the total and intended to be diluted and mixed to produce a supplement or a complete feed.

Table 3.A.2 (Continued)

Term	Definition or Descriptor
Forage	Aerial plant material, primarily grasses and legumes containing more the 18% crude fiber on a dry basis, used as animal feed.
Supplement	A feed used with another feed to improve nutritive balance or performance.
Photosynthesis	The formation of carbohydrates from the action of sunlight on plant chlorophyll, plus water from the soil and carbon dioxide from the atmosphere ($6CO_2 + 12\ H_2O$ + Action of sunlight on chlorophyll = $C_6H_{12}O_6$ (carbohydrate) + O_2).
Halophyte	A plant that tolerates high salinity in the soil, in irrigation water or salty spray, such as near an ocean.

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4

Physical Exam and Diagnostics

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The basic procedures of clinical diagnosis in camelids are like those used for livestock, horses, and even small animals, but anatomic differences necessitate some modifications. Unique characteristics and normal data are discussed.

Physical Exam

When examining sick animals, a good history (anamnesis) should be taken. Owners should be questioned regarding the duration of clinical signs as well as the onset of disease. Any response to previous therapy should be ascertained. The physical exam should commence with an observation of the animal from a distance if possible.

A physical examination is required for obtaining insurance and interstate or international health certificates, performing a pre- or post-purchase examination, conducting a soundness examination, or conducting an examination on a sick animal. The concepts are the same as for other species of domestic animals. Table 4.1 is a checklist of basic information that should be obtained on every examination. Table 4.2 is a checklist for a physical examination.

Physical examination should be systematic, beginning with simple, non-invasive, and inexpensive diagnostics and advance in a methodical manner to more invasive procedures. Every physical examination should commence with an assessment made by observing the animal. Observing the animal both at rest and in motion can be very valuable to the overall assessment. Unfortunately, depending on the circumstances, the examiner might not be afforded this opportunity. The attitude, mentation, and general demeanor should be the first assessment made. The mere catching and restraint of an animal may change the animal's attitude. Camelids are stoic animals, and they can mask disease in response to stress which can make diagnostics challenging. It may be beneficial to have a second "observation" period following the physical examination where the animal is placed alone in a box stall and observed from a distance. These observations are typically general findings but might also point to specific disease conditions. The confirmation, posture, and gait are important observations when presented with animals for lameness evaluation, and it is important for camelid owners to consider confirmation in any breeding program. Additionally, watching the animal eat, chew its cud, urinate, and defecate can provide valuable insight. Observing the animal's environment or gathering information from a patient history may prove to be valuable.

South American camelids (SACs) are generally docile and often minimal facilities are necessary to perform non-invasive procedures like examination. Due to the social nature of the species, working with them as a group can lead to examination on a calmer animal. Working with the animals in a small enclosure can limit their movement and make examination easier. Most SACs will respond to a halter [1] (see Chapter 2 on general handling techniques).

Conformation

All species of SACs are similar in conformation, with differences primarily in size. Camels are larger still and different restraint procedures are required. Veterinarians conducting lameness examinations should have an understanding of the animal's confirmation. The previous edition of this text devoted an entire chapter to body confirmation and gaits and the reader interested in a more in-depth description is referred there. The high-head carriage is immediately noticeable, and it contributes to the functional balance of these unique animals. In a healthy llama, the neck is held almost vertical, while the neck carriage of an alpaca is at approximately 70°. With weakness or depression, the head is held lower. Due to

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Table 4.1 Checklist of basic data to collect for any examination.

Identity of the individual Date _____

Name _____

Registration number _____, Microchip number _____

Owner's name and address _____

_____

Age _____, Sex _____, Weight _____

Color: Body _____, Head _____, Neck _____,

Legs _____; Distinguishing markings _____

Sire _____, Dam _____

History

How long has the animal lived at the present premises? _____

Previous owner(s) _____

_____

Location of previous farms _____

_____

Type of housing: Barn _____, Pens _____, Pasture _____

Other animals on the farm: Horses _____, Cattle _____, Sheep _____, Goats _____

Other _____

How many other llama/alpacas on the farm? _____

Previous illness in this animal _____

_____

Basic information

Body temperature _____°F, _____°C

Heart rate _____, Respiratory rate _____

Body condition: Thin _____, Obese _____, Normal _____, Body score _____

Temperament

Easily caught: Yes _____, No _____

Easily haltered: Yes _____, No _____

Table 4.2 Checklist for physical examination of llamas and alpacas.

Identification Date _____

Name _____

Registration number _____, Microchip number _____

Color _____, Age (yrs) _____

Sex: Male _____, Female _____, Neutered _____

(*Continued*)

Table 4.2 (Continued)

Movement

Normal (Y/N)

Free-moving gait _____, Stiff gait _____, Lameness _____

Leads easily: Yes _____, No _____

Front limbs

Normal (Y/N)

Front view: Base wide _____, Base narrow _____; Carpal valgus: Slight (<5°) _____,

Moderate (<10°) _____, Severe (>15°) _____, Bowed out at carpus _____, Splay feet _____,

Pigeon-toed _____, Polydactyly _____, Syndactyly _____

Side view: Camped forward _____, Camped behind _____

Angulation: OK _____, Too straight _____, Too flexed _____, Buck-kneed _____,

Calf-kneed: Slight (<175°) _____, Moderate (170–175°) _____, Severe (<165°) _____

Cocked ankle (pastern angle >90°) _____, Down in fetlock (pastern angle <30°) _____

Comments

Rear limbs

Normal (Y/N)

Rear view: Base wide _____, Base narrow _____

Cow-hocked: Slight (<5° rotation) _____, Moderate (5–10°) _____, Severe (>10°) _____

Bowed out at hock _____, Splay footed _____, Pigeon-toed _____

Side view: Camped forward _____, Camped rearward _____

Angulation: Post-legged _____, Too much flexion _____

Sickle-hocked: Slight (hock angle <135°) _____, Moderate (hock angle <130°) _____, Severe (hock angle <125°) _____

Cocked ankle (pastern angle >90°) _____, Down in fetlock (pastern angle <30°) _____

Comments

Head

Normal (Y/N)

Wry face: Slight (<2°) _____, Moderate (2–5°) _____, Severe (>5°) _____

Nostrils: Air movement both nostrils _____

Ears: Normal _____, Long _____, Short _____, Gopher _____, Frostbitten _____, Curled (fused) _____

Eyes: Entropion _____, Ectropion _____, Laceration _____, Tearing (evidence of blocked tear duct) _____,

Corneal opacity _____, Cataract _____, Dilated pupil _____, Constricted pupil _____, Evidence of blindness _____

Teeth: Superior brachygnathism (undershot jaw): <0.5 cm _____, >0.5 cm _____

Inferior brachygnathism (parrot mouth, overshot jaw): <0.5 cm _____, >0.5 cm _____

Retained deciduous incisors _____, Permanent canine teeth erupted _____

Comments

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Table 4.2 (Continued)

Neck and Body

Normal (Y/N)

Throat latch: Swelling _____

Cervical spine: Symmetrical _____, Scoliosis _____

Thoracic and lumbar spine: Scoliosis _____, Lordosis _____, Kyphosis _____

Tail: Twisted tail _____, No tail _____

Height, alpacas: Juvenile <74 cm (29 in.) _____, Adult <86 cm (34 in.) _____, Adult >102 cm (40 in.) _____

Weight, alpacas: Juvenile: >48 kg (106 lb) _____, <48 kg _____

Alpaca adults (central incisor erupted): Must be above 48 kg (106 lb) sheared or 50 kg (110 lb) unshorn _____

No height or weight standards for llamas

Comments

Reproductive system

Normal (Y/N)

Male

Testicles: Both testicles in scrotum _____, Cryptorchid _____

Size (length): **llama** < 3.4 cm (1 yr) _____, <4.2 cm (2.5 yr) _____

alpaca: <1.8 cm (1 yr) _____, <2.4 cm (2 yr) _____, <3.0 cm (adult) _____

Consistency: Hard _____, Too soft _____

Scrotal edema _____

Female

Position of vulva: Vulvar slit <0.6 cm _____, Vertical _____, Horizontal _____

Clitoris enlarged (evidence of intersex) _____

Comments

Pulmonary system

Normal (Y/N)

Respiratory rate: _____ bpm; Abnormal lung sounds _____

Exercise tolerance _____; Upper airway sounds _____

Comments

Cardiovascular system

Normal (Y/N)

Heart: Murmur _____, Arrhythmia _____, Heart-beat rate _____ bpm

Comments

Miscellaneous characteristics

Body temperature: _____°F, _____°C

Teats: Normal (4) _____, <4 _____, >4 _____

Hernias: Umbilical (>1 cm) _____, Scrotal _____

Toenails: Normal _____, Elongated _____, Curled _____, Abnormal horn _____

Other defects: _____

their long neck, in severely weakened SACs, the nuchal ligament overpowers the muscles that cause ventroflexion of the neck, and the head and neck are frequently positioned over the thorax in an opisthotonos position (Figure 4.1).

The animal's gait should be assessed as part of confirmation. Camelids have four natural gaits: the walk, pace, trot, and gallop. More details on the gait of camelids are included in previous editions of this text [2].

Body Condition and Size

Physical examination should include an evaluation of body weight and body condition. Body weight determination is critical if therapeutic intervention is going to involve administration of pharmacologic agents. Table 4.3 gives approximate sizes for domestic camelids. Estimating weight by visual observation and palpation only has been shown to be very inaccurate even when performed by experienced camelid owners and veterinarians. These estimates tend to underestimate the true body weight of the animal, especially when they are shorn [3]. The mature body size of male and female SACs vary little [4], but this is not true of Old World camelids. There is a strong correlation between body weight and thoracic circumference measured directly behind the forelimbs (heart-girth) [5, 6] (Figure 4.2). Using a heart-girth tape to make weight estimates is reliable but

Figure 4.1 Severely weakened alpacas commonly develop opisthotonos. *Source:* Photo courtesy of Dr. Grace VanHoy.

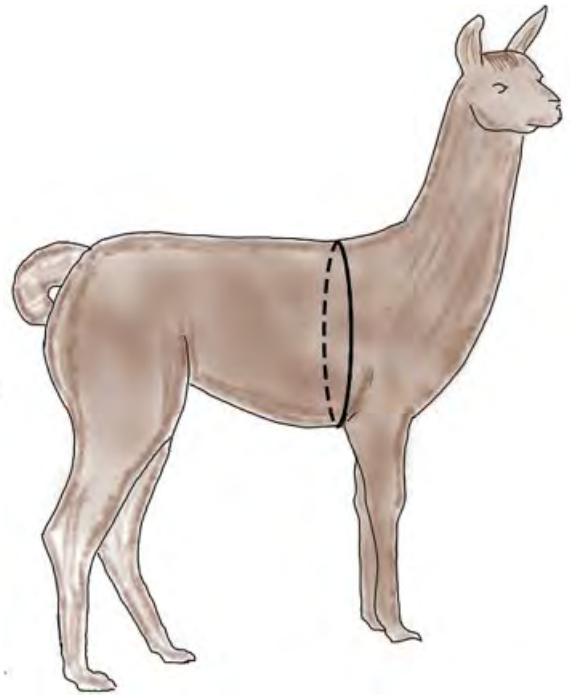


Figure 4.2 There is a strong correlation between thoracic circumference measured directly caudal to the elbow, represented by the line in the image, and body weight for llamas.

Table 4.3 Typical weight and height of camelids.

	South American Camelids				Camels		
	Vicuña	**Alpaca**	**Guanaco**	**Llama**	**Dromedary**	**Bactrian**	**Wild Bactrian**
Adult weight (kg)	45–55	55–90	100–120	113–250	661–1432[a]	992–1543	992–1521
Birth weight (kg)	4–6	6–9	8–15	8–18	57–100	55–120	Unknown
Adult height at withers (cm)	86–96	76–96	110–115	102–119[b]	71–83	71–77	70.8–78.7

[a] Males, castrated as juveniles, may grow taller and larger.
[b] Some imports may be as short as 97 cm.

one study showed that it tended to underestimate the weight by approximately 17.5% [3].

A llama's body weight can be estimated by the following equation [4], where body weight is in kg and thoracic circumference is in cm.

$$\text{Body Weight} = \left(1.005 \times 10^{-3}\right) \times (kg) \left(\frac{\text{Circumference}}{cm}\right)^{2.424}$$

Because body weight can be impacted by age, frame size, musculature, and pregnancy status, body condition scoring is a better way to assess nutritional status of the camelid. Determination of body condition score (BCS) is important at times other than when conducting a physical examination. It is important to assess body condition during pregnancy as well as early to mid-lactation [7]. Different body condition scoring systems have been used in camelids and have been described as the 10-point system [8] and 5-point scales adapted from small ruminant scoring systems [7]. Non-pregnant and non-lactating animals should have a BCS that is slightly lower (thinner) than pregnant and lactating animals which require greater energy demands [7]. The BCS is a subjective assessment and may vary with the observer, but a systematic assessment and experience will make the score more accurate and consistent. Evaluate the withers and loin area just caudal to the ribs and cranial to the pelvis (Figure 4.3). Evaluate for the presence of fat tween boney projections from dorsal spinous process and transvers processes. Determine how much shelf is formed by the transverse processes. Thin animals will have a prominent dorsal spinous process due to the atrophy of the epaxial muscles and lack of fat. Obese animals will have difficult to palpate dorsal spinous processes. Evaluate the area of the thorax just caudal to the point of the elbow for detection of the rib. Obese animals will also have excessive fat in the brisket, inguinal, and perineal areas, and these areas can be assessed to help separate the upper BCSs [8]. Care must be taken with extremely muscular animals, as muscle bulging can be apparent in these areas. Some fat females have excessive fat in the perineal region adjacent to the vulva. The paralumbar fossa is another place to evaluate; thin animals will have a "sucked-in" paralumbar fossa while fatter animals will have a paralumbar fossa that projects out and the transverse processes will be difficult to identify [7]. If scoring on a 10-point scale, 1 = thin, 5 = ideal, 10 = obese; if scoring on a 5-point scale, 1 = emaciated, and 5 = obese.

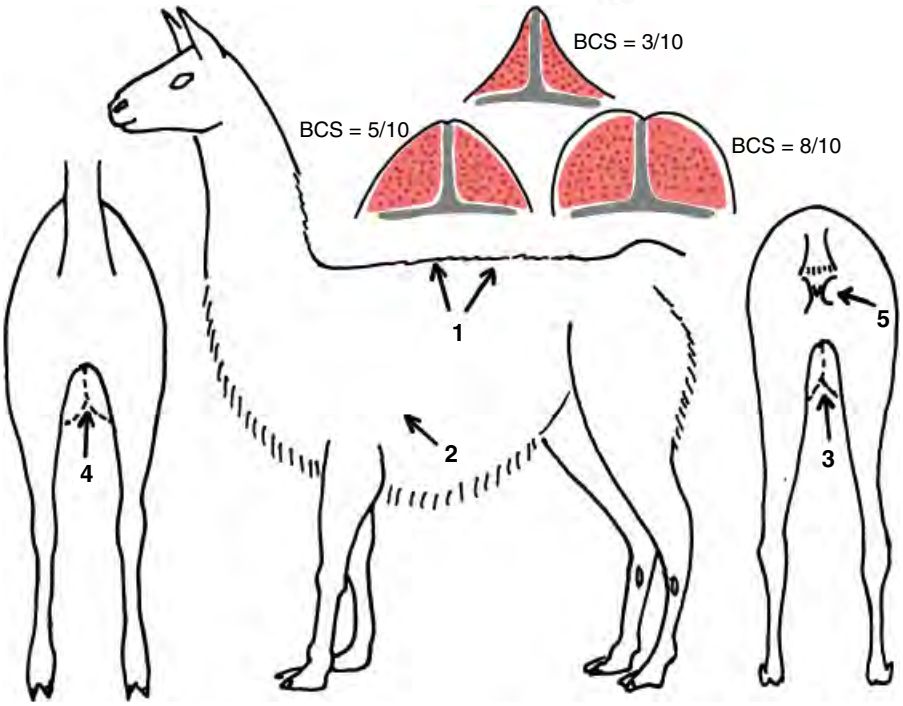


Figure 4.3 Key locations for evaluating body condition, in order of examination: (1) withers and loin, (2) over the thorax in the fiberless area behind the elbow, (3) between the rear legs, (4) chest (between the front legs), and (5) perineum lateral to the vulva. If scoring on a 10 point scale, 1 = thin, 5 = ideal, 10 = obese. Inset is a cross-section of spine taken in the thoracolumbar region. Thin animals will have a prominent dorsal spinous process due to atrophy of the epaxial muscles and lack of fat. Obese animals will have dorsal spinous processes that are difficult to palpate and excessive fat in the brisket, inguinal, and perineal areas.

Body Temperature

The resting body temperature of adult camelids varies depending on the environment. Normal body temperatures of neonate camelids fluctuate across a wider range because thermoregulatory mechanisms are less developed than those of adults. SACs evolved in harsh, cool climates and are well able to adapt to cold. Extremely hot and humid climates are less well tolerated, and special cooling systems should be incorporated into management programs to help them thermoregulate. Animals not able to thermoregulate in hot environments are prone to heat stress (see Chapter 8).

During hot summer seasons, some llamas are able to allow body temperatures to elevate and remain at 40 °C (104 °F). Though heavy fleece can be protective by insulating from hot summer sun, shearing is recommended to allow camelids to more efficiently thermoregulate [9].

The llama forced to remain recumbent because of trauma or disease is more subject to heat stress, because there is a fleece-free area of the body and the site for heat dissipation. If the llama is continually recumbent, neither normal nor fever-induced body heat can be dissipated (See Chapter 8).

The dromedary camel is able to endure diurnal fluctuations of body temperature from 36.5 to 42 °C (97.7 to 107.6 °F). The body acts as a heat sink during the heat of the day, thus conserving vital water that would otherwise be lost through evaporative cooling. During the cool night, body heat is dissipated by conduction and radiation. It should be obvious that evaluation of a fevered state in the dromedary is difficult. The vital parameters for camelids are listed in Table 4.4.

Cardiac Assessment

The heart rate of camelids are listed in Table 4.4. The heart is accessible for auscultation as in other mammals. By reaching under the fleece at the elbow, the stethoscope may be placed on a fleece-free area caudal to the triceps, which allows for both cardiac and thoracic auscultation (Figure 4.4).

Figure 4.4 Auscultation of the heart is best accomplished by placing the stethoscope on the fleece-free area just caudal to the triceps muscle.

Table 4.4 Physical exam parameters of camelids.

Parameter	New World camelids	Old World camelids
Body temperature, adults in moderate environment (°C/°F)	37.5–38.9/99.5–101.5	36–37/96.8–98.6, morning; 38/100.4, afternoon
Body temperature, adults in hot summer afternoon (°C/°F)	Up to 40/104	36.5–38/97.7–100.4, morning; 39.5–40.5/103.1–104.9, afternoon; 42/107.6 maximum
Body temperature, crias/calves	0.5–1 °C higher than adults	0.5–1 °C higher than adults
Heart rate, beats per minute	60–90	32–36, relaxed; 44, nervous
Respiratory rate, breaths per minute	10–30	5–8, cool weather; 10–12, summer
Feces	Pellated, use dung pile	Pelleted, void at random
Urine	Clear, pale yellow to amber	Clear, pale yellow to amber, dark brown and syrupy when dehydrated

Thoracic Cavity Assessment

The thoracic area for auscultation in SACs is bound by the caudal border of the triceps muscle, a line extending caudally for 20–25 cm (8–10 in.) below the top of the ribs and angled back to the olecranon. The diaphragmatic reflection is more caudal than this area, but lung sounds are not heard in the caudal thorax. Tracheal auscultation and respiratory rate is best established by placing the stethoscope over the trachea at the thoracic inlet. The landmarks for thoracic auscultation in the camel are similar to those of SACs with a larger area than in SACs (Figure 4.5).

The resting respiratory rates of camelids are listed in Table 4.4. Normal lung sounds are difficult to hear in camelids. With excitement and more rapid breathing, the sounds are vesicular rather than bronchiolar.

Abdominal Auscultation

Similar to the rumen, the camelid's first stomach compartment (C1) lies predominately on the left side of the abdomen; therefore, gastrointestinal (GI) sounds associated with C1 motility are going to be heard best on the left hemiabdomen. A fleece-free abdominal area is located just cranial to the thigh muscles of the hind limb and this is a good place to achieve good skin contact with the stethoscope for abdominal auscultation. To expose the area for auscultation, it is necessary to reach under the fleece in what would be the flank area in other species and lift the fleece up.

There are marked differences between rumen and C1 auscultation. Rumen contents consist of a dorsal fiber mat and a ventral fluid layer, while camelids have C1 contents that are homogenous and less fibrous. Ruminal sounds are produced from the dorsal fiber mat of the rumen rubbing over the ruminal papilla and are likened to distant rolling thunder. The C1 compartment is devoid of papilla and C1 contractions are less forceful compared to ruminal contractions. C1 contractions are rarely palpable as are true ruminations, and C1 motility produces sounds that are likened to gas and liquid churning. Normal C1 motility rate is three to four contractions per minute; the frequency increases slightly after feeding.

Eye

Similar to other herbivores, camelids have eyes positioned on both sides of their head allowing them to see approaching predators while they graze. The iris of SACs has a large corpus nigrum (granula iridica) on the dorsal aspect and a smaller one on the ventral. The function of the corpus nigrum is unknown. Unilateral or bilateral nonpigmentation of the iris (blue eye, glass eye, wall eye, or watch eye) is seen. The retina is characterized by a pronounced vascular pattern and no fovea. There is no tapetum lucidum. See Chapter 18 for more details on ocular examination, anatomy, function, and abnormalities.

A slight ectropion of the lower eyelid may be seen on some individuals when they are excited, frightened, or agitated. This condition should not be confused with anatomic ectropion, which may be either congenital or acquired.

Ear

The ears of llamas are long, variably shaped pinnae that are used to express mood. Alpacas have shorter spear-shaped ears. Close inspection of the pinna and external ear canal is difficult because llamas and alpacas resent having their

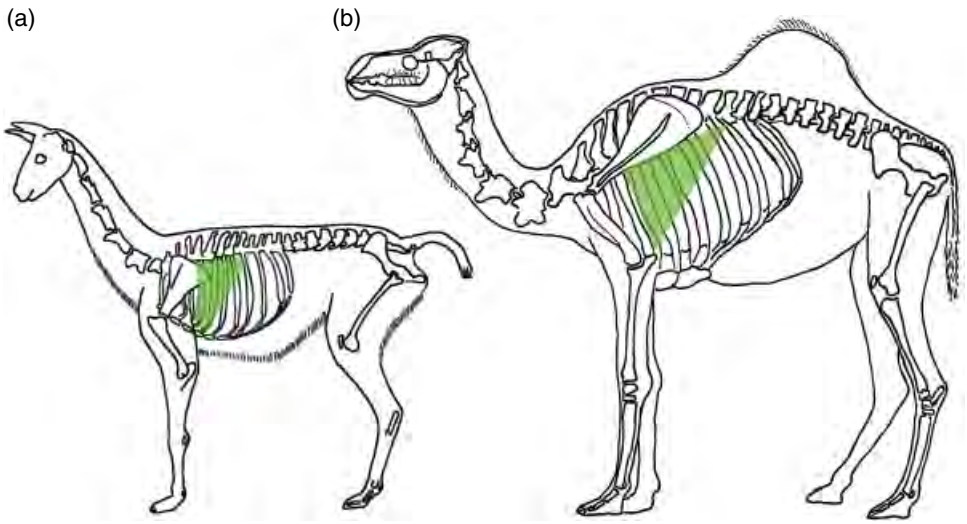


Figure 4.5 Diagram showing areas of thoracic auscultation in the llama (a) and the camel (b).

ears touched. Camels have even shorter ears but, on close inspection, they too are indicative of the mood of the camel. The external ear canal of SACs is small (3–5 mm in diameter), and a bend along the course of the canal precludes visualization of the tympanic membrane in many adults [10, 11].

Mouth

SACs are unable to open the mouth wide enough to make possible a more than cursory examination of the oral cavity. The mouth of the camel opens more widely. The incisor and canine teeth can be viewed and should be checked in every physical examination. Congenital malocclusion is prevalent in SACs (Chapter 21). The tongue rarely protrudes from the mouth. Nonpigmented gingiva may be used to determine a capillary refill time.

Diagnostic Procedures

Jugular Venipuncture

Jugular venipuncture can be performed cranially (high) on the neck near the ramus of the mandible or more caudally in the mid- to lower-cervical region [12] (Figure 4.6). Venipuncture can be challenging in camelids, and neither location is ideal for every situation. Motion of the patient, the dense fiber coat, the thick skin, the absence of a true jugular furrow and the minimal potential space within the cervical region for the vein to distend (compared to ruminants), the jugular vein's apposition to the carotid artery, the relatively small nature of the vein (compared to true ruminants), and the difficulty in restraining camelids without interfering with the procedure are factors that increase the difficulty with performing jugular venipuncture in camelids. As an evolutionary adaptation to protect against exsanguination from bites by males during fighting, SACs have evolved a deeper jugular vein that is partly protected by a ventral extension of the transverse process of the cervical vertebrae and the sternocephalicus muscle (Figure 4.7) making their jugular vein less apparent. Camels have larger and more distensible jugular veins compared to SACs, but their stature and the decreased ability to restrain these animals can add difficulty with the procedure (see Section 0).

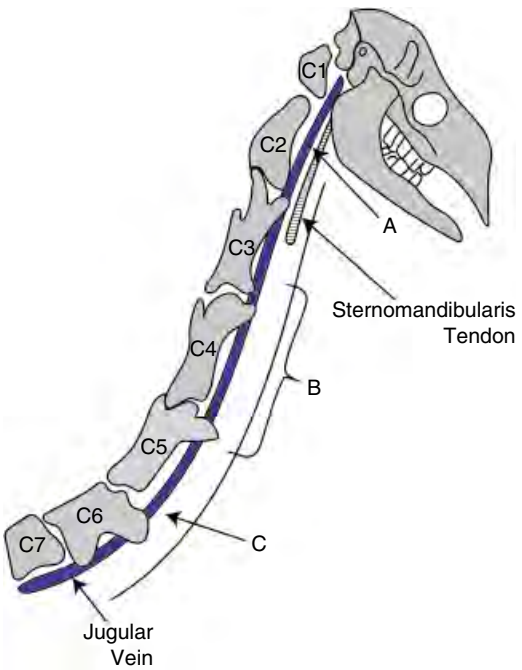


Figure 4.6 Sites for jugular venipuncture in a camelid. (A) High site, (B) Mid Cervical site, (C) Lower site.

Restraint and good body position are critical to successful venipuncture. Though many patients are used to being restrained by a halter, this alone is not an adequate restraint for invasive veterinary procedures. Often, proper facilities to restrain animals are not available, or the animals are unaccustomed to being restrained in these facilities. Yearlings are often more difficult to restrain for venipuncture than neonates or adults, because many are unaccustomed to being tied or having their heads restrained. They frequently rear up and struggle vigorously. Chutes and stocks are not usually designed for juveniles. See Chapter 2 for more information on safe and effective handling methods for venipuncture.

Inadvertent carotid penetration is more common in SACs than in ruminants. The jugular vein and carotid artery are in close apposition for much of their length with the carotid artery lying immediately deep to the jugular vein (Figures 4.8). Carotid penetration often results in hematoma formation around the vessels which can lead to respiratory compromise due to tracheal compression. Hematoma formation makes subsequent jugular venipuncture even more challenging. Caution must be practiced when injecting intravenous (IV) medications in the jugular vein, as inadvertent intra-arterial injection is possible. The carotid artery will have much higher and pulsatile blood pressure, whereas the jugular vein will normally not spontaneously bleed unless occluded proximally. This assessment can be used to determine that the carotid artery has not been penetrated and is especially useful when placing an IV catheter or performing an IV injection. If inadvertent carotid puncture occurs, apply pressure over the carotid puncture site for at least five minutes prior to continuing. A firm neck wrap can also be placed around the neck to minimize hematoma formation.

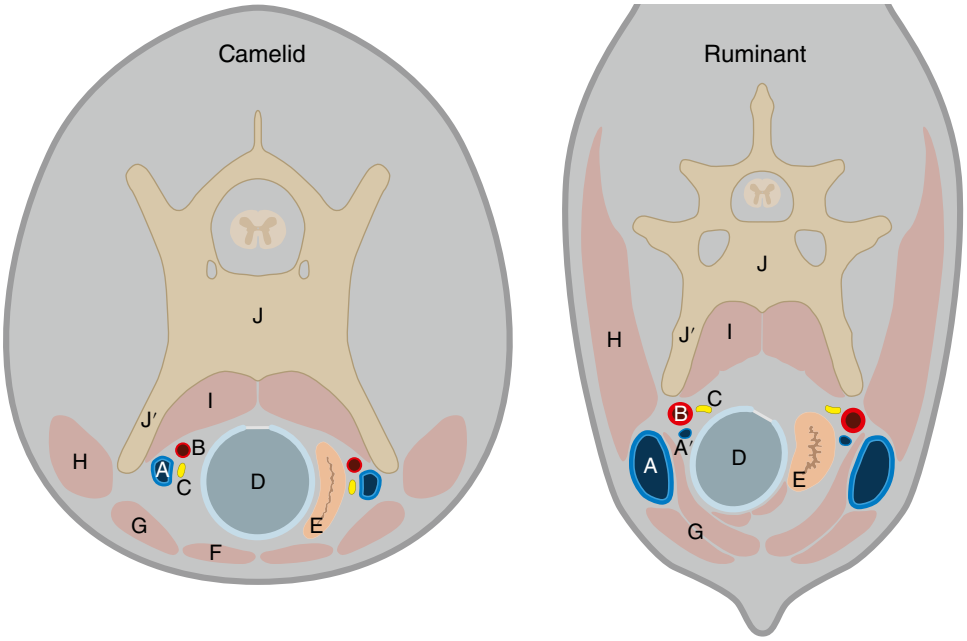


Figure 4.7 Cross-section of the cervical region of camelid (left) and true ruminants (right). Note that the transverse processes engulf the cervical vasculature and the jugular vein (A) is in close apposition to the carotid artery (B). The size of the jugular vein and carotid artery are close in size in camelids, making inadvertent carotid penetration easier in camelids than in true ruminants. Other labels include the vagosympathetic trunk (C), trachea (D), esophagus (E), sternothyrohyoideus muscle (F), sternomandibularis muscle (G), brachiocephalicus muscle (H), longus colli muscle (I), vertebrae (J), and the ventral extension of the transverse process of the vertebra (J′).

Figure 4.8 Anatomic dissection of the cervical region. The jugular vein and carotid artery run in apposition for much of its length. (A) Omohyoideus muscle, (B) common carotid artery, (C) jugular vein, (D) tendon of the sternomandibularis muscle.

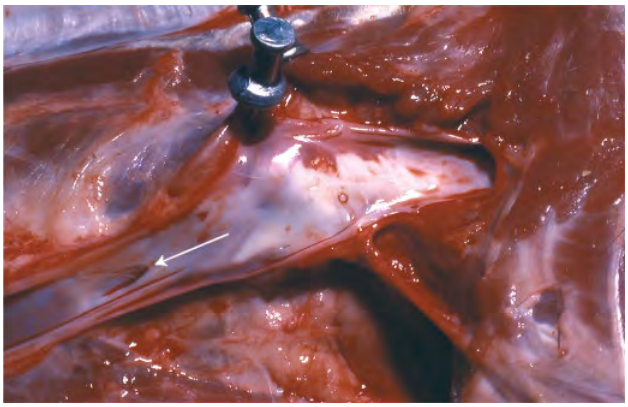


Figure 4.9 Valves in the jugular vein in the upper neck (arrow).

In long-necked animals such as horses, giraffes, and camelids, one-way valves in the jugular veins prevent backflow of blood to the head when it is lowered for feeding and drinking. In the llama, four or five of these valves are distributed from the confluence of the veins that form the jugular to the thoracic inlet (Figures 4.9 and 4.10). Though valves do not typically impede flow when injecting into the jugular vein, they may interfere with aspiration of blood.

Cranial (High-Neck) Jugular Venipuncture

The external jugular vein has a larger luminal diameter at its bifurcation into the linguofacial, **maxillary**, and caudal auricular vein near the caudal boarder of the mandibular ramus (Figure 4.11). In this area, the vein is separated from the carotid artery by the omohyoideus muscle and inadvertent carotid penetration is less likely with venipuncture in this cranial location. Disadvantages of using this cranial site include thicker skin in this region, coverage of the vein by the platysma muscle, and the apposition of the parotid

Figure 4.10 Valves in the jugular vein low on the neck (arrows).

salivary gland (Figure 4.12). This area is also more subject to movement as the animal moves its head.

Positioning of the head and neck is important to achieve success with this procedure. The author prefers to perform venipuncture from the right side, with an assistant standing on the left and grasping the right ear while pressing their body against the left neck to produce a slight bow, with the convex side toward the operator. The nose should also be pulled toward the left, making certain that the halter straps do not press on the vessels proximal to the penetration site (Figure 4.13).

An imaginary line should be scribed along the ventral border of the mandible onto the neck, with the head held in a slightly flexed position. The tendon of the sternomandibularis muscle should be palpated to locate the site of penetration, just dorsal to the intersection of those two lines (Figure 4.14). The vein should be occluded by deep, firm pressure at the ventrum of the vertebrae. By stroking the area of the vein toward the occluding hand, a wave may be felt against the fingers. Alternatively, correct selection of the site may be tested by placing a finger over the suspected location of the vein and releasing the occlusion to see if the dilated vessel empties.

Mid Cervical Jugular Venipuncture

Jugular venipuncture may be performed in the mid-cervical region. This location is also appropriate for jugular IV catheterization (See Figure 4.6).

The jugular vein begins at the confluence of the lingofacial, maxillary, and caudal auricular veins near the caudal mandibular ramus and continues caudally toward the thoracic inlet. As it courses caudally, it dives deep to the sternomandibularis muscle, and is incorporated in a fascial sheath with the carotid artery and the vagosympathetic nerve. All these structures lie on the ventrolateral surface of the trachea. The advantage to lower (caudal) jugular venipuncture is that the

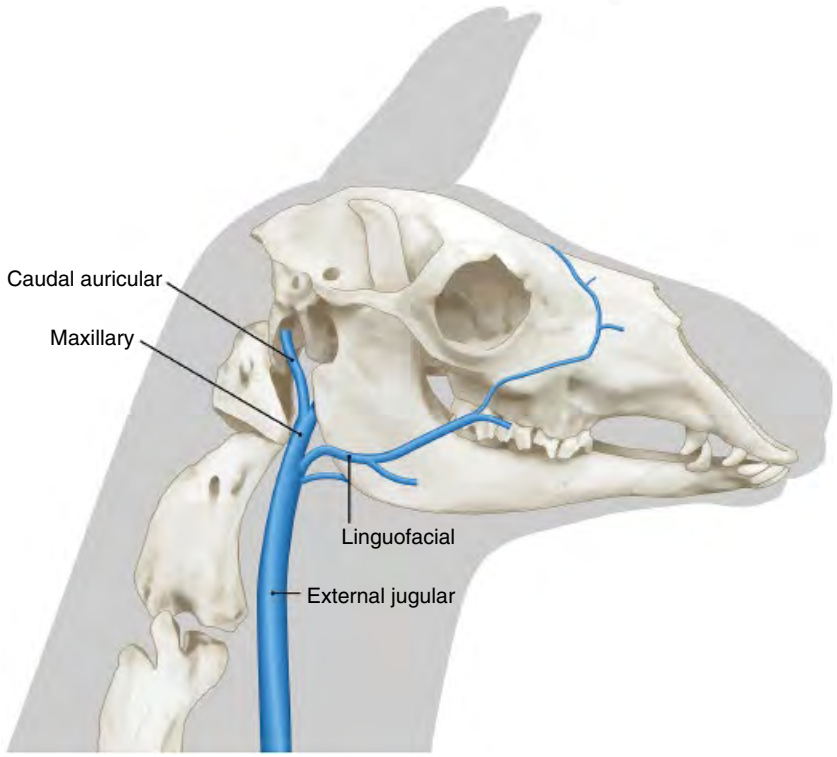


Figure 4.11 Proximally, the confluence of the linguofacial, maxillary and caudal auricular veins form the external jugular vein.

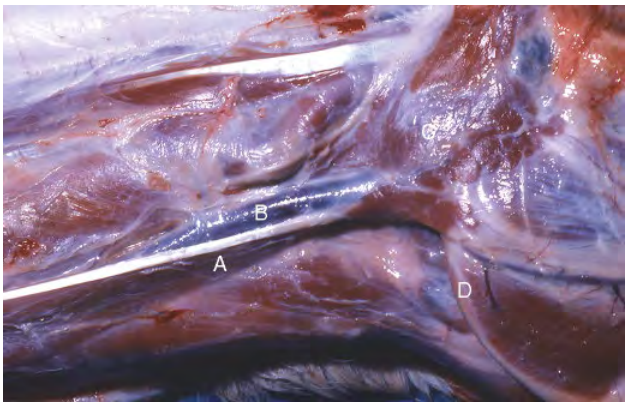


Figure 4.12 Anatomic dissection of the jugular vein in the cranial cervical region. (A) Tendon of the sternomandibularis muscle, (B) jugular vein, (C) parotid gland, (D) caudal ramus of the mandible.

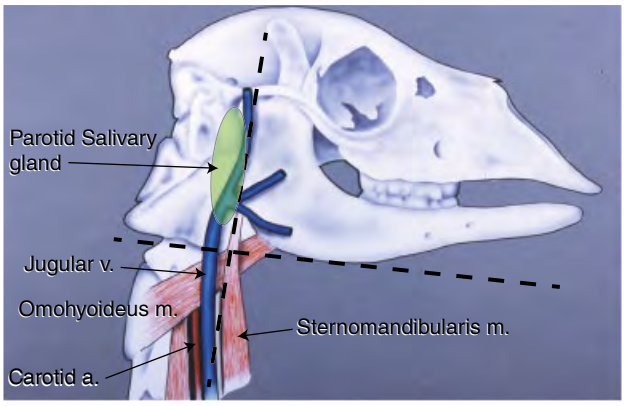


Figure 4.14 Diagram illustrating location for cranial (high) jugular venipuncture. A point slightly dorsal to the intersection between an imaginary line scribed along the ventral mandibular border and the tendon of the sternomandibularis m. gives an approximate location for high jugular venipuncture.

Figure 4.13 An alpaca is being restrained for right jugular venipuncture. The holder is standing on the left side of the animal and pulling the head toward himself, exposing the right cervical region for venipuncture.

skin is thinner, and movement of the head is less disruptive during the procedure; the main disadvantage is due to its intimate apposition to the carotid artery in this location.

Proper restraint is important to successful venipuncture. The holder must adequately restrain the animal without obscuring the site and interfering with the procedure. The animal is restrained so that the neck is straight and vertical, and the head is held with the ventral mandible horizontal.

The tight cervical skin prevents good palpation of the vein. However, in most animals, the distended vein can be palpated by repeatedly holding the vein, allowing it to distend and then allowing it to fall. Occlude the vein low on the neck and palpate in the mid-cervical region for a distended vein. Holding off too close to the area that is palpated will cause local interference of skin movement and will interfere with accurate palpation of the vein.

If the owner permits clipping of the fiber, this will aid in venipuncture or catheterization, but often owners object to clipping of the fiber. Clipping not only makes venipuncture much easier, it may reduce stress on the animal and permits the site to be aseptically prepped, an advantage if placing an indwelling IV catheter.

In adult camelids, occlude the jugular vein at the lowest palpable (C6) cervical vertebrae. Allow the jugular vein to fill with blood and distend. Lightly palpate in the space between the lateral transverse processes and the trachea in the mid-cervical region. Occluding and releasing the vein multiple times will aid in verifying the location of the vein prior to cannulation.

For routine blood collection, with the vein distended, penetrate the vein in the mid-cervical region with the needle aimed at a 45° upward angle. If unsuccessful, leave the needle through the skin and repeat the light palpation as described above to assess the exact location of the vein. Blindly sticking frequently results in a carotid penetration.

Low Cervical Jugular Venipuncture

The disadvantages of low jugular venipuncture is that the lower neck is usually more heavily fleeced than the upper

region, and the jugular vein and carotid artery are in juxtaposition at this location, increasing the risk of arterial penetration. Stocks or a chute is desirable to fix the head upward and prevent the animal from pushing forward.

The technique is like that described for mid-cervical venipuncture. The head should be elevated. The operator should palpate for the ventral projection of the transverse process of the sixth cervical vertebra and occlude the vessel at this site by wrapping the fingers around the projection. The pulsation of the carotid artery may be felt in this location. The needle should be inserted slightly medial to the projection, toward the center of the neck.

Intravenous Jugular Catheterization

A 14-gauge, 6-in. catheter placed in the mid-cervical region works well for most adult camelids. This size of catheter permits fluid delivery as well as administration of drugs. It also allows for diagnostic blood collection if repeated sampling is necessary. The length of the catheter increases the security so that if it backs out slightly, the majority of the catheter remains in the vein. Placement in the mid-cervical region allows adequate room cranially to secure the catheter, typically with just bandaging tape (Elastikon®). This location also affords versatility if a second attempt at catheterization needs to be made below a hematoma. For neonates, an 18-gauge, 4-in. catheter is recommended by the author.

The thickness of the skin prevents easy penetration. A guide (pilot) hole will prevent dulling of the tip or bending of the catheter. A guide hole with a needle of the same gauge as the catheter is useful (use a 14-gauge, hypodermic needle to place a 14-gauge catheter). The purpose of the guide hole is to merely make a pilot hole through the skin, not to penetrate the vein with the needle. A guide hole is not necessary in neonates. After the vein is located, place the guide-hole-needle through the skin while the vein is not distended to avoid inadvertent puncture of the vein. The needle is angled down the neck at an approximate 45° angle.

After the guide hole is made, the needle is removed, and a heparinized catheter is inserted in the hole just penetrating the skin. The angle of the catheter should be similar to that of the pilot hole (~45°). The vein is now occluded at the lowest palpable cervical vertebrae and time is allowed for the vein to distend. The catheter is monitored for a "flash" of blood. If a "flash" is present, the angle of the catheter is reduced (laying flat against the neck). The entire catheter and stylet is advanced slightly, making certain that blood continues to flow with the vein still occluded. The catheter should advance easily (no "pops"). Resistance to passage should be minimal. If necessary, the catheter is backed out slightly until blood flows, and advancement is attempted again changing the angle slightly.

The author likes to advance the catheter and stylet approximately 5–10 mm in the vein while the vein is occluded. If the catheter is still in the vein (based on continued blood flow), the stylet can be held stationary, and the catheter is advanced down (caudally) the vein.

Prior to placing the catheter, separate the catheter and stylet to make sure that the catheter and the stylet move freely from each other. Also do not occlude the end of the catheter while looking for the "flash." Occluding the end of the catheter will trap the pocket of heparinized fluid in the catheter and will slow the rate at which blood flows. Some prefer to use a small "bleb" of cutaneous lidocaine to block the area prior to catheterization; the author does not as he feels it obscures his ability to locate the vessel.

Jugular Venipuncture in Neonatal Crias

Restraint for venipuncture in a neonate up to four months of age may be difficult. Fortunately, the skin of the neonate is thin, and the jugular vein can usually be distended and visualized. Select a clean, dry location and place a clean towel beneath a newborn to protect the umbilicus. A simple method that enables a single person to perform venipuncture begins with straddling the neonate and folding the front legs with the hand as the cria is pushed down (Figures 4.15 and 4.16). The head should be pushed down with the other hand. As the cria lies down on the forelegs, a slight pressure on the rump encourages it to assume sternal recumbency. The cria should never be collapsed by sitting on it.

Figure 4.15 Position for venipuncture in a cria.

Alternatively, it may be lifted off its feet and tipped to lie on its side. Then the legs will fold up beneath it as the handler straddles it. While kneeling over the cria to keep it in sternal recumbency, the left arm should be placed on the right side of the neck to push the head and neck to the left. The left hand should also be used to occlude the vessel while the right hand inserts the needle to collect blood or give medication. Venipuncture may be accomplished at the cranial, middle, or caudal cervical locations.

Jugular Venipuncture in Camels

Jugular venipuncture in the dromedary camel is easier than in SACs if the head is restrained and side-to-side movement of the camel is controlled in stocks or a chute. The jugular vein may be distended using digital pressure as in the SAC (Figure 4.17), or a rolled-up towel may be placed on the ventrum of the neck, near the thoracic inlet, and held tightly with a cord or rope. The towel serves as a temporary tourniquet. The distended jugular vein is visible in the upper cervical region, and the distention progresses on to the facial vein on the lateral aspect of the mandible. Venipuncture may be performed at any location on the distended vein. During the winter months, the fiber of dromedaries increases in length and may diminish the visibility of the vein. The vein is easily palpated. Bactrian camels often have heavy neck fiber like the dromedary in cold weather. Venipuncture is performed the same in Bactrians as in dromedaries. The lateral thoracic vein is also accessible in camels, but the animal must be restrained to avoid kicking (Figure 4.18).

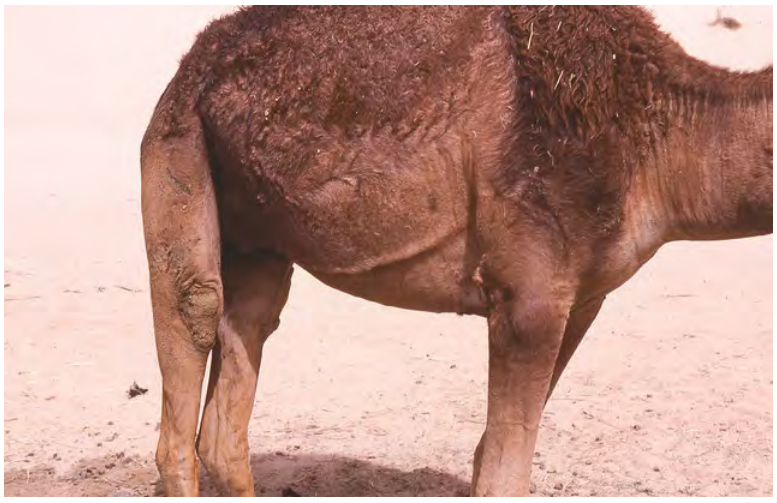
Venipuncture in Other Locations

Though the jugular vein is the largest and most readily accessible vein in most camelids, venipuncture may be performed at other locations as well. The saphenous vein is superficial and easily palpated on the medial aspect of the stifle in a recumbent animal. The artery and vein lie contiguous to each other, with the vein located cranially.

Figure 4.16 Alternate position for venipuncture in a cria.

Figure 4.17 Blood collection from the jugular vein of a camel.

Figure 4.18 Lateral thoracic vein of a camel, an alternate site.



A brachial vein is present on the cranial forearm as in a dog or cat. This vein may be useful if the fiber covering is not too dense.

Small volumes of blood may be collected from the middle coccygeal vein on the ventrum of the tail. In the camelid, the coccygeal vein is superficial, lying just beneath the skin. The tail should be elevated but pushing it too far up over the back should be avoided, because this will occlude the venous return. The procedure for blood collection from the tail of a camelid is easier than it is in the bovine, in which the vein is situated deep, near the body of the coccygeal vertebrae. Insert the needle through the skin, then establish negative pressure in the syringe, and continue the insertion until blood flows into the syringe. It is unwise to administer medication in the coccygeal vein because the long bevel on most needles may span the vessel and allow perivascular deposition of the medication.

There are also accessible veins on the ear (Figure 4.19). The caudal border of the ear may be nicked to allow collection of small blood samples. A vein can also be cannulated with a 23-gauge needle attached to a pediatric (butterfly) catheter. The largest auricular vein is located on the outer caudal border of the pinna. A temporary tourniquet (heavy elastic band) may be placed at the base of the ear to raise the vein.

Figure 4.19 Ear veins on a llama.

Hemogram and Blood Chemistry

Evaluation of hemogram and blood chemistry data, used in conjunction with clinical examination, is crucial to making a diagnosis in many disease conditions. A full discussion of normal and abnormal clinical pathology findings is found in Chapter 14, and the focus here will be collection of the samples.

Abdominocentesis

Peritoneal fluid evaluation is an important diagnostic tool for digestive and other abdominal disorders. A peritoneal fluid sample may be obtained from the ventral midline, just caudal to the umbilicus. The retroperitoneal fat layer of SACs may be as much as 6 cm (3 in.) thick and 14 cm (6 in.) broad lying lateral to the linea alba (Figure 4.20). Performing the abdominocentesis on the midline allows the operator to penetrate the peritoneal cavity without needing to traverse this fat layer. The sample may be obtained from either the standing or recumbent animal. The site should be clipped and surgically prepared and local anesthesia injected. A stab incision should be made through the skin with a scalpel blade, after which a 6-cm (3-in.), 14-gauge teat cannula can be inserted. When inserting the teat cannula, a quick thrust should be used to penetrate the peritoneum without pushing it away from the body wall.

In adult camelids, the glandular saccule area of C1, covered by the greater omentum, is located on the ventral midline craniad to the umbilicus. Because the omentum may occlude the ports of a teat cannula, it is recommended that abdominocentesis be performed caudal to the umbilicus.

A paracostal approach can also be used to collect peritoneal fluid. The site is located caudal to the last rib and approximately one-third to one-half of the distance from the ventral midline to the dorsal midline. The site of penetration in alpacas is 1 cm dorsal and 3 cm caudal to the costochondral junction of the last rib and, in llamas, it is 2 cm dorsal and 5 cm caudal to the costochondral junction of the last rib [13] (Figure 4.21).

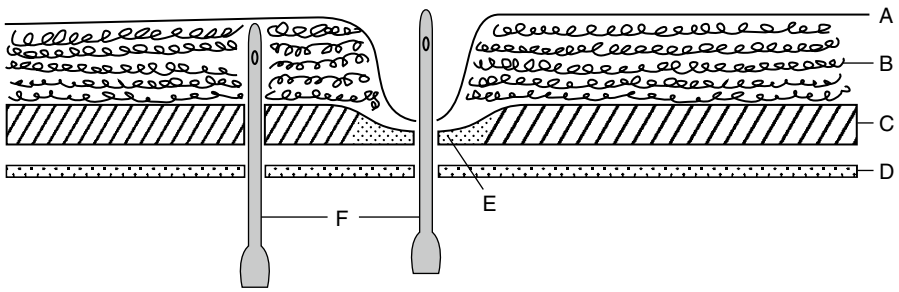


Figure 4.20 Diagram of the abdominocentesis location. (A) Peritoneum, (B) retroperitoneal fat, (C) abdominal muscles, (D) skin, (E) linea alba, (F) teat cannula.

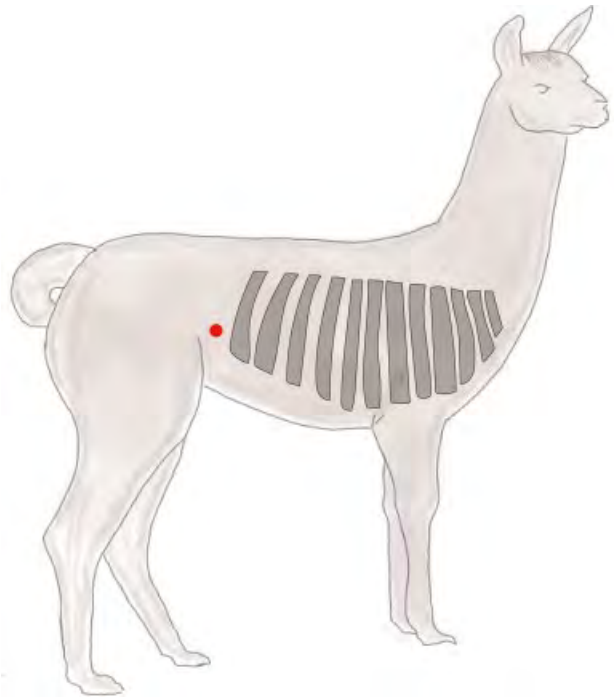


Figure 4.21 Diagram showing location for paracostal abdominocentesis (red circle). The site of penetration in alpacas is approximately 1 cm dorsal and 3 cm caudal to the costochondral junction; in llamas, it is 2 cm dorsal and 5 cm caudal to the costochondral junction.

In a normal animal, insufficient abdominal fluid may preclude collection. Unless there is 150 mL or more fluid in the cavity, it will not flow freely. If it does not, attaching a syringe and establishing slight negative pressure while repositioning the needle tip in the cavity may yield a sample. If the fluid accumulation is localized, successful aspiration will be fortuitous.

The technique for collection of peritoneal fluid from adult camels is described as follows: The site is 10 cm (4 in.) caudal to the xyphoid cartilage and 10 cm (4 in.) left of the midline. Naeini showed that the cellularity of abdominal fluid in dromedary camels was significantly lower than that of blood with the expectation of lymphocytes which was significantly higher than that of blood. Additionally, the total protein, cholesterol, triglyceride and calcium and activities of AST, ALP, CK and amylase in serum were higher than that in peritoneal fluid [14].

Orogastric Intubation

Orogastric intubation is a common practice to administer oral medications or to restore normal C1 flora via transfaunation (See Chapter 12) (Figure 4.22). Passage of the orogastric tube differs little from the procedure in cattle and sheep. The cheek teeth are sharp, so a speculum is

Figure 4.22 Positioning for gastric intubation of a neonate llama.

Figure 4.23 Small ruminant mouth speculum.

necessary to guide the gastric tube through the oral cavity and into the oropharynx. A small ruminant mouth speculum works well for this procedure in SACs (Figure 4.23). A standard cattle Frick speculum is suitable for camels but too large except for the largest llama. A 20- to 25-cm (8- to 10-in.) segment of rubber garden hose slightly larger in diameter than the orogastric tube makes an excellent guide. A similar length of polyvinyl chloride pipe, wrapped with adhesive tape to prevent accidental shattering, is also suitable. Camelids may be intubated either standing or in sternal recumbency, but the head and neck should be elevated to reduce the risk of regurgitation and aspiration

Table 4.5 Orogastric tube sizes for llamas and alpacas.

Body weight	French gauge	Outside diameter (mm)	Outside diameter (in.)
4–8 kg (8–18 lb)	18–22	6–7 (Stallion catheter or feeding tubes)	1/4
9–20 kg (19–44 lb)	24–30	8–10	1/3 to 3/8
30–90 kg (66–198 lb)	30–40	12–13 (Equine stomach tubes)	1/2
100–200 kg (220–440 lb)	40–45	13–16 (Equine stomach tubes)	1/2 to 5/8

(Figure 4.22). Table 4.5 is a guide to choosing an appropriately sized esophageal tube.

The head should be secured with a halter that allows opening of the mouth sufficiently to insert the speculum over the base of the tongue. The head should be slightly flexed. Once the tip of the tube is in the oropharynx, gentle pressure should be exerted to stimulate the animal to swallow. Indiscriminate jabbing should be avoided to prevent pharyngeal trauma, but gently rotating the tube or adjusting the position of the head will encourage the animal to swallow the tube. There should be a slight resistance to passage of the tube.

The tube should be palpated as it traverses the left ventral cervical region to ensure intubation of the esophagus rather than the trachea. Aspiration on the tube should reveal negative pressure while in the esophagus. When the tube passes into the C1 compartment, aspiration may reveal C1 contents, fluid, or gas that have a characteristic ruminal smell which can also be used to support passage of the tube into the C1 compartment.

The tube may be inserted into the lumen of C1. However, if it is desired to bypass deposition of fluid or medication into C1, the tube should be kept within the esophagus. This is particularly important when feeding orphan neonates via stomach intubation. Milk deposited in C1 may remain and ferment rather than be digested in C3.

Intubation may stimulate regurgitation (Figure 4.24). This is not serious, but it does make the process disagreeable and may necessitate starting over again. Regurgitation is more likely if the animal is stressed or excited prior to intubation. Attempting to keep the animal as calm as possible during this procedure is advantageous.

Nasogastric Intubation

Nasogastric intubation can be used to an alternate to orogastric intubation. The nasogastric tube is less likely to induce the gag reflex and regurgitation. However, epistaxis may result from nasogastric intubation, especially if the animal struggles. (See video on companion website Courtesy of Dr. Brianna Jordan). Many animals resist passage of the tube through the rostral few centimeters of the nares. Lidocaine gel can be used as a lubricant and has the added advantage of desensitizing the nares, making the procedure tolerable by some patients [15]. If passage of a nasogastric tube is causing excessive stress or causing trauma to the nasal cavity, orogastric intubation may be a preferred alternative.

Figure 4.24 Regurgitation accompanying gastric intubation of a llama.

The nasal cavity is narrow and may preclude passage of larger tubes. If a larger diameter tube is necessary, as will likely be the case if attempting to aspirate contents from the C1 compartment for analysis, orogastric intubation should be performed. Most adult llamas and alpacas will accommodate a tube around 1 cm in diameter [15]. A semi-rigid catheter like a stallion urinary catheter usually works well for nasogastric intubation in llamas or alpacas.

A lubricated tube is directed into the ventral nasal meatus and the tube is gently passed until it encounters resistance at the glottis. Gentle pressure on the tube should initiate a swallowing reflex and the tube can be advanced into the esophagus. Verification of passage into the esophagus and not the trachea is performed as with orogastric intubation.

Collection of a Urine Sample

The easiest way to collect a urine sample is to make a free catch while the animal is urinating. Camelids use a communal dung heap for both urination and defecation. Both the male and female eject urine caudally from a partial squat position. Unless extremely wild, most llamas will permit an approach to catch the urine in an open cup, or the cup may be attached to a stick approximately 1.2 m

(4 ft) long. Complete urination requires 30–60 seconds, allowing ample time to obtain a sample.

As with ruminants, male camelids have a urethral diverticulum dorsal to the ischial arch. Retrograde urethral catheterization is difficult in males because this blind pouch entraps catheters prior to bladder entry.

It is possible to catheterize the female. The external urethral orifice is easily palpated as a groove on the floor of the vestibule at the region of the hymen. However, a suburethral diverticulum in the female may also complicate catheterization (Figures 4.25 and 4.26). After the vulva has been cleansed, a sterile gloved finger should be inserted through the vulva to palpate the orifice. Withdrawing the finger slightly and inserting the catheter dorsal to the finger should direct the catheter into the urethral opening, avoiding penetration into the more ventral, blind, diverticulum. A 5-French polypropylene catheter is suitable for urine collection. The bladder should be found at a depth of 25 cm from the lips of the vulva.

Cystocentesis has been performed on SAC neonates and juveniles. The bladder is situated on the floor of the pelvis. The site for penetration of the abdomen is just cranial and slightly lateral to the pelvic brim. Clip a spot of fiber and disinfect the area before inserting the needle. The procedure may be performed on a standing animal or one in lateral recumbency.

Miscellaneous Procedures

Enema

An enema is indicated in cases of constipation, heat stress, or dehydration. In the juvenile, a feeding tube may be used attached to a funnel for the fluid (Figure 4.27), or with a syringe (Figure 4.28). Adults may be given an enema with an orogastric tube and pump or a garden hose. It is not necessary to insert the tip of the hose into the rectum. The tip is pressed against the anus and held with a hand to allow water to run in and out as the stimulus to evacuate occurs (Figures 4.29–4.32.)

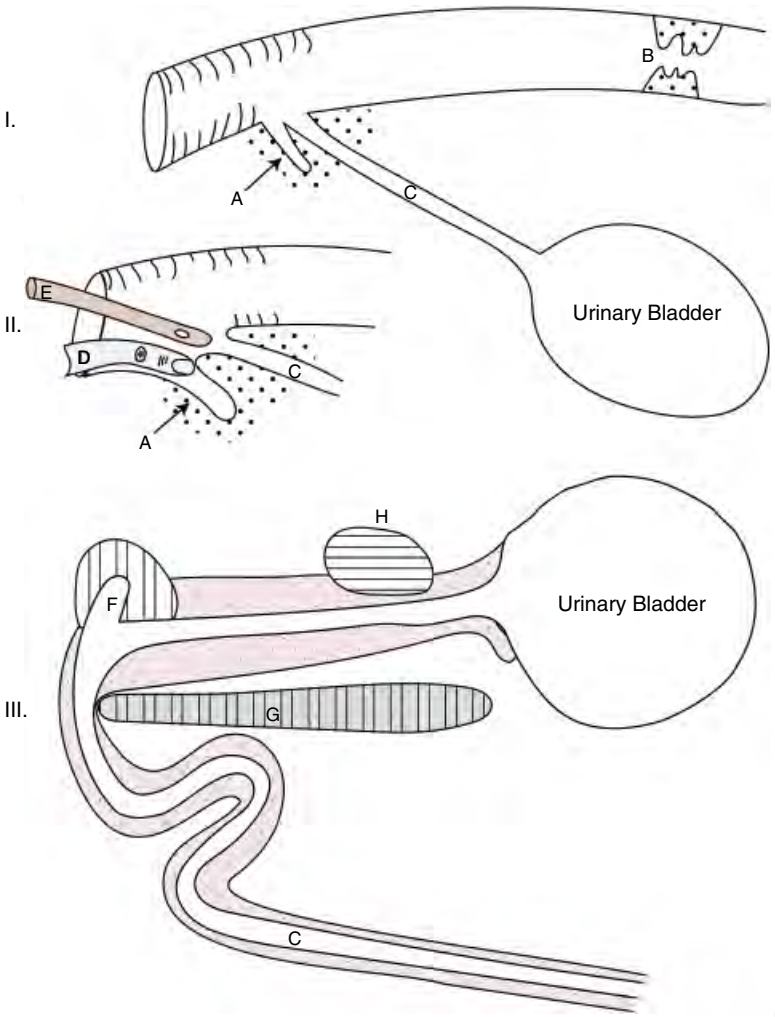


Figure 4.25 Diagrams of the female and male urethra. I. Schematic of the female urethra. (A) Suburethral diverticulum, (B) cervix, (C) urethra. II. Demonstration of passage of urinary catheter in the female. Covering the suburethral diverticulum with a finger (D) will prevent the catheter from entering the suburethral diverticulum. (E) urinary catheter. III. Schematic of the male urethra. (F) Urethral recess, (G) pubis, (H) prostate gland.

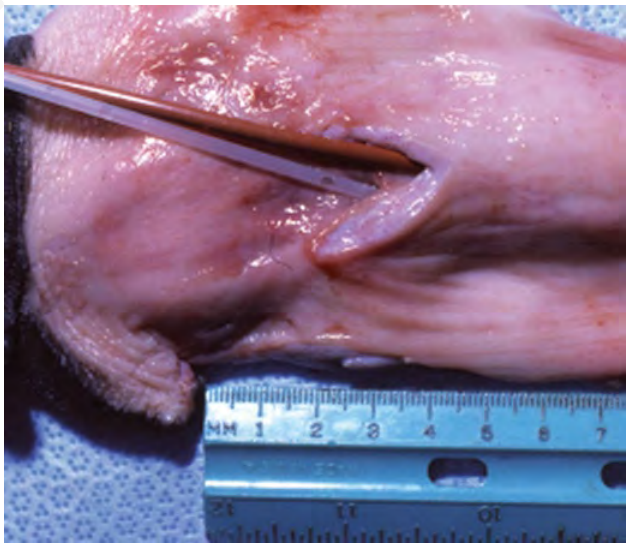


Figure 4.26 Postmortem dissection of a female camelid's vestibule and vagina showing the urethral opening. The urethral opening has been enlarged longitudinally. A red rubber catheter has been inserted into the urethra. A polypropylene catheter is inserted into the suburethral diverticulum.

Figure 4.27 Giving an enema to a juvenile with a tube and funnel.

Figure 4.28 Giving an enema to a juvenile with a tube and syringe.

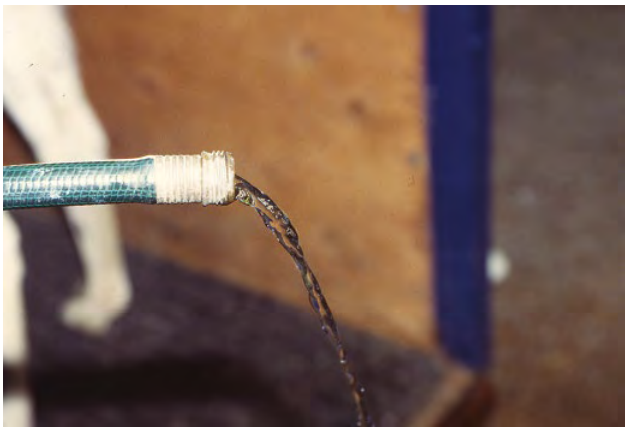


Figure 4.29 Using a garden hose to give an enema to an adult, light flow of water.

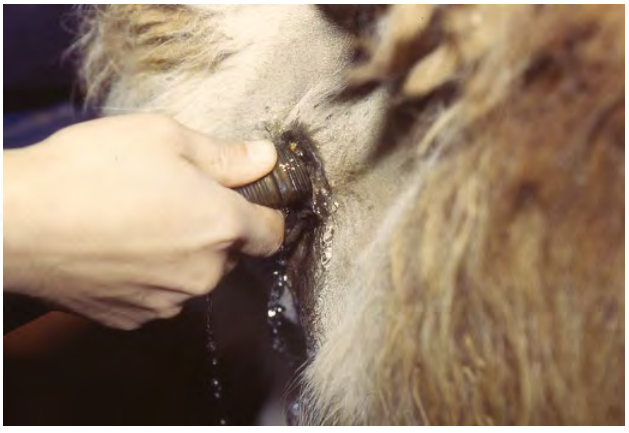


Figure 4.30 Hose to the anus but not inserted.

Figure 4.31 Using a finger to open the anus.

Figure 4.32 Holding the hose next to the anus to allow water to flow in and out of the rectum.

Thoracocentesis

Thoracocentesis may be indicated to obtain samples of pleural fluid for diagnostic purposes, remove excess fluid or exudate, or remove air from the pleural spaces. Penetration can be made from either side. The preferred site is at the sixth or seventh intercostal space (there are 12 ribs), 10–15 cm (4–6 in.) dorsal to the ventrum of the sternum or 2–4 cm dorsal to the costochondral junction of the ribs (Figure 4.33). The area should be clipped and prepared for aseptic surgery.

Selection of the needle or cannula to be used is determined by the purpose for the thoracocentesis. If fluid is expected, a 14- to 16-gauge, 5-cm (2-in.) needle should be inserted near the cranial border of the rib to avoid the intercostal vessels. The pleural cavity may be penetrated at an approximate depth of 2–3 cm (1–1.5 in.). Fluid should flow from the needle. A spinal needle and stylet may be selected if air is to be removed.

If the lung has collapsed, a more dorsal and caudal position should be selected. Entrance should be made at the eighth or ninth intercostal space, approximately 25 cm from the dorsum of an adult llama. Distances for smaller individuals must be reduced proportionately. With a standard technique, an indwelling catheter may be placed within the pleural space.

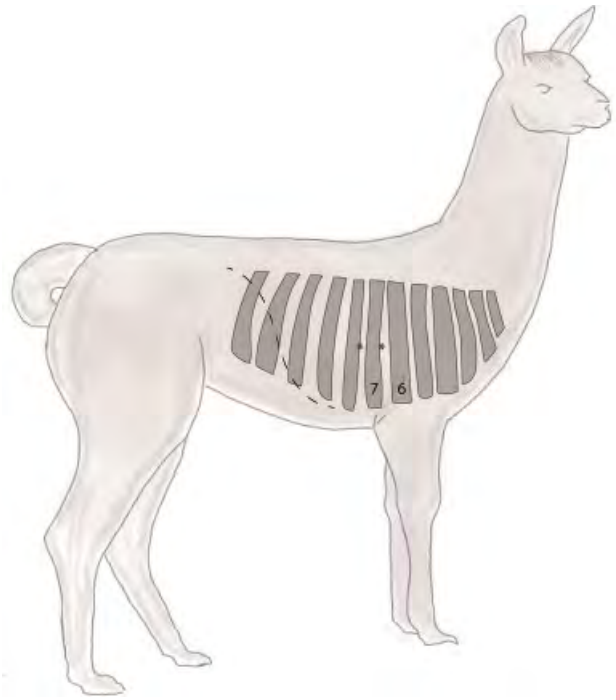


Figure 4.33 Schematic illustrating proper location for thoracocentesis, at the sixth or seventh intercostal space (*). To avoid neurovascular structures on the caudal aspect of the ribs, puncture should be made in the caudal intercostal spaces. The dashed line represents the pleural reflection.

Figure 4.34 Needle position for collecting a bone marrow sample from the sternum.

Bone Marrow Aspiration

Bone marrow is collected from one of the sternebrae. The wing of the ilium is too thin to allow penetration of the marrow cavity, and the location of rib marrow is less consistent than sternebrae marrow. An appropriate site is approximately 3–4 cm dorsal to the callosity on the ventrum of the sternum (Figure 4.34). Some clinicians prefer to insert the needle vertically through the sternal callosity. The author prefers the lateral approach. The needle should be directed medially and slightly dorsally to engage the bone. Significant pressure exerted on the needle is required to enter the bone. A grating may be felt with the thrust. The needle should not be twisted during the insertion or the bevel will be damaged.

Marrow may be obtained at a depth of approximately 2.5 cm in an adult SAC, and 3–5 cm in camels. After penetration, the stylet should be removed and a 12-mL syringe attached. Withdrawing the plunger with a pumping action should yield a sample. Marrow in the llama is diluted with

blood and is thinner than that of cattle and horses. A smear made of marrow shows fat droplets. Smears may be made directly, or the sample may be placed in an anticoagulant vial. The sample must be mixed thoroughly with the anticoagulant before the needle is removed from the vial to ensure collection of a proper sample.

If no marrow is obtained, the stylet should be replaced and penetration continued. After two or three unsuccessful attempts at aspiration, the needle should be withdrawn and moved 2–3 cm cranial or caudal, assuming that the initial insertion was made into an intersternebral space.

Liver Biopsy

Histopathology of liver tissue may be necessary to diagnose liver disease and determine the cause, and biopsy has been shown to be safe in camelids with minimal complications [16–18]. In the camelid, the liver lies entirely on the right side of the abdomen. The biopsy needle may be directed via ultrasonography, or a landmark approach may be used [19]. The technique for obtaining a biopsy entails penetration of the caudal thorax, through the pleural space and the diaphragm, into the liver. Clipping a large area may be avoided by parting the fibers and fixing them out of the operative site with masking tape. An area 2.54 cm (1 in.) square should be clipped and prepared for aseptic surgery (Figure 4.35). Using local anesthesia, a liver biopsy may be done with the animal in either a standing or a sternally recumbent position safely [17].

The recommended site for performing a liver biopsy in SACs is at the 9th or 10th intercostal space and approximately 20–22 cm (9–10 in.) from the top of the back. The intersection of a line drawn from the mid humerus to the tuber coxae intersected with the intercoastal space approximates this location (Figure 4.36) [17, 20]. The needle should be directed toward the midline, caudally and slightly ventrally (Figure 4.37). The chest wall is thin. The diaphragm is located immediately adjacent to the chest wall. To check proper site, let go of the needle. If the needle is in the diaphragm, it will move forward and back in synchrony with respiration. Because the liver lies immediately medial to the diaphragm, pushing the needle 2–3 cm deeper will penetrate the liver.

The sample should be collected as indicated for the type of needle being used. If the sample is desired for histologic

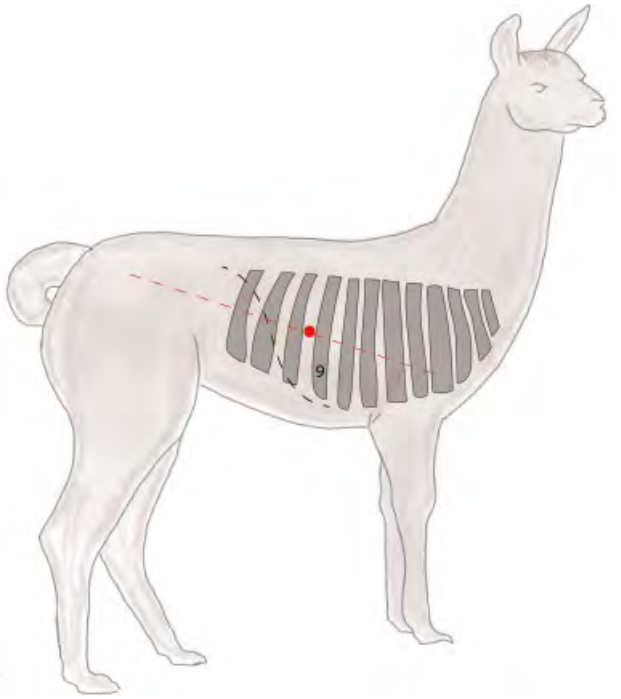


Figure 4.36 Schematic illustrating the approximate location for obtaining a liver biopsy in an alpaca. Biopsies are taken through the 9th or 10th intercostal space, approximately 20 to 22 cm from the top of the back. The dashed black line represents the pleural reflection. Alternatively, a line drawn from the tuber coxae to the mid-humerus (dashed red line) intersected with the 9th or 10th intercostal space is a good location for liver biopsy.

Figure 4.35 Tape can be used to retract the fiber to prepare the skin for sterile collection of a liver biopsy.

Figure 4.37 Insertion of needle for collection of liver biopsy.

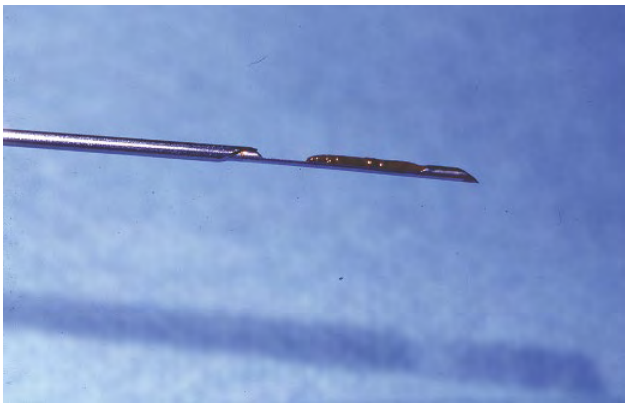


Figure 4.38 Liver biopsy sample collected with True-cut needle.

examination, a wide choice of needles[1,2] is available. Disposable needles used in human medicine are satisfactory (Figure 4.38). An automated, spring-loaded, biopsy instrument increases speed and patient comfort [19]. If larger samples are required for nutritional studies, special types of trocars and cannulas are required.

The technique has been described in sternally recumbent camels. Like SAC's, the ninth intercostal space works well for taking a liver biopsy. The biopsy instrument is inserted approximately 2 cm dorsal to the costochondral junction and aimed toward the contralateral olecranon [21].

Collection of Cerebrospinal Fluid

Laboratory analysis of cerebrospinal fluid (CSF) is a common diagnostic tool for animals with neurologic diseases. Neurologic diseases such as meningeal worm, meningitis, and listeriosis have classic CSF abnormalities (See Chapter 14). In addition to collection of CSF for laboratory analysis, the subarachnoid space may be cannulated to make a determination of CSF pressures or to perform myelography, a diagnostic imaging modality where radiopaque contrast media is injected in the subarachnoid space to highlight the course of the spinal cord.

CSF is contained within the subarachnoid space, which surrounds the spinal cord, and cannulation of the subarachnoid space with a needle typically results in spontaneous flow of CSF (Figure 4.39). The anatomic relationship of the subarachnoid space to the epidural space, meninges, vertebrae, and spinal cord is the same in camelids as in true ruminants and horses. The spinal cord and subarachnoid space extend from the head caudally to the level of the second sacral vertebral segment in the llama.

Collection of CSF can be performed from the lumbar cistern at the lumbosacral (LS) space or at the cisterna magna at the atlantooccipital (AO) space. CSF recovered from the LS space has been found to be more diagnostic for diseases

Figure 4.39 Needle inserted into the subarachnoid space of an alpaca. There is spontaneous flow of CSF from the needle.

of the caudal spinal cord, such as is typical with cerebrospinal nematodiasis. CSF collected from the LS space showed more significant elevations in protein and cellularity in llamas with paralaphostrongylosis compared to CSF collected from the AO space [22]. CSF collected from the AO space may have a higher diagnostic yield for intracranial or cranial spinal cord disease. Routine collection of CSF from the lumbar cistern at the LS junction is most commonly performed with the animal awake. This procedure can be done with the animal preferably standing or in sternal recumbency, but can also be performed in lateral recumbency. Collection of CSF from the AO space is not performed routinely, carries increased risk due to potential trauma to the brainstem, and requires complete immobilization with the animal under general anesthesia, which adds risk and expense [23]. AO taps are performed with the animal in lateral recumbency. The fiber should be clipped and the area aseptically prepared prior to performing the procedure at either location.

To collect CSF from the AO space, the animal should be anesthetized and placed in lateral recumbency. The head is flexed, and the wings of the atlas palpated to locate the narrowest width at the cranial border (Figure 4.40). Draw an imaginary line from one wing to the other and penetrate the skin directly on the midline. A 20-gauge × 6.35 cm (2.5 in.) spinal needle is advanced perpendicular to the skin through the funicular ligamentum nuchae and then the dorsal AO membrane and into the cisterna magna.

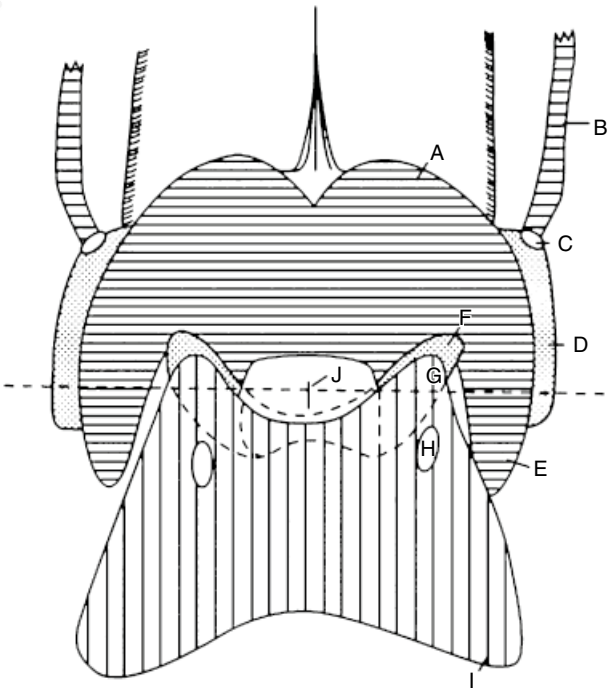


Figure 4.40 Diagram of the atlantooccipital space for cerebrospinal fluid collection. (A) Nuchal crest, (B) zygomatic arch, (C) osseous ear canal, (D) mastoid process, (E) jugular process, (F) occipital condyle, (G) cranial wing of the atlas, (H) alar foramen, (I) caudal wing of the atlas, (J) site for needle insertion.

The cervical dura mater lies closely adjacent to the membrane, and with the head held in tight flexion, the membrane and dura will be tense. The needle should penetrate the subarachnoid space with an audible or palpable "pop." The stylet should be withdrawn to check for fluid. If none is present, the stylet can be wiped on the back of the operator's sterile glove to check for moisture. If none present, the stylet should be reinserted and the needle rotated 90° and again a check made for fluid. If still no fluid is encountered, the stylet should be reinserted and the needle advanced 1 or 2 mm deeper. This may be continued until fluid can be withdrawn. The needle should never be manipulated without the stylet in place, otherwise the spinal cord may be lacerated or a plug of neural tissue removed. The depth of the subarachnoid space in an adult llama is approximately 4 cm (2 in.).

CSF collection from the LS space is safe and can be performed with the animal awake. The LS space can be located by the intersection of an imaginary line between the ileal wings and another imaginary line on dorsal midline. Cranial to this intersection, the dorsal spinous process of the last lumbar vertebrae (L7) can be palpated. The LS space is located approximately 2 cm caudal to the dorsal spinous process of L7 on the midline. An approximately 4 cm square is clipped and prepared centered over this area. 1 mL of local anesthetic can be injected at the site to desensitize the skin and subcutaneous tissues. An 18-gauge × 9 cm (3.5 in.) spinal needle is inserted perpendicular to the vertebral column in SACs. Camels will likely require an 18-gauge × 15.25 cm (6 in.) spinal needle. The interarcuate space between L7 and the first sacral vertebra (S1) is large (2 cm cranial caudal and 4 cm wide) (Figures 4.41 and 4.42).

The tissues penetrated by the needle are the skin, thoracolumbar fascia, interspinous ligament, interarcuate ligament, dura mater, and arachnoid, passing through the epidural space. Subtle "pop's" may be felt but they are not as prominent as those associated with CSF collection at the AO space. A reaction is often noted when the needle penetrates the dura [23]. Decreased resistance should be noted when passing through the interarcuate ligament. With each "pop" or change in resistance, the stylet can be removed to check for spontaneous flow of CSF. If spontaneous flow of fluid is not present, the stylet can be wiped on the back of the operator's glove to check for moisture. As previously indicated, the needle should never be advanced without the stylet in place. If no fluid flows, the jugular may be occluded to increase intraspinous pressure or gentle aspiration with a syringe may be performed (Figure 4.43). If still no fluid is encountered, the needle may be advanced through the conus medullaris of the cord to the floor of the spinal canal. Then the needle should be withdrawn, one millimeter at a time, until fluid flows.

The depth of the subarachnoid space is 6–6.5 cm in an adult llama and 2–2.5 cm in a neonate. The floor of the spinal canal is approximately 8 cm in an adult llama and 3.5 cm in a neonate.

Cytologic and biochemical values for CSF in camelids, as well as interpretation of abnormal values, is discussed in Chapter 14.

Radiography

Radiography is an important component of clinical diagnosis of the skeletal system and is discussed in detail in Chapter 5.

Ultrasonography

Ultrasonography is now employed in evaluating the reproductive system, heart, and digestive system, and is used to direct biopsy needles to the liver and kidney.

Endoscopy

Endoscopy can be a useful diagnostic technique in camelids. It can be used to evaluate the upper airway and trachea as well as the esophagus. Camelids have a smaller ventral nasal meatus than do other livestock species and which may preclude passage of larger diameters endoscopes. GI

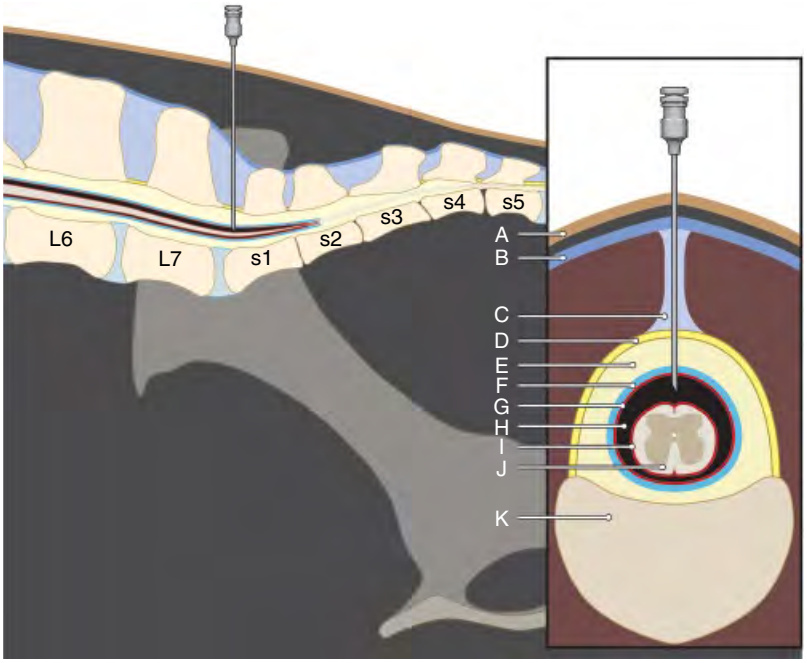


Figure 4.41 Diagram illustrating collection of cerebrospinal fluid from the lumbosacral area. The inset is a cross section at the lumbosacral space to illustrate layers penetrated during collection of CSF. (A) Skin, (B) Lumbosacral fascia, (C) Interspinous ligament, (D) Interarcuate ligament, (E) Fat within epidural space, (F) Dura mater, (G)Arachnoid meningeal layer, (H) Subarachnoid space, (I) Pia mater, (J) Spinal cord, (K) Vertebral Body.

Figure 4.42 Lumbosacral space, dorsal view.

endoscopy is typically limited to the esophagus as it is typically not useful in adults distal to the esophagus. Even with extensive fasting, the contents of the first gastric compartment typically obscures visualization of the C1 surface or GI structures abroad to C1.

Exploratory Laparotomy and Laparoscopy

Abdominal exploratory surgery may be a useful diagnostic tool that can also have therapeutic benefits. If a surgical lesion is high on the list of differentials, then surgical exploration is indicated if other diagnostics are inconclusive. Obstructive trichophytobezoars are among the most common GI lesions affecting camelids and warrant surgical exploration. Laparoscopy can be used as a less invasive alternative to laparotomy and may offer superior visualization. Laparoscopy does require specialized equipment.

Figure 4.43 A spinal needle has just been placed at the lumbosacral space. If spinal fluid does not spontaneously flow, a syringe can be used to gently aspirate. If unsuccessful, the syringe is removed, the stylet is reinserted, and the needle is redirected.

Radiographic Imaging

Radiographic imaging is a common diagnostic modality used for camelids. Limitations are like those encountered with other large animals and are often due to size or the temperament of the patient. Radiographs are useful for diagnosing musculoskeletal conditions but are also useful for abdominal and thoracic lesions. Most large, tertiary veterinary referral centers have computed tomography (CT) capabilities and the imaging benefits provided by this modality frequently outweigh the added costs of the procedure. Previously, it was thought that general anesthesia was necessary for the lengthy scan times [9], but in the author's experience, reduction of scan times with newer equipment permits the use of CT without general anesthesia for most patients and scans. CT, as a primary imaging modality, can be extremely rewarding; this is especially true when imaging areas of the body with complex anatomy and superimposing structures or regions with structures having similar radioopacities like the skull or abdomen (See Chapter 5).

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is a useful diagnostic tool in veterinary medicine. Several case reports on the use of MRI as a diagnostic imaging tool for certain conditions or to define the normal anatomy of camelids with MRI exist [24–31].

Arthrocentesis

Arthrocentesis is indicated when inflammatory processes involving the joint are suspected. Reference values for synovial fluid have been established in llamas, alpacas, and for multiple joints in dromedary camels. (Table 4.6) Cytological values have not been published for synovial fluid in the less athletic Bactrian camels.

Determination of the sites for arthrocentesis requires a working knowledge of their anatomy. These sites can also be used for joint lavage in cases of septic arthritis. When working with complex joints such as the carpus, tarsus, stifle, and fetlock, communication between each joint compartment must be considered because joint communication between compartments is highly variable among individuals [38].

Arthrocentesis should be performed using the aseptic technique. Light sedation, general anesthesia or a local block may be employed to restrain patients for this procedure. (see Chapter 6 on anesthesia) Joint volume and hence the amount of joint fluid within llama and alpaca joints is less than larger animals, and it may be difficult to obtain synovial fluid from a normal joint. Inflamed joints typically have an increased volume of synovial fluid produced in response to the inflammation. In SACs, the joints may be accessed as follows.

Shoulder Arthrocentesis of the shoulder joint can be safely performed from the cranial aspect of the shoulder joint. SAC's have a prominent acromion process and it can be palpated on the distal aspect of the scapula. The acromion can be palpated along with the tendon of the infraspinatus muscle as it attaches to the greater tubercle of the humerus. The greater tubercle has a notch just cranial to the tendon. The needle should be inserted through the notch held horizontally and directed in a caudomedial direction (Figure 4.44).

Elbow The needle should be inserted on the lateral side of the olecranon and directed cranioventrally in a line on the axis of the olecranon. The needle should be kept close to the olecranon (Figure 4.45).

Carpus The carpus is made up of three joints: the radiocarpal, middle carpal, and the carpometacarpal joint. The middle carpal joint and carpometacarpal joint communicate in llamas, however, communication between the middle carpal and radiocarpal joints is variable [38]. Arthrocentesis is performed on the dorsal aspect of the joint, and the dorsal aspect of the joint space is widened by flexing the carpus and separating the carpal bones. The tendons of the extensor carpi radialis (medial) and the common digital extensor (lateral), which pass over the dorsal aspect of the carpus, can be palpated and should be avoided during the procedure. A 3- to 4-cm, 20-gauge needle should be used for arthrocentesis.

Stifle The anatomy of the camelid stifle differs from other large domestic animal species. The tendons of the vastus lateralis and vastus medialis muscles form a sheet that inserts partly on the dorsal aspect of the patella, but also a free portion extends around the patella to insert on the tibial tuberosity. Instead of multiple patellar ligaments, there is a sheet of tendons composed of a combination of the quadriceps group plus the tendons of the biceps femoris muscle. The stifle joint is complex with multiple joint compartments. In llamas, it has been shown that consistent communication exists between the femoropatellar, medial femorotibial and lateral femorotibial joints [38].

Arthrocentesis of the stifle joint capsule may be accomplished in three locations. The distal compartment may be entered by inserting the needle just proximal and slightly lateral to the tibial tuberosity. This requires at least an 8-cm needle. The interfemoral tibial fat pad is freely moveable and may inhibit penetration of the joint capsule unless the joint is distended. As in the horse, another site for aspiration is alongside the lateral digital extensor muscle tendon as it passes over the tibial groove on the craniolateral border of the proximal end of the tibia. A third location for aspiration

Table 4.6 Synovial fluid reference ranges for camelids.

	SAC[a] normal reference ranges [32]		Dromedary camel normal reference ranges				
Variable	**Llama**	**Alpaca**	**Juvenile radiocarpal joint [34]**	**Juvenile tarsocrural joint [35]**	**Juvenile fetlock joint [36]**	**Adult elbow joint [37]**	**Juvenile dromedary with septic arthritis [33]**
Total NCC (/μL)	369.6 ± 308.3 [100–1200]	428.5 ± 417.0 [100–1400]	600 ± 500 [80–2300]	175.8 ± 136.7 [50–678]	500 ± 400	265 ± 260	7970 ± 5000 [2800-2820 000]
PMN's (%)	0.731 ± 1.5 [0–4]	2.0 ± 4.0 [0–12]	4 ± 5 [0–15]	3.5 ± 5.1 [0–15]	2 ± 2	29.76 ± 9.95	68 ± 19 [19–94]
PMN's (/μL)	1.8 ± 4.1 [0–16]	11.2 ± 37.8 [0–168]	24 ± 49 [0–250]	8.95 ± 19.0 [0–85.0]	1 ± 2	78.9[b]	5518 ± 3600 [893–12 000]
Monocytes (%)	17.9 ± 9.5 [4–48]	25.9 ± 19.2 [4–38]	36 ± 23 [7–88]	38.3 ± 20.8 [10–85]	72 ± 22	12.34 ± 7.20	26 ± 17 [4–46]
Monocytes (/μL)	63.3 ± 50.8 [8–192]	127 ± 197.0 [12–884]	222 ± 265 [14–1000]	70.6 ± 74.6 [7.5–306.5]	383 ± 323	32.7[b]	1600 ± 1120 [104–3000]
Lymphocytes (%)	80.8 ± 9.9 [52–92]	68.5 ± 22.2 [50–96]	60 ± 25 [0–90]	58.2 ± 21.55 [15–90]	26 ± 22	57.91 ± 12.83	8 ± 7 [0–25]
Lymphocytes (/μL)	319 ± 278.0 [68–1032]	304 ± 308.0 [40–1064]	310 ± 276 [0–1380]	96.3 ± 69.4 [8.7–339.0]	116 ± 167	153[b]	830 ± 140 [0–5000]
Protein (g/dL)	2.5 ± 0.35 [2–3.8]	2.3 ± 0.23 [2.1–2.5]	2 ± 1 [1–3]	2.1 ± 0.6 [1.0–3.0]	2 ± 1	2.54 ± 1.05	3.5 ± 1 [1–5]
Specific Gravity	1.017 ± 0.003 [1.010–1.026]	1.017 ± 0.001 [1.014–1.019]					
RBC's (/μL)							350 ± 130 [0–3000]

NCC, Nucleated Cell Count; PMN, Polymorphonuclear cells (neutrophils); RBC's, Red blood cells.
Values reported as Mean ± SD; [Range].
[a] Synovial fluid samples obtained from radiocarpal and tarsocrural joints. Significant differences between species (llamas/alpacas, or between limbs or joint were not noticed in this study).
[b] Calculated from total NCC's and percentage of specific type of NCC.

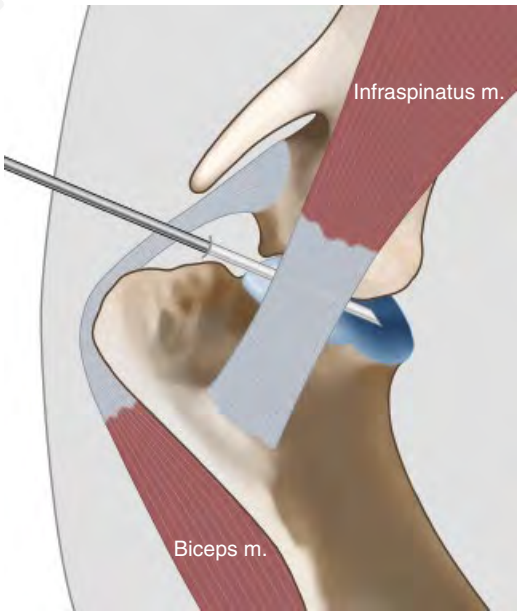


Figure 4.44 Diagram illustrating needle placement for shoulder arthrocentesis.

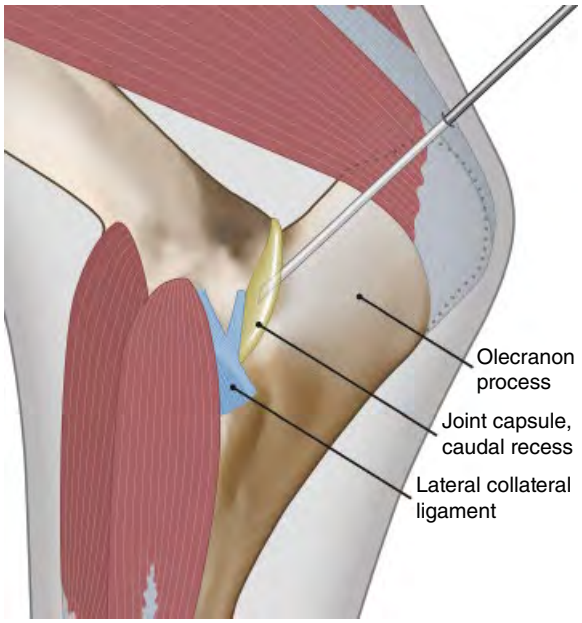


Figure 4.45 Diagram illustrating needle placement for elbow arthrocentesis.

is from the dorsolateral aspect of the patella. The needle should be inserted beneath the patella. The femoropatellar compartment is large and, if excessive fluid is present, provides the easiest access to stifle joint fluid (Figure 4.46).

Hock The needle should be inserted ventral to the lateral malleolus of the tibia and directed medially and ventrally to enter the tibiotarsal joint sac (Figure 4.47).

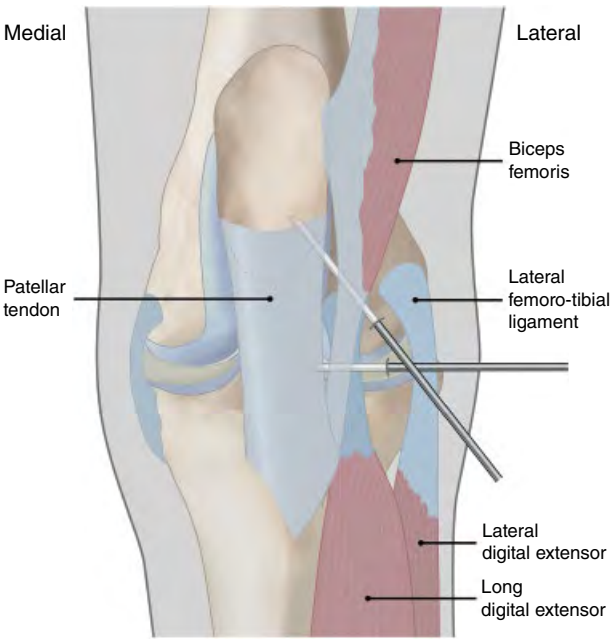


Figure 4.46 Diagram illustrating needle placement for stifle arthrocentesis.

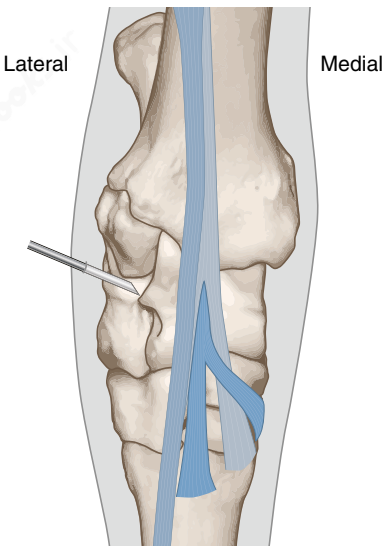


Figure 4.47 Arthrocentesis of the tibiotarsal joint as viewed from the cranial aspect of the joint. Lateral is on the left side of the image. Arthrocentesis is most easily approached by insertion of an 18-gauge needle just distal to the lateral malleolus. The needle is directed medially and slightly ventrally to enter the tibiotarsal joint space.

Fetlock and Interphalangeal joints

The fetlock and interphalangeal joints should be penetrated on the dorsolateral or dorsomedial aspect in areas devoid of tendons, vessels, and major nerves, all of which can be easily palpated beneath the skin. Flexing the joints will widen the dorsal aspect of the joints and aid in needle placement.

Notes

1 Monoject bone marrow biopsy needle, 5 cm (2 in.), 16 gauge, Sherwood Medical, St. Louis, MO, 63103.
2 Tru-cut disposable biopsy needle, 15.2 cm (6 in.), 14 gauge, Travenol Laboratories, Deerfield, IL 60015; ABC actuated biopsy needle, 15.2 cm (6 in.), 14 gauge, Monoject (Sherwood Medical), St. Louis, MO, 63103; Silverman biopsy needle, 15.2 cm (6 in.), 14 gauge, many medical suppliers.

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5

Radiology

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Introduction

This chapter focuses on radiography of camelids using normal anatomy as a reference and the basis from which abnormal radiographic findings are determined. Common disease conditions are illustrated and discussed. Computed tomography (CT) highlights challenging anatomic areas such as the skull and abdomen while ultrasound findings are presented in other chapters. In each subsection, normal anatomy is presented first and is followed by common abnormalities of that body part.

Making Radiographs

The majority of the radiographs can be made with a portable X-ray generator[1] and a digital detector[2]. In some instances, especially for larger camelids and thicker body parts (e.g. thorax, abdomen, and pelvis), a high frequency x-ray generator is needed. For extremities, a minimum of two (2) orthogonal X-ray projections are needed; this allows one to mentally create a three-dimensional image of an anatomic region. From the carpus or tarsus distally, the addition of oblique radiographs is strongly recommended due to the anatomic complexity of these regions. For larger camelids, making lateral projections of the thorax, abdomen, and parts of the spine is all that may be possible. Although, many camelids will cush on an X-ray detector, making dorsoventral (DV) projections of the thorax and abdomen possible. For some animals, sedation or general anesthesia is needed to facilitate patient positioning. Consistent, repeatable patient position is important for recognizing abnormal radiographic findings and for decreasing the number of X-ray exposures needed to complete the radiographic study.

Radiation safety is a vital part of radiography. All participants in the radiographic study – the radiographer controlling the X-ray machine, the person positioning the X-ray detector and the person holding/controlling the animal – should be wearing lead aprons, gloves and thyroid shields. The X-ray detector should be in a cassette holder that allows a person to increase their distance from the primary beam when the exposure is made. Sedation or general anesthesia facilitate patient positioning and decreases exposure of personnel to ionization radiation. The use of a radiographic technique chart minimizes the number of images made due to improper X-ray exposure. Table 5.1 is the technique chart used by The Ohio State Diagnostic Imaging Service and serves as a starting point for developing your own technique chart.

Degenerative Joint Disease

This is the most common abnormality detected in joints. Degenerative joint disease is often referred to as osteoarthritis or osteoarthrosis; these terms may be used interchangeably. Primary degenerative joint disease occurs without a specific, detectable underlying cause. Secondary degenerative joint disease occurs after an insult to a joint (e.g. osteochondrosis, ligamentous injury). In either

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Table 5.1 Technique chart used at The Ohio State University for making digital radiographs of camelids.

Region	kVp	mAs	mA	mSec	Comments
Front Leg					
Carpus	65	1			
Fetlock	65	1			
Phalanges	65	1			
Pelvis	80	40	1000	40	
Hind Leg					
Stifle – lateral	70	2–3			
Stifle – CC	70	4			
Femur	80	10–12			Use grid
Tibia	65	1–1.5			
Tarsus	65	1–1.5			
Fetlock	65	1			
Phalanges	65	1			
Skull					
DV	70	5–6			
Lateral	60	5–6			
Obliques	60	5–6			
Spine					
Cervical	70	4–5			
Thoracic	80	12–16			
Lumbar	80	12–16			
Thorax					
Cranial	80–95	12			Through elbows
Mid	80–95	6.3–8			
Abdomen	80–90	16 to 30			

instance, the radiographic changes of degenerative joint disease are similar.

Initially, degenerative joint disease begins as inflammation to the cartilage and synovium of the joint [1]. This manifests as an increase in joint fluid, or effusion, and/or thickening of the synovium (Figure 5.1). Images of a normal adult camelid (Figure 5.2) are provided for reference. Because joint effusion and synovial thickening are each soft-tissue opacities on a radiograph, it is not possible to differentiate the two. Small amounts of joint effusion may not be detected radiographically, even when detected using digital palpation on a physical exam. As the amount of joint effusion increases, it becomes easier to detect radiographically. Radiographically, joint effusion increases the soft-tissue thickness around a joint and distends the joint capsule. A normal stifle is provided for comparison (Figure 5.2).

As the joint capsule distends, the attachment of the joint capsule at the bone interface stretches. This leads to bone formation at the periarticular margins of the joint; these bone formations are called osteophytes [1]. At first, the osteophytes may be very small (Figure 5.1). In more severe cases of degenerative joint disease, the size and number of osteophytes increases (Figure 5.3). The periarticular margins of most joints also include ligamentous and tendinous attachments. Bone formation at these junctions are called enthesophytes [2, 3]. Due to the close proximity of joint capsule attachments on bone, and tendon or ligament attachment on bone, distinguishing between osteophytes and enthesophytes is not possible [1].

Changes to the subchondral bone may be seen. An increase in subchondral bone opacity, approximately 1–2 mm thick, is due to stress remodeling [1]. In severe cases of degenerative joint disease, subchondral erosions

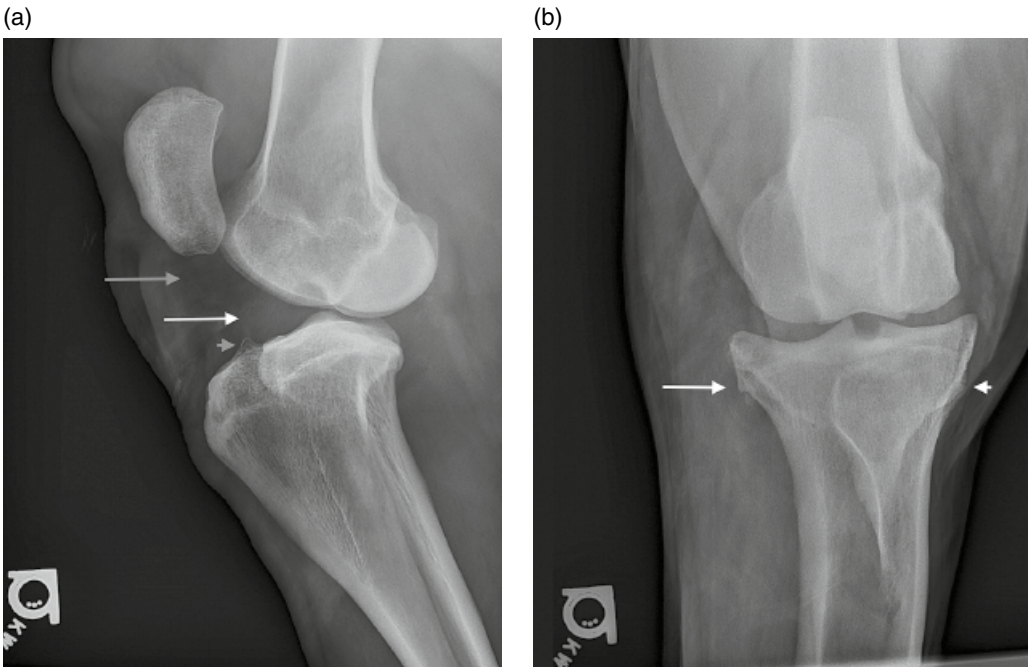

Figure 5.1 An adult llama with a left cranial cruciate ligament rupture and mild degenerative joint disease. (a) Lateral view: the proximal tibia is displaced cranially with respect to the distal femur. An increase in soft tissue (white arrow) representing joint effusion/synovitis, is within the cranial aspect of the joint causing cranial displacement of the infrapatellar fat pad (gray arrow). An enthesophyte is at the site of insertion of the cranial cruciate ligament on the proximal tibia (gray arrowhead). (b) Craniocaudal view: small osteophytes (arrows) are on the proximolateral (white arrow) and proximomedial (white arrowhead) aspects of the tibia.

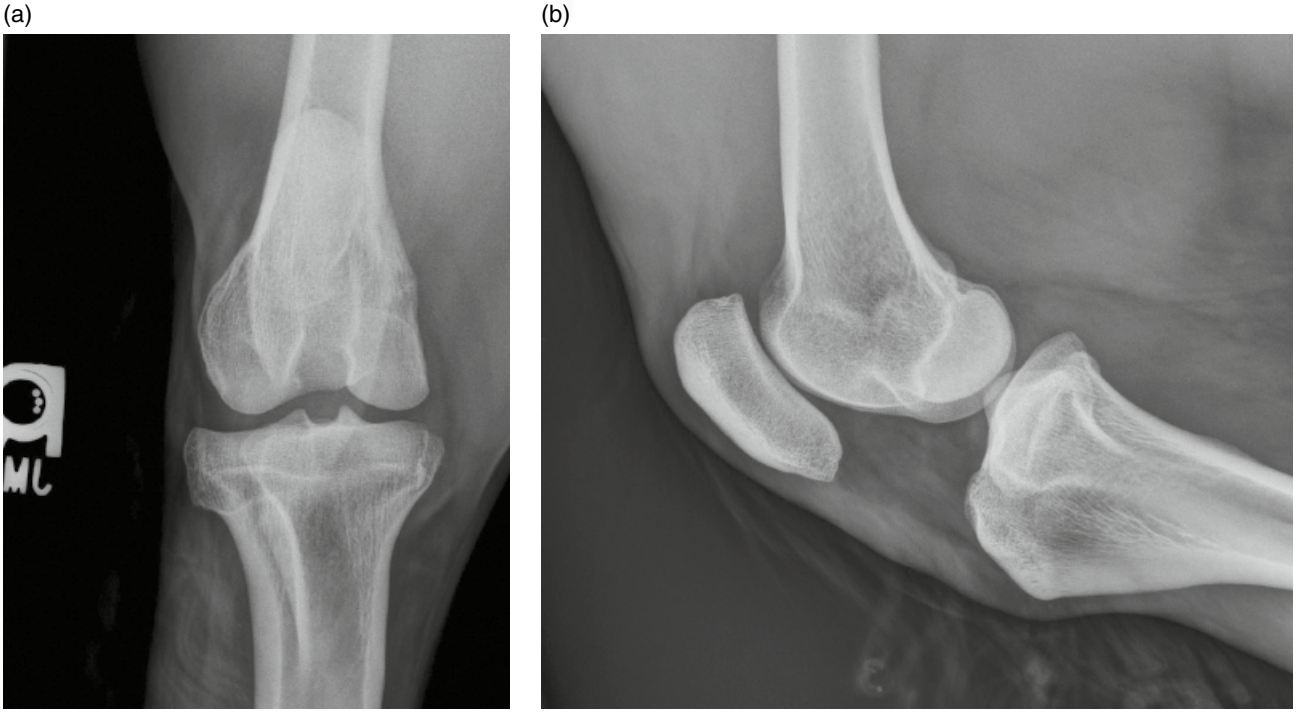

Figure 5.2 An adult alpaca right stifle. (a) Craniocaudal view: the R marker is on the lateral aspect of the limb. The patella is centered in the distal aspect of the femur. (b) Lateral view: the alpaca's fiber is seen cranial to the stifle and it summates with the stifle giving a slightly mottled appearance.

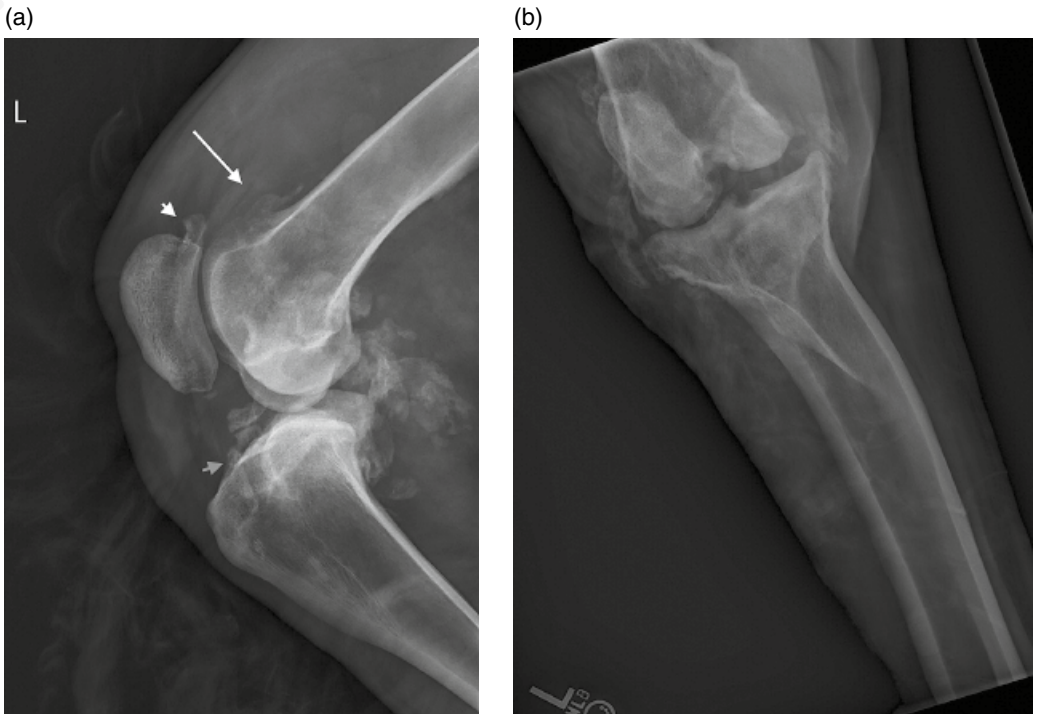

Figure 5.3 An adult llama with severe degenerative joint disease. (a) Lateral view: an increase in soft tissue, representing joint effusion/synovitis, is within the cranial aspect of the joint causing cranial displacement of the infrapatellar fat pad. Osteophytes are on the proximal aspect of the femoral trochlear ridges (white arrow). Enthesophytes are on the proximal (white arrowhead) aspect of the patella. An enthesophyte is at the site of insertion of the cranial cruciate ligament on the proximal tibia (gray arrowhead). Multiple irregular mineral opacities, representing synovial dystrophic mineralization, are in the stifle. (b) Craniocaudal view: osteophytes and enthesophytes are on the distal aspect of the femur and the proximal aspect of the tibia. Multiple irregular mineral opacities, representing synovial dystrophic mineralization, are in the stifle. The lateral aspect of the joint space appears narrower than the medial aspect. Because the patient was in lateral recumbency when the image was made (evidenced by the Mitchell marker in the bottom left corner of the image), the apparent joint space narrowing is due to patient positioning vs. cartilage erosion.

occur. Radiographically, these are rounded to irregular lucent regions at the ends of the bones that comprise the joint [1].

Occasionally, mineral fragments of various shapes and sizes are noted within a joint and they have a variety of causes [4]. Osteochondromas, or synovial dystrophic mineralization, occurs when chronic inflammation leads to mineralization of the synovium (Figure 5.3). These round mineral structures may move around within the joint, or they may be adhered to the synovium. In cases of secondary degenerative joint disease, the osseous bodies may be related to the pathology that incited initial inflammation within the joint. In animals with osteochondritis dissecans (OCD), the mineral in the joint is a mineralized cartilage flap. For animals with ligamentous injury, such as cranial cruciate ligament injury, the mineral in the joint may be an avulsion fragment.

Lastly, the appearance of degenerative joint disease is quite varied and can occur in every joint. The degree of degenerative change does not directly correlate to the degree of pain or lameness. Some animals with minimal degenerative joint disease are very sore/lame, while those with severe radiographic degenerative joint disease have minimal soreness/lameness.

Septic Arthritis

This condition affects any joint and the radiographic changes are similar for each joint. Introduction of microorganisms into a joint may be due to direct trauma (e.g. penetrating wound, spread from infection adjacent to the joint, iatrogenic due to arthrocentesis) or via systemic infection [5]. Direct trauma usually affects one joint, while systemic infection often involves multiple joints [5, 6]. In young animals, the systemic infection may be the result of failure of passive transfer [6].

Radiographically, the changes are similar in all species. Initially, the soft tissues in and around the joint are swollen. This may cause joint space widening, especially if the animal is non-weight bearing. As the disease worsens, articular erosion occurs and reactive bone develops around

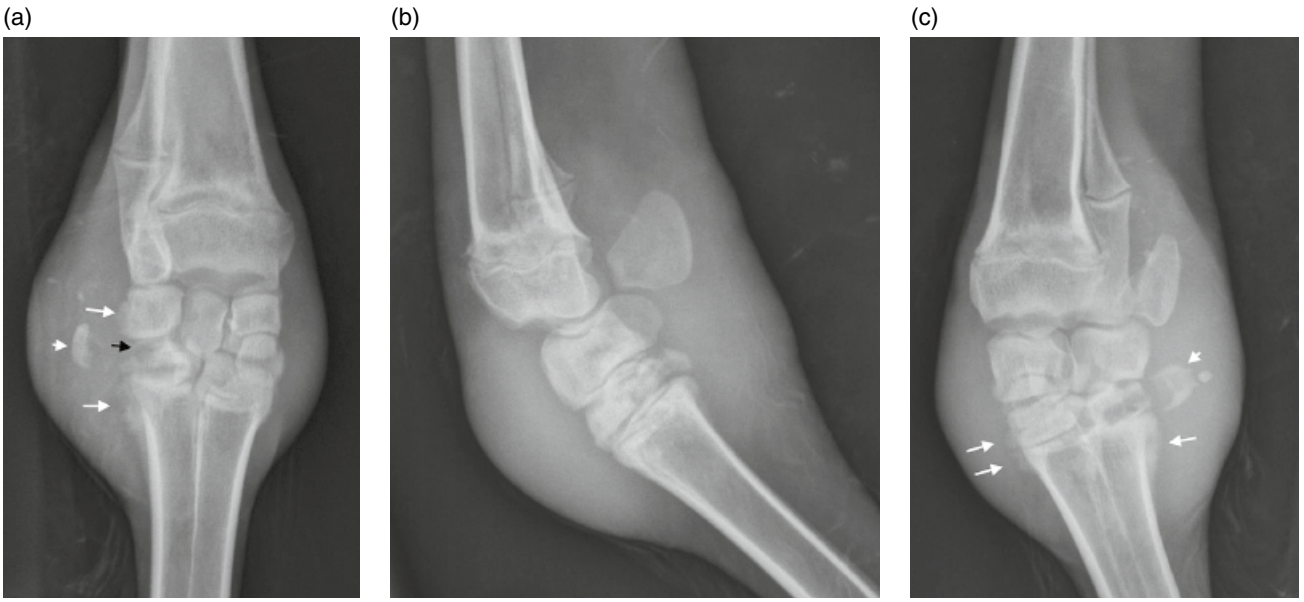

Figure 5.4 An immature alpaca with septic arthritis. (a) Craniocaudal view: the soft tissues surrounding the carpus are severely swollen, especially laterally. Osteolysis is associated with the fourth carpal bone (black arrow) and this bone is partially collapsed. Irregular periosteal reaction is on the lateral aspect of the ulnar carpal bone and proximal lateral fourth metacarpal bone, respectively (white arrows). A rectangular bone fragment (white arrowhead) with irregular margins is in the soft tissues lateral to the carpus; this is a pathologic fracture of the fourth carpal bone. (b) Lateral projection: the carpus is partially flexed because the patient was nonweightbearing. The soft tissues cranial and caudal to the carpus are thickened. (c) Craniolateral-caudomedial oblique projection: the soft tissue surrounding the carpus are thickened. Irregular periosteal reaction is on the cranial aspect of the third carpal bone, the proximomedial aspect of the third metacarpal bone and the proximal lateral aspect of the fourth metacarpal bone (white arrows). A pathologic fracture (white arrowhead) of the fourth carpal bone is displaced into the soft tissues caudolateral to the carpus. The round mineral opacity adjacent to the pathologic fracture is the fifth carpal bone.

the joint margins (Figure 5.4) [7]. Normal carpi from two immature animals are provided for comparison (Figures 5.5 and 5.6). The diagnosis of septic arthritis is confirmed using arthrocentesis [5–7].

Sequestra

A sequestrum is a devitalized fragment of bone that is avascular and non-viable [8]. These occur as part of bone trauma when a bone fragment, or sequestrum, loses its blood supply [9]. Interestingly, in camelids as compared to other livestock, infection, not trauma, is the most common cause for sequestra, especially in juvenile camelids [8]. The overlying periosteum is also devitalized and this area of bone is typically infected [8]. Because of the lack of blood supply to the sequestrum, it retains its sharp margins due to lack of remodeling. The region around the sequestrum, an area of necrosis, is relatively radiolucent on radiographs while the bone around the lucent region is sclerotic and has periosteal remodeling. Often, the periosteal remodeling is exuberant (Figure 5.7). Soft-tissue swelling also occurs at the site of sequestration. Sequestra are most commonly found in long bones, but are reported in the mandible, ribs and an ulnar carpal bone [8]. Surgical removal and debridement of the region is the treatment of choice [8, 9].

Shoulder

Lateral luxation of the humerus is an infrequent occurrence that happens more frequently in young, male camelids [9–11]. Shoulder luxation is usually secondary to trauma and may involve concurrent distal scapular and/or proximal humeral fractures (Figures 5.8 and 5.9) [10, 11].

Making shoulder radiographs is easier if the animal can be positioned in lateral and dorsal recumbency; this is facilitated by appropriate sedation and is similar to making shoulder radiographs in small animals [12]. Because the patient will be lying on the X-ray detector, placing the X-ray detector into a cassette tunnel protects the X-ray detector from the weight of the patient. Alternatively, smaller patients can be imaged on a small animal X-ray table. With the patient in lateral recumbency, the affected shoulder should be against the X-ray table or detector. The affected leg should be pulled forward and slightly distally so that the shoulder is not superimposed on the cranial thorax or

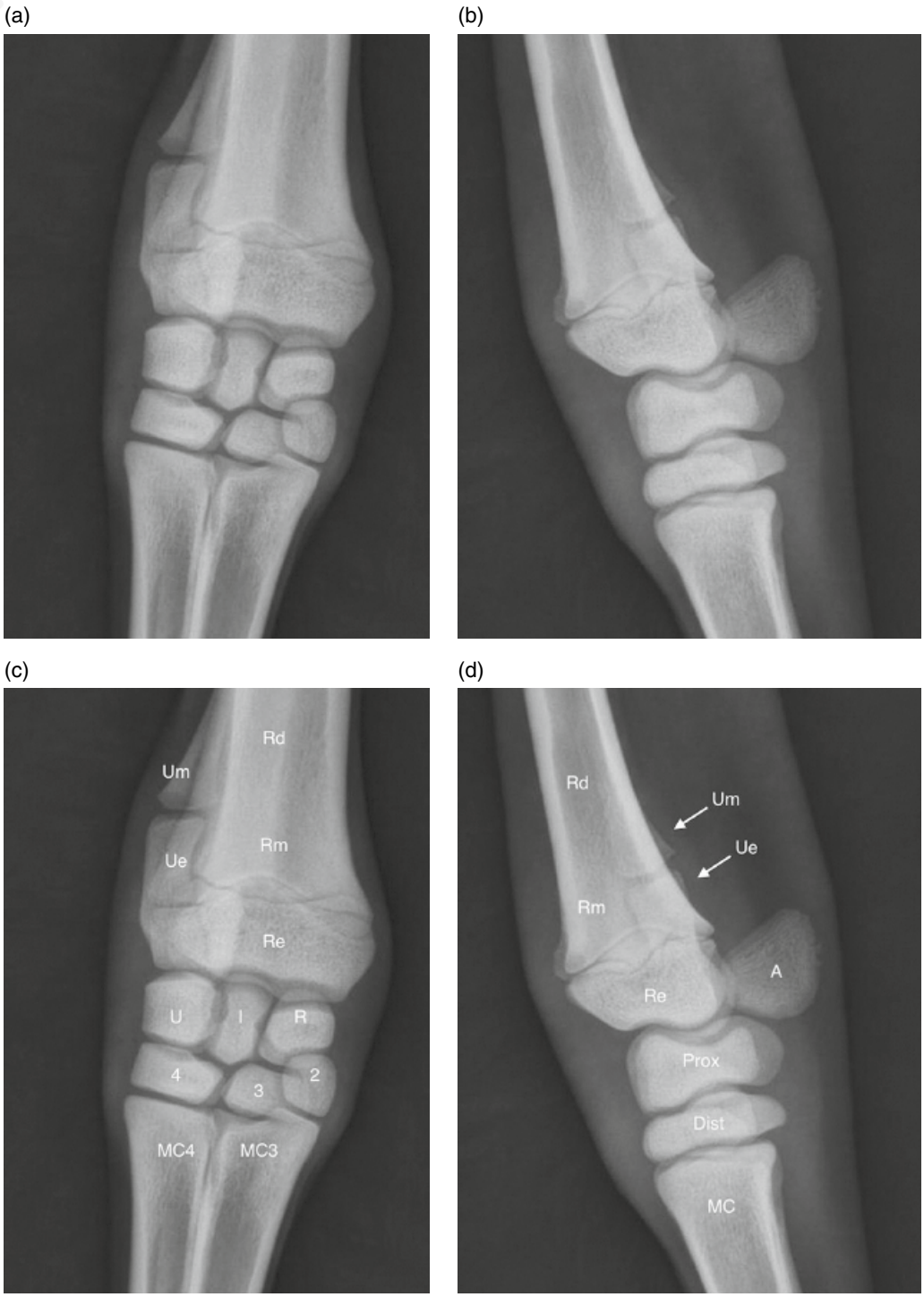

Figure 5.5 Normal carpus from an immature llama. (a and c) Dorsopalmar view. (b and d) Lateromedial view. Rd – radial diaphysis; Rm – radial metaphysis; Re – radial epiphysis (the wavy lucent line between the radial metaphysis and epiphysis is the physis or growth plate; Um – ulnar metaphysis; Ue – ulnar epiphysis. U – ulnar carpal bone; I – intermediate carpal bone; R – radial carpal bone; 4 – fourth carpal bone; 3 – third carpal bone; 2 – second carpal bone; MC4 – fourth metacarpal bone that is fused to MC3 – third metacarpal bone; A – accessory carpal bone; Prox – proximal row of carpal bones; Dist – distal row of carpal bones; MC – fused third and fourth metacarpal bones.

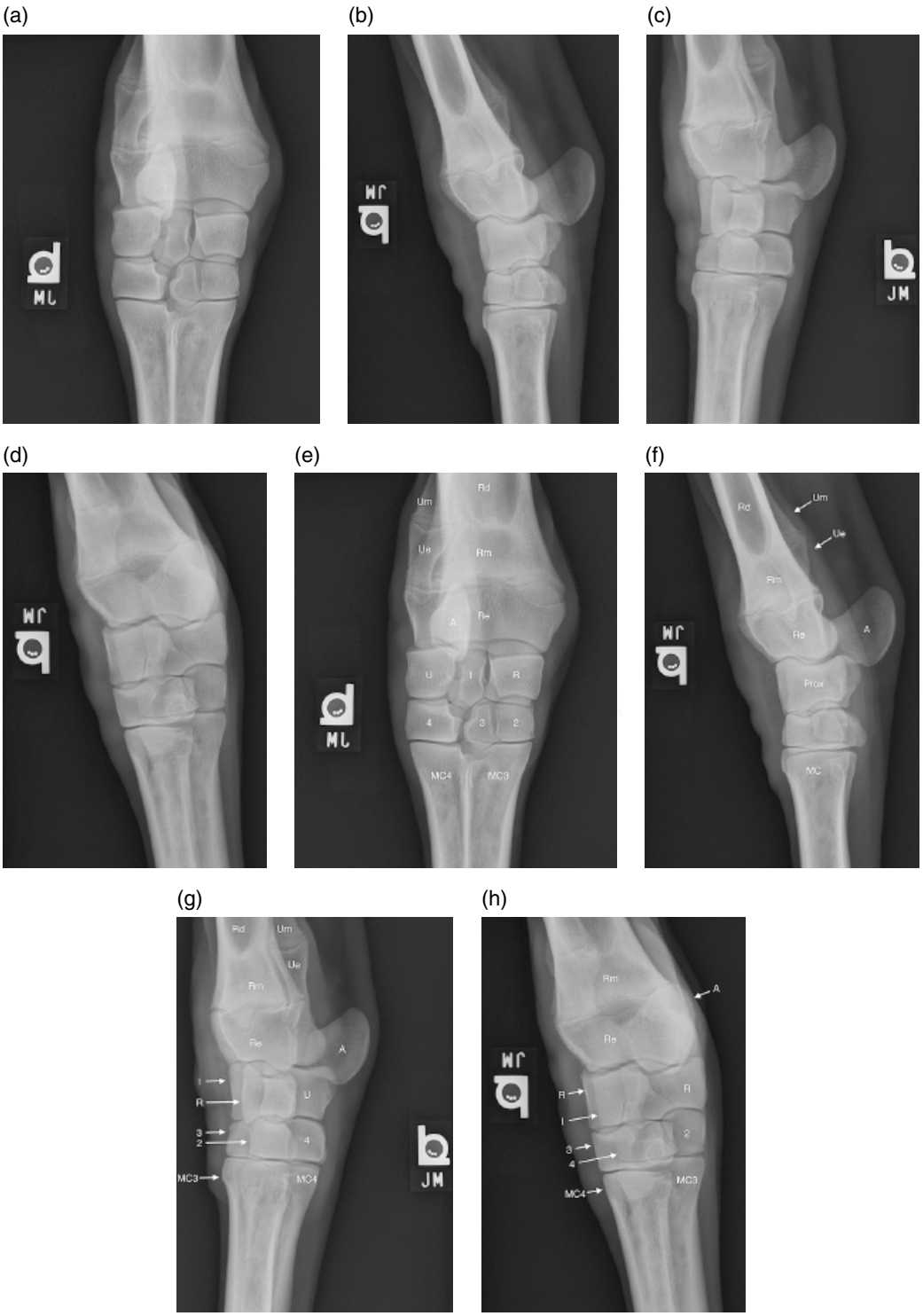

Figure 5.6 Normal left carpus from an immature llama. The left marker is on the lateral aspect of the joint except for the lateromedial view where it is typically placed dorsally. (a and e) Dorsopalmar view. (b and f) Lateromedial view. (c and g) Dorsomedial-palmarolateral oblique (DLPMO) view. (d and h) Dorsolateral-palmaromedial oblique (DMPLO) view. A – accessory carpal bone; U – ulnar carpal bone; I – intermediate carpal bone; R – radial carpal bone; 4 – fourth carpal bone; 3 – third carpal bone; 2 – second carpal bone; MC4 – fourth metacarpal bone that is fused to MC3 – third metacarpal bone.

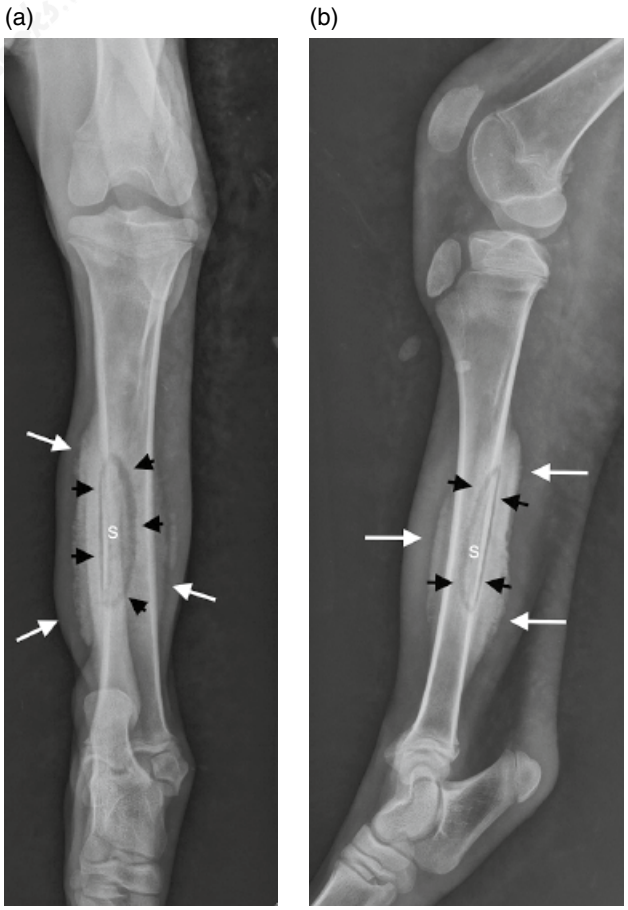

Figure 5.7 An immature llama with a sequestrum and osteomyelitis of the tibia. (a) Craniocaudal view with lateral on the left side of the image. A sharply margined, oblong bone fragment (S) representing a sequestrum is within the caudolateral cortex of the tibia. A zone of lucency, representing necrotic material (black arrowheads) surrounds the sequestrum. Smooth yet irregular bony proliferation (white arrows) is along the lateral and medial aspect of the tibia. The soft tissue surrounding the mid to distal diaphysis of the tibia are swollen. (b) A sharply margined bone fragment (S) is a sequestrum within the caudolateral cortex of the tibia. The sequestrum is surrounded by a zone of lucency representing necrotic material (black arrowheads). Smooth yet irregular bony proliferation (white arrows) is on the cranial and caudal cortex of the tibia. The soft tissues in the mid to distal diaphysis of the left tibia are thickened.

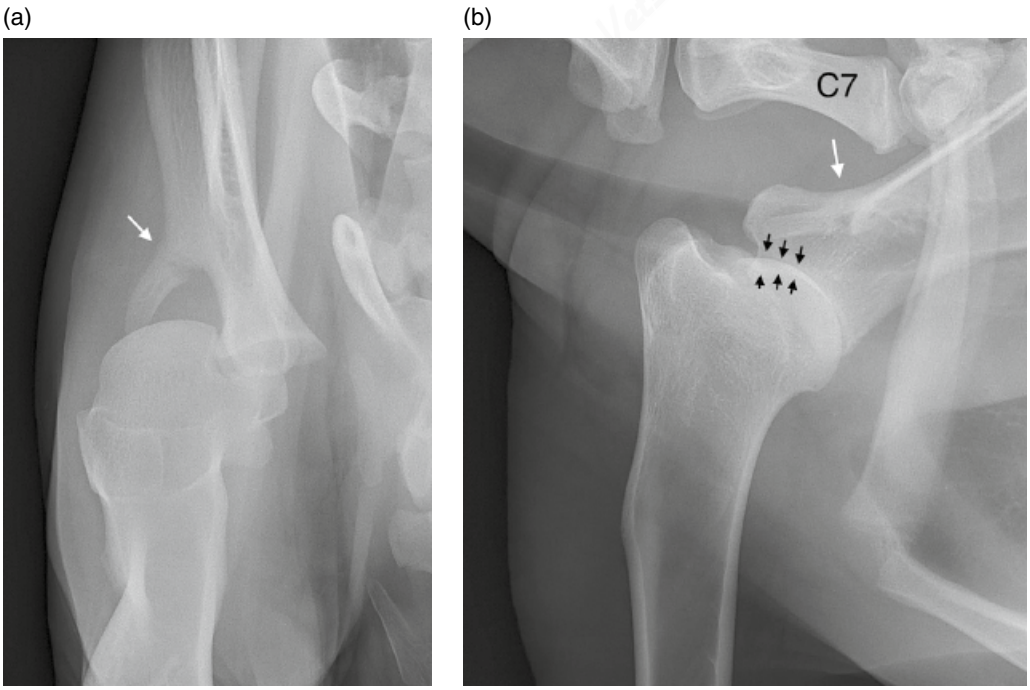

Figure 5.8 Shoulder luxation in a 6-year-old male alpaca. (a) Craniocaudal view: in the craniocaudal projection, the humeral head is laterally and mildly proximally displaced relative to the glenoid cavity of the scapula. (b) Lateral view: in the lateral projection, proximolateral displacement of the humeral head results in increased overlap between the humeral head and glenoid cavity. This overlap is seen as a thin curvilinear mineral opacity (between black arrowheads) that is due to summation of the humeral head and the distal scapula. There is also mild dorsolateral displacement and remodeling of the base of the acromion of the scapula (white arrow), consistent with a chronic fracture. (C7 = seventh cervical vertebra.)

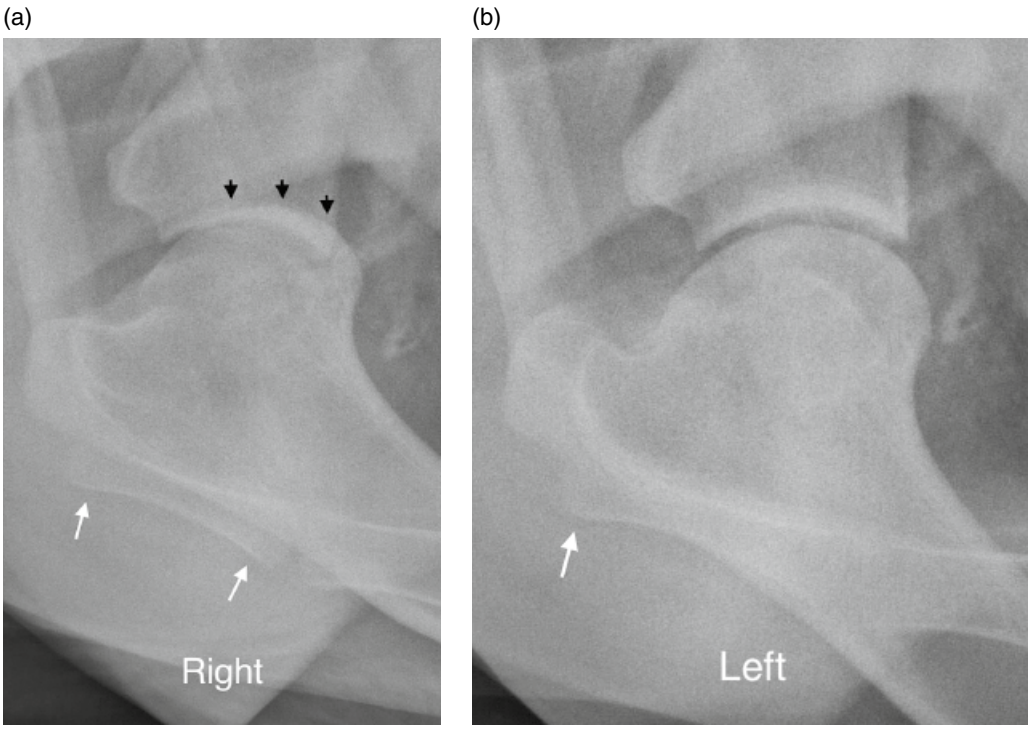

Figure 5.9 Right shoulder luxation in a llama. The lateral images were made with the patient standing and the shoulders superimposed on the cranial thorax. (a) Right: Proximolateral displacement of the humeral head results in increased overlap between the humeral head and glenoid cavity. This overlap is seen as a thin curvilinear mineral opacity (between black arrowheads) that is due to summation of the humeral head and the distal scapula. The cranial margin of the humerus overlaps the manubrium (white arrows). (b) Left: Normal. No overlap of the distal scapula and proximal humerus is seen. The cranial part of the manubrium (white arrow) extends to the cranial to the proximal humerus.

the caudal aspect of the cervical spine. Center the X-ray beam on the shoulder. For the caudocranial view, place the animal on its back (dorsal recumbency) and extend the affected leg cranially. Center the X-ray beam on the shoulder.

For a standing patient, the affected leg should be pulled forward and slightly distally so that the shoulder is not superimposed on the cranial thorax or the caudal aspect of the cervical spine. Place the X-ray detector against the lateral aspect of the affected shoulder and make the image with the X-ray beam aimed at the medial aspect of the shoulder joint. The craniocaudal/caudocranial image is challenging when the patient is standing. For a craniocaudal image, the X-ray detector is caudal to the shoulder, with one edge of the X-ray detector against the thoracic body wall (Figure 5.10). The X-ray tube may be angled slightly medially to facilitate inclusion of the entire shoulder in the image. Alternatively, one can make a caudocranial image by placing the X-ray detector cranial to the shoulder and the X-ray machine against the thoracic body wall. Regardless of the technique chosen to make a craniocaudal or caudocranial projection, the resultant image will be similar.

Making Skull Radiographs

Anatomically, the skull is a very complex structure, and this makes evaluation of the skull challenging. Proper radiographic technique and consistent patient (skull) positioning is very important as it helps increase the diagnostic value of radiography [13]. Adequate sedation greatly facilitates patient positioning (see Chapter 6 for sedation protocols). A minimum of four radiographic views are recommended for evaluation of the skull (Figures 5.11 and 5.12). These views are as follows: DV, lateral, right-to-left lateral 30°dorsal – lateroventral oblique (left-over-right oblique) and left-to-right lateral 30°dorsal – lateroventral oblique (right-over-left oblique) [13, 14]. For the oblique projections, center the X-ray beam half the distance between the ear base and lateral nostril angle on the line, parallel to the nasal margin which goes through the lateral nostril angle [14]. For the left-over-right oblique, the left aspect of the maxilla and the right mandible are highlighted, and vice versa. It is imperative that both the L and R markers are used when making these oblique images; for the left-over-right oblique, the L marker should be placed dorsal to the maxilla and the R marker should be ventral to the mandible. The opposite is true for the right-over-left oblique. With

Figure 5.10 Making shoulder radiographs. (a) Medial-lateral radiograph of the shoulder made with the patient in lateral recumbency. The neck is extended out of the way, the front leg is extended cranially and the contralateral front leg (not included in the image) extended caudally. The yellow/red area is the laser site indicating where the X-ray beam was centered. (b) Making a caudocranial, horizontal beam radiograph of the shoulder with the patient in lateral recumbency. The leg of interest is the non-recumbent leg. The X-ray detector is placed cranial to the shoulder and the elbow is extended. The right marker is taped to the X-ray detector. (c) Making a caudocranial radiograph of the shoulder with the patient in dorsal recumbency. The X-ray detector is underneath the patient and the leg of interest is extended cranially. The X-ray beam is centered on the shoulder joint.

the exception of the DV view, the addition of a mouth gag keeps the mouth open enough to separate the teeth [13, 15]. Because the camelid neck is quite flexible, the DV and oblique images can be made while the animal is cushed [13].

Tooth-Root Abscesses

Tooth-root abscesses are the most common dental disease in camelids [13]. Local swelling is the most common clinical sign in camelids with tooth-root abscesses with ~30% of animals having purulent drainage [16]. The likelihood of tooth-root abscessation in the mandibular teeth is 15 times higher than in the maxillary teeth [13, 15] and the likelihood of cheek-tooth involvement is 14 times higher than non-cheek teeth [13, 15]. Approximately 52% of animals have involvement of multiple teeth [16].

Radiographically, a tooth-root abscess is seen as a radiolucent zone around at least one tooth root (Figure 5.13). As the disease worsens, multiple teeth can be involved, osteomyelitis of the adjacent bone may occur, and the soft tissues may be swollen and have a draining tract. Acute

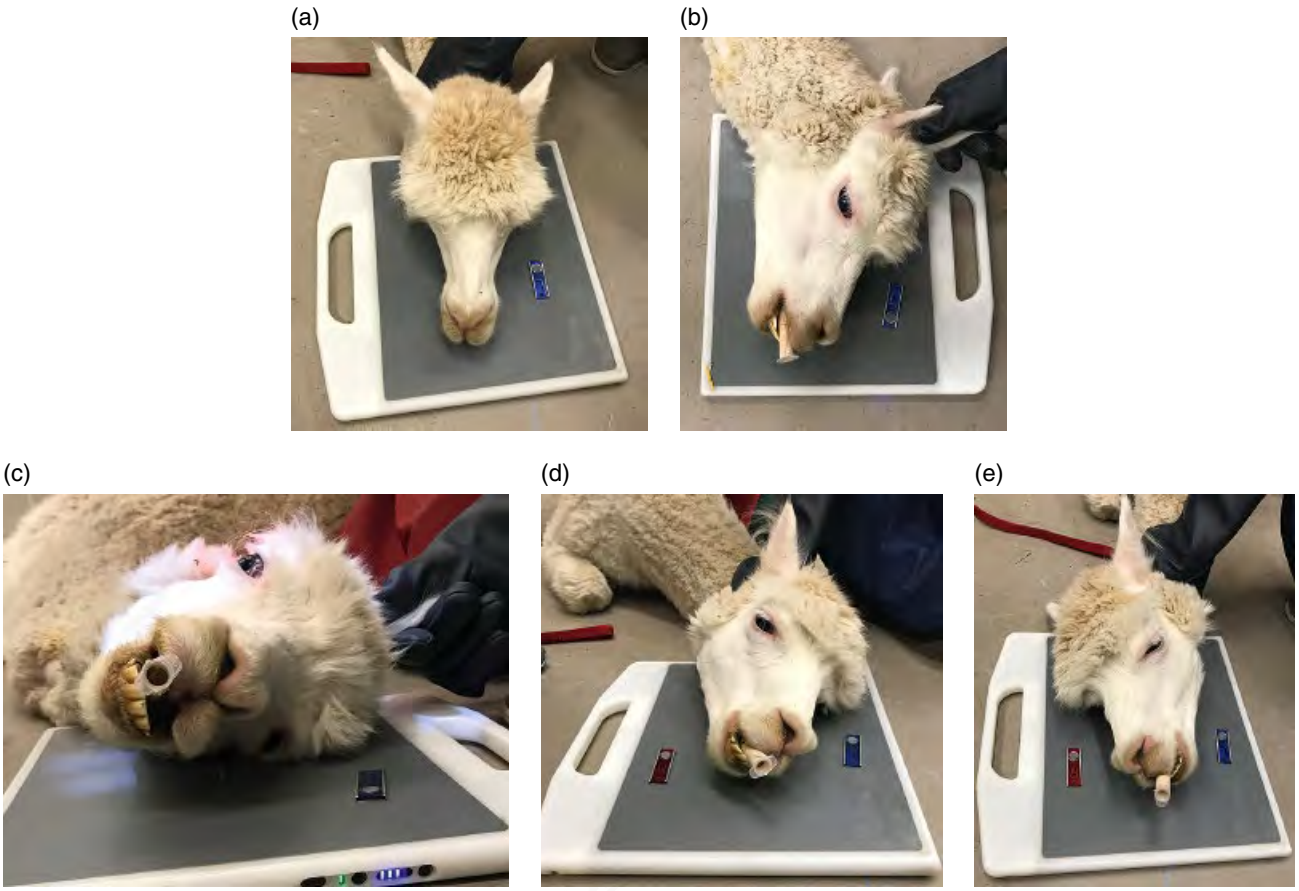

Figure 5.11 Making skull radiographs of an adult alpaca. With the exception of the dorsoventral view, the addition of a mouth gag keeps the mouth open enough to separate the teeth. Because the camelid neck is quite flexible, the DV and oblique images can be made while the animal is cushed. (a) Dorsoventral projection. The mandible (ventrum) is against the X-ray detector. The patient should not be tilted toward one side. The right and left sides of the skull should be symmetric. A marker denotes the left side of the patient. Using digital radiography, a few seconds after the exposure is made, one can evaluate the resultant image to determine if the dorsoventral projection is straight. If the patient is slightly tilted, then one can reposition the patient's head and make a second exposure. (b) Right-to-left lateral projection. An L marker is placed on the X-ray detector because the left side of the patient is against the X-ray detector. The head should be positioned such that the mandibles are parallel to one another and parallel to the X-ray detector. (c) This image shows how the patient's mandibles will be elevated from the X-ray detector so that they are superimposed over one another. The nose should also be parallel to the X-ray detector. (An X-ray exposure is not made from this vantage point). (d) Right-to-left lateral 30°dorsal – lateroventral oblique (left-over-right oblique). (e) Left-to-right lateral 30°dorsal – lateroventral oblique (right-over-left oblique) For the oblique projections, center the X-ray beam half distance between ear base and lateral nostril angle on the line parallel to nasal margin which goes through the lateral nostril angle. For the left-over-right oblique, the left aspect of the maxilla and the right mandible are highlighted, and vice versa. It is imperative that both the L and R markers are used when making these oblique images.

osteomyelitis is typically seen as amorphous osteolysis and irregular periosteal reaction. Chronic osteomyelitis has osteolysis and the periosteal reaction is typically smoother. Open fistulas are usually seen with gas in the soft tissues. If an abscess involves a maxillary tooth root, sinusitis may occur adjacent to the infected tooth.

Making skull radiographs can be laborious given all of the images needed for a complete study. Because of the complexity of the skull, interpretation of skull radiographs is challenging. CT, if available, simplifies patient positioning (typically in sternal recumbency) and provides rapid acquisition of images. Additionally, contrast resolution of the images is better than radiography and this cross-sectional modality does not have superimposition of anatomic structures [14]. Depending on the CT scanner, studies can be obtained with the patient under sedation vs. general anesthesia [17]. The imaging findings of tooth-root abscesses using CT are similar to the radiographic findings mentioned above.

Bulla Disease

For camelids with suspected otitis media, the radiographic series is as follows: DV, lateral, left-to-right lateral 30°dorsal – lateroventral oblique (right-over-left oblique)

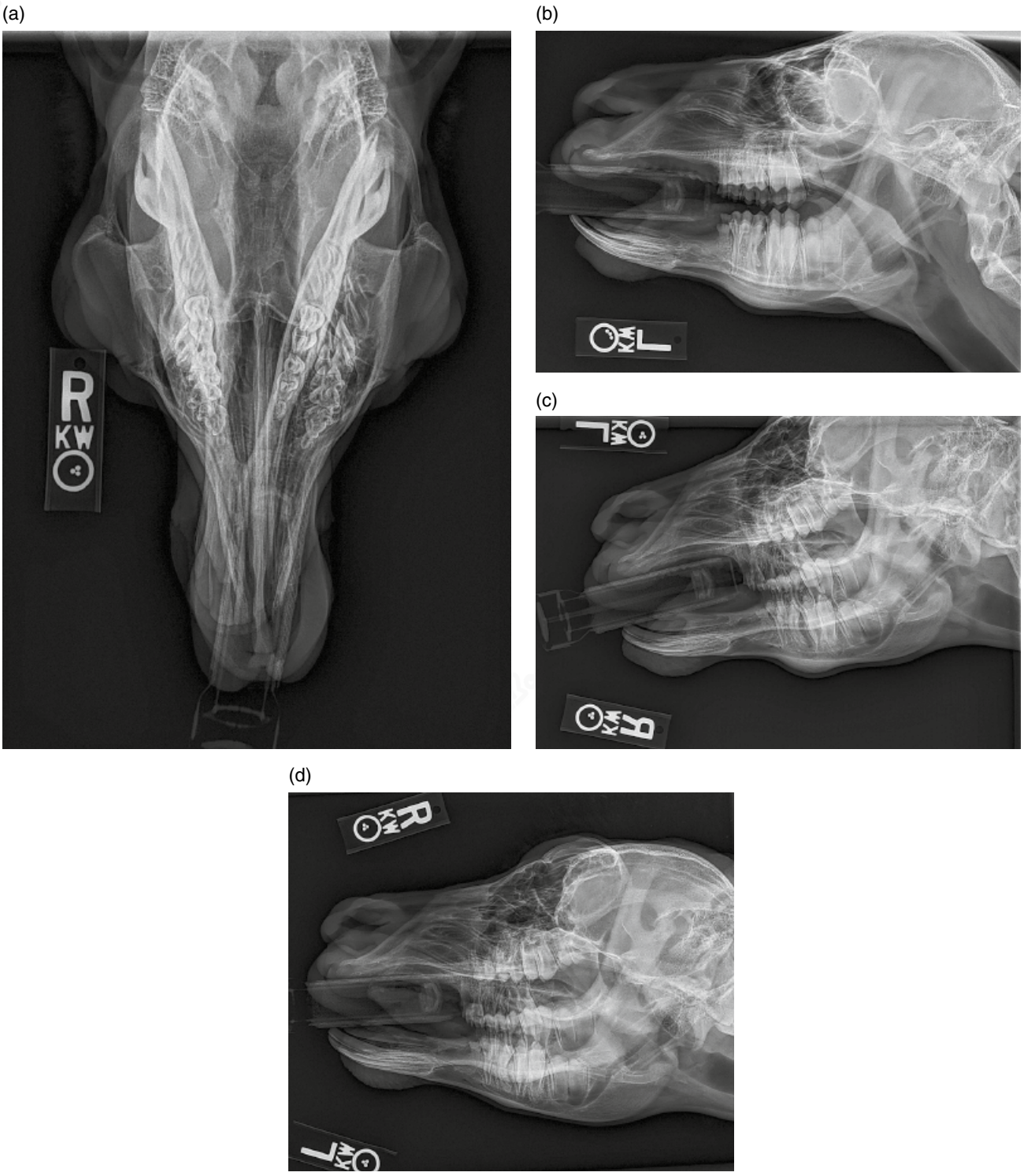

Figure 5.12 Normal skull of an adult alpaca. A mouth gag (an unopened 60 mL syringe in its plastic case) separates the molars. (a) Dorsoventral view. (b) lateral view made with the patient in left lateral recumbency. (c) Right-to-left lateral 30°dorsal – lateroventral oblique (left-over-right oblique). This view highlights the left maxilla, especially the left maxillary molars and the right mandible. (d) Left-to-right lateral 30°dorsal – lateroventral oblique (right-over-left oblique). This view highlights the right maxilla, especially the right maxillary molars and the left mandible. (e) Right-to-left lateral 30°dorsal – lateroventral oblique (left-over- right oblique) with left maxillary and right mandibular cheek teeth numbered using the Triadan system. (f) Left-to-right lateral 30°dorsal – lateroventral oblique (right-over-left oblique) with right maxillary and left mandibular cheek teeth numbered using the Triadan system.

(e)

(f)

Figure 5.12 (Continued)

(a)

(b)

(c)

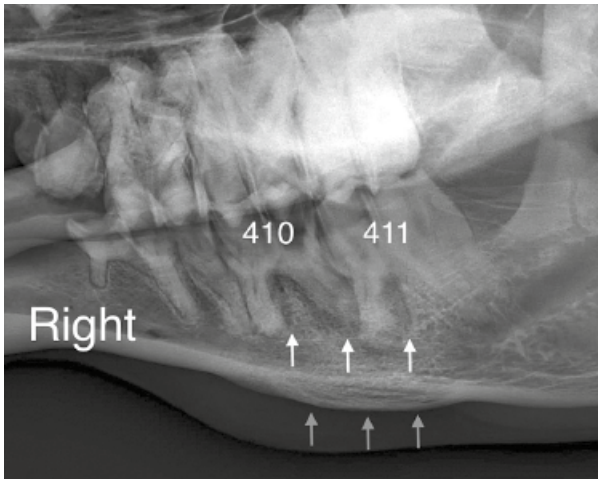

Figure 5.13 An adult llama with right mandibular second mandibular (410) and third mandibular (411) molar tooth root abscesses. (a) Right-to-left lateral 30°dorsal – lateroventral oblique (left-over-right oblique) view. A zone of lucency is around the roots of the right second mandibular molar (410) and the rostral root of the right third molar (411). The soft tissues ventral to the right hemimandible are mildly thickened. The mandibular cortex ventral to the affected teeth is thickened and mildly mottled. The left maxillary cheek teeth are within normal limits. (b) Left-to-right lateral 30°dorsal – lateroventral oblique (right-over-left oblique) view. The left mandibular molars and right maxillary cheek teeth are within normal limits. (c) Close-up view of the right mandible showing the zones of lucency (white arrowheads) around the caudal root of the second mandibular molar (410) and rostral root of the third mandibular molar (411). The mandibular cortex ventral to the affected teeth is thickened and mildly mottled (white arrows). The soft tissues in this region are mildly thickened.

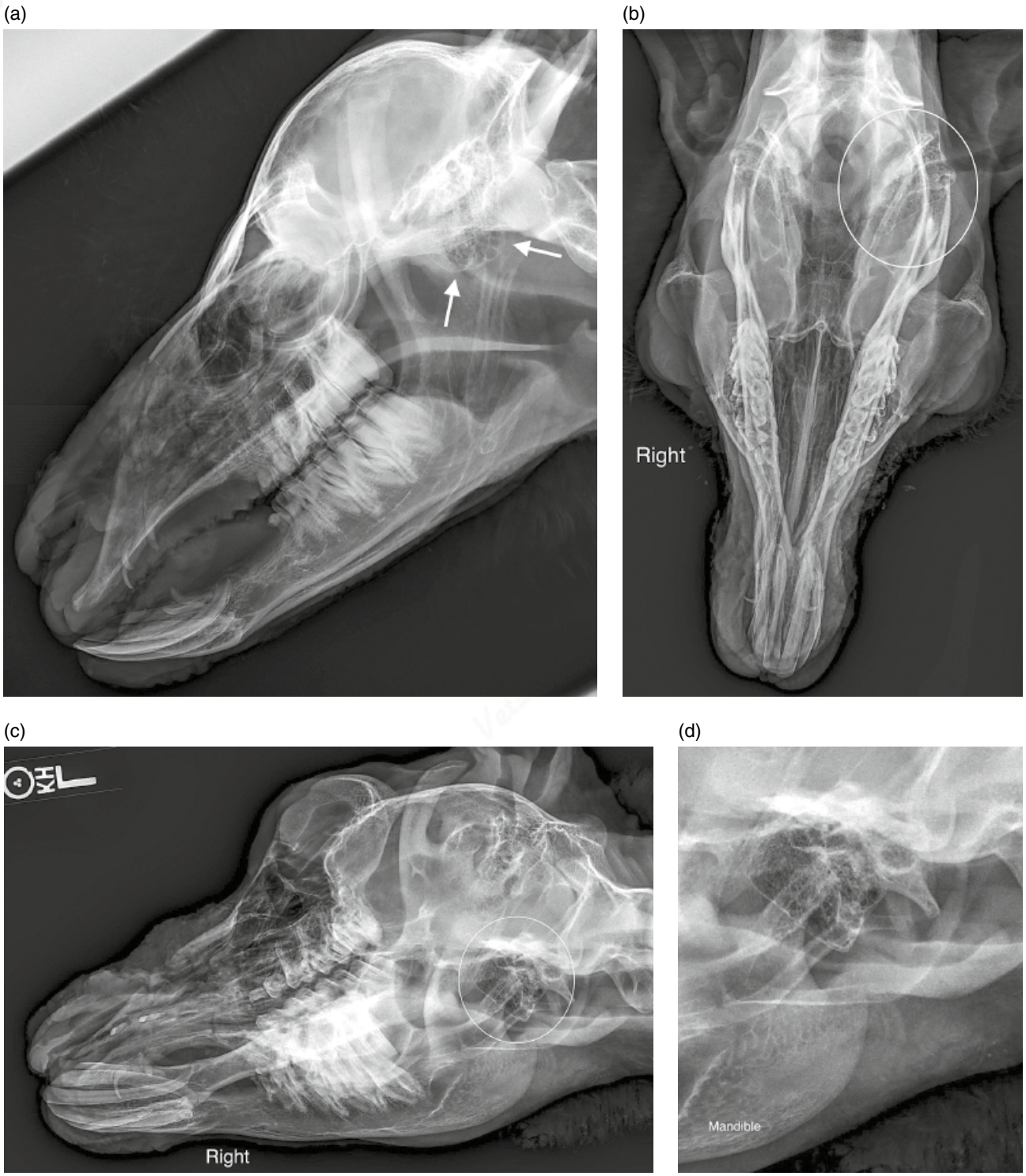

Figure 5.14 Normal skull from an adult llama illustrating the location and appearance of the tympanic bulla. (a) Lateral view with the tympanic bulla superimposed over one another (white arrows). (b) Dorsoventral view of the skull with a normal left tympanic bulla within the white circle. (c) Right-to-left laterodorsal – lateroventral oblique (left-over-right oblique) view highlighted in the right tympanic bulla (white circle). (d) Close-up of a normal tympanic bulla as seen on an oblique projection.

and right-to-left lateral 30°dorsal – lateroventral oblique (left-over-right oblique). The normal tympanic bullae are large, gas filled and have a thin, bony honeycomb appearance (Figure 5.14). In animals with otitis media, the tympanic bulla fills with soft tissue and various changes to the bony structures are seen (Figure 5.15). The bone may thicken and/

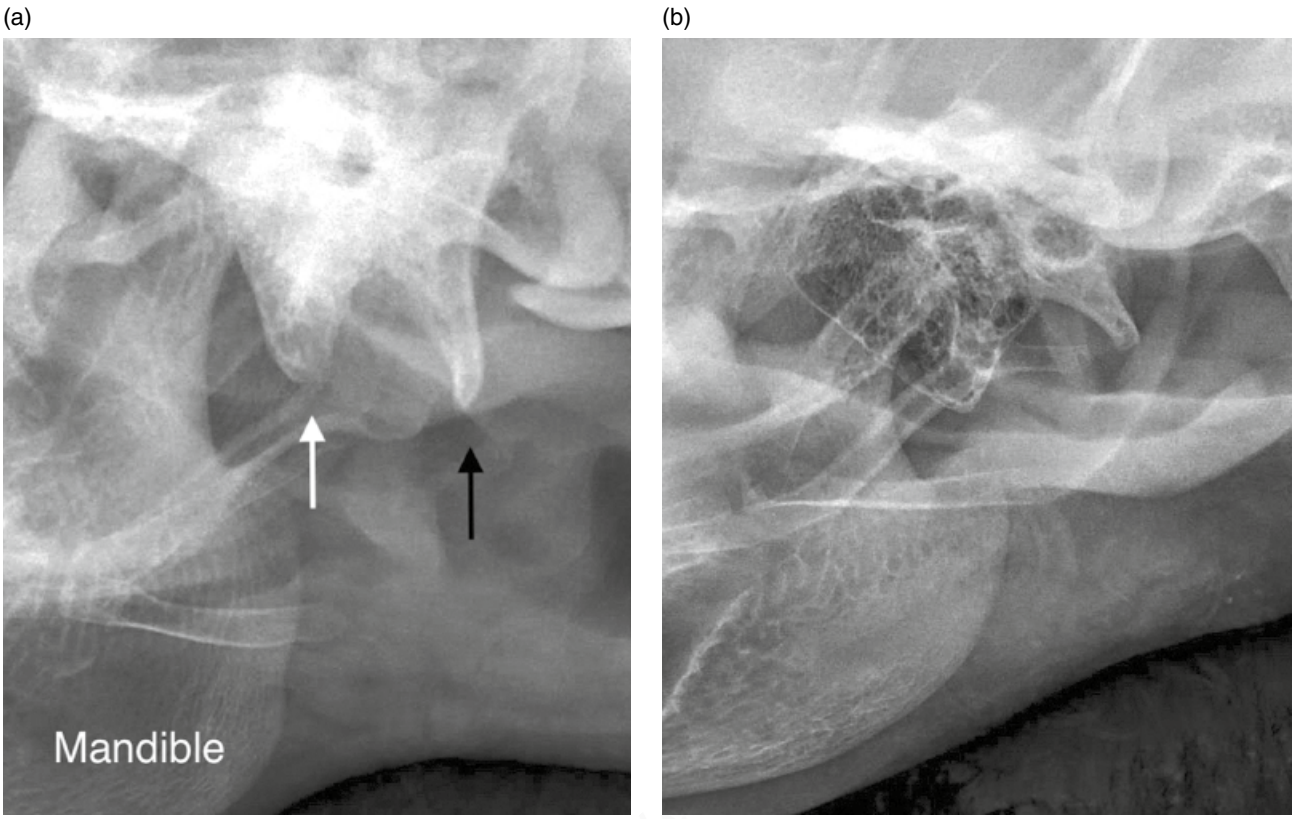

Figure 5.15 Adult llama with otitis media. (a) Oblique view highlighting a soft-tissue opaque tympanic bulla (white arrow). The normal honeycomb appearance of the tympanic bulla is not seen. The paracondylar process (black arrow) is caudal to the tympanic bulla. (b) Normal tympanic bulla for comparison.

or become lytic, often depending on the severity of the disease [18–20]. CT is often the modality of choice for imaging of the tympanic bulla as the extent and severity of the disease is often better assessed (Figure 5.16) [18, 20, 21].

Figure 5.16 A transverse CT image of a llama with left otitis media. The normal right tympanic bulla is gas filled and has multiple, thin bony septations. The abnormal left tympanic bulla has an increase in soft-tissue opacity and the thin septations are thickened with mineral.

Angular Limb Deformities

Carpal valgus is the most common angular limb deformity in camelids [22]. Multiple factors influence the development of angular limb deformities including heritability, trauma and hypovitaminosis D (rickets) [22]. The net result usually affects the distal ulnar growth plate (physis) resulting in asynchronous growth of the radius and ulna where the ulna grows slower than the radius. This means the central axis of the metacarpus angles laterally with respect to the central axis of the radius. Concurrently, the distal radius often bows caudally.

When making radiographs to assess for angular limb deformities, the X-ray beam is centered on the affected joint and collimation is extended as far proximally and distally as possible; the goal is to include as much of the long axis of the antebrachium and metacarpus in a single image if possible. Craniocaudal and lateral projections are the

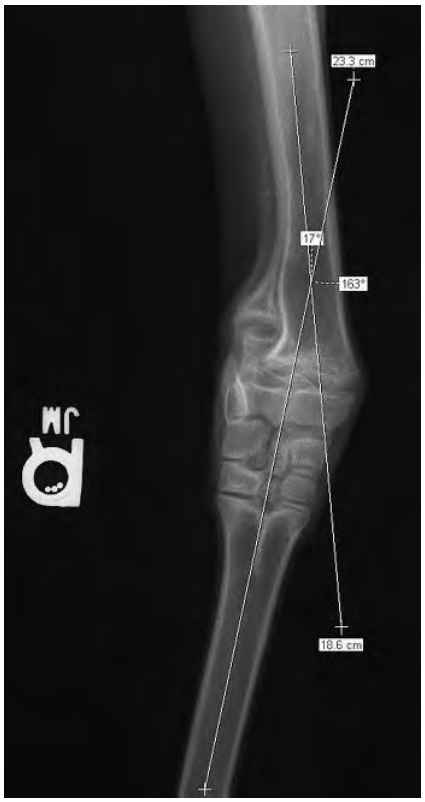

Figure 5.17 A craniocaudal view, centered on the right carpus, of an immature alpaca with rickets and a valgus angular limb deformity. To help determine the locus of the angular limb deformity, a line is drawn through the central axis of the right radius and the line is drawn through the central axis of the right metacarpal bone. The intersection of the two lines, in the distal radial metaphysis, the probable locus of the angular limb deformity. The growth of the distal right ulna is likely slower than the growth of the distal right radius allowing the carpus and metacarpus to angle laterally.

minimum views needed for this study. The degree of deformity is measured by placing a line along the central axis of the radius (bisecting the radius) and a second line along the central axis of the metacarpus (Figure 5.17). The angle of the intersection of these two lines is measured. Most digital radiography systems have an angle measurement feature that allows one to easily measure the angle. The degree of angulation inversely relates to the success of surgical intervention [22].

In addition to the angular changes to the limb, remodeling of the distal aspect of the radius and ulna occur [23]. The epiphyseal and metaphyseal regions flare while the physes are widened and rounded. The metaphyseal region also has sclerosis. The distal radial epiphysis is often wedge shaped with the lateral aspect of the epiphysis being narrow. The caudal cortices of the radius are often thickened due to stress remodeling [23]. The valgus deformities are commonly bilateral [23]. Carpal bones are usually unaffected [23].

The metacarpophalangeal and metatarsophalangeal joints can also have angular limb deformities [5, 23]. Similar to the carpus, a minimum of two orthogonal images of each joint is recommended. Angle measurements using the central axes of the distal metacarpus/metatarsus and phalanges would be made.

The angular limb changes in older camelids should be distinguished from valgus deformities of the front legs of newborns.

Patellar Luxation

Patellar luxation is a congenital abnormality in llamas [5]. Anatomically, the llama has a single patellar ligament that inserts on the tibial crest, and the patella resides in the distal femoral patellar groove [24]. In camelids with patellar luxation, the patella is displaced medially or laterally as seen on the craniocaudal view of the stifle. On the lateral projection, very little change may be noted, or the patella may be superimposed on the distal femur instead of cranial to the distal femur (Figure 5.18). In a small case series, the patella was luxated laterally in seven of eight cases [24].

Rickets

Immature camelids are susceptible to seasonal vitamin D deficiency resulting in a hypophosphatemic rickets syndrome [25, 26]. Crias with this syndrome grow more slowly, are reluctant to move, often have a humped back, shifting leg lameness and swollen joints [25]. The physes of crias with rickets are widened with flaring of the adjacent epiphysis and metaphysis (Figure 5.19). The distal antebrachium is most commonly affected, but rickets can also affect ribs [25]. In some cases, animals can develop angular limb deformities due to the physeal changes [22]. Appropriate treatment with vitamin D supplementation can correct hypophosphatemia and vitamin D deficiency in camelids [26].

Normal Images

Images of a normal alpaca foot (Figure 5.20), tarsus (Figure 5.21), and shoulder (Figure 5.22) are provided. When making radiographs of the foot, it is always important to put the laterality marker on the lateral aspect of the image. With the exception of the lateral-medial projection, anatomically, there is no way to tell medial from lateral, thus the marker serves a very important dual function.

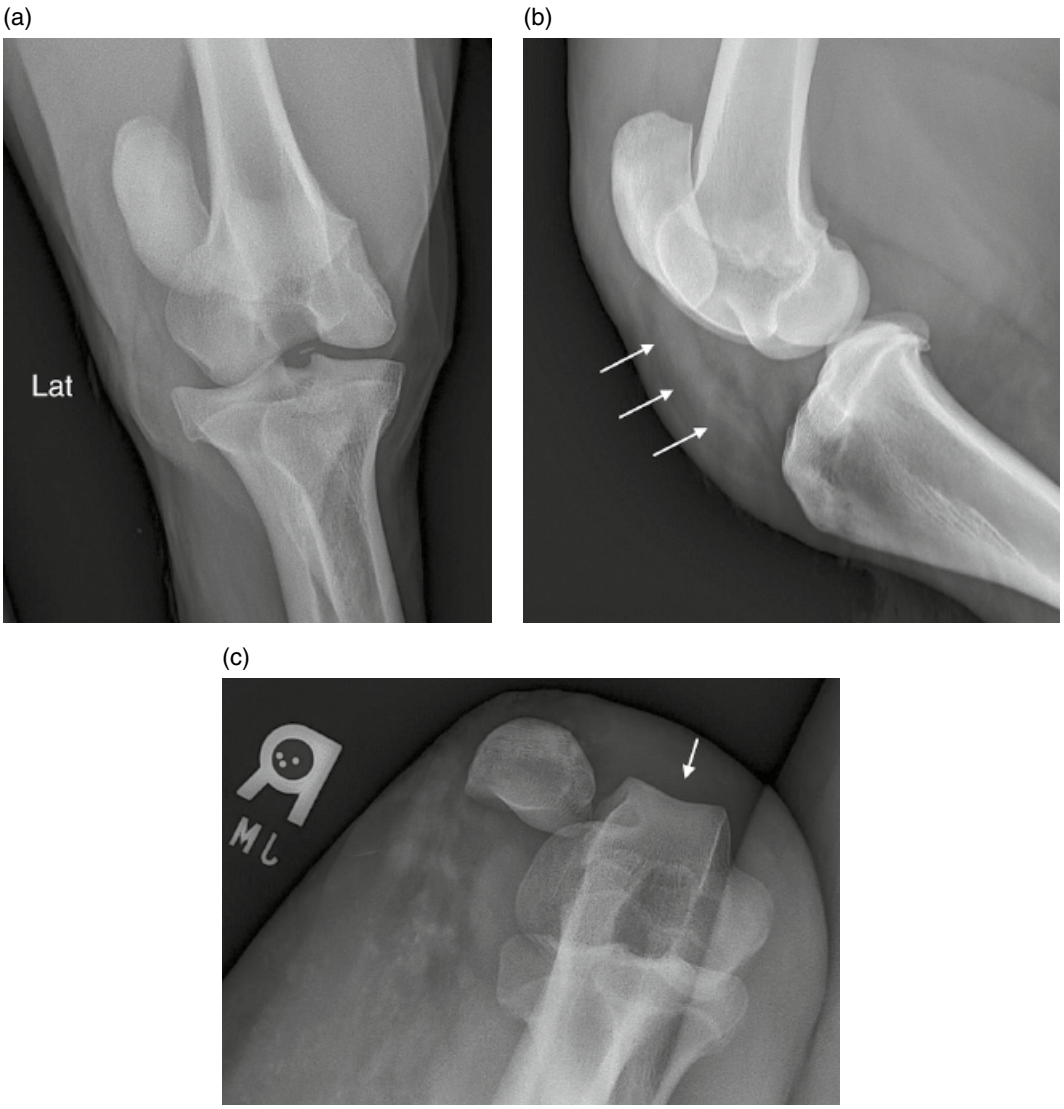

Figure 5.18 Lateral luxation of the right patella in an adult llama. (a) Craniocaudal view: the right patella is displaced laterally. A small mineral fragment of unknown origin is superimposed or within the right stifle. (b) Lateral view: the distal aspect of the right patella is superimposed on the craniodistal femur. An increase in soft-tissue opacity is within the cranial aspect of the stifle (white arrows); this bulges the stifle joint cranially. (c) A skyline view of the distal femur. The right patella is displaced laterally. The distal femoral groove (white arrow) is shallow. The patient's fiber causes mottling of the soft tissues lateral to the distal femur.

Figure 5.19 Immature alpaca with rickets (hypovitaminosis D). (a) Craniocaudal view and; (b) Lateral view: the distal ulnar physis is widened (white arrowheads) and the distal radial physis is widened (white arrows).

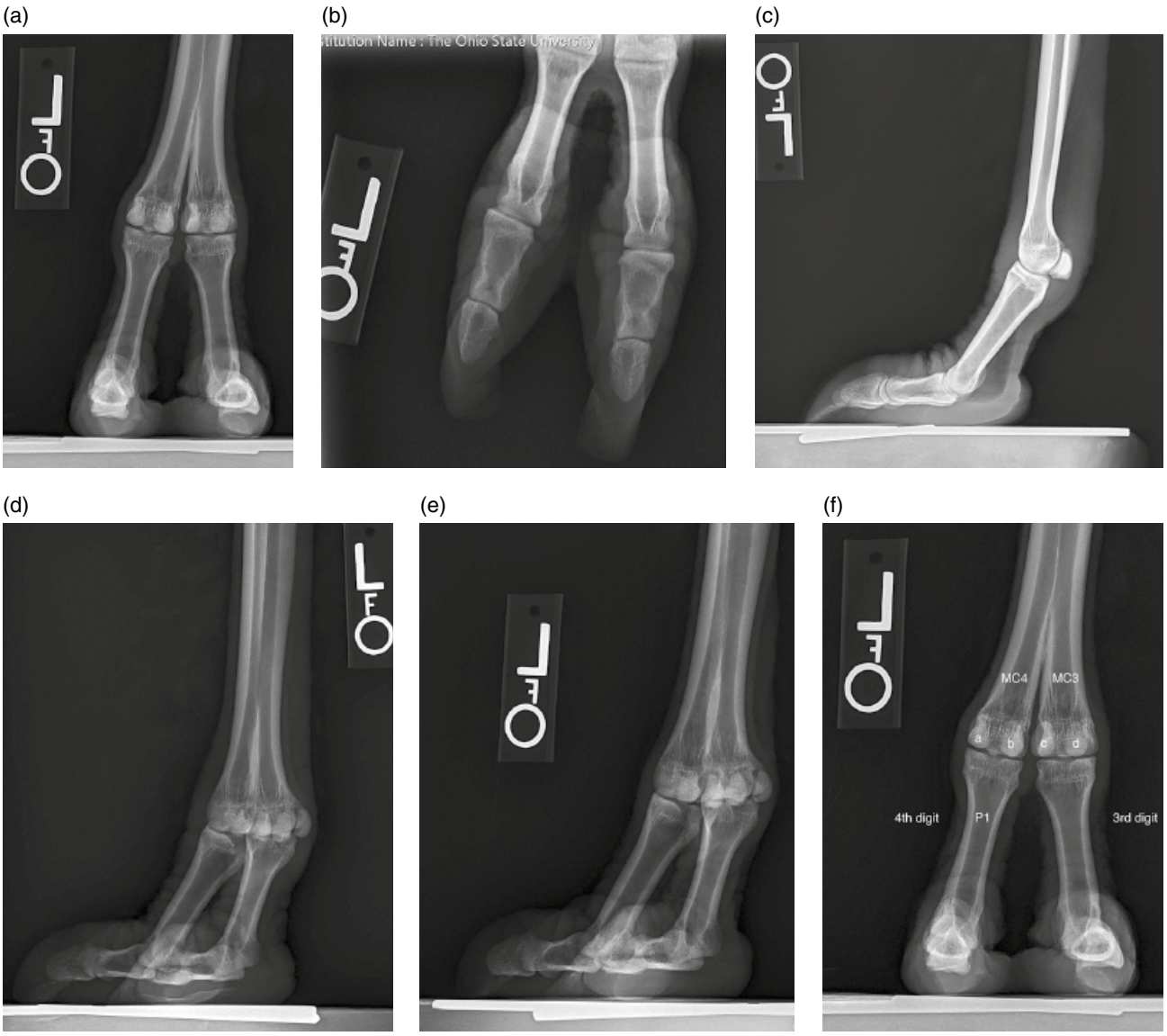

Figure 5.20 Normal alpaca foot. When making radiographs of the foot, it is important to always put the laterality marker on the lateral aspect of the image (The exception is the lateral-medial projection). Anatomically, there is no way to tell medial from lateral, thus the marker serves a very important dual function. (a and f) Dorsopalmar projection made with the X-ray beam parallel to the floor. (b and g) Dorsopalmar projection made with the X-ray beam perpendicular to the digits. (c and h) Lateral – medial projection. (d and i) Dorsolateral-palmaromedial oblique projection (DLPMO). (e and j) Dorsomedial-palmarolateral oblique projection (DMPLO). MC4 = fourth metacarpal bone; MC3 = third metacarpal bone; a = lateral proximal sesamoid bone, fourth digit; b = medial proximal sesamoid bone, fourth digit; c = lateral proximal sesamoid bone third digit; d = medial proximal sesamoid bone, third digit; PS = proximal sesamoid bones; P1 = proximal phalanx; P2 = middle phalanx; P3 = distal phalanx.

(g)

(h)

(i)

(j)

Figure 5.20 (Continued)

Figure 5.21 Normal immature alpaca tarsus. (a and e) Plantarodorsal projection. (b and f) Lateral-medial projection. (c and g) Dorsolateral-palmaromedial oblique projection (DLPMO). (d and h) Dorsomedial-palmarolateral oblique projection (DMPLO). Tib = tibia; Calc = calcaneus; T = talus; C = central tarsal bone; 4 = fourth tarsal bone; 2/3 = fused second and third tarsal bone; 1 = first tarsal bone; MT4 = fourth metatarsal bone; MT3 = third metatarsal bone.

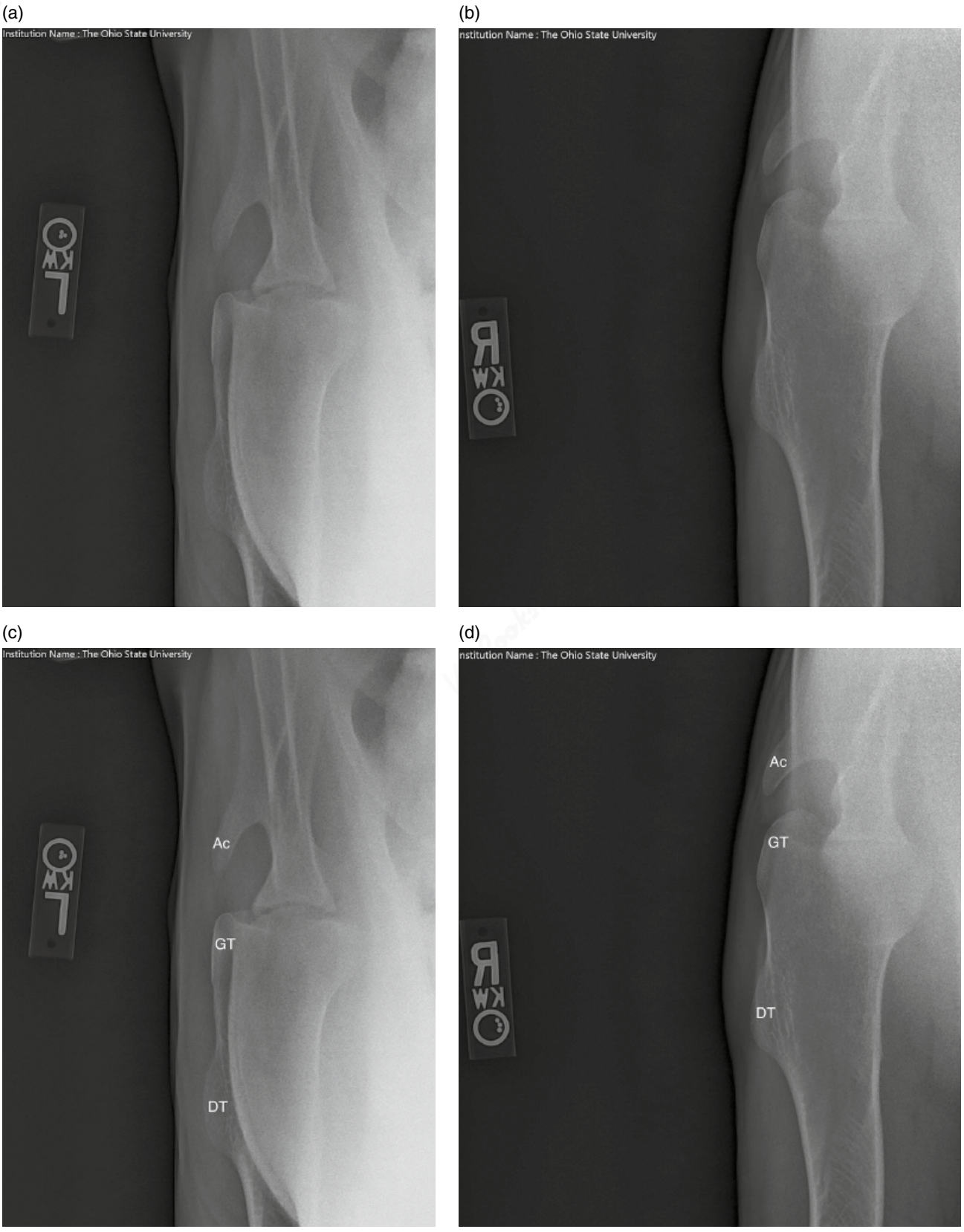

Figure 5.22 Caudocranial projections of a normal adult alpaca shoulder. (a and c) vertical beam projection made with the patient in dorsal recumbency (b and d) Horizontal beam projection made with the patient in left lateral recumbency. Ac = acromion process; GT = greater tubercle; DT = deltoid tuberosity.

Notes

1 The Ohio State Diagnostic Imaging Service use MinX.

2 The Ohio State Diagnostic Imaging Service use Canon G60.

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6

Anesthesia and Pain Management

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Pre-operative Assessment

Pre-Anesthetic Evaluation and Peri-Anesthetic Period

Pre-anesthetic evaluation should include a thorough physical examination and evaluation of blood work (packed cell volume and total protein in healthy animals, CBC/biochemistry in debilitated or sick animals). In addition, adult camelids undergoing sedation and/or general anesthesia should be held off feed for 12–24 hours prior to the procedure. Camelid patients have an increased risk of regurgitation and aspiration associated with heavy sedation/general anesthesia. Both passive and reflex regurgitation are potential sequelae to general anesthesia in camelids. The primary factors predisposing to passive flow of ingesta from the stomach are relaxation of the gastroesophageal sphincter, surgical positioning, pressure buildup in the stomach (either gaseous or external), and a large volume of ingesta in the stomach. Care should be taken to position animals in sternal recumbency with their head above their abdomen if possible (sedation) or with the pharynx elevated above the mouth (general anesthesia) prior to intubation. Camelids should be intubated if the duration of the procedure is greater than 20 minutes or in animals with an increased risk of regurgitation. In an anesthetized camelid, continuing contraction of C1 of the stomach moves ingesta toward the cardia. In a normal cycle, the cardia relaxes and ingesta is propelled to the mouth for rechewing. This cycle is a hindrance during anesthesia. These mechanisms are under parasympathetic control.

If regurgitation occurs, the poll of the head should be positioned to be the highest point to protect the airway and encourage drainage out of the mouth. In the event of regurgitation, suction may be utilized to remove gastric contents from the back of the mouth. Sometimes packed feed material may be present in the oropharynx, requiring long forceps for extraction. An intravenous (IV) catheter should be placed prior to sedation in order to provide IV access in the case of an emergency or in the event that general anesthesia is required.

Sedation

Sedation should be utilized in order to decrease stress of the patient, in patients that are fractious or aggressive, in order to limit the amount of induction drug (if general anesthesia is required), and to enhance analgesia and muscle relaxation.

Alpha-2 Receptor Agonists

Alpha-2 adrenoreceptor agonists are typically administered for sedative, analgesic, and muscle relaxant properties. Administration of alpha-2 agonists is associated with an increase in arterial blood pressure due to peripheral vasoconstriction. Heart rate decreases due to a baroreceptor reflex caused by the increased arterial blood pressure as well as a central effect due to reduced sympathetic outflow. This decrease in heart rate can manifest as second-degree AV block. Respiratory depression also occurs and can result in apnea. Additional effects include decreased cardiac output and aortic flow, depressed thermoregulation, increased urine production due to anti-ADH effects, and

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hyperglycemia due to insulin suppression. Animals sedated with alpha-2 agonists can override the sedative effect with sufficient stimulation. Additional side effects of alpha-2 agonists include muscle tremors and salivation. Intracarotid administration of alpha-2 agonists produces transient seizures and collapse [1]. Alpha-2 agonist administration can also decrease uterine blood flow.

Xylazine

Xylazine may be administered IV or intramuscularly (IM). Sedation occurs within three to 5 minutes following IV injection or 10 to 15 minutes after IM injection [2–4]. Analgesia lasts from 15 to 30 minutes. The level of sedation is dose-dependent, but the overall sedative effect can last for several hours. Common dosages administered to llamas and alpacas are 0.1–1.1 mg/kg IV or 0.2-0.9 mg/kg IM [2–4]. Xylazine administered to dromedary camels at a dosage of 0.2 mg/kg IV resulted in moderate sedation within five minutes which lasted for approximately 30 minutes [5]. Administration of 0.4 mg/kg IM of xylazine to dromedary camels resulted in sedation for up to 150 minutes and analgesia for 60–90 minutes [6]. Administration of 0.27–0.51 mg/kg of xylazine IM resulted in adequate sedation of various procedures including tuberculin testing, lymph node biopsy, and electroejaculation in Bactrian camels [7].

Detomidine

Detomidine administration in camelids results in variable sedation and restraint. In horses, it typically has a longer duration of action compared with xylazine. Detomidine may be administered IV or IM in dosages up to 0.04 mg/kg IV in llamas. Alpacas may require higher dosages than llamas for comparable sedation and/or restraint. A dosage of 0.07 mg/kg IV in alpacas resulted in onset of sedation in 1.6 ± 0.2 minutes and maximal sedation in 8.3 ± 1.3 minutes. This dose of detomidine also resulted in bradycardia [8]. Detomidine gel has been approved for oral transmucosal administration in horses and has been investigated for intravaginal use in alpacas [8]. The bioavailability was approximately 20% and maximal sedation occurred in 25 ± 4 minutes [8]. Detomidine-butorphanol combinations have been used for standing sedation in Bactrian camels at a dosage of 0.04–0.06 mg/kg of detomidine with 0.03–0.06 mg/kg butorphanol IM [9].

Romifidine

The sedative and analgesic effects of romifidine have been evaluated in dromedary camels, but not alpacas or llamas. In camels, romifidine administration resulted in dose-dependent ruminal atony, hyperglycemia, and bradycardia with no changes in respiratory rate. Profound sedation occurred at a dosage of 120 mcg/kg IV [10].

Dexmedetomidine

Dexmedetomidine has been evaluated in combination with tiletamine hydrochloride-zolazepam hydrochloride in alpacas at dosages between 5 and 20 mcg/kg (see injectable anesthetic combinations below) [11]. Dexmedetomidine at a dosage of 5 mcg/kg was administered IV to dromedary camels and resulted in moderate sedation within 5 minutes with a duration of more than 60 minutes [5].

Acepromazine

Acepromazine is a phenothiazine tranquilizer that provides mild sedation in camelids through its dopamine receptor antagonism. Acepromazine administration results in muscular relaxation and reduction of spontaneous activity, as well as antiemesis in small animal species. Cardiovascular effects are a decrease in arterial blood pressure caused by alpha-1 antagonist mediated vasodilation. This may be accompanied by a mild increase in heart rate. The vasodilation may also result in a decrease in body temperature. The respiratory effects of acepromazine are minimal. Dosages of 0.04–0.1 mg/kg IM or 0.01–0.02 mg/kg IV have been associated with reliable sedation for pre-anesthesia, catheter placement, and physical examination in case reports. When administered IV, effects are noted within one to three minutes. IM administration results in peak sedation in approximately 15–25 minutes. Acepromazine is a phenothiazine derivative and may potentiate the toxicity of organophosphate parasiticides, so inquiry should be made about the prior use of these products before administration.

Barrie reported that an adult female guanaco (approximately 100 kg) was tranquilized for an eye examination with 3 mg (0.03 mg/kg) [12]. Acepromazine maleate has been evaluated in combination with butorphanol and tiletamine-zolazepam in llamas (see injectable anesthetic combinations below). In that study, acepromazine and butorphanol were administered IM prior to IM administration of tiletamine-zolazepam. Sedation from acepromazine and butorphanol was not evident following any treatment [13].

Benzodiazepines

Benzodiazepines are GABA-agonists which act on the thalamus and hypothalamus, inducing calm behavior in many species. Muscle relaxation can be significant and result in transient ataxia. Benzodiazepines are also used as anticonvulsants.

Diazepam

Diazepam has not been evaluated for sedation in camelids. Diazepam was administered to treat seizures due to suspected tolazoline toxicosis at a dosage of 0.07 mg/kg IV [14].

Diazepam has also been investigated in studies evaluating combination protocols for anesthetic induction. In these studies, diazepam was administered at dosages of 0.1 or 0.2 mg/kg in combination with ketamine [15, 16].

Diazepam may be chemically incompatible with other immobilizing agents and it should not be mixed with them in the same syringe nor in IV solutions.

Midazolam

Midazolam is a short-acting benzodiazepine central nervous system (CNS) depressant, similar to diazepam, but more potent. IV or IM administration of midazolam is associated with variable sedation. Profound sedation progressing to lateral recumbency can occur following both IM and IV administration of midazolam at 0.5 mg/kg [17]. Cardiovascular and respiratory effects are minimal, and result in increases in heart rate and respiratory rate if any change is noted. Peak sedation occurs approximately 15 minutes after IM administration, and less than 1 minute after IV administration. Duration of sedation following midazolam administration may be as long as approximately 90 minutes [17].

Field Anesthetic Techniques

Sedation, Tranquilization and Chemical Immobilization

Many procedures can be carried out on nervous or apprehensive camelids by dulling the sensorium with tranquilizers or low doses of injectable anesthetic agents. In some instances, the animal remains standing whereas, at other times, recumbency may be necessary. Chemical immobilizing agents are routinely used to render wild, dangerous zoo animals immobile. Immobilization of obstreperous or vicious male camelids may be necessary to carry out even minor procedures as hoof trimming. Chemical immobilization of vicuñas or untrained guanacos may be preferable to physical restraint.

Individual drugs are described below, followed by a discussion of applications, either individually or in combination.

Butorphanol Tartrate

Butorphanol is a kappa-agonist, mu-antagonist opioid analgesic that may also result in sedation or can augment the sedative effects of other drugs. The analgesic effects of butorphanol are discussed later in this chapter. Butorphanol has been frequently administered IV, IM, or subcutaneously in dosages up to 0.1 mg/kg [13, 18].

Butorphanol has been used extensively in camelid clinical practice, either alone or in combination with other sedatives, to calm an excitable animal for examination or minor surgical procedures, though combination with acepromazine resulted in no significant sedation at a dosage of 0.1 mg/kg IM [13].

Ketamine Hydrochloride

Ketamine is a dissociative anesthetic agent. The animal usually retains normal pharyngeal-laryngeal reflexes. This desirable effect minimizes accidental aspiration of food or ingesta. However, regurgitation has occurred following ketamine use in camelids. It is difficult to say whether this was an effect of the drug or simply passive regurgitation from immobilization.

Ketamine produces an increased RR with a decrease in the tidal volume, but if ketamine is given IV at a too-rapid rate, apnea may be produced.

Animals experience transitory pain upon IM injection of the solution. Because ketamine crosses the placenta, anesthetic effects are noted in the fetus when ketamine is used as a sedative for cesarean section or dystocia. Ketamine is not known to produce abortion.

Ketamine produces a state of catalepsy with a fixed expression in the eyes, and eye reflexes are typically maintained. The eyelids stay open, yet the cornea usually remains moist. Occasionally, corneal ulceration has resulted from prolonged exposure. Palpebral reflexes persist. Ketamine is metabolized in the liver, and metabolites are excreted via the urine.

Ketamine does not produce skeletal muscle relaxation, rather a catatonia. There is profound analgesia at medium-to-high dosages, though analgesia of the visceral peritoneum may be less than optimal. Tonic-clonic convulsions are produced in some species but have not been observed in camelids except upon accidental intracarotid injections.

Tiletamine HCl and Zolazepam HCl (Telazol™)

Telazol is a combination of equal parts by weight of tiletamine and zolazepam, a benzodiazepine. The combination produces a rapid dissociative anesthesia typical of ketamine and with the relaxing action of zolazepam.

Telazol is used for chemical immobilization, anesthesia induction, sedation, and analgesia for minor surgical procedures. Indications for its use are the same as for ketamine, but less rigidity is associated with telazol administration. In practice, telazol functions similarly to a combination of midazolam and ketamine. Tiletamine-zolazepam, combined with other drugs, is commonly used for field anesthesia (see below).

When reconstituted per label, the 5-mL vial contains 50 mg/mL of tiletamine and 50 mg/mL of zolazepam. The administered dose is calculated from the combined (100 mg/mL) activity. Free-ranging adult male guanacos were immobilized with 4–6 mg/kg dosages [19]. The first signs of the drug's effect is incoordination, beginning at 2.9 minutes, with recumbency occurring at a mean time of 7 minutes (1.5–53 minutes). Immobilization lasts for 15–15.6 minutes. Characteristically, the duration of the recovery period is proportional to the dose administered.

In guanacos, there is some muscle twitching and rigidity at lower doses. Hypersalivation was common and pronounced in some individuals. Chewing movements, retching, and vocalization have been observed. Zolazepam effects may be countered with flumazenil.

Muscle Relaxants

Guaifenesin

Guaifenesin is a muscle-relaxing agent that is usually combined with ketamine and/or xylazine to provide analgesia and/or anesthesia. It has been found to be safe and effective in llamas. The dosage currently recommended in the llama is 1–2 mL/kg of a 5% solution [3]. The same dosage is used to induce general anesthesia when combined with ketamine such that the solution is 5% guaifenesin with 1 mg/mL of ketamine.

An IV catheter should be placed in the jugular vein before attempting to use guaifenesin due to the volume of drug required.

Injectable Anesthesia

Numerous injectable anesthetic agents or combinations of agents have been used in camelids. Refer to the pharmacodynamic discussions of the agents for details. In this section, applications for use in camelids are described.

Xylazine-ketamine

In one study, 0.8 mg/kg of xylazine combined with 8 mg/kg of ketamine IM was compared with 1.2 mg/kg of xylazine combined with 12 mg/kg of ketamine IM [20]. The higher dosages of xylazine and ketamine were effective for inducing sufficient restraint in most alpacas for approximately 30 minutes, with significant respiratory depression. The lower doses of xylazine and ketamine resulted in 30 minutes of sufficient restraint in half of the alpacas without significant respiratory depression [20]. Xylazine (0.25 mg/kg) and ketamine (5.5 mg/kg) IM was administered to dromedary camels and resulted in better analgesia, better muscle relaxation, fewer cardiorespiratory effects, and shorter recovery times compared to ketamine alone [21].

Acepromazine-Butorphanol-Tiletamine-Zolazepam

Administration of acepromazine 0.05 mg/kg and butorphanol 0.1 mg/kg IM, followed 20 minutes later by tiletamine-zolazepam at a dosage of 2 mg/kg IM resulted in a duration of anti-nociception of 41 $\pm$ 6 minutes with a smooth recovery quality and no statistically or clinically significant changes in cardiorespiratory parameters [13].

Dexmedetomidine-Tiletamine-Zolazepam

Administration of 10, 15, and 20 mcg/kg of dexmedetomidine IM, in combination with tiletamine-zolazepam at a dosage of 2 mg/kg IM, resulted in decreases in heart rate and respiratory rate, oxygen saturation, and arterial partial pressures of oxygen. Anesthetic duration was prolonged with increased dosages of dexmedetomidine. Recovery was smooth and occurred between 63 and 81 minutes, with longer recoveries occurring with higher dexmedetomidine dosages [11].

Xylazine-Tiletamine-Zolazepam

Xylazine 0.1, 0.2, or 0.4 mg/kg IM was investigated with 2 mg/kg tiletamine-zolazepam IM for anesthesia in llamas. Inclusion of 0.1 mg/kg of xylazine did not result in prolongation of anesthetic duration or decrease time of onset of sternal or lateral recumbency. Inclusion of 0.2 or 0.4 mg/kg of xylazine IM resulted in faster sternal and lateral recumbency and prolongation of duration of anesthesia in a dose-dependent manner. Respiratory depression and hypoventilation occurred in all llamas but was not clinically significant. Hypoxemia occurred, the severity of which was dose-dependent [22].

Propofol

Propofol is a GABA agonist anesthetic induction drug commonly used in small animal practice. Propofol administration is associated with an immediate onset and short duration. The use of propofol for anesthetic induction in camelids is often limited by volume and cost of administration. Propofol commonly causes respiratory depression leading to apnea following bolus administration and should not be used in animals that are not endotracheally intubated. Another common side effect of propofol administration is vasodilation leading to hypotension. Propofol has been investigated for use in camelids for anesthetic induction and maintenance. When used for anesthetic induction, a dosage of 2–3.3 mg/kg has been sufficient to achieve endotracheal (ET) intubation [16]. A dosage of 0.4 mg/kg/min was sufficient to maintain a stable surgical plane of anesthesia but resulted in significantly increased arterial partial pressures of carbon dioxide and decreases in arterial blood pressure [23].

Alfaxalone

Alfaxalone is a neuroactive steroid that also functions as a GABA agonist anesthetic induction drug. Alfaxalone administration results in similar effects to propofol in that it is associated with an immediate onset and short duration and, at higher doses, can cause respiratory depression and hypotension. The use of alfaxalone for anesthetic induction in camelids may be cost prohibitive. Alfaxalone induction was classified as excellent following administration of 2.1 mg/kg IV, which was also sufficient to achieve ET. Alfaxalone administration did result in hypoventilation, and recovery quality was poor [16].

Inhalant Anesthetic Techniques

Anesthetic Induction and Intubation

Premedication is advisable prior to anesthetic induction to reduce the dosage of drugs needed to produce recumbency and achieve ET intubation. The selection of the premedications may depend on the drugs available, the tameness of the camelid, the restraint facilities available, and the experience of the anesthetist.

Placement of an IV catheter is advisable for administration of drugs in the event of a light surgical plane of anesthesia and also the administration of resuscitative drugs in the event of an emergency.

To provide positive support for respiration and to avoid complications if the camelid should regurgitate, tracheal intubation is recommended whenever general anesthesia is selected, especially using inhalant anesthetics.

ET intubation is difficult in South American camelids because the restricted space in the oropharynx impairs visualization of the glottis with a laryngoscope while the endotracheal tube is manipulated into position. The restricted space is the result of a combination of factors, including the narrow space between the rami of the mandible, inability to open the mouth widely, the elevated mound on the dorsum of the caudal aspect of the tongue, and the elongated soft palate that may be situated either ventral or dorsal to the epiglottis (Figures 6.1 and 6.2).

Tubes designed for small animals are too short for use in llamas. Tubes designed for cattle and horses are too long. In adult llamas, the tube should be 50 cm long, with a diameter appropriate to the size or weight of the animal (Table 6.1). Use appropriately sized equine endotracheal tubes in camels.

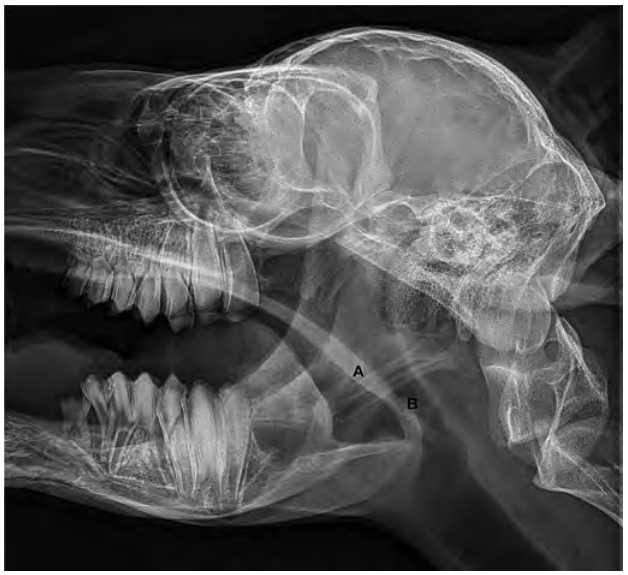

Figure 6.1 Lateral radiograph of the oropharyngeal region of a llama. The soft palate is ventral to the epiglottis. (A) Soft palate, (B) epiglottis.

Figure 6.2 Lateral radiograph of the oropharyngeal region of a llama with the soft palate dorsal to the epiglottis. (A) Soft palate, (B) epiglottis.

Table 6.1 Endotracheal tubes for camelids.

Body weight (kg/lb)	Inside diameter (mm)	Outside diameter (mm)	Length (cm)	French (size)
9–60/20–132	3	4.7	30	14
	4	6	30	16
	5	7	35	22
	6	8.7	35	26
65–160/143–350	7	10	50	30
	8	11	50	33
	9	12.3	50	37
	10	14.7	50	44
	11	15.3	50	46
	12	17	50	51
160+/350+	14	19	50	57
	16	21	50	63
Adult camel 700–1400/1543–3086	18	23.6	50	71
	20	31.8	50	76
	26	34.1	50	105

Laryngeal stimulation results in reflex closure of the glottis which, in turn, causes high negative intrathoracic pressure during inspiration. This induces passage of ingesta into the esophagus. It is essential that the laryngeal reflex be abolished before attempting ET intubation.

Prior to administration of anesthetic induction drugs, the patient should be positioned near the site for the surgery. Following induction, the animal should become recumbent.

Figure 6.3 Positioning a llama for endotracheal intubation following chemical immobilization.

An assistant should maintain the animal in a sternal position by straddling it at the withers and should extend the head by placing a finger on either side of the rostral maxilla. The anesthetist can extend the tongue and hold the mouth open to facilitate intubation (Figures 6.3–6.5).

For most camelids, placement of a long-bladed laryngoscope at the base of the tongue allows the anesthetist to visualize the glottis (Figure 6.6 and 6.7). If the soft palate is below the epiglottis, it can be released using either the laryngoscope blade or the stylet. At this point, the arytenoid cartilages may be sprayed with 2 mL of 2% lidocaine. An aluminum rod stylet or stiff polyethylene catheter should be placed in the ET tube to keep the tube rigid, and the stylet is inserted into the larynx (Figure 6.8). The ET tube is threaded over the stylet and into the larynx (Figure 6.9). The tube may be inserted through the laryngeal opening at the time of maximum opening. The stylet should be removed and a check made for air flow through the tube. The cuff is then inflated.

Some difficulty may be encountered in threading the ET tube through the laryngeal opening. If anesthesia is too light, there will be reflex closure of the laryngeal opening as the tube touches the epiglottic or arytenoid cartilage. The ET tube should be gently pressed against the larynx. The head may be repositioned slightly, or it may be helpful to fix the larynx from the exterior. If these adjustments fail, a slightly smaller tube should be tried (Table 6.1).

A check should be made for air movement through the ET tube before the catheter is removed. The cuff should be inflated and the tube tied or taped to the lower jaw or around the back of the head.

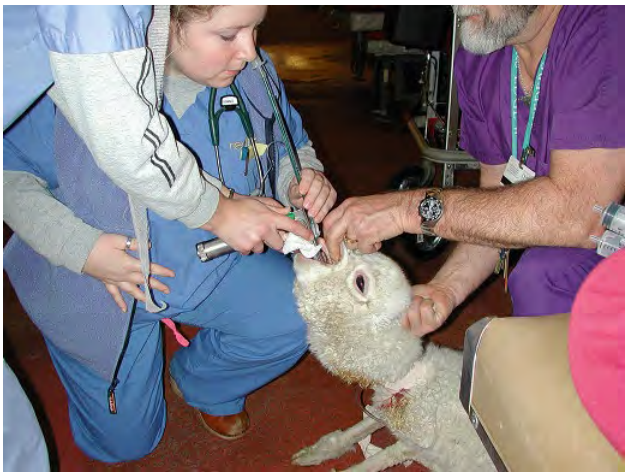

Figure 6.4 Positioning an alpaca for endotracheal intubation following chemical immobilization.

Following intubation, camelids should be placed in position for surgery (Figure 6.10). Care should be taken to ensure that appropriate padding is used. A head-down position may be helpful to encourage drainage of any regurgitated material. Be sure that eyes are appropriately moistened and not subjected to any regurgitated material.

Camel Endotracheal Intubation

Camel anesthesia is essentially the same as that for South American camelids. However, the oropharyngeal cavity is much larger and the mouth will open widely. However, the

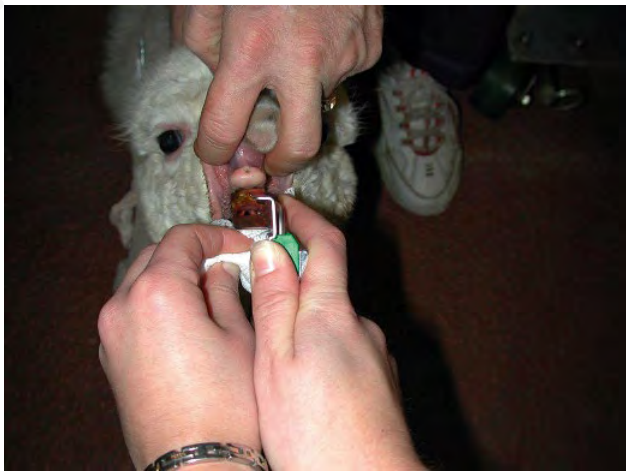

Figure 6.5 Visualization of the larynx in an alpaca for endotracheal intubation following chemical immobilization.

Figure 6.6 Long-bladed laryngoscope used for endotracheal intubation of camelids.

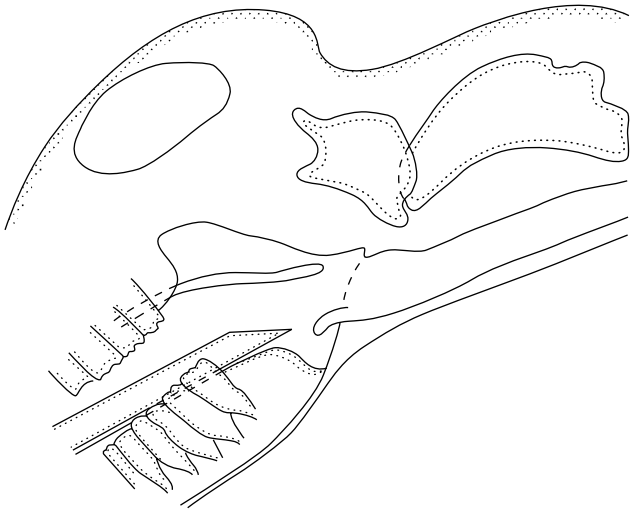

Figure 6.7 Diagram of a method of tracheal intubation. The laryngoscope blade is used to depress the tongue, providing visualization of the glottis.

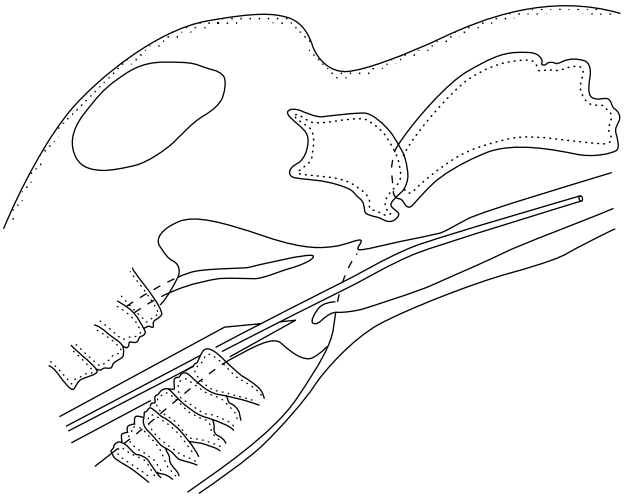

Figure 6.8 Diagram of a method of tracheal intubation. A plastic catheter is inserted into the trachea.

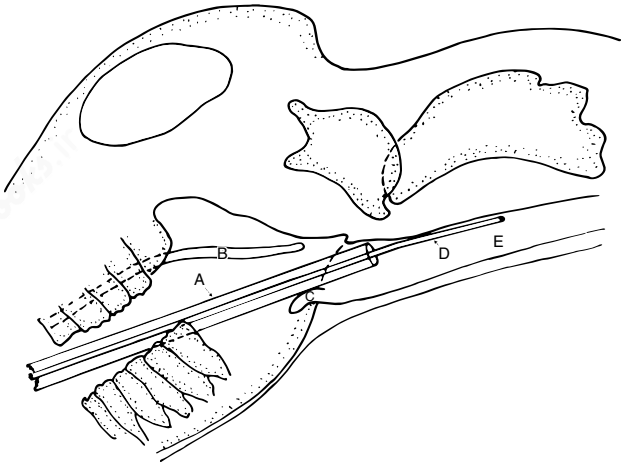

Figure 6.9 Threading the endotracheal tube over the catheter.

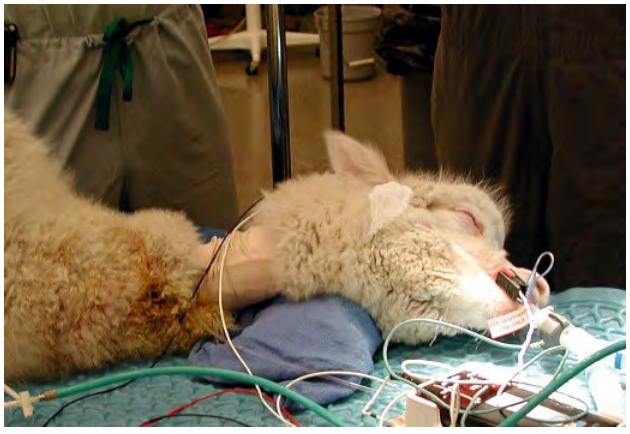

Figure 6.10 Inhalation anesthesia in an alpaca.

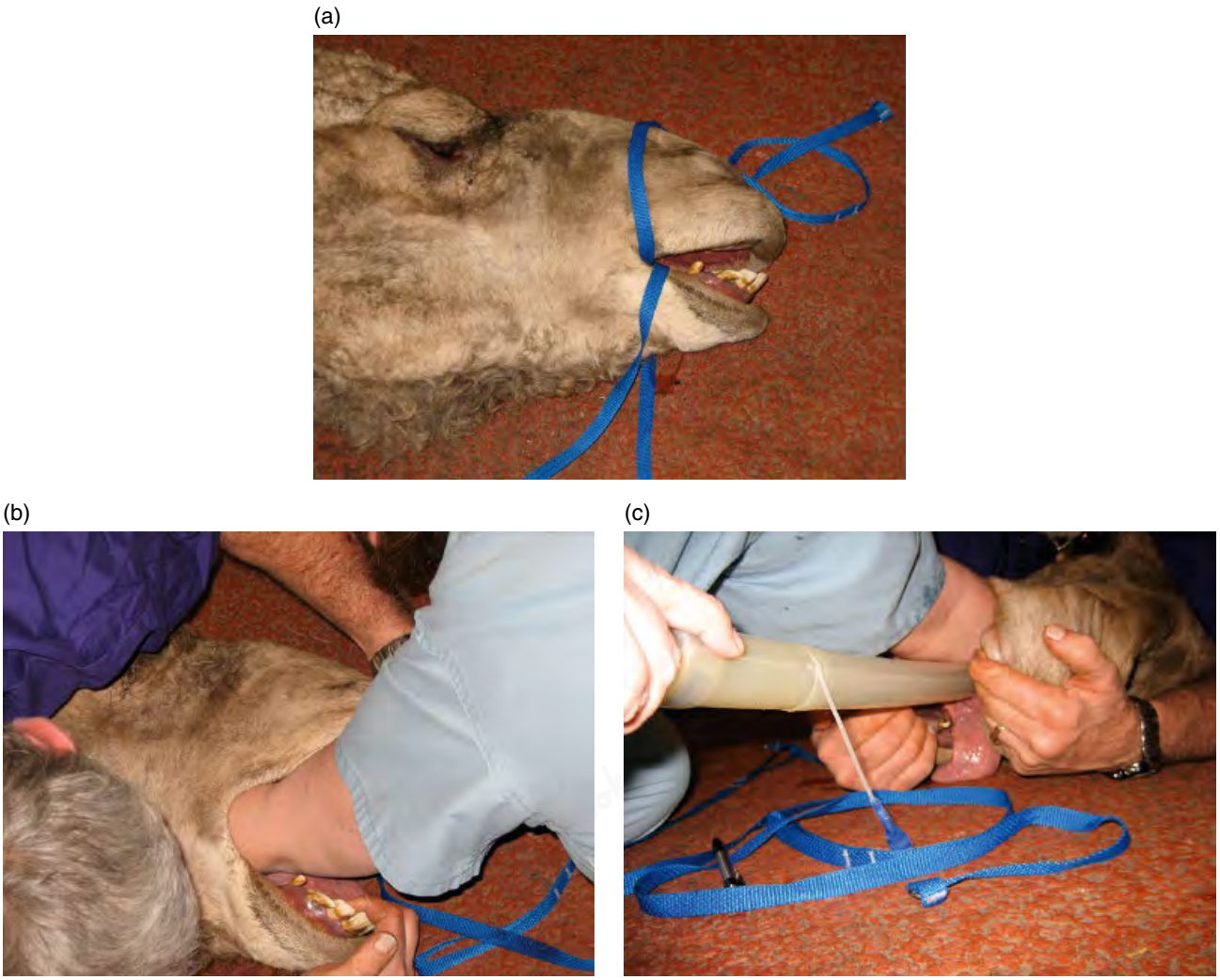

Figure 6.11 Intubation of a camel. Photo A – camel in lateral recumbency following anesthetic induction. Photo B – digital palpation of the epiglottis and arytenoids. Photo C – Passing endotracheal tube through the arytenoids. *Source:* Photos courtesy of Dr. Phillip Lerche.

male dromedary has a diverticulum of the ventral aspect of the soft palate called the dulaa (also called the dulah, doolah, or doola) that may protrude from the mouth during the process of intubation. This may startle the novice camel anesthetist but should merely be replaced or pushed aside to complete intubation. Camel intubation can be achieved using visualization (similar to llamas and alpacas) or through digital palpation of the epiglottis and arytenoids (Figure 6.11).

Maintenance of Anesthesia Using Inhalant Anesthetics

Inhalation anesthesia allows the anesthetist to control the depth of anesthesia, provide respiratory support, and reduce the risk of toxicity to the patient. The disadvantages are the cost of the agent, the expense of specialized equipment for administration, and the requirement for an anesthetist.

Agents

Isoflurane, sevoflurane, and desflurane have all been investigated for use in camelids. Isoflurane is the most commonly used inhalant anesthetic due to its ubiquity and low cost. The minimum alveolar concentration (MAC) of isoflurane in adult llamas has been determined as 1.05 ± 0.17% [24]. The MAC of isoflurane has not been determined in adult alpacas but is likely similar to that of llamas. The MAC of sevoflurane is 2.29 ± 0.14% in llamas and 2.33 ± 0.09% in alpacas [25]. The MAC of desflurane is 7.99 ± 0.58% in llamas and 7.83 ± 0.51% in alpacas [26]. The most notable side effect of inhalant anesthetics is vasodilation leading to hypotension. In addition, respiratory depression is common in patients maintained with inhalant anesthetics.

Physiology

The tidal volume of an adult llama is 10–15 mL/kg, similar to other species. Oxygen flow rates of at least 3 L/minute are recommended for induction. In a controlled ventilation system, this can be reduced to 10–12 mL/kg/minute for maintenance.

Due to the vasodilatory effects of inhalants, arterial blood pressure should be monitored in camelids anesthetized with inhalants. Intra-arterial catheters may be placed in an auricular or saphenous artery. Non-invasive blood pressure measurements using oscillometric monitors have been demonstrated to be inaccurate in camelid species and their use for determining trends in blood pressure measurements is also questionable [27].

Recovery time from inhalant anesthesia varies with the physiologic state of the patient, drugs used for premedication and induction, and the maintenance concentration of inhalant. Recovery times are longer in debilitated animals compared to healthy patients. In general, a llama should be able to remain in sternal recumbency within 60 minutes following cessation of administration of the agent and be able to stand within 90 minutes but may be depressed for several hours.

Monitoring and Supportive Therapy during Anesthesia

Heart rate, respiratory rate, and body temperature should be assessed periodically during anesthesia. Direct pulse palpation can be easily performed to determine a heart rate in anesthetized camelids. The auricular or saphenous arteries are typically used for pulse palpation or arterial catheterization. The saphenous artery lies on the medial aspect of the stifle, but the caudal articular and digital arteries may also be palpated.

Respiratory rate is often monitored by evaluation of chest excursion or movement of the rebreathing bag. Monitoring of end-tidal carbon dioxide tensions can be helpful and respiratory depression can be used as an indicator of excessive anesthetic depth as healthy camelids tend to maintain a normal end-tidal carbon dioxide tension at stable surgical planes of anesthesia.

Ocular reflexes are used to assess depth of anesthesia. The ventral eyelid palpebral reflex is suppressed when anesthesia is adequate, but the upper lid will likely retain the response to tactile stimulation. Nystagmus rarely occurs. The administration of ketamine may complicate assessment of ocular reflexes until the inhalant anesthetic agent takes effect.

If available, a pulse oximeter attached to the tongue is excellent for monitoring the oxygen status of the patient. An arterial catheter is desirable if facilities for arterial pressure assessment or blood gas analysis are available.

The head should be positioned to avoid overextension in order that saliva and stomach contents may be appropriately drained. Artificial tears should be instilled into the conjunctival sac and measures taken to protect the prominent eye from positional trauma.

An IV catheter should be placed and appropriate fluids administered during the procedure.

Hypothermia may be treated by the application of active warming such as warm water circulating blankets and forced air blankets.

If an injectable anesthetic is used, body and head positions are even more important. If a procedure is prolonged, the patient will benefit from supplemental oxygen, administered by placing a small tube up a nostril at a flow rate of 1 L/minute.

Recovery

Oxygenation is poorest when the patient is in dorsal recumbency, but hypoxemia is also common during the recovery stage, so administration of supplemental oxygen may be necessary following resumption of sternal recumbency. The animal should be recovered in sternal recumbency to encourage equal lung inflation and avoid regurgitation in the recovery period. The head is typically held up and nose pointed down to allow any fluid in its mouth to drain out until the patient can maintain this position on its own. In addition, camelids may be prone to edema of the nasal mucosa which can result in obstruction to breathing following extubation. Placement of the head above the heart will help to pre-emptively resolve any nasal edema and nasal pharyngeal tubes may also be placed until nasal edema is resolved (Figure 6.12).

Figure 6.12 Recovery of a llama from inhalant anesthesia. Note the nasal pharyngeal tubes inserted due to the intraoperative development of edema.

Removal of the ET tube should be performed following the return of the laryngeal reflex (patients will usually swallow when this has occurred). Care should be taken to ensure that any regurgitated material is not allowed to travel caudally once the ET tube cuff is deflated. If there is concern, the tube may be extracted while the cuff remains inflated as there is minimal risk of laryngeal trauma with this technique in camelids. It is also important to ensure that the ET tube does not remain in place after the animal begins to chew as camelids can bite the tube in two pieces and inhale the caudal piece back into the trachea, resulting in the need to re-anesthetize the patient to extract the tube.

Anesthetic Complications

Camelids are predisposed to developing nasal edema more than ruminants. Because camelids are semi-obligate nasal breathers, they cannot breathe efficiently through their mouth and nasal edema can cause severe respiratory distress. Placement of small tubes within the nose to allow passage of air through the stenotic opening is an effective treatment for nasal edema. Alpha-adrenergic agonists such as phenylephrine can be sprayed on the mucous membranes of the nose to cause vasoconstriction of the blood vessels and decrease swelling. A tracheostomy is a last resort effort to alleviate respiratory distress from an upper airway obstruction.

Camelids may produce copious amounts of saliva, and they are prone to regurgitation, which makes them at great risk for aspiration. To reduce regurgitation, animals should be fasted for 12–24 hours prior to anesthesia.

Field sedation and anesthetic techniques are typically not associated with hypotension in healthy camelid patients. Use of inhalant anesthetics and propofol may increase the likelihood of developing hypotension. Routine monitoring of blood pressure while in the field is rare. When necessary, blood pressure monitoring should be performed utilizing an invasive method (placement of an arterial catheter and monitoring utilizing a pressure transducer) as non-invasive (oscillometric) blood pressure monitoring has been demonstrated to be inaccurate in anesthetized camelids [27].

As with ruminants, fermentation occurs in the first stomach compartment. Gas production is a byproduct of this fermentation, and if an animal does not have the ability to eructate, bloat will result. Animals have a decreased ability to eructate when placed on their back or side. Recovery in sternal recumbency (cushed position) will encourage eructation. In severe cases, bloat will cause ventilation impairment if the C1 compartment or rumen pushes up on the diaphragm. A stomach tube can be passed to relieve this pressure.

Hypothermia is a complication seen mostly in young animals that undergo long duration of general anesthesia. Surgeries that involve an open body cavity are also prone to this complication. Cold lavage fluids can quickly reduce a patient's core body temperature. Hypothermia causes a decrease in a patient's metabolic rate and will prolong recovery times.

Analgesia

Opioids

Fentanyl Patches

Fentanyl patches are available in various sizes and have been associated with minimal side effects in healthy llamas. Pharmacodynamic studies have not been published on the use of fentanyl patches to provide analgesia in camelid patients. Placement of fentanyl patches on the antebrachium may be one option for provision of analgesia. Similar to other species, fentanyl patches do not provide analgesia for the initial 12 hours after patch placement. Therefore, patches should be placed a minimum of 12 hours prior to surgery or other analgesic should be provided for the first 12 hours if patches are placed at the time of surgery. Fentanyl patches have not been associated with sedation in healthy llamas.

Fentanyl patches are available in 25, 50, 75, and 100 mcg/hours sizes. Four 75 mcg/hour patches placed on llamas (approximately 150 kg body weight) provided consistent serum concentrations and no sedation [28].

A recent study on fentanyl patches in alpacas found consistent plasma concentrations of fentanyl when patches were placed at a dose of 2 mcg/kg/hr. Plasma concentrations peaked 29 hours after fentanyl patch placement and absorption occurred at a rate of 50 mcg/hour between 8 and 72 hours after patch placement [29].

A topical formulation of fentanyl, approved for use in dogs, has also been investigated. Administration of this product at the labeled dose in dogs is not recommended. A safe and effective dose of this product has not been determined.

Butorphanol

Systemic butorphanol administration has been associated with analgesia and sedation in llamas. Butorphanol (0.1 mg/kg) is safe and can be effective for somatic pain. The half-life of butorphanol in llamas following IV administration is approximately 15 minutes and is approximately 1 hour when administered IM [18]. An additional study determined that 0.1 mg/kg of butorphanol IV caused no significant changes in cardiorespiratory parameters or anesthetic depth in alpacas anesthetized with isoflurane [30].

Morphine

Systemic administration of morphine has been associated with sedation and analgesia in llamas. Level of sedation with morphine is directly correlated with dose, but lasted for less than 2 hours, regardless of dose. Analgesia was highly variable [31]. Suggested doses of morphine are 0.05–0.2 mg/kg IM or IV.

Buprenorphine

The pharmacokinetics of systemic administration of buprenorphine has been investigated in alpacas [32, 33]. In one study, a dosage of 0.005 mg/kg was administered IV or IM. The half-life of buprenorphine following IV administration was approximately 2 hours, and the half-life following IM administration was 3.3 hours [32].

A more recent study determined that administration of 0.02 mg/kg of buprenorphine IV was associated with plasma concentrations above 0.7 ng/mL (plasma concentration associated with analgesia) for 1.3 hours [33]. Mean elimination half-life of buprenorphine after IV administration was one hour. Plasma concentrations of buprenorphine after SC administration were above 0.7 ng/mL for 1.3 hours. Elimination half-life after SC administration was 2.7 hours. Administration of a sustained-release formulation of buprenorphine resulted in plasma levels of buprenorphine inconsistent with analgesia [33].

NSAIDS

Non-steroidal anti-inflammatory drugs (NSAIDs) are not as potent analgesics as opioids but typically are longer acting than opioids. NSAIDs have peripheral anti-inflammatory effects and centrally mediated analgesic effects. NSAIDs are very effective for treating pain when utilized with other analgesic modalities. Due to their effects on GI prostaglandins, NSAIDs may cause GI ulceration and should be used cautiously. Many sources recommend administration for a maximum of three days.

Meloxicam

Meloxicam has been evaluated for use in llamas. Oral administration of 1 mg/kg of meloxicam was associated with plasma concentrations in the therapeutic range (as determined for other species) for up to three days post administration. Minimal side effects were observed with this dosage regimen [34].

Flunixin Meglumine

Flunixin meglumine is likely the most used NSAID in camelids. The pharmacokinetics of 2.2 mg/kg of flunixin meglumine IV in llamas and alpacas demonstrated a short half-life, low clearance, and low volume of distribution [35, 36]. While the therapeutic window is unknown, a loading dosage of 2 mg/kg IV, followed by 1.1 mg/kg IV q24 hours is commonly used in practice. Flunixin provides excellent visceral analgesia but may have less potent effects for musculoskeletal pain. Flunixin is a nonsteroidal anti-inflammatory agent that has analgesic and antipyretic effects. It has proven to be an effective analgesic for colic pain.

Flunixin has been used extensively in camelids for analgesia in musculoskeletal disorders. It seems to be more effective than phenylbutazone in the relief of GI spasms. However, automatic administration of flunixin early in an episode of colic may mask clinical signs, delaying diagnosis and institution of appropriate therapy.

A transdermal formulation of flunixin meglumine has recently been approved for use in cattle and has been investigated for use in alpacas at a dosage of 3.3 mg/kg transdermal, which resulted in poor bioavailability and poor suppression of prostaglandin E_2; the authors did not recommend its use in alpacas [36].

Phenylbutazone

Phenylbutazone has been used extensively in a variety of species, including humans, to treat musculoskeletal disorders and reduce soft-tissue inflammation. It also has an antipyretic effect. Phenylbutazone seems to provide analgesia for musculoskeletal pain but may be less effective for treating visceral pain. Oral and IV administration of phenylbutazone has been investigated in llamas [37]. A dosage of 5 mg/kg of phenylbutazone IV resulted in a high clearance and short half-life, with a plasma concentration consistent with a therapeutic benefit occurring for one hour after IV administration. The oral administration of phenylbutazone in llamas demonstrated a variable absorption and bioavailability [37].

Phenylbutazone is also available in an injectable formulation, however injectable phenylbutazone must be administered IV, and perivascular injections of phenylbutazone may result in phlebitis and adjacent skin slough. Phenylbutazone has been reported to be ulcerogenic in the equine, however, there are no substantiated reports of ulcers in llamas following its use.

Local Anesthesia

In camelids, infiltration of a surgical site with a local anesthetic agent is a routine practice for lancing abscesses, suturing lacerations, laparoscopy, abdominocentesis, and thoracocentesis. Local anesthesia may also be used in combination with mild sedation for such surgical procedures as castration or a standing exploratory laparotomy.

Regional nerve blocks are less often selected in South American camelids because the anatomical course of the nerves is not well documented but has been shown to have wide variation from other livestock species. For instance, the major nerve supply to the foot is located only on the medial aspect of the metacarpus and metatarsus rather than bilaterally, as in the equine and bovine.

Any of the standard local anesthetic agents are suitable for use in camelids. Selection of anesthetic agent depends on many factors: rapidity of onset of the anesthetic agent, duration of anesthesia required, or the experience of the operator. Any local anesthetic agent may be toxic if the dose is excessive.

Intratesticular and Incisional Line Blocks for Castration

Use of intratesticular and incisional line blocks for castration in alpacas may be helpful to reduce the required dose of anesthetic drugs, diminishing or eliminating negative cardiorespiratory side effects during field anesthesia.

In alpacas anesthetized with ketamine (4 mg/kg), xylazine (0.4 mg/kg), and butorphanol (0.04 mg/kg) IM, use of intratesticular and incisional line blocks with either bupivacaine 0.75% or lidocaine 2% (2 mL in each testicle and 2 mL in subcutaneously along the line of incision) resulted in either fewer responses to surgical stimulation or improved cardiorespiratory parameters [38].

Epidural Anesthesia

Epidural anesthesia is indicated for any type of perineal surgery (suturing lacerations, penetration of a persistent hymen), replacement of a prolapsed rectum or vagina, and prevention of excessive straining during rectal palpation or obstetrical manipulation. In older camelids, a caudal epidural can be performed in order to provide anesthesia or analgesia to the hind limbs. In crias and young adult camelids, the lumbosacral space can easily be palpated and the technique performed similarly to a lumbosacral epidural in a dog.

Following surgical preparation of the site, the tail should be grasped with one hand and rocked up and down to locate the sacrococcygeal intervertebral space. The five sacral vertebrae (S1 to S5) are fused in most individuals, so the first moveable intervertebral space would be between S5 and coccygeal 1 (C1) (see Figure 4.40 in Chapter 4). In a few individuals, S5 is free and moveable. The space available between S4 and S5 for penetration into the neural canal is minimal. If difficulty is encountered when attempting an epidural, move caudally to the second moveable space to facilitate the procedure [39, 40].

A 1.5-in., 20-gauge needle should be inserted perpendicularly or slightly cranial to the slope of the tailhead, directly on the midline. The depth of the spinal canal is 1.5–2 cm in a 130-kg llama and deeper for camels. If the needle is positioned properly, the fluid should flow and drain from the hub of the needle when the syringe is detached, and a drop of the anesthetic agent is put on the hub. The needle should be left in position until motor control of the tail is lost, in approximately one to two minutes.

Solutions containing preservatives or epinephrine should not be used for epidural or spinal anesthesia.

Morphine

Use of morphine for epidural analgesia is associated with few systemic side effects and prolonged analgesia (up to nine hours). The dose of morphine is 0.1 mg/kg which can be diluted with saline to achieve a total epidural volume of 0.2 mL/kg.

Lidocaine

Caudal epidurals in adult alpacas using lidocaine typically result in a fast onset of analgesia (approximately three minutes) and a duration of approximately one hour. The dose used for caudal epidurals in adult llamas is 0.22 mg/kg using a 2% solution [40].

Similar results in adult alpacas occurred following administration of 1.5 mL of 2% lidocaine into the first coccygeal space, with onset in approximately minutes [39]. Alpacas did attempt to lie down.

Caudal epidurals in dromedary camels have utilized dosages of 0.22 mg/kg resulting in complete analgesia in the tail, anus, and perineum with no sedation for approximately 55 minutes [41].

Xylazine

Xylazine epidurals have been associated with mild sedation and up to three hours of analgesia. The dosage for a caudal epidural in an adult llama is 0.17 mg/kg using a 10% solution [40].

Xylazine and Lidocaine

Combination epidurals using xylazine and lidocaine have a fast onset (approximately three minutes) and a longer duration than either drug used alone (approximately 5 ½ hours). Xylazine 0.17 mg/kg and lidocaine 0.22 mg/kg in combination resulted in mild to moderate sedation in adult llamas [40]. In adult alpacas, administration of lidocaine as a 1 : 1 ratio of 20 mg/mL xylazine in a single 0.75 mL dose into the first coccygeal space resulted sedation and some analgesia, but no recumbency [39].

Ketamine

Ketamine epidurals have been evaluated in dromedary camels at dosages of 1 and 2 mg/kg. While 1 mg/kg of ketamine resulted in anti-nociception at the tail and mild to moderate sedation, administration of 2 mg/kg resulted in anti-nociception in the tail, anus, and perineum as well as deep sedation. Ataxia was noted at both dosages, with recumbency resulting from the 2 mg/kg dosage [42].

Ketamine and Lidocaine

A combination of ketamine (1 mg/kg) and lidocaine (0.22 mg/kg) for caudal epidurals in dromedary camels resulted in ataxia and complete analgesia of the tail, anus, and perineum for approximately 75 minutes [41]. Mild to moderate sedation occurred.

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7

Parasitology

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The diagnosis, treatment, and management of parasitic diseases in camelids can present significant challenges. Old and New World camelids share many of the same external and internal parasites that affect other livestock species, with some exceptions. In addition to the parasites themselves, emerging resistance to anthelmintics and the creation of integrated parasite management programs will be discussed.

External Parasites

See Table 7.1

Lice

Lice are wingless insects that live a complete life cycle on a single host [1]. They are generally host specific [2]. Both biting/chewing (*Bovicola* [*Damalinia*] *breviceps*) and sucking (*Microthoracius* spp.) lice have been identified on camelids [3, 4]. Of the *Microthoracius* spp., *Microthoracius mazzai, Microthoracius praelongiceps, and Microthoracius cameli* (Figure 7.1) are most commonly observed [5, 6].

Identification

Biting lice vary in size from 0.5 × 1.2 to 1.5 × 4 mm with sucking lice being two-thirds this size. They may be white or light tan in color. Lice can often be observed macroscopically but a hand lens (10×) can assist the observer. *Bovicola* spp. move when disrupted by parting of the fiber and are found near the surface of the skin or within the fibers compared to sucking lice that are more sedentary and partially embedded within the skin taking a meal of blood or lymph, which makes them more difficult to see. Biting lice have a blunted head that can usually be seen without magnification, but precise identification requires microscopic evaluation. Sucking lice have elongated mouth parts that are often too small for macroscopic identification [4].

Life Cycle

The life cycle of lice is simple (Figure 7.2). Adult lice copulate and the female deposits fertilized eggs (nits) on fibers and cements them in place. The eggs hatch within one to three weeks and tiny replicas of adult lice emerge. As the louse matures, it undergoes two or three molts but, in each case, it is growth not a change of body structure. The maturation phase lasts one to two weeks. The entire life cycle may be completed in as little as two to five weeks. Adults live for 15–40 days.

Epidemiology

Lice may complete the life cycle on a single animal. There is no free-living stage. Transmission from one camelid to another is by close body contact which may occur with maternal care of infants, during breeding, and confinement. Spread can also occur with co-mingling at shows, sales, or transport. Colder months often bring about the most clinical pediculosis cases due to close contact and longer fleece length, facilitating transmission. Transmission can also occur by the use of communal grooming equipment such as combs, brushes, and tack or by group use of blankets, scratching posts, dust-bath areas, and feed bunks.

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Table 7.1 External parasites of South American camelids [3, 80].

		Size (mm)			Anatomic location	
Scientific name	**Common name**	**Adult**	**Larva**	**Pupa**	**Adult**	**Immature**
Culex, Anopheles Aedes	Mosquitos				Neck, limb	Water
Vermipsylla spp., others	Fleas	1.5–4	<6	4 × 2	Head, neck, limbs	Soil
Tabanus spp.	Horse fly Deer fly				Head, neck, limbs	Organic debris
Musca domestica	Housefly	6.5–7.5	10–12		Around eyes, ears, nostrils	Feces, organic debris
Musca autumnalis	Face fly	Slightly larger than housefly			Around eyes, ears, nostrils	Cattle feces
Stomoxys calcitrans	Stable fly	11			Around eyes, ears, nostrils	Cattle feces
Calliphoridae (various spp.)	Blow flies	8			Free-living flies	Infected and necrotic wounds
Oestrus ovis	Sheep botfly	12	L-1 = 2 L-3 = 30		Free-living flies	Nasal cavity, sinuses
Cephenemyia spp.	Deer nasal bot fly	14	L-1 = 1–3		Free-living flies	Naso-pharynx
Otobius megnini	Spinose ear tick	8	2–3	Nymph 7–10	Around farm and ranch structures	External ear canal
Ixodidae (various spp.)	Hard ticks				Attached to body	Depends on species
Demodex spp.	Demodectic mange	0.1–0.4			Sebaceous glands and hair follicles	Same as adult
Sarcoptes scabiei	Sarcoptic mange mite	0.3–0.5			In tunnels in epidermis	Same as adult
Psoroptes bovis	Psoroptic mange mite	0.7–0.8			At periphery of scab on skin surface	Same as adult
Chorioptes ovis	Chorioptic mange mite	0.35–0.4			At periphery of scab on skin surface	Same as adult

Though the cycle may be completed in as little as two to five weeks, it may be extended many months by arrested development in the nit stage, or a few lice may lie dormant until environmental conditions become conducive to rapid development of a population, as lice have poor environmental hardiness especially in cold temperatures.

Clinical Signs

The syndromes of both types of lice are different but infestations are commonly found in heavily fleeced regions of the body. Clinical signs shared by both types include a poor luster to the fiber coat, ragged, unthrifty in appearance with matting or loss of fibers, pruritus, behavior changes such as restlessness, patchy alopecia, and excoriations with secondary infection, erythema, and scaling.

Biting Lice. The areas of the body most likely to be affected by pediculosis are around the base of the tail, dorsal midline, on the side of the neck, and along the sides of the body. To search for lice, parting of the fibers at regular intervals down to the skin, use of a bright light, and observing for movement are beneficial. The eggs (nits) may also be observed attached to the fibers.

Sucking Lice. Of the two types of lice, sucking lice are more pathogenic. In addition to the shared clinical signs noted, sucking lice may cause anemia as they feed on blood, lymph, and tissue fluid from the host. Anemic camelids are more susceptible to cold stress and secondary infections. Sucking lice prefer sites around the flanks, head, neck, and withers and may be found clinging to the fibers close to the skin or embedded in the skin taking a blood meal [6].

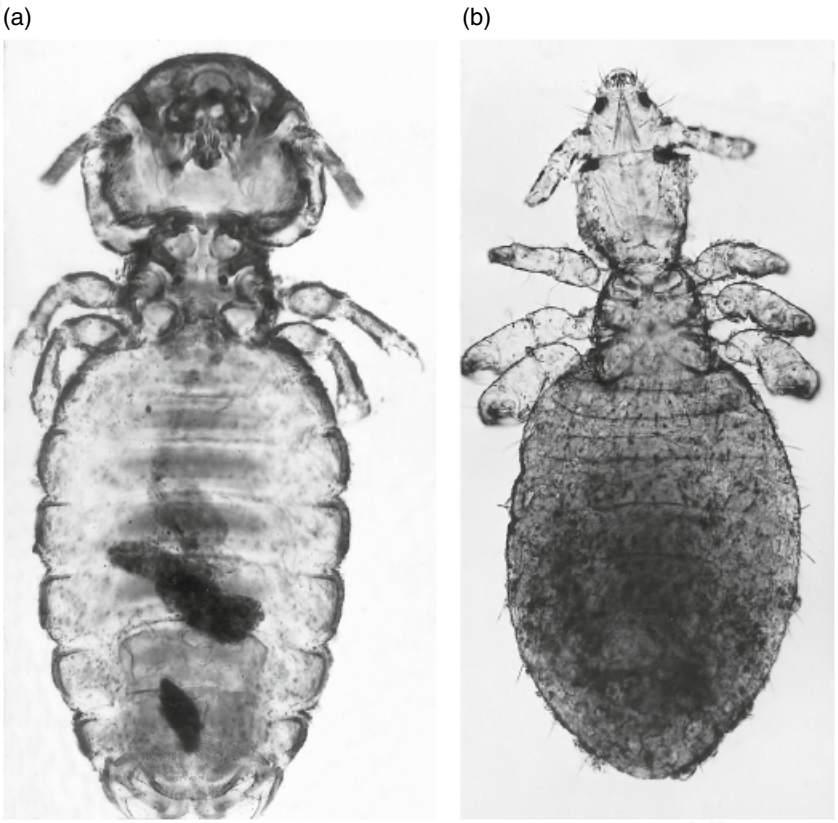

Figure 7.1 Camelid lice. (a) Chewing louse (*Bovicola breviceps*); note the round mouth parts and broad head and thorax, (b) sucking louse (*Microthoracius cameli*); note the narrow mouth parts and thorax.

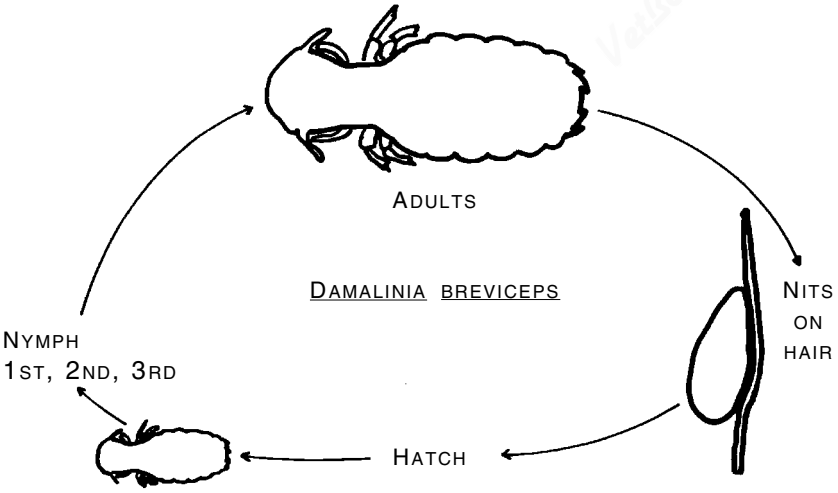

Figure 7.2 Life cycle of a louse.

Diagnosis

In addition to gross observation or use of a hand lens to identify lice within the fiber, close to the skin, or embedded within the skin, dead lice may also be observed upon examination of the feces which would support evidence the animal is chewing on its own irritated skin. Differentiation between the two types of lice can be performed using a hand lens to observe the head of the louse. Chewing/biting lice have a head width similar to the abdomen, compared to sucking lice that have a head width approximately one quarter of the abdomen.

Treatment

Lice may be destroyed by direct contact with numerous insecticides, including pyrethrins, chlorinated hydrocarbons, carbamates, and organophosphates. Though it may not be practical during certain times of the year, exposure of the skin to sunlight and fluctuating temperatures through shearing are effective for reducing the quantity of lice as well as improving contact with administered insecticides. Dusts and sprays applied superficially do not reach the depth of lice within fleece if not shorn. Drenching sprays or dips are

not always practical for temperate climates, especially because lice infestations are more pronounced in the winter.

The avermectin class of anthelmintics (ivermectin, doramectin, moxidectin, eprinomectin) are seldom effective against biting lice but have excellent activity against sucking lice at a dose of 0.2 mg/kg body weight administered subcutaneously. A combination of a topical and injectable avermectin may be required to achieve better results. It should be noted that though ivermectin has not been approved by the Federal Drug Administration (FDA) for use in llamas, it has been used extensively as a general anthelmintic in llamas and has proven to be safe. Currently, there are no reports of lice resistant to the avermectin class, but widespread reports exist of gastrointestinal (GI) trichostrongyle resistance to avermectins (see the nematode section later in this chapter). This may influence treatment recommendations.

Pyrethrin and permethrin-based topical sprays, powders, and dips, commonly used in other livestock, are proven to be safer and more effective treatments compared to cholinesterase inhibitors such as organophosphates and carbamates. Recommended dosing of topical and non-absorbable products should follow labeled direction for cattle and small ruminants. Data describing the efficacy of pour-on products in camelids is insufficient. Sprays and liquid solutions can be more focally applied to sensitive areas like the face if lesions are present.

Extinosad® Jetting Fluid (25 g/L spinosad suspension concentrate) has also been studied in a small herd of South American camelids in Australia affected by the biting louse. Following shearing, a shower dip system was prepared with a wetting agent (alcohol alkoxylate 1000 g/L) added at 1 mL/L of water followed by the addition of Extinosad (1 mL/L of water) to be applied as a total volume of approximately 8 mL/camelid. The shower dip was repeated 17 days following the initial application. Ten months following the final treatment, no adult lice, nymphs, or eggs were detected on the previously infested alpaca. Though not labeled for camelids, Extinosad, in conjunction with a wetting agent, has the potential to eradicate the biting louse [7].

The duration of treatment can be lengthy as treatments should be administered every 7–14 days to account for the larvae that are at various stages of the life cycle at the time of initial treatment. Treatment should be continued until all lice are gone and all camelids in contact with the one(s) affected should also be treated.

Fleas

Identification

Similar to lice, fleas are wingless insects with laterally compressed bodies [1]. They vary in size from 1.5 to 4 mm. Fleas are not host specific; thus, several different types may be found on camelids. No specific reports of flea infestations have appeared in the literature on South American camelids, but the genus *Vermipsylla* contains several species that infest camels [8].

Life Cycle

Adult fleas copulate and the female lays approximately 20 eggs at a time in organic debris on the host or in dust/dirt in the environment. Larvae hatch in 2–16 days and feed on dried blood, feces, or other organic matter. The larval stage is completed in 7–10 days, and the insect enters the pupal stage, which may last for 10–17 days or remain dormant for months. The adult emerges from the pupa to seek a suitable host.

Epidemiology

Fleas may be transmitted from one camelid to another by close body contact similar to lice. Fleas are active and may jump prodigious distances. Contamination of the environment with flea eggs and pupae enables infestation to be acquired by passing through any area previously exposed to animals with fleas. The life span of an individual flea may be more than one year. During this time, the female may lay as many as 400 eggs.

Fleas are notorious vectors for infectious and parasitic diseases; however, no such instances have been documented in South American camelids.

Clinical Signs

Adult fleas consume only blood and may cause anemia. In massive infestations, exsanguination from fleas may be fatal. While biting the host, fleas deposit saliva, which may stimulate an allergic response varying from mild irritation to marked pruritus, hyperemia, swelling, and dermatitis.

Treatment

As with lice, fleas can be treated using pyrethrins, permethrins, chlorinated hydrocarbons, carbamates, and organophosphates as well as ivermectin in a variety of formulations. Multiple treatments at 7–14-day intervals are recommended to cover the various stages of development within the environment or on the host.

Mosquitoes

Identification

Mosquitoes are slender-bodied, long-legged, winged insects with spherical heads and a long slender proboscis, which is used to cannulate capillaries to ingest blood meals from the host [9]. Numerous species exist but differ in habitat preference and have no significant host specificity.

Life Cycle

The gravid female lays eggs in water. The eggs hatch in less than a week to become air-breathing larvae. Larvae undergo four molts over a period of two weeks, progressing to the pupal stage that lasts two to seven days. The adult emerges from the puparium and must dry itself before being able to fly off within 24 hours. The entire cycle requires approximately one month or less in warm, moist habitats with stagnant water [3].

Epidemiology

Unlike fleas and lice, mosquitoes can travel long distances and easily reach a host, including camelids. They do not remain on the host for extended portions of their life cycle. Rather, they land, ingest a blood meal, and leave the host. Mosquitoes can be a nuisance and may stimulate an allergic-type reaction locally at the site where the mosquito took a blood meal, similar to that of fleas. Like fleas, mosquitoes are vectors for numerous infectious diseases such as West Nile virus and Eastern Equine Encephalitis, serving as an intermediate host for some and as a mechanical vector for others.

Clinical Signs

Mosquito bites, even in large numbers, are unlikely to cause anemia. However, the inflammatory response can cause individual bites to become pruritic, hyperemic, and swollen. The most concerning clinical signs are associated with the secondary diseases that they can transmit. Signs may be generalized: lethargy, depression, fever, anorexia, weakness, and musculoskeletal abnormalities ranging from staggering gait to muscle tremors and recumbency, seizures, coma, or death can occur with West Nile virus [10].

Treatment and Control

Reducing mosquito numbers requires management of the environment. Mosquitoes thrive and reproduce readily in regions with stagnant water and low oxygen content, allowing eggs to survive maturation to the larval stage. To reduce the density of the mosquito population, stagnant water should be drained, and water buckets changed frequently. For stagnant water sources that cannot be drained or removed, placing fountains or other devices to disrupt the water surface and increase the oxygen content of the water will decrease the mosquito population and, therefore, reduce their ability to propagate their eggs [3]. Mosquito repellents in water sources or topical human products are effective for a short period but should be used with caution on camelids or within water sources.

The West Nile Virus vaccine is considered extra-label use in camelids, but it has been shown to be safe and efficacious in camelids to produce protective antibodies that last for three to six months duration [11].

Myiasis

Common Flies

Identification

Llamas and other camelids are affected by the same types of flies that afflict domestic livestock. The house fly (*Musca domestica*) has four dark stripes on the thorax and yellow spots on the side of the abdomen and is approximately 7 mm long [3]. The face fly (*Musca autumnalis*) is slightly larger than the house fly and lacks yellow color on the abdomen. The biting stable fly (*Stomoxys calcitrans*) is also a nuisance to camelids. It is the same size as the house fly but has a long, stiff proboscis and gray-brown spots on the abdomen. Other species of flies may annoy or afflict camelids but their presence is regionally dependent.

Life Cycle

HOUSE FLY. The female lays eggs on manure or decaying organic matter. The larvae hatch in less than 24 hours and grow and molt twice in a few days to become third-stage larvae, which move to a drier area and pupate. Adults emerge in two to three weeks, climb to the surface, and spread their wings to dry. Adults live for six to eight weeks, during which time, a female will lay 2000 eggs. The entire cycle takes three to five weeks [1].

FACE FLY. The life cycle is similar to that of the house fly (Figure 7.3), except that the female lays eggs in fresh cattle feces, where hatching, larvation, and pupation occur. Hence, face flies are a problem only where camelids are housed near cattle.

BITING STABLE FLY. The life cycle is similar to those of house and face flies. Eggs are laid in decaying organic matter or manure. Both male and female ingest blood from the host and feed once or twice daily.

Epidemiology

House flies are found both inside and outside of barns and sheds. Face flies usually do not enter buildings, so camelids may gain relief by going inside. However, face flies may enter buildings to escape cold weather and may overwinter as adults, becoming active on days of unseasonably warm weather. All flies are more active on warm days than on cool days and cease activity at dark.

Clinical Signs

Biting flies may cause additional irritation. Excessive lacrimal secretion in the camelid is attractive for all fly species and may cause conjunctivitis.

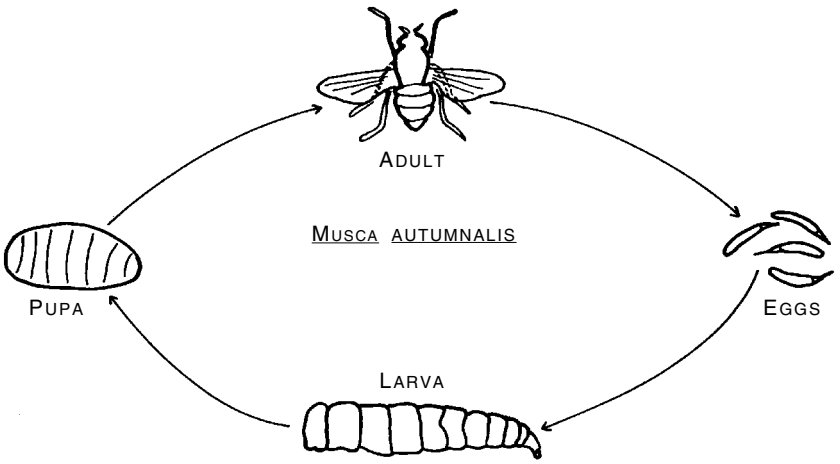

Figure 7.3 Simple interpretation of the life cycle of a fly (*M. domestica, M. autumnalis, and S. calcitrans.*

Management

Fly control is a never-ending challenge to livestock managers. Methods used to control files or reduce morbidity associated with flies in other livestock species like spray-on insecticides, cloth visors or head coverings, environmental fly traps, regular clearing of dung piles, and appropriate management of standing water should aid in fly management in camelid herds.

Black Flies (Buffalo Gnats)

Identification

Simulid flies are closely related to mosquitoes but have a short, piercing proboscis and a hump over the thorax. The legs are not as long as those of mosquitoes.

Life Cycle

The female black fly lays eggs only in running water, either on the surface or on submerged surfaces of stones, twigs, or vegetation. The eggs may remain in the water for months, overwinter in this state, or hatch in a few days. Larvae are mobile, with the lopping type of ambulation typical of an inchworm. Larvae molt six times before entering the pupal stage, which floats near the surface for their respiratory tube to take in the necessary air. Adults emerge from the puparium and may swarm in dense clouds if the population is large [1].

Epidemiology

Black flies are found throughout the world but are concentrated in warmer climates. Flies are active in the morning and evening. They do not live on the hosts but take blood meals from them.

Clinical Signs

Swarms of black flies are a nuisance to animals and may inhibit grazing on pasture or other feedstuffs. In sensitive animals, the bites may simulate those from mosquitoes and cause local pruritus, swelling, and hyperemia.

Treatment

Primary treatment of black flies is similar to that of mosquitoes where the population of flies is controlled by reducing water sources which limits the ability of these flies to propagate.

Tabanids (Horse Flies, Deer Flies)

Identification and Life Cycle

Tabanids are medium to large flies with powerful wings and large eyes. The eggs are glued in neat masses on vegetation overhanging or near a water source so the larvae that hatch in approximately one week fall into the water to continue maturation. Larvae and subsequent pupae may burrow into the mud at the bottom of a pond or stream and overwinter in the moistened dirt. Adults emerge from the puparium and females require a blood meal soon after to complete the life cycle. The full length of the life cycle requires a minimum of four months or may extend to the next season depending on environmental conditions [1].

Epidemiology

Tabanids are diurnal and are especially active on hot, bright, humid days. Only females take blood meals every three or four days. The bite from a tabanid is painful. Tabanids may be responsible for transmission of parasites and infectious agents, but this has not been documented for South American camelids. Tabanids are an important intermediate host for *Trypanosoma evansi*, the cause of trypanosomiasis (surra) in camels and other artiodactylids in Africa and Asia [12]. There is circumstantial evidence of *T. evansi* infection in South America, with a potential for infection in South American camelids.

Clinical Signs

The large mouth parts may pierce capillaries, which continue to flow after the tabanid has departed, attracting other non-biting flies. Sensitive individuals may develop

erythema and swelling at the bite site. As with other flies, tabanids can be a nuisance to animals.

Treatment

No treatment or prevention is available other than swatting the fly if observed near or on the animal.

Blow Flies

Identification

Many species of flies deposit eggs in fresh or necrotic wounds of animals. Camelids are equally susceptible to the attacks of these fly varieties which include screw worms and blow flies.

The primary screw worm (*Cochliomyia hominivorax*) invades healthy tissue whereas blow fly species prefer dead, necrotic tissue. The blue blow fly (*Calliphora* spp.) may be as long as 11 mm. The thorax is dull colored, and the abdomen is blue-green. The green blow fly (*Phaenicia* spp.) is approximately 7 mm long, with green to bronze coloration of the thorax and abdomen. The black blow fly (*Phormia* spp.) is approximately 8 mm long and is bluish – black in color [3].

Life Cycle

The female fly of the primary screw worm lays approximately 200 eggs in a fresh wound. The eggs hatch in 24 hours and the larvae feed on flesh, invading contiguous tissue. Mature larvae leave the wound in seven days to pupate. Adults emerge within one to several weeks. The entire life cycle may be completed in as little as two and a half weeks or continue for several weeks. The primary screw worm could be considered an obligatory parasite, meaning the organism cannot complete its life cycle without a host.

The facultative parasitic calliphorids have a similar life cycle but do not invade fresh wounds. They are attracted to suppurative or necrotic wounds or areas soiled with feces and urine. Examples of such sites include the neonatal umbilicus and downer animals that may develop decubitus wounds or ulcers on soiled areas and be subject to infestations.

Epidemiology

Primary screw worms have been eradicated from the United States but may be present in other areas of South America as they are endemic in tropical and subtropical regions of the Western Hemisphere. Blow flies are found throughout the world.

Clinical Signs

The main clinical sign of both the blow fly and screw worm is observing the larvae crawling and feeding in the wound.

Treatment

The wound should be cleaned and debrided to remove necrotic tissue. Fiber should be clipped to allow proper drainage of the wound and exposure to the air. Larvae may be removed mechanically or killed by the application of insecticides onto the wound. The vehicle used to solubilize the insecticide may be irritating to the tissue and may be systemically absorbed by the wounded tissue more readily than through healthy skin, so only as much fluid as is necessary to destroy the larvae should be applied. Secondary injury and trauma from treatment is also possible. Chloroform, ether, and hydrogen peroxide cause the larvae to retreat from crevices and cavities; however, each of these treatments have potential caustic effects to both the individual applying the product as well as the patient. Once larvae are destroyed, the wound should be properly treated and dressed. The wound should be monitored for additional larvae and control re-invasion. Ivermectin is effective for treatment of larvae.

Bot Flies

Identification

Only three species of bot flies have been reported from camelids: the sheep and goat nasal bot fly (*Oestrus ovis*), various species of the deer nasopharyngeal bot fly (*Cephenemyia spp.*), and the camel bot fly (*Cephalopina titillator*). The first two species are important for South American camelid owners and are discussed separately.

Oestrus ovis IDENTIFICATION. The adult fly averages 12 mm in length and is approximately the size of a small honeybee. Soulsby describes the fly as having a dark gray color with small, black, especially prominent spots on the thorax [1]. First instar larvae are whitish, translucent, and less than 2 mm in length. As the larvae mature, dark, transverse bands develop on the dorsal aspect of the segments. Mature larvae are approximately 3 cm in length, with a long, tapering anterior end and a flat posterior end. There are two black stigmal plates on the posterior surface (Figure 7.4), black oral hooks on the anterior end, and rows of small spines on the dorsal surface.

LIFE CYCLE. The bot fly deposits larvae around the nostrils of the camelid. Flies cause considerable annoyance to the animal which can cause them to take evasive action by pressing their muzzle close to the ground or against other animals. The larvae instinctively crawl upward into the nasal cavity and may stay in the turbinate area or crawl into a sinus. The cycle in the camelid has not been defined as to the location or length of time necessary to complete maturation, if, indeed, the larvae reach maturity in this species. In sheep, the first instar

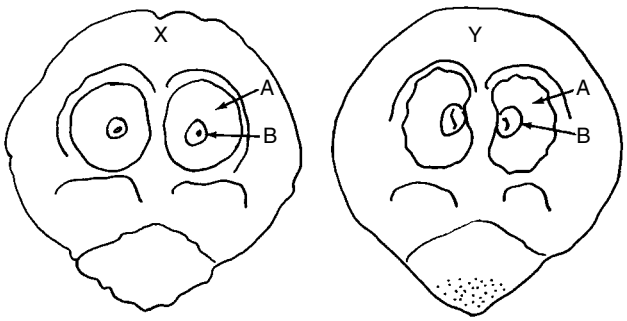

Figure 7.4 Third instar larvae. (X) *Oestrus ovis*, (Y) *Cephenemyia* spp., (A) peritreme (stigmal plates), (B) button, posterior view. Note that the buttons of *Oestrus* are centrally located in the peritreme, and the buttons of *Cephenemyia* are peripherally located in the peritreme.

progresses to the second in two weeks to as long as nine months. The second instar matures in 25 days or more. Mature larvae crawl out of the nasal cavity, fall to the ground, and pupate for three to six weeks in warm weather or remain viable in the pupal stage for months if protected from freezing.

EPIDEMIOLOGY. Bot flies are active during the day but spend most of their time resting in cracks or crevices. In temperate climates, the fly is active in all but winter months, and in warm climates throughout the year. This fly is a serious parasite in sheep and goats in many areas. It has only been circumstantially identified from a camelid. Proper identification should be pursued on any nasal larvae found in a camelid as the more common larva found has been the deer bot fly.

CLINICAL SIGNS. The syndrome seen in camelids has recently been reported [13]. A llama from within a herd of sheep and alpacas at an experimental station in Peru presented with clinical signs of dyspnea, respiratory stridor, and bilateral mucoid nasal discharge. Therapy, including antibiotics and anti-inflammatory medications, were initiated with no improvement in clinical signs. On necropsy, *O. ovis* larvae were identified in the nasal fossae and cranial sinuses. Affected llamas can also exhibit signs like those seen in sheep, with nasal exudation, sneezing, and head shaking. Visualization of the larvae with endoscopic examination would be fortuitous because the larvae are usually in the sinuses.

TREATMENT. Ivermectin therapy, at doses of 0.2 mg/kg, has been successful.

Cephenemyia spp. (Deer Nasopharyngeal Bot fly, Throat Bot fly, Deer Nasal Bot fly).

IDENTIFICATION. The adult fly superficially resembles a small bumblebee and is approximately 14 mm in length [14]. First instar larvae are approximately 1 mm long and may molt up to 3 mm. Second instar larvae vary in length from 3 to 13 mm. Third instar larvae vary from 12 to 40 mm. The puparium is 16–20 mm long. Third instar larvae of all species are similar, making it difficult to identify the species. First instar larvae are most easily identified but are rarely seen or recovered.

A third instar larva removed from the nasopharynx of a llama had the following characteristics: cream color, 2.5 cm long, and approximately 0.7 cm in diameter (Figure 7.5a and b). The peritremes (stigmal plates) were crescent shaped with the outer margins slightly sinuous. The button was on the inner margins (Figure 7.4). In contrast, the peritremes of the third instar *O. ovis* larva are circular with a central button (Figure 7.4).

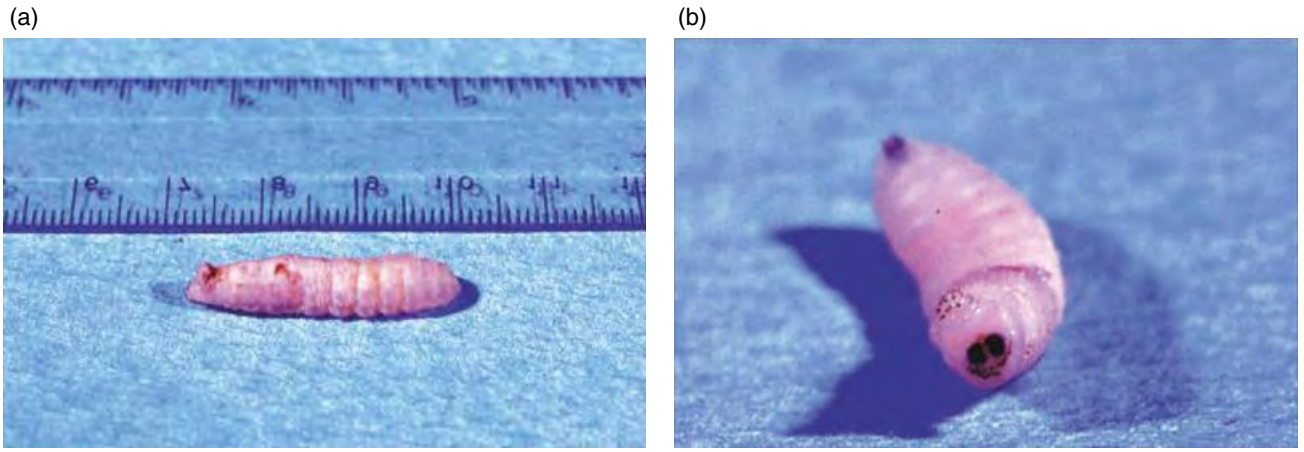

Figure 7.5 (a) Third instar larva of *Cephenemyia* sp. (b) Posterior peritremes of *Cephenemyia* sp.

Life Cycle. Primary hosts for *Cephenemyia spp*. are cervids. Whether the cycle is completed in camelids is unknown. In deer, the adult fly lives for two to three weeks without feeding. Deposition of larvae by the female is carried out by spraying larvae into the nostrils of the host (Figure 7.6). Larvipositing evokes a behavioral response in deer where the head is jerked back, and sneezing and snorting ensue, accompanied by head shaking. Older animals may lower the head, pressing the nose against the ground. A few animals will run a few yards immediately following a fly strike [3].

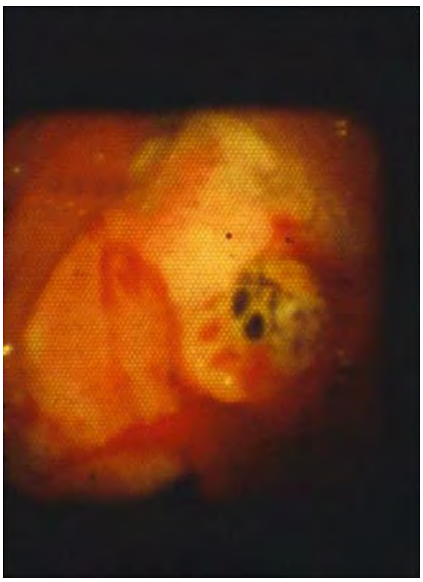

Figure 7.6 Endoscopic view of a *Cephenemyia* larva *in situ* in the nasal passage.

The larvae may remain for a time in the nasal passages but ultimately migrate to the nasopharynx where they take up residence in the retropharyngeal pouches found in cervids and develop to the second and third instar. At maturity, the third instar larvae migrate or are sneezed out through the nostrils, fall to the ground, pupate, and after 16–31 days, emerge as adult flies (Figure 7.7).

Epidemiology. Several instances of llamas infested with the deer nasopharyngeal bot fly have been described in the literature [14]. Camelids cohabiting pastures with deer are at risk. Various species of flies of this genus can be found in any area of North America where there are cervids. Camelids are aberrant hosts for this parasite, and it is not known whether the cycle is completed in the llama.

Clinical Signs. In deer, the oral hooks and spines of the larvae irritate the mucosa of the nasopharynx, inducing secretion of a viscous mucoid exudate that apparently provides nourishment for the larvae. Affected deer may have nasal exudate and sneeze periodically.

As the camelid is an aberrant host, there is a more significant reaction to infection with the development of a granulomatous swelling in the nasopharynx and nasal cavity [15]. Of the known affected camelids, none have died from infestation and no biopsies have been taken to establish the histopathology of this mass. However, the larvae can be seen embedded in the surface of the mass with endoscopic examination.

Prominent signs in the camelids are sneezing and coughing, with or without nasal discharge. The lesion is often observed in the nasopharynx with any exudation likely being swallowed and not expectorated through the nasal

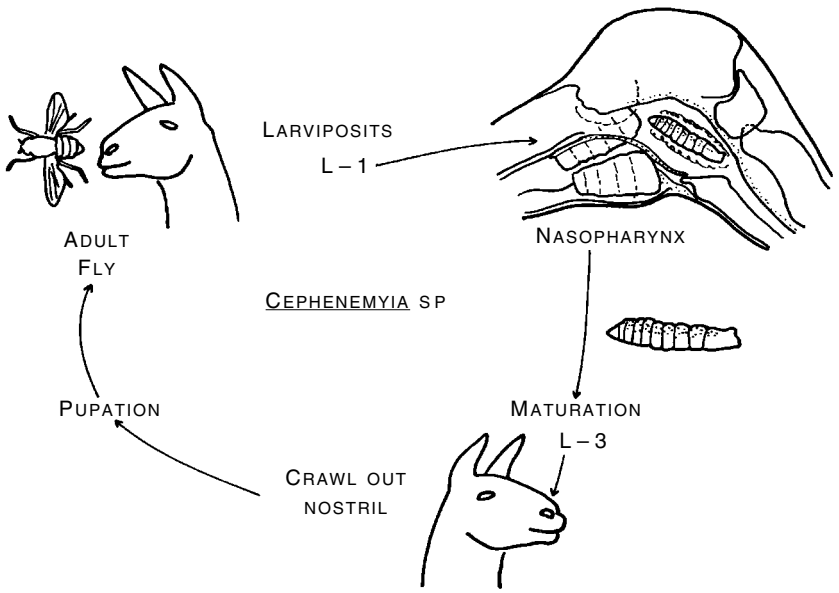

Figure 7.7 Life cycle of *Cephenemyia* spp.

cavity. Owners have reported that affected llamas develop shortness of breath or fail to keep up with others on the trail. Though the llama is an obligate nasal breather, if the lesion becomes obstructive, the animal will be forced to breathe through the mouth and will result in debilitating effects. Breathing will be audible. There is usually no febrile response or change in the hemogram. Due to the significant respiratory compromise and clinical signs that can occur with infestation, the bot fly of the *Cephenemyia* spp. is an important rule-out with other respiratory infectious diseases.

DIAGNOSIS. Primary evidence of larval infestation involves identifying the characteristic clinical syndrome and observing the environment in which the camelid lives as deer populating the region in conjunction with clinical signs is highly suggestive of deer nasal bots. The lesions, and perhaps the larvae, may be seen via endoscopic examination in an anesthetized individual (Figure 7.6). The mass appears as a radio-dense area in a radiograph (Figure 7.8). A final diagnosis may be made on the basis of response to treatment.

TREATMENT. Though too few cases have been seen and treated to be conclusive, there is evidence that ivermectin is effective at 0.4–0.6 mg/kg (double to triple the normal dose).

Camel Nasal Bot Flies

ETIOLOGY. The larval stages of the camel nasal bot fly (*C. titillator*) (family Oestridae) are common inhabitants of the nasopharynx of Old World camelids [6].

LIFE CYCLE. The female fly deposits first-stage larvae in the nostrils. The larvae are small and 0.7 mm in length. They migrate to the nasopharynx and attach to the mucosa. The first molt occurs in the labyrinth of the ethmoid bone and the second molt occurs in both the labyrinth and the pharyngeal cavity [16]. Maturation can take up to 11 months before larvae detach from the mucosa and are expelled out of the nostril by sneezing. Pupation occurs in the soil (Figure 7.9).

EPIDEMIOLOGY. The prevalence of *C. titillator* infestation is high in countries maintaining a sizable camel population, varying from 47 to 100%.

CLINICAL SIGNS. The adult flies are not as irritating to camels as the deer bot flies to South American camelids, and they may even be seen in large numbers resting on the heads of camels. Usually, camels exhibit no clinical signs of infestation except when mature larvae are migrating from their attachment sites to exit the nostril, during which time sneezing is common. Local inflammation may occur at the

Figure 7.8 Lateral radiograph of the head of a llama with a *Cephenemyia* granuloma in the nasal pharynx.

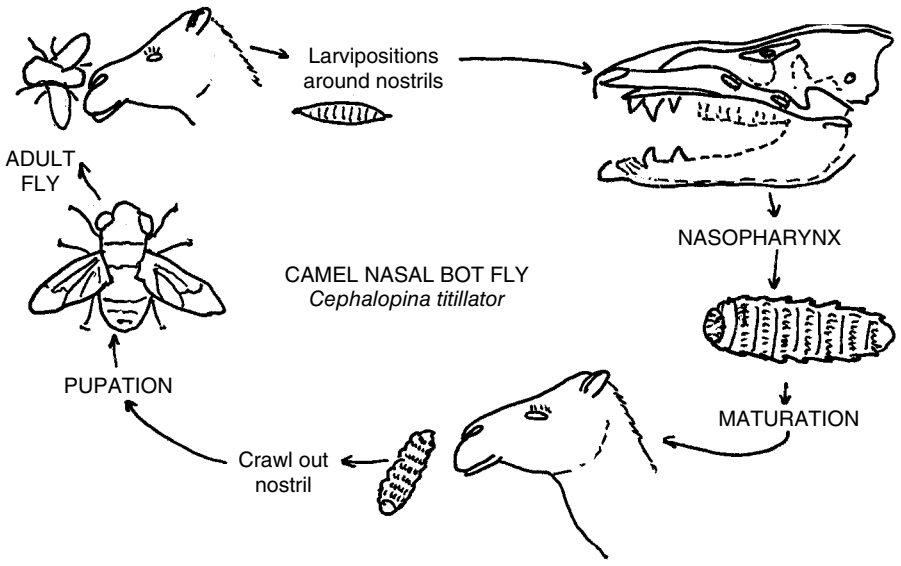

Figure 7.9 Life cycle of *Cephalopina titillator*.

site of attachment and heavy infestations may cause nasal exudation. Mortality is rare and is usually associated with larval penetration of the ethmoturbinates, resulting in meningitis. The camel is the primary host of *C. titillator* and not an aberrant host as is *Cephenomia* in South American camelids; thus, there is no granulomatous reaction or potential obstruction of the airways.

DIAGNOSIS. Larvae may be seen exiting the nostrils or are incidentally found in the nasopharynx at necropsy when camels die from other diseases. All larval instar stages have been found in the labyrinth of the ethmoid bone. Additional sites of larval location include the turbinates (first instar), pharyngeal cavity (second and third instar), and the nasal meatus (third instar) [16]. The larvae may be observed *in situ* by endoscopic examination.

MANAGEMENT. The larvae may be killed with ivermectin (0.2 mg/kg subcutaneously) or other parasiticides.

Ticks

Identification

There are two major groups of ticks: hard-bodied ticks (family Ixodidae) and soft-bodied ticks (family Argasidae). There are numerous genera of hard-bodied ticks that are characterized as having a scutum with mouth parts at the anterior margin. Further differentiation between male and female ticks can be made as the entire dorsal surface of males is covered by the scutum compared to females that have only a partial scutum. Though no reports in the literature list the species found in South American camelids, it is likely that non-host specific ticks are identified according to the region in which the camelid herd is located [17]. Tick paralysis (discussed later) has been reported in a llama as a result of hard-bodied tick attachment.

The only soft-bodied tick that has caused clinical disease in camelids is the spinose ear tick (*Otobius megnini*). Soft-bodied ticks lack a scutum and have mouth parts originating on the ventral surface. The adult tick, which is not parasitic, is about 8 mm in length and has a constriction at the middle, giving it a fiddle shape [9]. The larvae are pear shaped to spherical and about 2–3 mm in length. The nymph, which is the primary parasitic stage, molts twice, and when engorged measures 7–10 mm in length.

Life Cycle

All ticks have a life cycle that includes four stages of development (egg, larvae, nymph, and adult) and can be further characterized as a one-, two-, or three-host tick. Ticks drop from their host between life stages (except one-host ticks). Larvae hatch from eggs and are often present on vegetation for improved access to animals within the environment. When seeking a host, heat and carbon dioxide concentrations attract the tick. Once larvae attach to a host, they feed on blood, and can fall from the host to develop into the nymphal stage or stay on their host (if a one-host tick) to develop into a nymph. Mating occurs between adults while feeding, but eggs are laid by a female after detachment from the host [18].

SPINOSE EAR TICK. *O. megnini* adult ticks do not feed and may live for six months in cracks and crevices of barns, sheds, and fences or under feed bunks or stones (Figure 7.10). The female may oviposit 500–600 eggs in the previously mentioned areas during her lifetime. The eggs hatch to six-legged larvae, ready to attach to a host within 10 days. The larvae migrate to the ear canals and begin to feed on lymph. Larvae survive only two to four months without a suitable host [1].

The eight-legged nymphs molt twice within the ear canal, remaining on a single host for one to seven months, feeding on blood. The mature nymph climbs out of the ear canal, drops to the ground, and molts to the adult stage. Usually only one mating cycle is completed each year.

HARD-BODIED TICKS. Some species complete the entire cycle using a single host species; others require two hosts, and a few require three separate hosts, one for each of the life stages. One-host ticks do not commonly affect camelids. *Ixodes scapularis* (black-legged tick), *Ixodes pacificus* (Western black-legged tick), *Dermacentor andersoni* (Rocky Mountain wood tick), *Amblyomma americanum*

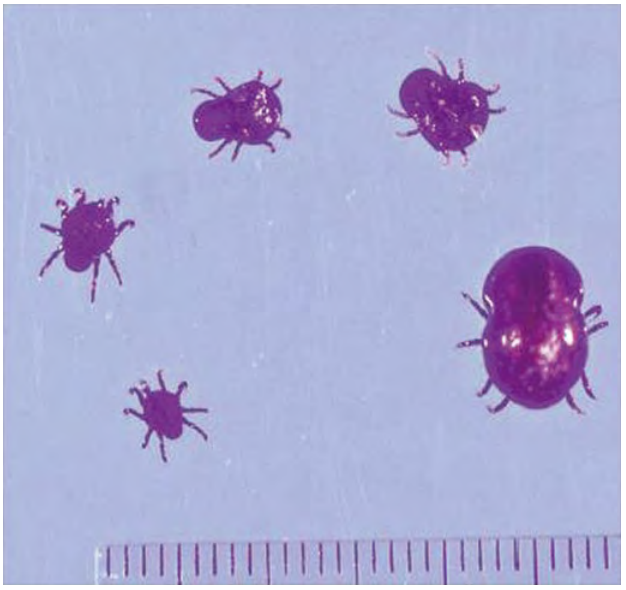

Figure 7.10 Spinose ear tick nymphs.

(Lone-Star tick), and *Amblyomma maculatum* (Gulf Coast tick) are examples of three-host ticks. The female oviposits only once, producing thousands of eggs. Larvae feed only once, and there is only one nymph stage, taking a single feeding. Hard-bodied ticks are more likely to be encountered in open grassland or shrubby areas rather than near sheds and barns, as is the case with soft-bodied ticks.

Epidemiology

Spinose ear ticks are a problem in camelids of localized regions of the western United States. Animals kept entirely on pasture or open range are less likely to encounter the ticks because the preferred habitat of these ticks is around buildings, sheds, wooden fences, cracks, crevices, and rough-barked trees, e.g. *Eucalyptus* in Australia [5].

Hard-bodied tick infestation is a problem in much of the West, especially with camelids used for packing into wilderness areas.

Clinical Signs

The most common signs of spinose ear tick infestation are head shaking and exudate from the external ear canal and is characterized as otitis externa. Otitis externa also has the potential to spread deeper into the middle and inner ear which could create a potentially life-threatening scenario. Additional signs include scratching at the head or ears and neurologic signs in chronic infestations that result in a head tilt, weight loss, circling, and possibly unilateral facial paralysis [5]. Examination should reveal the nymphs within the ear canal. Anemia may result in young llamas with heavy infestations due to the blood feeding nature of ticks.

Treatment Ivermectin (0.2 mg/kg) administered subcutaneously is effective for both the larvae and nymphs of the spinose ear tick but ticks that are not actively feeding will not be affected [5]. The ear canal may be physically cleaned with standard techniques and an insecticide solution can be instilled into the ear canal. Manual removal of the tick would be best but can be difficult due to the angled and narrow anatomy of the external ear canal in camelids. Additionally, a mixture of ivermectin and mineral oil, permethrin spray, and fipronil spray have been shown to be efficacious for treatment. Reinfestation with the spinose ear tick is possible and, therefore, it is recommended to routinely treat all animals to break the life cycle. There is little hope of eradicating the tick from an environment because the adults hide in crevices and under objects difficult to reach with sprays. Periodic inspection of the environment and treatment is necessary to prevent a buildup of infestation.

Tick Paralysis

EPIDEMIOLOGY. Tick paralysis has been recognized for more than 50 years, and 64 species in 10 different genera of ticks have been incriminated [19]. *Dermacentor* spp. are most commonly implicated in causing tick paralysis with the species varying depending on the geographic region of the United States. *D. andersoni* and *Dermacentor occidentalis* can cause tick paralysis in animals in the western Unites States while *Dermacentor variabilis* is more common in the eastern region. *A. americanum* can also cause tick paralysis. These two tick species are expanding their geographic distribution and are no longer restricted to southern regions in the US.

In general, only female hard-bodied ticks produce toxin, which is localized in the saliva and is injected into the host when the tick ingests a blood meal. No information is available as to why a given tick produces the toxin. In the United States, the neurotoxin from *Dermacentor* spp. interferes with acetylcholine liberation at the neuromuscular junctions, causing a lower motor neuron paresis. The paresis can be further described as slower nerve conduction, depressed amplitude of muscle action potential, and progressive ascending flaccid paralysis [20].

In Australia, *Ixodes holocyclus* is responsible for a serious form of tick paralysis in animals predominately on the east coast but has also been detected in affected animals on the west coast and inland regions. Even if the offending tick is found and removed, recovery may not occur unless specific tick antiserum is administered. The toxin produced by *I. holocyclus* results in myocardial depression and can lead to pulmonary edema and diastolic failure.

The bite from a single tick is sufficient to kill an animal if the tick is not removed. Based on work in other species, there is variable host susceptibility. There may also be seasonal or annual variability, since there have been reports of epidemics of paralysis in some years and none in other years. Duration of feeding, season, and stage of life cycle can cause variation in the clinical signs associated with paralysis that are observed due to the toxin released from the tick [21].

CLINICAL SIGNS. Tick paralysis has been diagnosed in the llama [20]. A one-year-old male llama was observed as normal on one day and the following day was completely paralyzed. A diffuse lower motor neuron disease was suspected. *Dermacentor* spp. ticks were found. The llama was sprayed with a pyrethrin insecticide, and ivermectin (0.2 mg/kg) was administered parenterally. Muscle tone reappeared within 12 hours, and the llama was standing in 36 hours. A documented case of tick paralysis induced by *I. holocyclus* in Queensland has been reported in a two- to three-year-old male llama. Clinical findings included unilateral facial nerve paralysis, right nostril collapse resulting in

inspiratory stridor, decreased cranial nerve reflexes, and decreased muscle tone and withdrawal reflexes of the hind limbs [21].

An affected camelid would be expected to follow a similar progressive course as noted in other species. After the tick begins to feed, signs may develop two to nine days later and would worsen over a few days. Paresis and paralysis are progressive and cause an ascending motor paralysis, beginning in the hind legs and moving cranially. Initial clinical signs may also include an abnormal gait of the hindlimbs, stumbling, increased time cushed, and eventual recumbency. Loss of all motor functions follows the development of ataxia.

The signs may progress rapidly over a few hours or more slowly over a period of 24–48 hours. Paralysis ascends to the forelimbs, neck, head, and face. In the early stages, the victim is bright and alert, able to eat and drink, but as the paralysis ascends, difficulty in chewing and swallowing develops. The cause of death is respiratory arrest from involvement of the respiratory centers of the brain.

Diagnostic tests for tick paralysis are often unremarkable or changes are generalized and can be found in a plethora of other conditions. Common abnormalities of bloodwork changes include a stress leukogram with hyperglycemia and hypokalemia, and increased muscle enzymes. Increases in inflammatory mediators and by-products can be secondary to aspiration pneumonia from a poorly functioning esophagus. A pre-renal azotemia and hyperproteinemia may also be observed but are often secondary to dehydration or heart failure.

Treatment and Control. Currently, no drugs can counter the effects of the neurotoxin. The only cure is to remove the tick from the animal. The difficulty lies in finding a tick on an unshorn llama or alpaca. Because only one tick may be sufficient to cause the paralysis, finding it is crucial.

Removal of the tick may reverse the signs in 2–12 hours. Assuming inability to find the offending tick, a standard insecticide dip may be employed. Supportive care in the form of a temperature-controlled environment, intravenous (IV) fluids, nutritional support, anti-inflammatory medications are also recommended. Amitraz (Mitaban, Zoetis, Parsipanny-Troy Hills, New Jersey) may be applied as a dip in a concentration of 250 ppm of the active drug (10.6-mL bottle/2-gal water) as well as a pyrethrin product to kill ticks that go undetected. Ivermectin and its related products are not recommended for treatment as it acts more slowly to kill the tick.

Controlling the tick population can be difficult, but preventative measures in the form of repellants or acaricides are recommended. Within the United States, ticks are typically seasonal, and females have the greatest activity in the late spring and early summer. Specific preventative measures include monthly moxidectin or ivermectin administration, permethrins, dog tick collars, fipronil sprays, or bovine ear tag repellents.

Mites

Four types of mites have been reported in camelids: *Sarcoptes, Psoroptes, Chorioptes*, and *Demodex* [5, 19, 22]. Of these, *Sarcoptes scabei var aucheniae* and *Chorioptes bovis* are the most common and troublesome. The identity of the mite must be established to institute proper therapy and determine whether the case must be reported to regulatory authorities. Psoroptic mange is a reportable disease in the United States and all forms of mange are reportable in certain states. Of production losses due to ectoparasites, sarcoptic mange is responsible for 95% of the total loss in South America. Though *Chorioptes* is less severe than sarcoptic mange, *Chorioptes* is more common and widespread outside of South America.

Sarcoptic Mange Mite

Identification. *Sarcoptes scabiei* is morphologically identical in all animals [8]. The identifying characteristics of the mite are a globose or round body with short legs [17]. The caudal two pairs of legs do not extend beyond the margin of the body. There are bell-shaped suckers (caruncles) on the long, unsegmented stalks (pedicels) on the tarsi of all the legs in the male but only on the anterior two pairs in the female mite. The male has no adanal suckers (copulatory discs). The capitulum (head or false head) is broad (Figure 7.11). The maximum size of the female mite is about 0.5 mm and of the male, 0.3 mm.

Figure 7.11 Gravid *Sarcoptes scabiei*.

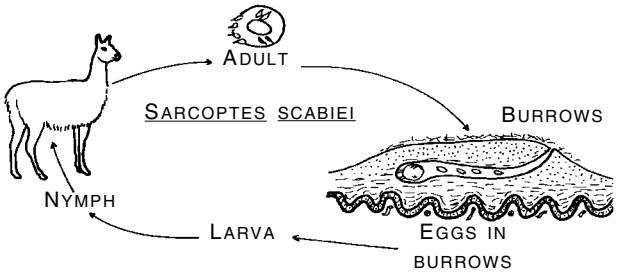

Figure 7.12 Life cycle of *Sarcoptes scabiei*.

LIFE CYCLE. The entire life cycle is completed in the skin of the camelid due to the deep burrowing nature of the mite (Figure 7.12). Adult male and female mites copulate on the surface of the skin. The fertilized female burrows into the skin, depositing eggs behind her as she tunnels. The eggs hatch in three to eight days. Larvae migrate to the skin surface and mature through the nymph stage to become adults in four to six days. The entire life cycle is completed in 7–14 days. Males and unfertilized females may also burrow or may follow the tunnels of the fertile female [1].

CLINICAL SIGNS. The burrowing mite causes hyperemia, papules, and pustules, which become encrusted as *Sarcoptes* mites burrow through all the epidermal layers except the basal layer [5]. Additional clinical signs include pruritus that can lead to alopecia, yellow crusting, skin thickening and bleeding, hyperpigmentation, erythema, and an overall generalized ill-thrift appearance [9]. The skin becomes thickened in affected areas and loses its vitality, thus becoming susceptible to secondary bacterial infection with subsequent production of exudate. Lesions are usually found on the limbs (between the toes), medial thighs, ventral abdomen, chest, axilla, and perineum of the female and prepuce of the male [23]. This distribution is similar to that seen with chorioptic mange. In severe cases of infestation, the entire head, body, and limbs may be involved (Figures 7.13–7.18). Though uncommonly observed in North America, this disease is known as *Surna* in South America.

Figure 7.13 Sarcoptic mange on the lips of an alpaca.

Figure 7.14 Sarcoptic mange in the interdigital space of an alpaca. There is a scent gland in this area that may have an accumulation of debris on it normally.

Psoroptic Mange Mite

IDENTIFICATION. The body of *Psoroptes* spp. is more oval shaped than that of *S. scabiei*, and the legs are longer, projecting beyond the margin of the body both in the front and back (Figure 7.19) [1, 3]. The pedicels of the tarsal suckers are long and composed of three segments (Figure 7.20). In the female mite, the third set of legs end in bristles instead of suckers (Figure 7.21). In the male mite, all legs have suckers, and the fourth set of legs (caudal set) are much shorter than the third. The male mite also has a pair of adanal suckers. The posterior margin of the abdomen of the male is bilobed, and the capitulum is elongated. The maximum size of the male mite is 0.7 mm, that of the female about 0.8 mm.

LIFE CYCLE. The entire cycle is completed on the host. Eggs are deposited on the skin at the periphery of the lesions and hatch in one to three days if the eggs remain in contact with the skin. If separated from the skin by crusts, hatching becomes delayed until four to five days, and if detached from the body along with fiber, the eggs will either hatch in 10 days or die. The larval stage lasts for two to three days, during which time feeding occurs. The nymphal stage lasts from three to four days. Adults copulate

Figure 7.15 Sarcoptic mange of the caudal abdomen and inguinal region in an alpaca.

Figure 7.16 Sarcoptic mange in the perineal region of an alpaca.

soon after maturation. A female mite lives for 30–40 days, laying about five eggs daily. The cycle may be as short as 10 days but is usually about three weeks.

Figure 7.17 Sarcoptic mange of the prepuce of an alpaca.

Figure 7.18 Sarcoptic mange and secondary deep pyoderma and cellulitis affecting the distal limb of an alpaca.

Clinical Signs. The mouth parts of mites penetrate the epidermis to suck lymph which stimulates a local inflammatory reaction that exudes serum. The dermatitis produced is characterized by erythema, thickening of the pinna and external ear canal, ceruminous crusting, and purulent exudate. Psoroptic mites have been found only on the pinna or within the ear canal in the United States, causing the clinical signs of head shaking, head tilt, ear droop, incoordination, ear scratching, and distracted

Figure 7.19 Male *Psoroptes* sp. mite. Note elongated capitulum (blue arrow), adanal suckers (orange arrow), rounded abdominal tubercles (green arrows) and bilobed abdomen.

Figure 7.20 High-power magnification (400×) of the segmented tarsal pedicel of the tarsal suckers of *Psoroptes* sp.

behavior [24]. Secondary bacterial infections often occur with the potential of neurologic signs to develop which would indicate rupture of the ear drum and infection into the middle and inner ear. When fibers are pulled, rubbed, or fall out, the mites move to the margins of the lesion. This habit should be kept in mind when scrapings are collected to check for the presence of mites.

Lesions are usually located around the shoulder and along the back, sides, and tail head. An early lesion or one at the margin of an established infestation consists of small papules about 5 mm in diameter. The papules are yellowish and have a moist surface. A mite is likely to be found in the center of a papule. Within four to five days, the serum will exude, congeal, and form the crusts characteristic of a scab.

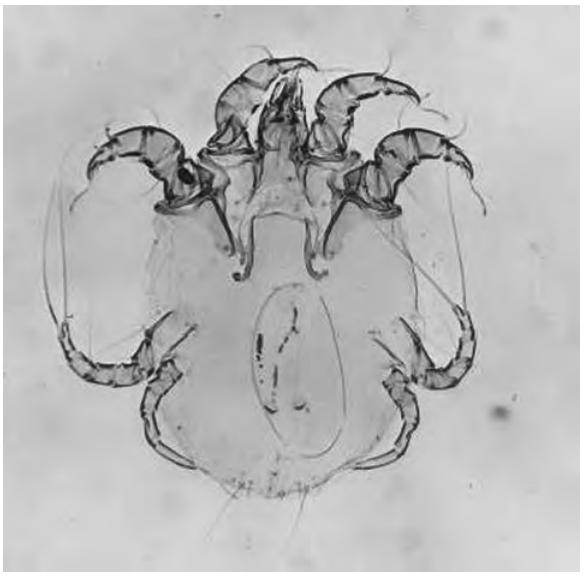

Figure 7.21 Gravid female *Psoroptes* sp. mite.

Though dermatitis is produced, the skin does not become thickened to the extent observed in sarcoptic mange.

EPIDEMIOLOGY. Little is known about the seasonality of psoroptic mange in camelids, but in sheep, it is most active during the fall and winter. Psoroptic mange is a reportable disease in the United States. One case involving a llama cria and his dam, has been reported in the United States, and only a few reports have come from South America. Identification to species was not possible in the case occurring in the United States.

Chorioptic Mange Mite

IDENTIFICATION. *C. bovis* closely resembles *Psoroptes* spp. except that the tarsal suckers are on short, unsegmented pedicels [3]. The capitulum is shorter and blunter than that of *Psoroptes* spp. (Figure 7.22). Female chorioptic mites are approximately 0.4 mm long, and males, 0.35 mm (Figure 7.23); larvae are 0.2 mm.

CLINICAL SIGNS. Chorioptic mites feed on epidermal debris on the skin surface. These mites have short chelicerae adapted for chewing, in contrast with the penetrating chelicerae of psoroptic mites. Lesions from the chorioptic mites are found on the ventrum of the tail, perineum (Figure 7.24), inner sides of both hind legs, and the ventral abdomen (Figure 7.25) [25]. The lesions can extend laterally up to more heavily fibered areas. Lesions can also be present in the axilla, again involving lightly fibered areas. Other sites include the tips and inner surfaces of the ears and between the digits, extending up to the fetlock. Mucocutaneous junctions of the face as well as the bridge of the nose are commonly affected. The basic lesion of chorioptic mange is similar to that of psoroptic mange.

Figure 7.22 Male *Chorioptes bovis* mite from a llama. Note the broad, shortened capitulum, adanal suckers, and bilobed abdomen.

Figure 7.23 Copulating adult male (left) and nymphal female *Chorioptes bovis* mites.

The skin is hyperemic, pruritic, thickened, and covered with incrustations 0.5–1.5 cm in diameter (Figure 7.26). Lesions slowly spread in size and can lead to dry, elephantine skin.

EPIDEMIOLOGY. Chorioptic mange is supposedly more ubiquitous and less debilitating to the host than psoroptic and sarcoptic mange.

Demodex Mite

Demodex mites have rarely caused a clinical manifestation in camelids but can be found within normal otic flora. Typical demodectic mange mites are 0.3–0.44 mm long with a cigar-shaped body. Clinical signs of infestation result in non-pruritic to mildly pruritic lesions that are pustular or nodular in appearance with alopecia that

Figure 7.24 Chorioptic mange on the perineal area of a llama.

Figure 7.25 Chorioptic mange on the ventrum of a llama.

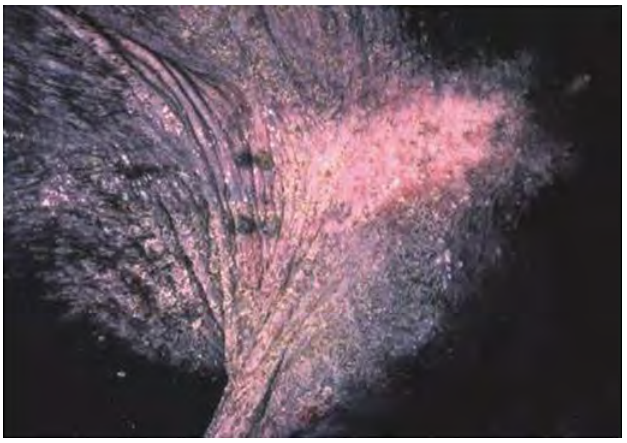

Figure 7.26 Encrustations typical of both psoroptic and chorioptic mange.

covers the face, neck, axillary and inguinal regions. As with other mite infestations, secondary bacterial infections may occur and create erythema, crusting, and excoriations [19].

Transmission

Direct contact is usually necessary for a new animal to acquire any of the mange mites. However, objects such as clothing, bedding, and grooming tools may serve as mechanical vectors. Dust bathing areas are also sources. Adult mites may survive off the host on epithelial debris for a maximum of 30 days. Eggs hatch in 4–10 days and larvae must feed within 10 days or they do not survive.

Bowman has shown that *Psoroptes* spp. and *C. bovis* are not host specific, and cross transfers are theoretically possible [3]. Sarcoptic mites are thought to be host specific. No cases of zoonosis from camelid sarcoptic mange have been reported, but it has been reported from camels in Australia.

Diagnosis

Any of the mange mites may be seen (but not identified) at 40× with either a dissecting or a compound microscope. At 100×, the mite will fill the field of view, and most of the identifying characteristics can be seen; however, the segmentation of the pedicel attached to the suckers in *Psoroptes* spp. can clearly be seen only at 400× magnification [3].

Lesion distribution for *Sarcoptes* and *Chorioptes* are similar but severity of the hyperkeratosis and pruritus experienced with *Sarcoptes* infections is more significant.

Scrapings for mange mite identification should be taken at the periphery of a lesion. When scraping lesions, it is necessary to penetrate deeply enough to draw blood especially for deep burrowing mites like *Sarcoptes* and *Demodex*. The debris should be scraped onto a slide for examination for adult mites or eggs. If no mites are observed, 10% potassium hydroxide (KOH) can be applied for 20 minutes to help clear debris to improve visualization. Superficial mites like *Chorioptes* can be diagnosed via superficial skin scrapes and rarely need KOH to clear debris. The most successful location for scraping for *Chorioptes* is between the toes even if the region clinically looks unaffected and void of any lesions. The feces should also be evaluated as dead mites may be present and indicate the animal is chewing due to pruritus. Histologic evaluation of biopsy samples of *Sarcoptes* lesions can show hyperkeratosis and epidermal hyperplasia, acute or chronic inflammation with neutrophilic, monocytic, or eosinophilic inflammation, and fibrosis.

Treatment

Regardless of the mite encountered, treatment regimens should be repeated every 7–14 days to account for the possible life stages on the host. Psoroptic and chorioptic mange respond readily to insecticide dusts, sprays, and dips, because mites live on the surface of the skin. Sarcoptic mange is more difficult to treat topically, but ivermectin (0.2 mg/kg) given subcutaneously has proven to be highly effective with two doses given 10 days apart clearing the infection within 24 days [26]. An alternative treatment includes topical amitraz solution. Environmental management is also encouraged for prevention of sarcoptic mange. Though part of the avermectin drug class, eprinomectin and doramectin products have not been shown to be effective treatments. Severe cases may require repeated treatment or an increase in the dose.

Specific treatment options for sarcoptic mange include subcutaneous injectable ivermectin, amitraz topical solutions, and environmental management. Though fipronil spray has not been reported in camelids for use against *Sarcoptes*, it may be effective and has been increasingly used as an acaricide in camelids. Fipronil is classified as a class C agent (possible carcinogen) by the US Environmental Protection Agency so caution must be taken by the applicator.

Chorioptic mange, on the other hand, responds more readily to topical treatments, while injectable ivermectin and eprinomectin are ineffective. Topical permethrin, topical lime sulfur (2%), and topical 2.5% fipronil spray (3 mL/kg) are good treatment alternatives. Several combinations of topical and injectable ivermectin, in conjunction with other medications, have been attempted and shown to have some efficacy in treatment. Dimethylsulfoxide (DMSO) (10 g) and water (60–90 mL) mixed with 1% injectable ivermectin (1 mL) applied weekly with concurrent 0.4 mg/kg ivermectin given subcutaneously has been shown to be successful at resolving lesions [27]. Additional treatments include weekly ivermectin (0.2 mg/kg subcutaneously) for four doses with a single topical application of fipronil (0.25% solution at 3 mL/kg) and environmental treatment resulting in the resolution of pruritus associated with mite infestations and improved fiber re-growth [28]. Eprinomectin at a dose five times that labeled for cattle (0.1 mg/kg) applied topically once a week for four weeks has been shown to reduce the number of mites infesting individual alpacas in a closed herd but was unable to completely eradicate the infestation [27].

Psoroptes infections within the ear canal can be treated with an injectable ivermectin formulation applied topically into the ear. Additional treatments for *Psoroptes* include intra-aural fipronil spray. Prior to the implementation of treatment, the ear should be cleaned to improve efficacy. Caution must also be taken to avoid use of neurotoxic

agents if the tympanic membrane is ruptured. Antibacterial agents should also be considered if a secondary bacterial infection is present.

Lesions associated with demodectic mange have been successfully treated with initial clipping and scrubbing of the affected areas with 2% povidone iodine in conjunction with amitraz (5–10 mL/L of 5% solution) for repeated treatments over an extended period of time.

Internal Parasites

Protozoa

Protozoal diseases (Table 7.2) are important throughout the world. *Mycoplasma haemolamae* was first classified as protozoa (an Eperythrozoan) but has since been reclassified as a haemotrophic bacteria [29]. Currently, the biologic or mechanical vector(s) responsible for transmission of this *M. haemolamae* are unknown; however, it has been hypothesized that biting insects transmit this agent in some herds. Experimentally, transfusion of *M. haemolamae*-infected blood was found to transmit this organism [30]. In that study, animals did not test positive by polymerase chain reaction (PCR) until at least four days post-transfusion, indicating that the timing of sample acquisition may be important for detecting and studying neonatal infection [30, 31]. *In utero* transmission is documented but rare [32]. Oxytetracycline is used to treat clinical cases associated with anemia along with supportive treatment for the hyperglycemia commonly associated with the acute clinical disease of *M. haemolamae*.

Trypanosomiasis (Surra)

Identification. Old World camels are highly susceptible to infection by several species of *Trypanosoma* [33]. Camels are not common in the region called the Tsetse Belt; illness and death caused by *Trypanosoma brucei*, known as "nagana," may be the key factor of their absence from the area. The Sudan is home to over four million camels, and *Trypanosoma evansi* is the major trypanosome affecting camels in this region, causing a disease called "Guffar." *Trypanosoma vivax* is the second leading cause of trypanosomiasis in the Sudan after *T. evansi* [34].

Trypanosoma species are morphologically similar and species identification may be impossible without specific diagnostic procedures. Unfortunately, clinical signs are not pathognomonic, parasitologic tests are not sufficiently sensitive, serologic tests have low sensitivity or specificity, and molecular tests cannot distinguish the taxa within the *Trypanosoma* subgenus [35]. Within a blood smear, the organisms have a leaf-like shape with a single flagellum attached to the cell by an undulating membrane (Figure 7.27) [3].

Life Cycle. *Trypanosoma brucei evansi* does not require a period of maturation in an insect vector and, thus, has a noncyclic transmission [36]. Any biting or blood-sucking insect or tick may serve as a vector. Flies of the genera

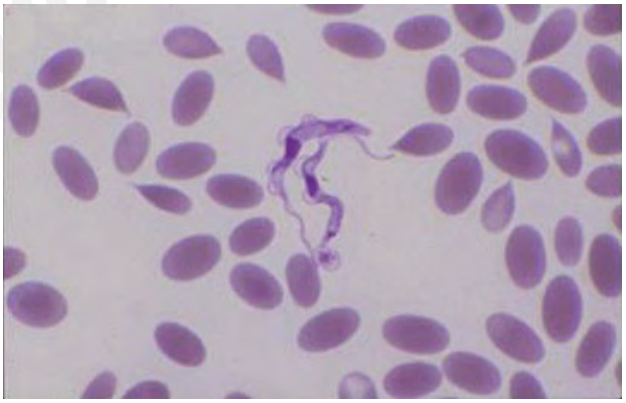

Figure 7.27 *Trypanasoma evansi* in a camel.

Table 7.2 Protozoan parasites of camelids [3, 80].

Protozoa				
Scientific name	**Common name**	**Diagnostic stage**	**Anatomic location – adult**	**Intermediate host**
Eimeria spp.	Coccidia	Oocyst – feces	Small intestine	None
Cryptosporidium spp.		Oocyst – feces	Distal small intestine, large intestine	None
Trypanosoma spp.	Surra	Trypanosome – blood	Blood, lymph, CSF	None
Sarcocystis spp.	Sarcocyst	Sarcocyst – muscle Oocyst – feces (DH)	Muscle	SAC
Toxoplasma gondii	Toxoplasma	Cyst – muscle Oocyst – feces (DH)	Multiple organs	SAC

Tabanus and *Stomoxys* are commonly implicated. Iatrogenic mechanical transmission by contaminated hypodermic needles is also possible. In *Trypanosoma* species that do not require an intermediate host, the trypanosomes are deposited in the blood by bloodsucking insects. In the camelid host, the organisms multiply by longitudinal binary fission in blood, lymph, and cerebrospinal fluid. When that fly takes another blood meal, it becomes a mechanical vector.

T. brucei and *T. congolensis* are the primary causes of human sleeping sickness and are transmitted by the tsetse fly (*Glossinia* spp.), which serves as an intermediate host. Old world camelids (OWC) may also be infected with these organisms. When the tsetse fly takes a blood meal from an infected animal, the organisms multiply within the hind gut of the fly. There, they undergo a metamorphosis and migrate to the salivary glands and reach an infective stage that is then transmitted when the fly takes another blood meal.

EPIDEMIOLOGY. Trypanosomiasis (surra, sleeping sickness, Chagas disease, nagana) is a blood parasitism affecting humans and numerous species of mammals including camelids. *T.b. evansi* is the primary species affecting OWC and is widespread throughout North Africa, Asia Minor, countries of the former USSR, Pakistan, Afghanistan, India, Burma, Malaya, Indochina, South China, Indonesia, and the Philippines [36]. Trypanosomiasis has also been reported from Central and South America in humans, though trypanosomiasis has not been reported in South American camels (SACs) in these areas despite the presence of the organism. Trypanosomiasis caused by *Trypanosoma cruzi* is an important disease of humans and animals in the New World. *T. cruzi* is transmitted by Reduviidae (kissing bugs) via excreted parasite stages in the arthropods' feces that make their way into a new host via small skin lesions or damaged mucous membranes; animals may acquire the infection by ingestion or crushing infected vectors [36]. Serologic studies of *T. cruzi* infection in the camelids *Llama glama* and *Vicugna pacos* have shown evidence of infection [37].

CLINICAL SIGNS. Acute surra is characterized by fever, progressive anemia, depression, weakness, ventral edema, and paralysis (Figure 7.28). The presence of pulmonary edema may contribute to the development of secondary pneumonia. Females may abort and the milk of lactating females may become caseous. Large numbers of trypanosomes are seen in peripheral blood samples. Death may occur within a few days. The subacute form is characterized by fever, edematous plaques, emaciation, high mortality, and death within a few weeks.

Figure 7.28 A camel ill with trypanosomiasis. Weakness, weight loss and increased time spent recumbent are features of acute and chronic trypanosomiasis.

Chronic surra is characterized by intermittent episodes of fever, anemia, ventral edema, and emaciation. Between episodes of fever, the parasite may be absent from peripheral blood vessels. A camel may live for three or four years, depending on the care provided.

DIAGNOSIS. Finding the organism in the peripheral blood of infected camels may be highly problematic because of the low numbers of the organisms and fluctuating parasitemia. The development of a suitable enzyme-linked immunosorbent assay (ELISA) serologic test has been found to be effective in decreasing the economic losses associated with surra. Other DNA technologies are being used in endemic countries.

Toxoplasmosis

Toxoplasma gondii is a ubiquitous protozoan parasite [38–41]. Camels acquire *T. gondii* infections through ingestion of sporulated oocysts shed by cats or wild felids in the environment. Antibodies against *T. gondii* in sera of dromedaries from different countries have been determined using various techniques, reporting seroprevalences as high as 67% [39, 42]. It has been estimated that 36% of camels in Africa have anti-*T. gondii* antibodies [43]. Abortion has been diagnosed in SACs and serum titers have been reported [39, 41, 44].

Neospora caninum is similar to *T. gondii* except *N. caninum* uses canids as the definitive host. Investigation of this parasite in camelids is limited [44–46]. *In utero* infections or abortions can result from this parasite, as in other species.

Coccidiosis

IDENTIFICATION. Like most ruminants, camelids are susceptible to coccidiosis [47, 48]. The species of *Eimeria* that parasitize camelids are unique to camelids and are not

transferable to livestock species or vice versa. Likewise, the species of *Cryptosporidium* and *Sarcocystis* are unique to camelids [49, 50]. Other species of protozoan parasites may be shared by the other species of farm animals. Six species of the genus *Eimeria* have been described from camelids (Table 7.3) [51, 52]. Five species have been identified by fecal flotation in llamas from Oregon, Wyoming, Colorado, Washington, Iowa, and California, so llamas and alpacas are hosts to the same six species of *Eimeria* [1, 53, 54]. *Eimeria* oocysts are frequently encountered on routine fecal flotations and the oocysts are morphologically distinct so identification can be made without the need of sporulation (Figure 7.34) [52, 55]. Coccidia identified from camels are listed in Table 7.4.

LIFE CYCLE. The life cycle of coccidia includes both sexual and asexual phases (Figure 7.29). The asexual cycle is called "schizogony" or "merogony." Sporulated oocysts are ingested by the animal and pass along the digestive tract to the small intestine. The oocyst frees sporozoites, which invade the epithelial cells of the intestinal mucosa. The sporozoite changes shape and becomes a trophozoite, which, in turn, grows larger and forms a schizont. Within the schizont, merozoites form and ultimately rupture the host cell and escape to infect other cells. This process may be repeated two or three times.

The sexual cycle is called "gametogony." The merozoites produced by the last schizogony cycle infect a cell and develop into either male (microgametocyte) or female (macrogametocyte) gamonts. The male gamont fertilizes the female gamont while it is still in the host cell, producing a zygote. The zygote matures to become an oocyst, which ruptures from the host cell and is shed in the feces. The oocyst sporulates in the environment in one to two days to become infective.

EPIDEMIOLOGY. Coccidial infections are generally self-limiting because asexual reproduction is repeated only two or three times [1]. Unless reinfection takes place, only one

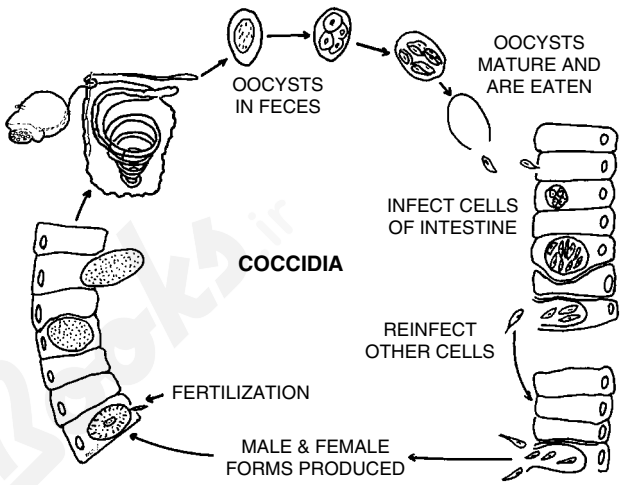

Figure 7.29 Life cycle of *Eimeria* spp.

Table 7.3 Coccidia of South American camelids.

Species	Size of oocyst (µm)	Shape of oocyst	Micropyle
Eimeria alpacae	22–26 × 18–21	Ellipsoidal	+
E. lamae	30–40 × 21–30	Ovoid to ellipsoidal	+
E. macusaniensis	81–107 × 61–80	Ovoid	+
E. punoensis	17–22 × 14–18	Ellipsoidal or oval	+
E. peruviana	28–37 × 18–22	Ovoid	–
E. ivitaensis	84–99 × 49–59	Ovoid	+ micropylar cap may not be visible in the histological sections

Source: Soulsby [1] and Dubey 2018.

Table 7.4 Coccidia of camels.

Species	Size of oocyst (µm)	Shape of oocyst	Micropyle	Sporulation time (days)
Eimeria bactriani	32 × 25–27	Spherical-ellipsoidal	+	10
E. cameli	81–100 × 63–94	Truncate	+	10–15
E. dromedarii	22–33 × 20–25	Ovoid	Micropylar cap	15–17
E. pellerdyi	23.2 × 12.6	Ovoid or ellipsoidal	–	5

cycle of development can occur, but in a contaminated environment, re-infection is the rule, and heavy build-ups can occur that may kill the host. Knowledge of this concept is important to the clinician, who must manage coccidiosis.

Ruminant livestock develop immunity to coccidia but may shed low numbers throughout their life. Whether or not this occurs in camelids is unknown. It is common to see *Eimeria* oocysts on fecal flotation, but coccidiosis with scours is uncommon in camelids in North America. However, it is a serious problem of recently weaned llamas and alpacas in Peru. The exception is with *Eimeria macusaniensis*, commonly referred to as *E. mac,* which can be lethal in adults.

Clinical Signs. Coccidia invade the epithelial mucosa of the small intestine, causing enteritis and diarrhea. The diarrhea may be mild and catarrhal in nature or more serious and hemorrhagic [53, 56, 57]. Adult alpacas have developed fatal coccidiosis within five weeks after transportation to a new farm [58, 59].
Treatment. Numerous coccidiostats are used in ruminants. Their use in camelids in North America has been recommended [19]. Ponazuril formulated for oral treatment of Equine Protozoal Myeloencephalitis (EPM) in horses demonstrated potentially effective concentrations to treat Apicomplexa when administered to llamas [60]. Ponazuril is the drug of choice to treat *E. macusaniensis*. Monensin and other ionophores should not be used because there is species sensitivity to these drugs. For example, 1000 alpacas were unknowingly fed a poultry feed rate of 66 ppm of salinomycin into their ration for two days. Acute deaths were noted with 5–17 deaths per day from day 3 through 15 [61].

Cryptosporidiosis

Identification. *Cryptosporidium parvum* and *C*. spp. are coccidial protozoan parasites that cause cryptosporidiosis and are of great concern to livestock managers and veterinarians [49, 54, 62]. The organism inhabits the distal small and large intestine. *Cryptosporidium ubiquitum*, *C. parvum*, and *Cryptosporidium cuniculus* have been identified in camelids and all three are zoonotic [63].
Life Cycle. The life cycle is similar to *Eimeria*, however the small oocysts (4–4.5 μm) sporulate within the host and thus are infective to other animals immediately upon passage in the feces. Autoinfection is possible. Transmission is via contaminated feed and water.
Epidemiology. Both OWC and New World camelids (NWC) have been infected. Bactrian camels seem to be more susceptible than dromedaries. Predisposing factors include failure of passive transfer of immunoglobulins and concurrent infection. Historically, *C. parvum* has been the most frequently recorded species of *Cryptosporidium* in molecular surveys of alpacas in the USA [50] and the UK [64, 65], and both *C. parvum* and *C. ubiquitum* have been detected in Peru [66].
Clinical Signs. Signs associated with cryptosporidiosis are those of gastroenteritis. Severe diarrhea, tenesmus, dehydration, and emaciation may lead to death. However, in one multi-farm study with 110 dam-cria pairs, 8% of alpaca dams and 7% of crias had detectable oocysts in their feces yet remained asymptomatic [67].
Diagnosis. Oocysts may be seen in smears made of fresh feces. Modified Ziehl-Nielsen stain is commonly used with counterstaining with carbo-fuchsin. At necropsy, there is villous atrophy leading to malabsorption. In crias and camel calves with severe diarrhea, other enteric pathogens must be considered, including *Clostridium perfringens* (enterotoxemias), *Escherichia coli*, *Salmonella* spp., *Coccidia* spp., coronavirus, rotavirus, and bovine virus diarrhea virus [68]. Cryptosporidiosis may also present as co-infections with other pathogens.
Management. Dehydration and acidosis are the main consequences of severe diarrhea. Both must be dealt with by rehydration with IV fluids and appropriate administration of sodium bicarbonate. *Cryptosporidium* oocysts are resistant to all commonly available, commercial antimicrobial agents and most disinfectants. Oocysts may persist in the environment for months to years.

Sarcocystosis

Identification. *Sarcocystis* spp. have an interesting two-host life cycle. *Sarcocystis* is a genus of coccidia, closely related to the *Eimeria*. The definitive host is a carnivore, in which gametogony, fertilization, and sporulation occur. Camelids serve as an intermediate host in which only schizogony and encystment occur. Both microcysts and macrocysts, indicating different species, can be found in camelids. Various names have been proposed to describe sarcocysts in SACs, resulting in considerable confusion regarding the nomenclature of *Sarcocystis* spp. infecting alpacas, guanacos, and llamas. Based on the current morphologic and molecular evidence, only two valid species of *Sarcocystis*, *Sarcocystis aucheniae* and *Sarcocystis masoni*, infect SACs that form macroscopic and microscopic sarcocysts, respectively [69].

EPM has only been recognized as an equine disease caused primarily by *Sarcocystis neurona*. The horse is a dead-end host for this parasite that normally cycles between wild mammals and opossums in the United States.

There are no clinical case reports that suggest *S. neurona* causes clinical disease in camelids.

LIFE CYCLE. Life cycles for all camelid *Sarcocystis* spp. have not been fully elucidated. Whether the dog, fox, cat, or human serves as the primary host is unknown. In general, the carnivore ingests the muscle of the herbivorous intermediate host (Figure 7.30). Zoites are released in the intestine and invade the epithelial cells. Gametogony is completed in the wall of the intestine of the carnivore up to and including the sporulated oocysts, which are excreted in the feces and are immediately infectious.

The camelid ingests the sporocyst; sporozoites are released in the intestine and invade vascular endothelial cells. One or more asexual cycles are completed in these cells before the merozoites enter muscle cells and form sarcocysts.

EPIDEMIOLOGY. Sarcocysts have been encountered as a secondary finding at necropsy in North American llamas. Clinical disease has been reported in an alpaca exported from Peru to the USA [70]. Location and feeding practices have been found to affect the prevalence of sarcocystosis in SAC anywhere from 23 to 100% [69]. Meat is downgraded as a result of the infection.

A carnivore or omnivore must serve as the definitive host, passing the sporulated oocysts with the feces. The camelid serves as the obligatory intermediate host.

CLINICAL SIGNS. Little or no damage is done to the definitive host. No signs may be noted in light infections in camelids. In high numbers, the schizogonous cycles in endothelial cells may produce acute febrile disease, resulting in abortion and death. Sarcocysts may cause mild myositis with myalgia and interference with muscle function.

TREATMENT. No treatment is available for the muscular form. Prevention requires disruption of the life cycle from the carnivore host.

Giardiasis

Giardia spp. cysts have been detected in immature, poorly conditioned llamas with soft stool or diarrhea [54, 71]. In one Australian study, six of 81 samples tested positive for *Giardia* [63]. In one US study examining potential pathogens in the feces of unweaned llamas and alpaca with diarrhea, *Giardia* was detected in 18% of the animals tested [72].

Trematodes

See Table 7.5

Fascioliasis

IDENTIFICATION. The common liver fluke, *Fasciola hepatica*, is locally ubiquitous and found throughout tropical and temperate regions of the world and will infect camelids [73, 74]. This fluke is leaf-shaped and reaches a maximum size of 30 × 13 mm. *Fascioloides magna* (large American fluke) is a parasite of deer in North America and immature specimens of this fluke have been found in hepatic cysts in a llama [75].

LIFE CYCLE. Adult *F. hepatica* live in the bile ducts (Figure 7.31). Eggs are discharged into the bile duct, carried to the intestine, and excreted in the feces (Figure 7.34) [3]. The eggs must fall into water for maturation to the ciliated miracidium stage to take place. This takes 10–12 days. The miracidium bores into one of many species of snail. *Lymnaea truncatula* is one of the more common intermediate hosts, but other species of *Lymnaea* may also act as hosts. While in the snail, the miracidium loses its cilia and

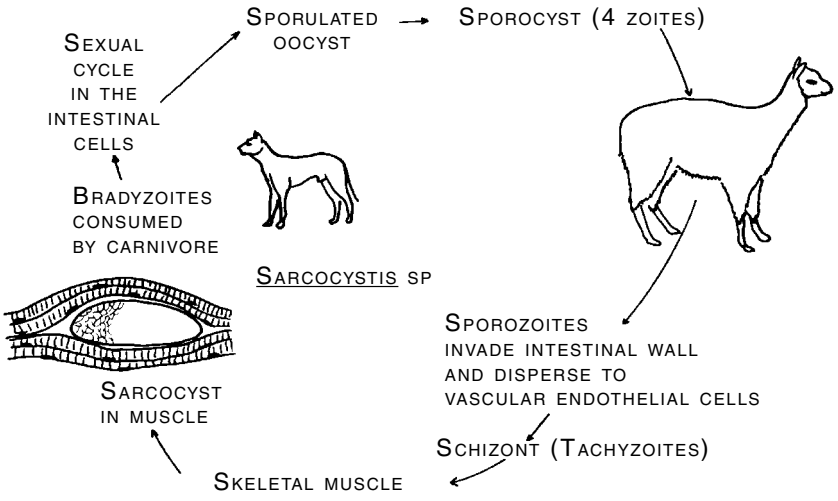

Figure 7.30 Life cycle of *Sarcocystis* spp.

Table 7.5 Trematode and cestode parasites of camelids [3, 80].

Scientific name	Common name	Size (mm)		Anatomic location		Intermediate host
		Adults	Egg (µm)	Adult	Immature	
Trematodes						
Fasciola hepatica	Liver fluke	13–30	130–15 × 63–90	Bile ducts	Small intestine, peritoneum, liver	Snail, *Lymnaea* sp.
Dicrocoelium dendriticum	Liver fluke	8–12	38-45x 22–30	Bile duct & gall bladder	Small intestine, peritoneum, liver	Land snail (1st IH), Ant (*Formica*) (2nd IH)
Cestodes						
Echinococcus granulosus	Hydatid	2–7	25–40 diameter	Intestine of carnivores	Lungs, liver	SAC (primary host is dog)
Moniezia expansa	Tapeworm	600 cm × 1.6 cm wide	65–75 diameter	Small intestine	Small intestine	Oribatid mite
Thysaniezia spp.	Tapeworm	200 cm × 1.2 cm wide		Small intestine	Small intestine	?

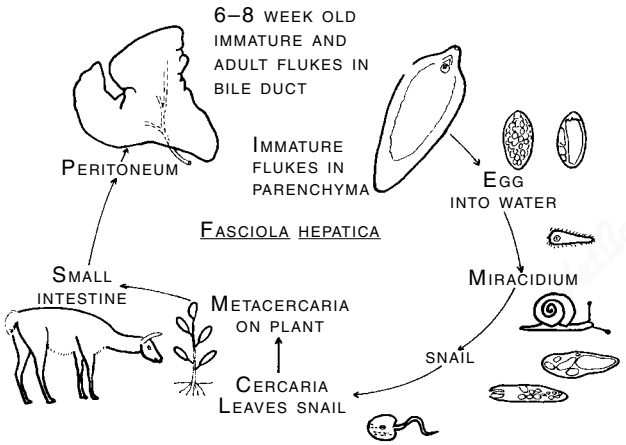

Figure 7.31 Life cycle of *Fasciola hepatica*.

matures to become a sporocyst, then a redia, and finally a cercaria. This phase requires four and a half to seven weeks.

The cercaria leaves the snail and is free swimming for a few minutes to two hours, attaches to a plant just below surface level, loses its tail, and becomes a metacercaria, which is the infective stage for the camelid. Metacercaria are ingested and immature flukes (marita) are released into the duodenum; they penetrate the wall of the intestine, enter the peritoneal cavity, and migrate to the liver. By three to seven days after infection, most of the young flukes will have reached the liver, where they penetrate the capsule. Following this, a migratory period of some five to six weeks in the liver parenchyma occurs before the flukes enter the bile duct and mature.

The prepatent period is about eight weeks, but development may be retarded, delaying maturity another two months. Adult flukes may live for nine months or longer.

EPIDEMIOLOGY. The completion of the *F. hepatica* life cycle depends on the presence of snail intermediate hosts, which, in turn, depend on an aquatic environment. The eggs will not develop at temperatures below 10 °C and there is a direct correlation of development time with temperature (at 13 °C, 60 days; at 15 °C, 40 days; and at 26 °C, 12 days).

Some snail hosts are capable of estivation for as long as 13 months under certain circumstances, e.g. during drought conditions. The infection may be maintained while the snail is buried in the dried mud.

Metacercaria can persist for a few days to a few weeks while encysted on the plant. Some of the cysts may fall to the bottom of the water and be stirred up and ingested when an animal walks into the water to graze or drink. The cyst may survive for as long as eight months on moist hay, but the usual drying process shortens life to a few weeks.

F. hepatica infection in camelids has been reported from South America [76] and occurs in localized problem areas in the western and southern United States [73, 75, 77]. If flukes are endemic in local cattle and sheep, and llamas or alpacas inhabit similar swampy, poorly drained pastures, it is reasonable to assume that the camelids will also have a fluke problem.

Dicrocoelium dendriticum is another trematode that will impact camelids with a similar but distinctive life cycle from *F. hepatica* [36]. This fluke lives in the bile ducts and gall bladder of the definitive host. Eggs pass through the bile ducts to the intestine and are excreted with the feces. There are two intermediate hosts. The first intermediate host is shell-bearing land snails which allows the parasite to undergo asexual replication and amplification. The land snails release the cercariae stage (up to 6000) encased within a snail slime ball. This stage survives only a few days unless consumed by

the various species of ants, mainly of the genus *Formica*. The parasite migrates within the ant including to the ant's brain. It takes at least 40 days at 25 °C for the *D. dendriticum* to reach an infectious stage in the ant. Naturally infected ants can contain 100–400 infective metacercariae. The presence of *D. dendriticum* in the ant's brain causes the ant to change its biologic behavior. The ant migrates and remains at the tips of plants, increasing the chances that it will be ingested by grazing animals. Once ingested by a susceptible definitive host, the metacercariae make their way from the small intestine to the liver bile ducts. It takes approximately seven weeks for sexual maturity to the adult stage with production of eggs. Adult *D. dendriticum* live 1.5–6 years.

D. dendriticum is regionally endemic in North America, South America, Europe, North Africa, and Asia. Distribution is dependent upon habitat conditions and the presence of the appropriate hosts. A variety of herbivores from alpacas to cottontail rabbits can serve as a definitive host and shed eggs, contaminating pastures.

Clinical Signs. Both acute and chronic forms of fluke-associated disease have been seen. The acute form occurs with overwhelming infections that produce signs of hepatic insufficiency similar to those caused by other agents. The chronic form is more often seen.

Chronic stasis of the bile, caused by *F. hepatica* obstructing the ducts, produces a hepatic fibrosis, which ultimately causes an elevation of intrahepatic blood pressure. A hyperplastic cholangitis, which allows leakage of plasma protein into the peritoneum, causing hypoproteinemia, also occurs. Adult flukes suck blood, causing intra-biliary hemorrhage, which results in anemia.

D. dendriticum induces similar liver lesions as *Fasciola* in NWCs, including abscesses, granulomas, and fibrosis, as well as bile duct proliferation [78]. In contrast to finding *Fasciola* eggs within the affected bile ducts, *D. dendriticum* eggs are found in the liver parenchyma [78].

The liver fluke affected NWCs becomes anorectic. Mucous membranes may be pale, and ventral edema may be seen. The fiber becomes dry and brittle, with "breaks" associated with intense periods of the disease. Depression and emaciation follow anorexia. Either diarrhea or constipation may be seen.

Treatment. Many different drugs have been used to treat fluke infestation. Currently, the only recommended drug in the United States is Clorsulon (Curatrem) at a dose of 7 mg/kg body weight. This is given per os, twice at 45- to 60-day intervals. Triclabendazole is also effective [36]. In Switzerland and Southern Germany, praziquantel has been described as being effective against *D. dendriticum* infestation, with treatment success of over 98% when used at a dosage of 50 mg/kg [78].

The unhealthy, fluke-infested liver may be susceptible to opportunistic secondary bacterial infection from clostridial organisms or other normal enteric bacteria. One fluke-associated disease of concern to livestock is Black's disease, caused by *Clostridium novyi* Type D, or *Clostridium hemolyticum*. Vaccination with a multiple-antigen clostridial toxoid may prevent development of these diseases.

Cestodes

See Table 7.

Hydatid Disease

Identification. Hydatid disease, caused by *Echinococcus granulosus*, is worldwide in distribution. The adult tapeworm resides in the intestine of a carnivore, with dogs, foxes, coyotes, and wolves all serving as potential definitive hosts. This is a small tapeworm, 2–7 mm long, consisting of only three or four proglottids, which usually disintegrate in the intestine, so only eggs are seen in the feces.

Life Cycle [1]. The feces of the carnivore host contaminate the feed of the camelid (Figure 7.32). The eggs are immediately infective to any of the many ungulate intermediate hosts, including camelids. The eggs hatch in the intestine, releasing oncospheres which penetrate the intestinal venules or lymphatics and migrate to the liver or lung (species dependent).

The hydatid cyst (metacestode) (Figure 7.33a and b) develops slowly over several months, reaching a diameter of 8–10 cm. The inner membrane of the cyst or bladder is composed of germinal epithelium and is surrounded by fluid enclosed in a capsule of inflammatory cells. Brood capsules, each with their own scolex, may develop within the outer capsule. Some of the brood capsules rupture, releasing scolices into the fluid ("hydatid sand"). The fluid is infective if the outer capsule is ruptured.

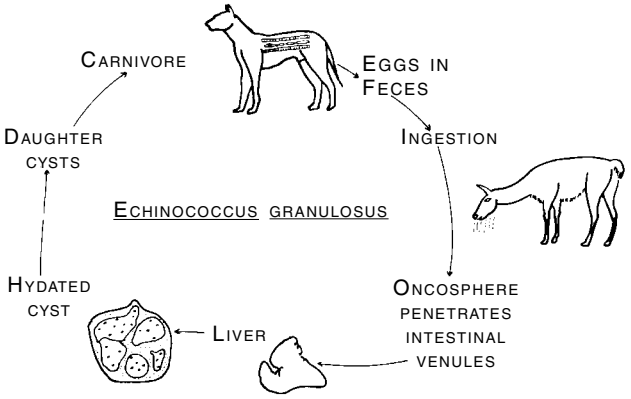

Figure 7.32 Life cycle of *Echinococcus granulosus*.

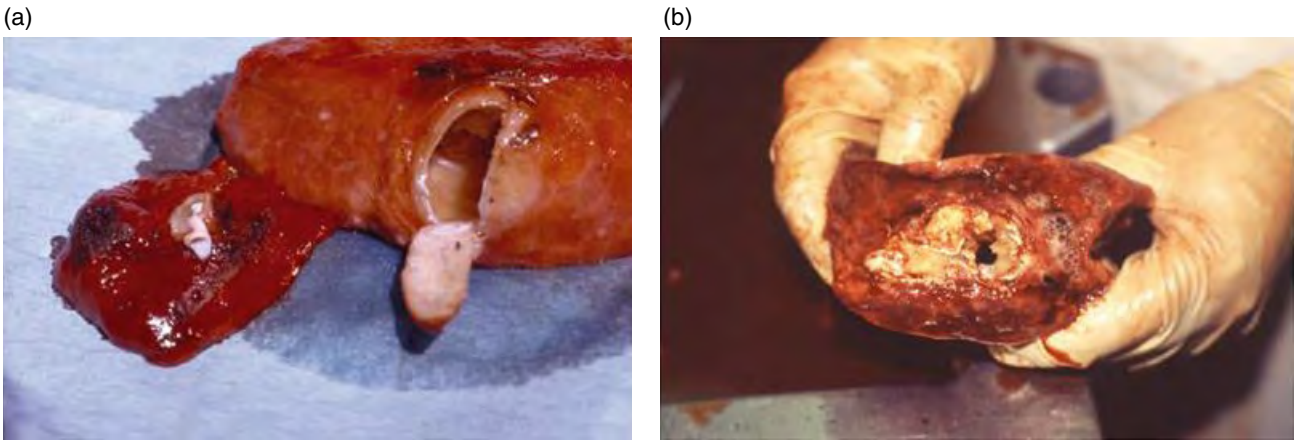

Figure 7.33 (a) Lesion of *E. granulosus* in the lung of a camel. (b) Lesion of *E. granulosus* in a llama.

The hydatid cyst is consumed by the carnivore when it scavenges a carcass or is fed offal. The protoscolices are released in the intestine, evaginate, and penetrate between the villi into the crypts of Lieberkühn, developing to maturity in about 47 days. Dogs may maintain an infection for two years.

EPIDEMIOLOGY. Only one species of *E. granulosus* has been identified, but in the field, various strains (based on molecular methods) or cycles (pastoral and sylvatic) exist between particular intermediate and definitive host species (e.g. sheep/dog, cattle/dog, camel/dog, wallaby/dingo [Australia], deer/coyote [California], and moose/wolf). The cycle in camelids in South America is camelid/dog or camelid/fox.

CLINICAL SIGNS. The adult tapeworm is usually harmless to the carnivore, and, except under unusual circumstances, a mild infestation in a herbivore produces no clinical signs. Cysts usually develop in the liver or lungs, but in humans and other hosts, they may also develop in the brain or heart. In any case, malfunction of any organ will occur if large numbers of cysts develop or if a cyst develops in a vital area. If the outer wall of a brood cyst ruptures, metastasis may occur, with numerous new cysts developing as a result.

TREATMENT. No treatment can destroy the hydatid cyst in the intermediate host. Cysts can be removed surgically after the cyst has been injected with formalin to obviate accidental rupture of the cyst and subsequent contamination of the surgical site. Prevention consists of ridding the carnivore of the tapeworm. This may be successful in a controlled situation involving a pet dog cycle but is not possible in sylvatic cycles or areas where feral dogs roam.

Monieziasis

IDENTIFICATION. *Moniezia expansa* has been recovered from camelids in the United States, and *M. expansa* and *Moniezia benedeni* are significant problems in some areas of South America. Adult tapeworms have been found in the small intestine of camelids. An intact tapeworm may reach 600 cm in length and be 1.6 cm wide. The scolex is 0.36–0.8 mm and has prominent suckers and no hooks [1].

LIFE CYCLE. Mature proglottids separate from the rest of the tapeworm and, along with the contained eggs (Figure 7.34), are passed with the feces. Cysticercoid

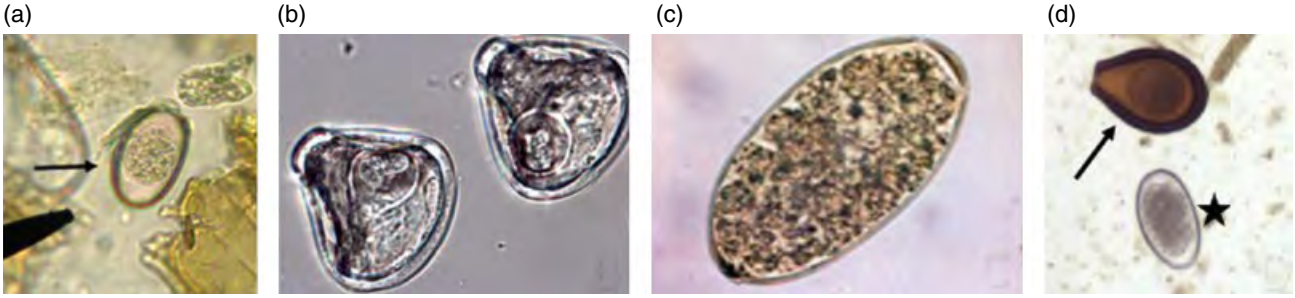

Figure 7.34 Appearance of miscellaneous parasite ova on fecal flotation: (a) *Eimeria* spp. (arrow) (b) *Moniezia* spp. (c) *Fasciola hepatica* (d) *Eimeria macusaniensis* (arrow), trichostrongyle (star).

development occurs in oribatid mites, which are a true intermediate host. Infective stages develop in approximately four months.

Camelids acquire the infection by consuming forage contaminated with the mites. Further maturation to the adult tapeworm in the small intestine takes 37–40 days.

EPIDEMIOLOGY. *Moniezia* occurs worldwide but is usually not a major disease-producing parasite. Heavy infestations could obviously impair nutrition and cause debility or even intestinal obstruction. There may be a seasonality of infestation in temperate climates because of the overwintering of the oribatid mites. Neonate camelids begin to nibble on grass early in life and could acquire an infestation and be passing proglottids by the age of six weeks.

CLINICAL SIGNS. Diarrhea and unthriftiness may accompany heavy infestations.

TREATMENT. Praziquantel (Droncit) at a dose of 2.5–10 mg/kg given orally or as an injection is probably effective. Fenbendazole at 10–15 mg/kg is effective in cattle and sheep and may be effective in camelids.

Thysanieziasis

Tapeworms of *Thysaniezia* spp. have been reported in llamas. Little is known about the life cycle or epidemiology. Adult tapeworms of the genus have been found in the small intestine of sheep, goats, and cattle. Little or no clinical significance seems to be associated with this parasite.

Taenia helicometra, T. hidatigena, T. omissa

Infections with larval (metacestode) stages of these *Taenia* spp. have been reported in alpacas and vicuñas in South America [3, 79].

Nematodes

See Table 7.6

Gastrointestinal Nematodes

Nematodes comprise some of the most numerous and detrimental parasites that infect camelids, leading to decreased production and economic impacts to the owner [9, 84, 85]. Gastrointestinal nematodes (GINs)

Table 7.6 Nematode parasites of camelids [3, 80].

Nematodes - GI					
		Size (mm)		Anatomic location of adults	
Scientific name	Common name	Adult	Egg (μm)		Appearance on fecal flotation
Gongylonema spp.	Cattle gullet worm	30–145 × 150–500 μm	50–70 × 25–37	Esophagus	Larvated egg; adult found in esophagus on endoscopy or necropsy
Camelostrongylus mentulatus	Stomach worm	6.5–9.27.5	65–100 × 34–50	C3	Egg (strongyle-type)
Marshallagia marshalli	Thread-necked worm	10–20	175–260 × 106–110	C3	Egg (similar to *Nematodirus*)
Ostertagia ostertagi	Medium brown stomach worm	6.5–9.2	65–100 × 34–50	C3	Egg (strongyle-type)
Haemonchus contortus	Large stomach worm	10–30	65–100 × 34–50	C3	Egg (strongyle-type)
Teladorsagia circumcincta	Brown stomach worm	6–10	65–100 × 34–50	C3	Egg (strongyle-type)
Trichostrongylus spp.	Stomach worm	4–7	65–100 × 34–50	C3, small intestine	Egg (strongyle-type)
Graphinema aucheniae	Stomach worm	5.5–12	65–100 × 34–50	C3	Egg (strongyle-type)

Table 7.6 (Continued)

Nematodes - GI						
		Size (mm)		Anatomic location of adults		
Scientific name	Common name	Adult	Egg (µm)		Appearance on fecal flotation	
Spiculopteragia peruvianus	Stomach worm	6.7–10.3	65–100 × 34–50	C3	Egg (strongyle-type)	
Cooperia spp.		4.5–6.2	65–100 × 34–50	Small intestine	Egg (strongyle-type)	
Nematodirus spp.	Thread- necked worm	10–23	175–260 × 106–110	Small intestine	Egg (very large strongyle-type)	
Strongyloides spp.		3.5–10	40–60 × 32–40	Small intestine	Larvated egg	
Lamanema chavezi		10–18	65–100 × 34–50	Small intestine	Egg (strongyle-type)	
Capillaria (Aonchotheca) spp.		8–20	45–50 × 22–25	Small intestine	Eggs with bipolar plugs (smaller than *Trichuris*)	
Bunostomum spp.	Hookworm	12–26	79–97 × 47–50	Small intestine	Egg (similar to trichostrongyle-type)	
Oesophagostomum spp.	Nodular worm	12–21.5	73–89 × 34–45	Cecum/colon	Egg (strongyle-type)	
Skrjabinema ovis	Pinworm	3–10 × 110–500 µm	47–63 × 27–36	Colon, rectum	Eggs on perianal skin	
Trichuris spp.	Whipworm	35–80	70–80 × 30–42	Cecum, colon	Eggs with bipolar plugs	
Nematodes – non-GI						**Intermediate host**
Parelaphostrongylus tenuis	Meningeal worm	39–91	Infection in camelids not patent	Larvae - spinal cord and brain	Larvae in central nervous system found on necropsy	Snail, slug
Thelazia californiensis	Eye worm	1–2 cm		Conjunctival sac, lacrimal duct	Larvae or adults in conjunctiva or lacrimal duct	Muscid flies
Dictyocaulus viviparus	Lung worm	40–80	82–88 × 33–38; L1 300–580	Bronchi	+/– larvated eggs in feces or tracheal samples; L1 on Baermann test	None

encompass a majority of nematode species and share similar life cycles and clinical signs (Figure 7.35). Virtually all camelids will become infected with GINs in their lifetime, but infection does not always correlate to clinical disease. Efforts should be made to identify populations of GINs present in individual herds, which can vary by geographic region, local climate, and historical deworming practices [86–90]. Once investigative diagnostics identify patterns of GINs in a herd, an integrated parasite control program should be implemented.

Etiology and Life Cycle

A large proportion of GINs are from the trichostrongyle ("strongyle-type") family and cannot be distinguished on a fecal flotation without additional investigative diagnostics. Since most GINs are trichostrongyles, they share a similar life cycle, and thus, similar control and prevention programs. Predominant GIN species in camelids can be organized by the segment of GI tract where the parasite can be found. Non-strongyle nematodes causing disease in the GI tract will be described in their respective sections.

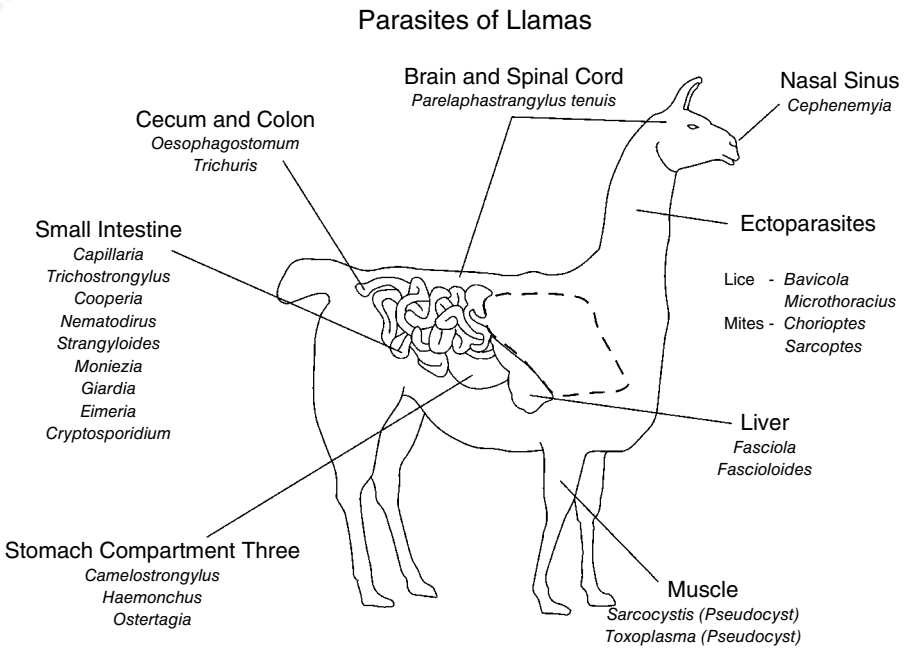

Figure 7.35 Predilection sites of common parasites in camelids.

The generic life cycle of a strongyle-type GIN begins with production of eggs by the female in the host GI tract. Female parasite fecundity varies by nematode species, with some highly fecund species (*Haemonchus contortus*) laying up to 10 000 eggs/female/day [91]. Eggs are passed in the feces of the host into the environment, develop into first stage larvae (L1) and hatch within the feces. Under ideal conditions, hatching occurs in one to two days. Ideal conditions for trichostrongyle eggs are generally between 10 °C–36 °C (50–96 °F), with individual species preferences [92]. Northern climates may not support the accumulation of clinically significant levels of trichostrongyle parasites on pasture. Moisture is as critical for larval development as temperature. For this reason, humid climates favor rapid development of trichostrongyles, though arid climates, which rely on irrigation, can become important [92]. The free-living L1 larvae feed on microorganisms in the feces and molt to second stage larvae (L2) and again to third stage larvae (L3), which is the infective stage, taking four to six days. L3s migrate out of the feces in about one week and climb onto vegetation. In addition, rain events can release larvae from fecal pellets and may require additional, specific monitoring of camelids a few weeks after significant rainfall [93]. The life cycle is direct, and the camelid ingests grass containing L3 larvae, which mature through L4 and L5 to become adults in the third compartment (C3) of the stomach or the intestine. The prepatent period is about 18–21 days [3].

Conditions which favor rapid development of the strongyle-type GINs include humid, mild temperatures. Conditions which slow or stop the life cycle include direct sunlight, desiccation, and extreme heat. Nematodes have a tough outer membrane called the "cuticle," which is replaced during each larval molt, but the infective L3 retains the L2 cuticle, making it resistant to poor environmental conditions. In addition, the cuticle of L3 prevents feeding, leaving the infective stage reliant on its stored energy source. If the L3 is not ex-sheathed before its energy stores run out, it will die. Cool, dry weather will decrease larval metabolic rates, and allow the larvae to persist in the environment for many months, whereas hot weather induces a high metabolic rate and depletes stored energy quickly [94]. These nuances become important for owners and veterinarians to understand in relation to pasture rest. A pasture will need to be rested for a longer period during cool, dry conditions since nematodes will be able to survive longer. *Haemonchus* and *Ostertagia* can protect themselves from unfavorable environmental conditions by undergoing a period of arrested development known as hypobiosis. Arrested L4 larvae of *Ostertagia* spp. can be found within the mucosa in C3, often occupying the gastric pits. Once conditions are favorable once more, larvae emerge and resume maturation. The emergence of many of these hypobiotic larvae can cause a clinical syndrome called Type 2 disease, discussed in the clinical signs section.

Epidemiology

Young, growing animals are most susceptible to clinical disease from GINs, since they lack acquired immunity [95, 96]. Though the presence of nematode-specific antibodies in colostrum and milk have been described in cattle and

small ruminants, it has not been determined whether these antibodies induce protective immunity. In older animals, any stressors or illnesses (pregnancy, lactation, weaning, shearing, overcrowding, inadequate nutrition, etc.) that may impair the immune response to GINs will increase their risk of clinical disease. A "periparturient rise" can be seen for most GINs, manifesting as increased numbers of eggs passed in the feces [9]. This can represent an additional source of infection for neonates, or increased pasture contamination for herd mates. Though patterns of GIN infection are generally specific to geographic region and climate, differing patterns may emerge due to the shipping of animals across country, the effects of global climate change on parasite life cycle, and the susceptibility of GINs to anthelmintics.

A description of common GINs of camelids follows, with important life cycle differences noted. GINs that produce strongyle-type eggs are noted with an asterisk (*), these eggs cannot be differentiated without additional diagnostic techniques such as coproculture, egg staining, or molecular techniques.

Esophagus

***Gongylonema* spp.** *Gonglyonema* is a spiruirda nematode with a broad-host distribution and is known as the "cattle gullet worm." Male *Gongylonema pulchrum* are 30–62 mm long and 150–300 μm wide; females are 80–145 mm long and 300–500 μm wide. The eggs are 50–70 × 25–37 μm [97].

Adults are embedded in the mucosa or submucosa of the esophagus and stomach. Eggs are passed in the feces and ingested by beetles, the required intermediate host. The eggs hatch and mature to infective larvae in about four weeks. The camelid must then ingest the beetle to complete the cycle. The migration route of the larvae to the esophagus is not known.

These parasites are nonpathogenic to cattle, and no reports of signs or pathology in camelids have been published.

Gastric Compartment (C3) Nematodes

Nematodes of C3 cause erosion and ulceration of the gastric mucosa and resulting gastritis. Damage to the gastric mucosa will decrease the amount of pepsin and hydrochloric acid produced, resulting in a potentially less acidic C3. Some nematodes of C3 cause profound anemia and ill-thrift.

Camelostrongylus mentulatus* This species is a common stomach (C3) parasite of camels in the Middle East, but it is also found in Australia, South America, and the United States. The life cycle and epidemiologic patterns are similar to those of other trichostrongyles [98].

Marshallagia marshalli* This species is closely related to *Ostertagia* and has a similar life cycle. When ingested, the larvae penetrate into the gastric mucosa of C3 and produce a nodule that is 2–4 mm in diameter [99]. Instead of one larva per nodule, as in ostertagiasis, each nodule contains two or three larvae that will mature in 15–18 days. The prepatent period lasts for up to three weeks, but there may also be arrested development (hypobiosis) in this species. The eggs of this parasite are large and frequently confused with that of *Nematodirus* spp. (Table 7.5). *Marshallagia* has a limited distribution, but pockets of infection are located in the western United States [89].

Ostertagia ostertagi* There are two types of cycle in these species (Figure 7.36). Type 1 is typical of a trichostrongyle life cycle, with larvae maturing to adults without passing through a developmental arrest stage. Type 2 involves developmental arrest in the mucosa of the stomach. Larvae become arrested in unfavorable environmental conditions and begin development again when favorable conditions return. Regional patterns in the United States have become increasingly erratic with the effects of climate change and changing parasite predominance [100, 101]. Larvae in the gastric glands of the C3 mucosa stimulate formation of grayish-white nodules. Maturation of previously arrested larvae can cause a buildup of pathogenic adults, which, in turn, cause gastritis and heavy egg levels in the feces.

Haemonchus contortus* The life cycle of this common parasite is similar to other trichostrongyles. Infective larvae in the pasture are present four to six days after the eggs have been passed in the feces. Eggs in the pre-hatch stage are more resistant to freezing and desiccation than the

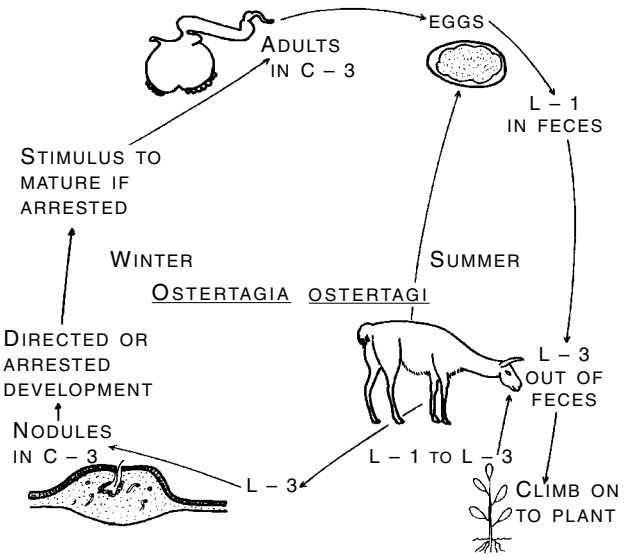

Figure 7.36 Life cycle of *Ostertagia ostertagi*.

larvae, but all stages of the eggs and larvae can tolerate some degree of desiccation and low temperature. Other species of *Haemonchus* may have slightly different cycles and be adapted to either hot/dry climates or moist/cool climates. Once the infective larvae have been ingested, they may enter an arrested development stage resembling that of *Ostertagia*. The nodules in C3 are not as diffuse or pronounced as in ostertagiasis.

H. contortus differs from other trichostrongyles in that it requires a blood meal for survival. The trauma to the mucosa produced by this parasite also allows seepage of blood into the lumen of the C3. The amount of blood loss caused by each nematode has been calculated to be 0.05 mL/day [1]. Blood may be detected in the feces through the use of occult fecal blood tests, but false negatives can occur, due to the concentrated nature of camelid fecal pellets.

In addition to erythrocyte loss, there is a significant loss of plasma protein through mucosal defects. Some compensation for the protein loss may be achieved by increased albumin synthesis, but ultimately, protein reserves are depleted and hypoalbuminemia develops. There are hyperacute, acute, and chronic forms of haemonchosis. The chronic form affects most camelids; affected animals become unthrifty, weak, and emaciated in addition to severe anemia. Camelids with severe clinical disease often require blood transfusions and intense supportive care. Faffa Malan CHArt (FAMACHA) scoring (observation of the ocular mucous membranes for signs of pallor indicating anemia) can be used to monitor for the development of clinical disease related to *Haemonchus* infection. This method is discussed in the "Treatment and Control" section in this chapter. Adult female *Haemonchus* have a characteristic "barber pole" appearance because the reproductive and GI system of the worm are entwined in a spiral pattern (Figure 7.37).

Teladorsagia circumcincta* *Teladorsagia* is a strongyle-type nematode that is usually found only in sheep but has been documented in a llama that had been pastured with sheep [102]. Nodules were found in the C3 on necropsy that had the gross and histologic appearance of Type 2 ostertagiasis. No adults were observed, and it is not known whether the larvae would resume development and produce clinical disease.

Trichostrongylus* spp. A generalized life cycle for *Trichostrongylus* spp. is as follows (Figure 7.38). The prepatent period is about 20 days [3].

Graphinema auchenia* This trichostrongyle has been reported in South America, Australia [86] and rarely in the United States. Adult parasites are found in C3. The life cycle and epidemiology are similar to those of other trichostrongyles.

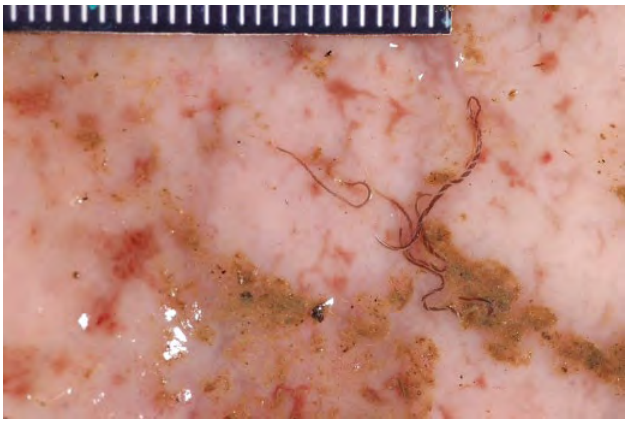

Figure 7.37 *Haemonchus contortus* found at necropsy. Note the distinct "barber pole" appearance characteristic of adult females.

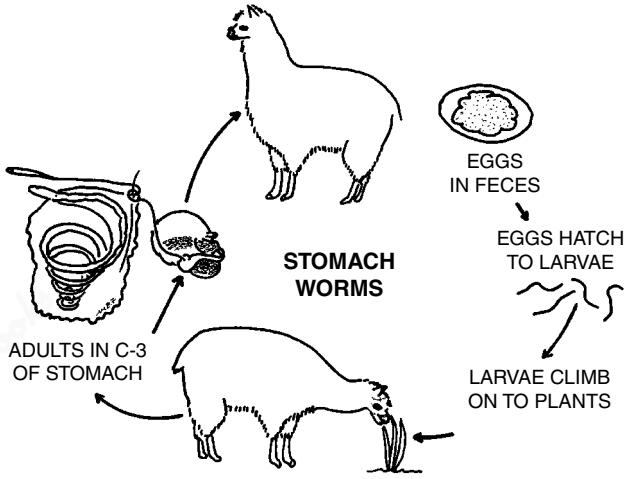

Figure 7.38 Life cycle of *Trichostrongylus* spp.

Spiculopteragia peruvianus* This trichostrongyle nematode is found in C3 of llamas, alpacas, and vicuñas on the Altiplano, near Lake Titicaca, in Peru [103, 104]. Little is known about the biology of this parasite other than its description (Table 7.6).

Small Intestinal Nematodes

Small intestinal nematodes will result in enteritis and varying degrees of protein-losing enteropathy depending on virulence and worm burden.

Cooperia* spp. *Cooperia* are small trichostrongyles living in the small intestine of ruminants and camelids throughout the world [88]. The life cycle and epidemiology are similar to those of *Trichostrongylus* spp. The infective larvae are able to survive on pastures 9–26 weeks and can overwinter. There may be an arrested development stage in the wall of the intestine [1, 105].

Nematodirus* spp. Eggs of *Nematodirus* spp. are passed in the feces and undergo slow development (over two or three months) to larvated eggs. The larvated eggs usually overwinter, and cold temperatures are required to ultimately induce the eggs to hatch and release the larvae. In the spring, when soil temperatures begin to rise, the mobile larvae climb onto vegetation and are ingested.

Following ingestion, infective L3 larvae penetrate the intestinal mucosa, where they molt to L4 and L5. L5 larvae leave the mucosa and become adults in the lumen of the small intestine. The prepatent period is 15 days under optimum circumstances. Adult parasites live for only a few weeks and seem to have a much lower fecundity than other trichostrongyles [106].

Since larvated eggs overwinter, an infection in a given year depends on contamination of a pasture the previous year. This parasite is most pathogenic to youngstock with no acquired immunity. Once larvae hatch, they only live for four weeks on pasture, so depending on the timing of larvae hatching and parturition, neonates may be at risk. Adults may harbor some parasites and contribute to contamination of the pastures, but overt disease is usually not seen in adults unless there is a heavy infection. Since this nematode has low fecundity compared with other GINs, lower egg counts may indicate a higher adult population. This is one of the only trichostrongyles with a distinctive egg. Species differences may also account for variation in pathogenicity: *Nematodirus battus* is infrequently encountered but may cause serious enteritis [87].

Trichostrongylus* spp. Some *Trichostrongylus* species are found in the small intestine instead of the C3. Life cycle is as above.

***Strongyloides* spp.** *Strongyloides* has been reported in racing camels in the United Arab Emirates, and has been occasionally reported on fecal examination in the United States [5, 107]. *Strongyloides* has a complex life cycle in which it passes rhabditiform larvae or larvated eggs in the feces and matures on pasture to become a free-living adult. Infection with *Strongyloides* can occur by ingestion of filariform larvae, or via percutaneous infection. Transmammary transmission is theoretically possible, based on *Strongyloides* infection in other species.

Lamanema chavezi* *Lamanema chavezi* is one of the more important and pathogenic parasites of camelids in the Andes [108]. Though identification of *L. chavezi* has been reported [109] outside of South America, it appears to be rare. Camelids being imported from South America should be carefully examined. The definitive host was reported as the mountain viscacha (*Lagidium viscacia boxi*) from

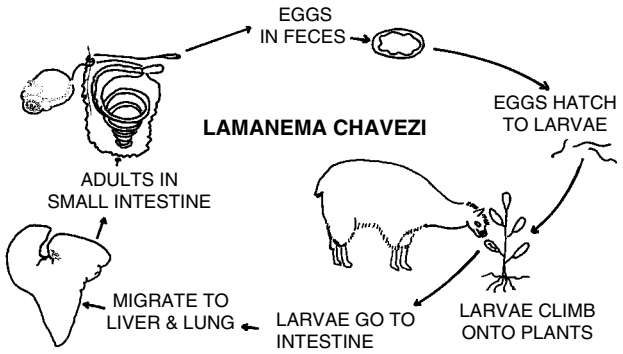

Figure 7.39 Life cycle of *Lamanema chavezi*.

samples recovered in Argentina [110]. It is highly likely that the alpaca or llama is an aberrant host. The life cycle of *L. chavezi* is unique (Figure 7.39). The adults are found in the small intestine rather than in C3 and the life cycle stages of egg–L3 are similar to those of other trichostrongyles. Ingested larvae penetrate the intestinal wall and migrate to the liver and lungs. It is in the liver that the most serious damage occurs. Ultimately, maturation is completed with migration back to the small intestine via the trachea [110].

Heavy infections cause hepatic and respiratory failure from involvement of the liver and lungs. The migration of the larvae through the wall of the intestine produces a catarrhal, hemorrhagic enteritis, with areas of necrosis of the mucosa. In acute cases, with recent migrations, the liver is congested with multiple foci of coagulative necrosis, petechial hemorrhage, and punctate abscessation. There are also areas of congestion in the lungs. Following maturation and remigration of the larvae, the liver lesions become fibrotic and may calcify. These residual foci have been seen in adult alpacas slaughtered for meat.

***Capillaria (Aonchotheca)* spp.** Capillarids are closely related to *Trichuris* but lack the slender and thick variation of the body [3]. Little is known about the species found in SACs, but eggs identified in the laboratory are presumed to be identical to species found in ruminants. This parasite has been detected on routine fecal examination, but no specific pathogenicity has been reported in camelids.

***Bunostomum* spp.** *Bunostomum* are hookworms that are rare in camelids; they are found only in SACs living in warm, tropical climates [111]. Infective larvae are susceptible to desiccation, so this parasite is a problem only where there is permanent moisture or high humidity. There is no encystment in the muscles nor is there transmammary migration as occurs in ancylostomiasis. See Table 7.6 for measurements.

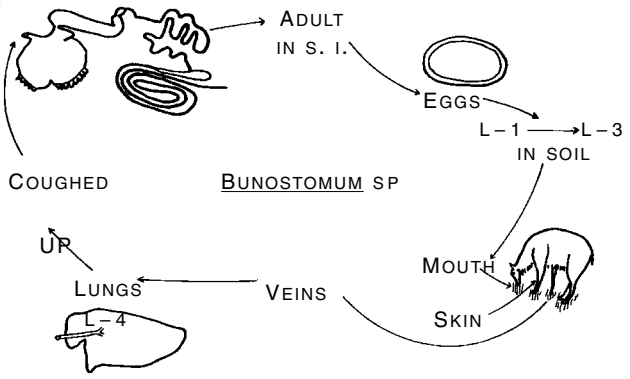

Figure 7.40 Life cycle of *Bunostomum* spp.

The life cycle is direct (Figure 7.40). Adults are attached to the mucosa of the small intestine and are blood suckers. The eggs require a few days after passage in the feces before infective larvae (L3) are produced. These larvae may enter the body via the mouth or through the skin. If via the skin, the larvae migrate to the lung via the venous or lymphatic vessels and mature to L4. These are coughed up and swallowed. L4 larvae migrate to the intestine. The prepatent period is 30–56 days [3].

Large Intestinal Nematodes

Oesophagostomum* spp. The nodular worm (*Oesophagostomum venulosum*) is a strongyloid nematode found in the small and large intestines of camels and ruminants. None of the parasites found in SACs have been identified as to species. A similar strongyloid, found in sheep, is *Chabertia ovina*, which has also been reported in camels and in vicuñas in South America [111]. See Table 7.6 for measurements.

The life cycle of camelid *Oesophagostomum* is as follows: the thin-walled eggs are passed in the feces and require six to seven days to reach the infective larval stage. The larvae climb onto vegetation, are ingested, and exsheath in the small intestine. Larvae penetrate the wall of the intestine according to species predilection and may or may not produce nodules. L4 and L5 maturation takes place in the wall of the intestine; then the larvae migrate back to the lumen and become adults, taking up residence in the large intestine. The prepatent period of *Oesophagostomum columbianum* is 41 days, and 21–38 days for *O. venulosum*. *O. venulosum* has less tendency to form nodules and thus is less pathogenic [3].

Trichuris tenuis, Trichuris ovis Whipworms (*Trichuris* spp.) are significant parasites of camelids and are resistant to treatment. They are characterized by a long, slender anterior and a thicker posterior segment. See Table 7.6 for measurements. The eggs are doubly operculated and thus are easy to identify, but they must be differentiated from those of *Capillaria* spp. Adult parasites are found in the cecum and large intestine [112]. Trichurids identified from camels include *Trichuris globulosa, Trichuris cameli, and Trichuris skrjabini* [113].

Trichurids all have a direct life cycle (Figure 7.41). Eggs are passed in the feces, requiring about three weeks to become infective. The infective eggs are ingested by the camelid and hatch. Larvae penetrate the wall of the anterior small intestine. Maturation proceeds for 2–10 days, and the larvae migrate to the cecum and large intestine and become adults. The prepatent period for *Trichuris ovis* is seven to nine weeks. The persistence of infective eggs in the environment makes management of this parasitism difficult [3].

These nematodes tunnel into the intestinal mucosa with the slender anterior ends (Figures 7.42 and 7.43). They

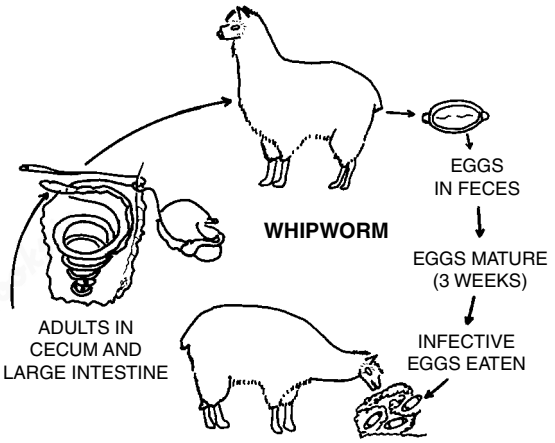

Figure 7.41 Life cycle of *Trichuris tenuis*.

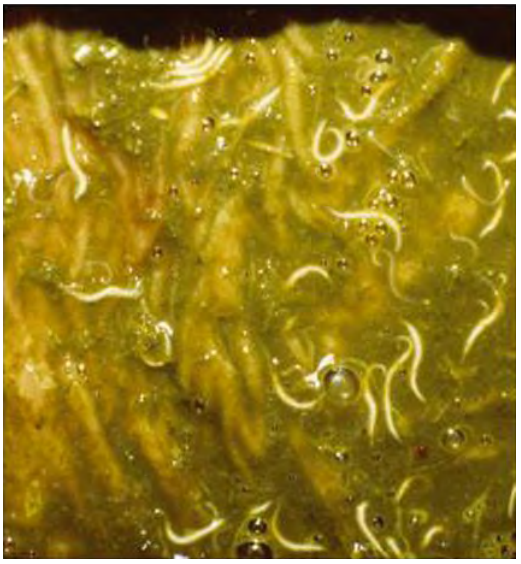

Figure 7.42 Whipworms in the colon of a llama. Ingesta surrounding parasites.

Figure 7.43 Whipworms in the colon of a llama. Ingesta washed away.

traumatize vessels, producing a catarrhal enteritis and cause hemorrhage, which is ingested by the parasites. The clinical syndrome is similar to that of haemonchosis.

Skrjabinema ovis This parasite is the sheep pinworm. It has been reported in the guanaco in Argentina [114]. The males are 2.3–3.7 mm long and 110–180 μm wide, making them wider than most other GI nematodes. The eggs are slightly flattened on one side and are 47–63 × 27 to 36 μm.

The life cycle is direct. The adults live in the rectum, but the female traverses the anal sphincter to deposit eggs around the anus at night. The eggs drop off and contaminate feed and water, infective larvae develop within the eggs. When ingested, eggs hatch in the small intestine, and the larvae migrate to the large intestine where they become mature worms in 17–25 days [3].

This parasite is not considered pathogenic to the host. Clinical signs include possible anal pruritus. Eggs will not usually be seen in a fecal flotation, adhesive tape applied to the anus and then applied to a glass slide is the customary diagnostic technique.

Clinical Signs

The majority of clinical disease related to GINs will manifest as a chronic, slowly progressive illness. Infection with a single type of GIN is rare, so clinical signs are typically non-specific and vague. The animal will experience progressive weight loss or decreased weight gain (in the case of young animals), ill-thrift, and poor fiber coat. Heavily parasitized camelids will be lethargic and weak, and may spend more time in a cushed position, or more time away from the herd. Emaciation may be seen in longstanding cases due to inappetence progressing to anorexia, combined with poor nutrient absorption due to heavy GI parasitism. Chronic GI parasitism may also predispose camelids to secondary infections (bacterial, viral, fungal) that they would normally be able to resist.

Diarrhea may be present, depending on which segment of the GI tract is most heavily parasitized. Camelids with predominant *H. contortus* infection of the third compartment may only have soft stool late in disease, while animals with predominant *Trichuris* spp. infection in the colon will have severe diarrhea. Occasionally, some camelids will display signs of colic (abdominal discomfort, kicking at abdomen, rolling, repeated bouts of standing up and laying down). Whether the camelid has diarrhea or not, GINs will cause a protein-losing enteropathy either from increased loss from diarrhea or decreased absorption from direct damage to the mucosa by the parasites. Clinical signs of hypoproteinemia might include submandibular or ventral edema, or periocular swelling. In addition to inadequate protein absorption, it can be expected that heavily parasitized camelids will also have poor absorption of critical vitamins and minerals from their diet. Lactating females will have decreased milk production, and this may predispose the cria to malnourishment.

Pale mucous membranes and anemia may be seen in heavy infestations, even with parasites that are not blood suckers. The cutting mouth parts used for attachment may result in leakage of plasma and cells from capillaries. Since the anemia is chronic, many camelids can compensate for slow blood loss until packed cell volumes (PCVs) are in the single digits (normal: 22–45%). Severe anemia will present as tachycardia, tachypnea, cold extremities, decreased blood pressure, and cheek-puffing or open-mouth breathing. South American camelids are uniquely positioned to withstand the effects of low oxygen tension, since they evolved at extremely high altitudes and gained the ability to shift their oxygen-hemoglobin dissociation curve further than animals that evolved at sea-level [115]. This adaptation may mask the severity of a chronic anemia until the animal is decompensating.

Acute decompensation of animals occurs when either hypoproteinemia, emaciation, or anemia (or a combination of these) has become so severe that the animal is not able to perform basic functions. Unfortunately, unless owners are diligent about checking herds for subtle signs and keeping good records, many cases go unnoticed until animals have heavy parasite burdens and chronic disease.

A notable exception to these patterns of chronic debilitation, is the acute/peracute Type 2 disease, associated mainly with *Ostertagia ostertagi* infection. Type 2 disease occurs when environmental conditions favor the resumed

development of arrested L4 larvae in the gastric mucosa. If a large amount of L4 larvae underwent hypobiosis at the end of the previous grazing period, all of these larvae will emerge simultaneously, creating devastating mucosal damage in the third compartment. Severe gastritis can cause clinical signs of profuse, watery diarrhea, anorexia, excessive salivation, acute collapse, and potential high mortality [116].

Diagnosis

Qualitative Diagnostic Techniques

Antemortem diagnosis of disease caused by GINs can be supported through microscopic examination of fecal flotations. A centrifuged fecal flotation can help give a qualitative assessment of the types of GINs shedding eggs in the feces, and a *subjective* assessment of the magnitude of infection. Fecal flotation relies on the differences in the specific gravity of parasite eggs or oocysts, and the specific gravity of the flotation solution. Parasite eggs or oocysts less dense than the solution will float to the top, and these can be examined by microscopy. Several salt and sugar flotation solutions are available, and each has its advantages and disadvantages. A sugar solution slide preparation can be kept at 4 °C for several weeks without disruption of the parasite egg morphology, but the solution has a shorter shelf life and can have fungal growth unless a preservative such as formalin is added. Salt solutions such as zinc sulfate have a longer shelf life because they resist bacterial and fungal growth, but slide preparations need to be examined within a short period of time since the solution will crystallize and distort egg morphology. A specific gravity of at least 1.24 should be used in order to recover most GIN eggs (Sheather's sugar solution specific gravity is 1.27).

Centrifugation flotation (Box 7.1) should be used for camelid feces; simple flotation without centrifugation will yield a sample with too much debris and may not be diagnostic. Box 7.1 outlines the basic centrifugation technique recommended for recovering parasitic eggs from camelid feces. Techniques such as simple flotation and direct smear will fail to recover heavy eggs such as *Nematodirus, Trichuris,* and *Eimeria macusaniensis* [117]. Collection of feces directly from the rectum will avoid contamination of the sample with feces from other animals, free-living nematode species or fungi, etc. Feces should be stored in a refrigerator at 4 °C in an airtight container and used for fecal flotation within five days to avoid larvation of the eggs.

Various parasitology textbooks can be used to identify nematode eggs to genus, except for strongyle-type eggs (Superfamilly: Trichostrongyloidea), which cannot be distinguished from each other (except *Nematodirus* and *Marshallagia*). Additional diagnostics are available to investigate genus- or species-level in strongyle-type eggs (*Haemonchus, Trichostrongylus, Ostertagia, Camelostrongylus, Cooperia, and Oesophagostomum,* etc.) (Fig. 7.44).

Box 7.1 Basic Centrifugation Flotation Technique

1) Mix 1 teaspoonful (1–2 g) of feces with a small amount water in a disposable cup. Strain through a nylon screen into a centrifuge tube.
2) Centrifuge for 3–5 minutes at 1000 rpm or about 400–1000 x *g*. You can repeat this wash step if necessary.
3) Decant the supernatant into a discard vessel and properly dispose.
4) Add a small amount of flotation solution and thoroughly mix into the pellet before adding more solution in incremental steps. If you do not mix the pellet well during this step, you diminish sensitivity.
5) Continue to add solution and mix until the tube is almost full (1–2 mm below the rim of the tube).
6) Centrifuge for five minutes.
7) Remove the tube from the centrifuge and place in a test-tube rack, add solution until a slightly bulging meniscus is created.
8) Place a coverslip on the meniscus and allow 10 minutes of flotation.
9) Remove the coverslip and place on a microscope slide to examine at 10X and 40X.

Quantitative Diagnostic Techniques

To objectively assess the magnitude of parasite infection, quantitative fecal egg counting techniques can be employed. Fecal egg counts (FECs) are assessed for the strongyle-type eggs as an estimate of adult worm burden within the GI tract by calculating the eggs per gram value (Figure 7.45). FEC can be a powerful tool for several reasons. It can serve as the basis of clinical decision making for a single animal and can provide information on a herd level to show seasonality in nematode egg excretion. The seasonality should be considered with other management factors for the design of an integrated parasite management plan. Over time, a robust clinical picture can be created. It can be expected that FECs from healthy camelids will be significantly lower than those found in sheep or goats grazing on the same pasture. This seems to be linked to camelids' ability to suppress fecundity more effectively than sheep with similar adult worm burdens [118]. Along the same lines, it can be inferred that camelids clinical for GI parasitism may not have as high a FEC as those found in clinical sheep or goats.

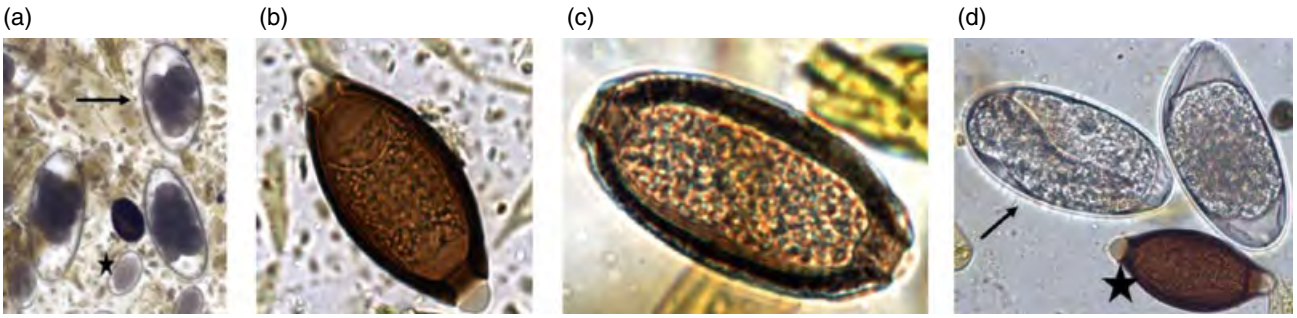

Figure 7.44 Appearance of common nematode ova on fecal flotation: (a). *Nematodirus* (arrow), *Eimeria macusaniensis,* trichostrongyle (star) (b). *Trichuris* (c) *Capillaria* (d) larvated trichostrongyle (arrow), *Trichuris* (star), trichostrongyle.

Figure 7.45 McMaster's counting slide with two chambers.

Factors do exist that impact the number of eggs present in a sample. Fecundity of female worms will affect the usefulness of a FEC; parasites with high fecundity such as *H. contortus* will have FECs that are highly correlated to adult worm burden, but parasites with low fecundity such as *Teladorsagia* are poorly correlated. In addition to differential fecundity, immature stages of parasite will not be producing eggs yet but may be causing significant damage to the GI tract. Diarrhea may dilute parasite eggs in a sample and underestimate levels of infection. Recently used anthelmintic compounds may also affect FECs.

There are several methods described for the quantitation of strongyle eggs in a fecal sample:

- McMaster
- Modified Stoll's
- FLOTAC or mini-FLOTAC
- FEKPAK
- Wisconsin or modified Wisconsin

Differences exist between the quantitative methods (e.g. McMaster is normally chosen for higher anticipated egg counts) and the sensitivity, specificity, and differences between these methods have been compared in the peer-reviewed literature for large and small ruminants [119, 120], but little work has been done in camelids. Regardless of the method chosen, consistency with the same chosen method will allow for comparisons over time.

Shortfalls in FEC techniques include its lack of ability to identify genus or species of strongyle-type eggs, the requirement of specialized equipment (centrifuge, special counting slides, FLOTAC devices, etc.), and shortfalls related to inconsistency of eggs in a sample as mentioned above. For these reasons, FEC techniques should not be the sole determinant of treatment and management of clinical GI parasitism, instead the clinician or owner should evaluate it in light of the entire clinical picture (clinical signs, body condition, FAMACHA, fecal consistency, weight gain/loss).

Genus- and Species-Level Diagnostic Techniques for Strongyles

Larval culture (coproculture) can be performed to hatch the strongyle-type eggs in the feces and microscopy and parasitology references can be used to identify the third-stage larvae. The L3 of all strongyle-type eggs has been described except for *Camelostrongylus*. Strongyle eggs can also be stained with a fluorescein-labeled peanut agglutinin and observed for fluorescence. Eggs of *H. contortus* will fluoresce, differentiating them from other strongyles, and an estimate of % can help guide management decisions and the use of monitoring tools such as FAMACHA scoring (described in the "Treatment and Control" section). Fecal PCR can also identify specific strongyles and is performed at various diagnostic laboratories.

Dr. John Gilleard's laboratory at the University of Calgary has developed a project called the "Nemabiome" which evaluates the complex communities of parasites unique to each animal or herd, much like the unique and complex bacterial populations in the GI tract. Using molecular techniques, the Nemabiome project can identify the proportions of each genus/species in the parasite community, providing a more detailed picture, and even identifying resistance alleles [121, 122]. This project may provide the

basis for more tailored approaches to parasite management unique to each farm but has limited applications in small herds at this time.

Anthelmintic-Related Diagnostic Techniques

An integral factor to the success of a parasite management program is the evaluation of drug efficacy. Severe, widespread and multi-drug resistance in the United States and other countries such as Australia, have resulted in treatment failure and increased morbidity and mortality in camelids. This reality has necessitated the monitoring of drug efficacy as well as monitoring for resistant parasites, which can be achieved with the Fecal Egg Count Reduction Test (FECRT) Box. 7.2, or other more advanced techniques such as the larval development assay (LDA).

The FECRT is a widely used and simple test that can be completed either by the veterinarian or by a diagnostic laboratory. Ideally, at least 10 animals are used, and camelids with FAMACHA scores >3 are optimal candidates, as they will generally have the highest worm burdens. Larger worm burdens will provide more accurate FECRTs. FECRTs should be performed every 2–3 years on a farm. Anything <95% reduction in the strongyle-type eggs is indicative of resistance of that parasite population to the anthelmintic used, according to the World Association for the Advancement of Veterinary Parasitology (WAAVP) [123]. To complete the FECRT:

Before administration of the anthelmintic, individual animals should be weighed, so that drug dosages are accurate. Further discussion regarding the FECRT as part of an integrated parasite management plan are discussed in the "Treatment and Control" section.

Larval development assay is performed by specialty testing laboratory services. A composite fecal from a herd is collected, parasite eggs are harvested from the sample and are exposed to increasing amounts of three drug classes. This provides information on the exact sensitivity of that subpopulation of parasites to drugs commonly used to treat GI parasitism and may help identify resistance trends [124–126].

Box 7.2 Steps to Perform the Fecal Egg Count Reduction Test (FECRT)

1) Pre-treatment sample is taken and FEC is performed using any of the quantitative methods.
2) An anthelmintic is administered the same day the fecal sample is tested.
3) A post-treatment fecal sample is taken and an FEC is performed 10–14 days later using the same quantitative method.
4) FECRT calculation:

$$\frac{FECpre - FECpost}{FECpre} \times 100 = \%\ reduction$$

Post-Mortem Examination

Camelids suspected of mortality due to GI parasitism should be examined for gross and histologic pathology. Sections of the GI tract can be tied off (to prevent leakage of contents from the lumen), separated, and examined. Special attention should be paid to mucosal defects, hemorrhages, or erosions/ulcers, as well as the presence of GINs attached to mucosa. Feed contents should be collected and examined for each section in order to identify larvae or adults.

Treatment and Control Programs

Historical treatment and control programs relied heavily on suppressive deworming (treating all members of a herd with an anthelmintic regardless of clinical signs or diagnostics) and rotational deworming (cycling through different classes of drugs on a schedule). Initially, this was highly effective because the anthelmintics were highly efficacious, cheap, and convenient. Over time, these suppressive deworming techniques created selection pressure for resistant genotypes and have resulted in debilitating treatment failures within all drug classes. These control techniques are no longer acceptable.

One of the critical components of GI parasite control is preventing infection, but we cannot prevent infection all the time. Eventually, we have to be able to detect infection with a monitoring program. A robust clinical picture can be created by using periodic FEC (or FECs during key events) to determine seasonal parasite patterns, and to assess risk during periods of time when camelids will be experiencing stressors such as weaning, shearing, or parturition. Once patterns are identified, recommendations can be made for strategic monitoring and control that integrate *all* aspects of husbandry and management to prevent *disease* from parasite infection. Integrated parasite management incorporates the following elements:

1) Prevention
2) Monitoring
   a) FECs, FECRTs
   b) FAMACHA
   c) BCS
3) Treatment
4) Biosecurity and Quarantine

Prevention

Management practices that decrease camelids' exposure to feces will help prevent clinical disease from GINs. Fortunately, voiding habits of camelids help concentrate urine and feces in communal dung piles. If dung piles are near common food or water sources, or if there is overcrowding or less dominant camelids in a herd, risk of fecal-oral contamination increases. Regular removal of feces on a schedule that exploits the general life cycle of GINs can decrease this risk. Under the right conditions, *H. contortus* can develop from an egg to an infective L3 in three to four days. Therefore, dung piles should be removed every two to three days in order to break the life cycle and prevent infection [94].

The proportion of parasite stages living inside the camelid is very small compared to the proportion living on pasture, which makes forage/pasture management an essential component of integrated parasite management. Third-stage larvae can only vertically migrate so far on pasture grass, so most larvae are found close to the ground [127]. Pasture with short forage (<4″) will increase exposure of camelids to infective larvae and should be avoided. Co-grazing with horses, who share no pathogenic parasite species with camelids, can help clear pastures of infective larvae before camelids are re-introduced. Pasture rotation should be carefully managed; pastures allowed to lay fallow for three to six months (depending on climate) can also exploit the parasite life cycle so that a minimal number of L3 are left before camelids are re-introduced to the pasture. Treatment with an anthelmintic, then movement to a "clean" pasture should be avoided at all costs. This will potentiate the survival of resistant alleles, since the only parasites left in the camelids will be those resistant to the anthelmintic administered (further discussed in "Anthelmintic Resistance" section). Dry lots or concrete stalls can be used during high-risk periods since desiccation, exposure to direct sunlight, and extreme heat can kill L3s.

All new herd additions or visiting camelids (breeding management, fairs, etc.) should have an FEC and qualitative flotation in order to determine parasite status. Quarantine procedures should be implemented (see below).

Monitoring

Regular FECs can be used to help make treatment and control decisions. Monitoring can be increased during high-risk periods such as weaning, shearing, and parturition and with enough information on seasonal patterns, thresholds for treatment can be created for individual herds. Choosing diagnostics that use simple laboratory methods or use methods that clients can do on the farm can lower costs of repeated FECs. Simplicity is key: if the technique is quick and easy, this will increase adoption of these techniques by the client. FECs should be combined with clinical signs, FAMACHA scoring, and body condition score (BCS).

Use of the FAMACHA scoring system is a way to monitor camelids for the development of anemia due to GI parasitism, especially *H. contortus*. The FAMACHA score evaluates the color of the ocular mucous membranes in relation to a scoring card and assigns increasing levels of pallor a score. The color of the ocular mucous membranes correlates to the animal's PCV. Though there are other causes of anemia than GI parasitism, the FAMACHA score can be combined with other diagnostics to help support a diagnosis. This score can be recorded for each animal and monitored over time to discover trends. Owners can also be trained to use this scoring system in order to perform regular herd monitoring. To perform the FAMACHA score, the examiner should compare the color of the eyelid conjunctiva to the five colors on the scorecard (red to white) while using the index finger to slightly/gently retropulse the globe through the upper eyelid. (Figure 7.46) This technique is widely used in small ruminants but has recently been validated for use in camelids [128].

Regular body condition scoring should be performed by owners throughout the year, and records should be maintained. Please refer to body condition scoring in Chapter 4. Since heavily fibered camelids can mask even severe underconditioning, owners and veterinarians should emphasize the importance of touching the animal to determine a BCS, instead of visual evaluation alone.

Figure 7.46 Example of FAMACHA scoring, this alpaca has a score of 5.

Treatment

Infection with GINs does not always equal disease. If diagnostics provide evidence that supports disease due to GI parasitism, and treatment is necessary, several treatment options exist. In the United States, camelids are considered a minor use species, and treatment decisions should be supported by the Food Animal Residue Avoidance Databank (FARAD) and the Animal Medicinal Drug Use Clarification Act (AMDUCA). Careful dosing should include accurate weights for each individual animal. Most dosage recommendations have been extrapolated from cattle or small ruminant dosages and have not been evaluated in controlled treatment studies for camelids. Resistance of the GINs have been documented with every drug class, so efficacy of the chosen anthelmintic should be monitored with post-treatment diagnostics and resolution of clinical signs. If resistance is found to a specific anthelmintic, it can be expected that other drugs in the same class will also have resistance, since the same mechanism of action exists. Rotational deworming is discouraged, as this will perpetuate multi-drug resistance. Combination treatments can be used for camelids on a farm where resistance is documented. Please see the "Anthelmintic Resistance" section below for additional information on combination deworming. Table 7.7 lists anthelmintics used in camelids.

Benzimidazoles: albendazole, fenbendazole, and thiabendazole

Drugs in this class interrupt parasite energy metabolism by binding to tubulin, which disrupts nutrient uptake and other functions. Fenbendazole has a wide margin of safety, and the dose and frequency can be increased in most situations. Studies show that the duration of exposure has a marked effect on efficacy against GI parasites so multiple day dosing also increases killing capacity [129]. Albendazole has a far narrower margin of safety; dosages or frequency higher than the label amount can cause teratogenesis and bone marrow suppression with pancytopenia and death [130]. Fasting animals before administration of benzimidazoles can help slow GI transit time and increase exposure of the parasite to the anthelmintic. Resistance to benzimidazoles is not uncommon, and once resistance has

Table 7.7 Anthelmintics used in camelids [5].

Anthelmintic		Dose mg/kg		
Generic name	**Trade name in US**	**NWC**	**OWC**	**Route of administration**
Albendazole	Valbazen	10.0	5.0–7.5	Oral
		15.0 for *F. magna*		
Amprolium	Corid	5	?	Orally × 21
Clorsulon	Curatrem	7	?	Oral
Decoquinate	Deccox	0.5	?	Orally × 28
Fenbendazole	Panacur	5–10		Oral
		15 for *Trichuris*		
Oxfenbendazole	Oxfenbendazole	5	5	Oral
Levamisole	Levasole	5–8	7.5	Oral
Thiabendazole	Equizole	100	100–150	Orally × 1–3
Mebendazole	Telmin	22	5	Orally × 3
Pyrantel pamoate	Strongid	18	25	Orally × 3
Ivermectin	Ivomec	0.2	0.2	Oral or SC
		0.4–0.6 for *Trichuris* and *Cephenemyia*		
Doramectin	Dectomax	0.2	0.2	SC or IM
Moxidectin	Cydectin	0.4	0.4	Oral
Toltrazuril	Baycox	20	20	Oral; no data for use in camelids
Diclazuril	Decocci	1	1	IM, SC; no data for use in camelids
Metronidazole	Flagyl	25 Twice a day		Orally × 5

Data from Ballweber 2009 [5], and personal experience.
Caution! Do not use ionophore coccidiostats (monensin salinomycin) in camelids [81–83].

developed, susceptibility is not regained even when the drug is not used for several years [94].

Nicotinic anthelmintics and tetrahydropyrimidines: imidazothiazoles (levamisole), morantel tartrate, pyrantel pamoate

Drugs in this class act as cholinergic agonists, resulting in sustained muscle contraction of the parasites, then neuromuscular blockade and paralysis. Since mammals also have acetylcholine receptors, these drugs have a narrow therapeutic index. Levamisole can be used at a dosage of 8 mg/kg orally in camelids [131]. Animals displaying signs of toxicity may have classic "SLUD" signs – salivation, lacrimation, urination, and defecation in addition to hyperexcitability, trembling, ataxia, collapse, and death from respiratory failure. Morantel and pyrantel both have a wider margin of safety than levamisole. Morantel is a feed additive, and like other feed additive anthelmintics, may not deliver an acceptable dose if the animal does not consume the entire amount. This can lead to underdosing and, eventually, parasite resistance.

Macrocyclic lactones: ivermectin, moxidectin, eprinomectrin

Drugs in this class act as gamma-aminobutyric-acid (GABA) agonists and bind to glutamate-gated chloride channels in nerve and muscle cells of invertebrates, which block nerve conduction and cause paralysis of nematodes and arthropods. Macrocyclic lactones (MLs) were able to control both internal and external parasites, are inexpensive, and were highly effective when first introduced. For these reasons, MLs were used heavily in suppressive deworming programs since their introduction and resistance is now widespread in the GINs. Though several pour-on formulations are available for use in cattle and small ruminants, these should not be used in camelids as they have not been shown to have efficacy in controlling GINs [132]. It is also not recommended to give pour-on formulations orally as efficacy has not been proven [133].

Amino-acetonitrile derivatives: monepantel

Monepantel's mechanism of action is similar to the nicotinic anthelmintics, causing sustained contraction of nematode muscles and paralysis. It is approved for use in countries outside of the United States. Though this new drug was promising, parasite resistance was documented soon after its introduction in Australia and South America [134, 135].

Biosecurity and Quarantine

Camelids with diarrhea or being treated for clinical GI parasitism should be isolated from the rest of the herd until diagnostics are complete and clinical signs have normalized. Efforts should be made to feed sick animals last, and to ensure that there is no cross-contamination of equipment such as feed/watering buckets or pitchforks.

Camelids from outside the herd should be confined for three to four weeks in a stall or dry lot, with no exposure to the established herd. Most GINs have a prepatent period between two to four weeks (exceptions: *Trichuris, Nematodirus,* and the protozoa, *Eimeria macusaniensis)* so most infections can be detected during this length of quarantine. During quarantine, the animals should be evaluated and treated accordingly, and efficacy should be confirmed with FECRT. Treatment with two or more different drug classes may be necessary.

Anthelmintic resistance

Between the 1950s–1980s, several classes of highly effective anthelmintics were introduced to the market. These drugs were highly efficacious, inexpensive, and convenient. For these reasons, parasite control for many decades relied solely on the frequent or strategic use of anthelmintics. Over time, these suppressive deworming techniques created selection pressure for resistant genotypes in nematodes. Resistant alleles in the surviving worms gave genetic advantage to the next generation of parasites and, eventually, the majority of individual parasites that exist in a herd will not be susceptible to treatment. Serious emerging resistance of camelid parasites to multiple classes of anthelmintics is present in the United States [131]. Complete anthelmintic failure (failure of every single drug class) has been documented in the United States [136]. Drugs within the same class will all show "side resistance" since they all share the same mechanism of action, even if a particular drug has never been used on the farm [137]. Resistance in parasite populations will continue to outpace the formation of new drugs (no new drug classes have been introduced to the market since the 1980s), so the anthelmintics currently in use should be considered a finite resource, and their efficacy should be guarded by every available method. Often, an owner or producer will not notice drug resistance until it is severe, making buy-in to integrated parasite control methods difficult since they require more time and effort than simple anthelmintic use. Every effort should be made to use preventative methods, forage control, and monitoring so that drug use can be judicious and sparing. An understanding of the concept of refugia can help guide individualized herd parasite control.

Refugia

Refugia-based strategies can help slow the development of anthelmintic resistance in a herd [138]. Refugia is defined as the proportion of parasites in a population that have not been exposed to a particular anthelmintic. Parasites in the refugia escape exposure to anthelmintics and will be able

to contribute alleles to the next generation of parasites on the pasture. The susceptible alleles will be maintained and will dilute the frequency of resistant alleles. Resistance evolution in a herd can be slowed in this manner.

Sources of refugia include:

1) Free-living stages of parasite on pasture – worm eggs and larvae.
2) Parasites living in untreated hosts.
3) Any stages of parasites living in a host that are refractory (but not resistant) to treatment.

Sustainable Anthelmintic Use

Sustainable control of GINs does not mean complete eradication of the parasites. Frequent use of anthelmintics on a whole-herd basis fails to preserve a large enough refugia. In addition, the practice of deworming and immediate movement to a "safe" pasture will also decrease refugia and increase resistance to drugs. To increase the useful life of a drug in a herd, rotational deworming should not be practiced. A drug should be used until parasites develop resistance, then a switch to a different drug class can occur [139]. Anthelmintic susceptibility should be monitored by performance of a FECRT every two to three years in a herd.

Parasites are not evenly distributed throughout a herd; often, 80% of the parasites are in only 20–30% of the animals in the herd. This small proportion of the herd is contributing the majority of pasture contamination with parasite eggs. The ability to identify these 20–30% with FAMACHA scoring, body condition scoring, and FECs will help identify animals that truly require treatment [138]. This practice is termed "targeted selective treatment" (TST). To increase refugia, only clinically diseased animals that would benefit from treatment should receive an anthelmintic. Parasite infection is not disease, and even though eggs are found on a fecal, the camelid may not require treatment. Otherwise, healthy animals that lack signs of disease from GI parasitism (emaciation, anemia, lethargy, etc.) DO NOT require anthelmintics to stay healthy.

Combination Anthelmintic Use

If multi-drug resistance is documented in a herd, using at least two classes of anthelmintics simultaneously, at full dosage, can improve efficacy through an additive effect. Increased efficacy leads to fewer resistant survivors and greater dilution from a larger refugia. In fact, using drug combinations can even slow the development of multi-drug resistance [140]. Herds that utilize combination deworming may retain drug efficacy for years compared with months using a single efficacious active drug, even though owners will not see a clinical difference. In order to convince owners of this reality, a strong relationship between the veterinarian and the owner is needed to create buy-in.

Alternative Control Measures

A wide variety of alternative (non-anthelmintic) control measures exist for camelids. "Resting" pastures can allow for natural attrition of infective larval stages but require an extended period of time based on the life cycle of most GINs [141]. In the temperate United States, pastures should be rested for at least six months in cooler weather, and at least three months in hotter weather. Providing adequate nutrition with a high amount of metabolizable protein can help camelids resist parasite infection and also support the immune response to a parasite challenge [142]. The addition of copper oxide wire particles to the diet has been shown to decrease *H. contortus* worm burden in sheep and goats, but care must be taken to avoid copper toxicity in these sensitive species [143]. The use of condensed tannin forages and predacious, nematode-trapping fungi (*Duddingtonia flagrans*) as feed additives have also shown promise [144, 145]. Genetic selection of animals based on their resistance to clinical parasitism or immune system response has been studied extensively in sheep and studies now exist in camelids [118]. It has been found that genetic selection of camelids more resistant to parasites can be achieved while avoiding lower birthweight or fleece weight. Vaccination of camelids with novel parasite proteins in order to stimulate a protective immune response has been studied [146].

Nematodes of Other Body Systems

Lungworms

Dictyocaulus filarial, D. viviparus, Dictyocaulus spp.

These lungworms are included in the superfamily Trichostrongyloidea because the life cycle is direct. See Table 7.6 for measurements. Adult parasites live in the lumen of the bronchial tree (Figure 7.47). The female lays a larvated egg, which is coughed up and swallowed. Hatching usually occurs during passage through the digestive tract. The free-living larvae, unlike other trichostrongyles, live on stored nutrients rather than on microorganisms. The larvae develop from L1 to the infective L3 in five days or more. Larvae then climb onto vegetation and are ingested. The larvae penetrate the wall of the small intestine and migrate to the mesenteric lymph nodes and via the blood vessels or lymphatics, to the lungs. About four weeks are required for maturation of L5 to the adult nematode. The prepatent period is four to five weeks [1].

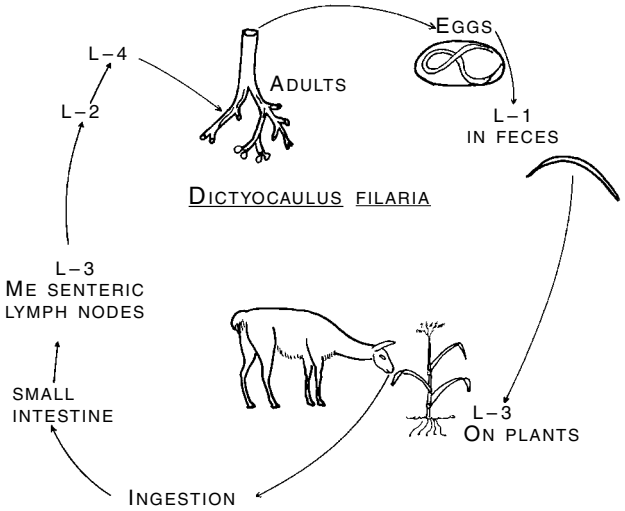

Figure 7.47 Life cycle of *Dictyocaulus filaria*.

Larvae require a moist environment and, therefore, are not a problem in hot, dry regions. Infective larvae can overwinter, but clinical signs are usually seen in the fall after a summer buildup of infection. The larvae are not usually evident on a standard fecal flotation. A Baermann sedimentation is necessary to evaluate feces for lungworm larvae. It is also important to remember that clinical disease may be seen before larvae appear in the feces. Larvae developing in the lungs produce a marked catarrhal bronchitis. Though this is a common parasite of SACs in South America and has been found in camelids in other countries [88], it is rarely encountered in the United States.

Clinical disease is seen more frequently in young animals or animals with a compromised immune system. This disease may be considered important when camelids are grazing in proximity with infected cattle and sheep. Clinical signs of *Dictyocaulus* infection are a dry cough, dyspnea, tachypnea, neck extension, and serous nasal discharge. Rectal temperature is normal unless a secondary pneumonia develops. In severe cases, pulmonary edema and emphysema cause increased mortality.

Treatment for *Dictyocaulus* is administration of ivermectin, fenbendazole, or albendazole. Prevention can be difficult in areas with high moisture. A vaccine is available for use in cattle but is not approved for use in other species at this time.

Angiostrongylus cantonensis

An alpaca in quarantine in preparation for importation to Australia, died, and at necropsy *Angiostrongylus cantonensis* nematodes were found in the lungs. This species is a rat lungworm in the Pacific Basin. The alpaca is an aberrant host.

Brainworm or Meningeal Worm

The meningeal worm, *Parelaphostrongylus tenuis*, is in the Protostrongylidae family. The white-tailed deer (*Odocoileus virginianus*) is the primary host for this parasite and rarely shows clinical signs. See Table 7.6 for measurements. The life cycle in the deer is shown in Figure 7.48. In aberrant hosts, the life cycle is rarely completed (non-patent infections). Adult worms are present in the central nervous system (CNS) in the venous sinus of the meninges. Eggs are laid into the blood stream and travel to the pulmonary parenchyma, are coughed up, swallowed, and are passed in the feces as L1. L1 mature in the intermediate host,

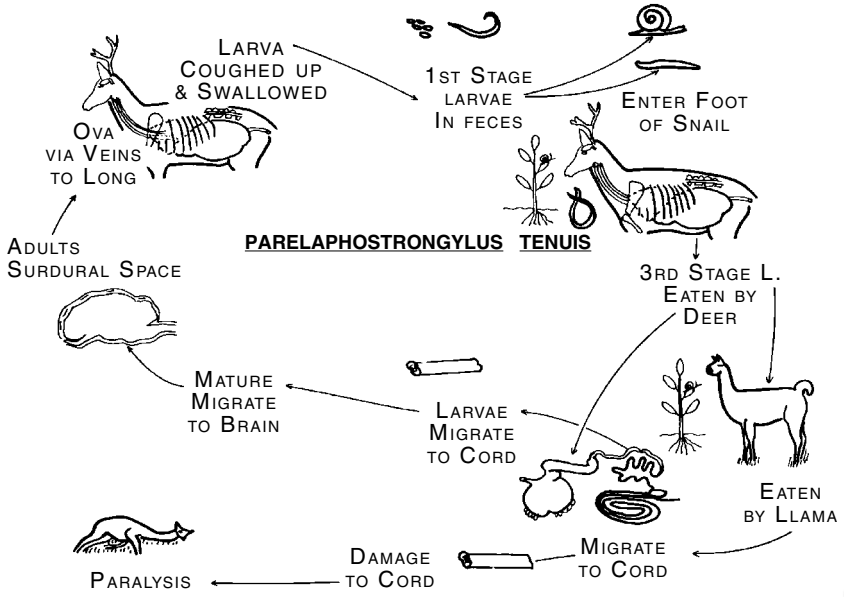

Figure 7.48 Life cycle of *Parelaphostrongylus tenuis*.

terrestrial snails or slugs (*Deroceras graciles, Deroceras reticulatum, Deroceras laeve, Cionella lubrica, Arion circumscriptus*, *Zonitoides nitidus*, or *Zonitoides arboreus)* to L3 within three to four weeks [147]. *P. tenuis* has been documented to survive and remain infectious outside the intermediate host in conditions of freezing and desiccation [148]. The snail, slug, or L3 in their slime trails is then ingested by the deer. Larvae are released in the stomach, penetrate the wall of the peritoneal cavity, and migrate to the spinal cord, usually via the spinal nerves, within 10 days. The larvae mature in the dorsal horns of the gray matter for 20–30 days. The adults then move to the spinal subdural space and migrate to the brain. They move into their ultimate location by penetrating the dura mater and passing into the venous sinuses. The prepatent period is 82–91 days.

The camelid is an aberrant host for this parasite and shows severe neurologic disease within 35–80 days of ingestion in experimental studies [149, 150]. The white-tailed deer has a broad distribution in the southern, eastern, and northern United States. The parasite is generally found wherever white-tailed deer are found. Camelids cohabiting in pastures with white-tailed deer are at risk. It should be mentioned that other ungulate species may also be aberrant hosts for this parasite, e.g. moose (*Alces alces*), wapiti (*Cervus canadensis*), caribou (*Rangifer tarandus*), black-tailed deer (*Odocoileus hemionus*), red deer (*Cervus elaphus*), cattle, sheep and goats.

The parasite is well adapted to the primary host, in which little or no clinical disease develops. In aberrant hosts, such as camelids, migration of the larvae in the spinal cord produces neurologic deficits commensurate with the location of the larvae (Figures 7.49 and 7.50) including lameness, proprioceptive (spinal) ataxia, stiffness, circling, blindness,

Figure 7.50 Alpaca with signs of intracranial migration of meningeal worm. Note the head tilt, unilateral ear and nostril droop.

(a)

(b)

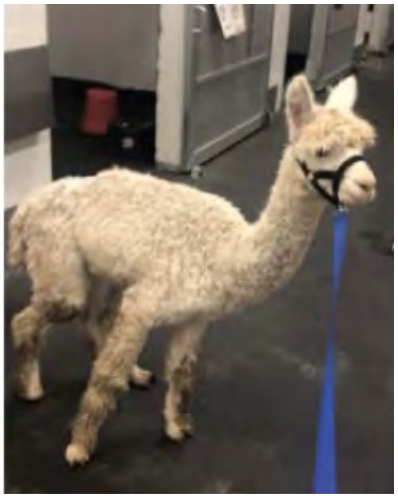

Figure 7.49 (a) Yearling female alpaca affected by meningeal worm. Animal is able to support her weight but has a crouched hindlimb stance indicative of hind limb weakness. (b) Knuckling at the right hind fetlock is indicative of proprioceptive deficits.

hypermetria, paraplegia, paralysis, and abnormal positions of the head [151].

Definitive antemortem diagnosis is difficult. L1 can be detected in white-tailed deer feces, however, the infection is rarely patent in aberrant hosts. Presumptive diagnosis is supported by clinical signs, exposure to the definitive or intermediate host, and eosinophilic pleocytosis in the cerebrospinal fluid [152, 153]. Serologic assays for use on serum and cerebrospinal fluid are currently being developed and show promise [154]. On postmortem exam, nematodes may be identified within the spinal cord or brain parenchyma but are not always present (Figure 7.51) [155]. Molecular confirmation of *P. tenuis* can be achieved with PCR.

Treatment of meningeal worm in camelids currently relies on the use of anti-inflammatories, physical therapy, and anthelmintics. Ivermectin is effective against the stages prior to entering the spinal cord, but ivermectin does not readily penetrate an intact blood–brain barrier [156]. Some clinicians report improvement and explain the results on the basis that penetration of the larvae through the meninges opens the blood–brain barrier. Efficacy of other drugs such as fenbendazole in the treatment of meningeal worm has not been evaluated. It is unknown whether spinal cord disease occurs due to the presence of the worm itself, or whether it is induced when the nematode cannot complete its life cycle and dies. For this reason, and because of the lack of controlled treatment studies, some veterinarians discourage the use of anthelmintics to treat meningeal worm.

Prevention is reliant on decreasing camelids' exposure to definitive and intermediate hosts. Barrier fencing, pea

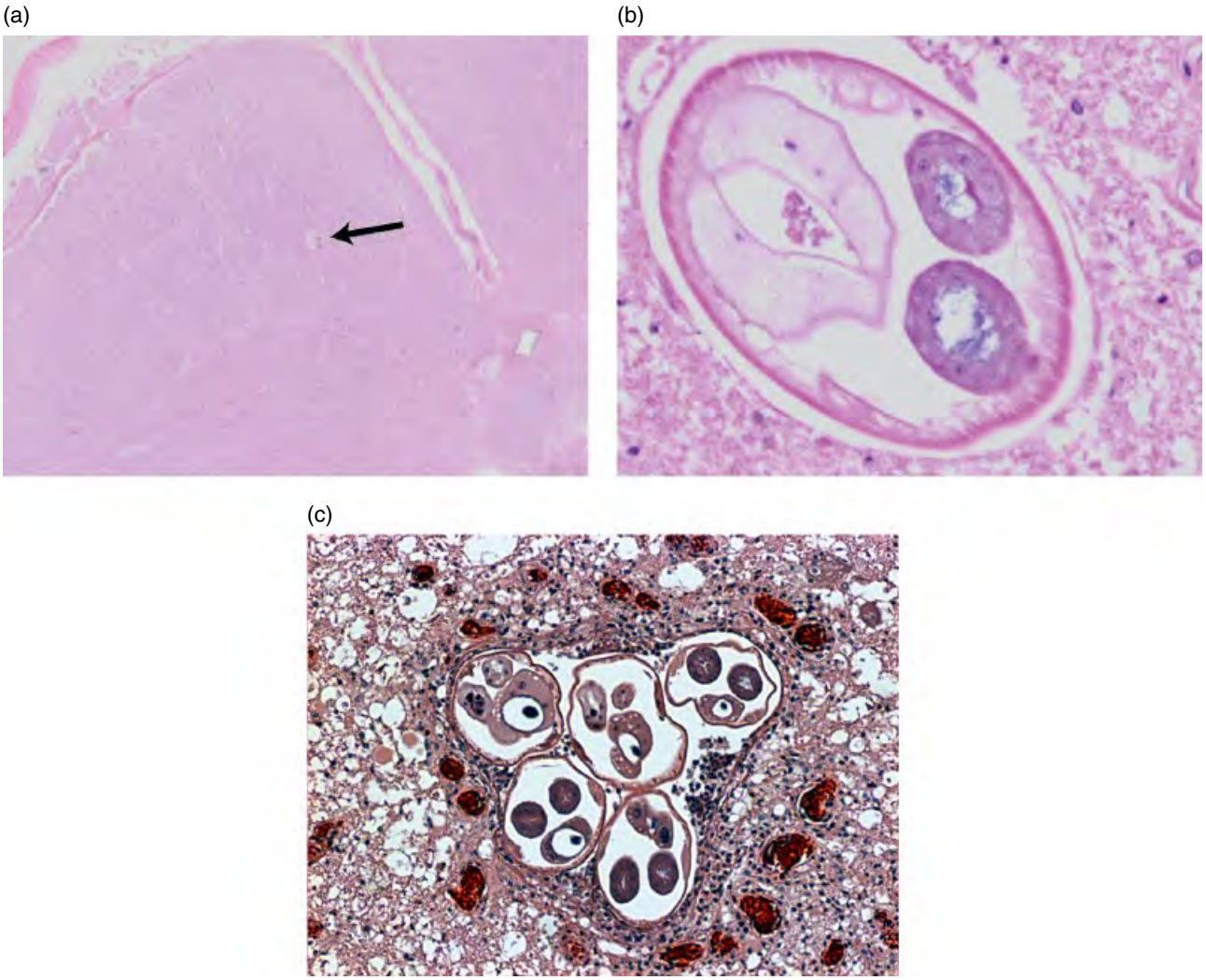

Figure 7.51 (a) (H&E X10) Cross section of a llama's spinal cord with *P. tenuis* (arrow). (b) (H&E X40) Higher magnification shows the *P. tenuis* organism in cross section. (c) Multiple cross sections of the worm are visible here along with a marked inflammatory response of the tissue surrounding the parasite.

gravel lining pastures, guinea fowl which consume the intermediate host, and elimination of moist pasture or access to wooded areas/ponds can help reduce risk. The administration of monthly "preventative" ivermectin is discouraged except on farms with a history of meningeal worm infection. The preventative use of ivermectin was supported by a single, small case study performed in white-tailed deer and has not been evaluated in aberrant hosts [156]. In addition to a lack of controlled treatment trials, the development of anthelmintic resistance, especially in macrocylic lactones, necessitates the need for judicious use of drugs.

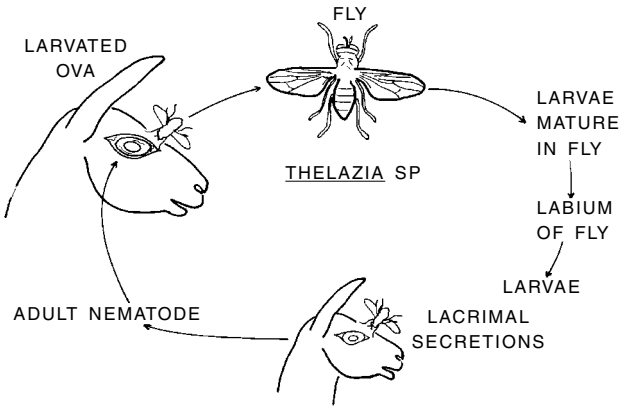

Figure 7.52 Life cycle of *Thelazia californiensis*.

Eye Worm

***Thelazia californiensis, Thelazia* spp.**

The eyeworm, *Thelazia californiensis*, is a spirurida nematode found in the conjunctival sac of deer, elk, cattle, sheep, dogs, cats, foxes, rabbits, llamas, and humans. See Table 7.5 for measurements.

Larvated ova are found in the lacrimal secretions. Muscoid flies, such as the face fly (*M. autumnalis*), ingest the ova or larvae. L2 larvae migrate to the fly's ovarian follicles and mature to L3 in 15–30 days (Figure 7.52). L3 larvae migrate from the follicles to the labia of the fly, and infective larvae are deposited in another eye when the fly feeds on lacrimal secretions. Prepatent period is 16–20 days. This parasite is transmitted during fly season [3].

In camelids with *Thelazia,* excessive lacrimation may be the only sign noted. Nematodes may be seen on the surface of the cornea or in the conjunctival sac (7.53). They may lodge beneath the nictitating membrane or be found in the nasolacrimal duct. With large numbers, a mild conjunctivitis may develop. The parasites can be mechanically removed under sedation with a local anesthetic. Diethylcarbamazine in a concentration of 2 mg/L may be instilled into the conjunctival sac. Ivermectin drops also may be instilled into the conjunctival sac.

Figure 7.53 *T. californiensis* in the conjunctival sac.

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8

Multisystem Disorders

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Neoplasia

Sporadic cases of neoplasia are widely reported in camelids with many types of neoplasia, affecting multi-organ systems [1–28]. Lymphosarcoma is the most common type of neoplasia reported [4, 28]. Only one report found squamous cell carcinoma (SCC) to be a slightly more prevalent malignant neoplasia [27]. More advanced immunostaining has revealed a small percentage of cases diagnosed as lymphosarcoma are better classified more broadly as malignant round cell tumors (MRCT) [1, 14]. For the purpose of this text and clinical practice, the term lymphosarcoma will be used to refer to all MRCT.

The clinical course for lymphosarcoma is usually very short from onset of clinical signs until euthanasia or natural death. Reported clinical signs of lymphosarcoma include anorexia [4, 23, 28], depression [4, 21, 23], weight loss [4, 21, 23, 26, 28], peripheral lymphadenopathy [4, 21, 26, 28], pleural effusion [26], dyspnea [21, 28], weakness/recumbency [17, 21, 28], acute death [21], polyuria/polydipsia [23], and diarrhea [23]. Most cases are multicentric with the reported sites including peripheral lymph nodes [1, 4, 17, 21, 23], liver [1, 4, 17, 21, 23], kidneys [1, 4, 17, 21, 23], lungs [1, 4], heart [4, 17], mediastinum [1, 21], mesentery [1, 17, 21], gastrointestinal (GI) tract (Figure 8.1) [17, 23], spleen [1], bone marrow [1], and pancreas [1]. Camelids can be affected by lymphosarcoma at any age but, especially alpacas, are often very young with the youngest reported age being six weeks [21] and a median reported age among alpacas and llamas being 4.5 years [4, 27]. One report of multicentric lymphosarcaoma in a juvenile alpaca also identified an infection with bovine leukemia virus (BLV) though causation could not be established [12]. Other reports failed to identify retroviral particles [4]. One case of acute myeloid leukemia with multilineage dysplasia has been reported in an 18-year-old alpaca [24]. Diagnosis of lymphosarcoma or other neoplasia is made by histopathology. No treatments have been reported.

Stress

Stress is the cumulative response of an animal to its environment and is an adaptive phenomenon [29]. All responses are primarily directed at coping with environmental change. Each reaction to a stressor has adaptive significance. Intense or prolonged stimulation induces detrimental responses that may be fatal in the camelid.

The lesions produced by harmful stress are difficult to document. Many of the effects of stress are functional, leaving no definitive lesion to mark their presence. Nonetheless, it is known that tissues and organs are weakened by prolonged insult, lowering resistance to disease. Classic lesions are lymphoid tissue atrophy, adrenal cortical hyperplasia, and GI ulceration. Though the actual cause of death may be pneumonia, parasitism, or starvation, stress may have paved the way for development of these terminal ailments [30].

Veterinarians providing healthcare for camelids should consider stress as a contributory factor to specific diseases. Husbandry practices should be evaluated and those that may be harmful corrected. Camelids are social animals. Isolation for therapy or recuperation may be counterproductive.

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Figure 8.1 Lymphosarcoma of abdominal organs.

Malnutrition is a stressor, as are repeated restraint episodes especially in animals unaccustomed to being handled (see Chapters 2 and 3).

Hyperthermia

Thermoregulation

South American Camelids (SACs), originally adapted to a cool environment, have been introduced to a wide variety of habitats in North America and other countries. They have demonstrated a general adaptability to a wide variety of climates. The ventral regions of the body are sparsely covered with hair and have a higher concentration of sweat glands [31], providing a thermal window for dissipation of heat. Evaporation is their primary cooling mechanism. Dirty, matted, excessively long, or wet fiber can prevent evaporation of water from the skin thereby inhibiting dissipation of heat [32]. Studies that compared sheared and non-sheared alpacas showed that sheared alpacas are more heat tolerant than non-sheared alpacas, especially in humid climates [33, 34].

Behavioral activities that aid SACs in thermoregulation include seeking shade, orientation to minimize radiant heat gain, standing to maximize evaporative cooling, recumbency when in sunlight to cover the thermal window, and standing in a stream or pond (Figure 8.2).

Predisposing Factors

Heat stress is a significant problem in llamas and alpacas, especially in the southern and western United States. Prolonged exposure to high environmental temperatures and humidity lead to heat stress illness. One formula that has been used for determining risk of a heat stress-related event is to add the temperature in °F to the % relative humidity; <120 equals low risk, >150 increases risk of heat stress significantly, and >180 poses extreme danger [35].

In addition to environmental conditions, predisposing factors for development of hyperthermia in SACs include muscular exertion, fever, dehydration, and drugs that depress thermoregulation. Activities that may contribute to the production of body heat include packing, racing, breeding, fighting, being transported, prolonged restraint, being chased by predators, or breeding males pacing a fence [32].

Some animals are at increased risk of hyperthermia because of low heat tolerance. Older animals may have decreased sweat gland activity, be in poor physical condition, or have deterioration of cardiovascular function. Obesity is another significant risk factor. Evaporative cooling is determined by the number of sweat glands per square meter of skin surface. Metabolic heat is produced in proportion to body mass but is dispersed in proportion to skin surface area. Thus, obese animals are at a distinct disadvantage in hot weather [36].

Figure 8.2 Hyperthermic alpaca cooling herself in a pond.

Clinical Findings

The normal core body temperature of adult SACs ranges from 99.5 to 101.5 °F [32]. The normal temperature of neonates may be a degree higher than that of adults. The degree of hyperthermia and effects of hyperthermia on organs and tissues may vary according to the duration of exposure to excessive heat. Body temperatures in llamas and alpacas are rarely noted above 110 °F; in fact, animals in severe heat stress may have temperatures of only 106–108 °F [32]. Protein denatures at temperatures of 113–116.8 °F, causing immediate death of tissues and organs [32].

Illness from heat stress has been described as a continuum from mild heat stress to heat exhaustion to heat stroke [37]. Clinical signs in camelids range from mild and non-specific to severe leading to death. Other than elevated body temperature, clinical signs of heat stress include anorexia [38], depression [38], tachypnea or respiratory distress [35, 39], tachycardia [35, 39], inability or unwillingness to rise [38, 39], sweating or anhidrosis depending on severity of hyperthermia and dehydration [35, 38], salivation [35], drooping of the lower lip [35], scrotal and/or ventral edema (Figure 8.3) [38], convulsions [35, 38], collapse [35], coma [35, 38], and death [35]. If the animal survives, wide fluctuations in body temperature often follow initial correction of hyperthermia [39]. Clinicopathologic changes often include increased CK and AST [39], hypoproteinemia [39], metabolic acidosis [39], severe hypokalemia and other electrolyte imbalances [39].

The following outlines the likely clinical progression of a severe hyperthermic episode in a llama or alpaca:

1) Elevation of the core body temperature
2) Accelerated heart rate
3) Increased respiratory rate
4) Redness of skin surface
5) Sweating
6) Hemoconcentration
7) Body fluid shift from viscera and muscle to skin (edema)
8) Decreased glomerular filtration
9) Decreased central venous pressure
10) Effects on the CNS, including cerebral hypoxia and coagulative necrosis
11) Coagulation defects (DIC)
12) Multi-organ dysfunction
13) Death.

Some long-term effects of hyperthermia can be seen, especially in the reproductive system. Heat stress may have a marked effect on the adult female, including diminished intensity of receptivity and anestrus. During pregnancy, the more profound effects are seen as fetal damage, including inhibition of embryonic cleavage and implantation, initiation of teratogenesis, and abortion [40, 41]. In males, excessive heat is spermicidal, at the primary spermatocyte stage. In much of the southern and western United States, it is common for males to become infertile during the hot weather of summer. The scrotum is not pendulous as it is in most domestic ruminants, thus core body temperature changes have a rapid and profound effect on developing spermatids. Scrotal edema is a classic sign of hyperthermia in male SACs.

Persistent low-intensity hyperthermia may also cause decreased digestive function which, in turn, may cause poor growth rates in juveniles and poor appetite and less efficiency in feed utilization in adults.

Necropsy Findings

The insulating fiber coat prevents dissipation of normal body heat, and when this is complicated by hyperthermia, autolysis is rapid. Gross lesions suggestive of death from

(a)

(b)

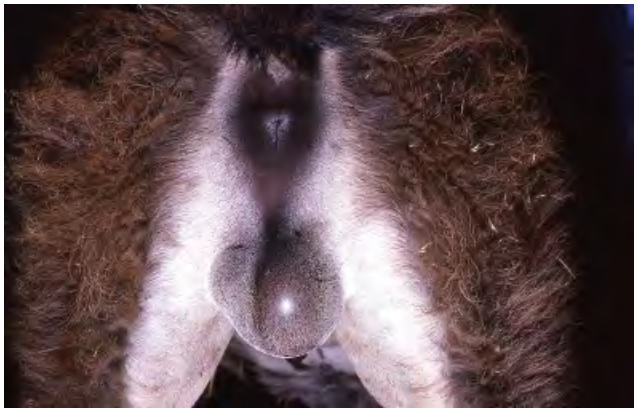

Figure 8.3 Bilateral scrotal edema caused by hyperthermia (a) compared to unilateral scrotal swelling caused by a scrotal hernia (b).

hyperthermia include serosal hemorrhages, evidence of DIC and congestion and autolysis especially of the brain, liver, and kidneys [38].

Therapy

Successful resolution of heat stress and its sequelae requires early recognition, rapid decreasing of the core body temperature and supportive care. The first step in cooling should be to remove the animal from the extreme environment into a cool environment. An air-conditioned room should be used if possible or at least provide shade and fans. Cold water or ice packs should be applied to the ventral abdomen, including the axillary and inguinal spaces. Soaking the fleece of heavily fibered animals is not effective in cooling. Shearing can be attempted if it can be done without excessive stress to the animal. Cold water enemas are sometimes recommended, but the ability to monitor the body temperature is lost. Immersion in very cold/ice water can be used for rapid cooling, however, in small animal medicine this is strongly recommended against as the vasoconstriction to the skin can stop core heat dissipation [42]. Human medicine literature acknowledges this, but still recommends ice baths in young people with exertional heat stroke [37]. If immersion in cold/ice water is used, massage of the skin is recommended to increase blood flow to the skin. There are no studies in large animal or camelids on the success of different cooling methods but this vasoconstriction mechanism should be considered. Using room temperature water in the immersion bath may be more beneficial.

Intravenous (IV) fluid therapy is very important, especially in more severe cases. Fluids should be tailored to address hemodynamics and electrolyte and acid-base derangements. Care should be taken not to be overly aggressive with fluids as many of these animals are hypoproteinemic and are prone to pulmonary and cerebral edema.

Anti-inflammatories will not directly affect body temperature in these cases but may be beneficial in combatting a secondary inflammatory response. Waiting until circulatory volume and pressure have been restored is advised. Vitamin E can be used as a free radical scavenger. Broad-spectrum antibiotics such as ceftiofur is indicated due to the risk of bacterial translocation from the GI tract. Once stable, diagnosing and treating any underlying condition should be attempted.

Prevention

The risk of heat-related illness can be mitigated by appropriate environmental provisions such as shade, fans in barns, and plenty of potable water. Restraint procedures should be avoided on warm days. Clients should be advised against heavy work for packers during the afternoons on hot days. Shearing before the hot season is recommended.

Hypothermia

Though SACs are adapted to the cool temperatures of their native lands, temperatures there do not reach the extremes found in some areas of North America. Neonates are particularly susceptible to hypothermia because of their greater proportion of skin surface area and minimal fat stores. Adult SACs under anesthesia or in shock are also at significant risk for hypothermia. Other predisposing factors include insufficient feed intake which reduces metabolic heat production, restricted muscular activity which prevents heat generation, and poor fiber coat that provides less insulation.

Many clinical thermometers record body temperatures only as low as 92 °F. With more sensitive thermometers, temperatures as low as 85 °F have been recorded in living SACs [30]. Other than decreased body temperature, common clinical signs of hypothermia include severe depression, recumbency, and signs of shock. Frostbite, most frequently of the ear tips, can occur with intense or prolonged skin exposure to cold.

Therapies for hypothermia should be aimed at rewarming and cardiovascular support. Rewarming can be accomplished by several means. Covering the animal with blankets and ensuring the animal is not directly on the cold floor or ground helps conserve heat. The next step is to add heat. Circulating water-type heating pads are effective, but electric heating pads have caused skin burns and sloughs. Hot packs can be placed directly on and around the animal or may be used to raise the ambient air temperature in a small, enclosed area. Be cautious when applying heat directly to the skin.

The air surrounding the patient may be warmed with infrared heat lamps, forced-air driers, or electric floor heaters. A hairdryer may be helpful in warming a neonate but be aware of evaporative cooling. A warm water enema is highly effective, but the ability to monitor the body temperature is temporarily lost.

Total body immersion in warm water (100–105 °F) is the fastest way to warm an animal, but the animal must be quickly dried afterwards to avoid evaporative cooling. This is difficult in animals with fiber.

IV fluid therapy is used for cardiovascular support. Warming IV fluid bags and administering through a standard IV administration line has been found to be unhelpful in preventing or treating hypothermia because the fluid's heat is lost to the environment as it passes

through the line [43]. Using an IV line warmer or passing the IV line through hot water immediately before it enters the patient may be more useful, but this has not been studied.

Fluid Therapy in Llamas and Alpacas

Fluid therapy is a fundamental component of medical and surgical therapy. When designing a fluid therapy plan, the first step is to determine the route of administration. In camelids, as with traditional ruminants, oral administration through orogastric intubation can be used effectively for mild dehydration when IV administration is not feasible. Oral administration of fluids is a stressful event that many camelids do not handle well. Repeated orogastric intubation is not recommended. Animals with severe dehydration, those requiring ongoing fluid therapy, those requiring ongoing electrolyte or acid/base support, or those in cardiovascular shock require IV fluid administration. IV fluid administration can be performed as a constant rate infusion (CRI) or fluid boluses can be given every 2–4 hours if circumstances do not allow for a CRI. When using fluids for bolus administration, care should be taken when supplementing potassium or calcium as rapid administration of these can have detrimental effects.

After determining whether to treat orally or IV, maintenance needs, dehydration/hypovolemia, ongoing losses, and electrolyte and acid base derangements need to be considered.

Water requirements for camelids is discussed extensively in Chapter 3 and represented in Table 3.2. In a neutral environment, daily water requirements have been determined to be 42.5 mL/kg$^{0.82}$, which for an average adult SAC is between 35 and 40 mL/kg/day [44]. As in other adult ruminants, 50 mL/kg/day (1 mL/lb/hr) is commonly used in clinical practice for maintenance fluids and 80–100 mL/kg/day is used in neonatal patients.

Dehydration can be difficult to assess on physical exam. Clinical signs associated with dehydration include muscle weakness, depression, enophthalmos, tacky, or dry oral mucous membranes, decreased urine production, elevated heart rate, decreased pulse pressure, and difficulty in distending a vein for IV therapy. Decreased skin turgor is used somewhat reliably in other species, but thickness of the skin makes this more difficult to assess in camelids. Change in body weight would be the most reliable indicator of dehydration but is impossible unless a recent, accurate, hydrated body weight is known. Hemoconcentration is a good indicator of dehydration; however, because hypoproteinemia and, to a lesser extent, anemia are very common in ill camelids. Normal hematocrit and protein concentration do not rule out dehydration. Animals with moderate or prolonged dehydration may have evidence of pre-renal azotemia on a chemistry panel. Severity of dehydration is usually estimated (somewhere between 5 and 12%), treated, and reassessed. Ongoing losses are primarily an issue only when high-volume diarrhea is present; if present, volume is estimated and added to the fluid plan (see Chapter 20).

As in other species, fluid volume to administer is calculated by adding maintenance requirements, the water deficit and estimated ongoing losses. The water deficit is calculated by the following formula:

$$Water\ deficit\ Volume\,(L) = Body\ weight\,(kg) \times \%\ dehydration$$

Some advocate for giving replacement (dehydration) volume over the first few hours, and the rest of the volume in the remainder of the 24-hour period. Because many sick camelids are hypoalbuminemic, slower replacement may be of benefit. This can be done by providing the total calculated maintenance and replacement volume as a CRI over the entire 24-hour period or, even slower, by providing maintenance fluids over each 24 hour period and spreading the replacement of dehydration over 48 hours. Slow fluid replacement is especially critical in neonatal crias (see Chapter 20).

In cases that require cardiovascular resuscitation, rapid bolus administration may be necessary. In these cases, 20 mL/kg boluses can be administered and repeated every 15–30 minutes until adequate perfusion is achieved. As previously mentioned, rapid administration of fluids is not without risks, namely worsening hypoproteinemia and edema.

In most cases, commercially available solutions used to treat other species of domestic animals are appropriate for camelids. Sick camelids are prone to sodium, chloride, and potassium derangements; therefore, concentrations of these electrolytes may need to be adjusted in commercially available fluids, especially in the case of hypernatremia or hypokalemia. Hypokalemia is commonly addressed by adding potassium chloride (KCl) at 20–60 meq/L to commercially available fluids. The rate of administration for KCl should not exceed 0.5 meq/kg/hr. At a reasonable CRI fluid rate, potassium supplementation is not usually a problem but administering fluid boluses with added potassium chloride can be dangerous.

In animals with significant hypernatremia, rapid decrease in plasma sodium can lead to osmotic influx of water into the cells, most importantly, the brain cells. To prevent rapid change in osmotic gradient, the goal is to reduce plasma sodium by no more than 0.5 meq/L/hr

(12 meq/L/day). This sometimes necessitates adding sodium, usually in the form of hyptertonic saline, to commercially available fluids to bring the sodium concentration closer to the plasma sodium concentration of the patient. See the section on hyperosmolar syndrome in Chapter 13 for a more thorough discussion on correction of hypernatremia.

Acid-base derangements are common but, unless severe, often resolve themselves with standard fluid therapy and treatment of the underlying disease. Commercially available balanced solutions that contain lactate, acetate or other buffers are slightly alkalinizing to the patient, while normal saline is slightly acidifying. Camelids and other ruminants are much more likely to become alkalotic in disease than acidotic; however, acidosis can be much more serious.

Significant acidosis often has to be treated by replacing the bicarbonate deficit directly using the formulas below. Once calculated, ¼ to ½ of the deficit can be administered rapidly, followed by correcting another ¼ to ½ over the following 12–24 hours. All deficit may not need to be replaced as improving perfusion through hydration alone can correct some level of acidosis.

$$Body\ weight_{Adult}\left(kg\right)\times 0.3\frac{L}{kg}\times Base\ deficit\left(\frac{mEq}{L}\right) = Bicarbonate\ deficit\left(mEq\right)$$

$$Body\ weight_{Neonate}\left(kg\right)\times 0.5\frac{L}{kg}\times Base\ deficit\left(\frac{mEq}{L}\right) = Bicarbonate\ deficit\left(mEq\right)$$

Failure of Predicted Growth and Weight Loss

Normal growth rates have been established for llamas but not alpacas [45–48]. Table 8.1 represents the mean body weight data of llamas from birth to 24 months of age from one study [46].

When weight loss is detected in an adult camelid, a detailed history, physical examination, and laboratory evaluation will often lead to a diagnosis. However, juvenile SACs often present with a syndrome of failure to grow and poor body condition commonly referred to as "failure to thrive" or "ill thrift." Differentials for this syndrome include inadequate milk production by the dam, failure to convert to a solid diet, rickets (hypovitaminosis D and hypophosphatemia), parasitism (coccidiosis, GI nematodiasis), immunodeficiency, chronic infection, and C1 or C3 bezoars. Efforts have been made to find a single etiologic agent causing these syndromes. Though

Table 8.1 Growth of the llama.

Age (months)	Mean body weight (kg)	Age (months)	Mean body weight (kg)
1	21.9	13	87.5
2	31.9	14	91.1
3	39.7	15	94.5
4	46.3	16	97.9
5	52.3	17	101.1
6	57.7	18	104.3
7	62.7	19	107.4
8	67.3	20	110.4
9	71.8	21	113.3
10	75.9	22	116.2
11	80.0	23	119.0
12	83.8	24	121.8

Source: Smith et al. [46].

suspected, an inherited immune deficiency has not been verified. Other studies have implicated thyroid dysfunction or iron deficiency anemia. These may contribute to a syndrome but have not been proven to be the primary cause. Many crias in this category have a common history of a poor start to life, being born in undesirable circumstances and receiving inadequate colostrum. Many "failure to thrive" crias struggle despite receiving appropriate intervention such as a plasma transfusion and are very susceptible to chronic infections or parasitism. Unfortunately, despite extensive medical work-up the outcome is often disappointing. There are some anecdotal treatments for unexplainable failure to thrive crias including cria shearing if weather appropriate (these crias often have very high fiber quality) and thyroid supplementation, especially if the thyroid panel is suggestive of inadequate levels.

An immunodeficiency syndrome (Juvenile Llama Immunodeficiency Syndrome, JLIDS) was first described in the early 1990s as a cause of ill thrift in llamas and has since been further characterized [49–51]. Signs that are suggestive of an immunodeficiency disorder included repeat infections (poor response to therapy), high susceptibility to low-grade pathogens, lymphoid hypoplasia/atrophy, and infections with organisms rarely observed in immunocompetent animals. Necropsy lesions included marked thymic hypogenesis/atrophy, lymph-node hypoplasia, and chronic inflammatory lesions in various organs. Lymphocyte blastogenesis assays indicated a severe defect in both T-cell and B-cell lymphocyte proliferative responses (see Chapter 14). This syndrome has not been described in alpacas.

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9

Integumentary System

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The integumentary system consists of the skin, hair (fiber), mammary glands, adnexal glands (sebaceous, sweat, scent), and toenails. Its main function is to act as a barrier to protect the body from external influences. It also functions to retain body fluids, protect against disease, ectoparasites and regulate body temperature. The ear and related issues will also be discussed in this chapter.

Normal Camelid Skin

The skin of the camel, llama, and alpaca have been studied and have been shown to be similar histologically to each other and to those of other domestic herbivores [1–3]. Even the camel, adapted to the hot, arid environment does not appear to have significant adaptations of the integument compared to that of their non-desert counterparts [3]. The physical characteristics of the skin vary with region of the body but tends to be thick and non-pliable. Males tend to have thicker skin than females, especially over the cervical region which provides protection from cervical trauma but can also make jugular venipuncture challenging [1]. Crias appear to have excessive skin manifest as wrinkles, most heavily seen on the neck and tail base. These are most prominent in Suri alpacas and are associated with a very high follicular density and hence a dense fleece, desirable by most producers. Clinically, this has application because it can make jugular venipuncture and IV catheter placement more difficult.

The skin serves as a protective barrier between the body and external environment and is an important organ for homeostasis, preventing excessive water loss and invasion of the body by pathogenic microorganisms. The flexibility and elasticity of the skin allow motion and provide shape and form. The skin also plays a vital role in thermoregulation and sensory perception.

The skin contains both sebaceous and sweat glands, which serve a secretory function. Melanin provides the variety of colors found in camelids. Precursors for vitamin D_3 synthesis in the skin are acted upon by ultraviolet light to produce the vitamin that is later converted to 1, 25 dihydroxycholecalciferol, the active hormone, via metabolism in the liver and kidney. Scent glands are located in the interdigital spaces and the medial and lateral metatarsal regions. Callosities form over the sternum, carpus, and stifle in response to recumbency patterns.

The layers of the skin are illustrated diagrammatically in Figure 9.1. The skin can be largely comprised of two layers: the dermis and the epidermis. The dermis, the deep layer, comprises the bulk of the skin's thickness, and is subdivided into the superficial, vascular papillary dermis and the thicker, dense connective tissue layer, the reticular dermis. In addition to the neurovascular structures, the dermis contains lymphatics and adnexal structures such as the hair follicles and glandular tissues. The epidermis covers the dermis and is attached tightly to the dermis by hemidesmosomes and type IV anchoring fibrils. The epidermis provides the water-tight barrier and first line of protection from the external environment. The epidermis is comprised of specialized epithelial cells called keratinocytes that form keratin. Keratin offers protection and forms a relatively water-impervious barrier.

The epidermis of most animals is comprised of five-layers of keratinocytes histologically. From deep to superficial, these are the stratum basale, stratum spinosum, stratum granulosum, stratum lucidum, and the stratum corneum. Though camelids share this same organization of keratinocyte layers, the stratum lucidum is variable and

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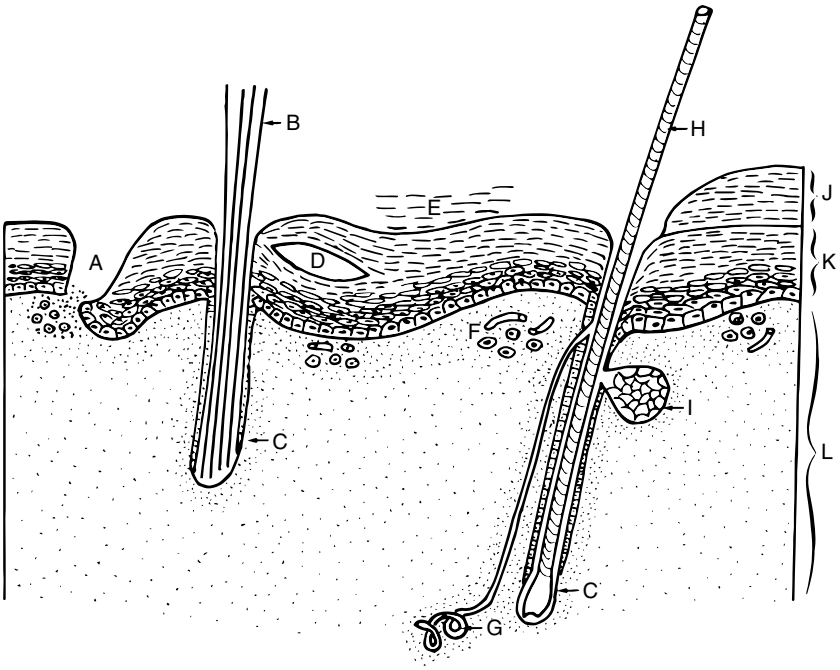


Figure 9.1 Diagram of the skin. (A) Laceration extending into the dermis, (B) multiple fibers emerge from a single hair follicle, (C) hair follicle, (D) vesicle, (E) desquamated epithelium (dandruff), (F) dermal blood vessels, (G) sweat gland, (H) fiber, (I) sebaceous gland, (J) hyperkeratosis, (K) normal epidermis, (L) dermis.

frequently absent in camelids [1–4]. The *stratum corneum* (horny layer) represents one-half to three-fourths of the total epidermal thickness. This layer consists of anuclear remnants of flattened, fully keratinized cells pushed up from basal layers. A *stratum lucidum* is occasionally seen in sparsely haired skin as a dense eosinophilic layer beneath the *stratum corneum*. The *stratum granulosum* (granular layer) is a single layer of cells in some areas and discontinuous in others. The nuclei are pycnotic, and most of the cytoplasm has been replaced with keratin. The *stratum spinosum* (prickle layer) is reduced in camelids but is composed of daughter cells of the basal layer and is one to three cells thick. These cells are viable and nucleated and actively synthesize keratin [5]. The *stratum basale* (basal layer or *stratum germinativum*) is the deepest layer of the epidermis and consists of a single layer of cuboidal or columnar cells, most of which are keratinocytes with a few melanocytes. The keratinocytes are capable of true regeneration as the epidermis is constantly renewed throughout the life of the animal. Because of this regeneration, partial thickness skin wounds that only affect the epidermal layers can heal without scarring back to completely normal tissue.

Melanocytes contain melanin pigment in pseudopods distributed between epidermal cells of the skin and hair. Skin color is determined by the number, size, arrangement, and dispersion of melanin granules [6, 7]. In chronic dermatitis, there may be overproduction of melanin, which causes darkening of the skin. Conversely, depigmentation may result from trauma, burns, or infection of the skin. Albinism occurs in camelids as it does in all species.

The dermis (corium) of camelids is thick (up to 1 cm in the cervical region of a mature male) and consists of a superficial layer comprised of loose connective tissue interdigitating with undulations in the epidermis and the deep dermis, comprised of dense fibrous tissue. The dermis contains hair follicles, blood and lymph vessels, nerves, and sebaceous and sweat glands. The middermis of camelids is characterized by a proliferation of blood vessels, in contrast to that in other domestic animals. Vessel walls are hyalinized. This normal histologic picture is frequently misinterpreted as an abnormal vascular proliferation. There is variation in the thickness of the epidermal layers in various areas of the body. Furthermore, the degree of vascularity and mononuclear infiltration may vary.

The hypodermis (subcutis) is comprised of loose connective tissue, which attaches the skin to the underlying bones or muscles. Some sweat glands extend into the hypodermis.

Hair (Fiber, Wool)

In North America most camelid producers refer to the haircoat as “fiber” and may call it the “fleece” when it is referred to as a whole unit on the animal or after it is shorn

Table 9.1 Definition of terms used to describe hair, wool, and camelid fibers.

Cotting – The entanglement of fibers into an irreversible matt.
Crimp – The natural waviness in a wool fiber.
Felting – Producing a compact, irreversibly entangled structure when subjected to friction in a soluble medium.
Fiber – The hair of animals other than sheep.
Fleece – The wool that covers a sheep or similar animal. The quantity of wool cut from a sheep at one time.
Kemp – Kemp fibers, guard hairs are coarse, relatively flattened fibers with a long tapering tip. They are periodically shed.
Luster – Silver luster from fine merino wool, silk luster in medium-to-long wool, and glass luster in mohair and other goat fibers. Luster – Suri fiber, Brightness – Huacaya
Staple – (i) Locks or tufts of wool shorn from a sheep. (ii) Natural fibers or cut lengths from filaments, also called staple length.
Suint – That part of the new fleece that is soluble in cold water. The dried residue of the sweat glands.
Wool – The fine, soft, curly hair forming the fleece of the sheep characterized by the property of felting due to the overlapping of minute surface scales.
Wool wax – The fatty product obtained from wool. Also known as wool fat or wool grease. Purified form known as lanolin. Source of allergies to wool. Produced by the sebaceous glands. Not seen in camelid fiber.

off. Technically, the term "wool" should be restricted to the fiber from sheep [6, 8–16]. Wool is the fine, soft, tightly crimped, usually non-medullated hair forming the fleece of sheep. Camelids have medullation like hair, with the finer fiber having an interrupted medullation, or none at all. This gives it a wool-like hair characteristic without the negatives of true wool (lanolin, "prickle-factor"). In South America, the term used is alpaca or llama fiber, and hair and fiber will both be used in this chapter. Table 9.1 defines terms used to describe hair.

The hair coat of llamas consists of two types, the coarse guard hairs (primary hairs) and a finer undercoat (secondary). The finer undercoat is 20–40 μm in diameter with the guard hairs comprising approximately 20% of the fleece and are 140–150 μm in diameter [17]. The finer undercoat can be used to make handicrafts and garments, etc. Guard hairs must be removed from the fleece prior to spinning and garment manufacture. Guard hairs can be used to produce rugs, wall–hangings and very sturdy ropes (Figure 9.2). North American llama owners use different terms to describe the types of llama with regards to how much fiber is on the llama, especially on the legs. Short/light wool llamas have guard hairs on the body/neck but minimal wool on the leg below stifle and minimal on the face. These are referred to the Q'aras' [18] type in Peru (Figure 9.3a and b).

Figure 9.2 Handmade rope of llama fiber from Peru.

Medium wool llamas have fiber that extends down the leg, with minimal coverage below the carpus and hock. Heavy wool llamas called Chaku [18] type in Peru (Figure 9.4), will have fiber on the front legs to the level of the carpus and somewhat less below, with rear legs coverage down to pasterns on the back of the leg. Additional terms used are single and double coat (those with guard hairs). Other phenotypes have been developed through careful breeding such as "Suri" and "Silky." Llamas come in a wide variety of colors and mixes of colors and patterns. Colors range from white to black with every color in between such as fawn, brown, gray, rose gray, cream, etc. No one color or pattern is preferred over another, just personal preference.

There are two breeds of alpaca. The Huacaya (Figure 9.5a and b) has a shorter fleece with moderate crimping of the hairs, while the Suri (Figure 9.6a and b) has a longer fleece with no crimping and it grows in locks. There are many different lock formations depending on genetics (straight, twisted, flat, fan, curled). The haircoat of most Suri alpacas consists of only finer undercoat fibers (secondary). Huacayas have a small amount of guard fibers and it is considered an undesirable trait to have too many. In alpacas, it is desirable to have fiber coverage extend down to the feet, though not all reach this goal. As in llamas, there is a wide range of colors and patterns present in alpacas. The Alpaca Owners Association (AOA) recognizes 16 official colors for alpacas ranging from white to black and all colors in between. The show industry is popular for both alpacas

(a) (b)

Figure 9.3 Q'aras' type llamas at Machu Picchu in Peru.

Figure 9.4 Chaku type llama. Photo courtesy of Donna Moore, Timberlane Llamas, OH.

and llamas. The fiber of alpacas is used in a wide variety of products, such as scarves, gloves, coats, sweaters, worsted men's suits, hats, rugs, fiber art, etc.

In Peru, alpacas are shorn every year. Previously, the fleece was allowed to grow up to 18 months, but it has been found that fiber production is increased with more frequent shearing. Also, the fiber was very matted and harder to process. Alpaca and llama fiber are slow growing, and this fact should be kept in mind when contemplating clipping the haircoat from an area for diagnostic or therapeutic purposes. When llamas first came to North America it was not routine to shear them every year but to harvest the fiber by brushing. Llamas have only a partial annual shed while wild South American Camelids (SACs) such as guanaco and vicuña and Old World Camels (OWCs) shed annually. Due to the hot humid weather in North America, shearing is currently a more common management tool with llama producers. However, some producers will only shear the barrel area (chest and abdomen) and leave the neck unshorn (Figure 9.7). As considerable heat is still trapped by the long fiber of the neck after several years' growth, it is advisable to shear the neck every two years. This is also helpful from a veterinary point of view when jugular blood draws and catheter placement are needed. Alpacas are routinely sheared annually for health reasons and to harvest the fiber for production of yarn and other products. Alpacas are what is termed "slick-shorn" with all the fiber removed from the neck and body and the slower growing fiber on their legs only trimmed (Figure 9.8).

The density of hair varies with the location on the skin, with the thickest coat over the back and sides of the animal and the neck [19]. Fiber management and harvesting are

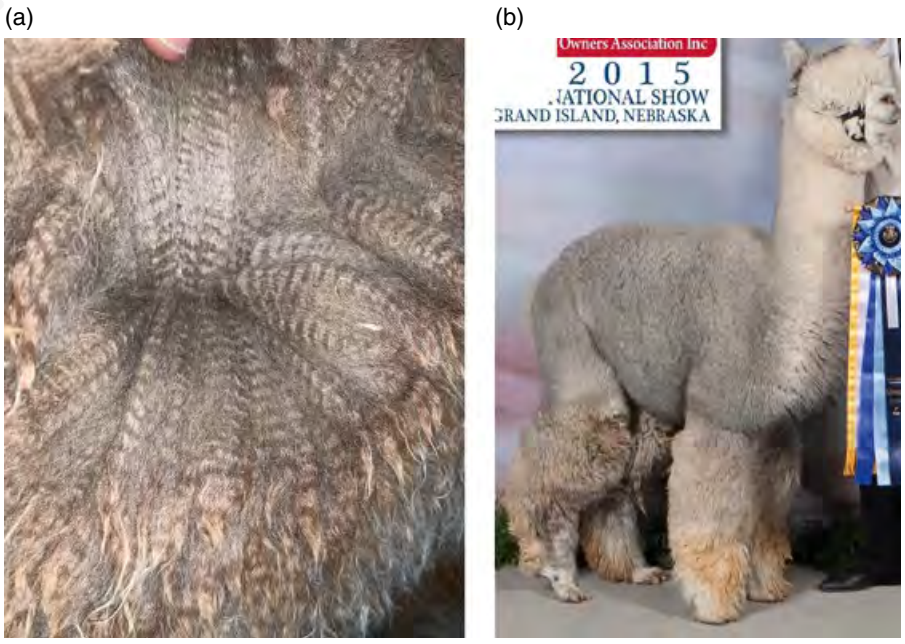


Figure 9.5 (a) Huacaya fiber showing crimp. (b) Huacaya in full fleece. Photo courtesy of Nancy Billington, Fun In the Country Alpacas, MI.

Figure 9.6 (a) Suri fiber showing locks. *Source:* Photo courtesy of Becky Camma, The Alpacas of Spring Acres, OH. (b) Suri in Full fleece. Photo courtesy of Lindsay Warne, The Alpacas of Spring Acres, OH.

important factors in alpaca management, but this has little medical significance other than to deal with hyperthermia. More detailed information about fiber production is found in *The Complete Alpaca Book* [17].

Hair follicles are tubular invaginations of the epidermis into the dermis. In camelids, there are two types: simple primary follicles containing a single coarse hair and complex secondary follicles containing multiple fine hairs (Figure 9.9a–c). The density of fibers varies with genetics, with greater density being desired for show animals. Both sebaceous and sweat glands empty into the hair follicles [1, 19]. All sebaceous glands are paired with hair follicles with the size and number increasing at mucocutaneous junctions and in the ear. There are none on the foot pads [1].

Figure 9.7 Llama with lion cut. Photo courtesy of Barb Baker, Baker & Company Llamas, VA.

There are sweat glands over the entire body with more located in ventral regions of the body where the skin is thinner (thermal windows).

The fine hairs of the undercoat of llamas and alpacas have three distinct layers: the medulla, cortex, and cuticle [11, 17, 20]. In contrast to sheep hair, camelid hairs are strongly medullated, except for most of the guard hairs. The cortex is circular to elliptic in cross section. The cuticular layer is evident within the follicle but becomes flattened as the hair emerges.

Crimp is the natural waviness in wool fiber. The crimp and scaled cuticle of sheep wool hairs provide excellent binding of the hairs as they are spun into yarn. The crimp also provides resiliency, the garment conforming to changing shapes as it is worn. Only Huacaya alpaca fiber shows crimp; it is an undesirable trait in Suri alpacas.

A pure alpaca hair garment does not resume its original shape if stretched. Special procedures must be used to spin alpaca hair and, frequently, a percentage of fine sheep wool (merino), bamboo or silk fibers are mixed with it to ease spinning and knitting and to improve garment usage. Llama hair is coarser and has no crimp. Garments made from llama hair have a harsher feeling than those made from alpaca hair. North American llama breeders are selecting individuals with finer fleece to improve the quality of the llama fiber produced.

A cottage industry for making llama-hair garments has developed in North America, while, in South America,

Figure 9.8 Alpaca "slick-shorn" Photo courtesy of Lindsay Warne, The Alpacas of Spring Acres, OH.

llama hair is handled locally by the native people and used to make blankets, ropes (Figure 9.2), bags (corresponding to burlap or jute bags) and inexpensive garments. Native people say that llama hair sweaters are warmer than those made from alpaca hair but recognize that llama hair is harsher and may cause the skin to itch.

Fine garments are made from pure alpaca hair or alpaca hair mixed with fine sheep wool or synthetic fibers. In North America, producers are using local mills to sort, grade, clean, and spin camelid fiber into yarn. Some of these mills go on to make products for sale (rugs, scarves, shoe inserts, etc.). Many producers have a farm store where products, yarn and roving's (unspun yarn) are for sale locally or on the internet. Much of the manufacturing of mass-produced finished products is still done in South America.

Hair Quality

Hair quality may be modified by such environmental factors as day length, temperature, elevation, and general nutrition, but the dominant factor in determining quality is heredity.

Hair Loss

It is often difficult to establish the etiology of patchy or generalized loss of hair/fiber in SACs. Certain specific diseases, such as mange cause alopecia, but pediculosis does not, unless the animal rubs the hair from such locations as over the spinous processes of the vertebrae [21]. Alopecia may also occur at other sites of excessive wear, such as alongside the necks of animals fed in a slatted manger or along the nose when using a slatted hay guard.

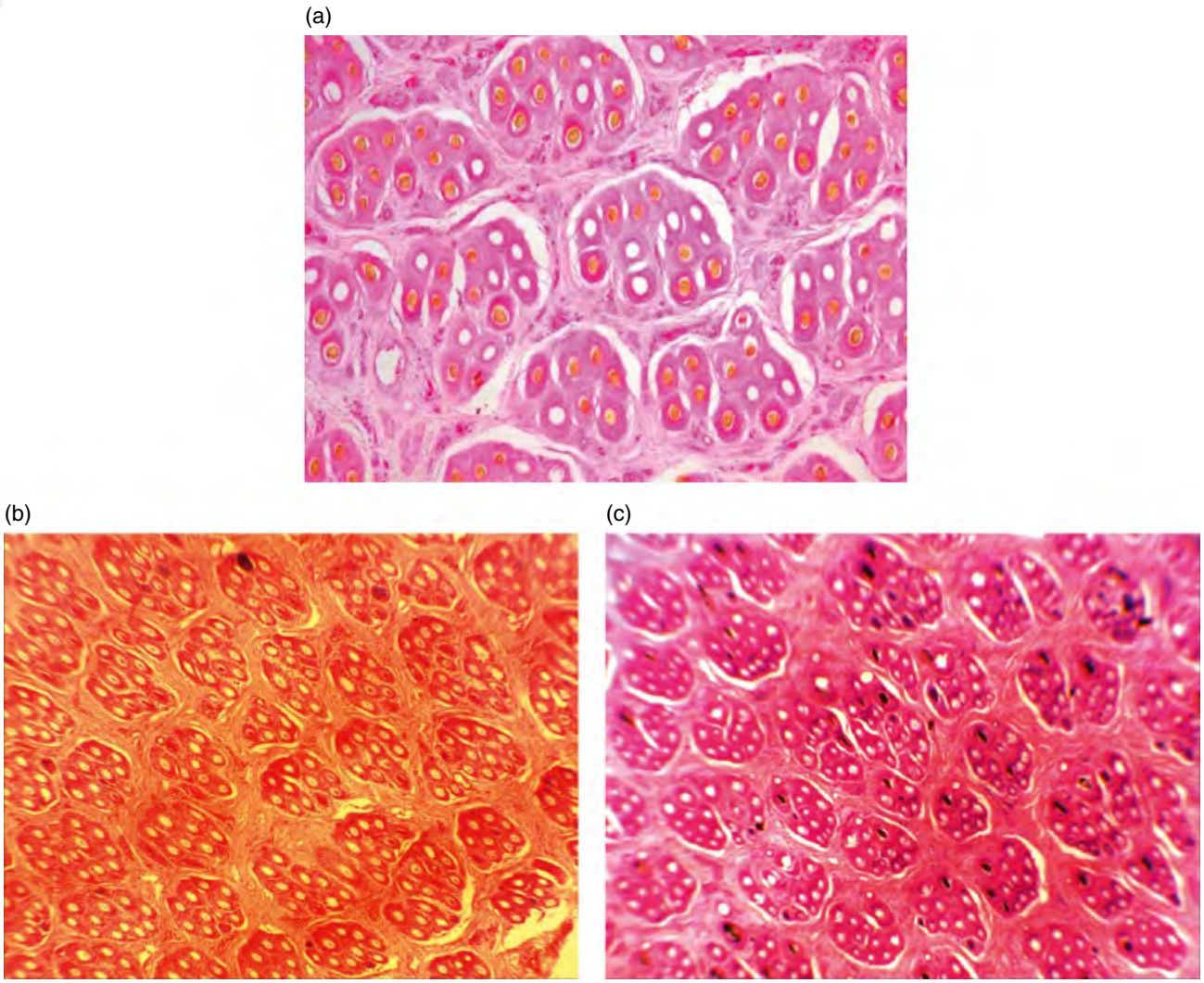


Figure 9.9 (a) Biopsy of alpaca skin with low-density follicles present with primary and secondary fiber distribution. (b) Biopsy of alpaca skin with medium- density follicles present with primary and secondary fiber distribution. (c) Biopsy of alpaca skin with high-density follicles present with primary and secondary fiber distribution. *Source:* Photos courtesy of Ian Watt.

Large patches of hair may be lost with no apparent inciting cause. There may be no evidence of an inflammatory response of the skin, yet when the skin surface is examined carefully, it will be noted that the hairs have broken off. This is likely similar to a condition called "wool break" in sheep. Sheep that have been stressed by illness or fever develop a break line in the hair that is growing in the follicle during that time. The entire fleece may be lost in one or two weeks. Camelid producers will complain that an animal has "tender" fleece and upon further questioning, it will be determined that the animal was ill earlier, possibly several months. This fleece will tend to come out during handling of the animal. The term "break" may be used to describe the actual breaking of the hair as a result of excessive thinning or the visible line on a fleece caused by the thinning of the diameter of the hairs. Thinning of the hairs can be produced by the administration of cortisol parenterally, indicating that any stressor may cause thinning of the hair and a potential break. Alpacas show this break line in the fleece when it is evaluated for micron testing. However, it is rare for them to lose the fleece entirely. Though formal studies have not been conducted to establish breaking as a phenomenon occurring in llamas and alpacas, routine yearly testing of fiber of individuals is done in many alpaca herds, especially if in the show and breeding portions of the alpaca industry. Fiber testing for fiber diameter micron determination and evaluation for consistency is done routinely by alpaca owners in North America. For current information on the process contact the AOA Inc. Testing of the fiber for normal distribution of micron diameter down the length of fiber and standard deviation of the micron along the length of the fiber will show peaks and dips

during the "year" of the fiber. In animals that show a great variation, this can sometimes be correlated with an illness earlier in the year. More testing information and correlation of health status will have to be done for this to be a formal evaluation of health on an individual or herd level.

To explain this phenomenon, it is necessary to understand that such a change in the diameter of the hair can occur only in animals that have a long growth cycle (anagen) (sheep, llamas, alpacas). Wild sheep and most other domestic animals shed wool and hair annually, and the fleece grows back to a genetically predetermined length. In anagen, various nutritional, metabolic, and environmental factors may influence the development of the hair while it is still in the follicle.

Sheep wool grown during the winter is finer (hairs are thinner in diameter) than that grown in summer. Studies have shown that this is not a result of cold weather but rather a response to the shorter day length. Poor general nutrition may also cause thinning. If both occur at the same time, the hairs may become fragile and break easily.

Lack of any essential nutrient in the diet may contribute to the production of hairs that are uneven and fragile. A deficiency of dietary copper weakens wool hairs. Because molybdenum has an inhibitory effect on copper metabolism, animals fed forage grown in areas known to have high concentrations of molybdenum may develop a copper deficiency. Low-protein diets, especially if deficient in sulfur-containing amino acids such as cysteine, result in thinning of the hairs. Alpaca producers have used this technique (feeding low protein diets) to have lower micron count in their show stock, unfortunately, to the detriment of their animals.

Thyroid hormone stimulates growth (both length and diameter) of hair. Estrogens tend to promote finer hairs, and testosterone, coarser ones.

In sheep, specific bacterial and fungal diseases of wool cause the hairs to deteriorate. Such diseases have not been identified in camelids.

Skin Glands

Adnexal Glands

In camelids, sebaceous (holocrine) glands are associated with each hair follicle, but the production of sebum (wool wax, wool fat, wool grease, lanolin) is low in comparison to that of sheep. Sebum is a mixture of waxes, cholesterol, and cholesterol esters that, when mixed with sweat, coat the skin and wool hairs to enhance water repellency, inhibit microorganism penetration, and inhibit dehydration via the skin.

Clippers used to shear sheep are naturally lubricated with wool wax as shearing proceeds. The lack of wool wax in camelids causes rapid overheating of clipper heads and clipper burns can happen with inexperienced shearers. Experienced camelid shearers will have several clippers in use and routinely dip or spray with oil for lubrication. This also allows time for the clippers to cool down in between use. Experienced shearers will take an average of three minutes to shear an alpaca that is stretched out properly for shearing. The clippers used to shear both sheep and camelids are the same but use different combs and cutters.

Epitrichial (formerly apocrine) sweat glands are generally found widely distributed over the surface of the skin but are more developed and denser on the ventrum, which is sparsely covered with hair (thermal window). The sweat glands consist of simple, tubular, or unilobular glands and are usually coiled. They are continuous with hair follicles and are also found on the muzzle, teat and with ceruminous glands associated with the fine hairs in the ear canal. Each eyelash follicle has several large glands of Moll present [1, 19]. In the most densely haired areas (cervical, dorsal thoracic, lumbar), pinna and chin only small, straight glands are found [1]. The slipper of the foot has eccrine (merocrine) glands with keratinized ducts emptying on to the surface of the foot [1].

Metatarsal Glands

Camelids have unique, oval-shaped, hairless patches on both the medial and lateral surfaces of the metatarsal regions of the rear limbs (Figure 9.10a and b) [1, 19]. Associated with the patches are multilobulated sebaceous (holocrine) glands, with ducts emptying on the surface. The dermis is markedly papillated, corresponding to invagination into the epidermis. The function of these glands is probably the excretion of alarm pheromones, perceived as "burned popcorn" odor to humans. Glandular secretions solidify upon excretion into leathery sheets that can be peeled off the surface of the skin (Figure 9.11). Some references describe these structures as chestnuts [1, 22] but histologically, they are not comparable to the chestnuts of horses [23].

Interdigital Glands

A line of hyperkeratotic hairless region extends down the length of the dorsal interdigital space on all four feet of SACs. These are termed the interdigital gland [1, 22]. One report claim them not to be glandular with no histologic evidence of glandular material present [22]. A second, more recent report, describes few, large circumscribed tubular glands present in the deep dermis of this area with

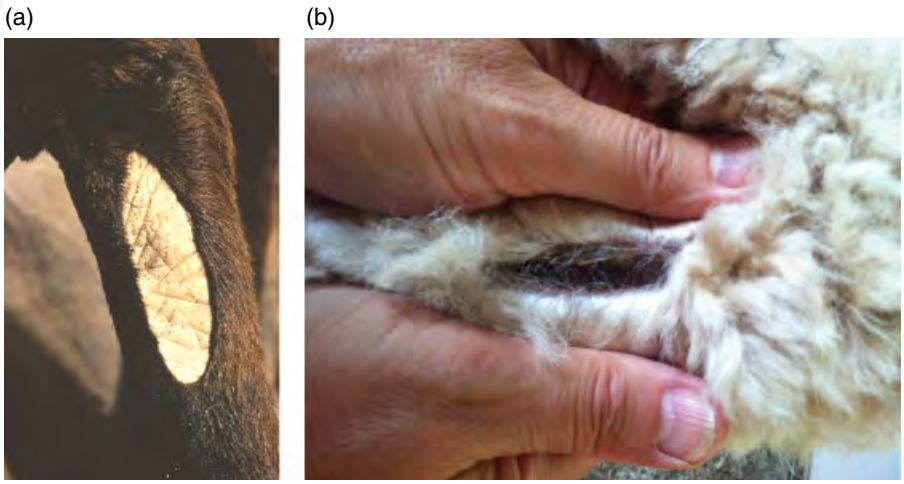


Figure 9.10 (a) Metatarsal gland in a llama. (b) Metatarsal gland in an alpaca. *Source:* Photo courtesy of Edy Dickson, Rocky Acres Alpacas, OH.

Figure 9.11 Metatarsal gland demonstrating excretions flaking off.

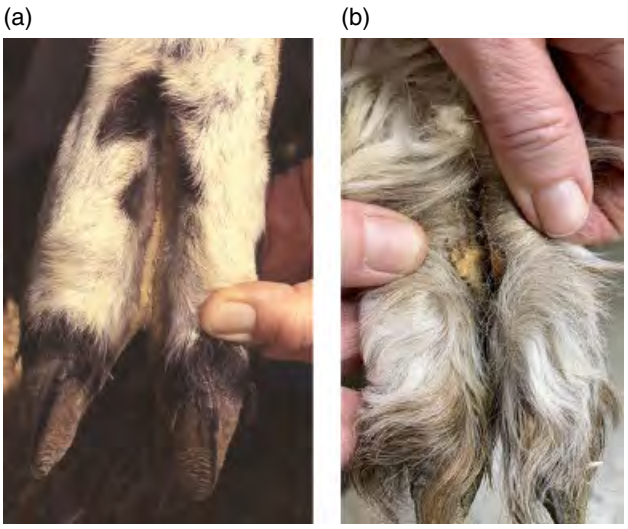


Figure 9.12 (a) Interdigital gland in a llama foot. (b) Interdigital gland in an alpaca foot.

tortuous ducts filled with keratin extending from the gland to the surface [1]. Regardless, the function of this area is unknown, and it has been suggested to be associated with individual and group identification (Figure 9.12a and b).

Poll Glands of Camels

Camels have a subcutaneous exocrine scent gland on the poll, between and just caudal to the ears. The gland is quiescent except when the male is in rut, at which time, the gland secretes a brownish-black fluid with an offensive odor (to humans). When in rut, the male camel may roll and rub the back of the head on the ground to scent mark.

Diagnosis of Dermatologic Conditions

A general protocol for differential diagnosis of dermatologic conditions should include the following in the approximate order of listing:

a) Conduct a complete physical exam to exclude systemic diseases that may produce a secondary dermatosis.
b) Obtain a complete history. In addition to information collected in the general exam, determine how long the dermatosis has existed. Is one animal or are multiple animals affected? Are the lesions enlarging or spreading to other areas of the body, face, or limbs? Is the

animal pruritic? Dermatoses that can cause pruritus to varying degrees include sarcoptic and chorioptic mange, pediculosis, food allergies, inhalant allergies, contact dermatitis, and insect bite allergy.

c) Establish and record the distribution and size of the lesions.
d) Pluck hair samples from the margin of the lesion and prepare a hot potassium hydroxide (KOH) preparation.
e) Perform a deep skin scraping from several sites to check for presence of mange mites.
f) If pyoderma is suspected, collect skin cultures as per same from dogs or horses.
g) Culture skin scraping material on Sabouraud dextrose agar (DTM culture)
h) Collect multiple skin biopsies from active and less active lesions.
i) Treat and observe the response to treatment.

Many camelid dermatoses are undiagnosed as to etiology (Figures 9.13a, b and 9.14a–d). Wounds similar, but not as severe as those depicted in Figure 9.13a were seen in other males being fed the same hay, so the oral lesions were thought to be due to irritation caused by feed. With chronic dermatitis of unknown origin, the condition and treatment can be frustrating and unrewarding for both owner and veterinarian. Once the condition has been worked up as described above, the author has used Amplimune® (a mycobacterium cell wall fraction immunostimulant) 1 mL, IV, every 30 days as an immunostimulant with moderate success. It has not completely cleared up unknown dermatologic conditions, but it has greatly improved the overall condition and comfort of the camelid.

Alopecia and chronic dermatitis over the bridge of the nose and ears (most commonly of dark-skinned llamas and alpacas) frequently occur (Figure 9.15a–c). The etiology may be multifactorial, including a vice of rubbing the nose, insect bite irritation, dermatophytosis, solar dermatitis, or an extension of some of the more generalized dermatoses. Frequently, these animals have no other fiber loss elsewhere on the head or body. The author had a yearling alpaca with both white fiber and black fiber in equal portions down the middle and sides of her nose. Only the black fiber was lost with a clear demarcation of where the white fiber started. Eventually, without any treatment, the black fiber grew back. Producers are often preoccupied with these cases, especially in show animals, and want to "do something." Rubbing in Tea Tree oil, Vitamin E oil or bag balm will moisturize the skin and fiber will grow back. This will be an ongoing, cyclical process in some of these animals.

Diseases of the Integument

Definition of Terms

The dermatopathologist uses special terminology to describe lesions of the integument. Following are definitions of a few terms, from Muller et al. [24], that have relevance to the discussion of integumental diseases in camelids.

Acanthosis is diffuse hypertrophy of the prickle cell layer of the epidermis. Acantholysis is loss of cohesion between epidermal cells. Hypergranulosis is increased thickness of

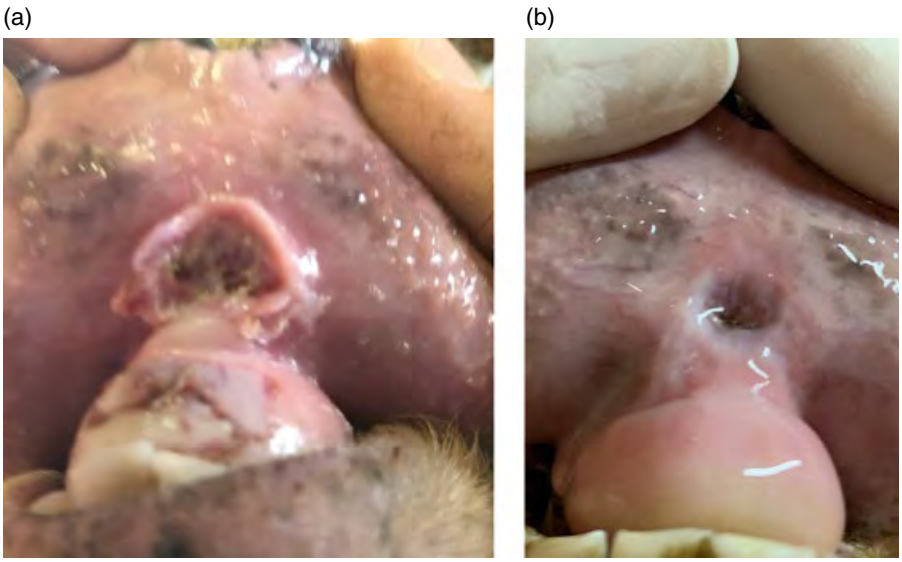


Figure 9.13 (a). Unknown lesion in mouth, probably due to irritation from feed. (b) Same mouth lesion four months later after repeated removal of hay particles and rinsing with saline.

Figure 9.14 Chronic nonspecific dermatitis. The animal pictured in (b), (c), and (d) are the same, (a) is different.

Figure 9.15 (a) Dark nose and ear "dermatitis" in a black alpaca. (b) Dark nose and ear "dermatitis" in a black alpaca. (c) Typical fiber loss on dorsum of nose in an alpaca.

the *stratum granulosum*. Hyperplasia is increased thickness of the noncornified epidermis. Hyperkeratosis is increased thickness of the *stratum corneum*. In normal skin of most species, the epidermal renewal time is 13–22 days. With inflammation, the renewal time may be reduced to three to five days, resulting in thickening (stacking) or excessive scaling. Parakeratosis is abnormal retention of keratinocytic nuclei in the *stratum corneum*.

The uniqueness of camelids must be kept in mind when interpreting skin biopsies [25]. There are some characteristics that may be normal but read out as abnormal by a pathologist not experienced with camelid skin. Mild orthokeratotic hyperkeratosis, mild perivascular inflammatory cells, small numbers of eosinophils and a prominent superficial cutaneous vascular plexus are likely normal in most cases [1, 22].

Parasitic Diseases of the Skin

Parasitic diseases of the skin include mange (sarcoptic, chorioptic, psoroptic, demodectic), pediculosis (*Damalinia* spp., *Microthoracius* spp.), and myiasis. In Peru, certain areas have problems with Trombiculosis (*Trombicula* spp.) and term it the "Red mange.[1]" These parasitic diseases are discussed in the Parasitology chapter (Chapter 7).

Viral diseases of the skin

Camelpox

Camelpox (abdarra, chechak, afturro, furuk) [26, 27] is the most frequently seen infectious disease of both dromedary and Bactrian camels [27], and has been produced experimentally in SACs. The disease primarily affects younger animals [30].

Etiology

The infective agent is *Orthopoxvirus cameli* (Family Poxviridae, Subfamily Chordoxvirinae). Camelpox virus is a large, complex DNA virus with a brick-shape appearance. There are numerous strains of the virus with differing degrees of virulence [27].

Epidemiology

Transmission is via viral exposure to abrasions or breaks in the skin, aerosols, or mechanical transmission by biting insects or other arthropods (mosquitoes, ticks). Recovery from the disease confers life-long immunity.

Camelpox is found throughout the Middle East, Asia, North Africa, Pakistan, Russia, and India, but not in the United States nor in the feral camel population in Australia.

Clinical Signs

The incubation period is 9–13 days. In mild cases, pustules develop on the muzzle, nostrils, eyelids, oral mucosa, and nasal mucosa. The pathogenesis of the pox lesion starts as an erythematous macule, progressing into a papule and then vesicles (Figure 9.16). The vesicle changes into a pustule with a depressed center and a raised erythematous border. When the pustules rupture, they become encrusted [27].

More severe cases are characterized by pox lesions over the entire body and limbs, accompanied by fever, anorexia, depression, and diarrhea (Figures 9.16–9.19). Lesions may occur in the trachea and on the surface of the lung. In severe cases, there may be up to 30% mortality, especially in the young and debilitated [28]. Abortion has also been reported. Secondary infection may complicate the clinical picture. Healed lesions leave patches of alopecia or a scar

Figure 9.16 Camelpox, acute, disseminated. *Source:* Photo courtesy of Dr. U. Wernery, U.A.E.

Figure 9.17 Camelpox, acute, scrotum. *Source:* Photo courtesy of Dr. U. Wernery, U.A.E.

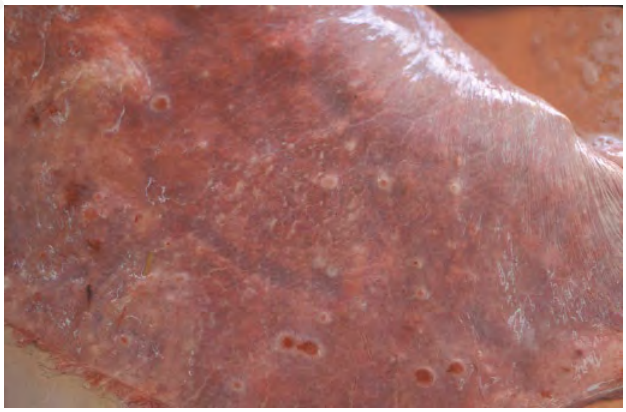


Figure 9.18 Camelpox, acute, lung lesions. *Source:* Photo courtesy of Dr. U. Wernery, U.A.E.

Figure 9.19 Camelpox, subacute.

Figure 9.20 Camelpox, healing lesions.

(Figure 9.20) [29]. Acute camelpox has been produced experimentally in a guanaco (Figure 9.21) [29].

Chronic camelpox may occur when a mild case develops in an immunodeficient individual. The lesions may take four to six weeks to heal with or without scars.

Figure 9.21 Camelpox experimentally produced in a guanaco.

Diagnosis

Clinical signs are characteristic, but a differential diagnosis must include lesions caused by a species-specific papilloma virus (warts) and parapoxvirus (contagious ecthyma [CE]). Laboratory diagnosis may include inoculation of embryonated chicken eggs, cell cultures, and inoculation into rabbits. Newer methods include enzyme-linked immunosorbent assay (ELISA), monoclonal antibody panels, and DNA restriction enzyme analysis and DNA probes [29]. A definitive diagnosis may be accomplished by electron microscopy, immunohistochemistry, and polymerase chain reaction (PCR) technology.

Management

There is no specific treatment for camelpox. In the generalized form, it is recommended to administer broad-spectrum antibiotics to minimize secondary infections.

A vaccine, Ducapox®, has been developed in South Africa and used successfully since 1994 in the United Arab Emirates to prevent camelpox. Wernery showed that a single dose given at one year of age would protect dromedaries for six years, possibly longer. He did stress that only a small number of camels were used in this trial [26]. When used in young camels, the manufacturer recommends a booster to be given in six to nine- month-old camel calves [27]. This vaccine was also shown to protect guanacos against camelpox [29]. In areas of the world where camelpox is prevalent some owners will take scabs from affected camels, dissolve them in milk and rub the mixture on scarified lips of camel calves [27]. Recombinant DNA technology may be used in the future to produce vaccines.

It has been thought that it was not zoonotic, but current evidence indicates that, under certain circumstances (non-smallpox vaccinated humans) Camelpox virus can be mildly pathogenic in humans [31] (Figure 9.22).

Figure 9.22 Individual pox lesion.

Figure 9.23 Contagious ecthyma (Orf) is a zoonotic disease.

Contagious Ecthyma

CE (contagious pustular dermatitis, sore mouth, Orf, auzdyk, ambarrur) is a parapox viral disease (*Parapoxvirus ovis*, Family Parapoxviridae). CE primarily affects the epidermal structures of the nose and lips of sheep and goats. Other susceptible species include musk-oxen (*Ovibos moschatus*), wild sheep (*Ovis canadensis*), Rocky Mountain goats (*Oreamnos americanus*), camelids, and humans [32].

Epidemiology

The natural reservoir host is probably the sheep. The virus may remain viable in crusts from a lesion for longer than a year. Transmission may be either by direct contact or insect vectors. In Africa and the Middle East, CE re-appears yearly in the early rainy season affecting camel calves in their first season of grazing. Eating of prickly plants will damage the lips allowing entry from the reservoir existing in the environment [31].

CE is a zoonosis that produces severe ulcerating lesions on the fingers, limbs, or face of affected people (Figure 9.23). A clinician should always wear gloves while examining a suspected case. One case involved a llama farm employee who had recently returned from working on a sheep ranch in New Zealand. Though she had a finger lesion at the time, it was not recognized as CE. One month later, a number of llamas at the farm developed lesions.

Clinical Signs

Clinical CE occurs in both OWCs and SACs. In Peru, classic lesions have been seen in alpacas at two to four months of age. Young camels are also susceptible or adults from naïve herds in endemic areas of the world. Initial signs include swelling of the lips, cheeks and possibly eyelids with a slight fever. These will progress in a few days to small nodules, then vesicles and papules. There may also be mild lymphadenopathy. Pustules may form from secondary infections as self-trauma from intense pruritus occurs. At this point, nursing and grazing may be affected [31]. Typical proliferative epidermal lesions at the commissures of the mouth have been seen in camelids in North America and New Zealand (Figure 9.24a and b) and in Africa and the Middle East. The lesion may be chronic, characterized by thickening of an area of the skin of the face or the perineum. Such lesions must be differentiated from those of idiopathic nasal/perioral hyperkeratotic dermatosis (Munge), mange, ringworm, or camelpox. History of exposure to sheep and goats will be an important determinant.

A nursing cria or camel calf may develop lesions on the lips and transmit the virus to the teats. Because of the pain associated with a lesion on the teats, the dam may refuse to allow nursing.

In sheep and goats, CE is a self-limiting disease. In camelids, the lesions may persist for months. In camel calves, the morbidity can reach up to 100% with severe lesions lasting for months [27]. In areas of the world with a high camel population, there have been reports of 60–100% morbidity with 9–38% mortality rates [31].

Diagnosis

The CE virus grows in tissue cultures but grows poorly on the chorioallantoic membrane of chick embryos. Microscopic lesions are diagnostic in early and acute cases [32]. Cytoplasmic inclusion bodies (4–8 μ) are found in swollen epidermal cells but disappear in older (six days or more) lesions [32]. Chronic lesions seen in camelids may be difficult to diagnose. Acanthosis, ballooning degeneration of spinose cells, hyperplasia of basal cells and edematous and granulomatous inflammation of dermal cells are seen histologically [31]. Differential diagnosis should include idiopathic nasal/perioral hyperkeratotic dermatitis (Munge), zinc-responsive dermatosis (hyperkeratotic lips), folliculitis, and Dermatophilosis.

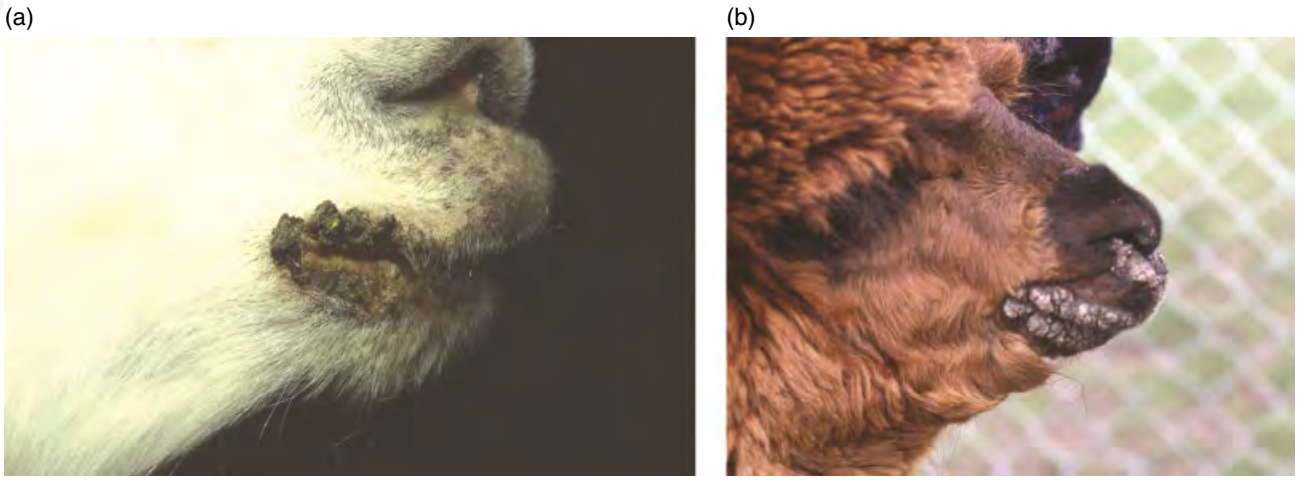


Figure 9.24 (a and b) Contagious ecthyma (orf) lesion in an alpaca.

Treatment

As CE is caused by a virus, there is no specific treatment. The animal(s) should be kept in isolation and the secondary infections controlled. It is usually self-limiting with scabs falling off in two to three weeks. To stimulate the immune system to shorten the infection, Amplimune (1 mL, IV, once per 30 days) can be attempted.

Prevention

Modified live virus (MLV) vaccines are routinely used in sheep and goats, but these vaccines are risky and may be unwise to use on SACs. Faced with an outbreak of CE in a herd, a clinician would have to weigh up risks against possible benefits. These vaccines are not effective on camels, however, an autogenous camel *parapoxvirus* vaccine has been used and was protective for at least six months [27]. There is not a long-lasting immunity to CE from either vaccination or natural infection. Any sheep or goats kept with camelids should be vaccinated and all traces of vaccinal lesions gone before such animals are allowed to mix with camelids. Vaccination using traditional Pastoral methods has been used in camel calves by taking scabs from infected camels, dissolving them in milk and rubbing the mixture on scarified lips of camel calves. It is recommended to do this during the dry season for a milder reaction [33].

Foot-and-Mouth Disease

Foot-and-mouth disease (FMD) (aftosa, aphthous fever, hoof-and-mouth disease) is a highly contagious viral disease, primarily of cattle, sheep, swine, and goats, but also affecting other artiodactyl domestic and wild animals. It is characterized by vesicular lesions and, subsequently, erosions of the epithelium of the lips, gums, soft palate, nares, muzzle, coronary bands, interdigital spaces, teats, and rumen pillar. Degenerative necrotizing lesions may have been observed in the myocardium of calves.

Etiology

FMD is caused by *Aphthovirus* sp. (Family *Picornaviridae*). Seven immunologically distinct types of FMD virus (FMDV) are known. Within the seven types, more than 60 subtypes have been identified over the years by complement fixation tests.

Epidemiology

FMD occurs in many countries but not in the United States and Canada. Mexico and other Central American countries have stringent regulations to prevent introduction of FMDV. FMD is of interest to camelid owners because the virus is present in many of the countries in which camelids are indigenous. Many species of artiodactyl are susceptible to FMDV, but they vary markedly as to whether or not clinical manifestations develop. Some species (e.g. the African buffalo [*Syncerus caffer*]) may harbor the virus in the pharynx for as long as 28 months, but vesicular lesions do not develop [34].

Many species of wild animals seem to be affected when an epizootic of FMD occurs in areas of Africa, but no susceptibility studies have been conducted. There are anecdotal reports, but no confirmed records, of natural infection in camels [35]. Experimental infections of FMD in camels have been reported [36]. However, in an epizootic of FMD in Ethiopia, where camels were in intimate contact with cattle, they neither developed lesions of FMDV nor produced antibodies to the strains involved [37].

During a severe epizootic of FMD in Peru, Peruvian regulatory veterinarians dealing with cattle reported that similar lesions had been seen in alpacas [38]. Also in Peru, a case of FMD in alpacas was confirmed by laboratory test (type A24) in 1971 [39]. A similar type of isolation made in

llamas in Peru that same year was not reported in the literature.

One study of camelid susceptibility to FMDV was conducted in 1952 in Peru [38]. One vicuña, 19 alpacas, and 16 llamas were exposed to FMDV by various methods, including scarification of the tongue, intradermal inoculation of the tongue, intramuscular (IM) and intravenous (IV) injection, and cohabitation with animals infected by other routes.

Animals became infected from all routes of exposure. Four animals died in the groups given IM or IV injections. The incubation period from exposure to the development of lesions was 48–72 hours. The first manifestations were vesicles on the tongue, with generalized vesicular formation occurring elsewhere within three to four days.

The four llamas exposed by cohabitation developed no lesions, but these same animals became infected when inoculated intramuscularly. Cattle exposed by cohabitation suffered 100% morbidity. One of the alpacas in the cohabitation experiment developed lesions in 3 days, another in 5 days, and a third in 10 days. Two other alpacas failed to develop lesions throughout cohabitation, but did so 20 days later when injected intramuscularly.

It was concluded from this Peruvian study that camelids are susceptible to the virus, but less susceptible than cattle and sheep. In a study conducted at the USDA National Foreign Animal Disease Laboratory at Plum Island, New York,[2] transmission was shown to be possible from the bovine to llamas and vice versa [40, 41]. The virus could not be isolated from llamas after 14 days following infection.

A more definitive study involving dozens of llamas was conducted in Argentina as a cooperative project with the Argentine government and the US Department of Agriculture. Previous findings were confirmed, demonstrating that llamas are quite resistant to infection with the FMDV and do not remain carriers.

The route of field transmission may be by respiratory aerosol or direct contact with infected animals, or exposure to contaminated feed and water. FMDV may be inhaled in aerosol droplets by people working around infected animals. The virus lodges in the pharyngeal mucosa and may be exhaled for as long as 24 hours post exposure. Thus, humans may contribute to the spread of FMD. Actual FMDV infection in humans is rare, and the disease is not considered to be a public health risk.

Clinical Signs

The incubation period varies from three to five days. In cattle, early signs include fever, depression, and anorexia. Stomatitis causes salivation, and lameness is caused by lesions on the coronary bands and interdigital spaces. Pain may cause the animal to tread, shake or kick out the feet, or lie down. Ultimately, the vesicles rupture, forming erosions, and a mucopurulent nasal discharge may develop. Pregnant cows may abort, and calves may die acutely. The overall mortality rate in cattle is usually less than 5%, but 50% of affected calves may die from myocardial degeneration.

Experimentally infected camelids in Peru developed fever up to 40 °C (104 °F). This was the first sign noted in all affected animals. They became totally anorectic approximately 24 hours following inoculation and chose to lie down most of the time. Vesicle locations and progress of the lesions followed patterns seen in cattle and sheep.

In the North American study, 30 llamas were exposed to FMD-infected pigs. The affected llamas were depressed but had no fever. Vesicles formed on the tongue, lips, dental pad, coronets, and interdigital glands. When the vesicles ruptured, the erosion healed rapidly. Footpads became undermined. These llamas were then placed with 30 control llamas that did not show any clinical signs of FMD, did not isolate any FMDV nor develop antibodies to FMD. The conclusion was that SACs can contract the disease after experimental infection or in close contact with FMD diseased livestock but are not very susceptible to FMD overall [34]. The consensus is that SACs and Dromedaries are generally resistant to FMD unlike Bactrian camels which have contracted naturally occurring FMD infections and may transmit infection to other artiodactylids [27].

Diagnosis

The diagnosis of any suspected vesicular disease is under the control of state and federal regulatory veterinarians in the United States and comparable authorities in Canada, Mexico, and other countries. Suspected cases must be reported promptly. Suspect facilities must be quarantined until a diagnosis is confirmed or disproved by a variety of sophisticated laboratory tests. A provisional diagnosis will be made within 24 hours of receipt of lesion material at the diagnostic laboratory. Specific vesicular diseases (i.e. vesicular stomatitis [VS]) cannot be differentiated by clinical signs nor at necropsy.

Pathology

Vesicles, followed by erosions, may occur on the tongue, dental pads, gums, cheeks, hard and soft palates, lips, nostrils, muzzle, coronary band, interdigital space, teats, and stomach pillars. Degenerative, necrotizing myocarditis may cause death, especially in young animals. Because camelids are also susceptible to experimental inoculation with VS virus, this disease must be considered in a differential diagnosis.

Treatment

There is no treatment for FMD. By law in the United States, both infected and exposed animals must be euthanized. This practice is also followed in Canada, Mexico, and Central America.

Prevention

Vaccines are used to control FMDV in livestock in countries in which FMD is enzootic. Vaccines are strain-specific and must be prepared for a given geographic area. Animals immune to FMD, after vaccination, can still be carriers as they can carry the virus in their pharynx. Cattle can carry FMDV for up to three years [27]. Vaccination is illegal in North America and many European countries.

Papillomatosis

Papillomas (warts) occur on a variety of animals worldwide. In most animals, the lesions are a benign neoplastic proliferation of the skin epithelium and mucous membranes. Cattle are the most common of the domestic livestock that develop warts with six types of *bovine papillomaviruses* identified. Warts are very rare in SACs.

Etiology

Papillomas are caused by a species-specific *Papillomavirus* (Family *Papovaviridae*). The particles are spherical with icosahedral symmetry and about 50 nm in diameter [27].

Epidemiology

Papillomas have been reported in OWCs and rarely in SACs. The disease is rare and is of little economic significance in camels. The lesions may be difficult to differentiate from camelpox and CE in the early stages of the disease. Warts usually occur in camels less than two years of age.

Papillomatosis is found in the Middle East [42], North Africa, and India. Transmission is by direct contact of abrasions of the skin or mucous membranes with active lesions on other animals. Handling equipment may become contaminated if used on infected animals. Insects may be responsible for mechanical transmission.

Clinical Signs

The lesion is characterized as a round cauliflower pedunculated mass varying in size from 0.3 to 4 cm in diameter. White, gray or light tan in color located on the nose, chin, lips, neck or brisket. The papilloma does not affect the general health of the camel. The mature wart is not likely to be confused with camelpox or CE, but the early lesion is a hyperemic elevation of the skin similar to the macula stage of camelpox.

Diagnosis

Microscopically, the lesion is a hyperplastic epithelium with excessive folding. Electron microscopy may identify viral particles. An immunohistochemistry method has become the definitive diagnostic technique [42].

Management

The disease is generally mild and self-limiting with the warts falling off on their own in three to six months. No preventative or treatment is considered necessary. However, in a herd outbreak, an autogenous vaccine was prepared and administered. Healing time was markedly diminished with the warts receding in 8–10 days [27].

Vesicular Stomatitis

Vesicular stomatitis (VS) (erosive stomatitis, pseudo aftosa) is another vesicular disease; it has a broader host range than FMD, including horses and omnivores. VS is enzootic in a number of locations in the western hemisphere.

VS virus is classified as *Rhabdovirus* spp.(Family Rhabdoviridae rhabdovirus). There are two major types, New Jersey and Indiana. There is no known reservoir host. It is possible that this virus primarily attacks a plant or an insect and only infects mammals secondarily.

Epidemiology

Definitive information on the epidemiology of VS is not complete. Epizootics occur seasonally in various geographic locations, affecting horses, cattle, swine, and deer in the United States [43]. One natural case of VS in camelids has been reported [44]. Experimentally, alpacas and llamas are susceptible to intradermal inoculation into the dorsum of the tongue, which produces localized vesicles [23]. Other methods of inducing infection were unsuccessful. Attempted exposure routes included IM injection, rubbing the virus on the surface of the tongue, and cohabitation with infected animals. Only 50% of those given intradermal inoculations developed vesicles (in 48–96 hours).

Fluid taken from vesicles of camelids was infective to susceptible cattle. Camelids that were unaffected in the cohabitation trials were subsequently susceptible to intradermal tongue inoculation one month later.

No reports exist on VS in OWCs [27].

Clinical Signs

Clinical signs of VS are indistinguishable from those of FMD [45]. In experimental llama cases, a transient, slight elevation in temperature (0.5 °C [0.9 °F]) occurred 24–48 hours after inoculation, followed by anorexia. The animals became recumbent and developed vesicles. No foot

lesions were seen. In the naturally infected llama, signs were similar to those described for experimental cases.

Pathology, diagnosis, and treatment are the same as for FMD. Animals should be treated supportively.

Bluetongue

Bluetongue was first described in domestic sheep but is now known to affect many species of ruminants [46]. There is serologic evidence that camelids respond to bluetongue virus (BTV) with the formation of antibodies, but clinical disease is questioned.

Etiology

Bluetongue is an insect-transmitted viral disease caused by bluetongue virus (BTV) (*Orbivirus* sp. Family *Reoviridae*). BTV is antigenically related to the epizootic hemorrhagic disease virus of deer. BTV exists in a multiplicity of serotypes. The degree of cross-protection against heterologous serotypes has been shown to be limited and variable.

Epidemiology

Small, blood-feeding gnats of the genus *Culicoides* biologically transmit BTV. After a female gnat has taken an infective blood meal, the virus replicates in the salivary glands. Ten days later, BTV may be transmitted by the gnat while taking another blood meal. The gnat will remain infective for life and live for about 28 days or more, feeding every 3–5 days.

BTV antibodies have been found in numerous species of wild animals. One alpaca in Germany had a fatal infection [47], but there is no evidence that alpacas are a reservoir for the disease in sheep. Twenty percent of the alpacas on one ranch in Peru had a positive antibody response to BTV [48]. In Africa and the Middle East, BTV can be considered endemic in the cattle, sheep and goat population. In those areas, seropositive dromedaries can range from 1 to 81% with no clinical signs of BTV demonstrated in any of the camels. There is a possibility however that the dromedary might aid in spread of BTV [27].

BTV is probably present in most regions where sheep, cattle, and camelids coexist. SACs in North America can be seropositive in areas where BTV is known, however with one exception, do not develop clinical disease. This disease should be included in a differential diagnosis of any camelid showing lesions in the mouth, especially in an area known for BTV.

Clinical Signs

Clinical manifestations of bluetongue vary widely according to species. In sheep, it is characterized by fever, nasal discharge, pneumonia, congestion of the lips, oral and nasal mucosa and epithelium of the tongue, necrosis of the dental pad, edema of the ears, and necrosis of skeletal and cardiac muscle. Mouth lesions may progress to shallow ulcers on the lips and gums. In a few individuals, a similar inflammatory response will occur on the coronary bands of the hoofs. Lameness is an early clinical sign in flocks of infected sheep on pasture.

Cattle may show fever, a stiff gait, drooling from the mouth, small ulcers on the oral and nasal mucous membranes and the dental pad, and/or nasal discharge. The muzzle may be hyperemic. The coronary bands may become inflamed and swollen, resulting in lameness similar to that seen with sheep. Reproductive abnormalities such as abortion, deformed calves, or unthrifty newborn calves have been associated with BTV infections in cattle. Also, many cattle apparently acquire an infection that is undetectable or not observed. Cattle usually recover 7–10 days after clinical signs appear.

A case of suspected BTV has been reported in North America in a female llama. A pregnant female had an episode of respiratory distress followed by an abortion. Paired serum samples taken after the abortion demonstrated a fourfold increase in BTV antibody titer [49]. A case of mortality in an alpaca was attributed to bluetongue in the United Kingdom [47].

Diagnosis

Virus isolation and serologic tests are necessary for diagnosis. Gross and microscopic lesions are suggestive but must be differentiated from vesicular diseases. A competitive ELISA test was developed and tested in two llamas that had been experimentally infected with BTV. Both animals remained clinically normal but developed titers within 2 weeks post inoculation that remained high for the 10 weeks of the experiment. The authors suggested that a 50% inhibition would be indicative of exposure to the virus in llamas in New Zealand [50].

Treatment

Supportive therapy and administration of antibiotics to prevent secondary infections are recommended.

Prevention

Sheep are routinely vaccinated. Currently available MLV vaccines are strain-specific and have not been tested for camelids. The use of these vaccines in camelids, with the present state of knowledge, is not recommended. It is difficult to completely prevent camelids from coming into contact with gnats, however, in problem areas, it may be beneficial to have the animals enclosed at dusk to minimize exposure.

Fungal Infections

Dermatophytosis (Ringworm)

Various dermatophytes produce superficial infection of the epidermis in many different species of animals [51, 52]. Fungal dermatitis is uncommon in camelids, but can occur in both SACs and OWCs.

Etiology

Ringworm (robbi, taat, robi) can be caused by *Trichophyton* spp. and by *Microsporum* spp. [52]. Only *Trichophyton verrucosum* and *Trichophyton mentagrophytes* has been documented in SACs [49]. *T. verrucosum* (Figure 9.25) is the common cause of ringworm in cattle and goats and more rarely in horses, donkeys, dogs, sheep, camels, and llamas. Arthroconidia are seen lining the surface of hairs in KOH preparations of material scraped from lesions. The arthroconidia are approximately 5 μm in diameter. Microscopic chains of chlamydospores are typical (Figure 9.25).

T. mentagrophytes var. *mentagrophytes* (Figure 9.26) has also been isolated from skin lesions in llamas as well as from cats, dogs, cattle, sheep, pigs, horses, rodents, and monkeys. The organism has a ubiquitous geographic distribution. Arthroconidia on the surface of hairs tend to cluster. Growth on artificial media is characterized by microconidia; macroconidia; coiled hyphae; and branched, septate hyphae. *Microsporum* spp. have also been isolated from llamas.

Species that have been isolated from OWCs include *Trichophyton verrucosum, T. mentagrophytes, Trichophyton schoenleinii, Trichophyton sarkisovii, Trichophyton dankaliense, Microsporum gypseum,* and *Microsporum canis*.

Clinical Signs

Clinical cases may simply be alopecic areas with limited distribution on the head and neck or more generalized over the body and limbs. In the clinical cases seen, the lesions in camelids caused by both organisms were similar to those observed in cattle, with raised, crusty, circular plaques around the poll and face (Figure 9.27a and b). The areas are not pruritic. The age of the lesions was unknown in the cases reported, but the plaques varied from 2 to 4 cm in

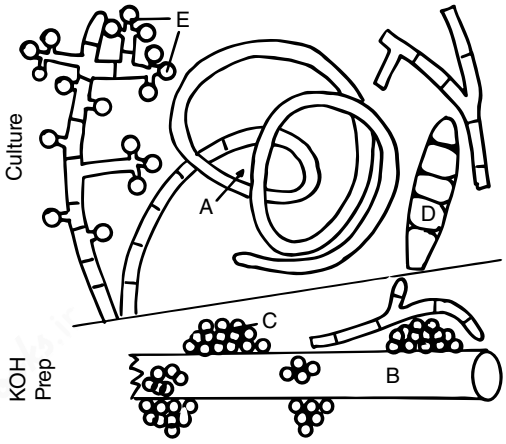


Figure 9.26 Diagrams for identification of *Trichophyton mentagrophytes*. (A) Coiled hypha, (B) fiber, (C) arthroconidia, (D) macroconidia, (E) microconidia.

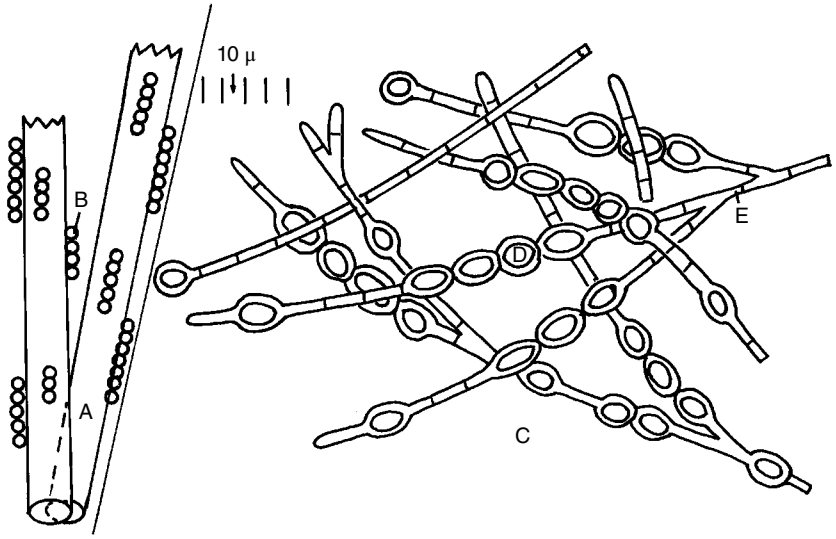


Figure 9.25 Diagrams for identification of *Trichophyton verrucosum*. (A) Fibers, (B) arthroconidia on the surface of the fibers, observed on KOH preparation of skin scrapings, (C) growth on artificial media, (D) chlamydospores, (E) septate hyphae.

Figure 9.27 (a) Ringworm in a llama. (b) Ringworm in an alpaca. *Source:* Photo courtesy of Carmon Koenigsknect.

diameter. In camels, ringworm is most common in animals under three years of age. The lesions are generally on the head and neck and will be small, round areas gray-white in color. These areas of hair loss may coalesce into larger areas resulting in a severe generalized infection involving the head, neck, chest and limbs with emaciation and debilitation occurring. This more severe involvement can be easily confused with mange [27].

Epidemiology

The epidemiology of ringworm in camelids is as yet unexplored, but direct and indirect contact are the usual modes of transmission of dermatophytes. The chlamydospores of both *T. verrucosum* and *T. mentagrophytes* may remain viable for up to 4.5 years in hair and cellular debris scraped off the animal and left attached to barn walls, fence posts, trees, feed bunks, halters, blankets, packs, brushes, and combs. Other camelids and humans may become infected from contact with these fomites. Both organisms may be zoonotic, causing an acute inflammatory response on the scalp and other areas of skin, with possible scarring.

Pathology

Early lesions have not been observed in camelids, but in other species, the dermatophyte produces toxins and allergens that evoke an inflammatory response in the susceptible host, with resulting erythema, exudation, and alopecia. The organism tends to move peripherally toward unaffected tissue, enlarging the lesion in a circular pattern. In cattle, the lesion becomes thicker as it enlarges peripherally.

Diagnosis

Direct microscopic examination of hair and crusts taken from the periphery of a lesion is the most rapid method of diagnosis. The material should be placed on a slide and a few drops of 10% KOH solution applied and allowed to react for 10–15 minutes, preferably accompanied with gentle heating to facilitate clearing. Fungal spores may be observed clinging to hairs when viewed microscopically at 100X, and irregular, septate hyphae may be identified.

Both *T. verrucosum* and *T. mentagrophytes* may be cultured on Sabouraud's dextrose agar. In contrast with most dermatophytes, *T. verrucosum* grows better at 37 °C (98.6 °F) than at room temperature. Growth is slow, requiring 10–14 days before colonies become evident. Chlamydospores and mycelia are produced on unenriched media, but numerous microconidia and more regular mycelia grow on thiamine enriched media.

A number of nonspecific and generally undiagnosed keratin-proliferative dermatoses have been seen in SACs. Any thickened lesion should be checked carefully, with multiple deep skin scrapings for dermatophytes and mange mites (Chapter 7).

Treatment

Lesions of ringworm are usually self-limiting in most species. Iodine (2% tincture) may be applied directly to a lesion daily for two weeks. A less caustic povidone-iodine preparation (diluted 1:4) may be equally effective. Iodine/betadine is used extensively to treat ringworm in livestock, horses, and dogs, and has been effective in treating camelids. Depending on the time of the year, consider a bath with an antifungal shampoo.

Captan® is a fungicide for ornamental plants. As a caution, captan has not been approved for use on animals intended for food, and gloves should be worn by people mixing and applying the solution. Captan may be purchased as a 50% wettable powder from nursery suppliers. The recommended dilution is two tablespoons of the powder to 4 L (1 gal) of water. The mixture is stable for one week after mixing. The solution should be applied liberally to the lesion(s) and the surrounding area twice weekly for two weeks. The animal should not be rinsed off after application of the mixture.

Topical antifungal preparations can be applied such as Miconazole containing products, Lyme sulfur sprays/ointments or Tresaderm® solution. Thiabendazole is no longer readily available as an anthelmintic, however, it has antifungal action and may be applied as a 2 to 4% ointment to lesions of ringworm at three-day intervals. The author has no experience with the use of oral griseofulvin in camelids but it has been reported to cause gastrointestinal (GI) upset and diarrhea in camels [27]. A vaccine (Camelvac Tricho®) has been used in OWCs with good results for both prevention and treatment [27].

Coccidioidomycosis

Coccidioidomycosis (San Joaquin Valley fever, valley fever) is a fungal infection primarily of the respiratory tract of humans and susceptible animals [53, 54]. Coccidioidomycosis is caused by *Coccidioides immitis* or *Coccidioides posadasii* [55]. It may appear in a disseminated form or as a dermatitis.

C. immitis was first described as a protozoa, similar to coccidia, but it is actually a dimorphic fungus. Its life cycle (Figure 9.28) is usually completed in the absence of animals. The literature on this organism is extensive because of human infection, but this pathogen is generally not spread from animals to humans except under unusual circumstances. There are no reports of coccidioidomycosis in OWCs.

Epidemiology

SACs appear to be highly susceptible to coccidioidomycosis, which is acquired by inhalation of arthrospores from a contaminated environment. Arthrospores are the infective stage; they convert to the tissue invasive spherules seen in animal tissues. The thick, refractile-walled spherule sporulates and becomes filled with endospores. When the spherules rupture, endospores are released, spreading the infection.

Coccidioidomycosis is an infectious disease, but it is not transmitted by direct contact from animal to animal. Infection is generally considered to be restricted to specific geographic areas, but in camelids, with extensive movement of animals from one location to another, the disease may be diagnosed in any area.

Llamas and alpacas maintained in endemic areas are at risk. The known areas in which *C. immitis* is endemic include the southern desert areas and the San Joaquin Valley of California; the southernmost parts of Utah and Nevada; and most of southern Arizona, New Mexico, and southwestern and west-central Texas. The climatic conditions of these hot, arid areas of the lower Sonoran life zone are conducive to maintenance of the fungus [56].

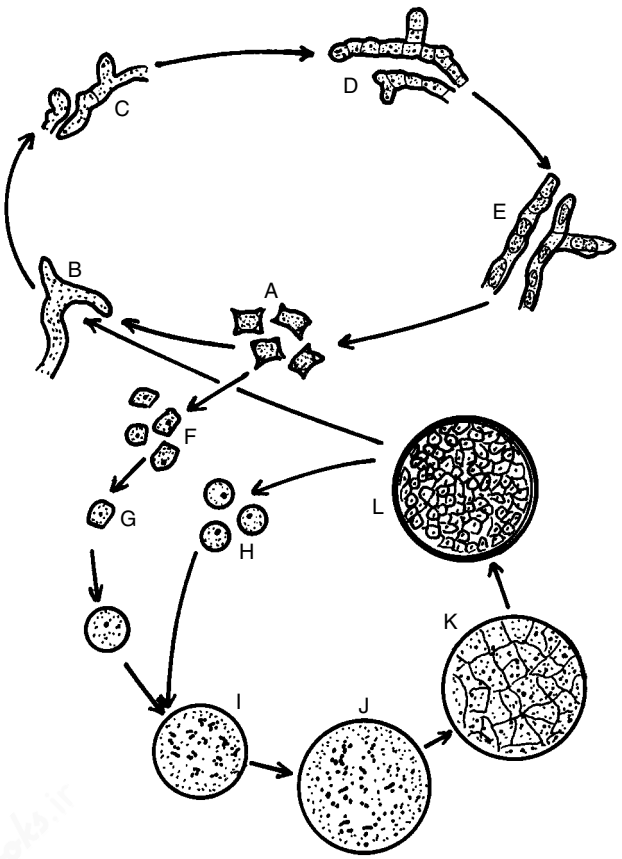


Figure 9.28 Life cycle of *Coccidioides immitis*. (A) Arthroconidia, (B) emerging fungal hypha, (C) nonseptate hypha, (D) septate hypha, (E) arthroconidia with hypha, (F and G) maturation of arthroconidia to spherule, (H) endospores, (I to K) development of spherule, and (K and L) endosporulation of spherule.

Disruption of the soil, such as for road building or excavation for construction, exposes the organism to winds, creating dusty aerosols suitable for inhalation of the arthrospores.

The arid regions of northern Mexico are known to harbor *C. immitis*. In South America, endemic areas are located in northwestern Venezuela and the Gran Chaco Pampa region of Argentina and Paraguay, with possible extension into southern Bolivia. Endemic areas have also been identified in Honduras, Guatemala, and Nicaragua in Central America.

Transplacental infection has occurred in a llama. Previously, this was only known to occur in humans [23].

Clinical Signs and Pathology

The dermal form is characterized by nodular lesions from 1 to 3 cm in diameter or extensive raised plaques (Figure 9.29a and b). They may be exudative and crusty or they may appear to be covered with intact skin and like a subdermal abscess (Figure 9.29c). Lesions have been observed over

Figure 9.29 (a) Dermal coccidioidomycosis. (b) Dermal coccidioidomycosis. (c) Subdermal coccidioidomycosis. The granulomatous lesion is completely covered with intact skin. The large lump under the skin (arrow) was initially mistaken for an abscess.

most of the body surface, e.g. on the perineum, face, and limbs. Pruritus is not evident.

Lesions on the skin may appear as discrete granulomas ranging from 1 to 5 cm in diameter or coalesce in large, irregularly shaped masses. The nodule is usually gray and firm. The microscopic lesion is characterized by granulomas or pyogranulomas, with numerous spherules present.

Diagnosis

Diagnosis is facilitated if a lesion is accessible by needle aspirates from abscesses, skin nodules, or biopsy samples. The organism can be readily identified by direct microscopic identification of the refractile, double-walled spherules that contain endospores (10–80 μm in diameter) [23].

The interpretation of serologic data from llamas is difficult because as yet, insufficient numbers of individuals and herds have been tested to establish sound normal data as a basis for comparison. Serologic and immunologic tests used in the study of coccidioidomycosis include precipitin, complement fixation, fluorescent antibody, latex particle agglutination, and agar gel diffusion precipitin tests. Some of these tests are presently being used to evaluate the status of infection in herds in endemic areas of Arizona.[3] Intradermal tests, at dilutions of 1:5, 1:10, and 1:100, have been negative in known infected animals.

The organism may be cultured on selective media such as cycloheximide-chloramphenicol agar incubated at room temperature. On artificial media, hyphae will be seen sprouting from the spherule. The hyphae are septate. Growth is rapid (three to five days) (Figure 9.30). Arthrospores develop within hyphal segments. An investigator should refer to standard texts for details of safety precautions before attempting to culture *C. immitis* because of the risk of human infection from the arthrospores produced in culture.

Treatment

Amphotericin B is the treatment of choice, however poor efficiency was reported when used with a pregnant llama. The llama was treated over a six-week period and appeared to deliver a healthy cria. The neonate was examined for evidence of coccidioidomycosis, including thoracic radiographs; all tests were normal. Approximately one month later, the cria became dyspneic and thoracic radiograph indicated a cystic lesion in the lung; serologic tests were positive for coccidioidomycosis. The female's complement fixation titer was positive at 1:256. Both the neonate and the female were euthanized. Extensive diffuse lesions were found throughout the thorax and abdomen of the female. Lesions were also found in the lungs of the cria. It was

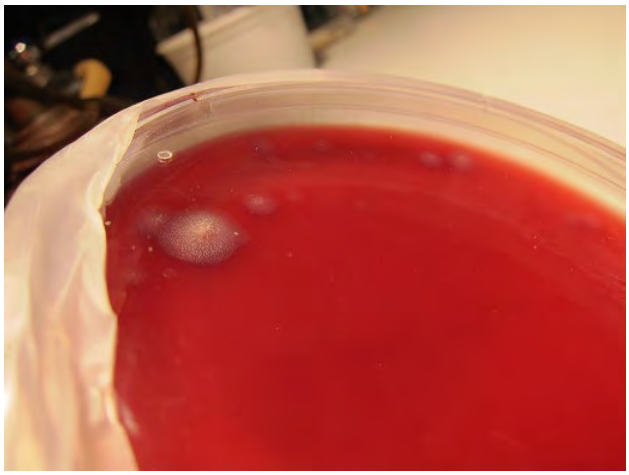


Figure 9.30 Culture of *Coccidioides immitis* grown from swab of submandibular granulomatous mass removed from a llama. *Source:* Photo courtesy of Dr. Josh Daniels.

concluded that therapy was not successful in eliminating the infection from the female nor in preventing transplacental passage of the organism to the fetus [23].

Several other antifungal imidazole drugs or combinations of drugs are used to treat coccidioidomycosis in humans and other animals. These include fluconazole, ketoconazole, miconazole, and itraconazole. There are no reports of use in camelids, nor dosages for large animals.

Prevention

Vaccines are being developed to protect humans who must work in high-risk areas. Similar vaccines have also been used in nonhuman primates in highly endemic areas with apparent success. However, vaccines for camelids have not been evaluated but may become important if camelids are to be kept in endemic areas. Since a primary mode of transmission is via dust, avoiding dust is a recommendation for prevention of coccidioidomycosis. Since camelids routinely dust themselves, this is hard to accomplish. In warm, arid areas of North America, camelid owners struggle with this disease.

Candidiasis

Candidiasis (moniliasis, thrush) caused by *Candida albicans* can be found normally in the respiratory, alimentary and gastrointestinal tracts [57]. In the camelid, it is rare to uncommon. Typically seen in the interdigital space, axilla, groin, perineum and with dermal plaques seen on the surface of the pinnae. The lesions are variably erythematous, thickened, and covered with waxy to greasy accumulations. Pruritus is not common and there may be marked to severe hair loss [58].

One report discusses the case of an eight-year-old male llama with history of an unknown skin disease of a "few years" duration. The owner was treating with zinc oxide ointment with no results. On the same farm, there was a female with the same lesions, though less severe [57].

The male llama had extensive areas of thick, coalescing crusts on the axillary, inguinal, and perineal areas and on the muzzle. The crusts could be removed, leaving the underlying skin moist and red and with a foul odor. Within the area of the removed crusts there were scattered small pustules. This llama was weak, had poor body condition, and was depressed.

Skin biopsy revealed marked parakeratotic hyperkeratosis with thick serocellular crusts of degenerated neutrophils and numerous yeast organisms. In the underlying dermis, there was multifocal perivascular infiltrate of neutrophils, lymphocytes, plasma cells and macrophages. A pure culture of *C. albicans* was cultured from the fresh skin biopsy sample [57].

Diagnosis

As part of a routine work-up for dermatologic diseases, Gram stain should be done. In the case of candidiasis, Gram stain may show characteristic rounded or oval budding cells (blastospores) 3–6 μm in diameter. The budding cells may be linked, forming a chain (pseudohyphae), with indentations between the buds. True mycelia may also be found. Culture on Sabouraud's agar can be attempted at either room temperature or 37 °C (98.6 °F) but may be unsuccessful.

Treatment

In the case reported, the male was treated with nystatin and chlorhexidine acetate ointments applied topically to the affected areas. Healing occurred in 60 days with fiber growing in affected areas. The female was treated similarly also with resolution of lesions. Because antemortem diagnosis is difficult and the disease rare, it seems unlikely that a treatment regimen can be properly developed. However, nystatin has been recommended for intestinal infection in pigs.

Prevention

Prevention of candidiasis can best be accomplished by minimizing predisposing factors such as stress and prolonged antibiotic therapy. Lack of response to antibiotic therapy after three to four days should prompt reevaluation of the treatment regimen.

Miscellaneous Fungal Infections

Infection caused by *Entomophthoramycosis conidiobolae* [59] and histoplasmosis [60] have been reported in

llamas. One case of Sporotrichosis was seen in a llama. Lesions were multiple firm papules, nodules and plaques on a distal limb. Diagnosis was by culture and histopathology [58].

Zygomycosis, caused by the normally saprophytic fungus *Conidiobolus coronatus,* has produced localized infection in humans, horses, and one llama. The lesion consisted of a nodular dermatosis involving the external nares, which had been present for three years. In addition to the nares, the face and muzzle were involved [54]. Encroachment of the nares had caused dyspnea to develop. The mass obstructing the nostril was excised, but total extirpation was not possible. The llama lived another three years following the surgery [54].

Bacterial Infections

Abscesses

Abscesses are local collections of purulent exudate buried in tissues, organs, or other confined spaces. They occur commonly in all animals including camelids. Abscesses may occur in any organ system but are most common in subcutaneous tissue, lymph nodes, and the parenchyma of the viscera. Infection may arise from breaks in the epidermis or from hematogenous sources.

Etiology

Many different bacterial species have been isolated from abscesses. *Trueperella pyogenes* and *Corynebacterium pseudotuberculosis* are the most common isolates. Other species that may be isolated include *Staphylococcus aureus, Streptococcus* spp., *Rhodococcus equi, Pseudomonas aeruginosa, Salmonella* spp., *Nocardia asteroides, Actinomyces* sp. *Actinobacillus* spp., *Corynebacterium ulcerans, Escherichia coli*, *Enterococcus* spp., *Proteus vulgaris, Fusobacterium necrophorum, Bacteroides* spp., *and Klebsiella* spp. Many of these organisms are opportunistic in that they may be normal inhabitants of the skin or mucous membranes, and only become pathogens when an animal is stressed, there is a break in the skin, they are in combinations with organisms that enhance each other, or there is overwhelming contamination.

Epidemiology

Abscesses occur worldwide. An abscess may be a pure culture of one of the species mentioned, but mixed infections occur. *T. pyogenes* infection in combination with *Fusobacterium* or *Bacteroides* is common [27].

Clinical Signs

Abscesses may be small (pustules, 1 mm) or large 12–15 cm (5–6 in.), soft and fluctuant, firm, cold or hot to the touch, thin- or thick-walled, painful or not to palpation. The exudate may be thin, thick, caseated, colored (whitish, yellowish, greenish, reddish), or have an odor or no odor.

Diagnosis

Superficial abscesses may be observed or palpated. A large bore needle (12 or 16 gauge) may be used to aspirate the exudate after surgical preparations of the skin. Appropriate culture and sensitivity procedures should be performed. Sometimes, the exudate is inspissated and may not be aspirated. In those instances, a small amount of sterile saline (after sterile preparation of site) can be added to aid in obtaining a sample. Occasionally, if the infection is contained to the wall of the abscess, the exudate is sterile.

Management

Most superficial abscesses are treated by surgical drainage and lavage. The incision should be triangular and not an ellipse or simple stab incision (Figure 9.31). The goal is to delay the closing of the incision until it is fully healed. In a large abscess, packing with gauze (brown roll gauze) soaked in straight betadine solution will help to cauterize the pyogenic membrane lining the abscess capsule. The packing should be removed the next day and repeated as

Figure 9.31 Large abscess on shoulder of an alpaca. Red triangle illustrates the preferred method to lance.

necessary until the cavity is too small to pack. At that time, straight betadine solution can be instilled with a syringe until the opening closes. It may be necessary to bring the abscess to maturity with a counter irritant such as hot compresses or ichthammol ointment. The use of antimicrobial therapy is debatable unless vital organs are nearby. Frequently, antibiotic therapy will diminish abscess activity but when the antibiotic is discontinued the activity will resume. This is particularly true of thick-capsuled *C. pseudotuberculosis* abscesses. The antibiotic simply doesn't reach the organisms. After the abscess is lanced and drained, if it is deep and there is much soft-tissue involvement, then a systemic antibiotic can be used. If possible, sensitivity patterns should be used to establish the appropriate antibiotic. Frequently, an antibiotic with action against anaerobes such as Procaine Penicillin G (300 000 IU/mL) is an appropriate choice at 44 000 IU/kg, once daily, subcutaneously (SC), until culture results are received.

Pseudotuberculosis (Caseous Lymphadenitis – CLA)

Pseudotuberculosis, caused by *C. pseudotuberculosis,* is worldwide in sheep and goats [27, 61–66]. It is extensive in OWC and has been reported in alpacas in Peru [66] (Figure 9.32a and b).

Etiology

It was first described by the French veterinarian Nocard in 1888. It is a short, gram positive rod that is unevenly ovoid. Two or possibly more toxins are produced. One is a toxic cell-wall lipid associated with virulence of the bacterium and the second toxin is an exotoxin (phospholipase D) that damages endothelium which increases vascular permeability and enhances bacterial invasion [27]. Two distinct strains have been identified: sheep/goat and horse/cattle strain.

Epidemiology

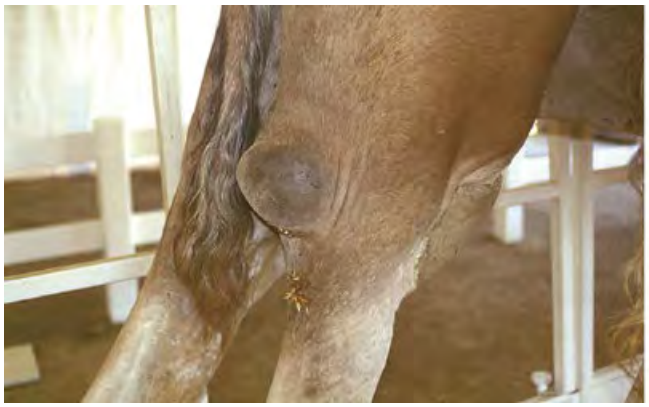
Camel CLA has been reported in Africa, the Middle East, Asia [67], Russia – anywhere camels can be found. Only the sheep/goat strain has been isolated in camels. Unlike sheep and goats, where pure cultures of *C. pseudotuberculosis* are isolated from affected lesions, it is not uncommon in camels to have a mixed infection with such bacteria as *Streptococcus* spp., *Staphylococcus* spp., *T. pyogenes* [27], or *C. ulcerans* [68]. Lymphadenopathy with or without abscesses can be seen in camels. Camels are exposed to harsh environments predisposing to skin abrasions from thorns, dry hard stems out in the desert, ticks, contaminated needles used for injections, skin parasites (the nodular worm), all creating opportunities for entry of *C. pseudotuberculosis*. Because of this type of environment, it is more common for CLA to appear in free grazing camels (greater than three years old), rather than racing camels that are kept in enclosures [27].

In Europe, as SACs travel and are imported from country to country, the report of CLA infections in alpacas have increased with mortality reaching 22% in SACs, mainly when internal organs are involved [69]. Cases of CLA in North America continue to be rare and easily managed on an individual basis.

Clinical Signs and Treatment

In sheep and goats, abscesses form in about 25–40 days, this is similar in camels. Unlike sheep and goats, it is rare to have internal abscesses in camels. The classic "onion" ring of caseous, inspissated material contained within CLA abscesses of small ruminants, is not found in CLA abscesses

(a)



(b)

Figure 9.32 (a) Corynebacterium pseudotuberculosis abscess on a camel's leg, (b) Abscessed submandibular lymph node caused by pseudotuberculosis in a camel.

in camels; the purulent material is thick, yellow/cream-colored. The abscesses are closed, painless swellings, in the external lymph nodes at the base of the neck, prescapular and submandibular lymph nodes (Figure 9.32b). They can be quite large 7–15 cm [27]. These animals serve as a source of infection to the rest of the herd and should be isolated. The abscess should be, lanced and drained in a controlled manner to contain the purulent material. Procaine Penicillin G (300 000 IU/mL) at 44 000 IU/kg, SC is the treatment of choice for these gram-positive bacteria. Once the abscess is opened and drained, the Penicillin should be more effective as the Penicillin will not penetrate the thick-walled off abscess. Adding Isoniazid (20 mg/kg, orally, daily for 30 days) may help the Penicillin to penetrate the abscess. However, the potential contamination of the environment from the draining abscess is a biosecurity problem for the remaining animals in the herd. Many treatments have been proposed to minimize the contamination of the environment, namely injecting the abscess with formalin, waiting approximately 10 minutes, then removing the formalin along with any purulent debris. This may kill the bacteria, but there is a great risk of causing necrosis and a deeper infection. Another proposed treatment is IV injection of 20% sodium iodide in 500 mL NaCl as a single treatment which can be repeated two to three weeks later. The abscess will slowly regress [69]. Careful use of sodium iodide in camelids is warranted, as some can become profoundly anorexic.

A study involving sheep and goats has been done using closed intralesional lavage with saline plus intralesional injection of tulathromycin (2.5 mg/kg). The saline and purulent material were removed (16-gauge needle) and tulathromycin was injected into the empty abscess cavity. A second group had the same intralesional lavage and evacuation and then a subcutaneous injection of tulathromycin (2.5 mg/kg) [70]. The lesions in both groups treated without opening the abscess resolved similarly to those in a third group who had the abscess lanced and flushed with betadine solution and a single injection of Procaine Penicillin G (20 000 IU/kg, SC) administered. The advantage of treatment without opening the abscess is that it greatly decreases the risk of environmental contamination [70].

Control

Many different approaches have been used to develop vaccines against CLA in camelids around the world. Based on the varied results, it appears there are genetically different *C. pseudotuberculosis* populations and the use of autogenous herd-specific vaccines from other host species may not work in different herds or group of camelids. This will require the development of herd-specific autogenous vaccines. Research is ongoing to try and control this debilitating disease [69].

Folliculitis and furunculosis (*S. aureus* dermatitis, botryomycosis, pyoderma, contagious skin necrosis)

Etiology and Clinical Signs

A bacterial skin infection caused by *S. aureus* and *S. intermedius* most often, with other bacteria sometime implicated [71]. It is known by many different names depending on which species it affects. *S. aureus* is a ubiquitous, Gram-positive bacterium found on the skin and nasopharynx in animals and humans. It can become pathogenic in certain circumstances. It causes a variety of suppurative infections and has always been a major cause of wounds and abscesses and mastitis in animals. It can be seen in OWC, especially in the young [27]. Botryomycosis (defined as a chronic purulent granulomatous infection) has also been diagnosed from an alpaca with many dermal abscesses with positive culture for *S. aureus* [71]. The abscesses in the alpaca varied from 0.5 to 4 cm in diameter and were located on the medial aspect of the thigh (Figure 9.33a and b). Treatment was successful and consisted of surgical

(a)

(b)

Figure 9.33 (a and b) Botryomycosis (Staphylococcus) in an alpaca.

extirpation of the abscesses followed by broad-spectrum antibiotic therapy for four weeks [49].

Botryomycosis was first thought to be a fungal infection, hence the name, but was subsequently identified as a local skin infection caused by *S. aureus*.

Lesions can occur anywhere on the body but seem to be most common on the head (Figure 9.34a and b), extremities and between the front legs (camel calves). There may be areas of hair loss, progressing to crusts and scabs. If abscesses are present, they are usually found in clusters in affected areas. When the scabs are removed, there may be a small depression filled with purulent debris. In larger abscess, there is thick, whitish-green purulent debris.

Treatment and control

Because there is potential for antibiotic resistance of *S. aureus*, culture and sensitivity should be done. If possible, surgical excision of the complete abscess should be undertaken. With true Botryomycosis there are frequently multiple abscess clustered together, and this may not be possible. When lancing the abscesses or if the abscess is already ruptured, strict biosecurity is important to minimize contaminating the environment. Lavage the abscess with dilute betadine, and pack the cavity if necessary, with betadine-soaked gauze. Remove the packing the next day, and repeat packing as needed until cavity is small. Depending on the severity and number of the abscesses, placing the animal on antibiotics until the sensitivity is known is recommended. Procaine Penicillin G at 44 000 IU/kg, SC, daily for 7–10 days or Ceftiofur at 4–6 mg/kg, SC, daily for 7–10 days are good initial antibiotic selections. If it is a diffuse skin infection as seen in Figure 9.34a and b, then systemic antibiotic and topical cleaning with a gentle disinfectant will resolve the lesions.

In outbreaks of *S. aureus* dermatitis in camels in the Middle East, autogenous bacterins have been made and used successfully.

Differential diagnoses include Pseudotuberculosis, dermatophytosis, secondary bacterial infection from chorioptic/sarcoptic mange, and zinc responsive dermatitis in early stages.

Dermatophilosis (streptothricosis, rain scald, rain rot)

Etiology and Epidemiology

Dermatophilosis, caused by *Dermatophilosis congolensis*, is found worldwide, but infection is more prevalent in areas of high rainfall (and snow) and in tropical areas. Also, to be seen where animals spend most of the time outdoors in bad weather. There is generally some type of skin damage present to allow the organism to proliferate. The disease is characterized as an ulcerative, exudative dermatitis [72]. It can be seen in cattle, horses, small ruminants and some exotic animals if the conditions are correct. Dermatophilosis is a zoonotic condition, though rare, can be transmitted to humans working with infected animals. In cattle, there does seem to be a breed susceptibility with European descended cattle being most susceptible. It can be known by different names depending on the species it infects, such as lumpy wool disease and strawberry foot rot in sheep. It does not appear to be common in SACs though reported in camels in Africa, India, and the Middle East [27].

Lesions can be anywhere, but most commonly on the dorsum where there is heavy fiber cover in camelids and moisture will tend to collect. It can also be seen on the sides and distal extremities. As stated above, there must be some break in the skin to allow the organisms to proliferate as it

Figure 9.34 (a) Staphylococcus aureus dermatitis (severe) involving the entire face. (b) Same animal nine days later after systemic antibiotics and gentle cleansing of areas involved. There was complete healing after several weeks of topical treatment.

is thought that *D. congolensis* is unable to invade healthy skin. Depending on the climate and time of year, external parasites such as flies, lice, and ticks may cause enough damage to the skin and help spread the infection. Other sources of skin injury include abrasions from thorns, scratching, rubbing, licking or shearing.

Clinical Signs and Treatment

D. congolensis is a Gram-positive, facultative anaerobic actinomycete that produces motile spores (zoospores) that are released in the rainy season. They can be transmitted by direct contact or by flies and ticks or fomites. The exudative reaction of the skin to the organism releases a serous exudate that forms a crust. If long hair/fiber is involved, it becomes matted with this exudate and forms the classic "paint-brush" effect with the hair/fiber clustered together. When these tuffs of hair are removed, underneath is a moist, pink, hyperemic wound that may, in some animals, be painful to the touch. To help with diagnosis, direct smears should be made with these crusts and stained with Diff-Quick or Gram stain looking for the "railroad" tracts of bacteria. In camelids with short hair, the lesions may present as nodules or raised areas with scabs. Underneath the crusts are the same moist lesions described above. Scabs or crusts can be submitted for bacterial culture or histopathologic examination.

Treatment consists of removing the crusts, clipping the long fiber away to allow air and drying. The area should be cleaned daily with dilute Betadine solution or Chlorhexidine, dried and Betadine ointment or topical antibiotic applied. Depending on the severity of the case, administering parenteral Procaine Penicillin G (44 000 IU/kg, SC, daily for 7–10 days) or Ceftiofur (4 mg/kg, SC, daily for 7–10 days) will shorten the healing time. Keep cleaning and using topical disinfectants until healed. Examine the rest of the camelid carefully for additional lesions and remaining animals in the herd. Review nutrition and ectoparasite management to see if any underlying predisposition can be determined.

Differential diagnoses include bacterial folliculitis, dermatophytosis, botryomycosis.

Nutritional Skin Diseases

Zinc-Responsive Dermatosis (Idiopathic Hyperkeratotic Dermatosis)

Zinc responsive dermatosis has been described in goats [73] and zinc deficiency in sheep, goats, dogs, and laboratory animals. The condition is apparently a common disorder in SACs but challenging to recognize and to treat. It may be seen in SACs of any age but is usually found in mature adults. It can occur in both sexes, but males seem to predominate. Only one or just a few individuals in a herd may be affected. Black SACs may have a predilection for the disease and there are indications that colored hair contains more zinc and copper than white hair [74]. In goats, it has been suggested that there is a heritable component with zinc-responsive dermatosis [73]. Pharmacologically, zinc is an immune system modulator and also has some anti-inflammatory effects.

Skin biopsies in SACs with zinc deficiency show papillated epidermal hyperplasia, predominantly lamellar orthokeratotic hyperkeratosis (epithelial, follicular), and mild to moderate superficial lympho-eosinophilic perivascular dermatitis [22, 58].

Zinc homeostasis is primarily controlled by absorption in the intestinal epithelium. There is not an identified storage pool of zinc in the body and tissue concentrations remain relatively steady across a wide range of zinc intake. If absorption is inadequate by the intestinal epithelium, then a relative zinc-deficiency will result in the face of adequate zinc concentrations in the diet [75]. Therefore, zinc deficiency may be the result of an absolute dietary deficiency, poor absorption from the intestine, or the binding of zinc with calcium in the intestinal tract. Zinc is a required trace element. A wide range of individual zinc requirements seem to be present in camelids, however, usually only one or a few animals in the herd are affected. To complicate the scenario, successive cuttings of hay within a season seem to be associated with declining zinc concentrations [75]. As a routine, camelids prefer second and third cutting grass hay which, for some individuals, may have inadequate zinc present.

The clinical syndrome in SACs is initially characterized by papules progressing to plaques, alopecia, dermal thickening, scaling (Figure 9.35) and hyperpigmentation with tightly adherent crusts. Early areas of involvement are the ventral abdomen, inguinal region, medial thorax and thighs, and axilla – relatively hairless areas. As the disease progresses, lesions will appear on the face, especially the bridge of the nose, pinna, and periocular region [22, 58]. Animals may be mildly pruritic or nonpruritic.

Care must be taken in obtaining samples for zinc analysis. Erythrocytes contain large amounts of zinc, so if red cells hemolyze, falsely elevated plasma levels will be found. Plasma or serum can be used, with possibly plasma being superior to reduce the chance of hemolysis. Rubber stoppers are frequently lubricated with a zinc-containing substance, so contact with the stopper must be avoided at all costs. There are trace mineral tubes available, Royal blue tubes with a plastic stopper [75]. If plasma is used, a heparinized tube (green top) with a layer of plastic wrap

Figure 9.35 Acute facial zinc-responsive dermatitis.

around the stopper should be used. Even with the plastic layer, avoid contact with the stopper. Keep the tube upright, spin immediately, and harvest the plasma for analysis. Ideally samples should be transported to the lab in Royal blue tubes. In North America, several diagnostic laboratories can run zinc concentration (trace mineral panels) with many laboratories sending samples to a referral laboratory (Michigan State University Veterinary Diagnostic Laboratory). Check with your states/countries diagnostic laboratory to confirm availability of tests.

A study done with 20 normal adult llamas in Colorado showed plasma zinc concentrations with a mean of 0.45 ppm and a range of 0.27–0.81 ppm [22]. Though obtaining zinc concentrations is part of the work-up for zinc-responsive dermatosis, it is typical, even expected, to find a normal concentration of zinc in the camelid being evaluated. This does not mean the diagnosis is faulty, just that this individual requires more zinc than average camelids. Biopsies of the affected areas will help to determine cause. If, however, plasma zinc levels are less than 0.45 ppm and biopsies show typical lesions, the diagnosis is confirmed.

Zinc-responsive dermatoses are treated by supplying zinc sulfate (36.3% zinc) in the diet at a dosage rate of 1–2 g/day. Improvement may be seen in as few as 30 days of supplementation but more likely two to three months. A more easily assimilated zinc supplement is zinc chelated to methionine, using a ZinPro® product. Zinc methionine has more effective absorption independent of calcium content of the diet. ZinPro comes in different concentrations and feeding 2–4 g/day of ZinPro 40 (providing 80–160 mg zinc) will be beneficial in camelids that have zinc-responsive dermatosis. This therapeutic concentration of zinc should be fed daily to effect and once the clinical signs have improved, zinc supplementation can be decreased to a lower daily maintenance dose. ZinPro has another zinc supplement (TruCare®) that has llama dosing on the bag. This dose represents only a minimal required amount and does not provide the therapeutic amounts needed for camelids that have zinc-responsive dermatosis. Zinc is not palatable and may be refused if top-dressed on the feed. To ensure administering the complete amount and for palatability, it should be individually dosed using apple sauce or juice. Daily maintenance feeding of the zinc supplement is required to eliminate clinical signs; a maintenance dose of 1 g/day (ZinPro 40) may be all that is required after complete resolution of the clinical signs [74]. Non-specific dermatoses of camelids are often treated empirically by adding supplemental zinc to the diet. Colleagues report variable success as a result of this treatment, possibly because a precise diagnosis has not been made and all dermatoses are treated alike.

Miscellaneous Dermatoses

Follicular/Sebaceous Gland Cysts

When doing routine physical exam of alpacas, both Suri and Huacaya, it is common in some individuals, to find masses just under their skin [22, 76]. These masses will generally be along either side of the thoracic and lumbar spine and sometimes cervical spine. Though not as common, they can also be found over the thoracic area, hips and thighs. They are nonmobile masses that appear in adults, with more developing as the alpaca ages. These are follicular/sebaceous gland cysts and are innocuous. On occasion, one will have to be surgically removed if they are in problematic area or appear atypical (Figure 9.36a–c). Figure 9.36c shows a Trichoepithelioma. Trichoepithelioma are differentiated from follicular cysts by the presence of neoplastic follicular cells [77]. The producer may be unaware of these masses until shearing, when removal of the fiber reveals numerous bumps/lumps (Figure 9.37). On occasion, they will rupture on their own and release a yellow, tan or black waxy plug, leaving a crater behind. This is also what will be found in some animals after they are shorn, and the clippers have removed the skin over the cyst (Figure 9.38).

Treatment consists of cleaning the crater with hydrogen peroxide, betadine and spraying with Catron® IV wound-fly spray to control flies. It would be rare to have a secondary infection if fly control is maintained until healing, even if there are numerous open cysts after shearing. They will heal in five to seven days. They may or may not come back in the same spot after healing. Once ruptured and healed, it seems there are fewer present the next year when sheared (personal communication by experienced camelid shearer).

Figure 9.36 (a) Follicular/sebaceous gland cyst. (b) Site of surgical removal of follicular cyst in Figure 9.34a. (c) Atypical location of an atypical follicular cyst (side of thigh – Trichoepithelioma) – it was surgically removed.

Figure 9.37 Typical follicular cyst.

The cysts are an accumulation of the normal waxy sebum produced by the sebaceous gland associated with each hair follicle. There has not been a formal study to determine why they are only seen in certain individuals and multiply as they age. It may be a genetic predisposition or possibly seen in alpacas with denser fleece.

Figure 9.38 Follicular cyst after shearing ruptured the cyst.

Idiopathic Nasal/Perioral Hyperkeratotic Dermatosis (Munge)

As the name "idiopathic nasal/perioral hyperkeratotic dermatosis" suggests, this is a catchall dermatosis that may have multiple etiologies.

It is most often seen in crias and juveniles under two years of age, however, it can occasionally be seen in adults. It does not appear to be contagious as not all crias are involved. When first noticed by producers, they thought that the crias mouth was just dirty. It is not uncommon to see this when doing cria show checks for the first time in the season.

Clinical Signs

It appears as heavy crusts (hyperkeratotic) that are strongly adherent to the perioral and sometimes the perinasal area.

Under the crusts, the skin is hyperemic, inflamed and tender to the touch. In some cases, lesions may be on the bridge of the nose, and around the ears and eyes. On occasion, crusts may be so severe as to cause nasal obstruction [76]. In mild cases limited to the mouth, it appears as if the cria has a local, temporary, sensitivity to their saliva and a secondary bacterial infection has occurred in the irritated tissue at the commissures of the lips. Once these lesions are treated and fully healed, they do not seem to recur as the crias grow older.

Diagnosis

A seropurulent crust on the surface of the skin is common and made up of alternating layers of degenerative nuclear and parakeratotic/orthokeratotic debris with a tendency to palisade. Microabscesses may develop in the substratum corneum. Presence of these histologic lesions does not confirm the diagnosis; however, it may rule out other etiologies. Differential diagnoses include CE, bacterial folliculitis, dermatophilosis, mange mites, viruses and underlying immunodeficiencies.

Treatment

In milder cases only topical therapy is needed (Figure 9.39a and b). As it seems there is an inflammatory as well as a bacterial component to the condition, topical products with both antibiotics and steroid activity can be applied. One simple topical treatment is made by taking over-the-counter triple antibiotic ointment and combining equal parts with over-the-counter hydrocortisone ointment. The compounded ointment is applied daily until lesions resolve. It is helpful to clean the area every two days with a mild disinfectant before applying the ointment. This will also, over time, encourage the crust to loosen and come off. In some crias it can be painful, so gentle handling is warranted. In more severe cases (Figures 9.40a, b and 9.41a–c), secondary bacterial infection must be treated with systemic antibiotics (Procaine Penicillin G 44 000 IU/kg, SC, daily for five to seven days, or Ceftiofur 4 mg/kg, SC, daily for five to seven days). In severe cases, affected crias may develop fatal sepsis.

Idiopathic Neutrophilic/Necrolytic/Hyperkeratotic Dermatosis (Generalized Munge)

As with munge described above, "idiopathic neutrophilic/necrolytic/hyperkeratotic dermatosis" is a catchall dermatosis that may have multiple etiologies. It is also seen in young camelids under two years old. Generalized munge may just be a more severe condition, involving more of the body than plain munge. Though the author does still see munge, generalized munge is not as common as previous.

Clinical Signs

Erythema, crusting is seen in the perinasal and perioral areas as in munge, but in this case the lesions extend to involving the axilla, inguinal area, ventral abdomen, distal extremities, and perineum (Figure 9.42). Animals are usually not pruritic. Erythema, crusting, and vesicle-like structures indicate necrotic epidermis. It is common to see lesions like those shown in Figures 9.43 when doing reproductive examinations on females. This will be the only area of the skin involved. It does not seem to change much over time but may wax and wane in severity in some individuals.

(a)

(b)

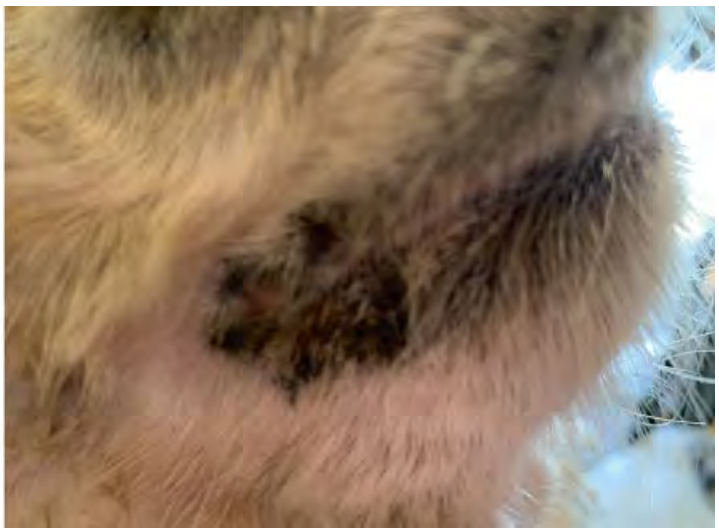


Figure 9.39 (a and b) Mild cases of munge in crias.

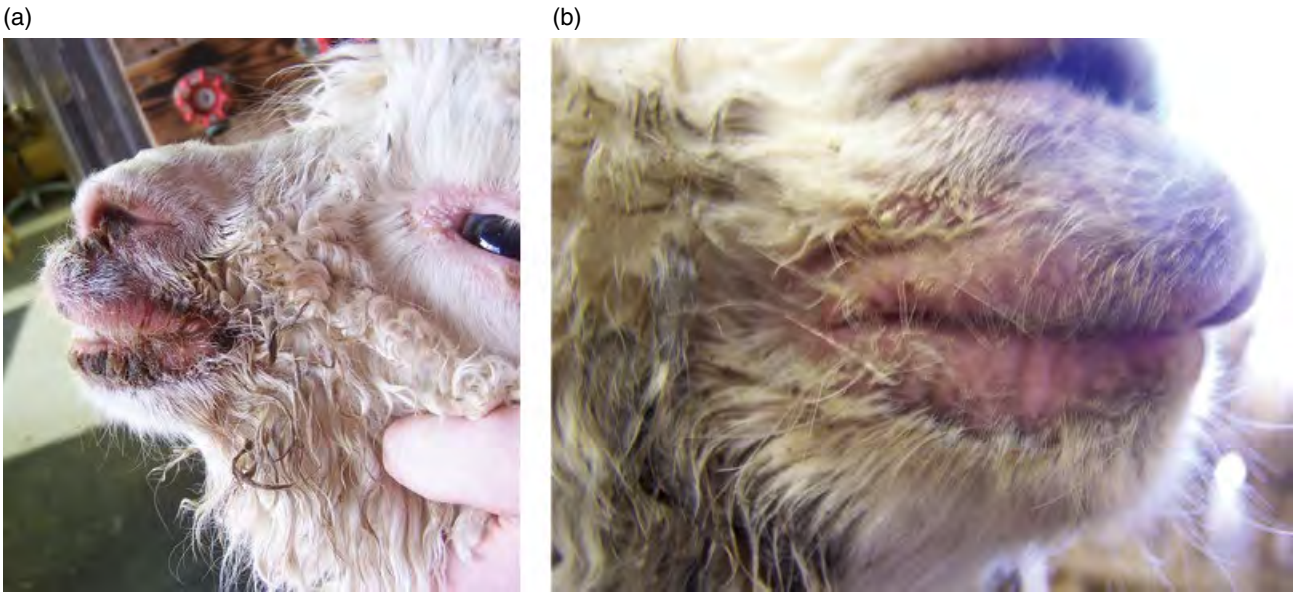


Figure 9.40 (a) Moderately severe munge in a cria. Note the fissures and inflammation. (b) Same cria 14 days later after systemic antibiotics and topical treatment with triple antibiotic ointment and hydrocortisone ointment.

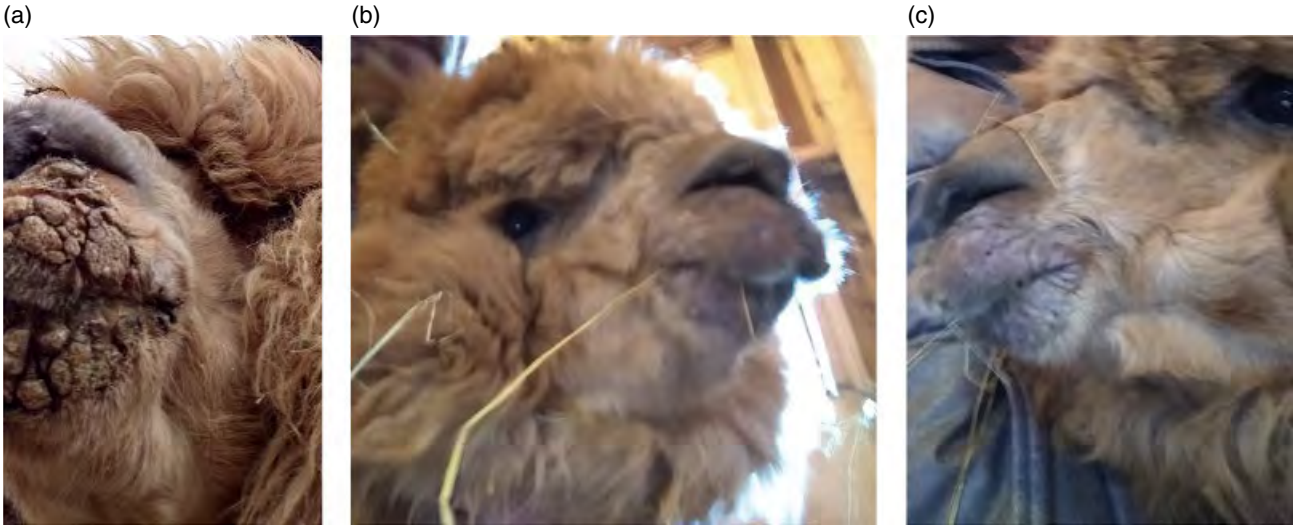


Figure 9.41 (a) Severe case of munge in a yearling alpaca. (b) Same yearling 17 days later after treatment with Animax ointment. (c) Same yearling 17 days later after treatment with Animax ointment. *Source:* Photos courtesy of Dr. Beth Sondgeroth.

Diagnosis

Microscopic lesions include ballooning degeneration of the superficial epidermis followed by a neutrophil infiltration. Micro-abscesses may develop in the substratum corneum and eosinophils may be present in increased numbers. Additional lesions include orthokeratotic and parakeratotic hyperkeratosis with a tendency to palisade. Nuclear and cellular debris may alternate with hyperkeratotic debris. Differential diagnosis is bacterial folliculitis, dermatophilosis, mange-mite infection, autoimmune disease and pemphigus.

Therapy

Secondary bacterial infection must be handled with antibiotics. Some of the more severe cases respond to oral corticosteroid therapy. A suggested regimen is to administer prednisone 2 mg/kg/day orally for one to two weeks. If responding well, decrease to 1 mg/kg/day orally for an additional two weeks, then administer this dose every other day for an additional two weeks, and then two times per week for two weeks, then discontinue therapy. Monitor for appetite and attitude carefully when using this therapy. When only local areas are affected, one can use chlorhexidine

Figure 9.42 Acute nonspecific dermatitis.

Figure 9.43 Nonspecific dermatitis, seen during reproductive exams in otherwise normal females.

scrub to clean the areas. The areas should be rinsed, dried and chlorhexidine ointment applied to affected areas. This can be done three times a week until lesions resolve.

It has been suggested to add oral zinc to the treatment and some animals do seem to respond to the higher doses. See Zinc-responsive dermatitis in the section above. As we do not know the cause of this condition, recurrence after cessation of treatment may occur and the producer should be warned.

Burns (Photosensitization, actinic dermatitis, solar dermatitis)

Burns are caused by contact with dry heat (fire, friction), moist heat (steam, hot liquid), chemicals (caustic or corrosive substances), electricity (faulty wiring or lightning) or electromagnetic energy (sunburn, X-rays). In camelids, burns are most likely sustained in barn or forest fires or automobile accidents, or they result from entanglement and struggling in ropes or electrified fencing (Figure 9.44). Light-skinned camelids are at risk of sunburn after shearing or if there is minimal hair on the pinna. The same is true of animals that have undergone a surgical procedure necessitating close clipping of an area that may be exposed to sunlight. Photosensitization may occur with hepatopathy (Figures 9.45 and 9.46) or ingestion of primary photosensitizing agents.

(a)

(b)

Figure 9.44 (a) Alpaca burn wound on footpad resulting from a forest fire. The third phalanx is exposed. (b) Footpad after the wound has healed. *Source:* Photos courtesy of Dr. Julie Dechant.

Figure 9.45 Photosensitization of the muzzle associated with fascioliasis.

Figure 9.46 Photosensitization of the ears of a llama cria.

Clinical Signs

Burns range in severity from reddening of the skin (first-degree burn), to blistered, swollen skin (second-degree burn), to charred skin with damage to deeper structures (third-degree burn). The surrounding fiber may become matted with fluid escaping from the injured skin. First- and second-degree burned areas are painful to the touch. In first-degree burns, the superficial layers of the skin will slough in three to five days. It may take two to three weeks for third-degree burns to start to slough depending on the size.

Severe burns open the skin to the invasion of infectious agents and the loss of fluids. Danger depends on the area of the body affected and the degree of damage. Depending on the number and location of the third-degree burns, the burned areas will coalesce to become larger areas.

Management

If the burn is witnessed, cooling the area by spraying or immersing in clean cold water may be beneficial. The application of an ice pack is indicated if available. Second- and third-degree burns should be covered with a nonadherent dressing to minimize fluid loss and prevent contamination and infection of devitalized tissue. Do not open blisters.

Chemical burns present different challenges. Any residual substances should be removed with clean water. Avoid using soap or disinfectant solutions unless the specific caustic chemical is known. Phenolic disinfectant burns may be neutralized with rubbing alcohol. Silver nitrate, found in some styptic sticks, should be neutralized with a saline solution.

The author had a case of third-degree burns that involved a mature male llama. This llama was outside of a barn fire and unbeknownst to the owner, had numerous ashes fall into his fiber. A blanket was put on him for the night and he was taken to the referral hospital the next day. He was bright with normal parameters upon admission. During his examination, ashes kept falling out of his fiber. Once he was shorn, it was estimated that 40% of the body was involved, including the face, ears, brisket, scrotum and pads of the feet in addition to his top-line, sides, and thighs. Initially, there were discrete areas that had a "bulls' eye" type appearance with red centers that enlarged, and over the next three weeks, these areas coalesced to become even larger areas. As soon as it was apparent which tissue was devitalized, surgery was done to debride the dead tissue. One area on the back did not heal and it was determined that the dorsal spinous process of the vertebrae was necrotic and required removal before healing proceeded. When the patient was first admitted, he was placed on Ceftiofur 4 mg/kg, SC, twice daily for two weeks. This was repeated after surgery. With much nursing care, this llama was able to go home after three months. Since he sustained third-degree burns, he had minimal fiber left on the burned areas. As he was mainly a white llama, the owner was warned to keep him covered to prevent sunburn. Most livestock die due to smoke inhalation from being involved in barn fires; in this case, the llama did not have smoke inhalation and was able to be saved. No secondary infection occurred, and he maintained a good attitude and appetite for the duration of treatment.

Ichthyosis

There are not many cases of Ichthyosis in the literature. Two cases involve congenital ichthyosis, one in a llama cria and one in an alpaca cria [78, 79]. The llama was a one-month-old cria that had diffuse hyperkeratosis and

conjunctivitis. Both had been present since birth and the entire body was covered by irregular hyperkeratotic plates. There was some fiber present. The most severely affected areas were mucocutaneous junctions, and the nostrils were partially occluded. The skin had deep ridges and did not have normal pliability. Ambulation was difficult due to involvement in the axillary and inguinal regions. The conjunctivitis was due to hyperkeratosis involving the eyelids. Biopsies were taken and showed excess compact to laminated orthokeratosis involving the epidermis and follicular infundibula. There was mild hypergranulosis and epidermal hyperplasia in the stratum corneum. The hair follicles were actively growing but had keratin plugging of the follicles. Secondary infection with *Malassezia* spp. was also present. Electron microscopy showed excess inter- and intracellular vacuolation in the stratum corneum and accumulation of greater than normal numbers of liposomes. Attempt at treatment was tried, but the cria had complications and was humanely euthanized. The diagnosis of Congenital Ichthyosis was made based on the clinical, histopathologic and ultrastructural findings [78].

The case involving the alpaca was a four-month-old cria that was born small and had an unknown skin condition since birth. She was small for her age and had never thrived. She had generalized, dry scaly, lichenified skin with a rough haircoat. There were crust and pustules at the upper lips, conjunctivitis and deformed toenails. The cria was humanely euthanized due to poor condition. At necropsy, there was scattered serocellular crusts containing degenerative neutrophils in the epidermis with marked orthokeratotic hyperkeratosis. The hair follicles had hair shafts present and were in various stages of growth. Most were filled with keratin. No other pathogens were detected in stained sections of the skin. Beneath the crusts, there was multifocally mild perivascular dermal infiltration by lymphocytes and plasma cells. The epidermis and infundibular stratum corneum were thickened but there were only two to four nucleated cell layers present in the epidermis. This cria also was diagnosed with Congenital Ichthyosis based on the clinical and histopathologic findings [79].

Ichthyosis is rare in livestock, and most reported cases are congenital. One, unreported case involved an adult female Suri, alpaca (Figure 9.47a–c) that had chronic, generalized hyperkeratosis for several years. The signs were not present at birth and slowly developed around four to five years of age. She had several crias before any problems were noted. Only one of her crias went on to have mild clinical signs similar to her dam. The owners tried many treatments, both at home and at a referral institute with no improvement. She was euthanized and the body submitted for necropsy. The diagnosis at necropsy was Ichthyosis. This case is atypical and may represent a variation of the condition.

Pemphigus Vulgaris

Pemphigus is a rare autoimmune skin disease [80]. One reported case involved a three-year-old llama male that had seborrhea, with mild pruritus for nine months. Examination showed alopecia and hyperkeratosis with pinpoint crusts on the muzzle, periocular, ears and inside portion of all four legs. Lesions progressed over the next three weeks to heavy crust on the neck and trunk with numerous vesicles on the ventral abdomen and medial thighs. These vesicles showed numerous neutrophils, some eosinophils, but no

(a)

(b)

Figure 9.47 (a and b) Ichthyosis in an adult alpaca.

bacteria. Biopsies showed acantholytic vesicular dermatitis with intraepidermal separation in the suprabasilar location. The antinuclear antibody test and LE cell preparation were negative, but the indirect immunofluorescence testing was positive to a titer of 1:320. Oral prednisolone (4.3 mg/kg, orally, daily) was started, but the animal died of unrelated reasons three days later. Diagnosis, based on the histological findings, was Pemphigus vulgaris [80].

Insect bite hypersensitivity (Idiopathic urticaria)

Camelids live outside and are exposed to many biting insects. There is an individual sensitivity to the bites of various insects, just as with individual humans (mosquitoes). *Culicoides* spp. gnats seem to be the most common culprit. Generally symmetrical, involving the ears, periocular area, bridge of nose and ventral surface of body to varying degrees. The lesions will initially be small, raised bumps progressing to alopecia, some crust and licenification with occasional papules in severe cases. The animals may or may not be pruritic. Skin scraping should be done to rule out common causes such as mange mites [81]. Frequently, adults will become symptomatic annually with resolution of clinical signs in the winter. In crias in their first summer, they may be the only one of a group affected, even though everyone is outside at the same time.

Cytological exam will reveal eosinophils, with possible bacteria. Histopathologic findings will be eosinophilic interstitial dermatitis and folliculitis. In pruritic cases, there may be orthokeratotic hyperkeratosis indicating rubbing and scratching [81].

Treatment is based on severity of clinical signs. Topical steroids, with or without antibiotics is the basis for therapy. Chlorhexidine ointment can be used to provide topical relief, and one or two doses of systemic anti-inflammatory (Flunixin meglumine 1.1 mg/kg, SC) is often beneficial. Permethrin-containing sprays can be used, and animals should be brought in before dusk. If the areas are limited to the ears, a protective coating of coal/pine-tar can be applied as a barrier.

Adverse Drug Reaction

Adverse drug reactions will not be directly related to the pharmacologic, physical or chemical properties of the drug and are independent of dose. They are rare and unpredictable. A hypersensitivity is probably the cause, though hard to document. They can resemble many skin conditions and can involve any part of the integument [81]. A history of contact with a novel product or a recent history of drug injection will support the diagnosis.

A presumptive diagnosis of cutaneous drug reaction was reported in a five-year-old male alpaca after receiving an injection of ivermectin. The owners reported bumps on the ears that progressed to ulcers and swelling of the pinna. The alpaca become depressed, and anorexic with low-grade fever. The lesions started to appear on the ventral abdomen and perineum, inguinal and axillary area. Cytologic exam of the exudate showed eosinophils, nondegenerative neutrophils and no bacteria; cultures were negative. Biopsies showed eosinophilic and neutrophilic epidermitis and luminal folliculitis and furunculosis. He was treated with methylprednisolone (1 mg/kg, orally, daily) and fully recovered. After a 12-month follow-up period and no ivermectin, he remained normal [81]. It was not indicated if he had received ivermectin previously.

To confirm the diagnosis, a repeat injection/application of the drug in question would need to be done; however, this is not advisable, as subsequent reactions can be more severe. Scott recommends the follow criteria to diagnose an adverse drug reaction: (i) a drug not received previously is given for more than seven days prior to onset of reaction (ii) the reaction occurs while the patient is receiving the drug or the drug has only recently been stopped (iii) stopping the drug stops the reaction, usually within 7–14 days (iv) if repeat exposure occurs, a similar, more rapid reaction occurs (v) other causes for the dermatitis are ruled out (vi) characteristics of the dermatitis are consistent with a drug reaction and correlate with the reaction the suspect drug is known to cause [58].

Neoplastic and non-neoplastic tumors

Tumors of the skin are rare in camelids but include squamous cell carcinoma [58], lipoma [58], rhabdomyosarcoma [58], metastatic mammary carcinoma, sebaceous carcinoma of the eyelid [82], retinoblasoma [83], and tricoepithelioma [77, 81] (Figure 9.36c), fibroma [81], melanocytoma [81], lymphoma [81], and mast cell tumors [81].

The Foot

The Normal Foot

The camelid foot is unique, with two digits on each foot [84, 85]. The plantar surface is covered with a soft, cornified layer of epithelium (called the slipper) similar to that of the bulb of the heel in sheep and goats (Figures 9.48 and 9.49). In SACs there is a separate slipper for each digit, while in the camel, a single slipper covers the entire weight-bearing

Figure 9.48 Bottom view of llama foot.

Figure 9.49 Bottom view of adult alpaca foot.

surface. Deep to the slipper is a layer of dense connective tissue, the corium, containing blood vessels and nerves.

Camelids are modified digitigrades. In SACs phalanx 2 (P-2) and P-3 lie horizontally within the foot and P-1 upright at approximately 45° (Figures 9.50–9.55). In camels, P-2 and P-3 are more vertical. A small, non-weight-bearing nail, similar to a human nail, is located at the extremity of each digit and closely attached to P-3 via the corium [84]. Primary nail growth occurs at the coronary band (Figure 9.56).

The suborder name for camelids is Tylopoda, meaning padded foot, so named because of the digital cushion interspersed between the slipper, corium, and P-2 and P-3 [84]. The digital cushion consists of a poorly vascularized meshwork of collagenous and elastic fibers with interspersed masses of fat and, occasionally, cartilage tissue, all encased in a fibrous sheath (Figure 9.56) [22, 76]. The toenail has laminae which attaches the nail to P-3, (Figures 9.57a, b and 9.58). The camel foot is illustrated in Figures 9.59 and 9.60.

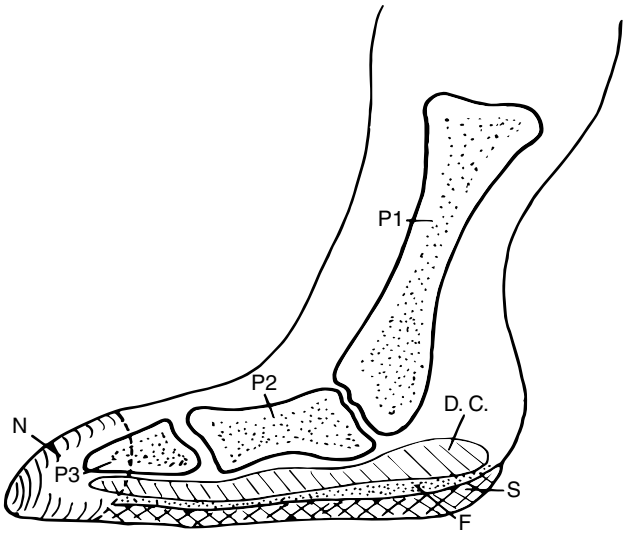


Figure 9.50 Diagram of SAC foot and pastern. (N) Toenail, (DC) digital cushion, (F) sole corium, (S) sole (slipper).

Figure 9.51 Lateral radiograph of the foot, pastern, and fetlock of a llama. (A) Digital cushion.

Foot Diseases of Camelids

Diseases of the toenail

Overgrowth of the toenail is the most common disorder of the SACs foot (Figures 9.61–9.65). The cause may be insufficient wear or congenital curling of the toenail in which the nail is pushed out of position for normal wear. The toenails should be trimmed as needed, using Saboten hoof-trimming shears (alpacas, Figure 9.66) or ARS sheep

Figure 9.52 Normal llama foot, lateral view.

Figure 9.53 Normal alpaca foot, lateral view.

Figure 9.54 Normal llama foot, dorsal view.

Figure 9.55 Normal alpaca foot, dorsal view.

Figure 9.56 Digital cushion, lateral view.

hoof-trimming shears (alpacas, llamas, Figure 9.67), Some llama feet will require more heavy trimmers like equine hoof nippers (Figure 9.68). Some SACs have a heavy ridge on the dorsal surface of the nail. This appears to contribute to the curling. The author recommends that this ridge be trimmed or rasped off to give the nail more flexibility. Camel toes will also have to be periodically trimmed depending on their environment or they can grow into the foot pad (Figure 9.69a and b).

When trimming the toes, proper restraint is important for the safety of the handlers, and for the animal. Frequently, llamas are acclimated to a chute and that will be the best place to trim llama toes. Alpacas may be held in a chute but are rarely comfortable and will usually fight being restrained in this manner. With many alpacas, it is

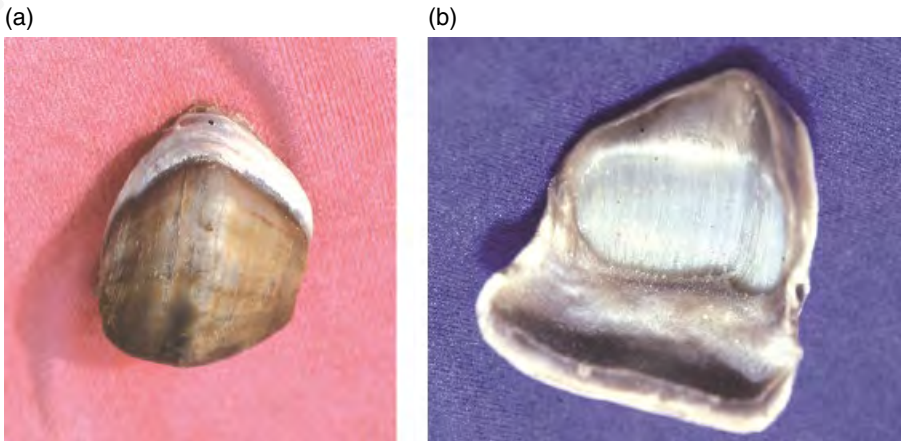


Figure 9.57 (a) Toenail of a camel. (b) Lamina of a toenail of a camel.

Figure 9.58 Lamina of toenail of a llama.

Figure 9.60 Camel foot dorsal view.

Figure 9.59 Bottom of a camel foot.

Figure 9.61 Elongated toenails on a llama.

Figure 9.62 Elongated toenails on a llama.

Figure 9.63 Elongated toenails on an alpaca in Peru. Extreme inward curling of the toenails is present.

Figure 9.64 Elongated toenails, both curved outward.

best to hold them near a solid wall in case it is needed for balance. Regardless of how the animal is restrained, proper handling of the legs and feet are important (See Chapter 2). The person trimming should stay on the same side as the

Figure 9.65 Elongated toenails, both curved inward.

Figure 9.66 Saboten hoof-trimming shears.

handler and start with the front foot. The trimmer should face away from the animal and work their way down to the foot, starting at the shoulder. The foot can be picked up, but care should be taken not to abduct the leg. The leg can be braced on your thigh with the foot held in your left hand, to facilitate access to the toes. The goal is to have the toes even with the foot pad. Blood stop powder can be used if the toe is quicked. As mentioned above, there are some toenails that show extreme curling (Figure 9.63); in those cases, trim off the top/dorsal ridge of the toe. When done, put the foot down and move to the back leg. Still facing away, start high at the hip and work your way down to the foot, lift the

Figure 9.67 ARS sheep hoof-trimming shears.

Figure 9.68 Equine hoof nippers.

(a) (b)

Figure 9.69 (a) Obstetrical wire is being used to trim an overgrown toenail on a camel foot. (b) Same camel foot after nail is trimmed.

foot with your left hand and brace on your thigh, again, do not abduct the leg. It is important to keep the animals balance underneath them for them to feel comfortable. When finished, place the foot down and work in the reverse direction on the other side. Have the handler also switch sides. If you start a cut, but it is not placed properly, the cut must be finished as leaving a partial cut/wound will allow bacteria to enter and possible infection. Some animals will not want you to trim their toes so you will have to work with them calmly to achieve your goal. If they cush (common), you can pull out a foot and work on it or can attempt to roll on their sides to get at the feet. Be cautious if the animal is on its side because it is easy for the animal to kick in this position; be careful to not get kicked in the face if this method is chosen. In alpacas, most white or light-colored animals will need to have their toes trimmed three to four times per year depending on their environment. Toenails on camelids with dark pigmented toes grow more slowly. All camelid feet should be examined at shearing and toes trimmed as needed.

The toenail of a camelid may be avulsed from the digit (Figures 9.70a–c). The nailbed must be protected by a light bandage until the nail regrows, usually within two months. Apply copper naphthenate (Kopertox®), to help toughen the remaining tissue and protect from dirt.

Onychia is inflammation of the corium beneath the nail. Paronychia involves the tissue at the margins of the nail. Both conditions may occur in camelids as a result of contusion or laceration of the nail. Neglected toenail trimming may allow infection to migrate dorsally under the nail from

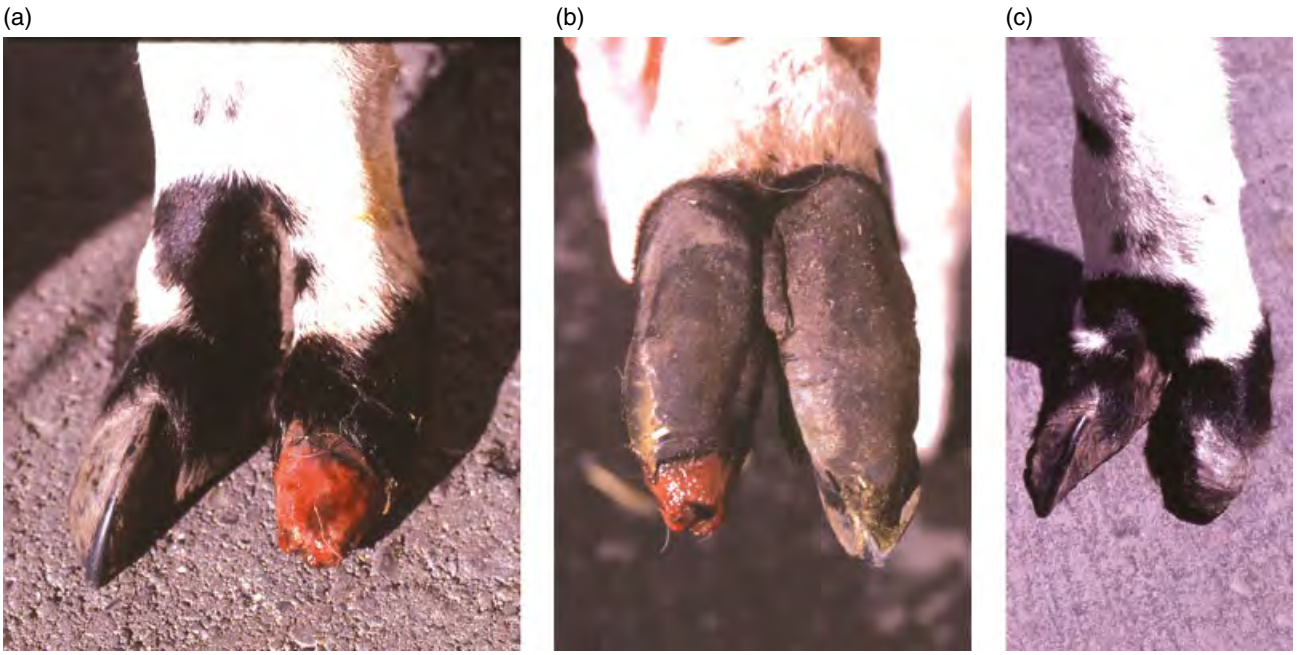


Figure 9.70 Avulsed toenail of a llama. (a) Dorsal view, (b) Palmar view, (c) Toenail following healing.

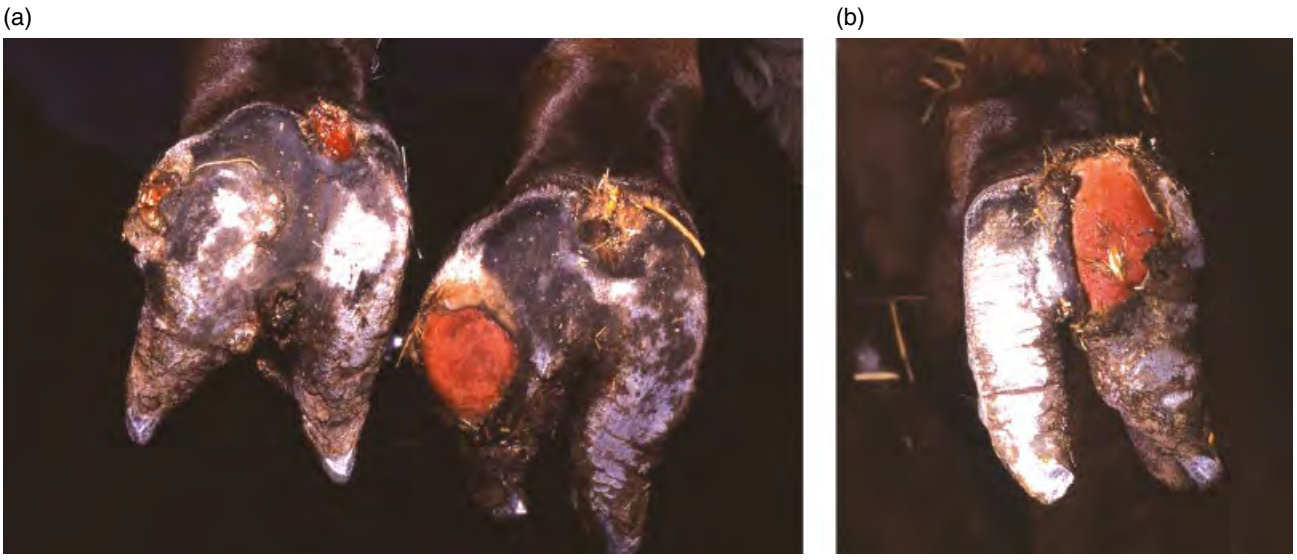


Figure 9.71 (a and b) Infectious pododermatitis.

a broken off nail. With infected toenails, drainage should be established, and treatment continued with local disinfectants and foot baths if possible.

The footpads are subject to laceration, contusion, foreign-body penetration, erosion, and ulceration. Sub-footpad abscessation may result in complete undermining of the pad. To repair this ailment, the detached slipper must be removed, and the sensitive underlying tissue protected by bandaging until recornification takes place (Figure 9.73b). In alpacas, large dog booties can be used for protection with daily cleaning, changing the bandage, etc.

Pododermatitis, Interdigital Dermatitis and Traumatic Injury

Pododermatitis is uncommon but can occur in camelids (Figure 9.71a and b). Causes may be multifactorial and attributed/complicated by trauma, bacterial folliculitis, mange, and yeast infection. In South America, it is felt that *F. necrophorum* is the primary etiologic agent [86]. In North America, a variety of organisms have been isolated: *Staphylococcus* spp., *F. necrophorum, T. pyogenes,* and *Bacteroides* spp. *F. necrophorum* is not highly invasive and requires a break in healthy epithelium or devitalized tissue

to provide a portal for entry. In mixed infections, *F. necrophorum* forms a symbiotic relationship with other organisms such as *T. pyogenes*, with the toxins of each enhancing the effects of the other. *Bacteroides* spp. are particularly difficult to eradicate because they are resistant to most antibiotics. If camelids are kept on a rough, abrasive surface they will more likely have small wounds or penetrations of the footpad; this is especially true in a wet environment. Lesions associated with chorioptic mange in the interdigital and heel bulb area can lead to secondary infections in the interdigital space or heel bulbs.

Interdigital dermatitis does happen and is usually associated with heavy fiber around the feet and bottom of legs and between the toes (Figure 9.72). In the author's experience, interdigital dermatitis is most frequently seen in younger camelids; however, it can be seen in adults with heavy foot fiber. In the alpaca industry, heavy leg and foot fiber is a desirable trait, and those individuals will be predisposed.

Traumatic injury can occur from aggressive interaction among males with untrimmed fighting teeth or poor environment such as large gravel, sharp stones, edges from broken concrete, etc. used as substrate. In some instances, serious injury can occur with minimal to no explanation as to how it occurred. Degloving injuries or pressure trauma can occur if a foot/leg is trapped and the camelid is unable to free itself (Figure 9.73a–e)

Figure 9.72 Interdigital dermatitis with abundant foot fiber.

Clinical Signs

Foot lesions can be in one or all four feet and interdigital dermatitis is more frequent in alpacas with abundant fiber on their feet and between the toes. If they are kept in a wet, dirty environment, unable to find a dry area, they will be predisposed. Continual moistness will lead to maceration making it more susceptible to damage and infection. Lameness may result from necrotic lesions in the interdigital space and on the footpad (pododermatitis) (Figures 9.71a, b and 9.72)

Depending on severity, lameness may be mild. If there is an abundance of fiber between the toes, the infection will be harder to detect unless the camelid is lame. Frequently there is no lameness at all, and the problem is detected when the toes are trimmed. There may be a foul odor, reminiscent of foot rot in cattle. The skin can be inflamed with waxy accumulations and frequently there is thickening of the interdigital tissue with production of crusts and ulcers Figure 9.74a–c).

If the foot pad is affected (pododermatitis) then there may be one or multiple lacerations, punctures or ulcers in the pad. Depending on the environment, there may be more than one camelid affected and the rest of the herd should be examined.

Diagnosis

Culture should be attempted but may be hard to interpret as a variety of aerobic and anaerobic bacterial and fungal organisms can be found from normal interdigital skin [87]. A study was conducted to determine the normal flora of the interdigital skin of healthy alpacas. Of the 60 feet cultured, 25% of the samples had one or more of the following bacteria: *Enterococcus* spp., nonhemolytic *Staphylococcus* spp., *Pantoea* spp., *Bacillus* spp., *E. coli*, *Klebsiella* spp., α-hemolytic *Streptococcus* spp., and *Proteus* spp. Five species of anaerobic bacteria were isolated: *Bacteroides* spp., *Porphyromonas* spp., *Clostridium* spp., *Peptostreptococcus* spp. and *Fusobacterium* spp. (1.7%). Twelve different fungi were isolated (one foot had none): *Mucor* spp., *Geomyces* spp., *Penicillium* spp. were found in 25% of the samples [87].

Treatment

Many of these infections will have to be managed by clinical signs and response to treatment. Treatment consists of cleaning the foot/feet, trimming excess fiber away and assessing how deep the infection goes. Any loose tissue should be trimmed, and the toes trimmed as necessary. If the camelid is willing, soaking the feet in dilute betadine or chlorhexidine solution will help in the early assessment period and clean the feet. Then the feet should be thoroughly dried and treated topically as needed. The animal should be maintained in a clean, dry environment. If the feet need to be bandaged, the bandage must be changed

Figure 9.73 (a) Trauma to foot after degloving injury to leg and foot. (b) Slipper sloughing five days after injury. (c) Bandage of foot with protective tape on bottom. (d) Five weeks into the healing process. Slipper has recornified. (e) Six months after the injury.

daily and reassessed. In severe cases or with all four feet involved, treatment with Penicillin is advised. Though it may be necessary in the early period to keep the feet covered, that should be done for as short of a time as possible as the foot needs to stay dry and in the open for complete healing. If the animal does not have a clean place to be housed, it must be stressed to the producer that healing will not take place in a dirty, wet environment. Once healing begins, Kopertox can be squirted onto the exposed surface to aid in recornification.

Prevention

Monitor crias and adults with abundant feet fiber carefully and trim away excess fiber as needed. Provide a dry, clean environment for them to loaf in during inclement weather. Keep their dry lots free of large stones to avoid punctures of the foot pads. If several cases of pododermatitis occur, examine the environment for possible threats.

Mammary Gland

Mammary Gland Anatomy

There are four teats on the mammary gland of both Old and New World camelids [88–93]. There may also be supernumerary teats, either cranial or caudal to the normal teats, some of which may connect with glandular tissue. The teats in alpacas are about 1.6 cm (0.6 in.) long and in llamas 2 cm (0.8 in.) long [94]. In SACs, each teat has two (or three) streak canals that enter into separate teat and gland cisterns (Figures 9.75 and 9.76). Camels have three streak canals per teat. Variable numbers and sizes of milk ducts

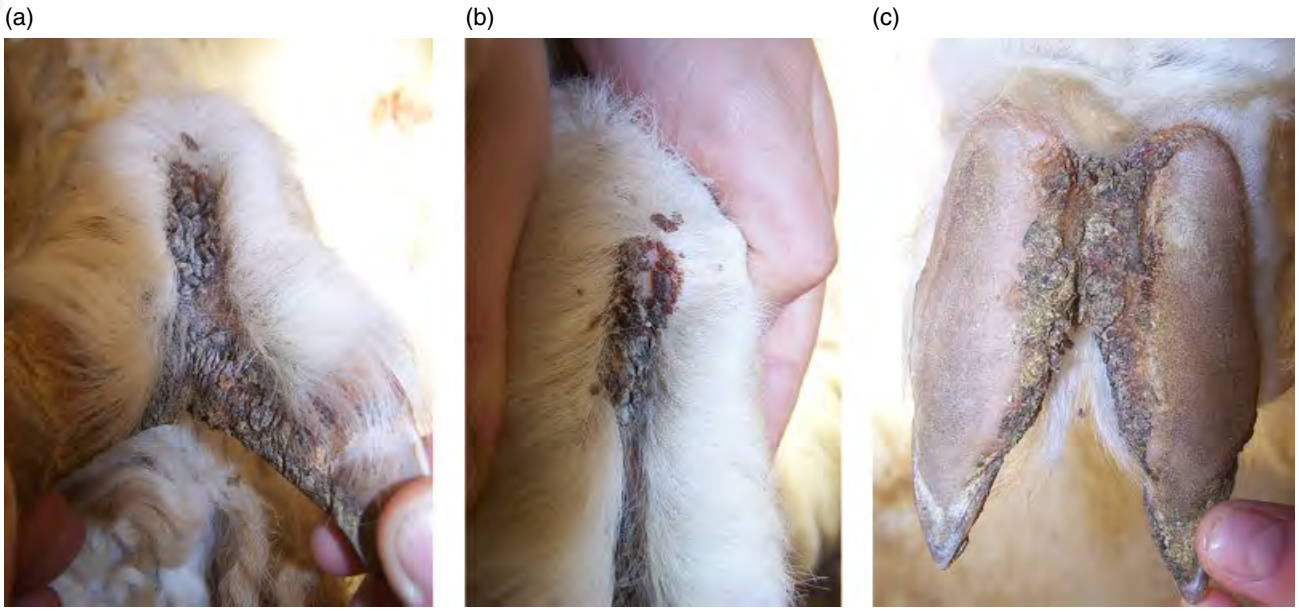


Figure 9.74 (a and b) Waxy accumulations and crusts of the interdigital tissue. (c) Waxy accumulations and crusts bottom of the foot.

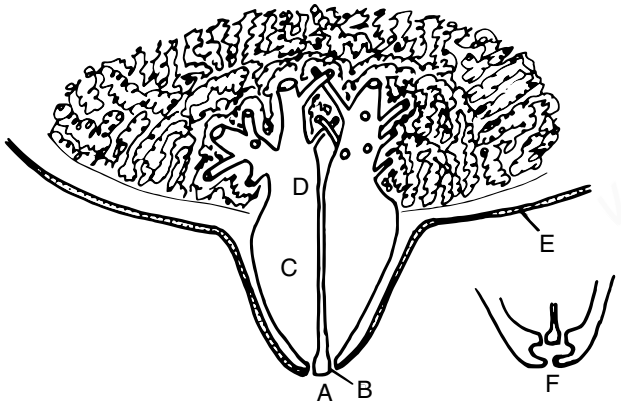


Figure 9.75 Diagram of a camelid teat and collecting system. (A) Streak canals opening on tip of teat, (B) streak canal, (C) teat cistern, (D) mammary gland cistern, (E) skin, (F) alternate morphology – streak canals emptying into a sinus with a single external orifice.

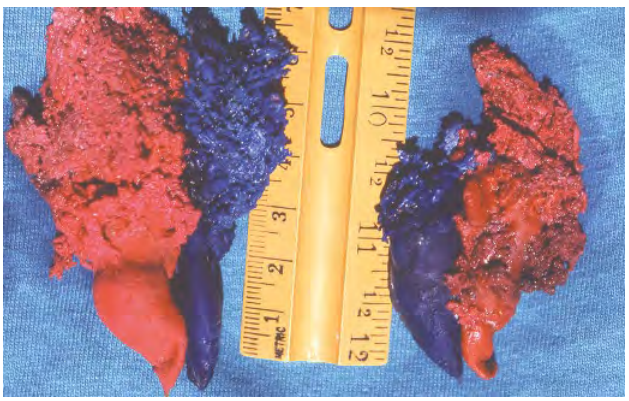


Figure 9.76 Latex cast of mammary gland-collecting system.

collect milk from the gland and empty into the gland cistern.

Though the glands and milk ducts draining into a single teat overlap and interdigitate with one another, they are separate and distinct, with no anastomosis as determined by evaluation of latex cast preparations (Figure 9.76) and contrast radiography. Thus, in effect, there are eight mammary glands. Various teat malformations occur (Figures 9.77 and 9.78). Conjoined (double) teats have been identified (Figure 9.79).

The streak canals of camelid teats are short (2 mm) and have such a narrow diameter that conventional tubes used to instill antibiotics into the teats of a cow or sheep are too large. A 1-mm (3 French, 18-gauge catheter) catheter is about the maximum usable size. The streak canals may exit the teat on the conical surface of the tip of the teat, or both canals may exit into a sinus at the tip of the teat, giving the erroneous impression that there is only one orifice (Figure 9.75). The teat and gland cistern are not separated. The combined cistern is approximately 2.5 cm long and 1 cm in diameter.

The glands are compound tubuloalveolar glands similar in form and development to those of cattle (Figure 9.80). In SACs, the right and left halves of the mammary gland are separated by one medial ligament that originates in the abdominal tunica (linea alba) [91]. The two lateral ligaments form an incomplete suspensory ligament from the caudal abdominal wall [91, 94]. The front and rear quarters

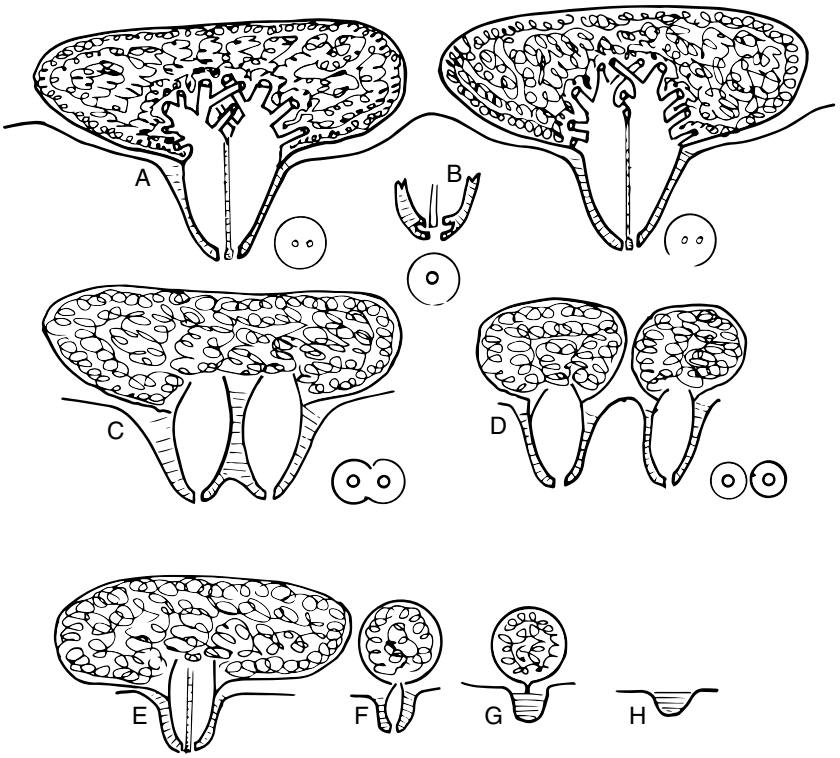


Figure 9.77 Diagrams of the side view of teats: (A) Normal, (B) alternate orifice, (C) partial separation of teats at a normal location, (D) double teats at a normal location, (E) small teat, may be one of the four normal teats or a supernumerary teat, (F) supernumerary teat with a functional gland, (G) supernumerary blind teat associated with glandular tissue, (H) supernumerary teat with no attachment to glandular tissue.

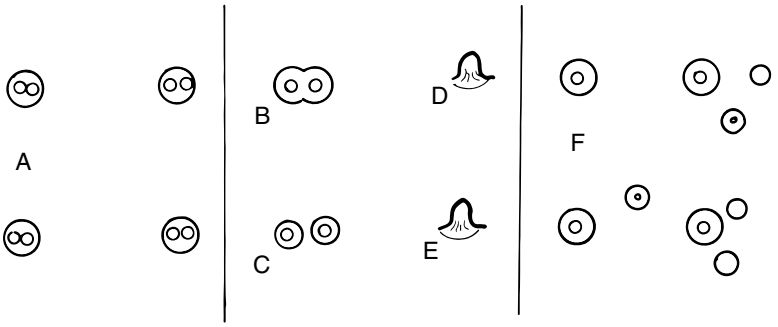


Figure 9.78 Diagram of the ventral view of teats of a camelid: (A) normal configuration, two openings at the tip of each teat, (B) fused double teat, (C) separate double teat, (D) lateral deviation from the vertical axis, (E) medial deviation from the vertical axis, and (F) possible locations of supernumerary teats.

cannot be visually or surgically separated, but there is no connection between the collecting systems of the two quarters. The udder of the camel is similar, except that there are two or three teat ducts per teat with independent teat cisterns leading to the gland cistern [95].

The recognition that there are eight separate glands is important clinically in the management of mastitis. If infusion of antibiotics into the gland is prescribed as a treatment for mastitis, it is crucial that the appropriate gland be infused.

Hand milking a camelid is a challenging task because of the short teat. At best, a thumb and one or two fingers are all that can be used. Even then, some have such short teats that they are difficult to grasp. Because the teat and gland cistern are not separate, it helps to express milk by starting up in the gland to build up volume to express from the teat. Hand milking quickly chaps the teats, so it is best to use a "wet technique" by utilizing milk on the fingers to aid in milking out the teats. Because the teats are so short, some weak crias may also fail to grasp such short teats. This is

Figure 9.79 Double teat in a llama.

particularly true if there is edema of the udder at the time of parturition. Though a technique using a 10- or 20-mL plastic syringe as a "breast" pump has been described [96] it is not tolerated by most camelids and should not be utilized.

OWCs are milked routinely to provide milk for people; they are an especially common milk-producing animal in drought-stricken areas of the world. Camel milk has been utilized by Bedouin, nomad and pastoral cultures as a source of nutrition since the domestication of camels. Camel milk is an important nutritional essential across Africa and the Middle East with the dairy industry providing fluid milk, cheese, ice cream, and other dairy products.

Milk Production

An interesting sidelight for North American breeders is that a high plane of nutrition (particularly excess energy) just prior to puberty results in lower mammary secretory tissue weight and is associated with low milk production in subsequent lactations. This is similar to what is seen in dairy heifers when they become over conditioned before their first lactation.

The total weight of the SAC udder in a nonpregnant mature female is approximately 250 g in llamas and 162 g in alpacas [94]. Udder enlargement may begin approximately two months before parturition, especially in a multiparous female. In some maiden females, the udder may not be very noticeable until soon before they give birth. The udder of a fully lactating female is relatively small, weighing only 450 g, and may appear empty all the time if nursed properly. As in all species of mammals, a genetic component determines mammary gland size and milk production. Llamas and alpacas have not been bred with milk production as a primary trait; and there is a wide variety between females with regards to milk yields [97]. A study utilizing deuterium oxide to measure water kinetics in

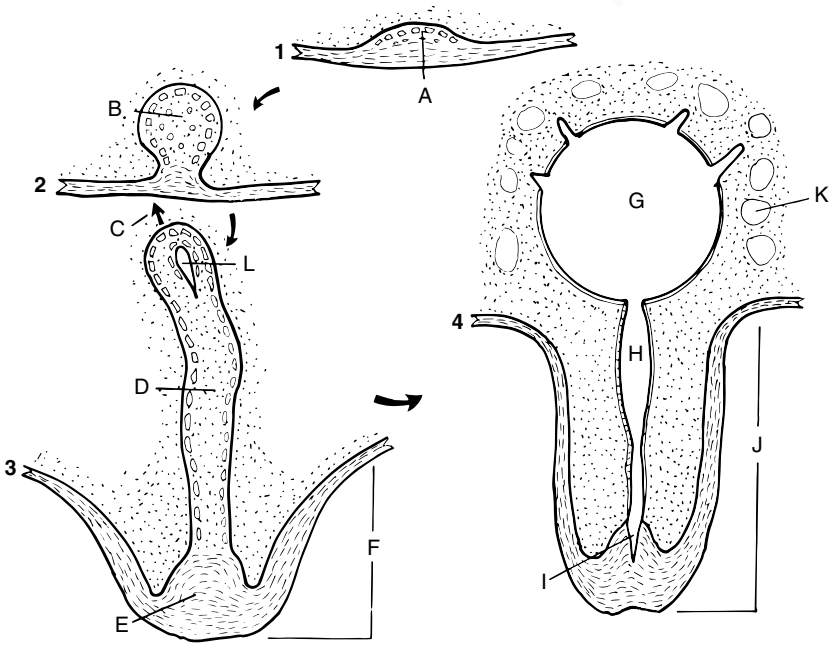


Figure 9.80 Diagrams of early embryologic development of the bovine mammary gland and teat. (1) Embryo size 2.5 cm, (2) embryo size 3.8 cm, (3) embryo size 5 cm, and (4) embryo size 27.5 cm. (A) Mammary hillock, (B) mammary bud, (C) direction of growth, (D) duct tissue, (E) skin cone, (F) developing teat, (G) gland sinus (milk cistern), (H) teat sinus (teat cistern), (I) teat canal (lactiferous duct, streak canal), (J) developing teat, (K) developing fat tissue, and (L) beginning of formation of the duct system. *Source:* Diagram modified from Hibbitt et al. [88].

suckling llama crias was performed to estimate daily milk intake in 11 llama crias. Peak lactation was approximately three weeks post-partum and lasted for about one to two weeks. At peak lactation, the llama crias on average were nursing 2.5 L/day. This was observed to be similar to meat sheep and goat offspring. At peak lactation, the dams were drinking 9.8 L/day, which decreased to 5.5 L in late lactation [97].

There is a vast difference in production by the camelid female. Due to their unique anatomy and behavior, it is hard to milk out any female fully. The study by Riek showed a mean volume consumed by the llama cria daily during peak lactation of 2.5 L with a range of 1.6–3.97 L [98]. This volume seems unlikely to anyone who has milked out a camelid. However, crias are much more efficient at draining the udder than humans.

Milk Composition

Reports of camelid milk composition in the past have been variable. Recent studies, using large numbers of llamas from different regions of the United States, give more credibility to milk analysis [99]. More detailed information can be found on other species [100–107].

Alpaca and llama milk are similar and may be considered together [101, 108]. Morin describes alpaca milk as having a white porcelain color and an odor like that of cow's milk. Alpaca milk has a sweetish taste and is more viscous than cow's milk. Riek reported that by week five, the fat and protein concentration had a steady increase until the end of lactation, with the fat content varying from a minimum of 3.09% up to 6.94% during a lactation period. The lowest fat content was in colostrum with a mean of 0.75% [97]. Morin did not find such variation in llama milk in her study [99]. Her study did not follow lactating dams throughout a lactation, but rather a sample was taken on a single occasion from each dam at different stages of lactation. This may also have accounted for the differences in the two studies as well as differences in locations and nutrition for each female. Llama milk has a pH of 6.4–7.2 [97].

Table 9.2 lists the composition values for the milk of camelids and other species. Numerous factors affect the composition of milk, including genetic predisposition, nutrition, stage of lactation, age of the female, season, ambient temperature, and presence of chronic subclinical mastitis.

Camel milk has been described by Farah in 1993 [103]. Camel milk is used extensively for human food. Camels are uniquely adapted to subsistence on harsh, dry, desert herbage and intermittent supplies of drinking water while continuing to produce milk that is a suitable source for both moisture and nourishment for camel calves and human infants [27]. The camel, when provided with optimal water, produces milk with 4.5% fat and 84% water and is able to continue to produce milk that is highly dilute with 90% water content and 2% fat content unless chronically dehydrated [27, 110]. This is unique, as cattle, sheep and goats either cease lactation or produce milk for a only limited time when subjected to drought conditions, and the milk they produce will be very high in fat and low in water [109, 111]; which is not healthy for nursing offspring. A camel that has not drank water in 10 days can still produce 20 L of milk in a day [27, 112].

The normal composition of camel milk is similar to that of SAC milk (Table 9.2). Milk yields of camels vary from 3.5 to 14 kg per day, with lactation yields (9–18 months) totaling 1000–4000 kg. Camel milk has a sweet, sharp taste and is sometimes salty. Milk taste and odor are modified by plant ingestion and other environmental conditions. Camel milk is rich in vitamin C, three times higher than cow's milk and 1.5 times higher than human milk. It is an important source of vitamin C for humans in a vitamin impoverished environment. The fat droplets in camel milk are small and do not rise to the top of standing milk [109, 111].

The secretion of dilute milk by a hot desert-adapted camel is explained, based on the effect of the pituitary antidiuretic hormone on secretion of water by the mammary gland (the mammary gland is a modified sweat gland). Camels have minimal sweat glands otherwise, which is

Table 9.2 Milk composition (as fed).

Species	Water (%)	Total solids (%)	Protein (%)	Fat (%)	Lactose (%)	Kcal/L
Llama	84.6–86.9	13.1	3.4–4.2	3.1–6.9	5.9–6.5	822
Camel	84–90	13.6	3.0–3.6	2.0–5.7	4.3–4.4	665
Goat	87.0	13.2	2.9–3.3	3.1–4.5	4.1–4.2	1680
Sheep	81.0	19.3	4.1–6.2	7.4	4.8–5.0	1138
Cow	87.3	12.7	3.4	3.7	4.8	653

Source: Morin et al. [99], Yagil [109], Riek [97].

one of the many physiologic adaptations that aid them in dealing with high temperatures and dehydration [109, 111].

Teats

Supernumerary teats (SNT, polythelia, accessory teats, rudimentary teats) are those in excess of the normal number of four teats. They occur in all species of livestock, companion animals, and humans. The incidence of SNT vary in llamas (17%) and alpacas (6%) [94]. Some of these SNT may be connected to a variable amount of mammary tissue or no mammary tissue. The concern is that the cria will fixate on these abnormal teats and will not receive the valuable colostrum needed in the first few hours of life. One Alpaca organization, The Suri Network, developed a breed standard for Suri Alpacas in 2016 that included, as an ideal trait, to have only four teats, uniformly positioned. This has recently been adopted by the AOA for all alpacas.

Though quite rare, a lesser number of teats may be present (subnumerary teats, hypothelia). The absence of teats (athelia) was determined to be 0.9% of 223 alpacas in one study [94]. Supernumerary teats have been determined to be an inherited, simple recessive trait in cattle and are assumed to be recessive in other species [92]. Other types of teat anomalies are seen (Figures 9.77 and 9.78):

1) Short teats
   a) Functional, associated with glandular tissue
   b) Nonfunctional, associated with glandular tissue, but no teat canal
2) Introverted
3) Excessively long or bulbous
4) Small dermal enlargement (rudimentary)
5) Divergence from the vertical axis
6) Partial separation of paired teats at a normal location (Figure 9.78)
7) Complete separation of paired teats at a normal location

Mastitis

Mastitis is not common in llamas and alpacas [97, 113, 114]. Even though mastitis is not common in SACs or camels, when it does occur, prompt attention is necessary to avoid loss of function in one or more quarters or even death. In cases of clinical mastitis, manual palpation of the udder may reveal the affected quarter to be warmer when compared to the non-affected quarters. There may also be enlargement of this quarter and abnormal secretions. Due to the unique camelid anatomy of the udder, one or more glands may be involved. Camelid mastitis occurs in the same forms as seen in dairy cattle, namely, subclinical, peracute, acute and chronic.

Predisposing Factors

In camelids, the udder is not pendulous, and the teats are relatively short, so trauma is minimized. Nonetheless, trauma (laceration, contusion, abrasion) of the glands and teats are possible in specific circumstances (dog attack). Trauma to the tip of the teat may weaken the sphincter of the streak canal, which in the normal state tends to prevent access to the gland of pathogenic bacteria. Even though the location of the udder minimizes exposure to filth, when the female is recumbent, she may be forced to lie down in mud if there is no access to clean, dry areas. In a hot climate, they seek out water to lay in an effort to cool. Milk is an excellent medium for bacteria, and any condition that allows milk to accumulate within the gland fosters growth of any organism that may have gained temporary entrance. Failure of the cria/calf to nurse, blockage of a streak canal with a waxy plug, pre- and post-parturient udder edema, and stricture of the streak canal are factors that may cause milk to accumulate in the gland.

Etiology

There are reports on specific bacteria that cause mastitis in camelids [93]. A study evaluating 100 lactating llamas revealed that 21% (76 of 369) of samples had indications of infection with 57% having positive bacterial culture in at least one quarter. None of the animals exhibited clinical mastitis and minimal evidence of inflammation was present [114]. In that study, the most common minor pathogen isolated was *Staphylococcus* spp. with major pathogens as *Streptococcus uberis*, *Streptococcus equinus* and *Enterococcus* spp. Isolates from peracute camelid mastitis have included *E. coli, Klebsiella pneumoniae*, and *Enterobacterium* spp., which are the same organisms causing peracute mastitis in cows. In camels *S. aureus, Pasteurella haemolytica* and *Streptococci* were found in mastitic milk samples and are considered to be the primary cause of mastitis, however, these organisms have also been isolated from the milk of healthy camels. Minor pathogens isolated were *Micrococcus*, *Trueperella*, *P. aeruginosa*, and *Bacteroides*. In the many studies done on camel milk, the consensus is subclinical mastitis is more frequent than realized [27].

Clinical Signs

Subclinical mastitis may be detected only by culturing for an organism in the milk or testing the milk with one of the mastitis tests such as the California mastitis test (CMT). In the case of subclinical mastitis, indurated areas may be palpable within a gland, but there are no systemic signs, and the gland is not swollen or hot. Chronic mastitis results in periodic changes in the quality of the milk.

Acute mastitis is usually seen just before or within a few days after parturition and is characterized by heat, swelling, hardness of the affected gland, and noticeable pain on palpation. The secretion may be watery, hemorrhagic, thickened, stringy, or malodorous. The female may refuse to allow the cria to nurse because of the pain, or the cria may refuse to nurse because of the unpalatable secretion. The cria may also develop gastroenteritis or septicemia from ingestion of the pathogen. The female may be anorexic and have a low-grade fever.

Peracute mastitis may have all the signs of acute mastitis (Figure 9.81) plus severe depression and endotoxemia. Peracute mastitis may even precede gangrenous mastitis (Figures 9.82 and 9.83), septicemia, and death. Peracute mastitis is usually seen within a few days of parturition.

Diagnosis

The diagnosis is based on a thorough physical exam, evaluation of the secretion for consistency, color, viscosity, presence of debris, and sediment, culture and sensitivity, and evaluation with the CMT. In camels, it has been shown that there is an increase in somatic cell count (SCC) when a bacterial mastitis is present and that using the CMT is appropriate when testing for subclinical mastitis in camels. However, the CMT and the SCC are ineffective at diagnosis of mastitis in llamas. Llamas lack of significant glandular inflammation and high cell counts when compared to the bovine [97, 114]. The volume of milk produced or obtained from a camelid may also contribute to the lack of a positive CMT. When mastitis is suspected, all quarters should be examined, not just the obviously affected gland. Ultrasonography may be used to locate walled-off abscesses.

Therapy

The objectives of treatment are to save the life of the female, restore the function of the gland, and improve milk quality. Treatment consists of removing the abnormal secretions, use of non-steroidal anti-inflammatory drugs (NSAIDs) to reduce inflammation and endotoxemia and broad-spectrum antibiotics such as Ceftiofur. As a general practice, it is not recommended to instill intramammary preparations due to the complicated teat anatomy discussed earlier in this section. If local infusion is selected, both streak canals must be cannulated simultaneously to ensure treatment of the affected portion. The streak canals are tiny, and 1-mm (3 French, 18-gauge catheters) catheters or smaller in alpacas are required. The streak canal is

Figure 9.81 Acute mastitis and dermatitis caused by *E. coli* infection.

Figure 9.82 Acute mastitis with abscessation.

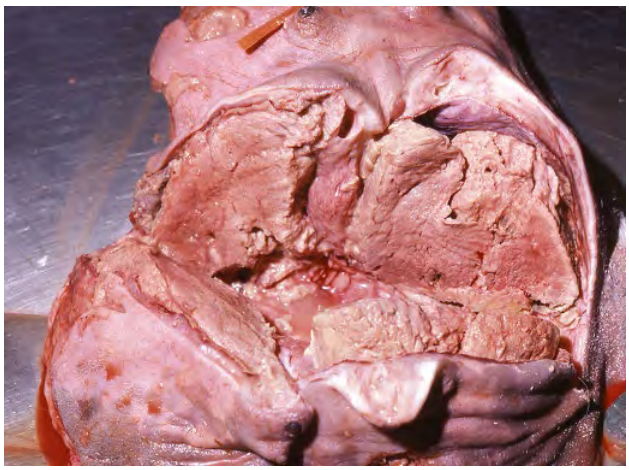


Figure 9.83 Necrotic mastitis caused by *E. coli* infection.

easily traumatized, causing lifelong complications. Do not attempt to infuse with a bovine teat cannula or commercial infusion tubes, nor have owners do infusions.

Peracute and possible acute mastitis cases require parenteral administration of antibiotics along with careful local infusion. In both instances, the selection of an antibiotic should be based on culture and sensitivity. The endotoxemia should be treated aggressively with steroids or NSAIDs and IV fluids. The exudate should be removed from the gland three to five times daily. Polymyxin B can be used to bind endotoxins. The use of Polymyxin B (250 000 units diluted in 40 mL sterile saline) infused into affected glands twice daily has been described in successfully clearing a case of peracute mastitis caused by *E. coli* and *Klebsiella* spp.

In acute and peracute cases of mastitis, it is essential that the cria be temporarily fed by bottle as a protection to itself and to minimize painful manipulation of the affected gland and teat by the normal act of nursing.

Prevention

Careful attention to the nursing behavior of the cria and observation and palpation of the udder allow early detection of problems. A weak or premature cria will likely not remove enough milk to empty the udder. The female should be milked regularly until the cria is strong enough to nurse unassisted. If possible, this milk should be fed to the cria, especially if it is colostrum. The cria generally removes milk from all four teats at each nursing episode. A single swollen teat should be investigated.

Udder edema

Pre- and post-parturient edema occurs in camelids, especially maidens, and must be differentiated from mastitis. Edema is symmetric and uniformly distributed over the gland. The swelling is not hot or very painful, and the secretion quality is not altered. Edema may cause swelling of the teats, which may mechanically interfere with the cria's ability to grasp the teat in its mouth. On occasion, there may be edema proximal to the udder. Edema will usually go away as the dam comes into milk and the blood flow to the udder becomes fully established. Massage and manual milking may be necessary to reduce the swelling. Colostrum should be saved and offered to the cria via a bottle or by orogastric feeding. These teats are especially hard to milk, and continued engorgement of the gland may predispose toward mastitis. Teats like this chap easily, so keep that in mind when milking and keep some milk on your fingers to moisten the teats.

The Ear

Normal Anatomy

The external pinna is elongated and highly mobile (Figures 9.84a–d and 9.85a, b). The ears are expressive of health and emotional state (See Chapter 2). Llama ears are longer than alpaca ears and the pinna in llamas is described as "banana" shaped, (Figure 9.84a); though at times they can be rounded (Figure 9.84b) or straighter, with an

Figure 9.84 (a) Pinnae of a llama, banana ears. (b) Pinnae of a llama, rounded ears. (c) Pinnae of a llama in Peru, straight ears. (d) Pinnae of a llama, straight ears.

Figure 9.85 (a) Pinnae of alpaca ears – cria. *Source:* Photo courtesy of Becky Camma, The Alpacas of Spring Acres, OH. (b) Pinnae of alpaca ears – adult male.

outward lateral curve (Figure 9.84c and d). The alpaca pinna is straight and "spear" shaped (Figure 9.85a and b) [115–117].

The external acoustic meatus is sigmoid shaped with a narrow (5 mm) vertical lumen and narrow (6 mm) horizontal lumen. The canal narrows as it courses deeper vertically [118]. The ear canal is located on the lateral aspect of the annular cartilage of the pinna. When inserting an otoscope cone, it is easy to mistake the conchal eminence for the ear canal (Figure 9.86).

An extensive study has been done by Computed tomography (CT) to characterize the structures of the external acoustic meatus, tympanic cavity and tympanic bulla of llamas [118]. The osseous external acoustic meatus curves ventrally as it tracks medially in a sigmoid shape. There is a bend of approximately 147° halfway to the tympanic annulus with its narrowest portion located at the level of the tympanic annulus. The tympanic bulla is divided by the stylohyoid fossa into a larger caudolateral and a smaller caudomedial process. Unlike dogs and cats with a hollow tympanic bulla, the tympanic bulla in the llama has a multicompartmental honeycomb-like structure with pneumatized cells (Figure 9.87). The tympanic cavity is contained within the petrous and tympanic portions of the temporal bone [118]. The irregularly shaped tympanic membrane is located on the medial side of the distal canal and is approximately 0.8 cm in diameter [23].

The canal is essentially straight in a neonate. Apparently, the external auditory canal is partially composed of cartilage in the neonate and ossifies at an unknown later time [23].

It is not possible to view the tympanic membrane in most adult llamas because of the bend and narrowness of the external ear canal. Edema and inflammation of the canal also obstructs viewing. Most otoscope cones are too wide and too short to be useful in detailed examination so a small endoscope should be utilized. The inner ear lies between the middle ear and the cranial cavity [23].

Diagnostic Procedures

An unsedated camelid is reluctant to allow even a casual examination of the ear. Deep examination of the canal is virtually impossible without sedation. If exudate oozes from the ear, it is important to exclude the presence of foreign bodies (grass awns) or ticks. Radiographic examination can demonstrate sclerosis of the tympanic bulla of the petrous temporal bones (Figure 9.87) in chronic cases of otitis interna. Clinical evaluation of cranial nerve deficits in camelids is the same as for other mammals [119].

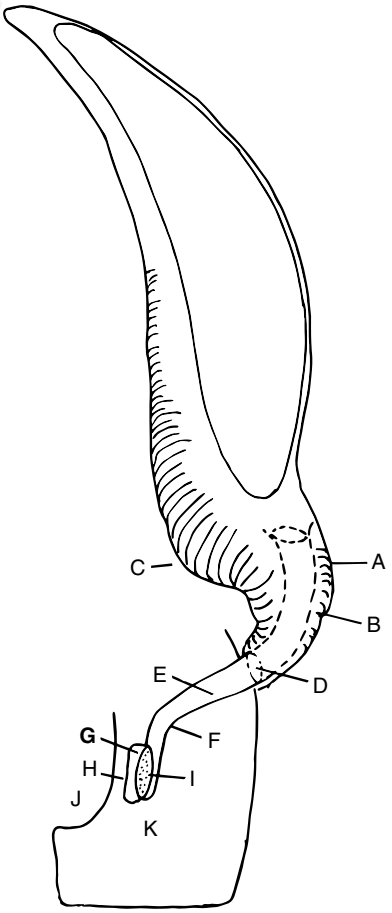


Figure 9.86 Diagram of the relationship of the pinna, external ear canal, tympanic membrane, and middle ear. (A) Lateral side of the left pinna, (B) annular cartilage of the pinna, (C) conchal eminence, (D) external orifice of (E) osseous ear canal, (F) bend of canal, (G) middle ear, (H) inner ear, (I) tympanic membrane, (J) cranial cavity, (K) tympanic bulla.

Figure 9.87 Dorsoventral radiograph of the skull with sclerosis of the tympanic bulla. (A) Affected bulla.

Recently, a study was completed to determine the cytology and resident flora of clinically normal alpaca ears [120]. The study consisted of 50 privately owned alpacas with one ear canal (vertical) sampled in two different seasons, August and January. Results were as follows: Bacteria isolated in August – *Bacillus* (44%) *Arthrobacter* (40%) and nonhemolytic *Staphylococcus* (26%); in January – *Bacillus* (42%) and *Pantoea* (38%). Yeast was found in 2–4% of samples, with 6% found in August and 30% found in January. Rod-shaped bacteria were seen in 50% of alpacas in August and 26% of alpacas in January. Coccal bacteria were seen in 32% of alpacas in August and 16% of alpacas in January. No ectoparasites were seen in any of the alpacas (expected finding in normal ears). Their conclusions were that inflammatory cells and ectoparasites are not normal findings in the external ear canals of healthy alpaca ears. However, rare non-nucleated epithelial cells, and yeast organisms may be observed with some Gram-negative rods and Gram-positive cocci as typical bacteria [120].

Diseases

Lacerations

Lacerations of the pinna are common in adult males because of inter-male aggression. These lacerations can be caused by fighting teeth if left untrimmed, or even by incisors when the opponent pulls away from the aggressor. It is important to trim fighting teeth to decrease injury among the males of the herd (See Chapter 12). If the event causing the laceration is observed and immediate attention can be given, such wounds may be cleansed, debrided, and sutured with reasonable success. Lacerations of the poll may transect the ear muscles, preventing motor control of the pinna. Every effort should be made to realign severed ends of the muscles of the pinna. Due to contamination with oral bacteria and dirt, it may be necessary to administer systemic antibiotics (Procaine Penicillin G @ 44 000 IU/kg, SC, daily, for five to seven days). Also, if it has been more than three months since the animal received a tetanus vaccination, a booster should be administered.

Other Diseases of the Pinna

Dermatophytes may infect the pinna. Sarcoptic, Psoroptic, and Chorioptic mange and demodectic mites have been detected on camelid pinnae (without causing a dermatitis). Hematoma of the pinnae is not common but may be seen in association with head shaking in ear tick infestation. A blow to the ear may cause a similar lesion. On rare occasion, crias can be born with hematomas of the pinna. It is usually associated with a specific

male known to carry the trait and is infrequently seen due to selective breeding. Treatment is similar as for small animals.

Otitis Externa

Inflammatory lesions of the external ear canal are common, but the cause of the inflammation is not always readily apparent. The spinose ear tick, *Otobius megnini*, may be a regional problem (Chapter 7) and can be associated with otitis externa. Grass awns may lodge in the canal and scarify the epithelium, providing a portal of entry for opportunistic pathogens.

The signs of otitis externa are head shaking, scratching the ear with a hind foot or against a post or barn, head tilt, peculiar positioning of the pinna, and exudation from the external ear canal.

Diagnosis is based on clinical signs and a thorough physical examination of the outer ear canal following cleansing of the ear and removal of all debris and ear wax. The epithelial surface may be hyperemic and ulcerated. Frequently, there is a foul odor. Cultured samples taken from the external ear canal may not be helpful, since the normal flora may harbor many opportunistic pathogens.

The external ear canal should be cleansed and irrigated once to twice daily depending on severity. Common small animal cleaning products can be used such as TrizEDTA Aqueous Flush,® Epi-Otic® or dilute Chlorhexidine. Instillation of a broad-spectrum antibiotic, anti-fungal otic ointment used in small animals or intramammary infusion preparation for lactating cows should follow cleansing.

Otitis Media and Interna

Infection of deeper structures is not uncommon and may be caused by a direct extension of infection in the external ear canal [121–123] or from ascending infection from the Eustachian tube with an upper respiratory infection. In many cases however, there is no previous history of either condition being present. Grass awns have been known to penetrate the tympanic membrane and initiate infection [116]. Many bacteria have been isolated from cases of otitis media/interna: *Proteus* spp., *Pasteurella* spp. [116], *Actinomyces* spp., group D *Streptococcus* spp. [123], Nocardia spp. [123], and *Propionibacterium* spp. [124] *Listeria monocytogenes* was isolated from an otitis interna infection that subsequently spread to the brain [123].

Otitis media/interna is usually associated with head tilt, ear droop, flaccid lower lip, collapsed nasal opening with deviation to opposite side of ear droop (Figures 9.88–9.91). Facial nerve paralysis may extend to decreased facial sensation, loss of a menace response and palpebral reflex leading to corneal ulceration. Horner's syndrome may be present [116]. The cardinal signs of Horner's syndrome are

Figure 9.88 Facial paralysis.

Figure 9.89 Facial paralysis due to inner ear infection.

slight ptosis of the upper eyelid, inability of the pupil on the affected side to dilate, and the nictitating membrane pushing out over the bulb as a result of retraction of the bulb deeper into the orbit, which makes the eye appear smaller. Horner's syndrome with facial paralysis, is virtually pathognomonic of a lesion in the middle ear because the sympathetic fibers involved in Horner's syndrome lie in juxtaposition to the facial nerve only near the middle ear. Additional signs of otitis media or interna may include disorientation, ataxia, and circling which can be misinterpreted as Listeriosis (Chapter 19).

With otitis media/interna more advanced diagnostics such as radiographs and CT scans may be needed to determine the

Figure 9.90 Head tilt due to middle ear infection.

Figure 9.91 Chronic facial paralysis.

extent of the infection and damage. Initial treatment should take into consideration the most common bacteria probably involved. Several antibiotics have been used successfully by the author: (i) Procaine Penicillin G 44 000 IU/kg, SC, daily for 20–30 days, (ii) Enrofloxacin 5 mg/kg, SC, daily for 10–15 days (warn of possible toxicity), (iii) Ceftiofur Hydrochloride 6 mg/kg, SC, daily for 20–30 days, (iv) Ampicillin 10 mg/kg, IV, four times a day for 7 days, followed by Procaine Penicillin G for an additional 7–10 days. As an adjunct treatment to each of the antibiotics listed above, Isoniazid 20 mg/kg, orally, daily for 30 days can be used. Isoniazid is widely distributed in the body and useful in combination with antibiotics to help carry them to hard-to-reach places, especially in the face of caseous material.

In cases that will respond to medical therapy, the clinical signs should improve within 10–14 days. It may be necessary to change the antibiotic being used if, after five to seven days, there are no signs of improvement. If during treatment, the clinical signs worsen or the camelid becomes anorexic, then further diagnostics should be pursued.

Detailed CT scans have been done on osseous structures of the llama ear canal [118]. Lateral bulla osteotomy and ear canal ablation has been done successfully on camelids [122, 125]. Owners should understand that the nerve damage may not resolve and may become worse, either temporarily or permanently, after surgery. Many of the long-term deficiencies can be handled easily on a well-managed farm.

Miscellaneous Conditions

The shape of the ears is a hereditary trait. Figures 9.92 and 9.93 illustrate some ear pinna shapes. Congenital diseases of the ear include agenesis of the pinna also known as "gopher ear" (Figure 9.94a and b) and shortened pinnae, curled tips (Figures 9.95 and 9.96). Some animals are born without ear canals, or a cauliflower-like appearance of tissue at the place of the ear canal. Others can be born with a very narrow ear canal opening. Frequently, these are never noticed unless a thorough exam is done by an experienced camelid veterinarian. These conditions are predisposed to ear infections and can be challenging to cure without corrective surgery. Bent ears (floppy pinna, airplane ears, helicopter ears) may be congenital or acquired (Chapter 20). Some crias may have the pinna bent and even completely folded. This can be seen in crias born prematurely or from *in utero* positioning. Straightening requires a bandage (use paper tape), wrapping the ear around a gauze roll or tampon for simple folds (Figure 9.96b). In more severe cases, one may have to use a 6- or 10-mL syringe case or similar stent for support for several days. If only the tip of the ear is bent, a simple bandage (use paper tape) should correct. This condition corrects itself with support as the cartilage strengthens/hardens with time. The most common noninfectious, nonparasitic disease is frostbite (Figure 9.97).

Wound Healing in Camelids

The body's ability to repair damaged tissue is a fundamental process of vertebrates, and it is a very complex process rivaling any other functional process in the body in its complexity. There is much to be learned regarding the biologic processes that orchestrate proper wound healing but

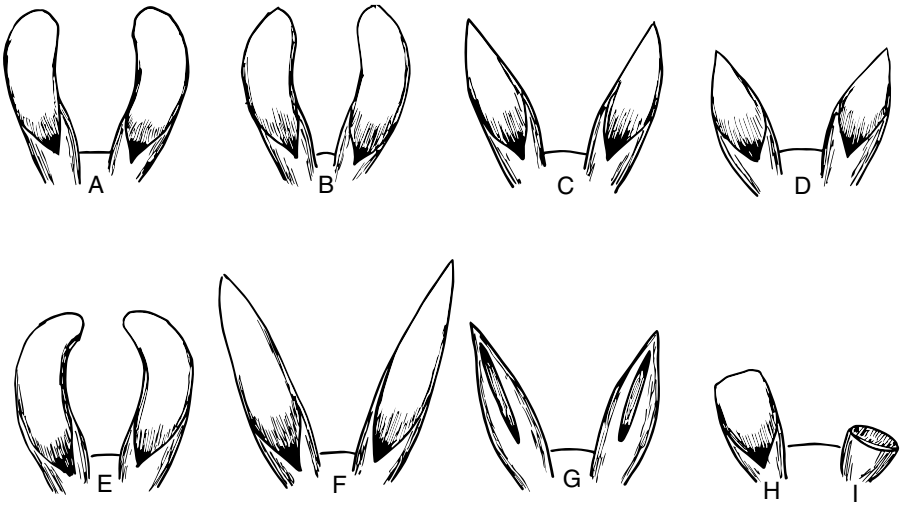


Figure 9.92 Diagrams of llama ear shapes. (A) Normal banana ear, (B) sharp-pointed banana ears, (C) straight medial border, (D) short ears, (E) exaggerated banana ears, (F) elongated, spear-shaped ears, (G) curled ears, (H) congenital shortened ear, (I) congenital gopher ear.

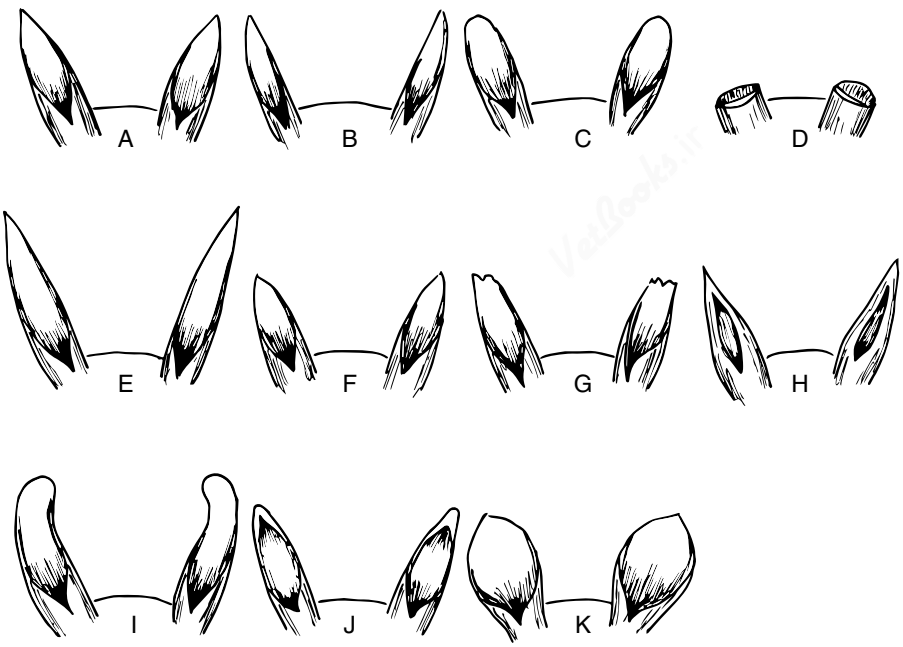


Figure 9.93 Diagrams of alpaca ear shapes. (A) Normal spear-shaped ears, (B) straight medial border, (C) round-tipped ears, (D) congenital gopher ears, (E) elongated ears, (F) short spear-shaped ears, (G) congenital-tip anomaly (multiple configurations), (H) curled ears, (I) banana-shaped (llama ears), (J) tented tip, (K) cauliflower ears.

undoubtedly involves a complex array of cellular and biomechanical mediators.

There is a dearth of veterinary research regarding wound healing in camelids, yet a plethora of literature involving research on small laboratory animals and humans exist. There seems to be common cellular and biochemical mechanisms of wound healing conserved among the mammalian species and we commonly use our knowledge of basic processes and clinical management in other species to direct our therapy in camelid patients. However, notable differences in wound healing have been observed even in closely related subspecies such as horses and ponies [126]. It is not inconceivable to think that differences in wound healing would be found among different species of camelids and caution must be used to extrapolate too aggressively from other species to camelids or even among the

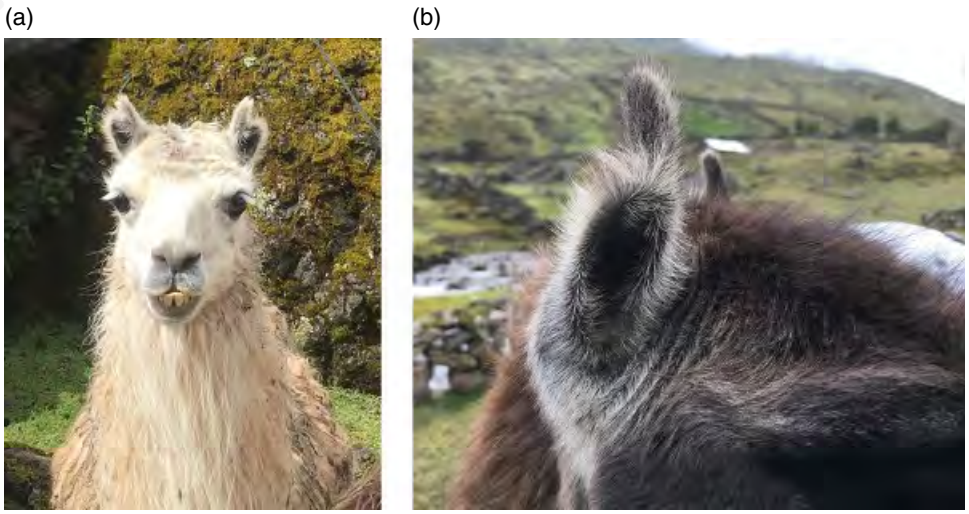


Figure 9.94 (a) Gopher ear of a llama in Peru. (b) Gopher ear of a llama in Peru.

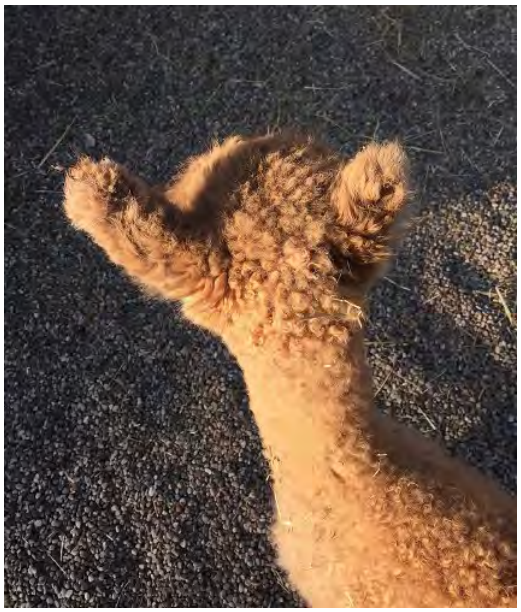


Figure 9.95 Pinna defect, one short and one normal ear.

various camelid species. Selection pressures on camelids living in different environments and selective breeding of domesticated camelids may provide for evolutionary differences among even the most basic of biologic functions.

The goals of wound repair are to re-establish an epithelial covering to protect deeper tissues and restore strength and function to the wounded part. However, wound healing generally results in the formation of tissue that is biologically inferior to unwounded, healthy tissue. There are multiple stages of wound healing, and the overall process is similar in camelids to other species. Each stage involves numerous biologic processes, and these processes are typically associated with a given stage. Though the process of wound healing is generally divided into broad phases, it is important to realize that many stages happen concurrently and the transition from one stage to the next is seldom distinct. Wound healing begins with hemostasis and formation of a blood clot and is followed by an inflammatory response, which serves to clean the wound and prepare it for the cellular proliferation and maturation.

Phases of Wound Healing

Hemostasis

The primary and immediate objective of this phase is to limit blood loss. The damaged endothelial cell membrane releases phospholipids which are converted to arachidonic acid which mediates vascular tone and permeability. Vasoconstriction ensues to limit blood loss and causes local tissue hypoxia. This enhances activation, adhesion, and aggregation of platelets and activates the intrinsic clotting cascade which helps to form a blood clot. The clot provides the provisional matrix which serves as a scaffold for cell migration. Integrins (special surface receptors) on inflammatory and stromal cells bind to the matrix. Chemoattractants, released by degranulating platelets, mediate cellular influx into the matrix. Mast cells also release mediators that further regulate vascular tone and permeability [127].

Inflammatory (Debridement) Phase

The inflammatory phase serves to clean up the wound and prepare it for the proliferative phase. In the early inflammatory phase, neutrophils are recruited to the site of wound healing. Leukocytes are recruited from blood

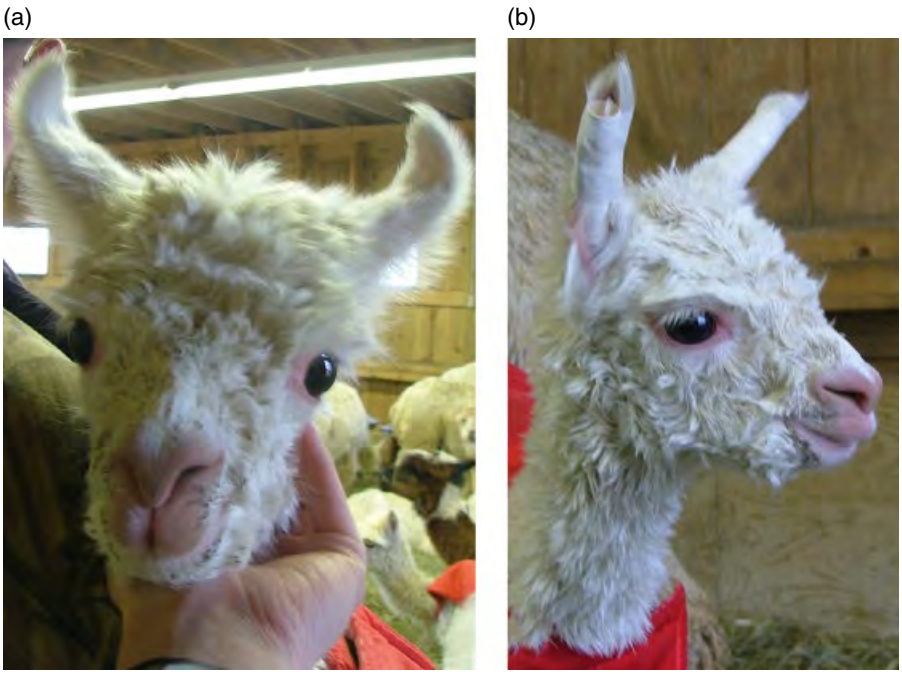


Figure 9.96 (a) Pinna in a newborn alpaca, temporarily curled tips but normal cartilage thickness. (b) Ears of same cria taped around a tampon to support cartilage into proper shape. *Source:* Photos courtesy of Carmon Koenigsknect.

Figure 9.97 Frostbite ears.

vessels. Numerous chemoattractants and vasoactive mediators from platelets and mast cells attract leukocytes to the site of inflammation and promote diapedesis from local vessels into the wound. The leukocytes clean up the wound mostly through phagocytosis and free-radical production. Leukocytes peak at the wound 1–2 days post injury. Neutrophils produce pro-inflammatory cytokines propagating the inflammatory response [127, 128]. Neutrophil migration stops after the bacteria are dead. Interestingly, neutrophils are not a necessary component of wound healing with sterile wounds.

The transformation of monocytes marks the late inflammatory phase. Macrophages have important roles throughout all phases of wound healing, and they orchestrate the overall process. There are resident macrophages present in the tissues and others are transformed from monocytes that enter the wound from the bloodstream. Macrophages have several roles during the inflammatory phase: antigen presentation, phagocytosis, production of inflammatory cytokines, production of growth factors [127, 128].

Much research has shown a difference between wound healing in infected wounds and sterile wounds. The inflammatory cells, neutrophils and macrophages seem to be less important in sterile wound healing. Nude mice, incapable of mounting a neutrophil and macrophage response, healed wounds in a similar time course to wildtype mice and with less scarring [129].

Once the inflammatory cells are no longer required at the site of inflammation, the inflammatory cells die via apoptosis.

Proliferative (Repair) Phase

The goals of the proliferative phase are to form granulation tissue and an epithelial covering over the wound. Experimental camel wounds have shown that this process can begin as early as three days following creation of the

wound [130]. The restoration of the vascular network is another critical component of this phase. The main cellular player during the proliferative phase is the fibroblast. Fibroblasts enter the scene within the first few days following wounding. Endothelial and epithelial cells also invade the wound during this time. Cytokines and growth factors produced by macrophages stimulate fibroblasts to produce extracellular matrix. The formation of new blood vessels (angiogenesis) is also stimulated by these growth factors [131]. Fibroblasts migrate through the clot aided by a proteolytic system. The fibroblasts precede advancing capillary buds and the tissue becomes highly vascular. The extracellular matrix, produced by fibroblasts, replaces the provisional matrix adding strength to the wound [127, 128].

The end of fibroplasia is marked by downregulation of fibroblasts. The capillaries eventually regress and the fibroblast-rich granulation tissue is replaced by acellular scar tissue. The fibroblasts either die via apoptosis or turn into myofibroblasts which are responsible for wound contraction [127].

The wound should eventually be covered by epithelium via a process called epithelialization. Epithelial cells can come from keratinocyte stem cells at the periphery of the wound, from underlying epithelium (in partial-thickness wounds) or from adnexal structures such as hair follicles or sebaceous glands. Epithelial cells migrate across the surface of the wound from the periphery. Proliferation of epithelial cells at the wound margin result in epithelial hyperplasia which serve to replace the cells that are migrating across the wound. A monolayer of epithelial cells covers the wound and is eventually replaced by a multi-layer epithelium similar to the original epidermis. The provisional matrix is eventually replaced by the mature epidermal cells which eventually attach to the basement membrane via hemidesmosomes. Once the surface of the wound is covered by epithelial cells, further migration of epithelial cells cease, mediated by laminin within the extracellular matrix [127].

Remodeling (Maturation) Phase

The goal of the remodeling phase is to increase strength and function of the new tissue. Continuing for up to two years following wounding, this process is the longest of all phases of wound healing. Factors such as the age and health of the animal as well as the size, location, and extent of the wound can affect the rapidity with which the wound remodels. Remodeling depends on proteolytic enzymes especially matrix metalloproteases (MMPs) which are kept in check by tissue inhibitors of metalloproteases (TIMPs). During the remodeling phase, there is no net increase in collagen content, but Type III collagen is replaced by Type I (mature) collagen, and it is arranged in a more structurally organized way. The glycosaminoglycan (GAG) content is decreased to that of normal skin. A remodeled wound is always weaker than the uninjured healthy tissue [127].

Wound contraction is a method by which the wound can be covered at least in part by normal, adjacent tissue that can be "stretched" across the wound, resulting in superior healing because the wound is covered by normal tissue with normal function. Superior cosmesis also results because normal skin architecture is retained without scar-tissue formation. Contraction occurs when fibroblasts within the granulation bed change their phenotype to myofibroblasts (induced by transforming growth factor (TGF) β-1) and begin to pull the edges of the wound together. Wound contraction stops when either the wound edges meet or the tension in the wound is equivalent to the tension in the surrounding tissue. Myofibroblasts, which is the predominant cell type of healing granulation tissue, begin to die off by apoptosis or they revert, back to quiescent fibroblasts [127, 128].

The granulation bed that remains following contraction will develop an epithelial covering (see epithelialization above) and will eventually turn to scar tissue in a process called maturation. Initially, the granulation tissue that fills the defect has poor tensile strength, as the collagen first deposited in the wound by fibroblasts is arranged in a haphazard manner. During the maturation phase, new collagen fibers are formed along lines of tension, form cross-links between one another, and join to existing (normal) collagen fibrils at the periphery of the wound. The remodeled tissue, albeit much stronger than the tissue formed during the proliferation phase, always remains weaker than uninjured tissue, and the bonds between scar and normal tissue serve as a "weak link" [127, 128].

Wound Evaluation

When evaluating a wound on a camelid patient, many factors need to be considered. Chronicity, size, location, involvement of adjacent structures, and degree of contamination of the wound are important factors (Figure 9.98). The age, health, and disposition of the patient are patient factors that may influence wound healing. Facilities, resources available, and economics must also be considered with every case. Because of their long fiber, accurate assessment of camelid wounds can be challenging, and the fiber surrounding the wounds should be clipped in order to achieve an accurate evaluation and to perform proper management of the wound.

A thorough physical exam is paramount. Animals that present with severe wounds may also suffer from blood loss or shock. A packed-cell volume and total protein should be

Figure 9.98 Deep, contaminated wound, distal to the hock involving multiple structures including the flexor and extensor tendons. A large portion of the lateral metatarsus is exposed and denude of its periosteum. Involvement of these structures will reduce the prognosis for success. *Source:* Photos courtesy of Dr. Matt Miesner.

assessed if the clinician feels that significant blood loss could be a factor, and he should be prepared to restore circulating blood volume if necessary. Patients with chronic wounds may develop bacteremia or septicemia and an assessment of white blood cells with a complete blood count (CBC) may be useful.

Wounds can involve other structures such as neurovascular structures, tendons and ligaments, synovial structures or body cavities which can greatly influence treatment and prognosis. A sterile probe or teat cannula can be used to probe wounds and assess their depth and involvement with other structures. Wounds caused by animal bites are often deeper than what is initially apparent (Figure 9.99). Radiographs with a probe inserted in the wound or following injection of iodinated contrast material can be useful to further assess wound depth and communication with boney or synovial structures. Ultrasound imaging may also be useful, though gas within the tissues will limit ultrasound penetration. If there is concern of synovial involvement, the synovial structure should be distended with sterile saline; if saline is observed leaking from the wound, communication is confirmed. The appearance and temperature of the soft tissues surrounding the wound can provide a reasonable assessment of the tissue perfusion and viability. However, caution should be used if evaluating wounds in the acute stages, as it may not be readily apparent as to what tissue is viable or non-viable.

Wound Management

Primary, Delayed Primary, & Second Intention Healing

Primary wound closure involves surgical closure of acute wounds that are clean or have minimal contamination. The current medical literature suggests that primary closure results in the least amount of patient morbidity, faster healing, and an optimum functional and cosmetic outcome [132]. It was once thought that wound chronicity was the main determining factor dictating the success of wound healing and if wounds should be closed primarily, managed with delayed closure, or allowed to heal by second-intention healing [133]. The "golden period" usually thought to be six to eight hours was an arbitrary time before which, the recommendation was to close wounds primarily, and after which wounds would become infected if

(a) (b)

Figure 9.99 This male alpaca was attacked by a dog and sustained multiple wounds in the inguinal area. Bite wounds are deeper than what might be apparent initially and should be probed. (a) A Penrose drain has been placed to facilitate drainage. (b) The same alpaca after treatment.

closed [134]. Recent work has suggested whether a contaminated wound becomes infected is multifactorial, depending on factors like age of the patient, blood flow to the site, immunocompetence, and degree of contamination with time following wounding as a less important determinant. It has been shown in humans and animals that wounds closed after the once-thought 'golden period' can heal without infection [135, 136].

Delayed primary closure involves cleaning up the wound and providing time for the inflammation to decrease prior to primary closure. Delayed primary closure occurs before granulation tissue has formed but after the acute inflammatory response and tissue edema has resolved. Wounds with minimal contamination are candidates for delayed primary closure [137].

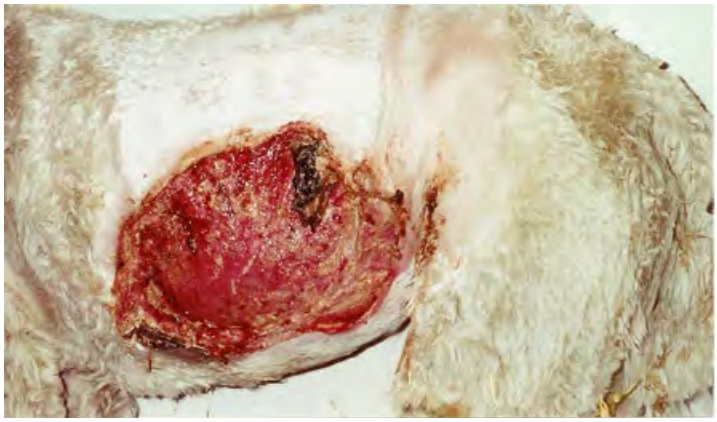
Closing contaminated wounds primarily gives organisms a more favorable environment to propagate and increases the risk of infection. Though the exact number likely varies with immune status of the host and virulence of the bacterial species, it is estimated that it takes significantly fewer organisms to elicit infection in open wounds (10^6 organisms/g of tissue) compared to wounds closed primarily (10^5 organisms/g) [138–140]. Either delaying closure in severely contaminated wounds or allowing them to heal by second-intention healing may be advantageous. Delayed primary closure (closure after cleansing and debridement of the wound), delayed secondary closure, (closure after granulation tissue has started forming) or second-intention healing (allowing the wound to heal without surgical closure), may be better options for severely contaminated wounds. Wounds that heal by second-intention healing generally form the largest scar, take the longest to heal, and result in the weakest tissue following healing, however, the result can be very acceptable especially if there is extra, healthy skin surrounding the wound (Figure 9.100). In some cases, the wound is too large with inadequate skin available to close the defect primarily and allowing the wound to heal by second-intention closure is the only option (Figure 9.101).

Wound Cleansing

Most all wounds, unless created in a surgical environment, are contaminated by the time they are treated, necessitating cleaning and possibly debridement of damaged tissue. Wounds on the distal extremities are closer to the ground and are often thought to be more contaminated than wounds on the body [141]. While cleansing the wound, care should be taken to not disrupt healthy tissue and new healing tissue; granulation tissue and new epithelium is friable and is easily damaged. The goals of wound cleansing are to remove gross debris, microorganisms, and exudate from the wound. If damaged and necrotic tissue is present, most wounds will benefit from surgical debridement of this tissue (Figure 9.101).

With many traumatic wounds, it may be beneficial to perform cold-water hydrotherapy with tap water using a hose to lavage the wound with copious amounts of fluid. Potable tap water has been shown to be an acceptable and cost-effective irrigant for open wounds [142]. Benefits include removing gross debris and exudate dried to the wound, softening the eschar to help with debridement, and vasoconstriction from the cold water to control hemorrhage and thwart inflammation. The benefit of this therapy is greatest in the early stages of wounding. Hydrotherapy has also been shown to encourage granulation tissue formation in horses [143] and after a granulation bed is established, continuing hydrotherapy may be contraindicated. Warm water therapy may have benefit in

(a)



(b)

Figure 9.100 (a) Second-intention wound healing of a left flank wound resulting from a skin dehiscence after surgery. The flank region has abundant skin which results in excellent wound contracture. (b) Wound has healed and resulted in a small scar.
Source: Photos courtesy of Kathy Zabar.

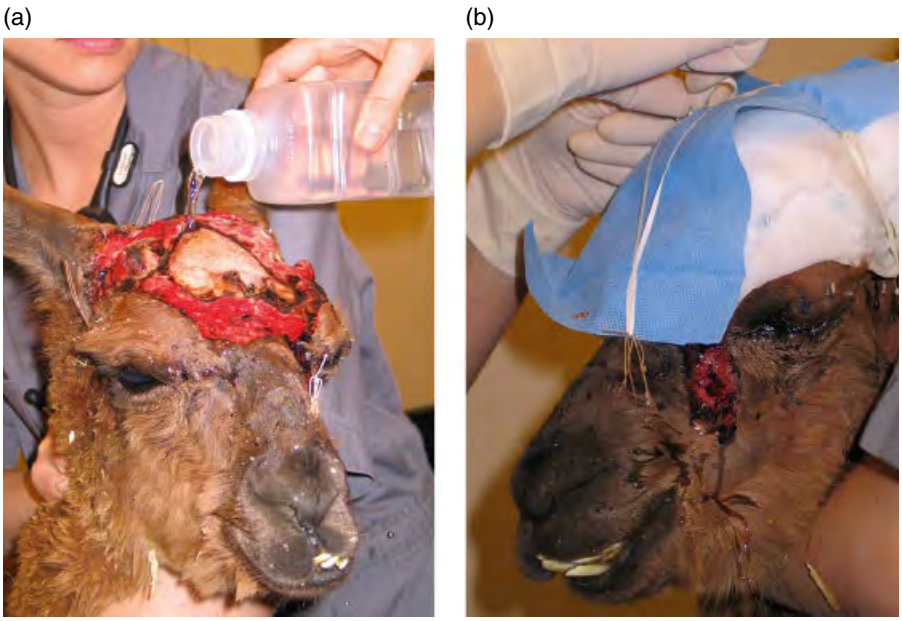


Figure 9.101 Alpaca with extensive head wound. (a) There is not enough surrounding tissue to close primarily and the wound is encouraged to heal by second-intention healing. Granulation tissue is forming around the periphery of the wound. The wound is lavaged with sterile saline to remove gross debris and exudate. (b) A wet-to-dry, tie-over bandage is placed to facilitate debridement (*Source:* Photos courtesy of Dr. Claire Whitehead).

chronic wounds to vasodilate and help promote blood flow to the region.

While hydrotherapy will reduce gross contamination, low pressure water lavage has been shown to be ineffective at reducing the surface bacteria to below infective levels. It has been shown that higher pressure lavage and pulsating jet lavage is significantly better than gravity flow or low-pressure irrigation using a bulb syringe or fluid pressure bag at reducing the bacterial count of wounds [144]. Studies in horses have shown that the ideal pressure to lavage wounds is between 10 and 15 PSI. Below 10 PSI, the fluid does not have enough pressure to adequately clean the wound; above 15 PSI, the fluid can drive debris and microorganisms deeper into tissues [145]. A syringe or gravity flow is insufficient at achieving this pressure; a 35- or 60-mL syringe attached to an 18-gauge needle can achieve lavage pressures within this range [146].

There are multiple wound cleansers available, which contain surfactants to break down biofilm and are more effective in highly contaminated wounds than tap water or isotonic fluid. However, some products may be detrimental to cells in the wound.

Local Wound Therapy

While elemental silver is inert, the ionized form of silver possesses broad-spectrum antimicrobial properties. It is included in ointments designed for wound like silver sulfadiazine. Silver's antimicrobial effects are thought to be related to the silver ions binding to sulfhydryl groups and proteins on cell membranes of the organism. Ionized silver inhibits key enzymes in the organism, causes destruction of the cell wall, and disrupts the bacterial cell DNA. Silver free radicals are also thought to play a role in its antibacterial properties [147]. It has efficacy against common wound pathogens including Staphylococcus, Enterococcus, Pseudomonas, and *E. coli*, but its effect is short-lived as the silver ions are rapidly inactivated.

Iodine containing antiseptics like povidone iodine cadexomer Iodine are commonplace in veterinary medicine. Cadexomer Iodine is a newer and less toxic product and is thought to reduce the bioburden and stimulate epidermal regeneration. A study also showed that there were no negative effects of cadexomer-iodine ointment on granulation tissue formation, neovascularization, or wound contraction [148]. Povidone iodine is more commonly used and is relatively safe, however, it has been shown to impair collagen synthesis and have toxic effects on keratinocytes but there is no direct evidence that it is detrimental to wound healing. Its antibacterial efficacy is related to the content of available iodine and it is most efficacious when diluted to a 10% solution which gives 1% available iodine by adding 10 mL of povidone iodine to 990 mL of saline solution.

Chlorhexidine is another commonly used antiseptic solution. Chlorhexidine binds to proteins in the stratum corneum and thus has more residual activity than povidone iodine. It also has more efficacy in the presence of

Figure 9.102 Llama with chemically induced keratitis secondary to chlorhexidine exposure. *Source:* Photo courtesy of Dr. Anne Metzler.

organic debris compared to povidone iodine. Proteus and pseudomonas may be resistant to chlorhexidine. It is typically well-tolerated; however, cytotoxicity is possible and has caused occasional contact dermatitis. Chlorhexidine is extremely irritating to the cornea and causes coagulation necrosis of the corneal epithelium [149] (Figure 9.102). It is also ototoxic if it gains access to the inner ear [150], and appears to be harmful within synovial cavities leading to synovial ulceration and fibrin accumulation [151].

Hydrogen peroxide is used commonly as a disinfectant for wounds because of its availability and cost, but its use is controversial as it has been shown to cause free-radical induced cytotoxicity at higher concentrations [152]. It has been shown to significantly increase graft acceptance in chronic-colonized burn wounds in people at 2% without detriment [153]. It is unstable in long-term storage and its antimicrobial efficacy will decrease with time [154]. It is thought to have sporicidal properties and does provide some mechanical debridement from the bubbling action.

Sodium hypochlorite and hypochlorous acid are compounds that possess effective, broad-spectrum antimicrobial efficacy. Sodium hypochlorite is the active compound in household bleach, and is available as a wound antiseptic, marketed as Dakin's® solution. Dakin's solution is available as full strength, Half strength, and quarter strength. Full strength is 0.5% sodium hypochlorite solution and is the highest concentration tolerated by intact skin (compare to bleach which is 5% solution). Quarter strength Dakin's solution is 0.125% and may be more tolerated by open wounds but still may have cytotoxic properties at this strength. Hypochlorus acid is a broad-spectrum antimicrobial with a rapid bacterial kill. It has more antibacterial efficacy [155], and is less corrosive than the hypochlorite ion. Its antibacterial mechanism of action is similar to the biologic action of the leukocyte *in vivo*, and has less cytotoxicity compared to the hypochlorite ion [156]. Vetrycin® is a commercially available product containing a mix of hypochlorous acid and sodium hypochlorite along with electrolyzed water, sodium chloride, and phosphates.

Mild acidic solutions have been shown to have antibacterial properties. These seem to be mostly efficacious against Gram-negative organisms like pseudomonas. Products containing acetic acid, boric acid, citric acid and ascorbic acid are available and can be used on wounds. Acetic acid is thought to be cytotoxic with limited bacteriocidal activity [157], however a study has shown that a 0.25% acetic acid solution had low cytotoxicity compared to hydrogen peroxide and a 10% povidone iodine solution [158].

Scarlet oil is a wound antiseptic that contains parachlorometaxylenol (pcmx) as the active ingredient combined with alcohol and eucalyptus and pine oil base. It is one of the oldest antimicrobials but has relatively weak antiseptic properties [159]. A study looking at parachlorometaxylenol alone as a surgical scrub found it to have poor efficacy as a surgical hand preparation, though it did not differ significantly from chlorhexidine as a surgical hand scrub in their study [160]. Studies looking at scarlet oil itself are lacking but it is still widely used in large animal veterinary medicine as a wound antiseptic. In addition to its antiseptic properties, scarlet oil is an irritant and is thought to promote granulation tissue formation. It can also be used as a fly deterrent around wounds.

Triple antibiotic ointment contains a mixture of bacitracin, Polymyxin B, and Neomycin and provides broad-spectrum protection except for pseudomonas. It is commonly used because of its availability and broad-spectrum action. It may promote epithelization [157].

Nitrofurazone ointment is available and still used in equine practice as a topical antimicrobial. It is prohibited from use (even topically) in food producing animals in the United States but may still be used in camelids not intended for food. It has been shown to have carcinogenic properties [161, 162] and can cause allergic contact dermatitis [163], and is no longer recommended for use.

Topical agents can be used to accelerate wound debridement. Enzymatic debridement involves using exogenous enzymes to break down necrotic tissue while preserving viable tissue [157]. Bacterial collagenase can be used to break down collagen bundles that bind non-viable tissue to the wound's surface. These agents are selective for non-viable tissue while being gentile on viable cells. Streptokinase breaks down fibrin and may help to digest the biofilm making other antibiotics more effective. Papain is a proteolytic enzyme and is the active ingredient in meat tenderizer. It is combined with urea and marketed as an ointment to break down proteolytic debris on wounds. Papain has non-selective action and is reported to be

painful when used in wounds. However, it has been shown to be safe for wounds in various stages of healing and to promote wound healing without any specific contraindications [164, 165].

Maggot debridement therapy uses larvae from the green bottle fly (*Lucilia sericata*) that are produced sterilely to facilitate wound debridement, a process known as biosurgical debridement. Indications for maggot debridement therapy include chronic, non-healing wounds containing necrotic tissue, exudate, and metabolic wastes where complete surgical debridement is not possible. Maggots are considered the "ultimate wound debriding agent" [166]. The larvae preferentially feed on necrotic over healthy tissue [167]. Added benefits include the production of proteolytic enzymes, up-regulation of the genes involved in wound healing, antimicrobial effects and inhibition on biofilm formation [168, 169].

Steroids are known to have generally negative effects on wound healing [20, 170]. Steroids reduce fibroblastic proliferation, decrease angiogenesis, decrease epithelialization, decrease wound contraction, and decrease collagen synthesis [171]. In wounds demonstrating an abnormal inflammatory response [172], most notably equine wounds associated with formation of exuberant granulation tissue [171, 173, 174], topical steroids have shown to have beneficial effects to wound healing. Though the formation of exuberant granulation tissue is less common in camelids than horses, it does occur and is most prevalent on the distal extremities because these areas have less redundant skin and therefore wound contraction is minimal. Also, wounds over areas with chronic motion (like joints) are prone to exuberant granulation tissue formation (Figure 9.103).

Figure 9.103 Llama with chronic wound over the carpus. Wounds on the distal limbs cannot contract well because there is no redundant skin to stretch across the wound. Also, the chronic motion caused by the joint prevents epithelization of the wound. These are often forced to heal by production of exuberant granulation tissue.

It has been demonstrated that biomaterials rich in sugar are safe and promote wound healing [175]. Ordinary sugar (sucrose) can be used in the inflammatory phase of wound healing to facilitate debridement and to provide a wound environment that is non-conducive for microbial growth due mostly to the hypertonicity of the sugar, depleting cellular organisms of needed water [176]. Added benefits include lack of toxicity, encouragement of angiogenesis, reduction of wound odor, and prevention of scaring [177]. Granulate sugar is very hypertonic and can be used in wounds to limit bacterial growth. Pseudomonas organisms are very susceptible to the effects of sugar. Sugar has been used successfully in the treatment of superficial wounds and deep cavity wounds. Its use as a topical wound therapy is safe, efficacious, and relatively inexpensive [178–180]. The hypertonicity of the sugar also cause a fluid shift through the tissues promoting debridement and reducing edema. As fluid is drawn into the sugar, the hypertonicity of the sugar solution decreases and eventually will begin to promote bacterial growth; frequent dressing changes are required. Sugar can be combined with povidone iodine, to increase its antibacterial efficacy and to modulate the activity of keratinocytes and fibroblasts [181, 182]. This combination, commonly referred to as Sugardine, is created by mixing povidone iodine with either granulated sugar or icing sugar until a workable paste is created [157].

Honey has been used for centuries as a wound treatment. Like sugar, honey has hypertonic properties and, as such, it does not promote bacterial growth. Its viscous, paste-like properties make it easier to apply to wounds than granulated sugar. It also has a low pH and contains glucose oxidase which enhances its antibacterial properties by converting glucose to gluconic acid and hydrogen peroxide [183, 184]. In addition to its direct antibacterial effects, honey has been shown to have anti-inflammatory properties which promote wound debridement, fibroplasia, and epithelialization [175]. It immunomodulatory mechanisms have not been fully established but is theorized to have effects on the production of cytokines from monocytes and macrophages, the production reactive oxygen species from neutrophils, effects on keratinocytes, fibroblasts, and endothelial cells [185]. Specifically, manuka honey, produced from the Leptospermum tree found in New Zealand and Australia, contains methylglyoxal which further enhances its antibacterial properties. Manuka honey can withstand substantial dilution with exudate and still retain antimicrobial properties [183]. Manuka honey is considered the most effective form of honey for "multifunctional activity in wound management" [186], though both Manuka honey and ordinary "pasture" honey has been

shown to have similar antibacterial efficacy against prevention of pseudomonas growth on wounds [187].

Wound Dressings

Until relatively recently, the dogma regarding wound healing was that wounds should be kept dry to optimize healing. That dogma has largely been refuted and it has been shown that moist wound healing encourages epithelization of wounds [188]. In addition, moist wound healing promotes delivery of antimicrobials, analgesics, and other bioactive molecules such as growth factors to the wound [189–191].

Moist wound healing necessitates a covering that prevents the loss of moisture from the surface of the wound. Many wound dressings are available to the veterinarian and no single dressing optimizes the wound healing environment for all stages of wound healing. Therefore, a knowledge of the stages of wound healing, as well as the properties of the dressings, are important for selection of the proper product for a given wound. The veterinary literature regarding the use of wound dressing in camelids is sparse to non-existent; most of the evidence is extrapolated from other domestic species or humans, and even these reviews provide weak evidence on the clinical efficacy of dressings for wound management [192].

Occlusive dressings prevent gas exchange at the wound surface including loss of water vapor. This type of dressing provides a moist wound environment with low-oxygen tension and a mildly acidic wound environment. The low-oxygen tension promotes angiogenesis [193]. Examples of fully occlusive dressings are hydrocolloid and silicone gel dressings. Indications for these types of dressings include wounds in the inflammatory stage when granulation tissue is desired [194].

Wet-to-dry (or dry-to-dry) bandages are dressings used to promote debridement of necrotic tissue and exudate [166] (Figure 9.101). Saline soaked gauze is allowed to dry on the wound. Exudate sticks to the bandage, and fibrin absorbs into the bandage. The bandage sticks to the wound and provides non-selective debridement when removed. This type of bandage can be very useful in wounds that have a lot of necrotic tissue but shouldn't be used when healthy granulation tissue or epithelialization is present because damage to the healthy tissue can result. In addition to the non-selectivity regarding tissue debridement, these bandages are painful to remove [166].

Chitin and chitosan are polysaccharides produced from fungi, yeast, or the exoskeleton of crustaceans. This is an absorbable material and provides hemostasis. Chitin and chitosan also attract leukocytes and increases their function, mainly phagocytosis and chemokine production [194].

Alginates are formed from seaweed. They are extremely effective at absorption and can absorb up to 20 times their weight in fluid. They are also very effective at debriding necrotic wounds and are more effective than other dressings at reducing wound area [192].

Hydrogels are hydrated polymers, consisting of 90%–95% water. Hydrogels are considered occlusive. Because of their high-water content, they can donate water to the wound and help to rehydrate dead and desiccated tissues, thereby promoting autolytic debridement [194]. However, they still retain capacity for water absorbency [166]. They will help to keep wounds cool in the face of infection. They are not usually recommended for infected wounds as they propagate infection, and in horses they are known for promoting exuberant granulation tissue formation so their use on granulating wounds is not recommended [194]. It is unknown whether hydrogels will promote excessive granulation in camelids.

Foams are non-adherent and semi-permeable. They are designed to maintain appropriate moisture at the wound bed, decreasing the risk of maceration. They have the potential to absorb a lot of exudate [166]. They provide variable degrees of absorption without drying out the wound. In humans they are reported to be comfortable to wear and remove.

Polyurethane films and silicone dressings provide a semi-occlusive to occlusive wound barrier. These dressings are waterproof and provide little to no absorptive capacity; therefore, they should not be used for exudative wounds, else maceration of the wound and adjacent skin can result. Polyurethane films will allow water vapor to escape, and both polyurethane films and silicone will provide for moist wound healing. Polyurethane films are coated with an adhesive; silicone will not stick to the wound surface but will lightly adhere to the surrounding skin.

Biologic dressings promote healing by creating a moist wound environment that allows migration of epithelial cells and retards exuberant granulation tissue. They are designed to be incorporated into the wound. Examples of tissues that can be used as biologic wound dressings include amnion, peritoneum, collagen, and skin. These can be challenging to obtain which adds to the cost of therapy [194].

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10

Musculoskeletal System

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Anatomy and Conformation

No text describes the anatomy of the musculoskeletal system of South American camelids. However, modern anatomy text of the dromedary camel is available [1]. There are many differences, but much of the information is applicable to South American Camelids. Only those clinical important aspects of anatomy unique to camelids are discussed here. Figure 10.1a–c are diagrammatic representations of the skeletal system of a llama, alpaca, and Bactrian camel, respectively.

Thoracic Limb

The angulation of the pastern and fibroelastic digital cushion of the foot absorb most of the force during ambulation (Figure 10.2). Normally, phalanx 1 (P1) is semivertical. Alpacas have a more vertical pastern (P1), approximately 80°, while the llama has more slope to the pastern (55–60°). P2 and P3 lie horizontally within the foot. Camels have a slightly different arrangement of the phalanges. P1 is essentially vertical, P2 at 45°, and P3 horizontal. Figure 10.3 illustrates a condition called "down in the fetlock," "weak pastern," or hyperextension of fetlock. More on fetlock hyperextension can be found later under "Disorders of Joints." Alpacas may be more inclined to develop hyperflexion of the fetlock (cocked ankle). When standing relaxed, alpacas appear to be slightly base wide in front. The appearance is enhanced by the fiber on the limbs and may even give the impression that an animal is knock-kneed. Careful observation must be used to make certain that the legs are tracking straight in these patients.

Pelvic Limb

The overall stance of the rear limb of camelids is straighter than that of the horse or cow; however, many domestic camelids tend to stand slightly sickle-hock (Figure 10.4a and b).

Gaits

All camelids use the same basic gaits: walk, pace and gallop. South American camelids, especially alpacas, may also trot, though this gate is uncommon in camels. A fifth gait, or playing gait, of South American camelids is called the pronk or stott.

The walk is an evenly spaced four-beat gait in which each foot strikes the ground separately and in sequence. Three feet are always on the ground. It is the slowest gait and can be sustained for long periods. It is also the most stable gait, providing the greatest base of support.

The pace is a medium speed, two-beat gait in which the fore and hind limbs on the same side of the body move in unison. Though the pace is the least stable of all animal gaits, it is designed to permit an animal to move swiftly over open country. Though lateral stability is reduced while pacing, camelids are uniquely conformed to use it. The unique foot of the camelid may be an adaptation to increase the stability of the animal. All other two-toed

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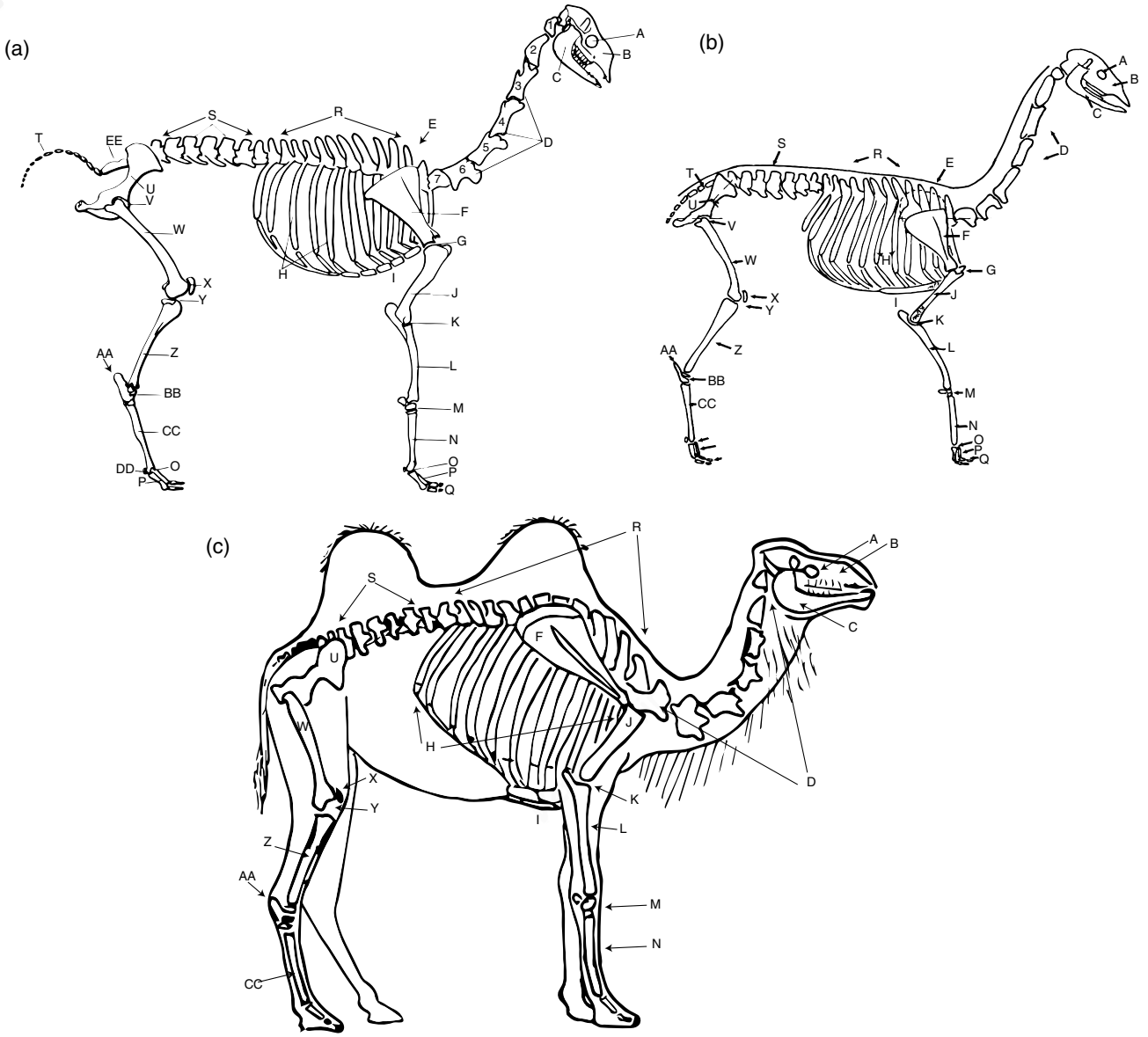

Figure 10.1 Diagrams of camelid skeletons. (a) Llama, (b) Alpaca, (c). Bactrian Camel.(A) Orbit, (B) maxilla, (C) mandible, (D) cervical vertebrae, (E) withers, (F) scapula, (G) shoulder joint, (H) ribs, (I) sternum, (J) humerus, (K) elbow, (L) radius, (M) carpus, (N) metacarpus, (O) fetlock, (P) pastern bones, (Q) P3, (R) thoracic vertebrae, (S) lumbar vertebrae, (T) coccygeal vertebrae, (U) pelvis, (V) hip joint, (W) femur, (X) patella, (Y) stifle, (Z) tibia, (AA) tuber calcis, (BB) tarsus, (CC) metatarsus, (DD) sesamoids, (EE) sacrum.

ungulates have ligaments that tie the digits together. Camelids have a splay-toed foot (not to be confused with splay footed) that spreads and provides a strong base of support. This, combined with the padded foot, makes the camelid one of the more surefooted ungulates. Adaptations proximal to the foot also permit this gait. Camelids have relatively long legs, with each limb being longer than the trunk, allowing the animal to develop a long stride. The cranial aspect of the thorax is narrow, permitting the proximal thoracic limb to freely move forward and backward. Also, in camelids, the attachment of the pelvic limb to the pelvis is narrow, and the abdomen is less rounded than in other livestock species, allowing the pelvic limb freer motion. Camelid limbs are also set more closely to the midline than in other species, eliminating some of the side-to-side rolling that occurs when the center of body gravity is changed with each stride. All of these adaptations make the pace much more stable in camelids than in other ungulate species.

Some llamas and alpacas routinely trot instead of pace. The trot is also a two-beat gait, in which the diagonal fore and hind limbs move in unison.

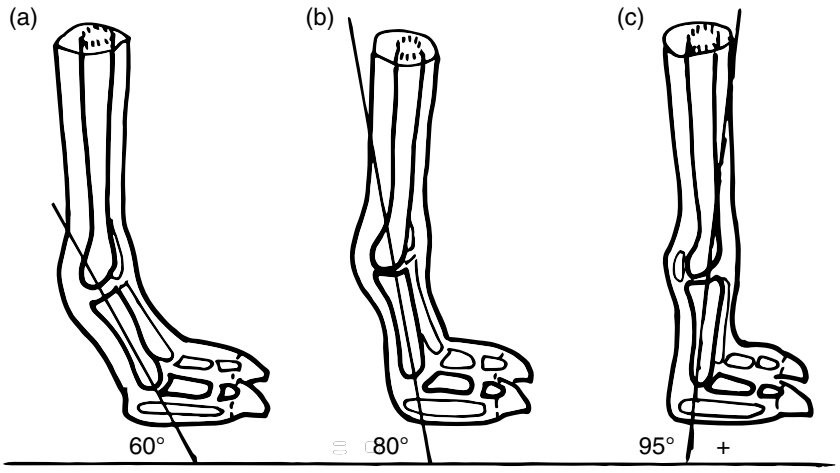

Figure 10.2 Diagrams of pastern angulation. (a) Appropriate angulation for a llama, (b) appropriate angulation for an alpaca, (c) over at the fetlock (hyperflexion of the fetlock).

Figure 10.3 Hyperextension of fetlock.

The fastest gait is the gallop, or run. It is the most fatiguing and can only be sustained for a few moments because, unlike horses, camelids lack heavy propulsion muscles in the rear limbs. Alpacas have a shorter stride than llamas and they tend to gallop more than pace when excited or running away from pursuers. Dromedary camels alternate between a pace and a gallop when racing.

Pronking (stotting) is a form of locomotion in which the SAC thrusts the body upward and forward, landing on all four feet at the same time in a stiff-legged stance. This is a playing gait and is more commonly seen in young animals, especially in the evening or when new animals are introduced into the herd.

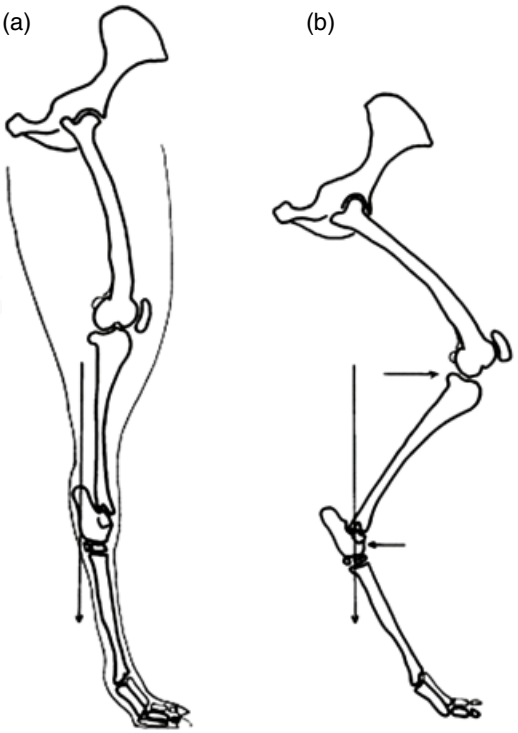

Figure 10.4 Hind limb, lateral view. (a) Straight leg, (b) excessive angulation (sickle-hock).

Specific Conditions

Long Bone Fracture

Fractures in camelids are caused by the same types of trauma reported in other species of livestock and companion animals [2]. Any of the fixation techniques practiced in other domestic animals are applicable in appropriate situations in camelids [3]. The size of llamas and alpacas places them between small animals and horses or cattle, and often permits a large array of management options to be utilized. Similar to cattle [4], camelids tolerate orthopedic surgery and application of various orthopedic devices well. Therefore, selection of a particular procedure should depend upon the bone involved and the nature of the fracture, available anesthesia, equipment and instrumentation, and the skill and experience of the surgeon. Though sophisticated procedures, such as open reduction and

internal fixation, are valuable for many fracture repairs, they may be contraindicated in certain cases where less sophisticated procedures are valid and effective.

Principles of Fracture Repair

Numerous factors must be considered when determining optimum treatment for long bone fractures. Patient age and size, fracture configuration, location, whether the fracture is open or closed, tissue damage, and owner expectation and financial position are all important aspects to consider. Having the knowledge of available treatment options for various fractures is imperative during the decision-making process when presented with a fracture. Techniques used to stabilize fractures are grouped either as external stabilization or internal fracture fixation.

External Stabilization

External stabilization takes advantage of the patient's capacity of secondary bone healing by stabilizing the bones in relatively close apposition.

Splints and Casts Casts and splints are the simplest form of external stabilization. Basic principles of fracture immobilization dictate that at least one joint above and one joint below the fracture must be immobilized for adequate healing to occur. As it is very challenging to immobilize the elbow or stifle with a cast or splint in camelids, only fractures of, or distal to, the metacarpus or metatarsus should be managed in this way. There are exceptions to this rule, however. Salter-Harris types I and II fractures interdigitate when reduced and help to prevent displacement. Therefore, a simple cast may be used successfully in the case of a distal radial or distal tibial Salter-Harris type I or II fracture.

Thomas Splints Thomas splints consist of a ring placed in the axillary or inguinal region which is connected to sidebars and a distal footplate (Figure 10.5). They may be combined with a cast and act similarly to a crutch, helping to transfer the weight of the patient from the axillary or inguinal region through the sidebars and past the fracture site to the footplate and ground. They may be used to stabilize fractures as proximal as mid-radius or mid-tibia and are also useful in comminuted fractures lacking inherent stability which may preclude the use of a plain cast. Their construction and application can be found elsewhere [5, 6]. Thomas splints can be cumbersome, and the patient may need assistance standing on occasion [7]. Other complications associated with Thomas splints include sores caused by the construct or cast, poor alignment, malunion, and delayed union. Some fractures may even become open in these constructs [5, 7].

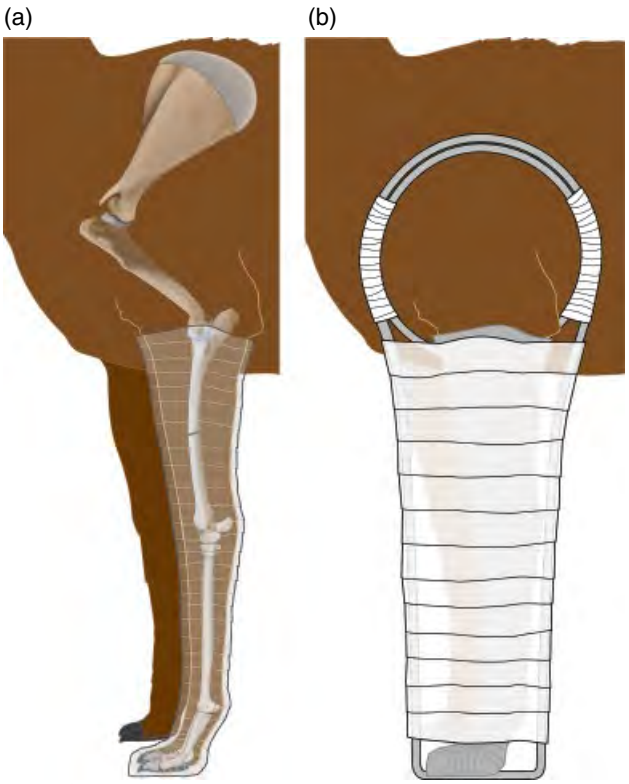

Figure 10.5 Treatment of a radius fracture with a Thomas splint/cast combination. Cast tape is used to incorporate the entire limb up to the elbow (a) and then used to secure the limb to a metallic Thomas splint placed in the axillary region (b).

External Fixators and Transfixation Pin Casts External fixators are another form of external stabilization which may be adapted to use in camelids. Transfixation pin casts are a modification of the traditional external fixator design and are particularly useful in large animals including camelids. The design consists of two pins placed proximal to the fracture in the frontal plane (medial to lateral or lateral to medial). A cast, incorporating the foot, is then applied. This cast then acts as the sidebars to the external fixator, and transfers the force to the ground, obviating the need for distal pins. See Figure 10.6. That is, as the patient bears weight, this force is transferred through the pins to the cast, and through the cast to the ground. The limb distal to the pins is suspended (hanging) by the pins within the cast, which is why this construct is sometimes referred to as a hanging-limb cast. Like the Thomas splint, this technique allows external stabilization management of fractures as proximal as the mid-radius or mid-tibia, as well as those with a configuration not lending themselves to plain cast application. There are fewer complications with a transfixation pin cast than with a Thomas splint but include cast sores and possible pin-tract fracture [7].

Figure 10.6 A transfixation pin cast is used to stabilize a metacarpal fracture. The weight of the animal is diverted through the pins to the cast and finally the ground, bypassing the site of the fracture.

However, this technique does typically require general anesthesia, unlike a Thomas splint, and additional surgical expertise is necessary.

Open Reduction and Internal Fixation

Internal fixation may be indicated in some fractures. Displaced humerus or femur fractures are unlikely to heal with conservative management, and external stabilization may not be possible. Mid-to proximal radius and tibia fractures may be impossible to effectively stabilize with most external stabilization techniques. Tibial fractures may be particularly challenging to reduce and manage with external stabilization, as the reciprocal apparatus tends to result in overriding of the fragments which cannot be overcome with traction. Principles and techniques for internal fixation can be found elsewhere [8–12].

Specific Limb Fractures

Though surgical approaches to the long limb bones are basically similar to those performed on dogs, horses, and cattle, there are slight variations [2, 13, 14]. A description of recommended approaches and treatment options for selected fractures are as follows.

Humerus

Humeral fractures may represent up to 18% of camelids presenting to referral institutions with fractured long bones and, most commonly, have a long oblique orientation [15]. Successful repair has been achieved with dynamic compression plate application, intramedullary pinning combined with lag screws or circlage wire, and lag screws alone [15–17]. Many humeral fractures would also be amenable to repair with an interlocking nail [7]. Camelids tolerate application of a Velpeau sling well, and this may be useful in the convalescent period to reduce implant strain/cycling, regardless of fixation type [15]. Application of a Velpeau sling can be found later, under "scapulohumeral luxations." Conservative management of non- or minimally displaced fractures is also an option, though this is associated with a higher rate of complications including limb contracture, persistent lameness, delayed union, and malunion [7, 15]. The use of a Velpeau sling at least in the early stages of conservative management of these fractures should be considered.

The distal humerus should be approached from the craniolateral aspect of the limb, directly over the bone. The skin and fibrous sheath over the muscles should be incised from the point of the greater tubercle to the lateral epicondyle. The biceps brachii muscle may be reflected away from the cranial border of the humerus. The radial nerve should be identified and avoided as it curves diagonally around the caudal and lateral aspects of the distal third of the humerus. If the caudal border must be exposed, it is necessary to transect the attachment of the deltoideus muscle from the deltoid tuberosity.

The major blood supply to the forelimb is the median artery, which lies on the medial aspect of the humerus and is protected by tissue. For this reason, fractures of the humerus are less likely than femoral fractures to result in transection of major blood vessels.

Following surgery, the patient should be fitted with a Velpeau sling for two to three weeks, and stall-rested in a small pen for six to eight weeks. A good prognosis can be expected following repair of humeral fractures [15].

Radius/Ulna

Successful management of radius/unla fractures in camelids has been reported with the use of a Thomas splint, transfixation pin cast, lag screw repair, and dynamic compression plate application [3, 7, 13, 16–19].

Location of plate placement on the radius in camelids depends largely on the fracture configuration. The radius is superficial throughout its length on the medial aspect of the limb, and provides a suitable surface plate application, albeit less soft-tissue coverage following the procedure. To expose the bone medially, incise the skin and the heavy subcutaneous fascial sheath and reflect the extensor carpi radialis muscle from the cranial border of the radius along with the major vessels and nerves to the lower limb. The flexor carpi radialis muscle is firmly attached to the caudal border of the radius. The craniolateral approach may also be used by making a curvilinear incision from the lateral epicondyle of the humerus to the lateral tuberosity of the radius, following the cranial curvature of the radius. The dissection is then continued between the common digital extensor and the extensor carpi radialis to the radius.

One study found the outcomes of radius/ulna fractures to be poorer than fractures of other long bones [16]. Despite this, radius fractures carry a good prognosis in camelids except in the presence of comorbidities such as radial nerve or articular damage [7].

Metacarpus/Metatarsus

A wide variety of treatment options are available for fracture of the metacarpus/metatarsus depending on the fracture configuration and economics available for therapy. Successful reports of external stabilization of fractures include casting, transfixation pin casting, and other external fixation techniques [2, 3, 20, 21]. Open reduction and internal fixation has been reported with the use of dynamic compression plating [19]. There is one successful report of a minimally invasive repair using a locking plate in a guanaco [22].

Both medial and lateral aspects of the metacarpus and metatarsus are superficial and accessible for surgery. However, the major blood supply to the foot lies on the medial side in the groove between the suspensory ligament and the deep digital flexor tendon; thus, if the fracture configuration permits, this is the approach that should be used to allow visualization and avoidance of the vessels. Exposure begins by incising the skin and reflecting the extensor tendons dorsally. The flexor tendons, suspensory ligament, vessels, and nerves are encased in a heavy fibrous sheath, which must be incised to expose the bone. At the level of the junction between the middle and distal third of the metatarsus, the vessels and nerves leave the sheath and track to the palmar/plantar aspect of the flexor tendons, continuing distally in the space between the paired tendons, ligaments, and digits of the foot.

The two nutrient foramina, one for each marrow cavity, are located slightly proximal to the middle of the bone.

Femur

Successful repair of femoral fractures has been reported with the use of intramedullary pins with and without circlage wire, interlocking nail application, and dynamic compression plating [16, 17, 23].

To expose the femur, the skin should be incised on the lateral aspect of the limb directly over the bone. The fascia lata lies immediately beneath the skin and should be incised in the same direction. The fascial sheet should be identified and incised between the biceps femoris muscle (caudal) and the vastus lateralis muscle (cranial). The biceps femoris muscle fibers run diagonally to the long axis of the limb, and those of the vastus lateralis run vertically.

The femur may be palpated at the distal end, but more proximally it is covered by the vastus intermedius muscle on the lateral and cranial borders. To completely expose the femur, it is necessary to sever the attachment of the vastus intermedius muscle on the lateral and cranial borders of the bone.

The sciatic nerve lies 3–5 cm caudal to the femur, well out of the way of the surgical site. The femoral artery and veins lie medial to the caudal border of the femur. Because of the proximity of these vessels to the femur, they are at high risk of laceration in a fracture of the femur, and exsanguination could presumably occur in camelids.

Closure should include placement of sutures in each layer incised, especially in the fascia lata.

Femur fractures generally carry a good prognosis for healing when surgically repaired in camelids, but they are at increased risk of chronic, persistent lameness following healing compared to fractures in other long bones [7, 14, 23].

Tibia

Stall rest, transfixation pin casts, and dynamic compression plates have all been used successfully in the management of tibial fractures [3, 16, 17].

As with the radius, approach to the tibia will depend largely on the fracture configuration. The tibia is free of muscle covering throughout its length on the medial side of the limb. Incising the skin and fascial layer exposes the bone. The cranial tibial muscle lies on the cranial border of the tibia and can be reflected out of the way. The deep digital flexor muscle is firmly attached to the caudal border of the proximal two-thirds of the tibia. This attachment must be released to completely free the tibia. In the craniolateral approach, a curvilinear incision is made through the skin from the tibial crest to the lateral malleolus. The long digital extensor, peroneus longus, and cranial tibial muscles are retracted cranially

while the lateral digital extensor is retracted caudolaterally.

The major blood vessels and nerves to the lower hind limb lie in the fascia between the gastrocnemius muscle and the deep digital flexor muscle and thus are well out of the way.

Complications

Complications associated with fracture healing are common and have been reported in up to 23–84% of cases. Complications are varied and often depend on the class of fracture as well as the management method selected. Open fractures, in particular, are associated with complications in healing, are more likely to become infected, and have a poorer prognosis for return to function [14, 16]. Many complications are minor requiring little to no further treatment. Table 10.1 represents information collated from two retrospective studies with the percentage of animals experiencing various complications following limb fracture treatment [14, 16].

Some fractures are not amenable to repair due to the severity of trauma, chronicity of fracture and soft-tissue trauma, lack of financial resources, or previously failed repair attempt. In these cases, amputation may be performed. Many camelids tolerate amputation well. However, some are unable to adapt, and it is often challenging to identify these patients prior to the procedure. Limb prosthesis may be an option in these cases.

Table 10.1 Complications following fracture repair in SACs.

Complication	Percent of treated animals experiencing
Chronic lameness	32
Joint hyperextension	28
Delayed union, malunion, nonunion	18
Implant failure/loosening	14
Infection/osteomyelitis	14
Osteoarthritis	14
Decreased range of motion	12
Peripheral nerve injury	12
Muscle, tendon, ligament fibrosis	8
Abnormal cush	8
Peroneus tertius injury	6
Tendon/ligament laxity	4
Shortened limb	4
Angular limb deformity	4
Sequestrum formation	2

Vertebral Fractures

Etiology

Spinal injuries to the cervical region are more common than other spinal segments. The probable cause of trauma to the cervical vertebrae and associated ligaments is a blow to the neck or a struggle against restraint when first being haltered and tied. Lumbar vertebral fractures may be caused by another large animal rearing up and striking down on the back of the victim during inter-male aggression or dominance behavior. A large breeding male may injure a small female as he attempts to force her to recumbency.

Clinical Signs

Variable degrees of ataxia, incoordination, paresis, and paralysis have been observed secondary to trauma that involve the vertebral canal and produce compression on the spinal cord or emerging nerves. Fractures of the articular facets of the cervical vertebrae cause the head to be held in an abnormal position. There may be a palpable deformity at the fracture site and evidence of pain on digital pressure. Trauma to the intervertebral ligaments, intervertebral discs, and contiguous muscles produces signs indistinguishable from those caused by osseous lesions.

Diagnosis

Imaging such as radiography or computed tomography (CT) is required for definitive diagnosis. Figure 10.7 is a radiograph of an alpaca that has a fracture of the dens of C2.

Management

Vertebral fractures in large animals are difficult to manage. Conservative therapy consisting of stall rest and anti-inflammatories should only be elected in skeletally mature patients with the absence of neurologic signs [24]. Camelids

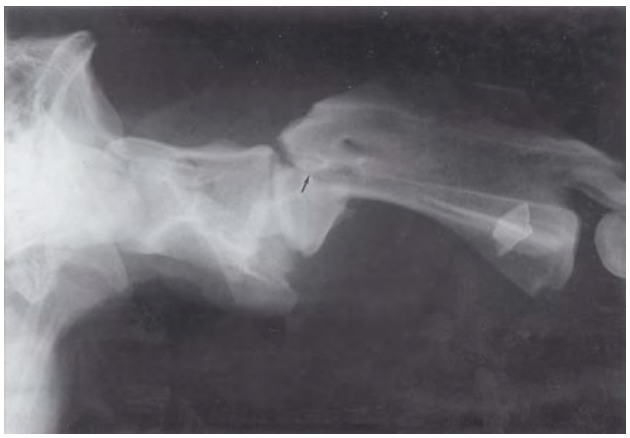

Figure 10.7 Radiograph of a fracture of the dens of C2.

do not tolerate neck casts well, and the cervical vertebrae of camelids have no flat surface suitable for plating. External fixation may be the best surgical option for these fractures, but secondary luxations may occur adjacent to the site of injury later in life [24].

Fractures in Camels

Fractures of the long bones of camels present a unique challenge, as their body weight is typically significantly greater than that of South American camelids. Despite this, many reports suggest that external coaptation using casts and splints alone have good results in both young and mature camels in fractures as proximal as the tibia and radius/ulna [25–29]. The high success rate of external coaptation in these species likely indicates a tolerance for these devices, as well as a willingness to remain recumbent for prolonged periods. These are similar attributes found in South American camelids and cattle, and are cited as contributors to successful management of fractures in these species [4, 7]. Open reduction and internal fixation have also been described [26, 30].

Mandibular Fractures

Mandibular fractures are the most common fracture encountered in adult camels [26, 28, 31]. These fractures most frequently occur in bull camels secondary to fighting behavior during the mating season or rut, during which the bulls either bite another camel or object. Their repair is well described in literature [25, 32, 33].

Anatomy

The adult hemimandible holds three incisors, one canine, a tusk-like premolar (PM1) 20 cm caudal to the canine tooth, and a second premolar (PM2) 20 cm caudal to PM1. PM2 is the first of four cheek teeth, with 3 M immediately caudal to PM2. The ramus of the mandible has three processes: the angular process, which is the most caudal and ventral in line with the occlusal surface of the cheek teeth, as well as a condylar process, and coronoid process [1].

Management

Mandibular fractures occur bilaterally at the level of PM1 or between PM1 and the canine teeth [25, 31–35]. Most of these fractures are best managed by the use of intraoral wiring (Figure 10.8a). A drill should be used to create holes through the gingiva between the first two cheek teeth. Stainless steel wire with a diameter of 0.8 mm is passed between PM2 and M1. One end of the wire is passed along the lingual aspect of the teeth and between the first incisors. The other end of the wire is passed along the buccal aspect of the teeth rostral to the incisors. The wire ends are then twisted together toward the roots of the incisors rostrally. The same procedure is then performed on the contralateral hemimandible [25, 28, 31–33, 35].

If the fracture is not transverse and/or lacks inherent stability, a U-bar can be used in combination with wires (Figure 10.8b). A 5 mm aluminum bar is bent to follow the contour of the mandible ending caudally at the last molar bilaterally. The bar is then placed along the buccal aspect of the teeth, and 0.8 mm stainless steel wire is passed around PM2 and twisted around the bar bilaterally to hold it in place. The fracture is reduced and the central and lateral incisors are wired to the bar rostrally [35, 36].

Patients should be fed a slurry for two weeks following the procedure, at which point, they may resume normal diet [25]. The fracture should have adequate stability by two to three months, and the wires may be removed at this point [25]. A good to excellent prognosis can be expected with rostral fractures treated with either wires or U-bars [25]. Common complications include small wounds or ulcerations at the points where the implants contact the gingiva [25]. Other methods to repair more complex

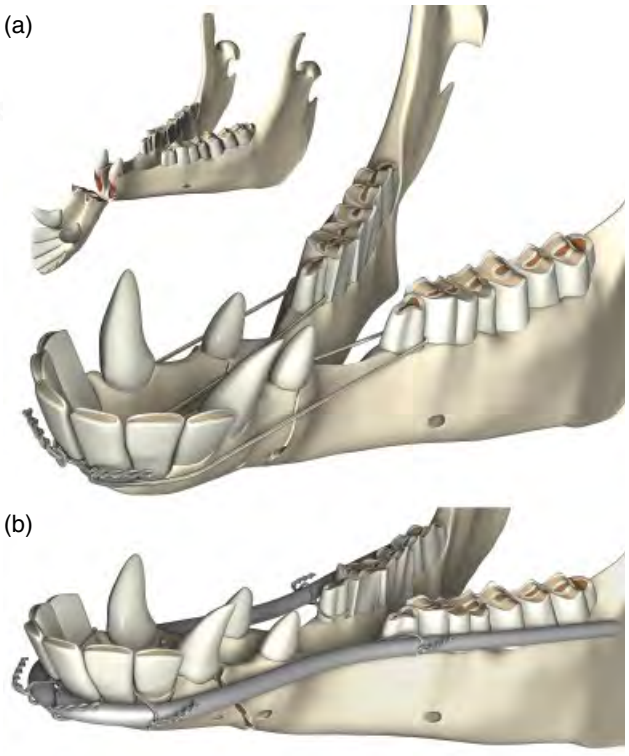

Figure 10.8 Diagram of the use of wire to stabilize a rostral mandibular fracture. (a) Orthopedic wire is passed between the caudal premolar and the first molar and then wrapped around the incisors to form a loop to stabilize the fracture. (b) A U-bar is used to stabilize the fracture. The bar is contoured to the lateral aspect of the mandible and is secured with wire around the cheek teeth caudally and the incisors rostrally.

mandibular fractures in camels are described, and include external fixation via pins and cast material, cortical screws, and dynamic compression plates [35, 37].

Disorders of Joints

Fetlock Hyperextension

Metacarpo/metatarsophalangeal (fetlock) hyperextension, frequently referred to as suspensory apparatus breakdown, is a condition that is fairly common in camelids. Clinical impressions are that it is found more in llamas than alpacas, though it can be seen in both. Normal phalangeal to ground angle in adult llamas is approximately 55° [38] (Figure 10.9). Affected animals can reach angles of less than 15°. (Figure 10.10), placing abnormal stress on the joints of the distal limb.

There are two main syndromes of fetlock hyperextension. The first is breakdown occurring in a singular limb. This can be initiated by abnormal stress from injury to the affected limb's suspensory apparatus [39] or, more commonly, from a severe injury to the contralateral limb causing prolonged excessive weightbearing in the hyperextended limb. Similar to support limb laminitis in horses, owners of camelids with significant injuries causing changes to weightbearing should be warned of potential contralateral limb breakdown. Attention should be paid to measures such as excellent fracture stabilization in the injured limb to increase weightbearing as soon as possible, as well as appropriate pain control to help prevent excess stress of the contralateral limb. The second fetlock hyperextension syndrome is idiopathic, affects multiple limbs in the same animal and is not related to a known injury or changes to weightbearing.

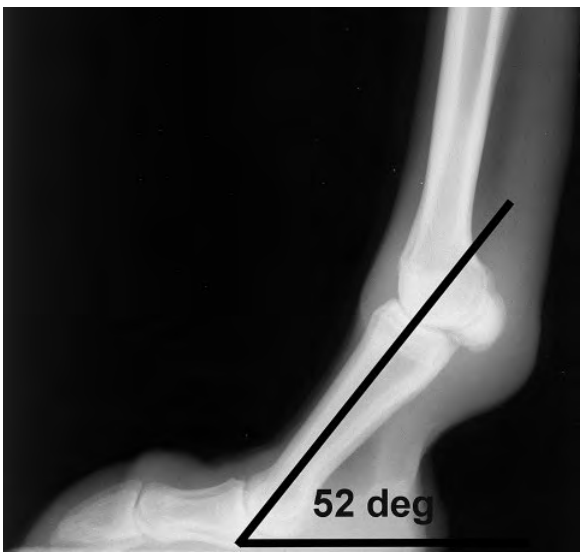

Figure 10.9 Radiograph of a llama fetlock with fetlock angle measuring approximately 52°. Normal fetlock angle (between P1 and the ground) in adult llamas is approximately 55°.

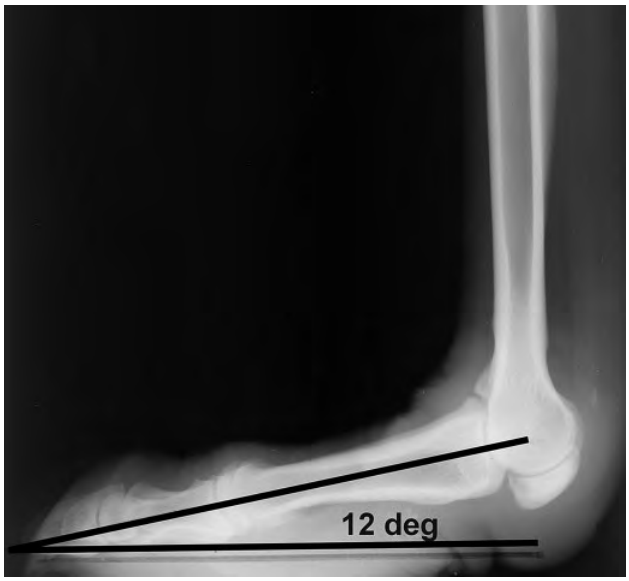

Figure 10.10 Radiograph of llama with severe hyperextension of the fetlock. The fetlock angle is approximately 12°.

Multi-limb fetlock hyperextension is not well understood. Llama owners will frequently express the belief of a heritable factor, but this information is anecdotal and has not been proven or published. Extensive examinations of groups of affected juvenile and adult llamas including ultrasound and radiographic examinations as well as morphologic, biochemical, molecular, histologic, and trace mineral characterization did not produce a concrete mechanism for this syndrome [40–42]. In these studies, hyperextension of the metacarpophalangeal or metatarsophalangeal joints in llamas did not appear to be caused by degeneration or inflammation of the suspensory ligament or flexor tendons. Though focal proteoglycan accumulation existed in the suspensory ligaments of two affected adult llamas, widespread abnormal connective tissue proteoglycan accumulation was not found [41]. Lower copper concentrations coupled with higher zinc concentrations in affected adult llamas may be indicative of secondary copper deficiency [40]. However, in the juvenile group of llamas, the same trace mineral imbalances were not seen, but rather increased serum molybdenum and decreased liver cobalt concentrations, compared with values for control llamas. Increased collagen type III gene expression and proteoglycan content in suspensory ligament samples of affected juvenile llamas provided evidence of ongoing suspensory ligament matrix repair [42].

Affected animals are likely to experience continued and worsening hyperextension. Many of these animals remain pasture sound for an extended period of time, though eventually osteoarthritis of the distal joints as well as mineralization of the soft tissues can affect comfort levels enough to necessitate euthanasia. Companies that market support braces for dogs and cats are frequently willing to modify their systems for camelids, but these supports need to be instituted before the angulation becomes severe enough that the system cannot be fitted to the animal. Additionally, because of the pressure placed on points of the limb, they are frequently impractical to wear constantly. Animals with only one limb affected may be candidates for fetlock arthrodesis [43–45]. In these situations, loss of palmar/plantar support can make it difficult to maintain hardware through healing in larger animals (>150 kg) and implant failure may be more likely. Animals with osteoarthritis may benefit from treatment with polysulfated glycosaminoglycan [46] (1–2 mg/kg IM Q4 days for seven doses then once monthly and/or meloxicam [47] (1 mg/kg PO Q3 days), neither of which are labeled for use in food animals though it may be applicable for minor species such as camelids. Veterinarians should review the diets and supplements of affected animals to ensure mineral imbalances are not influencing tendon cross linking. One source of excess dietary zinc is over supplementation to treat suspected zinc responsive dermatosis. Herds with more than one affected animal should be scrutinized for both dietary and genetic factors that may explain the condition.

Scapulohumeral Luxation

Anatomy

The shoulder joint in camelids is similar to those of other species [1]. The joint is a simple spheroidal joint made up of the glenoid cavity of the scapula and the head of the humerus, and movement is largely restricted to flexion and extension [1, 48]. The infraspinatus muscle inserts on the caudal part of the major tubercle of the humerus and lacks a bursa. The tendon of the infraspinatus muscle, along with the fused teres minor muscle, provides lateral stability to the joint [1]. The deltoideus arises from the scapular spine and acromion and inserts on and near to the deltoid tuberosity [1]. The supraspinous muscle has a lateral and cranial part. The lateral originates at the supraspinous fossa and inserts on the cranial aspect of the major tubercle while the cranial part originates at the proximal border of the scapula and inserts on the minor tubercle [1]. Medially, the joint is stabilized by the teres major, subscapularis, coracobrachialis, and articularis humeri muscles [1].

Etiology

Though rare in most large animal species, scapulohumeral luxations are relatively common in male South American camelids due to fighting behavior [46, 49–52]. Lateral luxations are most common, though medial luxations are occasionally diagnosed [46].

Diagnosis

Diagnosis of a scapulohumeral luxation can be challenging. The patient typically presents with severe, nonweightbearing to toe-touching lameness, often without swelling, and variable muscle atrophy [46, 53]. A painful response is often elicited upon manipulation of the shoulder joint. To rule out a fracture of the humerus, one hand may be placed on the point of the elbow, and one is placed on the proximal end of the humerus. Internal and external rotation is applied. If there is crepitus with a lack of proximal humerus movement, a fracture should be suspected. If humeral head movement is detected, luxation is more likely. The most important tool for diagnosis of scapulohumeral luxation is radiology. Radiographs may be difficult to interpret and must be carefully examined for congruity of the humeral head and glenoid cavity of the scapula (Figure 10.11).

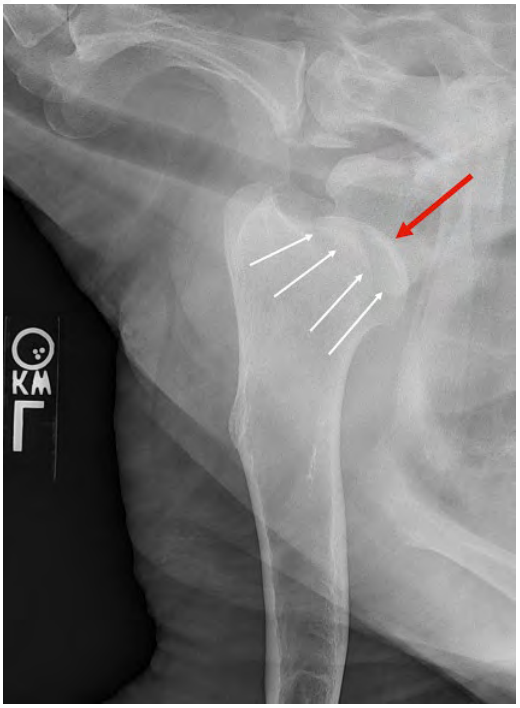

Figure 10.11 Medial to lateral projection of a luxated scapulohumeral joint of an alpaca with a lateral shoulder luxation. The red arrow points to the humeral head, while the white arrows point to the glenoid cavity, highlighting the lack of congruency of the joint.

Treatment

Closed Reduction

Closed reduction is performed by placing the animal in lateral recumbency under sedation or general anesthesia with the affected limb uppermost. Traction is applied caudodistally on the distal limb with countertraction in the pectoral region. Manual pressure is applied to the humeral head in a caudal direction. Alternatively, traction can be applied distally and cranially with pressure on the humeral head upon slow release. If successfully reduced, a Velpeau sling should be utilized for three to four weeks, and the patient should be stall rested for three months.

To apply a Velpeau sling, the limb is first flexed at the carpus and padded from the foot to the carpus. Following this, the limb is held in flexion and cotton padding is wrapped around the torso. Layers of gauze and elastic tape are used to hold the limb in flexion. If the shoulder was laterally luxated, as is most common, the bandage material is applied from the ventral aspect of the thorax/limb to the lateral aspect of the affected limb. The wrap is continued to the dorsum of the animal and around the contralateral thorax and around again. This medial to lateral wrap applies proximal and medial force on the joint, keeping the shoulder reduced. If, however, the limb was medially luxated, the limb should be wrapped in the opposite manner, from lateral to medial. This applies lateral force on the joint, opposing the forces that would tend to reluxate the shoulder.

Initially, after removal of the sling, the limb will be contracted and stiff. With physical therapy consisting of controlled walks, function of the limb will return. Recurrence with closed reduction is common in males [46]. Frequently, this is secondary to continued fighting behavior, and males should always be housed apart from other males to avoid this complication.

If closed reduction fails, open reduction may be pursued. In the absence of radiographic evidence of osteoarthritis, open reduction with shoulder stabilization can be performed. In the presence of severe osteoarthritis or failed shoulder stabilization, a shoulder arthrodesis may be the best option to decrease pain in these patients.

Open Reduction and Stabilization A curvilinear incision is made laterally from mid-scapula to the level of the deltoid tuberosity over the scapular spine, across the joint, and along the lateral humerus. The aponeurosis of the deltoideus is incised to access the infraspinatus tendon. A tenotomy is performed providing access to the joint capsule, which is incised parallel to the rim of the glenoid cavity. The joint is debrided as necessary and the luxation reduced. A screw and washer is then placed in the neck of the scapula, and a second in the greater tubercle

Figure 10.12 Postoperative radiograph of an alpaca following open reduction and stabilization of the scapulohumeral joint to treat a lateral shoulder luxation. Screws and washers are placed in the neck of the scapula just proximal to the glenoid and the greater tubercle of the proximal humerus to allow placement of a tension band to provide stabilization of the shoulder joint.

(Figure 10.12). Number 5 USP-braided non-absorbable suture is used as a figure-of-eight tension band around the screws. Joint capsule, fascial layers, subcutaneous tissue, and skin are closed routinely. The limb should be placed in a Velpeau sling prior to anesthetic recovery and maintained for at least 10 days, and the patient should be stall rested for 30–60 days following the procedure. Provided there is minimal joint damage prior to the surgical procedure, excellent prognoses can be expected with this procedure [46, 52, 53].

Scapulohumeral Arthrodesis Arthrodesis of the scapulohumeral joint of camelids is similar to what is described in the horse [54]. With the patient in lateral recumbency, a craniolateral curvilinear approach to the joint is made with an incision from mid-scapula to mid-humerus. A tenotomy of the supraspinatus muscle is performed close to its insertion. The articular cartilage is debrided from the joint, and the intermediate tubercle is removed with an oscillating saw or rongeurs to provide a flat surface for the plate. An appropriately sized plate is contoured to the cranial scapula and proximal humerus at roughly 120°. At least one screw should be inserted in lag fashion across the joint through the plate from the humerus through the glenoid. The rest of the plate holes are filled

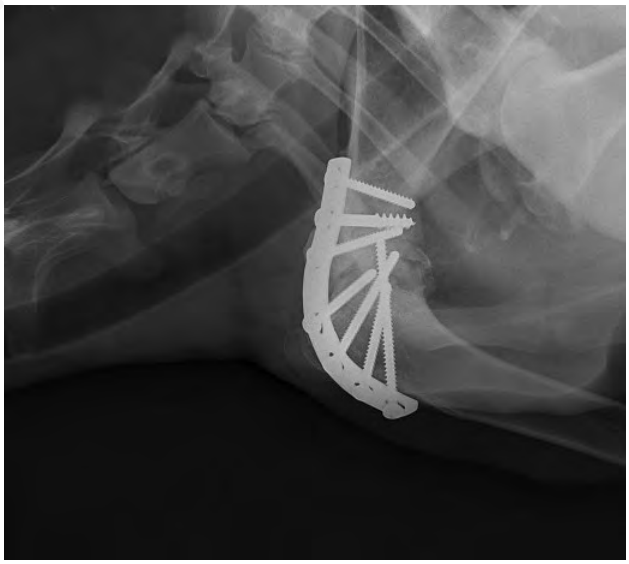

Figure 10.13 Lateral radiograph of an alpaca six-months following scapulohumeral joint arthrodesis.

with screws and closure is routine (Figure 10.13). A good prognosis can be expected, but a functional lameness will remain [46].

Disorders of the Stifle

Anatomy

The camelid stifle joint is made up of a medial and lateral femorotibial articulation as well as a femoropatellar articulation [1, 55]. All three of these articulations share a joint capsule and communicate freely [56]. There are two menisci, medial and lateral. The lateral meniscus is attached to the femur caudally via the meniscofemoral ligament, and cranially with meniscotibial ligament. The medial meniscus has a caudal and cranial meniscotibial ligament, as well as an intermensical ligament, and an attachment to the medial collateral ligament [1, 55]. Ligaments of the femorotibial articulation include the medial collateral ligament and the cranial and caudal cruciate ligaments [1]. The camelid lacks a lateral collateral ligament but has a substantial lateral retinaculum which is continuous with the patellar ligament and the gluteobiceps insertion on the patella and lateral aspect of the tibial tuberosity [1, 55]. The medial patellar retinaculum runs between the medial epicondyle and the patella. The medial and lateral patellar retinaculum are considered extensions of the single patellar ligament [1].

Cranial Cruciate Ligament Rupture

Etiology and Clinical Signs

The cranial cruciate ligament arises from the cranial intercondylar tibia and inserts on the lateral intercondylar area of the femur [1]. Rupture of the ligament occurs sporadically secondary to trauma or mounting behavior in males, and usually results in acute, severe lameness of the affected limb [57–60].

Diagnosis

Physical exam should localize the lameness to the stifle. Effusion is often present on palpation of the limb, and manipulation of the joint may reveal tibial thrust and/or cranial drawer [58–60]. Radiographs are often needed to confirm the diagnosis. Normally, the intercondylar eminences should be overlapped by the femoral condyles on a lateral to medial projection. In the case of cranial cruciate ligament disruption, the eminences are seen radiographically cranial to the condyles on a lateral to medial projection (Figure 10.14) and widening of the medial aspect of the femorotibial articulation may be appreciated [57–59]. Osseous fragments representing avulsion fractures of the tibial insertion may also be observed [58].

Treatment

In the case of incomplete rupture or minimal lameness, stall rest and anti-inflammatories may be utilized. The patient should be allowed at least six months of rest in a confined area with good footing.

Figure 10.14 A lateral to medial projection of an alpaca with a ruptured cranial cruciate ligament of the left stifle. The intercondylar eminences (white arrow) of the tibia can be seen cranial to the femoral condyles (red arrows), between which they should normally lie.

In cases of complete rupture and joint instability, surgical stabilization of the joint is necessary. Stifle imbrication has been utilized with fair results in our clinic. This technique is described in cattle [61], and relies on the formation of periarticular fibrosis for joint stabilization. This technique is more successful in smaller cattle [62, 63], and may be used in most SAC's. The patient is placed in dorsal recumbency and the affected limb is elevated. An s-shaped incision is created beginning 3 cm proximal to the patella, moving first medially, and ending 3 cm distal to the tibial tuberosity. The skin flaps are elevated, and dissection is continued through the subcutaneous tissue to the level of the periarticular fascia. An arthrocentesis should be performed to reduce the outward pressure of the joint on the periarticular tissue and to maximize imbrication. The limb is extended, and Lembert sutures are placed in the fascial tissue on either side of the patella and patellar ligament oriented in a caudoproximal to craniodistal direction. The imbrication suture pattern is similar to that described for treatment of patellar luxation. After one row is placed on either side, the joint laxity can be assessed, and more rows placed as required until instability is no longer elicited. The surgical incision is closed routinely. Immediately post-operatively, the patient will be equally or more lame than immediately pre-operatively, with a stiff gait. The patient should be stall rested for four to six months with steadily increasing activity. Lameness will continue to improve during this period, but improvement beyond six months is unlikely.

Ligament grafting is described and may be considered in larger patients such as adult llamas, as imbrication may be less effective [57, 58]. Tibial plateau leveling osteotomy is reported in two camelids with good results [59, 60], though supporting literature on plateau angle, anatomic and joint mechanic differences, and long-term outcomes for this technique are lacking.

Luxating Patella

Etiology and Clinical Signs Both medial and lateral congenital patellar luxations have been diagnosed in camelid crias. Congenital bilateral patellar luxation causes the cria to assume a crouched position (Figure 10.15), making ambulation difficult, and patellar luxation is evident upon palpation.

Acquired patellar luxation can be medial or lateral, but lateral is most common [55]. Traumatic luxation follows an incident that ruptures the medial or lateral femoropatellar ligament. These are often intermittent luxations, but while luxated, the animal typically ambulates with the limb locked in extension and outwardly rotated.

Figure 10.15 Bilateral medial patellar luxation in a llama neonate. Cria has a crouched stance characteristic of affected crias' inability to extend their stifles.

Table 10.2 Grades of patellar luxation with their respective description.

Grade	Description
I	Patella can be manually luxated, but reduces spontaneously
II	Patella luxates on manipulation of the joint, but can be reduced manually
III	Patella is luxated, can be reduced, but reluxates spontaneously
IV	Patella is non-reducibly luxated

Diagnosis Diagnosis is usually made via palpation of the patella in an abnormal position, but radiographs may be used to confirm the diagnosis or to detect additional osseous damage. The various compartments of the stifle joint may be distended, depending on the severity and chronicity of the trauma. Patellar luxations are graded on a four-point scale described in Table 10.2.

Treatment Treatment modality depends on the grade of luxation as well as evaluation of the stifle anatomy including the trochlear groove and the alignment of the tibial tuberosity. Low-grade luxations may be treated by retinacular release and imbrication. A curvilinear incision is made beginning close to the central axis of the limb, running just abaxial to the patella, and back toward the axial aspect of the limb. The fascia, retinaculum, and joint capsule on the luxated side just abaxial to the patellar ligament are incised. The release incision should be long

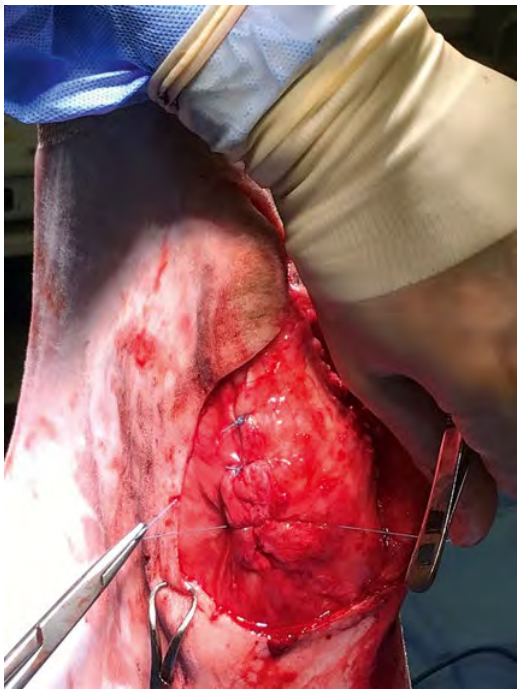

Figure 10.16 Imbricating sutures being placed alongside the patella and patellar ligament in a Lembert pattern.

enough (20 cm in the mature llama) to alleviate tension and prevent recurrence. Imbricating sutures are placed in the retinacular tissue on the opposite side of the joint in a Lembert pattern with braided, non-absorbable material alongside the patellar ligament (Figure 10.16). If trochlear groove hypoplasia exists, a trochlear block recession should be performed, deepening the groove [64]. Trochlear block recession may also be beneficial in cases of grade III or IV patellar luxation to prevent recurrence [55, 65]. Tibial tuberosity transposition should be considered in cases where the extensor mechanism is not in alignment. If tibial tuberosity transposition is deemed necessary, it should be noted that a high failure rate is reported when the tibial tuberosity is stabilized with pins or screws in combination with a tension band. For this reason, reinforcement with a plate should be considered [64, 66].

Upward Fixation of the Patella

Upward fixation of the patella is an acquired condition in older llamas. A predisposition may be a congenital conformational weakness (straight rear limbs and laxity of the tendons and ligaments). The mechanisms for upward fixation in a camelid is different from that in a horse. Camelids have a broad patellar ligament rather than the three ligaments of the horse. Fixation in the camelid likely occurs when the patellar ligament is stretched or the bones of the rear limb are overly straight. This is accompanied by a stretching or rupture of the medial or lateral femoropatellar ligament, which allows the patella to move medially or laterally to catch the dorsal aspect of the ridges of the trochlea. Camelids tend to have minimal curvature to the proximal bones of the pelvic limb, but this may be exaggerated to produce a straight or post leg, which causes the patella to ride high on the trochlea. Trauma to the patellar ligament may cause weakening and stretching, which, in turn, allows the patella to ride higher.

If the patella is locked dorsally, the limb will be fixed in extension, and if forced to move, the llama must drag the limb. With intermittent upward fixation, the signs may vary from an audible click as the patella momentarily locks during ambulation to a lock prolonged for a few seconds, followed by an exaggerated flexion of the limb as the lock is released. Lameness may be evident during periods of quiescence, depending on the cause of the upward riding of the patella.

Diagnosis Clinical signs and palpation are the primary means of diagnosis. Radiographs may be used for confirmation.

Treatment Upward fixation may be released by extending the limb fully while manipulating the patella. Mild upward fixation may be controlled by restricting activity to permit a stretched patellar ligament to heal. Surgical imbrication may be required in severe cases.

Tibiotarsal Luxation

Bilateral luxation of the tibiotarsal joint has been diagnosed in a number of SACs, including a seven-month-old female llama [67]. This condition in SACs may be congenital or acquired via trauma [67].

In a SAC suffering from luxation, the tibiotarsal bone lies in a horizontal rather than a vertical position, and the articular grooves of the tibia articulate with the caudal aspect of the tibiotarsal bone rather than the trochlea (Figure 10.17). Externally, the cranial surface of the tarsus bulges where the tibiotarsal bone projects forward. Movement of the tarsus is restricted, causing a mechanical lameness.

The condition in llamas is similar to congenital convex pes valgus (congenital vertical talus, congenital flat foot, teratologic dislocation of the talonavicular joint) in human infants [68]. The etiology in human infants is unknown, but it is thought to be genetic. Peculiar fetal positioning within the uterus has also been considered. Surgical realignment and pinning of the tibiotarsus may correct the defect in human infants but has not been performed in llamas.

Figure 10.17 Lateral radiograph of tibiotarsal luxation. Note that the tibiotarsal bone lies in a horizontal position, and the tibia is articulating with the caudal aspect of the tibiotarsal bone.

Figure 10.18 A dorso-plantar radiographic projection showing sequestrum in the metatarsus of an alpaca.

Bone Sequestra

Bone sequestration may be found in the appendicular skeleton and in the mandible. Unlike most cases of bone sequestration in other species, cases seen in SACs often have no evidence of trauma [69, 70]. Hematogenous infection is thought to be a factor [71].

Clinical Signs

Unexplained lameness is the major presenting complaint. The lameness may be mild or non-weightbearing. A firm swelling of a bone may be palpated later in the course of the condition. Occasionally, a fistulous tract to the bone may be seen.

Diagnosis

Radiographs of the affected limb reveals a sequestrum within an involucrum (Figure 10.18). It is important to have multiple views, as clear demarcation of the separated segment may be difficult to see, especially in the early disease. Radiographic evaluation is also necessary to ensure the removal of the bone segment will not compromise the stability of the bone. If this is threatened, the surgeon should be prepared to apply stabilization.

Surgery

The method for removal of the sequestrum depends on its location, size, and presence of contiguous periosteal and new cortical bone formation. The approach should be through and adjacent to the existing cloaca. Only enough bone should be removed so as to remove the sequestrum without disrupting the supporting new bone. Light curettage of the involucrum is used to eliminate diseased bone and the lining of the involucrum. It may be necessary to leave a drain in the wound. Broad-spectrum antibiotics are appropriate for 7–10 days. Recurrence, persistent infection, abnormal appearance of the limb, and neurologic deficits are all possible complications following treatment and were seen in roughly 20% of treated patients in one study [69].

Juvenile/Congenital Limb deformity

There are multiple limb deformities seen in camelids. The most common is angular limb deformity (ALD) which represents limb deviation in the frontal plane. A distal lateral deviation is termed valgus deformation and a distal medial deviation is termed varus [72]. The most commonly reported ALDs in camelids are carpal valgus followed by metacarpophalangeal valgus [73]. Bilateral ALD is generally an

acquired deformity from disproportionate bone growth. A historically common reason for this in camelids was hypophosphatemic rickets syndrome [74]. The incidence of rickets has decreased since producers have become more aware of the susceptibility of camelids to seasonal vitamin D deficiency. However, it can still be seen in growing camelids between three and six months of age and should be considered by clinicians. It is more common in animals born in the fall and winter [75]. Though the presenting complaint can be angular limb deformities, these animals are frequently affected in multiple areas resulting in multiple other clinical signs including enlarged joints, reluctance to move, humpbacked stance, and shifting limb lameness. Radiographically, the physes will be widened and irregular [74]. Other types of limb deformities that can be observed in camelids are rotational deformities, laxity of periarticular structures creating an observed angular change that does not originate in osseous structures (Figure 10.19), and flexural limb deformities [76] (Figure 10.20).

Figure 10.19 A juvenile llama cria with ligamentous laxity that could be straightened by manipulation to normal confirmation. No radiographic abnormalities were appreciated in this animal, and the limb angle discrepancy is due to soft-tissue laxity rather than osseous changes.

Figure 10.20 A juvenile alpaca cria with flexural contracture in the hind limbs and laxity in the forelimbs. The hindlimbs cannot be straightened by manipulation due to severe contracture. The forelimbs can be straightened and supported by manipulation into a normal orientation. No osseous abnormalities are appreciated on radiographs.

Diagnostic workup for animals with limb deformities should begin with a complete amnesis including dietary questions, with emphasis on energy density and vitamin and mineral supplementation. The next step is a thorough physical examination with evaluation of gait and lameness, assessment of mobility of joints evaluating the soft-tissue structures (assess whether the limbs can be straightened by hand) and note appearance of joint angles [38]. Animals with the visible appearance of angulation that can be manually straightened by pressure on one side of the limb are likely suffering from periarticular or soft-tissue laxity and are not candidates for surgical physeal intervention. Radiographs of the affected limb(s) are necessary. Radiographs should be taken on large cassettes, centered on the joint of angulation, and taken at a right angle relative to the frontal plane in the forelimb and to the sagittal plane in the hind limb. If possible, the bone above and below the affected joint should be aligned in one plane. The pivot point is established by the bisection of lines drawn through axis of the long bone on either side of the affected joint [72]. Establishing the pivot point will help determine if the deviation originates in the physis or the joint. A pivot point centered over the joint, without any evidence of osseous abnormalities, raises suspicion of periarticular laxity as the cause of angulation. The pivot point is also the focus for intervention. The angle formed by the bisecting lines is termed the pivot angle, which indicates the degree of limb deviation. Animals with rotational deformities may benefit from computed tomography (CT) and a comparison between affected and contralateral limb to better determine the angles and origins of the rotation.

Treatment

The determination to treat ALD is based on the severity of the angulation and the age of the animal. Animals with mild ALD (5–8° angulation) likely do not need intervention, though breeding those animals in the future should be discouraged. Alpacas with their smaller adult weight may have more tolerance for mild ALD than larger llamas, particularly those bred to be pack animals. Historically, techniques to expedite growth on the concave side of the physis (periosteal stripping) were used. The efficacy of those techniques has been questioned and the technique is not currently recommended. If a neonate (less than four weeks) is diagnosed with extreme angulation associated with lax ligaments, physical therapy and the use of articulating supporting orthotics/braces for the joint modified from small animal prototypes may help to straighten the limb in the early, rapid growth period. Weekly rechecks are recommended with this therapy to ensure the laxity isn't worsening and that the limbs are not incurring skin damage from inadvertent focal pressure.

In cases of severe angulation, surgical intervention may be necessary. The type of intervention will depend on the potential for additional physeal oriented growth and the complexity of the angular limb deviation. For young animals with angular deformities without rotation, surgery to retard growth on the convex physis of the affected bone (ex: medial physis for carpal valgus) is performed to provide static compression. For this to be effective, the affected physis must be open with active growth potential. Growth retardation is accomplished by spanning the physis with either figure 8 wires/Fiberwire suture connected to proximal and distal screws on either side of the physis, a single transphyseal screw, or a bone plate. Transphyseal screws are frequently used in equine patients [72], but have not yet been reported in peer-reviewed camelid literature. The principles are similar, should apply across species, and have been anecdotally reported for singular cases. Growth retardation techniques require that patients be monitored carefully, and the implants removed as limbs approach (for the transphyeal screw) or reach (for the screws and wires and plates) straightness. Not removing the implants can cause overcorrection, particularly in young fast-growing animals. Older animals without available physeal growth and animals with rotation and angular deformities will need corrective osteotomy/ostectomy [77–81]. Those animals with carpal valgus will likely also need an ulnar ostectomy [73]. Correction of the limb deviation should be aimed at improving animal comfort; affected animals should not be used in breeding programs as the deformity is potentially heritable.

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11

Respiratory System

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Overview of the Camelid Respiratory System

Nostrils and Nasal Cavity

Both camels and South American camelids (SACs) possess a highly mobile philtrum which allows them preferential selection of feedstuffs like sheep and goats [1]. Unlike camels, SACs are unable to completely close the nostrils to exclude dirt or dust (Figure 11.1). The nasal cavity has a ventral, middle, and dorsal meatus (Figure 11.1c). Conchae (turbinates) are arranged in a pattern similar to those of cattle and sheep. The ventral aspect of the cranial ventral meatus is very narrow. In addition, a bulge in the ventral septal mucosa partially occludes the lumen of the ventral meatus (Figure 11.1c). The dorsal section of the ventral meatus is also very narrow. The relatively large surface area and the narrowness of the spaces between nasal turbinates in desert camels (and potentially SAC), plays an important role in the conservation of water and reduction of brain temperature. The high surface area and nasal secretions of the nasal cavity help to reduce the temperature and moisten the warm dry air. During exhalation, water-saturated air (100% relative humidity) passes over the nasal turbinates, which are relatively dry during the warmer periods of the day. The moisture collected from exhaled air over the narrow channels collects on the mucosa. This results in significant conservation of body water [2]. Regulation of body temperature while conserving water during exhalation of air allows camels a way to maintain brain temperature and water resources in hot, arid environments.

The turbinates are very delicate and easily traumatized, so passage of a nasogastric tube or endoscope should be performed carefully. In most adult SACs, passage of the tube in the most ventral and medial aspect of the ventral meatus can be productive. In this author's experience, the use of a 137 cm long, 6.6 mm diameter urinary catheter in adult alpacas is possible. Alternatively, an orogastric tube can be used with a mouth speculum. Smaller endoscopes (<5 mm) may also be inserted carefully to visualize the nasopharynx, proximal trachea and esophagus. Young crias can be managed more safely by passage of an orogastric tube.

Nasolacrimal Duct

The nasal punctum of the nasolacrimal duct is commonly located less than 1 cm dorsal from the floor of the nostril near the mucocutaneous junction (Figure 11.2). However, it is not uncommon for SACs to possess nasal punctum more proximally in the nasal passage, or present with atresia of the nasal punctum, inferior or superior puncta. Congenital lesions of the nasolacrimal duct system is not uncommon in SACs [3]

The hard palate ends approximately 6 cm caudal to the leading edge of the first maxillary premolar. The soft palate is exceptionally long in camelids. The caudal edge of the soft palate should reside ventral to the epiglottis (Figures 11.3a). In stressed camelids, dorsal displacement of the soft palate often results in open mouth breathing

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Figure 11.1 (a) Left nostril of a Dromedary camel showing narrowness of the external nares. (b) Normal alpaca with relaxed appearance and open nostrils. (c) CT slice caudal to the incisor teeth showing dorsal, middle and ventral conchae, the nasal septum and vomer bone. The mucosal surface of the ventral surface extends approximately 2 mm into lumen.

Figure 11.2 Anesthetized alpaca in right lateral recumbency for passage of a 3.5 Fr polyethylene catheter into the left nasolacrimal duct. Notice the additional opening just rostral to the catheter (blind ended). The catheter can be advanced 1–2 cm proximally into the nasolacrimal duct and the duct opening occluded by finger pressure to irrigate the ipsilateral nasolacrimal sac and the inferior and superior nasolacrimal puncta (*Source:* Photo courtesy of Dr. AJ Niehaus).

providing the appearance of severe respiratory dysfunction (tachypnea, dyspnea) as they are obligate nasal breathers (Figure 11.3b). While SACs may develop clinical pneumonia, stressful procedures such as restraint, (restraint for intravenous (IV) catheter placement, obtaining blood for DNA analysis [parentage and registration], passage of an orogastric tube, ultrasound evaluation, etc.) may cause dorsal displacement of the soft palate. While auscultation of the thorax may be indicated, the examiner should consider altering their original plan by allowing the animal to recover in a quiet area by itself or consider other less stressful approaches [4].

Sinuses

The maxillary and frontal sinuses are delineated in Figure 11.4a. Imaging the skull and sinuses is sometimes required to evaluate trauma, facial deformity, draining of mucoid/muco-purulent discharge from lesions of the face or skull. Figure 11.4b depicts a single CT slice from the skull of a 12-year-old female alpaca. This animal had delivered a healthy cria approximately one month prior to imaging. The owners reported this animal has had a draining wound below the medial canthus of her left eye for six months during pregnancy. It is evident that the frontal sinus is not involved on either side, whereas the maxillary sinus on the left side is filled with a soft-tissue opacity with evidence of boney destruction in this area. There is a soft-tissue opacity below and medial to the orbit on the left side. After trephining the maxillary sinus, irrigation with physiologic saline promoted clearance of the fluid in the maxillary region. Figure 11.4c depicts a similar case in a nine-year-old male alpaca with both frontal and maxillary sinus involvement. The animal was in poor condition and was humanely euthanized after CT examination. Postmortem evaluation revealed an invasive squamous cell carcinoma (SCC).

Lungs, Trachea and Bronchi

The anatomy of the SAC respiratory system remains incompletely described in the literature [5]. The anatomy of SAC lungs share some similarity to that found in the horse [6]. However, detailed evaluation of the airway branching patterns of Old-world camelid (OWC) and SAC lungs demonstrate similarity to ruminant lungs [7, 8]. A cardiac notch separates the apical portion, but there are no additional individual lobes except for the small accessory

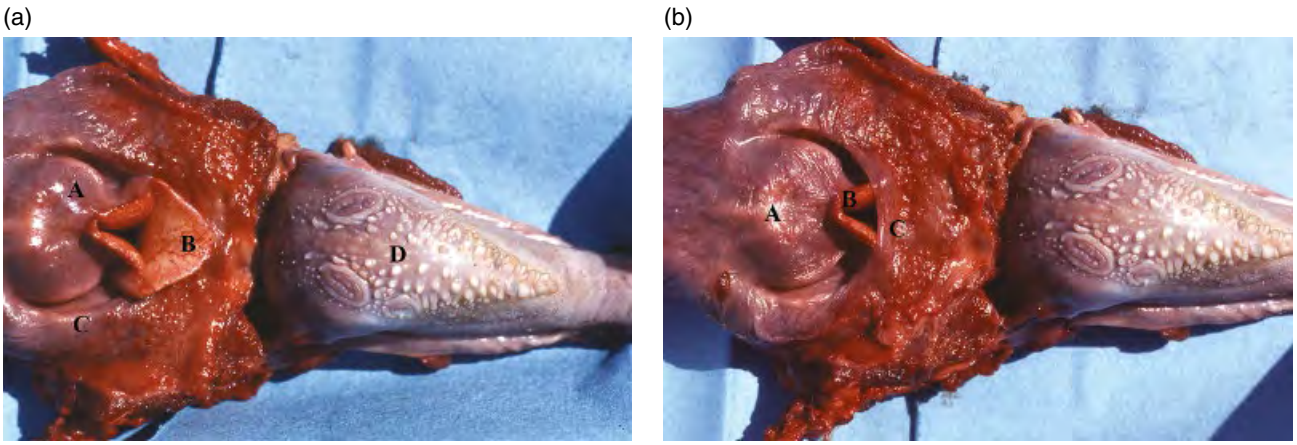


Figure 11.3 (a) Soft palate situated ventral to the epiglottis. (A) Larynx, (B) epiglottis, (C) aryepiglottic fold, (D) base of the tongue. (b) Simulated dorsal displacement of the soft palate in a cadaver. Anatomic dissection of the relationship of the soft palate dorsal to the epiglottis. (A) Larynx, (B) arytenoid cartilage, (C) caudal margin of the soft palate.

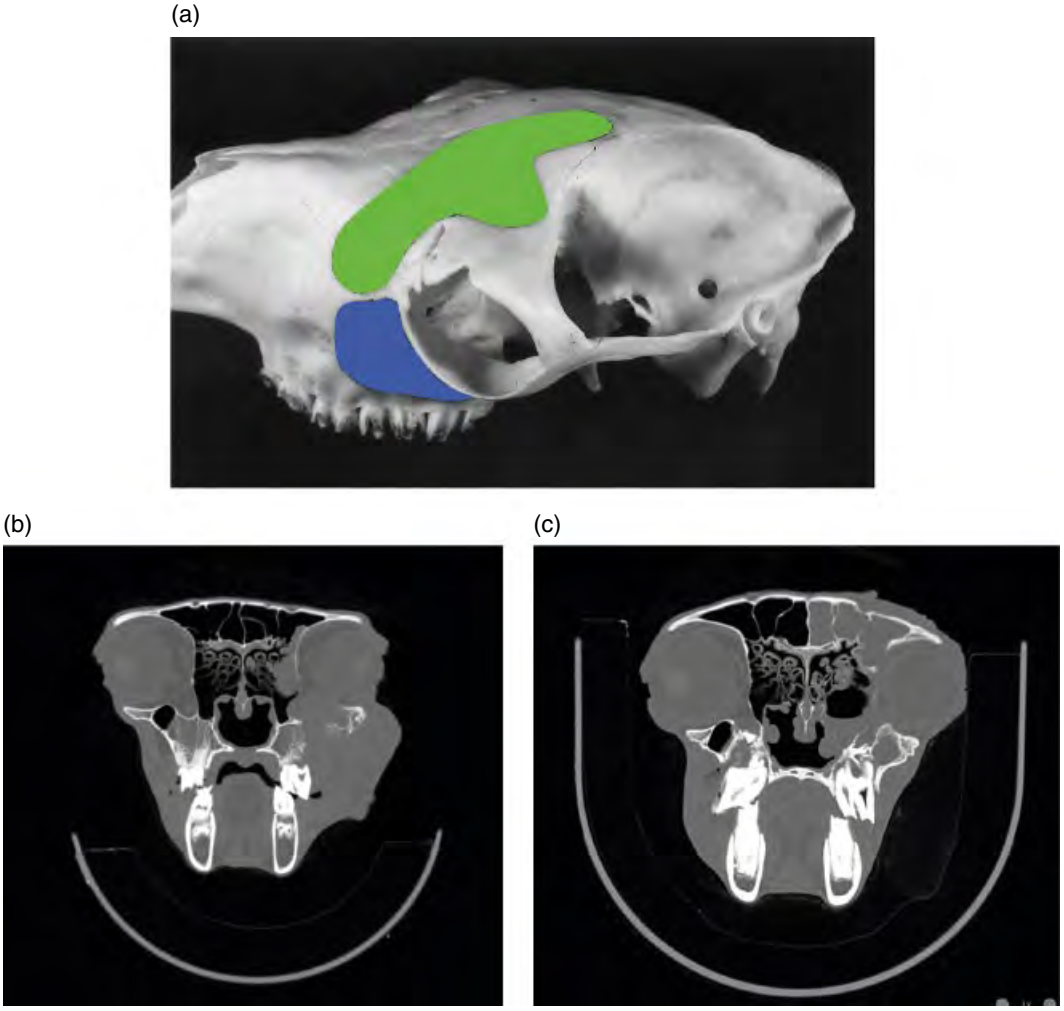


Figure 11.4 (a) Skull illustrating the outlines of the maxillary (blue) and frontal (green) sinuses, (b) CT slice from the skull of a 12-year-old female alpaca with draining tract ventral to left eye. Left maxillary sinus is filled with soft-tissue opacity and the bone in this area is lytic and irregular. (c) CT image of the skull of a nine-year-old male alpaca with both frontal and maxillary sinus involvement. Post-mortem evaluation revealed an invasive squamous cell carcinoma.

lobe on the right lung that surrounds the caudal vena cava. There is a tracheal bronchus (right) that originates from the trachea just cranial to the bifurcation of the lobar bronchi. Each main stem bronchus divides into a cranial bronchus and three caudal bronchi bilaterally with termination of the lobar bronchus caudally. In alpacas, the tracheal bifurcation occurs between the fourth and fifth thoracic vertebrae [8].

Cooley et al. [8] also demonstrated that the mediastinal portion of the caudal lung lobes extend further caudally than the lateral aspects of the caudal lung lobes in alpacas [8]. This peculiarity may explain clinical difficulties in auscultation of the caudal lung lobes due to enhanced gastrointestinal (GI) sounds at these locations [8]. As in small ruminants, Cooley et al.'s [8] findings also suggest the cranial portion of the right lung may be auscultable at the thoracic inlet as it appears, a portion of the right cranial lung lobe extends cranial to the heart [9].

Quantitative evaluation of lung anatomy, including airway branching patterns and fine structure of the airways in Camels and SACs have been conducted [7, 8]. These studies demonstrated bronchus associated lymphoid tissue (BALT) in the camel as well as the distribution of various airway epithelial cells (ciliated, goblet, subepithelial glands, basal, Clara, squamous epithelium) from trachea to alveolar ducts [10] The Clara cell is a nonciliated epithelial cell that occurs in the bronchiolar region of the lung. It appears to be a secretory cell, but its function in camelids is unknown. The Clara cells of other species function as a non-mucinous secretory cell and possess xenobiotic metabolizing enzymes and appear to function as progenitor cells to maintain the epithelial lining layer after injury [11]. Interestingly, Clara cell numbers per millimeter of bronchiolar epithelium are markedly different between llamas kept at high elevations (55–106 Clara cells/mm) and those kept at sea level (8 Clara cells/mm). Presumably, the high elevation environments result in injury to the lung epithelia. Proliferation of these secretory cells may be necessary to maintain lung function in animals living at high altitude [12, 13].

While difficult to appreciate at the light microscopic level, one study suggests camel lung alveoli possess pores of Kohn. This observation suggests that inflation of other alveoli via the pores of Kohn (collateral ventilation) can stabilize lung inflation via recruitment [14]. Collateral ventilation may also stabilize regional ventilation/perfusion ratios. This separates camelid lung design from bovine lung parenchyma which do not possess these openings [15].

The mediastinum of camelids is believed to be complete. The line of diaphragmatic pleural reflection follows a line approximately 23 cm cranial to the costochondral junction and crossing the midportion of the 12th (last) rib to finish dorsally approximately 4 cm caudal to the 12th rib.

Physiology

SACs are known to be well adapted to the hypobaric environment of the high Andes [16–24]. Numerous physiologists have studied this phenomenon to establish the mechanism(s) for this adaptation. It is probable that a combination of mechanisms involving the respiratory, cardiovascular, muscular, and hemic systems are involved. Some of these mechanisms may have relevance to clinical syndromes and clinical evaluation of the respiratory system.

SAC Adaptations to Altitude

1) High hemoglobin concentration of blood, elevated mean corpuscular hemoglobin (MCH) (45%)
2) Large numbers of erythrocytes that are small, ellipsoid, and thin. This red blood cell (RBC) morphology gives them a very high surface area: volume ratio.
3) Moderate to high degree of pulmonary hypertension at altitude, which does not progress to right heart hypertrophy or thickening of the pulmonary arteries. This may be related to enhancement of nitric oxide metabolic pathways [25].
4) High-oxygen affinity of SAC hemoglobin [26].
5) Efficient myoglobin utilization of oxygen by tissues – SAC blood oxygen dissociation curves are shifted to the left more than in species adapted to low altitudes [27].

Diagnostic Procedures

The proximal nares (alae) and lips of camelids are highly movable. Examination of camelids should include facial symmetry and determination of the symmetry of air flowing from each nostril. Movement of both alae, during successive inhalation and exhalation, should be similar. Lack of airflow from one or both alae may suggest obstruction or narrowing of the nasal passages or dorsal displacement of the soft palate. Unilateral dysfunction of one alae may suggest middle-ear infections or central nervous system (CNS) disease (Listeriosis, edema, space-occupying lesions). Localizing CNS dysfunctions of camelids are associated with concurrent dysfunction of the ipsilateral ear and an inability to close the ipsilateral palpebra. Swelling of the nose, face, eyelids may also reduce the function of nares. This is common in patients with trauma, over-tight neck

Figure 11.5 (a) A 12-year-old female alpaca presented for anorexia and abdominal distension. External palpation of the caudoventral abdomen indicated a grapefruit sized mass. Ultrasound of the abdomen indicated a mass in the right caudal portion of the abdomen. Malodorous nasal discharge from one nostril was associated with anorexia and dehydration. The abdominal mass was diagnosed as lymphoma. (b) A three-year-old intact male alpaca demonstrating clean nares with one drop of clear nasal secretions at the top of his nasal philtrum.

bands, venomous snakebite or in the presence of an inappropriately fitted halter.

Nasal discharge may be present emanating from one or both nostrils (Figure 11.5a and b). In normal animals, the discharge is small volume and clear (Figure 11.5b). The presence of limited unilateral or bilateral nasal discharge (serous) is not generally associated with dyspnea [4]. Unilateral or bilateral, viscous discharge may reflect rhinitis, pneumonia or systemic disease (Figure 11.5a). Malodorous nasal discharge sometimes becomes copious, white-green (increased white blood count [WBC]), bloody or blood-tinged discharge can indicate chronic inflammation of the nasal passages, pharynx or pneumonia. Unilateral, thick, persistent nasal discharge from one nostril may indicate a CNS disease, a foreign-body obstruction of one nasal passage, sinusitis, or megaesophagus. Discharge present in both nostrils suggests bilateral nasal disease or the source is caudal to the nasal septum (pharynx, larynx, lungs). Animals with proximal GI obstructions (trichophytobezoar; neoplasia, etc.) may regurgitate and the nasal discharge may contain GI contents. In neonates, the reflux of milk from the nostril may also indicate palatoschisis [4].

Complete blood counts (CBCs) in camelids with respiratory disease may be useful in clinical cases. It is often associated with mild to moderate anemia. However, red-cell regenerative responses are not robust in SACs [28]. Circulating leukocyte numbers are generally higher than those of ruminants and horses. Likewise, camelids often have a higher N : L ratio (normally >1.4) than is present in cattle (N : L 0.75–1.0) [28]. This may be associated with stress-induced catecholamine release leading to a greater percentage of circulating neutrophils or increased cortisol levels resulting in reduced numbers of lymphoid cells [28].

Development of markers associated with inflammation in camelids with disease has been reported in the literature. In an lipopolysaccharide (LPS)-induced inflammation model, one study demonstrated the effect of LPS was to alter circulating lymphocyte and neutrophil numbers resulting in markedly different N : L ratio [29]. They further

noted that during infusion of LPS, serum concentrations of iron were significantly lower than those of the non-LPS challenged group. Significant increases in serum Haptoglobin, but not serum amyloid A (SAA) were also determined in challenged alpacas [29]. Evaluation of serum cortisol has also been used in camelids to define stress responses in camelids with a variety of disorders [30]. In another study, animals that died from cerebrospinal nematodiasis had elevated serum levels of fibrinogen in comparison to those that survived [31]. Further study is needed to validate the use of these and other markers in camelids.

Lung auscultation is described in Chapter 4. The area available for auscultation is far more restricted than would be anticipated by the more caudal line of pleural reflection. Normal respiratory sounds auscultable in the cranial, ventral and caudo-dorsal lung fields are commonly bronchovesicular as in ruminants. In some animals suspected of having pneumonia, clipping the fiber from one or both thoracic lung fields may improve audibility of lung sounds, improve US imaging quality and, in some cases, remove radio-dense debris (sand, dirt, plant material, fecal pellets) present within the fiber or on the skin of a patient. Camelids with clinical signs of respiratory dysfunction should be evaluated for thoracic and abdominal effusions. Unilateral and bilateral effusions will compromise the ability to auscultate normal lung sounds. Thoracocentesis is also described in Chapter 4.

When evaluation of animals suspected of lung dysfunction is required, arterial blood gas analysis is useful to determine how well the animal is oxygenating itself. In severely compromised individuals, administration of oxygen via mask or using nasal canulae can improve arterial oxygen tension. Arterial blood gas sampling has been described and can be very useful in cases to evaluate oxygenation of the blood [4, 32].

Ultrasound (US) evaluation of the larynx, proximal trachea and thorax are commonly utilized in clinically ill camelids. For the thorax, one major limiting factor is the width of the ultrasound probe relative to the width of the intercostal spaces. In adult alpacas, llamas and camels US evaluation of the thorax without shaving can be performed; wetting the fiber with alcohol (70% isopropanol) or water with or without US gel improves the evaluation. A higher resolution examination follows clipping the thoracic cage with a #40 blade. Imaging of thoracic effusion, cardiac structures and function of the lung are readily available with most portable US units in practice.

Radiographic evaluation of the thorax is not limited to the lateral view as has been reported in the adult alpaca or llama (Figure 11.6). A dorsal-ventral (DV) view can be produced by making the animal cush onto a radiolucent offset

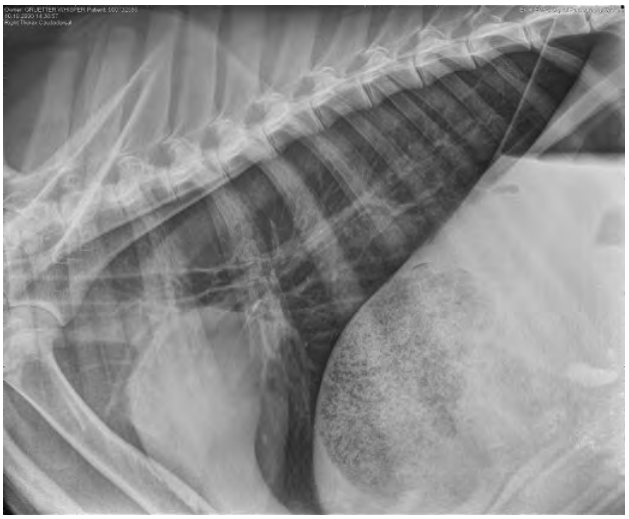
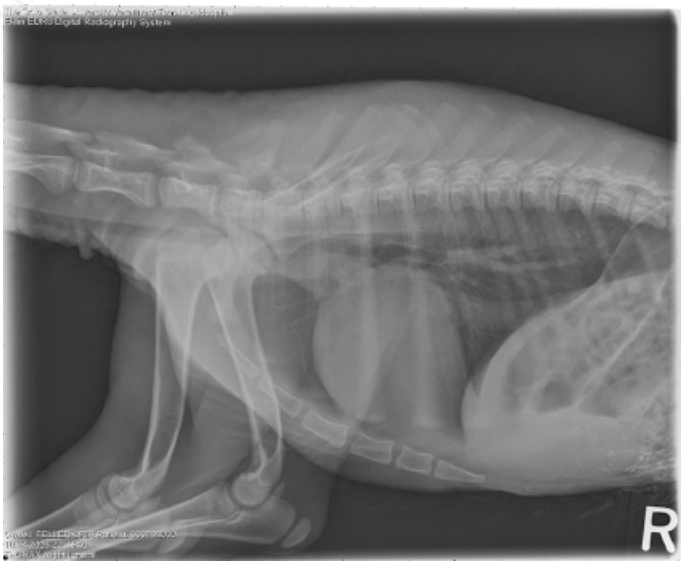


Figure 11.6 Unremarkable thoracic radiograph (right lateral projection) of an 18-month-old, female llama referred for possible megaesophagus. The animal has history of salivation, dysphagia and weight loss over a two-week period. Animal was azotemic (BUN 79 mg/dL), creatinine (5.1 mg/dL), hyponatremic, hypokalemic with normo-chloremia. Thoracic radiograph suggests megaesophagus is not present.

(V-shaped foam pad) on the floor above a cassette, with or without a grid in place. This often requires sedation to limit animal movement, and depending upon animal size and cassette size, multiple exposures to visualize all parts of the thoracic cavity may be necessary. Both lateral and dorsoventral projections are easily performed in neonates (Figures 11.7a and b). The availability of digital systems has made the acquisition of diagnostic-quality images possible and faster than previously.

With the development of high-resolution CT equipment, the use of CT in the assessment of many camelid disorders is also a very viable path for evaluation of sick camelids. This is especially true when one considers imaging of the mediastinum, vascular architecture and diaphragm. As an example, an approximately 2.5-year-old female alpaca with a history of weight loss and reproductive issues was referred to a veterinary teaching hospital. She had been bred three consecutive times with consistent loss of the pregnancy at approximately 90–120 days of gestation. The animal had a body condition score of 2/5 and body weight of 98 lbs. The heart and respiratory rates were within normal limits. Auscultation of both hemithoraces demonstrated abnormal lung sounds in all quadrants. Lateral radiographs of the thorax revealed large numbers of radio-dense nodules dispersed throughout all lung fields (Figure 11.8a). CT of the thorax was used to locate a nodule within the lung at the dorsolateral aspect of the pulmonary parenchyma adjacent to the lung surface (Figure 11.8b). Trans-thoracic US guidance provided the location for aspiration and cytologic

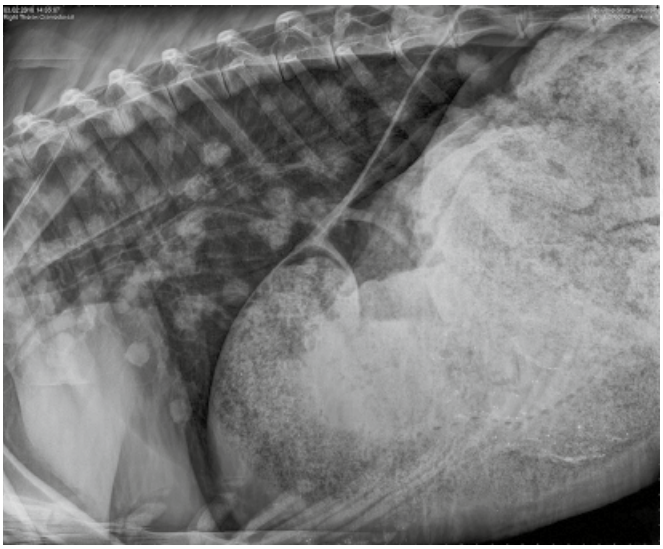
(a)



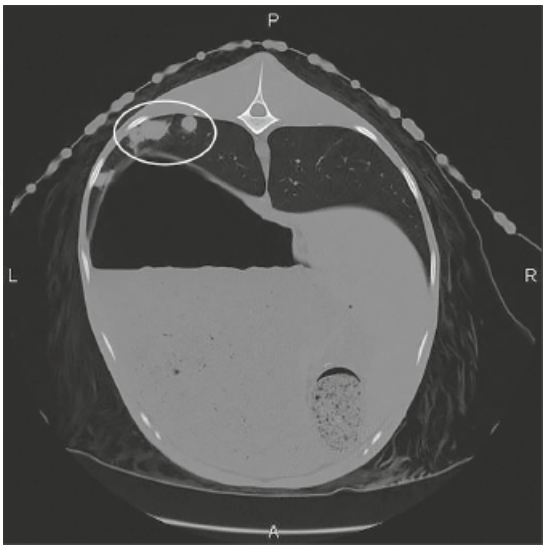
(b)

Figure 11.7 A three-day-old alpaca cria (male) with failure of transfer of maternal antibodies referred for possible pneumonia. Right lateral and VD view of thorax are relatively normal.

(a)



(b)



(c)

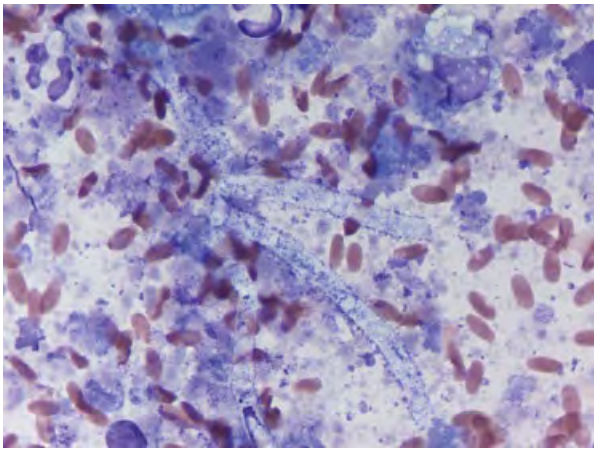


Figure 11.8 (a) Standing lateral image of thorax of three-year-old female alpaca presented for weight loss and repeated abortion. The image reveals relatively uniform-sized nodules throughout the lung parenchyma. (b) CT slice in dorso-caudal lung region with peripheral lung nodule identified for aspiration cytology. A white circle has been drawn around the superficial pulmonary nodules. (c) Cytologic preparation of FNA of nodule demonstrates negatively staining, branching, filamentous fungal hyphae. Polymerase chain reaction (PCR) analysis of cultured fungal organisms indicated *Aspergillus fumigatus*.

analysis of the superficial nodule (Figure 11.8c). Culture of lung tissue produced growth of characteristic fungal hyphae and confirmed as *Aspergillus fumigatus*.

Profound respiratory efforts (marked nostril flair, abdominal efforts) may suggest severe respiratory dysfunction. In newborn camelids delivered prematurely, surfactant deficiency may be the cause. Choanal atresia or other facial distortion may also produce respiratory insufficiency. In young camelids (2–12 months of age) with marked nostril flaring and markedly increased abdominal effort, dysfunction of the diaphragm associated with phrenic nerve dysfunction or damage has been observed [33–35]. Most of these animals are bilaterally affected and pathologic changes to the phrenic nerve are present at post-mortem examinations. While the inciting causes have not been determined in most cases, damage or degeneration of the phrenic nerve and diaphragmatic muscle(s) has been demonstrated. In humans, damage to the cervical vertebrae (falling with trauma to neck) or damage to brachial plexus have been associated with similar problems [36–39]. The long cervical spine of the camelid is prone to similar injury and may result in diaphragmatic denervation. Further, in some human patients, elevated serum antibody titers have indicated potential viral inflammation [38, 39]. This aspect of diaphragmatic paralysis in camelids has not yet been evaluated. Diagnostically, the use of fluoroscopy is considered the imaging method of choice for demonstration of this clinical entity. The prognosis for these cases is guarded to poor.

Transtracheal aspiration/wash, bronchoalveolar lavage and transthoracic fine needle aspiration (FNA) for cytology and/or culture have been useful in some clinical cases of pneumonia. Reference values and cytologic analysis, of healthy llamas has been reported [40]. Similar analyses of bronchoalveolar lavage have been reported in alpacas [41].

A 10-year-old female alpaca presented for acute respiratory distress and diarrhea. The camelids respiratory rate was elevated (60 breath/minute) with a prominent abdominal component to breathing. She had fetid breath and lung sounds were reduced bilaterally. US examination of the thorax indicated pronounced lung hepatization with some pleural effusion. A 300 cm long silicon broncho-alveolar lavage (BAL) catheter (4.2 mm diameter) was placed into the lung via the nasal passage and approximately 30 mL of physiologic saline was infused. The cytologic evaluation of this recovered fluid indicated marked mononuclear inflammation and hemorrhage with polymorphic rods and few cocci/coccobacilli (Figure 11.9). Autopsy revealed severe aspiration pneumonia with multi-focal hemorrhage and edema throughout the GI tract and other organs. Aspiration pneumonia was considered likely.

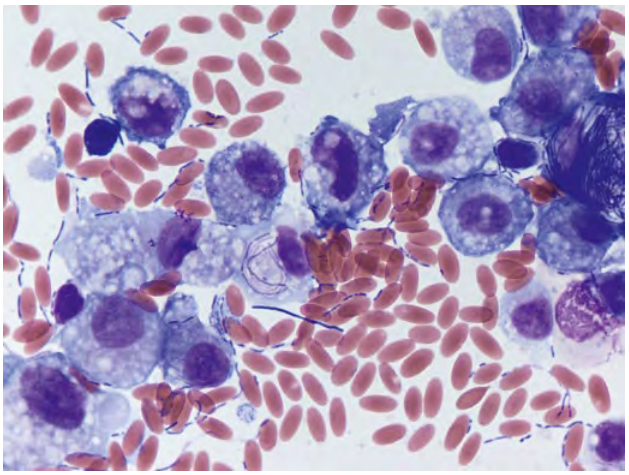


Figure 11.9 Cytospin preparation of lung lavage fluid from an alpaca with severe aspiration pneumonia. The fluid revealed hemorrhage, large numbers of reactive macrophages and numerous bacterial rods, coccobacilli, and cocci.

Infectious Diseases

Infectious causes of respiratory disease include bacterial, viral, and fungal etiologies. Of importance is that while these agents do cause disease in camelids, primary pneumonia is not frequently observed in camelids, unlike ruminants and horses. It is more common for diseases in other body systems to alter lung function leading to clinical signs. This makes it necessary to carefully examine the signalment, age, historical information prior to developing a diagnostic plan and deciding on what medications should be administered.

A common presentation at our hospital is provided. A 12-year-old adult female llama presented for diarrhea, loss of body weight and elevated respiratory rate and effort. On presentation, the llama had obvious nostril flaring, tachypnea and reluctance to move (walk). Auscultation of the thorax revealed very quiet lung sounds in the cranioventral quadrant. Ultrasound evaluation of the llamas right and left ventral lung fields revealed massive pleural effusion bilaterally. On evaluation of the animal's blood, the total protein was 4.6 g/dL and serum albumin 1.4 g/dL. After analysis of a fecal sample for parasite ID, numerous Trichuris spp. and several Nematodirus eggs were present in the fecal. In this case, hypoproteinemia led to effusion precipitating the respiratory embarrassment.

The literature of camels and SAC suggests that animals with weight loss, partial to complete anorexia, and respiratory signs can be afflicted by wide variety of causal agents. Tuberculosis and many commonly encountered fungal agents gain access to the camelids body via inhalation including Tuberculosis [42], Coccidioidomycosis

[43, 44], Aspergillosis [45, 46], Blastomycosis [47, 48], Cryptococcosis [49, 50], Histoplasmosis [51].

Coccidioides immitis, is highlighted in this chapter as it is highly geographically localized to the Southwest USA, Mexico, and Central America. An adult male castrated llama was presented to veterinary hospital at The Ohio State University for a possible abscessed mandibular tooth (Figure 11.10a). Since the mass was moveable, a surgical biopsy was taken for histopathologic evaluation, and spherules characteristic of C-immitis were observed in a granulomatous matrix (Figure 11.10b). After determination of the causative organism, thoracic imaging was conducted (Figure 11.10c) because inhalation of these organisms by the animal typically results in proliferation and dissemination. It is likely the submandibular nodule reflects dissemination from the lung. It was later determined the llama had traveled from the arid southwestern US to Ohio. Similar case descriptions (for *Blastomyces dermatitidis and Histoplasma capsulatum*) could be deciphered based upon the origin of these animals and their history. One could anticipate less geographic localization for other fungi like Cryptococcus spp., (found globally).

Viral diseases of the SACs are also associated with the respiratory system. While they are not commonly associated with severe illness or occur in outbreak forms, there are exceptions. One viral agent commonly isolated from SAC is adenovirus. Clinical signs associated with adenovirus infection in camelids included lethargy, anorexia with progression (in some) to respiratory illness and in some cases, development of CNS disease [52]. Lesions described by Galbreath et al. [52] include fibrinous pleuritis, peritonitis and pericarditis were associated with a prominent perivasculitis. Hepatocellular lesions were also documented with viral inclusions [52].

Bluetongue virus infections have also been documented in llamas and alpacas in association with thoracic effusion and lung edema [53–56]. Altered climatic norms have allowed advancement of the vector and virus to more northern regions.

Infectious bovine rhinotracheitis virus (IBR) presenting as respiratory illness in llamas has been described [57]. Lungs of this llama demonstrated congestion and hemorrhage and culture of the lungs indicated large numbers of *Pasteurella multocida*. In many of the studies from South America, serum antibody titers indicate that camelids respond to exposure to many viruses of ruminants and horses [58, 59].

More recently, an acute upper respiratory syndrome in alpacas was determined to be the result of a Coronavirus infection. This was most prominent when animals were taken to shows and developed mild clinical signs (including fever, nasal discharge [copious] and reduced appetite). When returned to their farms, co-mingling of these young animals with older pregnant female alpacas resulted in morbidity and mortality. Pathologic lesions in one study were characterized by severe pulmonary congestion, edema, and pleural effusion. Histologic examination confirmed edema as well as bronchointerstitial pneumonia, some with hyaline membrane formation [60, 61].

Bacterial Pneumonia

As mentioned previously, pneumonia is often a presumptive diagnosis in camelids, especially neonates, based on increased respiratory sounds and some degree of dyspnea.

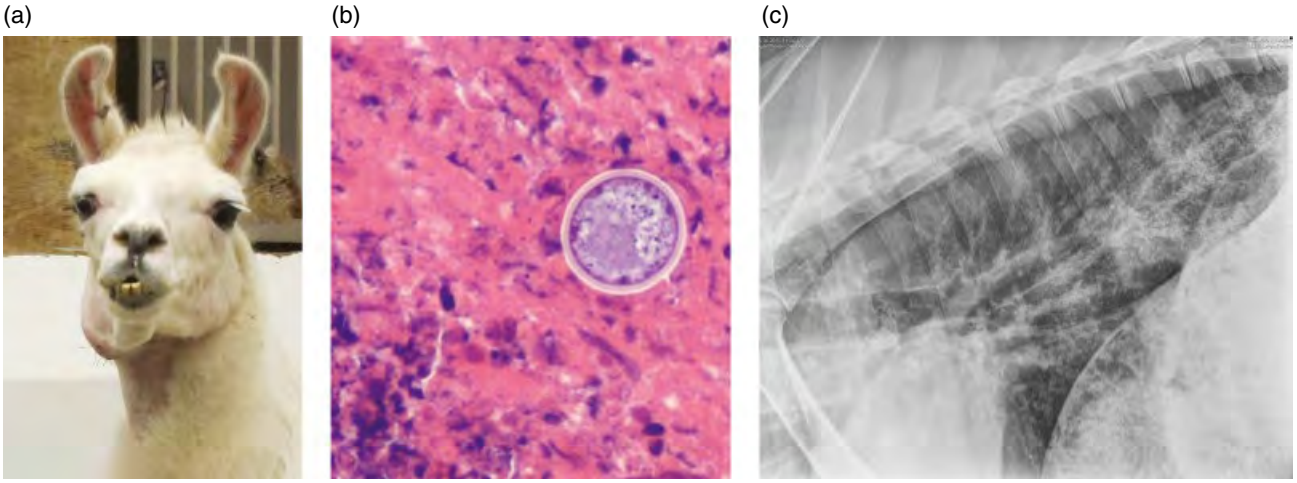


Figure 11.10 (a) Llama presented for swelling superficial to right mandible. (b) Histopathology of the mass following surgical removal demonstrated the characteristic tissue form of *C. immitis* granuloma. Spherules characteristic of C-immitis were observed in the granulomatous matrix. (c) The lung evaluation confirmed dissemination of the organism.

Pneumonia is also a frequent diagnosis at a gross necropsy because the normal lung tends to be slightly edematous and hyperemic. Complete examination and evaluation are necessary to exclude diseases of other organ systems. Pulmonary edema is a common terminal lesion seen in animals dying from numerous diseases.

Etiology

The causal bacterial agents of camelid pneumonia can be similar to those causing pneumonia in livestock and horses. In a very recent review of bacterial organisms cultured from pneumonic lungs of alpacas and llamas, 130 cases grew a variety of bacterial genera (*Escherichia coli*, Streptococcus spp., Pseudomonas spp., Klebsiella spp., Enterobacter spp., Bordetella spp., Rhodococcus spp., Actinobacillus spp., Nocardia spp.), along with fewer cases having Arcanobacterium spp., Corynebacterium spp., Salmonella spp., Pasteurella spp., Staphylococcus spp., Bacillus spp., Listeria spp. [44].

A review of 93 cases of pneumonia in llamas and alpacas evaluated by veterinarians at the authors' practice showed similar findings based on physical examination, radiology, and microbial culture of lung fluids, lung tissue, and thoracic effusion. The following isolates (n = 111) were obtained from adults and young crias: *E. coli* (16%), Streptococci (21%), Bordetella spp. (3%), Actinobacillus spp., (3.6%), Pseudomonas (4.5%), Fungal agents (6.3%). The remainder of bacteria were single isolates from individual animals. The previous study indicated 23% of the cases were from animals with aspiration pneumonia [44]. The Ohio State University (OSU) caseload also continues to see aspiration pneumonia cases. Milk aspiration by neonates nursing from a bottle or misplacement of a stomach tube can occur. Passive regurgitation at induction of anesthesia or during long anesthetic episodes when an airway is not secured with a cuffed endotracheal (ET) tube, remains a significant risk. Camelids with proximal GI obstruction may also be at increased risk for aspiration due to possible regurgitation of C1 contents. These polymicrobial lung infections require both aggressive antimicrobial and anti-inflammatory therapy.

Streptococcus equi, subsp. zooepidemicus infections are an important disease in camelids. There are numerous publications which indicate co-mingling of camelids with horses or housing of camelids in stalls also used for horses involving either inhalation of bacteria or potentially wound-induced infections which gain access to the systemic circulation [62–69]. The disease can be acute or chronic; systemic disease is common in young camelids, whereas the more chronic cases are generally found in adult camelids. Experimental induction of Streptococcus equi, subsp. zooepidemicus used young llamas (10–19 months of age) was characterized by anorexia, depression and fever [63]. Cases reporting systemic disease in adults are not uncommon. Antemortem clinical findings may include fever, depression, anorexia or altered GI function, prolonged recumbency and sudden death. At surgery or postmortem, polyserositis is a major finding. One report characterized the lesions as "strands of fibrin attached to the pleural surfaces, pleural effusion with congested, edematous, hemorrhagic and non-aerated portions of the lung" [69]. Effusions and fibrin may also extend to the abdomen as well as the pericardial sac with similar lesions. In most untreated cases, isolation of Streptococcus equi subsp. zooepidemicus from the lung, pleural effusion and liver is common.

The patterns of isolates from clinical cases of bacterial pneumonia in camelids suggest infections with coliform and Gram-positive cocci are common. These findings support treatment using broad-spectrum antimicrobials, at least until bacterial identification and antimicrobial susceptibility is determined or empirical therapy results in clinical improvement. Combination therapy is commonly used in many locations. IV therapy is recommended as it is likely to provide the most appropriate response. While a second-generation fluoroquinolone (enrofloxacin) may be efficacious against Gram-negative isolates, it would be unlikely to successfully treat a Streptococci infection.

While serologic evidence of antibodies to *Mycoplasma mycoides* "cluster" suggests this group of pathogens could produce lung disease in camelids [70], no reports of these disease agents causing pneumonia have been reported in SAC.

Hypertrophic Osteopathy

Hypertrophic osteopathy has been reported in an alpaca. The definitive identification of the inciting cause was not determined; however, at necropsy, a chronic pneumonia was found in the ventral aspect of the left apical lobe of the lung. There were also sarcocystis lesions in the skeletal muscle of this animal [71].

Parasitic Diseases

Camelids are susceptible to lungworm (*Dictyocaulus viviparous*) [72] and nasopharyngeal bots (*Cephenemyia* spp.) [73, 74], both of which are described in Chapter 7.

Congenital Disorders

Congenital disorders include facial deformities (Wry face), cyclopean cebocephaly, choanal atresia, cleft palate, are discussed in Chapter 21.

Miscellaneous Diseases [75–77]

Trauma

Injuries to the muzzle (dog bite, contusion) or nasal cavity causing swelling of the tissue may occlude the air passageways, resulting in dyspnea. Venomous snake bites of the nose or face can cause edema and lead to occlusion of the nares. Performing a temporary tracheostomy may be prudent in these cases. Llamas and alpacas may suffocate if they are inappropriately haltered or tied to an immovable structure. Collapse of the nostrils from impingement by a halter or trachea if a rope is tied around the neck may impair the ability of a camelid to breathe. Most camelid experts would suggest that halters should not be left on the animals when they are not required. It is generally unwise to tie camelids when hauled in trailers. This is especially true when there are multiple animals present during transport. Because camelids are primarily nasal breathers, any impairment of the air passage through the nostrils results in dyspnea and open mouth breathing. Partial occlusion of the upper airways may result in stertorous breathing.

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12

Digestive System and Abdomen

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The digestive system is one of the most intensely studied organ systems in the camelid. It has unique characteristics, which constitute some of the major differences that separate camelids from ruminants in the order Artiodactyla. Both Old and New World camelids evolved in harsh but differing environments. The two environments had in common a characteristic of sparse, poor-quality forage for at least part of the year, and both species developed adaptations to thrive in these environments.

It appears that the common ancestor of both camelids and ruminants was simple stomached. A foregut fermentation system and a rumination cycle evolved in parallel with the digestive systems of ruminants. Numerous other morphologic differences of the digestive system affect diagnosis, treatment, and management of diseases of the digestive system in these animals.

Anatomy and Physiology

The digestive system begins with the oral cavity and includes the lips, teeth, salivary glands, and pharyngeal structures. Continuing aboard, the esophagus leads into a complex gastric compartment consisting of three distinct, separate compartments. The small intestines begin at the pylorus and include the duodenum, jejunum, and ileum. Camelids have a cecum like other domestic mammals. The large intestines serve as the final intestinal segment and include a spiral colon like ruminants. The rectum is the terminal segment of the gastrointestinal (GI) tract and ends at the anus. Other closely associated structures within the abdominal cavity include the greater omentum, the pancreas, and a liver devoid of a gull bladder.

Peritoneum, Omentum, and Mesentery

Most of the digestive system is included within the abdominal (peritoneal) cavity and thus a discussion of the peritoneal cavity must be included when thoroughly discussing anatomy, function, and diseases of this organ system. The abdominal cavity is lined by the peritoneum, a smooth lining made up of specialized epithelial cells known as mesothelium [1, 2]. The peritoneum is subdivided into the parietal peritoneum that lines the abdominal wall and the visceral peritoneum that covers the abdominal organs (viscera). The peritoneum provides a low-friction interface for the sliding of the abdominal viscera. The peritoneum produces a hypocellular, low-protein fluid that aids in the lubrication of the abdominal viscera [2].

Camelids' greater omentum is less robust than that in cattle and sheep. It is thin, friable, and easily torn during abdominal surgery. There is no omental sling, and it does not support the weight of the intestines as it does in ruminants but merely rests on the abdominal floor. It does not accumulate large adipose stores as in ruminants. The greater omentum has attachments to all three gastric compartments. It attaches to the saccular region of C1, the greater curvature of C2, and the right surface of C1. The epiploic foramen enters the sac formed by the greater omentum ventral to the liver, near the entrance of the caudal vena cava. The lesser omentum attaches to

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the lesser curvature of C3 and extends from C3 to the liver.

Blood is supplied to the abdominal viscera via vessels contained within the mesentery. Multiple anastomosing vessels provide collateral circulation to the various parts of the GI tract. Branches of the celiac artery supply blood to the gastric compartments, while the cranial mesenteric artery supplies blood to the jejunum and ileum as well as the cecum and ascending colon. The cecal artery gives off two branches, which supply blood to the ascending and spiral colon. The caudal mesenteric root supplies blood to the descending colon and rectum [3].

Lips and Oropharynx

The upper lip of camelids is split by a philtrum (labial cleft) (Figure 12.1). Each side of the lip can be manipulated independently by the elevator nasolabialis muscle under the control of the facial nerve. The upper lip is highly tactile, useful for fine discrimination, and the camelid uses it as a sense organ to investigate potential feed. As a result of this fastidious feeding behavior, gastric foreign bodies [4], with the exception of bezoars, are rarely found in SACs, however, Old-World camelids (OWCs) are less discriminate eaters and gastric foreign bodies appear to be more common in these animals [5, 6]. The lower lip has no unique characteristic, but it is less mobile than the lower lip of the caprine or ovine.

The oral cavity of the llama is small, and the cheeks do not permit wide opening. The rami of the mandibles are set close together, and the tongue occupies the ventral space.

Figure 12.1 Split philtrum in an alpaca. The split philtrum present in camelids permits manipulation of each side independently.

The lips are tightly opposed to the teeth. It is rare for a cud to become trapped in the buccal cavity of a normal animal. Camels have a larger oral cavity and the mouth is capable of opening widely.

The camelid soft palate is elongated. The soft palate forms a partial seal with the epiglottis impeding passage of air from the oropharynx into the trachea. Camelids are therefore semi-obligate nasal breathers and become dyspnoic if they cannot pass air through their nose. With nasal obstruction, simultaneous eating and breathing are difficult. Breathing takes immediate precedence, and the animal will slowly starve if nasal obstruction persists.

The tongue of camelids is relatively immobile. It is rarely extended beyond the lips and is not used for prehension of feed. The camelid does not lick the neonate nor does it remove any attached fetal membrane. The caudal third of the tongue has a pronounced dome (approximately 5 cm in adult llamas) which can impede visualization of and access to the caudal pharyngeal area.

Esophagus

The camelid esophagus is similar to that of ruminants. It extends from just dorsal to the larynx through the thoracic mediastinum and penetrates the esophageal hiatus of the diaphragm. It joins the first stomach compartment at the cardia. The outside of the esophagus is covered by serosa throughout the thorax and abdomen. The muscular wall is made up of an outer circular layer and an inner longitudinal layer. The inner mucosal layer separates the muscular layer from the lumen. Muscular sphincters of the esophagus are present at the proximal and distal ends. The inner surface is lined by stratified squamous epithelium [3, 7].

Teeth

Comparative Dental Anatomy

As with other herbivores, camelids have dentition that is well adapted to grinding of forages. They have well developed molars and premolars and less developed rostral teeth. The specific dental anatomy however, is different than that of ruminants. A comparison of the deciduous and permanent dental formulas for camelids and ruminants is given in Table 12.1. Though the dental formula of camels differs from that of SACs, all SACs have the same dental formula.

The single upper incisor of both SACs and camels has migrated caudally and evolved to a caniniform shape and function. With the mouth closed, this tooth rests immediately caudal to the corner lower incisor. All four canine teeth are present in adult males; these canines along with the upper incisors are referred to as “fighting teeth.”

Table 12.1 Dental formulas of camelids and ruminants.

		South American Camelids (SACs)					Camels (OWCs)					Ruminants				
		I	C	PM	M	Total	I	C	PM	M	Total	I	C	PM	M	Total
Deciduous	Maxillary	1	1	2 → 3	–	x2 = 8 → 10	1	1	3	–	x2 = 10	0	0	3	–	x2 = 6
	Mandibular	3	1	1 → 2	–	x2 = 10 → 12	3	1	2	–	x2 = 12	3	1	3	–	x2 = 14
	Total					18 → 22					22					20
Permanent	Maxillary	1	1	1 → 2	3	x2 = 12 → 14	1	1	3	3	x2 = 16	0	0	3	3	x2 = 12
	Mandibular	3	1	1 → 2	3	x2 = 16 → 18	3	1	2	3	x2 = 18	3	1	3	3	x2 = 20
	Total					28 → 32					34					32

I, Incisors; C, Canines; PM, Premolars; M, Molars.

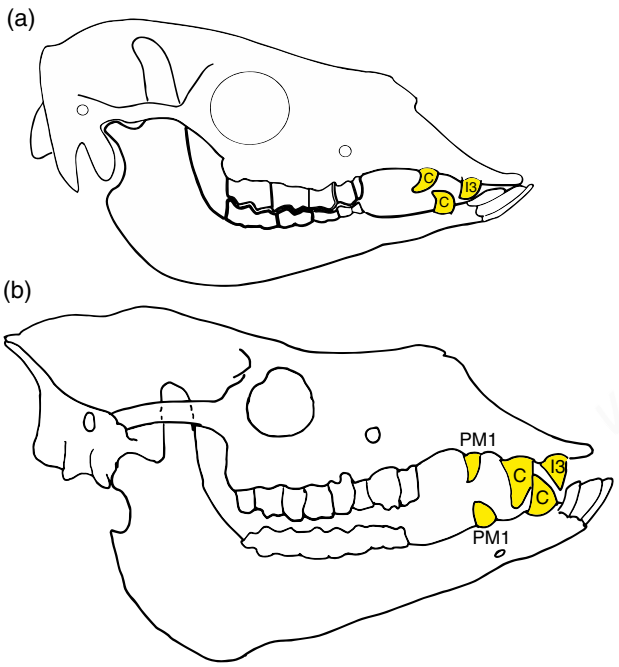


Figure 12.2 Diagram illustrating fighting teeth of male camelids. (a) Lateral view of a male llama skull. The canines and the maxillary third incisor (highlighted in yellow) comprise the fighting teeth. (b) lateral view of a male camel skull. The canines, first premolars, and the maxillary third incisors (highlighted in yellow) comprise the fighting teeth.

Therefore, intact male SACs appear to have two upper and one lower canine on each side (Figure 12.2a).

Camels have more premolars than SACs, but the first upper and lower premolars have also migrated forward in the jaw and become caniniform. Thus, the camel appears to have three upper and two lower canines on each side (Figure 12.2b). In contrast, cattle, sheep, and goats appear to have four lower incisors and no canines, but the corner incisor is actually a canine tooth that has migrated forward and become incisiform in ruminants.

Incisors

Camelid incisors are anchored firmly into the alveolus, as in sheep, but in contrast to cattle in which the teeth are movable in the alveolus. Incisor morphology varies by species.

Vicuña incisors differ in shape and structure from those of other artiodactylids. Permanent vicuña incisors are more like the continuously erupting incisors of rodents. The permanent incisors of the vicuña are parallel sided, ten times as long as they are wide, with a square cross section. The crown enamel is confined to the labial surface, extending down the root to within 2–3 mm of the wide, open base. The lingual occlusal surface is comprised of dentine, which is softer than the labial rim of enamel; thus, the tooth wears into a chisel shape (Figure 12.3).

The deciduous incisors of vicuñas are more elongated than those of the llama, but they are spatulate. The base of the incisor remains open. There is thin enamel on the lingual surface of the tooth, and the enamel of the labial surface extends halfway down the tooth [8].

Incisors of the llama and guanaco are spatulate, with the greatest width ranging from one-fifth to one-fourth the greatest length (Figure 12.4). The root tapers rapidly to a closed base. Both labial and lingual surfaces of the crown are enameled, and on the lingual side the enamel extends one-third the length of the tooth. Deciduous and permanent incisors are similar in shape and structure but can be differentiated by size and amount of wear.

Dental structure and DNA evidence demonstrates that alpacas have evolved from vicuñas and are now classified in the genus Vicugna (See Chapter 1). Alpaca incisors are intergrade between those of the vicuña and the llama. Enamel is present on both the labial and lingual surface of the tooth, though the enamel covering the lingual surface is much thinner [9]. The alpaca's incisor is narrow. The adult alpaca incisor is nonspatulate but has a rectangular cross section rather than the square cross section of the

Figure 12.3 Permanent incisors of a vicuña and alpaca. (a) vicuña. Incisors are parallel sided and much longer than they are wide. The crown enamel is confined to the labial surface, the remainder of the occlusal surface is comprised of dentine. The dentine of this tooth has worn more than the labial enamel creating a chisel-shaped tooth. (b) Permanent alpaca incisors have similar chisel shape to vicuñas, though they have enamel present on both the labial and lingual sides of the tooth.

Figure 12.5 Occlusal surface of mandibular incisors following trimming. Note the occlusal surface is no longer chiseled, and has assumed a rectangular shape.

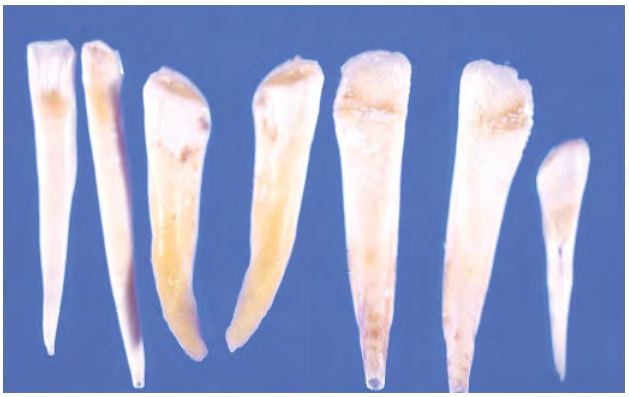


Figure 12.4 Incisor teeth of a mature llama. The incisors are spatulate and root tapers to a closed base. Enamel covers the entire occlusal surface.

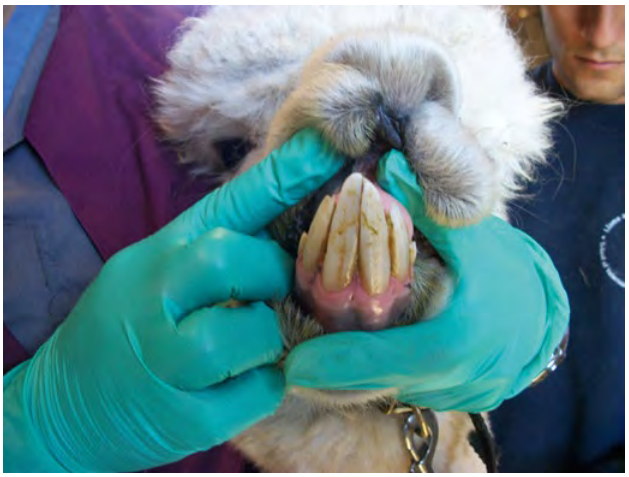


Figure 12.6 Overgrown incisors. The teeth protrude rostrally and do not contact the dental pad leading to decreased wear. The initial etiology is overcrowding which results in the pushing out of rostral incisors by those caudal to them.

vicuña. The apex of the incisor remains open, and there is good clinical evidence that the incisor continues to grow well into maturity. As in the vicuña, alpaca incisors retain a chisel shape on the occlusal surface. If alpaca incisors are trimmed to avoid having them protrude past the lips, the occlusal surface will appear flattened and have a rectangular or square shape (Figure 12.5).

Deciduous incisors may be difficult to differentiate from permanent incisors. Alpaca deciduous incisors are spatulate, but as the animal begins to grow and wear off the spatulate crown, the teeth assume more of the shape of an adult alpaca incisor. Deciduous incisors are slightly smaller and have a chalky color, in contrast with the permanent incisors that have a translucent sheen. Deciduous incisors taper to the gingiva, whereas permanent teeth have parallel sides into the gingiva. Some clinicians identify vertical ridges on permanent alpaca incisors by running a fingernail across the labial surface of the crown. If permanent canines are present in a male, it is an indication that the animal is mature and the incisors are permanent.

Overgrowth of the incisor teeth is common and is considered by some to be normal (see Chapter 21). Overgrowth is caused primarily by overcrowding and malocclusion and therefore lack of normal wear (Figure 12.6). See 12.4.4.6 Trimming Incisors to Prevent Tooth Drift for more details. If the incisors occlude properly with the dental pad, the teeth will wear off and not grow out past the lips.

Canines

The permanent canines of the camelids are adapted to intraspecies aggression, especially between breeding males. The canines are sharp and are anchored into the jaw by a curved root and are formidable weapons in the intact male (Figure 12.7). Permanent canine eruption in males is associated with puberty and a surge of testosterone. In females and geldings, it varies as to whether or not the permanent canines erupt. The deciduous canines are small, peg-shaped teeth and erupt rarely in females and in only about 5% of the males.

Cheek Teeth

The premolars and molars are collectively referred to as the cheek teeth. The roots of the cheek teeth are closed, and there is no continuous growth throughout life. Similar to other herbivores, they will undergo wear and their morphologic characteristics will change throughout life. The cheek teeth are selenodont (cusps have crescentic outline). It is sometimes challenging on clinical examination and during dental surgery to identify where a tooth begins and ends. The occlusal surface of the tooth exhibits the sharp ridges and points typical of a herbivore that is accustomed to eating harsh grasses, shrubs, and forbs (Figure 12.8). The cheek teeth of the mandible are slightly narrower than those of the maxilla. This uneven apposition commonly leads to the development of points on the lingual side of the mandibular arcades and the labial side of the maxillary arcades in aged animals. Though development of this uneven wear is common, this infrequently leads to problems with mastication or oral trauma. If problems arise, the uneven cheek teeth can be floated to create uniform apposition.

Figure 12.7 Rostral teeth of a male SAC. The maxillary and mandibular canine teeth as well as the upper third incisor comprise the "fighting teeth." They are curved caudally, well anchored into the bone and are formidable weapons in the intact male. C = canine tooth, I1, I2, I3 = first, second, and third incisors respectively.

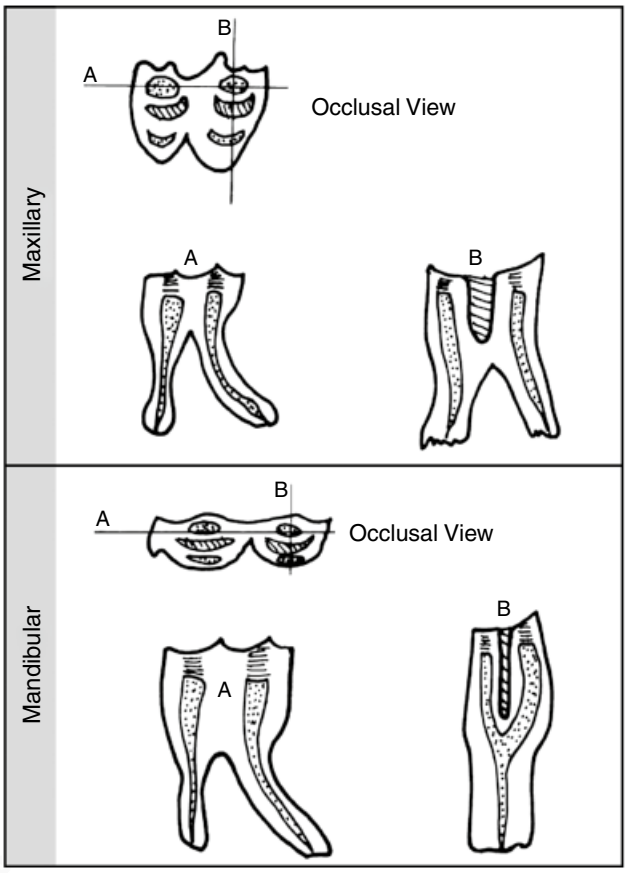


Figure 12.8 Diagrams of the maxillary and mandibular cheek teeth of a SAC. The top illustrations represent the occlusal view. The bottom illustrations are longitudinal sections taken along the lines marked "A" and "B". (a) Section of the tooth taken from the crown through the root in a mesial to distal direction, (b) Section of the tooth taken from the crown through the root in a buccal to lingual direction.

Aging

Determining age by dental eruption times and wear of the teeth has been studied in alpacas and llamas only in South America [8]. Eruption times may be controlled by a genetic trait, but wear depends on the harshness of feedstuffs and the degree of grinding necessary to masticate them. An approximation of age in the living animal can be derived by incisor eruption up to about five years (Table 12.2). After that, it is extremely difficult [10]. Figures 12.9 through 12.18 illustrate the aging of llamas and alpacas.

Salivary Glands

There are three pairs of major salivary glands (parotid, mandibular, and sublingual). Four other glandular regions are less well defined but also contribute to salivary secretion (buccal, palatine, lingual, and labial).

Table 12.2 Eruption of SAC and Camel teeth.

		South American Camelids	Camels
Deciduous Teeth	I1	Birth	2 weeks
	I2	Birth → 90 days[a]	5 weeks
	I3	Birth → 107 days[a]	6 → 12 weeks
	C	(Only present ≈ 5% of males)[b]	
	PM1	N/A	
	PM2	Birth[b]	
	PM3	Birth	
	PM4	Birth	
Permanent Teeth	I1	2 → 2.5 years	4 years
	I2	3 → 3.25 years	5 years
	I3	3.1 → 6 years	6 → 7 years
	C	2.5 → 3.5 years[c]	7 → 8 years
	PM1	N/A	5 → 6 years
	PM2	N/A	5 → 6 years
	PM3	3.5 → 5 years	
	PM4	3.5 → 5 years	
	M1	6 → 9 months	12 → 15 months
	M2	1.5 → 2 years	24 → 36 month
	M3	2.75 → 3.75 years	5 → 5.5 years

[a] Eruption delayed in premature crias.
[b] Presence of tooth variable.
[c] SAC canine eruption can vary from 2 → 7 years.

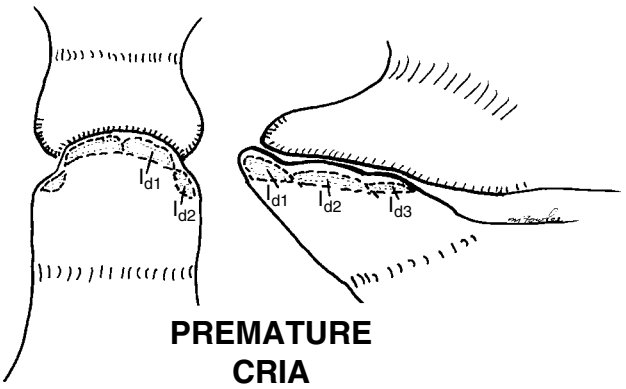


Figure 12.9 Diagram of the teeth of a premature cria. Deciduous alpaca incisors should be erupted at birth. Unerupted incisors at birth are a sign of prematurity.

The salivary glands of camelids are similar in location, number, and histology to those of cattle, sheep, and goats. The parotid gland is much larger than the others and produces only a serous secretion [11], while the other salivary glands are both serous and mucous producers.

The parotid duct arises on the ventral rostral border of the parotid salivary gland. The duct traverses the side of the

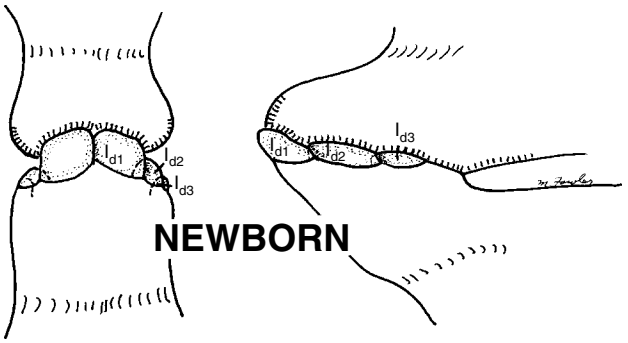


Figure 12.10 Diagram of the teeth of a newborn llama. Deciduous incisors should be erupted at birth.

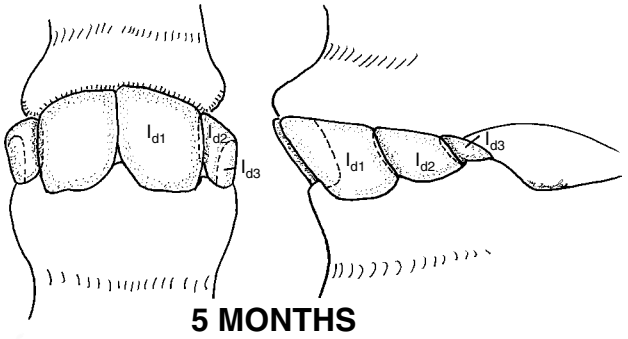


Figure 12.11 Diagram of the teeth of a five-month-old llama.

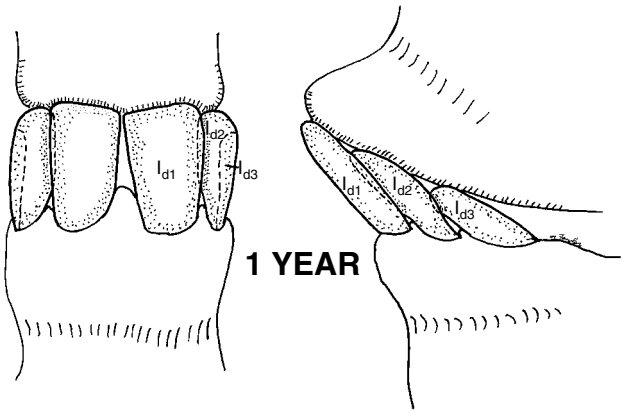


Figure 12.12 Diagram of the teeth of a one-year-old llama.

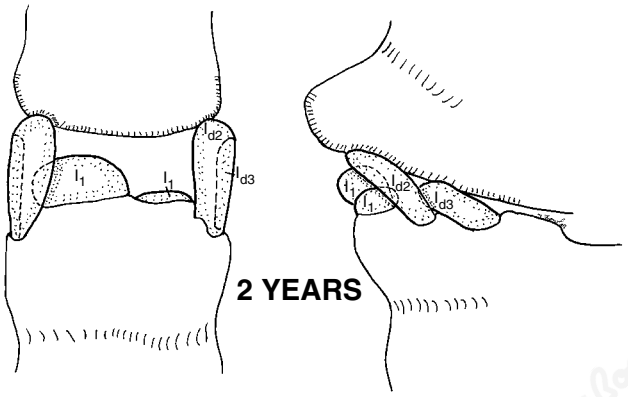


Figure 12.13 Diagram of the teeth of a two-year-old llama.

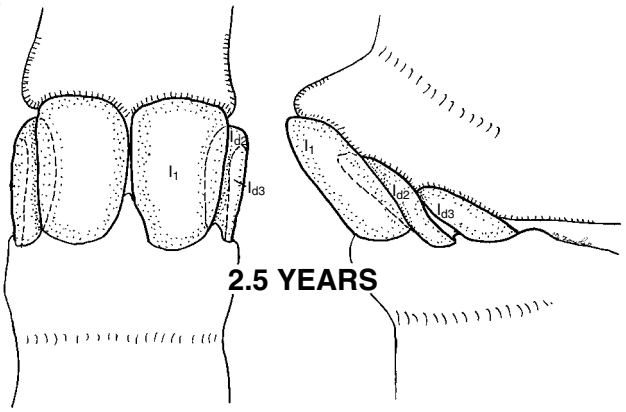


Figure 12.14 Diagram of the teeth of a two-and-a-half-year-old llama.

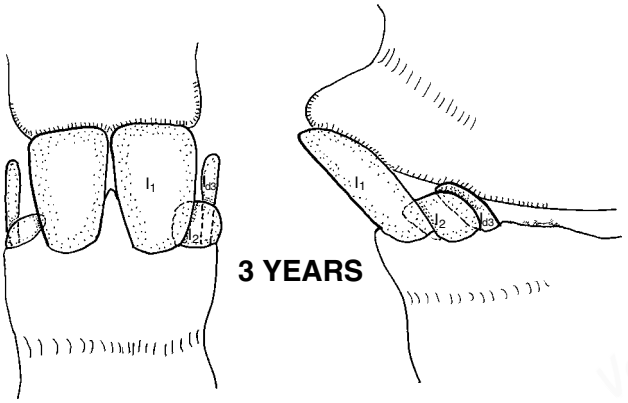


Figure 12.15 Diagram of the teeth of a three-year-old llama.

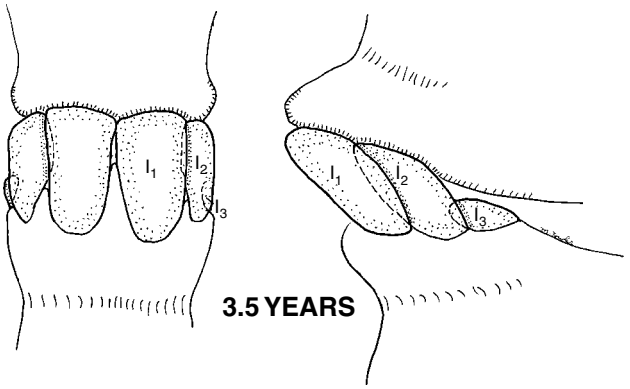


Figure 12.16 Diagram of the teeth of a three-and-a-half-year-old llama.

face mesial to the platysma muscle, approximately 1–1.5 cm dorsal to the facial vein. The parotid duct empties into the oral cavity through an orifice on a flattened papilla that is located 1 cm dorsal to the gingiva at the junction of upper cheek teeth 2 and 3 (Figure 12.19). The parotid duct of the camel empties opposite the fourth upper cheek tooth. In both groups of animals, a line dropped from the rostral margin of the orbit gives the approximate location of the

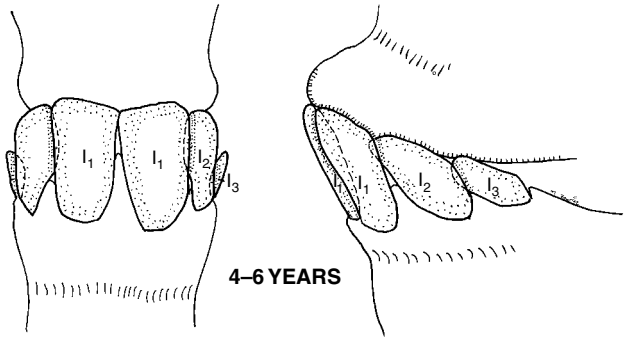


Figure 12.17 Diagram of the teeth of a four- to six-year-old llama.

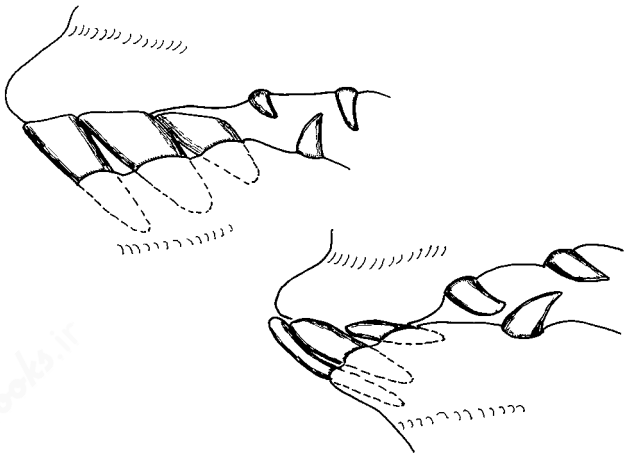


Figure 12.18 Diagram of the teeth of a seven-year-old llama (left) and an 11-year-old (right).

Figure 12.19 Cadaver alpaca specimen with a catheter inserted through the stoma of the parotid salivary duct. Note that the opening is opposite the maxillary third cheek tooth. Water stained with new methylene blue dye is being injected causing distension of the duct.

orifice. The duct may be cannulated with a 3.5 French catheter, but it is difficult to access it from the oral cavity because the mouth cannot be opened widely enough. The mandibular duct empties at the sublingual caruncle. The polystomatic sublingual gland has numerous openings alongside the tongue.

Salivary calculi (sialoliths) are composed of calcium carbonate and are common in horses but rare in camelids. Salivary calculi may form in the gland and be carried through the duct system. If the calculus is tiny, it may pass into the mouth and cause no problem. A calculus may lodge in a narrow segment of the duct and continue to enlarge. With total obstruction, the salivary gland may swell because of the backed-up saliva. The duct may dilate as well, causing a cystic formation called a ranula. Ultimately, the affected salivary gland will atrophy.

Gastrointestinal Tract

The GI tracts of all the *camelidae* are basically the same except for size. Though it shows many similarities, there are some distinct differences compared to the tract of true ruminants. A diagrammatic comparison of the digestive tract of camelids and ruminants is illustrated in Figure 12.20.

Stomach

Camelids are foregut fermenters and show similarities to members of the suborder "Ruminantia," frequently referred to as "true ruminants." Both undergo a process termed "rumination" marked by eructation, regurgitation, insalivation, mastication, and deglutition, and therefore may be referred to as "ruminants." In this chapter, members of the suborder "Ruminantia" will be referred to as "true ruminants." Camelids, as with true ruminants, chew their cud and have foregut motility associated with the rumination process (See Chapter 3). However, camelids possess many unique characteristics placing them in a separate family (See Table 1.1). The evolutionary evidence suggests that true ruminants and camelids are not two separate taxonomic classifications along an evolutionary continuum, rather underwent parallel evolution from a common simple-stomached ancestor [12] (See Chapter 1).

The nomenclature for the camelid gastric anatomy is debated, and various anatomists have used different terminology [13]. The author has chosen to use the terminology of Vallenas et al. [14] Camelids are foregut fermenters but the anatomy of the forestomach of camelids differs significantly from true ruminants (Table 12.3) [12, 16–21]. The three compartments of the camelid stomach (C1, C2, C3) contrast with the four-compartmented stomach of the true ruminant. Figure 12.21 illustrates the relative volume of the gastric compartments of cattle, sheep and camelids. Though the forestomach compartments of the camelid are often thought analogous to those of the ruminant, there are distinct morphologic and physiologic differences [22–24]. Major differences include the absence of papillae in nonglandular regions of C1 and C2, and the presence of glandular tissue in all three gastric compartments. Motility patterns are markedly different in camelids, and the timing of rumination and eructation during the motility cycle also varies from that of the true ruminant stomach [21, 25, 26].

The evolution of the various regions of the stomach can be traced from the primordial simple stomach by the structure of the tunica muscularis (Figure 12.22) [19]. Basically, the anatomy of the stomach and intestines is the same in

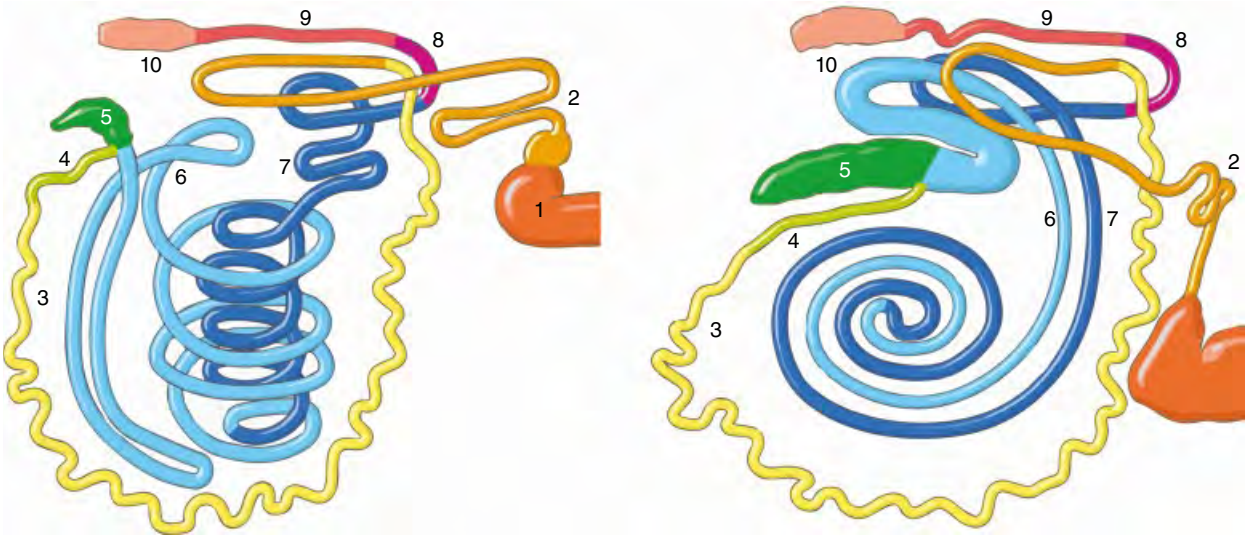


Figure 12.20 Diagram of the intestinal tract of a camelid (left) and that of a cow (right). Note the complex helical nature of the spiral colon and the rudimentary cecum of the camelid. 1. C3/abomasum, 2. Duodenum, 3. Jejunum, 4. Ileum, 5. Cecum, 6. Centripetal loops of spiral colon, 7. Centrifugal loops of spiral colon, 8. Transverse colon, 9. Descending colon, 10. Rectum.

Table 12.3 Comparison of ruminant and camelid stomach compartments.

	True Ruminant	Camelid	References
Gastric Compartments	4	3	[12]
Glandular distribution	Abomasum only	All compartments	[12]
Esophageal Groove Anatomy	Double lipped	Single lipped	[12, 14]
Gastric Maturation	Neonatal rumen (C1) proportionally small and becomes proportionally larger as animal develops fermentation capacity. Camelid develops mature fermentation capacity sooner than ruminant.		
	Rumenoreticulum	**C1**	
Function	Fermentation chamber	Fermentation, absorption of water, VFA and other solutes	[12]
Contents	Stratified (Gas, Fibrous, and Liquid layer)	Homogenous contents	
Esophageal Entrance	Enters between rumen and reticulum	Enters C1 only	[12]
Pillars	Pillars pronounced, horizontal	Pillars minimal, horizontal	[12]
Papilla	Papillated (Rumen only)	Non-glandular areas nonpapillated	[12]
Contraction frequency	2–4/min	3–5/min	[12]
Mucosal epithelial histology	Keratinized, stratified squamous	Nonkeratinized, squamous	[12, 14]
pH	5.8–7.0	6.4–7.0	[15]
	Omasum	**C2**	
Function	Reduce the particulate matter of the content of the omasum to a finer state	Fermentation, absorption of water, VFA and other solutes	[12]
Mucosal epithelial histology	Stratified squamous	Stratified squamous (1° crests); Glandular mucosa (2° crests)	[14]
pH	5.8–7.0	6.4–7.0	
	Abomasum	**C3**	
Function	Secretion of digestive juices that continue the digestive process	Absorption of water and solutes Proximal 4/5, digestive Distal 1/5, enzymes, acid	[12]
Mucosal epithelial histology	Glandular; entirely covered with enzyme and acid-secreting epithelium	Only distal one-fifth covered with enzyme and acid-secreting epithelium	[14]
pH	3.0	6.5 (cranial); <2–3 (distal)	

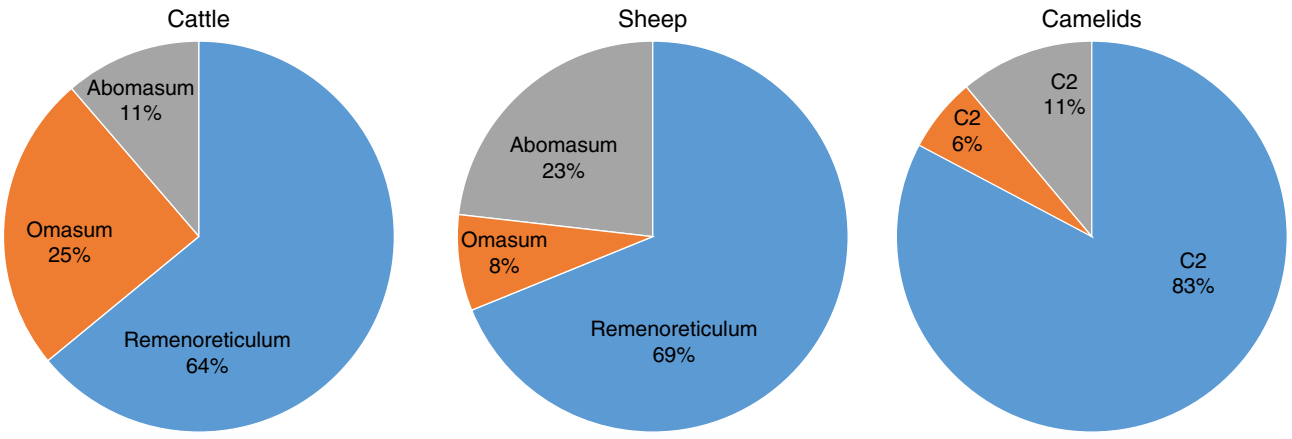


Figure 12.21 Relative volume of the gastric compartments of adult cattle, sheep and camelids.

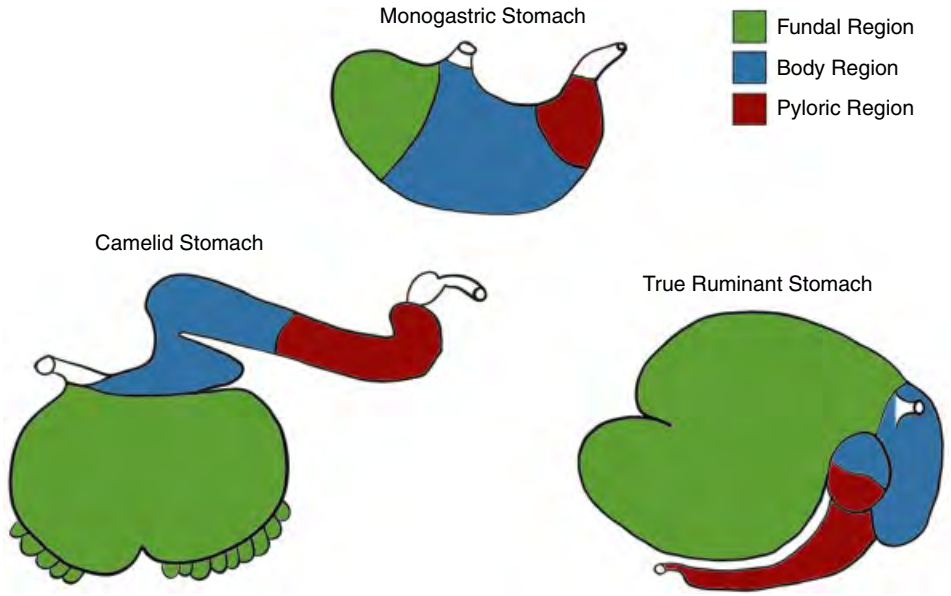


Figure 12.22 Comparative origins of different regions of the monogastric stomach, the camelid stomach, and the true ruminant stomach.

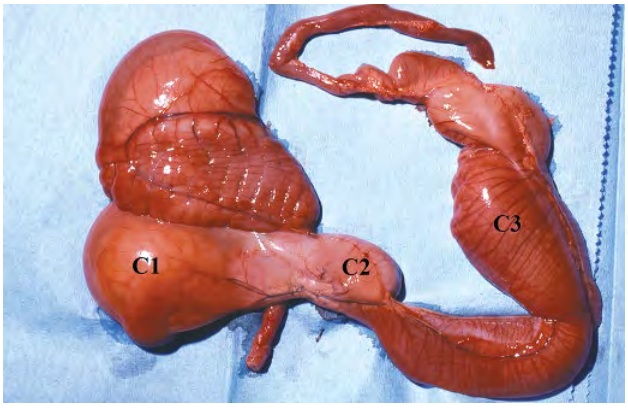


Figure 12.23 Stomach of a neonate llama. Similar to the rumen true ruminants, the first gastric compartment is proportionally smaller, and the last stomach compartment is proportionally the largest compartment in the neonate, similar to the abomasum.

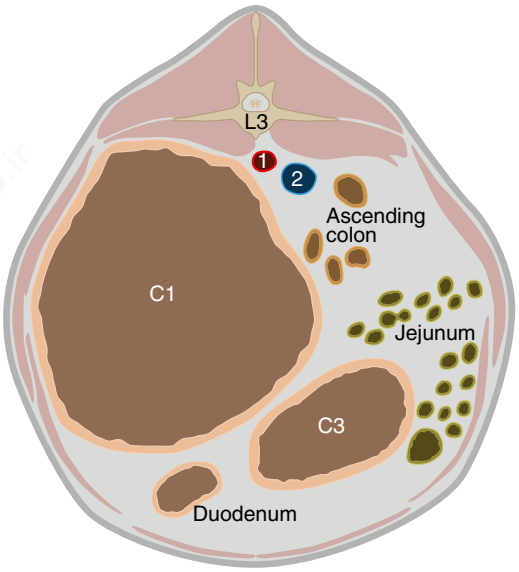


Figure 12.24 Diagram of the cross section of an alpaca abdomen taken at the level of the third lumbar vertebrae. Note the C1 compartment occupies the vast majority of the left side of the abdomen. C3 is seen on the ventral right abdomen. The jejunum and the ascending colon also occupy the right side of the abdomen. 1. abdominal aorta, 2. caudal vena cava.

all camelids. Similar to the true ruminant, the camelid neonate is physiologically a monogastric and has a poorly developed first compartment and a large true stomach (Figure 12.23). However, the juvenile camelid matures into an efficient foregut fermenter faster than juvenile true ruminants. Table 12.3 compares the true ruminant and camelid gastric compartments.

C1 occupies the majority of the left abdomen, while the other stomach compartments and GI viscera lie primarily on the right side (Figure 12.24). A cranial and caudal sac are weakly divided by a horizontal pillar. The esophagus enters C1 on its craniodorsal midline. C2 is situated on the right craniodorsal surface of C1. The tubular C3 arises on the cranial, medial aspect of C2. It curves to the right, caudal and ventral to the liver, and lies on the right surface of C1 in the right midventral abdomen (Figures 12.25 and 12.26).

The nonglandular mucosal surface of C1 and C2 is not papillated and is comprised of unkeratinized, stratified squamous epithelium [27–29]. The ventral surfaces of both the cranial and caudal sacs of C1 contain saccules (Figure 12.27) lined by papillated glandular mucosa [14]. The epithelium varies slightly from the orifice to the depths of the saccule, but it is generally a mucinous glandular

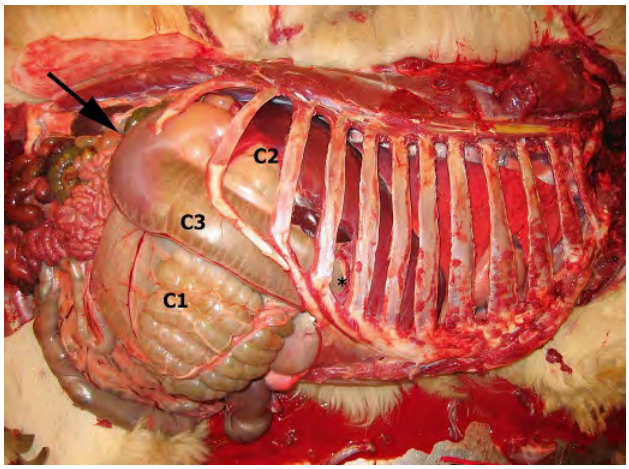


Figure 12.25 Stomach of a llama as viewed from the right lateral aspect. The right side of the image is cranial and the left side of the image is caudal. All three stomach compartments are visible. C3 is in the foreground of this right view and is tubular. C1 has prominent saccules and lies predominately in the left hemiabdomen. The orad aspect of C3 arises on the cranial, medial aspect of C2 (asterisk) and ends at the pyloric region. The distal (aborad) aspect of C3 is glandular and thicker (arrow). *Source:* Photo courtesy of Jeff Lakritz.

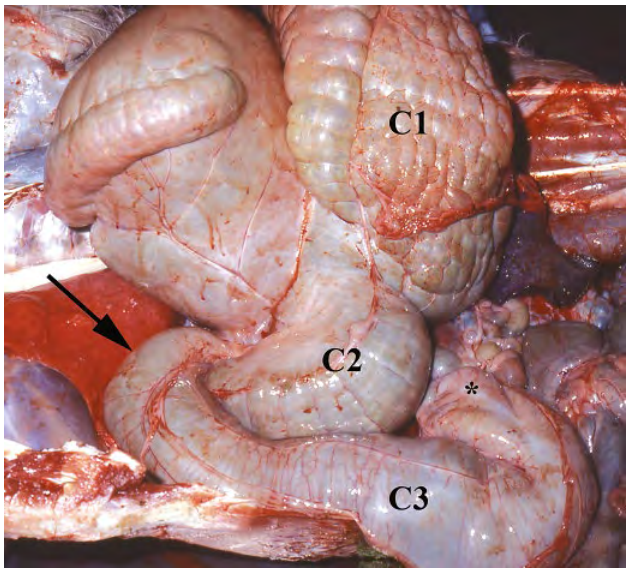


Figure 12.26 Stomach of a llama. The left side of the image is cranial and the right side is caudal. All three stomach compartments are visible. C3 is in the foreground of this right view and is tubular. C1 has prominent saccules and lies predominately in the left hemiabdomen. The orad aspect of C3 arises on the cranial, medial aspect of C2 (arrow) and ends at the pyloric region (asterisk).

epithelium. In the camel, glandular epithelium is found only on the bottom and lower walls of the saccules.

In earlier studies, it was reported that this glandular epithelium was the source of sodium bicarbonate [27], but later studies, using a Pavlov pouch, did not confirm this [30, 31]. Current studies indicate that the glandular epithelium also provides for rapid absorption of water and solutes [17, 28].

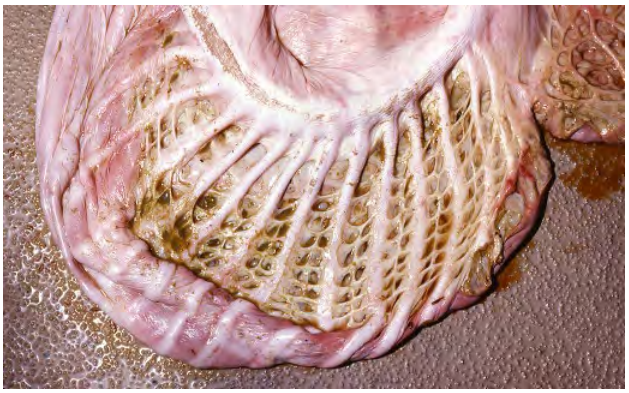


Figure 12.27 Glandular saccules of C1.

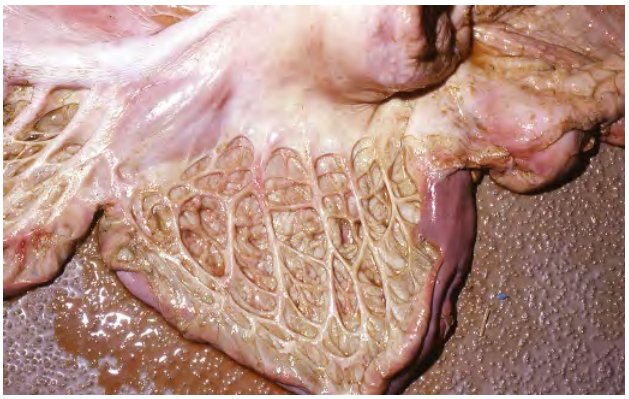


Figure 12.28 Mucosal surface of C2. Saccules along the majority of the surface of C2 are lined with glandular epithelium. Non-glandular epithelium lines the lesser curvature at the esophageal groove.

The mucosal surface of C2 is glandular except for a small area on the lesser curvature that constitutes the esophageal or ventricular groove (Figure 12.28). The glandular area is subdivided by a series of intersecting crests that produce a retiform pattern and is covered by a papillated glandular mucosa. The retiform pattern is not analogous to the pattern seen in the reticulum of ruminants [23, 32]. The primary crest margins are covered by stratified squamous epithelium extending from the ventricular groove, but the secondary crests are covered with glandular mucosa [23, 32]. The depressions of C2 are not deep and do not evert during the contraction phase of the motility cycle of the stomach.

Though grossly C3 appears as a single anatomic gastric compartment, an understanding of the physiology requires subdivision of the compartment into fifths. The mucosa of C3 is entirely glandular (Figure 12.29) however, the orad

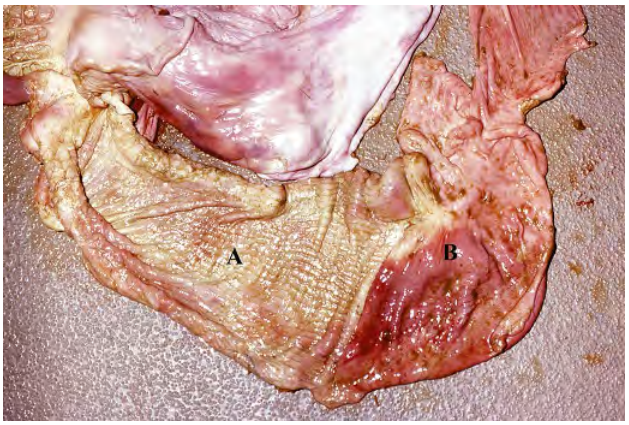


Figure 12.29 C3: (A) mucinous glandular tissue lines the proximal (orad) 4/5 of the compartment, (B) the terminal (aborad) 1/5 of the compartment contains true gastric glands and the mucosa is reddish-brown and the walls are thicker.

four-fifths of C3 is lined by a mucinous glandular tissue, similar to the glandular tissue of C1 and C2, while the most aborad (terminal) one-fifth contains true gastric glands (Figure 12.30).

The first one-fifth has a retiform pattern with short crests and shallow depressions on the lesser curvature and nonpermanent folds on the greater curvature. The mucosa of the middle three-fifths consists of approximately 50 permanent longitudinal pleats.

The terminal one-fifth of C3 contains the true gastric glands. The mucosa is reddish-brown, in contrast to the lighter pink of the proximal four-fifths. The wall of this area is thickened and the mucosal surface smooth. Digestive enzymes and acid are secreted by these glands. The pH in the cranial segment of C3 is 6.5, which decreases to less than 2 in the terminal fifth [33]. In OWCs, the mucosa of the terminal fifth of C3 may have coarse rugae [32].

The camel stomach is basically the same as that of the SAC except for size (Figures 12.31 through 12.33).

Gastric Motility

The basic contraction cycle of the stomach of the llama and guanaco was established by Vallenas [34, 35]. A contraction cycle is initiated by a single rapid contraction in C2, followed quickly by a contraction of the caudal sac of C1. The direction of the contraction is from caudal to cranial. Next, the cranial sac of C1 contracts in the reverse direction, cranial to caudal.

In the resting llama, the sequence of contractions of C1 is repeated six to eight times before another contraction of C2 occurs. The cycle is from one contraction of C2 to the next (of C2), and the average length of a cycle is 1.8 ± 2 minutes.

To the clinician, this equates to three or four audible contractions per minute in a resting animal and four to five contractions during feeding or the immediate postfeeding state. The amplitude of the contractions also increases during feeding.

The contents of the glandular saccules of the caudal sac are extruded just prior to the contraction of the caudal sac of C1. The contents of the caudal sac and dorsal area of the cranial sac of C1 are quite dry (see Figure 12.34). The more fluid and smaller particulate matter is found in the ventral cranial sac. The motility pattern tends to dump ingesta back and forth between the cranial and caudal sacs. This is

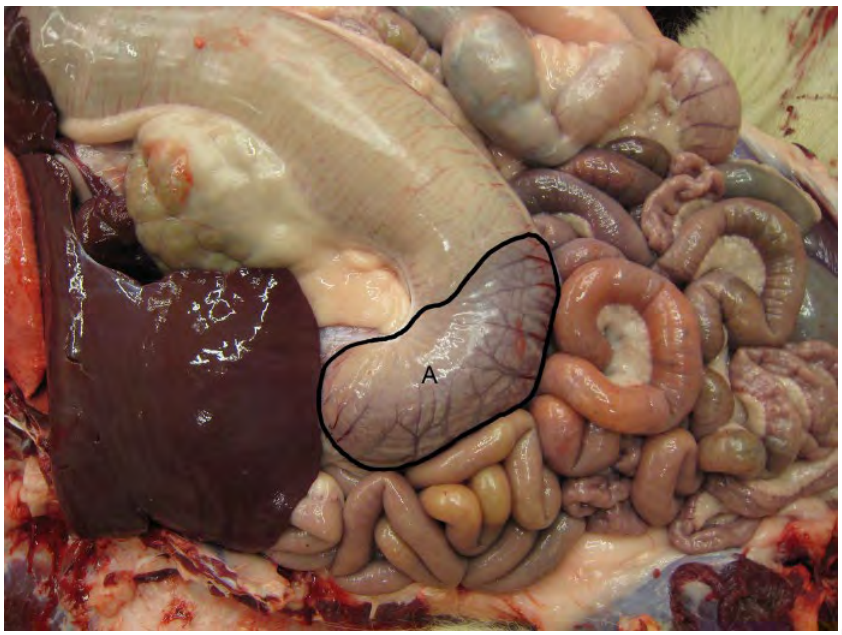


Figure 12.30 Serosal view of C3. The acid secreting portion is the terminal 1/5 of the stomach. The walls in this area are thicker and slightly darker as apparent in this photograph. *Source:* Photo courtesy of Jeff Lakritz.

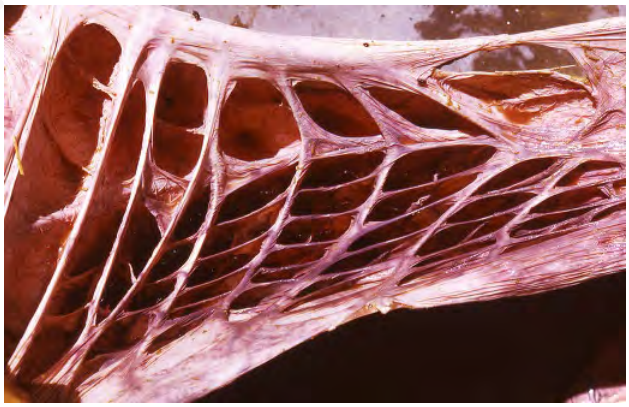


Figure 12.31 Camel stomach, C1.

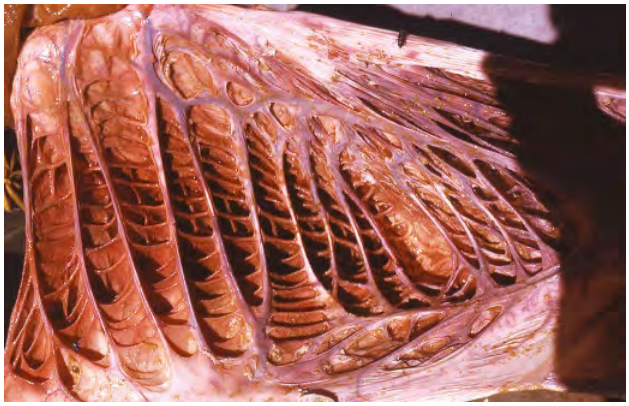


Figure 12.32 Camel stomach, C2.

Figure 12.33 Camel stomach, C3.

a highly efficient mechanism for mixing the ingesta and enhancing the fermentation process. The ingesta in C3 is rather dry; a result of the water absorption that takes place in the cranial four-fifths.

Volatile fatty acids (VFA) are rapidly absorbed from C1, perhaps at two to three times the rate of absorption from the rumen of sheep and goats [33, 36, 37]. Similar absorption of VFA occurs from the proximal four-fifths of C3.

Figure 12.34 Homogeneous ingesta, characteristic of C1. The contents of the dorsocranial sac are quite dry.

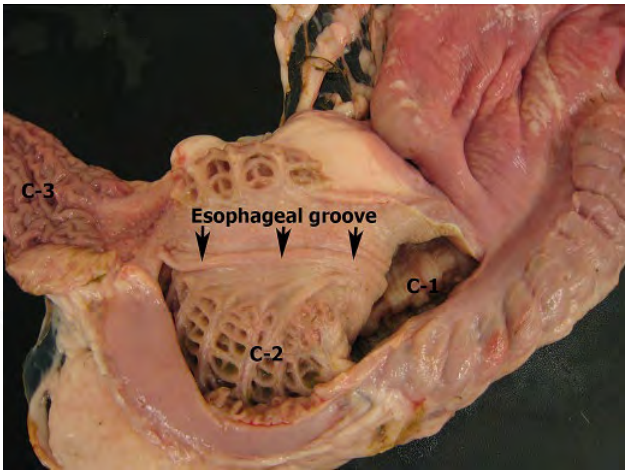


Figure 12.35 Camelids' esophageal groove has one lip and extends from C1 into C3. *Source:* Photo courtesy of Jeff Lakritz.

C1 microflora and fauna are described by Harmeyer and Hill [38] and Sillau et al. [39] and in Chapter 3.

Camelids are capable of adapting to low-protein diets by recycling the urea produced as an end product of nitrogen metabolism [40, 41]. Urea is used by stomach bacteria to form their own protein. The bacteria are subsequently digested and the protein absorbed further along the intestinal tract and utilized by the camelid. The presence of VFA enhances the efficiency of urea recycling; thus, it is desirable for the camelid to consume a source of carbohydrate, if at all possible, when on a low-protein diet. When food is withheld from a llama for 48 hours, the plasma-urea nitrogen (BUN) concentration will increase indicating that less urea is being recycled in the carbohydrate-deprived camelid.

Camelids have an esophageal groove, but it is less well developed compared to that of true ruminants (Figure 12.35) [42]. There is only one single lip as contrasted with two in the ruminant. The functional groove extends

from the cardia to and through the lesser curvature of C2 and functions to divert milk in the neonate directly from C1 into C3 (see Chapter 20).

Transit times for ingesta have been studied [43], but no conclusions have been reached. A study on eructated gas expulsion in alpacas was conducted in 1968 [44].

Intestine

Small Intestine [45–47]

There is a dilated ampulla at the entrance of the duodenum which is similar in size to the aborad portion of C3. The duodenum quickly decreases in diameter as it travels distally, coursing dorsally on the right side of C1 to the dorsum of the abdominal cavity just caudal to the liver. The jejunum is folded around the root of the mesentery, in the right caudal abdomen. The ileum begins ventrally and courses medially and dorsally to enter the large intestine at the cecocolic orifice.

Large Intestine [46]

The cecum lies approximately on the midline and is directed caudally toward the pelvic inlet, or it may curve ventrally and laterally to the left. The camelid colon is similar to that of the bovine. The ascending colon begins as a proximal loop that courses cranially and ventrally to enter the spiral loop (Figures 12.36 and 12.37). The most proximal loop of the ascending colon is loosely attached by a mesentery to the more compact spiral colon. There are five and a half centripetal coils (including the proximal loop) and four and a half centrifugal coils [48]. There are only two coils in bovines and three in ovines. The centrifugal coils are buried within the spiral, deep to the centripetal coils. The spiral colon lies on the midventral abdominal wall and is likely to be the first organ encountered on a ventral midline laparotomy.

The colon narrows from a 5-cm diameter at its beginning at the proximal loop to 2 cm within the first centripetal coil of the spiral loop. The spiral colon is the primary site of fecalith impaction in camelids.

The distal (centrifugal) loop of the ascending colon is juxtaposed to the proximal (centripetal) loop. The transverse colon passes from right to left cranial to the mesenteric artery and continues on as the descending colon to the rectum and anus.

The intestinal length appears to vary significantly between camels and SACs. One source cites the small intestinal length of the dromedary camel to be approximately 40 m in length [49] compared to 12 m for the small intestines of an adult llama. And the length of the large

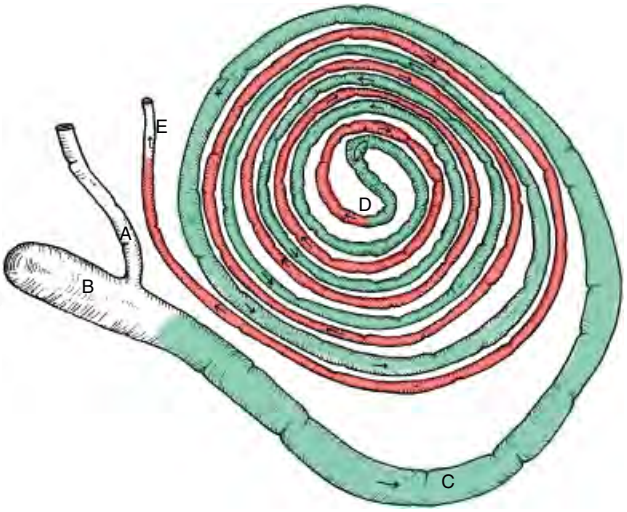


Figure 12.37 Diagram of the spiral colon of a llama. The centripetal loops are colored green and the centrifugal loops are colored red. (A) Ileum, (B) cecum, (C) proximal loop of spiral colon, (D) central flexure, (E) transverse colon.

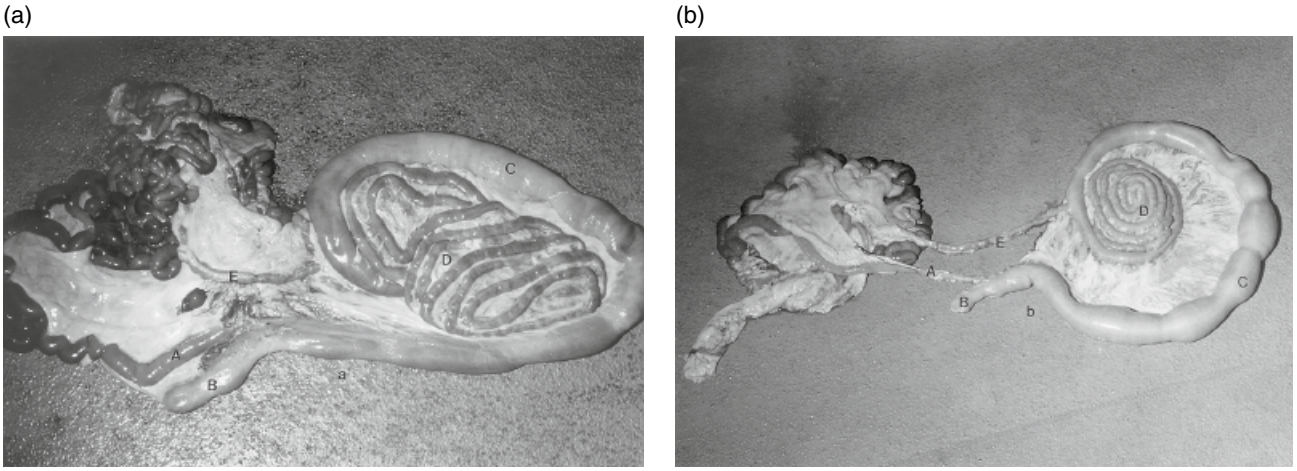


Figure 12.36 Intestines of a llama. (A) Ileum, (B) cecum, (C) proximal loop of spiral colon, (D) spiral colon, (E) transverse colon.

intestine was reported to be approximately 20 m for a dromedary [49] compared to 7.5 m long for a llama.

Camelids void feces in pellet form. These begin to form in the proximal spiral colon. Desiccation of the pellet continues throughout the colon. OWCs have the capacity to pass totally desiccated feces when water intake is restricted. The llama fecal pellet varies with the size of the animal, from 7 × 12 to 20 × 30 mm. There is generally a sharp teat on one end of the pellet. Some individuals pass teardrop-shaped pellets (7 × 12 × 20 mm). The pellets of a healthy llama may be voided as separate pellets or in a compressed cylindric mass, which easily breaks apart into individual pellets. The color of the stool varies with the diet but is generally greenish-brown, blackening after voiding [8]. The stool of the dromedary camel is passed in irregularly shaped pellets. A medium-sized female may void pellets that range in size from 15 × 15 × 15 to 20 × 25 × 25 mm. A large male may pass pellets as large as 25 × 30 × 35 mm.

SACs generally use communal dung piles (Figure 12.38), however OWCs do not. In inclement weather, a dung pile may be started in a barn and the use of it continued into summer. Numerous methods have been tried by owners to change the location of a dung heap. In the Arabian desert during the summer, freshly passed feces can be used for fuel. The fecal pellets of SACs are also used for fuel after they are allowed to dry further [8].

Liver

Like other species, the camelid liver has multiple lobes, (Figure 12.39) and the liver is located entirely on the right side [50]. The dorsocaudal border extends caudal to the cartilage of the last rib. The liver covers C2 and C3 of the stomach, though there is a notch on the cranioventral border where a small portion of C3 is visible. The caudal border of the normal camelid liver is fimbriated (Figure 12.40).

Figure 12.39 Liver of a llama.

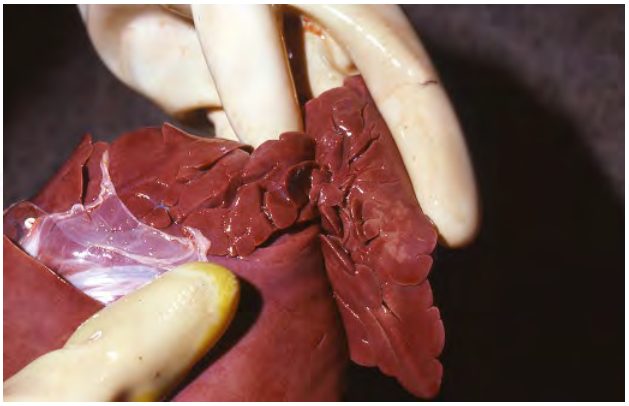


Figure 12.40 Fimbriated caudal border of a SAC liver.

Figure 12.38 Dung pile used by llamas for urination and defecation. SACs will use communal dung piles.

Bile Duct

The collecting system ends in the major bile duct, 4 mm in diameter, at the caudal aspect of the liver. The duct is 3–4 cm long and penetrates the antimesenteric surface of a loop of duodenum that courses toward the liver. The opening into the duodenum is 16–20 cm from the pylorus. The gallbladder is absent except in rare instances.

Clinical Signs Associated with Digestive Disorders

Diagnosing digestive system disorders can be challenging and frustrating even for experienced veterinarians. Clinical signs are often non-specific and can result from problems unrelated to the digestive system. Some digestive functions like GI motility are one of the first physiologic processes to be suppressed with multiple bodily dysfunctions. Even excessive pain and stress can cause dysfunction of the GI tract. Like many prey species, camelids are frequently stoic animals and can mask discomfort until the disease is chronic which leads many conditions to go unnoticed and undiagnosed until late in the course of the disease.

Determining if a condition can be managed medically or requires surgical intervention is one of the fundamental questions that should be ascertained early in the diagnostic workup. Exploratory laparotomy or laparoscopy may be employed in cases where this question is not clearly defined. See section on abdominal and gastrointestinal surgery for more information on techniques and procedures.

Signs of colic that are unresponsive to treatment, low serum chloride, high serum lactate, abnormal intestinal distension, lack of progressive intestinal motility, and lack of fecal production can be signs suggesting that surgical exploration of the abdomen is indicated. Determining whether an animal requires surgical intervention is frequently challenging, and clinicians inexperienced with camelids may benefit from a flow chart. Though no algorithm can encompass all of the considerations that must be considered with complex biologic systems, Figure 12.41 is a flowchart that might be useful for management of camelids with GI disease.

Anorexia

Anorexia is the lack or loss of appetite for food. It is a vague clinical sign that can stem from multiple primary etiologies. It is challenging to differentiate between a lack of desire to eat or a physical inability to eat due to weakness or a disorder that prevents eating.

Physical pain during mastication is a common cause of anorexia in camelids. Dental pain, stomatitis, temporomandibular arthritis, oral or pharyngeal abscesses, and glossitis can cause severe pain and prevent an animal from eating. Facial nerve paralysis, brachygnathism, or dental malocclusion may or may not result in physical pain but still might inhibit an animal from being able to prehend. Colic, resulting from lesions of the GI or reproductive tracts, can also lead to anorexia. Central nervous system (CNS) injury, anemia, septicemia, pyrexia, and metabolic derangements can also lead to anorexia in camelids. Changes in the social order among individuals, unpalatable feed, and inaccessibility to food are external factors that may also affect intake.

Anorexia can result in hypokalemia due to low potassium intake. Hypokalemia can be exacerbated if an animal is given parenteral fluids lacking in potassium, if potassium is lost due to vomiting or diarrhea, or if an animal is given potassium-wasting diuretics or corticosteroids. Clinical signs of hypokalemia include muscular weakness, depression, and altered cardiac function. These are non-specific signs often seen in an ailing patient and a case must be carefully evaluated to determine the cause. Serum chemistry analysis may be necessary.

Dysphagia

Dysphagia refers to difficulty or inability to swallow. Dysphagia may be the result of hypoglossal or glossopharyngeal nerve paralysis, pharyngitis, esophagitis, pharyngeal abscesses, pharyngeal tumors, esophageal obstruction (choke), megaesophagus, gastric overload, bloat, infectious diseases (rabies, botulism), or congenital defects such as palatal defects. In newborn crias, chonal atresia can cause an inability to suckle because the cria cannot eat and breathe simultaneously.

Signs of dysphagia include excessive drooling, retention of feed in the oral cavity, a fetid odor from the oral cavity, gagging, and retching. These signs may elicit panic in the owner and can lead to other problems like aspiration.

Regurgitation and Emesis

It is important to differentiate between regurgitation and emesis. Regurgitation may be voluntary in camelids. It is used as a threat or as a defense against aggression between males or by the female if she is pregnant or unwilling to submit to the male. This behavior can be anticipated if the ears lie back over the neck, followed by a gurgling noise in the pharyngeal region as the camelid prepares to eject the bolus. On the left side, the bolus can be seen traversing the cervical region. Stomach contents can be spewed a distance of 3 m quite accurately.

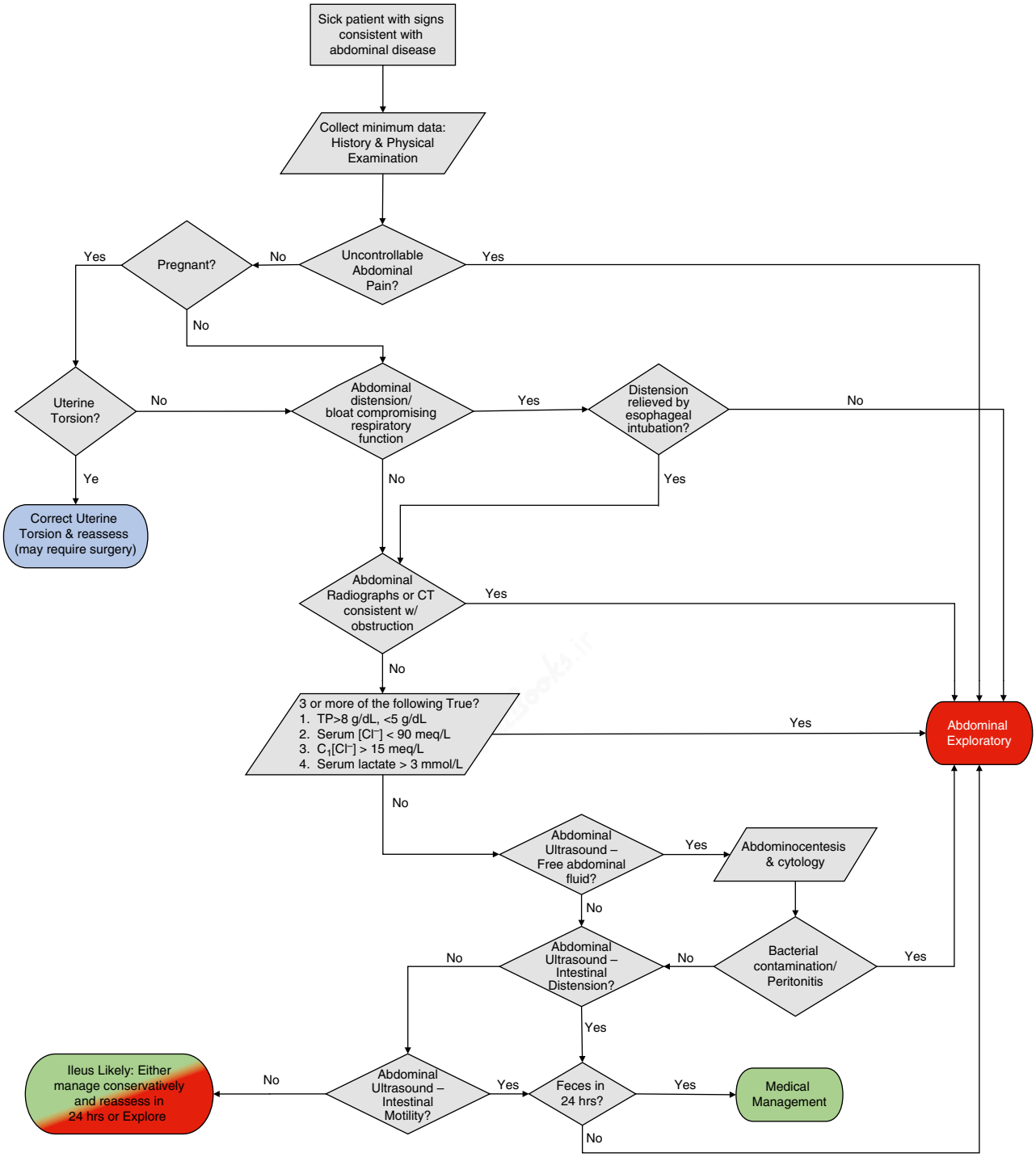


Figure 12.41 Decision-making algorithm for performing abdominal surgery in camelids.

Active regurgitation may occur as a result of esophageal stimulation as a gastric tube is passed or from laryngopharyngeal stimulation when an endotracheal tube (ET) is inserted. Passive regurgitation may occur from relaxation of the stomach cardia during anesthesia or from improper positioning during recumbency. If possible, the animal should lie on its right side. Abdominal pressure, either externally applied or as a result of bloat or other abdominal distention, may also cause regurgitation.

Emesis is the involuntary projection of stomach contents by a reflex action, and ingesta is ejected by a strong contraction of all the abdominal muscles and antiperistaltic contractions of the esophagus. It consists of an initial deep inspiration followed by closing of the glottis to prevent aspiration of ingesta. At the same time, the soft palate should automatically be elevated to seal off the nasopharynx, however, forceful emesis can push ingesta into the nasal cavity, causing rhinitis, obstruction, or aspiration. Emesis in camelids should be considered a grave sign and commonly results in sequella-like aspiration.

Some of the causes of emesis include overloading of C1, gastritis, diaphragmatic hernia, arsenic poisoning, partial esophageal obstruction, and the ingestion of poisonous plants. A list of such plants includes oleander (*Nerium oleander*), corn lily (*Veratrum californicum* or *V. viridis*), lily of the valley (*Convallaria majalis*), foxglove (*Digitalis purpurea*), sneezeweed (*Helenium* spp.), and various members of the Ericaceae family, all containing the same andromedotoxin (a glycoside), including rhododendrons and azaleas (*Rhododendron* spp.), laurel (*Leucothoe* spp.), labrador tea (*Ledum* spp.), and kalmia (*Kalmia* spp.) (see Chapter 22).

The clinical evidence of regurgitation or emesis is obvious. The management of these conditions depends on correction of the primary cause. Any obstruction should be corrected and irritants removed from the stomach, which may require gastrostomy. In addition, C1 electrolyte and microbial balance should be reestablished.

Abdominal Distention

Abdominal distention is not pathognomonic for one disease and can be caused by anything that takes up excessive space within the abdomen. It may be caused by an enlarged GI structure like gastric or intestinal distention. An enlarged uterus due to pregnancy or excessive abdominal fat or abdominal fluid may lead to distension. Abdominal distension is commonly referred to as "bloat." In ruminants, "bloat" refers to rumen tympany, a condition where the rumen is distended with gas that is unable to be eructated. Though this condition rarely occurs in camelids due to differences in their gastric physiology, the author prefers not to use the term "bloat" to describe abdominal distension.

Abdominal distention is frequently caused by overeating, late pregnancy, or excessive abdominal fat. However, it may be a sign of a more serious problem like a gastric or intestinal outflow obstruction, intraabdominal pathology, or uterine disease. A thorough physical examination may lead to identification of the cause. Diagnostics such as biochemistry profile and abdominal ultrasound (US) can also be useful. Due to their smaller size, a thorough rectal examination might not be possible in camelids as in cattle. Regardless of the cause, excessive or prolonged abdominal pressure pushes the diaphragm forward and can cause dyspnea. Additionally, compression of the abdominal vessels interferes with circulation, and shock may ensue (Figures 12.42).

Diarrhea

Diarrhea is the condition where feces are irregularly formed with a higher water content than normal and are passed more frequently than normal. Camelids have adapted to arid environments by maximally dehydrating their feces to a water content of approximately 50%. This is in comparison to cattle which have around an 85% water content [3]. The camelid colon is very efficient at extracting water, passing only small, dehydrated pellets. Even significant pathology affecting only the proximal GI tract may not result in

(a) (b)

Figure 12.42 Abdominal distension in an alpaca. (a) Left Lateral view. (b) Top view.

diarrhea because a healthy colon can effectively dehydrate the ingesta. Diarrhea in camelids is usually a sign that the intestinal pathology is widespread, or the colon is involved. However, diarrhea remains a non-specific finding and its presence does not indicate a specific etiology.

In neonates, the major cause is dietary upset, usually seen in orphan neonates experiencing rapid changes in the milk supplied. Overfeeding is also a factor. Infectious causes include colibacillosis, coronavirus, and rotavirus infections. Coccidiosis may be seen within the first few weeks of life. Poor sanitation contributes to the exposure of the neonate to opportunistic or pathogenic microorganisms. If the neonate failed to receive the transfer of adequate immunoglobulins, the animal is a prime target for infection. Septicemic neonates commonly have diarrhea.

In mature camelids, enteritis is the most common cause of diarrhea and may be initiated by clostridial toxemia, parasitism, gastric overload (C1 acidosis), or toxicities (arsenic, copper, lead, molybdenum). Additional causes are overhydration, consumption of too much lush feed, and sudden change in feed. Nondigestive-tract causes of diarrhea include excitement, hepatopathy, and poisoning from organic phosphate insecticides. Infectious diseases causing enteritis, hence diarrhea, include salmonellosis, paratuberculosis, colibacillosis, rinderpest, and tuberculosis.

Constipation

Constipation is the abnormally infrequent or difficult evacuation of feces that have remained in the rectum and are usually more firm or dry than usual. The term "obstipation" is a more severe condition and implies intractable constipation. In the usual clinical sense, constipation refers to any slowness or absence of passage of feces. It is important to differentiate between true constipation, in which feces pass into the colon and rectum, but the rate of passage is slow and fluid is absorbed, making the feces hard and dry, and the more serious obstipation, in which an obstruction or other cause prevents passage of feces into the terminal colon.

Because camelids pass a relatively dry fecal pellet, the dryness of feces may be a difficult sign to interpret. The constipated llama is not likely to be straining. When feces are collected from the rectum, the pellets or the fecal mass may be covered with mucus, giving the feces a shiny appearance.

Some of the causes of constipation include malnutrition or starvation, dehydration, painful condition around the anus, pelvic abscess, pelvic fracture, pelvic tumors, acorn poisoning (*Quercus* spp. tannins), paralytic ileus, general debility, spinal injury, lack of exercise (downers), and toxicosis (lead, fluorine, chronic zinc poisoning).

Ileus

Ileus is the lack of the normal progressive peristaltic movement of the intestines. Ileus can be mild or severe, segmental, or complete. This motility problem can lead to or result from a mechanical obstruction. Even in the absence of a true (mechanical) obstruction, ileus can be indistinguishable from a mechanical obstruction as this condition creates a physiologic obstruction that will prevent the passage of ingesta aborally.

Ileus may be caused by severe abdominal inflammation (peritonitis), intrinsic disorders of bowel motility, ischemia, or overstretching of the intestine from ingesta or gas. Metabolic derangements like acidosis, and hypokalemia may also initiate ileus.

C1 Atony

Atony of the first stomach compartment can be caused by numerous disorders including grain overload, simple indigestion, sudden changes of feed, prolonged antimicrobial therapy, spoiled feed, gastric ulceration, and obstruction of the cranial intestinal tract [51]. Other causes might be hypomagnesemia, septicemia, and other types of colic.

Microbial activity is crucial to proper stomach activity, and any condition that destroys normal microorganisms predisposes to gastric atony. Atony is usually accompanied by anorexia.

Lack of motility is generally ascertained by auscultation with a stethoscope. The contractions of C1 are not as pronounced as contractions of the rumen in the ruminant, so palpation in the left paralumbar fossa is not helpful. The rate and rhythm of C1 contractions vary with the proximity to feed ingestion. Recent ingestion of a meal will cause the contractions to be more frequent (two to four per minute, with no intervals between in llamas). In the normal llama, the only abdominal sounds are from C1. With obstruction or delay of movement of ingesta along the intestinal tract, gas formation may mix with fluid and produce intestinal borborygmus.

Stomach atony is usually only a sign of other clinical problems, so medical management of atony should deal with the primary disorder. However, prolonged atony of C1 (48–72 hours) may initiate a change in the pH of the ingesta which, in turn, will destroy the microflora and fauna. Ultimate reestablishment of gastric motility may necessitate transfaunation.

Colic

Colic refers to abdominal pain and is a non-specific clinical sign much like fever, tachycardia, or depression. Colic can result from multiple etiologies and a thorough

examination may be necessary to diagnose the primary problem [52–60]. Most digestive tract disorders, at one time or another during the course of the disease, produce colic pain. Non-GI lesions like uterine torsion, cystitis, urethral obstruction, ruptured bladder, pleuritis, peritonitis, liver pathology, back pain, and certain infectious diseases (salmonellosis, rabies, anthrax) frequently have colic as a clinical sign. Severe thirst or starvation may, in the early stages, cause stomach contractions that will be expressed as painful colic. Neonatal colic may be caused by retained meconium, intestinal atresia, and hernial incarceration of an intestinal loop. Other conditions that may mimic signs of colic include normal rolling behavior, weakness, incoordination (insecticide and heavy metals poisoning, head injuries, spinal cord compression), septicemia, and arthritis.

Colic has been a much-studied condition in horses, and it is known that ruminants with colic present much differently than their equine counterparts. Due to similarities in their anatomy, camelids are susceptible to similar conditions as ruminants, however, camelids seem to be more like the horse in expression of the pain of colic. Signs include groaning, bruxism, getting up and down, rolling, refusing to get up, kicking at their abdomen, peculiar stances, pressing into a corner, looking back at their abdomen, frenzy, depression, anorexia, arched back, tenesmus, tense abdomen, stomach atony, increased heart rate, frequent passage of small amounts of urine, tenseness of facial muscles, and a pained expression of the eyes (Figures 12.43 and 12.44).

The sensory response is via the afferent visceral fibers accompanying the sympathetic pathway. Normal GI mucosa is insensitive to touch and sharp pain like cutting, pinching, and tearing. However, GI pain arises from stretching of the muscular layer and serosa, as occurs with distention of the viscus or with powerful muscular contractions [61].

Figure 12.43 Pressing and abnormal position of a llama with colic.

Figure 12.44 Depression and recumbency of a llama with colic.

Acute inflammation or vascular engorgement decreases the pain threshold. As a result, mild stimuli that would not initiate a pain response in the normal bowel cause pain. Tissue hypoxia also decreases the threshold; thus, obstructive lesions compromising circulation cause significant pain. Excessive production of acid in C1 may produce a direct inflammation of the gastric mucosa and initiate a pain response. Acid may also stimulate muscle spasms.

Diseases of the Digestive System

Lip and Tongue Disorders

Laceration of the lips and tongue may be caused by barbed wire or other sharp objects in the environment or by dog bites. The lips may also be lacerated by the incisor teeth as a result of a blow to the muzzle. The camelid tongue is protected because it does not protrude through the mouth but may be lacerated by the large canines in the male. Labial swelling may result from dependent edema from hypoproteinemia or anemia, cellulitis, a tumor, constriction of venous drainage, contusion, or rattlesnake bite [62].

Erosions and ulcers of both the external and internal surfaces of the lips and the tongue may be caused by vesicular diseases (foot-and-mouth disease, vesicular stomatitis), rinderpest, or grass awns (yellow bristle grass, *Setaria lutescens*; foxtails, *Hordeum* spp.). Proliferative lesions of the lips may be caused by contagious ecthyma (Chapter 9).

Cranial nerve deficits can cause paralysis of the lips or tongue. Paralysis of the lips may follow damage to the facial nerve, as in listeriosis (Chapter 19). Hypoglossal nerve injury can lead to tongue paralysis. Secondary trauma to the tongue may result if the tongue protrudes from the mouth.

Stomatitis

The term "stomatitis" encompasses the inflammatory response to injury or disease of any oral tissue. It includes glossitis (inflammation of the tongue) or gingivitis (inflammation of the gums and gingiva).

Etiology

Any of the factors causing lip problems may also affect the mouth. Additional damaging agents may include foreign bodies, candidiasis, actinobacillosis, trauma from gastric tube passage, cellulitis arising from focal infections within the mouth, and developmental defects (malocclusion).

Signs

Signs of oral ailments include anorexia, peculiar chewing motions, excessive salivation, halitosis, hyperemia of the mucosa, erosions, ulcers, and vesicles.

Diagnosis

Careful physical examination of the mouth usually allows visualization of the lesions, though it may be difficult in SACs because the mouth is narrow and will not open widely. The camelid usually resents manipulation of the mouth, so sedation may be necessary to fully examine the mouth and carry out diagnostic procedures, including inspection, cultures, and biopsy. Because some oral diseases are foreign animal diseases, a definitive diagnosis is mandatory. Any case suspected of being a vesicular disease should be reported immediately to state or federal regulatory agencies. Procedures necessary to arrive at a definitive diagnosis will then be conducted by them.

Treatment

Diagnosis and correction of the primary condition is necessary. Since many oral lesions manifest as anorexia, supportive care includes providing soft, palatable feed. Using a dose syringe with a long nozzle, flush the mouth with water to cleanse it of feed particles or exudate. Antibiotics and anti-inflammatories should be provided as needed to treat bacterial infections and inflammation.

Oral Infections

Etiology

Oral abscesses may be caused by numerous opportunistic bacteria. Direct inoculation from the oral cavity that enters through a break in the oral mucosa is the mostly likely route of infection. *Trueperella* (formerly *Arcanobacterium) pyogenes* is the organism most frequently cultured from oral abscesses; Streptococci, staphylococci, actinobacillus, fusobacterium, and actinomyces have also been encountered.

Infections within the oral cavity may spread to the adjacent bones of the mandible or maxilla. Dental infections are commonplace in SACs and are most commonly thought to be the result of spread of normal commensal organisms from the oral cavity.

Necrobacillosis is a serious problem in SACs in South America caused by *Fusobacterium necrophorum*. Necrobacillosis causes severe abscessation and necrosis of the soft tissues and the bones of the mandible and maxilla.

Dental and Boney Disease

Dental Disease and Dental Care

Dental pathology is a common ailment among New World [59, 63, 64] and OWCs [65, 66]. Camelids share similar dental pathology to other herbivores. Possible conditions include dental plaque, discoloration of the teeth, alveolar periostitis, pulpitis, tooth fractures (Figure 12.45), mandibular fractures, sharp enamel points caused by malocclusion, excessive wear, elongated teeth associated with superior brachygnathism, (Figure 12.46) and developmental defects causing malocclusion, tooth abscesses, mandibular osteomyelitis, and sequestra. Often, the etiology is multifactorial and idiopathic. Dietary factors, genetics, and environmental factors like stocking density have been advocated to play a role in dental disease [67].

The signs of dental problems may be non-specific and include inappetence, and weight loss or point to a more localized disease process such as abnormal chewing, dropping of a cud, boney swelling of the mandible or maxilla, mandibular fistulae, and nasal discharge.

Figure 12.45 Fractured mandibular cheek tooth.

Examination of the teeth is challenging, but necessary, to accurately diagnose dental disease. A thorough oral exam is necessary to check for loose or missing teeth or packing of cud in the mouth which can resemble boney swelling.

Figure 12.46 Alpaca cria with superior brachygnathism. This congenital defect leads to malocclusion and overgrowth of the lower incisors. *Source:* Photo courtesy of Dr. Andrew Muir.

Figure 12.47 is an example chart for recording dental problems.

Radiographic imaging is necessary for diagnosis of boney or tooth root pathology, and high-quality, properly positioned films are required if radiography is to be of benefit [68]. Radiographic imaging necessitates heavy sedation or general anesthesia. Oblique projections of the cheek teeth must be taken to prevent override from the opposite jaw. Placement of a radiolucent gag in the mouth further helps with decreasing superimposition of the mandibular and maxillary arcades. Radiographs of dental lesions of camelids are evaluated in the same manner as those of other animals. Loss of the lamina dura, radiolucent areas with abscessation, fractures and increased density with sclerotic bone, and osteomyelitis are conditions that may be seen. Fistulography can be performed by injecting iodinated contrast material through a draining tract and can provide valuable information concerning the extent of the fistulous tract and underlying mandibular or maxillary involvement. If available, advanced imaging modalities like computed tomography (CT) can be useful to better define pathology of skull.

Plaque rarely interferes with oral function and does not require removal unless it is causing gingivitis. Pulpitis and

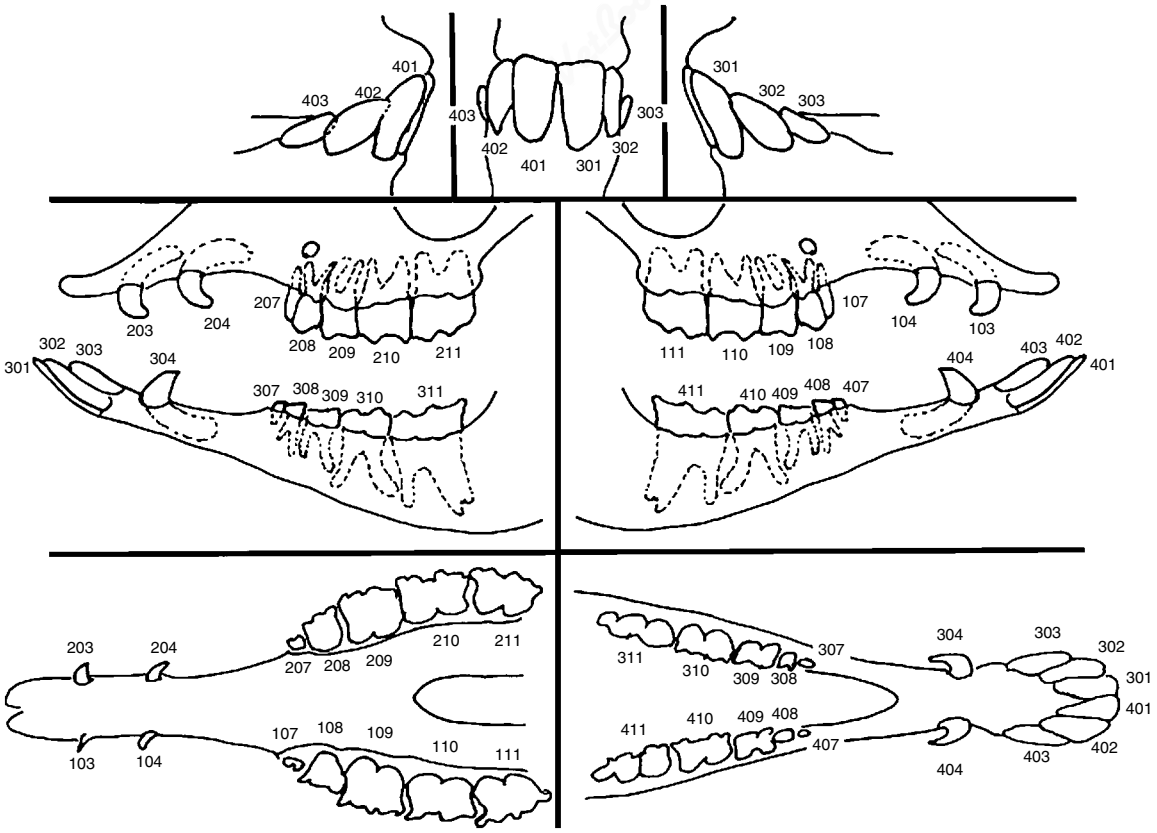


Figure 12.47 Diagram for dental examination using Tridan nomenclature.

alveolar periostitis may necessitate extraction or repulsion of the tooth. If a dental fracture opens the pulp cavity, a pulpotomy may be done [69]. If the pulp has become infected, the tooth should be removed [70].

Tooth root abscessation and Osteomyelitis and Sequestra

Tooth root abscessation is a commonly reported dental condition afflicting South American camelids (SACs) [59, 63, 71]. Most camelid dental abscesses involve the mandibular teeth, however any tooth, mandibular or maxillary, can be abscessed [59, 71]. Camelids of all ages can be affected. Geriatric animals have more issues with dental growth abnormalities such as excessively worn teeth, malocclusion, and sharp enamel points, however, tooth root abscesses are reportedly more prevalent in middle-aged camelids when the permanent teeth are erupting [63, 71].

Theories on the pathogenesis of tooth abscesses include mastication of rough forages leading to oral abrasions allowing invasion of commensal organisms, premature removal of deciduous teeth, fractured teeth allowing exposure of the pulp cavity, and hematogenous spread of bacterial pathogens [64]. Fractures of the teeth can result from fighting or can be iatrogenic from trimming of fighting teeth [59].

Clinical signs associated with tooth abscesses include a hard, boney swelling around the tooth root. Palpation of the abscess may be painful, and the patient may be head shy. Chronic cases may have a fistulous tract to the skin. Drainage of purulent material may be obvious or there may be a scab over the fistula. Chronic abscesses are generally associated with boney swelling and boney reaction evident on radiographs (see Chapter 5). Late-stage disease may be accompanied by mastication abnormalities, anorexia, and weight loss [64].

Generalized osteomyelitis (lumpy jaw) that is not associated with a specific tooth can cause swelling of the jaw like lumpy jaw in cattle and should be considered a differential and is often clinically indistinguishable from tooth root abscessation. Actinomycosis caused by *Actinomyces bovis* has been reported in new word camelids [72, 73] and camels [74], but mandibular osteomyelitis can be caused by other organisms like *T. pyogenes* [71]. Radiographically lumpy jaw appears similar to tooth abscessation since both have lytic and reactive new bone, and commonly leads to secondary tooth involvement. The prognosis for generalized mandibular osteomyelitis is poor since a targeted tooth removal or debridement is unlikely to be curative. Medical (antibiotic) therapy may be attempted but usually is not curative.

Boney sequestra are common with primary or secondary dental disease. The disease process is indistinguishable pathologically since the non-vital tooth roots in cases of tooth root abscessation can be thought of as sequestra, and the patients present with identical clinical signs. Sequestra can be due to a previous mandibular or tooth root infection, mandibular fracture, or can be idiopathic.

Treatment of dental and boney infections involves long-term medical therapy consisting of broad-spectrum antibiotics and anti-inflammatories. Antibiotics must be able to penetrate the tissue in order to be effective. In chronic cases, the diseased tissue may be "walled-off" from the rest of the body or be non-vital, lacking blood supply inhibiting effective penetration of antimicrobials. A common scenario is that the lesion appears to resolve but then recurs following discontinuation of medical therapy.

Surgical management is frequently required to remove the diseased or non-viable tissue that is a nidus for continued infection. Options for surgical management are tooth extraction, sequestrectomy, resection of the tooth root, pulpotomy, and bone debridement. General anesthesia is employed for most of these surgical techniques, however, regional or local anesthesia can be an adjunct to general anesthesia and provide for a multimodal anesthetic/analgesic approach [64] (see Chapter 6). A mandibular (alveolar) nerve block is routinely employed in the authors' practice to supplement general anesthesia for routine dental surgeries.

Cheek Tooth Extraction

Tooth extraction in camelids patients pose unique challenges. SACs have a narrow mouth opening and the mouth is deep, limiting access through the opening of the mouth to all but the most rostral teeth (incisors and canines). The technique that the author most commonly uses to extract cheek teeth is a modification of the ventrolateral technique described previously [70]. A challenge commonly encountered is correct placement of the incision so that it is centered over the affected tooth to be removed. Intraoperative radiographic imaging may help with placement of the incision.

A horizontal skin incision, centered over the affected tooth (teeth) to be removed, is made on the ventrolateral aspect of the mandible. A 5–6 cm incision is usually sufficient for a single tooth. If draining tracts are present, an elliptical incision can be made around the draining tract to completely excise the tract (Figure 12.48). The incision is continued through the subcutaneous tissues and mandibular periosteum. A periosteal elevator or blunt dissection is used to reflect the periosteum dorsally exposing the mandible bone (Figure 12.49). Hemostasis can usually be accomplished with electrocautery during this part of the procedure.

Ideally, a motorized burr is available and can be used to remove lateral alveolar bone until the tooth roots are

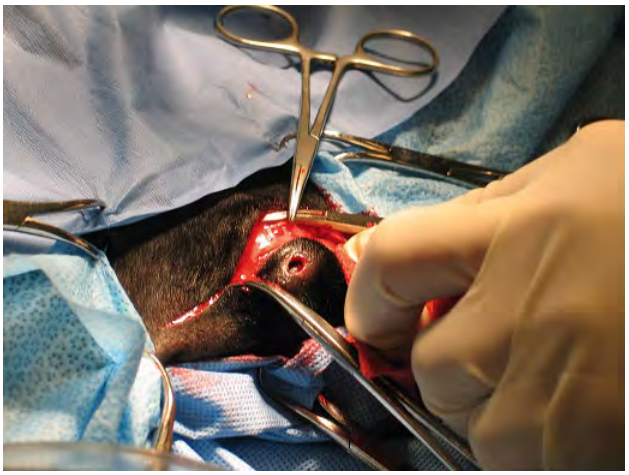


Figure 12.48 A ventrolateral skin incision is made around a draining tract. The incision is continued through the soft tissues to expose the mandibular bone.

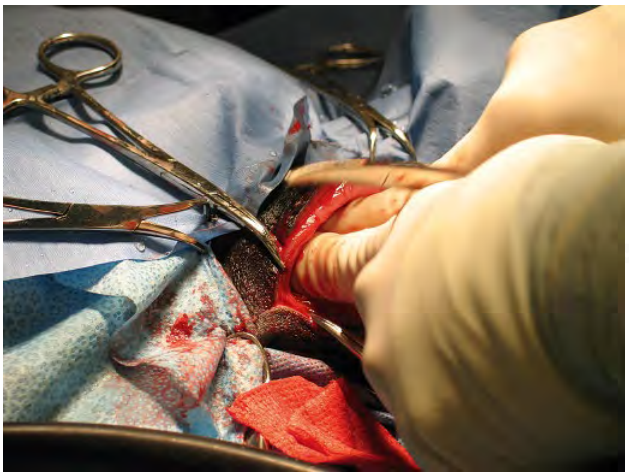


Figure 12.49 The soft tissues are elevated from the mandibular bone to expose the lateral alveolar bone plate.

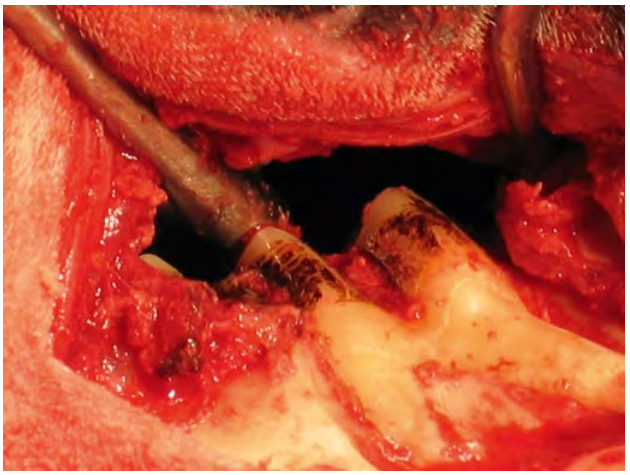


Figure 12.50 The lateral alveolar bone plate has been removed with a pneumatic burr to expose the tooth roots.

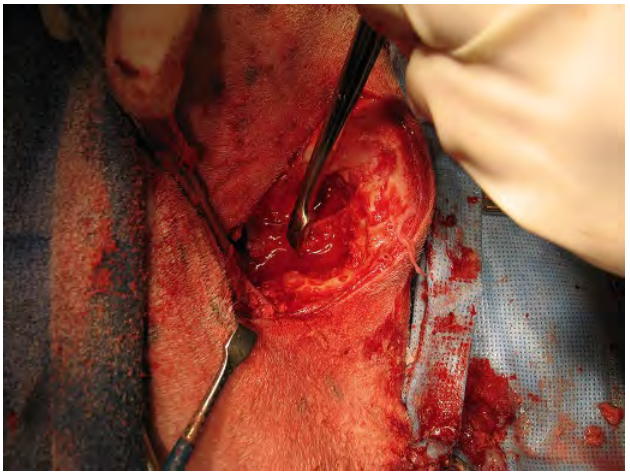


Figure 12.51 Curettage of the socket is performed following removal of the tooth to remove diseased bone and any small sequestra or fragments of tooth root that remain.

visible (Figure 12.50). A good working knowledge of the anatomy is important as the tooth roots can often be challenging to identify and correlate with radiographic images. Intraoperative radiographic imaging may be beneficial in identifying the affected roots. If a motorized burr is not available, osteotomes or bone chisels can be used to remove the lateral bone, but the risk of mandibular fracture using this technique is high.

Healthy tooth roots are attached tightly to the alveolar bone via the periodontal ligament. Disease and abscessation will loosen this attachment, but it is often deceiving because even teeth that appear to be diseased radiographically, with large areas of lucency surrounding the roots, can still be firmly attached and require significant force to free. A curved dental elevator should be used to separate the tooth root from the alveolar bone and pry the tooth free. Fracturing the root is common, but care should be taken to remove any fragments to prevent sequestrum formation. Curettage of the alveolar socket is usually enough to remove fragments and prevent sequestration [69].

Following removal of the tooth, the surrounding bone (assumed to be diseased) is debrided (Figure 12.51). Following debridement, lavage of the area using sterile saline or a dilute antiseptic solution is performed to remove blood, contamination, or other diseased tissue at the surgical site. During removal of the lateral alveolar plate or boney debridement, hemorrhage from the bone is common. Bleeding from the bone can be brisk and difficult to control because individual vessels often cannot be identified or clamped. Bone wax or dental wax can be used to plug small defects in the bone to control hemorrhage.

Camelids have a favorable prognosis following removal of cheek teeth. Also, removal of multiple teeth does not significantly affect the outcome [71]. However, large spaces created in the mouth from missing teeth can lead to malocclusion, overgrown teeth on the opposite arcade, problems with mastication, and packing of cud in the mouth. Dental impression material can be used to fill the defect, however, complications including dislodgment of the material are possible.

A mucosal flap can be created on the oral side of the defect to prevent feed contamination of the alveolar socket, but these commonly dehisce. The ventral part of the incision can be partly closed but should be left at least partly open to allow for ventral drainage. Packing material can be used temporarily to provide hemostasis and prevent salivary losses. If the ventral incision has been left partly open for drainage, packing can be inserted and removed via this ventral incision avoiding the need to open the patient's mouth. Packing is usually unnecessary after three to four days. Packing should be changed at least daily, and lavage with saline or dilute antiseptic solution can be used to flush debris from the incision when the packing is changed. Following removal of the packing, the incision heals by second intention.

Alternatively, an intraoral approach for tooth extraction can be effective for the rostral teeth. A curved dental elevator can be used to break down the periodontal ligament separating the tooth from the alveolar bone circumferentially. The tooth eventually becomes loose in its socket and can be rocked loose [69, 70]. Curettage of the socket is then performed in a similar manner as with the cheek tooth removal.

Maxillary tooth abscessation is uncommon. Maxillary tooth extraction is performed in a similar manner to mandibular extraction. The surgical approach to the alveolar bone plate of the maxilla for tooth removal is closed primarily following surgery. Ventral draining of the extraction site is still achieved into the mouth.

Tooth repulsion is described for removal of llama teeth but should be used with caution. The mandible and maxilla in SACs is thinner than horses and cattle and mandibular fracture is a possible complication to aggressive repulsion [75].

Ideally, the tissue that remains following surgical debridement is healthy enough to respond appropriately to medical management, and treatment with antimicrobials should be part of the post-operative care of these patients. *T. pyogenes* has been cultured commonly from the non-vital tooth roots. Most organisms show antimicrobial susceptibility to penicillin, ceftiofur and florfenicol [71]. Adjunctive treatments like isoniazid and sodium iodine, which are known for penetrating walled-off infections, have been tried; no data on their efficacy have been reported.

Retained Deciduous Teeth

The crowns of the non-deciduous teeth should be positioned immediately deep to the deciduous roots and should force the deciduous teeth out during eruption. If the non-deciduous teeth are not lined up with the deciduous teeth, the non-deciduous teeth can erupt with the deciduous teeth left behind. This condition is known as retained deciduous teeth and is common in SAC's. Typically, retained deciduous teeth are a cosmetic blemish only but can lead to entrapped food particles predisposing to dental decay and infectious dental disease. Altered prehension and mastication is also possible.

Deciduous incisors are most commonly retained. Retained deciduous incisors are smaller, typically located rostral to their permanent counterpart, and are often loose.

Retained deciduous incisors are usually easily extracted compared to permanent teeth. With the patient heavily sedated, a curved dental elevator is used to separate the tooth's root from the surrounding alveolar bone. Intermittent gentle traction with back-and-forth motion on the tooth is usually sufficient to loosen the tooth from the bone and gingiva. Sedation or anesthesia may be necessary for some animals with some teeth that do not extract easily. Though rare, damage to the permanent tooth roots is a possible complication.

Teeth Trimming and Routine Dental Care

There are multiple indications for trimming teeth in camelids. Overgrown mandibular incisors can result in malocclusion and difficulty prehending food. Many owners perceive this as an unacceptable cosmetic blemish. The fighting teeth (upper and lower canines and the upper third incisors) are commonly trimmed in male llamas and alpacas to prevent injuries to herd mates and caretakers.

Trimming of the incisors or fighting teeth can be done with obstetric wire or a cutting wheel attachment on a handheld rotary tool. Some field practitioners feel that using obstetric wire is a safer alternative to a rotary tool when anesthesia and restraint are minimal.

Like horses, cheek teeth that do not properly oppose the opposite arcade can become overgrown with sharp points and steps which can interfere with occlusion. This condition is most common when teeth have been previously extracted. The author does not recommend routine floating of cheek teeth in SACs, but if mastication is impaired due to overgrowth, floating can be done. Floating is best accomplished with small motorized dental floats. Normally, the maxillary arcade is wider than the mandibular arcade which will lead to enamel points on the buccal side of the maxillary check teeth and the lingual side of the mandibular check teeth. These points do not usually interfere with normal mastication and do not require routine floating. Malocclusion can lead to pathologic enamel points and may require floating as routine dental care.

Trimming Incisors to Prevent Tooth Drift

Frequently, malocclusion and rostral protrusion of the incisors are caused by overcrowding. It appears as if breeding for shortened-faced animals has exasperated the situation [76]. The second mandibular incisor overlaps the caudal surface of the first incisor, pushing the first incisor forward away from the dental pad.

Similarly, the third incisor frequently grows forward against the second incisor and increases the rostral pressure on the second and first incisors away from the dental pad (Figure 12.52). These abnormal forces on the first incisors can cause the rostral incisors to rotate so that the labial surfaces rotate toward midline (Figure 12.53). Incisors should not have overlapping edges, and the first and second incisors should not be pushed forward by the third incisor [76].

Figure 12.52 Overcrowding of mandibular incisors of an alpaca. The third incisor is growing forward pushing on the second incisor, and the second incisor is pushing forward on the first incisor.

Figure 12.53 The front view of an alpaca. The rostral pressure on the first incisor has caused rotation such that their labial surfaces are turning toward each other. The inset is a top view of mandibular incisors showing inward rotation of the labial surfaces of the first incisors.

Many trimming strategies trim the incisors flat to increase contact with the dental pad. This shortens the rostral incisors but the overcrowding is not improved and the procedure will need to be repeated. This also does not improve their ability to graze and creates a blunt leading edge of the tooth. Strategic shaping of the incisors removes this forward pressure and allows the first incisor to retreat caudally and contact the dental pad. This increases wear on the first incisor and promotes the development of a cutting edge which improves function and occlusion [76, 77].

The second incisor is shaped and trimmed such that when the teeth move, the strategic gaps made between teeth close (Figure 12.54). The smooth, enamel-contacting surfaces are able to slide past each other. This puts the entire second incisor lateral to the first incisor and removes any forward pressure on the first incisor allowing it to stay in contact with the dental pad (Figure 12.55).

Positioning the second incisor lateral to the first incisor greatly reduces the leverage that it would have on the first incisor. As long as it is able to slide freely past the first incisor, the first incisor will maintain its position against the dental pad.

The third incisor is shaped and trimmed so that it will slide lateral to the second incisor. This puts considerable force against the caudal surface of the second incisor and eventually the first incisor. By trimming and shaping the third incisor so that it slides freely, lateral to the second incisor, as the third incisor grows, this force is removed. In some instances, the third incisor becomes "tusk" like (grows horizontally). In these cases, it may have to have its length shortened to avoid contacting the second incisor (Figures 12.56 and 12.57).

Figure 12.54 Strategic gaps are created between the incisors so that the caudal incisors are not creating abnormal force on the rostral incisors.

Figure 12.55 Second incisor is lateral to the first incisor without contact. Abnormal force on the first incisor has been removed. Inset is view from the top.

Figure 12.56 The third incisor is growing horizontally and pushing rostrally on the second incisor.

Final trimming of incisor length is dependent on absence of root pulp in the portion of the tooth to be trimmed. All care should be taken to NOT cut live root pulp. Root pulp can be partially visualized within the tooth by shining a very bright light through the tooth. Root pulp will look pink inside the tooth (Figure 12.58). Root pulp often extends past the visible pink area.

Allowing the first incisors to retreat caudally and wear against the dental pad eventually exposes an empty root canal. The wear against the caudal surface promotes the retreat of the root pulp away from the area of wear. Once the empty root canal is visible, the worn area of the tooth demarcates a safe line for trimming.

Figure 12.57 The same alpaca as in Figure 12.5. The length of the third incisor has been trimmed so that it is not pushing on the second incisor.

Figure 12.58 A bright light is used to illuminate the translucent pulp canal.

The overall strategy of this method is to narrow the teeth by reducing the lateral edges and to shape and smooth any surfaces that will eventually contact another tooth so that the teeth are able to move freely (Figure 12.59).

The "gaps" between teeth opened by this procedure allow the incisors to move independently. As the teeth move, the "gaps" close (Figure 12.60). The first incisors move caudally. When the "gaps" close, if the second incisor still overlaps the first incisor pushing it forward, then the procedure is repeated until the incisors no longer overlap. The number of repeat treatments depends primarily on how much overlap exists between the incisors. Three or four treatments are common.

The rate at which correction is made depends on the growth rate of the animal's teeth. This will vary with the health and age of the animal. The best time to make these

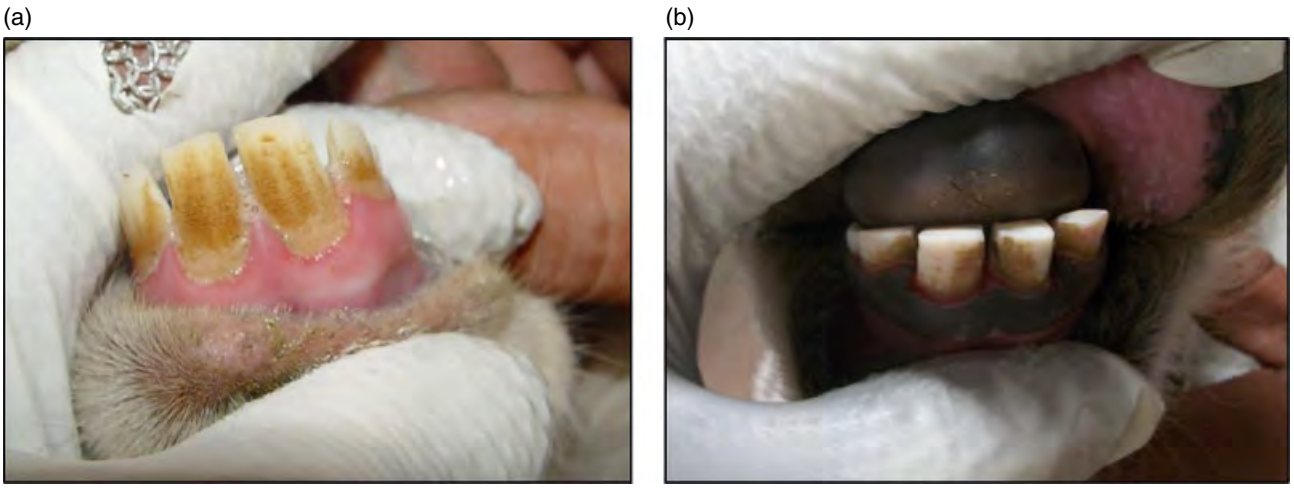


Figure 12.59 Gaps are created in between the incisors allowing them to more freely alongside one another.

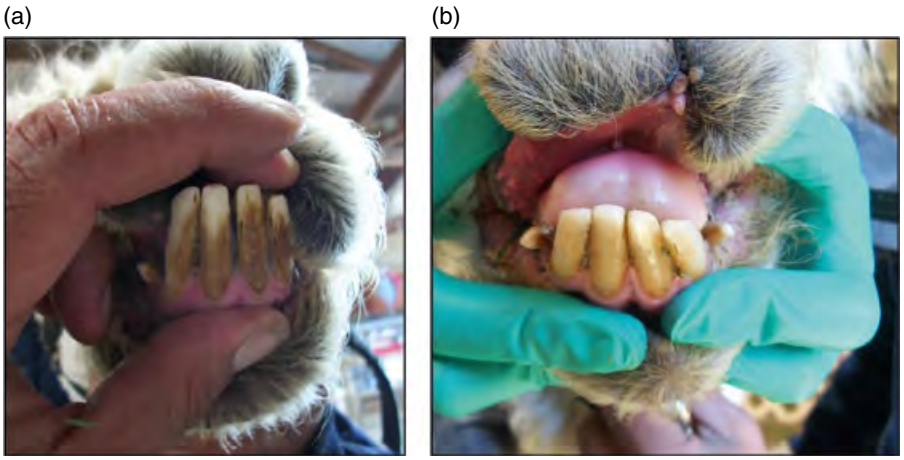


Figure 12.60 (a) Incisors of an alpaca following corrective trimming. Note the gaps between the incisors created to allow the incisors to grow independently of one another. (b) Same alpaca following continued tooth growth. The gaps have closed; however, the caudal incisors have not pushed rostrally on the first incisor, allowing the first incisor to retreat caudally and contact the dental pad.

corrections is when the permanent second incisor is beginning to contact the permanent first incisor.

An individual animal's deciduous incisors will foretell the positions of the permanent incisors. If they overlap as juveniles, they will overlap as adults. The condition is temporarily alleviated in the juveniles when the deciduous incisors fall out and the forward pressure is removed until the permanent second incisor eventually contacts the caudal surface of the first incisor, pushing it forward.

Salivary Gland Disorders

Etiology

The salivary glands of camelids are affected by problems similar to those of ruminants. Abscesses caused by *T. pyogenes,* within or next to the salivary glands, are most common.

Generalized parotiditis has not been described. Salivary fistulae may occur from lacerations of the side of the face or abscessation and rupture of a duct. A laceration of the body of the gland is possible, but the skin thickness over the neck and face is such that a severe slashing blow would be required to penetrate the skin. A parotid salivary fistula may occur from a facial laceration. Sialiths have not been reported but are likely to occur.

Signs

The primary sign is swelling over the gland, either firm in the case of cellulitis or parotiditis or fluctuant with an abscess. The area is hot and the skin hyperemic. The llama is likely reluctant to eat because of the pain in the angle of the jaw.

In the case of a salivary fistula, an excessive flow of saliva is observed, especially while the llama is feeding or ruminating.

Diagnosis

A physical examination usually discovers the problem. A needle may be inserted into a fluctuant swelling to establish the presence of an exudate, serum, or blood. If there is question that the fluid is saliva, the pH of the fluid should

be measured. Saliva will be very alkaline due to its high bicarbonate content.

Treatment

Primary parotiditis requires broad-spectrum antimicrobial therapy. However, this is of little value in chronic abscesses. It may be desirable to wait for an abscess to mature and localize before lancing but, otherwise, a needle should be inserted at the ventral aspect of the abscess. When the exudate begins to flow, the needle should be left in place and an incision made along the needle in a vertical direction through the skin. If possible, blunt dissection should be continued with a hemostat to open the abscess. The cavity may be explored with a gloved finger and irrigated with a 1:4 dilution of povidone-iodine solution. The irrigation should be continued daily until the cavity has healed from the inside out.

Diseases of the Pharynx and Esophagus

Pharyngitis and Esophagitis

Etiology

Pharyngitis and esophagitis may result from repeated gastric intubation, especially with a rough-ended tube. Chronic choke and feed accumulation within the esophagus may result in esophagitis. Acute or chronic bacterial infections, especially those of the respiratory system, abscesses, *Cephenemyia* spp. [78], larvae infestation of the nasopharynx (Chapter 7), and developmental defects such as agenesis of the soft palate or cleft soft palate (Chapter 21) can result in pharyngitis.

Diagnosis

Signs of pharyngitis and esophagitis include anorexia, weight loss, nasal discharge, coughing, retching, fluctuant swelling behind the ramus of the mandible, extended head, pain response to palpation of the throat and neck region, and mucosal hyperemia. Visualization of the pharynx may be accomplished by insertion of a long-bladed laryngoscope or via nasal endoscopy; visualization of the esophagus requires endoscopy with insufflation. These procedures require heavy sedation or anesthesia. Without adequate sedation or anesthesia, the animal may bite causing injury to the examiner or damage to the equipment (i.e. endoscope), or the animal may reflexively regurgitate. An oral speculum may be fashioned out of an appropriately sized, smooth-ended piece of polyvinyl chloride pipe. The nasopharynx may be explored with an endoscope via the nasal cavity, and the oropharynx, via the oral cavity.

Treatment

Supportive care may be the most important aspect of treatment. Soft, palatable feed and suitable quantities of water should be provided. Dehydration may be more of a problem than lack of food. If normal eating and drinking habits do not return within a couple of days, it may be necessary to institute intravenous (IV) feeding or enteral feeding via a C1 fistula (see 12.6.8).

Parenteral antibiotics may be indicated. Antibiotics should have efficacy against Gram positive and anaerobic bacteria, common inhabitants of the oral and pharyngeal cavity.

Due to their walled-off nature, chronic abscesses are often refractory to parenteral antibiotics due to lack of adequate penetration; it is usually necessary to lance abscesses. Sweating helps to bring an abscess to maturation. This may be done by frequent applications of moistened hot towels or by topical applications of salves such as ichthammol or an Epsom salts poultice.

Esophageal Obstruction (Choke)

Etiology

SACs are more discerning than cattle and OWCs [6] when eating and rarely choke from large foreign bodies. Choke in SACs is usually due to rapid consumption of dry feed that packs into the esophagus or esophageal motility disorders like megaesophagus. Cervical masses, abscesses, vascular ring anomalies, and large hematomas, secondary to carotid artery puncture, may compress the esophagus and lead to obstruction [79, 80]. Esophageal obstruction from foreign bodies including mostly plastic bags and sheets has been reported in dromedary camel calves [81].

Obstruction may be partial or complete. Any foreign body that becomes lodged in the esophagus will initiate muscular contractions with the purpose of moving the object one way or the other. If neither peristaltic nor antiperistaltic action is effective, the end result may be a spasm on either side of the obstruction that complicates the situation further, turning a partial into a complete obstruction.

Signs

Choke usually initiates alarming behavior that indicates the animal is in discomfort. The signs include retching, coughing, dysphagia, head shaking, salivation, nasal discharge, and anxiety. If the blockage is prolonged, dehydration may result from the inability to swallow and water lost through saliva. If the animal continues to eat, food may pack in the pharyngeal area, and subsequent retching may drive feed particles into the nasopharynx and out the nose. Aspiration pneumonia is a common sequel to choke.

The obstruction may be lodged anywhere from the origin of the esophagus to the cardia. If the obstruction is in the

cervical region, a mass may be observed or palpated in the esophagus. The esophagus of the camelid lies deeper than that of other species and is somewhat obscured by the ventral tuberculum of the transverse process of the cervical vertebrae making it less obvious, and palpation may be necessary to locate the obstruction. The object causing the obstruction need not be larger than the bolus the animal would normally ingest or regurgitate for rechewing. The exact reason for initiation of a spasm causing an obstruction is not entirely known.

Diagnosis

Clinical signs are characteristic in the early stages, but if the blockage persists, the animal will become depressed, and retching and coughing will diminish or cease. If blockage is suspected, a large gastric tube should be inserted into the esophagus and gently advanced to determine the patency of the esophagus. Vigorous force should not be used to push the object as this may cause esophageal trauma. Radiography, both plain and contrast, is helpful, particularly in diagnosing obstructions within the thoracic esophagus. The diagnosis may also be aided by response to spasmolytic therapy.

Treatment and Prevention

The most commonly observed cause of choke in the author's practice is due to rapid ingestion of dry feed. Though this condition can be distressing to watch, the majority of cases will self-resolve within an hour. The animal should be placed in a non-bedded stall, and any feed should be removed from the pen. Spasmolytics such as atropine or muscle relaxants like acepromazine should be given to relax the esophageal musculature and allow peristaltic activity to resume. Analgesics may be given because this condition is painful. Once the esophagus is relaxed, a large equine stomach tube may be used to gently push the obstruction onward.

Difficult cases that cannot be resolved by passage of a stomach tube may benefit from a gentle water lavage under direct endoscopic visualization. The animal should be anesthetized, and endotracheal intubation performed. Water or isotonic saline flushed through the endoscope following visualization of the obstruction, may break up feed boluses.

The foregoing stimulation of the pharynx and esophagus may initiate a reflex regurgitation attempt, and sedation or anesthesia may be necessary to continue. Aspiration of feed and saliva into the lungs is always a risk, and endotracheal intubation may be desirable when dealing with a difficult case. Antibiotics should be administered for potential aspiration pneumonia.

Complete obstruction of the esophagus prevents eructation, which may lead to the development of bloat from gas production within C1. If an esophageal tube cannot be passed and bloat appears to be life threatening, bloat may be relieved by inserting a large-bore needle into C1 through the left paralumbar fossa or surgically via a C1 gastrotomy. Life-threatening peritonitis and cellulitis of the body wall are likely complications from these procedures.

Esophagotomy can be performed to remove foreign material from the esophagus. Risks with esophageal surgery include esophageal stricture, fistula, cervical cellulitis, and mediastinitis. Esophagotomy was successful in 6 out of 7 camel calves with esophageal obstruction from foreign bodies [81].

Prevention of choke is mainly due to limiting animals' access to potential foreign bodies. Animals unaccustomed to dry feeds should be watched carefully and the amount of feed given at a time should be limited. Highly competitive animals are at greater risk of eating too fast and developing choke. Large rocks (too large to eat) can be placed in feed pans; forcing animals to eat around these large objects will slow the consumption of feed material and decrease risk of choke.

Megaesophagus (Esophageal Dilatation, Esophageal Paralysis and Esophageal Achalasia, Cardiospasm)

Megaesophagus is a disease characterized by dilatation of all or a part of the esophagus, especially the intrathoracic segment. It is reported in multiple species including humans, and many domestic animals including camelids. The veterinary literature does not report megaesophagus in OWCs, but this condition is a commonly cited cause of dysphagia and regurgitation in SACs.

Etiology

Megaesophagus in SACs is typically idiopathic. Llamas with megaesophagus have abnormal peristalsis with over half of the distal esophagus being aperistaltic suggesting a primary denervating disease as the cause [82]. However, hereditary causes such as hypothyroidism, GI obstruction, anticholinesterase toxicity, and vascular ring anomalies have been associated with megaesophagus in SACs [83, 84]. In humans and dogs, failure or relaxation of the esophagogastric junction (cardiac sphincter) has been associated with megaesophagus [85, 86].

Signs

The major clinical signs relate to the animals' inability to swallow. Regurgitation, especially immediately after eating, dysphagia and drooling are the primary clinical signs. The material regurgitated is undigested and will not have the characteristic smell of C1 contents observed when a healthy animal eructates or spits following regurgitation of C1 contents. In the early stages of disease, the clinical signs are often intermittent, and regurgitation may be only

observed when the animals' head and neck are down. Often, the disease progressively worsens with time. In chronic states of disease anorexia, weight loss, stunted growth, decreased libido, and aspiration pneumonia are observed. Esophageal waves can be observed reflecting the abnormal esophageal motility.

Diagnosis

An abnormally dilated esophagus is observed on lateral thoracic radiographs (Figure 12.61). Commonly, a fluid line will be observed because feed and fluid tend to sit in the esophagus. The abnormal dilation will be more prominent in the thoracic esophagus than the cervical portion of the esophagus. More definitive delineation is obtained by using contrast radiography. Fluoroscopy may also be used to evaluate the peristaltic activity of the esophagus. Figures 12.62a and b show the dilated esophagus *in situ* at necropsy. Mild rhabdomyolysis of the skeletal muscle component of the distal esophagus was present on histologic examination of these specimens.

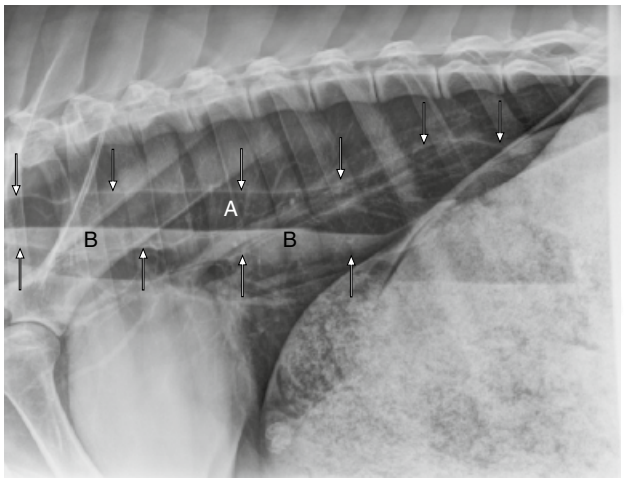


Figure 12.61 Lateral radiograph of dilated thoracic esophagus (arrows outline the wall of the esophagus). (A) Gas, (B) food.

Treatment

If a primary etiology can be identified, the megaesophagus may resolve with correction of the primary problem. With most cases of idiopathic megaesophagus, treatment is aimed at supportive care. Affected animals should be provided a soft diet with small stem fiber that is easy to move through the esophagus with a low likelihood of packing. The animal can be fed on an inclined plane with the forequarters elevated to allow gravity to aid in esophageal emptying. If lower esophageal sphincter disease is suspected, drugs with muscle-relaxant properties may be tried (acepromazine, spasmolytics), but their efficacy has not been established in camelids. The presence of pneumonia may necessitate antimicrobial therapy.

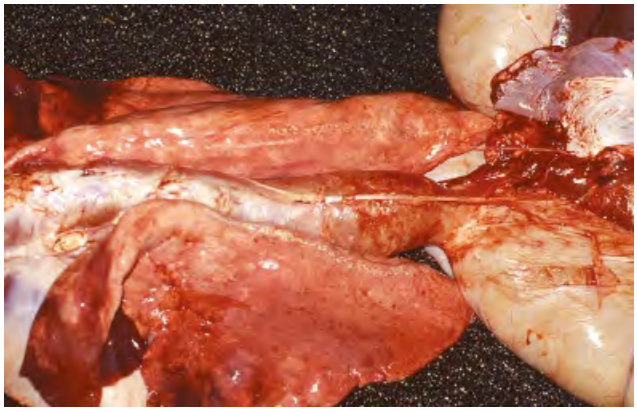
Gastric Disorders

Disorders of the gastric compartments include ulcerative gastritis, dysbiosis of the first compartment, trichophytobezoars, and gastric overload. SACs are more discriminate than cattle when they eat and therefore, they do not usually get traumatic gastritis like cattle.

Gastric Ulcers

Ulcerative lesions have been found in all three of the stomach compartments at necropsy, however, lesions affecting C1 and C2 are rare and the majority of lesions occur on the parietal surface of the lesser curvature of C3 and the proximal duodenum [58, 87]. Primary gastritis is usually subclinical and diagnosed as a lesion at necropsy. They are often included as a differential causing anorexia, weight

(a)



(b)

Figure 12.62 A necropsy photo of a llama with megaesophagus. (a). A dilated thoracic esophagus is observed in the middle of the photo. (b). The esophagus has been opened. Feed material is packed into the dilated thoracic esophagus.

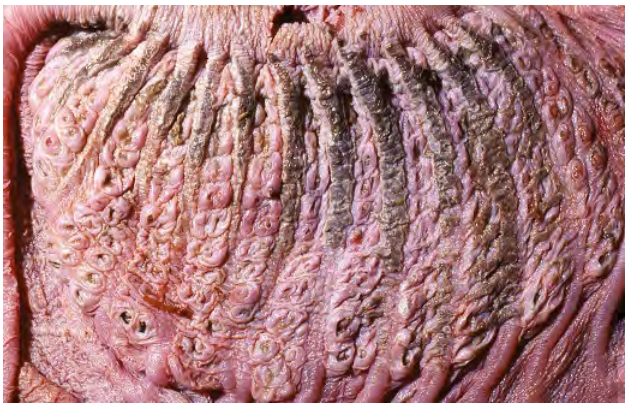


Figure 12.63 Mucosal ulceration of C1 in a llama.

Figure 12.64 Mucosal ulceration of C2 in a llama.

loss, ill thrift, and peritonitis in camelids of all ages. The number, location within the stomach, severity of the lesions, and concurrent disease determine the clinical signs observed. Ulcers can be a primary lesion or secondary to other disease.

C1 and C2 ulcers likely involve the margins of the saccules in the glandular areas (Figures 12.63 and 12.64). The ulcers in the proximal four-fifths of C3 tend to be linear along the ridges of the longitudinal pleats (Figure 12.65). Ulcers of the distal one-fifth are more focal, deeper into the submucosa, and more likely to perforate (Figures 12.66–12.68). Bleeding ulcers like those seen in ruminants, resulting in anemia and melena, have not been reported, and fecal occult blood tests are almost always negative [58].

ETIOLOGY. The etiology of ulcers is unknown. The role of stress in the development of the syndrome is unknown. However, many animals with gastric ulcers have concurrent systemic disease suggesting stress as a predisposing factor [58, 87]. No dietary association or association with GI parasitism has been made.

SIGNS. Signs of gastric ulcers are generally non-specific. Atony of C1, with inappetence and scant feces, may be the

Figure 12.65 Linear mucosal ulceration of C3 in a llama.

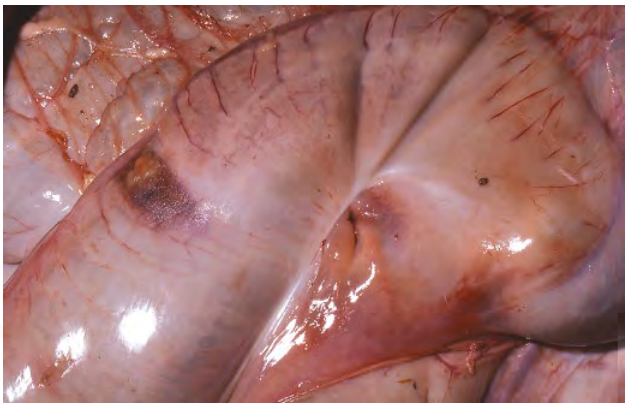


Figure 12.66 Serosal surface of C1 with gastric ulcers.

Figure 12.67 Ulceration of C1.

major syndrome. Non-perforating ulcers do not result in changes in the hemogram or change the composition of the peritoneal fluid or serum. The temperature, heart rate, and respiratory rate are usually normal. The animal may be depressed and stand off from the herd. Most animals do not exhibit colicky signs. The owner will frequently comment that the animal is isolated, lying down, and has a decreased appetite. Animals exhibiting these mild signs

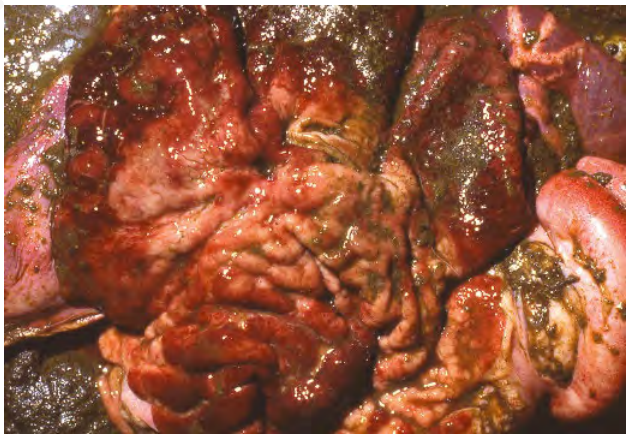


Figure 12.68 Abdominal contamination from perforating gastric ulcer in C3.

Figure 12.69 Acute, fibrinous peritonitis caused by perforating gastric ulcer.

should be given a thorough physical examination. If an ulcer has perforated, signs of peritonitis will be seen (Figure 12.69). Ulcerative gastritis can also be the cause of disseminated disease due to translocation of organisms across a compromised gastric mucosal barrier. Cases of mycotic pneumonia have been reported in SACs with gastric ulceration. Though the pathogenesis cannot be verified, presumably hemotogenous spread from the site of gastric ulceration caused the pneumonia [88, 89].

DIAGNOSIS. Definitive antemortem diagnosis of the lesions is challenging because endoscopy is not feasible, as in monogastrics, due to the voluminous first gastric compartment and the character of its content. Even animals that have been fasted will retain enough gastric contents in their C1 compartment to obscure visualization. Diagnosis of gastric ulceration is usually made only after elimination of other, more serious digestive disorders.

TREATMENT. Treatment of any underlying disease is of utmost importance. Nourishment and fluid must be provided as appropriate. An IV catheter may be placed to hydrate the llama and give parenteral nutrition. Electrolyte imbalance is not usually a major factor unless concurrent disease is present.

Pharmacologic treatment of ulcers is more challenging than in monogastric species. Orally omeprazole paste, a proton pump inhibitor, is marketed in the US (Gastroguard[1]) and has been shown to be efficacious in treatment of gastric ulcers in horses [90]. However, when orally administered, it has not been shown to achieve therapeutic plasma drug concentrations in llamas [87, 91], and another study showed that it was absorbed poorly when administered rectally [92]. Omeprazole has only been found to decrease gastric acidity when administered IV [93]. Rantidine reduced acid production in the third compartment suggesting the presence of H2-histaminergic receptors but was not therapeutic at the dosage studied, and based on pharmacokinetic calculations, the effective dose would be unpractical and possibly toxic [93]. Cimetidine (3.3 mg/kg IM) appeared to increase gastric pH within the first 30 minutes following administration but was not effective after that time [94].

Pantoprazole given at 1 mg/kg IV or 2 mg/kg SC decreased gastric acidity in alpacas [95]. Injectable pantoprazole is available in the US (Protonix[2]) and currently represents the most practical and efficacious way to pharmacologically increase the pH of the third compartment in camelids.

Gastroprotectants like Sucralfate and bismuth subsalicylate can be administered without untoward side effects, but their efficacy is unknown. These products have a local protective effect and likely would be more effective treating ulcers in the first compartment rather than the more common C3 ulcers, as oral medication given via the stomach tube may take a long time to reach C3 and be diluted below its effective concentration. Due to the large volume of the C1 compartment compared to the gastric volume of a monogastric animal, a relatively high volume of medication likely must be given to achieve a protective effect.

Forestomach (C1) Acidosis (Grain Overload)

Forestomach acidosis is much less common in camelids than in cattle, and this is likely due to intrinsic feeding behaviors and different management practices. SACs reared in group housing may increase overindulgence tendencies. A report of six SACs with forestomach acidosis were group housed and recently introduced to new feed [96]. Racing camels in the United Arab Emirates are fed a diet high in fermentable carbohydrates making them susceptible to forestomach acidosis [97].

PATHOGENESIS. Camelids are thought to possibly have more physiologic protective mechanisms compared to ruminants such as a high propensity for forestomach buffering by C1 saccules [98], further protecting against forestomach acidosis. The pathogenesis is similar to that of

rumen acidosis in true ruminants. The normal microflora of C1 ferments carbohydrates, with short-chain VFA (acetic, propionic, butyric) as end products, which are absorbed and used for energy. If the llama consumes an overabundance of readily digestible carbohydrates, the fermentation pattern changes, a new microflora emerges as predominant, and lactic acid and other long-chain fatty acids are produced.

Lactic, formic, valeric, and succinic acids are produced by microbial fermentation and change the osmolarity of C1, resulting in fluid being drawn in from the circulatory system and other tissues. Thirst causes excessive water intake and C1 distension. The pH of C1 may drop to 4, and there is gastric stasis and destruction of the normal C1 microflora. The excess fluid drawn into the GI tract produces a profuse, watery diarrhea. In cattle, the acidic forestomach environment causes gastritis permitting bacterial invasion of the wall of C1 leading to sequela-like bacteremia and liver abscesses. Liver abscesses do not seem to occur secondary to C1 acidosis as in ruminants [99]. In cattle, absorption of lactic acid can contribute to the systemic acidosis, however, in an experimental induction of C1 acidosis in a dromedary camel, increased systemic levels of lactic acid were not observed [97].

Signs. The signs of C1 acidosis are similar to cattle with grain overload. Signs of toxic indigestion may not develop until 12–36 hours after engorgement. Signs of engorgement include weakness, incoordination, depression, anorexia, C1 atony, colic (grunting, grinding teeth), abdominal distention, dehydration, a fetid diarrhea, and recumbency. The respiratory rate increases because of the acidosis. Without treatment, death may occur in 24–48 hours. Some of the signs will persist during recovery. Camelids are not likely to develop laminitis as in equids and ruminants.

Diagnosis. The history of access to concentrates is vital to the diagnosis; however, it may be difficult to establish the amount consumed. Clinical signs are valuable in making a diagnosis. The pH of the ingesta of C1 is normally around 6.4–7.0. If the pH of the first compartment of the stomach of a camelid drops below 5.5, the microenvironment is affected and a transfaunation may be necessary to re-establish C1 flora.

Treatment. If possible, evacuation of the stomach will decrease the amount of fermentable carbohydrates and accumulated acids and will prevent other orally administered products to work more effectively without dilution. Cases of acute forestomach acidosis are often accompanied by C1 contents that are more liquid than normal making evacuation via stomach tube more successful. A C1 gastrotomy may be necessary to achieve significant evacuation.

The administration of enteral antacids to correct the forestomach acidosis and parenteral fluids with or without sodium bicarbonate to correct systemic dehydration and acidosis may be necessary. Magnesium carbonate or magnesium hydroxide should be given in large volumes of warm water so as to disperse throughout C1; 3 L may be used in an adult llama. The dose of the magnesium powder should be 1 g/kg in early acute cases and 0.5 g/kg if the stomach has been evacuated. Additional therapy should include thiamine HCl, IV fluids, antibiotics, and flunixin meglumine.

Intestinal Disorders

Ailments of the intestinal tract of camelids are similar in etiology to those of ruminants. Conditions diagnosed in llamas include enteritis, obstruction, ulceration with and without perforation, colitis, proctitis, rectal prolapse, and GI parasitism. Making a diagnosis can be challenging due to the stoic nature of the species and the similarity in clinical signs with many diseases of the abdominal viscera. Exploratory laparotomy and laparoscopy may be useful in making a diagnosis and have an added benefit of being therapeutic for some conditions.

Enteritis

The inflammatory response of the camelid intestinal mucosa varies from hyperemic to catarrhal and hemorrhagic, progressing to necrosis of all layers of the intestinal wall. Specific names may be given in reference to the specific area involved (duodenitis, jejunitis, ileitis, cecitis [typhlitis], colitis, and proctitis).

Etiology. Enteritis may be primary or secondary to many other diseases. Infectious diseases that often result in enteritis include salmonellosis, colibacillosis, paratuberculosis, tuberculosis, rinderpest, and clostridial enterotoxemias. Numerous parasites can also cause enteritis and are discussed in Chapter 7. Eosinophilic enteritis is an idiopathic condition associated with eosinophilic infiltration into the lamina propria and occasionally into the submucosa [99]. Secondary enteritis may occur with any of the obstructive lesions and also with septic metritis and mastitis. Proctitis and rectal tearing may result from a too vigorous rectal examination.

Signs. The primary manifestation of enteritis is diarrhea, however, proximal enteritis without colonic involvement may not result in diarrhea due to the high water-extracting capability of a normal camelid colon. The nature of the feces aids in establishing the location and/or severity of the enteritis. The feces may have a fetid odor and contain excessive mucus, blood, undigested feed particles, and even shreds of sloughed mucosa.

Other signs of enteritis are dehydration, electrolyte imbalance, tenesmus, anorexia, colic, depression, stomach atony, plus or minus fever, and increased intestinal

borborygmus. No intestinal sounds can be heard in the healthy llama; the presence of intestinal borborygmi are a sign of intestinal fermentation, resulting in a mixture of gas and fluid in the intestine.

DIAGNOSIS. Clinical signs should determine diagnosis. An etiologic diagnosis requires cultures, fecal examination for cytology and parasitic ova, and hematology and blood chemistry evaluations. Colitis and proctitis may be evaluated by endoscopic examination. Colonic biopsies may be a diagnostic aid.

TREATMENT. In management of enteritis, it is crucial to monitor and correct any fluid and/or electrolyte imbalance. It is particularly important to provide glucose and sodium. Acidosis is common, so giving sodium bicarbonate is usually necessary. Intestinal protectants are frequently given, but their value is questionable. These include kaolin, pectin, bismuth, and subsalicylate (Pepto-Bismol). Broad-spectrum antibiotic therapy is indicated, not only to treat a primary infection, but to treat or prevent generalized secondary infections, since the permeability of the intestinal wall may be increased by enteritis.

Obstruction

ETIOLOGY. Partial and complete intestinal obstruction frequently occurs in camelids. It is reasonable to assume that any obstructive etiology affecting other species can occur in camelids. Specific causes of obstruction described in camelids include obstipation, sand impaction, bezoars, enteroliths torsion, intussusception, compression from the gravid uterus, strangulation from hernias, neoplasia, abscessation, infarction, and intestinal atresia (Figures 12.70–12.72) [5, 52, 55, 60, 100–102].

SIGNS AND DIAGNOSIS. The signs of blockage vary with the location and completeness of the obstruction,

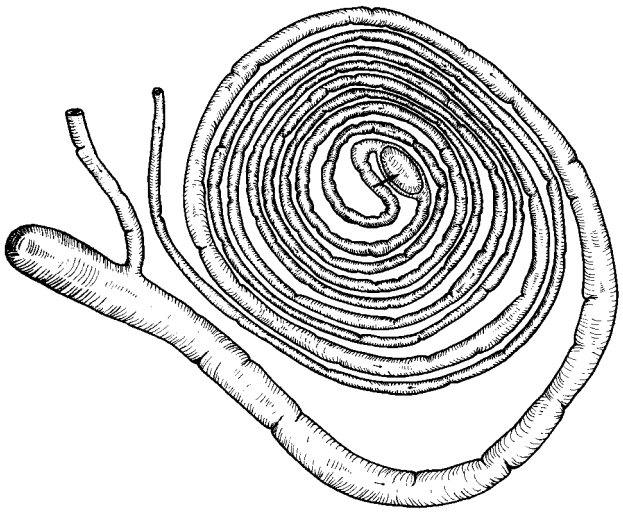


Figure 12.70 Diagram of spiral colon impaction in a llama.

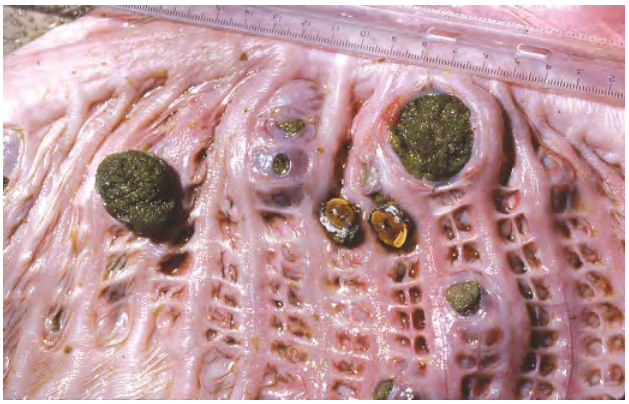


Figure 12.71 Gastroliths in the glandular saccules of C1 in a llama.

Figure 12.72 Postmortem examination revealing jejunal torsion.

the rapidity of development, the degree of enterotoxemia produced, the degree of vascular occlusion, and the production of gas. The major sign is colic, with little or no passage of feces. Proximal obstruction tend to be associated with higher degrees of metabolic derangements and have poorer prognosis than distal obstructions [55].

Strangulating obstructions also compromise blood flow to the affected bowel. When obstructions are strangulating, the ensuing sequence of events significantly alters the integrity of the bowel. In a partial vascular compression, the venous drainage is adversely affected.

Arterial blood continues to flow into the area, causing edema. If compression is complete, there will be tissue anoxia and increased capillary permeability, followed by intramural and mucosal hemorrhage. This is accompanied by edema and loss of the integrity of the epithelium, which allows bacterial invasion of the intestinal wall and, ultimately, bacteremia and peritonitis. Ischemia becomes complete and necrosis occurs. Tissue break-down products from the necrosis may be absorbed from the peritoneal

cavity and contribute to cardiovascular collapse (shock). This is an important clinical entity, characterized by a syndrome called the acute abdomen [55].

Gastrointestinal Bezoars

GI bezoars have been reported in both SACs [60] and camels [5]. Bezoars represent one of the most common etiologies necessitating abdominal surgery in camelids. These can be comprised of hair (trichobezoar), plant material (phytobezoar) or a mixture of both (trichophytobezoar). Bezoars can also have a mineral component (see Mineral Concretions below). Bezoars may be an incidental finding or the cause of intermittent to complete obstruction. Objects identified at the Holocene archeological sites of the Argentine Pampas share many resemblances to bezoars from present-day guanacos suggesting that bezoars were likely present in prehistoric camelids [103].

It is not normal camelid behavior to groom themselves by licking, however, animals with ectoparasites or pruritis may ingest large quantities of fiber from excessively biting themselves. Crias with dams deficient in milk, may exhibit aberrant suckling behavior, inadvertently ingesting fiber.

Bezoars within the gastric compartments are often incidental findings as the luminal diameter is large enough that obstruction does not occur. However, the gastric compartment can act as a repository for intestinal bezoars. Other signs include anorexia, cessation of fecal passage, colic, refusal to drink, and depression. Hematologic and blood chemistry findings may vary from normal to those of an inflammatory syndrome in the face of peritonitis or severe gastritis. Dehydration is reflected in an elevated packed cell volume and hyperproteinemia. Evaluation of abdominal fluid obtained via abdominocentesis is a valuable adjunct, particularly in establishing the presence of peritonitis.

Common small intestinal locations for obstructions resulting from bezoars are areas where the intestinal lumen rapidly narrows at the pylorus and descending duodenum (aborad to ampulla). The diameter of the large intestine decreases by five times in the spiral colon and the central flexure of the spiral colon is another common location for obstruction [55]. Bezoars located at the reflection of the spiral colon may not be obvious at surgery or necropsy because the intestine is hidden by the fibrous tissue that binds the spiral colon together.

Clinical signs vary depending on the location of the obstruction with proximal (orad) obstructions being associated with more severe clinical signs and metabolic derangements. Animals with proximal obstructive lesions have higher forestomach and lower serum chloride concentrations compared to normal animals and those with distal obstructive lesions [55]. Animals with distal obstruction commonly present for non-specific signs of lethargy [101].

Figure 12.73 Multiple trichophytobezoars removed from C1 of an alpaca cria. A quarter is included in the photograph for size reference.

The optimal surgical approach for removal of the offending bezoar is dependent on the location of the obstruction within the GI tract [104]. Left flank surgery may be required to empty C1 of accumulated hair. Often, the first stomach compartment can allow for many bezoars to accumulate that can cause an obstruction if they pass down the intestinal tract (Figure 12.73). The spiral colon can be accessed through a ventral midline celotomy [52, 101] or a caudal right flank (paralumbar) approach. A right paracostal approach will provide superior access to the descending duodenum, pylorus, or C3 [60] (see 12.6).

Mineral Concretions

Most of the mineral concretions found in the stomach or intestine of camelids are the result of precipitation of consecutive layers of mineral matter (phosphates) around a central kernel of plant fiber or seed awn. Mineral stones are common in the saccules of the camelid stomach [105–108] (Figures 12.71).

Sizes of gastroliths vary from 1 mm (1/32 in.) in diameter to roughly the size of a medium chicken egg. The surface of the gastrolith may be smooth or rough.

The composition of GI concretions is complex. Preliminary studies show that the mineral composition includes various forms of calcium and phosphate. Factors that may contribute to the formation of phosphate concretions in the intestinal tract may include diets high in magnesium or high pH of the ingesta. One report showed llama C1 concretions comprised of two different hydrates of magnesium phosphate (Bobierrite and Newberyite) [108]. Similar concretions in other species have yielded struvite (magnesium ammonium phosphate) on analysis. Frequently, a nidus of plant fiber serves as the nucleus for precipitating the mineral from the normal stomach contents. The mechanism that triggers the process is unknown.

Bicarbonate is secreted by the saccules producing a slightly alkaline environment within the saccule. This

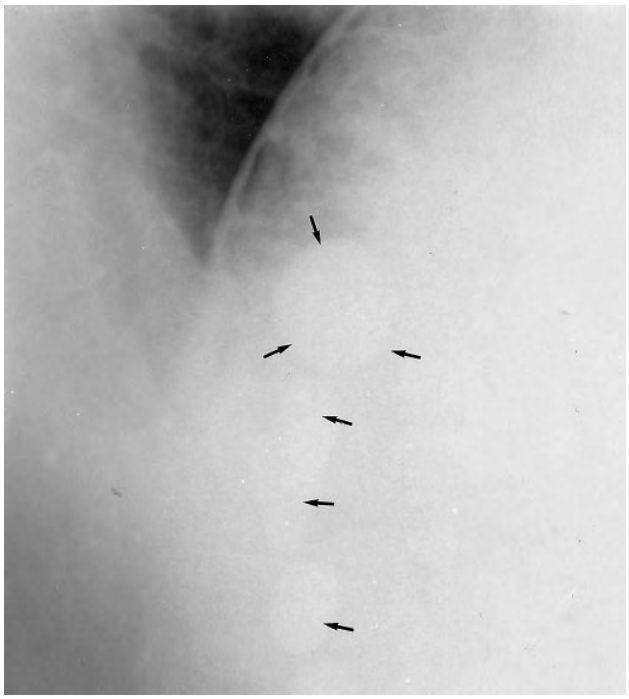


Figure 12.74 Lateral abdominal radiograph of a llama revealing gastroliths *in situ*.

alkaline environment is conducive to the precipitation of phosphates and carbonates, forming concretions over a period of months or years. Decreased motility of C1 may allow mineral to accumulate as the saccules do not evert at regular intervals [108].

Concretions located in the saccules of C1 cause no impairment of gastric function and do not require therapy. The stones are seen as incidental findings on radiographs (Figure 12.74) of the midventral abdomen or at necropsy. It is rare for them to cause GI obstruction. Ulceration or perforation due to the concretion is possible but rare.

Intestinal Ulceration

Etiology. Ulceration of the intestinal mucosa may follow any severe enteritis and develop at any location from the duodenum to the rectum. Common sites are the proximal duodenum and the spiral colon. In many cases, no indication of the cause can be determined, even at necropsy.

Ulcers can result from ischemic necrosis caused by an intestinal spasm around an impaction of dried fecal matter. Multiple ulcers may develop in such a case because the intestine may temporarily relax sufficiently for the impacted mass to move on, only to spasm again. This is sometimes observed in equine small-colon impactions, when chemotherapy results in temporary relaxation of the muscles. Such ulcers frequently necrose through the entire wall, perforating the intestine.

Diagnosis. It is extremely difficult to diagnose a primary intestinal ulcer. Elimination of other intestinal disorders should precede the tentative diagnosis, but an antemortem diagnosis of intestinal ulceration is always questionable. Treatment follows similar principles to gastric ulcers (see Section 0).

Rectal Prolapse

Etiology: Rectal prolapse is uncommon in camelids. The most common cause is tenesmus, associated with prolonged, severe diarrhea. A mild protrusion, lasting only a few minutes, may be observed following defecation. A slight protrusion of both the rectal and vulvar mucosae has been noted in females near term, especially when lying down; pressure from the large fetus pushes organs into the pelvic canal.

Signs. The extent of the protrusion is important because potential disruption of the blood supply to the invaginated segment may occur with large prolapses. This is very rare but there has been a report of a llama with 6 ft of the colon prolapsed initially on presentation with an additional 3 ft protruding within the following few minutes. The llama was euthanized, and necropsy findings demonstrated that the mesentery and vascular supply had been stripped from the colon [109].

A protrusion of 5–15 cm may not compromise the vascularity of the rectum, but if the prolapse is 20–30 cm, ischemic necrosis of the bowel may occur from compression of the vessels. In less severe cases, the mucosa may be traumatized by laceration or abrasion from the tail or dirt when the animal is lying down. Edema is a likely result, and necrosis of the mucosa may occur.

See 12.6.10 on rectal prolapse later in this chapter for treatment.

Rectal Laceration

Etiology. A rectal tear may occur during breeding, but camelid copulation is not accompanied by as vigorous a pelvic thrust as is seen in cattle or horses. Prolonged dystocia, especially with manual manipulation, may traumatize the rectal wall and allow either immediate or delayed rupture of the wall. The most likely cause of a rectal tear is an accident at the time of a rectal examination [110]. The pelvis of even a large llama is small, and the size of the rectum is commensurate with the size of the animal. The rectum of adult camels will usually permit palpation, but similar caution should be used in camel calves.

Straining against an inserted hand and arm may be lessened by using epidural anesthesia or by mixing local anesthetic with the lubricant. Even under epidural anesthesia, a colonic spasm may trap an arm caudal to the wrist. It is necessary to wait until the spasm relaxes before removing the arm to avoid splitting the wall of the colon.

DIAGNOSIS. The mucosal split may be felt at the time it is happening, but it is more likely that blood will be seen on the hand when it is withdrawn. Camelids are intermediate between mares and cows in terms of susceptibility to trauma of the rectal mucosa. As a result of tiny splits in the anal mucosa, it is not uncommon to see traces of blood on the back of the hand or wrist when finishing an examination. After a prolonged examination, the mucosa may be slightly abraded, and a small quantity of blood will be seen on the fingers. Neither of these conditions should be confused with the large quantity seen if a laceration has occurred. Copious quantities of blood in the palm of the hand or on the fingers should alert the clinician to the possibility of a mucosal tear.

If a laceration is suspected, the rectum should be carefully explored with a speculum, under epidural anesthesia, to determine the extent of the laceration. The rectum should not be explored with a digit, because this may enlarge a tear.

TREATMENT. Lacerations that penetrate deeper than the mucosa should be sutured. See section 12.6.10 on surgical treatment of rectal lacerations later in this chapter.

Diseases of the Abdominal Cavity

Peritonitis

Peritonitis is inflammation of the serosal surface of the abdominal viscera or the wall of the abdomen. The inflammation may be focal or diffuse. Peritonitis results in fibrin deposition in the abdomen, which functions to limit spread of contamination but also results in adhesions. Fibrinous adhesions can become fibrous adhesions and are a natural consequence of an inflamed serosa in chronic states of disease. Adhesions can be incidental findings at surgery or postmortem examination. Adhesions can limit the intestines' natural ability to slide along one another limiting their ability to push ingesta through its lumen. Widespread inflammation of the peritoneum can cause a functional ileus or adhesions can result in a physical obstruction.

There are numerous causes of peritonitis. Peritonitis can result from uterine and vaginal tears secondary to the normal process of criation or iatrogenic from fetal manipulation during a dystocia [111]. Ruptured bladder can result from urethral obstruction or blunt trauma to the abdomen. Abdominal abscessation can result from peritonitis or can result in peritonitis if the abscess ruptures or leaks. GI perforation can result in peritonitis in SACs and camels [112, 113]. Perforation can be due to GI blockage, foreign body [4], gastrolith [112], or can be iatrogenic from a traumatic rectal examination [114]. Full-thickness GI ulceration [58, 87] can result in abdominal contamination and is a common cause of septic peritonitis in camelids. Also known as "alpaca fever," *Streptococcus equi subsp zooepidemicus* can cause multisystemic disease which can include peritonitis as part of its disease complex [115–117].

Signs of peritonitis include colic, tense abdomen, gastric atony, ileus, weakness, diarrhea, painful movement, and increased recumbency. Fever may be present but is an inconsistent sign observed in animals with peritonitis. In severe cases, or if left untreated, death can occur in hours to days.

The collection of abdominal fluid by abdominocentesis (see Chapter 4) can provide diagnostic value. Peritonitis results in excessive peritoneal fluid accumulation and the peritoneal fluid will have increased numbers of inflammatory cells. However, with localized peritonitis, the abdominocentesis may not reflect the true inflammatory process. Abdominal ultrasound (US) can be useful to locate pockets of fluids and guide aspiration (Figure 12.75). An inflammatory leukogram may be present with diffuse disease.

At necropsy, peritonitis may be acute or chronic (Figures 12.76 and 12.77) and must be differentiated from serous atrophy of fat.

The prognosis for diffuse peritonitis is grave, especially if accompanied by perforation of the bowel, and euthanasia is recommended in these cases. If treatment is elected, the source of the peritonitis should be identified and corrected if possible. Abdominal lavage is possible but, because of the complexity of the abdomen, it is difficult to remove all of the contamination. Indwelling abdominal drainage systems are often unrewarding because they quickly become occluded with fibrin clots. Administration of broad-spectrum antibiotics and supportive therapy, including IV fluids will be necessary. Because of the loss (or sequestration) of high protein fluid, affected animals often become hypoprotenemic. Administration of plasma or blood products may be necessary to restore protein levels.

Figure 12.75 Transabdominal ultrasound image in an adult alpaca. Excessive abdominal fluid accumulation is evident at the top of the image.

Figure 12.76 Acute peritonitis, with fibrin tags.

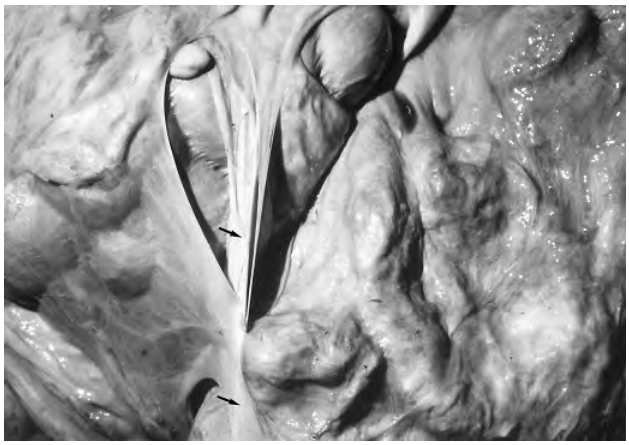


Figure 12.77 Chronic peritonitis, with fibrous bands that may incarcerate a loop of bowel.

Intraabdominal Hemorrhage

Hemorrhage may result from the rupture of any major vessel in the abdominal cavity or from extensive capillary oozing. Primary coagulopathy is rare in camelids. Common sources of hemorrhage are a ruptured liver or spleen from abdominal trauma. The cranial uterine artery is in the freely moveable broad ligament and may be subject to trauma, particularly late in pregnancy. The umbilical arteries may rupture too close to the body wall in the neonate and retract into the abdomen without closing. If the spermatic cord is under excessive tension at the time of transection of the cord with an emasculator, the artery may retract into the abdomen and continue bleeding.

The severity and rapidity of development vary with the rate of blood loss. The llama first becomes weak. The heart rate increases. There may be dyspnea and pallor of the mucous membranes and cool extremities. A hemogram is of little help until 8–10 hours have passed.

Even though the packed cell volume will not change immediately, it should be done to provide a base for monitoring and to evaluate the response to therapy. Also, the blood should be checked for possible clotting defects. If blood loss is thought to be caused by rupture of an abdominal vessel, it may be necessary to investigate the site via laparotomy. In one case, the liver had been ruptured, and it was possible to correct this at surgery.

Blood transfusions are possible if a suitable camelid donor is available. Various blood types have been identified in the llama and alpaca [118], but usually a patient can receive blood at least once (prior to formation of antibodies against the donor blood) without complications, but risk of a transfusion reaction increases with subsequent transfusions. A retrospective study of 22 alpacas that received a whole blood transfusion without crossmatching reported adverse events (pulmonary edema, hyperthermia, irregular heart rhythm) in four animals that responded to therapy or discontinuation of the transfusion [119].

Diseases of the Liver

Hepatic Insufficiency

Hepatic insufficiency (HI, liver failure) is a syndrome common to all vertebrates. The liver has great reserves (80% of the hepatocytes must be damaged before the liver fails) and great regenerative ability. However, once the critical threshold has been reached, a process begins that may cause the death of the animal within 36–48 hours. Lesions of the liver may be toxic, degenerative, proliferative, or inflammatory.

The most commonly reported hepatic disease in SACs is hepatic lipidosis [120–124]. Other causes include parasites (*Fasciola hepatica*, *Lamanema chavezi* [125]), infections (adenovirus, *Rhodococcus equi*, tuberculosis, coccidioidomycosis), hyperthermia, obstructive processes (fibrosis, cholelithiasis [126]), anesthetic agents [127], elemental phosphorus, halogenated hydrocarbons, copper [128–130], pentachlorophenols, tannins, cresols, plant toxins (lupinosis, blue-green algae, alsike clover, lantana, mushrooms, cocklebur, sneezeweed), hepatic cysts [131, 132], and neoplasia [133, 134]. Additional causes include hematogenous abscesses, gastritis (C1), septic metritis and mastitis, peritonitis, rupture of the liver, malnutrition (cobalt deficiency, selenium deficiency), and mycotoxins (sporidesmin, aflatoxin). Portal hypertension caused by cardiac insufficiency also causes decreased hepatic function.

The syndrome produced by HI is similar among species and similar regardless of the inciting cause. Clinical signs of HI are non-pathognomonic, similar to signs of most sick camelids: depression, recumbency, and weight loss. Different from the horse, the ruminant and camelid are generally more depressed and comatose rather than violent and active. Hepatoencephalopathy can be associated with

seizure-like activity [122]. Additional signs that may be seen at certain stages of the syndrome or in various species include anorexia, decreased growth rate, unthrifty appearance, icterus, photosensitization [135, 136], hemorrhage, tenesmus, rectal prolapse, hemoglobinuria, and diarrhea.

Photosensitization is a sign seen in some hepatopathies. Normally, the liver degrades chlorophyll to phylloerythrin, which is further degraded to inactive substances. In HI, the liver fails to decompose phylloerythrin, which then accumulates in the peripheral circulation. Phylloerythrin is a photodynamic agent. For a SAC to be photosensitized, nonpigmented skin must be exposed to the ultraviolet rays of sunshine. Heavily fibered areas of the body are protected. Clinical signs of photosensitization are similar to those of sunburn. Photosensitization is only one sign of HI and is relatively insignificant in the overall management of the case. Placing SACs inside a barn prevents progressive skin damage from the effects of sunlight.

Hemoglobin, bile pigments, or both may be seen in the urine of animals suffering from HI. Hemolysis is not of a magnitude to produce anemia.

Though icterus is a consistent sign associated with HI, it may be absent in a small percentage of terminal cases and is rarely observed in simple hepatic lipidosis. Diseases other than primary hepatic disease, such as hemolytic conditions and obstructive intestinal disorders, also produce icterus.

Routine hematology and serum biochemistry may be highly indicative of liver pathology. It is not usually possible to establish an etiologic diagnosis based on gross necropsy findings. The liver may be large, small, soft, firm, yellowish, darkened, or mottled. Postmortem decomposition begins quickly if the bodies are not kept cool. It is not uncommon for a diagnosis of hepatopathy to be changed after microscopic examination (Figures 12.78–12.80).

Figure 12.78 Gross postmortem examination of a camel revealing hepatitis.

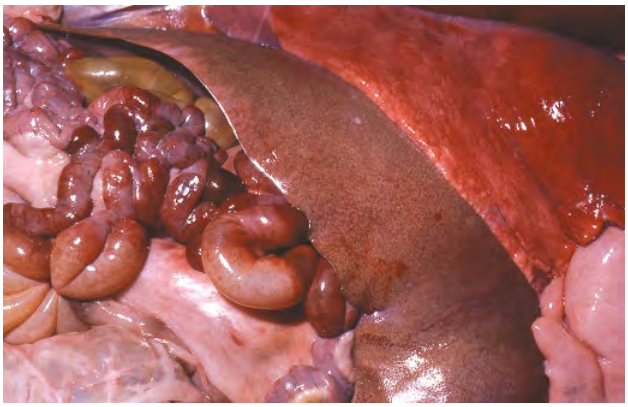


Figure 12.79 Gross postmortem examination of a llama revealing hepatic necrosis.

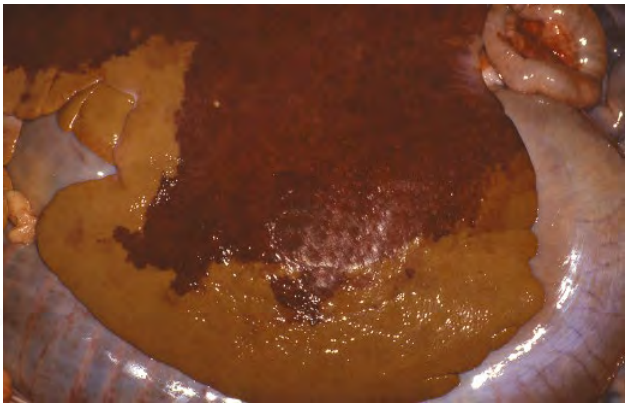


Figure 12.80 Gross postmortem examination of a llama revealing hepatic lipidosis.

Hepatic Lipidosis

Hepatic lipidosis is not a specific disease but rather the end result of one or more of the metabolic processes:

1) Increased mobilization of depot fat
2) A decreased rate of oxidation of mobilized fat
3) An increased formation of fat
4) A reduced rate of removal from the liver

Though camelids are often made physiologically analogous to ruminants, they respond differently to states of negative energy balance. While ruminants tend to develop ketosis during states of negative energy balance, camelids are less prone to ketosis but more commonly develop hyperlipemia and hepatic infiltration of the liver (hepatic lipidosis) (Chapter 3).

In order to begin to understand the cause of hepatic lipidosis in SACs, it is necessary to know how they subsist in their native countries. In South America, these animals are never fed concentrates or supplements and rarely are fed cured hay. They feed on native grasses and forbs. Llamas and alpacas are concentrated in the Altiplano, which is an

area of broad, high-altitude valleys and plateaus at elevations over 12,500 ft (3800 m). The climate of this area consists of a long dry season and a short, wet season, with 75% of the rainfall occurring from December through March. The dry season is from May through October.

The growing season (wet) is characterized by low temperatures (300+ nights of frost per year) and intense solar irradiation. Daytime temperatures reach a maximum of 18.3 °C (65 °F) with nighttime temperatures of −12 °C (10 °F). Available forage varies from the wet to dry seasons, but the animals adapt to the "feast and famine" routine by depositing layers of fat in the subcutaneous, muscular, and retroperitoneal tissue during the wet season and mobilizing these reserves in lean times. This is an evolutionary adaptation to good foraging followed by seasonally deplorable feeding conditions, and this adaptation is usually completely forgotten by North American managers. It is generally assumed by owners that SACs consume only enough to satisfy their requirements. This is not true, and some animals may become obese because of overfeeding (Chapter 3).

The generic causes of hepatic lipidosis include starvation, low-protein diet, high-fat diet, insulin insensitivity, deficiency of thyroxine, deficiency of pituitary hormones, and stress. Stress, and increased energy demands from pregnancy and lactation also predispose. Toxins, prolonged hypoxia or anoxic episodes may contribute.

Signs may not point directly to liver malfunction. Icterus is not a sign of uncomplicated lipidosis. Approximately 30 pregnant llamas died over a period of two months on one farm. The first signs noted were blindness and staggering. The animals became recumbent but continued to eat and drink up to the time of death. Most of the llamas were in fair to good condition at the onset of signs, but if they lived for days to weeks, they gradually lost weight. Later, in the course of the outbreak, abortion became a factor in the syndrome. Forty-five females aborted; eight of the forty-five died. Three stillbirth deliveries occurred.

Clinical pathology findings in many of the cases were not helpful. A few llamas had hyperlipemia [137]. Table 12.4 lists values for animals with hyperlipemia. Glucose levels were generally elevated (300–650 mg/dL), but this is true with any SAC that is anorectic. The CNS signs are explainable on the basis of hepatoencephalopathy generally thought to be caused by increased blood ammonia levels. Ammonia levels were measured in one of the above llamas and found to be elevated (550 μg/mL [normal, <5 μg/mL]). The only consistent lesion found at necropsy was hepatic lipidosis and renal necrosis. If renal function and hepatic function were diminished, the prognosis was grave. Late in the course of the disease outbreak, hypoinsulinemia was considered. Histopathology of the pancreas revealed a paucity of β cells. Glucose tolerance tests indicated insulin resistance.

Table 12.4 Selected biochemical parameters and blood gases from SACs with hyperlipemia.

Parameter	Alpaca	Llama
Glucose mg/dL	436	635
Creatinine mg/dL	6.3	4.8
S urea N mg/dL	27	132
Potassium mEq/L	2.6	1.3
Phosphate mg/dL	0.5	1.3
Triglyceride mg/dL	1564	5658
Cholesterol mg/dL	193	297
AST U/L	1331	888
SDH U/L	37	
GGT U/L	217	
Blood pH	7.36	
PO_2 mmHg	30	
PCO_2 mmHg	30	
Bicarbonate mEq/L	16.9	
Base excess mEq/L	−6.5	

The prognosis for acute hepatic necrosis is unfavorable [124]. Treatment is based on nutritional support [138] (see Chapter 3). In mild cases, the therapeutic rationale is to correct any dietary deficiency and electrolyte imbalance. Administration Vitamin B complex and rumen transfaunation is thought to be an appetite stimulant. Broad-spectrum antibiotic therapy is indicated because secondary bacterial infection is frequently seen in any hepatopathy. Hyperglycemia and insulin insensitivity is common in camelids and is thought to contribute to the hepatic lipidosis syndrome [139–141].

Abdominal and Gastrointestinal Surgery

Surgical Anatomy

To perform abdominal surgery, the anatomy of the abdominal musculature must be understood. The cutaneous trunci muscle is limited to the specialized preputial muscles in the male and a few fibers on the ventral midline in the female. The abdominal tunic (deep fascia) is less well developed in SACs than in the horse or cow. It is comprised of fibroelastic tissue that adds support to the muscles and their aponeuroses. The tunic covers the external abdominal oblique muscle dorsally and cranially and is an integral part of the aponeurosis of the external oblique muscle ventrally and caudally. The abdominal tunic extends into the

inguinal area and helps to form the superficial inguinal ring.

A specialized section of the abdominal tunic becomes the medial suspensory ligament of the mammary gland. The suspensory ligaments of the mammary gland are paired, but the right and left halves are not as easily separated at surgery as they are in cattle.

The external abdominal oblique muscle is the most superficial of the abdominal muscles. Its fibers are directed caudally to slightly ventrally. This muscle originates on the caudal border and lateral aspect of the last few ribs and the fascia over the intercostal muscles. The body of the muscle is comprised of short fibers and is approximately 1 cm thick. An extensive aponeurosis inserts onto the tuber coxae, prepubic tendon, and linea alba. The fascia of this layer is the most important structures when closing an abdominal incision as it provides the greatest support to the closure of the body wall.

The internal abdominal oblique muscle lies beneath the external abdominal oblique muscle (Figure 12.81). Its fibers are directed cranioventrally and medially. This muscle has its origin on the tuber coxae and the deep lumbar fascia. The body of the muscle is comprised of short fibers and is 1–2 cm thick. Ventrally and cranially an aponeurosis blends with the aponeurosis of the external oblique muscle. These combined aponeuroses (abdominal tunic, internal and external abdominal oblique muscles) insert on the caudal border of the last rib and the linea alba and are the main fibrous support for the abdominal organs. They form the outer sheath of the rectus abdominis muscle.

The fibers of the transversus abdominis muscle are directed ventrad and mediad (Figure 12.82). The aponeuroses of the transversus muscle lie dorsally and ventrally. The dorsal aponeurosis originates in the deep lumbar fascia and the transverse processes of the lumbar

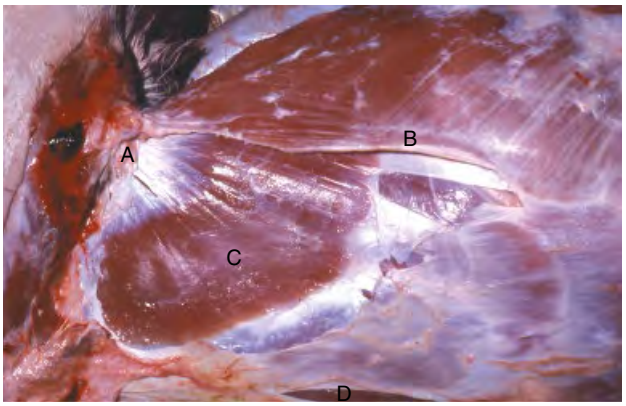


Figure 12.81 Position of the internal abdominal oblique muscle. (A) Tuber coxae, (B) external abdominal oblique muscle, (C) internal abdominal oblique muscle, (D) rectus abdominis muscle.

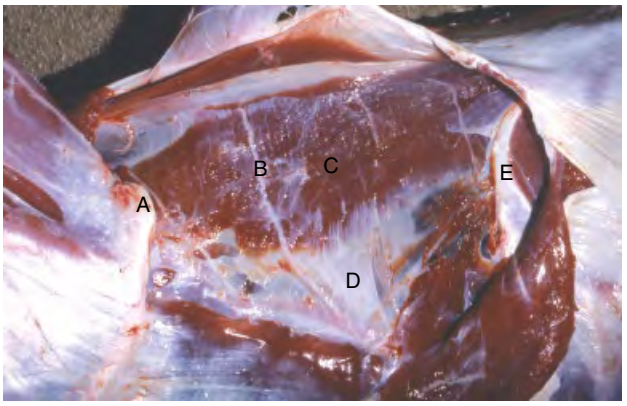


Figure 12.82 Lateral abdomen of a llama demonstrating the position of the transversus abdominis muscle. The external abdominal oblique and the internal abdominal oblique muscles have been removed. (A) Tuber coxae, (B) lumbar spinal nerve, (C) body of the transversus muscle, (D) ventral aponeurosis of the transversus muscle, (E) costal arch.

vertebrae and the costal arch; it is 10 cm long cranially and 4 cm long caudally. The length of the body of the muscle is only 9 cm, continuing on as the ventral aponeurosis, which inserts on the linea alba deep to the rectus abdominis muscle.

The abdominal wall of the camelid has unique features. There is no fold of skin from the lateral abdominal wall (flank) to the limb, above the stifle as there is with ruminants. The paralumbar fossa is not prominent. The only muscle fibers over the area bordered by the last rib, the transverse processes of the lumbar vertebrae and the tuber coxae, are those of the external abdominal oblique muscles at the cranial aspect and some of the internal oblique muscle fibers caudally.

In the female camelid, the mammary gland is situated cranial to the pubic attachment of the rectus abdominis muscle. If it is necessary to perform a caudoventral midline incision in a female, such as that used to perform an ovariohysterectomy, elevation and reflection of the gland caudally may be necessary to allow adequate exposure.

In the male camelid, the prepuce and penis are located in a similar area. The external preputial orifice of a mature alpaca male is located 15 cm cranial to the rim of the pelvis. The prepuce opens caudally in a sexually unaroused male. The prepuce is under the control of two pairs of muscles that are modifications of the cutaneous trunci muscle (Figure 12.83). The cranial preputial muscle pulls the preputial orifice forward when the male is aroused sexually. The caudal preputial muscle pulls the preputial orifice caudally as arousal subsides.

It is necessary to transect one of the cranial preputial muscles when making a ventral midline approach to the caudal abdomen. The muscle segments must be accurately

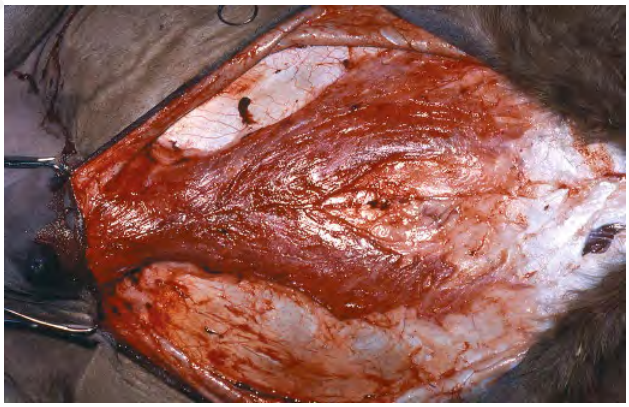


Figure 12.83 Preputial muscles of the llama.

Figure 12.84 Cradle used to restrain llama in dorsal recumbency.

re-apposed and sutured during closure to avoid subsequent malfunction.

The umbilicus is located slightly cranial to the midpoint of the distance between the xiphoid cartilage and the pubis. The linea alba is indistinct in the llama, particularly caudal to the umbilicus.

Patient Positioning

A ventral midline approach requires dorsal recumbency. Though elaborate surgical tables are employed in university and private clinics, a simple cradle may suffice to hold a llama in the correct position (Figures 12.84 and 12.85). Smaller patients may be lifted onto an elevated platform composed of bales of straw to avoid working on the floor or having to bend over during the surgery. It is possible to do some high flank surgeries with the animal standing as in cattle; however, camelids are more likely to lay down during restraint, which can complicate the surgical procedure. Many surgeons recommend camelids being in lateral recumbency for a high-abdominal-wall approach [142].

Presurgical Fasting

Camelids should be fasted prior to episodes of general anesthesia or long durations of recumbency. There are no published research investigating the optimum duration of fasting of camelids prior to these procedures. Often extrapolation is made from ruminants where recommendations vary but are typically between 24 and 48 hours prior to an anesthetic episode [143]. In adult camelids, food should be withheld for 12–36 hours prior to the anesthetic episode [142]. Water should be withheld for 12 hours prior to elective surgery on an adult SAC. Withholding feed for longer duration may be counterproductive because it tends to make the stomach contents waterier and may actually increase the incidence of spontaneous regurgitation. Feed and water should not be withheld from neonates.

Camelids are less likely to develop tympany of C1 than ruminants. Regurgitation under anesthesia seems greater when the patient is excited and is actively regurgitating as a defense mechanism prior to anesthesia induction. Animals should be kept as calm as possible prior to

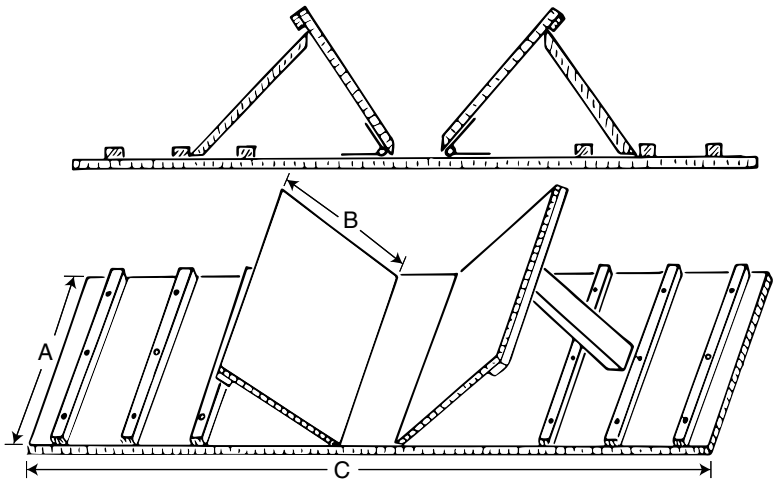


Figure 12.85 Diagram of camelid cradle. (A) 30 cm, (B) 38 cm, (C) 125 cm.

anesthesia induction. If the animals are extremely agitated and spitting (regurgitating) prior to anesthesia, elective procedure should be postponed.

Anesthesia

See Chapter 6.

Approaches to the Abdomen

Ventral Midline

The tissue layers penetrated vary with the site of the incision. Cranial to the umbilicus, the layers encountered are the skin, subcutaneous areolar tissue, a single layer of fascia comprised of the aponeuroses of the abdominal muscles, deep abdominal fat, and the peritoneum. Caudal to the umbilicus, layers found are the skin, subcutaneous tissue, cutaneous trunci muscle, superficial fascia (aponeuroses of the external and internal abdominal oblique muscles), deep fascia (thin aponeurosis of the transversus abdominis muscle), deep abdominal fat, and peritoneum. Lateral to the midline, the two fascial layers are separated by the paired bodies of the rectus abdominis muscle. Ideally, the incision should be made directly on the midline to avoid incising the muscles that will complicate entrance of the incision with paramedian incisions.

Closure also varies with the incision site. Cranial to the umbilicus, the suture layers include the peritoneum, fascia, and skin. Caudal to the umbilicus, the body wall is significantly thinner than the body wall cranial to the umbilicus [144], and in this location, it is important to ensure inclusion of all the fascial layers into one or more suture layers. The muscle body of the rectus abdominis muscle is usually included in the suture of the deep fascia, but the fascia is the tissue that provides support for the abdominal wall, and it is necessary that the external rectus fascia be included in the closure.

Often, the author will place an interrupted tension relieving suture in the middle of the incision to oppose the edges and facilitate the reminder of the closure. The body wall of ventral midline incisions can be closed with any appositional suture pattern. Inverted cruciate sutures have been recommended for linea closure (Figure 12.86) [142]. Biomechanical testing has been shown that the linea alba when closed with a simple continuous pattern has similar breaking strength to those closed with inverted cruciate patterns and may be preferable due to ease and speed of closure [144].

The linea can be closed with a prolonged absorbable suture such as polyglactin 910 (Vicryl), a braided, absorbable suture. The linea should not be closed with chromic gut as this suture is prone to damage from mishandling and loses tensile strength quickly, making the patient prone to incisional herniation. Postsurgical dehiscence and herniation are more frequent complications with caudal midline incisions.

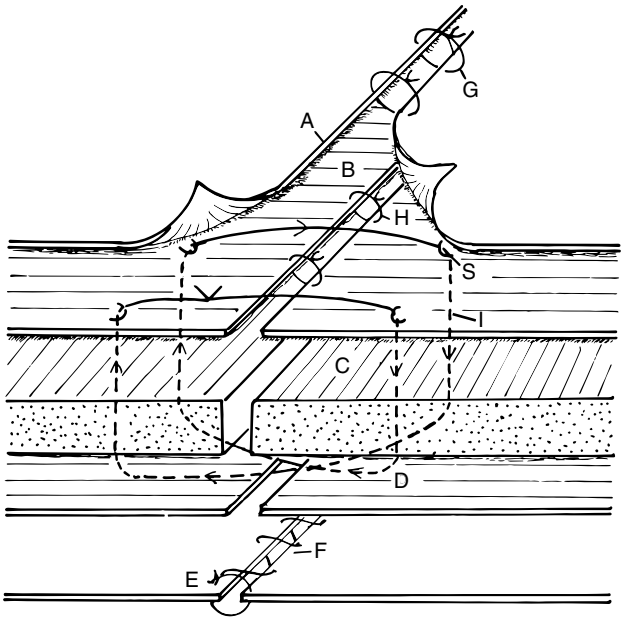


Figure 12.86 Diagram of the placement of an inverted cruciate suture for a ventral midline closure. Inverted cruciate sutures cross deep to the closure. (A) Skin, (B) superficial fascia, (C) rectus abdominus muscle, (D) deep fascia, (E) peritoneum, (F) continuous suture to close peritoneum, (G) interrupted sutures to close skin, (H) interrupted sutures to close superficial fascia, (I) inverted cruciate suture pattern, (S) suture.

Camelids do not have extensive subcutaneous tissues and generally, this layer does not require suturing. The skin can be closed using nonabsorbable suture. If suture removal is not practical, polyglactin 910 can also be used for skin.

Flank

Many surgeons have comfort with the flank approach because of the commonality of this abdominal approach in ruminants. The flank provides the surgeon with good access to much of the GI tract. If the animal will remain standing, it can be performed with the animal standing (use caution in camelids because of the high propensity of the animal lying down) or can be done with the animal awake and restrained in lateral recumbency. Though the fascial holding layers are not as substantial in this location as they are with the ventral midline approach, it has the advantage that catastrophic surgical dehiscence is rare because the entire weight of abdominal viscera are not supported by this musculature. Also, when the camelid is cushed, the flank incision is not in contact with the ground as is a ventral midline incision.

The flank approach can be performed on the left or right sides. A left flank approach is necessary for access to the C1 compartment. The left flank is also useful for access to the uterus during a Cesarean section as it is in ruminants, and C1 helps to retain other abdominal viscera during this procedure. C2 and C3 can be accessed from the right flank, as can the duodenum, and the spiral colon. The location of the flank incision can vary depending of the desired location within the abdomen.

A vertical incision should be made midway between the tuber coxae and the last rib and 6–8 cm ventral to the transverse processes of the lumbar vertebrae. If maximal intraabdominal exposure is required, the horizontal fibers of the external abdominal oblique muscles must be incised. The internal abdominal oblique and transversus abdominus muscles can be separated longitudinally in the direction of their muscle fibers. The deep abdominal fat layer, which is typically thin in this location, may be bluntly dissected and the peritoneum picked up with thumb forceps. The peritoneum should be held taut while it is penetrated with blunt dissection taking care to protect the abdominal viscera beneath.

The body wall is thin in camelids and can often be closed with a single layer incorporating the peritoneum, transversus, internal, and external abdominal oblique muscles. If the layers can be identified, they may be individually apposed with an appositional suture pattern using absorbable suture. The outer muscle fascial layer is the most important layer to include in the closure, as this layer provides strength to the abdominal wall.

A caudal flank incision can be made cranial and slightly ventral to the tuber coxae. This approach is indicated for simple ovariohysterectomy or intraabdominal orchidectomy. A diagonal incision should be made 6–10 cm ventral to the tuber coxae, ventrad and craniad in the direction of the fibers of the internal abdominal oblique muscle. The fibers of the external abdominal oblique muscles are minimal or absent at this site. The incision will penetrate the abdominal tunic, fibers of the internal abdominal oblique muscle (blunt dissection), aponeurosis of the transversus abdominis muscle, and peritoneum. Closure is similar to that for other flank incisions.

Parainguinal

A parainguinal approach can be used for accessing caudal abdominal structures such as the reproductive organs for an ovariohysterectomy. The animal is placed in lateral recumbency with the pelvic limb flexed and abducted. A 10–15-cm skin incision is made approximately 8 cm cranial and dorsal to the inguinal canal. Sharp dissection is continued to the internal abdominal oblique muscle, which is bluntly dissected in the direction of the long axis of the muscle fibers. The transversus abdominis muscle and peritoneum are penetrated in the plane of the muscle fibers. This approach is similar to the grid approach used in the horse. Using this approach, the uterus and ovaries can be identified and withdrawn from the incision for evaluation and/or amputation.

Paracostal

The duodenum and C3 are readily accessible and able to be exteriorized via a right paracostal approach [60]. With this approach, the animal is restrained in left lateral recumbency and an incision is made, parallel and immediately caudal to the last rib. In this location, the external abdominal oblique, internal abdominal oblique and transverse abdominal muscles and peritoneum are incised to gain entry into the abdomen [60].

Herniorrhaphy

Umbilical hernias, inguinal hernias, body wall hernias, and diaphragmatic hernias have been found in camelids. Hernias maybe acquired or congenital. Traumatic hernias, and those secondary to incisional dehiscence, are possible. Congenital umbilical hernia is likely to be of genetic origin, and appropriate recommendations should be made to owners who ask about breeding affected individuals [57, 145–152].

Umbilical hernias are the most common type of hernia found in crias and have been reported to represent 16% of the pre-weaning morbidity experienced by SAC crias, which is second only to diarrhea [153]. Crias are born with patency in the body wall around the umbilical vessels, but this ring should be closed within one month after birth. Usually, the tip of a finger can be inserted through the ring. Rings allowing insertion of two or more fingers or persisting for longer than one month are unlikely to close and will likely require surgical correction. Herniorrhaphy may be delayed for two to three months until the body wall musculature has become stronger. It is uncommon that the bowel will become incarcerated in the umbilical hernia.

Surgical techniques to repair hernias in other domestic animals are applicable to SACs. If the defect is less than 4 cm in diameter, the hernia may be repaired without entering the peritoneal cavity. In general, the edges of the ring in the abdominal musculature should be excised or scarified to facilitate scar tissue formation between the sides of the ring. Everting patterns like vertical mattress, overlapping patterns like “vest-over-pants” and appositional patterns like simple interrupted, simple continuous, and cruciate have been used successfully. Closure is similar to that of other ventral procedures (See closure described above for ventral midline)

GI structures and greater omentum may be present in an umbilical hernia. However, incarceration of GI structures are rare and umbilical hernias are rarely an emergent condition. Care must be taken to avoid causing iatrogenic trauma to these structures during herniorrhaphy surgery.

Closure of defects with a lot of body wall tension are challenging and the risk of re-herniation is high. Non-absorbable mesh can be used to close the defect. Rejection and infection of the mesh are possible complications with surgical use of mesh. Tension-relieving patterns such as near-far-far-near, vertical mattresses, or cruciate can be used to relieve tension on the primary closure. Large absorbable sutures like number 1 or 2 polyglactin 910 or polydiaxonone are recommended. Chromic gut should be avoided due to its friability and premature loss of tensile strength. Some or all of the sutures may be pre-placed loosely before tightening. Excessive tension on individual sutures may be avoided by pulling several sutures taut at the same time, minimizing the chance that a tension suture may pull out.

Inguinal hernias are less common than umbilical hernias in SACs. Inguinal herniation is typically thought of as a condition in males, but females can also develop herniation in this location. Indirect herniation through the inguinal rings or direct herniation through a rupture in the body wall can occur in this location [150]. Techniques recommended for cattle or swine are satisfactory for repair. In males, testicular sparing techniques where the hernial contents are reduced into the abdomen followed by partial inguinal ring closure can be performed. Breeding of affected animals should be done cautiously, as the heritability of this condition is unknown in camelids. Alternatively, complete closure of the ring can be performed following castration.

Antibiotics are not indicated for postsurgical care of a routine herniorrhaphy. In complicated hernia repair, hernia repair in animals with known infection or where non-absorbable implants (mesh) is used, perioperative antibiotics are recommended.

Proper post-operative care is important to reduce excessive tension on the body wall closure. During recovery, the animal should be kept where the footing is secure to avoid slipping, stretching, or falling. An attendant may soothe the animal to keep it in sternal recumbency until all effects of the anesthesia have dissipated. While healing, activity should be restricted to a small, clean enclosure or box stall. Controled movement should be encouraged to aid in the disbursement of edema. An elastic body support bandage may be applied to reduced tension on the primary closure and to keep the incision clean. Some patients refuse to stand after a full abdominal bandage is placed, and relationships of body surfaces are different in recumbent verses standing animals, therefore, a permanent support bandage should be applied only after the patient is standing. The abdomen of a camelid narrows toward the rear, making it difficult to keep a support bandage in place. Adhesive tape works better than Vetrap as the latter tends to gather, making its effectiveness unpredictable.

C1 Gastrotomy

A gastrotomy is indicated to remove foreign objects (hair balls, concretions, pieces of plastic, etc.) from C1 or to relieve a concentrate overload. This surgery can be performed in the left paralumbar fossa with the animal standing or under anesthesia with the animal in right lateral recumbency. A ventral midline approach with the animal under general anesthesia is also possible. The procedure is performed in a similar manner to that of a rumenotomy in a cow.

The incision site can be desensitized with local anesthetic, using an inverted L block or local infiltration. Even if general anesthesia is employed, local or regional anesthetic techniques can be used to augment general anesthesia so that the plane of general anesthesia can be kept lighter.

A 15-cm incision should be made in the left flank beginning 15 cm (6 in.) ventral to the transverse processes of the lumbar vertebrae, midway between the last rib and the tuber coxae, to avoid the pleural reflection, which may extend 2–3 cm caudal to the last rib. The spleen is usually visible deep to the incision in this area and caution should be taken not to inadvertently hit it. The incision should penetrate the abdominal tunic, and the fibers of the external oblique muscle should be separated as they traverse the incision in a ventrocaudal direction. If present, the fibers of the internal abdominal oblique muscle should also be separated in the direction of the fibers. The fibers of the transversus abdominis are vertical and should be separated in that direction. The peritoneum may be penetrated with a finger or Mayo scissors.

The dorsal, mobile part of C1 should be pulled through the hole and the seromuscular layer of C1 sutured skin prior to incising the wall of the compartment. Though not possible for emergent conditions like toxin ingestion or grain overload, fasting the animal for 24 hours prior to the procedure will cause a void in the dorsal sac of C1 and aid in pulling it through the body wall hole. Suturing C1 to the skin forms a seal between the compartment and the skin and avoids contamination of the peritoneal cavity while exploring and evacuating C1.

Ingesta should be removed as appropriate. In the case of ingestion of oleander, C1 should be washed out with water to remove all possible ingesta. Even small particles of leaves may be sufficient to produce toxicity.

The incision in C1 should be closed with a Cushing or Connell pattern with an absorbable suture. Prior to release of the C1/skin stay sutures, the exposed wall of C1 should be thoroughly lavaged with physiologic saline. Following release of the sutures, an attempt to keep C1 exteriorized for another lavage should be made. The author recommends a second layer of inverting sutures placed lateral to the suture holes created by the stay sutures. This second layer will provide added protection against leaking and will invert any damaged C1 caused by stay sutures. The abdomen should be closed as for a laparotomy.

Gastrostomy (Permanent C1 Fistulation)

This surgery is usually done as an experimental procedure for basic nutrition studies, but it may also be indicated for maintenance of a donor animal for transfaunation of other animals with digestive disorders. The basic incision is made as for gastrotomy in the left flank. A circle of skin should be removed from the skin. Usually, the cap of a commercial cannula to be placed following the procedure can be used as a template for the skin excision. The resultant circular opening should be the size of the cannula to be inserted later.

The dorsal aspect of the caudal sac of C1 should be pulled up into the skin incision and the serosa of C1 sutured to the skin with a single continuous suture. The seal at the ventrum must be secure. It may be appropriate to scarify the serosa of C1 prior to the suturing to enhance the adhesion of the stomach wall to the skin.

Permanent C1 fistulation can be performed as a one- or two-stage process. If a one-stage process is performed, C1 is now opened and the mucosal surface everted and sutured to the skin outside the original C1/skin sutures. If a two-stage fistulation procedure is performed, following the suturing of the serosal surface of C1 to the skin, the exposed serosa of C1 is covered with a moist dressing to preclude drying. One to two weeks is allowed for adhesion formation between C1 and the skin before C1 is incised for insertion of the cannula.

Surgery of the Spiral Colon

The spiral colon is a frequent site of pathology in camelids [55, 154]. The intestinal lumen quickly decreases in size at the spiral colon and is, therefore, an area prone to obstruction. Large fecal pellets, trichophytobeazoars, and fecoliths, can become entrapped within the centrifugal loops of the spiral colon [55]. Impaction of inspissated ingesta [101] and sand impaction [100] have been reported in crias. Torsion (volvulus) of the spiral colon [52] and obstruction of the spiral colon due to adhesions from a previous surgery [55] has also been reported.

The camelid spiral colon has a long mesenteric root compared to ruminants [154] and will usually be readily accessible from a ventral midline incision unless it is displaced by gaseous or fluid distention of other segments of the intestine or by torsion of the mesenteric root (Figure 12.72). The large centripetal loops of the spiral colon will frequently obscure the smaller centrifugal loops making lesions within the latter difficult to locate despite the surgical accessibility of the organ [55]. The long mesenteric attachments also make it prone to entrapment and mesenteric volvulus [52, 154].

The proximal colon and the distal small intestine may become distended with spiral colon lesions. It is usually necessary to decompress loops of bowel distended by gas or fluid prior to complete exam of the digestive tract or before enterotomy or resection. Gas and fluid may be withdrawn via a large-gauge needle or a rubber tube inserted through a stab incision and surrounded by a purse-string suture. A more rapid and thorough decompression can be accomplished using a technique frequently employed in equine colic surgery. In this technique, the distended bowel is laid on a sterile tray inclined to a collecting basket that, in turn, is connected to the surgery's drainage system. A longitudinal incision is made along the antimesenteric border of the bowel. Regular tap water may be used to wash ingesta down the tray and lavage the surface of the intestine. A common cause of ileus is failure to adequately decompress the intestinal segments before closure. This intestinal incision should be closed using an inverting pattern, preferably a Cushing pattern, before progressing further with surgery.

Spiral colon volvulus may be corrected by simple surgical correction following determination of the direction of the twist. In severe cases of compromised intestines, resection of the spiral colon with anastomosis of the proximal and distal segments of the ascending colon is possible [52].

Medical therapy for colonic impaction may consist of fluid therapy, intestinal mucostimulatory therapy, lubricant and osmotic laxatives, lidocaine CRI, enemas, antibiotics and anti-inflammatories. Cases of spiral colon impaction that do not resolve with medical therapy may require surgery. A study of 12 juvenile alpacas with spiral colon impaction showed that cases which were not able to be resolved by medical management had a poor prognosis [101].

It may be possible to gently knead a semisolid impaction and reduce it to particles small enough to be moved through the remainder of the intestine by normal peristalsis. However, if the wall is ischemic and friable, more harm may be done by using this technique than by performing an enterotomy. Before an enterotomy can be performed, the segment of colon containing the obstruction must be

isolated from other segments of the spiral colon with care to avoid the blood vessels.

If enterotomy is necessary, a small longitudinal incision should be made over the mass, which is then withdrawn with forceps and discarded. The incision should be closed with a single-layer inverting pattern. Infolding of the bowel wall should be strictly limited to minimize the potential for future stricture. All exteriorized loops of intestine should be lavaged with sterile physiologic saline and replaced in the normal position. The abdominal wall should be closed as for any laparotomy.

Rectal Prolapse

An effort should be made to identify the inciting cause of the rectal prolapse and this should be corrected if possible. Chronic enteritis resulting in persistent diarrhea is the primary cause of rectal prolapse. However, the rectum may also evert during tenesmus associated with prolapse of the vagina or uterus. A weak or relaxed anal sphincter may allow a few folds of the rectal mucosa to prolapse, especially while a late pregnant female is lying down.

This condition can be handled as it would be for a ruminant. Correction in the standing position is most desirable, but because camelids have a propensity to lie down, it may be necessary to operate with the animal recumbent. Epidural anesthesia (see Chapter 6) provides analgesia and relaxation sufficient to replace the prolapse. However, general anesthesia is an alternative if superior restraint is necessary.

The mucous membrane should be gently handled to avoid tearing of the friable tissues. The rectal tissue may be cupped in the palms of the hands and the prolapse gently replaced. If edema is severe, it may be desirable to wrap the prolapse tightly with a warm moistened towel or sheet until the swelling is reduced. Internal telescoping may be reduced by gentle insertion of a lubricated plastic or cardboard vaginal speculum. Care must be taken not to damage the rectal mucosa during prolapse reduction. Previous recommendations have been to apply sucrose (sugar) to the prolapse to draw fluid through the mucosa, which it does; however, investigations have demonstrated that the sugar granules can traumatize the mucosa. When the swelling is reduced, the prolapsed tissue should be gently replaced, avoiding point source pressure.

The invagination should be completely corrected, and a purse-string suture should be placed in the perineal tissue approximately 2–3 cm peripheral to the center of the anal orifice to maintain reduction. Chronic rectal prolapse or a prolapse with necrotic tissue may require a submucosal resection or rectal amputation, similar to the procedure for cattle. The purse-string should not be tied too tight or it will not permit the passage of feces. Approximately two fingers should be able to be inserted through the anus when the purse-string is tied. It should be remembered that a camelid excretes pelletized feces and may need a proportionally larger orifice than a cow to allow fecal passage. The animal should be observed for recurrence of the problem or the build-up of feces behind a purse-string suture.

Post-operatively, the patient should be observed for passage of feces and persistent tenesmus. If needed, the purse-string suture may be untied to allow for evacuation of feces. The purse-string should be retied to prevent subsequent prolapse. Purse-string suturing is a good temporary fix, but eventually will need to be removed because it will elicit infection in the perineal tissues. Stricture of the rectum or anus may occur from trauma to the mucosa, but stricture more frequently develops following amputation. Peritonitis, resulting from dehiscence of the suture line, is a possible complication.

Rectal Laceration

Though rare, laceration of the rectum is possible during rectal palpation in camelids [110, 114, 155]. Veterinarians with large hands or arms may have difficulty in conducting a thorough rectal examination because of the small size of the pelvis, rectum, and colon of SACs. Strong peristaltic waves may push against the hand. The palpator must give and take with these waves. A sudden movement of the animal, such as falling or rearing, when the rectum is under tension may result in a perforation.

A small mucosal laceration may heal without incident, or streaks of fresh blood may be seen on some of the fecal pellets. If the laceration perforates the colonic or rectal wall, peritonitis will ensue. Signs indicative of peritonitis appear within 24–36 hours. Without treatment, the animal will die in five to seven days. If a laceration is suspected, it is unwise to reenter the rectum to investigate. An epidural and an equine plastic or cardboard vaginal speculum may be used to visualize the rectum without imposing excessive further stretching of the mucosa. Full-thickness rectal laceration is an emergency. Parenteral administration of broad-spectrum antibiotics should be begun as soon as a laceration is suspected.

Evaluation and suturing via the anus necessitate epidural or general anesthesia. Requirements for trans-anal suturing is that the laceration is caudal enough to be reached and sufficient space within the rectum is present to manipulate the instruments. Tears occurring more cranial require suturing through a laparotomy which also provides the opportunity for peritoneal lavage. However, if peritoneal contamination is present, prognosis for long-term survival is poor [114]. It is important to be aware that the pelvic reflection of the peritoneum extends further caudally between the uterus and the rectum than it does ventral to the uterus and bladder,

increasing the likelihood that full-thickness rectal tears will enter the peritoneal cavity. In the llama, the peritoneal reflection is located approximately 4 cm cranial (orad) to the anus. A penetration caudal to that location will be retroperitoneal. However, it is more common to have lacerations that are cranial to the peritoneal reflection.

Fecal pellets should be gently removed from the rectum and the rectum packed cranially with cotton or gauze to avoid subsequent contamination of the field. The mucosa should be carefully but gently cleaned with physiologic saline and precautions taken to avoid enlarging the laceration. A dilute (1:10) povidone-iodine solution may be used to provide some degree of asepsis.

If it is determined that the laceration penetrates only the mucosa and is less than 1 cm in length, no treatment may be needed other than parenteral administration of broad-spectrum antibiotics for 7–10 days. Lacerations longer than 1 cm or lacerations that penetrate the muscle layers of the rectum should be sutured. It is difficult to obtain exposure via the anus. If the laceration is within 10–15 cm of the anus, a human plastic vaginal speculum, with a spoon length of 10 cm, may provide access to the surgical site.

A mucosal laceration may be closed with simple interrupted sutures using a swedged, taperpoint needle with absorbable suture. Picking up the underlying muscle layer adds strength to each suture.

If the laceration is full thickness into the abdominal cavity, a laparotomy should be performed to close the laceration and evaluate the peritoneal cavity for evidence of fecal contamination. This is an emergency procedure; therefore, it is not possible to fast the llama to partially empty the stomach. Exposure of the surgical site is the major problem. The caudal colon and cranial rectum may be approached from a ventral midline incision; however, a high-flank incision is usually recommended for optimum exposure. Advanced pregnancy further limits the surgeon's exposure.

Fecal contamination greatly affects prognosis. If there is extensive fecal contamination or peritonitis is already present, the prognosis is unfavorable. Placing an intestinal clamp (Carmalt) on the laceration may prevent further contamination during the procedure. Particulate matter should be removed manually, and the abdominal cavity flushed with physiologic saline in sufficient quantities to remove any residual contamination. A final flushing with povidone -iodine solution (diluted 1:10) is important.

The defect should be closed with an inverting Lembert or Cushing pattern using absorbable suture. The abdominal wall may be closed as for any laparotomy. Broad-spectrum antibiotics should be administered for 7–10 days post-operatively.

Post-operatively, the diet should be adjusted and a mild, osmotic laxative like magnesium hydroxide can be given to soften the stools. Enemas are contraindicated, because the fluid could seep through the incision and the pressure would be detrimental to healing. The patient should be monitored for signs of peritonitis. Continuing broad-spectrum antibiotics for at least one week post-operatively is recommended. A hemogram should be performed at least every other day for six days to monitor peritonitis. In addition to peritonitis, rectal scarring and stricture is also possible.

Atresia Ani and Atresia Coli

Atresia ani or atresia coli is a congenital/hereditary defect of the neonate in which the anal opening is lacking (atresia ani) or there is a stricture in the rectum or colon (atresia coli). Both forms have been reported in llamas and alpacas but are uncommon [156–158]. This may be a hereditary condition, so client communication is of paramount importance, and these animals should not be used for breeding.

With atresia ani, the absence of the anal opening is apparent, and there is usually a bulge at the anus. Atresia coli is more difficult to diagnosis and clinical signs of abdominal distention and straining to defecate may not be noted until a few days of age.

Contrast radiography with a barium enema may be required to determine the site of a lesion in the colon. It is possible for an older animal to acquire a rectal or anal stricture leading to identical clinical signs.

Sufficient anesthesia for the correction of atresia ani may be achieved by infiltration of local anesthetic into the operative site. General anesthesia is required for repair of atresia coli, which necessitates a laparotomy.

With atresia ani, a circular disc of skin approximately 2 cm in diameter should be removed from the center of the bulge. Feces may be allowed to escape by puncturing the rectal mucosa. The opening may be enlarged as seems appropriate. The mucosa should be sutured to the skin using fine absorbable suture, Figure 12.87.

Figure 12.87 Alpaca following repair of atresia ani.

Animals that have had previous atresia ani are prone to anal stricture. Some patients will lack an anal sphincter; these animals will be incontinent. Following surgical correction of atresia ani, the orifice should be stretched at least every other day for two weeks to prevent stricture of the surgical site.

Notes

1 Gastroguard®, Omeprazole Paste 37%, Merial, Boehringer Ingelheim Animal Health.

2 Protonix®, Pantoprazole sodium injectable, Pfizer.

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13

Endocrine System

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Endocrine Organs

Pituitary Gland

The anatomy and function of the camelid pituitary gland is not unique. Similar to the bovine, it is located at the base of the brain in the pituitary fossa of the basisphenoid bone. The growth hormone of the alpaca has been isolated and characterized and found to be chemically similar to other mammalian growth hormones [1]. This hormone has not been studied in a clinical setting to determine if it is involved in the failure to thrive syndrome.

Thyroid Gland

The paired thyroid glands are situated on the dorsolateral surface of the trachea. They are approximately 4 cm long and 2 cm wide and occupy the space from the cricoid cartilage of the larynx to the third or fourth tracheal ring. Blood is supplied by a cranial and caudal thyroid artery, directly from the carotid artery [2].

Reference ranges for serum triiodothyronine (T_3) and thyroxine (T_4) in llamas have been established using radioimmunoassay techniques and were found to be significantly higher than other domestic species (Table 13.1) [3–6]. Hypothyroidism has been put forth as a differential for failure to thrive and anemia in camelids [6] and thyroid supplementation has been anecdotally used to treat failure to thrive animals, especially if levels are suggestive of a deficit.

Parathyroid Glands

The parathyroid glands are embedded in the caudal pole of each thyroid gland, as they are in other ungulates. No direct parathyroid dysfunction has been reported in camelids; however, nutritional secondary hyperparathyroidism is seen with some frequency in old world camelids (OWCs) leading to "big head" as seen in horses (see Chapter 23).

Adrenal Glands

The right adrenal gland is elongated and lies medial and craniad to the cranial pole of the right kidney. The left adrenal gland lies in approximately the same position relative to the left kidney but has a square shape [2]. Adrenal gland production of cortisol is an important player in energy metabolism disorders of camelids, but no primary adrenal dysfunction has been reported.

Pancreas

In SACs, the pancreas is a flattened gland approximately 15–20 cm long and 3–6 cm wide that lies caudal to the liver, not well encapsulated in the duodenal mesentery. The pancreatic duct exits the cranial aspect of the gland and unites with the bile duct about 3 cm before it enters the intestine [2]. The pancreas is both an endocrine and exocrine gland. Pancreatic necrosis has been reported but is likely a comorbidity and supportive care is the only treatment.

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Table 13.1 Comparison of T_3 and T_4 values from various studies.

Age	McLaughlin et al. [3]				Smith et al. [4]				Fowler and Zinkl [5]			
	T_3 ng/dL[a] U.S. units	T_3 nmol/L SI[b] units	T_4 ng/dL[c] U.S. units	T_4 nmol/L SI units	T_3 ng/dL U.S. units	T_3 nmol/L SI units	T_4 ng/dL U.S. units	T_4 nmol/L SI units	T_3 ng/dL U.S. units	T_3 nmol/L SI units	T_4 ng/dL U.S. units	T_4 nmol/L SI units
0–2 d	513 (280–750)	7.7 (4.2–11.3)	61.0 (39.2–82.8)	785 (505–1066)	–	–	–	–	–	–	–	–
3–4 d	554 (400–700)	8.3 (6.0–10.5)	56.3 (50.2–62.4)	725 (646–803)	–	–	–	–	–	–	–	–
5–6 d	543 (470–620)	8.1 (7.1–9.3)	35.0 (19.1–51.0)	450 (246–656)	–	–	–	–	–	–	–	–
7–8 d	422 (380–460)	6.3 (5.7–6.9)	42.1 (19.3–64.9)	542 (248–835)	–	–	–	–	–	–	–	–
9–10 d	427 (290–560)	6.4 (4.4–8.4)	28.9 (16.7–41.1)	372 (215–529)	–	–	–	–	–	–	–	–
11–30 d	327 (80–560)	4.9 (1.2–8.4)	20.6 (15.6–25.6)	265 (201–329)	–	–	–	–	(274–686)	(4.1–10.3)	(18–39)	(232–502)
31–90 d	156 (90–220)	2.3 (1.4–3.3)	14.5 (9.6–19.2)	187 (124–247)	–	–	–	–	(42.0–326)	(0.63–4.9)	(10.0–25.9)	(129–333)
<1 yr	–	–	–	–	–	–	–	–	(63.0–321)	(0.94–4.8)	(10.0–20.0)	(129–257)
Adult Y	–	–	–	–	145 (45–414)	2.2 (0.67–6.2)	12.4 (7.0–22.0)	160 (90–283)	(108–277)	(1.6–4.2)	(7.8–18.7)	(100–241)
Adult X	–	–	–	–	136 (45–412)	2.0 (0.67–6.2)	8.8 (4.1–18.9)	113 (52.7–243)	(70.0–197)	(1.1–2.9)	(12.1–19.8)	(156–255)
Pregnant X	–	–	–	–	145 (48–440)	2.2 (0.72–6.6)	10.0 (3.3–30.6)	129 (42–394)				

[a] Triiodothyronine.
[b] SI = Système International.
[c] Thyroxine.

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Gonads

Reproductive endocrine anatomy and function are discussed in Chapter 16.

Endocrine Disorders

Physiology of Energy Metabolism in South American Camelids

South American camelids (SACs) differ significantly from traditional domestic ruminants in their handling of energy substrates. Llamas and alpacas clear glucose more slowly than other domestic species after a glucose challenge [7]. In most domestic herbivores, post-challenge serum glucose concentrations are indistinguishable from baseline values within 60–90 minutes. Llamas and alpacas had higher blood glucose values at baseline through 180 minutes post challenge. In most species, after a glucose challenge, the insulin increases fourfold and the insulin/glucose ratio increases and may not return to baseline values until normal blood glucose concentrations are achieved. In a camelid study [7], only a 50% increase in insulin concentration was noted, and the insulin/glucose ratio decreased after the challenge. In another study [8], exogenous insulin administration did not increase glucose tolerance. In fact, insulin-assisted glucose clearance in camelids was shown to be slower (mean 69 minutes) than unassisted clearance in clinically normal dogs (25 minutes) or cattle (35 minutes). However, when evaluating the effects of stress on glucose, insulin did counteract the hyperglycemic stimulus of hydrocortisone [9]. The conclusion of these studies is that camelids clear glucose slowly because of poor insulin response and because of a limited cellular capacity to increase glucose uptake in the presence of insulin (insulin resistance), but insulin may still be beneficial in treating glucose derangements.

SACs also store and mobilize energy (adipose stores) in a manner unique to most domestic animals. The reason for this unique handling of stored energy is likely because of how they subsist in their native environments. In South America, these animals are never fed concentrates or supplements and are rarely fed cured hay. They feed on native grasses and forbs. Llamas/alpacas are concentrated in the Altiplano, which is an area of broad, high-altitude valleys and plateaus at elevations over 12 500 ft. (3800 m). The climate of this area consists of a long dry season and a short, wet season, with 75% of the rainfall occurring from December through March. The dry season is from May through October. The growing season (wet) is characterized by low temperatures (300+ nights of frost per year) and intense solar irradiation. Daytime temperatures reach a maximum of 18.3 °C (65 °F) with nighttime temperatures of −12 °C (10 °F). Available forage varies greatly from the wet to dry seasons. The animals cope with the "feast and famine" routine by depositing layers of fat in the subcutaneous, muscular, and retroperitoneal tissue during the wet season and mobilizing these reserves in lean times. This mechanism allows for good foraging followed by seasonally deplorable feeding conditions [2].

These unique metabolic features of SACs are often forgotten by North American managers. Owners often assume that camelids consume only enough to satisfy their requirements. This is not true, and because of this, these animals are at risk of metabolic dysregulation [2].

Ketosis, Hyperlipidemia, and Hepatic Lipidosis

A syndrome of ketosis, hyperlipidemia, and hepatic lipidosis is a serious but under-diagnosed disease process in SACs. Camelids are at risk for this syndrome because of their natural handling of energy substrates as discussed previously.

There are several reports of this metabolic syndrome. The most comprehensive retrospective study (31 cases) [10] reports varied signalments and histories. Many of the animals were late pregnant, lactating, had concurrent disease or significant stressful event, or a combination of these things. Hyperlipidemia, ketosis and metabolic acidosis were common findings. Hyperlipidemia was likely the result of increased hepatic export of very low-density lipoproteins (VLDL) or decreased lipoprotein lipase activity. Hyperglycemia was common which indicates insulin resistance leading to cellular negative energy balance. The liver values (GGT, AST, SDH, and bile acids) were also increased.

Stress is thought to play a major role in the induction of this syndrome. The effects of hydrocortisone on substrates of energy metabolism have been tested [9]. Hyperglycemia developed 90–120 minutes after injection of hydrocortisone, which indicates that during the stress response, glucose utilization decreases quickly. Administration of insulin counteracted the hyperglycemic stimulus of hydrocortisone. This finding suggests that insulin resistance is not the only factor that leads to this cellular negative energy balance, and that administration of insulin may be helpful in impaired tissue utilization of glucose in camelids with hypercortisolemia.

A consequence of the ongoing carbohydrate and lipid metabolism dysregulation is hepatic lipidosis (Figure 13.1). Hepatic lipidosis is not a specific disease but rather the end result of one or more of the metabolic processes:

1) Increased mobilization of depot fat.
2) A decreased rate of oxidation of mobilized fat.
3) A reduced rate of removal from the liver.

Figure 13.1 Camelid liver with severe hepatic lipidosis. The yellow discolored areas of the liver represent areas of fat deposition.

Hepatic lipidosis occurs in cattle, sheep, cats, dogs, and, to a lesser extent, horses. The mechanisms of development vary from species to species. The generic causes of hepatic lipidosis include starvation (whole body negative energy balance) or a cellular level negative energy balance due to glucose dysregulation.

In one study [11], hepatic lipidosis was induced in llamas by feed restriction. Five of the eight animals developed hepatic lipidosis. Concentrations of AST, SDH, and GGT were significantly higher in animals that developed hepatic lipidosis than those that didn't. All animals lost weight, but the percentage of body weight loss did not significantly differ between hepatic lipidosis and non-hepatic lipidosis animals. Non-esterified fatty acid (NEFA) concentration increased in all animals by day two, but there was no correlation between concentration of NEFA and development of hepatic lipidosis. Beta-hydroxybutyrate (BHBA) also increased in all llamas by day two, but llamas with hepatic lipidosis had significantly higher BHBA than unaffected llamas. Insulin concentration was not significantly different between groups. Initial body condition did not seem to affect which animals developed hepatic lipidosis.

Clinical signs of this metabolic syndrome are fairly nonspecific and include anorexia, depression, dehydration, weight loss and unwillingness or inability to rise. Clinicopathologic findings include hyperglycemia, hyperketonemia, and sometimes gross lipemia as noted by a milky white appearance of the serum.

Animals exhibiting this metabolic syndrome often need significant supportive care. Fluid therapy should focus on cardiovascular support without over-dilution of protein and treatment of acid base and electrolyte derangements. The ultimate treatment for this syndrome is return to eating; however, initially this is not often possible. Initial therapy is aimed at improving the cellular uptake of glucose thereby reducing the cellular negative energy balance and decreasing fat mobilization and ketogenesis. Insulin administration has been shown to improve tissue utilization of glucose [9], to aid in the clearance of triglycerides and inhibit the release of NEFAs and BHBA into the bloodstream [12]. Long-acting insulin (0.4 U/kg SC once or twice a day) will improve cellular uptake of glucose [13]. Blood glucose must be monitored as significant hypoglycemia can result. Another, more aggressive yet easier approach is an insulin CRI (regular insulin starting at 0.02 U/kg/hr) with regular monitoring and adjustments to maintain an improved blood glucose [14]. The prognosis, especially with hepatic lipidosis, is poor.

Diabetes Mellitus

One report [15] presented an alpaca with the primary finding being chronic hyperglycemia. In this case, the clinical signs were consistent with diabetes (chronic hyperglycemia, polyuria/polydipsia, lens opacities, and foot ulcers). This animal was diagnosed with type I diabetes mellitus based on low serum insulin and response to insulin therapy. This is not common.

Hyperosmolar Syndrome

Hyperglycemic, hypernatremic, hyperosmolar syndrome is a disease process seen primarily in neonatal SACs. Camelids are at risk for this process because of their previously mentioned unique energy metabolism often combined with one or more predisposing factors (sepsis, trauma, premature birth, death of the dam, etc.). These stressful events lead to a significant cortisol response [16]. As discussed earlier, this cortisol response often leads to severe hyperglycemia and glucosuria. Glucose diuresis, in conjunction with decreased fluid intake, causes significant dehydration and hypernatremia.

Clinical signs of this disease process include dehydration, lethargy, hyperthermia, weakness, wide-base stance, poor-to-absent suckle response, altered mentation, ataxia, seizures, and coma [14, 16]. Diagnosis is made by measuring serum glucose (often above 600 mg/dL but has been reported as high as 1360 mg/dL) and serum sodium (>160 meq/L) [16]. Additionally, CSF sodium can be measured.

The goals of therapy include treatment of any underlying disease, controlling the hyperglycemia and decreasing the serum sodium to the normal range. Hypernatremia must be corrected at a rate that does not lead to cerebral edema (0.5–1 meq/L/hr). The hyperglycemia can be improved by hydration, but often insulin therapy is necessary. Regular insulin can be injected periodically or an

insulin CRI can be used, adjusting as needed to gain control of blood glucose [14]. Blood glucose should be monitored frequently during insulin therapy. Fluid therapy to relieve hypernatremia, while being careful to not reduce the serum sodium concentration too quickly, can be challenging. Traditionally, giving a fluid that has a sodium content that is the same or slightly lower than the serum sodium of the patient is recomended [16].

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14

Hematology, Clinical Biochemistry, and Fluid Analysis

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Laboratory testing is often an integral component of patient evaluation to assess health, establish a diagnosis, and determine severity of disease or risk prior to surgery. The complete blood count (CBC) and serum biochemical profile are commonly performed laboratory tests because they provide information about multiple body systems in assessing inflammation, neoplasia, and organ dysfunction, and can be used to monitor effectiveness of therapy and disease progression. Though there are selected references related to the hematology and clinical biochemistry of New World Camelids (NWC; llamas, alpacas, guanacos, and vicunas) and Old World Camelids (OWC; Bactrian and Dromedary camels), most of the information in this chapter will be about domestic llamas and alpacas, unless otherwise indicated. Other species-specific reference intervals (RI) should be used for hematologic and serum biochemical testing when available. It is important to remember that RIs also vary with instrumentation, methodology, and environmental conditions.

CBC

Blood for a CBC is collected in ethylenediaminetetraacetic acid (EDTA), which prevents clotting by binding calcium. EDTA preserves cell morphology unless samples are stored for prolonged periods. Commercially available lavender top blood collection tubes containing EDTA are available in several different sizes. The 2-mL blood collection tubes are used most often, and this volume is sufficient for a CBC. Collection tubes should be filled with the correct volume of blood because overfilling or underfilling can cause erroneous results. It is important to gently invert the tube several times after collection to make sure the blood thoroughly mixes with the anticoagulant. Blood samples should be inspected grossly for small clots before a sample is processed for a CBC, and if clots are present, a new sample should be collected. The tube should be inverted 10–15 times before the blood is processed through an automated hematology analyzer, a microhematocrit tube is filled, or blood smears are made, to ensure that red blood cells (RBC), white blood cells (WBC), and platelets are evenly distributed throughout the volume of blood.

The CBC typically includes a PCV (packed cell volume, or hematocrit [HCT]), RBC count, hemoglobin concentration (HGB), mean cell volume (MCV), mean corpuscular hemoglobin concentration (MCHC), platelet count, WBC count, and WBC differential (Table 14.1). Newer instruments with laser technology may include RBC hemoglobin content (CH), hemoglobin content of reticulocytes (CHr), corpuscular hemoglobin concentration mean (CHCM), hemoglobin concentration distribution width (HDW), mean cell volume of reticulocytes (MCVr), platelet volume distribution width (PDW), and plateletcrit (PCT). As benchtop hematology instruments designed for the practice setting become more sophisticated, it is important to understand their limitations in regard to some of the unique aspects of camelid blood [1].

Evaluating a blood smear is an important part of a CBC, especially for sick animals, because some hematologic abnormalities are recognized only on the blood smear. Examination of the blood smear is also an important quality control measure for confirmation of quantitative information obtained from automated hematology analyzers.

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Table 14.1 CBC reference intervals for adult llamas, alpacas, and camels.

Measurand	Units	Llamas[a]	Alpacas[b]	Dromedary camels[c]	Bactrian camels[c]
Plasma protein	g/dL	5.1–7.1[d]	6.5–7.5	NA	NA
PCV	(%)	27–45	22–45	20–33[e]	36–43
RBC	$\times 10^{12}$/L	10.0–17.0	9.4–18.1	7.9–12.6[e]	10.2–13.2
HGB	g/dL	11–19	8.8–15.4	11.4–15.0	11.7–13.7
MCV	fl	21–31	21–28	22–30[e]	25.3–31.6[f]
MCHC	g/dL	39–48	42–49	42.1–49.6[f]	37.0–47.0[f]
WBC	$\times 10^9$/L	7.5–22	7.1–18.6	6.0–13.5	10.0–15.8
Band neutrophils	$\times 10^9$/L	0–0.4	0	0	0
Segmented neutrophils	$\times 10^9$/L	4.6–16.0	3.5–12.1	3.0–8.1	2.0–13.1
Lymphocytes	$\times 10^9$/L	1.0–10.0	1.5–4.7	1.8–6.1	1.9–8.8
Monocytes	$\times 10^9$/L	0–1.0	0–0.9	0.1–1.1	0–1.1
Eosinophils	$\times 10^9$/L	0–5.0	0.4–4.0	0–0.8	0–2.8
Basophils	$\times 10^9$/L	0–0.1	0–0.1	0–0.3	0–0.5
Platelets	$\times 10^9$/L	150–800	220–947	129–606[e]	220–526[f]
Reticulocytes	$\times 10^9$/L	12–79	NA	0–77[f]	0–68[f]
Fibrinogen (heat precipitation)	mg/dL	121–482[g]	211–486[g]	200–400[f]	210–270[f]
PT	Seconds	7.0–10.0[g]	7.0–8.5[g]	10.48–11.72[e]	NA
APTT	Seconds	10.0–18.0[g]	9.0–17.0[g]	18.69–19.51[e]	NA

Abbreviations: APTT, activated partial thromboplastic time; CBC, complete blood count; HGB, hemoglobin; MCV, mean corpuscular volume; MCHC, mean corpuscular hemoglobin concentration; NA, not available; PCV, packed cell volume; PT, prothrombin time; WBC, white blood cell count.
[a] Unless otherwise indicated, all data for llamas are from Vap and Bohn [1].
[b] Unless otherwise indicated, all data for alpacas are from Dawson et al [44, 45].
[c] Unless otherwise indicated, all data are from Fowler and Zinkl [2].
[d] Van Houten et al. [3].
[e] Hussein et al. [4].
[f] Elitok and Cirak [5].
[g] The Ohio State University Veterinary Medical Center Clinical Pathology Laboratories reference intervals.

The numbers of RBC, WBC, and platelets can be estimated from a blood smear, and the morphology of all cell types can be evaluated. Infectious agents and circulating neoplastic cells sometimes can be detected.

Erythrocytes

A unique feature of llamas, alpacas, guanacos, vicunas, and Bactrian and dromedary camels is their elliptical RBC that lacks a central zone of pallor (Figure 14.1) [1, 2, 6, 7]. Camelid RBCs are smaller, flatter, and have a higher surface area to volume ratio compared to RBC from other species [1, 8–10]. They are more resistant to mechanical shearing and shape changes related to decreased adenosine triphosphate (ATP), and have a higher resistance to osmotic lysis, higher oxygen affinity, and higher membrane protein to lipid ratio [8, 11]. A different distribution of membrane

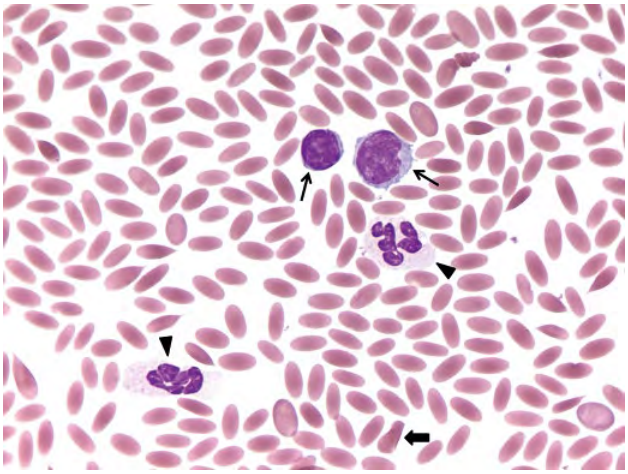

Figure 14.1 Blood from a healthy alpaca. There are numerous elliptical RBCs, one folded RBC (block arrow), two neutrophils (arrowheads), one small lymphocyte, and one intermediate lymphocyte (arrows). Wright-Giemsa stain. 1000X.

proteins also has been described, e.g. a higher proportion of band 3 protein and increased linkage of band 3 protein with other membrane proteins may contribute to decreased deformability and resistance to osmotic lysis [11–14]. These differences in RBC morphology may allow for more efficient surface gas exchange in high altitudes and increased resistance to osmotic lysis in desert environments where there is limited access to water.

These same morphologic variations are challenging when assessing RBC using conventional automated hematology analyzers, emphasizing the importance of using instruments that have been appropriately validated for the species of interest. For example, some analyzers that use optical flow cytometry (e.g. ADVIA 2120, Siemens Healthcare Diagnostics, Tarrytown, NY) depend on isovolumetric sphering to count RBC and measure MCV, from which HCT is calculated. HGB is measured by lysing RBC and using a modified cyanmethemoglobin method. A recent study compared CBC results from the ADVIA 2120 with impedance and manual reference methods using different species settings on the analyzers. Results showed that RBC from alpacas were resistant to isovolumetric sphering but were susceptible to lysis with the reagent used to determine HGB [9]. This study concluded that when using the ADVIA 2120 for CBCs for alpacas (and presumably other camelids), RBC counts and HGB concentration should be determined using the equine setting, and PCV should be determined manually instead of using the HCT generated by the instrument. MCV should be calculated using the RBC count from the instrument and the manual PCV, and MCHC should be calculated using the HGB from the instrument and the manual PCV utilizing standard formulae: MCV = (PCV/RBC) × 10 and MCHC = (HGB/PCV) × 100 [9].

The calculated MCV is lower, the calculated MCHC is higher, and RBC are more numerous for camelids compared to most other domestic animals (Table 14.1) [1, 15]. Juveniles may have a lower PCV and HGB than adults, and male llamas can have a higher PCV than females [16–18]. Perhaps because of their flat, elliptical shape, folded RBC can occur in health and in increased numbers in some anemic animals and are considered an artifact (Figure 14.1). Rhomboid or hexagonal hemoglobin crystals sometimes form, but these are nonpathogenic (Figure 14.2) [3, 14]. Cabot rings, which are narrow, red to violet thread-like structures in a ring or figure-eight shape, likely from remnant mitotic spindles, occur rarely and are of no clinical significance [14]. Reticulocytes, which are less mature RBC, are larger and more round than mature RBC, and stain more polychromatophilic with routine Romanowsky-type stains like Wright-Giemsa (Figure 14.3). Small numbers of reticulocytes can be present in health; higher numbers can occur in healthy animals living in high altitude and in response to some types of anemia [1, 14, 19]. In alpacas made anemic by phlebotomy, reticulocytes increased to a maximum of 1.5% with a peak response at 10 days [20]. Manual reticulocyte counts can be performed using supravital stains like new methylene blue (NMB). Low numbers (2–3 per 100 WBC) of nucleated RBC (nRBC)

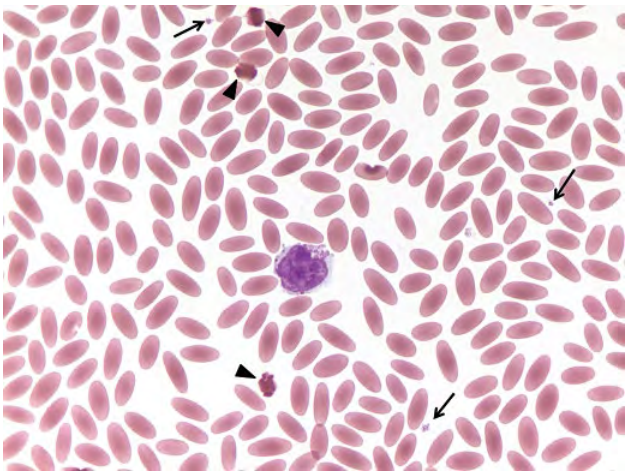

Figure 14.2 Blood from a llama. There is a large granular lymphocyte in the center. The granules in the cytoplasm can be eosinophilic, as shown here, or more basophilic. There are three RBCs with hemoglobin crystals (arrowheads). Platelets are small, discoid to irregular in shape with central granules (arrows). Wright-Giemsa. 1000X.

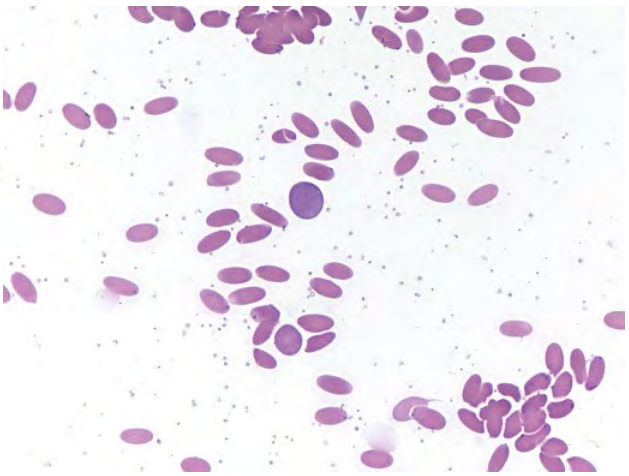

Figure 14.3 Blood from an alpaca. Numerous *Mycoplasma haemolamae* organisms are visualized on the surface of most of the RBC and free in the background between RBC. Organisms often dissociate from the RBC *in vitro*. *M. haemolamae* organisms are coccoid, rod-shaped, or ring-shaped inclusions <0.1 μ in diameter on the surface of RBC. There is a polychromatophilic cell in the center, which could be consistent with a regenerative response. However, most llamas and alpacas with *M. haemolamae* infection have nonregenerative anemia. Wright-Giemsa. 1000X.

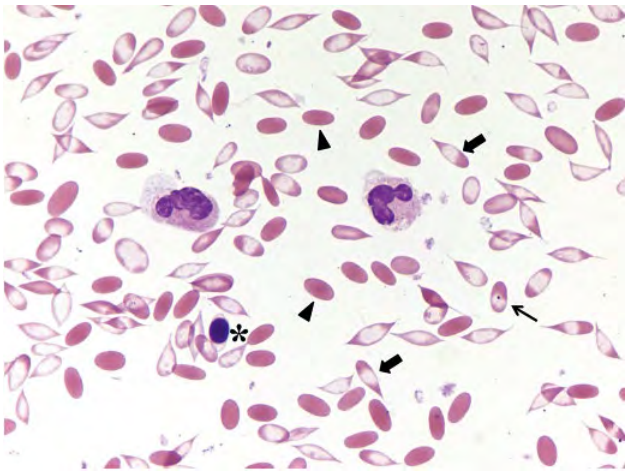

Figure 14.4 Blood from an alpaca that was markedly anemic and was treated with a blood transfusion. There are two populations of RBCs. The more normal appearing RBCs (arrowheads) are from a donor alpaca. The other RBCs are from the patient. Notice the marked central zone of pallor, moderate numbers of dacryocytes (block arrows), and occasional RBCs in which the hemoglobin is distributed at the poles. There is one Howell-Jolly body, which is a nuclear remnant (arrow) and a nucleated RBC (asterisk). These changes are all consistent with iron deficiency, which in this alpaca was from *Haemonchus contortus*. Wright-Giemsa. 1000X.

can also be present in health; increased numbers of nRBC can occur with anemia, depending on the cause (Figure 14.4) [1, 2, 6, 14].

Anemia can occur from hemorrhage, hemolysis, or decreased bone marrow production. Clinical findings depend on the cause of the anemia, but typically include dyspnea, depression, and pale mucous membranes. Hemorrhage can be from multiple causes, including lacerations, bleeding in the gastrointestinal (GI) or urinary tract, and coagulopathies. Acute severe blood loss can cause dyspnea and tachycardia associated with hypovolemic shock, whereas chronic blood loss can lead to iron deficiency. Anemia has been reported with some ecto- and endo- parasites, (see Chapter 7) but there is limited information about whether the anemia is regenerative or nonregenerative, or if iron deficiency occurs [21, 22]. Chronic hematuria and anemia associated with neoplasia involving the bladder has been reported with bracken fern toxicity in a llama [23].

Though uncommon, red maple leaf toxicity can cause marked intravascular hemolysis, which is characterized by reddish discoloration of the plasma and an increased MCHC. Oxidative damage by the toxins in the leaves causes Heinz body formation and methemoglobinemia. Methemoglobinemia can cause a brownish discoloration of blood and mucous membranes [24]. Methemoglobinemia can be determined using co-oximetry, in which a spectrophotometer is used to evaluate arterial blood at multiple wavelengths. On blood smears, Heinz bodies appear as teal blue RBC inclusions when blood is stained with NMB [25]. Other differentials for hemolytic anemia that have been documented in camelids include ingestion of other plants (e.g. onions, rape, and kale) [25]. Nitrate toxicity is rare, and causes methemoglobinemia, but does not cause Heinz body formation or hemolysis [25]. Hemolysis does not appear to be a part of copper toxicity in llamas [26] (see Chapter 22).

Hemorrhage and hemolysis typically cause regenerative anemia, characterized by increased reticulocytes, increased anisocytosis and, in some cases, increased nRBC, but the classification of anemia as regenerative or nonregenerative is not always clearly indicated in case reports of various causes in which anemia has been reported. Nonregenerative anemia occurs most often from chronic inflammatory disease, though *Mycoplasma haemolamae* (*M. haemolamae*) is another important cause of nonregenerative anemia (see below). Nonregenerative anemia has also been reported in llamas with hypothyroidism [27].

Abnormal RBC morphology is sometimes associated with anemia, but is variable, depending on cause and duration. Iron deficiency from chronic external hemorrhage can cause microcytic, hypochromic RBCs, which are characterized by decreased MCV and MCHC, and low RBC with an increased central zone of pallor when visualized on a blood smear. There may also be increased numbers of folded RBC, dacryocytes (tear-dropped shaped RBC), spindloid cells, and irregular hemoglobin distribution, which can be recognized on a blood smear (Figure 14.4). However, some of these morphologic abnormalities also occur with anemia due to other causes [1, 14, 27–29]. For example, hypochromasia can occur with copper deficiency or chronic inflammation [1, 17].

Hemotropic mycoplasmas occur in llamas, alpacas, and camels [30]. The organism, *M. haemolamae*, forms coccoid, rod-shaped, or ring-shaped inclusions <0.1 μ in diameter on the surface of RBC that can be recognized on blood smears stained with Wright-Giemsa (Figure 14.3) [1, 31–33]. Organisms loosely adhere to the RBC and can be found free in the background surrounding RBC on a blood smear, especially if smears are not made shortly after blood collection. Except for ring forms, organisms resemble stain precipitate. Parasitemia is cyclical, and the number of organisms is quite variable. Polymerase chain reaction (PCR) testing can be used to confirm infection and may be positive before organisms are apparent on a blood smear [34]. The mode of transmission is unknown. Transmission *in utero* or during parturition likely is rare [35]. Organisms can be seen in healthy animals. Mild to marked anemia occurs more commonly in stressed, immunocompromised, and debilitated animals. Animals can be PCR positive without clinical signs, anemia, or

presence of organisms on blood smears [34, 36]. Anemia is often nonregenerative [1]. Appropriate antibiotic therapy can be effective in reducing clinical signs and laboratory abnormalities, but does not consistently clear infection based on PCR [34].

Trypanosoma evansi (*T. evansi*), a protozoan parasite transmitted by hematophagous insects, is the causative agent of Surra disease in camelids in certain regions of Asia, Africa, and South and Central America, where prevalence can be up to 26% [30]. The disease is zoonotic and of public health concern in some countries. Chronic disease is more common than acute disease, which is usually fatal. Clinical findings include intermittent fever, anorexia, weight loss, abortion, edema, and anemia. The anemia is presumably regenerative, based on macrocytic, hypochromic indices, and may be due to oxidative damage to RBCs associated with the parasitemia [37]. The circulating trypomastigote stage of the parasite is slender, 20 × 2.2 μ, with a long free flagellum, a thin posterior extremity, and a small subterminal kinetoplast. Diagnosis by visualization of the organisms on blood smears is not as sensitive or specific as serology or PCR [38].

Increased PCV (HCT), HGB, and RBC count, or erythrocytosis (sometimes also called polycythemia) can be relative or absolute. Relative erythrocytosis due to dehydration is relatively common and is often associated with an increased plasma protein concentration. Relative erythrocytosis should resolve with appropriate fluid therapy. Absolute erythrocytosis due to epinephrine release and splenic contraction can increase PCV up to 10% in llamas, but this quickly decreases to normal [18]. There is minimal change in PCV in racing camels [39]. Absolute erythrocytosis can also occur with cardiovascular or pulmonary disease and some types of renal disease, or in response to hypoxemia from living at high altitudes [24]. Absolute erythrocytosis has been reported with a granulosa theca cell tumor [40], but sometimes a cause is not determined [41]. Clinical signs and laboratory changes, including erythropoietin levels, are variable, depending on cause.

Severely anemic animals are sometimes given blood transfusions as supportive care, depending on the primary disease. There are seven blood types in camelids. No naturally occurring antibodies have been identified, which means that any camelid blood from the same species can be safely transfused for a first-time transfusion, after which a crossmatch should be performed [42, 43].

Leukocytes

Total WBC are determined by most automated hematology analyzers. The RI for WBC is higher in camelids than in other domestic animals and is slightly higher for llamas than alpacas and camels [1, 14, 44]. Some automated hematology analyzers also perform a leukocyte differential that includes neutrophils, lymphocytes, monocytes, eosinophils, and basophils. Though median absolute neutrophil and monocyte counts were lower and median absolute lymphocyte counts were higher using an automated analyzer than those determined by a manual differential count, the differences were small and not clinically relevant [45]. Monocytes may be classified as large unstained cells. Many automated hematology analyzers do not accurately classify eosinophils and basophils, and most cannot accurately classify band neutrophils or immature hematopoietic cells. Also, these instruments cannot recognize most morphologic abnormalities in leukocytes, emphasizing the importance of blood smear evaluation, especially in sick animals.

In contrast to RBC, there are few unique morphologic features of camelid leukocytes. Neutrophils in camelids resemble those of other species and are the majority of the circulating leukocytes in health. The neutrophil to lymphocyte ratio is 1.5, which is similar to most domestic animals except some ruminants [2, 3, 14, 46]. Mature neutrophils have segmented nuclei with condensed chromatin and pale cytoplasm with fine, faintly staining granules (Figures 14.1 and 14.4). Mature neutrophilia can be associated with inflammation or can occur in response to increased endogenous or exogenous corticosteroids or catecholamine release [2, 7]. Mature neutrophilia from increased endogenous corticosteroids is often a component of a stress leukogram. Band neutrophils may be slightly larger than mature neutrophils and are characterized by minimally segmented C- or S (band)- shaped nuclei with parallel sides and cytoplasm that may appear slightly more basophilic (Figure 14.5). Band neutrophils increase with inflammation. In severe inflammation, less mature stages of neutrophils such as metamyelocytes can be released from the bone marrow. Metamyelocytes are slightly larger than bands, have indented nuclei, and have more abundant cytoplasm that is more basophilic. A degenerative left shift occurs when the number of immature stages of neutrophils is higher than the number of mature neutrophils and is a poor prognostic indicator. Toxic changes in mature and immature neutrophils include basophilic, vacuolated cytoplasm and Döhle bodies, which are aggregates of ribosomes that appear as round to irregularly shaped aggregates in the cytoplasm (Figure 14.5). Hyposegmented or ring-shaped nuclei are less common toxic changes in neutrophils. Toxic changes are due to markedly increased granulopoiesis, often associated with severe inflammation. In some animals, severe inflammation overwhelms the granulocytic capacity of the bone marrow, resulting in leukopenia [1]. The neutrophil to lymphocyte ratio is often reported to be 1.5, but lower values can occur in young animals, and

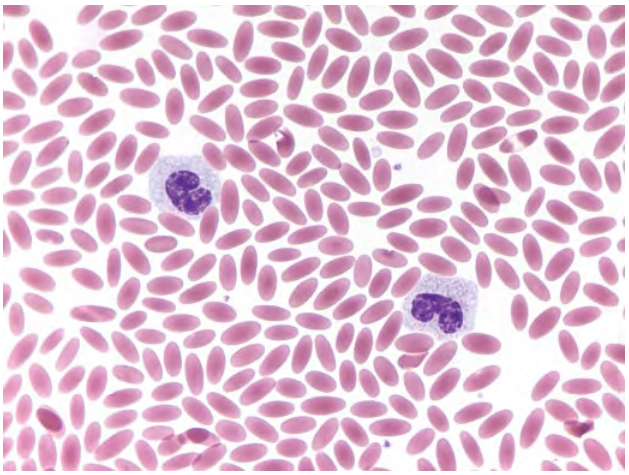

Figure 14.5 Blood from a llama with marked neutrophilic inflammation. These two band neutrophils have cytoplasm that is more vacuolated and more basophilic than is typical for mature segmented neutrophils. These are toxic changes consistent with increased granulopoiesis in the bone marrow and are associated with a poor prognosis. Wright-Giemsa. 1000X.

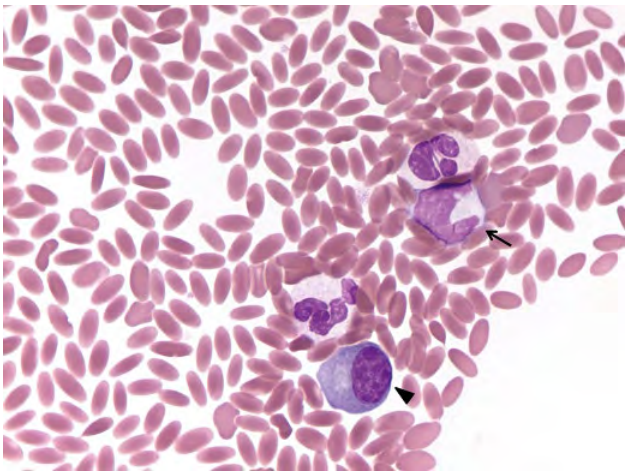

Figure 14.6 Blood from an alpaca. There are two neutrophils, one monocyte (arrow), and one reactive lymphocyte (arrowhead). The reactive lymphocyte is larger than a mature lymphocyte and has abundant basophilic cytoplasm. Reactive lymphocytes indicate antigenic stimulation. Wright-Giemsa. 1000X.

higher values have been seen in health in some reports [14, 47]. Increased fibrinogen, increased neutrophil to lymphocyte ratio, increased haptoglobin, and decreased serum iron can also be useful indicators of acute and chronic infections or other inflammatory diseases in camelids [17, 47–49].

Lymphocytes are the next most numerous circulating leukocytes and include small, intermediate, and large lymphocytes in health. Small lymphocytes are smaller than neutrophils, have round nuclei with condensed chromatin and inconspicuous nucleoli, and a high nuclear to cytoplasmic ratio (N:C) (Figure 14.1). Intermediate lymphocytes are similar in size to neutrophils (Figure 14.1). Nuclei can be round or slightly irregular, with smooth chromatin and inconspicuous nucleoli. Large lymphocytes are larger than neutrophils. Nuclei are round to irregular with finely stippled chromatin; nucleoli may be present. Increased numbers of large lymphocytes with intensely basophilic cytoplasm are sometimes called reactive lymphocytes and can occur with antigenic stimulation (Figure 14.6). Granulated lymphocytes may be up to 30% of circulating lymphocytes, which is a higher proportion compared with other domestic animals [3, 6, 46]. Granulated lymphocytes are often intermediate to large and have small numbers (2–20) of red to dark purple pleomorphic granules, often in one focal area of the cytoplasm (Figure 14.2). Younger animals may have a higher proportion of lymphocytes [2, 17, 18, 50], whereas sick animals may have lymphopenia, as part of a stress leukogram associated with increased endogenous glucocorticoids.

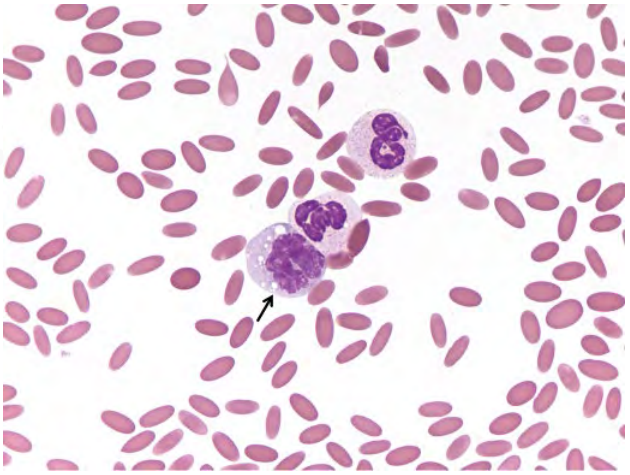

Figure 14.7 Blood from an alpaca. There are two neutrophils and a monocyte (arrow). Monocytes are larger than neutrophils and have indented or irregularly shaped nuclei. Abundant cytoplasm often is more basophilic and often contains vacuoles. Wright-Giemsa. 1000X.

Monocytes resemble those of other species. They are the largest circulating leukocyte, comprising 5–16% of circulating leukocytes in adult llamas, and 3–20% of the leukocytes in juveniles [51]. Nuclei are round, oval, indented, or irregularly shaped and have wavy or irregularly condensed chromatin. There is abundant cytoplasm that often appears grayish blue, and sometimes contains clear vacuoles (Figures 14.6 and 14.7). It may be difficult to differentiate monocytes from bands or metamyelocytes with toxic changes. Monocytosis can occur with inflammation or hemolysis.

Eosinophils circulate in low numbers in health, but the RI is higher than in other domestic animals [2, 14, 38].

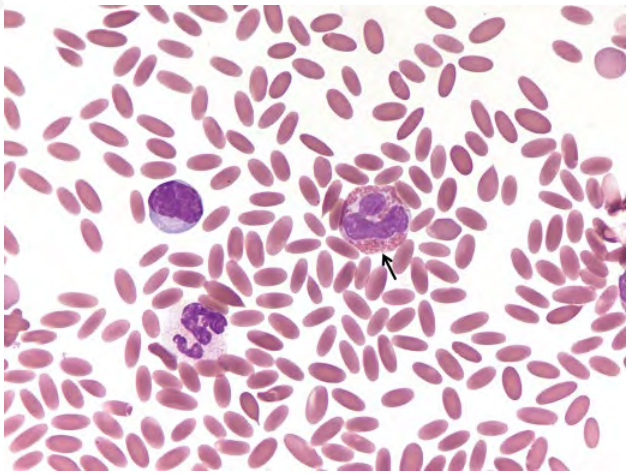

Figure 14.8 Blood from an alpaca. There is a neutrophil, a small lymphocyte, and an eosinophil (arrow). Eosinophils are characterized by a less segmented (shown here) or bilobed nucleus and cytoplasm that contains numerous eosinophilic granules. Wright-Giemsa. 1000X.

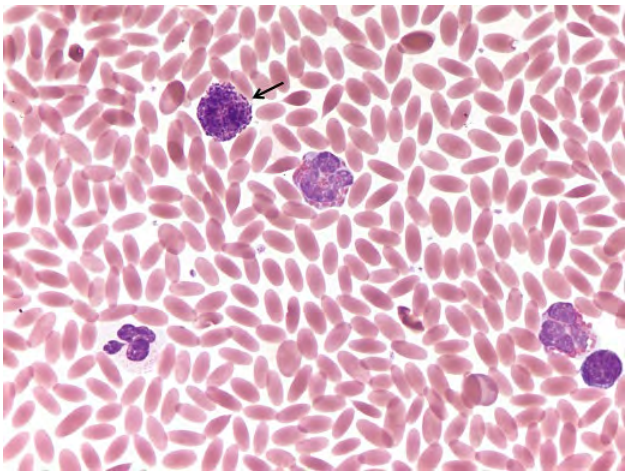

Figure 14.9 Blood from an alpaca. Though this is a thicker area than would be typically evaluated, it nicely demonstrates a neutrophil, small lymphocyte, two eosinophils, and a basophil (arrow) for comparison. Basophils are characterized by a twisted or segmented nucleus that often is obscured by numerous dark purple granules. Wright-Giemsa. 1000X.

Juvenile alpacas have fewer eosinophils than adults; female juvenile alpacas have fewer eosinophils than males [17, 50]. In most species, nuclei are segmented and have condensed chromatin, but in camelids, nuclei may be bilobed, band-shaped, hyposegmented, or occasionally ring-shaped [3, 6]. The prominent feature is the presence of numerous small reddish, round, oval, elongate or slightly-irregularly shaped granules in the cytoplasm (Figure 14.8) [6, 52, 53]. Eosinophils are important mediators of hypersensitivity reactions and contain substances that can damage and kill some parasites. Eosinophilia can occur with allergic reactions and in some parasitic infections, while eosinopenia (decreased eosinophils) occurs in response to corticosteroids.

Basophils also circulate in very low numbers, and often none are encountered during routine evaluation of blood smears from healthy animals. Basophilia may be present in hypersensitivity reactions or parasite infections, similar to eosinophils. The morphology of basophils is species dependent. In camelids, basophils appear slightly larger than neutrophils and have a twisted or irregularly shaped nucleus with less condensed chromatin (Figure 14.9). The cytoplasm is more basophilic than in neutrophils, and there are variable numbers of dark blue granules.

Platelets and Coagulation

Camelid platelets are small, discoid to irregular in shape, have clear cytoplasm, and contain multiple azurophilic granules (Figures 14.2 and 14.10). RIs for platelet count for camelids are wide, and platelets are more numerous than in

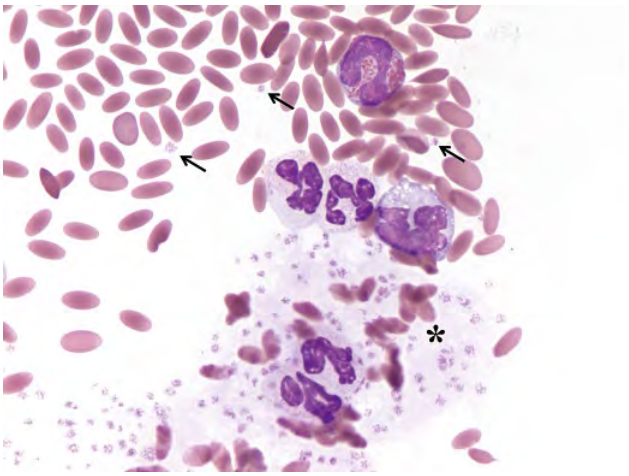

Figure 14.10 Blood from a normal alpaca. Individual platelets are small with central azurophilic granules (arrows). Blood films should be evaluated for platelet clumps as seen here (asterisk) as clumping can artifactually lower the platelet count. Also present are four neutrophils, one eosinophil, and one monocyte. Wright-Giemsa. 1000X.

many species (Table 14.1) [1, 17, 45, 54–56]. Juveniles (<18 months of age) have transiently higher but overlapping platelet counts compared with adults [17]. Camelid platelets have a mean platelet volume of 4.4–6.9 fl for alpacas [45] and 4.8–6.4 fl for dromedary camels (Table 14.1) [4, 57]. Because of the small size of camelid platelets, some may fall below the gated region for platelets on automated analyzers and, therefore, numbers may be undercounted by some hematology instruments [44, 45]. Evaluation of the peripheral blood smear to confirm the platelet estimate and to

examine the slide for platelet clumping is warranted (Figure 14.10). To estimate platelet numbers, one study used the average number of platelets observed in five oil immersion fields (1000 × magnification), in which 1 platelet = 15 000 platelets/μL (15×10^9/L) [45].

On ultrastructure, camel (*Camelus dromedarius*) platelets contain alpha granules, dense bodies and a microtubular system, similar to other species. The canalicular system is not open to the platelet surface, similar to cattle [57–59]. It has been suggested that platelets from camels may be resistant to activation by heat, thus conferring some protection against hyperthermia as an adaptation to a hot desert environment [58]. Similar studies investigating alpaca or llama platelet properties are not available.

Thrombocytopenia has rarely been reported in NWCs. Rattlesnake envenomation is associated with thrombocytopenia in NWCs [60, 61], and listeriosis with sepsis and secondary thrombocytopenia has been reported in a llama [62]. Thrombocytopenia was observed in sick camels (*C. dromedarius*) from Pakistan infected with various *Anaplasma* species [63], but thrombocytopenia was not noted in case reports of two alpacas and one llama infected with *Anaplasma phagocytophilum* in the United States [64–66]. Thrombocytosis may be seen in some cases of ectoparasitism, inflammation, or hemorrhage [17, 23]. Marked thrombocytosis was observed in an alpaca with acute myeloid leukemia (AML) [67].

Coagulation testing, including prothrombin time (PT), activated partial thromboplastin time (APTT), antithrombin activity, and fibrin degradation products, have been described in alpacas and llamas [45, 54, 68, 69]. PT and APTT are similar between llamas and alpacas and are most similar to dogs and cats (Table 14.1). Some differences in RIs for PT and APTT are apparent between studies, which is likely due to methodology and reinforces the need to establish RIs for the various camelid species in the laboratory performing these tests. Alternatively, blood from an age-matched normal control can be submitted simultaneously for comparison to a patient. Use of age-matched controls is particularly important when evaluating very young crias. A single case report suggested that as in other species, neonatal alpacas may have lower concentrations of coagulation factors and thus prolonged PT and APTT compared to adults [70].

Coagulopathies are rarely reported in camelids. Factor VIII deficiency (hemophilia A) was documented in a newborn male alpaca [70]. In dromedary camels, prolongation of PT and APTT is associated with *T. evansi* infection and is thought to be due to decreased factor production secondary to liver damage [37]. While it has been suggested that disseminated intravascular coagulation (DIC) may contribute to mortality in cases of hyperthermia [71], clear support for development of DIC was not seen in a case series of 15 NWCs (7 llamas, 8 alpacas) with heat stress [72]. Platelet counts were estimated to be within normal limits and coagulation testing was not performed. Of the eight animals that died and were necropsied, one had evidence of muscle hemorrhage and one had petechiae on the costal pleura.

Hematopoietic Neoplasia

Hematologic malignancies are uncommon in camelids. Most reported cases have been lymphoid lineage and typically present as lymphoma with involvement of multiple lymphoid tissues [1, 16, 73–80]. Multicentric lymphoma is the most common hematologic neoplasm in NWCs [81]. Lymphoma can occur in alpacas younger than one year of age, whereas lymphoma more commonly occurs in llamas greater than four years of age [74]. Clinical signs include weight loss and palpable masses of involved lymphoid tissues [81]. The most common laboratory changes are anemia, a left shift, hypoalbuminemia, and hyperglycemia. There is diffuse involvement of multiple tissues including lymph nodes, liver, kidney, heart, bone marrow, stomach, and the central nervous system (CNS) [77]. Neoplastic lymphocytes are often large and have moderate to abundant basophilic cytoplasm. Nuclei have stippled or irregularly condensed chromatin and one to several visible nucleoli (Figure 14.11). Though immunohistochemical labelling of normal alpaca lymph node tissue showed a typical distribution of B- and T-lymphocytes [74], too few cases of lymphoma in llamas and alpacas have been evaluated with

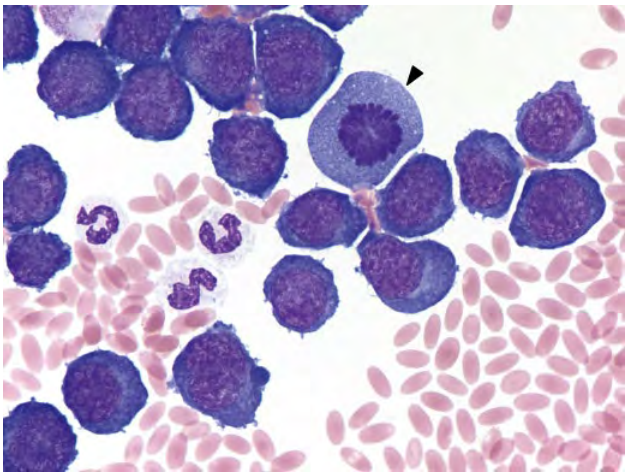

Figure 14.11 Abdominal fluid from an alpaca with lymphoma. There are three neutrophils and numerous neoplastic lymphocytes. The neoplastic lymphocytes are much larger than the neutrophils and have a moderate amount of basophilic cytoplasm. Nuclei are round to slightly irregular and have irregularly condensed chromatin and several nucleoli. There is a mitotic figure present in this image (arrowhead). Wright-Giemsa. 1000X.

immunodiagnostics to determine if they more commonly involve B- or T-lymphocytes. One case of bovine leukemia virus seropositivity based on PCR has been reported in a juvenile alpaca with multicentric lymphoma [81]. AML with multilineage dysplasia in an alpaca has been reported [67]. This alpaca had >20% blasts in the bone marrow, which is the threshold used by the new World Health Organization classification for AML in people, and has been proposed for animals with AML [67, 82].

Bone Marrow

Examination of bone marrow is sometimes helpful in the diagnostic evaluation, especially if there are abnormalities in more than one cell lineage as determined by a CBC. Bone marrow can be collected aseptically from the midline of the sternum with animals standing or in lateral recumbency, at the level of the caudal aspect of the elbow placed in natural position (llamas and alpacas) or from the fifth to eighth ribs in camels using 11–16-gauge bone marrow biopsy needle [14, 83–85] (See Chapter 4). Samples are collected in EDTA or with no anticoagulant if smears are made quickly. Bone marrow smears can be air-dried, stained with Wright-Giemsa stain or with Perl's stain for iron. In health, cellularity is 50–85% compared to the proportion of adipose tissue and stromal tissue [83, 85]. Morphology and maturation of erythroid, myeloid, and megakaryocyte lineages are similar to other species. The majority of the erythroid cells are metarubricytes (40%) and polychromatophilic rubricytes (30%), with fewer numbers of basophilic rubricytes (17%) and rubricytes (4%). Rubriblasts are less than 1% [83]. Erythrocytes maintain a round shape through polychromatophilic RBCs, after which they become oval. The majority of the myeloid cells are neutrophils, most of which are segmented (68%) and band neutrophils (16%). There are fewer metamyelocytes (9%), myelocytes (5%), and progranulocytes (2%). Myeloblasts are less than 1% [83]. There is a higher proportion of eosinophils (up to 14%) compared with other species, reflected by the higher numbers of circulating eosinophils [14, 83, 85]. The myeloid to erythroid ratio (M : E) is 0.9–2.9 in llamas [2, 83], 0.47–1.01 in alpacas [84], and 1.21 in dromedary camels [85], but may be lower in alpacas, llamas, and vicunas in high altitude [19]. Lymphocytes are <5%, plasma cells are <1%, and macrophages are <1% of the nucleated cells [83]. Megakaryocytes are most commonly associated with bone-marrow particles, with numbers ranging from 0–10/particle. Hemosiderin may be abundant and appears as dark greenish pigment with Wright-Giemsa stain within macrophages and golden pigment with hematoxylin and eosin [83].

Bone-marrow suppression can be associated with inflammation and chronic kidney disease, which typically cause erythrosuppression from inflammatory cytokines and decreased erythropoietin production, respectively [24]. This could result in an increased M : E ratio from myeloid hyperplasia and erythroid hypoplasia with inflammation, and from erythroid hypoplasia with chronic kidney disease.

Flow Cytometry and Immunohistochemistry

Comparative studies have shown that the structure, function, and pattern of expression of major histocompatibility complex (MHC) and leukocyte differentiation molecules are highly conserved among species, with few exceptions. The composition of leukocyte subsets in peripheral blood and lymphoid tissue appears to be similar in llamas and alpacas. Characterization of leukocyte subsets using antibodies directed against leukocyte differentiation molecules, often referred to as cluster of differentiation (CD) molecules can be helpful in the diagnosis of certain types of diseases involving hematopoietic cells and lymphoid tissues. CD molecules typically expressed on leukocyte subsets are shown in Table 14.2.

Table 14.2 Antigen expression on selected leukocyte subsets[a].

Surface antigen expression	Leukocyte subset
CD3	Pan T-lymphocytes
CD4	Helper T-lymphocytes
CD5	αβ and γδ T-lymphocytes
CD8	Cytotoxic/suppressor T-lymphocytes
CD11*a*	Panleukocyte marker, similar to CD18
CD14	Monocytes/macrophages
CD44	Panleukocyte marker, RBC, platelets
CD45	Panleukocyte marker
CD79α	B-lymphocytes
CD204	Macrophages
Iba1	Macrophages
Pax-5[b]	B-lymphocytes
MUM-1[b]	Plasma cells, some B-lymphocytes
Foxp3[b]	Regulatory T-lymphocytes

Abbreviations: CD, cluster of differentiation; Iba1, ionized calcium binding adaptor molecule 1; MUM-1, multiple myeloma-1; FFPELN, formalin fixed paraffin embedded lymph nodes.
[a] Uhde et al. [86].
[b] May not work in FFPELN in camelids.

B-lymphocytes comprise 15–41% of the lymphocytes in peripheral blood in adult llamas, based on expression of surface immunoglobulin (Ig), whereas T-lymphocytes comprise 21–61% of circulating lymphocytes [51]. The ratio of T-lymphocytes that express CD4 or CD8 is close to 1, with <1% that express both CD4 and CD8. αβ T-lymphocytes comprise 15–39% and γδ T-lymphocytes comprise 5–27% of CD8+ T lymphocytes. The number of circulating γδ T-lymphocytes is similar in juveniles and adults, which differs from cattle, sheep, and goats, but there is lower expression of CD5 on γδ T-cells than on αβ T-cells, similar to cattle and pigs. Expression of MHC class II on resting T-lymphocytes is low or absent [51]. Composition of T and B cells is similar in juveniles and adults, and similar patterns occur in alpacas [51].

Camelids have four Igs comprised of light and heavy chains similar to other mammals: IgM, IgG_1, IgA, and IgD, but also have two unique classes of two heavy chain immunoglobulins with no light chains. IgG_2 and IgG_3 are missing the C1 domain that binds the light chain portion of Igs, which likely helps direct the specificity and affinity that accompanies B-lymphocyte maturation. There may be two lineages of B cells that secrete these different forms of antibodies. These unique heavy chain antibodies comprise approximately 75% of antibodies in serum [51]. These have recently become useful in the development of new immunologic molecules for diagnostic, therapeutic and biochemical purposes in other species [87].

Immunophenotyping by flow cytometry can be used to assess expression of cell surface antigens on leukocytes from peripheral blood, lymph nodes, or other lymphoid tissue, which may be helpful in evaluation of animals with hematologic malignancies. Though some commercially available antibodies can be used for multiple species, some are species specific. Flow cytometry was used to support a diagnosis of AML in an alpaca [67]. Neoplastic cells expressed CD14, which is expressed on granulocytes; CD172a, a signal regulatory protein on granulocytes, monocytes, macrophages, and dendritic cells; and a high-affinity receptor for the complex of lipopolysaccharide (LPS) and LPS-binding protein expressed on myeloid-derived cells [51, 67, 88, 89].

Immunohistochemistry is also helpful sometimes in evaluation of animals with suspected hematologic malignancies, using monoclonal or polyclonal antibodies for antigen expression in formalin-fixed paraffin embedded lymphoid tissues. Similar to other species, B-lymphocytes from camelids express CD20 and CD79α, and T-lymphocytes express CD3 [86]. There may be a subset of T lymphocytes that express CD20 [90, 91]. In normal lymph nodes, mature and immature B-lymphocytes are concentrated in lymphoid follicles. T-lymphocytes are concentrated in the dense anodular lymphoid tissue, which resembles the paracortical region in other mammalian lymph nodes [86]. In the spleen, B-lymphocytes are located in follicles. T-lymphocytes are concentrated in the periarticular lymphoid sheaths (PALS), but are also dispersed in the splenic cords and sinuses. In the thymus, B-lymphocytes occur primarily in the medulla, but are also scattered throughout the cortex. T lymphocytes are closely packed in the cortical region of the thymus and are more loosely arranged in the thymic medulla. Monoclonal antibodies for expression of Pax-5, multiple myeloma 1 (MUM-1), Foxp3, and CD205 used in other species may not show specificity for these cell types in camelids [86].

Monocytes and macrophages express ionized calcium-binding adaptor molecule (Iba1), CD204, and myeloid/histiocyte antigen (calprotectin) [86]. Iba1 occurs on all cells of monocyte macrophage lineage, CD204 is expressed by macrophages in splenic red pulp and sinuses, and myeloid/histiocytic antigen is expressed by monocytes that have recently trafficked into tissues, depending on species. The distribution of macrophages in lymph nodes, spleen, and thymus in camelids is similar to other species [86]. Macrophages are distributed in the diffuse lymphoid tissue regions of lymph nodes, with fewer macrophages in follicles and dense anodular lymphoid tissue. In the spleen, macrophages are most numerous in the red pulp, with fewer macrophages in splenic follicles. Macrophages specifically expressing Iba1 surround small blood vessels and central arteries in the PALS regions of the spleen. In the thymus, macrophages occur in both the cortex and medulla.

Immunodeficiency Disorder in Juvenile Llamas

Immunodeficiency syndrome in juvenile llamas is characterized by very low numbers of B-lymphocytes and low Ig concentration in peripheral blood. The proportion of B-lymphocytes is only 1–5%, compared to 27–53% in healthy juveniles, which is accompanied by a higher proportion of T-lymphocytes that express CD8 (up to 50%). The syndrome may be due to an autosomal recessive genetic defect in B-lymphocyte maturation. The median age of onset is 11.6 months. Affected animals have poor growth, weight loss, anemia, and persistent and/or recurrent infections. A similar syndrome has not yet been reported in alpacas [51].

Biochemistry

Chemistry panels can be performed using serum (red top collection tube) or heparin (green top tube) [92]. It is important to use species specific RIs for clinical biochemistries for llamas, alpacas, and other camelids (Table 14.3).

Table 14.3 Biochemistry reference intervals for adult llamas, alpacas, and camels.

Measurand	Units	Llama[a]	Alpaca[a]	Dromedary camel[b]	Bactrian camel[c]
BUN	mg/dL	13–33	12–33	8–28	7–29
Creatinine	mg/dL	1.5–2.9	1.3–2.7	0.8–1.9	0.9–2.5
Phosphorus	mg/dL	4.2–10.6	2.5–8.6	2.2–10.5	2.7–8.7
Calcium	mg/dL	8.0–10.5	8.0–10.4	4.0–11.2	6.4–10.3
Ionized calcium	mg/dL	4.5–5.6	4.2–6.2	NA	NA
Magnesium	mmol/L	0.7–1.1	0.7–1.1	0.6–1.4	0.7–1.0
Ionized magnesium	mmol/L	0.5–0.7	0.5–0.7	NA	NA
Sodium	mEq/L	146–154	142–156	148–180	147–156
Potassium	mEq/L	4.1–6.4	3.6–6.5	4.9–7.6	3.6–4.7
Chloride	mEq/L	109–120	108–122	NA	107–115
Anion gap	mEq/L	2–18[d]	16–22[e]	NA	NA
Osmolality (calc)	mOsm/kg	295–312	286–314	NA	NA
Bicarbonate	mmol/L	19–31	19–29	NA	22–30
AST	IU/L	120–240	110–250	37–114	69–98
ALP	IU/L	20–150	20–150	56–407	33–196
GGT	IU/L	20–60	10–42	6–48	NA
SDH	IU/L	3–15	3–10	NA	NA
CK	IU/L	30–625	40–500	13–114	35–92
Cholesterol	mg/dL	15–70	12–58	15–54	NA
Triglyceride	mg/dL	5–55	5–30	18–97	NA
Total bilirubin	mg/dL	0.0–0.3	0.0–0.2	NA	NA
Direct bilirubin	mg/dL	0.0–0.1	0.0–0.1	NA	NA
Bile Acids	μmol/L	5–38	5–50	NA	NA
Total protein	g/dL	4.8–7.7	5.3–7.6	5.6–7.8	5.5–7.0
Albumin	g/dL	3.0–4.9	2.6–4.7	2.6–4.6	2.8–3.3
Globulin	g/dL	1.8–2.8	2.7–2.9	3.0–3.2	2.7–3.7
A:G		0.8–3.0	0.9–2.3	NA	NA
Glucose	mg/dL	100–149	100–132	58–167	NA
BHB	mg/dL	0.1–1.5	0.2–1.1	NA	NA
NEFA	mEq/L	0–0.24[d]	0.02–0.27[e]	NA	NA
Amylase	IU/L	420–1200[d]	824–1686[e]	NA	NA
Iron	μg/dL	70–148[f]	86–203[e]	84–112[c]	84–151

Abbreviations: A:G, albumin to globulin ratio; ALP, alkaline phosphatase; AST, aspartate transaminase; BHB, β-hydroxybuterate; BUN, blood urea nitrogen; CK, creatine kinase; GGT, γ-glutamyl transferase; NA, not available; NEFA, nonesterified fatty acids; SDH, sorbitol dehydrogenase (also called iditol dehydrogenase).

[a] Unless otherwise indicated, data for llamas and alpacas are from The Ohio State University Veterinary Medical Center Clinical Pathology Laboratories, established using serum with the Roche Cobas 6000.

[b] Unless otherwise indicated, data for dromedary camels are from Romdhane et al. [93].

[c] Elitok and Cirak [5].

[d] Oregon State University Veterinary Diagnostic Laboratory (https://vetmed.oregonstate.edu/sites/vetmed.oregonstate.edu/files/biochemistry_reference_intervals_1.pdf).

[e] Cornell University Animal Health Diagnostic Center (https://www.vet.cornell.edu/animal-health-diagnostic-center/laboratories/clinical-pathology/reference-intervals/chemistry), established using serum on the Cobas 501.

[f] Weiser et al. [15].

While RIs for clinical biochemistries of camelids have been published [2, 5, 46, 92–95], it is strongly recommended that each reference laboratory generate RIs specific to the species and instrumentation. Differences observed in published RIs can be due to analytic differences (test methodology, instrumentation) or to pre-analytic factors (age, sex and breeding state, husbandry, environment such as altitude). The components of a chemistry panel for camelids are similar to those used for other large animal species and include blood urea nitrogen (BUN), creatinine, electrolytes, liver and muscle enzymes, and glucose. In some cases, it may be useful to run additional indicators of energy status and lipid metabolism, including β-hydroxybutyrate (BHB), nonesterified fatty acids (NEFA), lactate, triglycerides, and cholesterol [56, 96].

BUN and creatinine are expected to increase with pre-renal, renal, and post-renal causes. The most common cause of pre-renal azotemia is dehydration, but other differentials can include causes of circulatory shock. Renal azotemia has been documented in cases of toxicoses (vitamin D, oak leaves), developmental abnormalities, infections, nephrotoxic drugs (non-steroidal anti-inflammatory drugs, aminoglycosides) and neoplasia [97–104]. Post-renal causes include urolithiasis and cystitis in males, urinary tract abnormalities in crias, and ruptured bladder [105–109]. In camelids, pre-renal azotemia can result in a disproportionate increase in BUN compared to creatinine. One author has suggested that if BUN is 20 or more times creatinine (both measured as mg/dL), this is most compatible with pre-renal azotemia [56]. Whereas urea nitrogen is primarily excreted in urine, it can be utilized by the forestomach microbes as a nitrogen source. Consequently, any expected rise in BUN may be blunted if the animal is also anorexic due to a combination of decreased protein intake and microbial utilization. Creatinine concentrations are influenced by muscle mass so creatinine may be lower than expected in emaciated animals or in young crias. Newborn crias can have an elevated creatinine but this should return to normal during the first week after birth [110].

Electrolytes in camelids follow similar patterns as observed in other species. Hypernatremia, often with hyperchloremia, accompanies disorders that result in free water loss, decreased water intake, increased renal sodium retention, or increased sodium intake. Hyponatremia and hypochloremia occur with loss of fluid and electrolytes, through mechanisms such as renal or GI losses, uroperitoneum [105], hypoaldosteronism [111], or decreased intake. A disproportionate change in sodium and chloride suggests an acid/base disorder. In camelids, proximal GI obstruction can present with hypochloremia without hyponatremia [112]. (Chapter 12) Decreased feed intake is an important cause of hypokalemia. Though hyperkalemia may accompany metabolic acidosis, whole body depletion of potassium with anorexia may blunt any increases in blood potassium [113].

Calcium and phosphorus are regulated through a complex combination of intake, absorption from the bone and gut, renal excretion, cellular translocation, and hormonal control. Phosphorus concentration is higher in young camelids compared to adults and shows seasonal variation in all ages [114]. The highest concentrations of serum phosphorus in camelids occur in summer months and likely relate to solar-enhanced production of Vitamin D [114]. Conversely, high calcium to phosphorus ratios, low serum phosphorus, and low vitamin D are documented in the winter months, a time during which growing crias are most at risk for developing hypophosphatemic rickets [115] (see Chapter 3). Hypocalcemia is commonly seen in sick camelids and is often related to hypoalbuminemia and decreased feed intake. Growing or lactating animals on rapidly growing pasture are at risk for developing grass tetany, characterized by hypocalcemia and hypomagnesemia. Hypermagnesiumia and hyperphosphatemia may be associated with azotemia of diverse causes.

Aspartate transaminase (AST) is commonly used as an indicator of hepatocellular damage; however, it has limitations. AST can increase with hepatocellular or myocyte damage so it is best interpreted with other enzymes such as creatine kinase (CK). CK is released from myocytes so concurrent increases in CK and AST are suggestive of muscle damage. Increased CK activity can result from intramuscular injections, trailering, uterine torsion [116], or when an animal is down and unable to rise, a common occurrence with sick camelids. Marked increases in CK can occur with myopathies associated with heat stress, vitamin E/selenium deficiency (See Chapter 3), toxicities, or myositis [72, 97, 117, 118]. Because of its shorter half-life, CK is expected to fall more rapidly than AST following acute muscle damage. Prolonged increases in AST after CK has decreased may suggest ongoing hepatocellular injury after muscular injury has begun to resolve. Sorbitol dehydrogenase (SDH, also called iditol dehydrogenase) is liver specific, and a simultaneous increase in SDH and AST supports hepatocellular damage. Because of its shorter half-life, SDH activity is expected to decline more rapidly than AST following acute hepatocellular damage [119]. The disadvantage of SDH is that it is less stable [119], and activity may deteriorate with storage or shipping of the sample. SDH activity in llama serum significantly decreased by 24 hours when stored at room temperature, but proved stable in serum and heparinized plasma for up to one week if stored at 4 °C or − 20 °C [119]. In that study, serum and plasma were harvested within one hour of sample collection. A liver specific and more stable alternative to SDH is glutamate

dehydrogenase (GLDH); however, it is not as commonly available on routine chemistry panels [92].

The biliary enzymes γ-glutamyl transferase (GGT) and alkaline phosphatase (ALP) are indicators of cholestasis. GGT is considered to be a more sensitive indicator of cholestasis than ALP in camelids [56]. Both GGT and ALP increase with hepatic lipidosis [120]. Transient increases in GGT can be observed in neonates due to absorption from colostrum; however, GGT is not a good indicator of adequate passive transfer of immunoglobulin [121, 122]. ALP is higher in young growing animals compared to adults. Total bilirubin concentrations are low in normal camelids and do not appear to increase with anorexia, so even mild increases in bilirubin are considered significant [56, 123]. Some newborn crias will have an elevated bilirubin for up to two weeks after birth.

Bile acids have been minimally evaluated as indicators of hepatocellular function and are not affected by feeding or time of day in llamas [124]. Increased bile acids have been observed in camelids with hepatic lipidosis [120, 123].

Normal adult llamas and alpacas have higher blood glucose concentrations compared to other species. This appears to be due to lower circulating insulin concentrations in health, partial insulin resistance and glucose intolerance, a sluggish insulin response to hyperglycemia, and poorly controlled gluconeogenesis [96, 125–127]. Increased production of endogenous stress hormones or administration of corticosteroids and catecholamines can result in hyperglycemia [96, 128–130]. Hyperglycemia occurs with a wide variety of pathologic conditions including stress, GI dysfunction, hepatic disease, uterine torsion, hepatic lipidosis, neoplasia, envenomation, parasitism, and infectious disease [60, 96, 98, 116, 120, 123, 131–138]. Though seemingly counterintuitive, hyperglycemia can sometimes be seen with conditions that result in a negative energy balance, including pregnancy and lactation, especially if the animal is stressed [96, 120, 123, 131, 139]. If the renal threshold for glucose of 180 mg/dL is exceeded, glucosuria and subsequent loss of water through osmotic diuresis can occur. If water intake is inadequate to keep up with renal water loss, the animal is at risk of becoming dehydrated and developing hyperosmolar syndrome characterized by marked hypernatremia and hyperglycemia [96, 140]. (See Chapter 13)

Camelids in negative energy balance mobilize fat and enhance ketogenesis. Concentrations of NEFA >1 mEq/L and BHB >5 mg/dL suggest a high risk for developing hepatic lipidosis [56, 96]. Hepatic lipidosis is characterized by increases in NEFA, BHB, cholesterol, bile acids, and hepatocellular (AST, SDH) and biliary (GGT, ALP) enzymes. Hyperglycemia is common except in pregnant and lactating camelids that may be hypoglycemic. Many camelids with hepatic lipidosis develop metabolic acidosis and are hypoproteinemic and hypoalbuminemic. Triglycerides increase with severity of disease. With marked hypertriglyceridemia (> 500 mg/dL), serum will appear lipemic. Hypertriglyderidemia and lipemia can occur independent of hepatic lipidosis, but if left untreated may contribute to the development of hepatic lipidosis [141].

Hypoglycemia is less common than hyperglycemia in camelids, but may occur with anorexia during pregnancy, sepsis, liver disease, *M. haemolamae* infection, and sick crias [31, 96]. Delayed separation of the serum from the cells prior to assay results in decreased measured glucose and should be considered a potential reason for low measured glucose [142].

Total protein and albumin are routinely measured as part of a biochemical profile. Refractrometry can be used to provide an estimate of total protein except in animals with lipemic serum or plasma. Serum protein electrophoresis has been reported for camelids, and the number of globulin peaks may vary with the methodology used. Using agarose gel technique, alpacas have 7 peaks (albumin; alpha 1 and 2; beta 1, 2, and 3; and a single globulin peak) [92], whereas llamas have 8 peaks (albumin; alpha 1, 2a, and 2b; beta 1, 2, and 3; and gamma) [143]. Dromedary camels are reported to have four (albumin, alpha, beta, gamma) or six (albumin; alpha 1 and 2; beta 1 and 2, gamma) peaks [5, 144]. Hypoproteinemia and hypoalbuminemia are common, non-specific findings observed in a variety of illnesses in camelids. This may relate to protein loss with GI diseases or parasitism and/or protein catabolism with excessive gluconeogenesis. Hyperproteinemia most commonly is due to dehydration.

Peritoneal and Pleural Fluid Analysis

Analysis of peritoneal and pleural fluid includes assessment of color and clarity, nucleated cell counts, protein determination, and evaluation of stained smears of the fluid. Cell counts in pleural and peritoneal fluids can be determined using a hemocytometer or an automated analyzer [145]. Protein concentration is determined by refractometry or biochemical analysis. Smears made from pleural and peritoneal fluids and stained with Romanowsky type stains or quick stains are used to determine the differential cell count to assess for evidence of inflammation, organisms, intestinal compromise, or neoplasia. If the cell count is high or the sample is bloody, direct smears are made; however, because cell counts are often low, concentration by either sedimentation or cytocentrifugation can facilitate slide preparation and evaluation. Based on these

parameters, fluids are classified as low protein transudates (protein <2.5 mg/dL and <3000 cells/μL), high protein or modified transudates (protein >2.5 mg/dL and <5000 cells/μL) or exudates (protein >3.0 mg/dL and >5000 cells/μL). Volumes of peritoneal fluid obtained from healthy llamas and alpacas are 1–10 mL [146] and 5–7 mL in healthy dromedary camels [147]. Peritoneal fluid is clear to hazy and colorless to yellow or straw-colored in healthy camelids. A pink to red color occurs with blood contamination or hemorrhage. Much smaller volumes (<1.0 mL) are obtained from the pleural cavity in healthy adult llamas, and blood contamination of the sample imparting a pink to red color is common [148].

Peritoneal and pleural fluids in healthy llamas, alpacas and dromedary camels are typically low protein transudates (Table 14.4) [46, 146–148]. Though most peritoneal fluid total nucleated cell counts are <3000 cells/μL with a median of 780 cells/μL, a wide range has been reported (110–20 500 cells/μL) in healthy NWC [146]. The highest cell counts and highest protein concentrations (up to 4.0 g/dL) were observed in samples with the lowest collected volume [146]. In pleural fluid from healthy llamas, cell counts are typically <1000 cells/μL with a reported range of 200–1500 cells/μL and 95% confidence interval of 385–768 cells/μL [148].

Evaluation of peritoneal and pleural fluid can be diagnostically useful in evaluating a variety of conditions. Increased cell counts can occur with either inflammation (peritonitis, pleuritis) or neoplasia. Peritonitis characterized by an increased nucleated cell count, increased percentage of neutrophils, and increased protein concentration can be observed in some cases of proximal and distal obstruction or rupture, septic peritonitis without viscus rupture, necrotizing enteritis or pancreatitis [146, 149]. In general, the highest cell counts and protein concentrations were observed in camelids with sepsis from either leakage from a compromised intestinal tract or hematogenous spread. The presence of degenerate neutrophils suggests a septic process, and the smear should be carefully examined for bacteria (Figure 14.12). Infection with *Streptococcus equi* subsp. *zooepidemicus* is an important cause of bicavitary exudates characterized by increased cell counts, increased protein concentration, and intracellular and extracellular bacterial cocci in NWC [49] and dromedary camels [150]. However, culture is more sensitive in determining bacterial infection than identification of bacteria on the cytology smear. Increased nucleated cell counts with a predominance of large mononuclear cells were observed in alpaca crias with spiral colon impaction [151].

Cytologic evaluation of fluid can sometimes provide a diagnosis of neoplasia [134]. Lymphoma, while rare, is the most common neoplasm in NWC and can result in increased cell counts and presence of neoplastic lymphocytes in pleural [152] or peritoneal fluid (Figure 14.11) [73, 153]. Routine

Table 14.4 Peritoneal and pleural fluid analysis in clinically healthy adult llamas, alpacas and dromedary camels.

		Peritoneal fluid		Pleural fluid
Measurand	**Units**	**Llamas and Alpacas[a]**	**Dromedary Camels[b]**	**Llamas[c]**
Appearance		Clear to hazy Colorless to light yellow	Clear Pale cream	Hazy to opaque Pink to red
Protein	g/dL	<2.5	1.6 ± 0.6	<2.7
Nucleated cell count	cells/μL	<3000	270 ± 30	576 ± 361
Neutrophils	%	65 (15–98)	30.11 ± 1.49	0–10
Large mononuclear cells	%	32 (2–83)	6.85 ± 0.48	0–20
Lymphocytes	%	2 (0–26)	62.64 ± 0.73	80–100
Eosinophils	%	0 (0–9)	0.40 ± 0.24	0
Glucose	mg/dL	140 ± 39	89.3 ± 38.3	135 ± 9.02
Lactate	mg/dL	0.72 ± 0.61	NA	2.95 ± 1.34
Sodium	mEq/L	147 ± 3	146 ± 19	NA
Potassium	mEq/L	4.4 ± 0.6	5.2 ± 0.7	NA
Chloride	mEq/L	116 ± 3	NA	NA

Abbreviations: NA, not available.
[a] Cebra et al. [146]. Data are presented as median (range) or mean ± standard deviation.
[b] Naeini and Nazifi [147]. Data are presented as mean ± standard error of the mean.
[c] Gerros et al. [148] Data are presented as range or mean ± standard deviation.

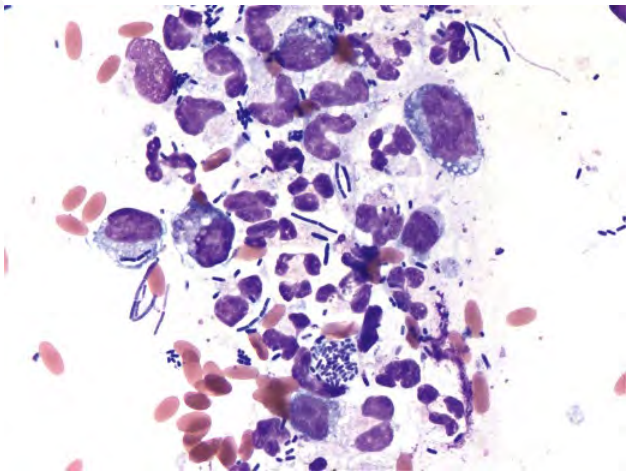

Figure 14.12 Abdominal fluid from a four-year-old male alpaca with septic peritonitis. The fluid appeared light yellow and cloudy and had a protein concentration of 3.1 g/dL and a WBC of 72/μL. The majority of the cells are degenerate neutrophils with five large mononuclear cells and one lymphocyte. Numerous intracellular and extracellular bacteria are present and consist of a mixed population of long and short rods. Wright-Giemsa. 1000X.

cytologic evaluation can be supplemented with immunocytochemistry or immunohistochemistry [153].

Normal peritoneal fluid contains slightly lower concentrations of glucose and lactate compared to venous blood [146, 147], whereas pleural fluid glucose and lactate concentrations are not significantly different from plasma [148]. Interpretation of peritoneal fluid glucose is problematic in light of the hyperglycemic stress response in sick NWC which may exaggerate the differential between blood and peritoneal fluid glucose concentrations or mask consumption of peritoneal fluid glucose during septic peritonitis [56]. Bladder rupture can occur secondary to urolithiasis or trauma in NWC and dromedary camels. Uroperitoneum is characterized by a low protein transudate with concentrations of creatinine, urea nitrogen, and potassium higher in the fluid than the serum [105, 107]. Amylase and lipase activities are lower in peritoneal fluid compared to serum in NWC (<1000 IU/L and <250 IU/L, respectively) [154] and dromedary camels (amylase 23.96 ± 5.96 IU/L) [147]. Though changes in abdominal fluid cell count and protein in camelids with pancreatic necrosis are inconsistent, amylase and lipase enzyme activities in the peritoneal fluid are reported to be higher compared to serum [149].

Cerebrospinal Fluid (CSF) Analysis

CSF analysis is often helpful in the diagnostic workup of animals with evidence of disease involving the CNS or fever of unknown origin. Results should be interpreted with other clinical findings and with the understanding that not all neurologic diseases are accompanied by changes in CSF, abnormalities may be non-specific, the magnitude of abnormal results can vary with location of the lesion and disease progression, and CSF analysis may not always correlate with results of imaging studies. A small volume (2–3 mL) of CSF typically is collected from the lumbosacral space, but also can be collected from the atlantooccipital space. The sample can be split into a tube containing EDTA for cytology and a plain tube for determination of additional analytes. Culture and susceptibility testing for infectious agents requires submission in a sterile tube [155, 156].

CSF analysis should be performed promptly after specimen collection to prevent cell degradation, which occurs rapidly because of the low protein concentration in CSF. Samples should be kept at refrigeration temperature prior to analysis. Though some analysis can be performed in a practice setting, samples are often sent to a reference laboratory for complete analysis. Ideally, the reference laboratory should be contacted prior to submission to confirm sample submission requirements, and to ensure prompt analysis once the sample is received [157].

CSF analysis includes assessment of color and clarity, WBC counts, RBC counts, microprotein determination, and evaluation of the types of cells present. Normal CSF is colorless and clear. Nucleated cell and RBC counts are performed using a hemocytometer and are reported as cells per microliter. Though some automated analyzers have been validated for nucleated cell counts in CSF from dogs with increased CSF cell counts (pleocytosis), they do not perform as well for CSF with normal or low cell counts and have not been validated for other species [157]. Nucleated cell counts in CSF from healthy camelids are listed in Table 14.5 [155, 158].

In reference laboratories, CSF microprotein is determined with an automated analyzer using protocols that are specific for the low protein concentration in CSF. CSF protein concentration in healthy camelids is shown in Table 14.5. Disturbances of the blood–brain barrier due to inflammation, trauma, parasite migration, meningitis, and otitis can increase CSF protein concentration [156]. CSF proteins can be further evaluated using protein electrophoresis or immunoelectrophoresis, but this is not performed routinely.

Cytologic evaluation is a critical component of CSF analysis to determine the types of cells present even if the total cell count is normal. In house methods to prepare glass slides for cell evaluation have been described [159]. Slides are prepared in reference laboratories using cytocentrifugation. Slides are air-dried and stained with conventional Romanowsky stains for microscopic evaluation and

Table 14.5 CSF analysis in clinically healthy adult llamas and dromedary camels.

Measurand	Units	Llamas[a]	Dromedary camels[b]
Appearance		Clear, colorless	Clear, colorless
Protein	mg/dL	31.2–66.8	48.7–60.8
RBC	cells/μL	<370	<80
WBC	cells/μL	<3	<9
Neutrophils	%	0–8	5–21
Large mononuclear cells	%	4–45	7–18
Lymphocytes	%	37–92	43–74
Eosinophils	%	0–10	0–3
Glucose	mg/dL	66–73	54–70
Sodium	mmol/L	151–157	152–154

[a] Welles et al. [155] Data are presented as reference intervals.
[b] Al-Sobayil [158]. Data are presented as 95% confidence intervals.

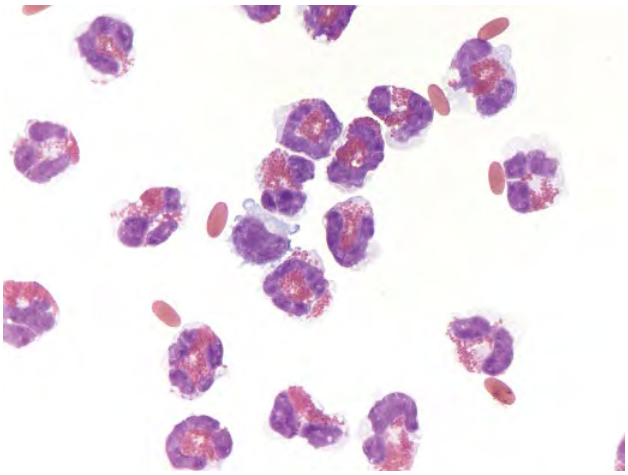

Figure 14.13 CSF from a seven-year-old male castrated alpaca with neurologic signs. The CSF was clear and colorless and had a protein concentration of 416 mg/dL, a RBC of 65/μL, and a WBC of 60/μL. The WBC differential was 97% eosinophils, 2% small lymphocytes, and 1% large mononuclear cells. In this image, there are numerous eosinophils, and there is one lymphocyte in the center. Several RBCs also are present. Wright-Giemsa. 1000X.

determination of the differential cell count. Cell differentials for CSF from healthy llamas and Dromedary camels are shown in Table 14.5. Alterations in the distribution of leukocytes can be helpful in some CNS diseases. For example, most bacterial meningitis or meningoencephalitis are associated with an increased proportion of neutrophils, whereas infection with *Listeria monocytogenes* is characterized by an increased proportion of monocytes [156].

Eosinophilic pleocytosis (Figure 14.13) occurs most commonly in camelids with *Parelaphostrongylus tenuis* (*P. tenuis*), also known as meningeal worm. Other causes of eosinophilic pleocytosis include toxoplasmosis, and in United Kingdom, infestation with *Elaphostrongylus cervi* [156]. Eosinophils greater than 17% of the nucleated cells in CSF had a sensitivity of 85% and a specificity of 92% for the diagnosis of *P. tenuis* using identification of parasites on histology as the reference method [160]. In this study, the median percentage of eosinophils was 83% (range, 0–95%) and the range of eosinophils was 1080–2034/μL. These findings are similar to a recent retrospective study of *P. tenuis* in 20 camelids [48].

Additional testing can include bacterial culture and sensitivity, fungal culture, enzyme-linked immunosorbent assays for antibody detection, and PCR-based assays for nucleic acid detection, but it is important to remember that false-positives and false-negatives can occur. Measurement of glucose, sodium, neurotransmitters, enzymes, and other substances can be performed, but other than decreased glucose concentrations in bacterial meningitis and increased sodium concentrations in salt toxicity and in crias with hyperosmolar syndrome, limited information is available about the clinical significance of these other measurands in camelids [156].

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15

Cardiovascular System

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Anatomy and Physiology

Overall, the cardiac anatomy of camelids is similar to other domesticated mammals in regard to chambers, valves, and great vessels with some mild variations in coronary anatomy and arterial branching [1, 2]. The apex of the left ventricle tapers to a pointed conical shape, similar to other large animal species.

Hemodynamics are similar to other domesticated species with some specialized cardiovascular adaptations. South American camelids (SACs), who naturally evolved in the high-altitude Andean region, have developed specialized cardiovascular adaptations that allow them to survive and thrive in hypoxic environments [3–5]. A natural tolerance to hypobaric environments is supported by the resistance of the pulmonary vasculature to pulmonary hypertension. In low-land species, including humans, the response to chronic high altitude and hypoxemia is pulmonary vasoconstriction with secondary pulmonary hypertension and right ventricular hypertrophy [6]. Studies in llamas living at high altitude have demonstrated that they do not develop pulmonary arterial medial hypertrophy or have increased right-heart weights [4]. Camelids also have unique small elliptical-shaped red blood cells with specialized adaptions including a relatively high hemoglobin concentration and oxygen carrying capacity [7, 8].

Special Diagnostic Procedures

Physical Exam

Physical exam findings for camelids with heart disease are similar to other species including murmurs, arrhythmias, dyspnea, tachypnea, cyanosis and history of syncope; though, they can also present without any clinical signs or physical exam abnormalities. As in other species, innocent or functional murmurs are common in young camelids. In general, an innocent or functional murmur (a murmur in which no structural disease is present) should be a grade 3/6 or less, localized to the heart base, occur in patients with no other cardiovascular clinical signs, and decrease in intensity as the patient matures [9, 10].

Other Testing

Specialized diagnostic procedures to evaluate for cardiovascular disease are similar in camelids as other veterinary species including electrocardiography, radiography, echocardiography, angiography, computed tomography (CT), and specialized hematologic testing (specifically cardiac troponin I). These tests typically require specialized equipment and a cardiologists' interpretation.

Diseases

In a retrospective study of the Veterinary Medical Database from Purdue University and Colorado State University, the prevalence of heart disease in llamas was 7.06 and 12.37%, respectively [10]. Of the 663 llamas that presented to Colorado State over a seven-year period, 58 (8.7%) were diagnosed with acquired heart disease, vascular disease, or ECG abnormalities, where 24 (3.6%) had congenital heart disease with ventricular septal defects being most common (14/24 cases). It is hypothesized that their evolutionary adaptations to hypoxic conditions may allow them to compensate for complex defects as compared to other veterinary species, allowing pre-mortem diagnosis and reporting [9].

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Congenital Heart Diseases

Diagnosis of congenital heart disease (CHD) is important clinically as some diseases can be incidental and hemodynamically stable, while some can cause significant morbidity and mortality. Many CHDs are hypothesized or proven to be heritable in other species, making the diagnosis of CHD especially important for counseling owners and breeders about potential breeding risks. See Chapter 21 on congenital and heritable conditions in camelids.

Echocardiography (ECG) is the gold standard for antemortem diagnosis of CHD in veterinary species. Ventricular septal defects (Figure 15.1) are most commonly reported [10–16]. They typically result in a loud systolic murmur, though this can vary depending on the presence of concurrent defects. Other CHDs reported in camelids include atrial septal defects [10], patent ductus arteriosus [10, 11, 14], persistent right aortic arch [17, 18], tetralogy of Fallot (Figure 15.2) [9, 10], tricuspid atresia [19], atrioventricular septal defect [20], pulmonary atresia [16], transposition of the great arteries [9], truncus arteriosus [13, 16], mitral valve stenosis [10], double-chambered right ventricle [15], double-outlet right ventricle [16], and transient peripheral pulmonary artery stenosis [21]. Treatment of CHD is not usually required or feasible, though successful interventional closure of a patent ductus arteriosus (Figure 15.3) has been reported [11].

Acquired Heart Diseases

Nutritional Muscular Dystrophy

Also known as white muscle disease, this metabolic disease is caused by a dietary selenium or vitamin E deficiency in a variety of species [22] and has been reported in an Arabian camel [23]. This disease can be mild and subclinical or severe resulting in myocardial fibrosis, failure, and arrhythmic instability. If identified early, treatment should be aimed at correcting the nutritional deficiency and supportive care.

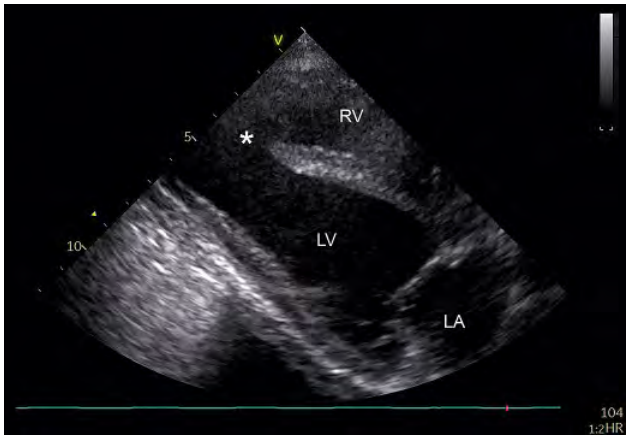

Figure 15.1 Two-dimensional ECG image from a one year old alpaca with an apical ventricular septal defect; LA, left atrium; LV, left ventricle; RV, right ventricle; *, interventricular communication.

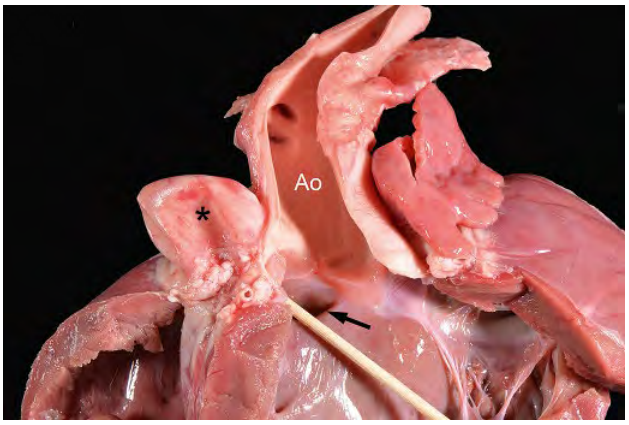

Figure 15.2 Tetralogy of Fallot in an alpaca cria. The pulmonary artery is almost completely atretic and is present only as a blind-ended cap (*). An overriding aorta (Ao) and perimembranous ventricular septal defect (arrow) are also present (*Source:* Photo courtesy of Dr. Rebecca Kohnken).

Infectious Diseases

Infectious valvular and mural endocarditis can be caused by acute or chronic bacteremia. Typically, the right-sided valves are affected more commonly in large animal species, while the left-sided valves are more commonly affected in small animal species [24–26]; though infections may also occur in the mural myocardium[27]. In a report of 10 alpacas with thrombotic endocarditis, six had only mural lesions, while four had a combination of mural and valvular lesions [27]. An infectious etiology was not found in these cases. It is hypothesized that either the body's host defenses sterilized the lesions or they were potentially affected with a noninfectious etiology as seen in people with chronic nonbacterial thrombotic endocarditis [28]. Infectious agents have been identified as the causative agent in mural and/or valvular endocarditis in camelid species including *Listeria monocytogenes* in an alpaca [29], *Streptococcus equi subspecies zooepidemicus* in alpacas [30], *Mycoplasma hominis* in alpacas [31], *Clostridium perfringens* in a llama [32], *Toxoplasmosis* in a llama [33], and *Escherichia coli* in a dromedary camel [34]. Sarcosporidiosis is very common in Arabian camels resulting in subclinical infections [35]. Sarcocysts have also been reported in cardiac tissue of wild and domesticated SCAs [36, 37]. Fibrinous endocarditis can also be secondary to other noninfectious causes such neoplasia (Figure 15.4).

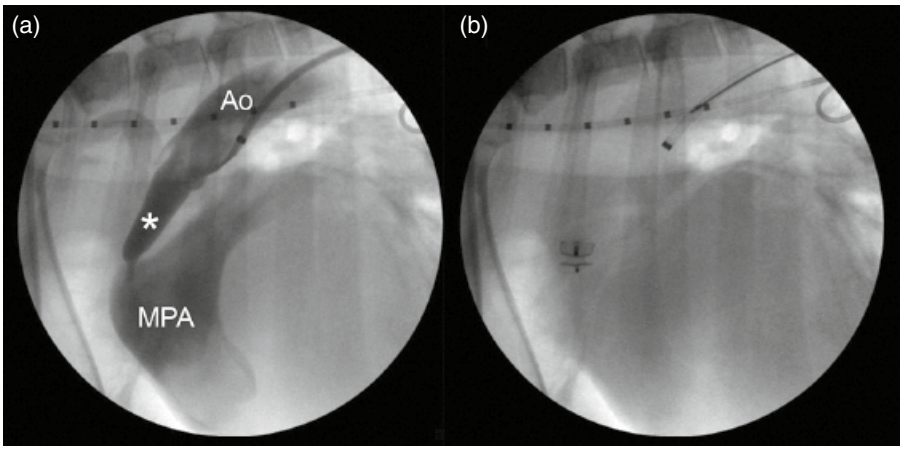

Figure 15.3 (a) Angiographic images from a six month old alpaca cria with a patent ductus arteriosus; (b) Angiographic image demonstrating closure of the patent ductus arteriosus with a Amplatz canine duct occluder; Ao: descending thoracic aorta; MPA: main pulmonary artery; *: patent ductus arteriosus.

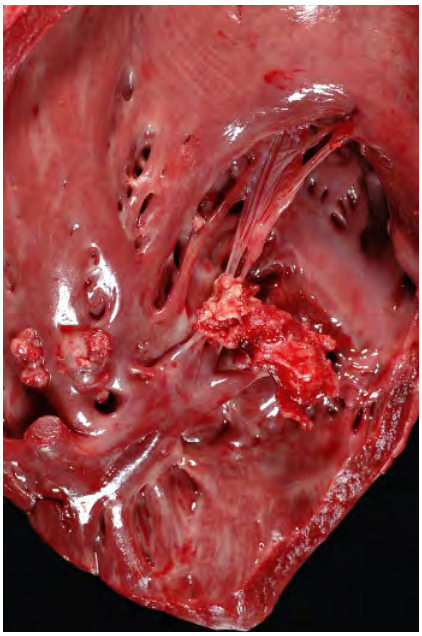

Figure 15.4 Metastatic carcinoma in the myocardium of an alpaca. Fibrinous endocarditis was also affecting the mitral valve (*Source:* Photo courtesy Dr. Lauren Himmel and Dr. Tracey Papenfuss).

Toxins

While there are numerous potential cardiac toxins that affect large animal species, oleander is the most commonly reported in camelids [38–40]. Oleander is a small flowering tree that contains deadly glycosides that inhibit the Na^+/K^+ ATPase in the heart resulting in deadly concentrations of calcium within the cells [41]. Sudden death is common [40]. In a report of eight camelids (three alpacas and five llamas) with atrial fibrillation, oleander intoxication was suspected or confirmed in three of the cases [38]. Though decontaminant and supportive treatment can be attempted, this toxicity commonly results in significant arrhythmias and myocardial necrosis and dysfunction (see Chapter 22).

Pericardial Disease

Pericardial disease has rarely been reported in camelids [10, 42]. Causes included pericarditis (infectious and/or inflammatory), traumatic hemopericardium, and pericardial effusion of unknown origin. Clinical findings suggestive of pericardial disease include a history of weakness and collapse and the presence of muffled heart sounds, weak arterial pulses, and distended jugular veins. Depending on the chronicity and cause, other non-specific signs include lethargy, poor appetite and weight loss can be seen. ECG is the best test to identify the presence of pericardial effusion and may assist with etiologic diagnosis. Pericardiocentesis with cytologic examination of the fluid is recommended to identify a possible cause.

Cardiac Neoplasia

Primary and secondary cardiac neoplasms appear rare in camelids (Figure 15.4). There is a report of primary pulmonary neoplasia in two llamas. Evidence of metastasis within the myocardium was present in one of the two cases [43].

Cardiomyopathy

Primary cardiomyopathy appears to be rare in camelids. Dilated cardiomyopathy has been diagnosed in an adult alpaca [44]. Though this alpaca was assumed to have primary or idiopathic dilated cardiomyopathy because an inciting cause was not found on pre- or postmortem examination, the diagnosis of primary dilated cardiomyopathy can be challenging because of the wide array of potential causes [45]. A detailed history and blood work (including cardiac troponin I) should be performed to evaluate for toxin exposure and myocarditis as the potential underlying cause. Hypertrophic cardiomyopathy has been diagnosed on postmortem examination in an adult alpaca that died suddenly [46]. Pre-mortem diagnosis of hypertrophic cardiomyopathy has been diagnosed by ECG in an adult

dromedary camel that presented with syncope [47]. Given the rarity of these diseases, specific recommendations for treatment are not available. If secondary cardiomyopathy is identified or suspected then treatment should be aimed at the underlying cause (infectious, inflammatory, neoplastic).

Figure 15.5 ECGs from a one-month-old alpaca cria. (a) Complete third-degree atrioventricular block with a slow ventricular escape rhythm (heart rate: 38 bpm); (b) Complete third-degree atrioventricular block with multifocal accelerated idioventricular rhythm (heart rate: 94 bpm) followed by a 10 second ventricular pause followed by a slow ventricular escape rhythm (heart rate: 50 bpm).

Arrhythmias

Primary rhythm disturbances are rarely reported in camelid species [10, 38, 42]. Arrhythmias are typically secondary or associated with other conditions (cardiac toxin, cardiomyopathy, pericardial disease, endo/myocarditis). Lone atrial fibrillation has been reported in a llama in which attempted cardioversion with quinidine was unsuccessful [38]. The author has diagnosed complete third-degree atrioventricular block and accelerated idioventricular rhythm (Figure 15.5) in an alpaca cria with aspiration pneumonia.

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16

Reproduction and the Reproductive System

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South American camelids (SACs) occupy some of the most inhospitable habitats in the world. The progenitors of the four species now found in the Andean countries developed unique reproductive strategies that enabled them to survive and flourish. Reproductive anatomy and behavior are similar in both SACs and Old-World camelids (OWCs). During the last few years, there has been an increasing interest in studying camelid reproductive biology and, as a result, useful information is available to students and clinicians.

Consideration of the reproductive strategies of vicuñas and guanacos may aid in understanding problems of llamas and alpacas. The anatomy and physiology of the reproductive system will be addressed, followed by a discussion of infertility.

Reproductive Strategies

Vicuñas

Vicuñas are limited to ranges at high elevations in the Andes of Peru, Bolivia, and the border between Argentina and Chile. They are nonmigratory. Family groups are highly territorial. The dominant male defends a family feeding territory against intrusion by strangers, either male or female. The borders of the territory are strictly delineated. The family group also maintains a sleeping territory, to which the family retreats in the evening. Corridors between sleeping and feeding territories may be neutral territory for several family groups.

Vicuñas are seasonal breeders in Peru, with 90% of all births occurring between mid-February and early April [1–6]. Though females will be bred within two weeks of parturition, only approximately half of the females will give birth the following year [7].

Vicuña social structure consists of family groups of two to five females and their crias, which are dominated by a single breeding male, and male groups (bachelor herds), varying from two to 100 animals. Both male and female offspring are driven from the family group by the dominant male (males at 4–9 months of age, females at 10–12 months). Females rarely conceive before they are 2 years old, but a few become pregnant at 12–14 months [1].

Males driven from the family group form temporary associations with other males and do not attempt to establish their own territories until four years of age. Juvenile females driven from the family group may attempt to enter another family group, but they may be repulsed by the male so as to maintain the optimum size of the group. Unattached females ultimately become part of a new male's family group.

In the Andes, more than 90% of vicuña births occur in the morning hours. This is probably an evolutionary adaptation to the weather patterns of that area [1]. The frequent afternoon storms could interfere with adequate drying of the cria before the nightly drop to near-freezing temperatures occurs.

Guanacos

Reproductive strategies and the social organization of guanacos are similar to those of the vicuña. However, guanacos have a broader distribution in more diverse habitats, from sea level to 3000 m [1]. Guanacos are more flexible than vicuñas; some populations are sedentary, whereas others are migratory. Migratory behavior necessitates some modifications of family group and male group patterns.

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In addition to these, aggregates of females may form during the winter, while each breeding male remains alone in the family group territory. There may even be mixed groups of males and females that come together during the winter.

Reproduction takes place only within the family group. Juvenile males and females are evicted from the family group, as in the vicuña strategy, but the age differs at which dispersal occurs. In guanacos, juvenile males and females are weaned at approximately 7 months but begin nursing again when the next cria is born, continuing until evicted at 13–14 months of age. Thus, both juveniles and the crias of the year are present in a family group for a few months. It is thought that enhanced social maturity is fostered by this biologic strategy [1].

Wild Bactrian Camels

Wild Bactrian camels inhabit some of the most inhospitable areas of the world, with climactic extremes, sparse vegetation, and only salty water to drink. The males are highly territorial, controlling 12 ± females and their offspring. Males fight one another for breeding rights, and such fights may be vicious. Breeding and birthing is seasonal to coincide with the availability of forage for mother and calf.

Normal Reproduction

Male Llamas and Alpacas

Anatomy

The penis of the male SAC has a prescrotal sigmoid flexure (Figures 16.1 and 16.2) and (Figures 16.1 and 16.2a, b). The nonerect penis of a small llama, 135 kg (300 lbs.), may vary in length from 36 to 45 cm (14–18 in.), measured from the tip to the ischial arch. The diameter is relatively small, 0.8–1 cm (0.3–0.4 in.), at the region of the preputial reflection, increasing to 1.2–2 cm (0.5–0.8 in.) at maximum diameter near the ischial arch. The penis does not expand appreciably in diameter during erection but becomes firm and elongated. The erect alpaca penis varies from 35 to 40 cm (14–16 in.) in length [8].

In the adult male, the glans is long (9–12 cm) and tapers to the tip, merging into a firm cartilaginous projection that has a slight clockwise curvature (Figure 16.3). The distance from the tip to the preputial reflection is approximately 11 cm (4.3 in.). The penis penetrates the cervix and deposits semen into the uterine horns [9]. The cartilaginous tip may be an adaptation to facilitate threading the penis through the ringed cervix.

The prepuce of the SAC is triangular and non-pendulous. In an unaroused male, the prepuce is directed caudally,

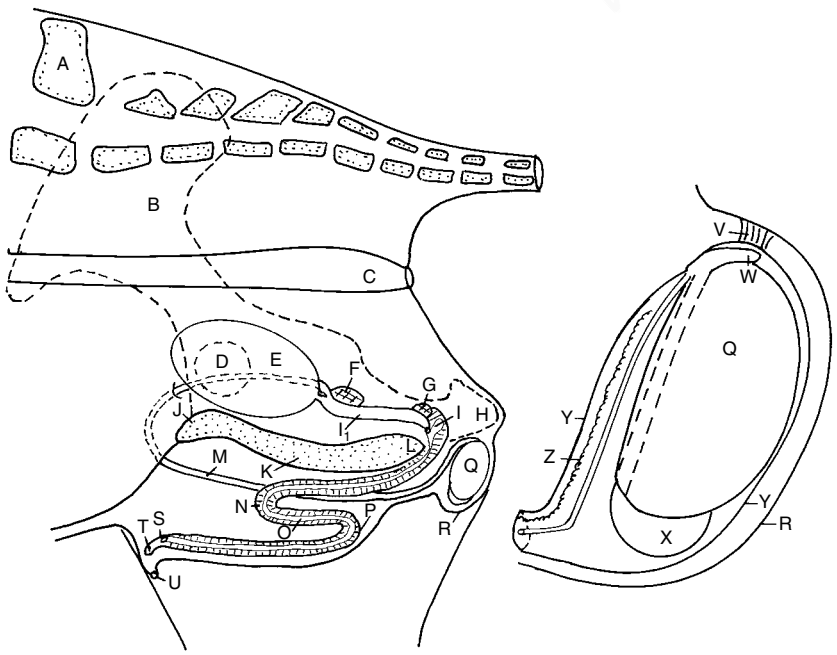

Figure 16.1 Diagram of lateral view of male camelid genitalia. (A) Spinal column, (B) ileum, (C) rectum, (D) acetabulum, (E) bladder, (F) prostate gland, (G) bulbourethral gland, (H) ischiatic tuberosity, (I) dorsal urethral recess, (I1) pelvic urethra, (J) brim of pelvis, (K) pubic bone, (L) ischiatic arch, (M) ductus deferens, (N) corpus cavernosum penis, (O) penile urethra, (P) sigmoid flexure of the penis, (Q) testicle, (R) scrotum, (S) urethral orifice, (T) cartilaginous tip of the penis, (U) prepucial orifice, (V) scrotal ligament, (W) tail of epididymis, (X) head of epididymis, (Y) tunica vaginalis communis, (Z) pampiniform plexus.

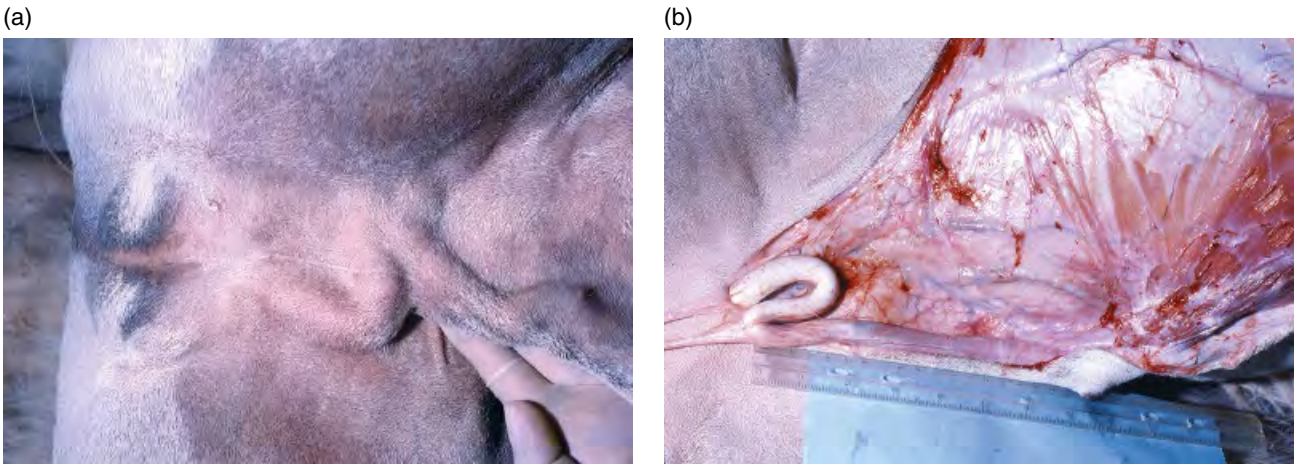

Figure 16.2 (a) Sigmoid flexure of a llama penis. (b) Sigmoid flexure of a llama penis.

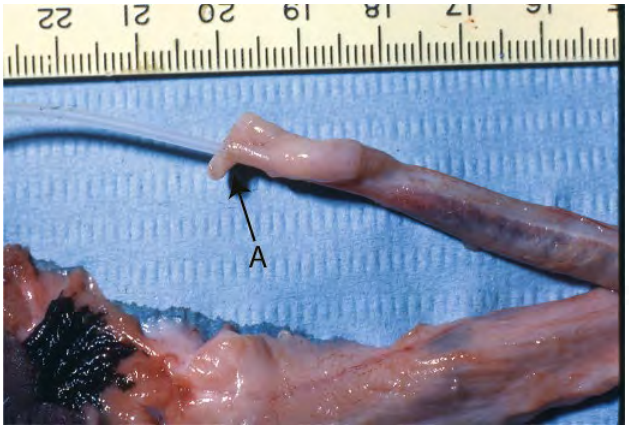

Figure 16.3 The tip of the llama penis has a slight clockwise curvature (B). The urethral opening is marked by the arrow labeled "A". A catheter has been inserted into the urethra.

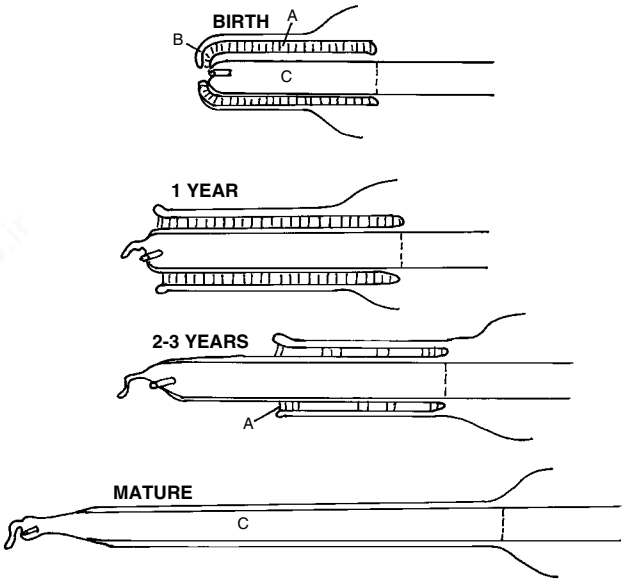

Figure 16.4 Diagram of preputial adhesions of the camelid. (A) Preputial adhesions, (B) prepuce, (C) penis.

and urine is projected backward between the hind limbs from a semisquatting position. When the male is sexually aroused, the cranial preputial muscles pull the prepuce cranially, and the penis is extruded in the same manner as in the bull. Movement is controlled by a double set of preputial muscles, cranial and caudal. Both arise from the cutaneous trunci muscle and lie on either side of the linea alba. These muscles are relatively larger in SACs than in domestic livestock.

The prepuce of the neonate and juvenile male adheres to the glans penis, making it impossible to extrude the penis from the prepuce until as late as two to three years of age (Figures 16.4–16.7). In a study of male alpacas, 8–12% were free of adhesions at one year of age, 70–78% were free at two years, and by the age of three, all were free of adhesions [8, 10]. When selecting future alpaca sires in Peru, early maturation is desirable, and yearlings free of adhesions are preferred. If a male is castrated prior to the onset of testosterone production, these adhesions may not be released or may be only partially released (Figure 16.8).

Urethral catheterization is difficult in the SAC male neonate because the glans cannot be extruded. Under sedation, the urethral opening may be protruded through the folds of the prepuce. In neonates, the cartilaginous process of the adult male glans penis is not present, and the tip of the penis is blunt. Attempts to peel the prepuce from the glans usually causes hemorrhage. Successful catheterization may be accomplished by gently probing for the urethral orifice with a number 3 French urinary catheter.

In the adult, the urethral opening on the glans is located at the base of the cartilaginous projection (Figure 16.3) and will accommodate passage of a number 5 or larger French

Figure 16.5 Inability to protrude penis, three-month-old alpaca.

Figure 16.6 Preputial adhesions of a yearling llama.

Figure 16.7 Adhesions of prepuce to the glans penis.

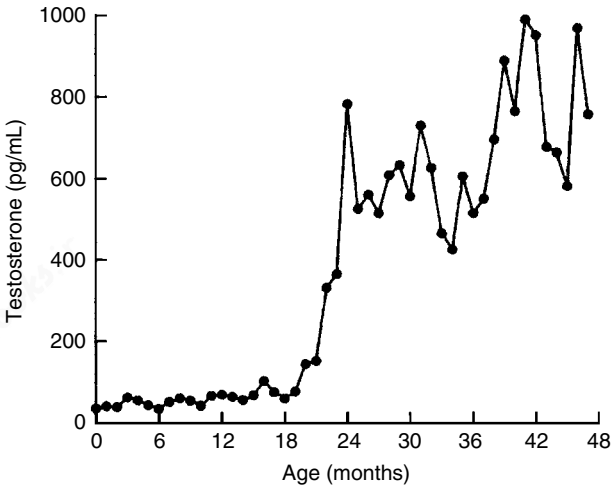

Figure 16.8 Testosterone concentration in the male llama from birth to maturity [11].

catheter. The pelvic urethra is approximately 7 mm in diameter, narrowing as it enters the penile segment. The urethra narrows further at approximately the level of the preputial reflection. Uroliths may lodge at this site.

It is impossible to pass a catheter through the tip of the penis into the bladder because of a dorsal urethral diverticulum just cranial to the ischial arch because the catheter would automatically be directed into the diverticulum (Figure 16.9). Even with a finger inserted into the rectum to deflect the catheter ventrally, bypassing this recess would be strictly fortuitous.

Accessory sex glands are limited to a pair of bulbourethral glands and a small prostate (Figure 16.10). There are no seminal vesicles. The bulbourethral glands lie dorsally and laterally to the pelvic urethra at the ischial arch and are approximately 2 cm (0.8 in.) in diameter. The prostate gland is small (3 × 3 × 2 cm) and situated on the dorsum of the urethra near the trigone of the bladder.

The retractor penis muscles arise from the anal sphincter muscle and pass around the ischial arch in close association with the body of the penis. In a perineal urethrostomy, the paired muscles must be parted or pulled to one side to enhance exposure of the urethra. The distal insertion of the muscle on the tunic of the penis is at the level of the preputial reflection.

The testicles of llamas and alpacas are roughly ovoid (Figure 16.11), but a few may be globose. There is great variation in the size of the testicles [12]. The ultrasonographic anatomy of the testicles is important because this technique is used daily and it provides a clear view of the testicular parenchyma. The testicular width and depth may be adequately measured, but not so the length because the length of the probe may not be enough to cover the whole testicle. The most important feature to look for is the echogenecity of the parenchyma. Figure 16.5 depicts a transversal view of a normal testicle. The parenchyma's appearance is homogenous and hypoechoeic without any hyperechoeic and nonechoeic spots. It is useful to move the ultrasound

Figure 16.9 Radiograph of male urethra containing contrast medium. Notice the dorsal urethral recess at the ischial arch.

Figure 16.10 Male camelid genitalia. (A) prostate gland, (B) bulbourethral gland.

Figure 16.11 Measuring the length of a testicle.

probe along the length of the testicle to see any variation in echogenicity. This sequence gives a better assessment of the testicular parenchyma. In a longitudinal view the testicular mediastinum may be clearly assessed and observed. Similar to cattle, testicular size influences fertility of the male SAC (Table 16.1). Forty-five percent of females became pregnant when bred to males with small testicles, in contrast to 75% of pregnancies in females resulting when bred to males with normal size testicles [14]. In Peru, future alpaca sires are selected at one year of age. The length of the testicles at this time ranges from 1.1 to 1.4 cm. The males with larger testicles are preferentially chosen.

The non-pendulous scrotum is situated just ventral to the ischial arch (Figure 16.12a and b). The testicles and scrotum protrude only slightly from the surrounding surface. Having the testicles closely apposed to the body may

Table 16.1 Mean testicular size (length × transverse axis) and weight in llamas, alpacas, and vicuñas of different ages (n = 158 alpacas, 54 llamas, 6 vicuñas).

Age (months)	Llama [11]		Alpaca[a]		Vicuña [13]
	Size (cm)	Weight (g)	Size (cm)	Weight (g)	Size (cm)
6	2.4 × 1.4	na	na	na	na
12	3.4 × 2.3	5.1	2.3 × 1.5	2.9	na
18	3.5 × 2.6	14.0	2.8 × 1.9	6.6	na
24	3.9 × 2.3	17.4	3.3 × 2.2	9.9	na
30	4.4 × 2.5	17.8	3.6 × 2.3	13.9	na
36	4.5 × 2.7	18.2	3.6 × 2.4	13.6	na
Sires	5.4 × 3.3	na	3.7 × 2.4	17.2	3.3 × 1.9

na = not available.
[a] Bravo, unpublished data.

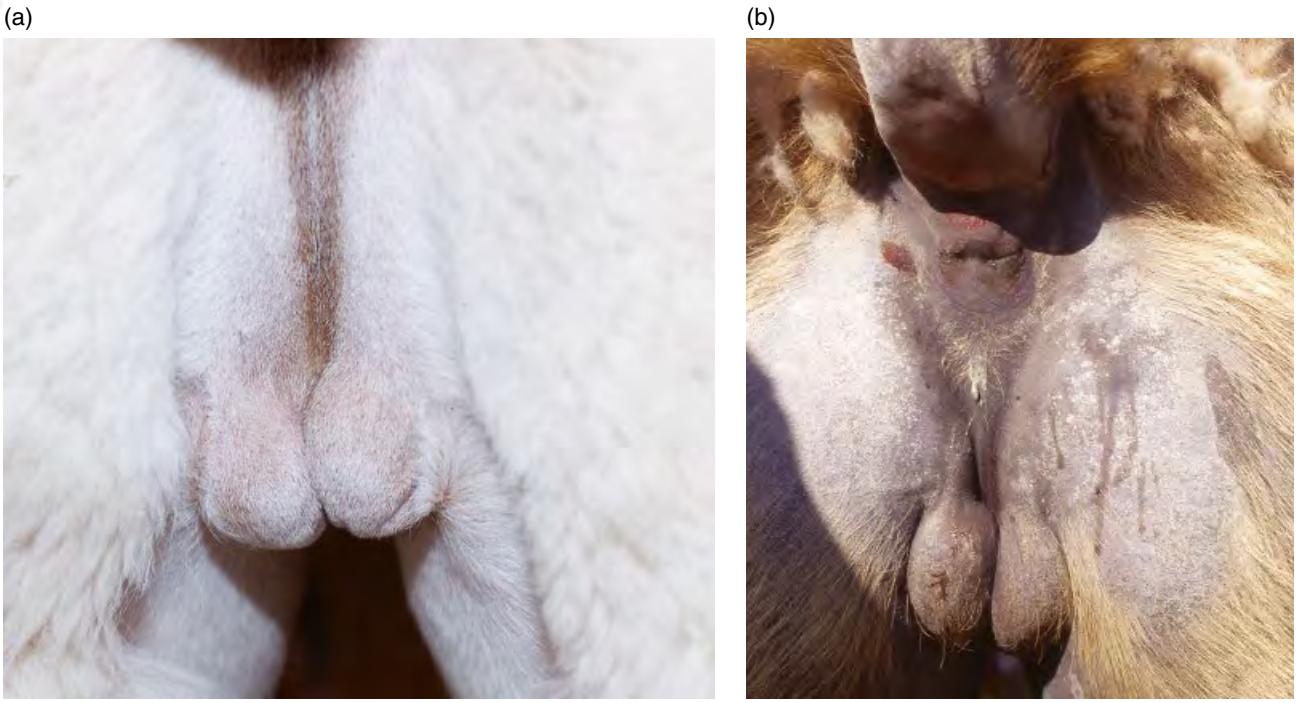

Figure 16.12 (a) Scrotum of an alpaca. (b) Scrotum of a camel.

be an important adaptation to prevent accidental castration during male–male fighting. Also, the skin over the scrotum is thick, providing additional protection for the testicles. Scars are often seen on the scrotum, a result of lacerations from the bites of other males.

The testicles are usually in the scrotum at birth but are tiny, flabby, and difficult to palpate. Adult testicles should be turgid, not mushy, and freely moveable within the scrotum. The epididymis is closely attached to the testicle, and the head, body, and tail can be palpated after some practice. The head of the epididymis is cranial-ventral and the tail caudal-dorsal as it is in the boar but not in the bull, ram, and stallion, where the head of the epididymis is dorsal. The epididymis should be resilient, with no nodules.

The microscopic characteristics of the testicles and epididymis are essentially the same as for other livestock species [15]. The diameter of seminiferous tubuli is between 174 and 237 μm [15, 16]. The shape of SAC spermatozoa is similar to that of livestock. Characteristics of SAC semen, from some limited studies, are noted in Table 16.2 [17]. The cycle of the seminiferous epithelium for the male llama has been described as eight stages with relative frequencies as 9.8, 12.5, 17.7, 14.1, 5.8, 8.1, 13, and 18.9%, respectively [18].

Table 16.2 Semen characteristics of llamas and alpacas [17].

Characteristics	Llama	Alpaca	Range
Volume (mL)	1.6 ± 0.8	1.9 ± 0.4	0.8–3.1
Concentration (number/mm^3)	1 388 000 ± 54 000	147 500 ± 7894	82 500–250 000
Total concentration (millions)		289 000 ± 15 200	61 785–750 000
Motility (%)	61.7 ± 23.5	85 ± 5.2	69.0–91.1
Live sperm (%)	82.7 ± 7.3	69.6 ± 3.7	58.0–83.1
Normal sperm (%)	na[a]	75.9 ± 2.1	70.6–84.1
Abnormal heads (%)	na	6.7 ± 1.4	2.6–12.6
Abnormal tails (%)	na	12.3 ± 0.8	9.4–15.2
Cytoplasmic droplets (%)	na	3.8 ± 0.8	1.3–7.3

[a] na = not available.

Physiology

To understand SAC breeding behavior and physiology, the general biology of the wild species should be reviewed (also see Chapter 1). Wild SAC males are basically territorial polygamists [1]. A successful breeding male will defend a territory and a set number of females against all intruders, male or female. Fights between males may be vicious. Though domestication of the llama and alpaca has diminished territorial behavior, if alpaca or llama males are kept near vicuñas, they revert to wild behavior and become difficult to manage as a domestic herd.

Testicular Function

The testicle has two functions: spermatogenesis and hormone production. As in other mammals, testosterone is responsible for the development of secondary sexual characteristics such as greater muscular mass, appearance, and libido. Testosterone concentrations in the male llama and alpaca have been determined from birth to adulthood (Figure 16.8) [11]. Basal concentrations of testosterone are observed from birth up to 19–20 months of age, after which testosterone levels increase rapidly, reaching adult values when males are approximately 2 years old. This increase in testosterone coincides with exacerbated sexual libido in male llamas and alpacas.

When male alpacas were separated from females as a management practice, testosterone concentration dropped to 3900 pg/mL in contrast to 9000 pg/mL during the breeding season. Similar observations were made for male vicuñas: 288 pg/mL of testosterone in the nonbreeding season in contrast to 1009 pg/mL in the breeding season [13]. This apparent change in testosterone concentration has not been observed in the United States in llamas maintained year-round with females. Testosterone concentrations fluctuated between 900 and 1200 pg/mL throughout the year [11].

Assessment of spermatogenesis is difficult. When SAC semen contacts air, it is converted to a gelatinous mass, which defies attempts to produce a satisfactory smear for evaluating motility and morphology. It is now known that the gel appearance of semen is due to the presence of a thick gelatinous material secreted mostly by the bulbourethral glands. Several protocols for semen collection have been devised; the most successful and suitable for clinical purposes is collecting it from the vicinity of the external cervical os of the female after breeding has taken place. For research purposes, the most adequate method of semen collection is using an artificial vagina (AV) that has been specifically modified for camelids. Briefly, this modified AV has two important characteristics: a stricture on the latex liner simulating the cervix and an electric heating pad to maintain liner warmth for the length of copulation [17].

Semen is whitish-cream in color, becoming reddish when collected after copulation from the female, with a pH between 7.2 and 7.8. Most of the biochemical components of the seminal plasma of alpacas [19] and llamas are similar to those of other livestock species, with the exception of small amounts of fructose (5 mg/dL) and citric acid (4.3 mg/dL), perhaps as a result of the absence of seminal vesicles. Fructose concentrations did not vary among semen samples collected from February through September [20, 21], nor between males of three and six years of age. Semen characteristics of alpacas and llamas appear in Table 16.2 [17].

Ovulation does not take place for at least 24 hours after copulation in SACs. The gelatinous semen mass probably provides some protection to the sperm until liquefaction of semen takes place. It is not known when liquefaction (liquefaction is the process of a solid becoming liquid, and since ejaculated semen is not solid, rather a gel, the correct term should be degelification) takes place *in vivo*, but *in vitro* semen samples begin to liquefy eight hours following collection. The influence of season on seminal production has been partially defined in llamas. Male alpacas lose libido when maintained in continuous association with females. Also, in the northern hemisphere, sperm production in llamas and alpacas decreased to negligible values after undergoing heat stress during the summer, then later returned to normal. Normal spermatozoa morphology was around 74% during spring in contrast to 24% in summer, rising again to 71% during the autumn. In addition, the proportion of abnormal spermatozoa increased during a period of approximately one to one and a half months. Thus, a temporary loss of fertility during the summer may be normal.

Sexual Behavior

Male llama crias (neonates is a term only for crias for the first week) begin play fighting at a few weeks of age (Figure 16.13). Some precocious weanlings will rear up on females and, if they can catch a nonpregnant female lying down, may attempt to mount her. Weanling males and females should be kept separate from each other because, in a few instances, males less than one year of age have impregnated females. Llama breeders in North America may tend to push young males into breeding too soon. Some individuals are anatomically capable of breeding when they are one and a half to two years old, but until they are three years old, most individuals lack sufficient libido and necessary body weight to force the female down and to breed vigorously. In some very young males, the testicles do not produce viable sperm, and there may still be preputial adhesions that prevent full extension of the penis.

Figure 16.13 Young male llamas play fighting.

Guanaco males do not become territorial until they are four to six years of age [1]. This does not mean that male guanacos are physically incapable of breeding earlier than this, but social and behavioral restrictions prevent earlier breeding.

In Peru, male alpacas are put into breeding service at three years of age [22]; however, it would be unwise to pronounce a male llama or alpaca as infertile (precluding any anatomic defects) before the age of three and a half to four years.

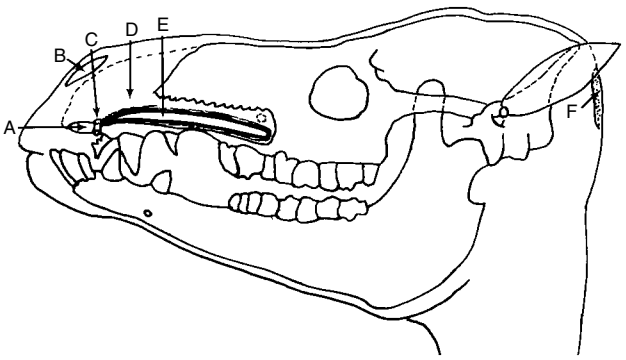

Figure 16.15 Diagram of the vomeronasal organ in a camel. (A) Palate, (B) opening to the vomeronasal organ, (C) nostril, (D) nasal septum, (E) Vomeronasal organ, (F) poll gland.

Both male and female (to a lesser extent) camelids exhibit flehmen, but in a slightly different manner than other mammals (Figure 16.14a and b). Many males approach a dung heap recently used by a female. He sniffs the area and then lifts his head and elevates the upper lip and opens the mouth slightly. The upper lip does not curl back as it does in other artiodactylids. Some, but not all, males sniff the perineal area of the female. The volatile pheremone enters the duct of the vomeronasal organ for evaluation (Figure 16.15).

(a)

(b)

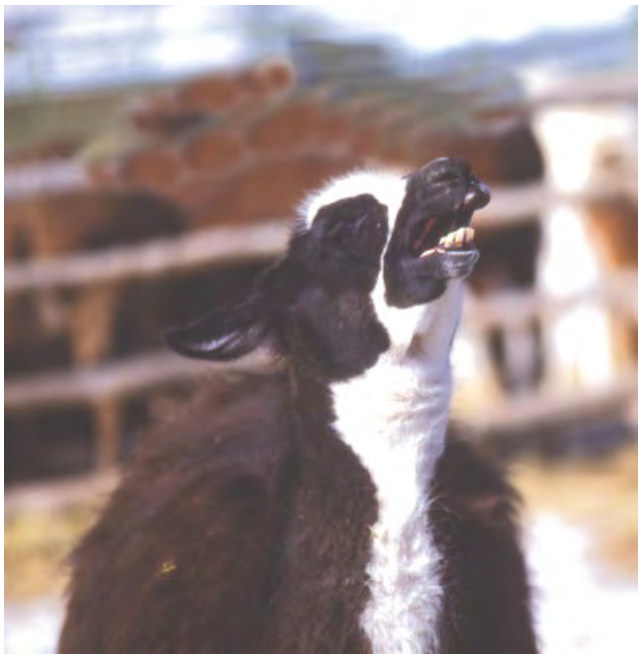

Figure 16.14 (a) Alpaca male exhibiting flehmen. (b) Llama exhibiting flehmen.

The usual breeding behavior of a male SAC placed in the same enclosure with a receptive female begins with pursuit of the female. Some females become recumbent immediately, but most move away from the male until he rears up and puts pressure on the hindquarters in an attempt to force the female to lie down (Figure 16.16a and b). He may do this repeatedly if she continues to move away. The rearing is not an attempt to copulate but merely to force the female to lie down. The size and body weight (not fat) of the male is important in successful copulation, as is the libido of the male. A male with good libido will chase a female for at least five minutes before giving up.

Once the female is recumbent, the male positions himself at her rear, in a half-sitting position, and begins intromission (Figures 16.17–16.19). The pelvic thrust of SACs is not as vigorous as it is in other domestic livestock. It may be difficult to ascertain whether or not intromission has actually occurred unless the penis is palpated or the fiber coat (wool) is pulled to the side to allow observation. With some experience, it is possible to perceive whether or not the male is in proper position. The male may be too far behind the female's pelvis. This is often observed when the male first mounts a recumbent female. At this stage, erection begins, and the tip of the penis may be visible. As the male begins to adjust his position by shifting from side to side or forward, working closer to the female, erection is completed. The perineal area of the female will be probed with the penis, almost as if the male is exploring to find the vulva. Upon close observation of the penis at this stage, it should be noted that the glans may alternately assume a corkscrew and a straight shape. This alternate configuration of the glans also occurs during electroejaculation and should not be construed as penile malformation. Perhaps this shape changing aids in penetration of the vulva and in threading the penis through the cervix.

When intromission has occurred and effective copulation is underway, the male will be positioned with his pelvis close to the pelvis of the female, and the hocks of the male are lateral to the hocks of the female. A practical approach to assess penis intromission into the genital tract of females is following the penis of the male and if it is positioned into the vulva of the female. This is done from behind the animals copulating. It could be done from the side, but the presence of the legs of the male and his heavy coat make it more difficult to evaluate than from behind.

The male constantly vocalizes during copulation with a guttural sound that is called "orgling."[1,2] The exact source of the sound is unknown, but it may be the result of vibrations of the relaxed, elongated soft palate as air rushes past during rapid breathing.

Copulation may continue from 5 to 50 minutes, with an average time of 20–30 minutes. The elapsed time of copulation and conception rates have not been correlated. It is impossible to ascertain exactly when ejaculation occurs, because ejaculation is continuous from two to three minutes after intromission until the end of copulation. There is no tail flagging, as in the stallion, or hyperpelvic thrust, as in the bull. It is known however, that ejaculation is a continuous (pulsating ejaculation). It is common to see the male reposition himself during copulation by moving

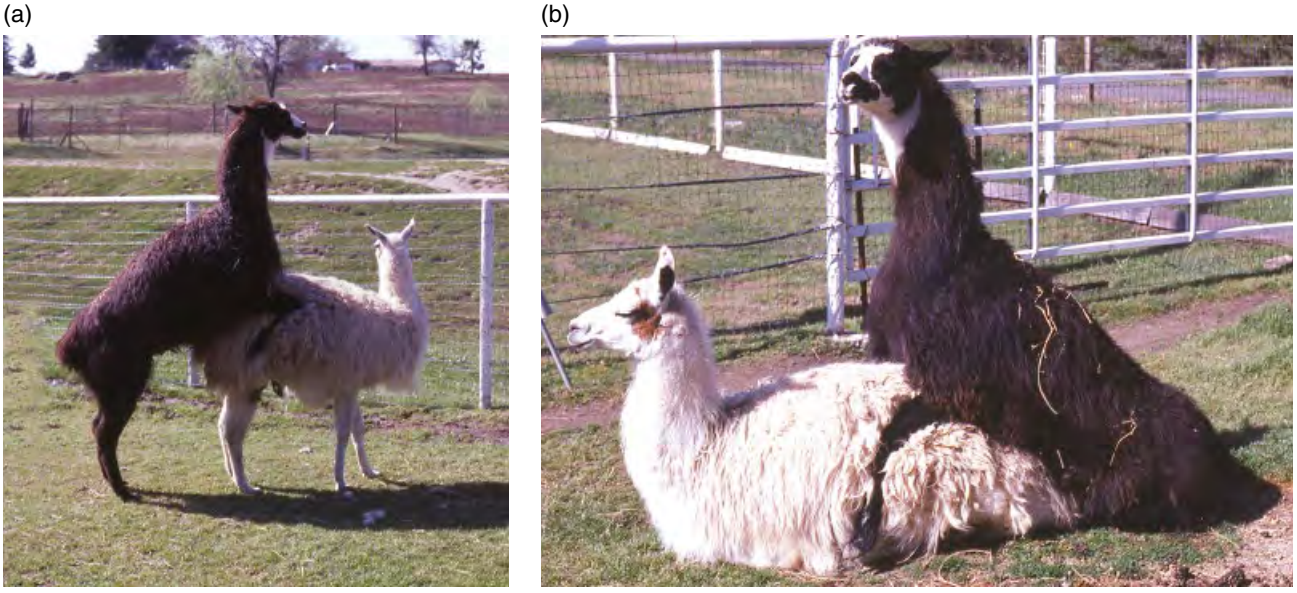

Figure 16.16 (a) Male llama attempting to force a female to sternal recumbency prior to copulation. (b) Beginning copulation, not close enough for intromission.

Figure 16.17 Proper breeding position in alpacas.

Figure 16.18 Inexperienced llama male attempting to breed a female.

slightly backward, then shifting right or left before moving forward again. It is probable that he is changing the position of the penis from one uterine horn to ejaculate into the other. It has been demonstrated that the penis reaches the uterine horns during copulation [9].

Occasionally, a male will cease copulation for no apparent reason, stand up, and begin the positioning sequence once again. When copulation is completed, the male rises and wanders off. The penis will have been retracted into the prepuce. The female may remain recumbent for a few minutes or may also rise and move away.

If the male and female are left together, copulation may be repeated the same day but usually not until the next day, and perhaps it will be repeated on a third day. If ovulation has occurred, the female will refuse to lie down and will spit at the male to discourage him. This is called "spitting off."

Figure 16.19 Camels breeding.

If other receptive females are in the enclosure, a vigorous male may breed as many as five or six females in one day. Reports from Peru indicate that a vigorous alpaca male may breed as many as 18 times in 1 day, for the first 3 days of the breeding season, but this activity quickly drops off on subsequent days, and if the male is left with the females for 10–14 days, he will ignore newly receptive females [8]. The length of copulation decreases when males are subjected to consecutive breeding on the same day. Males will copulate for 16 minutes for the first two or three matings, in contrast to 12 minutes for a fourth mating and 10 minutes for a sixth [23]. In addition, the length of copulation also decreases on consecutive days of breeding. Males copulate for 17 minutes on the first day, 13 minutes on the third day, 11 minutes on the seventh day, and six minutes on the eighth and ninth days of breeding. The number of copulations affects the fertility rate of the male: the percentage of pregnant females was 77% for males copulating two to four times a day, in contrast to 59% for males copulating six times a day. Likewise, the fertility of the male decreases with the progressive number of days of breeding: 88% of females became pregnant on the first day of breeding, 84% on the third day, 71% on the seventh day, and no pregnancies resulted from breedings on the ninth day.

A male SAC may take a dislike to a particular female and refuse to breed her. He may even become somewhat aggressive with her. Varying degrees of aggressiveness have been observed in breeding males. Some will even "rape"

pregnant females. Others are so shy that they almost have to find a female lying down before they attempt to copulate. Shy males may be intimidated by dominant females. Libido may be an inherited trait, so it may be undesirable to use a shy breeder.

Intermale aggression can be violent and damaging to both combatants, especially if each has a full complement of canine teeth. The objective of the fight is to establish dominance. The males may run at each other, rear up, and ram chests, each trying to knock the other off balance. If an advantage has been gained, the stronger will try to position himself to the side, with his neck over the neck of his opponent, in an attempt to force him to the ground. If the weaker animal cannot dislodge the stronger, he may reach around and bite the leg of his opponent. This "necking" may continue for some time, with each alternating at being the stronger.

Males may position themselves side by side in a head-to-tail position. Each male may try to bite the other on the hind legs and the scrotum. As one male is being bitten, he may lean into the aggressor and almost sit down on the aggressor's head. Severe wounds may be inflicted by intact canine teeth.

Particularly aggressive males may charge from the side in an attempt to knock an opponent down or rear up and come down on the body to force recumbency. They may also bite at the neck, ears, or jaws of an opponent. During the fight, the animals may constantly spew stomach contents at one another.

The interaction is over when one of the males remains recumbent in a subordinate posture. Both males will stand with their mouths partly open for several minutes after breathing has returned to normal (Figure 16.20). This is not a form of open mouth breathing, necessary to increase air flow into the lungs, but rather a behavioral trait exhibited after any aggressive or disagreeable interaction. The authors have also observed this behavior after a strenuous restraint episode.

Figure 16.20 Open mouth stance (pouting) assumed by male llama after aggressive encounter with another male.

The anatomy and basic reproductive physiology of the male camel is similar to that of New World Camels (NWCs) [24, 25]. However, both Bactrian and dromedary male camels undergo a seasonal rut period characterized by heightened aggression, secretion from the poll glands, dribbling of urine, flicking of the tail to splash urine onto the back, and vocalization. Weight loss is usual because the male expends so much energy in establishing and defending his harem. Inappetence may also be a factor in weight loss. Rut may last for weeks or months.

The poll gland is a scent gland that is unique to OWC males. The gland secretes a brownish to black fetid fluid that mixes with dirt and urine during rut to mat the hair on the back. If allowed to do so, a male camel in rut may roll and attempt to rub the poll gland against the dirt to scent-mark. The testicles increase in size during rut, accompanied by an increased testosterone level in the blood.

Male dromedary camels have a diverticulum on the lingual aspect of the soft palate (dulaa, dulla, dulah) which may be inflated with air and be extruded from the oral cavity during rut. The camel produces a gurgling sound accompanied with excessive salivation and foaming. This behavior is a threat to other males. The dulaa may be protruded at times other than rut, when the camel is excited or angry. It may also come out of the mouth during anesthesia induction and may cause alarm to the novice camel anesthetist. At such time, the dulaa may simply be pushed aside and intubation completed.

Sexual behavior may be evident in dromedary males at two years of age, but puberty usually does not occur until the animal is two to five years old. Dromedary males are usually not used for breeding until they are five years of age. Breeding may continue for ± 20 years.

Bactrian males mature more slowly, with sexual behavior beginning at three years of age and puberty at five to six years of age. Copulation is in the recumbent position as in SACs. In dromedaries, the duration of copulation may be 7–22 minutes; in the Bactrian, the duration is only an average of 3 minutes. In contrast, alpacas copulate for 5–50 minutes and llamas for 5–65 minutes.

Camel semen is similar to that of SACs and is white and creamy to grayish. Camel semen develops the same gelatinous consistency shortly after ejaculation as does that of the SAC. The volume of semen produced varies from 1 to 15 mL. Semen pH is alkaline (7.6–8). Motility varies from 40 to 80%.

Diseases of the male camel are similar to those seen in SAC males. Specific conditions include scrotal laceration, orchitis [26], testicular hematoma, testicular hypogenesis, cryptorchism [27], neoplasia, and epididymal cysts.

Female Llamas and Alpacas

Anatomy [28–31]

See Table 16.3 for a comparison of measurements of llama and alpaca reproductive organs.

Table 16.3 Measurements of female SAC reproductive organs.

	Llama[a] (cm)	Alpaca (cm) [32]
Vulva		
Length of labia	5	
Depth of hymen	6–8.5	
Vagina		
Hymen to cervix		
Length	15–21	13.4 ± 2
Diameter	5	3.4 ± 0.7
Cervix		
Length	2–5	
Diameter	2–4	
Rings	2–3	2–3
Body of uterus		
Length to septum	3–5.5	3.05 ± 0.71
Diameter	3–5	
Horns of uterus		
Tips of septum to tip of horn	21–22.5	
Bifurcation to tip of horn	8.5–15	7.9 ± 1.3
Diameter	2.5–4	
Oviduct	10.5–18.3	20.4 ± 4.2
Ovary		
Right		
Length	1.3–2.5	1.6 ± 0.3
Depth	1.4–2	1.1 ± 0.2
Width	0.6–1	1.1 ± 0.2
Weight		1.87 ± 0.94 g
Left		
Length	1.5–2.5	1.6 ± 0.3
Depth	1.5–2.5	1.1 ± 0.2
Width	0.5–1	1.1 ± 0.2
Weight		2.4 ± 1.34 g

[a]Measurements collected at University of California, Davis.

VULVA. The labia of the vulva lie in a slightly slanted to vertical position approximately 4–6 cm ventral to the anal orifice (Figure 16.21). In aged or emaciated llamas, or those with congenital malformations of the reproductive tract, the labia may lie in a more horizontal plane (vulvar shelving) (Figure 16.22). The vulvar orifice (a slit) is approximately 3–5 cm long. The labia do not swell during the reproductive cycle. The depth of the vestibule (vulva to the hymen) is approximately 3–4 cm.

VAGINA. The vagina, from the hymen to the cervix, varies in length from 15 to 25 cm (6–10 in.) and is approximately 5 cm in diameter.

Figure 16.21 Normal llama perineum. The labia of the vulva lie in a slightly slanted to vertical position approximately 4–6 cm ventral to the anal orifice.

Figure 16.22 Perineum of llama with abnormal, near horizontal relationship of vulva to anus (shelving).

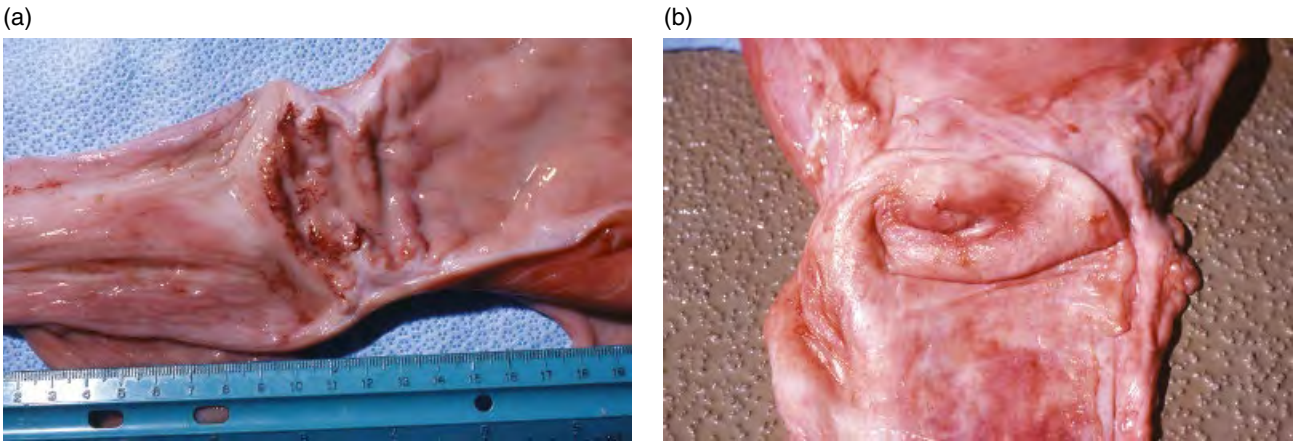

Figure 16.23 (a) Llama cervix showing the rings. (b) External cervical os.

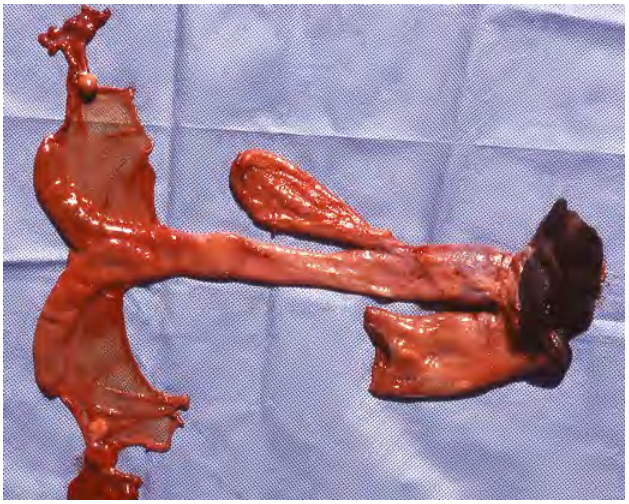

Figure 16.24 Female reproductive tract of a nulliparous alpaca.

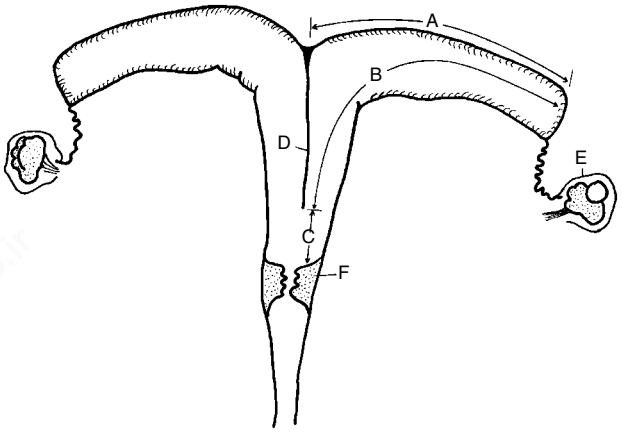

Figure 16.25 Diagram of camelid female reproductive tract. (A) Palpable length of uterine horn, (B) actual length of uterine horn, (C) body of uterus, (D) intercornual septum, (E) ovarian bursa, (F) cervix.

CERVIX. The llama cervix is 2–5 cm long and 2–4 cm in diameter. The cervix has two to three ring-like structures (Figure 16.23a and b). The cervix relaxes with estrogen stimulation and opens for several days, then closes and remains closed, which precludes the insertion of uterine catheters and or insemination pipettes.

UTERUS. The SAC uterus is bicornate. The body of the uterus is short (3–5.5 cm) and approximately the same diameter throughout. Externally, the length is not apparent because the horns are fused together for a short distance, being separated by a septum (Figures 16.24–16.26).

The left uterine horn may be slightly larger than the right, especially after the first pregnancy. The uterine horns of a llama, from the end of the septum to the tip of the horn on the convex surface, vary from 20 to 22.5 cm in length; in alpacas, the length is 6–7 cm. The proximal segments of the

Figure 16.26 Uterus and adnexa of a multiparous llama. (A) ovary encased with the ovarian bursa, (B) left uterine horn, larger than the right, (C) cervix, (D) mesosalpinx.

horns diverge at approximately 180°. The distance from the notch at the divergence to the tip of each horn varies from 8.5 to 15 cm (3.3–6 in.). The diameter of the horns varies from 2 to 3 cm with age and the number of previous pregnancies. The tip of the uterine horn is rather blunt.

Uterine mucosa is similar to that of other species, consisting of a columnar epithelial layer. The fibrous tissue of the submucosa is more dense than in the mare, and there are fewer and smaller uterine glands [33]. There are no glands in the cervical submucosa, and the epithelial layer is more cuboidal. This information is necessary to enable differentiation of the site from which a biopsy specimen has been taken.

The penis penetrates the uterine horns. Thrusting during copulation causes mechanical friction and may initiate an inflammatory response in the uterine endometrium, including hemorrhage and edema in some cases [34]. This inflammatory reaction is normal, but if a female is bred more than once during a follicular phase, the damage to the uterus may be correspondingly greater. Histologically, there may be lymphocyte infiltration, plus plasmocytes and eosinophils. Increased branching of the endometrial glands of the endometrium may also be observed.

OVIDUCTS. The oviducts are rather long and tortuous and are embedded within the mesosalpinx. The duct measured 10.5 cm in one llama, with a diameter of 3 mm. Bravo and Sumar measured the ducts of alpacas at 20.4 ± 4.2 cm [35]. The oviduct empties into the tip of the uterine horn on a papilla that contains a sphincter (Figure 16.27) [8]. It is impossible to flush fluid retrograde into the oviduct from the uterus, but the reverse is possible.

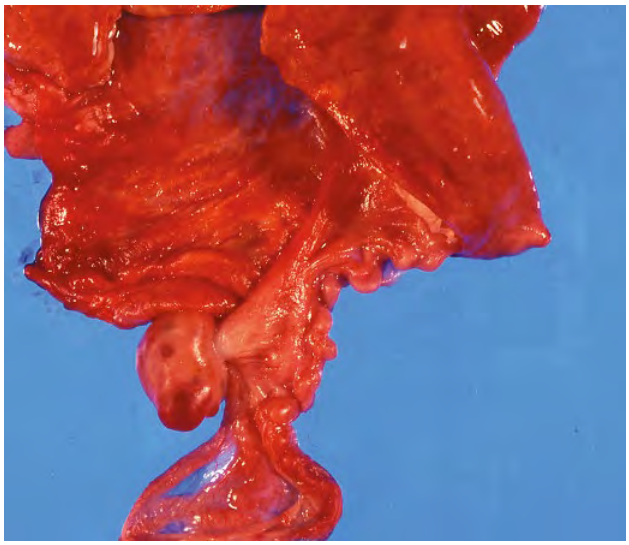

Figure 16.27 Tip of uterine horn everted to expose oviduct papilla.

The ovarian bursa is large (2.5 × 2.5 × 5 cm) and completely surrounds the ovary. Ovarian structures must be palpated through the bursa, or the bursa may be gently moved to expose the surface of an ovary. At laparoscopy, the bursa is lifted off the ovary with forceps.

OVARY. The ovaries of the llama are ellipsoid to globular, while those of the alpaca are more globular. Numerous follicles, varying in size from 2 to 10 mm, can be seen on the surface of the mature, normal ovary (Figures 16.28, 16.29a and b) [28]. A follicle ready for ovulation may reach 12 mm. Any follicle larger than 13 mm is considered to be pathologic [8, 36, 37]. Measurements of the ovaries from six llamas were as follows: length, 1.3–2.5 cm.; depth, 1–2 cm; and width, 0.5–1 cm. These measurements may be compared with those of alpacas (Table 16.3). Llama ovaries have not been weighed, but alpaca ovaries weigh approximately 2 g. A composite of sonograms of the reproductive organs of a female alpaca has been assembled to give the reader a perspective of the organs visualized using ultrasonography (see Figure 16.51 later in this chapter).

Puberty

The assessment of puberty in female SACs is difficult because of the lack of an estrous cycle. Nonetheless, some progress has been made in assessing onset of sexual maturity based on body weight and ovarian activity. Female alpacas 12–13 months of age with body weights of 33 kg successfully delivered and cared for crias. Under South American conditions in which nutrition is based on native pastures, 60% of yearlings attained weights of 33 kg, and of these, 70% conceived following copulation. At the next breeding season, there was no difference in the body weights of these females compared with those

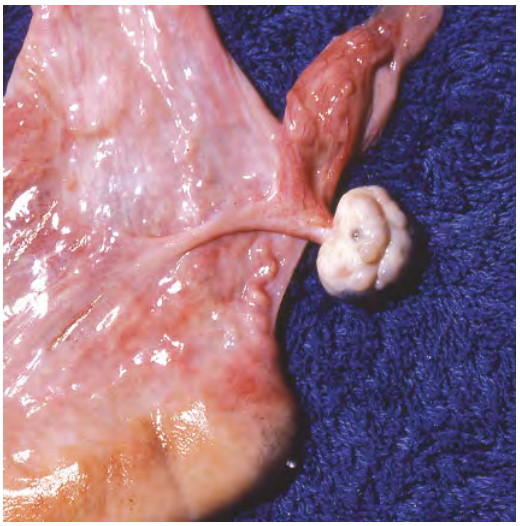

Figure 16.28 Inactive llama ovary.

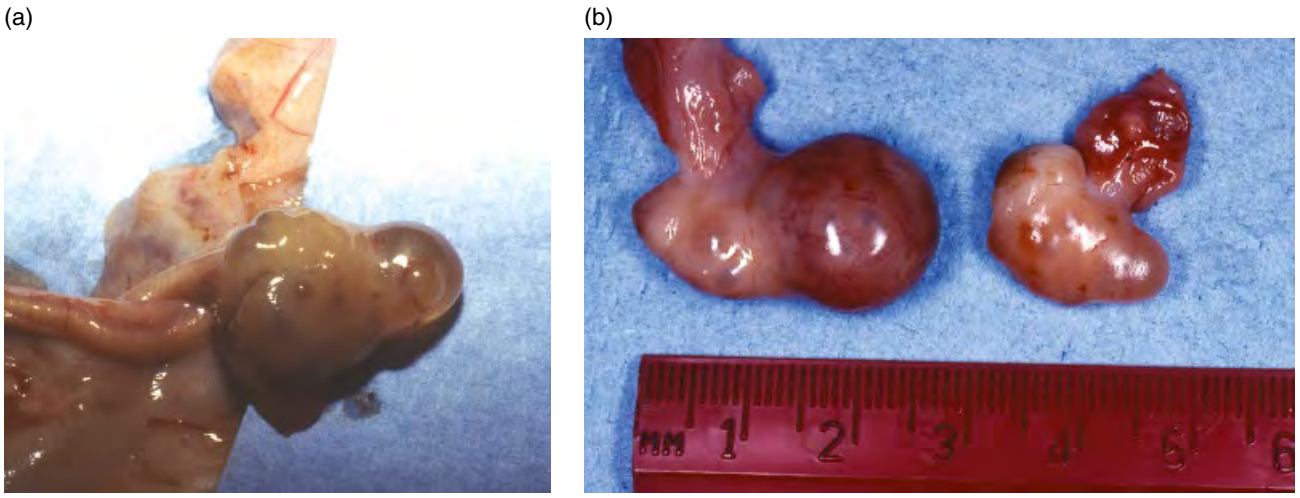

Figure 16.29 (a) Llama ovary with an ovulatory follicle. (b) Llama ovary with a corpus luteum.

of older females. Percentages of animals reaching maturity based on body weight may be greater where cultivated pastures and supplementation provide better nutrition. Yearlings did not differ from adults in sexual behavior.

Investigations of ovarian activity began with alpacas at 10 months of age [38]. Laparoscopic studies of ovarian follicular development showed little difference between yearlings and adults: 65 follicles 6 mm in size were counted in yearlings, and 80 mature follicles in adults.

Follicle development may begin earlier than 10 months. When follicle activity was monitored by measuring concentrations of estrone sulfate in urine samples collected weekly from female llamas, beginning with youngsters at five months of age, intermittent increases in estrone sulfate were apparent in the majority of females at six months of age [39].

Physiology

The endocrine relationships to the female reproductive tract are complex. The miracle is that in the vast majority of instances the correct correlation occurs (Figure 16.30).

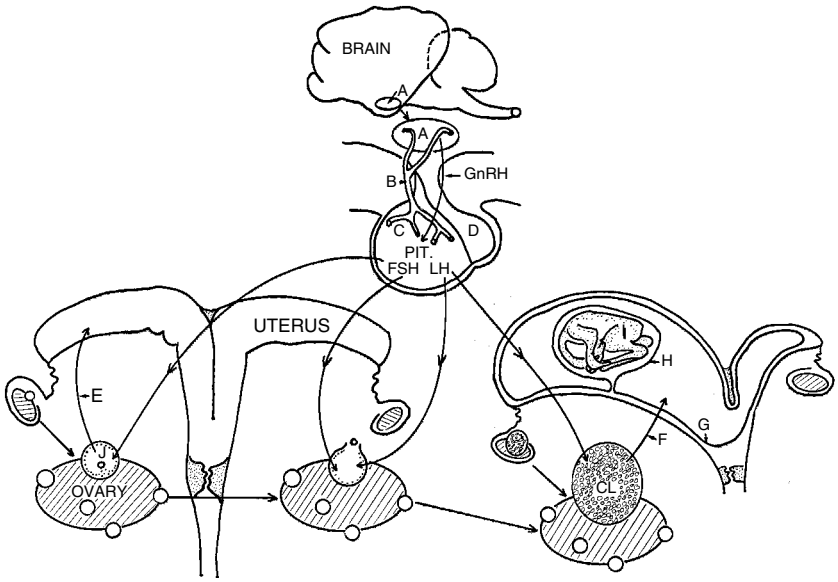

Figure 16.30 Diagram of the endocrine relationships of the female reproductive tract. (A) Hypothalamus, (B) blood flow from the hypothalamus to the pituitary, (C) anterior pituitary, (D) posterior pituitary, (E) estrogen stimulation of the uterus, (F) progesterone maintaining pregnancy, (G) chorioalantois, (H) amnion, (I) fetus, (J) ovarian follicle, (CL) corpus luteum.

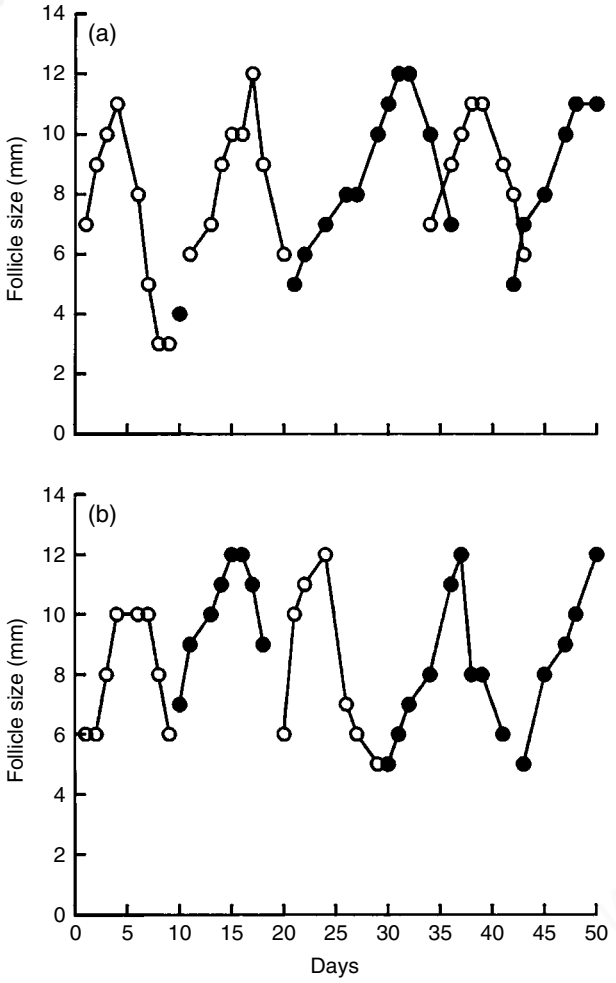

Figure 16.31 Ovarian follicle size in llamas (a) and alpacas (b) for a 50-day period without contact with a male. The open and shaded circles indicate follicles from different follicular waves. *Source*: Used by permission from W.B. Saunders Company.

Camelids have a unique ovarian cycle; they are induced ovulators but their hormonal physiology differs from that of other induced ovulators such as the rabbit or cat [30, 40]. The typical follicular phase of the ovarian cycle is charted in Figures 16.31 and 16.32.

The ovarian cycle of a noninduced ovulation species is illustrated in Figure 16.33. Unlike these species, SACs do not have an estrous period. Females are generally receptive to a male unless they have been recently bred or are pregnant. Camels are also induced ovulators and have follicular waves, but the wave interval is longer, so the waves do not overlap to produce estrogen, which would signal constant receptivity (Figure 16.34). Thus, camels exhibit periodic receptivity that appears to be an estrous cycle; however, the follicular dynamics are markedly different from those of a noninduced ovulator species [24, 41].

Follicular development in llamas and alpacas is similar [39, 40, 42–44]. Follicles grow and regress in an average time of 10–18 days, with ovaries alternating in 85% of the cases in which the presence of mature follicles were observed. Studies of ovarian follicle dynamics using laparoscopy and ultrasonography, correlated with hormone concentrations, revealed that follicles grow, mature, and regress in an overlapping wavelike pattern (follicular wave) (Figure 16.32). These waves are similar in adult and yearling females [45], and apparently these waves extend into the first half of pregnancy. The follicular phase resumes early in the postpartum period of the females, as early as five to seven days [46]. There was little difference in the number of follicular waves between the nonbreeding [44] and breeding seasons in South America.

For ovulation to occur, a follicle must be >7 mm in diameter. In a female with small follicles (<6 mm), copulation

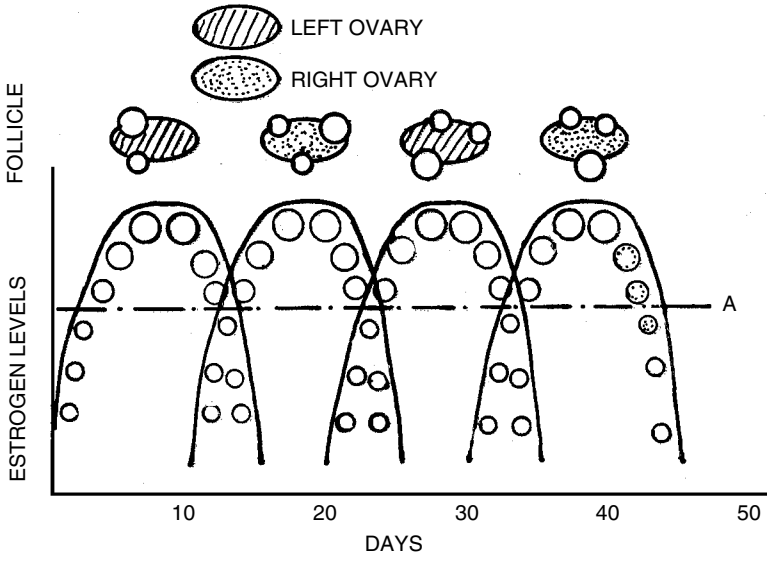

Figure 16.32 Diagram of the follicular wave cycle of an SAC female.

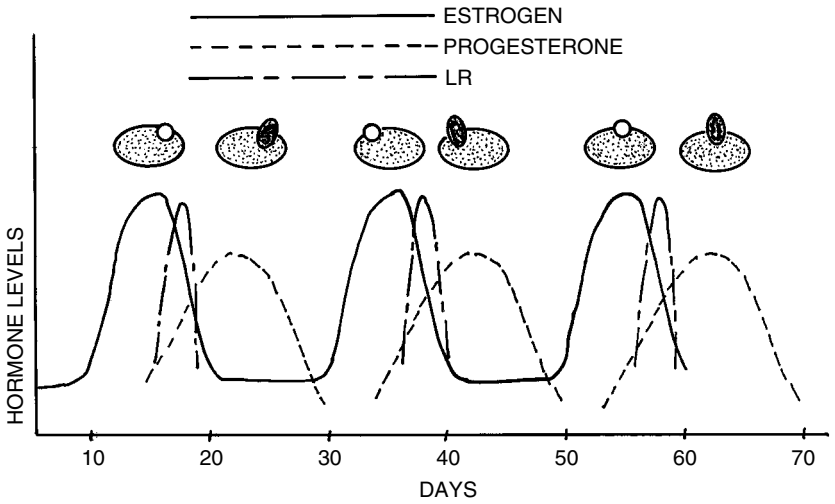

Figure 16.33 Diagram of the hormonal relationships of a noninduced ovulatory mammal [40].

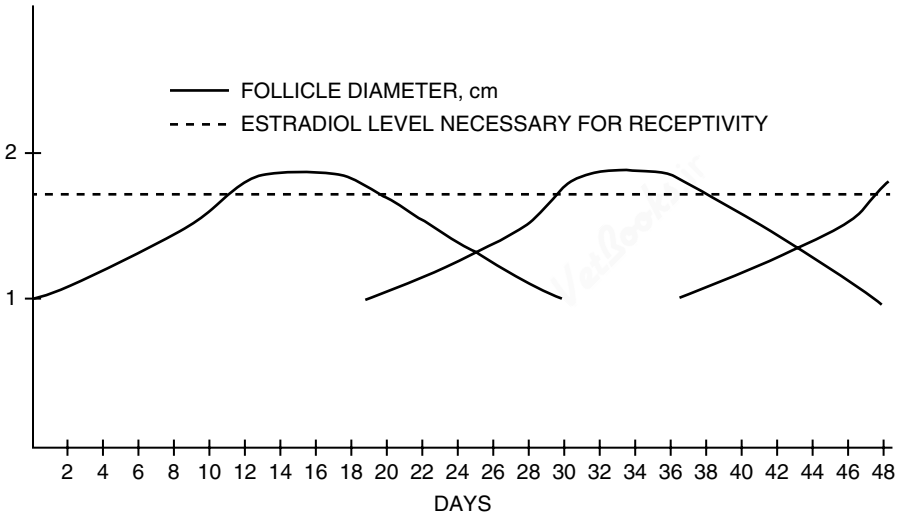

Figure 16.34 Diagram of the follicular wave of a camel. Dips indicate periods of nonreceptivity.

did not trigger ovulation; neither were follicular waves altered. If females with follicles between 7 and 12 mm in diameter were bred, ovulation was induced [47]. If females are bred during the regressive stage, copulation may initiate luteinization of the follicle, without ovulation. Progesterone is secreted, resulting in a short luteal phase lasting five to six days. In contrast, the luteal phase lasts 10–12 days when ovulation occurs but pregnancy does not follow. A short luteal phase may explain why some females refused the male on day 6 or 7 after copulation but were sexually receptive on day 9 or 10 after copulation.

Follicles of 7–9 mm in diameter are mature. Any follicle >13 mm is considered to be cystic. A puzzling aspect of normal reproductive physiology in the female SAC is that sexual behavior or sexual receptivity is not related to the size of follicles. There was no difference in sexual receptivity between females with small follicles and those with mature follicles.

Ovulation in the female SAC is induced by the combined inflammatory response to mating and the intrauterine deposition of ß-nerve growth factor (OIF/ß-NGF) in seminal plasma [48–51]. The neurotrophin, ß-NGF was originally known as "Ovulation Induction Factor" before it was characterized as NGF in 2013. This protein originates in the prostate gland of the male camelid and is carried by the seminal plasma into the female's uterus to induce ovulation. Peripheral reflex stimuli for ovulation may include penile penetration through the cervix, orgling of the male, and the male clasping his forelimbs around the body of the female, however ovulation will not occur without the

presence of seminal plasma. NGF from the seminal plasma is absorbed through the endometrium and elicits a preovulatory LH surge by action on GnRH hypothalamic neurons which, in turn, secretes gonadotropin-releasing hormone (GnRH) [51]. GnRH acts on the anterior pituitary to initiate release of luteinizing hormone (LH), which subsequently acts on the follicle, causing ovulation (Figure 16.35) [50, 53]. Ovulation usually occurs 30 hours following copulation, but sequential observation of the ovaries revealed that ovulation may occur between 24 and 48 hours following copulation. Time to ovulation is not influenced by follicular diameter [54]. It is also apparent that a single ovulatory input, either through copulation or administration of GnRH, is enough to induce ovulation. A second copulatory period and/or GnRH administration at 6, 24, or even 48 hours does not stimulate more release of LH from the pituitary [52]. After the initial surge, the pituitary gland becomes refractory to GnRH stimulation. It should also be noted that a small percentage of females may ovulate spontaneously, complicating breeding management.

If conception does not occur, the corpus luteum (CL) enlarges to its maximum size in seven to eight days and then regresses in response to the action of prostaglandin $F_2\alpha$ ($PGF_2\alpha$) [49, 55]. Pulsatile release of $PGF_2\alpha$, detected as its metabolite 15-keto-13,14-dihydro$PGF_2\alpha$, begins at days 9 through 12 after copulation. While the CL is active, the development of ovarian follicles is suppressed. As the CL regresses, follicle size increases, returning to normal by three to four days following the demise of the corpus luteum. The female becomes receptive to a male and remains in this state until copulation and ovulation occur again. More rarely, spontaneous ovulation may occur, or an ovarian quiescent state ensues.

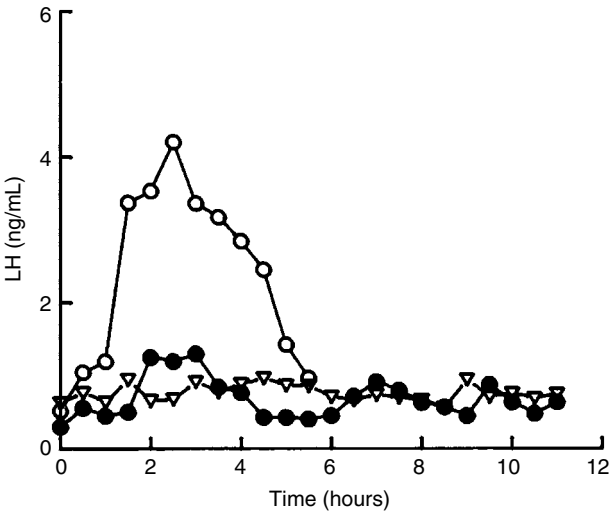

Figure 16.35 Luteinizing hormone response following copulation in a llama. Open circle = ovulating females, closed circle = nonovulating females, and triangle = control females that were not bred [52].

Hormone concentrations of estradiol follow follicular dynamics. Estradiol 17-β and estrone sulfate in plasma or urine are related to follicular waves. Concentrations of 10–15 pg/mL of estradiol, 20–30 ng/mg of urinary estrone sulfate, correlate with follicle sizes of 7–11 mm.

Pregnancy

The ovarian bursa completely encases the ovary so that transport of the ovum to the oviduct is easily accomplished. In livestock species, the ovum reaches the ampulla (midportion) of the uterine tube within two hours, where fertilization takes place and the zygote begins the cleavage process. The zygote remains in the uterine tube for three to six days. South American researchers have indicated that the alpaca embryo reaches the uterus by day 6 or 7 after copulation.

Once the zygote reaches the uterus, it will probably implant in the left horn. More than 95% of successful llama and alpaca pregnancies are implanted in the left horn. Some studies have indicated that 99–100% of pregnancies are implanted in the left horn [56]. Because there is an equal distribution of CLs in both the right and the left ovaries, and because each ovary is encased within a bursa, the ovum and subsequent zygote from the right ovary must migrate from the right horn to the left horn or be absorbed.

The precise time of fetal membrane attachment to the uterine mucosa in camelids has been reported recently. The approximation of embryo membrane to the uterus is initiated by 20–21 days, which coincides with elevated levels of estrone sulfate between 21 and 27 days after copulation [57]. In addition, appearance of the embryonic heartbeat 24–25 days after copulation may indicate implantation.

Early embryonic death occurs more frequently in alpacas and llamas than in other livestock species [1]. Estimates of 15–20% have been reported in North America; however, in South America 50% embryo loss within the first 30 days is seen. Late work involving ultrasonography and progesterone determination during the first 44 days of pregnancy indicates that 48% of embryo loss occurred in females that had previously failed to conceive for a year and 29–30% of embryo loss in lactating or maiden females. The precise cause of early embryonic death is unknown but is suspected to be heavily related to progesterone. About 42% of embryonic loss was correlated to a drop in progesterone levels. A second 21% of embryo loss occurred when progesterone concentrations dropped two to three days before the disappearance of the embryo (progesterone insufficiency).

And one third 37% of embryo loss occurred when progesterone concentrations remained elevated (persistent corpus luteum). In addition, failure of the right horn to maintain a suitable environment for the conceptus, or chromosomal aberrations may be factors. Infections have not been identified as abortifacients in SAC.

Fetal development proceeds in SACs as it does in other livestock species. Prenatal growth in alpacas has been described [58]. It was found that 85% of fetal weight was gained after 210 days of gestation. The usual length of gestation ranges from 335 to 360 days [55], with the occasional female carrying a fetus for more than a year. Gestations as long as 13 months have been reported; however, the authors have dealt with a number of female llamas suspected of pregnancies of more than a year, only to ultimately find a discrepancy in breeding dates. Carrying twins to term is rare [59, 60].

Twinning had once been considered rare, however more recent research has shown that multiple ovulations are relatively common in SAC's. Incidences of 6–10% have been reported in alpacas, and 12% in guanacos [61]. Twinning may be an important cause of early pregnancy loss as nearly all twin pregnancies are reduced to a single pregnancy by 33 dpm. In one study, bilateral ovulations had a higher incidence of twin pregnancy (62.5% of bilateral ovulations compared to 37.5% of unilateral observations) [62]. Spontaneous twin reduction may also result in complete loss of both conceptuses. Females with multiple CL's at pregnancy diagnosis should be monitored until at least day 45–50 dpm for evidence of twin pregnancy. If twins are diagnosed beyond this stage, it is recommended the pregnancy be terminated by administration of a luteolutic dose of cloprostenol to prevent later abortion or complications [62].

The growth of the embryonic vesicle is illustrated in Figure 16.36. The growth of the fetus is illustrated in Figure 16.37. The crown-rump length (CRL) progresses uniformly, with an increase in rate during the last three and a half months. The primary increase in fetal weight and development occurs during the last three and a half months of pregnancy.

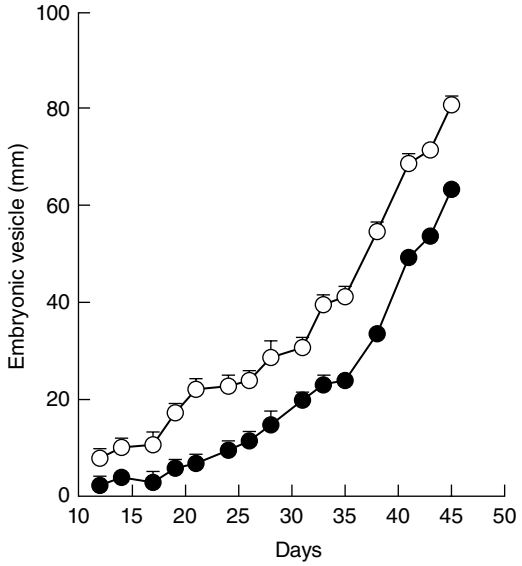

Figure 16.36 Growth of the embryonic vesicles in two alpacas from the day of detection to day 45.

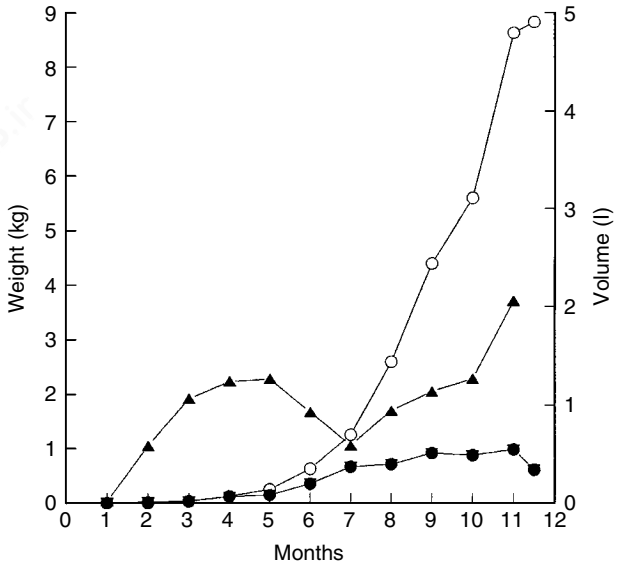

Figure 16.37 Fetal weight (open circle), weight of the placenta (triangle), and amniotic fluid volume (closed circle) during pregnancy [58].

Fetal Membranes and Fluid

Placentation of camelids has been classified as diffuse and epitheliochorial, similar to that of the mare and sow. However, camelid fetal membranes are unique. Early embryonic development of the fetal membranes, from the blastocyst stage to attachment to the uterus, is not unusual. Between 70 and 90 days, the surface of the chorion becomes dotted with numerous half-circular, domed projections that fit into corresponding depressions in the uterine mucosa (Figures 16.38 and 16.39). At 20×, a capillary network may be seen on the surface of the domed placentomes. The attachment is tenuous, and the chorion may be peeled from the mucosa with no resistance.

At this stage, the embryo is encased within an amniotic vesicle that is free-floating within the allantochorion, attached to the allantois by the umbilical cord and vessels (Figure 16.40) [63]. This relationship is maintained until the last two months of gestation.

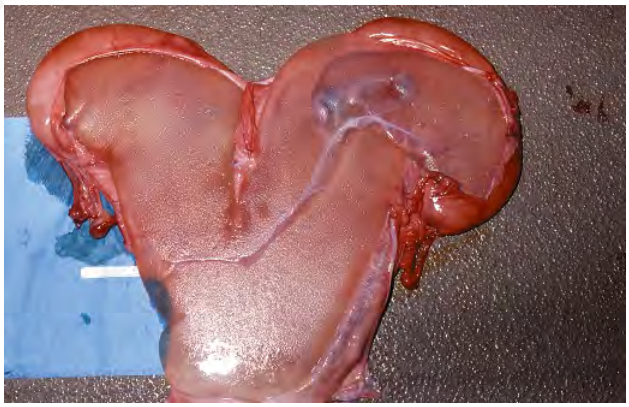

Figure 16.38 Llama placenta, *in situ*, approximately 60 days gestation.

Figure 16.39 Surface of a llama chorion at four months gestation.

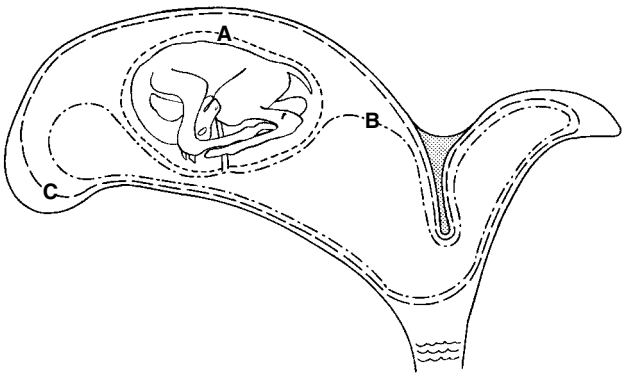

Figure 16.40 Diagram of early camelid placenta. (A) Amnion, (B) allantois, (C) chorion.

The allantochorion contains the fetal vascular supply. The surface of the chorion gradually changes from the domed placentoma to a multifolded papilla. It appears that each dome elongates, producing folds that expand to provide a greater surface area. The tip of each tuft is larger than the neck. If the placentoma becomes edematous as a result of disease, it looks like a miniature mushroom. The papillae fit into corresponding crypts in the uterine mucosa.

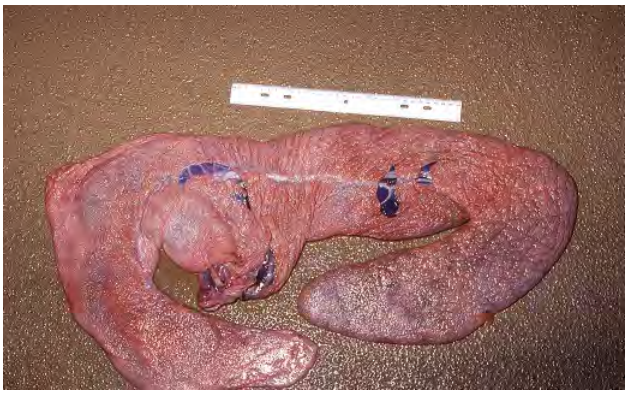

Figure 16.41 Full-term llama placenta, chorion outside.

Figure 16.42 Scanning electron microscope picture of the endometrial face of the chorion in a full-term llama placenta (Magnification 25×).

The papillae vary in size and shape, but generally the base is smaller than the tip. They may be columnar, rectangular, or shaped like an inverted cone and are 0.25–1.5 mm long. The density of the papillae is greatest in the patches, but a few papillae are found in the interspaces.

On visual inspection, reddish, irregularly shaped patches occupy most of the surface of the chorion, with lighter-colored interspaces (Figure 16.41). The surface appears to be a roughened, undulating membrane grossly, but when viewed through a dissecting microscope at 20×, hundreds of multifolded papillae per square centimeter may be seen. An electron microscopic picture emphasizes the multiple folds (hence more surface area) (Figure 16.42). Patches of papillae are most dense on the greater curvature and toward the center of the crescent-shaped placenta. The tips of the placenta have lighter patchiness, and a strip 2–3 cm wide, extending the length of the lesser curvature in juxtaposition to the large vessels of the allantochorion, is nearly devoid of papillae. A branching pattern of vessels may be

traced from the large vessels arising from the lesser curvature of the placenta to each patch of papillae. The relationship of the membranes to the fetus is illustrated in Figure 16.43.

The most unusual characteristic of the camelid fetal membranes is the development of an extra membrane of fetal epidermal origin. This membrane was originally described in the dromedary camel [64] but was not described in SACs until 1988 [65–67].

The epidermal membrane (EM) is an opaque whitish membrane, approximately 1–2 mm thick, covering the surface of the fetal body, head, neck, and limbs at near full term (Figures 16.44–16.47). The EM is attached to the neonate at the mucocutaneous junctions such as at the lips, anus, vulva, and prepuce. The EM is also attached at the junction of the skin and footpad, coronet of the nail, and umbilicus.

The EM is friable and easily torn or brushed from the surface of the neonate with only slight friction. Even with little movement or abrasion, it dries out and withers away soon after parturition. This is in contrast to the amnion of most mammals, which may completely envelope the nose and mouth, causing suffocation of a weak, nonstruggling neonate. Histologically, the EM consists of a layer of stratified squamous epithelial cells lying next to the fetus and an outer keratinized layer with indistinct cell outlines and no nuclei [65]. The fetus is usually delivered with the EM intact. However, if parturition is prolonged, with erratic or intense labor, or if manual assistance is necessary, the EM will split and disintegrate.

The precise function of the EM is unknown. The fetuses of noncamelid placental mammals float freely in the amniotic fluid. In the later stages of gestation in camelids, the EM separates the skin from the amniotic fluid. All of the SAC body orifices are unaffected by the EM, so excretory products from the digestive tract of the fetus are discharged into the amniotic sac, as in other mammals.

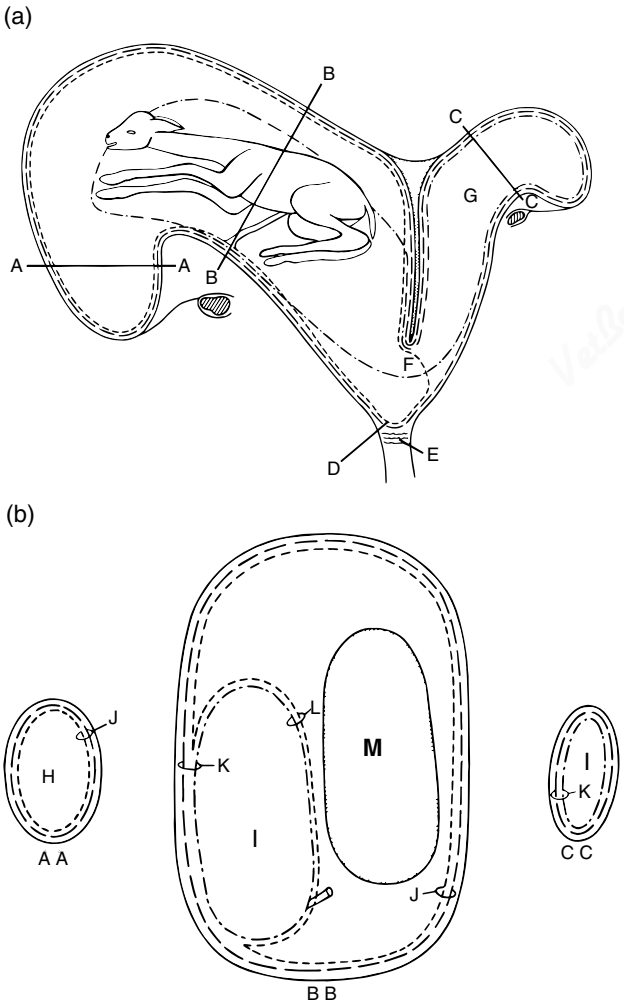

Figure 16.43 (a) Diagram of full-term llama placenta: (D) site of amniochorion dehiscence, (E) cervix, (F) body of uterus, (G) nonpregnant horn. (b) Diagram of cross section of full-term fetal membranes: AA, BB, and CC correspond to locations from A.; (H) amniotic cavity, (I) allantoic cavity, (J) amniochorion, (K) allantochorion, (L) amnioallantois, (M) fetus covered with epidermal membrane.

Figure 16.44 Epidermal membrane attached to the nostrils and lips.

Figure 16.45 Epidermal membrane attached to pads and nails.

Figure 16.46 Epidermal membrane over the pinna.

Figure 16.47 Epidermal membrane over the eye and eyelids.

The amniotic fluid of the llama remains watery throughout gestation, as does that of the camel, producing a slippery surface on the EM that facilitates delivery [63]. In contrast, the amniotic fluid of the mare and cow becomes mucoid toward the end of gestation; this also lubricates the fetus and facilitates delivery.

The EM originates from the epidermis of the fetus. The precise embryology of the membrane is not known in SACs, but the time of its development has been studied in the camel [64]. Microscopic changes could be identified with a fetal CRL of 17 cm, or approximately midgestation. Gross appearance of the membrane did not occur until just before the development of the hair, which seemed to elevate the membrane away from the skin surface. In llamas, the EM has not been observed grossly until the last two months of gestation. Prior to this time, aborted fetuses have been surrounded by the amnion, which had not become adherent to the chorion.

At full term, the SAC fetus is situated within the pregnant horn and the body of the uterus. The amniotic sac extends to the tip of both the pregnant and nonpregnant horns and occupies all the space not occupied by the allantoic sac. The amnion may be easily peeled from the fetal side of the chorion. The chorionic attachment to the allantois is more secure. There is a minimal amount of fluid in the amniotic sac.

The allantoic sac extends into the nonpregnant horn, though not to its tip, and occupies all of the space in the pregnant horn. One or more hippomanes (allantoic calculi) are present in the allantoic sac, varying in size from 1 × 1 × 1.5 cm to 1 × 2 × 5 cm and weighing 2–10 g (Figure 16.48a and b).

(a)

(b)

Figure 16.48 (a) Hippomanes from llama allantoic cavity. (b) Hippomanes from llama allantoic cavity.

Pregnancy Determination

Methods used to determine pregnancy in SACs include behavior, progesterone analysis, rectal palpation, ballottement, and ultrasonography [68]. All of the techniques described in this section have application for pregnancy determination in SACs. Currently, the preferred and most definitive method for pregnancy determination is ultrasonography, either per rectum or transabdominally.

Behavior

Usually, a mature, nonpregnant female will accept mating by a breeding male. In South America, refusal of sexual advances of the male by a female is considered to be a positive diagnosis of pregnancy. Refusal may be a good preliminary indication of pregnancy, with an 84% accuracy rate, but many factors may modify this behavior, and for the SAC owner or manager in North America, additional information must be obtained to verify pregnancy. Certain pathologic conditions simulate pregnancy (pyometra, retained CL, mummified fetus).

Breeding strategies that continually leave a male with females may foster development of a male that ignores even receptive females. Also, the male and one or more females may simply be incompatible.

An extremely aggressive male may overpower a timid female and force copulation, even though the female is pregnant. There is some danger if this should happen, since the male inserts the penis through the cervix and even into the horns of the uterus during copulation. Physical entry into the uterus after 90 days may traumatize the developing fetal membranes or introduce infection into the uterus. In either case, abortion may ensue.

Hormone Analysis

Serum progesterone analysis has been promoted as the definitive method for pregnancy determination in SACs [69]. While it is true that elevated levels of progesterone indicate the presence of a CL, this is not always confirmation of pregnancy. If this fact is kept in mind, progesterone levels may be useful as a management tool not only for determining pregnancy but also for evaluating various types of infertility problems. Elevated progesterone (>2 ng/mL) at day 7 after copulation is an indication of ovulation and presence of an active CL. A concentration level of progesterone >1 ng/mL (depending on the laboratory) 21 days post-copulation is an indirect indication of pregnancy.

Estrone sulfate and relaxin have also been studied in SACs [70]. Peaks in levels of estrone sulfate in plasma and/or urine are observed twice during pregnancy (Figure 16.49), the first at 21–27 days after copulation (45 ng/mL in serum) and the second at the end of pregnancy. At this time, concentration levels of 40 ng/mL in serum have been reported. The first peak probably indicates the beginning of implantation, and the second is an indication of fetal viability. The second measurement is useful in cases of prolonged pregnancy, as the amount decreases precipitously at the time of parturition [71]. Even though estrone sulfate is an indicator of pregnancy and potentially fetal well-being, its clinical use has not been well defined.

Elevated levels of relaxin concentrations in the pregnant llama or alpaca may also indicate pregnancy. At two months of pregnancy, the values are 26 ng/mL (basal, 2.4 ng/mL). The maintenance level is 5 ng/mL, increasing to >25 ng/mL by the end of pregnancy, making relaxin an accurate indicator of pregnancy at specific times [70]. Currently, there is no commercial assay for determination of relaxin in camelids and thus, it has not been used clinically.

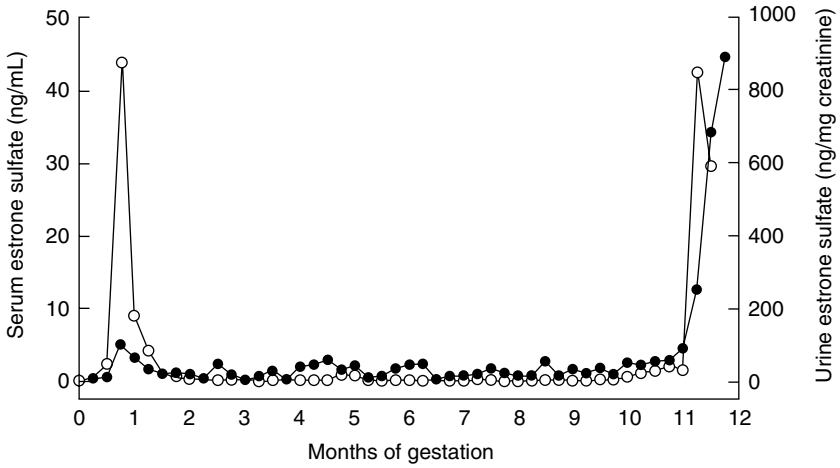

Figure 16.49 Estrone sulfate concentration in serum (open circle) and urine (closed circle) of the pregnant llama/ alpaca.

Radioimmunoassay (RIA) and enzyme immunoassay (EIA) are standard analytic techniques for determination of progesterone levels. There is some variation between laboratories, but as long as results are consistent and the same laboratory is used for all determinations, this should not cause great concern. Some laboratories using RIA report that levels of 2 ng/mL are indicative of the presence of the CL. Higher levels, 5–8 ng/mL, have been reported but do not correlate with better maintenance of the CL. A different laboratory may use 1 ng/mL as the cutoff point. It should be noted that cases of successful pregnancies have been reported in which progesterone levels were less than 0.5 ng/mL. At the other extreme, progesterone levels of over 2 ng/mL have resulted from a retained CL, with no fetus present.

Rectal Palpation

Rectal palpation has been the method of pregnancy determination in horses and cattle for decades. The smaller size of llamas and alpacas makes rectal palpation more difficult, but it is a valid technique and may be done safely if certain precautions are observed.

Adequate restraint is a prerequisite for rectal palpation and ultrasound examination. Several different types of chutes prevent side-to-side motion of the animal (Chapter 3). Some SACs lie down when subjected to an unpleasant situation; others constantly alternate between lying down and jumping up. The latter behavior makes examination difficult and dangerous. Some clinicians prefer to perform rectal examination with the animal in sternal recumbency, because abdominal pressure pushes the pelvic viscera into a more accessible location. To ensure continued recumbency, it may be necessary to sedate the SAC with 0.15 mg/kg xylazine intravenously (IV).

People with large hands (glove size larger than 7) or large forearms will find it difficult to examine any except the largest animals. Adequate lubrication is the key to success. Fecal pellets should be gently removed from the rectum and methyl cellulose or other lubricants instilled into the rectum with an irrigation-tipped 60-mL syringe or dose syringe. The rectum should be slowly dilated, with the hand-held in a cone position, palm facing dorsally. More lubricant should be added as needed.

Some examiners find it helpful to use a lubricant containing lidocaine to diminish mucosal irritation and reflex straining. Commercial lidocaine lubricants are available, but a less expensive mixture may be prepared by mixing 10 mL injectable 2% lidocaine with 100 mL methyl cellulose.

The anal sphincter may be the most restrictive site in the examination process. The anal epithelium may tear slightly during dilation. Excessive anal tone may be diminished by epidural anesthesia (Chapter 6). Lidocaine (1–2 mL, 2%) provides adequate relaxation of the sphincter but does not prevent peristaltic activity in the rectum. While a rectal examination using epidural anesthesia was being conducted, the female contracted the rectum around the author's wrist. The spasm did not subside for about 10 minutes. Nothing could be done except wait because any vigorous attempt to withdraw the hand would have split the rectal wall.

SACs are not as susceptible to rectal trauma as mares but are more sensitive than cows. See Chapter 6 for a discussion of rectal laceration.

The cranial half of the uterine horns diverge at approximately 180° from each other. Judicious palpation allows measurement and evaluation of the horns and the ovaries. It is important to remember that the ovaries are encased within an ovarian bursa, which may hinder evaluation (Figure 16.50). The relaxed ovary is ventral to the broad ligament. Retraction of the uterine horn aids in delivery of the ovary for palpation.

Examiners familiar with rectal palpation of the pregnant mare or cow may find slight differences in the uterine tone of an SAC. At 60–90 days, the left uterine horn is taut, resembling an inflated balloon, and is the key feature differentiating a nonpregnant from a pregnant female SAC.

At 30 days, pregnancy of a primiparous female may be determined through rectal palpation done by experienced SAC palpaters, who expect a unilateral increase in the diameter of the pregnant horn. In multiparous females, the left horn is usually larger than the right. If determination is to be limited to a single examination, it is preferable to examine at 40–45 days, at which time the pregnant horn should be 7–8 cm in diameter (palm width). Beyond 90 days, the uterus is positioned cranial and ventral to the brim of the pelvis, making it difficult to reach the uterus for evaluation. After eight months, the fetus may be accessible for palpation.

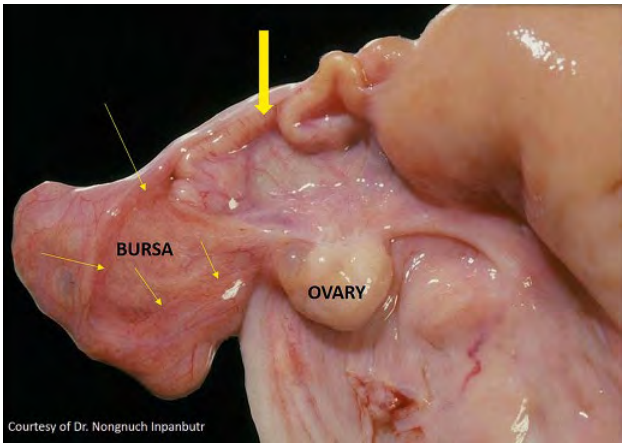

Figure 16.50 Alpaca ovary and oviducts. The ovarian bursa is indicated in the photo. *Source*: Photo courtesy of Dr. Nongnuch Inpanbutr.

Signs such as the inability to palpate the uterus, a flattened, taut vagina, and a firm cervix may be suggestive of pregnancy but not conclusive evidence. There are too many congenital anomalies that produce mucometra, especially in a nulliparous female. The middle uterine artery develops fremitus during the last trimester of pregnancy, but this is not the basis for determining pregnancy or differentiating between pregnancy and pyometra, as it is for the bovine. It is not possible to slip the fetal membranes in llamas due to the diffuse type of the placentation. The relative quantity of placental fluids is much less in SACs than in other livestock species. Some examiners have even made the mistake of concluding the presence of a mummified fetus because of the lack of fluids.

Determining the age of the fetus via rectal palpation is difficult after 90 days, even late in pregnancy, when various parts of the body and limbs may be palpable. Increases in the CRL are gradual, even during the last trimester, while the fetal body weight increases exponentially during the last three and a half months.

Ballottement

External signs of pregnancy may be lacking in female llamas. Udder development may be minimal, especially in a nulliparous female. The body contour changes in most females, but the fiber coat may mask changes. As late as eight months, the fetus weighs only 3.5 kg. By 10–11 months, the fetus may be balloted from the right side. Compartment 1 of the stomach occupies the entire left side of the abdomen, so the pregnant uterus is diverted to the right side. Ballottement is carried out as in cattle and may be done in two positions. In a standing animal, a clenched fist is pressed up against the abdominal wall, and a short vigorous push is exerted while the fist is held in place. If the female is pregnant, the push will cause the fetus to bounce away from the push, being cushioned in the fetal fluids, but it then drifts back and bumps the fist. In the lateral position, as practiced in South America immediately after shearing, pressure of both hands is applied on the abdominal wall, and the fetus bounces, indicating pregnancy and that the animal can be separated from open females. The procedure should be repeated at a number of locations on the right side before the conclusion is reached that the female is not pregnant. At time of ballottement, the development of the mammary glands should also be inspected. However, mammary glands usually do not become enlarged with colostrum until 20–30 days before parturition and, even then, some may not be enlarged.

Ultrasonography

The use of ultrasound (US) techniques for evaluation of the tubular reproductive tract and the ovaries has added a new dimension to veterinary medicine. The rapid improvements in technology in this field parallel those of the computer industry. Units are currently available that allow the transducer to be carried into the rectum and manipulated by the hand of the palpator. This is the most desirable method to use on pregnancies of less than 120 days [72–75].

More sophisticated units allow transabdominal evaluations, which are more applicable for pregnancy determination later in the gestation period. In one instance, repeated rectal palpation and rectal US evaluation led a clinician to conclude that a female was not pregnant, and he was ready to institute hormonal therapy. A last examination using the transabdominal approach detected a fetal heartbeat in the abdomen, ventral to the stomach.

The application of real-time rectal or transabdominal US has a great advantage, especially for early detection of pregnancy. Pregnancy can be confirmed with ultrasonography by the presence of the embryonic vesicle, as a hypoechoic circular structure within the uterus as early as 9 days after copulation, but it is best performed 16–23 days after mating, when the accuracy of diagnosis is 100%. Selected ultrasonograms of early pregnancy in llamas appear in Figure 16.51. Figure 16.37 provides information on the growth rate of an alpaca embryonic vesicle from its detection until day 45 of pregnancy.[3]

Adequate restraint is a prerequisite for ultrasonography (see earlier rectal palpation discussion). When manual palpation is not possible, the rectal probe should be rigid. The attachment of a polyvinyl chloride (PVC) pipe eases the process. An alternative is the use of a prostatic probe, as in humans and mares. The resolution of the scanner is also greatly enhanced when a probe of 7.5 MHz is used. Details on formation of membranes, heartbeat, and placental development are evident using the latter probe. To determine fetal age and possible due date, the biparietal distance may be measured transabdominally. There is a strong positive correlation between gestational age and the biparietal distance. The equation is formulated as follows: GA = 18.8 + 3.79 BPD, where GA is the gestational age in days and BPD is the biparietal distance measured in mm [76].

Parturition

The endocrinology of parturition in llamas and alpacas has been partially defined. It has been reported that progesterone levels decrease rapidly two days before parturition. The same profile was observed when urine samples were assayed for pregnanediol glucuronide, a metabolite of progesterone. A different profile is seen when estrone sulfate is evaluated. Estrone sulfate in urine decreased precipitously

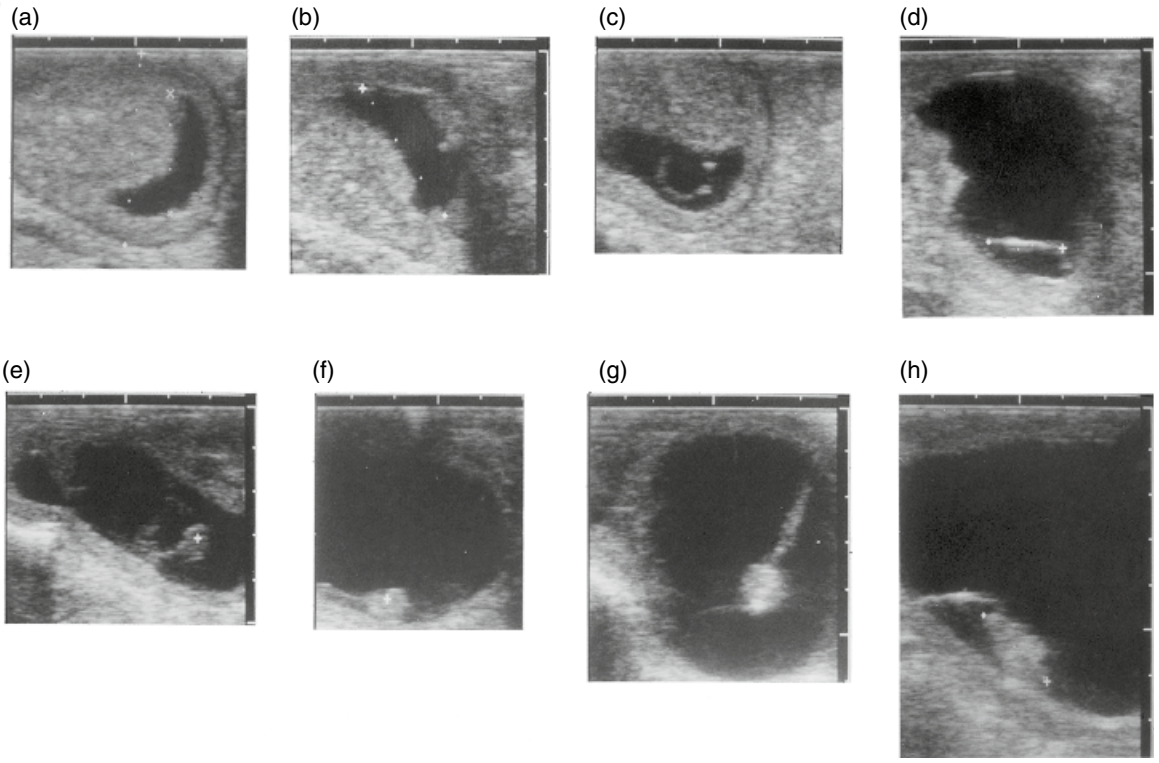

Figure 16.51 Ultrasound images of pregnant llamas. (a) 17 days, (b) 20 days, (c) 24 days, (d) 30 days, (e) 35 days, (f) 40 ays, (g) 50 days, (h) 60 days [71].

only at time of parturition, i.e. when the fetal-placental unit was disrupted [71].

Trying to predict the imminence of parturition is frustrating. Though the abdomen enlarges, growth may be hidden by the heavy fiber coat of many llamas and alpacas. Field biologists are able to assess pregnancy in guanacos and vicuñas by body contour, but these wild camelids lack the thick fiber coat of the alpaca and llama.[4]

Pregnant females within one month of parturition change behavior and appear restless and uncomfortable, with more frequent sniffing at the manure pile and possibly more voiding. Some females develop ventral edema, but not as pronounced as in mares and cows. Udder development is not necessarily correlated with nearness of parturition. The udder of a primiparous female may show little or no development until after parturition. The udder of a multiparous female usually enlarges two to three weeks before delivery, and the nipples may swell three to four days before delivery. Wax may form on the end of the teat, but its absence is not evidence that parturition is not imminent [77].

Normal parturition is initiated by hormonal changes and is divided into three stages. Stage one begins with cervical relaxation and uterine contractions that propel the fetus toward the pelvis, dilating the cervix. This stage may last from two to six hours and tends to be longer in primiparous females. The signs of stage one include slight discomfort, excessive vocalization (humming), and repeated defecation and urination.

Stage two is the expulsion of the fetus. The amniochorion may dehisce within the birth canal, or the chorion only may dehisce and the amnion protruding through the vulva as a fluid-filled sac. In either case, a little fluid is expelled, usually dripping from the vulva. It is possible that the fluid-filled sac may be a pouch of the EM, pushed ahead of the fetus. At this stage, the perineum will begin to protrude (Figures 16.52 and 16.53).

The normal presentation of the fetus is cranial longitudinal, with a dorsosacral position and the head extended and lying dorsal to the extended limbs (Figure 16.54). A slight variant of this commonly occurs, with the limbs positioned over the top of the head (Figure 16.55). The posterior longitudinal presentation, dorsosacral position with the hind limbs extended, is also an acceptable orientation that leads to a productive delivery but is much less common (Figure 16.56). The risk to the life of the fetus is greater in a caudal presentation dystocia because of the danger of occlusion of the umbilical vessels as a result of stretching or pressure against the pelvis of the dam.

Figure 16.52 Parturition, protrusion of the amniotic sac.

Figure 16.53 Delivery of the fetus.

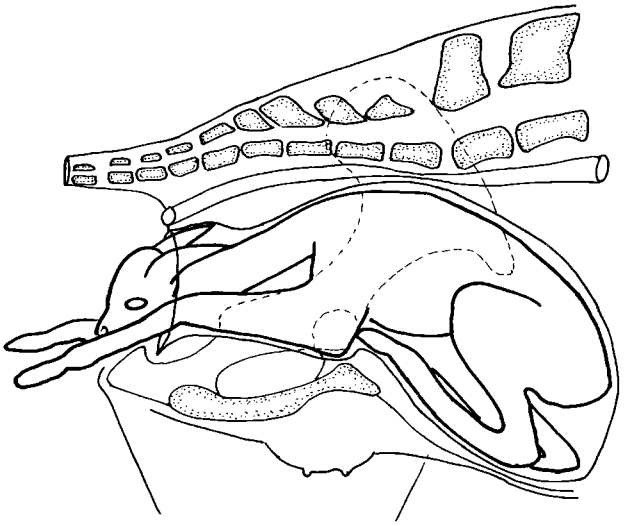

Figure 16.54 Normal llama delivery, cranial presentation, dorsosacral position.

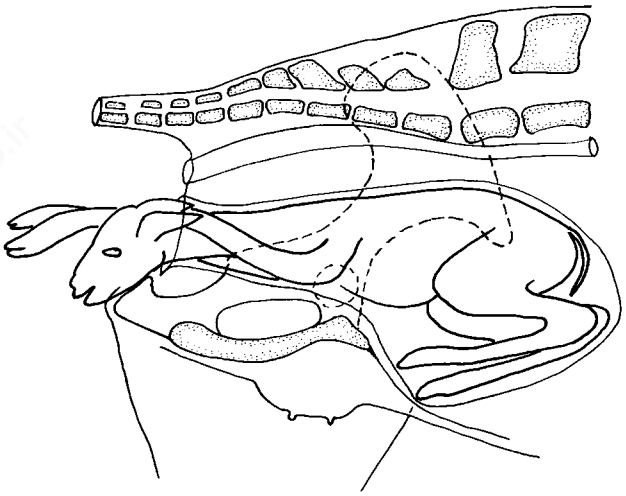

Figure 16.55 Commonly crias will be delivered successfully with legs above the head.

Uterine contractions initially occur every 10 minutes, increasing in frequency and intensity as expulsion progresses. At this stage, the female is definitely uncomfortable. She may lie down and rise repeatedly, lie on her side, or even roll. If the fetus has been partially delivered, it may be traumatized by such actions. The female may look back at her side, vocalize, and exhibit other signs of colic. In an uncomplicated parturition, the unassisted female should deliver the fetus within 8–25 minutes (24 minutes in llamas in the Altiplano). The majority of females deliver the fetus while in the standing position. In observations made on alpaca parturition in Peru, 65–73% of the fetuses were delivered while the female was standing. An additional 20–24% were delivered from sternal recumbency and 7–12% while the female was lying on her side.

The umbilical cord usually ruptures 15–20 cm from the abdomen of the cria. Maternal care is minimal. The mother does not attempt to remove the EM or lick the cria to dry it. There is usually nose-to-nose touching, with humming that apparently functions to establish the mother-cria bond.

The majority of births take place during daylight hours, with the preponderance occurring in the morning [1, 78]. However, llamas in North America have been known to

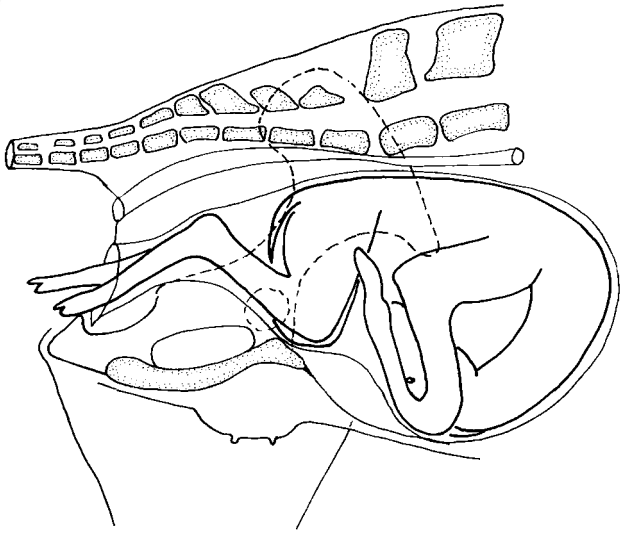

Figure 16.56 A caudal presentation, dorsosacral position is another alternative to allow for a successful vaginal delivery, however, this is less common.

give birth at night. SACs seem able to regulate parturition as a voluntary activity. Too much attention and excitement may actually delay parturition.

Stage three of parturition is expulsion of the placenta. The epitheliochorial placentation releases readily, and delivery of the placenta should occur within two hours postpartum. Retention of the placenta is rare in SACs. Dystocia or uterine inertia predisposes retention of the placenta. Failure to expel the placenta in six hours should be considered abnormal but, at this point, the only recommended therapy is the administration of oxytocin (5–20 units intramuscularly). If a segment of the placenta is protruding from the vulva, it should be grasped gently by a gloved hand and slight tension applied. Frequently, the membranes are sitting loosely in the vagina and the body of the uterus, and a slight tug is all that is needed to effect delivery.

It has never been necessary to manually separate the placenta from the uterine mucosa. If the placenta has not delivered by 12 hours, the protocols used for dealing with the problem in a mare are recommended.

The SAC placenta is crescent shaped. In the majority of cases, the membranes are reddish in color, with the chorion on the outside. In a few instances, the placenta is turned inside out, with the amnion exposed, and have a whitish, glistening surface. Each placenta should be inspected to determine the presence of the two tips, a good indication of complete expulsion.

The average llama placenta, exclusive of fluids, weighs approximately 3 kg (range 0.74–1.44 kg). The greater curvature measures 196–280 cm (77–110 in.), and the concave curvature measures 96–210 cm (40–83 in.).

Involution of the Uterus [79]

Discharge from the vulva postpartum (lochia) is scanty and is usually absent after six to eight days. Postpartum involution of the uterus occurs rapidly in SACs. In a study conducted with alpacas, the average weight of the uterus was 883 g 24 hours after parturition, with the pregnant horn being approximately 15 cm in diameter. Ten days postpartum, the uterus weighed only 155 g, and the pregnant horn was 3.5 cm, and in 20 days, 83 g and 3 cm, respectively. Such a rapid involution is necessary to maintain a 12-month birthing interval. Female llamas will accept copulation within two to three days postpartum, but the uterus is not involuted, and it is not desirable that breeding take place until 20 days following parturition [43]. Note that a male may even attempt to copulate with a female when she is recumbent and parturition is imminent.

Camel Reproduction

Female Camel Reproduction [41, 80]

Female camel anatomy and physiology are similar to those of SACs with due consideration for a larger size. Puberty may occur at two to four years of age, but breeding is usually not recommended until four years of age. Camels are seasonally polyestrous. The breeding season usually coincides with the rut in males. After the breeding season, the ovaries become inactive. Anestrous may extend up to six months.

The ovarian cycle is a follicular wave, similar to that of the SAC female. Camels are also induced ovulators. Contrary to SAC females, which are essentially receptive to the male unless they are pregnant, the camel follicular cycle is spread out, which allows estrogen levels to become low enough to cause the female to become non-receptive and later to become receptive again. This type of cycling is frequently but mistakenly, called an estrous cycle. There is still only a follicular wave and no ovulation unless the female camel is bred at an appropriate time.

The induction of ovulation is a neurohormonal stimulation brought about by an ovulation inducing factor present in the seminal plasma of males [81, 82]. This ovulation inducing factor has been identified as a beta-nerve growth factor that induces ovulation at similar rates similar to GnRH [82]. As in SACs, fertilization of the ovum occurs in the oviduct. The embryo enters the uterus at six and a half to seven days post breeding in camels. This is similar to the migration in llamas, but in alpacas the embryo may reach the uterus in three to four days. Organ differentiation occurs in 45–60 days. A heartbeat may be detected by 22 days. Gestation length varies from 355 to 419 days.

Camel Pregnancy

Camel caretakers claim the pregnancy is proven when the female extends her tail or holds it upward when teased by a male. Definitive diagnosis is established by rectal palpation and US examination as in SACs.

Signs of impending parturition include distention of the abdomen, mammary gland enlargement, relaxation of the pelvic ligaments, and edema of the vulva a few days prior to parturition. Parturition is similar to that of SACs. Uterine involution is complete in ± 20 days.

Postpartum Complications

Failure to Deliver the Placenta. Failure to deliver the placenta can occur but seems to be less common than it is in cattle. See discussion above.

Prolapse of the Uterus and Vagina. Too vigorous pulling on the placenta immediately after delivery of the fetus may evert the uterus. Replacement of the uterus at this time is easily accomplished.

Vaginal Prolapse

Causes

Vaginal prolapse is usually associated with pregnancy. In cattle, it often occurs at two to three months of pregnancy, at about the stage in which the placenta begins to secrete estrogens, inducing relaxation of the pelvic structure. Insufficient records have been kept to correlate similar happenings in camelids.

Prolapse occurs more frequently in multiparous females, indicating that stretching or injury to the pelvic structures as a result of a previous birth may predispose to vaginal prolapse. In cattle and some other species, it is thought that a hereditary factor may be involved and also that some animals may produce more estrogens than others [83].

Obesity, with excessive pelvic fat, may produce increased abdominal pressure while an animal is lying down. A preexisting relaxation of the pelvic structure may also contribute to vaginal prolapse.

Vaginal prolapse is a complication of parturition, particularly if dystocia has resulted in excessive manipulation of the fetus.

Clinical Signs

A slight prolapse of the mucous membrane of the vagina is sometimes seen during pregnancy near term, especially when the female is lying down. The membrane is usually pink, moist, and glistening, with no evidence of pathology. If the bulge disappears when the female rises, no treatment is indicated. However, a continually exposed membrane may become traumatized by the tail or become dry and necrotic.

The entire vagina may be exteriorized by persistent tenesmus. Edema or pressure may obstruct the urethra and cause urine retention. Other caudal abdominal organs may prolapse through the vulva via rents in the uterine or vaginal wall.

The animal should be standing, if possible, with the hindquarters elevated. An extensively prolapsed vagina should be wrapped with a moistened bath towel and encased in a plastic bag to keep the membrane moist and to avoid trauma and infection. Epidural anesthesia is recommended. The extent of the prolapse and degree of trauma to the mucous membrane determine treatment. The membrane should be thoroughly cleansed with physiologic saline and povidone-iodine solution (diluted 1 : 10). Any foreign material must be physically removed, and obviously necrotic tissue should be debrided. The vagina may be cupped in the hands and gently pushed back into place with the palms. Point pressure with the fingers should be avoided. The invagination must continue past the lips of the vulva. A person with small hands may be able to push beyond the vulva with bent fingers. A plastic or cardboard mare speculum may be gently inserted to complete the inversion process if the operator's hand and arm are too large to enter the female.

It is usually necessary to suture the lips of the vulva closed to prevent recurrence until the edema and inflammatory response have dissipated. The vulva is small, and a simple purse-string suture may be sufficient. However, the author prefers a lacing technique similar to that used on cattle.

In this technique, two or three loop sutures are placed on either side of the vulva, 2 cm lateral to the vulvar slit. Excessive slack should not be left in these loops (Figure 16.57). The loops are threaded like a shoe is laced, using 3-mm (1/8-in.) umbilical tape, and tied in a bow. The animal will be able to urinate. The threaded strand may be loosened or even removed to ascertain the degree of healing but can be easily replaced if the prolapse threatens to recur.

If this procedure is carried out on a pregnant female that is nearing the end of term, she must be closely observed so that the lacing can be removed as parturition begins.

Sprinkling the swollen membrane with sugar or applying glycerin to reduce edema of a prolapsed vagina is a time-honored practice, and there is little doubt that fluid can be removed in this manner [83].

It may be desirable to insert a catheter to instill a solution containing powdered tetracycline into the vagina, but an

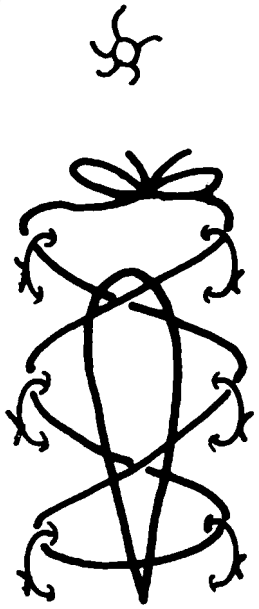

Figure 16.57 Truss (shoelace) pattern for retention of vaginal prolapse.

antibacterial bolus should not be inserted, because it may stimulate tenesmus.

Post-operatively, the animal should be observed closely for signs of continuing tenesmus or significant exudation. The major complication is recurrence of the prolapse; however, vaginitis and even metritis may follow a prolapse. If tenesmus persists, the vagina should be reexamined to ensure that the prolapse was completely repositioned.

Prolapse of the Uterus

Causes

Much of the discussion relative to vaginal prolapse applies to uterine prolapse. Prolonged and intense tenesmus induced by a retained placenta is a common cause. The uterus has also been known to prolapse during attempted delivery of the placenta immediately following parturition. Following an exhausting dystocia, a completely atonic uterus may prolapse through the relaxed vagina.

Clinical Signs

The protrusion of the inverted uterus is obvious. In camelids, there are no cotyledons on the placenta. It may be somewhat difficult to differentiate uterine from vaginal mucosa, but it should be possible to identify the cervical rings.

Much of the previous discussion on replacement of vaginal prolapse is also applicable when discussing uterine prolapse. However, it may first be necessary to deliver the placenta. Any portion of the placenta that remains should be delivered. The attachment is minimal, and delivery should not be difficult. It is more difficult to reduce the telescoping, because of the small size of the canal, but immediately after parturition, even a person with large hands can manually reposition the uterus, taking care to invert both horns. If the prolapse occurs at any other time, it may be repositioned only by a person with small hands and arms. As soon as the uterus is replaced, 30–50 units of oxytocin should be administered IV or intramuscularly (IM) to stimulate contraction of the uterine muscle.

A truss suture pattern may be employed to ensure retention of the uterus but, in many cases, this is not necessary. The animal should be observed closely for tenesmus and reexamined if it persists.

Rupture of the Uterus

Causes

In SACs, rupture of the uterus is usually associated with dystocia. Prolonged, strong contractions of the uterine musculature against an immovable fetus may result in a rent in the wall of the uterus. Other causes include uterine torsion and an oversized fetus. A limb may be pressed through the wall if a carpus or tarsus has become ankylosed and inflexible. Chronic peritonitis associated with uterine adhesions may result in a tear in the uterine wall when the contractions of parturition begin. Severe trauma to the abdomen near term may cause a rupture of the uterus.

Clinical Signs

External signs may be minimal and consist only of knowledge that dystocia has occurred. Frequently, the rent will be palpated after the fetus has been delivered and the uterus is being checked for a twin. If the rupture is not detected at parturition, it may be indicated as signs of peritonitis develop.

Dorsal recumbency is the preferred position for surgery, and general anesthesia is required. The surgical approach to correct a uterine tear is similar to that used for hysterectomy. Hemorrhage in the peritoneal cavity or peritonitis may be present. Because the tear may be in the body of the uterus near the cervix or in the uterine horn, careful palpation is necessary. A tear in the body is more difficult to visualize and suture.

A Lembert or Cushing infolding suture, as in a cesarean section, is satisfactory for closing the tear. The abdominal cavity should be lavaged with physiologic saline and treated as appropriate to prevent or alleviate peritonitis. The abdominal incision is closed as described previously.

A uterine tear maybe repaired using a controlled uterine prolpase to gain surgical access. With this technique, if the rent is detected during or immediately following

parturition, a hand and arm are inserted into the tip of the lacerated horn. An assistant administers 4–6 mL 1 : 1000 epinephrine HCl IV to induce relaxation of the uterine musculature. When the surgeon feels relaxation begin, the tip of the horn is grasped by the fingers and slowly everted and pulled through the vagina. The laceration may then be sutured outside the body and the uterus returned to its proper position. Oxytocin (20 units) should be administered to stimulate contraction of the uterus following the surgery.

Post-operatively, a five- to ten-day course of antibiotics, given parenterally, is indicated to prevent or treat metritis and peritonitis. If one of the large uterine arteries is ruptured concurrently, intraabdominal hemorrhage may result in exsanguination or severe anemia. Anemia would not be detectable via a hemogram for a few days after the incident. Peritonitis is a common sequel, along with metritis.

Uterine Infection

Metritis is rare except in cases of dystocia in which trauma has been inflicted by manipulations. A few cases have been associated with the obese female, postpartum.

Postpartum Hemorrhage. Hemorrhage can arise only from lacerations of the uterus or vagina. A persistent flow of blood should be investigated immediately.

Agalactia

Lactation failure following parturition is common in SACs (see Chapters 10 and 21). The problem is seen more often in primiparous females, but it also occurs in multiparous females. Lactation in SAC is presumably controlled by the same systems as in other livestock species. If the fetus is delivered prematurely, hormonal preparation of the mammary gland to begin secretion may not have been completed. Inadequate milk let down may be a factor. Placement of the neonate in the nursing position may stimulate the dam to allow milk let down, or administration of IM oxytocin in repeated doses of 10–20 units every two hours is appropriate therapy. Both actions should be accompanied by gentle massage of the udder with warm water and stripping milk from the teats. Domperidone (Equidone® oral gel) has been recommended for treating agalactia in camelids by giving three times the equine dose orally (~5 cc for a llama) for 7–10 days [84].

A careful watch of the baby is necessary to ensure that sufficient milk is being consumed. Otherwise, the cria must be supplemented or orphaned until an adequate milk flow has been established.

Rejection of the Cria

Rejection of the cria is extremely rare in SACs; in fact, female alpacas and llamas often accept crias other than their own. In one zoo herd, a particular female would accept every cria in the herd long after its own cria was weaned. However, this is uncommon in Vicuñas and guanacos which do not accept other crias.[5]

Obstetric Procedures [85]

Dystocia. Dystocia refers to any parturition that is more prolonged than expected. Breeders report a low prevalence of dystocia in SACs as contrasted with sheep and cattle [86]. In observations of large numbers of animals in Peru, it was determined that the prevalence of dystocia in alpacas was less than 2.4% and that the entire parturition process in the alpaca should be completed within three and a half hours [87]. Causes of dystocia may be related to fetal or maternal factors (Table 16.4).

Malposition. The factors that orient the fetus to the normal parturient presentation and position are unknown, as are those factors that cause malpositioning. Several malpositions are described and illustrated.

The most common malposition is cranial longitudinal presentation in the dorsosacral position, with the limbs extended but the head and neck flexed backward to the side (Figure 16.58). Others malpositions include:

1) Cranial longitudinal presentation, dorsosacral position, with the head and one limb extended but the other front limb retained (Figure 16.59)
2) Cranial longitudinal presentation, dorsosacral position, with one limb extended but the head and the other limb retained (Figure 16.60)
3) Cranial longitudinal presentation, dorsosacral position, with the limbs extended but the head retained between the limbs (Figure 16.61)

Table 16.4 Causes of fetal and maternal dystocia.

Fetal	Maternal
Malposition	Uterine torsion
Congenital defects	Uterine inertia
Schistosomus reflexus	Failure of cervix to dilate
Ankylosis of major joints	Malformed pelvis
Hydrocephalus	Stenosis of the vagina
Edema and maceration of fetus	Stenosis of the vagina

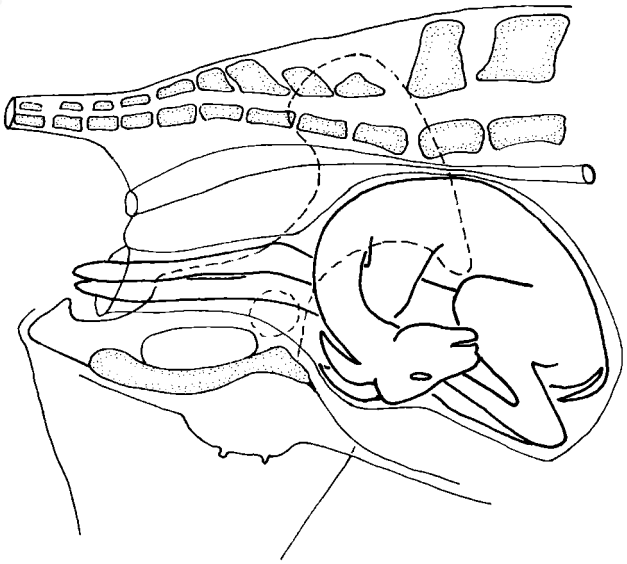

Figure 16.58 Camelid dystocia, cranial presentation, dorsosacral position, with head retained to one side.

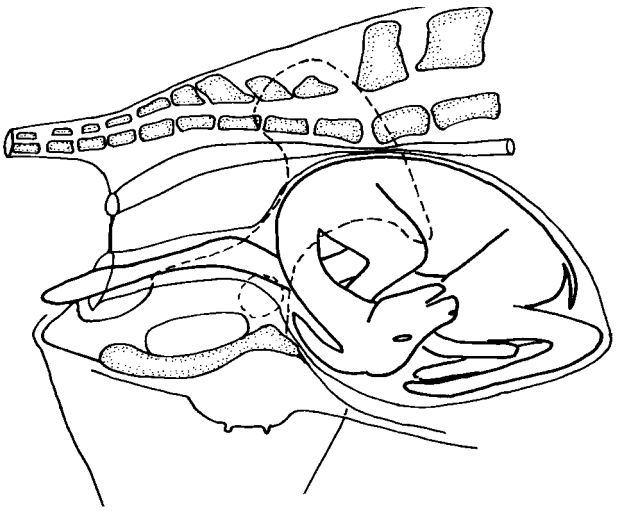

Figure 16.60 Camelid dystocia, cranial presentation, dorsosacral position, head and forelimb retained.

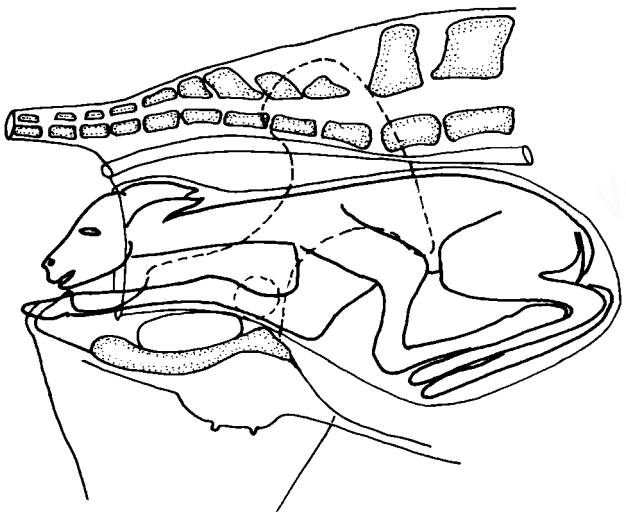

Figure 16.59 Camelid dystocia, cranial presentation, dorsosacral position, one limb retained.

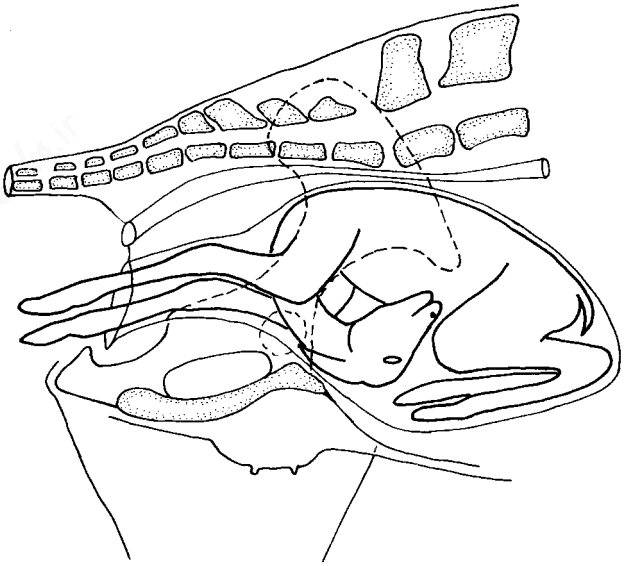

Figure 16.61 Camelid dystocia, cranial presentation, dorsosacral position, head retained between legs.

4) Cranial longitudinal presentation, dorsosacral position, with the head extended but both limbs retained (Figure 16.62)
5) Cranial longitudinal presentation, dorsosacral position, with both limbs and head retained (Figure 16.63)
6) Caudal longitudinal presentation, dorsosacral position, with the limbs retained (breech presentation) (Figure 16.64).

Other, less common dystocias are cranial longitudinal presentation, dorsopubic position, with head and forelimbs extended (Figure 16.65), and transverse ventral presentation, dorsoilial position, with all four limbs and the head extended (Figure 16.66).

Manipulation of Dystocias

Space does not permit detailed discussions of obstetric procedures. The reader is referred to a standard veterinary obstetrics textbook [83, 88]. A few unique facts should be kept in mind. SACs are small and there is little room for manipulation, even by a person with small arms and hands. It may be necessary to draft the smallest person available to

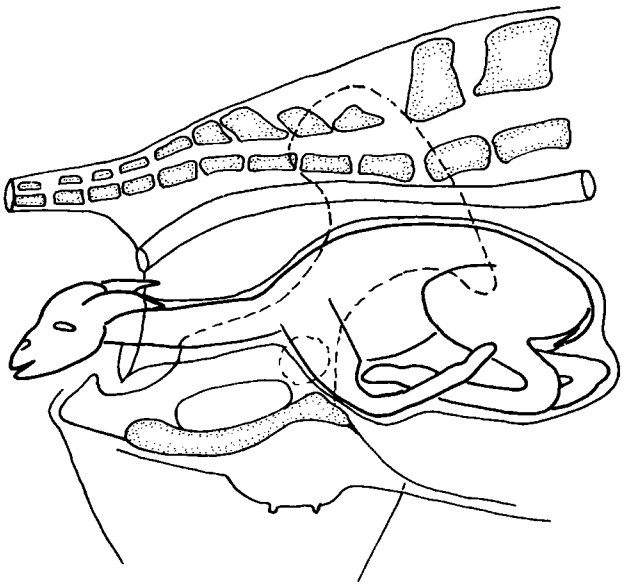

Figure 16.62 Camelid dystocia, cranial presentation, dorsosacral position, both forelimbs retained.

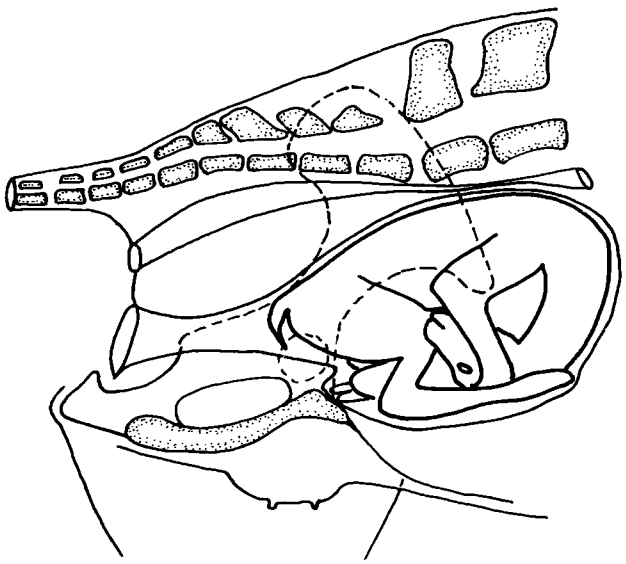

Figure 16.64 Camelid dystocia, caudal presentation, dorsosacral position, hind limbs retained (breech).

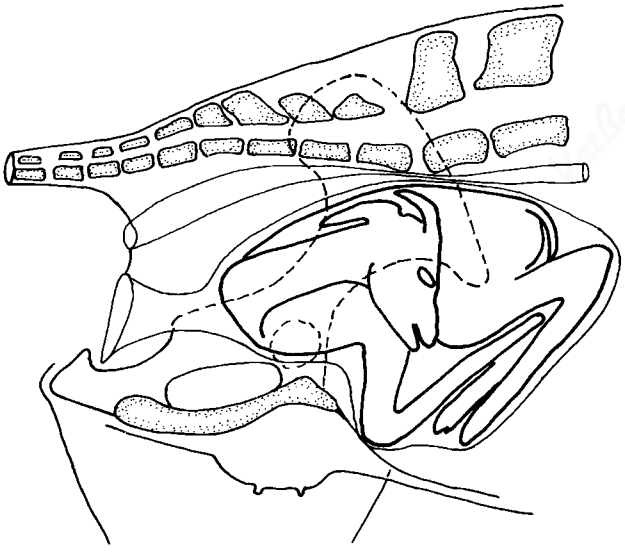

Figure 16.63 Camelid dystocia, cranial presentation, dorsosacral position, head and both forelimbs retained.

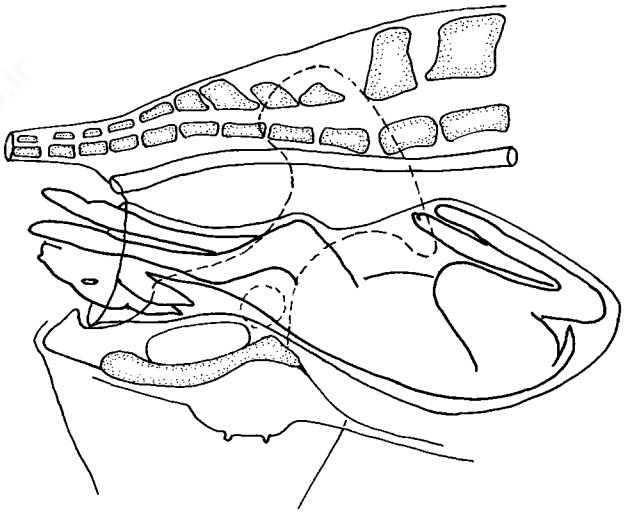

Figure 16.65 Camelid dystocia, cranial presentation, dorsopubic position, head and forelimbs extended.

perform any necessary internal manipulation, under appropriate direction. If manipulation by a large person is essential, epidural anesthesia should be administered to provide analgesia; additional relaxation may be produced with butorphanol (Chapter 6).

The limiting dimensions of the birth canal are not at the pelvic inlet but rather at the level of the caudal end of the sacrum and the ischiatic spines of the ilia (Figures 16.67 and 16.68). Measurements of one adult female weighing approximately 140 kg were as follows: on the cranial view, the vertical distance is 11 cm; the width, 10.5 cm; and the diagonal, 12 cm. The caudal view shows the relationship of the ischia ilia and sacrum and the corresponding measurements are as follows: the vertical distance is 12.5 cm, the narrowest width, 8.5 cm; and the diagonal, 14 cm. For comparison, Figures 16.69 and 16.70 illustrate cranial and caudal views of the pelvic inlet of a male.

Some ligamentous relaxation occurs at the time of parturition to allow the sacrum to tilt up slightly, but it is not likely that there will be any give in the width. The authors

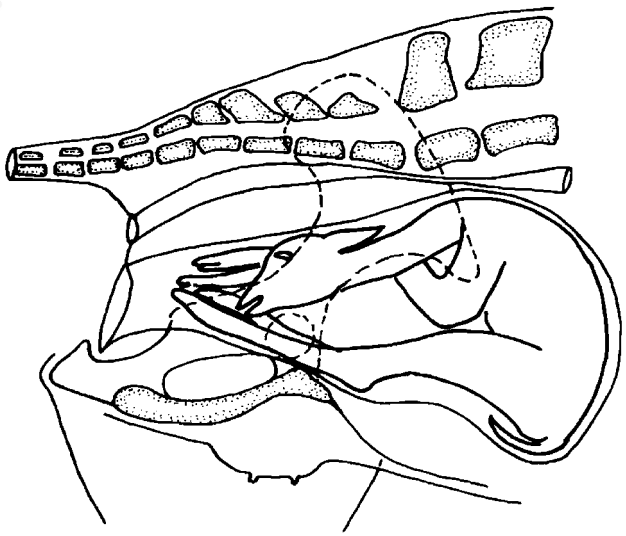

Figure 16.66 Camelid dystocia, transverse presentation, dorsoilial position, four limbs and head extended.

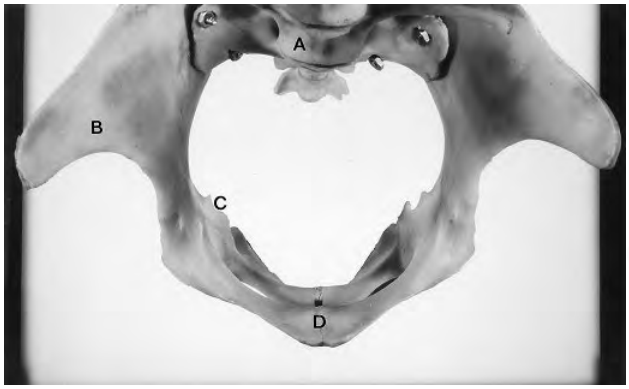

Figure 16.67 Female llama pelvis, cranial view. (A) Sacrum, (B) ileum, (C) ischiatic spine, (D) pubis.

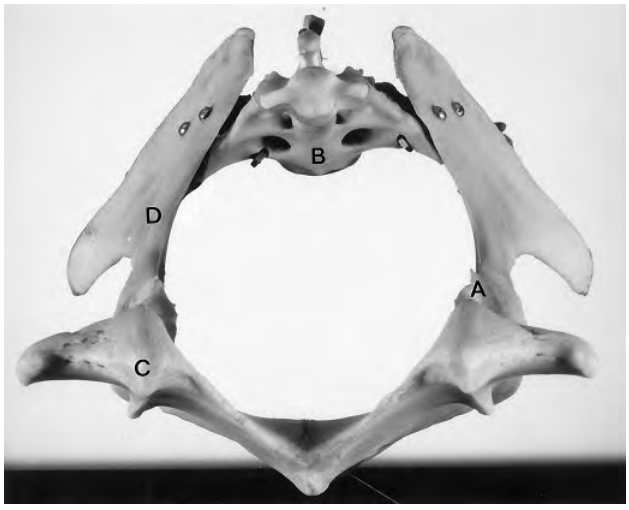

Figure 16.68 Female llama pelvis, caudal view. (A) Ischiatic spine, (B) sacrum, (C) ischium, (D) ileum.

Figure 16.69 Male llama pelvis, cranial view. (A) Ilium, (B) ischiatic spine, (C) pubis.

Figure 16.70 Male llama pelvis, caudal view. (A) Ilium, (B) sacrum, (C) ischiatic spine, (D) ischium.

have noted that the ischiatic spines may be so close that the hand must be inserted in a vertical plane to pass this anatomic obstruction.

Amniotic fluid is scanty, and if the EM has been rubbed off by manipulation, lubrication will be needed to facilitate delivery. Soap is contraindicated, because it is irritating to the mucosa and dissolves surface-protecting oils. Methylcellulose is an excellent lubricant.

In most livestock species, the fetus is likely to impact at the hips. In the llama, the shoulder girdle is more likely to impact. From a normal delivery presentation and position, before the shoulders reach the pelvic canal, it is helpful to twist the fetus 45° and gently pull on one leg at a time. The adult pelvic girdle is basically a vertical rectangle, with the greatest cross-sectional dimension on the diagonal.

The use of obstetric chains is not warranted. Usually, only manual grasping is necessary. If repulsion is necessary, a clean, disinfected lightweight nylon cord may be secured to a limb to allow later access to the limb.

If the hips lock, the fetus should be repelled and twisted on its longitudinal axis 45–90° before traction is applied dorsally. The fetal pelvis is wider than it is deep, and the twist serves to coincide the fetal pelvis with the greatest vertical dimension of the dam's pelvic girdle. Other manipulations are similar to those recommended in cattle and sheep dystocias.

Any of the caudal presentations carry more risk of fetal death with prolonged delivery. The umbilical circulation is quickly compromised because of the caudal position of the umbilicus on the abdomen. It is wise to advise any client who is conducting preliminary obstetric examinations that caudal presentations usually require professional assistance and should be considered emergencies.

Delivery from the caudal presentation must be quick and efficient. There must be adequate lubrication, and the cervix must be fully dilated before traction is applied. If the clinician feels that more than 10 minutes will elapse before the fetus can be delivered, a cesarean section may be preferable.

Many dystocias caused by congenital anomalies require delivery by cesarean section, or in the hands of a skilled operator, fetotomy may be performed.

Uterine Torsion Correction

Uterine torsion occurs in llamas and alpacas. The causes are unknown. The torsion may be in either direction. Ranges of 90–360° rotation in the vagina have been reported. Rectal examination, manual vaginal examination, or a vaginal speculum examination will identify the problem. The primary clinical sign of torsion is repeatedly assuming the sternal position and staying there for a little while, then standing and not eating. The female may look at her flanks and act colicky. The cervix may partially dilate, but relaxation cannot be completed because of mechanical inhibition.

The management of uterine torsion depends on the degree of rotation, the size of the female, and the distress experienced by the female. When rolling the female, she should be put in lateral recumbency on the side where the uterine torsion is directed, i.e. if the torsion is clockwise, the female should be in lateral recumbency on her right side. If the torsion is counterclockwise, the female should be put in lateral recumbency on her left side and correction of the uterine torsion begun (Figure 16.71).

A rectal and/or vaginal manual palpation is conclusive if the uterine torsion has been corrected, or there was an over correction. Rolling the female while pressing against the uterus through the wall of the abdomen with fists or a board may be of help in some cases (Figure 16.71). Some degree of sedation may be necessary. The torsion may be corrected by the female herself through such colicky maneuvers as getting up and down or rolling. One alpaca became upset while being restrained in a chute, reared up, and fell over backward. After she scrambled to her feet, reexamination showed that the torsion had been corrected.[6] Manual maneuvers should not be continued for more than 10–15 minutes. If the torsion persists longer than this, a cesarean section may be necessary to save the life of the fetus and the dam [89]. A practical observation is that after correcting the uterine torsion, the female will get up and go with other females and start eating. Uterine rupture is a possible sequel to torsion.

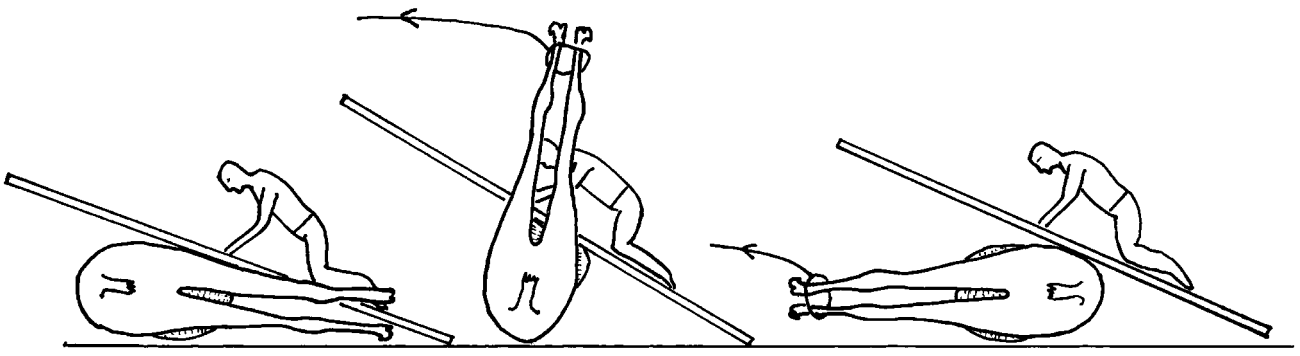

Figure 16.71 Diagram of the mechanical method of correcting a counterclockwise torsion of the uterus.

Uterine Inertia

Uterine inertia is usually caused by prolonged, unproductive effort during stage one or two of parturition. Following correction of malpositioning, neither uterine nor abdominal contractions will continue if the female is exhausted. In such cases, though delivery will be more difficult without the assistance of contractions, it is unwise to administer oxytocin until after the fetus is delivered.

Failure of the cervix to dilate is difficult to differentiate from false labor. A small female carrying a large fetus may experience colicky signs during the last month of pregnancy; the colic is caused by partial obstruction of the digestive tract by the massive uterus. The relaxation of the cervix is under hormonal control, which may be abnormal. Good breeding records, careful observations, and a physical examination are necessary to precisely identify this problem.

Prostaglandins ($PGF_2\alpha$) are the trigger for cervical relaxation and cloprostenol may be administered subcutaneously at a 125–250 mcg total dose. However, if a mistake has been made in the breeding date, this drug will cause abortion. Relaxation occurs within 24 hours following the administration of $PGF_2\alpha$. If the SAC is showing signs of stage one, it may be necessary to manually dilate the cervix. Alternatively, misoprostol, a synthetic prostaglanding E1, can be applied directly onto the cervix and cervical os by crushing a 200 mcg tablet and diluting it with some sterile lubricant. It may take 1–3 hours for the cloprostenol to dilate the cervix. Pelvic stenosis may be caused by abscesses, tumors, or healed but misaligned fractures. A cesarean section may be necessary to relieve the dystocia prior to correction of the basic disorder.

Cesarean Section

This surgical procedure is a valid alternative when manual manipulation to deliver the fetus is impossible, the fetus is too big, or the pelvic canal of the dam is too narrow. The general principles of cesarean section in other livestock species should be followed. Cesarean section is performed with the female in lateral recumbency or in sternal position; a C-section of a llama in standing position has even been reported. The uterus should be incised at the mesometrial angle, avoiding large blood vessels. If available, the use of a vacuum pump is helpful to avoid spilling of fetal fluids within the abdominal cavity of the patient. The placenta may be left inside the uterus, which should be expelled later through the vagina within eight hours after performance of the C-section. In addition, the placenta may be separated for a couple of centimeters along the incision side at the mucosal level of the placenta. This is helpful so the placenta is not caught when the double suture is placed. A person knowledgeable of neonatology should care for the neonate and assist it if it does not begin breathing and/or correct any other lack of vital signs (see Chapter 20).

Pregnancy Termination

The termination of pregnancy in SACs may be indicated for the same reasons as in other livestock. Accidental mating is the most common indication, but suspected prolonged gestation may be considered sufficient reason for the induction of parturition.

Early Gestation. The synthetic $PGF_2\alpha$ cloprostenos, at a total dose of 125–250 mcg, may be administered once IM from six to 100 days following copulation. The six-day lag allows time for the CL to begin formation. SACs that have been administered prostaglandins should be constantly observed for at least one hour to detect reaction. Should the llama become dyspneic or colicky, oxygen should be administered immediately.

Midgestation. In cattle, after 150 days gestation, it is recommended to induce abortion with a combination of dexamethasone and prostaglandin. Dexamethasone, even in doses of 5 mg, caused fetal death with delivery of a stillborn within seven days of administration, and females had a retained placenta. Placenta retention was not observed in $PGF_2\alpha$ administration [90].

Late Gestation. Observations indicate that dexamethasone, estradiol, and oxytocin did not induce parturition within 10 days of the due date. By contrast, $PGF_2\alpha$ was the most efficient way to induce parturition. Delivery occurred within 21 hours after $PGF_2\alpha$ was administered. The primary indication for late gestation termination or induction of parturition is a female suspected of prolonged gestation. The female should always be examined for pregnancy before inducing; clients have been embarrassed to find that the female they thought to be showing all the signs of impending parturition was just becoming fat.

A veterinarian should be cautious about acquiescing to a client's desire to induce parturition. Numerous instances of incorrect breeding dates have been reported. Furthermore, valid 13-month gestations are known. Prolonged gestation, as recognized in cattle, is not known in SACs. The fetus does not overgrow the ability of a normal female to deliver it successfully.

Infertility [91]

Female Llamas and Alpacas

Early breeding management strategies for alpacas and llamas in Peru yielded approximately 70% live births. The strategy used then was similar to that recommended for the management of sheep. The reproductive physiology of SACs is unique, not at all like that of sheep, and sheep methods were not successful with SACs. With better

management, the fertility rates of llama and alpaca herds and the individual animal has markedly improved, but numerous problems remain to be solved. Data are still incomplete on the precise causes of infertility in both males and females.

North American experiences with therapy and management are equally limited. A detailed discourse on these matters would lack factual foundation and simply be an extrapolation from knowledge of cattle, horses, sheep, and goats. Therefore, this discussion only outlines what is known about infertility in SACs and suggests some approaches to the problems. Experimental data is introduced when appropriate.

Etiology

Congenital defects [92, 93] account for much infertility in the female llama and alpaca, both in North America and the Andes. In one study in Peru, one or more anomalies that could compromise fertility were seen [8] in more than 10% of the females examined at slaughter. Buyers should be encouraged to require pre-purchase examinations of breeder animals if they are old enough that such an examination may be conducted. Otherwise, a written agreement relative to congenital anomalies should be part of the buyer–seller contract.

Following is a list of the anomalies that have been seen or reported in the female llama and alpaca reproductive tract: ovarian aplasia, ovarian hypoplasia, tubular hypoplasia, segmental aplasia (oviduct, uterine horn, cervix, vagina), persistent hymen, uterus unicornis, double cervix [92], and intersex [94] (see Chapter 21 for a full discussion). In addition, the presence of numerous small follicles, hypoplastic ovaries, occurrence of cystic follicles, and failure to ovulate compromise fertility. Numerous small follicles resemble a bunch of grapes with no follicle reaching maturity. This anomaly may last for 15 days and then resolve itself.

Hypoplastic Ovaries. Hypoplastic ovaries (less than 10–13 mm) represent 17% of the total anatomic abnormalities. Follicles may grow sporadically, but follicular waves are irregular, and follicles do not mature beyond a size of 6 mm. Estrone sulfate levels are lower than in females with normal ovarian follicles [37].

Cystic Follicles. The presence of cystic follicles, 13–25 mm in diameter, is common in llamas and alpacas [37]. Cysts may persist for an average of 9 days (range 4–15 days). Once a cystic follicle is present, the contralateral ovary is quiescent. Attempts to treat cystic follicles have had variable results. Manual rupture of cysts is not recommended and females may or may not respond to hormonal treatment. Luteinization of follicular cysts can be attempted by administering a GnRH analogue (e.g. gonadorelin, 100 mcg IM) or human Chorionic Gonadotropin (hCG, 1000–2000 IU IV). Breeding females with cystic follicles have variable results. Some females respond to copulation, and LH is secreted; however, luteinization of the follicle ensues, resulting in a short luteal phase (described above). Other females do not respond to copulation, and cystic follicles may increase in size. Erratic sexual behavior of the female may be seen, with some females refusing to permit copulation.

Ovulation Failure. Failure to ovulate occurs in approximately 15% of females. Some develop mature follicles but do not ovulate after copulation or the administration of GnRH [37].

Endometrial Cysts. Endometrial cysts are rare. Ultrasonography indicates they are irregularly shaped, hypoechoic structures, and randomly located in the endometrium. Females with large endometrial cysts do not become pregnant, even when ovulation occurs.

Failure to Maintain a CL. Failure to maintain a CL has been recognized as a clinical entity, and exogenous progesterone are being used to support pregnancy, with some reported successes. Synchromate B is a progesterone product used to synchronize estrus in dairy cattle, and it has been administered to llamas that continue to suffer early embryonic loss. To avoid perpetuation of poor reproductive performance in a possibly genetically inferior female, the product is recommended only for females that have successfully carried a fetus to term. An implant is inserted with a special needle gun at the base of an ear. Three weeks later another implant is inserted in the opposite ear, and three weeks after that, the implant from the first ear is replaced. The procedure is repeated every three weeks until a fetus is delivered.

A second source of exogenous progesterone is Hydroxyprogesterone caproate (commonly known as Hypoval), which is administered IM. This is a good alternative to maintain pregnancy in a female that is diagnosed pregnant by ultrasonography and that has generally lost the pregnancy within the first month of gestation. The caveat of using this drug is that when used up to term it sometimes induces prolonged gestation and may result in a dead fetus that must be delivered by C-section. Records of a clinic (Walter Bravo) indicate that of 12 females given an exogenous progesterone supplementation to maintain pregnancy, five delivered a dead fetus after the pregnancy was prolonged beyond a year, four of which required a C-section to deliver a dead fetus. Exogenous progesterone should only be administered up to 10 months of pregnancy, under strict veterinarian supervision. The role of a veterinarian is critical. Some owners who opted to administer exogenous progesterone to all females that failed to

conceive induced a serious disturbance of the ovarian cycle of the female. The author (Bravo) observed that 10 females did not resume ovarian activity for two to three months after an indiscriminate administration of exogenous progesterone. Most females do not need exogenous progesterone. Before a decision is made to administer exogenous progesterone, US examination should confirm the presence of an embryonic vesicle and most important, weekly doses of progesterone should be less than 0.7 ng/mL.

An important side note is that synthetic progesterone in the commercial product is not detected by standard laboratory progesterone analysis; thus, the only way to monitor effectiveness of treatment is the use of US periodically to confirm the presence of embryonic vesicle, embryo, fetus, or fetal components.

Miscellaneous Conditions. Physical abnormalities should be noted. Tilting of the vulva (shelving) may be seen in old, emaciated, or poorly conformed females. The labia may lack tone. Nothing is really known as to how or whether these may contribute to infertility. They are of concern in the mare or cow and can lead to recurrent uterine infections.

Inflammation of the reproductive tract includes vaginitis, cervicitis, metritis, endometritis, and pyometra. True venereal diseases are rare in SACs. Brucellosis (*Brucella melitensis*) has been reported in South America but not in North America.

The normal flora of the vagina and uterus is not known. Numerous species of bacteria have been cultured from infertile females, but Koch's postulates have not been demonstrated [95, 96]. Perhaps, as in other females, the organisms are opportunistic pathogens. It should be kept in mind that in about one-third of the infertile females cultured, samples show no bacterial growth simply because the cause of infertility is not a uterine infection, but rather some other disturbance involving the ovary or even the pituitary. This may either be accurate or a result of technique error.

In the clinic of the author (Walter Bravo) some years ago, cases of acute and peracute metritis were usually associated with a hemolytic *Escherichia coli* infection. *Arcanobacterium pyogenes* was the second most prevalent organism cultured. In 2006, the bacteria most often isolated from uterine culture were *E. coli*, *Enterococcus* spp., and *Klebsiella* spp. In 2007, 55% of isolates were *Pseudomonas aeruginosa*, 18% *Enterobacter* spp., 9% *E. coli*, and 9% *Streptococcus* spp. The remaining 9% yielded no bacteria. In 2008, 51% were *P. aeruginosa* and *Pseudomonas fluorescens,* 11% *E. coli*, 6% *A. pyogenes*, 6% *Streptcoccus* spp., alpha hemolytic, 6% *Proteus* spp., and 20% did not yield any bacteria. More than 50% of isolates are Pseudomonas. This bacterium is a normal inhabitant of the reproductive flora, but it may become virulent and wreak havoc in the endometrium. Unfortunately, it is resistant to most antibiotics. Some cultures showed sensitivity to Amikacin, Gentamycin, and Enrofloxacin, but others showed resistance to at least one of those antibiotics. Thus, a treatment protocol should be delayed until results from the laboratory are available.

After treatment with the appropriate antibiotics, a second culture may show the continued presence of Pseudomonas, requiring a second round of treatment. It is recommended to rest the female for two to three weeks following treatment to allow the uterine defense mechanism to rejuvenate.

Abortion

Abortion is defined as the loss of a fetus from 45 days gestation to term [97]. Prior to 45 days, the loss of a conceptus is considered embryonic mortality [98–100].

Signs of Abortion. Signs of abortion include a placenta protruding from the vulva, presence of a dead fetus in a paddock or pen, or indications of receptivity to a male.

Prevalence. In general, the percentage of abortion after 60 days gestation is about 2%. Recently, a study in New Zealand reported a 5% fetal loss after 100 days of gestation. In a separate study, a 10–17% loss was observed in alpacas.

Causes. Infectious causes reported include *B. melitensis*, listeriosis [101], chlamydiosis [102], toxoplasmosis [102], leptospirosis [102], and equine rhinitis virus [103]. Additional causes include stress, hormonal imbalances, and administration of corticosteroids [90, 104]. Dozens of other causes of abortion have been identified in horses and livestock, and given time, SACs will likely become the victims of many of these.

Unfortunately, a precise diagnosis will be made in less than 50% of abortions. Nonetheless, every effort should be made to collect samples and begin recording data.

Treatment. Every female that aborts should be examined to exclude an intrauterine infection. In most early gestation abortions, no special therapy is indicated.

Late-term abortions may require more attention. Often, there is a discharge from the vulva that may vary from odorless to foul smelling. A foul-smelling exudate may be associated with necrotic tissue. If the placenta or any part of it remains in the uterus, 20 IU of oxytocin should be administered, followed by voluminous flushing with physiologic saline. Antibiotics (parenteral and intrauterine) may be indicated.

Infertility Examination

It is vitally important that systematic, thorough examinations be conducted and that detailed records be kept. It is only through such records that it will be possible to amass sufficient data to serve as a sound basis for rational evaluation. Table 16.5 is a suggested examination form.

Table 16.5 Llama reproduction examination – female.

Identification Date _____

Name_____ Age _____ Owner _____

ISIS No. _____, Llama Registry No. _____

Color Body _____ Neck _____ _____ Head _____

Legs _____

History

Previous pregnancies—0, 1, 2, 3, 4

Life births—0, 1, 2, 3, 4

Abortions—0, 1, 2, 3

Breeding dates during past 6 months _____ _____ _____

Number of males used _____

Fertility of male—known _____ unknown _____

Response to male, past 2 months _____ _____ _____

Previous medical problems _____ _____ _____

Previous fertility treatments and response _____ _____ _____

Fertility rate of the herd _____ _____ _____

Rectal examination

Cervix diameter _____ cm, length _____ cm

Cervix tone _____

Right horn

Diameter _____ cm, length _____ cm

Left Horn

Diameter _____ cm, length _____ cm

Consistency _____

Right ovary _____ × _____ × _____ cm

(*Continued*)

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Table 16.5 (Continued)

Left ovary _____ × _____ × _____ cm structures present

Pregnant _____ yes, _____ no, _____ months

Vaginal examination

Vulva

Conformation _____

Discharge _____

Hymen _____

Vaginal mucosa

Color _____ Exudation _____

Cervical os

Cytology _____

Exudation _____ _____ _____

Cervical culture _____

Uterine culture _____

Uterine cytology _____

Uterine biopsy _____ ____

Hormone assay _____ Progesterone _____ ng/dL

Ultrasound evaluation _____ _____ _____

Other diagnostic procedures—laparoscopy, laparotomy _____ _____ _____

Conclusions _____

The exam should be preceded by a detailed history. Unfortunately, infertile females are frequently shifted from one owner to another, often without the new owner being aware of the problem. It has been reported that unscrupulous sellers have sold females without ovaries with a claim that they previously had multiple crias.

The vaginal exam may be conducted using a heifer glass vaginal speculum (2.5 cm outside diameter, 25 cm long). Specula constructed for use in goats are too short to reach the cervix (15 cm). In larger and multiparous females, a standard equine glass or cardboard speculum may be used, but it is longer than necessary (4 cm in diameter, 25 cm long). A piece of PVC plastic pipe of suitable diameter may be smoothed off on the end or fired and used as a speculum after sterilization. A last alternative is the use of a human rectoscope which accommodates itself very well to the vulva and vagina of the female alpaca. (Figure 16.72) This vaginal speculum could be attached to a light source to facilitate examination of the cervix. It should be remembered that the cervix opens when there is an ovulatory-sized follicle in the ovary, and then remains closed. A swab culture from the cervix should be taken when the cervix is open, rather than forcing the insemination rod and/or the tip of a swab culture and damaging the cervix. In some cases of uterine infection, the cervix is closed and there is no discharge from the uterus. It is wise to wait until the cervix opens to observe the nature of the discharge. If discharge is seen, its presence may be noted on the ventral aspect of the female's tail. Any thick, yellowish dry matter should be removed, because during the next examination, if the tail has the yellowish matter, the female is still infected.

It is possible, in multiparous females examined by a small-handed person, to manually palpate the vagina after suitable cleansing and gloving. Rectal palpation is a valid and necessary procedure for evaluation of the reproductive tract. It can be done safely on all but the smallest females and virginal alpacas. Obesity greatly restricts movement in the pelvic canal and may preclude an adequate examination. A detailed description of rectal palpation has been given previously. It is unfortunate, but some owners have been swayed by exaggerated rumors of the dangers of rectal palpation and will not allow such an examination to be made. It may then be impossible to evaluate an infertile female and recommend a course of action.

Figure 16.72 A rectoscope and light source is pictured here. The rectoscope is ideal for performing a vaginal examination on camelids.

Ultrasonography is a valuable aid not only for pregnancy diagnosis but also for conducting the infertility examination. The rectum should be lubricated with the appropriate gel to facilitate the conductivity of US waves. A systematic and organ-by-organ US should be conducted. The urinary bladder is visualized, and immediately the presence of urine as a hypoechoic structure serves as an initial landmark. As the probe is gently advanced cranially, the cervix becomes apparent, and if the animal is pregnant, the cervix shows two or three distinct hyperechoic areas (rings of the cervix). Then, the body of the uterus is visualized, and by moving the probe laterally, both uterine horns should be visualized. The uterine horns appear as round structures which are hypoechoic and homogenous. The diameter of the uterine horns should be observed. In maiden females, the diameter should be approximately 4–5 cm. When there is infection in the uterine horns, the presence of discrete and irregularly shaped hyperechoic structures are apparent. In addition, there are lymphatic vessels of irregular shape at the bottom of the uterine body. In the early postpartum female, lymphatic vessels are pronounced and manifested. Ovaries are usually located on the side of the uterine horns, but sometimes they are in the center. The presence of follicles, which are round, fluid-filled structures appear hypoechoic and serve as a basis to identify the ovaries. (Figure 16.73) The presence of a cystic follicle (greater than 13 mm in diameter) is unmistakable. Large, cystic follicles can pull the ovaries ventrally into the abdominal cavity and make it challenging to find them via US.

A corpus luteum will appear as large as the ovary itself, and as a clear hypoechoic rounded structure at the periphery of the ovary (Figures 16.74 and 16.75) When the female is losing its embryo, the embryonic vesicle appears with irregular hyperechoic structures, and the delimiting outside border is disrupted and not well defined.

Uterine cytology [33], endometrial cultures, and sensitivities are vital for selection of proper therapy for metritis. An equine uterine culture swab is recommended. It is useless, in fact misleading, to obtain a culture from the vagina or external os of the cervix. The cervix relaxes only when a follicle is present in the ovary, and therefore it is difficult to thread a tube through the cervix, but by gentle persistence it can be done and allows access to the uterus for proper location for obtaining a culture sample.

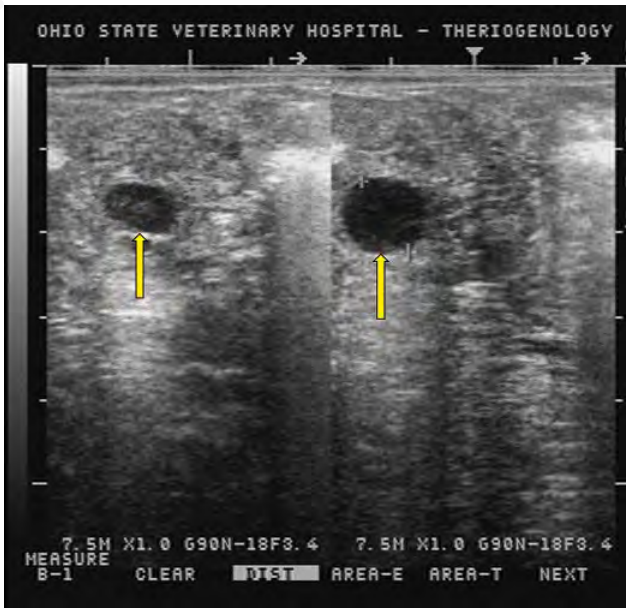

Figure 16.73 Ultrasound image of a mature follicle on an alpaca's ovary.

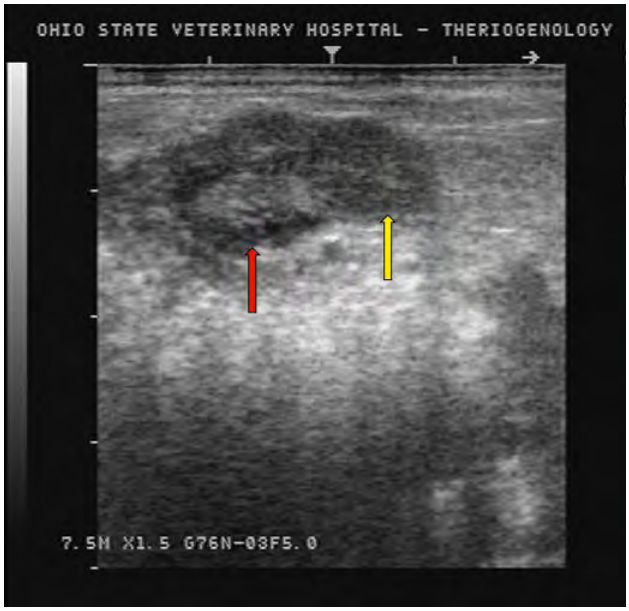

Figure 16.74 Ultrasound image of a corpus luteum (yellow arrow) on an alpaca ovary (red arrow).

Three approaches have been described for sampling uterine endometrium for cytology, histopathology, or culture. A small-handed person may enter the vagina with a gloved hand. A finger should be used to locate the cervical os and begin dilatation. The double-guarded swab should be inserted alongside the arm, up to the finger, and gently pushed forward with periodic changes of direction to move

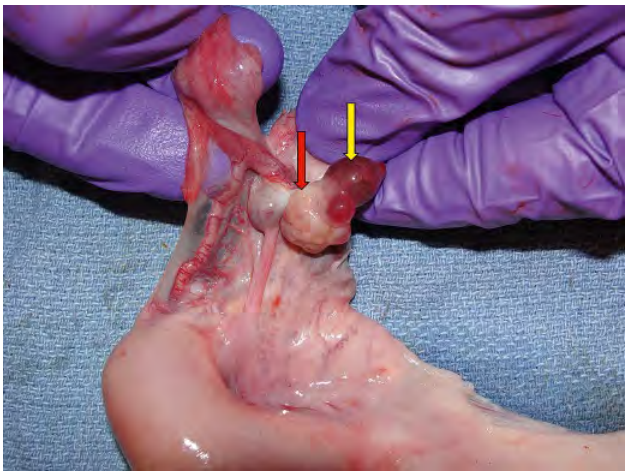

Figure 16.75 Gross image of a corpus luteum (yellow arrow) on an alpaca ovary (red arrow).

over the rings. In another method, a short vaginal speculum is pressed around the os of the cervix. The culture swab is inserted through the os and the manipulation continued by visual contact, or the speculum may be withdrawn and allowed to rest on the tube while the other hand is inserted into the rectum to grasp the cervix to aid in the threading process. There is usually insufficient room for both a speculum in the vagina and an arm in the rectum in the SAC. Endoscopy is now being used to guide pipettes and swabs, and hysteroscopy can be used to visually examine the endometrium and collect diagnostic samples [105].

Cultures should be interpreted in relation to the cytology. If no inflammatory cells or discharge are present, the organism cultured may not be a pathogen.

Endometrial Biopsy. The histologic evaluation of the uterine mucosa has become a standard diagnostic technique in mares and cows suffering from infertility. Much less experience has been gained in SACs, but the technique and histologic picture of the endometrium are known. The technique is similar to passing the swab for a culture. The tip of the biopsy forceps must be threaded through the cervix and, with a hand in the rectum to fix the uterus, a snip of tissue may be pinched off the mucosa. Some of the larger equine forceps have jaws that may be too large for an SAC; consequently, a heifer forceps is most appropriate for llamas and alpacas. Injudicious use may penetrate the uterine wall. Cultures can be obtained from the biopsy tissue before placing it in fixative.

Each clinician should be consistent in the general location from which the specimen is collected. Unless a uterine mass must be biopsied directly, the lateral wall of the left horn is an appropriate location.

The uterine mucosa consists of an epithelial layer of tall columnar cells and a connective tissue layer containing the glands. The density of the connective tissue is greater and the number of glands in the mucosa much less in an SAC than in a mare, leading to misinterpretation by inexperienced pathologists. Other lesions of inflammation are similar in both species.

Pigmented macrophages have been observed in the endometrium. This may be suggestive of abortion, parturition, or other causes of hemorrhage, as it is in mares. Fibrosis of the uterine glands is an unfavorable lesion. SACs do not cycle as most mammals do; therefore, a variable histologic picture associated with stages of the estrous cycle cannot be described.

LAPAROSCOPY [106, 107]. Laparoscopy has been used effectively in experimental work to document ovarian activity during various stages of the reproductive cycle [108]. It is possible to visualize the ovaries, oviducts, and uterine horns via a right-flank approach, with the animal in a standing position. Mild sedation with butorphanol and local anesthesia is used. The abdominal cavity must be inflated with an inert gas, and the distention may cause discomfort in the patient.

In the male, intraabdominal vasectomy may also be done using laparoscopy. The male is fasted for at least 24 hours. A general anesthetic is administered, and then the area ventral and lateral to the penis is prepared aseptically. The male is positioned in Trendelenburg position, with the rear legs up and the head down. Two incisions are made; one is used to insert a trocar cannula which is then replaced by the rigid telescope. The other is for a biopsy and/or cutting forceps. The abdominal cavity is insufflated moderately until the vas deferens are located on the sides of the male. They are white and glistening. Using the cutting forceps, a 2-cm segment of each vas deferens is removed. Surgical material is withdrawn, air is pushed out by manual pressure, and the two skin incisions are sutured with simple interrupted sutures [107, 109].

HORMONE ANALYSIS. Progesterone levels have been used extensively to determine pregnancy in llamas, but it should be reiterated that positive levels only indicate the presence of a CL. Repeated or periodic progesterone samples can be helpful in evaluating certain infertility problems such as recurrent early embryonic death. Levels of progesterone have been detected in milk and urine.[7] Estrone sulfate and relaxin have also been studied in pregnant llamas and alpacas. Estrone sulfate shows a bimodal peak, the first during the first 21–27 days of gestation and the second during the last month of pregnancy. Relaxin can also be an indicator of pregnancy after month two of pregnancy, however, there is no commercial assay for determination of relaxin in camelids. (see description under Pregnancy Determination).

Treatment of Infertility and Metritis

Techniques currently being used to treat infertile mares and cows are being applied to SACs. Statistics are not available as to the efficacy of various therapies.

Sexual rest is an important management tool. Animals that have experienced any difficulty with parturition should be given at least 30 days for optimal uterine involution before being exposed to a male.

Various hormones have been administered to livestock and horses to correct deficiencies based on actual measurement of hormone levels or on the basis of clinical signs and palpation examinations. No baselines have been established in llamas or alpacas for most of the hormones, so hormone therapy must be based on extrapolation from regimens developed for other species. Before these drugs are prescribed, it is important to be certain that the basic reproduction cycle of SACs is understood. It is important to use caution in the dosage of hormones.

Discussion of therapy for metritis usually includes a list of antibiotics that have proven useful. Such a list is inappropriate for SACs because no consistency of therapy has been established. Clinicians in different regions of North America administer the antibiotics they use in mares or cows, but the data have not been correlated to determine efficacy.

The trend is to minimize the use of uterine flushes with disinfectants or antimicrobials and instead to use physiologic saline to thoroughly wash out all exudate. One therapeutic regimen is to flush the uterus as described, finishing with a uterine infusion of llama plasma.

Infertility Conditions of Female Camels

Conditions causing infertility and treatments of those conditions are similar among all camelids. Tibary [80, 110] discusses the following conditions in detail: ovarian hypoplasia, ovarian bursal adhesions, ovarian neoplasia [111], laceration of the cervix, rectovaginal fistula, rectovaginal prolapse [112], uterine infection, retained CL, endometritis, early embryonic death, cystic ovaries, ovarian inactivity, uterus unicornus, infantile uterus, vaginal prolapse [113], retained placenta, uterine prolapse, and vulvar and vaginal lacerations.

Male Llamas and Alpacas

Etiology

Little investigation has been carried out to delineate the various causes of infertility in male SACs. Congenital defects are some obvious causes and include such conditions as cryptorchidism, ectopic testicle, hypogonadism [114], persistent frenulum, prepucial adhesion, small penis, curvature of the penis, and intersex.

HYPERTHERMIA. See Chapter 8 for a detailed discussion of this topic.

HYDROCELE. The author has seen two llamas with scrotal hydrocele. In one case, the cause was an abscess at the external inguinal ring, obstructing fluid flow into the peritoneal cavity. In the other case, the etiology was not determined. Sterile, normal fluid was contained between the visceral and parietal tunics. There was no evidence of an inflammatory response. US examination confirmed the presence of nonpurulent fluid. It is important to differentiate hydrocele from scrotal edema, which is a characteristic sign of hyperthermia in male SACs.

TRAUMA. The prepuce, scrotum, and testicles may be traumatized during aggressive intermale encounters. The wounds should be managed as any other lacerations. Unattended lacerations of the prepuce may result in posthitis and, ultimately, stenosis and phimosis (inability to extrude the penis).

A previously successful breeding male began to fail to complete copulation. He chased the females and assumed his usual breeding position, but at the time intromission should have occurred, progress stopped, and he arose and left. Upon examination, it was observed that a stricture prevented full extension of the penis. Attempts to forcibly extrude the penis elicited a pain response, even though the llama was sedated. This male exhibited a strong libido, but because erection had become painful, he simply quit.

Another source of trauma to the penis is entangling fibers from the female's perineal region. North American llama owners are reluctant to clip fleece from their animals, but the perineal area of all breeding females should be clipped sufficiently to preclude fibers from obstructing copulatory efforts. One or two caught fibers may lacerate the glans and cause balanitis. If fibers wrap around the glans and become twisted, ischemic necrosis may occur.

Prepubertal adhesions may persist into adulthood [10]. Stenoses of the prepuce are serious impediments to successful breeding; surgical correction is usually necessary. Standard techniques used on bulls have been successfully employed in SACs and may include multiple triangular release reconstruction surgery or removal of circumferential collar and resuturing of the preputial mucosa. Infectious diseases of the male reproductive system are either rare or have not been reported. Brucellosis, caused by *B. melitensis*, has been reported in South America but not in North America. Trichomoniasis and campylobacteriosis are unknown. Two cases of breeding males with Pseudomonas infection have been observed by Bravo. In both cases, a female was found infected with the same bacteria. Lavage with antibiotics was done in the following manner: The antibiotic was diluted with saline solution and 50 cc of the mixture was placed inside the prepuce. The opening was held tight for about five minutes. This procedure was repeated for five consecutive days. A second culture from the glans penis was negative to bacterial infection, and the male returned to the breeding program.

No specific abscesses have been observed in the testicle. Infections are frequently a sequel to lacerations and severe contusions.

HORMONAL DEFICIENCIES. Hypogonadism is likely the result of low levels of testosterone, but little is known about normal levels in male SACs. Certainly, some males lack libido, but whether this is caused by a hormonal defect or is a behavioral defect is unknown.

Hormonal treatment of hypogonadal testicles in adult males is questionable. If the testicles are small, there could be a genetic component which makes the male with small testicles a poor breeder prospect. It should be also considered that in South America only 5% of the males are selected as sires, which means that only outstanding males in every sense of the word should be sires. The same pressure of selection has been used for many years in other livestock species. Moreover, there is a relationship between fertility and testicular size.

BEHAVIORAL ABNORMALITIES. Males may be overly timid or hyperaggressive. Such behaviors may be inherited, as in other livestock species, or they may be developed. A young male just starting to breed may be intimidated by an aggressive female. The major known behavioral anomaly is that of the human-imprinted (rogue) male (Chapter 2). Such behavior does not make the male infertile, but it may preclude his usefulness as a breeding male because of the danger to humans who must handle him.

Infertility Examination

A fertility exam should begin with a complete history and a thorough physical exam to identify problems that may preclude breeding, such as arthritis, or that may inhibit mounting and copulation. In the fertility exam, the use of a chart such as that illustrated in Table 16.6 facilitates completeness. The individual should be identified by registration number (the number should be seen) or by color and markings.

Table 16.6 Llama reproduction examination – male.

Identification Date _____

Name _____ Age _____ Owner _____

ISIS No. _____, Llama Registry No. _____

Color

Body _____ Neck _____

Head _____ Legs _____

History

Born at present ranch—yes _____, no _____

Age at time of purchase _____

Pastured or housed alone—yes _____, no _____

Number and sex of other animals in the group _____

History	**Prior to purchase**	**After purchase**
Number of crias sired	1, 2, 3, 4, 5, +	1, 2, 3, 4, 5, +
Number of females bred	1, 2, 3, 4, 5, +	1, 2, 3, 4, 5, +
Age at time of first breeding	1, 2, 3, 4, 5	1, 2, 3, 4, 5

Physical examination

General body condition Good _____, thin _____, fat _____

Conformation faults _____

Testicles _____

Right _____ × _____ × _____ cm

Left _____ × _____ × _____ cm

Consistency _____ firm _____ soft _____ hard _____

Nodules _____ Other _____

Epididymus _____

Abnormalities _____ Cryptorchid—yes _____ no _____

Penis

Length _____ cm, diameter _____ mm

Abnormalities

Preputial adhesions _____, curvature _____

Semen evaluation

Volume _____ mL, color _____

pH _____, density _____

Concentration _____ million/mL

Motility _____

Live/dead _____ %

Abnormal forms _____

Behavior

Hand reared—yes _____, no _____

Aggressive to people—yes _____, no _____

Precopulatory behavior

Aggressive _____, timid _____

Copulatory behavior

Proper position—yes _____, no _____

Penetration—yes _____, no _____

An exam for breeding soundness varies somewhat according to the age of the animal and the reason for the exam. An exam for sale of a breeding male should encompass a complete set of parameters. The exam of a weanling or yearling as a potential breeding male must, of necessity, be limited. The exam of a male with identified or suspected fertility problems is complex.

Breeding males may also be examined for insurance purposes. The veterinarian must ascertain whether the insurance is for mortality only or if the client has contracted for breeding assurance, in which case, much more detail is required in the way of exam and evaluation.

History is vital. Has the male bred previously? Has breeding resulted in living crias? Is he aggressive or shy? Is he intimidated by dominant females? What is the status of his libido? It would be desirable to see the male actually breeding a female, but most of the history must be obtained by judicious questioning of the owner. The owner may or may not have been able to determine whether or not the male has made penetration.

A thorough exam of the male reproductive organs should be conducted, including evaluation of the testicles for size and consistency. The testes should be evaluated by ultrasonography and measured. The epididymis should be palpated. Measurements of the penis can be made only with the penis extruded from the prepuce. This requires tranquilization or anesthesia. Some information may be obtained by palpation through the skin. Rectal palpation should enable exam of the accessory sex glands, especially the bulbourethral glands.

Semen Collection [17, 115, 116]. A complete fertility examination should include semen evaluation. This is difficult in SACs, since no method has been effective in collecting semen from every animal. AVs have been employed to collect semen from stallions, bulls, rams, and alpaca males. Males must be trained to use the dummy female, so this method is applicable only for research projects.

Electroejaculation has been of limited effectiveness. The male must be sedated and even lightly anesthetized. Various types of ejaculators have been used. The exact power, pulse frequency, and duration required have not been established. In the author's clinic, a variable-voltage bovine unit has been used, attached to a three longitudinal electrode ram probe. Before proceeding, the feces should be removed from the rectum. The technician must be prepared to collect semen at this point and also when the probe is inserted, since either stimulation may cause premature ejaculation. It is most desirable to manually extrude the penis from the prepuce prior to any rectal stimulation, so that semen may be collected from the penis rather than from the prepuce. Contamination of semen with urine and absence of a well-developed ampulla are factors that make electroejaculation less than optimal as a technique for semen collection.

Under clinical situations, the most reliable and effective method of semen collection is retrieving it from the vaginal fundus. This method takes advantage of three features of breeding. First, ejaculation is constant throughout copulation. Ejaculation begins after two minutes of penis insertion into the female reproductive tract, and ends right before male withdrawal. Second, semen is gelatinous and seminal plasma holds spermatozoa. Third, semen is also left outside the uterus when the male is repositioning between the two uterine horns. Hence, following copulation and with the aid of a warmed vaginal speculum and a warmed insemination pipette attached to a syringe, semen may be retrieved from the posterior os of the cervix. The evaluator must take into consideration that sperm has been in contact with the female reproductive tract and its secretions, which can affect sperm quality. A phase contrast microscope and a prewarmed glass slide are needed to determine motility. Then, the preparation may be stained for live/dead evaluation and morphology.

Concentration is difficult to assess but not impossible. Moreover, when retrieving semen from the female's vagina, the concentration may be meaningless since the total volume of ejaculate cannot be determined. Motility, live/dead, and morphology of spermatozoa may be determined. The presence of large numbers of red blood cells may impair a fair assessment of sperm morphology. Live spermatozoa can be determined using a drop of Hancock's stain. Table 16.7 collates the results of examinations of 22 alpaca and 8 llama sires.

Statistics are unavailable on the desirable ratio of normal to abnormal sperm, but in a fertile male, at least 70% of the sperm should be normal.

The morphology of the sperm should be evaluated at 1000×. It may be necessary to dilute the semen to separate the seminal fluid, which can be done during the staining process by mixing the sperm with a larger volume of stain A prior to making the smear. A variety of stains may be used.

Evaluation of the motility of SAC sperm in the viscid semen may be misleading. SAC spermatozoa are much slower than those of rams or bulls because of the thick seminal fluid. It is imperative that the semen be maintained at a constant temperature of 37 °C from the time of

Table 16.7 Sperm analysis of 22 alpacas and 8 llamas.

Species	Motility (%)	Live spermatozoa (%)
Alpacas	68 (50–90)	85 (70–95)
Llamas	71 (50–90)	78 (68–90)

collection until motility is evaluated; otherwise, motility should not be considered in the fertility examination.

Assisted Reproductive Techniques [17, 25, 117, 118]

Progress has been achieved in some assisted reproductive techniques (ART), such as synchronization of ovarian follicular development, ovarian superstimulation, and embryo transfer (ET), while others present fewer advances, for example, artificial insemination (AI), especially when using cryopreserved semen, *in vitro* fertilization (IVF) and intracytoplasmic sperm injection (ICSI) [25, 118–120].

INDICATIONS. AI is used primarily to increase the production of crias from a clearly superior male. It may also be used in cases of physical incapacitation of the male or female.

APPLICATION IN SACS. AI was first performed in Peru as early as 1966 [121, 122]. Subsequently, AI has been investigated as a possible adjunct to natural breeding in both North and South America [119, 123, 124]. The question as to the characteristics of a clearly superior SAC remains unresolved. The following account is a summarized discussion of the state of AI in llamas and alpacas in Peru, and mimics the procedure of semen extension and preservation as has been developed in other livestock species.

Semen degelification was achieved using mainly two enzymes, trypsin and collagenase. Semen dilution has also been accomplished with phosphate buffer, egg-yolk citrate, skimmed milk, and Tris buffer with egg yolk. The best extender is Tris buffer with egg yolk citrate. Spermatozoa were maintained alive for more than 24 hours under refrigeration. Glycerol has been used successfully for semen freezing and a concentration of 7% of glycerol is adequate. Semen was also frozen successfully using vapors of liquid nitrogen but freezing must be done slowly. A rapid freezing killed spermatozoa. The survival rate of spermatozoa in frozen–thawed semen is between 30 and 50%. Insemination with fresh diluted semen and thawed semen have both resulted in the birth of live crias. Semen preservation requires considerably more experimentation and improvement before AI becomes practical.

Embryo Transfer

INDICATIONS. ET is primarily valuable for enhancing the production of a superior female. It is suggested that female recipients could be of poorer quality or could have nonreproductive system congenital defects.

APPLICATION IN SACS. ET was first attempted by surgical recovery of embryos in 1968 by Novoa and Sumar. Subsequently, a number of investigators have used both surgical and nonsurgical procedures with variable success [123–132]. Jane Vaughn in Australia, in 2008, succeeded in producing more than 1000 crias through ET with fresh embryos. In this case, embryos were collected after breeding and without manipulation of the ovaries. Recently, ET has became more popular in North America.

Protocols to induce production of multiple follicles in the ovaries are many in other livestock species. In llamas and alpacas, follicle stimulating hormone (FSH) and eCG have been used effectively. Embryos have been collected surgically as reported by Novoa and Sumar, but the best option is to flush the uterus with a catheter inserted through the cervix (Figure 16.76). In this sense, it is easy to make the ovaries grow multiple follicles (Figure 16.77); these follicles will also ovulate after breeding with an intact male plus the administration of either gonadorelin of human Chorionic Gonadotropin. Freezing embryos is being reported by scientists not only in South America but also in other countries of the world. *In-vitro* maturation, fertilization, and culture of SAC oocytes have been also explored. Protocols from other species have been extrapolated to llamas and alpacas. It is an exciting technology and the future will bring tangible results of the application of these technologies for llamas and alpacas. At the moment, these techniques produce unacceptable results for commercial use.

Figure 16.76 Embryo transfer flush is being performed on this sedated alpaca.

Figure 16.77 This alpaca ovary has multiple corpora lutea as a result of multiple ovulations.

Selection of Breeding Camelids

A prepurchase exam of a breeding animal should include a complete soundness examination, with special emphasis on the reproductive system. A veterinarian is likely to be restricted to noninvasive procedures, but the checklists provided should guide the clinician in obtaining pertinent information (Tables 16.8 and 16.9).

Reproductive Surgery

Cesarean Section

A cesarean section is indicated when the fetus is unable to traverse the birth canal. The fetus may be enlarged (emphysema or anasarca) or malformed (ankylosis of carpus or tarsus, schistosomas reflexus).

If an immature female is bred accidentally (cases are on record in which a female has become pregnant at five months), parturition will occur before the pelvis and the genital tract have reached maturity. The cervix may fail to relax and dilate properly, or the uterus may clamp around the fetus so tightly that forced extraction will rupture the uterine wall. A fetal mummy lacks flexibility, and the cervix may not relax sufficiently to allow expulsion of a mummified fetus.

Narrowing of the birth canal may occur as a result of previous trauma, causing fibrosis and stenosis, or from pelvic tumors. Correction of an acquired vaginal stenosis may result in a passageway that allows penetration by the penis, but not sufficient space to allow natural delivery. Uncorrected uterine torsion mandates a cesarean section. In addition, prolonged manipulation may jeopardize the life of the fetus, and a cesarean may be appropriate to obviate the threat.

The surgery may be performed either via a left flank approach or on ventral midline [133]. Dorsal recumbency is required for the ventral midline approach. For a flank incision, lateral recumbency with the animal lying on its right side is appropriate. General anesthesia or heavy sedation is highly recommended.

Left_Flank Approach. Because more than 95% of camelid pregnancies are in the left horn, there is a gravitational twist counterclockwise that pulls the uterus toward the right side. If the right side is selected, it is more difficult to exteriorize the uterus for extracting the fetus because the uterus is already pulled toward the right side. Furthermore, if the uterus contracts during suturing, the uterus will pull away from the right incision. The low-flank area is devoid of muscle mass and should be avoided. A midflank incision is appropriate. There is no greater omental sling to bypass as in cattle, so it is easy to grasp and exteriorize the pregnant uterine horn.

The uterine wall should be incised on the greater curvature, avoiding large vessels. The fetal membrane arrangement in camelids differs from that of other artiodactylids. With a cranial presentation, the hind limbs are encountered first in the uterus; they may be grasped to deliver the fetus. A clamp should be placed on the umbilicus. The uterine-chorial attachment is minimal, and it may be possible to deliver the placental membranes through the uterine incision. If much pressure must be exerted to free the placenta, it should be left *in situ* for later vaginal delivery. The second horn of the uterus should always be examined for the presence of a twin. Any uterine torsion should be corrected and the uterus palpated externally for possible uterine rupture.

The uterine incision may be closed with one or two layers using a Lembert or Cushing inverting suture pattern and any absorbable suture. The uterus should be replaced in the abdominal cavity and the cavity flushed with physiologic saline if there has been contamination with infected fetal fluids. The presence of uninfected amniotic or allantoic fluid in the peritoneal cavity is not harmful.

The abdominal wall may be closed as for a laparotomy.

Ventral Midline Approach. The initial incision should be made from the cranial border of the mammary gland forward for a distance of 30 cm. The operation may then proceed as for the flank approach.

Retained placenta, metritis, endometritis, peritonitis, and herniation at the incision site are potential complications. If dystocia was prolonged, or significant manipulation occurred before the decision to perform a cesarean section was made, it may be necessary to treat for

Table 16.8 Checklist for a female intended as a breeder.

Breeding History

Previous pregnancies: 0, 1, 2, 3, 4, _____

Live births: 0, 1, 2, 3, 4, _____

Date of last birth _____

Abortions/Stillbirths: 0, 1, 2, _____

Breeding dates during past months _____

Number of males used _____

Fertility of male: Known _____, unknown _____

Response to male _____

Previous medical problems _____

Previous fertility examinations, treatments and response: _____

Fertility rate of the herd _____

Noninvasive examination

Conformation of the vulva

Vertical _____, horizontal _____; Prominent clitoris _____

Discharge from vulva _____

Hymen: Present _____, absent _____

Mammary gland

Number of teats present _____, size of teats _____

Palpable swelling _____

Secretion from gland _____

Pregnancy status

Pregnant _____, nonpregnant _____

Method of determination: Rectal palpation _____, ultrasound _____, progesterone _____

Detailed examination

Rectal examination

Cervix: Length _____ cm

Right horn

Diameter _____ cm, length _____ cm

Left horn

Diameter _____ cm, length _____ cm

Tone of uterus _____, pregnant: yes ____, no ____

Right ovary

Size _____ cm; structures present _____

Left ovary

Size _____ cm, structures present _____

Ultrasound examination

Pregnant: yes _____, no _____

Abnormalities _____

Vaginal examination

Partial persistent hymen _____; tags _____

Vaginal mucosa

Normal: yes _____, no _____; Discharge _____

Cervical opening

Normal: yes _____, no _____; Discharge _____

Note: The extent of the examination depends upon an agreement between the buyer and seller, the expertise of the examining veterinarian, and the purchasing situation. A purchase at an auction rarely allows a detailed examination prior to purchase. This is a distinct disadvantage. I would push for an agreement that would be contingent upon a thorough examination either prior to purchase or immediately upon arrival at the new owner's premises.

Table 16.9 Checklist for a male intended for a breeder.

Behavior
Hand reared: yes _____, no _____
Aggressive to other llamas: yes _____, no _____
Aggressive to people: yes _____, no _____
Precopulatory behavior
Aggressive _____, timid _____
Copulatory behavior
Proper position: yes _____, no _____
Penetration: yes _____, no _____
Physical examination
Testicles
Right: Size_____cm, consistency_____
Left: Size _____cm, consistency_____
Abnormalities _____ _____
Penis
Length _____cm, diameter _____ mm
Abnormalities _____

endometritis or metritis. Oxytocin (30 units) should be administered IV to stimulate uterine contraction, expel the placenta, and assist in hemorrhage control. Oxytocin should not be administered until the uterus has been sutured and replaced inside the abdomen; 1–2 g tetracycline powder may be inserted in the uterine horn at the time of surgery. Boluses are contraindicated because these may stimulate straining. Excessive soft-tissue trauma or microbial contamination may indicate the administration of parenteral antibiotics for five days post-surgery.

Ovariohysterectomy

A uterine rupture with severe trauma to the uterus may be an indication for hysterectomy. In one case, small fetal mummies were identified by rectal palpation. A hysterotomy was performed, and three partially developed fetal mummies were found firmly embedded in the uterine mucosa. These were removed with difficulty. Postsurgically, this female suffered from a prolonged chronic metritis and, ultimately, hysterectomy was required.

One of the more common congenital anomalies of the reproductive tract of llamas and alpacas is segmental aplasia of the tubular tract anywhere along its length, preventing egress of normal uterine secretions. In such cases, uterine fluids accumulate, resulting in enlargement of the uterine horn, simulating pyometra. Eight to 10 l of milky fluid have been drained from some long-standing cases (Figure 16.78). Ovariohysterectomy is indicated to enable use of the animal for a pet, a packer, or fiber production.

Various experimental protocols may require either ovariectomy or ovariohysterectomy.

The animal is placed in dorsal recumbency under general anesthesia following a 24–48 hours fast. Surgery is best performed from the ventral midline approach in order to extirpate the cervix and mostly easily access both sides. The skin incision should begin just cranial to the mammary gland and extend forward 30–40 cm. The mammary gland should be reflected caudally for better exposure for penetration of the abdominal wall as close to the pelvis as possible. Both uterine horns should be exteriorized.

Fluid may be drained from the uterus with a large needle attached to a flexible tube for gravity drainage or attached to a suction apparatus. More rapid drainage may be accomplished by inserting a suction head through an incision in the uterine wall. For this procedure, a purse-string suture is placed at the intended site. With the suction head ready, a stab incision is made and the head quickly inserted through the opening. The purse string should be tightened around the suction head with a single throw and the tension held with a hemostat. Following removal of the uterine contents, the suction head should be removed and the purse-string suture pulled tight.

The uterus may then be manipulated to isolate vessels and the cervix. The ovarian and cranial uterine arteries must be identified and double ligated. Other uterine vessels are found in the broad ligament and on the ventral aspect of the uterus. These arteries and veins should also be secured by double ligation.

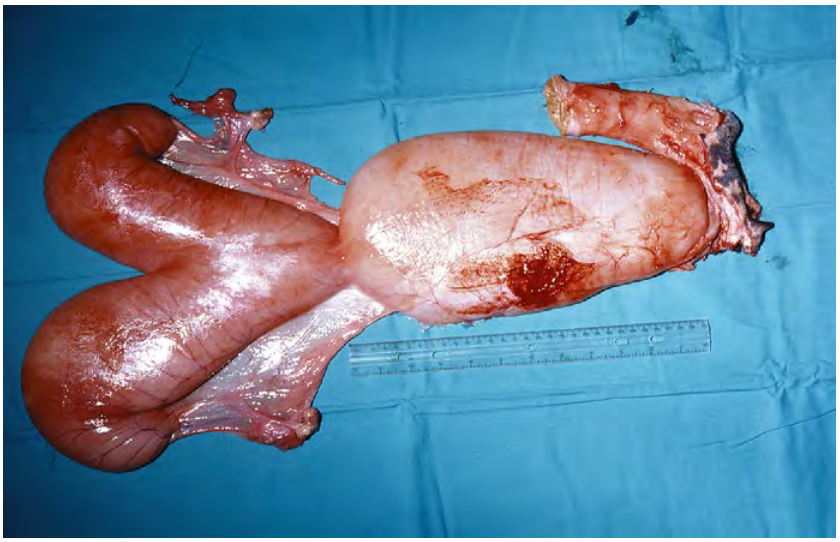

Figure 16.78 Mucometra, ventral view, emphasizing vascular distribution.

Following ligation of the vessels, the uterine ligaments may be transected to expose the vagina just distal to the cervix. Even with marked enlargement of the uterus, the cervix lies close over the brim of the pelvis or within the pelvis. Visualization of the vagina is enhanced if the animal can be tilted forward by raising the hindquarters, but it is difficult to achieve the desired exposure.

The vagina should be closed with overlapping mattress sutures and number-2 or -3 gut or other absorbable sutures of a similar size. Amputation of the reproductive tract proximal to the mattress sutures is best. Simple interrupted sutures may be used to approximate the serosal surfaces of the stump. A final check for hemorrhage should be made before closing the abdomen as described previously.

Routine monitoring during surgery is necessary, because shock may ensue from the removal of such a large organ. Standard postsurgical regimens are recommended, as for any laparotomy. Since uterine fluid is not infected, there is no indication for parenteral antibiotics if surgical asepsis is maintained.

Persistent or Imperforate Hymen

It is critical that this condition be differentiated from segmental aplasia of the vagina. A persistent hymen may be only one manifestation of a continuum of congenital anomalies of the tubular genital tract of the llama and alpaca. Because such anomalies are considered to be hereditary in certain breeds of cattle [83], the veterinarian should consider the ethical implications of performing surgery to permit breeding, especially if it is likely that the female is to be sold as a breeder.

Persistence of a segment of hymen may present a stricture at the vulvovaginal junction. This may be discovered by digital palpation. The orifice may be enlarged by gentle finger pressure. The urethral opening on the floor of the vulva should be identified either by digital palpation or inspection with a vaginal speculum before the hymen is manipulated. A small heifer speculum may then be used to enlarge the orifice further to visualize the cervix.

An imperforate hymen may be indicated by a bulge in the vulva caused by the accumulation of fluid in the vagina and uterus. On palpation, this may resemble palpation of a fluid-filled balloon. It may be thought that the hymen should break at breeding, but the male llama does not have a vigorous pelvic thrust, and penile penetration may easily be deterred by even a slight obstruction. In at least one case, the penis was deflected into the urethra. When penetration by the male was checked for by the breeder, it was noted that the urethra was markedly dilated. The female lacked a vagina; there was no passageway beyond the hymen.

An imperforate hymen results in the accumulation of uterine secretions, just as in segmental aplasia of the vagina. Rupture of the hymen and maintenance of patency allows drainage of the fluid and avoidance of the complications of a prolonged distention of the uterus.

The animal should be standing, with the tail secured up over the back or to the side, out of the perineal area. An epidural using 1–2 mL 2% lidocaine provides anesthesia if required.

The membrane may be easily ruptured by a quick thrust with a finger. A membrane too thick to rupture with finger pressure requires surgical intervention. The lips of the vulva may be spread, or a short speculum may be used to allow

visualization of the hymen. A stab incision, enlarged with a finger or forceps, is recommended. Palpation proximal to the hymen should ascertain the size of the lumen. The orifice may be enlarged by lateral incisions in the form of a cross or by a circular incision to remove a segment of the hymen.

It is important to maintain the patency of the orifice by continual stretching during the healing process. Otherwise, contraction and scarring will negate the benefits of the surgery. Stretching may be accomplished by inserting a heifer vaginal speculum once or twice daily for 10 days. Reclosure and stenosis of the vulvovaginal junction are the major complications. It may be possible to make an opening in the tract large enough to allow penetration by the male to inseminate the female. If, however, stenosis develops at the hymenal area, dystocia is probable.

Castration [134]

In addition to the usual indications for castration as seen in livestock, castration is highly recommended for llama or alpaca male crias that are hand raised with poor socialization with the herd. It should be understood that a cria may be bottle raised, but it should be given no special human attention other than feeding followed by return to association with other camelids to be properly socialized. Bottle-raised crias (both male and female) or crias with close human contact may become imprinted on humans and become pushy or aggressive as adults. Such animals should be castrated before sexual maturity, preferably before two months of age. The gonads are usually present in the scrotum at birth, and castration may be performed as early as two weeks of age or any time thereafter.

For a right-handed surgeon, lateral recumbency with the right side up is recommended. The upper leg should be tied to the neck and the tail tied to the fleece over the back. Xylazine (0.3 mg/kg IV), with local anesthetic infiltration of the skin 3–5 cm cranial to the scrotum on either side of the median raphe, is satisfactory. If sedation is insufficient, IV ketamine (25 mg/kg) may be administered.

Numerous methods have been employed in castrating camelids [134–139]. Any standard method recommended for livestock is suitable.

With the animal in lateral recumbency, two 4-cm incisions should be made 3–5 cm cranial to the scrotum and 1 cm to the right and left of the median raphe. Blunt dissection may be used to isolate the spermatic cord. The penis should be palpated to avoid exteriorizing it. A finger may be placed under the cord to lift it out of the incision and pull the testicle forward from the scrotum. The testicle should be grasped in one hand and the fat stripped from the tunic as far craniad as possible (toward the external inguinal ring).

A transfixation ligature should be placed around the tunic near the external ring and the cord transected with either an emasculator or a scalpel. Alternatively, an incision may be made through the common tunic longitudinally to isolate the vessels and the ductus deferens, which should be ligated separately, transected, and released. Then the common tunic should be ligated.

After both testicles have been removed, the incisions should be plucked to remove any strings of fat that may protrude from the incisions after the animal rises. Any pockets may be eliminated to ensure unimpaired drainage. Incisions are not sutured.

Administration of antibiotics is unnecessary, and equine-origin tetanus antitoxin serum is not recommended. Swelling is rare. Llamas do not lie in their own feces; therefore, the incision remains clean. It is recommended that the animal be returned to pasture or kept in a clean, straw-bedded box stall.

Alternative Techniques

1) The same procedure may be followed, except incisions are sutured as in a canine castration. Surgical asepsis must be employed throughout.
2) The initial incision may be made through the scrotum. Using a closed technique, the testicle and surrounding tunics are isolated and separated from the surrounding fascia as far craniad as possible. An open technique may also be performed, in which case, the common tunic is incised, freeing the testicle, epididymis, and ductus deferens. The ductus deferens and other vessels should be ligated prior to transection. The common tunic is left *in situ*.
3) In standing castration, the camelid should be placed in a narrow stock or squeeze chute, with the tail held or secured up over the back. The area should be surgically prepared, with local anesthesia administered subcutaneously over each scrotal sac. Additional local anesthetic may be injected into the spermatic cord, craniad to the scrotum, or an open procedure is employed, with incisions left unsutured.
4) In single incision, the animal is anesthetized and placed in lateral recumbency. A rope is placed around the upside fetlock and the rope threaded over the back to the opposite hock and tied at the fetlock. This method of restraint places the rear limbs in a frog-legged flexed position. A testicle is pushed craniad as far as possible (10–15 cm from the scrotum). A 5-cm incision is made over the testicle, approximately 3 cm lateral to the midline, to the common tunic. The testicle is grasped and stripped of areolar tissue and the cord ligated as close to

the inguinal ring as possible. The cord is then transected distal to the ligature. The opposite testicle is pushed craniad, dorsal (beneath in this position) to the penis and extracted from the same incision. The skin is closed with the surgeon's preferred suture pattern

Early Castration – Pro or con

It has been known since the time of Aristotle that boys castrated in infancy (eunuchs) developed elongated limbs when they grew up. The same is true for many other species of animals, but the degree of effect varies from species to species. In llamas and alpacas, castration before puberty may delay physeal closure of the long limb bones, allowing growth to continue [140].

The physiology behind physeal closure should be understood. In males, the physes of the humerus, radius, metacarpus, femur, tibia, and metatarsus begin closure with the onset of testosterone production. In females, estrogen production stimulates closure of the physes. If hormonal stimulation is delayed or prevented by neutering, physeal closure is delayed. The limbs continue to elongate, which changes the normal balance between body depth and limb length conformation (Chapter 24).

Reasons for early castration:

1) Prevention of aberrant behavior in human-imprinted males.
2) Elimination of intermale aggression so that males may be housed together or the gelded male may run with females.
3) Avoidance of accidental breeding of inferior males to receptive females.
4) Prevention of the use of inferior males, sold for pets or to packers, as breeding males.
5) Preference of packers for a tall llama.

Reasons to avoid early castration:

1) Avoidance of long-legged adults.
2) Tendency of long-legged camelids to be post legged on both the fore and hind legs, which may predispose them to patellar subluxation, especially if a shallow trochlear groove is present.
3) Potential for development of dropped fetlock, especially if the animal becomes obese, and arthritis or ligamentitis.
4) Concern of veterinarians with food animal experience about the possibility of urolithiasis in gelded animals. To date, this has not been a problem; most urolithiasis cases have occurred in intact males.

Young males with aberrant-behavior tendencies should be castrated prior to the onset of puberty. If facilities for maintaining intact males are available, it is desirable to delay castration until libido begins and the canine teeth have erupted fully, usually between two and three years of age.

Though it has not been experimentally confirmed, the parenteral administration of testosterone to a gelded male at the approximate time of maturation may initiate physeal closure.

Be aware that hypo and agonadal females may also develop elongated limbs due to lack of estrogen production.

Cryptorchid Castration [27]

Cryptorchidism is one of many genetic malformations of the camelid male reproductive system. Only rarely has the cryptic gonad been retained in the abdominal cavity. More frequently, the gonad has been located in the fascia, anywhere from the external inguinal ring to near the scrotum and alongside the penis. In a few cases, the gonad has been located in the subcutaneous fascia on the medial aspect of the rear limb. The cryptic gonad may be difficult to palpate because it may only be 0.5–1 cm in diameter and may be confused with local lymph nodes.

Because cryptorchidism is a genetic trait, such an animal should not be used for breeding, and both the cryptic and normal testicles should be removed. Bilateral cryptorchidism is extremely rare.

The cryptic gonad should be located and an incision made where necessary to amputate. If the gonad is intraabdominal and the side can be identified, a paralumbar fossa grid approach may be used after incising the skin. If it is impossible to determine the appropriate side, the abdomen may be entered via the ventral midline approach.

Vasectomy

The primary purpose for vasectomy is to facilitate study of reproductive physiology. Vasectomized males can be used to check females for pregnancy or to induce ovulation without fertilization.

The scrotum is not pendulous, so the spermatic cord is not as accessible as it is in the bull or ram. An incision is made cranial to the scrotum, similar to a castration incision. The spermatic cord is isolated and exteriorized. The tunic is incised and the ductus deferens isolated. Ligatures are placed 2 cm apart, and the segment between the sutures is excised. The tunic should be sutured with simple interrupted observable sutures.

Alternatively, a 2-cm segment of the ductus deferens may be excised via standard laparoscopy using forceps scissors [109, 141].

Acknowledgments

Drs. Murray Fowler and Walter Bravo authored the corresponding chapter on Reproduction in the 3rd edition of this text. Although this edition has been updated and restructured, some of their previous content remains and the current authors would like to acknowledge their contribution to this chapter.

Notes

1 Dr. P. Walter Bravo is a reproductive endocrinologist with vast experience in the study of reproduction and reproduction problems in South American camelids both in North and South America.
2 The term used in Peru is cutuneo.
3 P.W. Bravo et al. 1997, unpublished data.
4 W.L. Franklin, personal communication.
5 W.L. Franklin, personal communication.
6 P. Miller, personal communication.
7 J. Paul-Murphy, personal communication, 1987.

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17

Urinary System

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Anatomy

Kidney

Camelid kidneys are non-lobulated (Figure 17.1). Both kidneys are the same size (approximately 5 × 9 cm) and fixed in the dorsal abdominal area. The left kidney is located ventral to the transverse processes of the fourth to seventh lumbar vertebrae. The right kidney is located slightly more cranial, beneath the fourth to sixth transverse processes of the lumbar vertebrae. Kidney weight varies from 120 to 170 g in llamas weighing from 74 to 120 kg. Microscopic anatomy has been described.

Ureters, Urinary Bladder, and Urethra

Anatomy of the ureters and urinary bladder is similar to cattle and sheep.

In male camelids, a dorsally situated urethral recess at the ischial arch prevents passage of an urethral catheter into the bladder (Figure 17.2) [1]. The urethra ends at the urethral orifice and there is a fibrocartilaginous, spiral-shaped projection at the tip of the penis (Figure 17.3). In cases of dysuria, stranguria or suspected trauma, the penis has to be extended. This procedure is usually only possible with sedation or general anesthesia. In young males, adhesions between penis and prepuce make an extension of the penis impossible.

The female urethra has an external orifice located at the ventral border between the vagina and vestibule. A ventral diverticulum at the external orifice makes female urethral catheterization difficult but not impossible.

Characteristics of Camelid Urine

The composition of urine has been described [2–4]. The urine of 138 alpacas maintained at an elevation of 4200 m was analyzed in Peru (Table 17.1) [3]. In general, there was insignificant variation with age, sex, or physiologic state. Ketones can be found in camelids with metabolic disorders, especially hepatic lipidosis. In a group of alpacas, at 4200 m, Acetoacetic acid was detected in 42% of the nonpregnant alpacas and in 100% of the pregnant alpacas. Acetone was detected in only 2% of the nonpregnant alpacas but in 88% of the pregnant alpacas.

Urea is excreted by adult alpacas at 6.08 g/L urine. Urea excretion is slightly higher in juveniles as compared with adult animals, and in pregnant animals it is higher still. Animals consuming diets containing high concentrations of protein (>15%) have higher concentrations of serum urea nitrogen (SUN) and likewise have higher urinary nitrogen concentrations. In general, alpacas have higher rates of urea excretion than any other animal except humans. The amount of ammonia excreted (0.13 g/L) does not vary with age or sex [5]. Renal function has been discussed for camelids [5–7]. Urine collected from normal llamas in California had similar characteristics to those of alpacas.

Camel urine is different than South American camelid (SAC) urine [8]. The physiology of urine production, storage, and voiding are important considerations for the camel clinician. The composition of camel urine is highly variable depending on the availability of water, dehydration status and environmental factors such as ambient temperature and availability of plants resistant to drought.

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Camel urine is normally clear and pale yellow in color. The pH varies from 6 to 9. The specific gravity is highly variable depending on the degree of dehydration. Extreme dehydration results in a urine of a syrupy consistency. Calcium oxalate and calcium hydrogen phosphate crystals may be observed in urine sediment.

Diagnostic Procedures

Urine may be collected as a free catch during urination at a communal dung pile, by urethral catheterization in the female, and by cystocentesis in both the female and the male SAC by fixing the bladder per rectum and inserting a 10–15-cm (4–6-in.) 18-gauge needle on the midline just cranial to the pubis. The area is surgically prepared and, in the male, the penis and prepuce are pushed laterally to allow the midline insertion of the needle. Cystocentesis should be performed using ultrasonographic guidance.

Urinalysis may be conducted in the field using dipsticks to evaluate common parameters (glucose, pH, ketones, protein, blood). A refractometer may be used to determine specific gravity. More sophisticated procedures are done in the laboratory, including electrolyte concentrations, quantifying protein and other solutes, and cytology.

Hematuria or hemoglobinuria may be observed in renal trauma (automobile accident), ruptured bladder, copper poisoning, blood transfusion reactions, severe burns,

Figure 17.1 Longitudinal section of a camelid kidney.

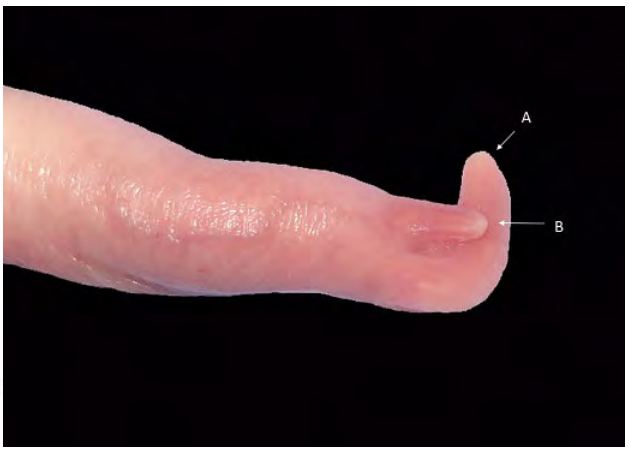


Figure 17.3 Penis of a llama, A: fibrocartilaginous process, B = urethral opening.

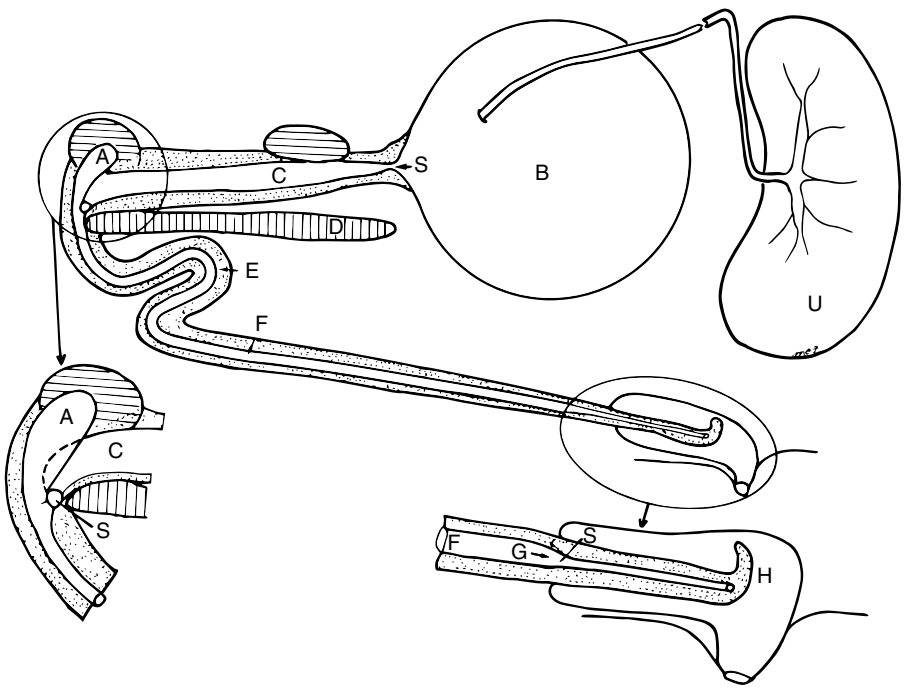


Figure 17.2 Diagram of the urinary tract of a male camelid. (A) Urethral recess, (B) bladder, (C) pelvic urethra, (D) pubic bone, (E) sigmoid flexure, (F) penile urethra, (G) beginning of the glans penis, (H) cartilaginous projection at the tip of the penis, (S) point of narrowing (potential site for urethral obstruction), (U) kidney.

Table 17.1 Normal camelid urinalysis.

Parameter	Alpaca kept at 4200 m N = 138	Llamas kept at sea level N = 15	Dromedary camels
Volume (l/24 h)	0.125–3.8 (1.06)		1.5–5.0
Color	Clear, yellow to amber	Clear, light yellow to amber	Clear, light yellow to dark yellow
Specific gravity	1.010–1.048 (1.021)	1.013–1.048 (1.023)	1.022–1.070
PH	Alkaline 94%, acidic 2%	7–8.5	4.5–8.7
Protein	Negative	Negative	Negative
Glucose	Negative	Negative	Negative
Ketones	50% + at 1	Negative	Negative
Urobilinogen	52% = at 1	Negative	Negative
Bilirubinogen	45% = at 1	Negative	Negative
Indican	45% +	Not run	Negative
Blood	Negative	Negative	Negative
Sediments	Triple phosphates common, ammonium urate rare	Calcium oxalate common in alkaline urine, uric acid rare	Calcium oxalate, calcium hydrogen phosphate, ammonium urate, triple phosphate

Table 17.2 Urinalysis in selected diseases.

	Dehydration	Urinary tract hemorrhage	Chronic nephritis	Cystitis
Gross appearance	Clear	Clear	Clear	Cloudy to hemorrhagic
Specific gravity	↑	↑	↓	↑
Refractive index	↑±	↑	↓	↑
Protein	0	+1–4	+1–2	+1–4
Color	Yellowish-amber	Reddish	Yellowish	White to reddish
Glucose	0	+1	+1–2	0– +1
Ketones	0	0	0– +1	0
Cells	±	±RbC	0– +1	Neutrophils-epithelial
Bacteria	0	0	0– +1	+ +

neoplasia, dicoumarol poisoning, or urolithiasis, or following urethral catheterization, or it may be associated with infectious diseases (cystitis, pyelonephritis, urethritis, prostatitis, septicemia).

Proteinuria may be observed in urinary tract hemorrhage, chronic nephrosis, glomerulopathy, inflammation of the urinary tract, urolithiasis, and neoplasms.

Table 17.2 provides data for selected urinary tract diseases. Urinary tract diseases are diagnosed by a combination of clinical signs, urinalysis, serum biochemistry, hemogram, and special procedures such as renal angiography, radiography, ultrasonography, and cystoscopy in the female.

A number of electrolyte excretion and clearance tests are used in human and other animal species to evaluate renal function. Reference ranges for camelids are now available for consideration by clinicians and diagnosticians. These tests include total excretion of electrolytes and creatinine, fractional excretion of the same substances, and electrolyte excretion compared with creatinine as an index to avoid having to collect 24-hour urine samples.

The presence of certain enzymes in urine, including γ-glutamyltransferase (GGT) and N-acetyl-β-D-glucosamidase (NAG), indicate tubular activity such as leakage or necrosis.

SUN is often used as an index for kidney function. Causes for an elevated SUN concentration may be prerenal in origin (reduced renal perfusion, hypovolemia, dehydration, starvation, high-protein diet, fever, burns, steroid administration, anabolic steroids, infection) or the direct effect of kidney malfunction (acute renal failure, chronic renal failure). Postrenal elevated SUN concentration may arise from urinary tract obstruction or a ruptured bladder. A mildly elevated SUN

concentration may simply be a reflection of anorexia and is seen as an ancillary finding in many sick camelids.

Low SUN concentrations are seen in neonatal animals and in camelids suffering from hepatic insufficiency.

Elevated serum creatinine concentrations are caused by the same factors that cause elevated SUN concentrations. The creatinine concentration is a better guide to renal failure because SUN is influenced more by diet. An elevated SUN concentration, associated with an elevated creatinine concentration, is significant. In neonates, the creatinine concentration is transiently elevated within the first 48 hours of life. Due to fluid ingestion and low muscle mass, the creatinine concentration can be reduced to concentrations below 1.5 mg/dL. Therefore, repeated measurement of the creatinine concentration is recommended, in order to assess neonates [9].

Percutaneous renal biopsy may be accomplished by guiding the needle with ultrasonography.

Ultrasonography

Ultrasonography has become a valuable additional tool to physical exam and laboratory tests. It provides important information to investigate dimensions and structure of both kidneys (Figure 17.4), urinary bladder and urethra.

The fiber can be parted with the fingers and 70% ethanol can be applied, in order to achieve good contact between probe and skin. The left kidney can be approached from the left paralumbar fossa directly ventrally to the fourth to seventh transverse processes of the lumbar spine. The spleen is located cranially from the left kidney (Figure 17.5). The right kidney can be found in the right paralumbar fossa, caudal to the last rib and ventral to the transverse processes. The dimensions of the right and the left kidney have been described [10].

Figure 17.4 Ultrasonographic image (longitudinal) of a normal kidney.

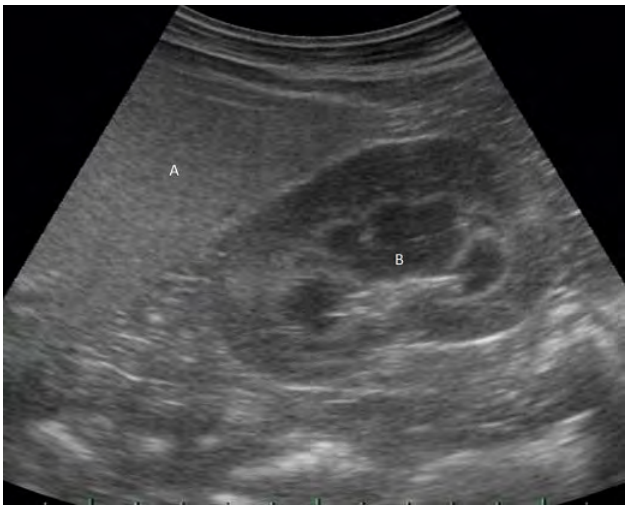


Figure 17.5 Ultrasonographic image of the normal spleen (A) and the normal left kidney (B).

The ureters cannot be visualized in healthy camelids. The urinary bladder can be imaged transrectally. In normal camelids, the urinary bladder cannot be visualized by transcutaneous ultrasonography. However, a distended bladder may be imaged. The urethra can be imaged transrectally or transcutaneously in male camelids.

In camelids with renal disease, ultrasonography is helpful to reveal focal lesions like in tuberculosis (Figure 17.6) or lymphoma (Figure 17.7). Also, diffuse mineralization at the cortico-medullary junction (Figure 17.8) and the medullary region (Figure 17.9) can be detected by ultrasonography and can be of value in light of other findings, suggestive of renal disease.

Cystoscopy

Cystoscopy can be helpful to evaluate the urinary bladder and to examine the urine coming from each ureter. In camelids with hematuria, cystoscopy can help to define which side is affected [11]. Polypoid cystitis can be diagnosed by cystoscopy [12].

A 3.3-mm-diameter flexible fiberoptic endoscope has been used for cystoscopy [12].

Urination Behavior

Camelids use communal dung piles for both defecation and urination. Camels defecate at random, but assume the same stance as SACs to urinate. With stimulation to void, both male and female SACs seek out a dung pile. At the approach, the animal will sniff the pile and, if satisfied, turn around and assume a squatting position, with the

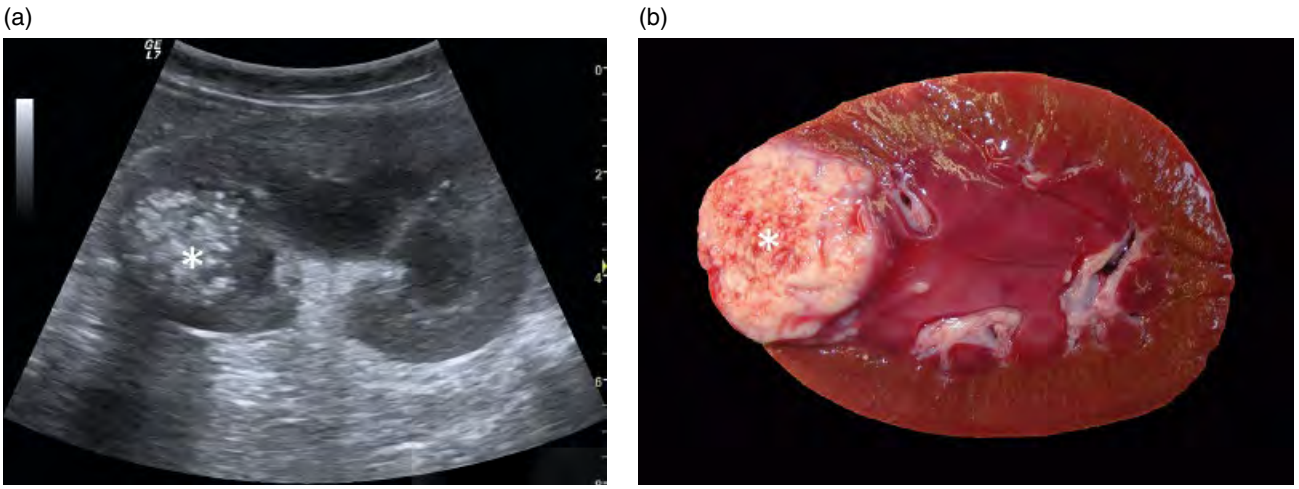


Figure 17.6 Ultrasonographic image and necropsy finding in a two-year-old female alpaca with a multi-organ granulomatous lesions (*), caused by Mycobacterium ssp.

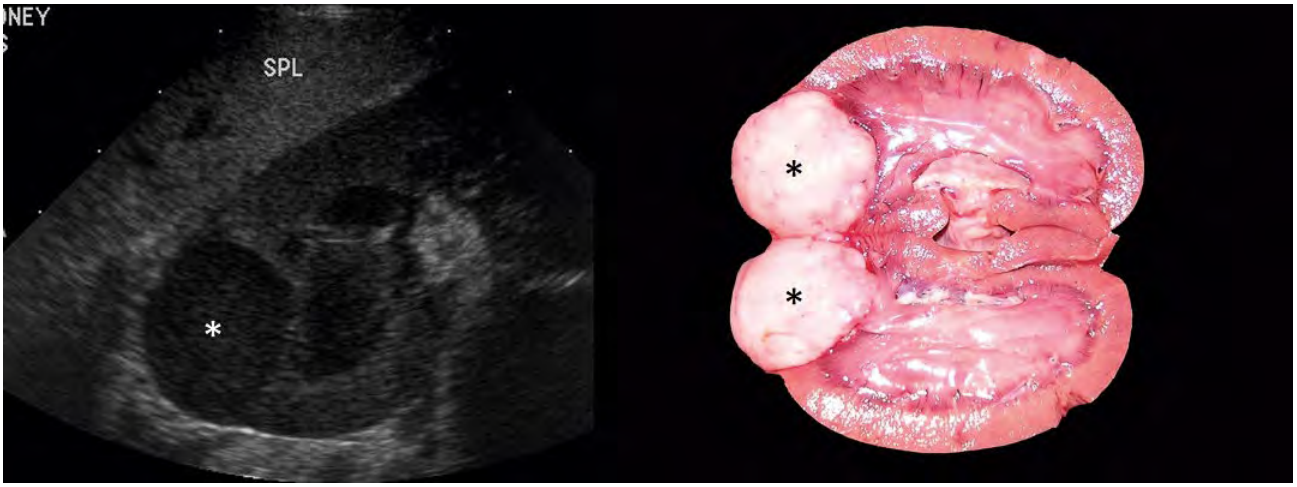


Figure 17.7 Ultrasonographic image and necropsy finding in a one-month-old alpaca cria with lymphoma (*).

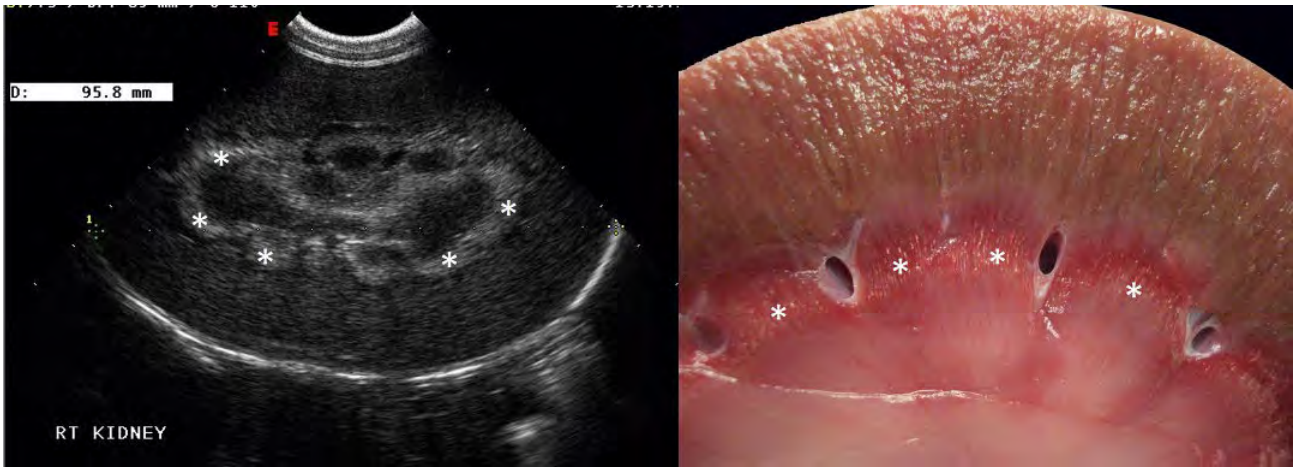


Figure 17.8 Hyperechogenicity at the cortico-medullary junction (*) in a two-year-old female alpaca with severe edema and diarrhea due to protein-losing nephropathy; Necropsy revealed severe diffuse membranous glomerulopathy with multi-focal medullary tubular mineralization and tubular necrosis.

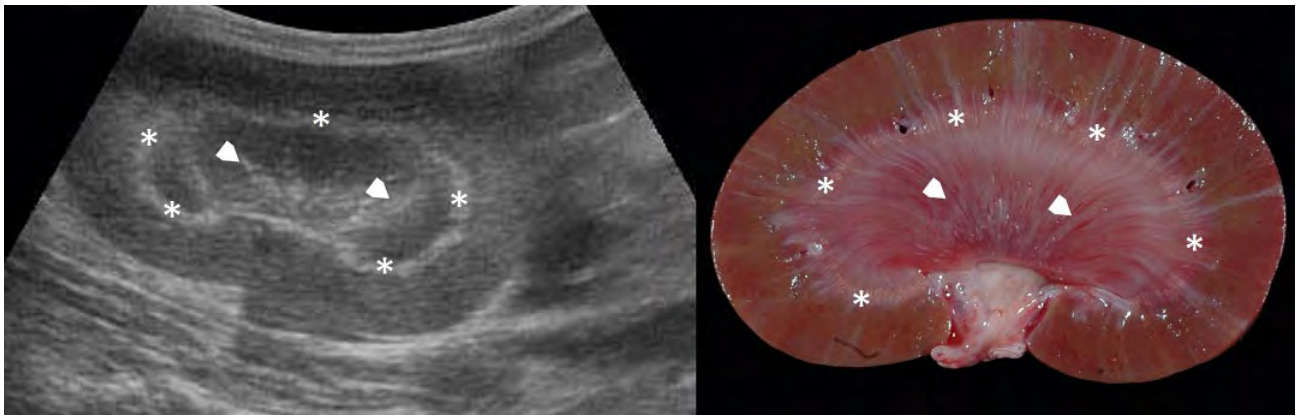


Figure 17.9 Hyperechogenicity at the cortico-medullary junction (*) and pelvic mineralization (arrowhead) in an eight-year-old female alpaca with multifocal interstitial fibrosis and interstitial nephritis.

hind legs spread apart and brought forward under the body. Defecation usually occurs first, followed immediately by urination. A llama or alpaca will usually urinate two to four times a day but while carrying a heavy load in the wilderness may urinate only upon arising in the morning and again in the evening.

An adult camel, with unlimited access to water, voids up to 7 L of urine per day in aliquots of 450 mL. The camel urination stance is similar to that of SACs, with the hind legs apart and in a semi-crouch position. Male camels may vary this position during heat stress and stand erect, spraying the hind legs, apparently to produce evaporative cooling.

A llama may refuse to urinate while being transported. This must be considered when a llama is transported by trailer to a clinic. If the bladder is distended with urine, any restraint method employing bands around the body may cause rupture of the bladder if the animal should struggle. Likewise, it is wise to proceed with caution when conducting a rectal examination on an animal with a distended bladder; rupture of the bladder has occurred from application of too much pressure. A newly hospitalized llama may refuse to urinate or defecate for a few hours if on a concrete or wooden floor. The animal should be moved to a lawn or dirt area to provide an opportunity to void before concluding that there is oliguria or no passage of feces.

Diseases

Renal disease can occur primarily or be secondary to underlying disease. Clinical signs of renal diseases are often non-specific and renal disease is not usually a primary complaint in camelids referred to a veterinary hospital. Renal problems are an important differential diagnosis in the work up of anemia, hypoproteinemia, weight loss, and hematuria. Therefore, a good history is crucial to obtain evidence of clinical signs accompanying renal disease or nephrotoxic drugs given to the patient previously. Common historical information in camelids with renal disease includes weight loss or failure to gain weight, weakness, anorexia, soft feces, dysuria, and anemia.

Unlike in other species, insufficient numbers of camelid cases of nephritis have been studied to categorize the disease (glomerulonephritis, interstitial nephritis, pyelonephritis). Iatrogenic toxicity from the administration of gentamicin has accounted for the majority of acute renal failure cases.

Other noninfectious causes of acute renal failure include hydronephrosis, urinary tract obstruction, neoplasia, autoimmune disorders, toxicities (heavy metals, oxalates, tannins, ethylene glycol), hemolytic crisis, and crotalid snakebite (see Chapter 22).

Stranguria and Dysuria

Dysuria is characterized by abnormal posture at the dung pile or by straining as if to urinate but with urine dribbling or no urine flow. It may be difficult to differentiate dysuria from tenesmus. Stranguria or dysuria are suggestive for inflammatory or obstructive problems of the urinary tract. The most common causes are cystitis, urolithiasis, neoplasia, congenital defects, pelvic displacement of the bladder [13], balanitis, posthitis, and prostatitis.

Dysuria has also been reported in an alpaca with severe chronic purulent discospondylitis and spondylosis narrowing the spinal canal at the level of L4 and L5. Diagnosis has been confirmed by magnetic resonance imaging (MRI) [14].

Nephrosis

Nephrosis is a degenerative disorder of either the glomeruli or tubules and may be caused by endotoxemia, renal ischemia, or nephrotoxic agents (oak tannins, oxalates,

ethylene glycol, lead, arsenic, aminoglycoside antibiotics, myoglobin, hemoglobin) or immune complexes [15]. Renal ischemia is associated with shock and may be exacerbated by general anesthesia. In horses and dogs, surgical manipulation of the abdominal viscera results in a reflex decreased blood flow to the kidneys. A similar phenomenon may occur in camelids. Partial or complete vascular occlusion to a segment of the intestine results in a buildup of endotoxins in the lumen. If the obstruction is corrected surgically, endotoxic shock and hypotension may occur as a result of the rapid absorption of endotoxins once circulation is restored. The animal may die from the direct effects of the shock or develop renal failure from renal ischemia.

Severe, prolonged dehydration, as may occur with diarrheal diseases, may predispose to ischemic nephrosis. Excessive quantities of myoglobin and hemoglobin in the urine produce degeneration of the renal tubules. Myoglobinuria may be seen following crushing injuries or violent struggling or it may be associated with white muscle disease.

Oak Toxicity

Oak toxicity was diagnosed in a llama that had access to large numbers of acorns. The affected llama was a 134-kg female in the fifth month of gestation. Eighty other llamas were in the same pasture with access to the acorns, but this llama was the only one affected. This is characteristic of the syndrome as seen in cattle as the affinity for ingesting acorns seems to vary based on individual animal.

Clinical Signs and Laboratory Findings

The llama was anorectic, depressed, weak, and recumbent when first examined. She exhibited signs of colic (rolling, refusal to rise) and no gastric motility. A large amount of mucoid material was excreted from the rectum, in the absence of feces. This female llama was pregnant but aborted her fetus. Temperature, pulse, and respiration were normal. She was anuric and had submandibular and perineal edema.

SUN (136 mg/dL), creatinine (11.5 mg/dL), and potassium (2.8 mEq/L) levels were elevated, and sodium (149 mEq/L) and chloride (117 mEq/L) levels were decreased. A metabolic acidosis was present. Urinalysis results showed a specific gravity of 1.023, a pH of 8.5, trace protein, no glucose, trace ketones, and initially a 1+ blood.

Treatment

She responded to the administration of lactated Ringer's solution at 80 mL/kg/day (two times maintenance) with 26 mEq/L of KCl added to the fluids. Ampicillin 10 mg/kg and cimetidine 2.2 mg/kg were administered twice daily.

Another report of oak toxicity described clinical findings and outcome in two adult llamas [16]. The animals were presented for depression, anorexia, and diarrhea. Upon exam, they were found to have severe azotemia and isosthenuria. One llama survived, after treatment with ranitidine, furosemide, sodium ceftiofur, and isotonic saline containing potassium chloride (20 mEq/L). The llama received fluid therapy over 30 days. After discontinuing fluid therapy, the creatinine concentration was within the normal range.

The second llama died after 22 hours of treatment. At necropsy, multiple fragments of oak leaves could be detected within all three gastric compartments. Histologic examination revealed multifocal, acute tubular degeneration and necrosis.

Gentamicin Toxicity

Two cases of gentamicin toxicity have been reported [17].

Clinical Signs and Laboratory Findings

Signs were similar to those seen in the oak toxicity, with the addition of weight loss, dehydration, bruxism, loose stool, and gait abnormalities.

SUN and creatinine levels were markedly elevated (SUN 280 and 396 mg/dL, creatinine 21.5 and 24 mg/dL). Other findings included a serum phosphorus of 13.6 and 33 mg/dL; sodium of 133 and 140 mEq/L; chloride of 89 and 105 mEq/L; and a severe acidosis (HCO_3 of 11 and 11.1 mEq/L). The urine was isosthenuric at 1.013 with a pH of 5.5.

Vitamin D Toxicity

Prophylactic treatment with vitamin D supplements is a common husbandry practice.

However, the appropriate dosage, dosage interval, and formulations of products containing vitamin D are often misunderstood. Recommended doses are 1000–2000 units/kg as a single dose or repeated in two to three months. Accidental single overdosing did not cause clinical consequences. However, repeated dosing within a short period of time can lead to renal insufficiency.

Two alpaca crias have been reported to be over-supplemented with oral vitamin D [18]. Cria 1 received a cumulative dose of 26250 IU/kg or 3750 IU/kg/day over seven days and cria 2 a cumulative dose of 46900 IU/kg or 12987 IU/kg/day over five days. In both crias, hypervitaminosis D lead to severe renal insufficiency and anuria. Both cria were presented with generalized weakness, anorexia, and depression. Laboratory analysis of the blood revealed the following results in cria 1: SUN 48.5 mmol/L (range 1.48–24.4 mmol/L), creatinine 557 µmol/L

(range 115–239 μmol/L), calcium 3.5 mmol/L (range 2.0–2.6 mmol/L), phosphate 4.5 mmol/L (range 0.8–2.8 mmol/L) and in cria 2: SUN 85.7 mmol/L, creatinine 548 μmol/L, calcium 3.8 mmol/L, phosphate 3.3 mmol/L. Despite aggressive therapy with fluids and furosemide, both crias were euthanized due to deteriorating conditions. In cria 2, peritoneal dialysis was unsuccessful; SUN (66 mmol/L) and creatinine (998 μmol/L) concentrations increased over three days. Urine production was absent during the entire time.

In both cases, necropsy revealed diffuse glomerular mineralization with tubular necrosis. No normal glomeruli were found.

Chronic Renal Failure

Some of the foregoing signs may be seen in chronic renal disease, but additional signs include anorexia, weight loss, depression, straining, and mild anemia. A low-grade fever of 40–41 °C (104–106 °F) may be seen with suppurative nephritis. The urine may contain bacteria, leukocytes, blood, and elevated levels of protein. Concentration of the urine varies, as does the quantity of urine produced, but usually the concentration is low and volume output is high.

Uremia is the terminal syndrome of renal failure and is characterized by anorexia, weight loss, depression, muscular weakness, muscular tremors, dyspnea, oliguria, albuminuria, hyperemic mucous membranes, uremic odor from the breath, tachycardia from dehydration, recumbency, coma, and death.

Nephritis

Inflammatory lesions of the kidneys are usually caused by infectious agents, which may produce focal or diffuse glomerulonephritis, interstitial nephritis, pyelonephritis, or embolic nephritis. Clinical signs are similar to those described for acute and chronic renal failure. Treatment should include antimicrobial therapy.

Renal failure must be treated early and intensively to avoid permanent damage to the glomeruli and tubules. The animal must be carefully rehydrated to overcome renal hypotension, renal ischemia, and oliguria. Urine production should be monitored to ensure that the kidneys are able to function.

Furosemide (0.5–1 mg/kg) may be given intramuscularly (IM) twice daily to cause diuresis. However, furosemide is ineffective if tubular nephrosis is severe. Any electrolyte imbalance must be corrected and administration of fluids continued until the animal is able to drink adequate quantities of water.

Appropriate parenteral antibiotics are indicated for infectious nephritis and may prevent secondary infection in nephrosis. Once the animal is stabilized and eating, protein intake should be minimized by offering grass hay.

Cystitis and Urethritis

Cystitis and urethritis are more common in the female because of a shorter urethra and the high probability of retrograde invasion by opportunistic bacteria. Cystitis and ureterolithiasis traumatize the mucous membrane, predisposing to infection. In one female llama with segmental agenesis of the vagina, repeated breeding by a male resulted in penetration of the urethra by the penis and traumatic urethritis. Bladder paresis and urine stagnation predispose to cystitis. One case of a tumor of the bladder has been reported to have caused cystitis.

Urination is painful and frequent, with only small quantities of urine produced. The urine may contain blood, leukocytes, bacteria, and excessive numbers of epithelial cells in the sediment. Clinical signs and urinalysis usually suffice to confirm this diagnosis. Urine should be cultured and a sensitivity test performed.

Diuresis and appropriate parenteral antibiotic therapy for 10–14 days are usually successful.

Posthitis

Posthitis is an inflammation of the prepuce. No specific infectious diseases cause this condition in camelids. Trauma, caused by biting during aggressive male interaction, is the usual cause. Lacerations may be minimal or severe. In male camels, posthitis may be caused by masturbation in the hot desert sand.

Lacerations are evident on physical examination. Heat, swelling, exudation, and dysuria may also be observed (Figure 17.10). The swelling may obstruct urine flow.

Recent lacerations may be cleansed, debrided, and sutured. Infected wounds should be treated locally by

Figure 17.10 Posthitis in a llama.

applying disinfectants (povidone-iodine) and alternating hot and cold packs to disperse the edematous, inflammatory response. Parenteral, broad-spectrum antibiotics may be administered.

Concretions of the Urinary Tract

Urinary calculi (urolithiasis, uroliths, nephroliths, bladder stones, cystoliths) are formed in either the calices of the kidney (Figure 17.11), or more commonly in the urinary bladder. Small uroliths may enter the ureter or urethra and cause partial or complete obstruction of urine flow [13, 19–33]. No specific studies on the pathogenesis of formation of urinary calculi in camelids have been reported. There are clinical reports of disease caused by the obstruction produced by the calculi. Camelid veterinarians must rely on information extrapolated from studies of cattle, sheep, and goats, which have urine of similar composition [34]. The composition of urinary calculi recovered from llamas at the University of California is listed in Table 17.3. Urethral obstruction may also occur with non-mineral concretions.

Urinary calculi are formed in males and females equally, but the lumen (diameter) of the female urethra generally allows free passage of a calculus that may enter the urethra. Thus, obstructive urolithiasis is rare in the female. Urolithiasis has been associated with a diet high in concentrated feeds, such as are often used in zoos. Cattle pastured on grasses containing high levels of silicates may sometimes develop silicate urolithiasis, and presumably camelids grazing on such pastures may also be at risk.

A basic understanding of the camelid urethra is required to locate sites of possible obstruction and develop approaches to management. Figure 17.2 is a diagram of the camelid urethra and associated structures. The prostate gland does not surround the pelvic urethra as in carnivores. The pelvic urethra is expansive, but at the reflection around the ischium, only a tiny orifice allows passage of urine beyond this point. The anatomy of this area is further complicated by a dorsal urethral recess, which precludes any possibility of passing a catheter into the bladder from the tip of the penis. Whereas the sigmoid flexure is the probable site of the majority of bovine urethral obstructions, this is not the case in camelids. The orifice from the pelvic urethra into the penile urethra is a common site of obstruction; another is where the penile urethra narrows as it enters the glans penis.

Table 17.3 Composition of urinary calculi recovered from llamas at the Veterinary Medical Teaching Hospital, University of California.

Silicon dioxide (SiO_2)	Crystobalite
Magnesium ammonium phosphate ($MgNH_4PO_4 \cdot 6H_2O$)	Struvite
Basic calcium phosphate ($Ca_5(PO_4)_3(OH)$)	Apatite
Calcium carbonate ($CaCO_3$)	Carbonate
Uric acid ($C_5H_4N_4O_3$)	Urate

Clinical Signs

Uroliths in the bladder or calices of the kidney rarely cause discomfort, though large and rough-surfaced uroliths may initiate cystitis. The signs of urethral obstruction caused by calculi vary with the stage of the disorder [5, 35, 36]. Signs prior to bladder rupture include colic, straining to urinate (Figure 17.12), dribbling urine, blood-tinged urine, anuria, distended bladder, and possible pulsation of the urethra.

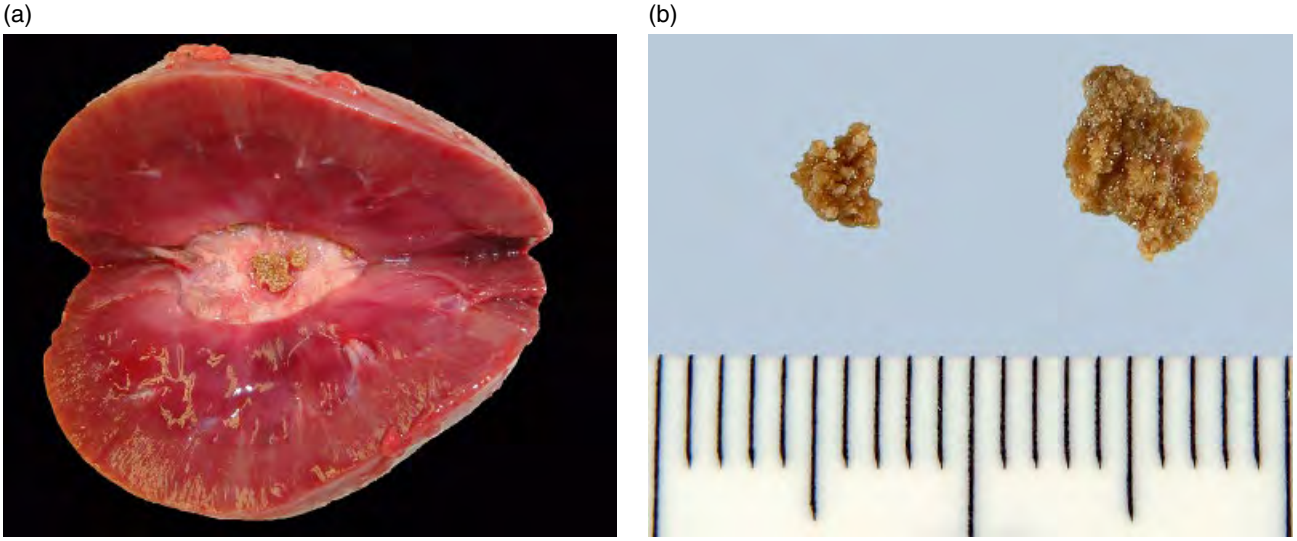


Figure 17.11 (a) Nephroliths in a six-month-old alpaca. (b) Stone analysis revealed 100% $CaCO_3$ (Calcium carbonate).

Figure 17.12 Stance of a llama cria with urethral obstruction.

Following bladder rupture, the animal typically does not display colicky signs. However, depression, anorexia, anuria, and uroperitoneum prevail. Additional signs of bladder rupture include distention of the abdomen and uremia. The uremia leads to muscular weakness, dehydration, dyspnea, tremors, uremic odor in the breath, tachycardia, recumbency, coma, and death [34].

Uroperitoneum may be caused by trauma when the bladder is distended or from rupture of the bladder following urethral obstruction. In the llama, the immediate response to urine draining into the abdominal cavity is severe pain. Llamas become frenzied and thrash about violently. This initial pain subsides, and the pain associated with a distended bladder disappears. Urine in the abdomen may arise from a single or multiple tears in the bladder wall but also from seepage through the stretched-thin bladder wall. A ruptured urethra has been reported in camels [19, 35]. A ureter may also rupture, but this condition has not been reported in a camelid.

Diagnosis

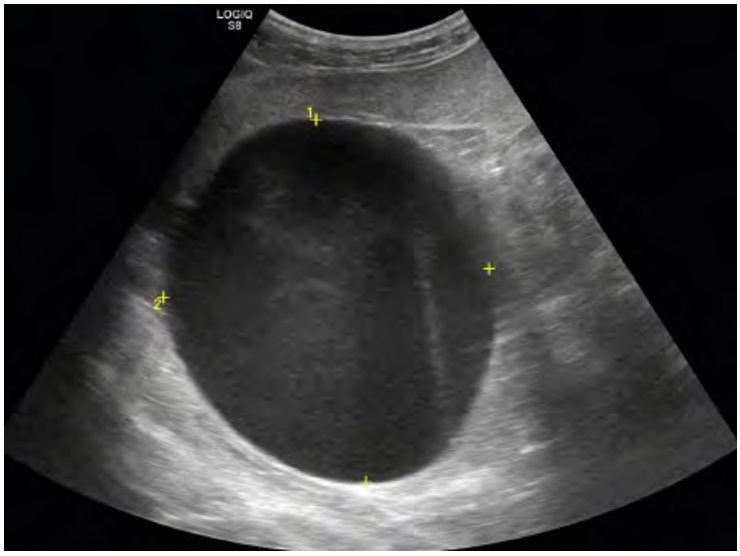
Diagnosis is based on assessment of clinical signs, pertinent history, and special diagnostic tests. Differential diagnoses should include any disease condition that can result in colic. The major hematologic and serum chemistry changes found in camelids with obstructive urolithiasis include hemoconcentration, elevated blood urea nitrogen, hypophosphatemia, hypercalcemia, hypermagnesemia, hyperkalemia, hypercreatininemia, and hypochloremia [19, 32, 34, 37].

It is not always easy to identify the source of fluid in the abdominal cavity. Urine should have the odor of ammonia, but it may be necessary to heat the fluid to concentrate the odor before it becomes perceptible. The ability of individuals to smell ammonia varies. In abdominal fluid caused by urine, the creatinine concentration is twice as high as the serum concentration. Urine potassium is >185 mmol/L (normal serum <5 mmol/L).

It is almost impossible to pass a catheter in a camelid male because of the dorsal urethral recess and restrictive diameter of the urethra.

Ultrasonography can be useful in order to identify a dilated bladder (Figure 17.13), dilated kidneys, uroliths in the kidney (Figure 17.14) or bladder, and an increased amount of fluid in the abdomen.

(a)



(b)

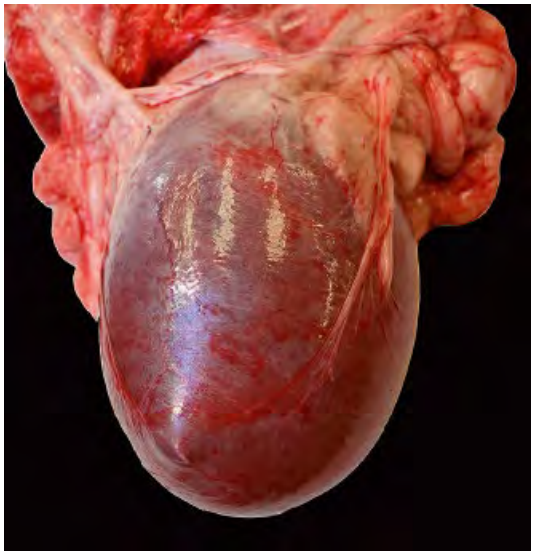


Figure 17.13 (a) Ultrasound image of a distended bladder (12 × 10.7 cm) from an 11-year-old male, intact llama with bilateral nephrolithiasis. (b) Image of the same bladder at necropsy.

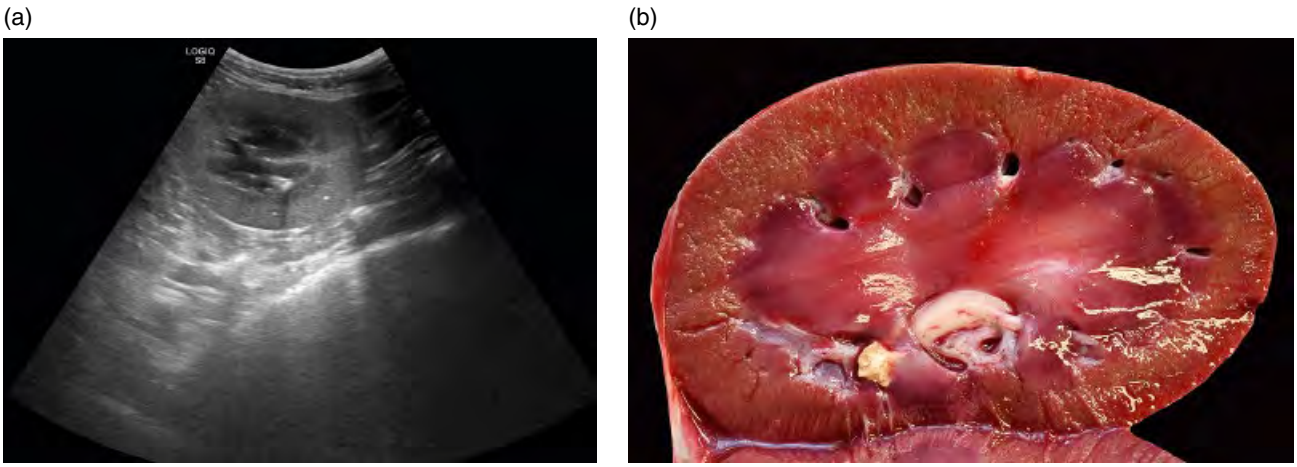


Figure 17.14 (a) Ultrasound image of a nephrolith in an 11-year-old male, intact llama with bilateral nephrolithiasis. (b) Image of the kidney at necropsy (same case as in Figure 17.13).

Figure 17.15 Preputial abscess due to urethral rupture in an 11-year-old castrated llama with obstructive urolithiasis.

The narrow urethra may predispose the accumulation of organic debris. A llama developed swelling of the preputial area that was diagnosed and treated initially as trauma and later as a cellulitis. At necropsy, the urethra was found to have ruptured proximally due to an obstruction at the glans penis caused by a plug of nonmineralized organic material. The urethra may also become obstructed by external pressure on the urethra caused by a hematoma of the corpus cavernosum urethra following penile trauma. As a sequela of a rupture of the urethra, a preputial abscess can develop (Figure 17.15).

Treatment

In most species, urethral obstruction caused by uroliths is more common in castrated males, but unfortunately, in llamas, breeding males are most frequently affected. The first task is to relieve the obstruction and restore urine flow if the bladder has not already ruptured.

In breeding males, an attempt is made to localize the obstruction and perform a urethrostomy over the urolith [38]. Unfortunately, the prognosis for return to breeding condition is poor to unfavorable. Concerning surgical options, the same techniques can be applied, as in small ruminants. Dietary management in herbivores is not highly successful.

Potential sequelae of urolithiasis include ruptured bladder [39], ruptured urethra, urethral stricture caused by trauma from the calculus, urethral stricture caused by scarring following surgical manipulation, chronic cystitis, and recurrence of calculi.

Neoplasia

Renal neoplasms have been reported in camelids, such as unilateral renal papilloma [11], multicentric urinary papilloma involving both kidneys and a ureter [40], both cases associated with anemia and hematuria. In one case, multicentric urinary neoplasia developed, associated with prolonged exposure to bracken fern. The llama developed multicentric transitional cell carcinomas of both kidneys, ureteral papilloma and urothelial hyperplasia [40].

In the case with unilateral renal papilloma, the involvement of only one kidney could be confirmed by cystoscopy, where blood could be seen flowing from one ureter. The alpaca was successfully treated by unilateral nephrectomy [11].

An urethral sarcoma has been reported in a seven-week-old female cria, presented with stranguria, pollakiuria, and hematuria [41].

Other renal neoplasms reported were renal teratoma in a llama [42], nephroblastoma in a guanaco [43, 44] and renal lymphosarcoma [44, 45].

Congenital Defects

Congenital abnormalities of the kidneys have been described in camelids. Unilateral [46] and bilateral [47] renal agenesis of the kidneys were reported in crias,

leading to azotemia and death. Both cases were diagnosed by abdominal ultrasound (US). Ureteral duplication was reported in a cria resulting in hydronephrosis [48]. Urinary bladder agenesis in a 10-day-old female alpaca resulted in bilateral hydronephrosis and hydroureter [49]. Defects of the urinary system can also occur in conjunction with other defects. Multiple congenital malformations have been reported in a three-day-old male alpaca with inguinal hernia, right renal and ureteral aplasia, and left testicular aplasia [50]. Bilateral ectopic ureters were detected in a six-month-old female alpaca, presented with dribbling urine, without an observed voided stream of urine [50].

Hydronephrosis due to malformations of the ureters can be accompanied with pyelonephritis [50, 51].

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18

Ophthalmology

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Anatomy of the Eye

Though the basic ocular anatomy is similar to that of other mammals (Figure 18.1), camelid eyes are relatively large with an axial globe length of approximately 38 mm. The globes are laterally placed in the skull, providing excellent peripheral vision and a broad field of view but limited binocular vision (Figure 18.2). Vision in alpacas is nearly emmetropic (normal vision) while llamas, especially females, have been found to be slightly myopic (near-sighted) [1].

Camelids are diurnal and have no need for a tapetum to intensify light [2, 3]. Both rods and cones are present in the retina indicating that camelids have color vision. Though llamas are not usually active at night, packers who have found themselves on the trail at night observe that llamas remain agile and have functional night vision [2]. Distant vision is likely similar to that of any grazing animals where well-developed motion perception is necessary to detect predators.

The periocular vibrissae and cilia are long and prominent. The eyelid margins are pigmented and do not contain meibomian glands [4]. A large, fleshy, haired caruncle of the third eyelid contains sebaceous glands that likely contribute to the pre-ocular tear film (Figure 18.3). The nasolacrimal anatomy is similar to sheep and horses. Paired canaliculi connect the ocular punctae of the upper and lower eyelids and converge at the lacrimal sac approximately 5 mm from the medial canthus (Figure 18.4). The duct traverses within the lacrimal bone for a distance of 2 cm in close association with the osseous canal of the infraorbital nerve (Figure 18.5), then exits the bone and continues submucosally within the nasal cavity to exit within the nares. The nasal puncta is 2–3 mm in diameter and may be visualized 15–20 mm proximal to the rostral border of the nasal vestibule (Figure 18.6) [5].

The cornea is 0.5–0.6 mm thick and ovoid in shape with a prominent curvature, a horizontal diameter of about 25–30 mm, a vertical diameter of 18–22 mm [6, 7]. The corneal epithelium is relatively thick, made up of 12–15 layers comprising 30% of corneal thickness. The relatively thick epithelium is thought to be protective against UV light, dehydration and trauma. The limbal junction of the cornea and conjunctiva is often heavily pigmented.

The iris of camelids is unique with large, pleated folds of posterior pigmented epithelium located dorsally and ventrally around the horizontally oval pupil rather than true corpora nigra (Figure 18.7). Pigmentation of the iris and fundus varies with coat color where more heavily pigmented coat colors are associated with brown irides and a darker brown fundus color and animals with white or variable coat colors may lack pigment in the iris causing a blue eye and a red fundic reflex when the pupil is illuminated due to lack of pigment in the retinal pigmented epithelium and choroid. Lack of pigment in the iris is associated with deafness in white cats and rabbits, Dalmatians and Australian shepherd dogs with double merle coloration. Deafness has also been recognized in white llamas and alpacas. However, not all white, blue-eyed llamas and alpacas are deaf, nor are colored animals with blue eyes. The lens and aqueous humor are similar to that of other ungulates [8].

The fundus of South American camelids (SACs) is characterized by a lack of a tapetum lucidum, a variably pigmented oval-shaped optic nerve head, a variably pigmented retinal pigmented epithelium, prominent retinal blood vessels, and commonly, a long, white hyaloid artery remnant (Bergmeister's papilla) extending from the center of the optic nerve. Retinal vessels converge on the optic disc, which is located in a medial ventral position (Figure 18.8).

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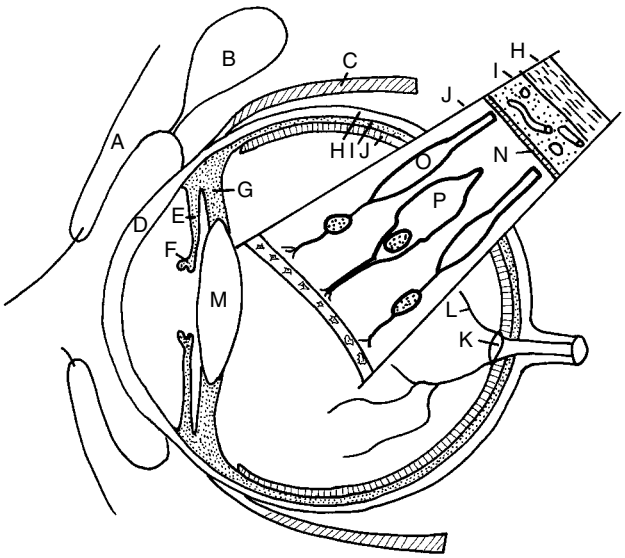

Figure 18.1 Diagram of a longitudinal section through the eye of a camelid. (A) Upper eyelid, (B) lacrimal gland, (C) extraocular muscle, (D) cornea, (E) iris, (F) corpus nigrum, (G) ciliary body, (H) sclera, (I) chorioid, (J) retina, (K) optic disc, (L) retinal vessels, (M) lens, (N) retinal pigmented epithelium, (O) rod, (P) cone. Diagram compliments of Dr. Murray Fowler.

Figure 18.2 Normal eyelid and globe conformation of an alpaca. Due to laterally placed globes, camelids have a very wide field of view but limited binocular vision.

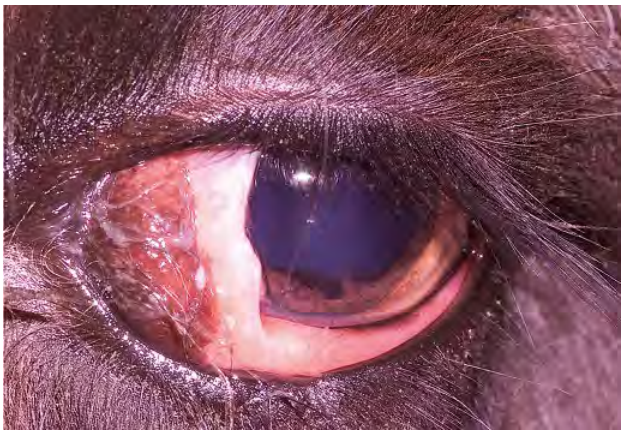

Figure 18.3 Camelids have long periocular vibrissae and cilia and the eyelids lack meibomian glands. The caruncle at the base of the third eyelid (nictitating membrane) is fleshy and haired and contains sebaceous glands. In this llama, the third eyelid is elevated and the conjunctiva is chemotic due to exophthalmia.

Figure 18.4 The nasolacrimal outflow apparatus consists of ocular puncta (identified with nasolacrimal cannulae) in the superior and inferior eyelid that drain tears through canaliculi to the nasolacrimal sac, the nasolacrimal duct and the nasal puncta. Photograph compliments of Dr. Murray Fowler.

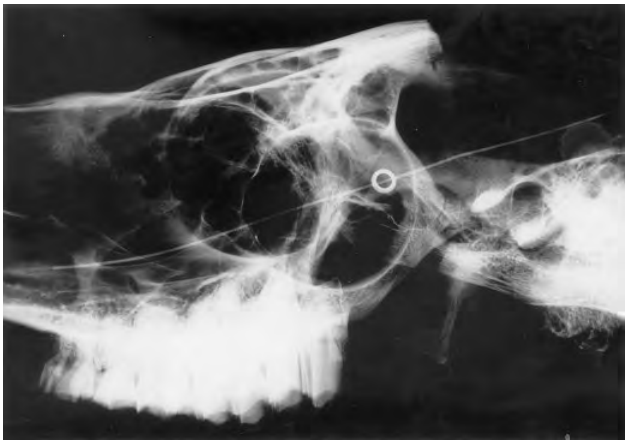

Figure 18.5 Lateral radiograph of a radiopaque cannula in the nasolacrimal duct illustrating the relationship to the roots of the maxillary cheek teeth. Photograph compliments of Dr. Murray Fowler.

Figure 18.6 The nasal punctum (arrow) is 2–3 mm in diameter and may be visualized 15–20 mm proximal to the rostral border of the nasal vestibule.

Figure 18.7 Heterochromic normal iris of an alpaca with a variegated coat color. Coat color dilution is associated with hypopigmentation and a blue color of the iris. The iris of camelids has large pleated iridal folds that are located dorsally and ventrally around the horizontally oval pupil rather than true corpora nigra.

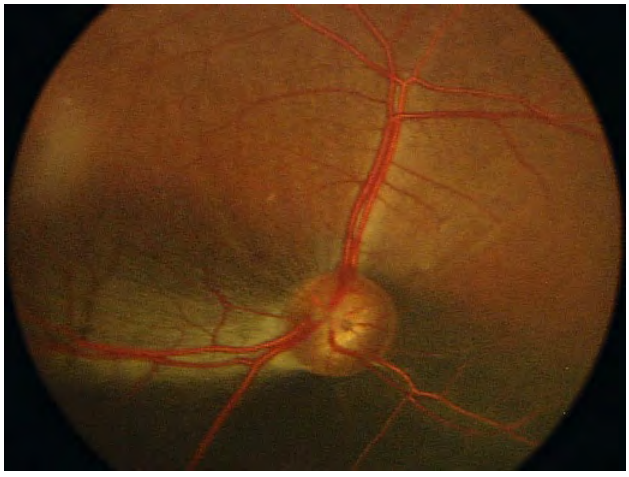

Figure 18.8 The ocular fundus of a llama. South American camelids do not have a tapetum lucidum. The optic nerve is variably pigmented, oval and well-myelinated. Blood vessels are prominant on the retinal surface.

Ophthalmic Diagnostic Procedures

Ophthalmic examination of camelids rarely requires sedation or eyelid nerve blocks and is conducted in a similar manner to that performed on other domestic large animals [3, 7, 9]. Cranial nerve examination, such as the pupillary light reflex and palpebral reflex, is an important part of a thorough evaluation. The menace response is learned and therefore not present in newborn crias.

The mean Schirmer tear test value is similar to other domestic species: 17–19 mm/min in llamas [4, 7, 10] and 21 mm/min in alpacas [11]. Topical application of fluorescein dye is used to rule out or assess the extent of a corneal ulcer as well as assess the patency of the nasolacrimal system. Appearance of the dye at the nostril rules out nasolacrimal obstruction; however, failure of the dye to appear at the nostril within five minutes does not confirm obstruction; confirmation requires nasolacrimal cannulation and irrigation (Figure 18.4).

Corneal culture and susceptibility are indicated for any corneal ulcer that is slow to heal, progressive and/or refractory to appropriate medical therapy and/or has other clinical features of infection including corneal cellular infiltrate or hypopyon. A cytology specimen may be collected with a Microbrush® dental applicator and stained with Diff-Quik to evaluate for evidence and type of inflammation and/or infection. The normal ocular flora is similar to other domestic species, where gram-positive bacterial species such as *Staphylococcus* and *Streptococcus* spp. are most common and the presence of some Gram-negative species such as *Pseudomonas* spp. and fungal species such as *Aspergillus* spp. is normal [12, 13].

The mean normal intraocular pressure (IOP) of llamas and alpacas using applanation and rebound tonometers is 12–15 mmHg [14, 15].

Pharmacologic pupil dilation is indicated for thorough evaluation of the lens, vitreous and fundus. One to two drops of tropicamide may be applied topically for complete pupil dilation within 15–30 minutes that lasts for 4–6 hours.

Anterior segment examination requires a bright light source such as a Finoff transilluminator, ideally with a source of magnification. Fundic examination can be performed using a direct ophthalmoscope. For a wider field of view, a PanOptic retinoscope can be used or indirect ophthalmoscopy can be performed using a transilluminator or flashlight with a 20 Diopter condensing lens.

Nasolacrimal irrigation may be performed in a normograde or retrograde fashion. The normograde approach requires cannulation of the ocular puncta with an irrigating cannula or a 3.5 French tomcat catheter (Figure 18.4) and irrigation is first performed from ocular puncta to ocular puncta, then after digital compression of the cannulated puncta, continues through to the nasal puncta [16].

Normograde irrigation is required to diagnose nasolacrimal anomalies, most of which are located distally (e.g. lack of nasal puncta) in camelids [16–19]. Retrograde irrigation may be performed using a 5 French 20″ urethral catheter through the nasal puncta (Figure 18.6). When normal nasolacrimal anatomy and outflow cannot be verified, cannulation with a radiopaque cannula or dacryocystorhinography may be performed (Figure 18.5). Survey lateral skull radiographs are performed, then the ocular puncta is cannulated and approximately 3–5 mL of a radio-opaque contrast agent is injected into the nasolacrimal duct to identify the site of the obstruction. Alternatively, a 5 French 20″ urethral catheter can be advanced from the ocular puncta to the site of the obstruction and is usually palpable beneath the nasal mucosa.

Ocular ultrasonography is indicated when the anterior and/or posterior segment cannot be visualized or for evaluation of orbital disease. A 10–20 MHz probe is preferred. Electroretinography has been performed in camelids presented for blindness to assess retinal function. Waveform amplitudes are higher in camelids than in most normal cats and dogs [20]. Computed tomography (CT) and Magnetic Resonance Imaging (MRI) have been performed in camelids for assessment of intracranial, orbital or sinus disease processes.

Diseases of the Eye

A retrospective study found at least one ocular lesion in 6% of llamas presented to veterinary teaching hospitals [17]. The prominent nature of the camelid eye predisposes it to traumatic injury [3]. Corneal lacerations and abrasions are the most common ocular lesions seen. Uveitis is also relatively common as a sequela to systemic infection. Camelids also appear to have a relatively high prevalence of congenital eye lesions.

Systemic infectious diseases involving the eye include toxoplasmosis, aspergillosis [21], coccidioides [22] and equine herpesvirus-1 [23]. Uveitis is the most common ocular manifestation of these diseases. Panophthalmitis may be seen in septicemic animals, especially immune-compromised crias [24]. Uveitis is responsive to standard therapy, but the underlying condition may be refractory to treatment. Protection from light, topical atropine administration once daily, topical and/or systemic corticosteroids or non-steroidal anti-inflammatory agents, and specific antibacterial or antifungal agents may be useful to address the ocular manifestations of these disorders. Severe vision-threatening sequela such as posterior synechia, cataracts and retinal detachment may occur (Figure 18.9).

Figure 18.9 Alpaca with chronic severe anterior uveitis. Diffuse corneal edema, active superficial corneal vessels, aqueous flare, posterior synechia and mature cataract can be seen.

Figure 18.10 *Thelazia* spp. in the conjunctival sac and on the corneal surface. Photograph compliments of Dr. Murray Fowler.

Parasitic diseases are discussed in Chapter 7 and include those caused by *Thelazia californiensis* (Figure 18.10) and various species of flies attracted to lacrimal secretions. The most common sequela of ocular parasitic infections/infestations are conjunctivitis and blepharitis.

Corneal ulcers are usually caused by trauma and often become secondarily infected [7, 17]. Primary corneal pathogens and associated herd outbreaks are rare in camelids. Clinical signs of corneal ulceration include squinting, light sensitivity, periocular swelling, tearing and focal to diffuse corneal opacity. Focal corneal edema, characterized by a bluish "ground glass" appearance, indicates a break in the corneal epithelium. Corneal cellular infiltrate indicating inflammation and/or infection appears white to yellow (Figure 18.11). Fluorescein staining is helpful to identify the location and extent of ulcers. Culture specimens should ideally be collected prior to staining or placement of any substances onto the eye but proparacaine can be applied if severe blepharospasm impedes precise sample collection.

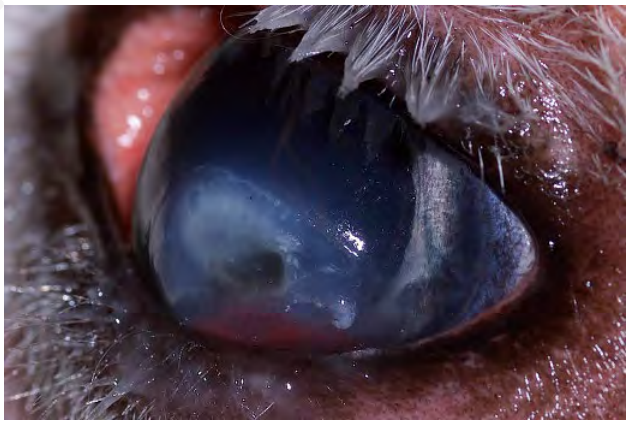

Figure 18.11 Infected corneal ulcer in an alpaca with facial nerve paralysis. Note focal corneal edema surrounding an oval-shaped area of white cellular infiltrate in the axial cornea. Focal stromal loss has occurred at the ventronasal margin. Active corneal vessels can be seen extending from the ventral limbus. Fungal hyphae were found on a cytology specimen and *Aspergillus* spp. were cultured.

Figure 18.12 Young llama with chronic keratoconjunctivitis attributed to equine herpesvirus. Marked conjunctival hyperemia, chemosis third-eyelid elevation and active superficial corneal vessels are seen.

Cultures should ideally be processed anerobically as well as aerobically, because up to 20% of samples have yielded anerobic pathogens [25]. Cytology samples should be performed to evaluate for neutrophils, rod or cocci bacteria and fungal hyphae to direct initial therapy pending culture results.

Uncomplicated superficial corneal ulcers usually heal within five to seven days when treated prophylactically with topical broad-spectrum antibiotics such as bacitracin-neomycin-polymixin B (q6h). Topical atropine (q12-48h) and systemic anti-inflammatory medications such as flunixin meglumine administered early in the treatment course may decrease discomfort and uveitis. If fungal infection is suspected or confirmed, topical antifungal agents should be added to the treatment plan. Miconazole ointment (q6h) may be used, but it does not adequately penetrate intact corneal epithelium so is not effective against stromal abscesses. Voriconazole penetrates the cornea and has a wide spectrum of efficacy when applied topically q 4-8 hours. Voriconazole may be obtained from a compounding pharmacy or the powdered form for intravenous (IV) use can be reconstituted with 19 mL sterile saline to make a 1% solution.

With deeper and/or infected corneal ulcers, more aggressive and longer-term treatment is necessary. Subpalpebral lavage systems may be warranted in camelids requiring frequent treatment, or who become refractory to direct application [26]. Topical antibiotics may be given more frequently (up to 6 times/day) and/or a fluoroquinolone antibiotic such as levofloxacin or ofloxacin may be selected for better corneal penetration [16]. Serum may be collected from the patient to be administered topically (q2-4h) or topical oxytetracycline-polymixin B ointment may be applied (q6h) for anticollagenase effects to slow or stop progression. If the ulcer appears to be worsening, or the depth exceeds 50% of the stroma, the affected cornea may be surgically debrided and a conjunctival pedicle graft applied to provide structural support and blood supply to the defect. The graft is left in place for at least 8–12 weeks but can be trimmed even months to years after the surgery. Perforating ulcers or wounds may also be repaired surgically [27]. Residual corneal scarring may result from chronic or severe ulceration.

Corneal opacity without superficial ulceration most commonly occurs due to a bacterial or fungal stromal abscess [22], but differential diagnoses should include lipid deposition associated with hypercholesterolemia [28] or corneal fibrosis. Corneal tumors have not been reported. Diagnosis via corneal culture or a cytologic specimen is precluded by intact corneal epithelium, but a bacterial or fungal corneal stromal abscess will recruit corneal vessels and elicit ocular pain and inflammation whereas non-infectious infiltrate usually will not. Corneal vascularization in camelids is typically characterized by thick caliber superficial vessels with prominent distal branching (Figure 18.12).

Camelids appear to be prone to developing bullous keratopathy associated with corneal disease, anterior uveitis and cataract surgery [17, 20]. Compromise of the endothelial function of pumping fluid from the corneal stroma results in severe edema, swelling and deformation ("hydrops") of the cornea. It is presumed that the endothelium of camelids is more susceptible to dysfunction after insult but the pathogenesis has not been fully elucidated. The density of camelid endothelial cells is similar to that of other species, but morphologic heterogeneity of the cells has been described [6].

Figure 18.13 Llama with congenital persistant hyperplastic primary vitreous/persistent hyperplastic tunica vasculosa lentis (PHPV/PHTVL). A white retrolental plaque with patent vascular remnants can be seen. Multifocal posterior synechia are sequela to lens-induced uveitis associated with the compromised posterior lens capsule integrity.

Some congenital eye anomalies are discussed in Chapter 21 and are the second most common ophthalmic abnormality found in camelids presented to veterinary teaching hospitals [17]. Lesions range from mild to blinding and include microphthalmia, entropion, ectropion, dermoids, cataracts, persistent embryonic vasculature (Figure 18.13), lens and optic nerve coloboma, cyclopia and nasolacrimal atresia [3, 17, 29, 30].

Atresia of the nasal puncta of the nasolacrimal duct may be unilateral or bilateral [17–19]. The main clinical sign is tear overflow with periocular wetting, usually beginning soon after birth. Discharge persists and may become more mucopurulent with chronicity as secondary conjunctival bacterial overgrowth occurs. Failure of fluorescein to pass from the eye into the nose, inability to flush the nasolacrimal duct, or contrast radiographs may be useful diagnostically. Correction involves passing polyethylene tubing from the lacrimal puncta as far as it will go, injection of lidocaine through the tubing to form a bleb in the nostril, incising the mucosa over the bleb, and either pushing the tubing through the submucosal tissue out the incision, or grasping the tip with forceps and pulling it out. Tubing can be sutured to the outside of the nostril and the eyelid and left in place for two to six weeks to allow the new orifice to epithelialize. Topical ophthalmic antibiotics, or antibiotic-steroid combinations should be administered 2–4 times daily while the tube is in place. When the tube cannot be palpated or advanced to the expected length of the nasal passage, dacryocystorhinography is helpful to identify the more proximal site of the atresia (Figure 18.5). In such cases, alternative corrective surgical procedures such as conjunctivorhinostomy, routing lacrimal outflow from the conjunctival fornix into the nose, may be necessary.

Figure 18.14 Congenital nuclear cataract in a mature alpaca with apparently normal vision function. The focal white opacity is visible through the undilated pupil. Ocular adnexa, cornea and iris are normal.

Congenital cataracts are commonly diagnosed in camelids and, in most cases, are presumed to be heritable [7, 17, 30]. Small or nuclear cataracts may not obscure vision as the globe and lens grow (Figure 18.14) and may be found incidentally during exam. However, congenital cataracts in neonates may be diffuse and cause blindness. Progressive nuclear and cortical cataracts may partially spontaneously resorb without surgery, but the animal is left with far-sighted vision, and some degree of residual lens opacity is common [16]. Progressive cataract resorption often leads to an immune-mediated uveitis recognized by conjunctival hyperemia, miosis, iris hyperpigmentation and posterior synechia. During resorption, the affected eye should be treated with a topical non-steroidal anti-inflammatory agent such as diclofenac 0.1% or a topical corticosteroid such as 1% prednisolone acetate every 12–48 hours as needed to control the inflammation. Atropine may be applied topically to prevent formation of posterior synechia.

Cataract surgery can be performed in some cases but camelids may have an increased risk of corneal endothelial decompensation with subsequent development of severe corneal edema and bullous keratopathy [7, 16, 17]. However, young alpacas may have less risk than llamas and use of specific irrigation solutions during the cataract removal procedure may help reduce this risk [31]. Another reported complication of cataract surgery is development of secondary glaucoma [31, 32]. Currently, there is no artificial intraocular lens implant available for camelids; the resulting post-operative vision is far-sighted.

Acquired cataracts are uncommon but may occur in camelids under similar conditions as in other species [3, 17]. Previous inflammatory or traumatic lesions (Figure 18.15a and b), advancing age, and persistent hyperglycemia are possible risk factors.

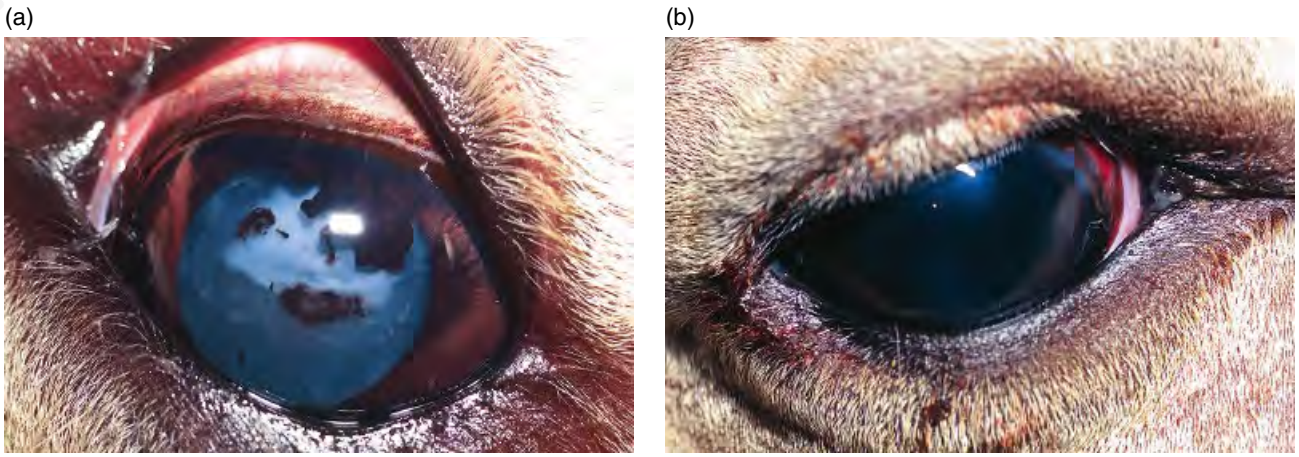

Figure 18.15 (a) Traumatic cataract in a llama. The iridal folds have avulsed from the superior pupil margin and multifocal iridal pigment deposition is noted on the anterior lens capsule. A mature cataract with a focal dense capsular opacity in the dorsal paraxial lens resulted in blindness. (b) Vision was restored after phacoemulsification. Photograph was taken one day postoperatively.

Glaucoma has rarely been reported in camelids but may occur in association with multiple congenital ocular anomalies or secondary to chronic uveitis [7, 17], or intraocular surgery [31, 32]. Topical treatment with IOP lowering drugs such as 2% dorzolamide or the combination of 2% dorzolamide-0.5% timolol q 8–12 hours may effectively lower the IOP.

Retinal detachment may occur secondary to traumatic globe rupture or can occur subsequent to severe chorioretinitis associated with systemic disease [7, 17]. Blindness due to retinal detachment is typically irreversible. Other ophthalmic conditions include trauma to the eyelids or cornea [33, 34] (laceration, abrasion, ulceration, foreign body penetration) and intraocular tumors [3, 35–39]. Enrofloxacin-induced retinopathy has also been reported [40].

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19

Nervous System

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Primary neurologic disease in camelids is widely reported in the literature, some diseases being common in clinical practice, but many being uncommon. Having an understanding of common neurologic diseases that affect camelids as well as a practical treatment approach is important for the veterinary practitioner.

The general morphology of the nervous system of camelids does not differ from that of other domestic ungulates, nor does the basic distribution of peripheral nerves [1]. The location of nerves important in surgical procedures have been discussed in the appropriate sections of this book.

Diagnosis

The neurologic examination of camelids is similar to that of other domestic species and lesion localization can be performed in a similar manner as in traditional small ruminants (brain and spinal cord) and dogs (spinal cord), though camelids tend to respond more slowly to many of the stimuli.

Further routine evaluation of the central nervous system (CNS) can be performed using plain film radiography and cerebrospinal fluid (CSF) analysis. The usefulness of radiography without contrast is limited to osseous changes such as fractures, malalignments, osteomyelitis, discospondylitis, or vertebral body abscesses. Cytologic analysis of CSF is an important tool to help diagnose inflammatory diseases of the CNS and is especially useful in diagnosing *Parelaphostrongylus tenuis* (meningeal worm) infestation [2, 3]. The normal composition of CSF from healthy adult llamas has been identified and reported [4]. CSF may be obtained from the atlanto-occipital (AO) or the lumbosacral (LS) space (Chapter 4). The LS space is much more commonly used as it is easier and requires only local anesthesia and little or no sedation.

Advanced diagnostics that can be useful when evaluating a neurologic patient include CT, MRI, myelogram, fluoroscopy, EMG, and EEG. While these tools may help provide a diagnosis, their use in clinical practice is often unavailable or impractical. See Chapter 5 for further discussion on advanced imaging modalities.

Common Neurologic Diseases

Polioencephalomalacia

Polioencephalomalacia (PEM, polio) is a histopathologic diagnosis that means softening or necrosis (-malacia) of the gray matter (polio-) of the brain (encephalo-) [5]. The most common cause of PEM is thiamine deficiency and the term "polio" is often used to describe thiamine deficiency, even though there are other causes of the lesion.

Thiamine (vitamin B1) and other B vitamins are produced by microbes in the rumen/C1. Thiamine deficiency, in its simplest form, is a result of dysbiosis in the fermentative compartment, leading to decreased production of thiamine. Thiamine deficiency is often the consequence of a rapid feed change, especially a shift to highly fermentable feed (grain overload). When this occurs, thiamine producing bacteria start to die off and the population of thiaminase producing bacteria thrive.

Thiaminase activity can also be increased by ingestion of preformed plant thiaminases or possibly by the administration of certain medications (promazines, levamisole, benzimidazoles, and others) that can act as cofactors in thiaminase production [6]. Additionally, amprolium can act as a thiamine analog and over administration can block the activity of thiamine. The net result of all of these is significantly decreased thiamine absorption from the

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fermentative compartment. These causes of PEM are considered thiamine responsive.

Non-thiamine responsive causes of PEM include sulfur toxicity, lead toxicity, and water intoxication. All causes of PEM result in sodium and water dysregulation, either directly or by interfering with the Na^+/K^+ ATPase pump. When intracellular sodium concentrations increase, water follows and swelling occurs which, in turn, leads to necrosis due to the lack of space for the brain to swell.

Clinical signs of PEM are associated with symmetric cerebrocortical damage and include dull mentation, separation from the herd, head pressing, recumbency, opisthotonos, and pathognomonic cortical blindness (absent menace response with intact pupillary light reflex) and dorsomedial strabismus. In camelids, reported clinical signs include cortical blindness [7, 8], wandering aimlessly [7], depression [7], tremors of the head and neck that were exaggerated by external stimuli [7], ataxia [7], head pressing [7], circling in stall [7, 9], sternal or lateral recumbency [7, 8], convulsions [7, 8], opisthotonos [7, 8], muscle fasciculations [8], and seizures [9].

The cornerstone of treatment for PEM, no matter what the cause, is thiamine at 10 mg/kg q six hours SC or IM. In severe cases the first dose can be administered IV, either very slowly or diluted as some animals will have a reaction if given rapidly IV. Another option is to administer the total daily dose of thiamine over 24 hours as a continuous rate infusion (CRI) if IV fluids are being administered. Polio patients should also be treated with anti-inflammatories such as dexamethasone or flunixin meglumine. Treatment with thiamine and anti-inflammatories should be continued for at least three days or until signs resolve and eating and cud chewing have resumed.

For severe cases, an osmotic diuretic can be used in the first day to reduce brain swelling. Mannitol has historically been recommended and is likely the best option for reducing brain swelling in camelids with PEM. The preferential use of hypertonic saline to reduce intracranial pressure is often discussed and may be a good choice for other species with other causes of cerebral edema such as traumatic brain injury [10]. One of the advantages of hypertonic saline in other situations is that the sodium gradient is regulated by the Na^+/K^+ ATPase pump, whereas mannitol can sometimes "leak" across the blood–brain barrier, diminishing and eventually reversing the gradient [10]. Because in many cases of PEM the Na^+/K^+ ATPase pump is not functioning, using sodium to set up the osmotic gradient may not be effective and may worsen the problem, therefore mannitol is likely a better choice. Another caution for using hypertonic saline in camelids is their propensity to retain sodium and become hypernatremic.

Meningeal Worm

Meningeal worm or parasitic myelopathy/encephalopathy is caused by the migration of *P. tenuis* larva from the GI tract to the spinal cord or brain of an aberrant host. The definitive host for *P. tenuis* is the white-tailed deer (*Odocoileus virginianus*) and the intermediate hosts are snails and slugs. In the definitive host, the parasite causes no known clinical signs. In aberrant hosts, including camelids, sheep, goats, and other species of deer, migration of the larvae causes disruption of the normal neural tissue and neurologic signs associated with the migratory path. Histopathologic lesions have been well described and include identification of the parasite and linear tracts of hemorrhage and inflammation [3, 11, 12] (Figure 19.1a and b).

Meningeal worm is one of the most common neurologic diseases of camelids in the United States [13–15]. Clinical signs can be varied but most commonly include wide-based stance and decreased proprioception in the early or mild forms of the disease. Ataxia and severe weakness progressing to the inability to stand is frequently seen in more advanced stages (Figure 19.2). Often one leg is worse than the other and can sometimes be mistaken for lameness. Ascending migration to the cervical spine and brain sometimes occurs with accompanying clinical signs [14, 16].

Diagnosis of meningeal worm is often empirical, and treatment is commenced without a definitive diagnosis. Antemortem diagnosis can be achieved by demonstrating eosinophilic inflammation in the CSF. One study indicates that >17% eosinophils or >1.4 eosinophils/μL in CSF is appropriately sensitive and specific for the antemortem diagnosis of meningeal worm [2]; however, the presence of any eosinophils in the CSF is highly suggestive of meningeal worm. If blood contamination is present, the eosinophil percentage should be compared to the percentage of eosinophils in the blood.

The most common treatment protocol recommended is fenbendazole 50 mg/kg orally once daily for five days [3, 14, 15, 17], non-steroidal anti-inflammatories such as flunixin meglumine once or twice a day or corticosteroids [3, 14, 15, 17], supportive care [14, 15], and physical therapy. Physical therapy, i.e. helping the animal to rise multiple times a day, is critical to recovery. One study showed that the administration of corticosteroids significantly decreased survival [3], though it is unclear whether low numbers or case bias affected this outcome. Other therapies that are sometimes implemented include dimethylsulfoxide (DMSO) [3, 14, 15, 17], and Vitamin E [3, 14].

Prevention strategies often discussed include environmental manipulations to control deer and mollusks, but these strategies are very difficult and likely unrewarding. Monthly ivermectin injections (0.2–0.3 mg/kg) are possibly

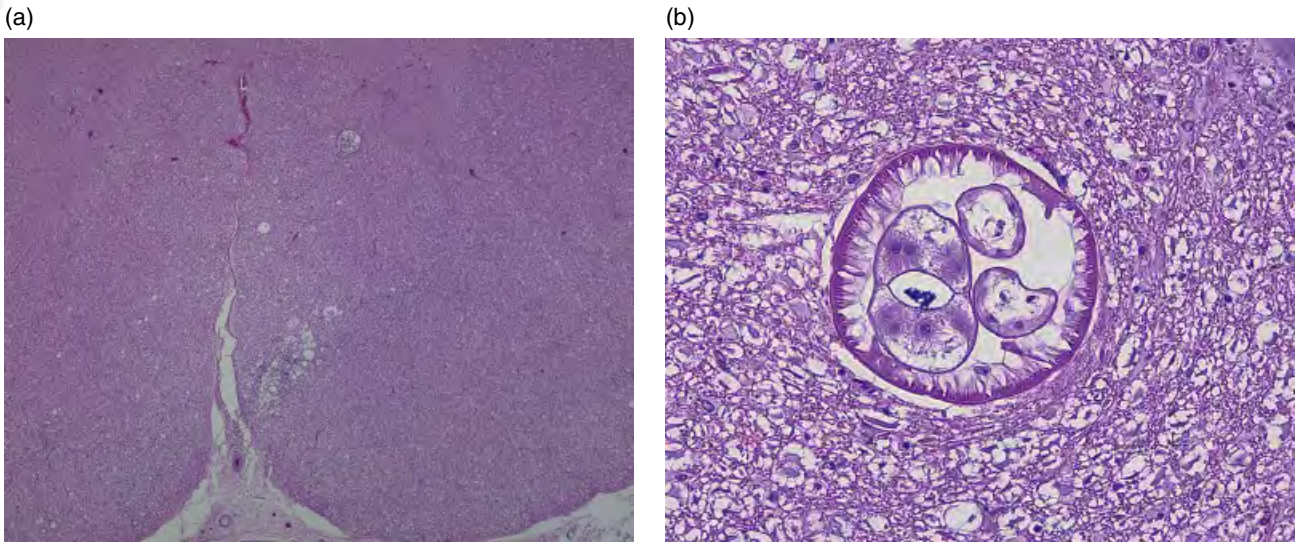

Figure 19.1 Histopathologic section of a spinal cord containing inflammatory cells and a cross section of a *P. tenuis* larva. (*Source:* Photos courtesy of Troy A. Brick, DVM, MS).

Figure 19.2 Llama exhibiting crossed rear limbs and posterior ataxia secondary to aberrant migration of meningeal worm.

the most useful preventive [15]. This strategy will hasten the development of ivermectin resistant gastrointestinal (GI) parasites, which must be considered when devising a GI parasite control program (See Chapter 7).

Encephalitic Listeriosis and Otitis Media/Interna

Encephalitic listeriosis is a very common neurologic disease in traditional small ruminants, but has only been reported a few times in camelids [18, 19]. When the gram-positive bacteria, *Listeria monocytogenes*, gains access to the brainstem, microabscesses can form leading to unilateral cranial nerve deficits. The widely accepted pathophysiology is that the bacteria gain access through retrograde travel up the trigeminal nerve from the mouth to the brainstem. The other possibility is that bacteremia leads to seeding of the brainstem with the bacteria. The organism is ubiquitous in the environment and is commonly ingested without consequence. Outbreaks of this disease in cattle are often associated with ingesting poorly ensiled feedstuffs but, since small ruminants and camelids are rarely fed silage, it is a more sporadic disease.

Clinical signs commonly associated with encephalitic listeriosis suggest a unilateral brainstem lesion and include circling, head tilt, decreased tongue tone, weak jaw closure, signs of facial nerve paralysis (ear droop, decreased/absent facial sensation, inability to blink one eye), and vestibular signs (head tilt, nystagmus and inability to stand or sit sternal) [5, 20]. Reported clinical signs in camelids include weakness [19], head tilt [19], facial paralysis (ear droop, inability to close one eye, flaccid lips) [19], ataxia [19], and recumbency [19], but deficits in any of the cranial nerves V-XII should make encephalitic listeriosis a differential.

Treatment for listeriosis consists of antimicrobial and anti-inflammatory therapy as well as supportive care. The most common antibiotics used are penicillin or oxytetracycline. Flunixin meglumine or dexamethasone can be used to treat the inflammation. The primary differentials for this disease are meningeal worm and otitis media/interna. Because meningeal worm migration can cause similar signs, adding fenbendazole to the treatment regimen is advised.

Figure 19.3 Llama with head tilt and right-sided facial paralysis typical of animal with listeriosis or otitis media/interna.

Otitis media/interna is commonly an extension of otitis externa. Clinical signs are very similar to those of listeriosis, often starting with a simple ear droop and progressing to cranial nerve deficits as the infection worsens (Figure 19.3). The treatment of otitis media/interna, for the general practitioner, is similar to the treatment for listeria (antibiotics and anti-inflammatories), possibly with the addition of ear flushing and topical ear medications. If resolution is not achieved, more advanced diagnostics and surgery may be required.

Tick Paralysis

Tick paralysis has been recognized for more than 50 years, with many different species of tick being able to cause paralysis and many different mammalian species, including humans, being affected. Reports of tick paralysis in camelids have all come from the western United States [21, 22] associated with *Dermacentor* spp. and Australia [23] associated with *Ixodes holocyclus*.

In the United States, the neurotoxin from *Dermacentor* spp. interferes with acetylcholine release at the neuromuscular endings, causing a lower motor neuron paresis. The bite from a single tick is sufficient to kill an animal if the tick is not removed.

Reported clinical signs include lethargy [21], weakness [21], recumbency [21–23], paresis progressing to motor paralysis [21–23] with intact sensation [21, 23], opisthotonus [21], head tremors [21], and seizures [21]. The onset of clinical signs in these cases was rapid and progressive. After removal of the offending tick, many cases started responding within a few hours with all animals being able to stand after one to five days and fully recovered in three to nine days [21, 22]. The case from Australia was improving but ended up succumbing [23]. It is unclear whether this animal died because of the known poor prognosis associated with *Ixodes holocyclus* tick paralysis or because of the secondary metabolic disease that had developed.

In general, affected camelids follow a similar progressive course of paresis and paralysis as noted in other species. Ataxia develops, followed by a loss of all motor functions. Pain perception remains. The cause of death is respiratory failure.

No drug can counter the effects of the neurotoxin. The cure is to remove the tick from the animal and provide ancillary care until the signs resolve. Removal of the tick may start to reverse the signs within 2–12 hours [21, 22]. The difficulty lies in finding a tick on an unshorn llama or alpaca. Because only one tick may be sufficient to cause the paralysis, finding it is crucial. A topical permethrin, fipronil, or other anti-tick medication can be used to kill unlocated ticks or, potentially, as a preventive.

Less Common Neurologic Diseases

Viral Encephalitides

Rabies. Rabies is a viral encephalitic disease endemic in many areas of the world. Like all other mammals, camelids are susceptible to this rhabdovirus. The virus has been studied extensively because of its zoonotic aspect and its high mortality rate once clinical signs appear.

Rabies virus is primarily spread by bites from infected animals. The virus has been found in saliva and other body excretions, but it cannot penetrate the unbroken skin. Reservoir hosts vary with location. In the United States, rabies is controlled in dogs and cats by vaccination, but several distinct rabies virus variants have been identified in terrestrial mammals including raccoons, skunks, and foxes. In addition to these terrestrial reservoirs, several species of insectivorous bats are also reservoirs for rabies [24].

Over the years, several multi-llama and alpaca cases have been reported associated with rabid dog attacks in Peru [25–26]. Rabies has been reported in dromedary camels in all dromedary raising countries [25]. In the United States, rabies in camelids is reported at a rate of one to two cases every one to two years with no apparent regionality [27–32].

The reported incubation period is 15–31 days, with death occurring six to eight hours after clinical signs [26]. Early

signs of rabies in camelids include lameness, ataxia, and posterior paresis. Early signs are followed by either an aggressive syndrome (furious rabies) or a paralytic syndrome (dumb rabies) [26].

In the aggressive form, a normal body temperature is usually recorded until the animal becomes aggressive and there is excessive muscular activity. The major signs in this form are attacks on pen mates, offspring, people, and themselves. The rabid animal may also bite inanimate objects, chew foreign objects, and become self-destructive by pushing into and over obstacles. Other signs that have been observed during the course of the disease include vocalization changes (alarm cries without cause), bloat, pruritus, muscle tremors, aimless running, sexual hyperactivity (spontaneous ejaculation), recumbency, convulsions, coma, and death in one to four days. Females may refuse to permit nursing. Camels characteristically yawn in the terminal recumbency stage [25].

Paralytic rabies in camelids is characterized by anorexia, depression, head droop, ptosis, tenesmus, salivation, circling, facial paralysis, mild fever (39.4 °C [103 °F]), flaccid muscles (face, anus, bladder), and pharyngeal/laryngeal paralysis [25].

There is no officially recognized rabies vaccine for use in camelids, however, killed rabies vaccines have been used in endemic areas. Clinical research has demonstrated that llamas respond to killed vaccines with titers that are considered protective in other species [25]. Modified live vaccines for rabies should not be used [26].

Equine Herpesvirus 1. Herpesviruses are highly evolved and usually well adapted to one or more hosts. Interestingly, no herpesviruses unique to camelids have been identified [25]. In 1984, blindness was diagnosed in 21 alpacas and llamas of a herd of approximately 100 animals [33]. The condition was ultimately attributed to infection with Equine herpesvirus 1 (EHV-1). Blindness was complete and irreversible. Four animals exhibited neurologic signs in addition to blindness. Two of those animals died of encephalitic complications. Subsequently, neurologic disease and death was induced in two of three llamas that were experimentally infected with EHV-1 that was isolated from the brain of an alpaca that died in the previously described outbreak [34]. Camels exhibit neurologic signs but not blindness [35]. Acute and convalescent titers can be measured as camelids do mount an immune response to the virus [34]. There are very limited reports of this disease, therefore it is considered rare.

Bornavirus. Borna disease is a viral disease primarily of horses and donkeys but has been sporadically reported as causing non-purulent meningoencephalitis and death of new world camelids (NWCs) in Germany [36, 37]. This disease is considered rare.

Eastern Equine Encephalitis. The Eastern Equine Encephalitis virus (EEE) is endemic in reservoir hosts which are birds, especially those found in freshwater swamplands in the western hemisphere. EEE is primarily seen in the eastern states of the United States, in Central and South America, and the Caribbean. Horses, humans, and South American Camelids (SAC) are dead-end hosts [25]. The disease was first described in eight alpacas and one llama in a retrospective study from 2007. All cases were from the east coast of the United States from New Hampshire to North Carolina in late summer/early fall. Most of the cases were ≤1-year-old.

Clinical signs were consistent with intracranial disease and included fever, lethargy, ataxia, mentally inappropriate behavior, seizures, recumbency, torticollis or opisthotonos, vestibular signs, and cranial nerve deficits (only one case). Only one animal survived. A subsequent study showed that vaccinating alpacas with a killed bivalent (eastern and western equine encephalitis virus) vaccine approved for use in horses induced a humoral antibody response against EEE after three doses given at four-week intervals [39]. Vaccinating alpacas for EEE may be considered in endemic areas.

West Nile Virus. West Nile Virus (WNV) is a mosquito-borne flavivirus that causes encephalitis primarily in birds, equids, and humans and is maintained naturally through a mosquito-bird-mosquito transmission [39]. The virus is indigenous to Africa, Asia, Europe and Australia but has, more recently, spread to other parts of the world including the United States [39]. The virus first appeared in the United States in 1999 [40] and was first recognized in a camelid patient in 2002 [41].

Reported clinical signs in alpacas include lethargy [42], inappetence [42], fever [42], torticollis or opisthotonos [41–43], pain on palpation of abdomen, spine, and pelvis [42], ataxia [41, 42], stiff gait [42], decreased proprioception [43], hyperesthesia [41, 43], recumbency [41–43], dull mentation/obtundation [41–43], head and facial tremors [43], unilateral miosis or mydriasis [42], respiratory distress [41], and death [41–43]. Analysis of blood and CSF showed non-specific changes [41–43]. Acute and convalescent serum titers can help with the diagnosis but may be difficult to interpret if animals have been vaccinated [43].

There is no specific treatment. Supportive care and anti-inflammatories are indicated but mortality is high. In one report, two animals were treated with hyperimmune llama plasma containing antibodies for WNV [43]. The animal that was treated early in the clinical disease survived.

Vaccines for WNV are used effectively in horses and humans. Using commercially available equine WNV vaccine has been evaluated in alpacas and llamas [44].

Administering three doses of commercial equine WNV vaccine at three-week intervals to alpacas and llamas is able to establish titers similar to those observed in horses after two doses.

Louping Ill. Louping ill is an encephalomyelitis, primarily of sheep, caused by a flavivirus [45]. The louping-ill virus is endemic to specific regions of Great Britain [25, 45]. The infection is seen in many species of wild birds and mammals including sheep, humans, horses and camelids. The virus is transmitted by ticks, primarily *Ixodes rincinus* [25, 45]. In camelids, Louping ill virus produces an encephalitis characterized by anorexia, lethargy, hyperexcitability, ataxia, goose-stepping gait, and progressive paralysis [46, 47]. Supportive care and anti-inflammatories are recommended. Prevention is by the control of ticks and vaccination where available. This disease is rare in camelids.

Tetanus

C. tetani occurs worldwide as a soil saprophyte, but it can also be found in the feces of horses, humans, and cattle. Tetanus develops when wounds have been contaminated with soil or feces containing *C. tetani* spores. Contaminated deep wounds with devitalized tissue are most at risk. Such wounds are poorly aerated, providing optimum conditions for growth of the anaerobic organism.

Tetanus has been described several times in camelids [48–52]. Clinical signs are similar to those in other species and include prostration, muscle rigidity/sawhorse stance, dyspnea, tonic muscular contractions, joint stiffness, a fixed stare, erect ears, a locked jaw, protruding nictitating membrane, and a fever (Figure 19.4).

Treatment is also similar to that of traditional ruminants which includes wound debridement and aeration, high doses of penicillin, high doses of tetanus antitoxin, and supportive care as well as muscle relaxants, tranquilizers, or sedatives, and providing a stimulus free environment.

Figure 19.4 Llama with tetanus. Animal has an inability to masticate and swallow resulting in an accumulation of hay in its mouth.

Tetanus toxoid vaccines are readily available. Studies have demonstrated that llamas respond to tetanus toxoid vaccination with a rise in titer, but challenge studies have not been conducted [53]. Tetanus toxoid is part of the vaccine that is considered "core" in small ruminants and camelids, *Clostridium perfringens* types C and D and *C. tetani* (CDT).

Botulism

C. botulinum is distributed worldwide. No cases have been reported in NWCs but has been reported in a group of dromedary camels in Chad [54].

The syndrome is believed to be similar to that seen in most other mammals, consisting of a progressive flaccid paralysis of all muscles including respiratory muscles. Body temperature is not elevated. The pupils of the eyes become dilated. Once signs have developed, little can be done other than respiratory support. Antitoxins used to treat human cases are not available for animals. No vaccines against botulism are available for protection of animals or humans.

Dysautonomia

Dysautonomia is a disease that is characterized by degeneration of the motor neurons of the autonomic nervous system, most notably of the enteric nervous system [46]. Equine Grass Sickness, a form of dysautonomia, has been recognized in horses since the early twentieth century [55] and is considered a polyneuropathy because the skeletal lower motor neurons are also affected [5]. Dysautonomia has been diagnosed associated with GI ileus in several camelids [56–58].

Clinical signs include lethargy [56–58], colic signs (laying in lateral recumbency and rolling) [56, 58], posturing to urinate and deficate [57], lack of fecal output [56–58], inappetence [57, 58], weakness [57, 58], tremors [58], absent GI sounds [57, 58], dilated pupils with absent pupillary light reflexes [57], dilated anal sphincter and rectum [57], distended urinary bladder [57], and dysphagia [58]. None of the animals in the reported cases survived. Gross necropsies revealed distension of some or all of the GI tract with no mechanical obstruction. Histopathology revealed neuronal degeneration consistent with dysautonomia.

Other Reported Neurologic Diseases

While meningitis is an important neurologic disease and has been reported in camelids, it is almost always a sequela to neonatal sepsis. A diagnosis of meningitis significantly

worsens the already guarded prognosis of sepsis. The diagnosis and treatment of sepsis is discussed in Chapter 20.

There are also some important congenital neurologic lesions that have been reported which include hydrocephalus, meningocele or myelomeningocele associated with spina bifida, vertebral anomalies such as hemivertebrae, and complete agenesis of the spine and spinal cord. Congenital lesions are discussed in Chapter 21.

Other reported neurologic diseases in camelids include degenerative myeloencphalopathy in two llamas [59], phrenic nerve degeneration leading to diaphragmatic paralysis in a llama [60], intervertebral disk extrusion leading to tetraparesis in a llama [61], intracranial teratoma in an alpaca [62], discospondylitis in an alpaca and a llama [63, 64], cerebellar abiotrophy in an alpaca [65], cerebral injury from intracarotid injection of anesthetics in an alpaca [66], intracranial cyst in an alpaca cria [67], hepatic encephalopathy in three cases of hepatic lipidosis in llamas (see Chapter 13) [68], and pituitary adenoma in an alpaca [69].

Non-specific Therapy Plan for Neurologic Disease

Veterinary practitioners are often called on to make a "most likely" diagnosis and come up with a treatment plan based on limited information. In reviewing these common and uncommon neurologic diseases of camelids it becomes clear that many have similar clinical presentations as well as similar therapies. Below is a "shotgun" therapy plan that will cover most, if not all, of these diseases whether a definitive diagnosis is made or not.

1) Thiamine – As previously discussed, thiamine is the cornerstone for treatment of PEM, but including thiamine in the treatment plan for other diseases is recommended. Thiamine is well-documented as a neuroprotectant and the therapeutic index is very large. The dose is a 10–20 mg/kg SC or IM every six hours or the daily dose can be administered throughout the day if included in the IV fluids.
2) Anti-inflammatories – In almost all neurologic disease (polio, meningeal worm, listeria, otitis, trauma, etc.), anti-inflammatories are either recommended or at least not contraindicated. The most common anti-inflammatories used are flunixin meglumine at 1.1–2.2 mg/kg or dexamethasone at 0.05–0.1 mg/kg. Multiple days of anti-inflammatories may be required.
3) Antibiotics – In neurologic disease with a bacterial cause, including antibiotics in the therapeutic plan is important. The use of antibiotics is not contraindicated in any neurologic disease as long as the antibiotic choice isn't contraindicated for another reason, e.g. nephrotoxicity in a dehydrated patient. Antibiotics most commonly used to treat listeriosis are penicillin and oxytetracycline, while other broad-spectrum antibiotics such as ceftiofur are often used for other indications.
4) Anthelmintic drugs – Because meningeal worm is common and an important differential for neurologic diseases in camelids, including fenbendazole in the non-specific treatment plan is advised. The recommended dose is 50 mg/kg orally once daily for five days.
5) Supportive care, physical therapy, and rehabilitation – No matter how good the medical or surgical intervention is, without good supportive care and physical therapy, recovery from neurologic diseases is very unlikely.

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Neonatology

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South American Camelids (SACs) are natural mothers. It is a rare alpaca or llama that does not bond with their cria. "Cria" is the word the Spanish use for a baby SAC. Though it was probably originally a Quechua word, it is commonly what North American camelid owners use, and is the term used throughout this book to refer to young SACs. Camel neonates however, are termed "calves." While the following discussion is based on the SAC cria, the same general principles of care and conditions that may occur are the same in camel calves. Crias (birth to weaning) are very rewarding and enjoyable to work with, but the health of the neonate starts with a healthy dam and proper care before, during and after breeding and parturition.

Prepartum Care

A thorough physical and reproductive exam including a vaginal exam should be performed on the dam to determine health and the presence of any factors that would negatively impact the birth and health of the cria (see Chapter 16). As the gestation is long with a wide variability (330–360 days, average 340–346 [1]) it is best to booster any necessary vaccinations before breeding. The most common vaccine administered to camelids is *Clostridium perfringens* type C, D and Tetanus (CDT). Other vaccination requirements will vary depending on location and diseases in that area (i.e. West Nile, Rabies). In some herds, a CDT booster vaccination is given to pregnant females four to six weeks before expected parturition. This is routinely done in most livestock without problems. However, in some camelid herds, abortions have occurred after the CDT booster is given due to possible endotoxemia and lysis of the corpus luteum. It is possible that it is not the vaccine itself, but the stress of catching and restraining to administer the injection that is responsible for the abortion. A general recommendation is to avoid administering medications the first and last 60 days to pregnant females. This does not, however, pertain to use of an Avermectin in the prevention of Meningeal worm. Avermectins have been used for years in areas of the North America that have white tail deer without any known repercussions to pregnant females or their fetuses. It is also best to avoid other stressors such as transport, toenail trimming, or other routine husbandry practices during the last portion of the long pregnancy. Many owners question if it is safe to shear their camelids in late pregnancy. As heat stress is a major problem in most of the North America, it is recommended to shear rather than subject the pregnant female to the long-term stressor of heat and humidity that can induce heat stress (and possible premature birth). If an experienced person is doing the shearing, the female should be restrained for less than 5–7 minutes, which is minimal when compared to weeks of hot, humid weather (Figure 20.1). Many llama owners will shear only the blanket area (fiber over the back) and the top of the hind legs (Figure 20.2). This is usually done standing and can be done in stages if the female starts to show signs of fatigue with standing. As pregnant females are more prone to heat stress, provisions must be made ahead of time for shade and other ways to cool off (fans, sprinklers, misters, etc.). Depending on how many pregnant females are in a barn/area, multiple fans, etc. should be provided to avoid overcrowding. Females should be grouped by stage of gestation in order to be fed more

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Figure 20.1 Pregnant alpaca recently shorn. *Source:* Photo courtesy Lindsay Warne, The Alpacas of Spring Acres.

Figure 20.2 Llama with a barrel and hip cut. *Source:* Photo courtesy of Barb Baker, Baker & Company Llamas, VA.

specifically. It is best to breed females close together to allow for a shorter window the following year for monitoring. The most rapid growth occurs in the last trimester; consequently, there is less room for hay in the first Compartment (C1). Therefore, the maternity group needs to be fed better quality hay as it is hard for them to eat enough poor-quality hay. Be aware that older females (and some young ones) are prone to thinness when nursing a cria. Frequently, this is also when the owner is trying to get them pregnant again. If there is a specific maternity area, they should be moved approximately 60 days before the 330 "due date." The maternity group/barn needs to be monitored closely, ideally every hour when there are females near their expected date of parturition. If it is cold, they should be monitored every 15–20 minutes. It is advantageous to cluster breeding dates to make management of the expectant dams easier.

In South America, there can be high cria loss (20–80%). In North America, though the exact number is not known, the death loss is far lower, and farms should strive for less than 3–5% loss. Well-managed farms may have less than a 1% loss. Encourage owners to keep good records of breeding dates and to have ultrasound (US) confirmation of pregnancy done by 60 days of gestation. Owners become obsessed with their pregnant camelids, and when they do not give birth when they expect, immediately think something is wrong. Camelids are known to put the gestational maturation of the fetus on hold leading to prolonged pregnancy. Causes of prolonged gestation can be attributed to extreme winter conditions, nutritional deficiencies, abnormal placentation leading to intrauterine growth retardation, fetal abnormalities, and fescue toxicosis [2]. Due to this phenomenon and possible errors in record keeping, the female and the cria must be assessed carefully before intervention is considered (see Chapter 16).

Characteristics of the Camelid Neonate

The body weight of newborn alpaca crias varies from 3.8 to 11.3 kg (8–25 lb) [3], and llama crias vary from 8 to 20 kg (18–45 lb) [2, 4–7]. Birthweight is determined by genetic factors, age and size of the mother, degree of maturity, and nutrition of the dam during gestation [8]. The neonate is unlikely to gain weight during the first two to three days of life; in fact, it may lose up to 0.5 kg (1 lb) [9, 10]. Greater weight loss than this may indicate dehydration or lack of milk intake. After the first few days, weight gain should be about 125–250 g (0.25–0.5 lb) per day for the first two weeks for alpaca crias and 0.5 kg (1 lb) after that for llama crias. Weight of the cria at birth is also influenced by age of the dam. The heaviest crias (8–9 kg) are born from dams that are between 8 and 10 years old. Dams between two and seven years have crias weighing between 7 and 8 kg. As nutrient conditions change in the Andes, dams older than

Figure 20.3 Newborn with epidermal membrane.

Figure 20.4 Late-term aborted fetus showing epidermal membrane and umbilicus.

11–12 years old produce crias weighing less than 8 kg [11]. These weights may be increased in other countries wherein health and nutrition has been greatly improved.

The neonate is usually covered by a thin, semitransparent epidermal membrane attached at the mucocutaneous junctions, at the coronets of the nails, and the umbilicus (Figures 20.3 and 20.4). Its purpose is not conclusively known, but possibly aids delivery and keeps the cria from being saturated with uterine fluid and thus less likely to be chilled. Even though it is "shrink wrapped" to the cria, the cria is still born reasonably wet. If *in utero* stress of the cria has occurred there may be yellow to brownish coloring to the membrane and the crias' fiber. The eyelids are open, and most of the time the incisor teeth are erupted.

The body temperature of the recent newborn should be the same as that of the dam, 37.7–38.9 °C (100–102 °F) (Figure 20.5). The crias' rectal temperature drops to 35.5 °C

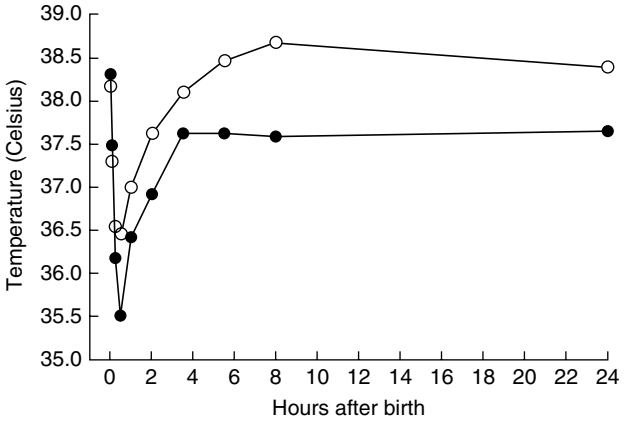

Figure 20.5 Rectal temperature in neonate alpacas for the first 24 hours following birth. Normal crias (open circles), hypothermic crias (closed circles).

(95.9 °F) during the first 30–45 minutes after birth, then rises to the homeothermic stage (37.5 °C, 99.5 °F) Once the cria has stabilized to the ambient environment, body temperature may fluctuate more in a cria than in an adult and normally may rise to 39.2 °C (102.5 °F). In South America, most alpacas and llamas give birth between 7 a.m. and 1 p.m. This allows the cria time to warm up, dry, move around and nurse before the coldness of the night. In altitudes greater than 4000 m, it is common to have freezing temperatures and snow [12]. In North America the camelids have kept to a similar schedule. If a cria is born late in the day, there is an increased chance of problems.

Immediate Care of the Newborn

Several sources discuss care of the newborn cria [13-16]. Camelid owners vary greatly in the care given to the neonate. Some allow the entire process to proceed naturally, while others wish to be present at each delivery, assisting in the parturition and lavishing care on the cria. Ideally, each birth should be attended, but this is not always feasible nor practical. The weather may have a significant bearing on whether or not special care must be given, but with the value of some camelids, it is logical to give maximum care in all cases. The following instructions are based on the owner/manager or veterinarian being present at the time of parturition. An evaluation of the cria should be made shortly after birth (Table 20.1) [3, 5, 7, 18–20].

At birth, place the cria on a clean towel, make certain that the cria is breathing and clear the airways and keep them in sternal (cush) position. This allows both sides of the lungs to inflate. A simple and gentle squeeze of the nostrils eliminates any fluids present in the distal nasal cavity and generally makes the cria sneeze and snort. Suction with a bulb

Table 20.1 Cria scoring system.

Score for each sign	Cardiac rate	Respiratory effort	Irritant response	Activity/Muscle tone
0	Undetectable	Undetectable	Unresponsive	Limp, lying on side
1	Less than 80 beats/min.	Irregular, noisy, open mouth, flared nostrils	Grimace, with mild rejection	Some tone, lying on side, but with effort can move to sternal
2	Over 80 beats/min.	Steady, nonlabored	Sneeze/cough, pulling away from irritant	Sternal or actively trying to get sternal

Note: Scoring[a]

0–3 Critical – Notify veterinarian of an emergency.

4–6 Marginal – Notify veterinarian if second score at 10 minutes has not improved.

7–8 Normal – Give usual supportive care.

[a] A total score is the accumulation of the signs observed in each of the four columns. Example: If a crias heartbeat was less than 80 (1 point), was limp and lying on side (0 points), breathing steady (2 points), and pulled away from an irritant (2 points), the total score would be 5 (marginal).

Source: Courtesy of L.B. Walker [17].

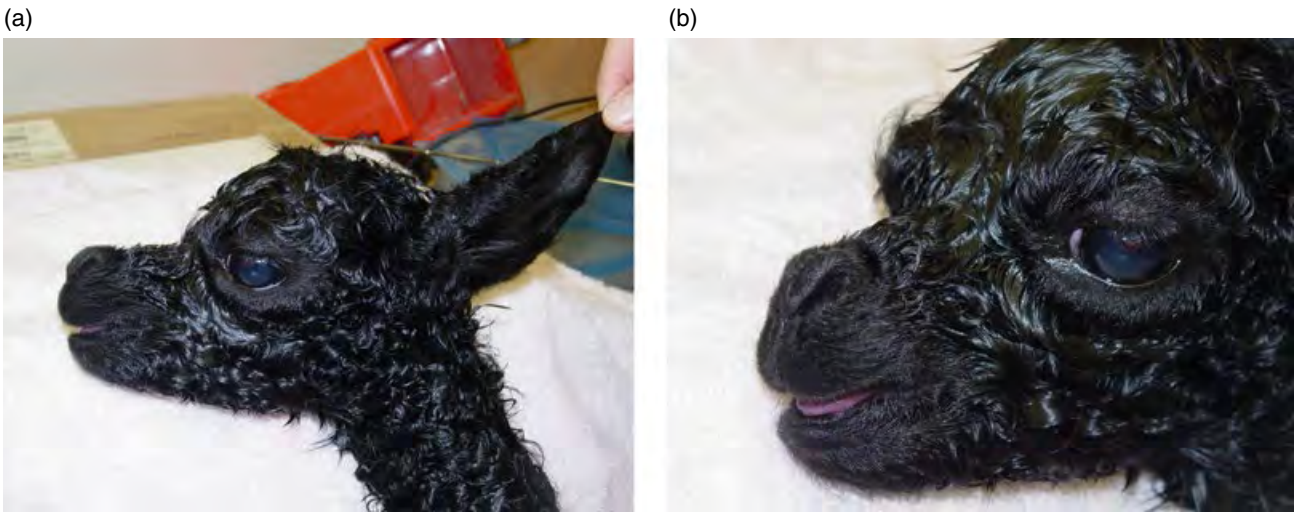

Figure 20.6 (a and b) Newborn crias with choanal atresia.

syringe or a human nasal aspirator can be used for removing excess fluids from the nostrils and mouth if needed. To assist with clearing the lungs of fluid, coupage (gentle tapping) with a cupped hand on both sides of the chest is effective. Traditionally, it has been suggested for neonates of livestock species, including crias, to be held upside down for a few seconds or gently swung in an arc to develop mild centrifugal action to clear the passageways. This is not necessary and can even be counterproductive as it forces the abdominal content to put pressure on the diaphragm preventing lung expansion. Also, the fluid is usually gastric in origin, not from the lungs. Most fetal fluid is absorbed from the lungs and is unable to be expelled successfully [2].

Once the cria is breathing well, the epidermal membranes can be removed from the cria by gentle rubbing with a towel. While drying the cria, evaluate if any abnormalities are present. Common congenital abnormalities that can be assessed readily include: atresia ani (no anus), lack/small vulvar opening, cleft palate, tongue abnormalities, abnormal limb development, choanal atresia (decreased or absent patency of nasal passages), and cataracts. (Figures 20.6–20.8, 20.46). Inserting a finger into the mouth to remove any collected mucus and check for a cleft palate may initiate a nursing reflex or stimulate the cria to move (Figure 20.9).

If the cria has still not begun to breathe, artificial respiration must be initiated. The most beneficial method is mouth-to-nose resuscitation since the camelid is an obligate nasal breather. Chest massage and compression are also useful. In a hospital situation, administration of humidified oxygen via a nasal tube or endotracheal intubation is appropriate. Also, examine the mucous membranes of the mouth and check for capillary refill time to ascertain the status of circulation. Cyanotic gums call for immediate oxygen administration and determination

Figure 20.7 Newborn with atresia ani. Note the lack of an anal opening.

Figure 20.9 Normal newborn, minutes after birth.

of its cause. If the cria is breathing loudly ("gurgly"), keep it in cush position and do coupage gently on both sides of chest. Stimulation by tickling the crias nostrils with a piece of straw or grass will also cause the cria to move its head and neck in avoidance. Over stimulation is counterproductive as the sensation will become refractory. Another method used to stimulate breathing is pressure just inside the nares with a finger on each side and pressing firmly.

Camelid dams do not lick their cria to dry it. Most may not nudge or otherwise stimulate the cria to rise, though some dams will put a foot on the cria as if to encourage it to stand. They do, however, perform bonding activities, and most dams nuzzle the cria and vocalize with a humming sound (Figure 20.10). Some camelid owners call this "kissing" and relate it to bonding between a human mother and baby (Figure 20.11). During this assessment of the cria, repeated dipping of the umbilical cord is appropriate (see care of umbilicus later in this Chapter). Though there are differences of opinion about when to weigh the cria, it is best to weigh it on the day of birth once it is fully dry. Frequently by this time, the cria has already urinated and passed its meconium (first feces) (Figure 20.12). Weight is

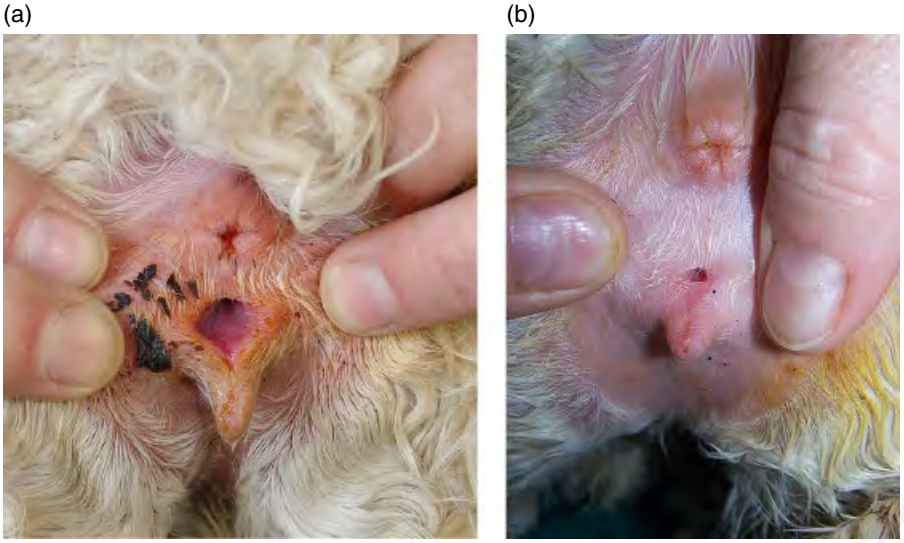

Figure 20.8 (a) Three-day old cria with a normal vulvar opening. (b) Newborn cria with hypoplastic vulvar opening.

(a)

(b)

Figure 20.10 (a and b) Dam bonding with cria.

Figure 20.11 Dam kissing cria. *Source:* Photo courtesy Becky Camma, The Alpacas of Spring Acres.

Figure 20.12 Normal meconium from a cria.

an important criterion to determine overall vigor and success of the crias' first day. Some crias are what are termed "closet nursers" and do not like to be observed nursing, so comparison of weight is important. As discussed above, it is not uncommon for crias to lose weight the first day or two, but generally gain it back by days three to four if not day two with a vigorous cria and a well-producing dam.

If, as a veterinarian, you are involved with a birth, examination should include taking the rectal temperature (encourage owners to do this also), assessing heart sounds to determine the presence of murmur, and recording the pulse and breathing pattern. The heart rate varies from 60 to 90 beats per minute and the respiratory rate from 10 to 30 breaths per minute. Camelids are obligate nasal breathers, so any upper respiratory obstruction causes dyspnea. Listen to the trachea for indications of the presence of fluids so corrective steps may be taken to expel excess fluids. Some normal crackles can be heard but should disappear quickly. The shape and direction of the ears should be assessed as well as the presence of an ear canal. Lack of ear canals have been attributed to some sires in both Suri and Huacaya, though less in recent years as the defect has been recognized and males taken out of the breeding pool. Some crias may have the pinna bent and even completely folded. Straightening it requires a bandage (use paper tape) wrapping the ear around a gauze or tampon for simple folds. More severe cases may require the use of a 6- or 10-mL syringe case or similar for support for two to three days. If only the tip the ear is bent a simple bandage (use paper tape) should correct it (Figure 20.13a and b). This is not a serious defect, rather positioning in the uterus. The presence of any birth defect as described above should be noted. See Camelid Birthing Record form (Table 20.2).

Table 20.2 Camelid birthing record.

Date: _____ **Staff:** _____

Dam: _____ **Sire:** _____

Owner: _____ **Cria Name:** _____

Time of birth: _____

Cria Weight: _____

Male: _____ Female: _____

Suri: _____ Huacaya: _____

Llama: _____ Color: _____

Teats: 4 _____ DNA/FTA card: _____

Placenta weight _____ lbs

Physical Findings (Date): _____

Temperature: _____

Heart Rate: _____

Respiratory Rate: _____

Lungs: Clear/Congested _____

GI Tract: _____

Vulva/Testicles: _____

Legs: Straight/Valgus: _____

Umbilicus/Hernia: Y/N _____

Tail Normal: Y/N _____

Cleft Palate: Y/N _____

Ears Normal: Y/N _____

IgG: (Date) _____ Level: _____ mg/dL

BVDV: (Test Date) _____ Results: _____

CBC: _____ PCV: _____ TP: _____

Treatments & Medications for Cria

	Date	Initials	
Iodine to navel	_____	_____	
Rota-Coronavirus	_____	_____	MUST BE GIVEN IMMEDIATELY
E-coli (5cc orally)	_____	_____	
Enema	_____	_____	Meconium passed: Y / N
C D & T (2cc SQ)	_____	_____	
Vit A & D	_____	_____	
Bo – Se (0.5 mL/20 lbs)	_____	_____	

Observations: **Due Date:** _____

_____

_____

A normal cria should soon begin to struggle shortly after birth, attempting to get up. Its first milestone is when it flops over lateral, and it rights itself into cush position. A normal cria is able to stand, albeit unsteadily, within an hour, and will immediately attempt to make contact with the mother. Directional instincts may be poor initially, but many crias will soon locate the udder and begin nursing (Figure 20.14).

Little has been written on llama and alpaca nursing behavior [21]. In a study of a few animals, first nursing occurred at about one hour [10]. The cria must learn to semi-crouch and extend the head upward with a "U" bend to its neck to reach the udder and assume the correct posture for suckling (Figure 20.15). The suckling posture is described as reverse parallel, with the cria standing at the side of the mother, its tail directed toward the mother's head. Generally, the mother will reach down to smell the crias perineum to make sure it is hers (Figure 20.16). In most crias that are latched and nursing well, they will curl their tail over their backs. Nursing may be stimulated in a standing cria by tickling its tail. Crias are very oral and will try to nurse on anything that stands still and frequently start their nursing career by trying the flanks, chest or fiber on the legs of the dam. Also, crias naturally seek a dark spot to find the udder and can be found trying to get under water buckets hung on a wall or a feeder. All breeders should preselect/plan maternity stalls with this in mind and have them well lit, even overnight. Be aware of "wall suckers." Some crias will keep going to the wall repeatedly to try to nurse. As soon as this behavior is observed, intervention is necessary to break this trend. Generally, a few nursing lessons will have this eager cria nursing its dam. Nursing lessons can be as simple as pointing the cria in the correct direction. Sometimes, the lesson is more complex and involves supporting the cria's weight while both hands are used to purse its lips around the teat, preferably while squirting milk in its mouth (Figure 20.17). Experienced dams will stand with their legs out behind to allow the cria better access to the udder (Figure 20.18). Of primary importance is for the cria to suckle the colostrum [5, 13, 22–24]. As previously stated, it requires an hour or two for the cria to be able to stand and search for the udder. Some crias are very vigorous and will be standing by five minutes and attempt nursing by one hour. In one study,

Figure 20.14 Cria attempting to nurse under chest. Note flipped up tail of cria.

(a)

(b)

Figure 20.13 (a) Simple bent ears in an alpaca cria at birth. (b) A simple bandage used to stent the ears has been placed to correct the deformity.

Figure 20.15 Cria in proper nursing position with "U" bend in neck.

Figure 20.17 Cria properly latching onto teats to nurse with some assistance. *Source:* Photo courtesy Becky Camma, The Alpacas of Spring Acres.

Figure 20.16 Dam smelling perineum of cria to confirm it is her.

Figure 20.18 Dam moving rear legs out behind to allow better access to nursing for cria.

approximately 7.5% of alpaca crias achieved suckling on the first attempt. At the second attempt, 33.4% were successful; 53% required three efforts [23]. Suckling occurred every half hour for the first four hours after birth, and then reduced to every hour; suckling lasted from 5 seconds to 4 minutes, with an average of 30 seconds. To maximize colostrum intake in a normal cria, it is recommended to get the cria up hourly during the first day of life. The goal before leaving the cria for the night is to have them spontaneously get up on their own and nurse their dam. Newborn crias spend about 5% of their time nursing, dropping to

about 3.5% by one month of age. With nursing episodes about 10 times per day for the first seven days, after which periods decreased to about eight times per day [22, 25, 26]. Though it is frequently stated that crias do not nurse at night, they do. They take advantage of when their dam gets up to participate in normal activities within the group (Becky Camma, The Alpacas of Spring Acres, Zanesville, OH, personal communication). Frequency of nursing varies with the age of the cria. During the first 10 days of life, it will nurse two to three times per hour during the daylight. Up to one month of age, nursing frequency may be twice per hour (Figure 20.19). Camelid crias begin nibbling at hay or grass at 10–14 days. As the intake of solid food increases, nursing episodes and time spent nursing decrease [19, 27]. Camel calves should be given free access to their dams to nurse colostrum and this is a common practice in a modern domestic setting. If raised in a more traditional, pastoral setting this free access may not occur with only limited colostrum ingested leading to possible sepsis and death. Depending on access to its dam, a camel calf may not feed on grass until two to three months of age. It is common to leave a calf on its dam until it is one year old. (Figure 20.20).

Meconium

Meconium is usually passed within 24 hours of birth or sooner. Colostrum is a natural laxative and successful nursing and normal activity should aid in passing the meconium unassisted. If the cria does not pass the meconium and is found straining but not passing feces (digital exam), then an enema should be given. Various products can be

Figure 20.19 (a) Newborn alpaca cria. *Source:* Photo courtesy of Carmon Koenigsknect. (b) Newborn alpaca cria indicating hunger with tail up. *Source:* Photo courtesy of Amber Kohl. (c) Typical position for cria to lay.

Figure 20.20 Camel calf nursing.

used including warm soapy water or Pediatric Fleet® enemas. The Pediatric enema works well and quickly, however it is an irritant-type enema, so should only be used twice. Proper restraint of the cria is required and insertion must be done with care as the rectal tissue is very thin and easily pierced. A report of inadvertent transvaginal delivery of a Fleet enema resulted in profound abnormalities and death in one of the two crias involved [28]. The tip of the commercial enema is designed for humans and is not recommended to be used. Using a 10 mL syringe with a plastic teat infusion tip (lubricated) is sufficient to allow easy insertion and proper placement of the enema solution (8–10 mL). Generally, the cria squirts out the enema solution followed shortly by the meconium (Figures 20.12 and 20.21). If the cria does not pass its meconium within one to two hours, one more enema can be given. If an enema is unsuccessful, orogastric tubing with electrolytes (60–120 mL) and mineral oil (5–10 mL) can also aid in passing the impaction. As a note, if enemas are given to newborn crias soon after birth and the cria has a very short or no umbilical stump, it is not uncommon for the stump to bleed a little. Usually, dipping the umbilicus again several times will normally stop the bleeding. On occasion, it can be tied off for a few hours. Wait until the end of day and monitor for the cria to pass the meconium on its own. Then, if it is determined to be necessary, give the enema. Some owners believe the cria nurses better after they pass the meconium (Linda Bader, Shady Hollow Suri Alpacas, Bad Axe, MI, personal communication).

All of the initial care procedures can be carried out in the presence of the dam as the majority tolerate human presence at this time. Only a few dams will become difficult and rarely are they aggressive, especially if eye contact is avoided.

Figure 20.21 Normal meconium from a cria.

Once initial care has been completed, it is best to leave the mother and cria undisturbed. Ideally, it is best to have them birth out in a clean pasture or in a clean area of the barn in inclement weather. Most females do not like to be isolated away from the group, even during labor. In fact, she will stop labor if she can until reunited with the group. If possible, in good weather it is nice to leave a stable pair out in the pasture for a short time as a good deal of socialization occurs, with other females gathering to nose the newborn.

In North America, it is common shortly after the cria is born, to place the dam and cria into a maternity stall with clean straw or small area by themselves so bonding can occur. This minimizes distractions for both mother and cria.

If the mother is disinterested (usually maidens), this isolation is critical for bonding to occur. Most females tolerate this well, but some nervous types will not stand for the cria to nurse in this isolation. Each case needs to be assessed individually but, on occasion, the pair may have to be put out with the group if the weather permits. These crias need to be monitored discretely to confirm nursing is taking place.

Though human interference should be limited to essentials, the cria should be kept under constant observation (discretely) to note when it can stand and when it begins nursing and for how long. A cria that has not nursed by three hours following birth requires intervention. The position of the head is a good indicator of a crias wellbeing. A head-up posture with attentive ears indicates that the cria is healthy. The parturient dam vocalizes with a guttural sound like clucking to communicate with her cria. Sometimes, this sound is a humming. The dam is always close to the neonate and when the cria is suckling, the dam may turn her head and sniff the perineum of the cria (Figure 20.16). Some dams make an episodic cluster of hummings when the crias are in their proximity and especially when they contact the head of the cria.

Care of the Umbilical Cord

If the female delivers while standing, the umbilical cord usually ruptures a few inches from the body wall during the descent to the ground. If the dam is recumbent during parturition, the umbilicus ruptures as soon as the cria begins to struggle or the dam stands. If the cord does not rupture, it should be squeezed and cut off 15 cm (6 in.) from the body of the cria. It is not necessary nor desirable to ligate (tie a string or suture around) the umbilical cord unless a pulsing stream of blood continues to flow. Even then, it is better to apply temporary pressure to the cord. A ligature may prevent normal drainage from the cord, and the fluid is an excellent medium for the growth of bacteria. If the cord is covered with dirt or other debris, it should be gently cleaned and disinfected on the outside only. If the cord is long, then it can be trimmed (after cleaning) and this fresh surface dipped. If hemorrhage is ongoing the umbilical cord can be ligated with umbilical tape, suture, or dental floss.

Several disinfectants to use for umbilical dipping or spraying are available and are generally chosen based on personal preference. The most common products used are 7% (or 2%) tincture of iodine, 7% (or 2%) povidone iodine or Chlorhexidine 0.5% solution. A study conducted on numerous foals compared the bacterial counts obtained from the umbilical cord before and following treatment with commonly recommended solutions (7% tincture of iodine, 2% tincture of iodine, tamed iodine [betadine, povidone-iodine], and chlorhexidine [Nolvasan®]).

Nolvasan (chlorhexidine) was the only solution that was shown to be both safe and effective in reducing the bacterial count. Though it is blue, it does not stain the fiber or fingers. Perhaps a psychologic drawback to its use is that the cord remains soft and pinkish, giving the appearance that nothing has been done. Nonetheless, following the use of Nolvasan, the umbilical cord dried and healed within an appropriate timeframe, and most importantly, umbilical infection prevalence was reduced. A one-time treatment with Nolvasan provided residual disinfection that none of the other solutions provided. It is recommended that Nolvasan be diluted to a 1:3 dilution to produce a 0.5% solution (one-part Nolvasan to three parts water). The diluted Nolvasan should be kept in a dark bottle, and any unused solution should be discarded after two weeks. With this information, it would be reasonable for veterinarians to switch from an Iodine-based product to Chlorhexidine. Iodine-based products have the advantage that it is visually evident the umbilicus has been treated.

Care Given to the Dam

As soon as a neonate is stabilized, the dam should be examined to determine that no serious lacerations of the vulva have occurred and to determine the status of the udder. It is not uncommon to have a few breaks and stretches in the skin of the vulva with a normal delivery. These will heal up without complications. On rare occasions, the female may have a tear into the anus, that might have been avoided if there was assistance available with the delivery. These females will need to be treated like a mare in similar situations. In most situations using lube, patience and gentle stretching of the tissue of the vulva around the head, the cria can be delivered without an episiotomy. On rare occasions, usually with maidens, an episiotomy may be needed to facilitate passage of the head from the vulva. Due the perineal body being so small in camelids, the positions of three and nine o'clock are used rather than the 12 o'clock position in mares. To avoid major vessels in the area, it is best to wait until the vulvar tissue is stretched around the head and only make the incisions long enough to deliver the head. Once the cria is out and stable, suture up the incisions with absorbable suture. If there was extensive obstetric manipulation, the female should be carefully assessed for development of vaginal adhesions in the next seven to 10 days. Camelids are prone to vaginal adhesions and if the adhesions become fibrous, her reproductive life is over.

The udder should be checked for development of glandular tissue and any excessive swelling (edema), heat or pain.

Check the udder soon after delivery as there is a short period of time after the birth when the dam is in a bit of a haze and will not mind too much. Check for the presence of colostrum as well as any indication of mastitis. The teats should be squeezed lightly to remove wax from the teat ends and the streak canals (Figure 20.22). This makes it easier for the cria to nurse for the first time, and it allows an assessment of the quality and quantity of colostrum. Good-quality colostrum should be sticky like glue and have a creamy color. Avoid vigorous and persistent stripping of the teats as camelids do not generally make an abundant amount of colostrum. Over aggressive stripping robs the cria of vital colostrum. Whatever colostrum you strip out of the teats, smear on the cria's lips and mouth to provide a taste of what is to come. Most crias lick their lips in response (Figure 20.23).

Agalactia is a common postpartum disorder and may be temporary or permanent. The etiology of agalactia may be as simple as failure of milk letdown because of lack of oxytocin stimulation, or it may be the result of lack of development of the glandular tissue or lack of patency of one or more teats. Other causes of agalactia or dysgalactia in camelids include hyperthermia, toxemia, mastitis, pain (lameness, arthritis), digestive upsets, ergot ingestion, stress, and genetic factors. If parturition is premature, glandular tissue lacks maturity. The degree of immaturity depends on the stage of gestation. Mastitis is more likely to occur in a female having had more than one cria but it may also occur in a first-time mother.

Simple failure of milk letdown may be treated by stimulation of the udder with a warm wash cloth and massage, working with the cria to nurse or by intramuscular (IM) injection of oxytocin (5–10 USP Units), which may be repeated at three- to four-hour intervals if necessary. Oxytocin, or any other drug, does not increase milk production in a nonfunctional gland.

Pain caused by simple engorgement with milk, mastitis, and udder edema may cause the female to move away, preventing the cria from nursing. Administering Banamine® (Flunixin Meglumine) at 1.1 mg/kg (1 mL/100 lb, 45 kg), subcutaneously will help with the pain of these conditions and the dam may allow the cria to nurse. In the case of engorgement of the teats (seen more frequently in older, multiparous dams) milking out the quarters to reduce the amount of colostrum will reduce teat size so the cria is able to latch on. Be careful to save that colostrum to give to the cria via bottle. Sometimes, a female may be a poor mother and refuse to stand still for the cria to nurse. Llamas can be put in a chute to work with, and some alpacas just cush when you start to work with their udder. If the dam cushes, put her lateral (easier said than done) recumbency and with proper restraint, work with the cria to nurse. It is not ideal to do this, and it is rarely needed. These difficult females can be sedated to encourage acceptance of the cria. Frequently, once a cria successfully nurses, the dam will be more willing the next time a cria attempts to nurse, especially if the cria is persistent.

Figure 20.22 Wax on teats.

Figure 20.23 Newborn after colostrum put on lips. *Source:* Photo courtesy Lindsay Warne, The Alpacas of Spring Acres.

Mastitis is rare in llamas and alpacas [29, 30]. A study evaluating 100 lactating llamas revealed that 21% (76 of 369) of samples had indications of infection with 57% having positive bacterial culture in at least one quarter. None of the animals exhibited clinical mastitis and minimal evidence of inflammation was present [30]. In that study, the most common minor pathogens isolated was *Staphylococcus* spp. with major pathogens as *Streptococcus uberis*, *Streptococcus equinus* and *Enterococcus* spp. The usual tests run on dairy cows for detection of subclinical mastitis such as the California

mastitis test (CMT) and the Somatic Cell Count (SCC) were demonstrated not to be effective on llama milk. This was theorized to be due to the lack of significant glandular inflammation and lack of high cell counts when compared to the bovine [29, 30]. Mastitis can happen and may involve just one gland or multiple. A report of five cases were confirmed by bacterial culture. *Escherichia coli* in three cases and *Staphylococcus aureus* in two cases. Manual palpation of the udder revealed the presence of hard tissue within the affected quarters. In cases such as these, the affected quarter may be warmer when compared to the non-affected quarters. There also may be enlargement of mastitic quarters and usually abnormal secretions [31]. The best approach to treatment is use non-steroidal anti-inflammatory drugs (NSAIDs) and broad-spectrum antibiotics such as Ceftiofur. It is not recommended to instill intramammary preparations as the teat anatomy is not straightforward as in other species (see teat anatomy in Integumentary System Chapter 9). On occasion, a dam will come fresh with a blind gland that was normal the previous lactation. This would normally indicate that some sort of disruption/disease took place in that quarter of the mammary gland. Palpation of the gland may reveal a hard mass(es) present. Yet, this female never showed any problem and her cria grew well in the previous lactation. As stated above, it is common to have minimal signs of inflammation even with bacteria present. When examining a cria for lack of weight gain, always remember that there are two parts to this equation and to examine the dam also, paying close attention to her udder. Most females do not like to have their udder palpated but, with patience, it can and should be examined.

Edema of the udder after giving birth is also present in some animals, especially maidens. The edema is like what is seen in heifers after freshening and is painful. The edema may be severe enough to involve the teats making them appear even smaller than normal and even harder to latch on to for the cria. On occasion, there will even be edema proximal to the udder. Edema will usually go away as the dam comes into milk and the blood flow for the udder becomes fully established. In the meantime, gentle massage with udder balm twice a day for several days has been effective as well as manually evacuating the glands and feeding the colostrum to the cria. These teats are especially hard to milk and generally only a thumb and forefinger can be used. Teats like this chap easily, so keep that in mind when milking and keep some milk on your fingers to moisten the teats.

Treatment of agalactia may also involve the use of Domperidone (Equidone®) orally twice a day for three to five days. As an antidopaminergic, Domperidone stimulates the secretion of prolactin, the hormone of milk production. The recommended dose is 330 mg, twice daily for alpacas and 550 mg, twice daily for llamas. This is a larger dose than in mares. Also, as is it a prokinetic drug, the dam should be monitored for signs of colic and diarrhea [18]. Unlike the mare, this drug should not be given before parturition. Domperidone should be used cautiously, because sometimes dams produce enough milk, but in the absence of a nursing cria, letdown of milk is not present. There is an herbal mix (Dr. Pollard's lactation stimulator) that some producers have used with mixed results. It is intended to be started about two weeks before the expected due date.

Another approach to agalactia is a constant stimulation of milk production. This can be done by a gentle massage of the udder and milking up to four times on a daily basis. Milking of the llama and alpaca udder requires patience. Only two fingers are used, and the production of milk yields may be as much as 120 mL. Repetitive milking is advised to continue milk production and when the cria is ready there should be no problems in milk production and milk letdown.

A major concern with agalactia is whether the female lacks the genetic potential to produce enough milk to raise a cria. It is hard to determine in maidens, as some just need more time. It is recommended that if a female is a poor producer for two lactations, she should be removed from the breeding program. We know in other species that lactation is a strongly heritable trait and there is no reason to doubt this is true for camelids.

Once the cria and dam are bonded, and nursing is well established, other procedures that were not performed during the last two months of pregnancy to avoid stress can be done. This will include trimming toenails, trimming incisors and shearing if the decision was made to delay. This is also an ideal time to perform a fecal floatation using concentrated sugar and centrifugation method. These females have been carrying around a "parasite" for almost one year and their immune system has been depressed. Rather than administering anthelmintics, possibly encouraging the formation of resistant parasites, a proper fecal exam will demonstrate if any drugs are needed. Not all females will need to be dewormed after a long pregnancy.

Prematurity

Intensive care of the premature infant has become commonplace in humans, horses, cattle, and companion animals. Numerous observations on premature crias have been made to establish certain factors, which, combined with extrapolation from known care for other premature ungulates, may save the life of a premature cria. Prematurity may be determined on the basis of birthweight, length of gestation, observable characteristics of the neonate, and laboratory analysis of blood. There is considerable variation in the length of gestation of llamas and alpacas

(330–360 days, with an occasional pregnancy continuing for more than one year). Further, an accurate breeding date is sometimes difficult to establish. When pasture breeding is practiced, unobserved early embryonic death may be followed by breeding within a few days. Such variation makes it difficult to establish definitive prematurity on the basis of the age of the fetus. The degree of maturity should also not be based entirely on the number of days in the uterus. Crias born after an excessively long gestation can show signs of prematurity and be termed dymature [32].

Signs of Prematurity

Unusually low birthweight is the most evident characteristic of lack of maturity. Generally, if a llama cria weighs less than 8.2 kg (18 lb), there should be concern; if it weighs less than 6.8 kg (15 lb), some degree of prematurity should be assumed. Alpaca crias should weigh at least 4 kg (8.8 lb). This minimal weight for alpacas was done in Peru and even stated that crias born at 9 kg (19.8 lb) had a greater chance of survival [11]. In developed countries, it is more typical for alpaca crias to weigh 7.3–8.2 kg (16–18 lb). However, an absolute weight cannot be considered as the sole determinant. If a crias birthweight is approximately 20% lower than the average cria on the farm, it should be considered at risk [32]. For instance, if the expected delivery weight of a mature fetus is 13.2 kg (30 lb), a cria delivered at 11 kg (25 lb) may be immature.

Premature crias are weak and may be unable to stand or hold the head up to nurse (Figure 20.24). Overextension of the fetlocks, from tendon and ligament immaturity, causes the cria to walk on the fetlocks (Figure 20.25a and b). The sucking reflex may be weak or entirely absent.

Premature crias have floppy ears, a result of immaturity of the aural cartilage (Figure 20.26). However, not all tipped or floppy ears are caused by prematurity; genetic factors may also be involved or *in utero* positioning (Figure 20.27). An immature fiber coat has a silky feel, especially over the back and rear quarters. The eyelids may be stuck together in crias that are one month or more premature.

The rubbery covering (eponychium) over the ungulate hoof is also present on the camelid fetal nail (Figure 20.28).

Figure 20.24 Depressed premature newborn alpaca cria.

(a)

(b)

Figure 20.25 (a) Relaxed tendons and muscles in this newborn cria is causing this alpaca to have a plantigrade stance. (b) Angular limb deformity and plantigrade stance in newborn cria. Normal movement and weightbearing usually will help to resolve these deformities.

Figure 20.26 Floppy "dog ears" of a premature cria.

In a mature cria, the rubbery mass is removed from the toe within 6–12 hours as the cria struggles to stand and takes its first steps. The covering persists for one to two days in premature crias.

The epidermal membrane is thin, friable, and easily rubbed off a full-term fetus within a few hours after birth. In the premature cria, the membrane is thicker, and remnants may cling to the lips, etc. for 24 hours.

The incisor teeth have erupted in the normal, full-term SAC fetus (Figure 20.29a). The incisors erupt in the last two to three weeks of gestation and failure of these teeth to be erupted at birth is a primary indication of prematurity (Figure 20.29b and c).

Occasionally, a sick dam, may release enough steroids in her system to help mature the crias lung and the cria will have a chance to live if born prematurely. Septic crias may be stressed enough to cause birthing to start. Umbilical cord torsions, depending on when it happens in gestation, may cause a catecholamine surge with elevated concentrations of cortisol and the cria may be born alive.

Premature crias sleep most of the time. They even adopt a lateral position and it may be thought that the cria is dead. Transition to life outside the uterus is an obstacle most crias can overcome in a normal progression. It is especially challenging to premature crias who transition more slowly. At birth, there is a normal catecholamine surge and elevated concentrations of cortisol, both of which help the cria to adapt to extrauterine life [2]. This

Figure 20.27 Flipped out ears in a normal newborn cria. *Source:* Photo courtesy Kathy Turk, Gaelic Glen Alpacas.

Figure 20.28 Eponychium (rubbery covering) on the toenail of a premature cria.

elevation in cortisol will have the newborn look bright for the first few hours after birth, but as this decreases, the cria will start to show the true state of its compromise [2]. This may also be true in cases of dystocia in which assistance is needed to deliver the cria. Due to this period of "normalcy," most producers will have a false sense of

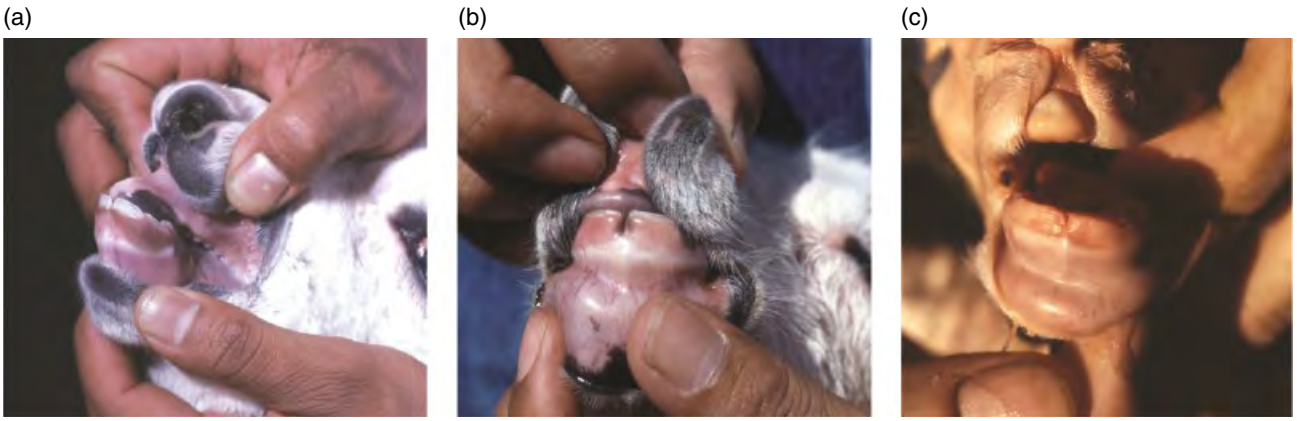

Figure 20.29 (a) Dental eruption at the time of birth - normal. (b) Premature cria, incisors covered with membrane. (c) Membrane over all the premature incisors at birth.

security and may not contact their veterinarian after what is suspected to be a premature cria. As the pulmonary system is the last to mature in the fetus, respiratory distress is a common sign in premature crias necessitating immediate intervention. Most neonates are born with enough surfactant to help them breath for 12–24 hours, then the Type II pneumocytes will produce more. This is probably true for normal crias as well. With premature crias, the Type II pneumocytes are not mature enough and the cria will be unable to produce more surfactant to help with oxygen transfer across the alveolar membrane. This again will have the cria seem "normal" in the first hours after birth and contribute to delay in seeking assistance from their veterinarian. Keeping the cria sternal will allow both lungs to inflate, supplemental oxygen by mask (or nasal cannula for longer term, preferably humidified) if the cria is not pink (if dark pigment skin, check vulva or prepuce for pink) may be needed if cria is showing signs of respiratory distress (open mouth breathing) or cyanosis. Bradypnea (less than 10/minutes) is common in the premature cria because they sleep so much. There is an absolute hypercapnia, but this fails to initiate respiratory effort, and PaO_2 is low. Because crias have such poor muscle tone, physical therapy in the form of passive range of motion is beneficial to help with muscle development. It has the added benefit to get the cria moving and help blow off the CO_2. This physical therapy (done several times a day) is best done in lateral recumbency, but once done, return the cria to sternal recumbency. To help with bronchodilation, diaphragmatic effort and possible surfactant formation give Aminophylline at 2 mg/kg (0.9 mg/lb), subcutaneously. Be careful because the toxic dose is close to therapeutic dose. Aminophylline should be given for three days at decreasing frequency [33].

Day 1 – every four hours
Day 2 – every six hours
Day 3 – every eight hours

Surfactant is another option, though expensive and not a routine item in most veterinary clinics. It has been demonstrated in a unique set of circumstances to be effective [34]. In crias with severely depressed respiratory rates, Doxapram can be administered at 5 mg in alpaca and llama crias and 50 mg in camel calves sublingual or IV. If there is minimal response, an additional dose can be administered, but monitor for convulsions or hyperventilation [32].

Frequently, premature crias that do not have a suckle reflex must be fed by orogastric intubation. This is safest done once they are strong enough to hold their head up. The tube should be passed slowly through the pharynx to make sure that it is in the esophagus. The best approach to confirm tube placement is to palpate the tube in the cervical region. As newborn crias do not yet have much muscle mass in the neck, the tube is easily felt, especially if it is moved up and down a short distance. There are other methods, but each has its drawbacks, this is the most certain of correct placement. There are a variety of tubes that can be used, Red rubber feeding tubes (18–24 Fr, 40 cm [16 in.] long) work well (Figure 20.30). Due to the long neck, it does not go into the first Compartment, possibly allowing some closure of the gastric groove and fluid to go to the third Compartment. Barium studies have shown that after orogastric intubation, the fluid rapidly entered all compartments and even into the proximal part of the small intestine [35]. Volume of colostrum in premature crias must be small as to not overload. Begin with 30–60 mL as a starting point and monitor how the cria does. Some cannot stand but want to nurse; if the dam is cooperative, hold the cria to udder; if the dam is uncooperative, offer them a bottle of colostrum using a Pritchard flutter valve nipple (red tip with yellow base) (Figure 20.31). The intestinal mucosa

Figure 20.30 Red rubber feeding tube. Sizes 18–24 Fr are suitable for orogastric intubation in crias.

Figure 20.31 Pritchard flutter valve nipple.

of the premature cria is refractory to absorption of immunoglobulin (Ig) macromolecules [13]. (see Immunoglobulins in this Chapter).

Crias are born the same temperature of the dam and cool off in the immediate post-partum period. How fast this happens is a function of the environmental temperature. The normal cria starts to generate heat within 15 minutes to 3 hours after birth while the fiber dries [12]. The premature cria has poor thermoregulatory ability and is thus at great risk of hypothermia. The body temperature of a cria suspected to be premature must be carefully monitored. When newborn crias are found outside in winter and found "frozen" but alive, it is important to warm them up slowly. It is not recommended to immerse them (in a trash bag) in hot water to warm them, they will inevitably become wet and remain chilled. Warming them up gradually is important so as not to shock them further. Sometimes they are only alive because they are so chilled. As they warm up, the body releases toxins built up in the tissues. If this happens too quickly, it will cause them to go further into shock. Use warmed towels (put in the dryer) and vigorous rubbing, blow dryer, heating pad (closely monitored), hot water bottles, etc. Move their legs (passive range of motion) to help get the muscles warm every 15–20 minutes. Then return to their towel "cocoon." Important to keep them on their chest (except for physical therapy as above) so both lungs inflate; coupage (lightly tapping on their sides with a cupped hand) if needed. Give them 3 mL at a time oral 50% Dextrose until warm enough to take colostrum ~36 °C (97 °F). It is important to either bring the dam inside with the cria while warming up, or to put the dam in an area where she cannot see other crias. She can become confused and "adopt" another cria as hers when her cria is out of sight. This happens quickly and she will refuse her cria after it has been warmed and returned. It takes most crias about three hours to warm up. During this time, they should have increasing ability to hold their head up and regain a suckle reflex. Once they start to stand, they warm up quickly.

Full-term crias may be hypoglycemic at birth and for a few hours thereafter; premature crias may remain hypoglycemic and lack the energy to stand or nurse [18]. Producers frequently want to give Karo syrup to weak crias. Though unknown in crias, calves lack sucrase to absorb sugar as sucrose. Also, too much Kayro syrup will give them diarrhea. As this may be possible in crias, a better source of sugar would be Dextrose which is biochemically the same as glucose and will have good absorption [36]. Leukopenia, caused by an absolute neutropenia, is characteristic of prematurity. The immature adrenal cortex is unable to respond to adrenocorticotrophic hormone stimulation to produce cortisol and deal with stress. Newborn crias have a higher packed cell volume that should normally drop some with absorption of colostrum and IgG [2]. Investigation for possible *Mycoplasma haemolamae* should be considered if the cria is anemic, with hypoglycemia. Though it is not predictable, there can be transplacental transmission of *M. haemolamae* to the cria with a positive dam [37].

Elevation of plasma fibrinogen in neonatal crias (<24 hours old) is regarded to be an indication of a bacterial infection developed *in utero* [2].

Premature foals have incomplete formation of the carpal and tarsal bones, predisposing to angular limb deformities. This phenomenon has not been investigated in premature camelids, but it warrants consideration, because angular limb deformities are common in llamas and alpacas.

It is important to recognize that the fully mature cria may exhibit one or more of the characteristics of immaturity. The overall morphology and activity of the cria must be evaluated on an individual basis. Intrauterine septicemia may simulate many of the signs of prematurity, as may congenital heart defects. A neonatal maladjustment syndrome has been reported in foals wherein the foal fails to adapt to extrauterine life, even though fully mature. It is possible that there is a similar syndrome in camelids.

Prematurity is life threatening. Prompt diagnosis and intensive care is mandatory [8, 18]. The veterinarian should work closely with clients to educate them on early recognition so that prompt, effective remedial action may be taken.

Dysmaturity is a term used for crias that have a complete time of gestation but nonetheless are still weak. They may lack a suckling response and the administration of colostrum is imperative to avoid a failure of passive transfer. These crias may become normal after three to four days and join other crias in socializing.

Immunoglobulins

Camelids have an epitheliochorial microcotyledonary placenta that does not allow transfer of immunoglobulins from the dam to the fetus. For this reason, crias are born hypogammaglobulinemic and depend on passive transfer of immunity through colostral intake [7]. Many reports confirm the importance of timely and appropriate intake of colostrum to prevent or lower death due to failure of passive transfer [13, 25].

Production of Immunoglobulins

Research has shown that when evaluating the concentration of immunoglobulin G (IgG) in mammary secretions of prepartum dams, it is almost 10 times higher than the concentration of IgG in serum [23]. Further study revealed that the IgG concentration in the serum remained unchanged in the pregnant female and it was theorized that transfer of IgG in camelids did not occur from transfer of IgG from the blood to the mammary gland just prior to parturition [23]. It was further theorized that, based on this evidence, there is accumulation and production of IgG in the mammary gland of pregnant camelids. The concentration of IgG decreased in mammary secretions for five days after parturition [23]. This appears to be unique and different than the bovine's mechanism for increasing IgG in colostrum. In the cow, preparation of the mammary gland for colostrum production begins about two to three weeks prepartum [38]. Special receptors on the epithelial cells of the mammary gland selectively bind serum IgG, which is, in turn, taken into the cell and ultimately transported to the lumen and hence the colostrum. Many factors can interfere with production, quality and quantity, of colostrum. As camelids have not been selected for milk production, there are genetic factors such as individual and breed differences. If a previous cria is allowed to nurse after supposedly being weaned, this will rob the newborn of valuable IgG. This may also happen if a pregnant female serves as a wet nurse for multiple crias. We know multiparous cows produce higher-quality colostrum than uniparous cows. This is also true of camelids as seen in a study by Bustinza [11]. Energy-deficient diets result in a decrease in the volume of colostrum produced but does not alter the composition. IgG content of the first milking is double that of the second milking, and content is halved with each succeeding milking [39]. Leakage of colostrum from an engorged udder and possibly other crias in the maternity stealing from a prepartum dam can negatively impact the amount of good-quality colostrum available for a newborn. In females that deliver prematurely, the udder may not have had time to prepare for the birth in both production of IgG and quantity of colostrum available. This will depend on how premature the cria is and if she has lactated previously. In females that have a history of mastitis, have a blind gland (s) or come into milk with mastitis in a quarter, the amount of colostrum will be less, however, three teats with good colostrum provide enough IgG at 48 hours. This may happen when one of the teats is missing or an accident happens at time of shearing. Immune status of the female prior to parturition can also be a function of the quality and quantity of colostrum produced.

Consumption of colostrum can also be an issue and may be due to factors related to the dam or to the cria, factors such as mastitis, edema or agalactia from reasons already discussed. Crias that are premature, dysmature, weak or have physical defects may be unable to nurse or absorb the colostrum given. A crucial component in the survival of the neonate is the concentration of immunoglobulin G (IgG). This specific immunoglobulin is the main source of antibodies for the neonate. Camelid crias are born without IgG (agammaglobulinemic) because the camelid placenta is refractory to the passage of IgG from the mother to the fetus. Crias must acquire antibodies from colostrum.

Upon ingestion of colostrum, IgG levels increase dramatically to 2500 mg/dL in serum. This value is maintained for the first 10 days after birth, and then it decreases slowly to 1500 mg/dL during the next 10 days [40]. There is no difference in the IgG concentrations between llama and alpaca crias.

Colostrum Composition and Absorption

Colostrum differs widely from milk [8, 14, 23, 41]. Colostrum is high in protein (immunoglobulins [IgG, IgA, IgM]), high in chloride, low in fat, low in carbohydrates (lactose), low in calcium and phosphorus, and higher in minerals and vitamins than milk. It contains a high number of white blood cells that are vitally involved in providing protection against infection (macrophages, B and T lymphocytes, neutrophils), and it is also high in complement, which inhibits infection of the lining of the stomach and intestines [42, 43]. Camel and SAC colostrums are similar in fat percentage and are sticky like glue in contrast to the thick and creamy colostrum of cattle (Table 20.3). The high protein content of colostrum is the result of the presence of IgGs. There is no exact match for camelid colostrum, but Table 20.3 provides information on three other colostrum's. Both cow and goat colostrum can be used as substitute colostrum. Sheep colostrum is too high in fat and will likely make a camelid cria develop diarrhea.

Camelids, like other ungulate species, obtain passive protection from IgGs via intestinal absorption of macromolecules in colostrum. Absorption is short lived and failure to ingest colostrum during the first 12–18 hours of life is probably the most important predisposing cause of mortality in camelid neonates. In Peru, serum IgG concentrations of alpaca crias that died were significantly lower than those of crias that lived [13, 40]. Colostral intake serves two purposes: (i) a source of IgGs for absorption into the circulatory system across the intestinal mucosa and (ii) local effects, protecting the mucosa against endotoxins and microorganisms. The first requires ingestion within 12–18 hours of birth, while the second benefits the animal for a number of days and is brought about by local action of the specific IgGs, nonspecific complement, transferrins, and lactoferrins. The common recommendation for the amount of colostrum to be given is 10% of their body weight within the first 12 hours, with 5% consumed within the first 6 hours of life. Though it is impossible to determine the volume ingested when a cria nurses its dam, it is unlikely to be 10% of its body weight. This is a guideline for when a cria needs to be fed in total or supplemented. There are many factors that can interfere with absorption of colostrum such as cold/heat stress, prematurity or dysmaturity, dystocia/cesarean section, and metabolic disturbances of the cria. The final outcome is that even if you feed the cria what seems to be a large amount of colostrum, they still may have a low IgG concentration.

Research done in Peru also indicates that the time of suckling colostrum by the cria is important to achieve more than 1000 mg/dL at 48 hours [13]. The highest IgG concentration happens when crias start getting colostrum between two and four hours after birth. Suckling colostrum at 6 or 8 hours after birth also provides marginal but close to 1000 mg/dL at 48 hours (Figure 20.32). The process of absorption can be delayed somewhat by withholding nutrients for no longer than 24 hours (Figure 20.32). Note the lower total IgGs absorbed. This is not to be relied upon as this is a non-selective process and whatever macromolecules arrive first, including bacteria/pathogens will be absorbed.

The IgGs contained in colostrum are large-molecule proteins. Normally, such proteins cannot be absorbed from the intestinal tract but are digested to amino acids, which can then be absorbed. During the first 12–18 hours of life (time varies), the wall of the intestine remains open to absorb IgGs but then closes to prevent infections from also gaining entrance to the body. The mechanism of absorption of IgGs is a biologic marvel (Figure 20.33). The cells lining the intestine contain vacuoles. The protein molecule and the

Table 20.3 Colostrum composition (as fed).

Species	Water (%)	Protein (%)	Fat (%)	Kcal/L
Llama	74.0	16.5	0.95	1341
Goat	73.5	12.0	6.0	1064
Sheep	58.8	20.1	17.7	2064
Cow	70.0	17.0	3.0	991

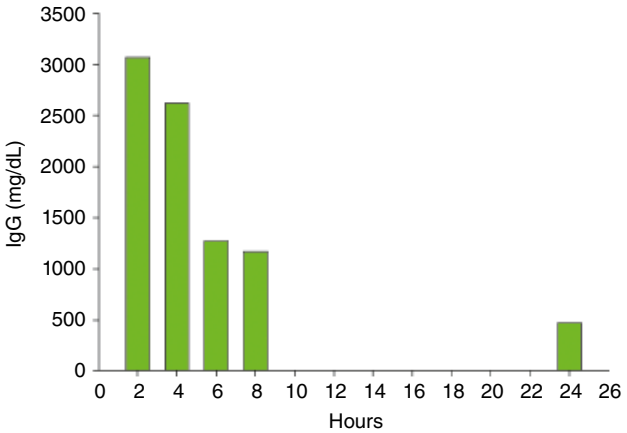

Figure 20.32 Concentrations of IgG in crias at 48 hours when suckling of colostrum begins at 2, 4, 6, 8, and 24 hours after birth. *Source:* Garmendia et al. [13].

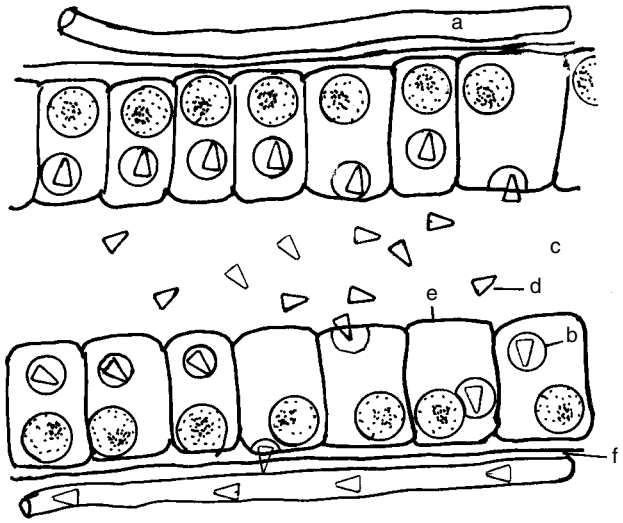

Figure 20.33 Diagram of the transfer of immunoglobulins through the mucosa of the intestine. (a) Lymph vessel, (b) IgG molecule in a vacuole, (c) lumen of the small intestine, (d) IgG molecules in colostrum, (e) epithelial cells of intestinal mucosa, (f) basement membrane.

vacuole line up at the cell wall. The protein passes through the wall and is engulfed by the vacuole and thus is spared the usual response of the cell to such a foreign body. The vacuole containing the IgG moves to the opposite end of the cell; the IgG passes out of the cell into the lymphatic vessels and then to the general circulation.

Information on the initiation of closure of the intestinal wall to IgG is extrapolated from studies conducted on cattle, sheep, and horses. In a normal full-term cria, closure begins soon after the first feeding and certainly started by six hours postpartum, to a certain extent whether or not the cria has nursed. Fifty percent closure has taken place by nine hours. A progressive change occurs in the cells of the intestinal wall that begins to destroy the IgG. The newborn has little protein digestion ability until a few hours after birth at which time the IgG, which is a protein, begins to be digested. The newborn stomach contains little acid until a few hours postpartum which also helps to protect the protein from the digestive process until a few hours after birth.

The intestinal mucosa of the premature cria is not prepared for absorption of IgG macromolecules, so even though colostrum is administered via stomach tube, there may be a failure of passive transfer of IgG (Figures 20.32 and 20.33). This is an extremely important factor, necessitating intravenous (IV) or intraperitoneal administration of plasma to avoid mortality from neonatal infections.

Colostral proteins are absorbed in the distal small intestine. IgGs of milk deposited in C1 of the stomach can be delayed, but there is radiographic evidence that, after orogastric feeding, the fluid does get to all compartments and even to the proximal small intestine [35]. However, the longer it is before colostrum is ingested by any method, the IgG may be denatured before it ever reaches the small intestine. A number of other macromolecules may be absorbed along with IgGs, including alkaline phosphatase (ALP) and γ-glutamyl transferase (GGT). Elevated levels of the two enzymes in the neonate may not be indicative of a disease state.

In cattle, a number of behavioral factors modify the ultimate levels of IgGs in the circulation of the calf. Mothering has a beneficial effect, with higher levels attained when the calf is kept with the mother and allowed to nurse freely, as contrasted with calves allowed to nurse periodically but kept separate from the mother. Calves that are bottle-fed but left with the mother are in the intermediate range of absorption. Lowest of all are levels reached in bottle-fed calves, even when given equal or greater volumes of true, first-milk colostrum. It is not known how these factors affect camelids, but it is reasonable to assume that crias are similar. Though the camelid female does not lick the cria as does a cow her calf, a bonding and nursing response nevertheless occurs. Other factors that may affect camelid immune response follow. Cold weather decreases a cria's vigor and hence overall colostral intake and absorption. Heat stress likewise inhibits IgG absorption efficiency [23]. A cria delivered following a dystocia may not have a depressed IgG uptake, but one delivered by cesarean section will. Such animals must be watched carefully. (Figure 20.34).

Absorption is directly related to the amount of IgGs ingested. There seems to be better absorption from a first ingestion with a higher concentration of IgG than from the same total quantity of IgG in a larger volume of milk.

Using experience from calf rearing as a guide, it can be assumed that crias deprived of all colostrum are at risk of dying from colisepticemia within three to four days of birth.

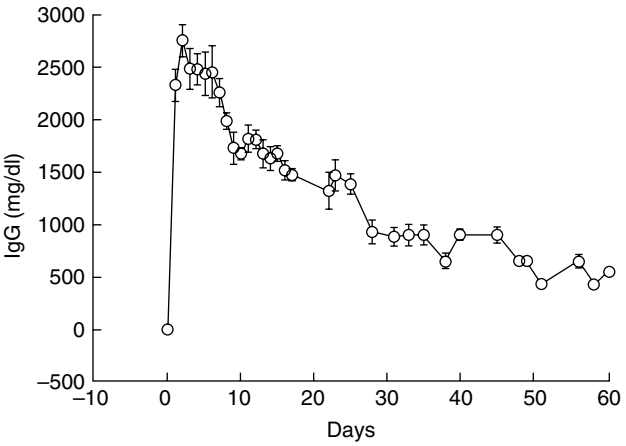

Figure 20.34 Immunoglobulin concentrations in the alpaca cria from birth to day 60. *Source:* Bravo et al. [23].

If some IgG has been absorbed, the risk of disease is still great but will occur later in the form of diarrhea, pneumonia, or chronic polyarthritis. If the dam has not been vaccinated against nor exposed to a specific pathogen, the cria remains at risk from that pathogen. Even crias absorbing adequate IgGs may succumb to an overwhelming infection.

Failure of Passive Transfer (FPT) of Immunoglobulins

Causes of the failure of immunoglobulin transfer are listed in Table 20.4.

Detection of FPT

Even with intense management, it is thought that 10–30% of calves are hypo- or agammaglobulinemic as a result of failure of passive transfer of IgG. This is significant in neonatal morbidity and mortality [2, 13, 23, 25, 44]. Veterinarians dealing with llamas and alpacas must also assume this to be true in camelids and act accordingly.

Early recognition of FPT is crucial to the well-being of the cria. A history of no ingestion of colostrum during the first 24 hours is clear evidence of FPT, but this may not always be known. The cria may have appeared to nurse without obtaining colostrum for reasons enumerated in the agalactia discussion. Also, the amount of colostrum intake is important. Important to mention is there are definite location, herd and time of year differences when evaluating crias that have low IgG. If the management, nutrition, etc. of the herd is excellent and the climate is moderate (dry, warm) crias with FPT can and do remain healthy.

Crias should be tested for passive transfer (PT) at 36–48 hours of age. Studies have indicated that at 24-hours old, some crias may be misidentified at FTP and thus the recommendation to wait until 36 hours old [45]. Single radial immunodiffusion (SRID) to measure IgG has been the gold standard in many species of livestock neonates including camelids, having high sensitivity and specificity (species specific). SRID plates are commercially available from Triple J Farms (part of Kent Laboratory, Bellingham, WA). Triple J has a long history of working with camelids and has been producing SRID plates for camelids since 1982. Due to the species-specific nature of the test, the SRID plate intended for camelids must be used in crias that received camelid IgG. There are SRID plates available for cattle, goat, and sheep IgG if colostrum from those species are used. SRID plates are considered a reliable measure of determining PT in camelid crias. Some comments on the use of the SRID plate: Manufacturer's directions need to be followed closely and, for consistency, the same person performing and reading the results each time. Small differences in measuring the circumferences of the precipitin rings can lead to large changes in the IgG concentration reported. Results using the Triple J SRID plate of >800 mg/dL are considered adequate PT; values <400 mg/dL are considered FPT. Though there are vast differences in the

Table 20.4 Causes of failure of passive transfer of immunoglobulins.

Conditions associated with the dam
Rejection of the cria
Not immune to common diseases
Poor colostrum production
Obesity (inhibits mammary gland development and may make it difficult for the cria to grasp the nipple)
Painful udder
Udder edema
Small teats
Plugged teat ducts
Overabundance of milk may cause dam discomfort, enlarged teats and reluctance to allow nursing. Heavy producers may also begin to leak colostrum from the teats because of the pressure
No milk letdown for 24–48 hours; in the meantime, the intestinal tract closes to the absorption of immunoglobulins
Mastitis—blood, pus, clumps, odor, color change
Older females may begin to have diminished colostrum and milk production
A pregnant female may allow a juvenile to continue nursing during the three to four weeks prior to parturition
Premature delivery—dam may not have produced IgG yet
Causes—unknown, heat stress, excessive stress of the pregnant female (physical restraint, medical procedures [vaccination, deworming, nail trimming, shearing], transporting, social stress [regrouping, new arrivals]), and prenatal infection
Conditions associated with the cria
Weakness—unable to stand (prematurity, fatigue from prolonged labor, prenatal infection).
Congenital defects
Facial (dental malocclusion, wry face, choanal atresia)
Limbs (carpal contraction, fused joints [arthrogryposis])
Trauma during delivery, Cesarean section
Prenatal septicemia/endotoxemia, *Escherichia coli* "dummy syndrome" cria
No nursing reflex (prematurity, septicemia, endotoxemia, hypoxemia)
Conditions associated with human interference
Too vigorous unplugging of teats
Failure to prevent late nursing of older offspring
Failure to properly immunize the dam prior to birth

conditions of crias born in Peru (Figure 20.35) compared to crias born in North America, in Peru, studies have shown greater survival rates if crias have an IgG of 1000 mg/dL [11, 13].

Other methods to determine PT include refractometer measurement of serum total solids, serum protein, globulin concentration, serum electrophoresis, sodium sulfite precipitation, zinc sulfate turbidity and serum γ-glutamyltransferase (GGT). All these methods have been used in camelids, but not all are reliable, easy to use, nor are available (Bethyl SSP). GGT activity has been determined to be unable to accurately predict PT [46]. Using a refractometer to measure total solids is one of the more practical tests available for practitioners. However, there are conflicting results in the literature [47, 48]. Generally, it is accepted that a refractometer reading <4.5 g/dL would indicate FPT, a reading >5.5 g/dL would indicate adequate PT. Reading between these two are challenging to interpret [48]. It is important that the refractometer be properly calibrated as that can greatly influence the readings. Physical exam of the cria and farm evaluation would need to be taken into consideration as to the next proper steps for treatment. Serum total protein concentration and globulin measurements are also methods used to determine PT. An earlier study considered the hand-held refractometer to be too variable for accurate detection of FPT and recommended use of serum total protein of >5 g/dL to indicate adequate PT and serum total protein of <4.5 g/dL suggest FPT [47]. Concentrations between 5 and 6 g/dL are adequate for most crias to indicate PT indicating probable successful passage of IgG. Serum globulin concentrations ≤2.3 mg/dL is an indication of FPT [47, 48].

Figure 20.35 Typical birth in Peru. Note fecal pellets in area.

Recently, a new commercially available analyzer has been described and compared to automated chemistry analyzer and SRID plate [49]. It is an immunoturbidimetric assay (DVM Rapid Test, Spring Valley, WI) and was determined to be more precise than the SRID assay. The researcher recommended to no longer consider the SRID assay to be the gold standard when measuring IgG [49]. A significant drawback is the high cost of acquiring the DVM Rapid Test analyzer, not to mention the cost of the individual tests and keeping reagents available. The Triple J SRID plate is still a reasonable choice when measuring camelid IgG in most veterinary practices.

Management of FPT

The management of FPT depends on the stage at which one becomes involved. Involvement at parturition is ideal [50]. If the cria has failed to nurse by three hours, colostrum obtained directly from the mother, or stored, frozen camelid or goat colostrum should be given to the cria, preferably by bottle. Mare and cow colostrum have been used, but goats are more likely to have been immunized for the diseases of most concern in camelids. Further, goat colostrum and milk composition are most nearly like those of camelids. It is virtually impossible to milk sufficient colostrum from the post parturient female to supply the needs of the cria. It is probably more satisfactory to rely on frozen goat or cow colostrum. However, the source of the colostrum must be scrutinized. The desired colostrum is the first-drawn milk only. Many livestock producers operate on the principle that since the milk is not normal for up to 72 hours after parturition, all fluid withdrawn during that time is colostrum. Unless it is specified that only first-drawn milk is to be used, producers may not supply suitable colostrum. Further, it is important to determine that the herd is free of disease: bovine viral diarrhea virus (BVDV), Johne's, caprine arthritis and encephalitis (CAE), brucellosis. Colostrum should be collected in clean containers and if intended to be frozen, to use a chest freezer not a frost-free freezer. Frost-free freezers undergo warming cycles to prevent buildup of frost. The colostrum (well mixed) should be divided into small portions of ~300 mL (10 oz) in freezer bags. This amount is in keeping with the 90–120 mL (3–4 oz) volume being fed at a time depending on size and maturity of the cria. Bags should be identified as to source and date, colostrum kept

in this manner will be good for one year. If a scientific freezer is available (−20 ° C to −70 ° C) it will be stable for longer. When needed, colostrum should be thawed slowly in warm water and never in a microwave as that will denature the proteins. When a bottle is warmed up to feed a cria, put the bottle of colostrum in a container of warm water (body temperature). Crias like temperatures slightly higher than human infants. This will be discussed further in the section on orphan crias. Studies done determining the success of using commercial colostrum replacements have not been done on camelid crias. One study did show that in calves, the Land O′ Lakes colostrum replacement did result in a serum total protein >5.2 g/dL, which correlated with an IgG of ≥1000 mg/dL [51]. There is a Land O'Lakes colostrum supplement product for goat kids and lambs. It would be interesting to see a study with camelid crias and this product. As the source of IgG is Bovine, a Bovine IgG test must be utilized.

As stated above, the common recommendation for the amount of colostrum to be given is 10% of their body weight within the first 12 hours, with at least half of that consumed within the first 6 hours of life. This is a minimum amount and it is best if the cria is willing to nurse from a bottle. An average size alpaca cria (7.7 kg, 17 lb) will be able to consume 90–120 mL (3–4 oz) every two hours; llama crias require more. If it is a premature cria, it may only be able to take 30 mL (1 oz) at a time. If it is unwilling to nurse, it will require feeding by orogastric intubation (see section on premature crias).

Treatment of FPT

Clinical diseases following FPT vary, but the most critical are bacteremia, septicemia, pneumonia, and enteritis (see Neonatal Disease section later in this Chapter) [50]. These require intensive care with appropriate antibiotics and fluid therapy. Partial or total parenteral nutrition may be necessary. It is best to start out treating FPT by preventing it by administering colostrum in a timely manner. As stated above, many things can interfere with the absorption of colostrum even if the cria receives what appears to be an adequate or even abundant amount. Also, having a high IgG does not guarantee the cria will not become sick. Crias with high levels of IgG may succumb to infection. The body may not be able to cope with overwhelming exposure to an infectious microorganism. Furthermore, immunoglobulins (antibodies) are produced in response to specific antigens, so if the dam was not vaccinated appropriately, the colostrum will lack certain protective antibodies, even though IgG levels are high. In addition, not all infectious agents stimulate the production of IgG.

The most effective method of treating FPT is administration of plasma to crias. Plasma may be given by three methods: IV, intraperitoneally (IP), and orally. However, if a cria is already showing signs of illness (lethargy, no suckle reflex, etc.), IV method is superior. IV plasma transfusion has a dual purpose of increasing total solids and plasma oncotic pressure plus added IgG to help the cria fight the cause of the illness. Administration of plasma (warmed, 37 °C) needs to be done carefully, using a blood filtration set. As it is likely the cria is dehydrated, conservative use of Banamine (Flunixin meglumine) at a half-dose of 0.5 mg/kg (0.5 mL/100 lb [45 kg]) can be given before the plasma transfusion to decrease the likelihood of a transfusion reaction. However, reactions are uncommon. Start slowly for the first 15–30 minutes and watch for any reactions (shaking, increased respiratory rate/distress, and increased rectal temperature). The rest can be administered over two to three hours (100–200 mL/hour). Too rapid administration can cause respiratory distress and possible death. The recommended amount of plasma is 30 mL/kg which is 270 mL for a 9 kg (20 lb) cria. Commercially available hyperimmune camelid plasma from Triple J Farms (Bellingham, WA) is usually about 250–280 mL per unit. Some crias benefit from a second unit of plasma during the course of fluid therapy if there was only minimal improvement in demeanor and desire to nurse ~24 hours after plasma transfusion. Llama crias will need two units to start with reevaluation after 24 hours.

When administering plasma IV to an extremely weak, debilitated, hypovolemic, and dehydrated cria, the placement of an IV catheter into the jugular vein can be challenging, especially if they have skin folds (wrinkles) as in Suri crias. The cria can be in lateral recumbency, with the head at the same level or in cush on a table with one person cradling the head/neck against their body and a second person keeping the hips still. Digital pressure over the jugular vein allows distension so the vein is more readily seen. In severely dehydrated crias, the jugular vein may have to be held off for a long time before filling and visualization is possible. Crias, even if depressed, do not like to be restrained and are prone to move at a critical moment. Restraint is very important to the success of the catheter placement, but it is best to have the cria only loosely restrained while preparing the site leaving the more confining hold until the time of actual catheter placement. This is a very stressful procedure for the compromised neonate and is essential to have all the supplies ready before the patient is even restrained. It is not uncommon for crias to look much worse after catheter placement. Traditionally, the right jugular vein is used, but if needed, the left can also be used. In crias, the jugular vein can usually be visualized after the fiber is removed. With Suri and some llama crias, the fiber can be lifted, taped out of the way and the fiber clipped underneath. In Huacaya crias, it is harder to hide that the fiber has been clipped. This is only important from a show point of view and it is

not the time to worry about cosmetics. After aseptic preparation of the neck, use a 15 blade (with 0.25 mL lidocaine bleb) to make a small longitudinal cut through the skin (Figure 20.36) over the jugular vein (tent the skin over the vein to avoid lacerating the jugular vein). This "cut through" eliminates skin drag on the catheter and facilitates placement of the catheter. Use 5 cm (2 in), 18 or 16 gauge for most crias and 5 cm (2 in), 18–20 gauge for premature crias. A larger catheter can be used in llama crias and camel calves. For security, the catheter hub and short (15–18 cm [6–7 in]) extension set (the author prefers JOR-467Z style) need to be sutured to the neck. After confirming blood flow from the catheter, use triple antibiotic ointment on 4 × 4 gauze pads over the catheter insertion site and tape everything in place. After the catheter is placed and a small bolus of fluids given (200–300 mL) put the cria back with its dam to "recover" for 30–60 minutes before any further diagnostics or treatments are done.

It is common for crias to be identified as having low IgG but are otherwise healthy. For insurance purposes, these crias will have to have a plasma transfusion as most insurance companies require an IgG of >800 mg/dL under three months of age. In one study, 10 of 25 llama crias were determined to have some degree of FPT, but none developed signs nor were treated for FPT [47]. As mentioned previously, crias in well-managed herds, in good weather may avoid serious illness even with low IgG. For these healthy crias, IP plasma administration may be done. There are several precautions with this method. It is not without risk, and the risk of complications increase if done improperly and by inexperienced personnel. Trauma to the kidney, intestines, or bladder is possible. There is controversy over this approach due to possible complications from potential formation of adhesions, even years later. The author (P. Walker) has been able to follow many animals out for 10 years that have had IP transfusions. None of these animals developed any abnormal clinical signs. There are several approaches to consider, left ventral abdomen and left or right paralumbar fossa. The choice is personal preference as all locations have inherent dangers. For the left ventral abdomen, it is best to have the cria lightly sedated and in dorsal to lateral recumbency. It is important to minimize movement as this will also decrease complications. For the left ventral abdomen, the preferred area is below and lateral to the umbilicus. There can be quite a few vessels in this area to avoid. For the paralumbar fossa approach, catheter insertion should be done in the center of the paralumbar fossa to avoid vital organs. Regardless of area, both should be shaved and surgically prepared. Use either a short, 18 ga catheter or blunt bovine teat cannula. When trying this for the first time, one may want to use the teat cannula, which will require a small 5-mm skin incision, then a "pop" through the peritoneum. With any method, the approach is to have the cria in a position to have the abdominal contents away from the insertion site. If using a catheter, the stylet is advanced to just through the abdominal wall muscles and the catheter advanced. If in correct placement, the catheter should advance smoothly. Once the catheter is placed and stylet is removed, the catheter hub should be observed for 60 seconds to determine if fluid returns. If you have inadvertently pierced the bladder, you will know immediately. In this case, stop and continue again in a few days. With correct catheter placement, the plasma should flow easily. If the catheter is too superficial, plasma will be deposited subcutaneously and the plasma will not flow well. In this case, the depth and possibly the angle of insertion should be altered to allow plasma to enter the peritoneal cavity. Warmed plasma is administered over 15–20 minutes using a blood filtration set. This greatly increases the volume of fluid in the abdominal cavity, and too rapid of an infusion (<15 minutes) can be uncomfortable. The skin incision may be sutured or glued and the cria returned to its mother. Though there is potential for adverse reactions (anaphylaxis), it is uncommon. As mentioned above, the IP route is best used only in healthy, growing crias so full dose Banamine can be used to premedicate the cria (1.1 mg/kg (1 mL/100 lb (45 kg) to reduce reactions.

Figure 20.36 Longitudinal cut through the skin for IV catheter placement.

Plasma may also be given orally by stomach tube. The decision to administer plasma orally is made when the dam does not have any colostrum or there is an impaired udder. The amount of absorption is minimal in most cases and the

resultant serum IgG is underwhelming. It is recommended to schedule an IP transfusion in these crias if the cria remains healthy. Unless the neonate becomes compromised, IP transfusions can wait for a week or two until the cria is growing and healthy. This is done with the understanding that the farm conditions are well managed and the cria remains healthy and growing at a steady rate.

Caring for the Orphaned Camelid

The cria may be orphaned at birth as a result of rejection by the mother, disease, injury, or death of the mother, or because of a variety of factors previously discussed in this chapter [6, 8]. Successful rearing of an orphan camelid is an art. Certain individuals within the camelid industry have made tremendous contributions to the knowledge of the rearing of orphans. Their experience and expertise should be sought when faced with the challenge of long-term hand feeding. However, the veterinarian should be able to deal with short-term problems and make recommendations on how to deal with FPT and neonatal infections.

Some basic information follows. Camelids rarely learn to drink milk from a bucket. Some crias will not go back and forth between a bottle and their dam, seeming to prefer one or the other, however some will. These crias can be hard to manage as it is important to keep them with their dam, but if she is a poor producer, will not have enough milk for the cria to grow and thrive. These crias should still be offered a bottle several times a day and will frequently take a bottle when they become very hungry. This will tend to be a trend in this particular cria. These crias (as many do) require much patience to succeed. If the female is slow coming into lactation, many producers feel if you feed the cria from a bottle, it will not learn to nurse its dam and prefer to supplement by orogastric intubation rather than bottle-feeding. With most crias this is not true, and it is best to give the colostrum by bottle as it is absorbed better. If a cria is bottle-fed, the bottle should be held level, even with the head, not above it, to avoid aspiration (Figure 20.37). The hole in the nipple used for a weak baby should be tiny, then enlarged with a crosscut as strength is gained. A lamb nipple or Pritchard nipple (Figure 20.31) is recommended. In a lamb nipple, use a crosscut, rather than a round hole for an orifice. The Pritchard nipple does have a hole, but the tip of the nipple must be cut off first. Cut off ½ of the tip when first introducing the nipple/bottle to a newborn. It may be possible to cut off more once the cria becomes experienced. Offer colostrum 90–120 mL (3–4 oz), (average size cria) every 2–3 hours up to 24 hours or until the cria is able to stand and nurse on its own or its dam starts to come into milk. To give these crias the best chance, it is best to feed them throughout the night for the first couple of weeks. Table 20.5 is a bottle baby feeding schedule to document milk feeding.

Figure 20.37 Bottle position for feeding a bottle to a camelid cria.

If the cria refuses or is unable to suckle an artificial nipple, it is necessary to use orogastric intubation to feed the cria. Orogastric intubation is easily carried out by straddling the recumbent cria in a kneeling position, keeping their head/neck braced against the humans chest. Many different tubes and methods are available to administer colostrum [24]. Red rubber feeding tubes (18–24 Fr, 16 in. long) work well in alpaca and llama crias (Figure 20.30). Due to the long neck, it does not go into the C1, possibly allowing some closure of the gastric groove and fluid to go to the third Compartment. Barium studies have shown that, after orogastric intubation, the fluid rapidly entered all compartments and even into the proximal part of the small intestine, but some was retained in C1 [35]. The tube should be lubricated with rectal lube or water. Hold the head in a semi-flexed position to insert the tube into the mouth. No speculum is necessary and they will fight less if not used. Gently push the tube to the pharynx and allow the cria to swallow it (Figure 20.38). Palpate the left cervical region for esophageal placement and continue insertion only to the thoracic inlet. Fluid traversing the thoracic esophagus may cause reflex closure of the esophageal groove and shunting of the colostrum to C3 of the stomach, where it belongs. Do not insert the tube passed the thoracic inlet because this will ensure placement of the colostrum into C1 where absorption may be delayed. Be aware that this tube can be "lost" in the cria if the end is not held onto or a catheter tip syringe acting as a funnel is not kept attached to the end. Because the tube is still within the esophagus, when administering the colostrum/milk do not use too fast of a rate as it will come up around the outside

Table 20.5 Bottle baby feeding schedule.

Cria:_____

Date	Time	Oz	Comments	Date	Time	Oz	Comments

of the tube possibly leading to aspiration. Also, bend the end of the tube over on itself when removing from the cria.

If it is obvious the cria will not be able to be fed by the mother (due to one of the many reasons listed previously in this chapter), then the first feeding should be given by four to six hours postpartum in the amount of approximately 1% of the body weight, 112 mL (4 oz) for an 11.34-kg (25-lb) cria. Repeat the feeding every 2 hours until 10% of the body weight is reached, ideally by 12 hours when most of the IgG will be able to be absorbed. For up to 24 hours, continue feeding with colostrum for any possible further absorption of IgG and local action of the colostrum and to make sure the cria receives the fluid and calories needed. The stomach capacity of most mammals is 25–50 mL/kg of the animal's body weight. For an 11.34 kg llama cria, this amounts to 280–560 mL, but it is undesirable to use the higher volumes until the stomach has expanded and is prepared to accept such amounts. If 24 hours have elapsed since birth, it must be assumed that the intestine is refractory to absorption of IgGs.

Another method for dealing with an orphan cria is adoption by another dam. In various instances, dams without crias may be available, especially when parturition occurs in a definitive season, and dams that lose their crias are good candidates to adopt an orphan cria. By nature, SACs are good mothers and they may adopt another cria if they have lost their own cria recently. In some areas of the world, the skin of the dead cria is put on the top of the orphan cria

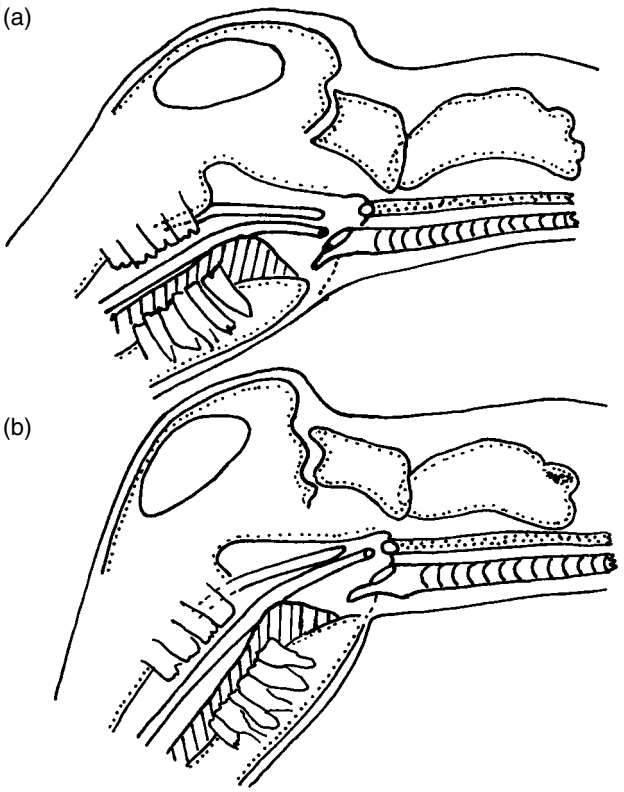

Figure 20.38 Diagram showing head position for orogastric intubation. (a) Head too straight, allowing insertion of catheter into the trachea, (b) proper head position which directs the catheter into the esophagus.

and then maintained with the surrogate dam separated from other animals. If the prospective dam has recently lost a cria and the placenta is still available, a portion of the placenta can be draped over the orphan cria. Some people try to rub the perineal area of the orphan cria close to the nose of the dam, and it may smell and adopt the cria. Others just put the orphan cria with the surrogate mother and see if the cria will attempt to nurse the adopting dam. Hopefully, the new mother will accept the cria and feeding the cria with milk replacement is not necessary. Keep in mind, the dam must allow the cria to nurse, but the cria also has to have the notion that it is acceptable to nurse another female that is not its mother. Some crias will not do this, so intervention will be needed. Regardless of technique used, the manager or owner must be patient during this process.

For a true bottle baby, it is best to start out with pasteurized whole goat's milk. Crias seem to prefer the taste and it is the commercially available milk that most closely resembles camelid milk [41]. The temperature of the milk is also important. Test it like you would for a human infant on your wrist, if it is just a little too warm for a human infant, then it is perfect for a cria. Remember their mom's milk is warmer than human's. Initially give 90–180 mL (3 oz to 6 oz) for normal size alpaca, llama crias respectively, every two to three hours (even during the night) for the first ~2–3 weeks. The amount can be increased gradually as the cria grows. The guideline is to give a minimum of 10% of their bodyweight (divided in many feedings) per 24 hours (Table 20.5). Full-time bottle babies can be slowly transitioned to goats milk replacer once they are drinking the whole milk well. Some producers add plain yogurt to the bottle to increase the calories. Crias are very particular about their bottles so bottle and nipple hygiene are very important. Use cold water for initial rinse, then very hot soapy water and clean with a bottlebrush. They do best with the Pritchard nipple. If only supplementing because of a poor producing dam, they can be fed just during the day. It is very important to understand that crias will not feel full for 5–10 minutes after being fed; they will always act as if they are still hungry. Overfeeding will cause discomfort that they may associate with bottle-feeding. If you are bottle feeding a stubborn cria that will not nurse its dam that has milk, bottle feed the cria for two to three days a full amount, then back off and skip a few bottles while urging the cria to nurse its dam. Generally, after a few days of being slightly hungry, the cria will learn that nursing its dam is a good idea. Encourage bottle crias to eat solid food as soon as possible, most crias start by 10–14 days. Bottle babies should be left with the herd, even if they do not have a specific mother. Social interaction between the bottle cria, the rest of the crias and the herd is very important for normal development, especially in males. Too much human "cuddling" can lead to aggressive behavior later when males reach puberty (see Chapter 2) [15].

Milk Replacement

A variety of milk substitutes (pasteurized, homogenized cow's milk; pasteurized goat's milk; commercial kid and lamb milk replacers) have been used successfully by caregivers who understand basic principles. Owners faced with the daunting task of having to provide milk for a cria are often given conflicting recommendations by other owners who may have tremendous or minimal experience themselves. Camelid owners sometimes feel that veterinarians confuse clients with conflicting information about what to feed and how to mix milk replacers for crias.

There is no difference between the composition of llama and alpaca milk, so the term "llama" applies to both. Table 20.6 provides an approximate composition of selected components of various milks. It should be evident that camelid, goat, and cow milks are quite similar in composition. Either goat or cow milk makes a satisfactory substitute for camelid milk, but some experienced orphan rearers recommend the use of goat milk (Meyenberg Goats Milk)

Table 20.6 Milk composition (as fed) [52].

Species	Water (%)	Protein (%)	Fat (%)	Kcal/L
Llama	86.9 (79–89.5)	3.4 (2.1–4.4)	2.7 (1.3–5.9)	822
Camel	86.4 (85.02–88.5)	3.6 (3.5–5.2)	2.9–5.5	–
Goat	87	3.3	4	680
Sheep	81	6.2	7.9	1138
Cow	87.3	3.3	3.6	653

because it most closely resembles camelid milk. Because goat milk fat is naturally homogenized, it may be more easily digested than the larger fat globules of the milk of other species. Any substitute fresh milk should be pasteurized. Orphans also satisfactorily tolerate kid and lamb milk replacers. Once a substitute is chosen, it is unwise to switch back and forth between milk products.

Consideration should be given to a cria's nutrient requirement. A 10-kg (22-lb) cria has a basic energy requirement (BMR) of 394 kcal/day to perform body functions in an optimal environment. Its maintenance requirements are BMR plus 1.5 BMR or 985 kcal/day. Camelid milk has approximately 822 kcal/L, and Land O'Lakes Lamb Milk Replacer, mixed with 720 mL (1.5 pints) of water, has 864 kcal/L (qt). Thus, a 10-kg (22-lb) cria requires approximately 1 L of milk per day (1 qt [32 oz]) or 10% of its body weight. A study conducted by Riek, et al. determined that at peak lactation (17 days postpartum) llama crias (18.7 kg (41.14 lb) would consume 2.6 kg (5.72 lb) [39].

Commercial Milk Replacers

Numerous products are advertised as being the ideal milk replacer. Most commercial products are satisfactory if basic principles are understood and followed [52]. Companies supplying milk replacers for camelids are listed in Table 20.7. Milk replacer should have a milk byproduct as their primary protein source (listed as the first ingredient on the label): dried whey protein concentrate, dried whey, dried whey product, etc. Do not use products that have non-milk sources as the primary protein source. Lamb milk replacer may be too concentrated, making kid milk replacer preferable. The mixing directions on the label of Land O′ Lakes Lamb Milk Replacer provide the appropriate reconstituted composition for lambs, but it makes a more concentrated milk than is normal for the camelid. Using 720 mL (1.5 pints) of water instead of 480 mL (1 pint) provides the closest approximation to camelid milk, but crias have been reared on replacer mixed according to the label directions. Note that kid milk replacer mixed according to label directions is close to camelid milk in composition (Table 20.8). Also, the "all species" products are not a good choice if a kid option is available as all milk is different for all species and camelid milk is most like goat.

Table 20.7 Some companies manufacturing lamb and kid milk replacers in the USA.

Company	Products
Land 0' Lakes Purina Animal Nutrition 100 Danforth Drive Gray Summit, MO 63039 Phone: (800) 227 – 8941 or (536) 742 – 6100	Doe's Match® Kid Milk Replacer Ultrafresh Lamb Milk Replacer ProNurse® Specialty Milk Replacer (llama listed)
MannaPro Milk Products 707 Spirit Park Dr. Suite 150 Chesterfield, MO 63005 Phone: (800) 690-9908 FAX: (636) 681-1799	Kid Milk Replacer Powder Lamb Milk Replacer with Probiotics Powder NurseAll Non-Medicated Milk Replacer (cria listed)
Sav-A-Caf® Chilton, Wisconsin Milk Products, LLC 855-227-9104	Sav-A-Kid® Milk Replacer Sav-A-Lam® Milk Replacer Ultra 24™ Multi-Purpose Milk Replacer (cria listed)
DuMOR Tractor Supply Brand	Blue Ribbon Kid Milk Replacer Blue Ribbon Lamb Milk Replacer
Milk Specialties Global 7500 Flying Cloud Dr., Suite 500 Eden Prairie, MN 55344 800-323-4274	Kid Milk Replacer Lamb Milk Replacer Multi-Species Milk Replacer (list crias)

Table 20.8 Composition of Land O′ Lakes milk replacers and llama milk.

Product	Protein (%)	Fat (%)	Kcals ME/L[a]
Lamb Powder	24	35	
114 g/454 g (1 pt)	27 g/454 g = 5.95	40 g/454 g = 8.8	1152
114 g/681 g (1.5 pt)	27 g/681 g = 3.96	40 g/681 g = 5.9	864
Kid Powder	26	20	
114 g/681 g (1.5 pt)	29.6 g/681 g = 4.3	22.8 g/681 g = 3.3	
Llama Milk	2.1–4.4	1.3–5.9	822

[a] Kilocalories of metabolizable energy per liter.

Unless you have sound data and/or experience to direct you, always follow the directions provided on the label. Even these may be confusing, so careful reading is necessary. Several products supply a plastic measuring cup to measure the powder. These cups are only to measure the

powder and are not meant to measure the water added. A standard liquid measuring cup (240 mL/cup) is intended to be used for the correct mixing.

Another consideration is that product ingredients may vary. Also, proximate analysis of some commercial products has been performed by users, and the levels as stated on the label have not always been accurate. Palatability factors may also enter into acceptance of any product.

Weaning

There are many factors that need consideration when weaning: weight, age and if the cria is chewing its cud. In North America, alpaca and llamas crias are around six months and 27 kg (60 lb) for alpaca and 45 kg (100 lb) for llama crias. Crias should double their birth weight (or more) by 30 days of age. Initially growth is slow, especially in larger neonatal crias, but will increase at an accelerated pace after the first week. This will be a reflection of the dam's lactation curve [53]. Also, crias should be weighed on a regular basis, the frequency will vary by farm and how many crias they have at one time (Table 20.9). If there are many crias in several barns, all crias under 23 kg (50 lb) should be weighed daily with the understanding that a few days may be missed on occasion. Crias are very stoic and can hide illness well just as adults. After 23 kg (50 lb) is reached, weekly will suffice for most crias. If the farm only has a few crias in total, they still need to be weighed on a weekly basis to keep track of growth. It is easy to forget how old a cria is and keep thinking it is just a "baby," when really it is undersized for its age. Any cria acting off should be weighed and compared to previous weights.

Frequently, crias wean themselves by six months of age, however, a few persist in nursing for more than one year if their dam will tolerate it. However, in these instances, the amount of milk consumption may be negligible, and the nursing has become a behavioral pattern. Some females

Table 20.9 Cria Weight schedule.

DATE	NAME	NAME	NAME	NAME	NAME	NAME	NAME	NAME

may kick the cria away at four to five months. In vicuñas, the crias are weaned at approximately 6 months and expelled from the herd at 9–12 months [26]. Guanacos have a slightly different weaning pattern, and yearlings may still be found with the mother while a cria of the year is nursing.

Mothers and their crias have a strong attachment and the time of weaning is very stressful for both. It is common for crias to lose weight the first week after weaning and then start to gain weight again [27]. Crias will pace, walk the fence line and vocalize in search of their dams. Some dams also do this to a milder degree, with a few looking to escape back to their crias. It is best to leave the cria in the same area and to move the dams to a different area. It is also best to wean as a group and not individually. The dams should be moved to an area not in visual range of the crias and not share the same fence line. On occasion, a cria will have to be returned to its dam for another week or so before trying again. On smaller farms, a procedure called "day weaning" can be done. For five to seven days, take the crias away from the dams for the day, then put them back together for night. Then one day, just do not put them back together. This will work for many crias, however, each cria must be handled as an individual.

Generally, four to six months should elapse before female crias are reintroduced to their dams. It is common for each to find the other again, and they must be observed if nursing behavior resumes, especially if the dam is pregnant again. Some families always remain close, even as adults with their own crias. Male crias must have a separate area to go apart from mature males. Mature males are not tolerant of the young and will force them down to "breed" them. Weanling males can usually be safely put with males two years of age and under.

If a cria has converted to pasture and hay, there is little advantage to continued nursing after six months, and there may be a decided disadvantage to the female if she is pregnant again. Some owners wean at five months, and this is entirely satisfactory if the cria is well adapted. Orphaned crias may be able to make it on their own at two months of age, but it is not advisable and there is less risk of stunting if the nursing period can be prolonged.

Neonatal Diseases and Conditions

Umbilical Infection and Associated Complications

Acute Umbilical Infection

Navel ill (omphalitis) is inflammation of the umbilical stump and associated structures, including the urachus, arteries, veins, and surrounding tissues (Figure 20.39) [54]. The infection may be localized in the region of the umbilical stump or spread to the bladder via the urachus, the liver via the umbilical veins, or the general circulation to become a septicemia, or it may spread directly to the peritoneum causing peritonitis.

The majority of infections result from contamination of the cord with ubiquitous organisms in the immediate environment (Figure 20.40). This is especially true if FPT of IgG

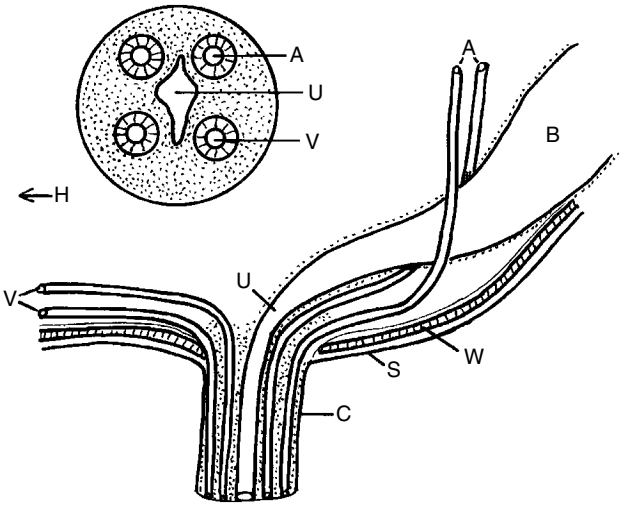

Figure 20.39 Diagram of the relationship of the bladder and vessels. (A) Umbilical artery, (B) bladder, (C) umbilicus, (H) cranial direction, (U) urachus, (V) umbilical veins, (W) body wall.

Figure 20.40 Camelid giving birth in Peru. Heavy environmental contamination increases the risk of omphalitis.

has occurred, rendering the neonate susceptible to otherwise nonpathogenic organisms. A variety of opportunistic Gram-negative or anaerobic organisms are the usual cause.

If infection is limited to the base of the umbilical stump, there may be swelling, heat, pain on palpation, and an exudate from the stump. Infection of the umbilical arteries, veins, urachus, or peritoneum may have no external manifestations, but the animal may exhibit colic, depression, or anorexia and spend an inordinate amount of time recumbent.

Diagnosis can generally be made with a thorough physical examination, but hematology and US examination of the umbilical region may be helpful. Physical exam should include a deep palpation of the abdomen above the umbilicus to detect thickening of internal structures.

If infection is detected early (within 24 hours), broad-spectrum antibiotics (Procaine Penicillin G, Ceftiofur) and supportive care may be sufficient. If infection is allowed to spread or if abscessation occurs, it is usually necessary to remove the infected tissue and structures surgically.

Chronic Infection

Chronic umbilical infection may lead to hemotogenous spread of the bacteria leading to bacteremia or septicemia, septic arthritis or polyarthritis (joint ill), septic physitis, and septic epiphysitis. The pathogenesis is similar to that for acute infection, but the manifestation of disease is delayed for varying reasons, including innate resistance of the animal, slow-growing abscesses, or localization of infection in various organs and tissues.

Signs are highly variable depending on the site of infection and may be delayed for three to six weeks. Urinary tract abscess may result from progression of infection from the urachus. Liver abscesses may occur secondarily to omphalophlebitis. Sudden lameness in one or more legs is the usual initial sign for secondary orthopedic infections. Joints may be swollen, hot, and painful upon manipulation. Arthritis caused by trauma is rare in crias.

Arthrocentesis and radiography are used to establish joint infection. Cultures may be negative even in an infected joint. Blood cultures may be more useful.

The prognosis of septic arthritis is guarded to grave. Aggressive therapy is crucial for satisfactory resolution. Systemic and intra-articular broad-spectrum antibiotic therapy is necessary. Antibiotic therapy must be continued for four to six weeks. Joint lavage with normal saline is used to remove fibrin and debris to minimize destruction of the articular cartilages and synovial membranes.

Umbilical Hernias

Umbilical hernias are congenital, but not necessarily heritable. They can occur if there is too much tension on the umbilicus during delivery or secondary to omphalophlebitis. It is not uncommon to be able to fit the tip of a finger through the "ring" surrounding the umbilical structures. This should close by one month of age. If the opening allows three fingers (~ 5 cm), it is unlikely to close on its own. With a large enough hernia, it is possible to entrap a portion of the gastrointestinal (GI) tract. Be aware of this if a cria shows abdominal discomfort with an umbilical hernia. If the hernia closes by three months of age, then it is not thought to be genetic as in other species.

Use of a hernia belt is common among camelid owners and the belts can be successful at facilitating closure of the hernia ring (even up to 3 cm [1.2 in]). Use of the belt or a pad with tape must be done with caution. If applied improperly, the cria could develop urine scald and or skin ulcers if the tape or belt is not checked at least weekly to ensure it is not too tight as the cria grows. Umbilical evisceration is rare but can happen when the cria is born. If this happens at an observed birth, the intestines can be kept clean and moist and attempted to be surgically replaced. There would be a high likelihood of peritonitis. The one case the author (P. Walker) has seen was in an unobserved birth early in the morning and the cria was found walking on its own intestines.

Patent Urachus

Failure of the urachus to close shortly after birth can result in urination from the umbilicus. It is usually congenital or on occasion a reopening of the urachus due to urachal infection. If this is present at birth, the umbilicus should not be tied off as this will only trap in whatever bacteria has already been wicked up into the umbilicus at and after the time of birth.

Routine care of the neonate includes aseptic dipping of the umbilicus (see Care of the Umbilicus previously discussed in this chapter). Repeatedly dipping the umbilicus with Tincture of Iodine (½ Alcohol & ½ Betadine solution or commercial product) may help the umbilicus to dry out and close down. As the urachus is in direct connection to the bladder, the cria should be started on Procaine Penicillin G (44 000 IU/kg, 20 000 IU/lb, subcutaneously, twice daily for five to seven days) to prevent a urinary tract infection. Using this approach, the patent urachus should resolve in a few days. Some crias may need surgery to resect the urachus and the tip of bladder if it does not close up after three to five days.

Osseous Sequestration

A sequestrum is a piece of dead bone that has become separated during the process of necrosis from normal or sound bone. This discussion will focus on the unique aspects of this condition as typically seen camelid crias.

The typical presentation is a healthy cria, with no other apparent problems, is noticed to be lame. Though the lameness is normally in one leg, but multiple legs can be affected at the same time or a few days apart. This lameness can come on acutely and there may or may not be swelling at the site of pain. Unlike other livestock, there is no history of trauma. Rarely is there a history of illness or FPT. It is possible there was a mild umbilical infection as a source of infection but given the stoic nature of crias, mild illness may have been overlooked. Radiographic evaluation of the bone in question is usually adequate for diagnosis, but if there is no evidence of a sequestrum at initial investigation, repeat the radiographs in five to seven days. Young animals have a quicker progression of boney changes than adults and the second image should show the sequestrum. It is rare for there to be a draining tract unless the lameness has gone unnoticed for a period of time. It is theorized that the cause of these sequestra are hematogenous in origin, even though a source of the bacteria cannot be determined in most of cases.

The majority of crias respond to sequestrectomy as a treatment with proper bone support in the follow-up healing period [55] (see Chapter 10 for further discussion).

Neonatal Septicemia

Septicemia is defined as the presence of a suspected or proven infection with evidence of systemic inflammatory response syndrome (SIRS). SIRS has many components and it has been proposed that if two of the following criteria are met, then sepsis exists [56]:

1) Hypothermia or hyperthermia (more common to have hypothermia)
2) Tachycardia (heart rate > 100 bpm
3) Bradycardia (heart rate < 60 bpm) with no evidence of concurrent heart abnormalities
4) Tachypnea (respiratory rate > 30 bpm)
5) Leukocyte count either elevated or depressed, > 10% immature neutrophils

Septic crias can have a wide range of presentations from vague, non-specific signs to a cria with obvious abnormalities. There is usually a history of no or inadequate colostrum intake by the cria. If the cria has lost weight or not gained weight for 24 hours, is more lethargic than previous and not staying with its dam, these are signs of possible sepsis. These crias can quickly go from being a "normal" cria to septic. Also, crias can be born septic from a healthy dam, and this may actually be the cause of a premature birth. These crias will usually appear normal at birth (catecholamine surge), but within a few hours (or less) be "limp rags" (Figure 20.24) and unable to stand or keep their heads up. These crias present similar to premature crias. Physical exam on septic crias will show tachycardia, tachypnea, injected sclera/conjunctiva (Figure 20.41) and gums, weakness, depression and dehydration. Rarely will a cria have a fever; it is more common to be hypothermic. Blood work can show either a leukocytosis or leukopenia. Hypo- or hyperglycemia can be present along with hypernatremia, hypoproteinemia, and hypoglobulinemia. Crias can have a good IgG and still become septic. If hypoglycemia is present, the cria can be presumed to be bacteremic. The key to treatment of septic crias is IV fluid therapy to provide circulatory support and to "flush" the system of endotoxins formed by the bacteria involved in the sepsis.

A balanced isotonic electrolyte solution (acetate containing) such as Normosol® or Plasmalyte® is the best to use. It is important to warm the fluids before administration to prevent further decrease in cardiac output. Even with this precaution, crias will frequently start shaking toward the end of the fluid administration. It is hard to determine based on physical exam alone if a cria is hypo- or hyperglycemic; solutions with higher concentrations of dextrose should be avoided until glucose concentration determined. If needed, start with 1–2% Dextrose added (for each 1% dextrose needed, add 20 mL/L 50% dextrose). The glucose concentration of the cria should be monitored while on IV fluids containing dextrose to detect which direction the glucose is going. If high glucose containing solutions are needed, slow weaning from glucose is required to avoid hyperglycemia or rebound hypoglycemia. Once the cria is partially rehydrated giving Banamine (Flunixin meglumine) half dose (0.5 mg/kg [0.23 mg/lb], IV, twice daily) will help with endotoxemia and make the cria feel better and hopefully willing to nurse. In addition to isotonic fluid

Figure 20.41 Injected conjunctiva of septic cria. *Source:* Photo courtesy Darlyn Shumaker, Alpaca Darlyn's Suri Farm.

therapy, a plasma transfusion is also required (see Treatment of FPT earlier in this Chapter). Crias are prone to hypernatremia (and occasionally hyperchloremia) with or without diarrhea, so the crias sodium concentration is important to determine before the type of fluid is selected. Crias may be hypokalemic with diarrhea, so potassium chloride (KCl) at 20 mEq/L can be safely added. If the cria has metabolic acidosis (pH < 7.2) then IV bicarbonate is the best to use. If the cria develops acidemia without diarrhea (rare) [57], correcting half of the base deficit along with rehydration usually results in restoring correct acid–base balance, then re-evaluate. If the acidemia is due to diarrhea, correction of ¾ to the entire base deficit may be needed, then re-evaluate the acid–base status.

To calculate the amount of bicarbonate needed to correct acidosis:

$$Body\ weight\left(kg\right)\times 0.5\frac{L}{kg}\times Base\ deficit\left(\frac{mEq}{L}\right) = Bicarbonate\ deficit\left(mEq\right)$$

Fluid calculation example:

9 kg (20 lb) cria with base deficit of 10 mEq/L

$$9\,kg\times 0.5\frac{L}{kg}\times 10\frac{mEq}{L} = 45\,mEq\,HCO_3$$

The concentration of HCO_3 available will determine the volume of HCO_3 needed.

- If using 1.3% HCO_3 (0.156 mEq/mL) then 45 mEq/0.156 mEq/mL = 288 mL HCO_3 needed
- If using 8% HCO_3 (1 mEq/mL) then 45 mL HCO_3 is needed – this small amount can be given along with Normosol fluids for correction of the dehydration

Fluids can be administered to correct half of the calculated deficit over two to three hours with most crias. Due to hypoproteinemia, camelids are very sensitive to pulmonary edema from rapid rehydration when receiving IV fluids, so monitor the cria carefully for pulmonary edema. If this is a concern, then replacement of the deficit should be done more slowly over six to eight hours.

Most crias with severe diarrhea cannot or are unable to nurse enough to maintain their hydration. After the dehydration, electrolyte and acid–base balance is restored, maintenance fluids need to continue until the diarrhea subsides. Maintenance needs are 2–4 mL/kg/hr; use the higher rate if the severity of the diarrhea results in continued weight loss despite IV fluids. If the diarrhea and weight loss continue for more than five days, consider partial parenteral nutrition (PPN).

Several challenges exist when administering IV fluids to camelids, one is that catheters can be positional depending on neck position; this cannot be controlled for with standard IV drip sets. Another challenge is there is usually a cria and a dam in the stall, so depending on how active the cria is, a drip set cannot be left attached to the cria. It is important to keep the cria and dam together, so an alternative is to create a small area within the larger stall with straw bales to house the cria until the most critical phase of treatment is done. Septic crias are frequently too weak to lift their heads and have a slow blink reflex, making them prone to corneal ulcers. If the cria is unable to hold its head up, be sure to wrap the bales in sheets or towels to prevent corneal trauma (Figure 20.42).

Once the cria is more stable and mobile, the best method is to calculate the maintenance needs for three hours and slowly "bolus" this amount over 20–30 minutes. Remember to warm the fluids before administering. It is also important to continue feeding the cria. If the cria is too weak to lift its head, it is better to wait to feed the cria until after it has responded to the initial round of fluids. If needed, the cria can be tubed if there is no suckle reflex or refuses a bottle. In a cria still able to stand, assist the cria to nurse its dam. The cria needs to be weighed twice a day to monitor success of fluid therapy. Tapering off fluids can be started when the cria has recovered and back to fully nursing its dam.

In addition to fluid and plasma therapy, antibiotics should be administered. Both Gram-positive and

Figure 20.42 Cria kept safely in bale enclosure.

Gram-negative organisms have been isolated from neonatal crias with sepsis [2, 44, 58]. Ideally, blood cultures should be taken, but the time to obtain results is too long for immediate action. So broad-spectrum antibiotics with good activity toward the most common bacteria involved (*E. coli, Enterococcus* spp., *Pseudomonas*, *Listeria*) should be administered. Naxcel® (ceftiofur sodium) at 4–8 mg/kg, (2–4 mg/lb) IV, twice daily is a good first choice as is it reasonably non-toxic. If an aminoglycoside (Amikacin 15–18 mg/kg [6.8–8.2 mg/lb] daily, IV for five days) is deemed necessary in combination with Naxcel, it should be given for a maximum of five days, in combination with IV fluid therapy and the creatinine concentration monitored every two days. Enrofloxacin at 5 mg/kg (2.3 mg/lb), IV, twice daily can also be used in combination with Naxcel [2].

Septic crias need to be kept warm, monitored for urination and development of further abnormalities (swollen joints, hypopyon, uveitis and corneal ulcers). If hypopyon develops or is present on initial exam (Figure 20.43), use of atropine ointment will help to decrease synechiae formation. Oxygen (humidified) is needed for some crias, for longer-term administration, nasal cannulas are preferred.

Hyperosmolar Syndrome

Hyperosmolar syndrome is characterized by severe hypernatremia, hyperchloremia and severe hyperglycemia (>700 mg/dL). There are no classic clinical signs, but affected crias usually are dehydrated and the cria may act as if its head hurts manifested as head pressing. Crias can be as young as one day old to several months (11 months) old [59]. Blood work is needed for diagnosis.

Treatment of hyperosmolar syndrome is complicated and requires minimal IV fluids combined with plain oral water 120–180 mL (4–6 oz), three times daily or more often, if [Na^+] is not decreasing. Insulin at 0.2 IU/kg (0.09 IU/lb), SQ is needed along with frequent monitoring of glucose concentration to determine if glucose is decreasing. Using diuretics with two different mechanisms of action will help to decrease the sodium and chloride concentration, subsequently reducing cerebral edema. The author (P. Walker) has used Furosemide at 4 mg/kg (1.8 mg/lb), IV, twice daily and Spironolactone at 2 mg/kg (0.9 mg/lb), PO, twice daily successfully. This condition requires regular monitoring of [Na^+], [Cl^-], and glucose to determine changes needed in treatment. This condition is frequently fatal, even with treatment.

Diarrhea

Diarrhea may occur when the cria ingests too much colostrum and/or colostrum becomes too acidic. The cria shows a yellowish and/or orange watery diarrhea that lasts for a day (Figure 20.44). Sometimes, the diarrhea is so severe that the cria becomes recumbent from pain. This type of diarrhea is generally temporary and the cria usually recovers by the following day. Usually treatment is not required. It is common for normal crias to develop a yellowish, low volume, normal frequency diarrhea about ~14–20 days old. These crias have normal behavior, weight gain and nursing frequency. This is a consistent finding on many of farms and appears to be similar to "foal heat" diarrhea. No treatment is required as it goes away on its own. If the producer wants to do something, they can give the cria 10–20 mL plain yogurt twice daily. It is important to check the dam to see if she has mastitis; if so, the cria is ingesting bacteria

Figure 20.43 Cria with hypopyon. Hypopyon is a sign of septicemia.

Figure 20.44 Diarrhea from a newborn cria. *Source:* Photo courtesy Rob Turk, Gaelic Glen Alpacas.

that may cause diarrhea. If any diarrhea continues for more than a few days and the cria is depressed and becoming dehydrated, the diarrhea may be caused by viruses (Rota, Corona), parasites (Cryptosporidium, Giardia), or bacteria (*E. coli*, etc.). When examining a cria, pay careful attention to the fiber on its legs and around the perineum. If it is very watery diarrhea, it may arc away from the cria initially, but then soil the fiber.

Viral Diarrhea

Both coronavirus and rotavirus have been recognized as causes of diarrhea in crias for many years [60, 61]. Parreño et al. determined that crias as young as two days of age can have severe diarrhea caused by rotavirus. In the same survey, it was determined that all the adult guanacos sampled were positive for antibodies to rotavirus [62]. Diarrhea caused by both coronavirus and rotavirus also occurs on alpaca farms in North America [63]. Recently, Jin et al. sequenced the genome of an alpaca coronavirus (ACoV) [64]. He determined that the sequence of ACoV was closely related to bovine coronavirus [64]. In an attempt to prevent viral diarrhea, alpaca owners are using oral vaccines made for calves; these vaccines are given orally to the newborn cria before nursing. Based on the findings of Jin et al., and clinical experience, it appears as if these vaccines should be effective. The author (P. Walker) has been using Pfizer's Calf-Guard® 3 mL, orally at birth before the cria nurses for years without problems with an apparent decrease of viral diarrhea. This is only recommended for farms that have had positive identification of viral cause of diarrhea. In the camelid show circuit, there have been several post-show outbreaks of both coronaviral and rotaviral diarrhea three to five days after returning from different shows. Most of these cases were self-limiting and could be controlled with common anti-diarrheal drugs (Kaolin, Imodium); however, several fatalities also occurred (personal communication). In neonates, vial diarrhea tends to be high volume and high frequency and frequently requires IV fluids to maintain hydration in addition to other supportive care. It is important to rule out other pathogens that may also be present as is the case in calves.

Bacteria Diarrhea

E. coli and *C. perfringens* spp. can cause diarrhea in crias, however *Salmonella* spp. is not common [44, 65]. *E. coli* diarrhea is often associated with neonatal sepsis and can be secondary to failure of passive transfer [44]. *E. coli* diarrhea can also occur secondary to viral diarrhea in older neonates as happens with calves. *E. coli* diarrhea typically occurs between three to seven days of age, usually before any of the other more common causes of diarrhea can start. The diarrhea may be profuse and liquid, with the cria very lethargic and may have abdominal distension. Crias can be leukopenic, but unlike calves are usually hypernatremic [65]. If the cria is also septic, blood glucose is usually low, but glucose concentration should always be verified before any dextrose is administered as the cria may be hyperglycemic. Treatment should be aimed at correcting the dehydration and electrolyte imbalances and broad-spectrum antibiotics administered. Many owners use oral vaccines made for calves to prevent diarrhea caused by *E. coli*; The author (P. Walker) has used Boehringer Ingelheim's Bar-guard 99®, 5 mL, orally, three to five hours after birth for years without problems.

Parasitic Diarrhea

Cryptosporidium parvum occurs in calves greater than seven days of age but in crias it can be as early as four to five days of age. Diarrhea in crias caused by *Cryptosporidium* spp. can cause profuse, liquid, green, uniquely foul-smelling diarrhea with some crias rapidly losing body weight (10–14%) requiring aggressive treatment with IV fluids. There are several ways to diagnosis Cryptosporidium in feces; acid fast stain and fecal floatation with Sheather's solution are two methods used with variable results due to the small size of the oocyst, variable staining characteristics and possible low numbers shed at the time the sample is taken. As a result, immunoassays have been developed to aid in diagnosis. One such test is a commercially available lateral flow immunoassay test called ImmunoCard STAT! for Crypto/Giardia distributed by Meridian Bioscience in Cincinnati Ohio. This test is very easy to use with results in 10 minutes. The author (P. Walker) has provided the company with positive samples for several years. Those samples are retested with a more sophisticated test (Merifluor DFA) at the lab with all the samples testing positive.

There are no labeled treatments for Cryptosporidium but many different drugs have been proposed, some with more success and others more toxicity. Paromomycin (Humatin®) is a nonabsorbable aminoglycoside that is used in immunocompromised humans [66], cats, and calves [67] in an attempt to treat the infection. The human trial had a response rate of 67% and even though there were few cures, the diarrhea improved more with the drug than with the placebo [66]. Grinberg et al. showed that neonatal calves started to shed oocysts and develop diarrhea only after the drug was withdrawn, suggesting strong anticryptosporidial effect [67]. Viu et al. demonstrated a significant reduction of oocysts in lambs after being treated with paromomycin at 100 mg/kg (45 mg/lb), orally for three days, plus resolution of diarrhea immediately after the medication stopped [68]. In the literature, there are positive responses and negative responses to Paromomycin. In the author's experience, in a clinical setting, with natural occurring

cases of diarrhea caused by Cryptosporidium, use of Paromomycin at 50 mg/kg (23 mg/lb), orally for 5–10 days has been very successful in shortening the duration and severity of diarrhea in crias. Other drugs used to treat diarrhea caused by Cryptosporidium (lasalocid, nitazoxanide, decoquinate, or halofuginone) have a narrow margin of safety when used with camelids or access to the drug is limited.

Giardia spp. can occur in crias as early as 14 days of age and is correlated most often with periods of high rainfall or problems with run-off water. Giardia is seen on farms where run off water is a problem or when water accumulates from use of hoses and sprinklers to cool off the animals in the summer. The dams will lie in the puddles, contaminating their udder, then the cria will nurse and ingest the oocysts if present; also crias seem to have an affinity for water puddles.

Diarrhea from Giardia causes soft, runny, slimy/shiny, slightly green feces that may have a frothy appearance (Figure 20.45). On occasion, it can result in profuse, watery diarrhea. Some crias will exhibit consistent straining to defecate before the onset of diarrhea.

Giardia cysts can be difficult to find in fecal flotations depending on the skill of the microscopist, so immunoassays such as the ImmunoCard STAT! above can be more reliable with quick results. Diarrhea from Giardia is usually seen in the fall and spring, but the oocysts can over winter and survive at 4 °C for several months. Once ingested, the trophozoite is released from the oocyst and invades the small intestines causing villous atrophy and subsequent malabsorptive diarrhea. Giardia diarrhea is an unusual finding in adults and is generally considered present on farms with overcrowded barns and pastures with a large population of young, immunologically naïve animals [63]. While this is true, the curious and very oral nature of an active cria also contributes to exposure to many parasites. Camelid crias can be treated with Fenbendazole at 50 mg/kg (23 mg/lb), orally, daily for five days for diarrhea caused by Giardia. The author has also used Metronidazole at 50 mg/kg (23 mg/lb), orally, twice daily for five to seven days without any problems, but only use in crias less than two months of age.

Figure 20.45 Feces from cria with diarrhea from Giardia. Giardia frequently causes soft, slightly green feces with a frothy appearance.

Diarrhea caused by Cryptosporidium and Giardia can be very frustrating and costly on some camelid farms. Most camelid owners clean their barns and even pastures daily, sometimes twice daily and are frustrated by still having crias develop diarrhea. Rulofson et al. looked at 354 llamas from 33 different farms in California, ages from 3 weeks to 23 years. Using the Merifluor DFA test, 12 (3.4%) of the specimens were positive for Giardia and no specimens were positive for Cryptosporidium [69]. His conclusions were that farms with larger numbers of crias born the previous year would result in greater numbers of yearlings, which provided the source of animals shedding the parasites, keeping continual exposure for young crias [69]. A study in Spain looked at asymptomatic cows, sheep, and goats, aged 3–13 years old. The study found Cryptosporidium in 8.4% of cows, 5.3% sheep and 7.7% goats and Giardia in 26.6% of cows, 19.2% sheep and 19.8% of goats [70]. This supports the idea that neonates of many ruminant species are exposed to these parasites by subclinical carriers. There is no reason to conclude alpaca farms would be different.

Nasolacrimal (NL) Duct Occlusion/Atresia

Nasolacrimal duct occlusion can be congenital (atresia/stenosis of puncta or duct) or acquired from debris accumulation in the duct [71]. Usually, history is long-term ocular discharge from one or both eyes refractory to topical ophthalmic ointments. Ocular discharge can be a new symptom or present since birth.

To flush the NL duct use a small animal NL catheter, 22–24-gauge IV catheter or a 3.5 French polypropylene catheter depending on the size of the patient to cannulate the NL duct. There is an inferior and superior conjunctival puncta as well as a nasal punctum (Figure 20.46a and b). All of these are very small in a young cria and can be challenging to cannulate. The cria should be deeply sedated and pre-medicated with Banamine (1.1 mg/kg 0.5 mg/lb, IM). It facilitates the procedure to use Proparacaine to numb the cornea and have a bright light. Locating the nasal punctum and being able to open the nostril enough to access it can be difficult in a small cria. The inferior conjunctival puncta may actually be easier to cannulate in small crias. Be careful not to damage the cornea if this

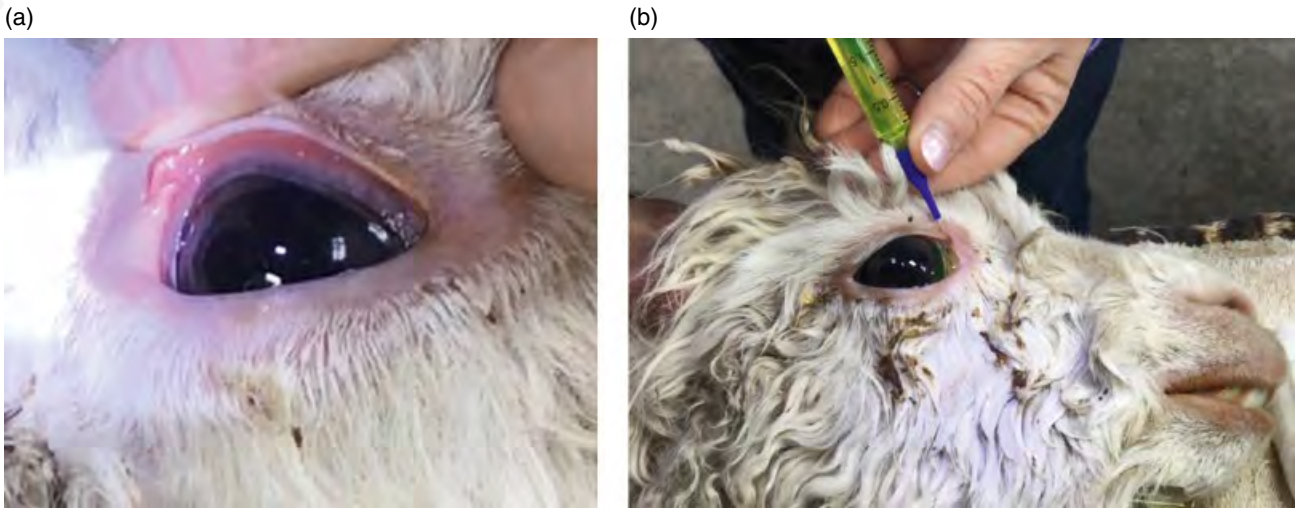

Figure 20.46 (a) Superior conjunctival puncta. (b) Superior conjunctival puncta, cannulated. Note the discharge on the fiber. *Source:* Photos courtesy Becky Camma, The Alpacas of Spring Acres.

approach is used. Using saline and a small 1 or 3 mL syringe pulse saline through the catheter and watch for saline to exit the other puncta. Initially, there will be cloudy debris, then a stream of saline, flush thoroughly. If there is a nasal puncta present, you should see saline coming through the nasal puncta. If unsure, locate the nasal puncta and observe if a membrane is bulging over the puncta. If so, take a #15 scalpel blade and cut away the membrane. Keep on flushing to clear the duct completely. There should be little trauma in most cases and follow up triple antibiotic drops with steroids can be used for a few days if no corneal ulcer is present (see Chapter 18).

Neonatal Maladjustment (Dummy Syndrome)

Lack of oxygen during delivery, or the latter part of gestation from placental insufficiency can result in cerebral hypoxia. Affected crias are often depressed but are still able to walk around. These crias have a difficult time finding the udder and seem more disoriented and wander away from the dam if left out in an open pasture. Proximity to the dam is helpful as the cria does not have far to look, so it is best to put the pair into a maternity stall. Dams will sometimes not pay close attention and bond with these crias until they start to nurse. These crias will probably have to be fed colostrum via orogastric intubation and treated as if a premature cria. Depending on a blood test, administration of glucose may also help. Because there may be hypoxia, administration of oxygen to the cria may prevent development of this syndrome. In Peru, it is possible these crias would not survive, especially in a maiden dam or in inclement weather. These crias can frequently be taught to be a normal cria, but the caretaker will have to have patience. If they do not respond by the end of their first day to normal care, they should be evaluated for common neonatal problems. If the cria seems healthy in all other regards, treatment with DMSO 20% solution (1 g/kg [0.45 g/lb], IV in fluids, once) to reduce cerebral edema may help. This pair should be kept confined until the cria is reliably nursing its dam and gaining weight.

Meningitis, secondary to sepsis, is uncommon and can happen without obvious signs of failure of PT. It is well recognized in the foal that the blood–brain barrier is not well established in neonates, and consequently any bacteria may enter the brain and cause inflammation and edema which, in turn, causes this behavior (dummy). That could also be the case for camelids. Clinical signs associated with meningitis are usually vague. Depression, seizures, unable to lift their head, unable to right themselves, tremors, knuckling, sleeping more than normal, etc. are some of the more common clinical signs. A cerebral spinal fluid tap with cytology and culture can sometimes be helpful in diagnosis. Treatment needs to be aggressive, using IV antibiotics that cross the blood–brain barrier. Naxcel at 6–8 mg/kg (2.7–3.6 mg/lb), IV, twice daily, Ampicillin 10 mg/kg (4.5 mg/lb), IV, three to four times daily, and NSAIDS (Banamine) are appropriate.

Seizures and depression can result from hyperglycemia or hypoglycemia and must be diagnosed and treated immediately and aggressively to save the cria. Both conditions can present in a similar fashion. Determination of glucose concentration is important before any dextrose is administered. Keeping a hand-held glucometer in your vehicle is recommended [72]. See Hyperosmolar syndrome and Septic crias earlier in this Chapter for more information.

Limb Deformities and Rickets

There are several types of limb deformities in camelid crias: ligament laxity, flexural limb deformities, and angular limb deformities. Laxity of the ligaments can be seen in premature crias. Physical therapy with the cria as part of its care will help strengthen the legs, so by the time the cria can walk on its own the laxity will quickly disappear in a few days. Flexural limb deformities or contracted tendons can be seen in the carpus, fetlock, or pasterns. Causes such as *in utero* positioning, heredity or possible toxins have been postulated. In milder cases, if the cria is able to bear weight and walk on its footpad, it stretches as the cria starts to move, bear weight and use the limbs. Some normal newborns can be born down on their fetlocks ("slipper foot") but normal movement and weightbearing resolves this in a few days. (Figure 20.25) In more severe cases, if the cria is walking on the dorsum of the foot or the legs cross over so severely the cria cannot bear weight, splints may have to be applied. Various splints can be used from the small animal world or a length of PVC pipe. Careful application of the splint is needed as the skin of the distal limb is not thick. The splints should be checked daily and removed and replaced if needed every five days. Arthrogrogryposis is uncommon in camelid crias [73].

Angular limb deformities can be congenital or acquired. Careful history from the owners will be helpful to determine if the cria was born this way or has developed over time. Carpal valgus is a very common deformity seen both in North America and sometimes in South America (Figure 20.47). Some degree of carpal valgus may be acceptable. Looking at the dam or sire may be indicative, as conformational issues will be heritable. There can also be trauma associated with joint instability in premature crias or possibly from rickets. Breeding decisions for the next offspring may be determined based on the suspected origin. Even though defects can be corrected (see Angular Limb deformity in Musculoskeletal System Chapter 10 for treatment), this does not make the animal acceptable to include in the breeding program.

Rickets [74, 75] (Vitamin D-responsive hypophosphatemia), though a metabolic condition, will present initially as a possible neurologic problem. Rickets is a lack of vitamin D in growing animals. Milk does not contain vitamin D, so if a nursing cria does not get exposure to sunlight because of weather, fiber cover or wearing a coat, they will be at risk for rickets. Clinical signs usually show up between four and seven months of age. Early signs are lameness, not wanting to play with herd mates and painful walking as if on "egg shells" progressing to a posture with a hunched back and a reluctance to rise. There is also swelling of leg joints (carpi, tarsi and fetlock joints) and

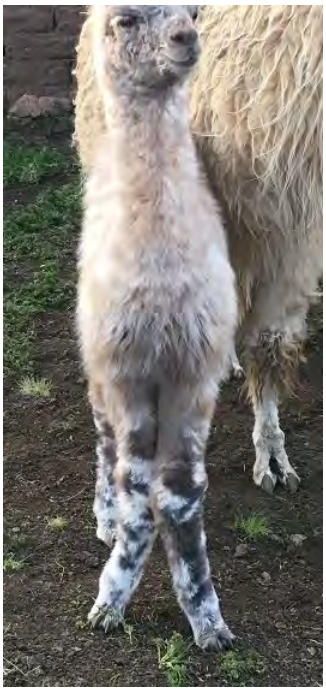

Figure 20.47 Carpal valgus in a llama cria in Peru.

the bone-cartilage junction of the ribs. Angulation of the front and/or back legs may also be seen in some animals. Diagnosis can be made by presence of classic clinical signs and lack of vitamin D supplementation with definitive diagnosis done by using radiographs and measuring serum vitamin D concentration. It is also important to measure serum Calcium and Phosphorous concentration as sometimes supplementation is needed. Serum Phosphorus <4.5 mg/dL, in an animal less than seven months old, with classic clinical presentation would be consistent with a diagnosis of rickets [75]. Treatment is vitamin D by injection at 2000 IU/kg (1000 IU/lb) subcutaneously, every two months. Prevention is easily done by giving vitamin D at the same dose as treatment, every two months OR 33000 IU, orally, every two weeks during times of minimal sunlight. Most signs will reverse with treatment if the disease has not progressed. There is a report of vertebral fractures in two crias [74] and the author (P. Walker) encountered a female with a hunched back with a history of rickets when a cria. She also had a deformed pelvis as a result and was unable to deliver a cria through the pelvic canal. Vitamin D as an injection comes in many concentrations and it must be communicated to producers to be careful when determining dose and also not to believe everything they read on the internet. Mineralization of tissues and renal failure after toxic amounts of vitamin D are given either by injection or orally is not reversible and will be fatal [76, 77].

Congenital Defects (Partial List)

Choanal atresia/stenosis is a lack of complete union of the nasopharynx and pharynx and is considered to be a heritable genetic defect. There can either be a bony or membranous barrier between the nasal and pharyngeal cavities, preventing air passage. Frequently, these crias are born looking anatomically normal, so it is not always immediately apparent (Figures 20.6a and b). Clinical signs are flared nostrils, "gurgling" breathing sounds, open mouth breathing, and puffing out cheeks when they exhale. As they cannot breathe through the nose, they also cannot nurse. If they try any activity, including nursing they may become cyanotic and collapse (can also happen with cardiac problems).

It is important to definitively diagnose this condition so normal crias are not mistakenly euthanized. To diagnose, several things can be done on the farm initially without advanced diagnostics. First, take a mirror or eye glasses and hold them up to the crias nostrils. Be careful that the cria does not breathe from the mouth on the glass. If there is fog on the glass from the nostril, then there is a passageway present. This does not test the size of the nasal passage and it is possible that there is choanal stenosis present. It is also possible that there may be an abnormal amount of mucus in the nares. In that case, time and activity will usually resolve the clinical signs. If there is no fog, further tests need to be performed. Next, using a small red rubber feeding tube (~ 16 French) and a small amount of lube, pass the tube along the ventral floor of the nasal cavity and pass down into the nasopharynx. If the tube does not pass and you are unsure of your technique, take a normal cria and pass the tube. It is not always easy to find the right place/angle, make sure the tube stays ventral and does not stop in the dorsal pharyngeal recess. Pay close attention and ensure the tube does not bend back on itself or coils up, mimicking being passed the full length. If the tube still does not pass, then further diagnostics should be done before the cria is euthanized. Contrast radiographs can be taken to confirm, though endoscopy (6 mm) is the definitive test. Put 5–10 mL of radiopaque contrast material in each nostril with the head and neck extended. Using a horizontal beam, keep the cria in cush position, head elevated slightly for the image. If there is a passageway present, the cria will swallow or cough and only small amounts of contrast will be seen on radiographs. If there is no passageway, the cria will not react and the contrast will be seen on radiographs, also the contrast will run out when the head is lowered. Any attempt at correction is an involved and painful procedure. Most crias are euthanized as they have poor quality of life and many develop fatal secondary problems (aspiration pneumonia, septicemia, malnutrition, etc.). Even if only a partial atresia/stenosis is present, they should not be used as breeders. Surgical correction can be done, but surgery is prone to complications and not very rewarding.

Wry face (maxillofacial dysgenesis) [78] is a condition where the maxilla and mandible are to differing degrees misaligned (Figures 20.48a and b). This is considered to be a heritable, congenital defect, though not definitively proven. Frequently, they can manage to adapt to their abnormality depending on how severe it is, and they survive in a managed herd situation. In severe cases, their quality of life can be marginal and they will need to be eventually euthanized.

Cleft palate can involve all of the soft or hard (or both) palate (Figure 20.49). This may be heritable or result from toxin exposure that interferes with normal movement of the tongue during gestation and the tongue "is in the way" during palette

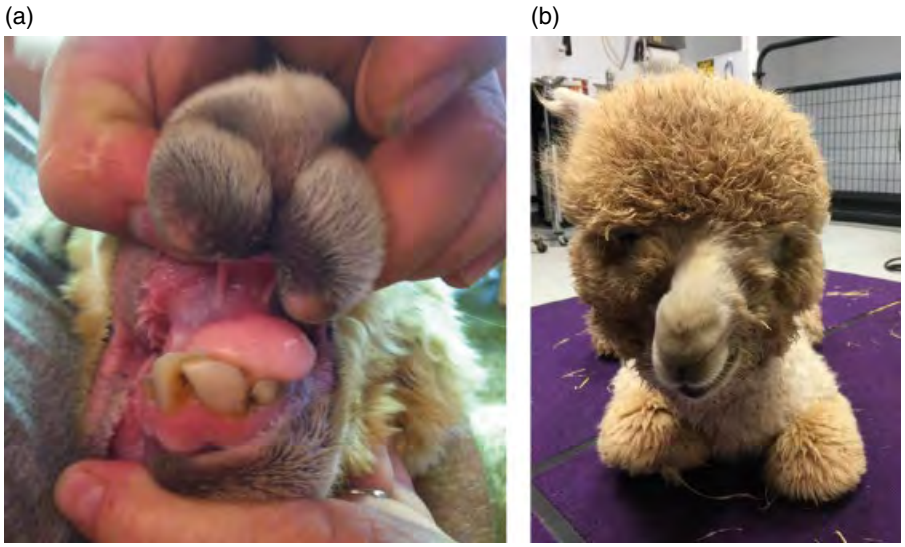

Figure 20.48 Jaw malalignment seen in a cria with Wry face. The mandible (a) and maxilla (b) can be involved.

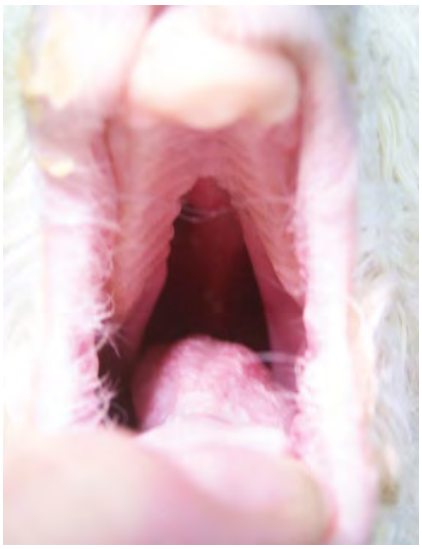

Figure 20.49 Newborn cria with a cleft palate.

formation. The cria may have a history of milk coming from the nostrils, or nasal discharge or aspiration pneumonia in the first few weeks of life. All newborn exams should include an assessment for cleft palette. These crias should be euthanized as treatment is long, painful and usually unsuccessful.

Atresia coli and Atresia ani are heritable conditions in calves and likely heritable in camelids. Crias with atresia coli can go an unbelievably long period of time with the condition without showing signs of pain. The author (P. Walker) had a bottle cria that went seven days, taking a bottle every three hours, with the abdomen progressively becoming more distended. It died at 3 a.m., after drinking its full bottle at midnight. Atresia ani can be seen alone or in conjunction with a rectovaginal fistula [79, 80]. Depending on the extent of the atresia, these conditions may be corrected with surgery, but frequently other abnormalities are present. These animals should never be bred and it is more humane to euthanize them. (Figure 20.7).

Vulvar hypoplasia is a defect where the vulvar opening is either partially (Figure 20.8) or completely absent (labial fusion) (Figure 20.50). It appears to be heritable in camelids, though not always expressed in subsequent matings. If it is complete, the cria will have stranguria and a "balloon" filled sac of urine at the vulva. (Figure 20.50) If not corrected, the cria will die. As a newborn, the "balloon" can quickly be incised allowing the cria to urinate. Use of triple antibiotic ointment with steroids topically for a few days and the normal frequency of the neonate to urinate (~6 times per day) will prevent closure. This opening will have to be surgically enlarged at puberty to allow the large amounts of urine produced as an adult to be passed. If the opening is left small, the urine will back up into the vagina and uterus causing secondary problems. On occasion, the cria will have a complete fusion of the tissue but is passing urine out of the clitoris (Figure 20.51). These crias should also have surgery because an adequate amount of urine cannot be passed in this fashion in a growing cria. These animals should not enter the breeding pool.

Hemivertebra is where one or more vertebra does not develop properly causing a wedged shape with subsequent misalignment of adjacent vertebra (subluxation) and pressure on the spinal cord (Figure 20.52). When this abnormality occurs in the cervical, thoracic or lumbar part of the spinal cord, it is a fatal condition, diagnosed by

Figure 20.50 Absence of vulvar opening. These animals quickly develop a "balloon" as urine fills and stretches the vagina. Creating a small opening in the vagina will allow these animals to urinate.

Figure 20.51 Two-month-old cria with a severely hypoplastic vulva. This cria was only able to pass urine through a small opening in the clitoris.

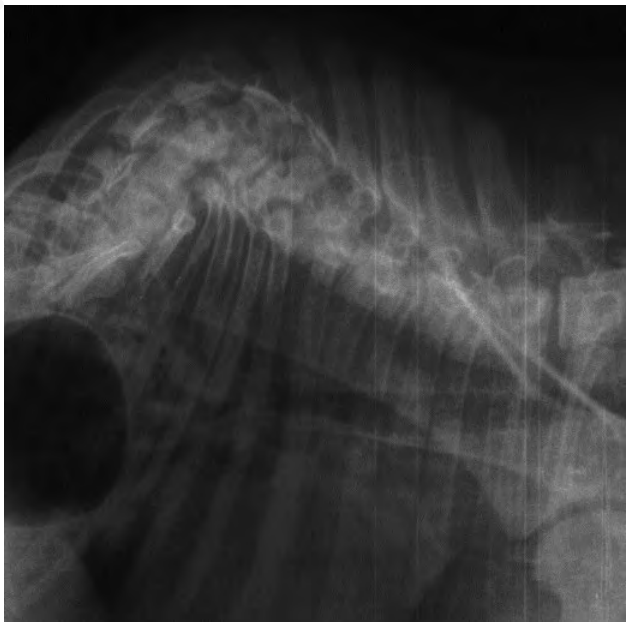

Figure 20.52 Radiographs of cria with Hemivertebra.

radiographs. Clinical signs (usually seen under one year of age) are usually an uncoordinated gait, possibly falling down when attempted to restrain, especially if the head and neck are manipulated. This trait has been associated with the cause of crooked (wry) tails. If it is expressed as only a crooked tail, there will not be any significant problem (with the gait). However, there seems to be a strong correlation between crooked tails and the other more serious vertebral deformity of hemivertebra. In one series of cases, many crias from the same sire who had a crooked tail also had crias born with crooked tails, short tails and some with short ears [81]. It is likely a heritable condition, but with the exception of crooked tails, these animals do not live to be a breeding age. It is recommended that this breeding match not be repeated and to eliminate from the breeding pool any animal that has an abnormal tail. When doing exams on camelids (of any age, but certainly newborns) make it a routine to exam the tail for any deviations.

There are many other congenital abnormalities possible, and camelids seem prone to them [73, 78]. This can be attributed to the Spanish nearly obliterating the SAC population and has been further compounded by selecting from this reduced population small groups of SAC to import into other countries [78]. The importation selection criteria were strict, any animal with obvious signs of abnormalities, including a poor bite, were to be rejected. This obviously did not eliminate genetic carriers of traits. Some congenital conditions not discussed above include pointed tongue, congenital cataracts, polydactyly, syndactyly, deformed uneven legs, numerous heart defects, ear pinna defects, lack of ear canal (cauliflower ear), urinary bladder agenesis, unilateral to bilateral renal agenesis, intracranial cyst, reproductive tract defects, hemophilia, etc.(Figures 20.53–20.58) [20, 71, 73, 78–86]. On rare occasions, crias can be born with a healed fracture. This happens for unknown reasons *in utero* (Figures 20.59a and b).

Immunoprophylaxis

There are no labeled vaccines for use in camelids, and it is unlikely this will change. There is little research done on vaccinations of camelids, however, there is one report out of Argentina on response to epsilon toxin of *C. perfringens* [87]. Their research showed that after vaccinating three- to five-month-old crias twice, 21 days apart, there

Figure 20.53 Cria with one short ear.

Figure 20.54 Cria born with blood on cornea. This condition self-resolved.

Figure 20.55 Cria with torticollis and arthrogryposis.

Figure 20.57 Cria born alive with uneven legs and deformed feet.

was a high titer in response. A third vaccination 21 days later resulted in a lower titer for unknown reasons [87]. This was not a challenge trial, so it is unknown if the crias developed a protective immunity.

Due to the lack of knowledge, there are many different protocols reported as to the "correct" timing of vaccination. Many of these extrapolate from other species or individual preferences. Based on solid knowledge from other species, the most protective regimen for the camelid cria is to make certain that the dam is immunized at least two months prior to parturition and that a booster vaccination is given four to six weeks before parturition. This ensures maximum production of specific IgGs in the colostrum. See discussion in Prepartum Care earlier in this Chapter. Because there is a cause for concern of abortion, on some farms, the dam is not boostered before parturition. In this case, it is common to vaccinate newborn crias for *C. perfringens* types C and D toxoids and *Clostridium tetani* toxoid (CDT) soon after birth (three to seven days) with a second vaccine given three to four weeks later and a third after six months. Crias are born immunocompetent, but the immune system lacks full maturity, and may be unable to respond to vaccination at an early age. Nonetheless, veterinarians with sheep experience

(a)

(b)

Figure 20.56 (a and b) Adult llamas in Peru with bilateral gofer ears.

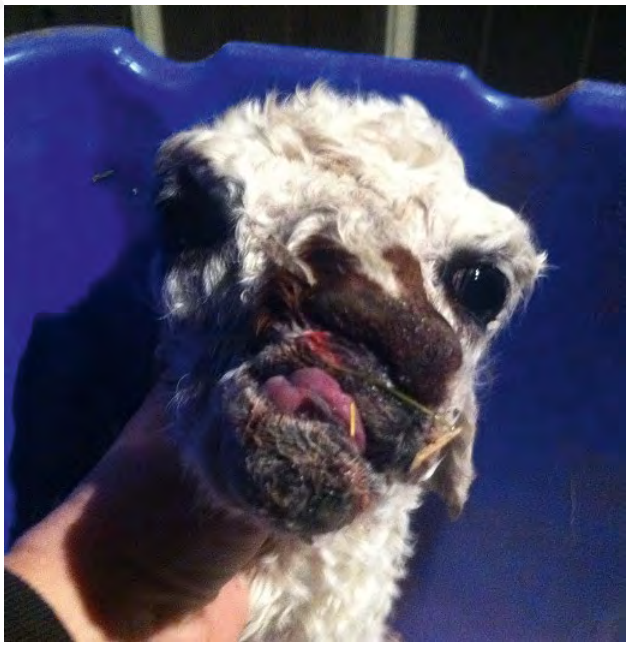

Figure 20.58 Cria born alive with severe wry face and choanal atresia.

recognize that enterotoxemia extracts a high toll in lambs that are not vaccinated by two weeks of age. Knowledge of clostridial diseases in camelids in North America is meager, but prudence dictates that vaccinations be administered within the first month of life, with boosters one month later and again at weaning. In South America, cria mortality rates from *C. perfringens* types A and C, can reach 50% [88]. Frequently, there are complications from other enteric pathogens contributing to the cause of death.

The specific vaccine recommended depends upon the region and prevalence of diseases in local cattle, sheep, and goats. Basically, it is recommended that CDT (3 – way) be given to the young crias. The multivalent vaccines with coverage for more Clostridial diseases (C. chauvoei, C. septicum, C. haemolyticum, C. novyi, etc.: 7 or 8 way) should only be used if deemed appropriate for the area/farm. If these are used, it is best to wait until shortly before weaning and not to administer to pregnant females. A study has been done to look at the immune response to tetanus toxoid vaccination with vaccinated crias showing higher titers than non-vaccinated. Titers of crias less than one month old had consistent titers with their dams, with older crias showing higher titers after vaccination [87].

In rabies-endemic areas, a killed rabies vaccine should be given at three to four months of age with yearly boosters. Do not use rabies vaccine in pregnant females. If anthrax may be of a concern, crias should be given only one-fourth to one-half the dose of anthrax spore vaccine recommended for the adult as deaths have occurred.

Vitamin E and Selenium (BO-SE® (1 mg/mL) –though not a vaccination, does need to be mentioned. Areas of the United States are selenium deficient and it is routine to give crias a dose of BO-SE® soon after birth. Generally, this is used in crias as a prevention of white muscle disease (Selenium deficiency). This should be given around seven days of age unless signs of

(a)

(b)

Figure 20.59 (a) This cria was born with a healed tibial fracture. (b) Radiograph of the cria's tibia showing the healed fracture. *Source:* Photos courtesy Dr. Samantha Coe.

muscle weakness are present and, in these cases, it should be given earlier. Be very careful only to use the BO-SE® product as Selenium overdoses occur with products containing a higher concentration of selenium and can be toxic. Anaphylactic reactions have been known to occur, so the animal should be monitored for respiratory failure and collapse after administration. The recommended dose is 0.055 mg/kg (0.025 mg/lb) which equates to 0.5 mL/9.1 kg (20 lb) of BO-SE® (1 mg/mL).

Routine Husbandry Practices

As a veterinarian, owners will look to you for guidance in many areas. Many producers will do most of the routine upkeep of their animals by themselves. However, a veterinarian will be required for certain practices and procedures. Unless properly trained, average camelid owners should refrain from drawing blood from the jugular vein. In crias, the jugular vein can be felt and sometimes visualized, especially in Suri alpacas. If needed, wet down the fiber with alcohol. Try not to shave the neck, but if needed, shave only a small area. Some crias have what is described as "wrinkles" on their neck and are a normal finding (Figure 20.60) but this can make getting blood even more challenging. These wrinkles represent extra skin and extra fiber density and will "stretch out" as they grow. Using anatomy, locate the 6th or 7th cervical vertebrae (largest) and work up the neck keeping in line with the transverse process. Place your thumb to hold off the vein medial to the transverse process, then "thrum" down the vein to feel it on your thumb (Figure 20.61).

In addition to drawing blood for testing for FPT and routine blood work, it is routine to DNA test and register alpaca crias. This is a requirement to show and if selling as a breeding animal. It is suggested of llamas, but not required unless registering as a breeder. To register, a DNA sample must be submitted using a DNA card that can be purchased from the Alpaca Owners Association (AOA) or the International Lama Registry. Frequently, owners will have cards available, but if planning to do camelids in your practice, it is a good idea to have some on hand. The jugular vein is commonly used to draw blood for DNA sampling; however, the tail or ear vein can also be used, and frequently is by owners. Using a 20-gauge needle put small spots of blood within the circle; do not just fill the circle with blood (Figure 20.62). If also using the DNA card for BVDV testing, make sure to date the card and have the owner send it in quickly as the BVDV testing window is only 60 days. The DNA will be good regardless of time.

BVDV has caused persistently infected crias to be born. It is not prevalent in most of North America but in an effort to keep it permanently out of the North America camelid population, it is recommended that all camelid crias be tested. PCR on whole blood is the test of choice and it is appropriate for the diagnostic labs to pool the samples for testing. The AOA DNA blood cards, if submitted by 60 days after drawn, can also be used for testing. BVDV testing is a requirement for alpacas to be shown, but not llamas.

Another routine practice is for microchip placement for identification. Many owners avoid doing this procedure and rely on their veterinarian. Some owners will put ear tags in their camelids, but the use of microchips is much more common and are necessary for an animal to go to a show. These do not have to be the type that needs to be

Figure 20.60 Wrinkles in cria skin.

Figure 20.61 Proper restraint for IV blood draw and catheter placement (alert cria). *Source:* Photo courtesy Becky Camma, The Alpacas of Spring Acres.

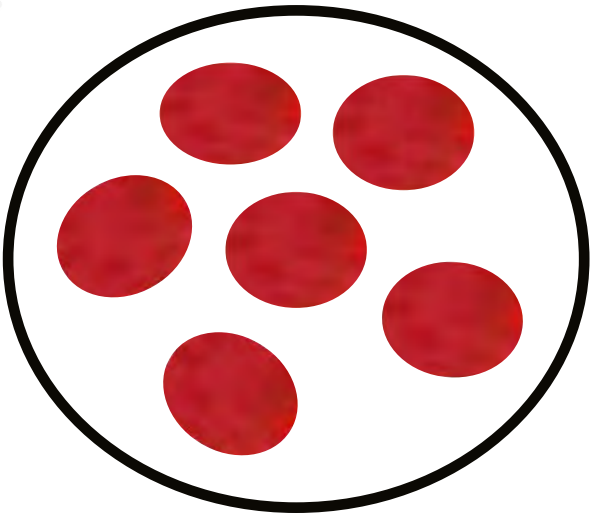

Figure 20.62 When drawing blood for DNA sampling, place small spots of blood within the circle; do not just fill the circle with blood.

Figure 20.63 Proper restraint for microchip placement.

registered, as they will be checked to confirm ID for health papers at a show or if traveling out of state. But they do have to be ISO compliant with 15 digits. The most common brands are Deston® and Smartchip®. It is standard for placement to be by the left ear or the base of the tail. The chip can be done at any time, even newborns, however, caution needs to be exercised when microchipping very young crias as movement of the head (crias are very wiggly) can result in the needle going through the skull. To avoid this, it is recommended that three people be used in very young crias. One to hold the cria up in the arms, one to "clam-shell" the head between two hands and one to place the chip (Figure 20.63). The chips should be placed by the left ear. Use the ear as a "handle" and hold the ear down to the side of the head and place the needle (facing forward) by the base of the left ear (side closest to center of head). Place the needle, no preparation is needed, make sure the tip of the needle is not all the way out the other side of the skin, and advance the plunger. Hold a finger over the needle site as it is being pulled out and keep pressure on the spot after the needle is removed. Sometimes, the site will bleed quite a lot, use pressure with gauze/towel to stop the bleeding. After placed, use a microchip reader to make certain the chip reads and verify the number is the same on the package. In some crias, you can actually feel a small "rice-like" bump under the skin. Make certain it is not just within the fiber, but under the skin. It is considered to be acceptable to place the microchip at the base of the tail. Do not use any other area, as it will be hard for a new owner or show check-in person to find the location if different.

Figure 20.64 Social interaction among crias.

Camelid moms and babies are very fun to be around! The dams need careful monitoring before and after giving birth and even more careful monitoring of crias after they are born. It is important to let them be crias, but if they are not up and active by three hours (or before), something is not right and they need to be checked out and at a minimum assisted to nurse. Interference is acceptable, but once they are okay, back off and let mom take over. Then enjoy watching the crias play!! (Figure 20.64).

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21

Congenital/Hereditary Conditions

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Congenital problems of llamas and alpacas occur more commonly than in livestock, companion animals and horses. Reasons cited for the overrepresentation of camelid congenital conditions include:

1) Culling of millions of camelids during the Spanish conquest from ~1500 – ~1800 CE. reducing genetic variation dramatically in South American Camelids (SACs) [1, 2].
2) Importation of small numbers of animals to develop national herds via "inbreeding" [1].
3) Selection of individuals possessing breeder-desired traits. In north America, selection for show qualities as well as fiber production in such small numbers is likely to narrow the genetic variation further [3]. Selecting for desirable coat color, fiber length and diameter and fiber production/animal could also be associated with selection for undesirable traits.

In addition to heritable defects, congenital lesions may be the result of exposure to toxicants or infectious agents. Toxins and viral agents are commonly associated with a variety of defects in neonatal livestock. Few similar lesions have been reported in camelids [4–7].

Plant and other toxicants exposures to camelids could also play a role, albeit in a small but growing number of documented cases. Plant and feed-related toxicities are commonly reported in camels.

Separating a congenital defect (present at birth) from a heritable trait (caused by genetic mutations or inheriting mutations from carrier dam and sire) is currently very difficult. Some defects are obvious at birth, whereas others may not be evident until later after significant growth. Further, some defects are lethal, causing early embryonic death, birth of a weak cria (dysmature) or dead full-term cria. At present, there are few examples of heritable traits that explain the defects present in camelids.

For other veterinary species, the presence of genes associated with heritable defects are well characterized. The ABCB1Δ mutation in collie dogs, pulmonary hypoplasia and anasarca, tibial hemimelia, complex vertebral malformation, spider lamb and mannosidosis in cattle, sheep and goats are a few of many described heritable defects [8–10]. In spite of the rapid production of the alpaca genome, the development of markers to identify clinically evident hereditary defects has not yet emerged.

After reviewing some terminology, the focus of this chapter will be to update the described congenital and heritable defects of camelids. Table 21.1 lists some of the congenital conditions seen in camelids.

Terminology

Congenital defects can be caused by single gene mutations, multiple genetic mutations and exposure of the dam to viral or toxic agents during pregnancy. Congenital defects may result from any of these individually or together. Congenital conditions are commonly, but not always obvious at birth. Some are obvious at birth (arthrogryposis, caudal vertebral defects, choanal atresia), while others may not be expressed until later in life (Wry face, angular limb deformities, hemivertebrae or vertebral fusion).

All camelids (Llama, Alpaca, Vicugna, Guanaco, Dromedary camel, Bactrian camel) possess a diploid chromosome number of 74. There are 37 sets of chromosomes from the sire and 37 sets from the dam. Alleles are the sequence of each gene, which can be identical or vary from one other. When two or more phenotypes are present within a population, differences in individuals may be called polymorphic traits. Fiber color and fiber length are examples of genetic polymorphisms. Genetic

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Table 21.1 Congenital conditions in lamoids.

Condition						
Skeletal	**Head and face**	**Reproductive**	**Digestive system**	**Cardiovascular**	**Ocular**	**Miscellaneous**
Ankylosis, carpus	Cerebellar hypoplasia	Cervix	Atresia ani	Atrial septal defect	Blindness, cause not determined	Dwarfism
Angular limb deformity [11]	Choanal atresia	Aplasia	Atresia ani + rectovaginal fistula	Aortic arch, persistent, right [12]	Cataract [13]	Ears, short
Carpal valgus	Cyclopia	Double	Atresia coli	Ductus arteriosus, patent	Ectropion	Hernia
Carpal varus	Meningoencephalocoele	Fallopian tube aplasia	Megaesophagus	Portocaval shunt	Entropion	Diaphragmatic
	Hydrocephalus, internal [14]					
Femorotibial valgus	Facial bones	Hymen, imperforate	Pyloric stenosis	Tetralogy of Fallot	Eyelid, hypogenesis	Inguinal
Metacarpophalangeal valgus	Agenesis	Intersex, pseudohermaphrodite		Transposition of great vessels	Iris, nonpigmented (glass)	Umbilical [15]
Arthrogryposis	Lateral deviation	Ovary		Ventricular septal defect		Renal agenesis
Femur, shortened		Aplasia				Polythelia, supernumerary teats
Hemivertebra	Mandible	Hypoplasia				Teat agenesis
Spinal agenesis	Brachygnathia	Penis				Toenails, crooked
Metacarpal, shortening	Prognathia	Corkscrew				Urachus, patent
Patella, medial luxation		Curvature				
Polydactyly		Hypoplasia				
Scoliosis		Persistent frenulum				
Syndactyly		Testes				
Tail, agenesis	Nares	Cryptorchidism				
Talus, vertical	Aplasia	Cystic structures				
Tendon contracture	Stenosis	Ectopic				
Carpus	Palate	Hypoplasia				
Stifle	Palatoschisis (cleft)	Twinning				
		Uterus				
		Segmental aplasia				
		Uterus unicornus				
		Vaginal fusion				

Note: Y = inheritance confirmed; S = inheritance suspected; U = occurs but etiology unknown; blank = no information.

variation is associated with single nucleotide polymorphisms (single nucleotide change (e.g. A > G) if this change in nucleotide results in expression of a different protein sequence, this polymorphism may be responsible for no effects (silent-synonymous) or a non-functional protein (non-synonymous) necessary for an important function (an example might be when histamine to proline change in a critical drug metabolism enzyme produces a non-functional enzyme [16] Other changes that can occur include indels (insertion–deletion mutation; small number of base pairs), variable number of tandem repeats (short sequences repeated one or more times in a sequence) rearrangements (deletion, duplication, inversion of sequence, translocation of sequence) and *de novo* mutations. Mutations are relatively rare events (<1% of the changes in DNA sequences) Many of these changes can be silent or may result in defective gene expression or loss of entire genes.

Recessive traits are difficult to identify clinically since they must be homozygous for that particular gene expression [17]. Since each gene is present in two copies, one copy of a recessive gene will be silent. When both copies of a gene are recessive, the mutant gene results in a loss of function, leading to a clinical disorder. Pulmonary hypoplasia and anasarca of cattle is an example of an autosomal recessive trait [17, 18].

Dominant traits will be clinical even if the animal has only one copy of the gene. In this case, expression of the gene alters some function that is clinically evident. Lack of horns in cattle is associated with an autosomal dominant trait [17].

Co-dominance is a form of mutation whereby the dominant trait is variable depending upon whether one (heterozygous) or both alleles (homozygous) are mutated. Polysaccharide storage myopathy in horses is an example of co-dominant inheritance [17, 19].

Sex-linked defects are more common in males than in females because they only have 1 X and 1 Y chromosome [17]. In males, half of the offspring of a single heterozygous dam will express the defective phenotype. If the affected males are bred to normal females, all females will be carriers of the defect. Factor eight deficiency has been reported in an alpaca cria and could potentially represent an X-linked recessive trait in alpacas [20].

Many defects are associated with more than one gene and influence of other genes or environmental conditions may shape the response of the individual based upon these interactions (in dairy cattle, [temperature, radiation] milk production and beef cattle, meat characteristics are influenced by multiple genes) [17]. For some of these characteristics, thousands of single nucleotide polymorphisms are associated with each trait [21, 22].

Management of Congenital/ Heritable Conditions

Waiting over 330 days and planning what will be done with a cria generates angst and enthusiasm. For many cases, the birth of a live but, perhaps compromised cria born is very traumatic for alpaca enthusiasts. It likely depends upon how functional the cria is (can stand, nurse and follows dam) relative to normal. I have seen many camelids with polydactyly, mild wry-face, bent tails and other defects who lived to be very old and were well cared for. Of greater importance, is whether these animals should breed if they can? In cases where one, or more births from the same sire produces deformed cria, should one consider limiting or ending the breeding career of the sire? Unfortunately, this is where we are currently. Some suggest that the presence of a defect (as yet undescribed) is reason enough to cull or no longer breed an animal(s). Currently, production parameters (growth rates, fiber production and quality, health status) may determine whether the genetics have enough merit to continue breeding a particular animal. However, there are many non-heritable reasons for poor growth rate, fiber quality, etc. that may also play role (hypovitaminosis D, poor mothering instinct, low milk production, etc.).

In terms of the national herd, it is imperative to limit the contribution of new animals to those with the most robust productivity. Until genetic markers are available, this may be the most robust means to improve productivity. Veterinarians should consider defects seriously and monitor the incidence of similar problems that have occurred in these animals. Repeated instances of the same or similar defects suggest heritability.

In the case of abortion, stillborn cria or euthanasia of a newborn with critical deformities, it would be prudent to investigate the morphologic abnormalities present in each case. Autopsy of affected animals should be performed on as many deceased animals as possible. There are likely many reasons for this statement, but herd health is one such reason. The autopsy of the sick or diseased animal can provide important information about the farm's management. An example might be provided through a case work-up we did on a young black fiber-coated male cria that was referred for a fractured femur in winter. The animal was brought to our hospital for surgical repair of the fracture. Prior to surgery, blood was obtained for routine CBC and biochemical profile. Serum was also sent to a reference laboratory for vitamin D concentrations. One week after the fracture was successfully repaired, the cria fractured his opposite femur and was euthanized. Vitamin D concentrations became available later and serum vitamin D was 14 nmol/L and considered deficient compared to

reference intervals (Reference: 50–150 nmol/L). The usefulness of this story is not the surgical repair; rather, about forgetting to provide appropriate medications to animals to prevent these injuries.

Historic information about the farm, management practices and potential exposures to drugs (dates, drug, and dose), chemicals as well as the nutritional program during gestation are also important. Using medications within the first period of pregnancy (when developmental changes occur rapidly) may lead to injury and result in loss of the cria or birth of a deformed cria.

Monitoring pastures for the presence of weeds, shrubs, potential access to flowers is prudent in many areas due to the potential for intoxication. Clean-up of on-site dumps (batteries, treated wood, ashes, etc.) to prevent exposure of animals to noxious plants or substances present on the farm.

Finally, record keeping should be used to monitor problems in the breeding herd. Productivity of individual animals can be measured in many ways. One measure of the productivity of the dam and cria involves serum IgG measured in cria 48 hours post birthing (efficiency of transfer of maternal immunity to cria). On one farm, 14 dams (out of ~500 alpacas), had cria whose serum IgG was consistently below 1000 mg/dL over 5 consecutive years. We found no correlation between cria IgG and the sire used in each breeding. Removal of these dams improved cria health for this farm. It was not possible to determine whether limited IgG production by the dam or absorption of IgG by the cria was involved. This farm had very good husbandry and ensured cria nursed within 2–3 hours of birth.

Another example involved breeding to a highly regarded male alpaca. This male was desired for superior confirmation and fleece quality. After several years of breeding, the author was consulted because the cria born to a wide variety of dams (sired by the male in question) began to have problems with chronic parasitism. This suggested a potential for heritability, especially when the female offspring of this sire began to produce cria (male and female) that had chronic parasitism issues. Pasture management on this farm included mowing pastures very short to enhance the appearance of the property, which confounds the problem by increasing exposure to trichostrongyle L3 as well as monthly deworming of alpacas to prevent aberrant migration of *Parelaphostrongylus tenuis* (this farm had never experienced a case of *P. tenuis*). Developing knowledge of a farm's animal management and use could produce information suggesting hereditary or other relationships. In the case of animals born deformed or dying unexpectedly, the morphologic changes present within the animal can be very useful to make a diagnosis.

For the practitioner, knowledge of documented or potential defects is important. While there are a limited number of mutations identified, curated online resources such as OMIA – Online Mendelian inheritance in animals (OMIA) is available through the University of Sydney, Australia [23]. After searching by species, llamas have 18 identified conditions, alpacas have 11 and there are nine such conditions provided for camels. This is not all encompassing but is instructive in terms of those defects commonly observed in these species.

For example, the genetically determined characteristics of llama fiber was studied to evaluate why some llamas have short fiber length and others longer [24]. Deleterious mutations of fibroblast growth factor 5 (FGF5) is shown to be associated with long fiber length in llamas. This is in comparison to that found in ancestral animals (wild guanaco), where they express the full length, 3-exon gene. Ancestral guanaco have short hair length from 1 to 5 cm, whereas modern South American llamas have much longer fibers (up to 15 cm). Selection for fiber production has resulted in selection of a mutation that controls fiber growth rate [24]. This polymorphism (complex rearrangement) with single-base deletion, 12-bp insertion and a nonsense (stop-gain, both in the FGF5 gene) is designated OMIA 000439-9844.

Teratogenesis

Etiology

The causes of congenital/hereditary defects can be summed up as: environmental (toxic, viral, chemical (drugs, toxins, minerals) and unknown. A large portion of the defects discussed remain unknown and likely represents some exposure of the fetus or a genetic disorder which has not yet been decoded. Realistically, congenital conditions are common and likely encompass all or most of the diseases/disorders described in other species. Viral agents associated with congenital defects are common in livestock species. While a number of reports indicate evidence of infection with disease or antibody production in animals (exposure to viral agents) against these pathogens, in camelids, few have been associated with congenital disease at present. Toxic causes, especially plant toxins are not uncommon in livestock and deleterious defects or death have been documented in Old world camelids (OWCs). Thus, it is likely that they also occur in South American camelids (SACs) (see Chapter 22). At least in livestock, toxin exposure is more commonly observed than genetic defects. Building differential lists by comparing livestock species lesions to those observed in camelids may benefit this effort. This is

especially true when evaluation of defects in young animals sired by one male suggests hereditary causes.

Some general principles for the disease and disorders provided in this chapter to consider:

1) The degree of susceptibility to the effects of a teratogen is determined by the animal's genotype, dose of toxin/virus, other initiators, age of animal (time of exposure), and nutritional status of the animal.
2) The teratogen must be able to traverse layers between the environment, gut and fetal membranes in order to alter development (cause defects).
3) The time of exposure (relative to pregnancy) is associated with specific defects.
4) The mechanism of action of teratogens dictate if a lesion occurs and when it occurs. Different teratogens may produce the same or similar lesions in multiple animal species.

Poisonous plant ingestion by a pregnant camelid is an ever-present hazard to the fetus [24, 25]. Camelids are fastidious in their eating habits, rarely consuming large amounts of strange plants, but they do investigate and try new plants. A low-dose intake may be a saving factor in camelids. Table 21.2 lists plants known to produce teratogenic defects in livestock.

Hereditary Traits

In spite of the publication of the alpaca genome and its updates, few definitive genetic studies have been conducted. Coat-color determination is currently one of the few mutations that have developed genetic markers for camelids [27, 28].

Chromosomal Aberration

A number of chromosomal abnormalities have been reported in domestic animals. These defects occur during meiosis and include fusion of chromosomes, translocations of segments of chromosomes, loss of a segment, among other alterations [29, 30]. The expression of a defect is determined by whether or not the change occurred on an autosome or a sex chromosome. Some chromosomal aberrations can affect the individual or can be perpetuated as an inherited trait. Identification of interspecific somatic cell hybrids, and cytogenetic studies, including karyotyping appear useful in determination of the mutation cause. Recent studies document the usefulness of these techniques [31–33].

Though most of the congenital defects reported in camelids are known to be inherited in one or more species of other domestic animals or humans, it is important to recognize that many may also be produced by other etiologic agents. In fact, such defects as arthrogryposis are more likely to be caused by exposure of the dam to a toxin or virus at a crucial time during gestation than by genetic damage (Table 21.2). At present, no congenital defects are known to be caused by viral agents.

Congenital Conditions

Table 21.1 lists more than 100 congenital conditions of SACs that have been identified by the previous author [34, 35],

Table 21.2 Poisonous plants with teratogenic effects in livestock.

Plant name Scientific	Common	Species affected	Stage of gestation, days	Type of defect
Astragalus spp. *Oxytropis* spp.	Locoweed	Cattle, sheep	1–100	Flexure of carpus, abortion
Conium maculatum	Poison hemlock	Cattle	40–70	Arthrogryposis
Datura stramonium	Jimson weed	Swine	60–120	Arthrogryposis
Leucaena leucocephala	Koa haole, lead tree	Swine, rats		Fetal resorption, polypodia
Lupinus spp.	Lupine, blue bonnet	Cattle	40–70	Arthrogryposis, scoliosis, cleft palate
Nicotiana tobaccum	Tobacco	Cattle, Swine, rats		Twisted limbs, dorsal flexure of hind digits, cleft palate
Sorghum vulgare	Sudan grass	Horses		Carpal ankylosis
Trachymene cyanantha	Wild parsnip	Sheep		Crooked legs
Veratrum californicum	False hellebore, corn lily	Sheep, SACs	Sheep: Day 14–16 Camelids: Unknown	Cyclopia

reported in the literature [11, 36–43], or reported by personal communications. The reader is reminded that these conditions may be hereditary; however, they could be caused by non-hereditary causes.

Skeletal Defects

Angular Limb Deformities

Angular limb deformities may be associated with incomplete ossification, uterine malposition, nutritional deficiency/excess or ingestion of toxicants during pregnancy and a more recent discovery associated with *in utero* limb fractures [44]. Postnatally, angular limb deformities may be associated with traumatic injury during bone growth or vitamin D, calcium, phosphorous, copper, zinc or molybdenum deficiencies [44]. Carpal valgus appears to be most commonly observed in llamas and alpacas; however, abnormal angulation of the more distal joints may also be observed. (Figure 21.1) Other potential causes of carpal valgus may include some form of trauma, or heritable traits (such as chondrodysplasia in sheep) [44].

It is also possible that angular limb deformities are associated with rickets in crias. However, mechanisms of limb deformities in rickets differ from those observed in carpal valgus. In carpal valgus, the prominent sign is inward bowing of one or both carpi (Figure 21.2). If the valgus is allowed to progress, the deformity will worsen and promote the development of joint diseases. Varus (inward deviation of the distal aspect of the limb) of the carpi is uncommon (Figure 21.3). Bowing of the fetlock has also been seen in camelids (Figure 21.4).

Figure 21.1 An aged adult llama with a congenital defect of the left rear metatarso-phalangeal joint of life-long duration.

Carpal valgus may be present at birth, but an evaluation and/or therapeutic intervention should be delayed for one month to allow the cria to develop strength and balance. The deviation may develop at two to six months of age and

Figure 21.2 Llama cria with carpal valgus. Valgus is defined as an outward deviation of the distal aspect of the limb.

Figure 21.3 Carpal varus (inward deviation of the distal limb) of a llama is pictured here. Varus deformities is much less common than valgus.

Figure 21.4 Angular (valgus) deformity of fetlock.

progressively become more severe, up to fifteen months of age. The severity of ALD is often described by the radiographically measured angles or more subjectively as mild, moderate or severe (corresponding to magnitude of the joint angulation) (see Chapter 5 for more information on radiographic imaging of angular limb deformities). Angulation of >5–7° in a young animal can be prone to development of DJD or other problems associated with the animals attempts to stabilize the joints [44]. The pathogenesis of carpal valgus is proposed to be associated with cessation of growth at the ulnar physis on the lateral aspect of the limb, allowing continued growth of the medial radial physis. There may be other causes involved in the development of this defect.

It is also commonly observed that a newborn cria or calf can have very angular limbs at birth. Thus, it is prudent to allow time for muscle and bone to become more stable prior to embarking on a surgical repair. In severe cases, splints, heavy bandages (Robert-Jones) or casts may help, though adding additional weight to the limbs complicates ambulation of the neonate. Don't make a diagnosis of congenital crooked legs on newborn camelids until 30 days post-delivery. Frequently, newborn ungulates have ligament and tendon laxity that corrects itself after a few days outside the uterus. Figure 21.5a and b show a newborn camel that, by 30 days, had normal camel conformation.

Lesions of metabolic bone disease are rarely observed in SACs but can be seen in camels. Inflammation of the radial physis, characterized by flaring and widening of the physis, may be caused by trauma though can often be explained by rapid growth associated with nutritional (energy, protein, macro- or micromineral) imbalances.

Surgical management of angular limb deformity is discussed in Chapter 10.

(a)

(b)

Figure 21.5 (a and b) Two examples of crooked legs on a newborn camel.

Arthrogryposis

Arthrogryposis is defined as permanent joint contractures resulting in ankylosis of the limbs. It is often accompanied with cleft palate, kyphosis or scoliosis. In the cria, it may be accompanied by polydactyly, tendon contractures and choanal atresia. The bones and joints are often malformed, and difficulty identifying the bones due to their abnormal shape and size is common. Arthrogryposis can involve a single limb (Figure 21.6), several limbs, be uni- or bilateral, with single or multiple joints involved (Figure 21.7).

The cause of arthrogryposis in camelids is unknown. Teratogens are frequently involved in livestock (Table 21.2). At birth, crias are unable to stand, it is not possible to straighten the limb and bizarre changes (aberrant direction, rotational changes) in the limbs can be prominent.

Overextension of the Carpus

Overextension of the carpus (calf-knee) may be a serious defect in racing camels (Figure 21.8). Like other species, this defect may be caused by poor conformation, which could be inherited.

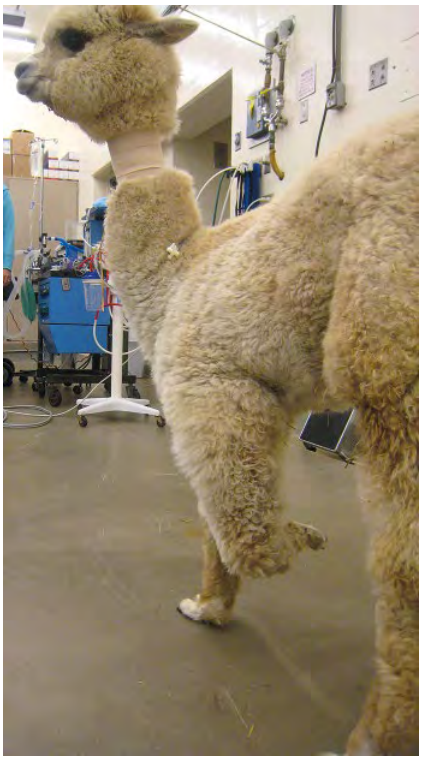

Figure 21.6 Yearling cria presented for evaluation of congenital fusion of left carpus with deviation of the metacarpal bones medial and cranially.

Shortening of Long Bones

Unilateral shortening of the femur and metacarpus has been reported as a congenital condition in camelids. The cause is unknown.

Figure 21.7 Cria born alive with multiple congenital contractures, bone rotations, with cleft palate and VSD.

Figure 21.8 Camel with over extension of the carpus (calf knee).

Luxation of the Patella

Luxation of the patella occurs in llamas, alpacas and camels. It is often present at birth as a congenital lesion. It may, however, develop later in life (as an adult). These animals often have significant degenerative joint disease. The luxations occur laterally or medially. Crias with patellar luxation (medial or lateral) have a characteristic inability to straighten the femorotibial and tibio-tarsal joints (Figure 21.9). Medial or lateral palpation of the limb should reveal the location of the patella outside of the trochlear grooves. Diagnostic imaging (lateral, cranial-caudal, and the skyline view) will demonstrate the position of the patella and ligament as well as document whether formation of trochlear groove is normal (deeper rather than shallow). In young animals, teratogens or heritability have not been diagnosed as a cause, but it would be prudent to consider these as potential causes.

Upward fixation of the patella is an acquired condition in older llamas (Chapter 10). A predisposition may be a congenital conformational weakness (straight rear limbs and laxity of the tendons and ligaments, shallow trochlear grooves).

Hemivertebrae

Hemivertebra [45] refer to lack of formation of a complete vertebral body. Depending upon which vertebral body is misshapen, it may be associated with few consequences or be associated with acute paresis or paralysis due to slipping of the vertebrae and spinal cord trauma. This is especially true when the cria begins to become active and more independent. Accidental injury may result in catastrophic failure and permanent paralysis. In humans, the genetics are complex including shortening of the limbs, hemivertebrae, genital hypoplasia and fetal facies [46–48]. Figure 21.10 demonstrates a lesion involving traumatic luxation of C7 and T1. The cria was turned out with other dams and their crias and was found down and unable to put weight on any limb. Figure 21.11 demonstrates defects in C1–C2 that produced a cria with abnormal head carriage due to the inability to move his head and neck normally.

Malformation of the spinal cord is often associated with spina bifida, which has been reported in a llama.[1] Hemivertebra of the fourth cervical (C4) vertebra has been diagnosed in association with subluxation of the cervical vertebrae and spinal cord trauma in a juvenile alpaca. Hemivertebrae of the coccygeal vertebrae is one cause of crooked tails.

Clinical signs included ataxia and falling when attempting to avoid capture for restraint. The ataxia was exacerbated by restraint, particularly if the head and neck were manipulated. The body of C7 was only partially formed as observed radiographically, confirming the diagnosis of hemivertebrae. There was a marked subluxation between C6 and C7 (Figure 21.12). Stenosis of the neural canal was confirmed by contrast myelography administered via the lumbosacral space.

Figure 21.9 Bilateral medial patellar luxation in a llama neonate.

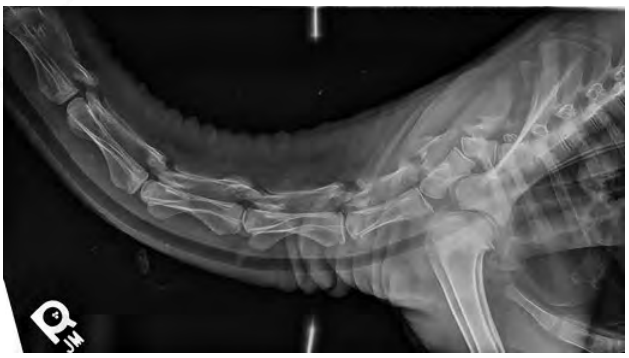

Figure 21.10 Lesion involving traumatic luxation of C7 and T1. The cria was turned out with other dams and their crias and was found down and unable to put weight on any limb.

Figure 21.11 Dorsoventral radiograph view of cria with C1–C2 malformation.

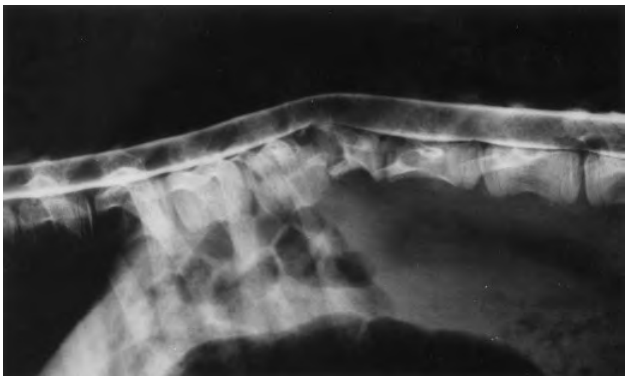

Figure 21.12 Lateral radiograph of hemivertebra of the first lumbar vertebra in a llama neonate. Spinal cord compression is noted by contrast myelography. (*Source*: Photo courtesy of Dr. Richard Cambre, Denver).

Polydactyly/Syndactyly

Polydactylism is a common dysostoses of camelids [49, 50]. These abnormalities occur singly or in combination of multiple extra digits. In animals, they are most commonly associated with the extremities where one to three accessory digits may occur on one or all four limbs (Figure 21.13a and b). Though not necessary to make a diagnosis, radiographs can be used to visualize boney abnormalities associated with this condition and is useful if surgical removal of the extra digits is to be performed. (Figure 21.14a and b)

Fusion of two normal digits (syndactyly) is less common (Figure 21.15a and b). Both conditions have been identified as inherited traits in cattle, dogs, and humans. The conditions are evident on clinical exam. The degree of development of the accessory digits varies, but it may be complete, with a full complement of tendons, ligaments, and bones, including metacarpals and metatarsals. In humans, polydactylism is often associated with other congenital anomalies. In some human studies, involvement of sonic hedgehog (SHH) along with Patched 1, smoothened, GLI (GLI 1/2/3) are involved in digit number and identity. Mutations or toxicants that interact with these factors regulating embryogenesis disrupt growth regulation in a manner that produces these defects [51]. Genetics of this trait are not known for camelids, but Figure 21.16a and b illustrate the trait in a dam and her fetus, suggesting heritability. Some native pastoralists in Peru believe it to be good luck to have a polydactyl animal. Recommendations for the removal of accessory digits to allow them to be used as pets has been recommended. This author suggests that accessory digits not be removed (unless necessary) as a reminder that they should not be bred.

Scoliosis [45, 52, 53]

Various forms of curvature of the spinal column occur as isolated incidents (Figures 21.17 and 21.18a and b). The etiology is unknown. Wry neck may be trauma induced from overzealous traction during dystocia. In true congenital scoliosis, changes that indicate permanent distortion of the vertebrae may be seen on radiography. Kyphosis is also seen (Figure 21.19).

Luxation of the Tibiotarsal Bone

Bilateral luxation of the tibiotarsal bone has been diagnosed in a number of SACs, including a seven-month-old female llama. This condition in SACs may be congenital or acquired via trauma.

In a SAC suffering from luxation, the tibiotarsal bone lies in a horizontal rather than vertical position, and the articular grooves of the tibia articulate with the caudal aspect of the tibiotarsal bone rather than the trochlea (Figure 21.20). Externally, the cranial surface of the tarsus bulges where

(a)

(b)

Figure 21.13 (a) Dorsal view of a cria foot with polydactyly. (b) Ventral view of a cria foot with polydactyly.

(a)

(b)

Figure 21.14 (a) Aged female alpaca with three claws on each digit. (b) Radiograph of a one year old alpaca digit demonstrating four digits on one claw.

(a)

(b)

Figure 21.15 (a and b) Two examples of syndactyly.

Figure 21.16 (a) Polydactylae in llama mother. (b) Polydactylae in a fetus from the previous mother.

Figure 21.17 Camelid spine from a camelid with scoliosis.

the tibiotarsal bone projects forward. Movement of the tarsus is restricted, causing a mechanical lameness.

The condition in llamas is similar to congenital convex pes valgus (congenital vertical talus, congenital flat foot, teratologic dislocation of the talonavicular joint) in human infants [54]. The etiology in human infants is unknown, but it is thought to be genetic. Peculiar fetal positioning within the uterus has also been considered. Surgical realignment and pinning of the tibiotarsus may correct the defect in human infants but has not been performed in camelids.

Face and Head Defects

Craniofacial Dysgenesis

A number of congenital defects of the face, nasal cavity, and pharynx (Table 21.1) may be lethal because of the obligate nasal breathing of the cria. The precise relationship between the various defects is unknown. The embryologic development of the nasopharyngeal region is complex, yet it seems logical that these conditions may be the result of interference with development at a specific time during *in utero* development.

The least evident defect may be stenosis of the nasal passages. Contrast radiography of the upper airway can be used to define stenosis or choanal atresia. (Figure 21.21) Choanal atresia is common and may consist of a membranous or osseous partition between the nasal and pharyngeal cavities (Figures 21.22 and 21.23) [40, 55, 56]. Agenesis of the facial bones causes variable shortening of the face and muzzle (Figures 21.24–21.26) and accentuates the doming of the forehead that may be mistaken as a hydrocephalic condition [14, 36, 57]. The nares may be totally occluded (Figure 21.27a and b). In extreme cases, agenesis of the facial bones, along with other tissue dysgenesis, may result in cyclopia (Figure 21.28).

The etiology of camelid facial dysgenesis is unknown. A familial relationship is known in humans with choanal atresia [58]. In animals, teratogens are known to produce similar congenital defects in sheep. Cyclopia has also been documented in alpacas. This condition is not inherited, rather due to consumption of plants containing a toxicant called 11-deoxojervine (cylcopamine). The ingestion of the toxicant early in gestation results in cyclopia in lambs (when consumed by pregnant ewes on day 14 of gestation), limb defects in lambs (when consumed by ewes on days 28–31 of gestation), and tracheal stenosis in lambs (when consumed by ewes on days 31–33 of gestation) [59]. The defects are somewhat variable but include the presence of one eye, reduced/absent maxilla, tongue protrusion, and a

Figure 21.18 (a) Scoliosis of thoracic vertebrae in a llama cria. (b) Scoliosis of the cervical region.

vestigial proboscis on the forehead. The toxin (cyclopamine) targets SHH (sonic hedgehog) and several downstream regulators (Patched, Smoothened, Gli3) that play important roles in regulating the development limbs, skin, teeth, eye, lung, and central nervous system (CNS) as well as differentiation of sperm and cartilage [59]. In elegantly designed studies, it was shown that cyclopamine inhibited or disrupted SHH signaling mechanisms associated with patterning of the neural tube [59]. In four neonatal alpaca cria, cyclopia was evident. (Figure 21.28) In each of these four cases (one each from PA, KY, two from OH), growth of Veratrum is known to be widespread in some areas of these states (see Chapter 22). Based upon USDA NRCS mapping, *Veratrum virginicum*, *Veratrum viride* and *Veratrum woodii* are native in all of the states where the cyclopian cria originated from (of interest, one prior case reported by a veterinarian in Alaska is also known to have native *V. viride* present), It is possible that the dams consumed these plants early in gestation resulting in the cyclopia. The actual day of gestation in alpacas has not been determined.

Clinical signs vary with the nature of the defect. All produce some impairment of respiration. Complete occlusion of the nasal passageways causes a characteristic breathing pattern in the cria. On inspiration, the mouth is opened slightly and filled with air. Then the lips are slightly closed, while air continues to be sucked into the mouth, ballooning the cheeks. The lips close tightly, and the cheeks compress to force air around the elongated soft palate, which is positioned ventral to the epiglottal cartilage. With expiration, air is forced out of the larynx into the oropharynx, and complete exhalation requires further effort to push the air around the soft palate and out of the mouth.

An affected cria stands with the head extended, because this position restricts air flow the least. Also, from the extended head position, it may be possible for the soft palate to flip dorsal to the epiglottal cartilage, allowing free flow of air into the trachea and more normal breathing.

Restricted expiratory airflow entraps excessive air in the pharynx, which may, in turn, be swallowed, causing tympanites. Affected crias not only have difficulty breathing but find it almost impossible to nurse. The time required to obtain sufficient oxygen to sustain life precludes time for swallowing milk. Aspiration pneumonia is a common sequel. Affected crias are known to chew at and ingest fiber from the mother. Numerous hairballs have been observed in compartment 1 of the stomach at necropsy.

Nasopharyngeal obstruction may be partial or complete and uni- or bilateral. Some affected animals are not detected as neonates but have respiratory deficiencies as adults. Such conditions are difficult to differentiate from acquired chronic respiratory diseases.

Figure 21.19 Kyphosis in a mature alpaca.

Figure 21.20 Lateral radiograph of tibiotarsal bone luxation.

Definitive diagnosis of these conditions may require radiographs. In a neonate with suspected choanal atresia, a 5-mm catheter should be inserted intranasally. The head should be maintained in an elevated position to prevent back flow of the medium from the nostril; 10 mL of an iodinated contrast medium (hypaque) should be deposited into the nasal cavity, followed by immediate exposure (Figure 21.21). Both nasal cavities should be evaluated.

The prognosis for the life of a cria with bony choanal atresia is unfavorable. When the atresia is associated with failure of the membrane to regress, successful maintenance can be achieved. However, multiple surgeries may be required and the cria often suffers from aspiration pneumonia and potentially from bacteremia (because they cannot drink colostrum due to respiratory embarrassment). Surgery has been performed in an attempt to salvage a cria as a pet, but results have not been favorable [56]. Euthanasia is a humane alternative.

Jaw Dysgenesis [43]

The most common congenital disorder of llamas/alpacas involves malformation of the mandible or maxilla (Figures 21.29–21.34). This is a significant problem within the industry. Overgrowth and protrusion of the incisors are considered by some to be normal, yet the teeth are cut off to improve appearance. Though alpacas have continuously growing incisors, with proper alignment, the teeth are naturally worn off, and overgrowth does not occur. There is little question that the various forms of shortening or elongation of the jaws are hereditary. Genetic transmission is known in other livestock, pets, and humans. Peruvian investigators have reported on genetic transmission in llamas and alpacas [43, 60].

Retention of deciduous incisors is common, and a familial tendency is suspected in camelids, but the precise etiology is unknown (Figure 21.35). Other skull defects are illustrated in Figures 21.36 and 21.37.

Dysgenesis of the Palate

Palatal defects of camelids have involved both the hard and soft palates, including clefting or complete absence (Figure 21.38). Cleft palate does not seem to be a part of the choanal atresia/facial deformity syndrome. The etiology is unknown. Clinical signs may be present at birth, with milk flowing from the nostrils while the cria nurses. With a

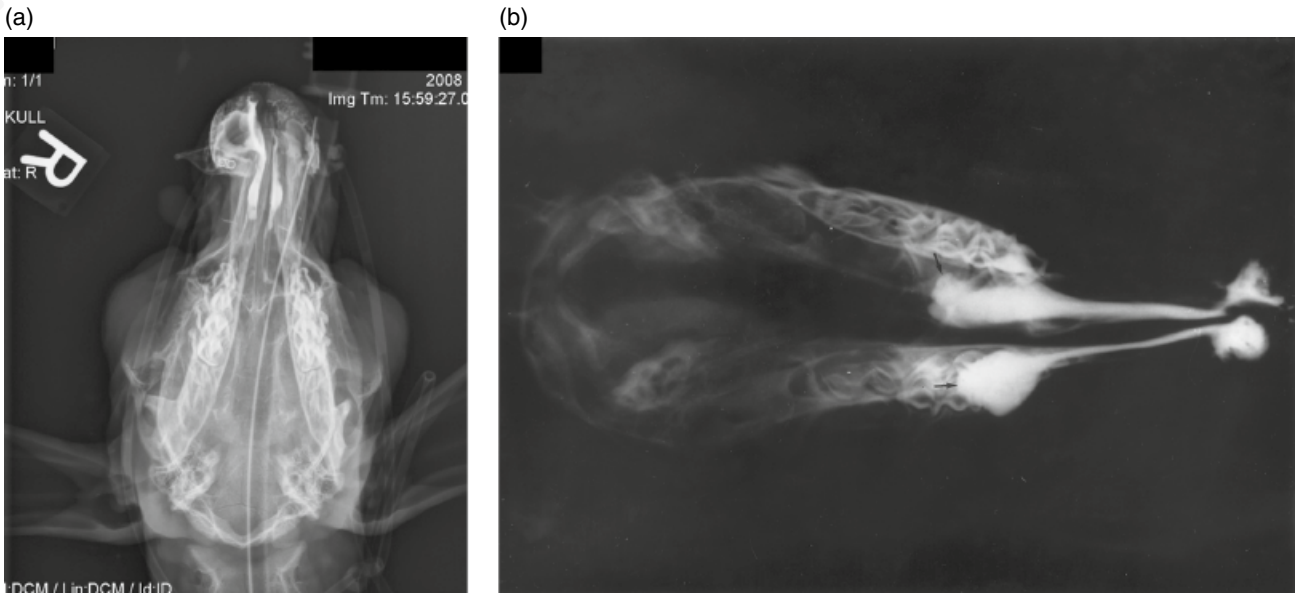

Figure 21.21 (a) Nasal stenosis in a young cria demonstrated by instillation of an iodinated contrast agent in each nares and show reduced diameter of the nasal passages bilaterally about 3–4 cm caudal to the nares. (b) Dorsoventral radiograph of choanal atresia, radiopaque contrast instilled into nostril. Arrows represent the caudal extension of the contrast media. No contrast has entered the caudal aspect of the pharynx, trachea, or esophagus.

Figure 21.22 Flared nostril, open mouth breathing characteristic of choanal atresia.

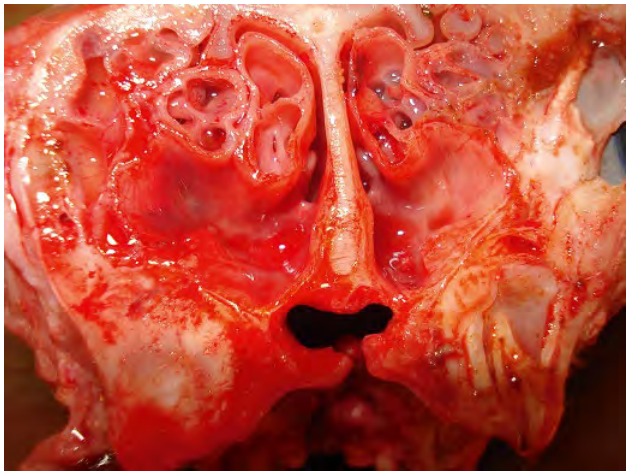

Figure 21.23 Cross section of the caudal aspect of the nasal passages demonstrating the complete obstruction of airflow (bilateral choanal atresia) through the nasal choanae in an alpaca cria. The opening to the pharynx is seen below the nasal septum.

small cleft, signs may not be evident until solid food ingestion begins and food particles enter the nasal cavity, producing a rhinitis (Figure 21.39). A nasal exudate that contains food particles is highly suggestive of a cleft, which may be confirmed by examining the oral cavity. A laryngoscope may aid in visualizing the extent of the defect.

Surgical correction of cleft soft palate has been reported [61]. Surgical repair of a cleft hard palate has been attempted in the past. This procedure is difficult, and the author is confident it has not been totally successful. Feeding management is likely to provide only temporary relief, and euthanasia should be recommended.

Reproductive System Defects

Much has been written about congenital/hereditary defects of the reproductive system of llamas and alpacas. Many of the defects seriously impair or prevent reproductive performance. The prevalence of reproductive defects in both North and South America is alarming. In one study in Peru, as many as 10% of the animals had one or more defects [62]. Veterinarians should be aware of the scope of

Figure 21.24 Craniofacial shortening in a llama cria.

Figure 21.25 Another example of craniofacial shortening in a llama cria.

the problem (Table 21.1) and consider these conditions on any soundness or infertility examination.

Hypogenesis/Agenesis of Reproductive Organs

Failure of development, particularly of female reproductive organs, is often seen. The etiology is unknown in camelids, but genetic transmission has been reported in cattle and humans (Table 21.1). The major clinical sign is

Figure 21.26 Often multiple congenital deformities occur simultaneously in an animal. Polydactylae occurred in this cria that also had craniofacial shortening.

(a)

(b)

Figure 21.27 (a) Choanal atresia with deformed face. (b) Nostril agenesis.

Figure 21.28 Cyclopia is evident in these alpaca crias. The dams in these crias were exposed to Veratrum during gestation.

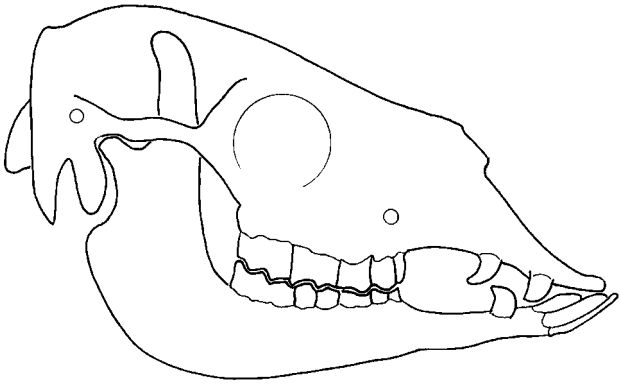

Figure 21.29 Diagram of normal jaw relationships.

Figure 21.31 Mandibular hypogenesis in an alpaca.

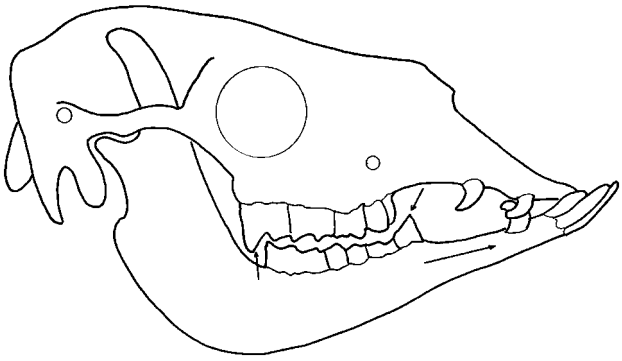

Figure 21.30 Diagram of jaw deformity, elongated mandible.

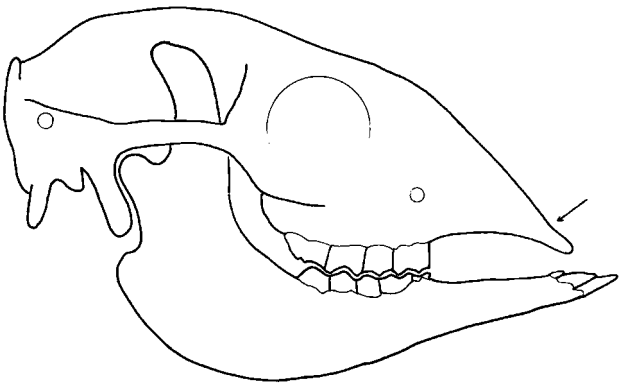

Figure 21.32 Diagram of superior brachygnathism in a neonate, which may prevent nursing.

infertility, though pregnancy is possible in some cases, such as uterus unicornis (Figure 21.40) or double cervix (Figure 21.41) [63].

Segmental agenesis of the tubular genital tract of the female is common. Stenosis or occlusion of the tract may occur at any location from the oviduct to the hymen. Unilateral agenesis of an oviduct may never be detected. Agenesis within the uterus or vagina prevents outflow of uterine secretions, resulting in accumulation of fluid (mucometria) and dilatation of the segments of the tract cranial to the agenesis (Figure 21.42).

The dilated uterus may be mistaken for pregnancy on rectal palpation. The fluid may be milky to slightly reddish in color and have the consistency of skim milk. The fluid is sterile unless organisms have been introduced by diagnostic

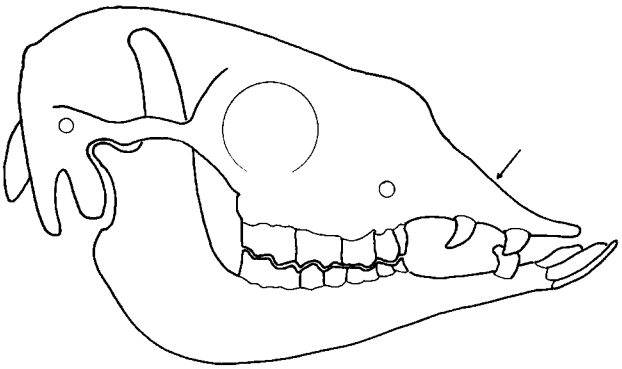

Figure 21.33 Diagram of superior brachygnathism in an adult male llama.

Figure 21.34 Maxillary prognathism in an alpaca.

Figure 21.35 Retained deciduous incisors on labial side.

manipulations. It is difficult to differentiate mucometria from placental fluids on palpation, but it is easy to differentiate it from pyometra on ultrasonography. Exudates caused by pyometra have a flocculent appearance, while mucometria and placental fluids are homogenously clear.

The diagnosis of segmental agenesis involves a combination of rectal palpation, ultrasonography, examination with a vaginal speculum, aspiration of fluid, and visualization at laparotomy or laparoscopy, depending on the location of the lesion. See Chapter 16 for discussion of the normal anatomy of the reproductive system.

Uncorrected tubular agenesis precludes pregnancy except for uterus unicornis and unilateral oviduct agenesis. The veterinarian may be asked to open the tract, especially

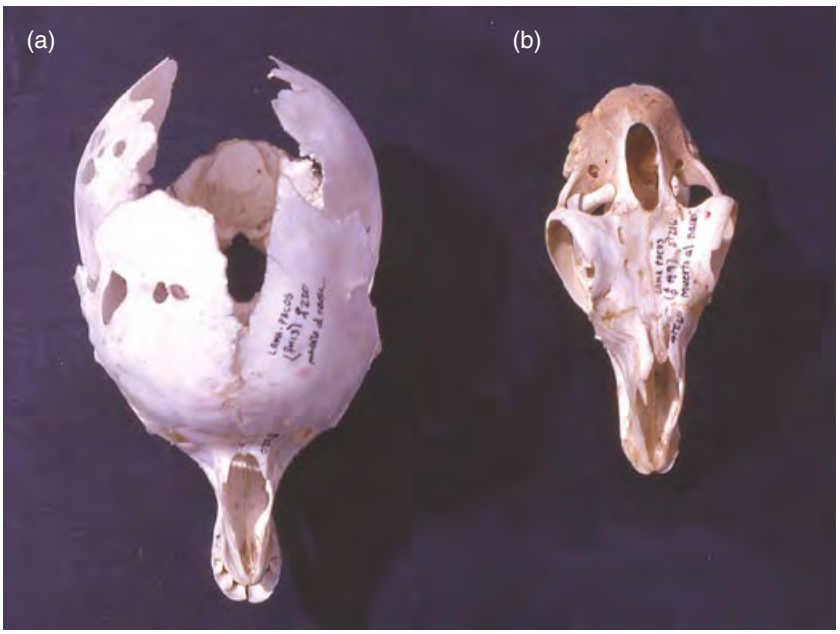

Figure 21.36 Alpaca skull deformities. (a) Hydrocephalus, (b) cerebromeningocele.

Figure 21.37 Skull deformities in alpacas.

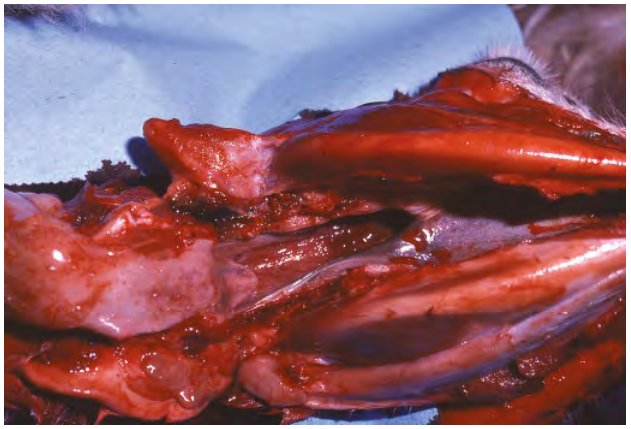

Figure 21.38 Pathologic dissection of a llama neonate with a cleft palate.

Figure 21.39 Nasal exudate containing feed particles, characteristic of cleft palate.

Figure 21.40 Uterus unicornis.

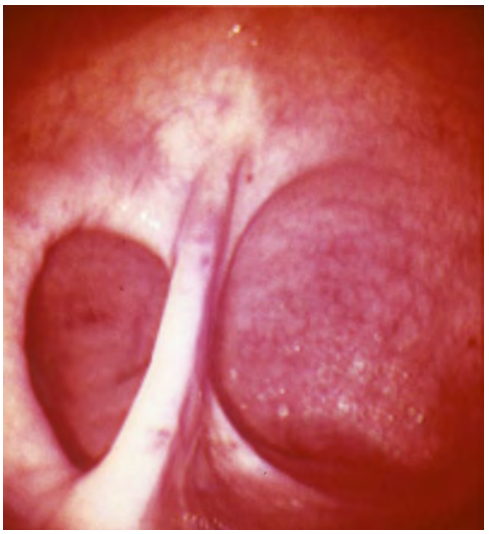

Figure 21.41 Double cervix in a llama.

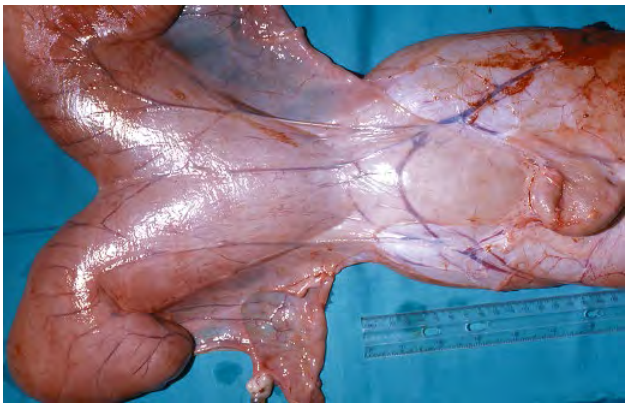

Figure 21.42 Mucometria caused by segmental agenesis of the vagina.

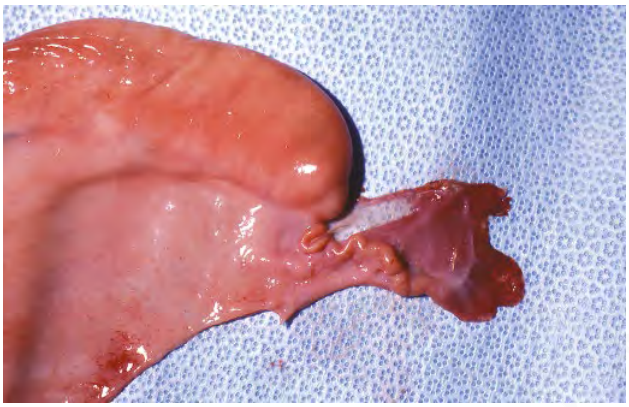

Figure 21.43 Ovarian dysgenesis, hypogenesis.

Figure 21.44 Ovarian agenesis.

if the occlusion is at the level of the hymen or caudal vagina. Such surgery is possible, but the client should be advised of the likelihood that this condition is hereditary and that such an animal should not be used for breeding. Further, depending on the length of time that the mucometria has been present, damage to the uterine mucosa may be irreparable. Ovariohysterectomy may be indicated to

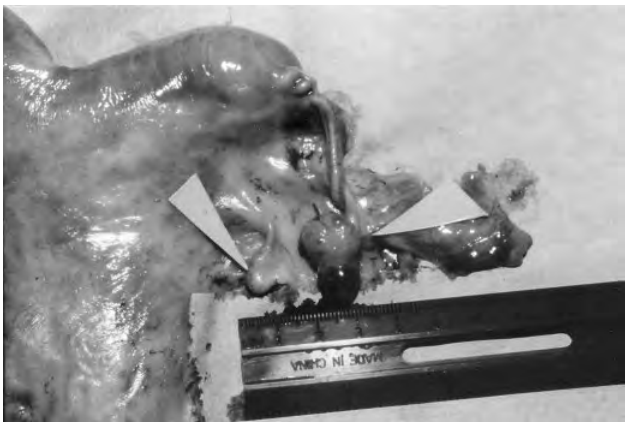

Figure 21.45 Persistent embryonic tissue that may be confused with the ovary on rectal palpation.

allow use of the female for fiber production, as a pet, or as a packer (Chapter 16).

Ovarian hypogenesis and agenesis also occur (Figures 21.43 and 21.44). Persistent embryonic tissue that may be confused with an ovary is illustrated in Figure 21.45.

Intersex

An intersex is any animal in which there is ambiguity in the structure of the gonads, reproductive tract, or external genitalia [64]. Various forms of intersex are known to occur in camelids. Anatomic variations are numerous, and clinical manifestations vary according to the hormone levels involved.

There are four stages of development of the male and female sexual organs: (i) chromosomal sex, as determined by the X and Y chromosomes at the time of fertilization, (ii) gonadal sex, which occurs as the ovary and testis develop from the undifferentiated embryo gonad, (iii) somatic sex, as other sexual organs develop, and (iv) psychic sex, as the animal develops male or female behavior patterns.

A definition of terms follows [65]. A pseudohermaphrodite has the gonads of one sex but an alteration in one or more of the other criteria of sex identification. A true hermaphrodite has both ovarian and testicular tissue, with intermediate external genitalia. A lateral hermaphrodite has an ovary on one side and a testicle on the other. A unilateral hermaphrodite has an ovotestis on one side and either an ovary or a testicle on the other. A bilateral hermaphrodite has ovotestes on both sides.

The embryologic anatomy of genital organs is complex and confusing, but basic understanding is necessary for a consideration of the topic (Table 21.3) [66]. Gonadal development begins with a swelling called the genital ridge, which forms on the Wolffian body in the dorsal abdomen. The testicle and ovary develop from the genital ridge. Initially, the gonad is indifferent, with a medulla and

Table 21.3 Homologies of male and female reproductive systems.

Undifferentiate stage	Male	Female
Internal genitalia		
Gonad	Testis	Ovary
Mesonephric tubules (Wolffian body)	Vas deferens	Epoophoron
	Paradidymus (rudiment)	Paroophoron
Mesonephric duct (Wolffian duct)	Epididymus	Duct part of epoophoron (Gartner's duct)
	Vas deferens	
	Ejaculatory duct	
Mullerian duct	Appendage of testis (rudiment)	Fimbria of oviduct
	Prostatic utricle (uterus masculinus)	Uterus
		Vagina (all or part)
Urogenital sinus	Prostatic, membranous and cavernous urethra	Urethra vestibule, vagina in part
	Bulbourethral glands	Vestibular gland
	Prostate	Paraurethral gland
External genitalia		
Genital tubercle	Glans penis	Glans clitoris
	Corpus penis	Corpus clitoris
Urethral fold	Raphe of scrotum and penis	*Labia minor*
Labioscrotal swelling	Scrotum	Labia majora

cortex. The medulla is the potential male portion, and the cortex the potential female portion. The Wolffian body is attached to the cloaca by the Wolffian duct, which will become the vas deferens and the epididymis in the male.

As sexual differentiation takes place in the female, the Mullerian duct develops, which eventually becomes the uterus, vagina, oviduct, and fimbria of the oviduct [66]. The Wolffian duct disappears, but vestigial structures may persist, which are called the epoophoron, paroophoron, and Gartner's duct.

The etiology of intersex in camelids is unknown, but certain forms are known to be genetically transmitted in all livestock species. Intersexes may also result from hormonal defects. Genital development may fail at any of three stages.

First, distributions of sex chromosomes during meiosis or even mitotic division may result in sex chromosome aneuploidy in gametes. Fragments of sex chromosomes or autosomes bearing sex-influencing genes may be abnormally distributed as a result of partial or complete deletion or translocation in chiasma formation. Second, gonadal morphogenesis may be disturbed because of abnormal corticomedullary relationships on the genital ridge. Third, secondary and accessory genital structures may develop abnormally under the influence of an irregular endocrine environment or as a result of teratogenic factors.

The clinical manifestations of intersex are highly variable. Behavioral patterns may be altered, and the external genitalia may appear as intermediates from expected morphology. A female pseudohermaphrodite llama had an enlarged clitoris, with the cartilaginous projection normally seen on the glans penis (Figure 21.46). Her tubular organs were normal, based on rectal palpation and digital vaginal examination. It was not possible to insert a vaginal speculum past the hymen, but digital palpation indicated a patent vagina cranial to the hymen. The ovaries were not located. This female mounted other females, attempting to force them into recumbency. This is normal male behavior, but it has also been seen in normal females.

The definitive diagnosis of intersex is difficult. The ultimate diagnosis is based on gonadal histology, but other clues include the appearance of external genitalia, morphology of the reproductive ducts, altered karyotype, and hormone analysis. The female pseudohermaphrodite described above had a normal karyotype but significantly altered testosterone levels. The female's estrogen level was 6.2 pg/mL, progesterone 0.1 ng/mL, and testosterone 325 pg/mL. The basal estrogen level of the domestic female mammal is usually less than 10 pg/mL. If there is any follicular activity, the levels are higher than 10. A level of 6.2 pg/mL estrogen in the female llama indicated no

follicular activity. A progesterone level of 0.1 ng/mL is basal. A testosterone level of 325 pg/mL is approximately 10 times the normal level for a female. A cow would have 10–15 pg/mL and a mare 25–40 pg/mL as basal testosterone. The level in this female llama was approximately what would be expected in a cryptorchid stallion (200–500 pg/mL).

The following defects of the female camelid reproductive system have been reported since the previous edition: urethrovaginal fistula [67], transverse vaginal septum [68], and vulvar deformations [69].

Male Defects

Any of a long list of hereditary defects of male livestock species warrants exclusion of the individual from breeding [70]. Congenital defects of male camelids include testicular hypoplasia (Figure 21.47), cryptorchidism [71], testicular cysts, penile hypoplasia (Figure 21.48), persistence of the penile frenulum, and curvature of the penis. It should be recommended to clients that such defects warrant exclusion of males for breeding.

Digestive Tract Defects

Atresia ani [39], atresia coli, and atresia ilei [72] are known to occur in camelids (Figure 21.49). The etiology is unknown. In cattle, atresia coli has been associated with

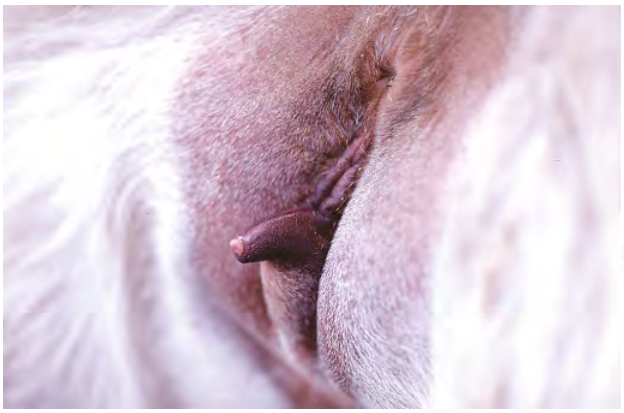

Figure 21.46 Female pseudohermaphrodite. Note the enlarged clitoris with a cartilaginous projection.

Figure 21.48 Penile hypoplasia with incomplete/imperforate urethra on a one-day-old male alpaca demonstrating swelling of the urethra caudally. Surgical drainage and urethrostomy were performed to allow cria to urinate.

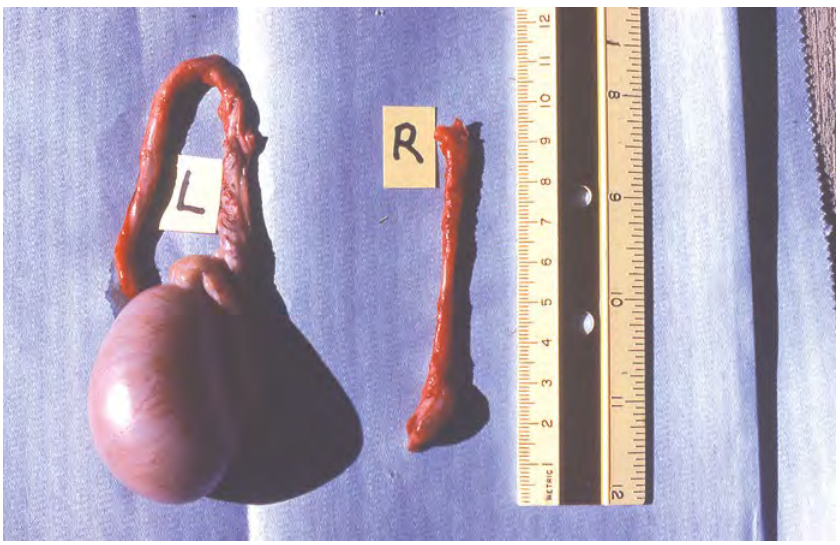

Figure 21.47 Testicular hypogenesis (R), normal (L).

excessive pressure exerted during rectal palpation. Megaesophagus may be either congenital or acquired (Chapter 13). Etiology is unknown. The colon emptied into the bladder of one llama fetus (Figure 21.50).

Figure 21.49 Atresia ani in a llama.

Cardiovascular Defects [12, 73]

Cardiovascular defects are seen sporadically in most animal species. None is unique to camelids (Table 21.1). Ventricular septal defect (VSD) (Figure 21.51a and b) is relatively common in llamas and may occur singly or be associated with other cardiovascular or congenital defects [53, 74, 75].

Clinical signs may be limited to auscultation of a holosystolic murmur or accompanied by cyanosis and exercise intolerance. The author is aware of an adult llama with

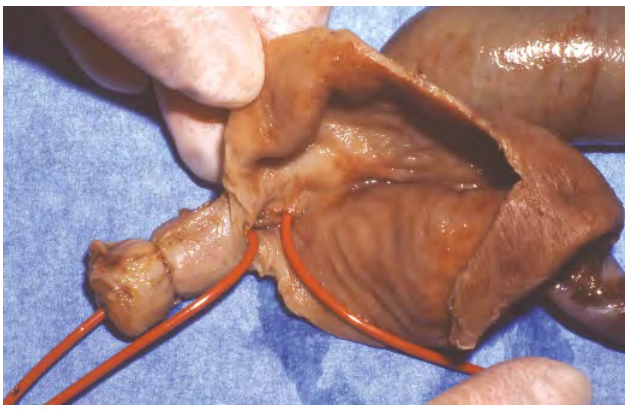

Figure 21.50 Congenital defect with colon emptying into the bladder.

(a)

(b)

Figure 21.51 (a and b) Two examples of ventricular septal defect.

VSD that lives a normal life. The presence of a murmur is a frequent, perhaps normal, clinical finding in the newborn. The murmur should resolve by one week of age. Transposition of the great vessels is rare (Figures 21.52a and b, 21.53) and would be lethal shortly after birth unless accompanied by ventricular or atrial septal defect.

The diagnosis of VSD should be suspected with the presence of a murmur but can only be definitively diagnosed with ultrasonography. Other cardiovascular defects may be diagnosed only though radiography, fluoroscopy, or angiography.

Miscellaneous Defects [15, 20, 76–78]

Many other defects are listed in Table 21.1. Similar defects have been reported in other livestock, pets, and humans. One other defect that is worthy of special mention is crooked toenails. Overgrown nails and crookedness are common. Some nails grow crooked despite judicious and regular nail trimming. Crooked hooves and nails are known to be genetically transmitted in cattle, horses, sheep, and pigs (Figure 21.54). Selection of breeding stock should include a consideration of sound feet and legs. An intrauterine defect caused fracture of the tibia in a fetus (Figure 21.55). Super- and subnumerary teats can occur (Figure 21.56). Wry face is also thought to be a hereditary defect (Figure 21.57a and b). Figure 21.58 shows a dwarf Bactrian camel.

Nonanatomic Defects

A discussion of congenital/hereditary defects would be incomplete without mention of physiologic characteristics that have a genetic basis. These are not known to be inherited

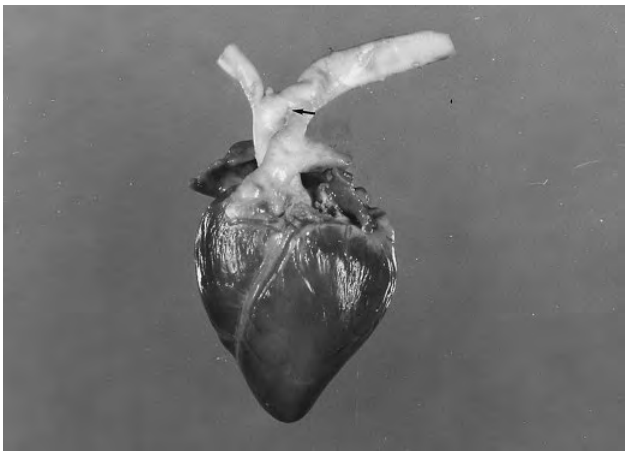

Figure 21.53 Normal crossover of aorta and pulmonary artery in a llama neonate.

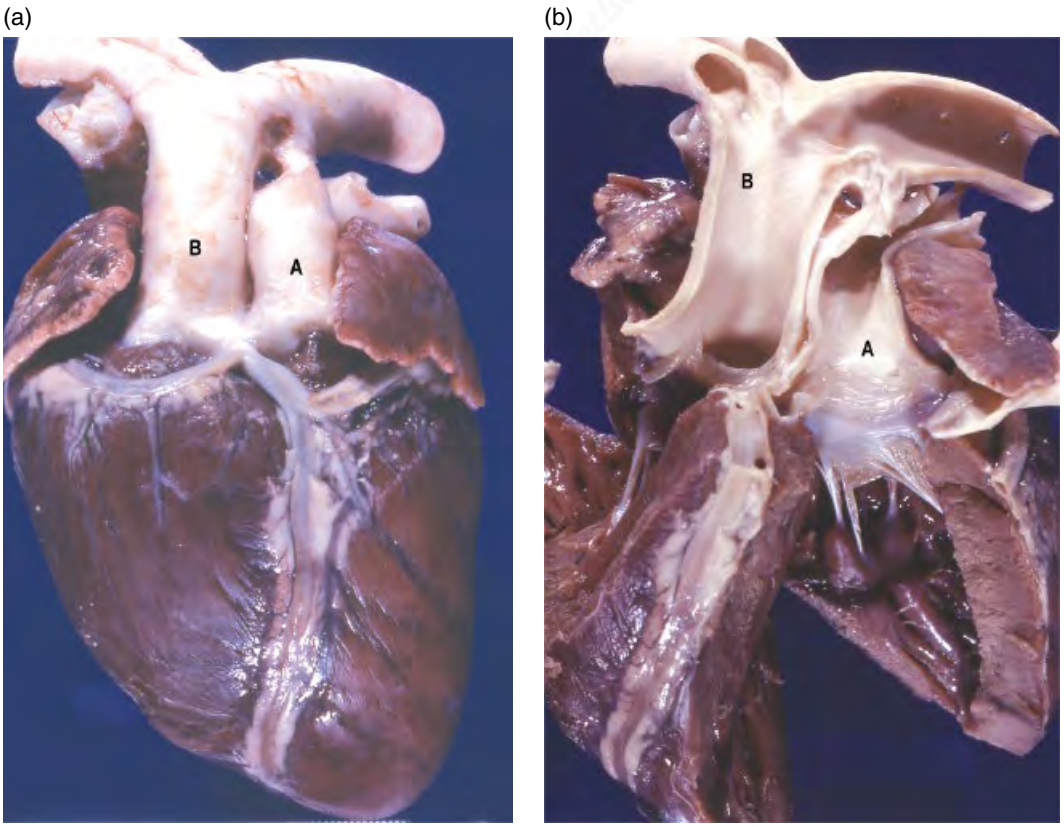

Figure 21.52 (a) Transposition of the aorta and pulmonary artery, closed. (A) Pulmonary artery, (B) aorta. (b) Transposition of the aorta and pulmonary artery, open. (A) Pulmonary artery, (B) aorta.

Figure 21.54 Curled toenails in a llama.

Figure 21.56 Supernumerary teats.

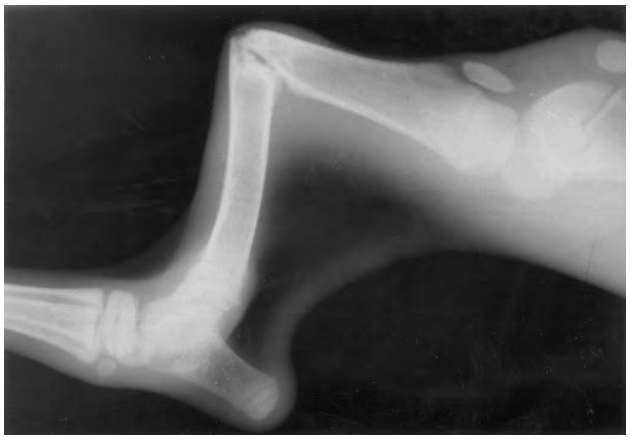

Figure 21.55 Lateral radiograph of the hindlimb of a cria. Severely contracted tendon of the gastrocnemius muscle resulted in a tibial fracture in the fetus.

Figure 21.58 Dwarf camel.

(a)

(b)

Figure 21.57 (a and b) Two examples of a wry face in a llama.

Figure 21.59 A paco-vicuna (alpaca × vicuna) (middle) produced by breeding a male alpaca (left) with a female vicuna (right).

Figure 21.60 Dromedary × Bactrian camel hybrid.

traits in camelids, but they are so important in livestock that mention should be made here. Consider the following: milk production, mothering ability, disease resistance, growth rate, thermoregulatory adaptability, feed utilization, birth weight, semen quality, neonatal mortality, fertility, early embryonic death, and resistance to neoplasia.

New Congenital Defects of SACs

The following congenital defects have been reported since the second edition: glaucoma [79], wry face [80], urethrovaginal fistula, renal agenesis [81, 82], cataracts [76], astrocytoma [83], ocular changes [75], vascular ring anomaly,

Figure 21.61 Stillborn alpaca × camel, Israel.

hepatoblastoma of the liver [84], Factor VIII deficiency of the blood [20], labial fusion of the vulva [85], cystic rete testis [86], vulvar deformities, cranial cruciate ligament aplasia,[2] and caudal vertebral malformations.

Camelid Hybrids

All SACs will inter-breed and produce fertile offspring (Figure 21.59). Likewise, OWCs interbreed (Figure 21.60). Hybrids between OWCs and NWCs are rare but have been produced using artificial insemination. A stillborn fetus resulted from an alpaca and camel cross in Israel, (Figure 21.61). Scientists in the United Arab Emirates have produced live offspring from a guanaco-camel cross (Figure 21.62a, b, and c). It is not yet known whether or not these crosses are fertile.

(a)

(b)

(c)

Figure 21.62 (a) Mother of Rama, a guanaco × camel, United Arab Emirates. (b) Rama the Cama (guanaco x camel hybrid), age ten months. (c) Rama the Cama age six years.

Notes

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22

Toxicology

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Intoxications in camelids have been associated with medicated feeds, plant intoxications, trace minerals, heavy metals, noxious gases, drugs, insecticides, and rodenticides via a variety of exposures. Numerous texts deal with the effects of poisonous substances in domesticated animals [1–7]. This chapter updates those reported agents associated with intoxication of South American (SACs) and Old-World camelids (OWCs). Where possible, we provide mechanism of action (MOA), diagnostics and treatments described in the literature.

Exposure of camelids to toxins, drugs or other noxious agents may occur as observed in cattle, sheep, goats, equines, and other species. While there are few reports of toxicant exposure in camelids compared with other animals, one can assume toxicants causing disease in ruminants can cause the same or similar disease in camelids. For example, consumption of red clover infected with *Rhizoctonia leguminicola* produces similar disease in camelids as seen in livestock. Approaching potential intoxication patients is the same as in any other case. History, clinical exam, supportive care appropriate to the clinical abnormalities and if available, the use of antidotes may be beneficial [8].

Clinical exam of affected and un-affected animals is a critical part of deciphering the cause of morbidity and mortality. Depending upon the clinical signs, their sequence of occurrence and response to therapy may provide information that narrows the list of potential toxicants. Lists of clinical signs observed after intoxications are available for food animals [9]. These can be useful in developing differentials for intoxications and can guide therapy [9]. To protect yourself and your staff, decontamination should be conducted using appropriate personal protective equipment (PPE), including true respirators if the intoxication involves volatile or other toxic compounds (OP, fentanyl). If there are suitable antidotes, this should be provided.

Necropsy evaluation of dead animals is critical to determine causal factors. While some toxicants may produce little or very subtle lesions (lead poisoning) many toxicant exposures are associated with specific lesions (ionophore toxicosis). Plant intoxications may be determined by examining the contents of the gastric compartments and finding characteristic plant leaves, fruits. When clinical signs and lesions suggest a specific toxicant, chemical analysis of gut content, body fluids or tissues may provide the identity of the potential toxic compounds. Focusing on the history, clinical signs (the range of clinical signs), clinico-pathologic evaluation, gross and microscopic evaluation of lesions present in the animal(s) and identification of a toxicant or the metabolites of the toxicant in animals is the overall goal. In general, these evaluations will take much longer and are usually performed by pathologists and toxicologists.

Adaptation to Toxicants

Prevention of exposure is critical to limiting the intensity of morbidity and mortality in animals. Animals (wild or domesticated) can learn to limit consumption of some toxic plants by avoidance, dilution, degradation, and detoxification [10]. Avoidance is an adaptive skill that must be learned early in life. It becomes less likely for camelids in their native habitat to ingest most toxic plants. However, if the quantity and quality of forage or concentrate provided is inadequate, the animal will seek other feedstuffs, some

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containing toxicants. Little is known about the sensitivity of camelids to most toxicants.

Feed scarcity forces animals to forage and consume those plants present. Failure to provide salt, especially in lactating animals puts them at risk for exposure to agents containing sodium (sodium fluoride containing pole wraps, oil well "produced water" containing saline) [11–13].

Transporting camelids from their native environment to new habitats, without the benefit of knowing which local plants to avoid may result in exposure. Thus, determining whether there are potentially toxic plants, waste materials (ash from treated wood fence posts, batteries, etc.) or other hazards prior to provision of free access to this forage is important. It is useful to provide stored hay, salt, loose mineral and feed initially to reduce their hunger prior to turn out. In general, acutely toxic plants (Water hemlock, Arrow grass, choke cherries) may cause sudden death, whereas other plant toxins causing chronic toxicity (Crotalaria spp., Senecio spp., Amsinckia spp.) may not be clinically evident for weeks to months after placing animals in new grazing areas. As much as 40% of the plants growing on native rangelands may contain secondary plant compounds (toxicants). Harvested hays and prepared feedstuffs (silage, haylage) may also contain plant based or other toxicants.

Animals with a choice of plants, may dilute a toxicant exposure by ingesting only small quantities that fail to reach the threshold level for production of acute toxicity. Animals smell and/or consume plants then will either stop eating due to aversive taste or digestive consequences or continue to eat due to lack of these factors. For camels, grazing or browsing a few plants and moving on to other plants, could dilute potentially toxic compounds in the first compartment. In contrast, their ability to tolerate significant water deprivation may also increase their susceptibility to toxicants (dehydration could lead to concentration of poisons in this compartment). Some ingested toxicants may be degraded within the digestive tract as occurs in ruminants. The multi-compartmented stomach of camelids and ruminants is well designed to degrade toxic compounds, but the animal depends upon crucial gastrointestinal (GI) microorganisms as well as xenobiotic pathways to assist in rendering some toxicants harmless. In contrast, other toxicants are remediated in the adult but cause deformities of the fetus [14].

Once the toxic agent is absorbed from the GI tract, the body must either excrete it unchanged, sequester it, detoxify it by enzymatic pathways, or suffer the ill effects caused by the toxicant. All vertebrates have general detoxification pathways that can deal with many different substances. Some have specialized mechanisms, unique to a given species. Specific mechanisms are not well described in camelids; however, it is likely that much detoxification is carried out by microsomal enzyme activities [15, 16].

Diagnosis of Poisoning

Historical information about the herd, exposure, environment and the individual patient are important. Signalment, health history, medications currently provided to the animals, how the animals are fed, watered, bedded down. Whether or not they are in stalls, dry lot pens or on pasture is also important. Where the supplemental feed is kept, whether a new shipment of feed was received, and any other changes should be collected [17].

Information such as animal movement from one paddock to another or one farm to another could prove useful. Movement of hungry animals into poor-quality pasture of questionable composition can provide opportunities for ingestion of toxic plants. Likewise, weather-related events may produce situations where previously unavailable plant materials are present within an otherwise safe pasture, or down fences allow animals to gain access to new areas. Characterization of the coincidence of illness and receipt of a shipment of feed concentrates could be important in understanding a disease occurrence. Thorough investigation of potential agents present and their use on a farm would prove useful when considering clinical signs of disease observed during the investigation. Further considerations include if an agent is present, if it can produce the observed clinical signs, does the exposure dose correlate with the observed clinical signs [9].

Clinical laboratory analysis may suggest a particular toxicant (elevated rumen pH with increased serum ammonia – urea supplemented feed; marked azotemia – Oak toxicity, anti-freeze containing liquids; hemolysis – copper toxicity, red maple leaf toxicity [18]). Convenient lists of clinical signs associated with intoxication of ruminants are also available [9].

For most intoxicants, there are no specific antidotes available, and clinical signs may represent the effects of multiple agents. Other intoxications are characteristic of specific agents, some of which may have specific antidotes.

Clinical signs associated with known toxicant exposure of camelids are not well defined compared with other species. Further, clinical signs may be representative of multiple disorders, for example, botulism intoxication can look like hypocalcemia. Published clinical reports document clinical information to accompany the known toxicants affecting camelids [18–30].

Prevention of Poisoning

Basic concepts are important in the prevention of poisoning in camelids. The most obvious is elimination of exposure to the toxicant. This may include engineering to prevent access of animals to plants growing in a pasture, up a fence, over a fence or after storms where trees/shrubs, etc. fall into an enclosure. Pastures should be examined for weeds, unidentified plants and periodically re-planted. Anything "dumped" into the pasture should be removed immediately. Most importantly, ensuring the pasture present is to provide space and forage safe for the animals.

Since there are very few medications approved for use in camelids, liabilities on the part of the prescriber may be significant. This includes those camelids that can be consumed by their owner or others. Use of medications with no or little knowledge of residues could result in significant liability for the prescriber.

Also, careful evaluation of purchased forage for weeds or foreign material present in hay. Animal feed supplements (grains, pellets, etc.) should provide for the needs of the animal (energy, protein, and trace/macro-minerals). If possible, reducing the stocking density will reduce competition among animals at feeding time. Lower stocking density should also limit exposure to parasites. Greater numbers of animals competing for feed and shelter will increase stress. Finally, provision of enough quantity and quality of nutrients in the diet to promote healthy populations of GI microflora and micro fauna. High-quality water for animal consumption is also particularly important.

Treatment of Poisoning

The basic principles of treating suspected poisoning cases include removal of the source of toxicant (decontamination; whether topically applied, ingested or parenterally administered) thereby limiting the absorption of toxicant. Of equal or greater importance is to provide appropriate supportive care.

Most toxicants do not have distinct toxidromes making diagnosis difficult and time consuming. Early, during most poisonings, symptomatic and supportive therapy should be instituted. Ensuring a patent airway as well as efficient oxygenation of blood is critical when providing oxygen to patients. Measurement of blood oxygenation (SpO_2 %) is critical in cases where respiration is impaired, either via toxic insult, bloat or pulmonary edema [31]. Hand-held pulse oximeters can be carried in a pocket and many can simultaneously monitor carboxyhemoglobin, methemoglobin, oxygen content and other parameters. Evaluation of the patient for systemic bleeding in cases of rodenticide toxicity and ensuring adequate cardiac output, pulse quality and CRT. Evaluation of cardiac rate and rhythm for arrhythmias and remediating tachy- or bradycardia are also critical to ensure adequate perfusion of vital organ systems. Maintaining hydration and body temperature with fluids and blankets or cooling to control temperature. Evaluation of consciousness and treatment for seizures or central nervous system (CNS) depression is also critical to the outcome of the intoxicated patients [31]. Treatment may also require normalizing acid–base disturbances, replacement of electrolytes, bicarbonate, dextrose and whole blood or blood components can be indicated depending upon the toxicant. Ethylene glycol and methanol intoxications can produce anion-gap metabolic acidosis which can be severe. Also, ethylene glycol is metabolized to oxalic acid leading to renal toxicity [32].

One toxicology textbook suggests "intravenous (IV) fluid therapy is always beneficial." [33] The clinician should recognize that IV fluids administered to camelids require careful monitoring since sick camelids are commonly azotemic from a wide variety of causes. Also, aggressive IV fluid therapy in camelids can result in serious edema formation which can compromise pulmonary and GI function leading to body cavity effusions [34].

The very low glomerular filtration rate (GFR) in the dromedary camel, compared to other species could result in fluid accumulation when fluids are administered rapidly IV (GFR estimated to be 1.33 ± 0.23 mL/min/kg in hydrated; 1.06 ± 0.21 mL/min/Kg after 34 days of water deprivation) [35–37]. Lower production of angiotensin converting enzyme by camelids may be involved in altered clearance of drugs and/or toxicants in camelids via renal vascular dilation. This peculiarity may also be associated with their ability to limit pulmonary hypertension at altitude [38, 39].

Dermal exposure to toxicants can be approached by washing. This can dilute the concentration of toxicant and potentially reduce the exposure of an animal to a toxicant. Washing can be difficult in camelids, due to significant coverage of the body by dense fleece. If shorn prior to exposure, dilution of the toxicant may be more effective (depending upon toxin exposure, shearing an animal may also reduce exposure to topically applied agents). Decontamination is important, though agreement upon methods of removing poisons from the animals' body remain controversial. First, for some intoxication's, decontamination may expose human caregivers to these toxicants. Second, controversy over which methods of decontamination employed and their ability to reduce exposure of the patient to toxins are significant.

Consideration of the suspected toxicant should also imply controling environmental contamination from run-off from the intoxicated animal(s), as well as exposure to those individuals involved in the decontamination process [31].

Emetics are inappropriate for use in horses and ruminants and presumably camelids [40]. Emetic administration does not reduce the patient's exposure to a toxin. This is likely to be more significant when considering animals that possess complex forestomachs. By the time the emetic is administered, it is unlikely that enough of the ingesta can be removed from the patient to lower the exposure, if it worked appropriately. The use of emetics is not safe in patients with depressed mentation. This often leads to major complications such as aspiration pneumonia. Practically speaking, the volume of ingesta in compartment 1 (C1) of the camelid cannot be effectively removed by emetics or gavage, even when the animal is anesthetized and intubated. Surgical approaches to the first compartment are like that in ruminants (rumenotomy), allowing one to physically remove contaminated ingesta, replacing it with normal flora and feed material that may help in more chronic cases (lead poisoning), but for most acute toxicants, exposure of the veterinarian and staff to toxins during the procedure may result in serious concerns. Similar comments could be made for gastric lavage and the inability to remove all contents from the first compartment [31].

Activated charcoal may be one of the more useful means of binding toxins which range in size from >100 Da or <1000 Da. This approach may be more successful for drugs and some plant toxins [31]. Use of this absorbent media is contraindicated if an irritant toxin is present, the toxin is a petroleum distillate or in dehydrated patients [31]. Activated charcoal is capable of adsorbing ingested toxicants after administration by orogastric intubation (1–2 g/kg body weight in water; 1-g activated charcoal/3–5 mL water). If multiple doses of activated charcoal are used, reducing additional doses by half may provide removal of additional drug in those cases where enterohepatic recycling are common (ibuprofen, digoxin, loperamide, diltiazem, doxyrubicin) [8].

Catharsis in camelids can be produced by administration of magnesium oxide or magnesium sulfate dissolved in water (0.25 g/kg body weight; 17 g/150 lbs. body weight diluted in water). Mineral oil can also be used to hasten the passage of gut contents and could decrease the concentration of lipid soluble compounds absorbed into the animal (100–250 mL by stomach tube in an adult alpaca). Careful placement of an orogastric tube is important to prevent serious lipoid pneumonia. Administration of a mixture of mineral oil and activated charcoal simultaneously is not advised.

For some drugs/toxins, altering the pH of the ingesta in the first compartment may reduce the absorption of toxins by increasing their polarity for those toxins that are weak acids or bases (acidifying the rumen of an animal with urea toxicity) [41]. Use of IV fluids while adjusting urine pH may also promote diuresis that increases urine production while urine pH has changed trapping a toxin and preventing reabsorption. Ammonium chloride and other salts (orally) acidify the urine and sodium bicarbonate given IV alkalinizes the urine [31]. A major caveat to these therapies is effective renal plasma flow and sufficient renal function of the patient as well as consciousness to swallow medications. Some intoxications (i.e. Vitamin D intoxication) are associated with compromised renal function.

Antidotes

Antidotes are substances used to counteract a specific poison or group of poisons. They may also bind toxins limiting absorption, inhibit metabolism of a toxin, compete with a toxin for receptor binding or antagonize the toxic effect or speed metabolism of a toxin [31]. While counteracting the effects of a potentially intoxicated animal, administration of appropriate supportive care is imperative.

Specific Antidotes

Calcium gluconate may increase ionized calcium necessary to support cardiac, CNS or GI function while also being useful to address the effects of dietary oxalates (given orally). Calcium gluconate and calcium chloride may be irritating when given IM/SQ so an IV line must be used for immediate results. Monitoring ionized or total serum calcium should be conducted to prevent bradycardia, myocardial depression and altered mentation [32].

Dextrose may be supportive providing carbohydrates needed to maintain energy production and has proven useful in Xylitol toxicity associated increased insulin release. Sodium nitrite and sodium thiosulfate are critical in cyanide toxicity. Sodium nitrite produces methemoglobinemia and vasodilation. Cyanide bound to cytochrome oxidases is then transferred to the methemoglobin producing cyanmethemoglobin. A variety of endogenous sulfotransferase enzymes then facilitate the reaction of cyanide to thiol producing thiocyantes which are easily eliminated in the urine. Monitoring the blood level of methemoglobin is important. Monitoring methemoglobin is critical to limit to less than 15% when using nitrites. Administration of thiolsulfates and their conversion to thiocyanate is more direct and does not rely on production of methemoglobin. Methylene blue has also been used to treat cyanide toxicity

when it is not known whether cyanide toxicity has been diagnosed [42]. Vitamin B12 (Hydroxycobalamin) detoxifies cyanide by production of cyanocobalamin and has also been used to chelate cyanide [43].

Calcium ethylenediaminetetraacetic acid (EDTA) is very useful for chelation of lead in lead toxicity and perhaps with cadmium and zinc. Excess chelation of calcium may also cause hypocalcemia. For organophosphates, atropine is a very specific treatment; however, it may have side effects in ruminants and horses due to the prolonged residence in the animal.

Animals suspected of the non-clinical phase of copper toxicity can be treated with ammonium- or sodium molybdate (100 mg Molybdenum) orally to reduce the absorption of copper from the GI tract. Sodium sulfate (1 g in feed daily for 30 days) or sodium thiolsulfate (6–20 mg/kg/day for 30 days) will reduce absorption of copper from gut. D-penicillamine at 50 mg/kg/day PO increases copper excretion. Ammonium tetrathiomolybdate (2–7 mg/kg IV or SQ daily for six treatments) diluted to 1 mg/mL in LRS has also be used to increase elimination of copper.[1] In addition to tetrathiolmolybdate, high doses of xylazine (0.77 mg/kg) to Cu intoxicated sheep results in nearly two-fold increase in copper loss to the bile [44]. This dose of xylazine is very high for sheep, though perhaps not too high in camelids.

Pharmacologic antidotes antagonize a toxin at the target receptor. Flumazenil competes with benzodiazepenes at benzodiazepine (BZD) receptors releasing the BZD and interrupting their clinical effects. Chemical antidotes like pralidoxime interfere with organophosphates by binding to the organophosphate (OP) moiety, thereby inactivating it as well as augmenting elimination of the inactive product.

Specific Toxins

Pesticides

Insecticides

More than 1800 pesticide products are listed, while only 35–40 pesticides are currently registered for home use in the USA [45]. Clinical signs of muscarinic action of many OP are salivation, lacrimation, urination, and defecation. Muscarinic activity of OP is also responsible for increased tracheobronchial secretions making respiration difficult. GI atony and miosis also occur. CNS signs include muscle fasciculations, muscle twitching, weakness, tremors, incoordination, dyspnea, paralysis of the respiratory muscles and seizures eventually lead to death.

Few cases of confirmed pesticide intoxication of SACs have been reported. Pearson et al. described a young (1.5 year old) male llama that was treated with Chlorpyrifos (topically) in combination with fenbendazole (doses unknown) [46]. Five days later, the recumbent, anorexic, hypersalivating, miotic, hyperglycemic, and acidotic animal presented to a veterinary hospital. Their exam revealed bradycardia, normal respirations, hypomotility of compartments and diarrhea. Analysis of blood demonstrated hyperglycemia and hypocalcemia. Determination of blood pseudocholinesterase activity demonstrated an ~50% decline in activity compared to healthy llamas. Rapid response to administration of atropine sulfate was observed (bradycardia, hypothermia, and hypocalcemia resolved). Unfortunately, when trying to administer oral liquids, alfalfa meal and starch, the llama regurgitated and ultimately died of aspiration pneumonia. Of note, this study provided insight into the observed changes in pseudocholinesterase activity of chlorpyrifos exposed llamas (topical application of 25 mg/kg), as well as established reference values of healthy, unexposed llamas (n = 29). Chlorpyrifos-exposed llamas (25 mg/kg topically) did not demonstrate clinical signs of intoxication even though pseudocholinesterase activity declined to 38–62% of baseline by five days. The authors suggested that pseudocholinesterase activity may be useful in diagnosis of chlorpyrifos toxicity, however, it likely does not correlate with clinical signs [46].

A second study characterizing megaesophagus in llamas evaluated a five-year-old male llama administered ivermectin, oxytetracycline, and chlropyrifos that became recumbent, anorexic and was observed to hypersalivate. This animal responded well to atropine to reduce salivary losses [47]. At a later re-check exam, the animal had developed renal failure with evidence of normal esophageal function and was euthanized. While there are few reports of intoxications associated with topical or ingested insecticides, one should be careful to keep these agents stored away from animals and their feed.

Administration of atropine sulfate is the standard therapy for OP and carbamate insecticide poisoning. Poisoned animals are resistant to the effects of atropine, so the initial dose should be 0.2–0.4 mg/kg, not the standard dose of 0.04 mg/kg. If possible, half the dose should be given IV and the rest subcutaneously. Pupillary dilatation and GI atony are significant effects and may last for days following atropine therapy. Keeping animals in the shade until pupillary constriction is normal. Pralidoxime (2-PAM) is effective against OP poisoning and is not recommended for carbamate poisoning. The dose is 20 mg/kg. Activated charcoal should be administered orally at 1–3 g/kg if the OP was taken orally. Administration of midazolam or diazepam will help control convulsions. Due to the muscarinic effects of OP, it is necessary to ensure a patent airway is

present. Oxygen insufflation or tracheal intubation may be required to provide positive pressure ventilation.

Rodenticides

Rodenticide toxicity in camelids is also uncommon. There is only one published report of the determination of brodifacoum concentrations in the liver of a naturally exposed llama that presumably died [48]. While no details of the exposure were provided for this intoxication, one would presume these second-generation anti-coagulants may function like those mechanisms in other species. This author has observed contamination of straw bales with rodent blocks in stalls where llamas and alpacas were housed. It was determined that vendors selling the straw baled loose straw left in a barn. If rodents have damaged bales, some will place rodenticide containing blocks to remove them. One could assume that either the camelids did not eat the blocks or that camelids were not susceptible to this toxicant.

Drug Toxicities and Adverse Drug Reactions

With the increased popularity of camelids in North America and their importance in countries around the world, the use of pharmaceutical agents has grown tremendously. While there are studies evaluating the safety, efficacy and uses of these agents in old world and SAC, few, if any are approved for use in these species by regulatory agencies. Thus, it is prudent to provide information regarding toxicity and adverse effects of these agents in camelids.

Prolonged administration of aminoglycoside antibiotics is reported to produce nephrotoxicosis as reported in other large animal species. Gentamicin has produced renal tubular degeneration in llamas and alpacas. The signs of gentamicin poisoning are similar to acorn poisoning, including weight loss, dehydration, and loose stool, along with elevated blood urea nitrogen (BUN) and creatinine.

Camels are very important in many countries for transportation of human's, goods as well as a source of food (meat and milk), and in some countries, use in sporting events [49]. Current estimates suggest there are approximately 35–36 million camels in the world today [50]. Trypanosomiasis is a common disease in many countries within the world (Africa, Europe, Asia, South America). Many camels suffer from infections with Trypanosomiasis (*Trypanosoma evansi, Trypanosoma vivax, Trypanosoma congolense, Trypanosoma brucei brucei*). The infections often result in wasting, infertility and death in many species (cattle, sheep, goats, horses, pigs and camelids). Morbidity and mortality of these diseases result in tremendous socio-economic impacts. Trypanosomiasis is commonly observed in dromedary camels and treatments used have been based upon those used in other species (Cattle, sheep, goats, horses). El Bahri et al. provided dosing and adverse effects of agents commonly used in camelids in Tunisia [36]. Ali also conducted a survey to evaluate the drugs used commonly in camels and the toxicity that can occur [36, 49]. Anti-trypanosomal drugs such as Suramin (Naganol), Quinapyramine (Antrycide), Isometamidium chloride (Samorin), diaminazine aceturate (Berenil) and melarsomine (Cymelarsan) are commonly used to treat animal Trypanosomiasis. Most were marketed as early as 70 years ago [51]. These drugs are limited to treatment and some preventative measures. Some agents have narrow. therapeutic windows Some of these agents (Quinapyramine sulfate and Melarsamine) when administered in an extravascular manner, can lead to significant tissue injury at the injection site [52]. Of importance, the trypanosomal-specific drugs are not commonly available commercially. Because of this, many compounded medications (non-approved) are on the market and adversities associated with the use of these medications are reported. Availability of low cost, approved medications would seem to be important to countries were camels are common and used for work and transportation.

Other drugs have also been observed to cause adverse reactions in camels. Though tetramisole is no longer available in North America as an anthelmintic, it was used in camels to treat GI parasites. Doses >10 mg/kg result in adverse reactions and the injectable form was particularly irritating to tissues [36]. Levamisole is sometimes available in North America for use in ruminants. Levamisole functions as cholinergic receptor agonist that produces parasite paralysis by persistent activation of nicotinic acetylcholine receptors. This results in depolarization of nervous tissue and it can interrupt metabolism of sugars in GI parasites. The resulting paralysis of the nematode produces an inability of the parasite to maintain attachment in the gut and they either die or are expelled by GI contractions. It is effective against a variety of parasites (stomach, intestinal, lungworms). The label dose (Prohibit®) in cattle, sheep and swine is 8 mg/kg per os and requires dilution in water. After dilution, it has a shelf life of up to 90 days. In previous editions of this text, a dose range of 8–12 mg/kg is provided. Care should be taken when dosing, especially in compromised patients (anemia, dehydration) as it can produce intoxication when administered to camelids at doses >15 mg/kg when given orally.

El Bahri et al. also indicated that Nitroxynil at doses exceeding 50 mg/kg SC is fatal to the dromedary camel [36]. Nitroxynil is an uncoupler of oxidative phosphorylation and disrupts adenosine 5'-triphosphate (ATP) production and is used to treat Fascioliasis. At label doses (7–13 mg/kg) no adverse effects are noted in cattle. In camels, 40 mg/kg Nitroxynil SC is well tolerated. Exceeding this dose, however, is reportedly fatal to camels.

Tolazoline Intoxication

Tolazoline hydrochloride is an α -adrenergic blocker used to reverse the effects of xylazine HCl. The label dose for horses is 4 mg/kg body weight. Pharmacologically, tolazoline functions as an α-adrenergic antagonist with some agonist effects. In addition, it may also have cholinergic, and histaminergic effects on the patient. Cardiac arrhythmias do occur when tolazoline is administered rapidly, in high doses. High doses of tolazoline have also been observed to cause similar effects in calves and horses [53].

Read et al. reported on an adult female llama (140 kg) that was sedated with xylazine hydrochloride for evaluation of rear limb injury [54]. To evaluate the lesion and provide treatment, the llama was given xylazine at 0.5 mg/kg IM. After determining the extent of the lesion, surgical debridement was conducted after re-dosing the llama with a second 0.5 mg/kg dose of xylazine IM. This provided approximately 40 minutes to treat and bandage the wound. To reverse the xylazine, the animal was given approximately 4 mg/kg of tolazoline hydrochloride IV which reversed the sedation. After becoming sedate again, a second (higher dose) of tolazoline was again administered (300 mg; half IV and half IM). Within minutes, anxiety, trembling, hyperesthesia, salivation, sweating, tachypnea, periodic dyspnea and diarrhea developed. Tolazoline toxicosis was suspected of causing these adverse effects.

The tolazoline label indicates approval for use in reversing the effects of xylazine in horses only. The dose rate is 4 mg/kg body weight (4 mL/100 kg). The label also indicates it should be given "slowly" by IV injection. Some practitioners split the dose giving half of the calculated dose IV and the other half IM. Some of the side effects listed on the drug label include elevated blood pressure, vasodilation of peripheral vasculature, sweating, hyperalgesia of the face, piloerection and nasal/ocular discharges.

Albendazole

Intoxication of alpacas with albendazole has been reported in the refereed literature as well as anecdotally. Albendazole is not labeled for use in llamas or alpacas but used in doses recommended in the range from 10 to 15 mg/kg PO once [55, 56]. At higher doses or after multiple doses, interference with microtubule formation may result in damage to bone marrow, or other rapidly dividing cells resulting in profound hypoplasia of the progenitor cells of the marrow and intestinal epithelial cell necrosis. In camelids, the use of albendazole as a substitute for fenbendazole or as a "more effective anthelmintic" than fenbendazole is not uncommon. In one report, nine alpaca crias and three adult male alpacas were treated daily for four (nine crias) or five days (adult male alpacas) receiving total doses of 33–100 mg/kg/day (nine crias; one to seven months of age) or 19–33 mg/kg/day (three adult males; two to three years of age). Clinical signs associated with these doses in the crias were: found dead (6 days after starting treatment; two Crias), severe watery diarrhea and hypoproteinemia. All seven crias were neutropenic. In the adults, two presented for lethargy, fever and diarrhea after the fifth day of albendazole. The third adult was brought into the clinic three days after receiving the fifth dose [57]. In the cases reported in this report, aggressive therapy was required to save some, but not all of the animals [57]. Parenteral administration of fluids, colloids, broad-spectrum antibiotics, antifungal agents and Granulocyte-colony stimulating factor (G-CSF) (Filgrastim) were utilized in these cases. Of the seven crias that were hospitalized, five died. Of the three adult animals, one died. Autopsy findings were similar in both age groups. Serosal and mucosal erythema of the GI tract, with consistent histologic changes were observed in all dead animals. Mucosal necrosis with regeneration in the young animals small and large intestine. In the adult male alpaca, lesions were more severe in the large intestine. Epithelial necrosis, with regeneration and areas of severe ulceration in the colon. Bone marrow hypoplasia was evident in all dead alpacas [57].

Since this publication was disseminated, additional cases have been anecdotally reported. One inhouse case presented off feed and dehydrated after receiving 49.5 mg/kg albendazole daily for three days. The animal was an adult female llama weighing 148 kg and was neutropenic and leukopenic on arrival. She became bacteremic and responded to antimicrobial agents, filgastrim and supportive care. She was discharged from the hospital; however, it was determined that this female had died some months after discharge. A more recent publication demonstrated severe fiber loss in young alpacas when treated for fascioliasis with albendazole (~37.5 mg/kg PO). Within approximately three days post dosing, young alpacas developed high fevers (40–41°C) followed 10 days later by fiber loss. Six months later, the fiber had grown back [58].

Propylene Glycol

A four-year-old pregnant female llama (123 kg) was presented for poor appetite, lethargy and weight loss of approximately six weeks duration [59]. The local veterinarian had diagnosed *Trichuris spp*. infestation which was treated with fenbendazole. The owner had also treated the llama with a propylene glycol paste (three tubes containing 270 g of propylene glycol). Analysis of serum revealed acidosis with low potassium, low total protein and albumin with increased aspartate aminotransferase (AST) and gamma-glutamyl transferase (GGT). A sample of C1 fluid was obtained which smelled like a petroleum product and after sitting, droplets reminiscent of oil appeared at the surface. Most of the

protozoal organisms were not viable at the time of exam. The pH of the C1 fluid was 6.0. After transfaunation of the llama and supportive care, the acidosis resolved and the female began to eat, drink, and gain weight normally. Acidosis, associated with excess propylene glycol, occurs in horses and cattle as well as small ruminants [60–62]. While there are established cases of propylene glycol intoxicated cattle and horses, there is no minimal toxic dose established for any species. When use of propylene glycol in ruminants and camelids is necessary, frequent evaluation of the patient is important to prevent overdosing with this agent.

Vitamin D Toxicosis

Since vitamin D deficiency is associated with rickets in camelids, owners are taught to provide adequate vitamin D to their animals (in feed, as a paste or by injection). Overzealous use of vitamin D has resulted in illness and death in these species [63]. In one report, two alpaca crias (two-day old Huacaya Male; eight-day old Suri Male) were presented for failure to thrive, anorexia, lethargy and potential failure of transfer of maternal IgG. The first cria was treated and discharged after nine days of hospitalization. However, at home, the cria was supplemented with 4.6 g of goat colostrum supplement to each bottle feeding for seven days. The goat colostrum powder contained a minimum of 2750 IU of vitamin D_3 per 4.6 g of powder. The cria was fed every two hours for seven days. The total cumulative dose of vitamin D_3 over seven days was 26 250 IU/kg of body weight (3750 IU/kg/day). After re-admission to the hospital, serum biochemical analyses revealed marked azotemia, hyperphosphatemia, hypercalcemia, and hyperkalemia. The cria did not produce urine, even following aggressive fluid therapy and diuretics (furosemide). Radiographs of the abdomen revealed mineralization of the third gastric compartment. At necropsy, the mineralization of the medulla of both kidneys was observed. Histologically, there was mineralization of the interstitium, tubules, and glomeruli. Serum vitamin D_3 levels were 663 nmol/L (reference range: 50–200 nmol/L). Kidney tissue vitamin D_3 concentrations were also elevated (282 nmol/L reference range: 2–11 nmol/L). Similar to the first, the second cria presented for anorexia and lethargy and the owners were providing 1 g of paste supplement containing 100 000 IU vitamin D_3 per day for the first five days of life. The total cumulative dose was 500 000 IU which was equivalent to 12 987 IU/kg/d over this time. Total serum calcium was 3.99 mmol/L, the cria demonstrated marked azotemia and did not produce urine. Peritoneal dialysis and diuretic treatment with IV fluids were not successful. Similar gross and histologic lesions occurred in the second cria. Serum vitamin D_3 was 602 nmol/L, and kidney tissue vitamin D was 350 nmol/L [63].

None of the products available for use in veterinary patients includes a label for camelids. Further, while it is known that crias require supplemental vitamin D to prevent orthopedic problems, animals are often treated with single large doses or multiple appropriate doses over longer periods of time [64–66]. More recent cases of vitamin D intoxication have been reported in the literature [67]. None of these cases were associated with successful outcomes.

Other causes of hypercalcemia and widespread mineralization include plants that produce weight loss, painful lameness, mineralization of soft tissues, kyphosis, polyuria, and dyspnea [68, 69].

The active factor in *Solanum malacoxylon*, *Cestrum diurnum* and other plants contain 1,25-$(OH)_2$D bound to a plant glycoside that is water soluble. This form of vitamin D rapidly increases the plasma concentrations of phosphorous and calcium leading to tissue mineralization [70].

Growth Promoting Antimicrobials

Ionophores are naturally occurring antimicrobial drugs that are widely used to control coccidial parasites of poultry, cattle, and swine. They also enhance feed utilization and thus are considered growth promoters. Three common ionophores most likely to be involved with camelid poisoning are monensin [71–73], salinomycin [74, 75], and lasalosid, though narasin has also caused the death loss of many camels in the world [76].

Modern animal feeding is commonly associated with the use of ionophore antibiotics to promote health by limiting GI parasites (coccidia) as well as improving the amount of feed provided for a greater amount of muscle growth. Sensitivity of a species to these feed additives varies greatly. Cattle have much higher tolerance of monensin and lasalocid compared with horses. Poultry tolerate higher feed additive levels than ruminants. There are no established tolerances for monensin, lasalocid, salinomycin, or narasin in camelids (camels, alpaca, vicugna, llama, guanaco). There are examples of intoxication of camelids for all ionophores except for lasalocid [72, 75–77]. Outbreaks of acute deaths, rhabdomyositis leading to myoglobinuria, associated with ionophores, have also been reported in alpacas [74].

Clinical signs, associated with ionophore toxicosis, center around the organ system most affected in the species of concern as well as the dose of specific toxins. Initially, anorexia and depression are observed, followed by muscle tremors, weakness and incoordination. In camels, profound weakness in the rear limbs may keep them recumbent. Muscle involvement is very common leading to respiratory, cardiac and GI dysfunction. In cases where

total dose in feed is high, CNS signs may also be present. Urine may be port-wine colored (dark red/purple). Clinically, pathologic, elevation of creatine kinase, AST, lactate dehydrogenase (LDH), alkaline phosphatase (ALP), azotemia, hypocalcemia, and hypokalemia are expected. Hemoconcentration is associated with anorexia, azotemia, and dehydration [74].

Diagnostically, analyzing the suspect feed(s), water, minerals and other potential products that may have been provided should be collected in large plastic bags (1 gal) or plastic containers and kept frozen or stored where rodents/insects cannot damage these samples. Identification of an ionophore in the feed supplied to the owner of these animals is critical. Analysis of animal tissue or plasma has not been as useful in the diagnosis due to extremely low levels present. Obtaining large quantities of GI content (1st compartment; 1 gal) may prove useful identifying an ionophore. For animals that die, it is prudent to obtain an autopsy to identify lesions such as chalky white streaks in muscle (heart, skeletal muscles), pallor of muscle or flabby heart muscle. In camels, myoglobinuria is a common finding in published reports [74].

In animals that survive, use of cathartics to remove GI content more rapidly as well as supportive care has been recommended. It is known that depletion of vitamin E can occur, therefore, supplemental vitamin E, selenium may be beneficial. Judicious IV fluids are likely important due to myositis and the effects of proteins on kidney function. One should also recommend the provision of "clean" palatable feed to the animals.

Prostaglandin Toxicity

Prostaglandin F2α is incorporated extensively into reproductive procedures in veterinary medical practice. Numerous commercial products result in different pharmacologic effects, depending on the form of prostaglandin employed. One such, $PGF_2\alpha$, is prescribed as a smooth muscle activator or for its luteolytic action. The effects of $PGF_2\alpha$ are vasoconstriction, bronchiolar constriction, intestinal muscle contraction, uterine muscle contraction, and luteolysis if a corpus luteum is present.

Dinoprost, tromethamine is an aqueous solution, with benzyl alcohol as a preservative. Generally, $PGF_2\alpha$ is considered nontoxic. However, there are species differences related to effective doses. The toxic dose for camelids is unknown, but three female llamas died shortly after $PGF_2\alpha$ was used to produce luteolysis. In all cases, the llamas were determined to have retained corpora lutea.

Case one was a 16-year-old female llama with a cria at side. Dinoprost tromethamine (15 mg total dose) was administered IM at 3 p.m. The llama was found dead the next morning. Excessive fluid was found in the lungs at necropsy.

Case two was a seven-year-old female. Dinoprost tromethamine (20 mg total dose) was administered IM at 7 p.m. She was found dead one hour later. Pulmonary edema and intestinal hyperemia were noted at necropsy.

Case three was a mature female. Miscommunication resulted in an overdose (50 mg) of dinoprost tromethamine administered IM. She was returned to the owner's ranch but died within one hour from what is presumed to be a direct toxic effect of $PGF_2\alpha$.

Three other females have exhibited dramatic, but temporary, clinical responses to therapeutic doses of dinoprost tromethamine.

The three llamas that died were not observed prior to death. The presence of pulmonary edema would indicate that they were dyspneic. The three females that developed transient signs reacted within five minutes. Two females rolled on the ground and foamed from the mouth. The client described periodic abdominal spasms, somewhat like hiccups. Camelids displaying open-mouth breathing could also be very seriously stressed, toxemic, have nasal edema or recently developed a dislike for a pen mate, and likely other causes. Historical information regarding the use of a luteolytic drug would be important.

Oxygen insufflation, via a face mask, may aid the llama with mild pulmonary edema or bronchiolar constriction. Prostaglandins are valuable therapeutic agents for dealing with infertility, but their use is not without risk. The animal should be kept under observation for at least one hour following administration of $PGF_2\alpha$. An apparatus for administration of oxygen should be available in the event of a reaction.

Heavy Metals

Lead, arsenic, mercury, copper, and molybdenum are used in agricultural products and paints. It should be assumed that camelids are susceptible to inadequate or supraphysiologic levels of copper. Lead, arsenic and mercury are very toxic to all animals (and humans) and may be of concern in some countries where consumption of contaminated soil and/or the feed produced from these soils can occur. Clinical signs and lesions are presumed to be like those of poisoned cattle and sheep. Table 22.1 lists heavy metals that may cause toxicity in camelids.

Selenium

A local alpaca enthusiast asked their veterinarian to examine two pregnant female alpacas for a complaint of "sweating" in very cold weather (see Figure 22.1).

Table 22.1 Heavy metal poisoning in camelids.

Metal	Source for llamas	Clinical signs	Diagnosis	Pathology	Management
Lead (Pb)	Contaminated forage, paint	Depression[a], anorexia, stomach atony, colic, diarrhea, ataxia, circling, blindness	Blood, liver, and kidney analysis	Encephalopathy, gastroenteritis	Calcium EDTA (110 mg/kg) for five days
Arsenic (As)	Contaminated forage, pesticides, herbicides	Colic[a], weakness, salivation, trembling, diarrhea, depression, dehydration, shock	Chemical analysis of liver, kidney, and stomach contents	Gastroenteritis	Removal, symptomatic, fluids, prevent secondary infection
Selenium (Se)	Plants containing high Se, therapeutic overdose	See Nutrition, Chapter 2	Chemical analysis of liver and kidney	Pansystemic, hepatic necrosis, renal necrosis	Removal, symptomatic
Copper (Cu)	Water contamination, insecticides, food supplements	See Nutrition, Chapter 2	Chemical analysis	Hemolytic anemia, hepatic necrosis	Nothing for acute toxicity, add molybdenum to diet
Molybdenum (Mo)	Plants high in Mo and low in Cu	See Nutrition, Chapter 2. Diarrhea, coat depigmentation, decreased fertility, emaciation, anemia	Response to Cu supplementation	Microcytic, hypochromic anemia, hemosiderosis, emaciation	Cu supplementation, Cu injection

Note: Signs listed are a composite.
[a] Clinical syndrome has not been described in lamoids. Signs are variable in livestock.

Figure 22.1 This alpaca presented after receiving too much selenium. Notice the wet fiber coat from sweat. Photo courtesy of Dr. Eric Shaver.

Both females were in full fleece and were approximately 120 days gestation. During discussion of these two females with their local veterinarian, it was apparent that the owners were feeding the animals appropriately (hay free choice, with additional energy and protein supplements during the wintry weather (average −16 °F; −26 °C). We discussed the possibility of excess selenium or iodine as a potential cause and serum from these two animals was submitted for CBC, biochemical profiles and trace mineral profiles were submitted. Both females had hyperselenosis (serum, 382 ng/mL, 507 ng/mL; reference range: 120–160 ng/mL). Hair samples were also submitted but came back within reference range 0.57 μg/g, 1.07 μg/g; reference range: <2 μg/g). In addition, serum T4 was elevated in one animal (20 μg/dL) with higher Se concentrations, while the second animal had T4 within the reference range- 10.3 μg/dL). After reviewing the diet, forage analysis and supplements, Se was being fed at a rate of 80 ppm per head per day. Because they were group fed, some may have gotten less, others higher levels of Se each day. The diets were adjusted to reduce selenium exposure, serum levels dropped over the remainder of their gestation. At birth of the two crias, one was completely normal, the second animal was born normal in appearance, but unable to stand. At autopsy, there were no gross lesions observed in this cria (spinal cord discarded). Selenium toxicity in young swine has been associated with paralysis due to malacia of the lumbar spinal cord [78].

Copper

Absorbed copper is stored in hepatocyte lysosomes. When the storage capacity of the liver is exceeded, copper is released into the cytoplasm of the hepatocyte causing hepatocellular necrosis. Once the hepatocyte cell wall has been destroyed, copper is released into the circulation, causing hemolysis, icterus, and anemia. The excessive amount of free hemoglobin in the circulation may occlude renal tubules and reduce kidney function, resulting in renal failure.

Copper poisoning has been reported in SACs [79, 80]. Four zoo llamas were fed a dairy pellet supplement containing 44.8 mg copper/kg of supplement [80]. The total dietary intake was estimated to be 25 mg/kg of feed (cattle tolerate relatively higher levels of copper in the diet as compared to sheep). Owners should be advised that pellets or mineral mixes intended for cattle are not suitable as supplements for camelids unless the total dietary intake of copper, including the supplement, is <15 mg/kg of feed. It is also necessary to maintain a copper/molybdenum ratio of at least 4:1.

In one animal, the first signs noted were lethargy and inappetence. The following day the llama was recumbent, markedly depressed, dyspneic, and hypothermic, and had pale, but not icteric mucous membranes. It died eight hours later. None of the poisoned llamas demonstrated hemoglobinuria or hemoglobinemia.

Necropsy of these animals demonstrated hepatomegaly, hepatocellular necrosis and bile-duct proliferation histologically. Serum copper levels varied from 4.3 to 5.7 μg/dL, and liver copper levels were 847–1700 mg/kg dry weight (reference ranges: 6.7–330 mg/kg in neonates and 10 to 36 mg/kg in adults) [81]. Serum enzymes associated with hepatic necrosis were elevated: AST (2525–5137 U/L [normal 179 U/L]), LDH (2286–8652 U/L [normal 287 U/L]), and GGT (177–216 U/L [normal 125 U/L]).

Toxic Plants [2, 82]

There are only a few published reports of plant poisoning in Old World camelids (OWCs) and New World camelids (NWCs). In North America, the most common plant poisonings are oleander (*Nerium oleander*) [83] and various species of the family Ericaceae [29, 84, 85]. No plant poisonings have been reported from South America. Camels and llamas browse a wide variety of plants and may be affected, in certain circumstances, by the plants listed in Tables 22.2–22.4. Alpacas are primarily grazers and hence are less likely to consume shrubbery. Other plants may also affect camelids. Knowledge of common poisonous plants is critical for the veterinarian and owner [10, 29, 83–90].

Ornamental plants are a special hazard for camelids [86]. Unfortunately, such plants may not appear in poisonous plant lists and booklets of the region, because many ornamentals are not native.

Rhododendron Poisoning

A number of genera and species within the plant family Ericaceae contain a resin, andromedotoxin, and a

Table 22.2 Poisonous plants that may affect llamas on trek.

Common name	Scientific name	Habitat	Poisonous principle	Signs of poisoning	Therapy
Arrowgrass	*Triglochin maritina*	Meadows at low to moderate elevations	Cyanogenic glycoside	Muscle twitching,-convulsions, dyspnea, bright red blood	Methylene blue, sodium-thiosulfate
False hellebore, corn lily (Figure 22.7)	*Veratrum californicum*	High mountain meadows	Alkaloids	Vomiting, salivation, convulsions, fast irregular heart beat	Supportive care and preventing access.
Death camas, sandcorn	*Zigadenus* sp.	Hillsides, fields, meadows in spring of year	Alkaloids	Foaming at mouth, convulsions, ataxia, vomiting, fast weak pulse	Supportive care and preventing access.
Nightshade	*Solanum* sp.	Ubiquitous	Alkaloid-glycoside solanine	Vomiting, weakness, groaning	Supportive care and preventing access.
Jimsonweed, thornapple	*Datura metaloides*	Fertile soils	Alkaloid-atropine, hyoscyamine, scopolamine	Dry mucous-membranes, dilated pupil, GI atony, tympany, tachycardia, anuria, convulsions, mania	Parasympatholytics
Chokecherry, wild cherry	*Prunus virginiana*	Streamsides	Cyanogenic glycoside (entire plant, not fruit)	Dyspnea, convulsions, rapid death, bright red blood	Sodium nitrite and sodium thiosulfate
Western sneezeweed	*Helenium hoopesii*	High mountain meadows	Sesquiterpene alkaloids	Vomiting, depression, frothing at mouth, coughing, weak -irregular pulse	S
Labrador tea	*Ledum glandulosum*	Around lakes, meadows, streams	Andromedotoxin (diterpene), arbutin	Vomiting, colic, paresis, anorexia, muscle twitches	S
Black laurel, Mt. laurel (Figure 22.2)	*Leucothoe davisiae*	Around lakes, meadows, streams	Andromedotoxin (diterpene), arbutin	Vomiting, colic, paresis, anorexia, muscle twitches	S
Western azalea	*Rhododendron -occidentale*	Around lakes, meadows, streams	Andromedotoxin (diterpene), arbutin	Vomiting, colic, paresis, anorexia, muscle twitches	S
Rhododendron (Figure 22.3)	*Rhododendron* sp.	Around lakes, meadows, streams	Andromedotoxin (diterpene), arbutin	Vomiting, colic, paresis, anorexia, muscle twitches	S
Oleander (Figure 22.4)	*Nerium oleander*	Ornamental	Cardioactive glycoside	Diarrhea, colic, cardiac irregularities, cyanosis	S, gastrotomy
Castorbean (Figure 22.9)	*Ricinus communis*	Ornamental, may escape	Ricin, water soluble albumitoxin	Anaphylactic shock, diarrhea	Treat for shock, fluids
Tobacco, tree tobacco	*Nicotiana* sp.	Waste places	Alkaloid, nicotine	Stimulations of CNS, then depression; sweating, muscle twitches, convulsions	S

Note: S = Symptomatic. In most cases of poisoning from ingestion of poisonous plants, there is no specific antidote; rather, it is necessary to treat symptomatically.

Table 22.3 Additional plants that may cause poisoning in SACs.

Scientific name	Common name	Poisonous principle	Habitat	Signs of poisoning	Management
Robinia pseudoacacia	Black locust	Alkaloids	Ornamental	Diarrhea, collapse, shock	Supportive
Digitalis purpurea	Purple foxglove	Cardioactive glycoside	Ornamental	Same as oleander poisoning, see text	Supportive
Taxus baccatta	Yew	Alkaloid	In gardens and parks as trees, hedges. Found wild in California.	Sudden death, dyspnea, collapse, diarrhea	Supportive
Lantana camara	Lantana	Hepatotoxins	Ornamental ground cover, escapes into waste areas	Hepatic insufficiency, photosensitization, icterus	Remove source, keep out of light, r
Asclepias spp.	Milkweed	Alkaloidal glycosides	Along roadsides, edges of cultivated fields, waste places	Depression, weakness, convulsions, diarrhea	Symptomatic
Senecio spp.	Ragwort, groundsel, fireweed	Pyrrolizidine alkaloids	Weed in hay and waste places	Hepatic insufficiency	None
Dieffenbachia seguine	Dumbcane, dieffenbachia	Oxalate crystals	Ornamental house plant	Head shaking, salivation, excessive drinking	Wash mouth out with water
Nitrate accumulator plants	Many species of forage grasses and weeds	Nitrate/nitrite	Harvested crops, waste areas	See text	Methylene blue, see text
Quercus spp.	Oaks	Tannins, tannic acid, pyrogallol	Trees planted for shade around homes and barnyards, also found in woods and forests	Renal failure, high BUN and creatinine	Symptomatic, prognosis poor
Osteospermum echionis	South African daisy	Cyanogenic glycoside	Ornamental, Australia	See text for HCN	See text for HCN
Acer rubrum	Red maple	Oxidant	Eastern USA	Hemolytic anemia	Blood transfusion

Table 22.4 Poisonous plants that may affect camels.

Scientific name	Common name	Country	Poisonous principle	Signs of poisoning
Capparis tomentosa	Magico medicinal plant	Africa	Glycoside	Torticollis, tremors, weakness, incoordination, dyspnea, convulsions
Erythrophloeum chlorostachys	Ironwood, camel poison	Australia	Alkaloid	Staggering, heart pounding, blindness, dyspnea, star gazing
Nerium oleander	Oleander	USA, Australia, India, Africa	Oleandrin, cardioactive glycoside	Diarrhea, salivation, tremors, convulsions, cardiac arrhythmia
Gastrolobium grandiflorium	Desert poison bush	Australia	Fluoroacetate	Tachycardia, dyspnea, muscle spasms, convulsions
Oxylobium spp.	Box poison	Australia	Fluoroacetate	Same as above
Gyrostemon granulosus	Camel poison	Australia	?	Coughing, nasal discharge, foaming, dyspnea
Dubosia hoopwoodii	Emu poison bush	Australia	Alkaloid, nicotine	Trembling, weakness, incoordination
Trema tomentosa	Poison peach	Australia	glycoside	Depression, twitching, staggering, coma
Homeria breyniana	Cape tulip	Australia, Africa	?	Tremors, colic, stiffness, incoordination, diarrhea
Peganum hirmale	African rue	Africa, India	Alkaloid	Paralysis
Datura stramonium	Thorn apple	Africa, USA, United Arab Emirates	Alkaloid, atropine	Bloat, colic, dyspnea, maniacal behavior
Iphiona aucheri	Composite	United Arab Emirates	Diterpine glycosides	Signs of liver failure from liver necrosis
Palicourea grandiflora	Mata	Brazil	Fluroacetate	Sudden death, cardiac failure
Diplotaxa harra		North Africa		Stiffness in the hind quarters
Cassia truncata				
Uvaria denhardtin				
Thasia gargancia	Apiaceae	North Africa	Organic acids	Excessive salivation, colic, diarrhea
Ipomoea carnea	Morning glory			

Figure 22.2 Black laurel, Mt. laurel (Leucothoe davisiae).

Figure 22.3 Azalea (Rhododendron spp).

glycoside, arbutin, which produce an identical syndrome in a broad host range. A description of the history and clinical signs of two llamas poisoned with Sierra laurel (*Leucothoe davisiae*) follows [29]. (Figures 22.2 and 22.3).

Two pack llamas were tied to some shrubs near a grassy area at the edge of a small lake during a lunch stop. The first clinical sign was noted one and a half hours later when one llama began to cough and appeared to be choking. Within

an hour, he was recumbent and refused to rise. The second llama began foaming (shaving cream consistency) from the mouth. He, too, began coughing and choking. Both llamas ultimately began projectile vomiting between episodes of rolling, retching, and groaning. Depression, anorexia, vomiting, groaning, and signs of colic persisted for two days. On the third day, the animals had recovered sufficiently to slowly move out of the mountain to a trailhead.

The signs observed in these llamas are similar to those seen by the author in cases of rhododendron poisoning in both llamas and domestic livestock. The composite of the clinical signs includes anorexia, repeated swallowing, salivation, depression, vomiting, bloat, colic (straining, rolling, groaning, grinding of the teeth), weakness, ataxia, prostration, dyspnea (aspiration pneumonia), bradycardia, and hypotension. Hepatopathy may be a sequel [27].

Treatment of rhododendron poisoning is non-specific. Administration of a cathartic such as magnesium oxide (10–20 g) is appropriate. Activated charcoal (100–200 g) is a general antidote and may be administered by stomach tube or as a drench consisting of a slurry of activated charcoal in water. However, such therapy is dangerous in a vomiting animal. Atropine (0.04 mg/kg) may be used to alleviate bradycardia. Other symptomatic treatment is indicated.

Oleander Poisoning

Oleander (*N. oleander*) is one of the most toxic shrubs to which a camelid or any other mammal may be exposed. The lethal oral dose of either green or dried leaves is 225 mg/kg. For a 150-kg (330-lb) llama, this would mean less than 35 g of leaves (15 medium-sized leaves or 1 leaf/10 kg body weight). Comparable quantities based on weight would kill camels.

Oleander is a beautiful ornamental shrub, native to the Mediterranean area and grown extensively throughout California and along the southern tier of the United States (Figure 22.4). As a potted shrub that can be moved inside during the winter, it may be found almost anywhere. Camelids may obtain oleander leaves in a variety of ways. Rarely will they ingest leaves directly from the living shrub. However, if cuttings or lawn clippings containing leaves are placed where llamas can get at them, there is great risk. A sprig of oleander that had been chewed on was found in the manger of an eight-month-old llama that died with signs of oleander toxicity.

In another instance, oleander leaves and branches were included with other tree cuttings and run through a shredder. The mulch was then spread along a path next to a llama pasture. Some of the dried mulch blew into the pasture. Within 24 hours, one of three llamas was sick and died four days later. Another llama became ill but recovered [83].

Figure 22.4 Oleander, (Nerium oleander).

Clinical signs of oleander poisoning are similar in all species of mammals. Llamas become anorectic and depressed and lie down unless forced to rise. The major observable sign is frequent, projectile, catarrhal to hemorrhagic diarrhea. Colicky signs may accompany the diarrhea. If the ingested dose is high, the animal may die of cardiac complications before diarrhea develops.

The cardioactive glycoside has a direct effect on the cardiac musculature. Various conduction abnormalities may be heard on auscultation over a period of a few minutes because of the rate and rhythm change. There may be bradycardia, tachycardia, dropped beats, and partial and complete blocks. Impaired circulation may result in cyanosis, muscle tremors, patchy perspiration, and dyspnea. Terminally, tachycardia progresses to ventricular fibrillation and agonal struggling.

Clinical signs in ruminants may develop 5–24 hours after ingestion. Camelids may develop clinical signs sooner because C1 of the stomach has an absorptive mucosa (Chapter 12). Death may occur within 10 hours or after three to four days. The severity of clinical signs is not directly correlated with possible mortality.

At necropsy, lesions are limited to enteritis plus petechial or ecchymotic hemorrhages of the GI serosa or of the epicardium, endocardium, or pericardium. Stomach contents of all three compartments should be carefully examined for the presence of leaf segments. Oleander leaves have a unique vein pattern, with a single midrib and parallel secondary ribs (Figure 22.5a). Finding even a small segment of a leaf in the stomach contents justifies a diagnosis of oleander poisoning. (Figure 22.5b). A two-dimensional (2D) thin-layer chromatography test has been developed that detects oleandrin in a 0.05-ppm concentration in ingesta.[2]

Treatment of oleander poisoning is difficult. The regimen for treating an overdose of digitalis in humans is not satisfactory for camelids (lowering serum calcium by

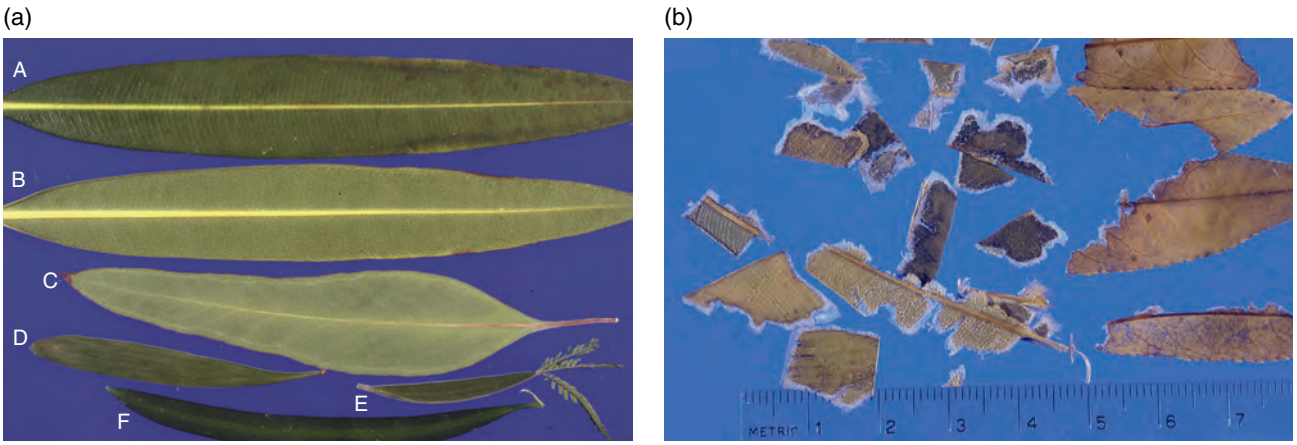

Figure 22.5 (a) Veination of leaves that may be confused with oleander. (A) Oleander, top view, (B) oleander, bottom view, (C) eucalyptus, (Eucalyptus spp.), top view, (D) acacia, (Acacia spp.), top view, (E) acacia, bottom view, (F) Oleander fruit. (b) Oleander leaf segments collected from the ingesta at necropsy.

titration with sodium versenate, administration of potassium glutamate). The use of various drugs to counter arrhythmias has not been established. If a definitive diagnosis is made, the logical recourse is to remove the remaining oleander material from the digestive tract. This means that a gastrostomy must be performed (Chapter 12). All of the contents of C1 must be removed and the lumen carefully washed. Small-leaf segments left in the stomach may be lethal. The microorganisms from C1 must be replaced by transfaunation from another camelid or ruminant.

Unfortunately, patients with circulatory deficiencies are not good surgical anesthetic risks. Gastrostomy may be performed in the standing camelid under local anesthesia, but it is more difficult.

Animals in a herd are not all equally curious and all may not consume leaves in their enclosure. However, all exposed animals should be examined for cardiac irregularities. A cathartic may be administered to hasten emptying of the GI tract. Oleander is so toxic it is dangerous to wait for signs to appear.

Pyrrolizidine Alkaloid Poisoning

Dozens of pyrrolizidine alkaloids are found in plants broadly distributed throughout the world. These alkaloids vary in toxicity from no effect to pronounced effect on the liver of the host. Many animals have some innate resistance to pyrrolizidine alkaloids. The alkaloid must be metabolically altered to an active metabolite before toxicity occurs, and certain species such as sheep degrade the alkaloid in the digestive tract and liver, avoiding production of the active metabolite.

Horses are the most susceptible species studied, followed by cattle, then swine. It is not known where camelids fit on the scale, but in an unreported study conducted on three llamas in Oregon, clinical signs and/or lesions could not be produced. The plant material used was tansy ragwort (*Senecio jacobea*), which is known to be highly toxic. The llamas would not eat the dried plant material, so the material was suspended in a slurry and administered by stomach tube or via a stomach fistula. The llamas were given 50% of their body weight of the dried plant material.[3]

It is a commonly held belief that pyrrolizidine alkaloid poisoning (PAP) occurs in alpacas in Australia from consuming fireweed (*Senecis quadidentatus*) and other species of *Senecio*. While this author has not been able to find any scientific report of PAP in alpacas, clinical veterinarians in Australia have reported that the lesions of PAP occur in alpacas with hepatic insufficiency.[4]

Dieffenbachia Poisoning (Dumbcane)

Some discarded ornamental dieffenbachia (*Dieffenbachia seguine*) was fed to a llama, which immediately began to salivate, spit, cough, shake its head, and rub its mouth on a fence post [90]. Dumbcane produces the same effect on any animal that takes it into the mouth. Both oxalate crystals and a proteinaceous agent may irritate the mucous membrane, but the speed of reaction indicates a toxic rather than a mechanical effect. The affected llama appeared to recover within 30 minutes.

More severe signs may occur if laryngeal edema is sufficient to partially obstruct the larynx. Glossal edema may inhibit tongue mobility and swallowing. These effects may last for a number of days. The only treatment is to wash the mouth out with water from a garden hose. The animal should be monitored for signs of severe dyspnea, in which case a tracheostomy is necessary.

Figure 22.6 Yew (Taxus spp.).

Yew Poisoning

Yew (*Taxus* spp.) has been known to be highly toxic to farm animals and humans for centuries. (Figure 22.6). Yews are common ornamentals but should not be planted at a site where animals may gain access. A group of llamas came in contact with some yew shrubs, and though they consumed only a small amount, it was sufficient to kill some animals.

Death is so rapid that signs may not be observed. Taxine, the poisonous agent, affects the CNS, causing vomiting, tremors, convulsions, and dyspnea. Further, no lesions are produced. Unless a history of ingestion is forthcoming, the only diagnostic aid is to find the characteristic needles in the stomach. There is no treatment for yew poisoning.

False Hellebore Poisoning

Veratrum californicum and other species of Veratrum are known to produce teratogenic effects in sheep, pigs, horses, and cattle fetuses (Figure 22.7). Veratrum spp. contain several alkaloids, some of which are responsible for the teratogenic effects and others for general systemic affects.

If pregnant sheep ingest *Veratrum* on days 14–16 of gestation the fetus is affected, and the lamb will be a cyclopian-cebocephalic (one eyed, monkey faced). The author has seen four cases affecting camelids in Ohio. Others have reported similar cases in Oregon and Alaska. Presumably *Veratrum* spp. or other teratogenic plants are also present in South America. (Figure 22.8).

Varying degrees of facial deformities and other congenital conditions are produced in sheep and swine depending upon the precise age of gestation. In sheep, days 14–16 were determined to produce cyclopian cebocephaly. The time of insult to the fetus in camelids is not known, however; based upon the longer gestation of camelids, it could be later in the first month of pregnancy.

Figure 22.7 Corn lily (false hellebore) (Veratrum californicum).

Figure 22.8 Cria born with a single, enlarged, centrally located eye from an alpaca exposed to Veratrum californicum during pregnancy. Ingestion of veratrum californicum has been shown to have teratogenic effects, specifically leading to a cyclops fetus when ingested by the dam during pregnancy.

Castor Bean Poisoning

Ricinus communis (castor bean, castor oil plant, palma Christi) is an erect perennial shrub of warm climates but is

Figure 22.9 (a) Castor bean (Ricinus communis) plant. (b) Castor bean foliage. (c) Castor bean seed. The seed coat contains the ricin toxin. Broken seeds are extremely toxic.

an annual herb in areas where frost is common, Figure 22.9a–c. Castor bean is native to the Mediterranean region but has become naturalized in many countries of the world. It is commonly grown as an ornamental throughout the world. The seeds of castor bean contain a high concentration of oil that has been used medicinally since 2000 BCE.

Chemically, ricin is a protein and classified as a toxalbumin in the toxicologic literature. It is similar in action to the toxalbumin found in rosary pea (*Abrus precatorius*). As with many proteinaceous substances, it is possible for humans to develop an allergic sensitivity to ricin. Ricin powder may be aerosolized and inhaled or ingested. It is readily absorbed by either route. It is not absorbed through the intact skin. Horses and humans are the most susceptible species but any animal may be poisoned.

Ricin is insoluble in oil so is not present in castor oil. The seeds may contain from 0.2 to 1% of ricin. The residue left after squeezing the oil may be rendered nontoxic by steam heating, which coagulates the ricin. If castor bean seeds are ingested without breaking the seed coat, they will pass through the digestive tract without causing harm. If the seed coat is broken, as little as one seed could be fatal to a child. Four seeds could be lethal for a horse. The toxic dose for a camelid is unknown.

Classical signs of ricin toxicity are a profuse watery to hemorrhagic diarrhea associated with colic and straining. However, a high dose of ricin may cause collapse and circulatory failure before diarrhea begins. Signs may appear in a few hours following ingestion, or with lower doses it may be a few days.

The syndrome in the horse begins with colic and a pounding heartbeat that may be observed at a distance. The inciting cause of the tumultuous heartbeat is fluid being withdrawn from the circulation to enter into the intestines. The packed cell volume (PCV) increases up to 60%. The blood essentially becomes a sludge that requires tremendous effort by the heart to circulate. The body

temperature begins to rise and there is a leukopenia. Clinical signs to this point would indicate a possible viral infection. Other signs include anorexia, dullness, weakness, profuse sweating, and dehydration. Extravasation of plasma from the circulation leads to hypovolemia, hypotension, and potential shock. Trembling, incoordination, and convulsions may result from the hypoxemia associated with poor circulation, or be incited by ricinin, an alkaloid also contained in the seeds.

In cattle, ruminal tympany and decreased lactation may be seen in a more chronic syndrome. In human ingestion of a biotoxin, mild poisoning causes nausea, vomiting, diarrhea, and colic. In more severe cases, the diarrhea may become hemorrhagic. Hypotension and liver and/or renal failure may lead to death. When ricin is inhaled, signs may appear in six to eight hours and include coughing, dyspnea, arthralgia, fever, and death, which may occur in 4–36 hours.

A history of exposure to castor bean seeds may be important. A hemogram indicates a leukopenia and elevated PCV associated with a fever. At necropsy, there may be evidence of seeds in the ingesta. The intestines are ballooned with fluid with petechial to ecchymotic hemorrhages on the serosa and/or mucosa. It may be possible to analyze for ricin, but this may require contacting the local poison control center to find a laboratory capable of running the test.

The only specific therapy is an antiserum that is unlikely to be available to veterinarians. Fluid therapy may be helpful in correcting the hypovolemia, hypotension, and elevated PCV. In humans, gastric lavage followed by activated charcoal may be beneficial. Oxygen insufflation is indicated.

Nitrate (Nitrite) Poisoning

Excessive nitrate may be found in water and fertilizers and from plants capable of accumulating nitrates. Nitrogen in various forms is frequently applied to forage crops as a fertilizer. All forms of nitrogen are converted to nitrates before being absorbed and utilized by plants. Nitrate poisoning is rare in camelids but has been reported as occurring in alpacas fed orchard grass hay (*Dactylis glomeratus*) containing high levels of nitrates.[5] In Australia, four alpacas died of nitrate poisoning after eating oat hay (*Avena sativa*) containing 3.2% nitrate [28].

The poisonous principle is nitrate, which is reduced in the compound stomach of foregut fermenters (cattle, sheep, goats, camelids) to nitrites. Nitrites are readily absorbed from the GI tract and disrupt hemoglobin metabolism by converting hemoglobin (Fe^{++}) to methemoglobin (Fe^{+++}). Oxygen is unable to bind to methemoglobin, thus preventing transport of oxygen to tissues. Signs of toxicity may be seen when methemoglobin levels reach 30–40%.

Clinical signs of acute poisoning usually appear within four hours of ingestion of a toxic dose of plants containing excessive nitrates. Signs include dyspnea, cyanotic mucous membranes, and a rapid, weak pulse. Venous blood is dark brown (chocolate) in color. Other signs of anoxia include muscle tremors, weakness, ataxia, and exercise intolerance. Terminally, there may be agonal struggling or convulsions.

The key to management is an accurate diagnosis. Differential diagnosis should include other hypoxic conditions such as pneumonia or cyanide poisoning. Cyanide poisoning should be eliminated as a possibility because therapy for cyanide poisoning exacerbates nitrite poisoning. Brown-colored (chocolate) blood is highly suggestive; however, poisoning with sodium chlorate (herbicide, defoliant) produces the same effect on blood.

Methylene blue is a specific antidote that reduces methemoglobin back to hemoglobin. The IV dose for SACs is 6–10 mg/kg or 1–5 mL of a 1% solution, extrapolating from the cattle dose. Be cautious because an overdose of methylene blue may actually produce more methemoglobin.

Cyanide Poisoning

A case of lethal cyanide poisoning has been reported in two alpacas that browsed heavenly bamboo (Sacred bamboo, *Nandina domestica*), an ornamental shrub which is not a member of the grass family as is true bamboo.[6]

The poisonous principle is p-Hydroxynadelonitrile, a cyanogenic glycoside. The glycoside is rapidly hydrolized in the compound stomach, to free CN^{-}.

Clinical signs are typical of signs of cyanide poisoning in livestock species, including weakness, incoordination, dyspnea, recumbency, paddling the feet, and tetanic seizures. Death may ensue within 15 minutes of the first signs. Venous blood is bright red in color. Evaluate this sign carefully because normal venous blood in SACs is brighter than in traditional livestock.

Alpacas exhibited depression, ataxia, head bobbing, dyspnea, bradycardia, and subnormal rectal temperatures (35.9 °C, 96.6 °F). Both alpacas in the cases cited above salivated profusely.

Management is difficult because of the peracute nature of the syndrome. However, cyanide poisoning is one of the few plant poisonings for which there is a specific antidote. Sodium nitrite at 10 mg/kg is administered IV to convert some of the hemoglobin to methemoglobin which, in turn, combines with CN^{-} to form a less toxic cyanomethemoglobin. The sodium nitrite is followed by the IV administration of sodium thiosulfate at 0.25–0.5 g/kg to form sodium thiocyanate from the slow release of CN^{-} from cyanmethemoglobin.

***Iphiona aucheri* (Boiss)**

Iphiona aucheri is a member of the Asteraceae (daisy) group of plants that grows well in Northeast Africa, Arabian Peninsula and Iran. In desert climates, these flowering plants grow in areas with stones, granite-based surfaces. They produce bright yellow flowers with pungent odor. During rainy seasons, they grow rapidly. When present in the paddocks of racing camels, their consumption may produce fatalities and illness in these animals [91, 92]. Pathologic analysis of these animals demonstrated centrilobular hemorrhagic liver necrosis, veno-occlusive disease and little evidence of inflammation [91]. After isolation of toxic compounds in pure forms, a diterpene glycoside was demonstrated to reproduce these same lesions in experimental mice. Other members of the Asteraceae are also associated with similar disease in ruminants [93].

***Capparis tomentosa* (Laturdei)**

Capparis tomentosa is a woody, climbing shrub growing along rivers in South-eastern Africa north as far as tropical Africa. (Figure 22.10). It has been shown to be consumed by camels, sheep, goats, and cattle resulting in a variety of clinical signs. In camels, weakness, limb stiffness, reluctance to move, fever, labored breathing, foamy discharge from the mouth. As the disease progresses, seizures may develop. Poisoning is associated mainly with consumption of the ripe fruits which look like tomatoes. The toxic principles include phytosterols, and a dipeptide acetate derivative of the sterol [94]. There are no known treatments though, B vitamins have been used with limited success.

***Trema tomentosa* (Poison Peach)**

Poison Peach is another evergreen plant that grows as small tree/shrub up to 5 m in height, with short branches, small ovate leaves and produces small dark clusters of fruit. In one report, five camels that were tethered near some roadside shrubs died within 48 hours after consuming this plant. Necropsy of two of these animals demonstrated diffuse subcutaneous hemorrhages of the thorax and abdomen as well as C1, C3 and duodenum. Hepatocellular necrosis was marked. While the authors indicated the presence of Lantana camara, Pteridium esculentum, and Poison peach plants present where the camels were tied, it was evident that the camels preferred the poison peach plants by their consumption.

Cyanobacteria

Associated with climate change and modern farming practices, the eutrophication of water bodies results in ever increasing harmful algal blooms [95]. While literature confirming the intoxication of camelids has not been forthcoming, it is suggested that all animal species, including fish, and reptiles, and humans are considered susceptible to the toxic effects of Microcystis spp., Oscillatoria spp., and Anabaena spp. [96]. Based upon the effects of cyanobacteria in dogs, decontaminating the exposed animals is very important.

Sodium Fluoride

Relative susceptibility to fluoride (F) toxicosis in the form of osteo-dental fluorosis was observed in domestic animals of diverse species living in areas with naturally fluoridated

(a)

(b)

Figure 22.10 (a) Capparis tomentosa plant that is found in southeastern Africa. *Source:* Photo by Purves, M., CC BY-SA 3.0, https://commons.wikimedia.org/w/index.php?curid=17974270. (b) The ripe fruits resemble tomatoes. Ingestion of the fruits is associated with toxicity. *Source:* Photo by Purves, M, CC BY-SA 3.0, https://commons.wikimedia.org/w/index.php?curid=16360182.

(>1.5 ppm F) drinking water. While many other species were evaluated, Camels (Camelus dromedaries) were also represented. Of these animals, evidence of dental fluorosis with varying grades were present in mature and young animals. The primary goal was to evaluate the incisor teeth (stained light-to-deep brownish color). After clinical exam, it was demonstrated that periosteal exostoses, intermittent lameness, and stiffness of tendons in the legs were associated with skeletal fluorosis. Camels were one of the less sensitive species studied whereas bovine calves were most sensitive [97].

Mycotoxins

Several mycotoxins produce disease in camelids.

Sporodesmiomycosis (Facial Eczema)

Sporidesmin is a mycotoxin produced by the fungus *Pithomyces chartarum*, and is a common contaminant on perennial ryegrass (*Lolium perenne*) [20]. The fungus is found worldwide. Sporidesmin causes hepatic necrosis resulting in photosensitization (Facial eczema). The disease is reported in camels in the UAE and alpacas in New Zealand and Australia [98]. Sporodesmins have been isolated from perennial ryegrass in the United States, but facial eczema has not been reported in livestock or camelids.

Diagnosis is based on clinical signs and history of possible exposure to plants harboring *P. chartarum*. At necropsy, the hepatic lesions are characterized by bile duct proliferation, fibrosis, and venooclusive lesions. Take note that this is a similar finding found in PAP, except that in PAP there is generalized hepatocytomegaly, which is not present in facial eczema. It is also the opinion of Australian veterinarians that both hepatic necrosis from sporodesmins and PAP may be present in the same animal.

There is no treatment for hepatic necrosis. If the necrosis is mild, the animal may live with diminished hepatic function. The animal should be kept in an environment devoid of exposure to the sun and the skin lesions treated with protectants.

Aflatoxicosis

Aflatoxicosis is caused by the mycotoxin aflatoxin which is produced by the fungi *Aspergillus flavus* and *Aspergillus parasiticus*. These fungi are ubiquitous and saprophytic, growing on a variety of grains and seeds including peanuts, corn, rice, sorghum, cottonseeds, and sunflower seeds. Fungal growth and subsequent aflatoxin production is enhanced by warm, moist, humid conditions and trauma to the seeds when harvesting. Humans are also affected by aflatoxins. Aflatoxicosis has been reported in camels [89].

Clinical signs are consistent with hepatic insufficiency. Hepatic tumors are also associated with aflatoxicosis. Immunocompromise caused by aflatoxins may foster secondary infection. Other signs include reduced appetite, weight loss, and variable icterus.

At necropsy, acute lesions consist of hepatic necrosis, bile-duct proliferation, and fatty infiltration of the hepatocytes. In chronic cases, the lesions are similar to PAP, including fibrosis and hepatocytomegaly.

Ryegrass (Ergot) Toxicosis

Perennial ryegrass is the second most popular pasture grass (second to tall fescue) in the middle and northern United States. Perennial ryegrass grows best in a cool, moist climate. It is also grown in drier climates where irrigation is available. Ryegrass is commonly infected with symbiont fungal agents that produce toxins to prevent predation by insects or other organisms (including mammals).

Perennial ryegrass toxicosis in livestock is reported commonly in the USA, Australia, New Zealand, Europe and South America. Some alpaca or llama cases with presumed ryegrass toxicosis improved over the course of hospitalization (anecdotal reports). Toxicity of forage is dependent upon the percentage of the ryegrass infected with fungal agents. Higher infection rates in grass result in higher incidence of clinical signs in the animals grazing it. Ergot and other fungi require specialized environmental conditions for maximum production. Ergot infects the ryegrass shoots as they germinate and ultimately replaces the seed head. Toxicosis is thought to occur more commonly during a rainy spring followed by hot/dry summer and fall, when the ryegrass has bloomed prior to consumption and prolonged grazing resulting in consuming grass lower to ground. In North America, ryegrass toxicosis is commonly observed from June to September and in southern hemisphere countries from December to June (the warmer months).

The clinical history of animals grazing ryegrass pasture, with evidence of improvement in the clinical condition of the animals after removal from infected pasture fits with this condition. Clinical signs may develop within 7–14 days of continuous ingestion of endophyte-infected ryegrass. Laboratory tests may be performed, but analysis of blood is generally not diagnostic of the toxin(s) and pathologic findings may be positive, but not necessarily diagnostic [99]. Prevention of over-grazing the grass during dry weather will reduce exposure to the toxin. Adding mycotoxin binders to feed was useful in reducing the impact of disease in one trial [100].

Ryegrass may be involved with different syndromes produced by separate mycotoxins:

1) Gangrene of the extremities: Ryegrass is host to ergot (*Claviceps purpurea*). Consumption of ergotized seed heads may result in gangrene of the extremities (feet, tips of ears, and tip of tail). Ergot-caused gangrene develops in the autumn following a wet spring that fosters growth of the fungus. Such gangrene has not yet been reported in alpacas or llamas.
2) Ryegrass staggers: Endophyte-infected ryegrass (*L. perenne*) can produce fungal mycotoxins (peramine, Lolitrem B, ergovaline and potentially others) that induce tremors, head nodding, ataxia, stiff gaits, that progresses to staggering and collapse [101]. Clinical disease has been diagnosed in cattle, sheep, horses, alpacas [102–104], and llamas in New Zealand, Australia (Victoria, Tasmania, South Australia, and West Australia), and the United States. For many years, the toxic agent(s) causing ryegrass staggers remained unknown. It is now believed that mycotoxins (lolitrem-B, etc.) produced by the endophyte fungus *Neotyphodium (Acremonium) lolii* is responsible for the clinical signs [99]. *N. lolii* is found concentrated in the stems, in a 2-cm (1-in.) section just above the root crown.

Affected animals may appear to be normal until excited or agitated, when the prominent sign, shaking of the head and neck, appears. Moderate signs include stiffness when walking or trying to run, swaying, incoordination, falling, and a high-stepping gait when forced to run. Severely affected animals may stand in a sawhorse stance and sway back and forth. Some animals may be unable to rise, may paddle, have marked head shaking, and have their heads pulled backward. Affected animals being driven may fall and roll down a hill or into a ravine and be unable to extricate themselves from a predicament. Clinical failures often result when animals are forced to move, and accidents occur leading to injury. Drowning is a particular hazard as animals may fall into a body of water.

Depending upon toxin uptake by plants, neurologic signs are usually observed within 7–10 days post introduction of animals. High infection rates are common in Australia and New Zealand. Initial clinical signs are mild with progressive worsening of clinical signs until the animal's removal from the pasture. Clinical signs in most animals gradually resolve with no residual effects if removed from pastures containing the source of the mycotoxin. Even animals exhibiting severe neuromuscular signs usually recover if left unmolested within one to three weeks after no access to the source of the fungus.

Phalaris Staggers

Phalaris grasses have been imported to the United States as pasture grasses and have become naturalized. Hardinggrass (*Phalaris aquatica, P. tuberosa*), reed canarygrass (*P. arundinacea*), and annual canarygrass (*P. canariensis*) grow in the United States.

Poisoning is usually seen when there is new growth of harding- or canarygrass, especially when new green shoots appear around standing dry grass. Animals seek out the tender green shoots.

In sheep, phalaris staggers is a progressive, usually irreversible, fatal brain disorder that develops two to three weeks after sheep graze on pasture, and it may develop days after removal from the pasture. In the early stages of this syndrome, the clinical manifestations are similar to those seen in ryegrass staggers, including tremors, head nodding, staggering, a high-stepping gait, and falling when excited. The differences are that an uneven heartbeat is prominent in phalaris staggers, the tremors don't abate, and the severity progresses until the animal dies. Affected sheep often walk stiff-legged and may drag their hindquarters (unable to flex the hocks). Some may walk on their knees. Animals that collapse exhibit convulsions, paddling, muscle tremors, nodding of the head, and salivation.

Cattle may also be affected, but the signs are usually restricted to stiffness of the hocks and dragging the hind toes. Only severely affected animals show intense nervous signs similar to those seen in sheep.

Phalaris poisoning has been diagnosed in alpacas in Australia in regions where sheep losses also occur from poisoning. Signs are similar to those seen in sheep. High mortality in affected animals is characteristic. Lesions in the brain are also characteristic.

No successful treatment has been found once the animal is poisoned. Many years ago, a report was published indicating that cobalt (Co) prevented the development of phalaris staggers in sheep. Though the use of Co as a preventive has been long practiced in Australia, recent investigations have cast doubt on the effectiveness of such supplementation for sheep and cattle in the United States. Nothing is known about the effectiveness of Co as a preventive in alpacas or llamas.

Fluroacetate Poisoning

Camels may be exposed to fluroacetate compounds by ingesting poisonous plants containing the compounds. Camels have been poisoned in Australia. Any camelid may come in contact with the rodenticide sodium monofluroacetate (Compound 1080). Monofluroacetic acid occurs naturally in several species of plants including *Dichapetalum cymosum* (gifblaar) in South Africa, *Gastrolobium grandiflorum* (desert poison bush) and *Oxylobium parviflorum* (box poison) in Australia, and *Palicourea margravii* (mata) in South America. No fluroacetate plants occur in North America.

Sodium monofluroacetate (Compound 1080) and Fluroacetamide were developed as rodenticides, which are extremely toxic and are banned in many countries. They may be used by professional exterminators for specific purposes in the United States. The only legal use is for collars on sheep and goats for the control of coyotes (*Canis latrans*). Unfortunately, unused packets of 1080 may persist in farm and ranch medical storerooms, or even be used illegally. In the Kreb's (Tricarboxylic acid) cycle, acetate combines with CoA to form acetyl-CoA which, in turn, is acted upon by enzymes that convert acetyl-CoA to citrate, then to isocitrate, and through systematic decaroxylation to carbon dioxide, water, oxaloacetic acid, and ATP. Fluroacetate, which itself is a relatively harmless compound, is metabolized to fluroacetyl-CoA and ultimately to the highly toxic (2R-3R)-1-flurocitrate which disrupts the cycle and causes a catastrophic loss of cellular respiration in all organ systems. A latent period of 4–24 hours allows for plant digestion in the stomach, absorption into the bloodstream, and subsequent disruption of the tricarboxylic acid cycle. Lethal dose varies with species. The western gray kangaroo (*Macropus fuliginous*) is immune to toxic effects from ingestion of fluroacetate-containing plants, while its cousin, the eastern gray kangaroo (*Macropus robustus*), a native to eastern Australia where the plants don't grow, is highly susceptible. Several species of wild antelope in South Africa are able to consume gifblaar with impunity. There is considerable difference in the lethal dose in various species. The latent period in 1080 poisoning is short (one-half to two hours). The major effects may either be on the CNS or the heart. Signs of nervous system involvement predominate in carnivores (dogs and cats), whereas cardiac effects are more common in cattle and sheep. Signs in cattle and sheep include marked cardiac arrhythmias, tachycardia, weak pulse, and ventricular fibrillation. Animals are often found dead. The clinical syndrome associated with exposure to 1080 in camelids has not been reported.

The action is biochemical and there is usually insufficient time for lesions to develop. Chemical analysis of ingesta may yield fluroacetates. Rapid death usually precludes an opportunity to treat.

Fescue Poisoning

Tall fescue (*Festuca arundinacea*) is an important pasture grass in the United States but, under certain situations, it may be responsible for mycotoxicosis from the fungus *Neotyphodium (Epichloe) typhina (Acremonium coenophialum*). The syndromes produced are as follows:

1) Fescue foot: Cattle and sheep grazing endophyte -infected pastures in the late summer and fall may develop gangrene of the extremities, similar to that caused by ergotism. Clinical signs include lameness, arched back, and diarrhea that develop within a few days to months after grazing of infected pastures. More severe signs appear as the disease progresses, including loss of body weight; emaciation; and gangrene of the tail tip, hooves, and ears.
2) Summer slump: Cattle, and possibly llamas, grazing tall fescue pastures in the summer may experience weight loss, a dull rough hair coat with failure of winter coat shedding, anorexia, and elevated body temperature (this is critical for llamas that normally may have a higher body temperature in general – the added effect of the mycotoxin could trigger hyperthermia).
3) Reproductive problems in mares: Prolonged gestation (13–14 months), abortion, difficult birth, weak neonates, high foal mortality, thickened placenta (foal may not be able to escape the amnion), retained placenta, and decreased milk production (agalactia) have all been observed in mares grazing endophyte-infected tall fescue pastures during late gestation. Foals from affected mares may have failure of passive transfer or starve from lack of milk. Agalactia has been observed in llamas in Kentucky.

Forty percent or more of the tall fescue pastures in the United States are thought to be infected with *A. coenophialum*. Pastures may be plowed up, fallowed, and replanted with endophyte-free tall fescue seed. Several endophyte-free strains of tall fescue have been developed by researchers in agronomy departments of agricultural universities. Pregnant females should be removed from endophyte-infected tall fescue 30–90 days prior to parturition. Mammary gland development should be monitored. Parturition should be attended and the cria monitored for colostrum intake and adequate nourishment. Use of domperidone in llamas and alpacas has anecdotally been useful in the increase of milk production of dams consuming fescue toxins the month before parturition. Use of the equine paste is adjusted for the animal size. One to 2 g dose for alpacas, up to 3–4 g for a llama.

Miscellaneous Plant Poisonings

See Tables 22.2 through 22.4.

Acorn Poisoning (Tannin Toxicity)

Acorn poisoning (tannin toxicity) has been diagnosed in our clinic. Predisposing factors include heavy acorn production and individual addiction. Clinical signs include constipation followed by a black, tarry diarrhea. The ultimate syndrome is that of renal failure. Other signs include

anorexia, depression, colic, GI atony, anuria, and perirenal edema. On clinical pathology, the BUN is elevated (136 mg/dL) as is creatinine (11.5 mg/dL).

Mechanically Injurious Plants

Many plants and grasses have fruits or seeds that contain barbs or hooks, which aid in their distribution within the environment. Grass awns, clover burs, and other plant parts catch on the fiber coat and work their way deeply into the coat, much to the chagrin of camelid groomers. Of greater concern are grass awns or plant foreign bodies that lodge in the conjunctival sac, ear, nostril, or mouth.

Foxtails (Hordeum spp.)

Foxtails (Figure 22.11a–c) are a special problem in the western United States. These grasses are widespread weeds of pastures and hayfields. If weedy hay is fed to llamas, the awns may lodge alongside the tongue and in the buccal cavity. The tongue of the llama is not highly mobile and rarely protrudes from the mouth. The immobility of the tongue may inhibit the clearing of foxtails from the mouth.

Foxtails have also been extracted from the conjunctival sac and the external ear canal, producing signs of head shaking, head tilt, exudation from the ear canal, lacrimation, photophobia, blepharospasm, and depression. In one case, the foxtail had penetrated the tympanic membrane, entered the middle ear, worked its way through the inner ear and the temporal bone, and finally became lodged in the contiguous brain stem, producing an abscess and subsequent death of the llama. See Chapter 9 for a discussion of ear disorders.

Bristle Grass

Another mechanically injurious plant is yellow bristle grass (*Setaria lutescens*) (Figure 22.12). In the eastern United States *Setaria* spp. is given the name of foxtail. The grass causes no problem in a pasture; in fact, it is probably good forage. The problem arises with dried grass in hay.

Figure 22.11 (a) Foxtail (*Hordeum jubatum*) plant. (b) Foxtail awn. (c) Small foxtail awns. These awns are known to migrate along the tongue in the oral cavity or become lodged in the conjunctival sac or ear canal.

The dried brittle awns of the seed head become tiny needles. Yellow bristle grass is a common weed of irrigated alfalfa hay. If the grass constitutes more than 25% of the hay, there is a risk of mechanical injury to animals that consume the hay.

The awns penetrate the mucous membranes of the tongue, lips, gingiva, and buccal cavity. Ulcers varying in size from 1 to 3 cm in diameter may be characterized by the protrusion of dozens of awn segments. The lips may be swollen. Salivation is common, and prehension and mastication are inhibited.

In a large herd of alpacas in Washington, 50 or 60 animals were affected.[7] The first sign noted in the alpacas was a major weight loss as a result of anorexia. Upon exam, the practitioner noticed ulcers and vesicles and alerted federal authorities. Fluid was withdrawn from some of the larger vesicles and submitted to an appropriate laboratory for evaluation for foreign vesicular diseases. All tests were negative for vesicular diseases.

Once the vesicle ruptured, a raw ulcer formed. Deep scrapings from these ulcers yielded awns of the grass embedded in the tissue, but awns did not protrude from the surface of the ulcer as the author has seen in horses and cattle. The awns of yellow bristle grass are normally straight, but in the moist environment of the ulcer they became spiraled like a coiled spring.

Ideally, the awn segments should be curetted from the surface of each ulcer. The application of disinfectants or other medication to the ulcers is superfluous. The weedy hay must be eliminated from the diet.

Figure 22.12 Yellow bristle grass (*Setaria lutescens*).

Tarweed (Hemazonia spp.)

Tarweed exudes a dark resin onto the leaves and stems that may stick to the fiber and skin of grazing animals (Figure 22.13a, b).

Snake Envenomation

Venomous snakes are found throughout the world and are likely to be as hazardous to camelids as to livestock species, horses, and humans. The prevalence of snakebite in camelids depends on the curiosity of the individual, the environment in which the camelid is found, and the species of snakes found in the area. Most venomous snakes are quick to move away from an encounter unless startled or in a situation wherein they are unable to retreat.

Four types of venomous snakes are found in the United States: rattlesnakes (*Crotalus* spp.), copperhead (*Agkistrodon*

(a) (b)

Figure 22.13 (a) Llama that has been grazing in tarweed (Hemazonia kelloggii). Plant resins can be seen on its face, neck, and limbs. (b) Llama with plant resins on face, neck and limbs from grazing in tarweed.

contortrix), water moccasin (cottonmouth) (*Agkistrodon piscivorus*), and coral snake, (*Micrurus* spp. and *Micruroides euryxanthus*) [4].

Copperheads are found in eastern North America, but their venom has low toxicity, and serious envenomation is unlikely to occur in camelids. Water moccasins are found in the swampy, marshy areas of southeastern North America, and camelids are unlikely to encounter them. Coral snakes are small, inoffensive, secretive snakes of Florida, Texas, and Arizona and pose little threat to camelids.

Camelids, especially juveniles, are curious and may be bitten on the nose while investigating a strange animal. Nose bites are especially hazardous to SACs because local swelling may occlude the nostrils. Because SACs are primarily obligate nasal breathers, dyspnea and suffocation may ensue.

Clinical signs of rattlesnake bite include local tissue swelling at the bite site that spreads proximally and may involve any tissue or organ. The local tissue reaction and swelling progressively becomes worse over hours to days. (Figure 22.14a–e) Sloughing of the skin may be seen in serious envenomation (Figure 22.15a and b). Bites on the limb show unilateral edema of the limb proximal to the bite [4, 105].

Systemic manifestations of rattlesnake envenomation are generally absent or minimal in large animals such as camelids. However, the bite from a large eastern diamondback rattlesnake (*Crotalus adamanteus*) could produce effects on the kidney and cardiovascular system (hypotension) as well as local necrotizing effects [4, 105].

Diagnosis may not always be clear if the bite was not observed, because trauma may produce similar signs. Supportive and symptomatic therapy may be instituted

Figure 22.14 This llama sustained a rattlesnake bite on its muzzle and developed progressive swelling. (a) Muzzle swelling four hours following bite. (b) Llama muzzle 24 hours following bite. (c) Swelling has progressed to involve entire face after llama was bitten by a rattlesnake 48 hours previously. (d) Extremely swollen face of llama 72 hours after rattlesnake bite. (e) Due to the extreme facial swelling, the animal developed respiratory distress 48 hours after the bite and a tracheostomy was performed.

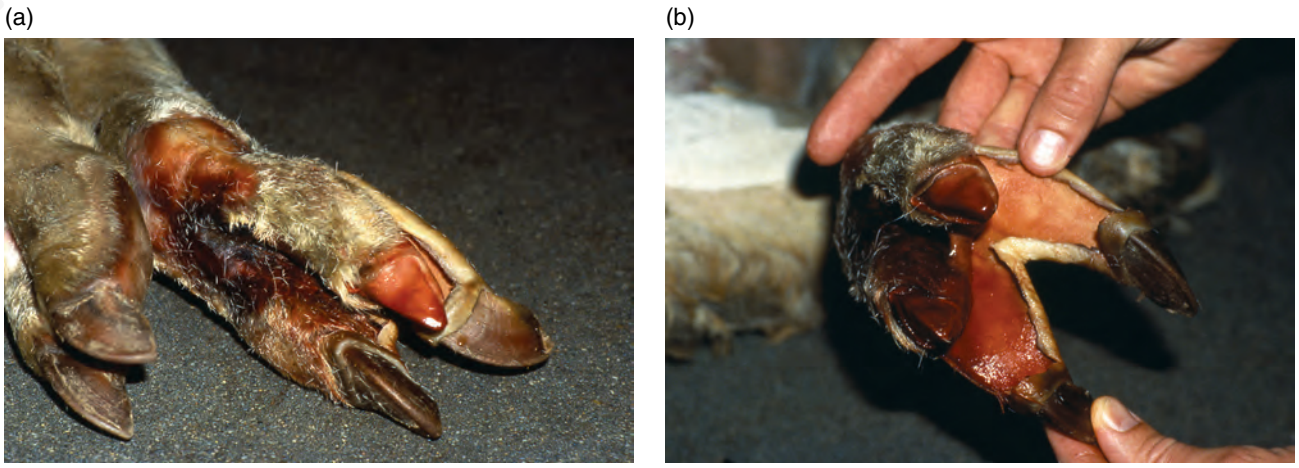

Figure 22.15 (a and b) Two examples of sloughing of the skin on the foot following a rattlesnake bite.

while monitoring the progress of the swelling. In some regions of the United States, malignant edema (*Clostridium hemolyticum*) may be a sequel to rattlesnake bite.

The only specific treatment for crotalid envenomation is the administration of polyvalent antivenin [4]. The amount of antivenin to be used depends on the amount of venom injected by the snake, not the size of the victim, and because that is not possible to determine, usually, multiple vials (2–4) should be administered IV. Antivenin is prepared from horse serum and may sensitize the camelid to future use of any equine-prepared serum product. Immediate medical treatment may require that a tracheostomy tube be placed and maintained until the animal can breathe properly with the orifice of the tube occluded; this usually occurs within two to three days. Most bites are on the muzzle, so a first-aid procedure that may be recommended is to place an 11-cm (4-in.) long, 1-cm (1/2-in.) wide tube in the nasal passageway. The swelling occurs around the tube, maintaining air flow [4]. Alternatively, the inside roller of a hair curler may be used.

Early in the course of a bite, cold water packs or spraying with water from a garden hose tends to restrict spread of the edematous process. After 24 hours, the use of warm water packs and spray is indicated to enhance circulation and drainage of accumulated fluid and tissue breakdown products. Corticosteroids have been administered but are of questionable value. Antihistamines are contraindicated because of their potential hypotensive action, which may be additive to the hypotensive action of the venom. For more details on snakebite and therapy, consult the reference cited [4].

Snakebite in camels and SACs may occur in any country where camelids and venomous snakes coexist. Camelids do not naturally inhabit the tropics or densely forested areas of the world so their being at risk from some venomous species is remote. Nonetheless, camelids may be maintained in zoological gardens in such habitats and veterinarians working in those areas should be aware of snakes in their area.

Australia, Africa, Asia, and the Middle East each have their own species of venomous snakes to deal with. The reader is referred to publications covering local snakebite or Fowler [4].

Miscellaneous Bites and Stings [4]

Occasionally, all animals are stung or bitten by insects or arachnids (wasps, bees, ants, mosquitoes, and spiders) [4]. Reactions vary but are unlikely to produce serious illness. The local inflammatory response produced by venom injection cannot easily be differentiated from contusions or foreign body penetration (slivers). Multiple bee or wasp stings may produce systemic manifestations, and horses and cattle have died from the envenomation when subjected to a swarm attack from a disturbed hive of bees or wasps. Africanized honeybees (*Apis mellifera adamsonii*) are now in the United States. They are no more toxic than European honeybees (*A. mellifera*) but are much more aggressive. Fire ants (*Solenopsis invicta*) found in the southeastern United States, have produced serious injury and even death to livestock tied near an anthill and unable to flee. Neonates and moribund animals are especially at risk. Camelids are at the same risk.

There have been no reports of scorpion envenomation in camels, but it is likely that stings have occurred or will occur if camelids are exposed to these arachnids. Likewise, envenomation from the black widow spider (*Latrodectus mactans*) or the brown recluse spider (violin spider,

Loxosceles reclusa) has not been reported. Both of these spiders are poisonous to humans but are unlikely to inflict other than transiently painful bites to animals. The bite of the brown recluse spider (*Loxocoles* sp.) causes a slow-healing ulcerous wound in humans, while the black widow spider causes muscle spasms and CNS depression.

Blister Beetle Toxicity

Blister beetle (Epicauta) intoxication was reported in two, 2.5-year-old alpacas from the same farm [22]. There were approximately 200 alpacas on this farm. The animals were fed grass pasture and alfalfa hay as well as oats and had access to trace minerals and water. The first was reportedly found down and not willing to stand for approximately eight hours. This animal was very colicky and vocalized. The second animal was also noted to prefer being recumbent but did not appear colicky. The first animal was tachycardic, tachypneic, and hypothermic, but the second animal was only hypothermic. GI sounds were reduced to absent in both animals. Clinical pathology noted in these two animals included: severe dehydration, hemoconcentration, and leukocytosis, acidosis, elevated AST, BUN, creatinine, glucose, creatinine phosphokinase and LDH. The first animal deteriorated within two hours and was euthanized. The second animal initially responded to therapy but deteriorated with dysuria and hematuria, eventually becoming leukopenic, hypoproteinemic, and hypocalcemic. Urine from the second animal was positive for cantharidin. Post-mortem exam of the first alpaca revealed lesions in the GI tract, kidneys and heart. In the second animal, lesions were most significant in the esophagus, gut compartments, lungs heart, kidneys, and bladder. In the midwestern USA, alfalfa production and the presence of beetles of the genus Epicauta are common. Feeding clean alfalfa is imperative. There is no antidote.

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23

Old World Camelids

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The old-world camelids (OWCs) include the dromedary camel (*Camelus dromedarius,* Figure 23.1), the domesticated Bactrian camel (*Camelus bactrianus,* Figure 23.2) and the endangered wild Bactrian camel (*Camelus ferus,* Figure 23.3). The Bactrian camel is adapted for the colder climate of Asia, while the dromedary thrives in the hot climate of Africa and the Middle East. Many dromedary camel breeds exist, and can come in a variety of colors, including white, brown, black, and piebald with blue eyes (Figure 23.4) [1]. The camel is highly valued in many Asian, African, and Middle Eastern cultures due to its versatility, utility, and hardiness in harsh, extreme climates. They are a beast of burden used for riding and packing purposes while also providing milk, meat, and fiber.

The worldwide camel population was estimated to be 35 million in 2019, and the rate of growth outpaces that of sheep and cattle [2, 3]. Demand for camel meat and milk products is on the rise within the camel's native locales as well as internationally. Many camels continue to be raised in traditional systems as multi-use animals, but increasing demand for camel products has driven advances in reproduction, management, and breeding strategies. The popularity and economic impact of exhibition, racing, and entertainment has also contributed to the modernization of camel husbandry.

Camel racing is a multi-million-dollar industry, where prize winnings and the sale prices of elite racing camels rival those of elite racehorses [4]. Additionally, camels are shown in pageantry events, where they are judged on their features and beauty. Competition is steep enough that entrants have been disqualified due to alleged cosmetic surgery and manipulation of the animals [5]. The races and shows are most popular in the Middle East, Africa, and Asia (Figure 23.5). Camels in North America are often used for holiday or religious scenes, as well as offering rides and attraction in zoos and parks (Figure 23.6) [6].

Introduced to Australia in the 1840s, feral camels have since thrived in this hot, arid climate and taken root as an invasive species. Primarily composed of dromedaries, the feral camel population has reached approximately 1 million, and is growing at a rate of 9% per year [7]. They are responsible for considerable environmental and economic damage. They consume and trample native vegetation, disturb natural water sources, and compete with humans and livestock for habitat [8]. Some wild camels are captured and sold, and find new purpose in tourism, racing, meat/milk production, or export to the Middle East. Despite the multiple avenues for use of these animals, the majority of population control is done through large-scale culling of adults, which often makes international news and raises animal welfare concerns from the public [9]. Managing the environmental and social impact of the feral camels of Australia continues to be a complex and controversial issue.

The medicine and management of domestic and wild camels continues to change due to their enduring and evolving relationship with humans. Modern veterinary practices such as embryo transfer and orthopedic surgery exist alongside long-standing cultural traditions and husbandry practices. The role and importance of the camel in the foundations of modern society is indisputable, and there is a continued need for veterinarians interested and knowledgeable in their care.

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Figure 23.1 Dromedary camels.

Figure 23.2 A Bactrian camel.

Behavior and Handling

Though trainable and potentially compliant companions, the camel provides significantly more challenges than the smaller South American camelids (SACs). Novice owners and handlers can be in danger of fatal injury if care is not taken to safely restrain and handle camels for veterinary procedures. If safe physical restraint is not possible for handling the camel, chemical restraint with sedation or anesthesia should be considered for the well-being of the animal and caretakers. Acceptable handling practices and restraint may vary depending on the culture and experience of the camel owners. Some of the handling techniques described in this chapter mirror those suggested for SACs in Chapter 2.

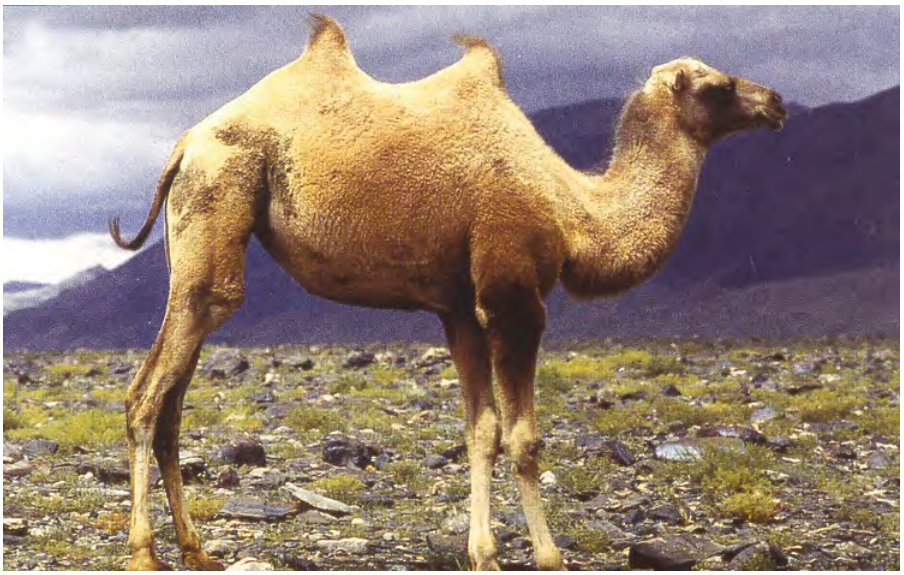

Figure 23.3 A wild Bactrian camel. *Source:* Used by permission rom George B. Schaller, Wildlife Conservation Society.

Figure 23.4 A piebald dromedary camel.

Body Posture

Camel ears are rather short compared with SACs, but ear position still reflects mood in camels. The usual ear position for a relaxed camel is with the ears vertical on the head and rotated laterally (Figure 23.7). An alert camel aims its ears forward. Frightened or angry camels position the ears rearward. The more angry the more rearward the position (Figure 23.8).

Head and neck position also reflect emotional state. A relaxed camel holds its head at approximately the same height as the hump. An alert or attentive camel positions the head 20–38 cm (8–15 in.) above the hump. When running or charging, the head is held low and extended. The tail of an agitated camel is elevated (Figure 23.9).

Vocalization

Camel vocalizations are frequent and varied. Even mild restraint is likely to elicit a roar of complaint. Unaroused camels emit a low-pitched grumble, but when annoyed, the pitch rises to a whine or roar. Handlers should become accustomed to changes in vocalization, as the escalating sounds are often indicative of escalating displeasure. These sounds usually precede spitting, kicking, or other aggressive behaviors.

Females and their offspring exchange a variety of "hums" similar to SACs, depending on the situation. The vocalization of adult females interacting with each other varies from a growl, to a roar, or bellow, depending on whether they are contented, angry, or frightened. The older females in a group are usually less vocal than the younger ones. Though camels do not have an alarm call as the SACs do, a camel may emit a bark-like sound when excited or nervous. Bull camels in rut have characteristic vocalization described later in this chapter in the reproduction section.

Offensive and Defensive Behaviors

Camels have been domesticated for thousands of years and are easily managed if accustomed to being handled properly; however, improperly trained adult camels may inflict

Figure 23.5 Camel racing.

Figure 23.6 Privately owned camels at a farm in Texas.

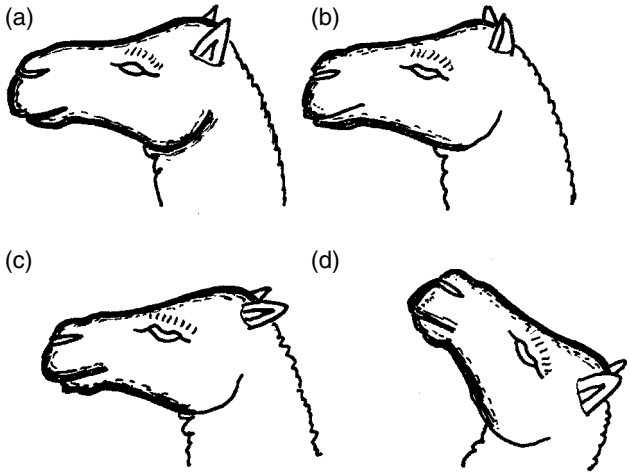

Figure 23.8 Diagrams of camel ear and head positions. (a) Usual ear position, (b) alert or attentive position, (c) upset or angry position, (d) threat display or flehmen.

Figure 23.7 Head of a relaxed camel, ears vertical and turned laterally.

serious or fatal injuries to an unsuspecting handler. Even well-trained adult bulls may become belligerent and dangerous while in rut, so intact male camels should always be regarded with caution.

Spitting

Though camels may project saliva from the mouth, more commonly gastric contents are spewed. Spitting is not used to intimidate or threaten herdmates, as in SACs, but both Bactrian and dromedary camels are capable of spewing first-compartment contents at people when they become

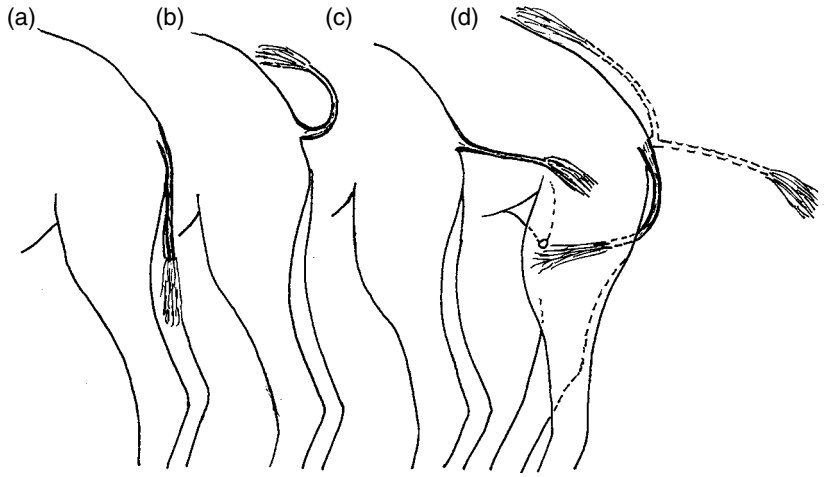

Figure 23.9 Diagrams of the tail set of camels. (a) Normal position, (b) excited, angry, or pregnant, (c) pregnant, (d) tail flipping of male camel in rut.

Figure 23.10 Camels can open their mouths widely.

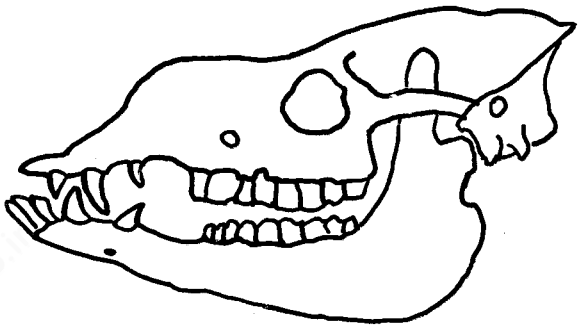

Figure 23.11 Prominent canine teeth in the camel.

angry or frustrated. While one may predict the projectile pathway in SACs, camels are able to spit out of the side of the mouth to spray a handler standing at their side.

Biting

Camels are able to open the mouth wider than any other ungulate, making them capable of grasping any part of the human body, including the head (Figure 23.10). Adult male camels have large canine teeth, which are employed in intraspecies aggression with other males vying for a female (see Chapter 12) (Figure 23.11). A prelude to biting may be the snapping together of the teeth, which is part of a threat display. When a camel bites, it clamps onto the object and begins to shake his head. This may cause considerable contusion and severe lacerations. There are reports of children being grasped by the head, lifted off the ground and killed by this behavior, in addition to severe injuries to adults [10]. When adult bulls in rut resort to serious fighting, they may bite their opponent on the head, neck, legs, and scrotum.

Orphaned male calves raised without other camels or with inappropriate human contact may exhibit aberrant behaviors similar to those seen in SACs (sometimes referred to as "berserk male syndrome") (see Chapter 2). It is theorized that when maladjusted males become sexually mature, they may treat a human as they would another male. Camel raisers should allow camel calves to live with other camels in order to develop appropriate social skills. If necessary, milk and veterinary care should be supplied by caretakers, but overly-affectionate interaction should be avoided.

Kicking

All four feet and legs may become formidable weapons. The front legs may strike out in any direction. The lack of a skin fold forming the flank provides extreme mobility for the rear limb giving the camel an extended range of movement compared to ruminants, and the rear legs are able to reach forward to scratch the head. Thus, there is no safe place to stand near an untrained or belligerent camel. Other aggressive behaviors include charging and bumping a person. If the victim is knocked down, which is likely, an aggressive camel may stomp on the person, as they would do to another camel that had been knocked down during a fight.

Restraint

Information on the restraint of camels is needed because these animals are now being raised more frequently by private individuals in North America who may not have experience with proper safe handling of these animals. Handlers must be continually alert when working around even tame camels, as with other large animal species.

Numerous types of chutes and stocks have been used to restrain camels. Camels can be handled in standard equine stocks (Figures 23.12 and 23.13). In this design, both the front and rear gates are padded to minimize trauma should the animal kick or strike and to protect the examiner. A heavy, soft cotton rope stretched between the bars and attached to the loops provided may restrict forward and rearward movement. Sturdy pipe should also be used. This stock will accommodate the average female camel. The design in Figure 23.14 was constructed at a private camel farm. The heavy pipe construction provided adequate strength for containing the camels, but its original design with double gates in front and back with 91 cm (36 in.) between the side frames allowed too much movement within the stock. It also did not provide adequate access to the rear of the animal for rectal examination. Fowler recommends a modification of the original design, with a single swinging gate at the rear and a curved front gate (Figure 23.15), only 76 cm (30 in.) between the side

Figure 23.12 Camel in equine stocks.

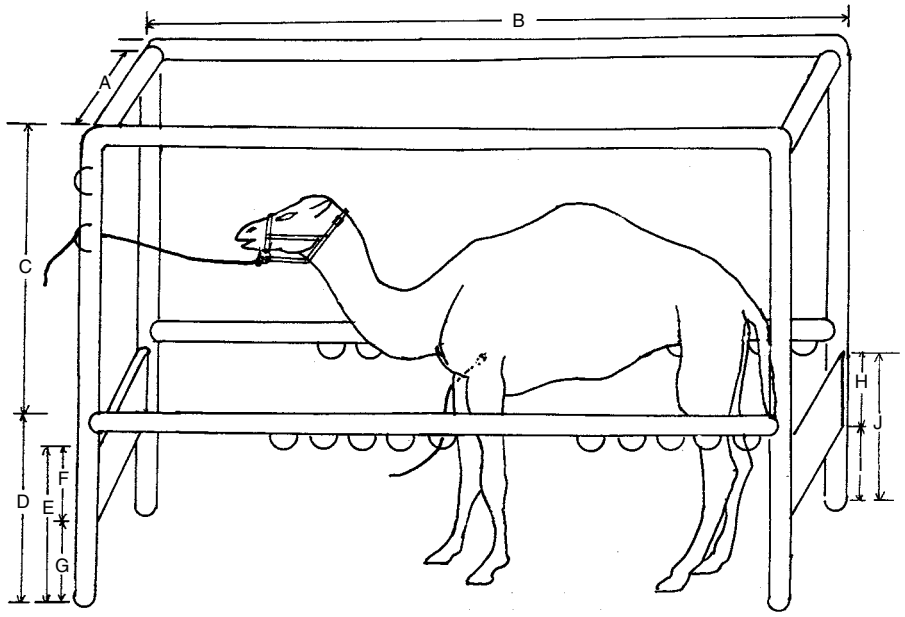

Figure 23.13 Diagram of an equine stock, including measurements. (A) 76 cm (30 in.), (B) 2.1 m (84 in.), (C) 1.8 m (70 in.), (D) 1.2 m (48 in.), (E) 1 m (41 in.), (F) 53 cm (21 in.), (G) 51 cm (20 in.), (H) 53 cm (21 in.), (I) 36 cm (14 in.), (J) 89 cm (35 in.). Pipe is 7 cm (3 in.). Front and back gates padded.

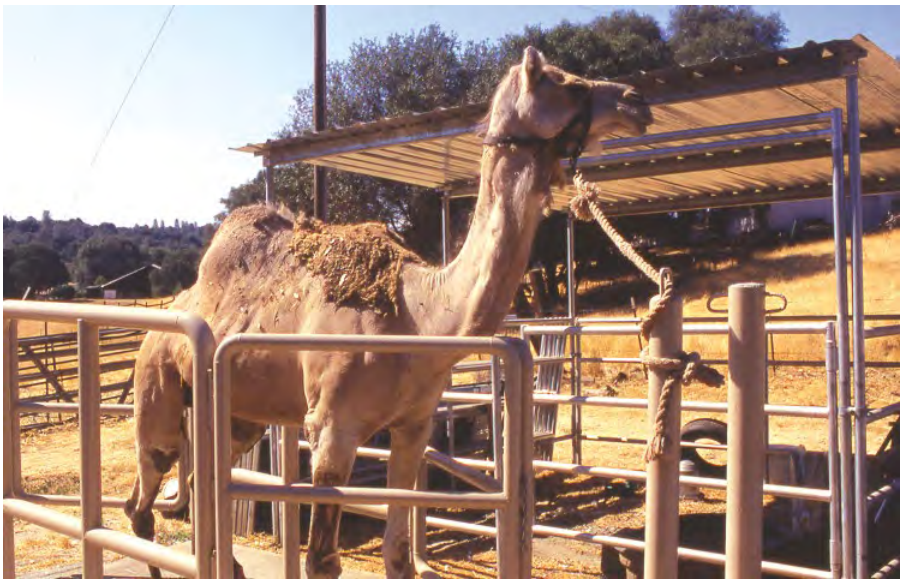

Figure 23.14 Camel in California-designed stock. There is too much room in which to move around.

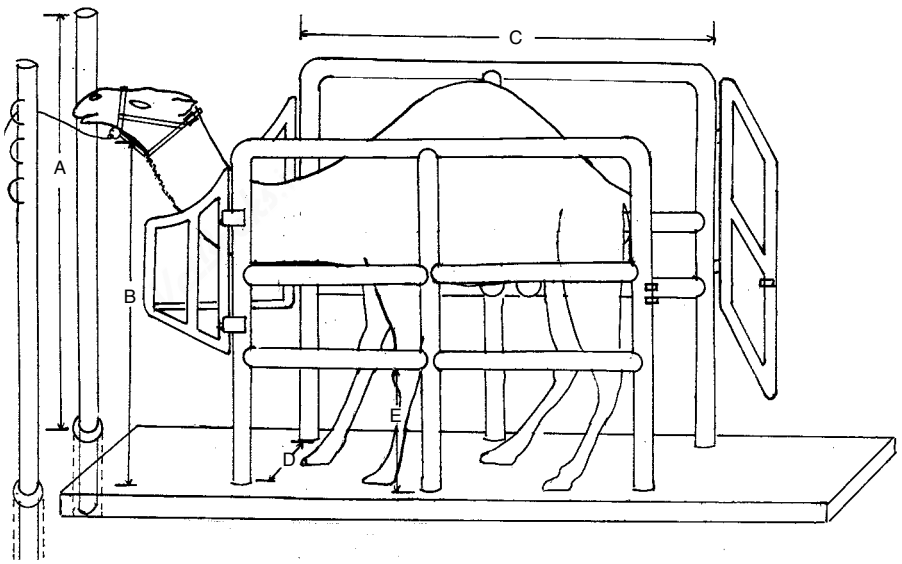

Figure 23.15 Diagram of a modified California-designed stock, including measurements. (A) 2.1 m (84 in.), (B) 1.6 m (64 in.), (C) 2.1 m (84 in.), (D) 76 cm (30 in.), (E) 61 cm (24 in.). Pipe, main frame, 8 cm (3 in.); gates, 6.4 cm (2.5 in.).

panels. Some extremely large animals may have difficulty in fitting into that narrow space, but for a stock to be effective, side-to-side motion must be limited. A stock used in a camel facility in the United Arab Emirates is illustrated in Figure 23.16. These were designed primarily to examine the reproductive tract and specifically to conduct embryo transfers. Fowler's preferred stock is a simple design that does not require the use of pipe. The Fowler design may be constructed next to a barn or solid wall, as illustrated in Figure 23.17. A heavy post is set approximately 45 cm (18 in.) from another post that is next to the wall. A 2.5-cm (1-in.) thick sheet of marine plywood 1.2 m (4 f) by 2.4 m (8 f), is hung on the post with heavy bolt hinges. An alternative type of construction is with both sides of the chute hung on posts so that access

Figure 23.16 Stock used for embryo transfer studies in Al Ain, United Arab Emirates.

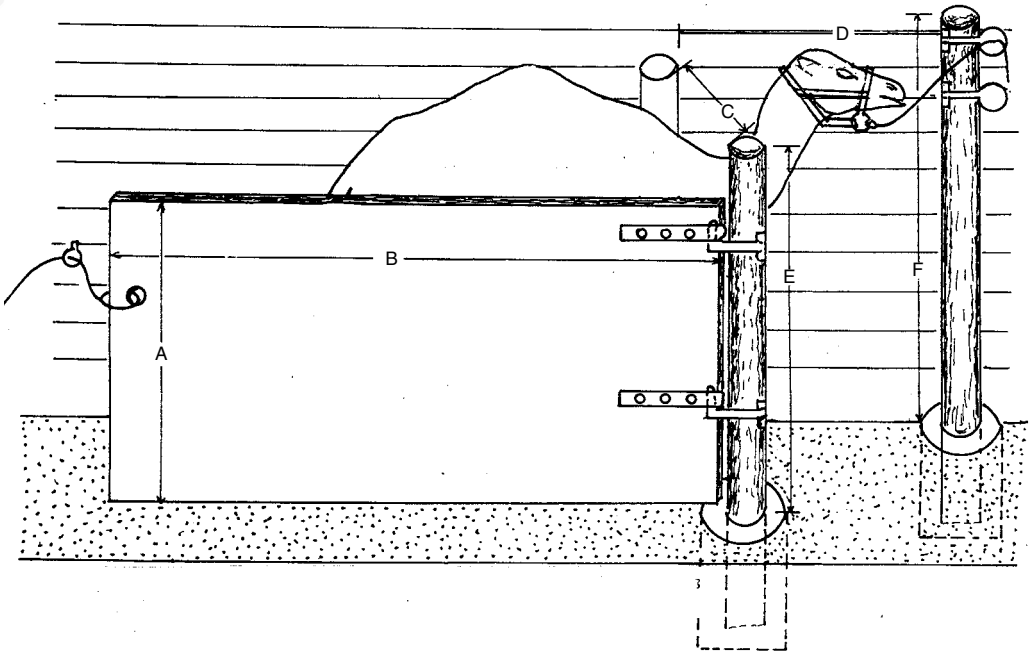

Figure 23.17 Diagram of Fowler-designed stock. (A) 1.2 m (48 in.), (B) 2.4 m (96 in.), (C) 46 cm (18 in.), (D) 91 cm (36 in.), (E) 1.8 m (72 in.), (F) 2.1 m (84 in.). Posts 15 cm (6 in.) in diameter.

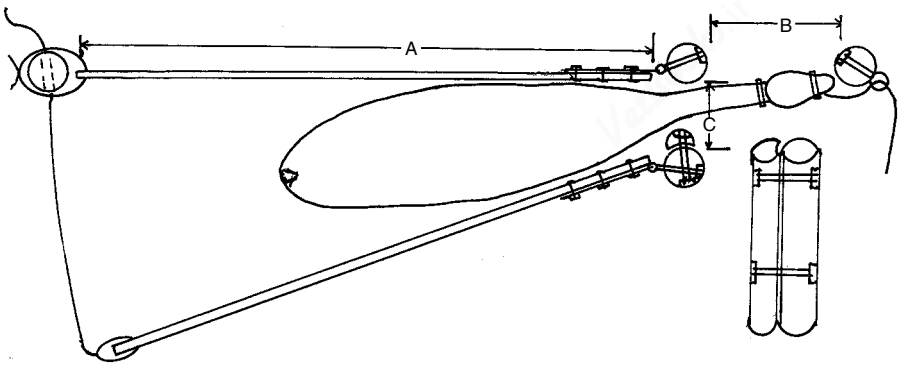

Figure 23.18 Fowler design, top view. (A) 2.4 m (96 in.), (B) 91 cm (36 in.), (C) 46 cm (18 in.).

may be from both sides (Figure 23.18). Arrangements may be made to attach another post on the inside of one post to narrow the opening for smaller camels. A post set in the front allows the animal to be tied, thus restricting backward movement. Once the camel is tethered, the gate may be swung closed, restricting side motion. Bales of hay or straw may be placed behind the camel if a rectal examination is necessary. Figure 23.19 illustrates the principle of this design using an aluminum gate, with the camel against a wire fence. The chance of an injury to the camel is greater with the aluminum gate. Any of the open-sided stocks may be made more kick-proof by lining the stock with plywood sheeting. Bales of hay or straw protect the examiner from kicking. Figure 23.20 illustrates another example of gates being used to create a chute for vaccinating a herd of camels. This set up is convenient for quick injections, but would be insufficient for more involved procedures.

The best-designed stock or chute is useless unless the camel will enter it. The key is the placement of the stock or chute in relation to the corral design. If the chute is located in an area that is strange to the camel, or if the camel is not trained to walk into the chute, handlers will be unable to coerce an adult camel to enter. Untamed camels are a special problem. This is often the situation in zoos or breeding facilities where little attention is paid to the animals except when something is to be done that is unpleasant for them. A special catch pen should be provided in these facilities to allow the animals to be brought from a larger enclosure or pasture to a more manageable enclosure. Preferably, the camels have been fed at least part of their feed in the catch pen, so they are willing and accustomed to entering it.

Figure 23.19 Temporary stock using an aluminum gate to press the camel toward a fence. Bales of straw protect the examiner.

Figure 23.20 Gates used for containment of camels for vaccination.

Physical Restraint

Positioning a camel in sternal recumbency (kushing) provides an opportunity to closely examine or collect laboratory samples without risk of being kicked or struck. Obviously, this necessitates a camel being trained to kush on command. For many camels, this includes the verbal command "kush" alone or combined with pulling the head gently toward the ground. The camel may be secured in the sternal position by using a body harness. Either a leather strap or rope is used to place a loop or figure-8 around the front limb when the leg is flexed at the carpus. Using only the loop around the front leg, the camel may still rise to its knees. Many camels are trained to allow hindlimb physical restraint as follows: A person on either side of the camel brings a soft cotton rope up behind the hind limbs as the camel is being directed to kush; optimally, the rope should be below the fetlock (Figure 23.21). Once recumbent, the rope is placed medial to the stifles and tied tightly over the back behind the hump (Figure 23.22). Rising on the forelimbs is prevented by placing a loop over a flexed limb, extending the rope over the top of the neck, and securing the opposite forelimb in the same manner (Figure 23.23).

Head Control

The method of catching a camel and controlling the head varies with the experience and culture of those carrying out the procedure. Camels may be roped with a lariat, but the roper needs to keep the loop as high on the neck as

Figure 23.21 The rope is brought up behind the pasterns or hind legs.

Figure 23.22 The rope is placed beneath the pasterns and on the medial side of the stifles, then tied over the back.

Figure 23.23 The strap is placed around both front legs and over the top of the neck.

possible to provide better control of the head. A loop may be placed around the neck to pull the head toward a barrier, thus allowing placement of some type of head restraint. In countries where camels have been beasts of burden for centuries, a head restraint may consist of a wooden peg placed through the side of the nostril (Figures 23.24 and 23.25). A small cord is attached to the peg. Pressure on the peg signals to the camel a desired response. Usually, the tactile contact is accompanied by a verbal command. If the peg is pulled too vigorously, it may be pulled through the skin. Sometimes, the wooden peg is replaced with a short rope loop, left permanently in place, to which a lead rope may be attached. Another means of head restraint is to secure a loop of rope around the lower jaw, caudal to the canine teeth and under the tongue. The foregoing restraint practices are controversial.

Figure 23.24 Wooden nose plug.

Figure 23.25 Nose plug in place.

Figure 23.27 Bridle on a driving camel. The strap from the bit over the bridge of the nose keeps the bit away from the lower canines.

Figure 23.26 Camel wearing a horse halter.

In North America, South America, and Europe, the head is usually restrained by placement of some type of halter. Camel handlers should learn how to tie a bowline knot because it is fundamental to producing a non-slip or non-tightening loop in many restraint situations. A camel should never be secured to a solid object with a lariat loop or any other knot that can tighten when pulled, as the risk of a camel becoming frightened, pulling back, and strangulating is great. Halter designs are varied. Halters may be constructed from rope, synthetic fibers, or leather. Camels are obligate nasal breathers and may suffocate if the nasal passageways are constricted from an ill-fitting halter. Horse halters can often be fitted appropriately to camels and often fit more comfortably than cattle halters (Figure 23.26).

The cheek pieces should be positioned to not rub against the orbit. The halter material and all buckles, snaps, and other attachments must be strong enough to hold the camel should it become frightened and pull back. Snaps are often the weakest link in the system. Quick-release snaps that can be released while under pressure are most suitable. Halters are rarely left on camels constantly for fear that they may catch on a post or other protruding object. Neckbands may be left in place, serving as a means of identifying or catching an animal.

The use of a bridle is controversial. The presence of the canine teeth allows a camel to grasp the bit, decreasing its function of producing pressure on the commissure of the mouth. The bit banging against the teeth may annoy the camel and cause it to continually shake its head. A chin strap over the bridge of the nose has been shown in some cases to hold the bit near the roof of the mouth (Figure 23.27) and may be used to eliminate head shaking.

A lip twitch, performed by grasping the upper or lower lip, may be used when examining a camel. The ears are too small to be used, as can be done for llamas or alpacas.

Tethering

Camels should be trained to stand quietly when tied to a post or other solid object. Handlers should learn to tie a quick-release knot, that allows release of the knot even while under pressure. Camels may be tethered from the halter, or by a front leg. A leather or fiber strap is attached to the leg above the ankle, and the tether is attached by means of a "D" ring on the hobble. The opposite end of the rope may be attached to a post, stake, or metal ring embedded in the ground. Commercial camel trekkers usually tether to a metal ring that is augured into the ground by means of a spiral coil. It is also possible to tether to a picket line (a rope stretched between two secure ends), either on the ground or elevated at 1 m above the ground.

Hobbles

Hobbles may be employed to minimize straying when animals are released to graze while on trek (Figure 23.28). Usually only the forelegs are secured to one another, but some camels may learn to gallop using a short stride, with only the front legs hobbled. In that event, it may be necessary to hobble one forelimb to a hind limb. Hobbles may be leather straps with buckles that surround each ankle and attach to the opposite leg by a short length of chain. They may also be fashioned from soft cotton rope, (1.5 m [5 ft] long and 1.27 cm [0.5 in.] in diameter). The rope is folded in half around one front leg. The two ends are twisted together for three or four turns, then the ends are tied around the opposite leg using a square - bow knot. The use of a hobble may be important in preventing a camel from striking a handler when protective barriers are not available. A hobble may also be placed between the hind legs, but extreme caution must be exercised to avoid being kicked while applying the hobble.

Figure 23.28 Rope hobble on front legs.

Sedation

Even with effective physical restraint, sedation may be necessary to perform certain tasks on camels. The doses and combinations of sedation do not vary wildly from those used in SACs, but some sample protocols (Table 23.1) and notes about specific drugs are included. An accurate weight should always be obtained if possible. Estimation of bodyweight by inexperienced practitioners is typically lower than the actual weight of the camel, and doses should be adjusted accordingly.

α-2 Agonists

Like SACs, camels are more resistant to the effect of α-2 agonists than ruminants. Xylazine, romfinidine, metdetomidine and detomidine have all been used with success in camels. They can also be combined with other classes of sedatives for synergist effects. The author frequently uses

Table 23.1 Sedation.

Drug/Combination	Dose	Route	Notes
Xylazine	0.05–0.25 mg/kg	IV, IM	Camel will typically be sedate but remain standing [11].
	0.25–0.5 mg/kg		Camel may become recumbent.
	0.5–1 mg/kg		Camel will typically become recumbent.
Xylazine + ketamine	0.15 mg/kg + 2.5 mg/kg	IM	Sedation and analgesia [12]
	0.4 mg/kg + 0.4 mg/kg	IV	Sedation and recumbency adequate for castration [13]
	1–2 mg/kg + 1–2 mg/kg		Induction of anesthesia [14]
		IV	
Butorphanol	0.2 mg/kg	IV	Butorphanol alone will produce sedation and analgesia in camels [15], but the author often uses it at lower doses in combination with xylazine and ketamine
Medetomidine	6 – 20 μg/kg	IV	Produces sedation, analgesia, and recumbency [16, 17], reverse with atipamezole 30 μg/kg IV [16]
Romifidine	40–80 μg/kg	IV	Standing sedation
	120 μg/kg	IV	Recumbency [18]
Detomidine	25–75 μg/kg	IV	Higher doses cause profound sedation [11]
Midazolam + ketamine	0.2 mg/kg + 2 mg/kg	IV	Produces recumbency and immobilization for short procedures.
Midazolam + propofol	0.2 mg/kg + 0.75 mg/kg	IV	Induction and maintenance of surgical anesthesia [11]

Table 23.2 Darting combinations.

Drug/Combination	Dose	Route	Notes
Medetomidine Ketamine	0.22 mg/kg 2.54 mg/kg	IM dart	Reverse with atipamezole 0.24 mg/kg [20]
Medetomidine Ketamine Butorphanol	0.12 mg/kg 2.3 mg/kg 0.05 mg/kg	IM dart	Reverse with atipamezole 0.23 mg/kg and naltrexone 0.17 mg/kg [20]

combinations of xylazine, ketamine, and butorphanol at similar doses of the "ket-stun" used in ruminants (with higher doses of xylazine typically selected). Individual animal response will depend on route (intramuscular [IM] versus intravenous [IV]) and excitability at time of administration. IM administration often provides longer duration of effect, but also a longer time to reach sedation and milder sedative effects than the IV route.

Xylazine alone at 0.4 mg/kg IM has provided sedation satisfactory for surgeries of short duration [19]. Xylazine at 0.4 mg/kg with ketamine at 0.4 mg/kg IV provides recumbency and sedation adequate for castration [13]. Lower doses of xylazine at 0.15 mg/kg with 2.5 mg/kg of ketamine IM can also provide sedation and analgesia in field conditions [12]. Additional combinations and dose ranges used in domestic camels are provided in Table 23.1. Published combinations for darting free-ranging camels are provided in Table 23.2.

Reversal of α-2 agonists may be preferred or indicated. Though tolazoline is not recommended for use in SACs due to potential for adverse reactions and cardiovascular collapse, it has been used and recommended at a dose of 1–2 mg/kg in camels [21–23]. The author has experience with a camel reacting adversely to tolazoline administration. Yohimbine at 0.1–0.25 mg/kg and atipamezole at 0.03 mg/kg–0.24 mg/kg (typically three to five times the dose of medetomidine) have been safely used for a variety of anesthetic protocols in camels [16, 21, 24].

Local anesthesia including epidurals and nerve blocks, can be performed similarly to other species (see Chapter 6). Lidocaine (0.22 mg/kg) alone or in combination with other drugs (ketamine 1 mg/kg, xylazine 0.05 mg/kg) has produced successful anesthesia in caudal epidurals [25].

General anesthesia is recommended for prolonged, painful, or involved procedures. Premedication, induction, and maintenance of anesthesia can be performed similarly to SACs (See Chapter 6). The dulaa, a diverticulum of the soft palate in the dromedary bull, may sometimes become exteriorized during intubation and is not cause for alarm. Camels are reportedly slower to rise after recovering from isoflurane anesthesia than horses and cows [26]. This has been the author's experience, as they typically remain quietly in a kush position until ready to rise of their own accord (Figure 23.29).

Figure 23.29 Camel recovering from anesthesia in a stall.

Routine Veterinary Care

Physical Examination & Normal Parameters

The thoroughness of a physical exam of a camel will depend on the tractability of the individual. They may resent handling of the mouth and head, so exam of mucous membranes can be challenging. The mouth is often pigmented, so the conjunctiva is recommended for accurate assessment of pallor.

Normal temperature may vary from 96.3–102° F (35.7–38.9° C), and is highest in the afternoon [27]. Heart rate is typically 35–50 beats per minute in the relaxed camel [27]. Frequency and strength of first-compartment contractions is similar to SACs and is best heard in the left paralumbar fossa. Stress during exam and handling may produce mild and transient diarrhea.

Vaccination

Many camels in the world are not routinely vaccinated, and there are no vaccines labeled for camels in North America. Vaccination of camels may be necessary in certain areas, dependent on disease and epidemiologic risk factors. Private camel owners frequently request vaccines due to familiarity with diseases of other species and concern about risk for their camels. Due to the lack of a core vaccination series established for camels, and the variability of disease exposure across many different countries, a herd vaccination plan will be dependent on the judgment of the veterinarian and the client's perception of risk factors.

Clostridial Diseases

Combined *Clostridium perfringens* type C&D and tetanus vaccines are the most commonly used vaccines in camelids in the United States, though other multivalent *Clostridial* vaccines are also utilized. Tetanus has been reported after castration and puncture wounds of the foot of camels, and routine vaccination against *Clostridium tetani* at a minimum is highly recommended [28, 29]. The choice of which combination of *Clostridial* bacterins to utilize will depend on management and environmental risk factors of the individual herd. Given that camelids are potential hosts for flukes, protection against *Clostridium novyi* and *Clostridium haemolyticum* can be considered in fluke endemic regions.

Rabies

Rabies infection can occur in camels in contact with infected wildlife, and outbreaks have been reported in the Middle East and Asia [30, 31]. Recently, a canine rabies vaccine was used on Bactrian camels in the face of a rabies outbreak in China, with vaccinated animals reportedly achieving satisfactory titers [30]. Many American owners request the vaccine due to their camels having contact with the public, but due to the lack of labeling and evidence of efficacy, an animal that bites a human may not be considered vaccinated appropriately by public health officials. In areas where camels have the potential to be exposed to infected wildlife, rabies vaccination is recommended. The author uses a product labeled for other large animals.

West Nile Virus

Camels are known to develop titers against West Nile Virus, but it had not been isolated from a camel until recently [32]. Though they may seroconvert, clinical disease is not reported in camels and evidence for routine vaccination is weak.

Bovine Viral Diarrhea Virus (BVDV)

While the effects of BVDV infection, including persistent infection, has been well documented in SACs, some question still remains about its role in camels. Though the effects of the virus on camels may be low, they could serve as a potential source of transmission to cattle and other ruminants. Routine vaccination is not currently advised until more information is available.

Equine Herpes Virus-1

Fatal herpesvirus encephalitis has been reported in a camel housed near equids [33]. Due to the more widely known pathogenic potential of EHV-1 and West Nile Virus in SACs, it has been the author's experience that some owners may request "all of the vaccines" or "the horse vaccines" when discussing camel care with their veterinarian. Risk of clinical disease from infection with equine herpes virus and West Nile virus is low, and routine vaccination does not seem warranted. A situation where the camel may be exposed to carrier equids could prompt additional consideration for vaccination.

Middle Eastern Respiratory Syndrome

The use of a killed equine herpes virus product would be appropriate (albeit, no safety studies have been conducted) where susceptible camels are in contact with horses. Vaccines against the coronavirus responsible for MERS are in development, and one vaccination has been shown to provide complete protection in alpacas and reduce viral shedding in camels. It is not commercially available or recommended at this time [34].

Anthrax

Anthrax is reported in camels in some regions and vaccination is employed for control and prevention.

Blood Collection

Blood collection and IV injection is most easily achieved via the jugular vein. Unlike the llama and alpaca, the vein is visible when occluded and distended, and is technically easier to access. Success is reliant on adequate restraint and safety, which is often more challenging than the act of venipuncture. IV catheterization is performed similarly to SACs, with respect for the thick cervical skin.

Fecal Examination and Deworming

Camels are vulnerable to severe clinical disease from endoparasitism, and vigilant and proactive monitoring of individual animal fecal egg counts is strongly recommended, especially in North American camels that are often subjected to resistant parasites and indiscriminate deworming practices. A quantitative fecal flotation should be considered part of a routine exam of a healthy camel and is absolutely required if illness is suspected. Whipworms and *Haemonchus contortus* infection are common causes of diarrhea, lethargy, inappetence, and weakness in North

American camels. Even very low numbers of whipworms on fecal egg counts are considered significant. An in-depth discussion of parasitism and management strategies in domestic camelids is available in Chapter 7.

Reproduction

Normal Reproductive Behavior and Physiology

The anatomy and basic reproductive physiology of camels is similar to that of SACs (see Chapter 16), with the main difference being the existence of a breeding season of both sexes. Bactrian and dromedary male camels undergo a seasonal rut period, lasting weeks to months, characterized by heightened aggression, secretion from the poll glands, dribbling of urine, flicking of the tail to splash urine onto the back, and vocalization. Weight loss is typical because the male expends so much energy in establishing and defending his harem. Inappetence may also be a factor in weight loss.

The poll gland is a scent gland unique to bull camels. The gland secretes a brownish to black fetid fluid that mixes with dirt and urine during rut to mat the hair on the back. If allowed to do so, a male camel in rut may roll and attempt to rub the poll gland against the dirt to scent-mark. The testicles increase in size during rut, accompanied by an increased testosterone level in the blood. Male dromedary camels have a diverticulum on the lingual aspect of the soft palate (dulaa, dulla, dulah) which may be inflated with air and be extruded from the oral cavity during rut (Figure 23.30). The camel produces a gurgling sound accompanied with excessive salivation and foaming. This behavior is a threat to other males. The dulaa may also be protruded when the camel is excited or angry.

Female camels are seasonally polyestrous. The breeding season usually coincides with the rut in males. After the breeding season, the ovaries become inactive. Anestrous may extend up to six months. The ovarian cycle is a follicular wave, similar to that of SAC female. Camels are also induced ovulators similar to SACs. However, contrary to SAC females, which are typically receptive to the male unless they are pregnant, the camel follicular cycle is spread out, allowing estrogen levels to become low enough to cause the non-pregnant female to become unreceptive to the male. When estrogen levels rise, the female will again become receptive. This type of cycling is frequently, but incorrectly, termed an "estrous cycle." Follicular waves are not associated with ovulation unless the female camel is bred at an appropriate time.

The induction of ovulation is via β nerve growth factor (βNGF) in the seminal plasma of male camelids [35, 36].

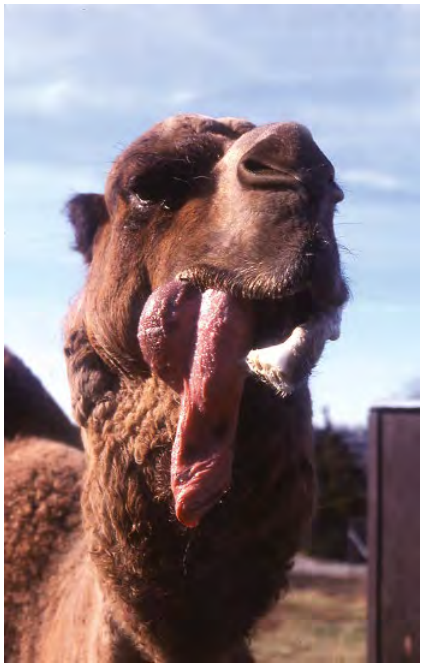

Figure 23.30 Protruding dulaa.

As in SACs, fertilization of the ovum occurs in the oviduct. The embryo enters the uterus at six and a half to seven days post breeding in camels. A heartbeat may be detected by 22 days. Organ differentiation occurs in 45–60 days. Gestation length varies from 355 to 419 days.

Assisted Reproductive Technology

Most camels in North America are bred by natural cover, but advanced techniques in reproduction have been developed and used with increasing frequency in the Middle East. Timed ovulation, artificial insemination, embryo transfer, *in vitro* fertilization and cloning have been utilized and well-studied in camels.

Commercial embryo transfer in the UAE has been used mostly for racing camels. Successful racing females often have a relatively long career, and their long calving interval limits their reproductive potential in traditional systems, thus creating a need for enhanced reproductive efficiency. Improvement of dairy camel genetics and milk production has also utilized embryo transfer [37]. Embryo collection and transfer procedures are performed similarly as in the bovine. Camel-specific challenges to successful embryo transfer include individual response to reproductive hormones (FSH, GnRH, and eCG), finding an appropriate recipient animal, and variable gestation length after transfer [36, 38]. Over a 10-year period, one commercial embryo transfer program had 27% of embryo transfers result in a weaned calf [38].

Figure 23.31 Hybrid female with shaved area due to dermatitis of the hump cleft.

Embryo transfer from Bactrian camels into dromedary recipients has successfully produced live calves, and may help with endangered species preservation [39]. Additionally, cloning of a Bactrian camel using dromedary oocytes and a dromedary recipient has been achieved [40]. Dromedaries have also been successfully cloned [41, 42].

Hybridization

All of the camel species can interbreed and produce fertile hybrids. First-generation dromedary and Bactrian hybrids are reportedly larger and stronger, with a larger, single hump that sometimes contains a cleft (Figure 23.31) [43]. They have been valued historically as draft animals, but may have a challenging temperament. Off-spring of two first-generation hybrids are reportedly extremely difficult and sterile animals that have not be useful [44].

First-generation hybrids may be bred back to Bactrians or dromedaries to produce more desirable animals. Mating choices are carefully considered depending on the desired characteristics of offspring, as the characteristics of the animal will vary depending on which species is the dam or sire. Producers in Kazakhstan have embraced hybridization in an effort to produce excellent dairy animals (a dromedary trait) that can withstand the cold (a Bactrian trait), with 80% of the country's camels estimated to be hybrids [45].

Camels can also be hybridized with SACs, and "camas" (camel/llama hybrids) have been produced via artificial insemination. The utility of these animals has yet to be defined.

Pregnancy Diagnosis

Multiple techniques for pregnancy diagnosis exist, and the preferred method will depend on the comfort of the veterinarian and the tractability of the individual camel.

Behavior

The pregnant camel will sometimes raise and curl her tail when approached by a male, and this behavior has been correlated with progesterone concentration. This behavior has been noted to occur as early as 15 days post-mating, but false positives are possible in non-pregnant animals. Female camels are not always receptive to the male, even when fertile, and lack of receptivity does not indicate pregnancy.

Rectal Palpation

Rectal palpation can be performed for pregnancy diagnosis in the camelid, but has some key difference from its use in ruminants and equids. A membrane slip will not be palpable due to the placentation type. The presence of a corpus luteum on the ovary is supportive of pregnancy. After approximately 45 days, changes in the uterus are detectable, starting with an enlarged left horn. An increase in fluid, and eventually a palpable fetus will become detectable by six months [46]. The ability to palpate the fetus may vary as gestation increases. As the uterus enlarges, it assumes a more ventral position in the abdomen, which may make transrectal palpation more difficult. Rectal tears are a potential risk in inexperienced or aggressive palpators, or if the patient is poorly restrained.

Ultrasound

Transrectal ultrasound (US) is commonly used to detect pregnancy in camels and is the most accurate method for early pregnancy. Accumulation of uterine fluid and a visible corpus luteum can support pregnancy status at 15 days. The conceptus can be imaged by 17–18 days, but is more easily detected after 20–21 days [46]. Imaging the fetus via the transrectal approach is challenging after approximately 60 days due to abdominal location. Transabdominal ultrasonography can be used to detect a fetus at six weeks. The fetus is typically imaged on midline in the caudal abdomen just cranial to the udder, and becomes visible more cranially as gestation progresses [47].

Progesterone

As in SACs, progesterone has utility in determining success of ovulation and conception. Progesterone will rise above 1 ng/mL at 3–4 days post-breeding if ovulation has been successful, and remain >1 ng/mL at 10–12 days post-breeding if pregnancy is successful [48]. An unsuccessful mating will result in a progesterone returning to below 1 ng/mL by this time. During pregnancy, serum progesterone is typically 3–5 mg/mL but may dip to 2 ng/mL [46, 49, 50]. Progesterone has also been successfully measured in milk using a bovine testing product and reflects similar values [51].

Cuboni Reaction

Chemical tests potentially provide a safer method of pregnancy detection in camels that are not routinely handled. The Cuboni reaction occurs when urine estrogens react with benzene, hydrochloric acid, and sulfuric acid. This method reportedly has a sensitivity of 83.3% and a specificity of 91% but is most accurate after 230 days of gestation [52].

Pregnancy and Parturition

As in SACs, normal gestation length in camels is variable, and ranges from 355 to 419 days. Rate of twinning is low and pregnancy is almost exclusively in the left horn of the uterus. The most common reasons for dystocia are fetal malpositioning and uterine torsion, though uterine inertia, incomplete cervical dilation, and fetal monsters are reported [53]. Pregnancy loss due to infectious causes is documented [54]. Cesarean section may be required in cases of dystocia, and is described later in the surgery section.

Neonatal Care

Care of newborn calves should be approached similarly to care of the cria. Prematurity, weakness, hypothermia, failure of passive transfer and sepsis threaten the viability of the calf. In some pastoral communities, colostrum intake is limited, which contributes to morbidity and mortality in the neonatal period [55, 56]. New camel enthusiasts may not understand the importance of colostrum ingestion for immunity and disease prevention, and producers should be advised on normal neonatal behavior and when veterinary intervention is indicated. Commercial llama plasma has been used for the treatment of failure of passive transfer in American camel calves.

Diseases of Camels

Metabolic Bone Disease

Camels may experience lameness and bone disease due to improper dietary management. The cause and presentation will vary based on the geography and diet of the camel. Appropriate nutrition and recognition of signs of deficiency are key in raising healthy camels in captivity.

Nutritional Secondary Hyperparathyroidism

Nutritional secondary hyperparathyroidism causes fibrous osteodystrophy in captive camels due to chronic dietary imbalances. This is typically driven by a dietary deficiency of calcium and vitamin D, which may be coupled with an excessive intake of phosphorous. Concurrent gastrointestinal (GI) parasitism may contribute to inadequate calcium absorption from the intestinal tract. The camel will increase secretion of parathyroid hormone, which promotes resorption and remodeling of normal bone, in an effort to maintain calcium homeostasis. Affected camels most commonly present with lameness or swelling of the maxilla or mandible. Multiple reports of camels diagnosed with nutritional secondary hyperparathyroidism are documented in the literature, and the author has encountered them occasionally in clinical practice [57, 58].

Radiographs frequently reveal osteopenia of affected bone. Serum biochemistry typically reveals normal or decreased total calcium, elevated phosphorous, and increased alkaline phosphatase (ALP). Diagnosis is typically made based on history, clinical signs and compatible changes on radiography. Biopsy or necropsy can be performed to definitively identify fibrous dysplasia.

Clinical signs may progress despite calcium supplementation and dietary modification [58]. Even if progression of the disease is halted via successful treatment, the acquired deformities are typically permanent. Utilizing alfalfa hay instead of grass hays, and reducing or eliminating grains to reduce to phosphorous has been successful [57]. Supplementation of additional calcium via limestone has been attempted.

Though renal secondary hyperparathyroidism may cause similar lesions in other species due to excessive serum phosphorous as a result of impaired renal function, there are no reports yet of this occurring in camels.

Rickets

Clinical signs of rickets and osteomalacia in camels are similar to the presentation in SACs described in Chapters 2 and 3. It has been attributed to phosphorous and copper deficiency in Bactrian camels in Asia, and has been successfully treated with supplementation of phosphorous and trace minerals [59].

Sway Disease

Sway, a disease of Bactrian camels in Asia, may present as lameness and predisposition to fractures. It is attributed to a relative copper deficiency, and is usually accompanied by microcytic anemia, pica, and ill-thrift. Camels in this region were found to have a low copper to molybdenum ratio, and treatment was successful with supplementation of copper sulfate [60].

Viral Diseases

Middle Eastern Respiratory Syndrome

MERS-CoV, the coronavirus responsible for this zoonotic disease, can cause a high mortality rate of approximately 35% in infected humans [61]. Dromedary camels serve as the natural reservoir of the virus and play an important role in transmission. Clinical signs in the camel typically present as a mild respiratory infection, though many infections are subclinical. Shedding is suspected to occur primarily in young camels via respiratory secretions [62].

Evidence of MERS-CoV circulation in camel populations is present in Africa and the Middle east, seropositivity is often reportedly over 90% of camels in these areas, though prevalence of viral nucleic acids in nasal swabs is typically quite low (estimated 5–12%) [62, 63]. No evidence has yet been found for circulation of MERS-CoV in camels in Australia, Japan, Kazakhstan, or North America [62]. MERS-CoV antibodies has been detected in llamas and alpacas [62]. Naturally occurring disease has not been observed in Bactrian camels or ruminant species, but Bactrian camels were recently experimentally infected and displayed clinical signs and shedding behavior similar to dromedary camels [64]. Bats are suspected to be the original source of the virus, as with other zoonotic coronaviruses [65].

Humans can acquire the virus from infected camels or humans, but the transmission between people is often limited to healthcare providers and other members of the household. Some MERS-CoV cases have no documented contact with camels or infected humans, and the source of infection remains unknown. Approximately 55% of infections are linked to contact with camels [66]. Infection can occur from direct contact with camel saliva or respiratory secretions, or contact with contaminated fomites. Raw camel milk and fecal transmission could theoretically transmit the virus, but the risk is considered low at this time [61]. Most human cases have originated in the Arabian Peninsula or from contact with people who had recently traveled there, despite circulation of the virus in Africa and other areas [63]. Though MERS-CoV has a high mortality rate associated with infection, some camel-handlers have evidence of prior infection and immune response without history of severe respiratory infection [67].

With the continued emergence of zoonotic diseases caused by novel coronaviruses such as severe acute respiratory syndrome (SARS), MERS and COVID-19, the camel may continue to serve an important role in global health and research. Development of vaccines, understanding of immunity, and continued monitoring of disease transmission may require an extended focus on camel health and physiology.

Foot and Mouth Disease

Bactrian camels can become infected and show clinical signs, but dromedaries have not been shown to contract or transmit the disease [68].

Bovine Viral Diarrhea Virus

Camels, like llamas and alpacas, can become infected with BVDV. Studies of healthy camels have confirmed the presence of antibodies and BVDV antigen in Bactrian and dromedary camels [69, 70]. Camels may represent a source of virus and mode of transmission to other ungulates, but the role of BVDV in disease in camels has not been as clearly defined as it has in SACs.

Bacterial Diseases

Caseous Lymphadenitis

Camels are susceptible to infection with *Corynebacterium pseudotuberculosis* and will show clinical signs of caseous lymphadenitis typical of the presentation in other species. Internal abscessation as well as subcutaneous and muscular abscesses are reported, and swelling of external lymph nodes is variable [71]. The author has observed recurrent abscesses of the prescapular lymph nodes and subcutaneous abscessation of the pectoral region. The ovis biotype has been exclusively isolated from camels [72]. Diagnosis, treatment, and control are the same as for other domestic species.

Streptococcus Equi Subspecies Zooepidemicus

Camels are susceptible to infection with *Streptococcus equi* subspecies *zooepidemicus*. Reports describe respiratory

infection, peritonitis, or polyserositis as seen in "alpaca fever" in SACs [73–75]. It is one of the most common causes of metritis and endometritis in camels [76]. Despite the potential for pathogenicity, it has also been isolated from the respiratory, GI, and reproductive tracts of healthy camels [75].

Parasitism

Like SACs, camels are susceptible to severe clinical disease and death from endoparasitism. They may present for non-specific signs such as lethargy, inappetence, and malaise and may have reached a point of severe compromise before veterinary attention is sought. Of particular importance in domestic camels is whipworm infection. Diagnosis, treatment, control, and prevention strategies are similar to those described in Chapter 7.

Surgery of Camels

Most surgical procedures of camels can be approached similarly as described in the previous chapters. With the exception of the dulaa, the majority of the anatomy and physiology is shared between the old and new world species.

Gastrointestinal Surgery

Camels are more prone to ingestion of foreign bodies than the SACs, likely due to their larger size and curiosity. Accumulation of foreign bodies such as a plastic and rope in the first two compartments of camels is common and well-documented [77, 78]. Camels will also ingest wires or metal objects and develop signs consistent with traumatic reticuloperitonitis [79]. Foreign bodies may cause weight loss, alterations in GI motility, pyrexia, anorexia, and GI perforation with local or diffuse peritonitis. Surgery may be required, and can be performed similarly to rumenotomies in ruminants.

Cesarean Section

Cesarean section is well-described in the literature and typically employs a left paralumbar or left ventrolateral approach [22, 80]. The ventral midline approach is regarded as having the most complications [22]. Though dystocia is uncommon in the camel, cesarean section may be necessary in cases of fetal malposition, fetal malformation, or uterine torsion. Outcome is variable and dependent on individual animal factors. Anesthesia, analgesia, antimicrobial therapy, and supportive care should be tailored to the individual case and recommendations do not vary from those given in Chapter 16.

Castration

Adult bulls can be dangerous and difficult to handle, especially during the rut, so castration is recommended for any camels that will interact with the public. The castrated camel is typically more compliant and docile.

The testicles typically lie outside of the inguinal ring by seven months of age, and are descended completely into the scrotum by two years of age. Attempts to castrate camels very young may incorrectly give the impression of cryptorchidism. Sexual behavior may be evident in dromedary males at two years of age, but puberty does not usually occur until the animal is four to five years old. Bactrian males mature more slowly, with sexual behavior beginning at three years of age and puberty at five to six years of age. Recommendations for age of castration varies between two and four years of age.

The surgical procedure is performed similarly to that described in alpacas and llamas (see Chapter 16). The most common technique is via two pre-scrotal incisions left open to drain. A single midline pre-scrotal incision with subsequent skin closure may also be performed with a low complication rate [13]. Standing castration under sedation is reported [81]. Camels should be current on tetanus vaccination when castrated.

Dulaa Surgery

Both male and female dromedary camels have a unique diverticulum on the ventral aspect of the soft palate. The diverticulum (dulaa, dulla, dulah) is more highly developed in the male and may be inflated and protruded from the oral cavity during rut, or when excited or angry (Figure 23.30). Bactrian camels do not have a dulaa.

Several conditions of the dulaa may require surgical intervention including lacerations, hematomas, erosions, ulcerations, necrosis, impaction, and paralysis. The dulaa may be lacerated by bites from other combative males or from its own canine teeth while the dulaa is protruded. If the dulaa remains protruded for a prolonged period of time it may be traumatized by objects in the enclosure or dry out and become infected or necrotic. The dulaa may become impacted with feed, preventing protrusion. The dulaa may be amputated in racing camels because of real or perceived interference with respiration while racing.

Clinical Signs

Signs of trauma that may be observed when the dulaa is protruded include erosions, ulcers, hematomas, and necrosis. Impaction interferes with swallowing feed and

drinking water. Large impactions may interfere with breathing. The swelling may be observed or palpated upon oral examination. Impaction may cause a swelling in the throat region that is visible or palpable.

Superficial lacerations and erosions may be managed with routine wound care. More serious conditions may necessitate amputation. Several methods for amputation of the dulaa are described in the literature [82–85].

The camel should be heavily sedated and maintained in sternal recumbency, preferably with a body rope harness in place to prevent the camel from rising during the procedure. Some surgeons prefer the camel under general anesthesia. The mouth is held open with a speculum. The oral cavity should be lavaged with water and finished with a disinfectant solution such as chlorhexidine or povidone-iodine solution. Intubation or a tracheostomy should be performed to prevent interference with respiration while working in the oral cavity. Four methods are described for amputation of the dulaa. In each of them, visible blood vessels should be ligated or cauterized by electrocautery.

1) Pull the dulaa from the mouth and spread the tissue. Cut the dulaa close to its attachment to the soft palate with a curved Mayo scissors. The wounds are left open to heal by second intention.
2) Clamp the tissue close to the soft palate with a large Carmalt forceps or an angiotribe. Incise on the distal side of the forceps with a scalpel. Leave the clamp in place for a minute. Then release the clamp and replace it on the next segment to be incised. Repeat the process until the entire dulaa is amputated.
3) The dulaa is stretched maximally and incised with a long Mayo scissor. Vessels must be ligated to prevent hematoma formation following closure. The margins of the mucous membranes are approximated and closed using an inverting Cushing suture pattern.
4) Impactions may be managed by mechanical evacuation of the impacted material. In severe cases, it may be necessary to use a blunt hook to pull the dulaa forward to allow amputation.

Following surgery, the administration of a broad-spectrum antibiotic such as long-acting tetracycline is recommended. The oral cavity should be irrigated with dilute hydrogen peroxide, chlorhexidine solution, or povidone–iodine solution for three to five days, and soft feed should be offered for two weeks.

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