CHAPTER **37**

Diseases of the Respiratory Tract

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Abnormal respiratory signs, including tachypnea and dyspnea, are common in sick or stressed camelids. Compared with other species of domestic hoofstock, these signs in camelids commonly arise from upper respiratory tract dysfunction or from multisystemic diseases, rather than from pneumonia. Thus, the veterinarian should be prepared to explore beyond thoracic auscultation and beyond the respiratory tract altogether, even if the camelid is demonstrating overt signs of respiratory tract dysfunction.

Anatomy

The alae of the nose are soft and fleshy, and the external termination of each nostril is slitlike. Camels can completely occlude their nostrils by depressing these alae, presumably as protection against blowing sand, whereas New World camelids cannot. However, the external nasal structures of all camelids are vulnerable to injury or external compression such as from snake bite, manual compression, or a too-narrow halter and may occlude the airway anyway.

The nasal passages and larynx are also relatively narrow. The nasolacrimal orifice is positioned on the medial aspect of the lateral cartilaginous wall of the nasal cavity and is usually just beyond what may be seen without an endoscope. This narrowness may be an adaptation to moisten and warm inspired air in the dry, hot, or cold native environments. Regardless of the cause, the narrowness of the airway allows small amounts of compromise to severely restrict air flow and also means that many diagnostic or therapeutic modalities such as paranasal endoscopy and nasogastric or nasotracheal intubation are more difficult to perform on camelids than on some other species. Of the passages, the ventral meatus is the largest, and its ventromedial component represents the widest, straightest path to the larynx. Its dorsal component is approximately half the diameter and often too narrow to allow passage of a tube or endoscope. Therefore, it is helpful to manually press either of those items as ventromedially as possible during entry in to the nose.

Camelids have a relatively long palate, with the soft palate component accentuated in camelids. Laryngeal and tracheal anatomy are relatively conventional; the caveat, however, is that the length and narrowness of the nose and mouth make direct visualization of the larynx, and hence tracheal intubation, a challenge.

The anatomy of the camelid lung is also relatively conventional except that the small accessory lobe in the right side represents the only example of separation into lobes. Similar to ruminants, the forestomach pushes close to the caudal border of the heart ventrally, making the dorsal lung field much more expansive for auscultation. The mediastinum is reported to be complete.

Examination

The alae of the nostrils should be thin and mobile. With each breath, whether the airway is patent or not, the alae should be pulled back to open the nostril. Collapse of one or both alae may represent a neuromuscular defect; decreased function may also come from edema or inflammation. The internal mucosa should be assessed for evidence of edema or discoloration. Airflow should be felt from each nostril. Air passage within the nostril may be assessed by placing a stethoscope on each side of the nose. The laryngeal region should be palpated for obvious swelling or mass lesions.

Nasal discharge from camelids is usually clear and noncopious. Thicker discharge may reflect increased mucus content or white blood cells (WBCs). These rarely reach sufficient quantity to restrict air flow. Hemorrhagic discharge typically reflects inflammation at some point in the airway or a bleeding disorder. Green-tinged or feed-containing discharge reflects difficulty swallowing or regurgitation. With cleft palate or abnormal regurgitation, the camelid may asphyxiate because of occlusion of the upper airway.

The most common serious upper airway sign is dyspnea. Affected camelids have increased nostril flaring, are exercise or stress intolerant, frequently mouth-breathe with the lips pulled away from teeth, and often have inspiratory or expiratory stridor. Whereas in other species with laryngeal dysfunction inspiratory stridor is more common, the sail-like nature of the camelid soft palate makes expiratory stridor common as well.

With added stress, the respiratory distress may worsen, with the head flopping to one side or over the dorsum. In such cases, the veterinarian should halt the stressful interventions and try to promote the camelid's breathing by delaying non–life-saving procedures, allowing the animal some space, keeping the animal's head and neck in an extended position, administering oxygen by oral or nasal insufflation, or, in extreme cases, performing a tracheostomy.

With lower respiratory tract disease, a variety of signs, including tachypnea, dyspnea, inspiratory or expiratory grunts, obtundation, weight loss, and lethargy, may be seen. The trachea may be auscultated along the neck, and the lungs are best assessed by auscultating from within the short-haired axillary window. Although this does not allow auscultation of the most dorsocaudal lung, moving the stethoscope around the dorsal extent of that window and stretching the skin more dorsally usually allows adequate assessment.

Whenever lower respiratory disease is suspected, information of lung function may be obtained from arterial blood gas (ABG) analysis. Camelids have a number of superficial arteries, but the most commonly used are found on the medial aspects of the limbs and are thus more accessible in neonates and debilitated older animals in lateral recumbency than in more vigorous individuals. The median artery is found between the shoulder and the elbow, running along the craniomedial aspect of the humerus. Its pulse is usually palpable, although the structure of the artery itself usually is not. It is usually approached perpendicularly (see Figure 37-1). The saphenous artery is often both visible and palpable from medial midthigh to below the stifle. It is usually approached in a near-parallel fashion, often from its dorsal aspect to avoid puncturing the accompanying vein (Figure 37-2). Both arteries are useful in neonates; the saphenous vein is also used extensively in adults.

Endoscopy and bronchoscopy allow direct visualization of the mucosa and discharges and are especially useful for diagnosing obstructions, aspiration, inflammatory conditions, or lungworms. The ventromedial aspect of the ventral meatus is most useful. Although externally narrow, it widens considerably at the level of the molars; with a small enough endoscope and sufficient lubrication, valuable information can be obtained. With persistence, a 6-millimeter (mm) external diameter scope will pass on most crias and adult alpacas, and adult llamas may accommodate 9-mm scopes fairly easily.

Figure 37-1 Obtaining blood from the median artery. Note the perpendicular approach on the dorsomedial aspect of the humerus.

Figure 37-2 Obtaining blood from the saphenous artery. Note the parallel approach on the medial aspect of the distal femur. The artery itself is visible and palpable.

In dyspneic camelids, endoscopy does reduce available airway and may lead to distress of the patient, so procedures should be completed as quickly as possible. Dorsal displacement of the soft palate may exacerbate respiratory compromise.

Transtracheal or transendoscopic wash or bronchoalveolar lavage (BAL) may be used to collect information on infections, parasitic infestations, and cytologic abnormalities. Transtracheal wash using catheters or commercial kits for foals may be performed after clipping and surgically preparing the ventral midline of the neck a few centimeters beyond halfway down the trachea and infiltrating the site with a local anesthetic.[1] Transendoscopic tracheal washes yield similar results, except possibly on bacteriologic culture, and the endoscope may be wedged in a bronchus for a BAL.

Pleural fluid may be obtained from healthy camelids or more readily from those with subacute to chronic hypoproteinemia.[1] Fluid is best obtained approximately one third to half way up the thorax, at the sixth or seventh intercostal space. The area should be clipped, aseptically prepared, and infused with local anesthetic. A needle, cannula, or chest tube may be used, depending on how much fluid is expected, and how flocculent it is likely to be. Normal pleural fluid contains less than 1500 nucleated cells per microliter (cells/µL), with lymphocytes making up at least 80% of these cells.[1] Pleural fluid usually has protein concentrations less than 2.7 grams per deciliter (g/dL), but as with abdominal fluid, outliers are possible.

Imaging studies are useful for assessing the upper airway and lung. Conventional radiography or cross-sectional techniques are useful for the bony and cartilaginous structures of the head. Intranasal contrast material may be used, if occlusion is suspected. Radiography and cross-sectional studies may be used to assess the lung, with the cross-sectional studies being more useful than radiography for finding small focal lesions such as tumors, abscesses, or granulomas. Transthoracic ultrasonography is most useful for identifying pleural fluid or when parenchymal disease is extensive or against the body wall.

Supplemental Oxygen and Ventilation

Although camelids often appear relatively vigorous in spite of hypoxemia, it is generally accepted that they are healthier and have more efficient body functions when oxygenation is closer to normal. Thus, similar rules apply as in other species: camelids with an arterial partial pressure of oxygen (PaO_2) less than 60 millimeters of mercury (mm Hg) are candidates for supplemental oxygen, and camelids with $PaCO_2$ greater than 60 mm Hg are candidates for mechanical ventilation. In neonates or other small camelids, oxygen cages may be used. For larger camelids or for crias kept near their mothers, intranasal oxygen is often effective and well tolerated. Soft rubber catheters or specialized breathing tubes may be tabbed into one nostril so that the distal end in the nostril is approximately at the level of the medial canthus of the eye. Pure oxygen may be delivered at approximately 0.75 to 1.5 liters per hour L/hr per 9 kilograms kg (20 lb), with the higher volumes used in acute or severe cases. Delivered oxygen may be gradually decreased over 1 to 5 days as the clinical condition or blood gas values improve.

Mechanical ventilation also follows the principles used in other species. The ventilator or breathing bag may be hooked to an endotracheal tube or tracheostomy tube, with the latter necessary for animals with an intact gag or chew reflex or a collapsed upper airway.

Diseases of the Respiratory Tract

Dorsal Displacement of the Soft Palate (Pharyngeal Collapse)

The most common cause of dyspnea is dorsal displacement of the soft palate, possibly compounded by dorsal pharyngeal collapse or epiglottic entrapment. In most cases, the underlying cause is unknown, but it is postulated that this is part of the stress response, especially in weakened or sedated camelids. In some cases, fatigue of the airway musculature caused by chronic high airway resistance or a neuromuscular defect may play a role. Clinical signs include dyspnea, stridor, open-mouthed breathing, dorsiflexion of the head and neck, regurgitation, nystagmus, and death. Definitive diagnosis requires upper airway endoscopy (Figure 37-3), but the diagnosis can often be inferred, as endoscopy of patients in respiratory distress may be unnecessarily life threatening. Possible treatments include administration of nasal oxygen, maintaining the head and neck in extension, placing a towel over the eyes to reduce sensory input, reversal of sedative medications, and tracheostomy. When stress is the causative factor, handling to administer medications, draw blood samples, maintain the neck in a certain position, or perform other diagnostic or therapeutic procedures may worsen the condition. Such camelids may improve if allowed to recover on their own. However, camelids with spontaneous dorsiflexion rarely improve without some degree of intervention. When this appears to be
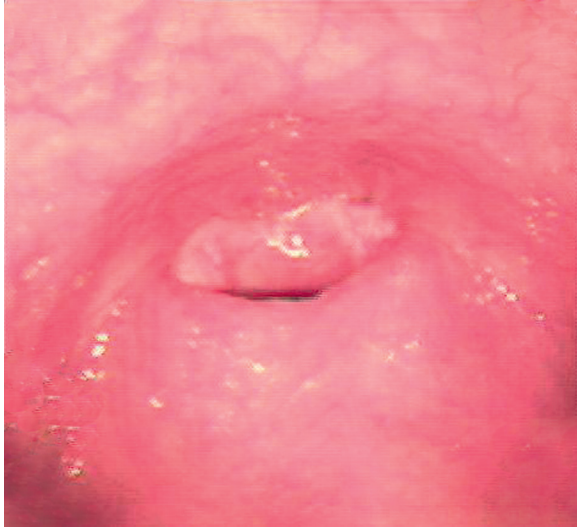
Figure 37-3 Dorsal displacement of the soft palate in a llama with respiratory distress. This condition may be permanent or transient and, in either case, is associated with respiratory distress.

a permanent condition, a permanent tracheostomy may be necessary and greatly improve the animal's quality of life.

Laryngeal Edema

Laryngeal edema has a similar clinical appearance to dorsal displacement of the soft palate. Causes include insect or snake bites, tracheal intubation, tight cervical wraps or collars, entrapment of the neck in gates or fences, hypoproteinemia, overhydration, jugular thrombosis, cervical hematoma or trauma, heat stress, ingestion of caustic plants or medications, or right heart failure. Clinical pathology data may reveal evidence of an underlying inflammatory disease or hypoproteinemia. Radiographic or ultrasonographic examination of the region reveals thickening of the soft tissue structures and separation of other tissue layers. If the camelid is stable, treatment should be directed at resolving the underlying condition. Antiinflammatory drugs or diuretics may aid in reducing swelling. If dyspnea is severe, intranasal oxygen or tracheostomy may be necessary.

Arytenoid Chondritis and Laryngeal Abscessation

Inflammation or infection of the laryngeal cartilages is much rarer and much more serious than simple edema of the region. Affected camelids usually have severe dyspnea with exercise intolerance and stertorous breathing. Increased recumbency and collapsing with exercise are seen. The application of mild external pressure to the laryngeal region exacerbates the signs. Endoscopic examination is usually necessary to confirm the diagnosis. Radiography of the region may reveal soft tissue thickening or mineralization. Antibiotics (for 2 or more weeks) and antiinflammatory medications (for a few days) usually reduce the regional swelling. Surgical resection or tracheostomy may be necessary in extreme cases.

Laryngitis with abscessation not involving the cartilage was identified in one 10-day-old cria with possible partial failure
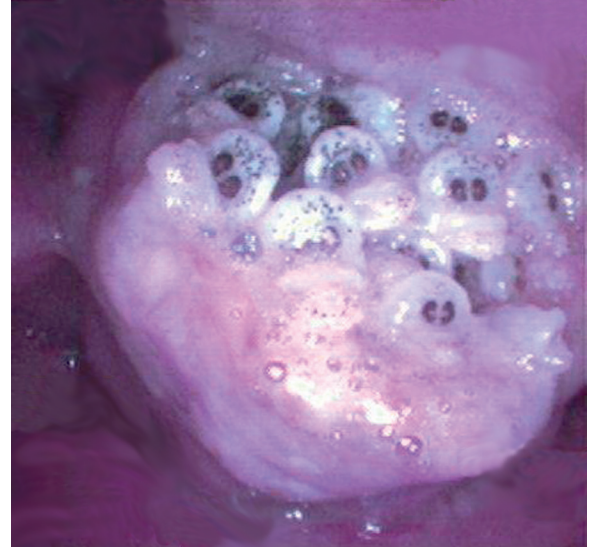

Figure 37-4 *Cephenemyia* larvae within the larynx of a llama.

of passive transfer.[2] No respiratory signs were noted, but it is likely that they would have developed had the cria lived longer. *Mannheimia hemolytica* was isolated.

Retropharyngeal Lymphadenopathy

Enlargement of the retropharyngeal lymph nodes may lead to occlusion of the upper airway and dysphagia. This finding is relatively rare in camelids and may be the result of neoplastic change or infection. The most common neoplasm of the area is lymphoma.[3] The most common infection is likely to be *Corynebacterium pseudotuberculosis*, although this is more likely to affect the submandibular lymph nodes, rather than the retropharyngeal nodes.[4,5] Penetrating foreign bodies may also lead to infection in that area with a variety of bacteria introduced.

Masses may become large enough to be appreciated from the outside or may require deep palpation of the upper neck. A small amount of external pressure may cause an exacerbation of stridor or dyspnea. Endoscopy, ultrasonography, or imaging studies may also reveal the mass lesion, and facilitate aspiration or biopsy. Treatment for the primary disease may be all that is necessary, but if the camelid has dyspnea or dysphagia, lancing of the abscesses or tracheostomy may provide relief.

Nasal Bots

Infestations of the caudal nasal passages and dorsal pharyngeal region by nasal bots larvae from sheep (*Oestrus ovis*) or deer (*Cephenemyia* sp.) have been reported.[6–8] Reports of deer nasal bots are currently confined to the western parts of the United States (California, Colorado, Oregon). Infestation occurs during fly season. Populations of the definitive host (sheep, deer) are usually found nearby. Both genera of fly are viviparous and deposit live larvae near the external nares of the host. Larvae colonize the upper airway leading to an exudative, granulomatous reaction and enlargement of adjacent soft tissue (Figure 37-4). On maturation, larvae are sneezed or coughed back onto the ground and develop into flies.

Most reports involve individual camelids, but herd outbreaks have occurred. Clinical signs are mainly from the physical narrowing of the upper airway. These include sneezing, coughing, respiratory stridor mainly on inspiration, nasal discharge possibly with mild epistaxis, exercise intolerance, and open mouth breathing. Pulmonary auscultation is usually normal but is complicated by sounds referred from the upper airway. Affected camelids are afebrile and have normal clinical pathology data except for the changes caused by stress. Most cases occur in adults, but one case of a 9-month-old llama has been reported. Many also have a history of treatment failure with antibiotics.

Definitive diagnosis of *Cephenemyia* infestation is by endoscopic examination. Individuals or groups of larvae may be seen. They are often found within a pouch of soft tissue on the dorsal nasopharyngeal wall. This pouch may also be seen radiographically. *Oestrus ovis* are less commonly reported but have a predilection for the dorsal nasal turbinates and sinuses in sheep. These areas are inaccessible to endoscopy. *Oestrus ovis* larvae may be distinguished from *Cephenemyia* sp. by examining the two dark peritremes at the posterior (exposed) end. Peritremes are circular in *Oestrus* with a central button and kidney-shaped in *Cephenemyia* with buttons aligned with the medially oriented lesser curvature.

Treatment depends on the severity of signs. Mildly affected camelids may be treated with ivermectin, which often resolves the infestation within 2 weeks. More compromised camelids may require more aggressive intervention. Removal of larvae one at a time using the endoscope's biopsy instrument is time consuming but effective.

Upper Airway Malformations

Congenital upper airway malformations are relatively common in New World camelids. Among the more common are choanal atresia (10.4% of reported congenital defects in one study), wry face (7%), and cleft palate (3%).[9] Subepiglottic cysts and hypoplastic trachea are less common. All of these impede air flow or heighten risk of aspiration.

Choanal atresia is anatomically well described. It results from failure of the buccopharyngeal membranes to completely rupture during the early second trimester of fetal development, leaving membranous or bony obstructions over one or both nasal passages (Figure 37-5). Camelids with unilateral lesions may thrive into adulthood, showing little more than a heightened respiratory rate, open-mouthed breathing, or dyspnea when stressed. Bilaterally affected neonates are born with little or no ability to breathe nasally. Thus, clinical signs start immediately or soon after birth. Affected crias typically display open-mouthed breathing, with the lips pulled away from the mouth, stertor (inspiratory or expiratory), and pronounced nostril flaring (Figure 37-6). They often have difficulty eating and are at high risk for aspiration. Air flow at the nostrils is absent or reduced, and soft rubber tubes cannot be passed nasally beyond the level of the eye. Definitive diagnosis may be achieved by endoscopic examination with a pediatric endoscope (up to approximately 6 mm diameter), plain radiography, contrast radiology, in which approximately 5 to 10 milliliters (mL) of contrast (preferably an organic iodide to reduce risk of aspiration complications) is instilled into each nasal passage and the nose held in dorsal extension
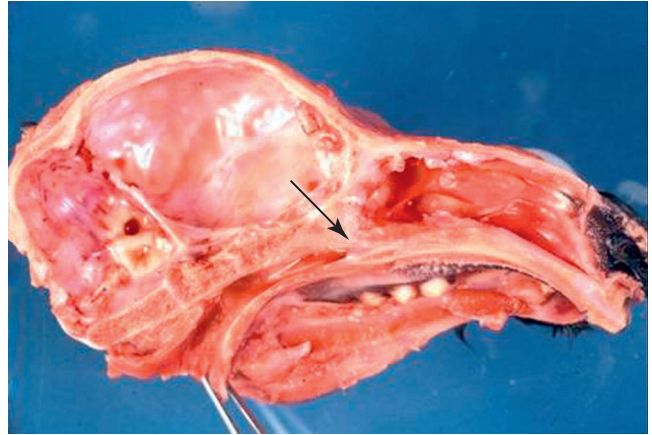

Figure 37-5 Choanal atresia is a neonate. The arrow marks the bony occlusion of the airway.

Figure 37-6 Open-mouthed breathing in an alpaca cria with choanal atresia. The nostrils moved in synchrony with inspiration in spite of no air flow.

(Figure 37-7), or more advanced imaging techniques that allow the nasal passages to be displayed in slices. Aerophagia and hyperinflated lungs may also be seen. Endoscopy or contrast radiography reveals imperforate membranes or bony obstruction. Mucoid fluid may obscure the actual obstruction. Plain radiography reveals bony obstruction only and may be difficult to interpret. Clinical pathology data reflect stress or secondary aspiration pneumonia. Blood oxygenation is usually poor. Tracheostomy or surgical repair are possible and provide immediate relief, but camelids frequently outgrow these stomata and repeated procedures may be necessary.[10]

Much of the controversy surrounding choanal atresia involves questions of heritability. Researchers have been unable to define a specific mode of inheritance. Camelids that have given rise to previous offspring with choanal atresia appear to be more likely to have subsequent affected offspring compared with camelids that never have had affected offspring. In contrast, breeding affected camelids rarely leads to the birth of affected offspring.

Similar problems are seen in camelids with severe facial distortion. They usually have a patent airway, although reduced

Figure 37-7 Contrast radiographs of a cria with choanal atresia. Five to 10 milliliters (mL) of iohexol are placed in each nostril, and the nose held in an elevated position.

in size. Diagnosis of the disorder is suggested by physical examination with possible endoscopic or radiographic confirmation. Surgical repair or tracheostomy may be necessary. Less is known about the heritability of this disorder, although a genetic basis is suspected.

Cleft palate is relatively common in camelids, some of which also have other congenital malformations.[9] The airway is not specifically narrowed with this disorder. Aspiration is the greatest danger. Affected camelids frequently cough after eating and may have milk come out the nose. Some clefts are large enough to be seen on oral examination, although many affect only the caudal palate and require endoscopy to diagnose. Surgical repair is uncommon but may be successful with the smaller clefts.[11] Camelids with large defects tend to aspirate at a very young age, whereas those with smaller defects may thrive for a longer period. No information is available concerning heritability in camelids.

Fungal Rhinitis and Sinusitis

True fungal infections of the nasal passages are rare in New World camelids. Turbinate infection by *Rhizopus* spp., together with nodular pneumonia and meningoencephalitis, was identified in a single llama with various cranial nerve deficits and eventually weight loss.[12]

Sinus infection with osseous proliferation and facial deformation was associated with an *Aspergillus*-like organism in a llama.[13] Infections of the sinus or nasal passages may be difficult to diagnose. Most lead to bone deformation, bone lysis and proliferation, and soft tissue densities that may be seen on radiographs or cross-sectional imaging studies. Smaller masses or those involving the airway may be identified by endoscopy. Biopsy with or without fungal culture is necessary to distinguish these masses from tumors.

Infection on the skin of the nares by *Conidiobolus coronatus* in two llamas led to proliferation of tissue, chronic nasal discharge, and eventual occlusion of the nasal passages.[14,15] *Conidiobolus* is a tropical fungus and most common in the Gulf Coast region in the United States, but one infected llama was a lifelong resident of Illinois. Diagnosis was achieved by histopathologic examination of tissue sections and fungal culture. Systemic antifungal medications have been used successfully in other species, but in the single treated llama case report, infected tissue was surgically removed after iodides and topical fungicides failed to resolve the lesion.

Snake Bite

Although snake envenomation affects several organ systems, the propensity for camelids to be bit on the lips or nose warrants the discussion here. Bites occur during seasons of snake activity, usually late spring and the summer. Most reports involve the Western diamondback in California or the smaller prairie rattlesnake in Colorado.[16,17] The venom of these snakes contains a combination of enzymatic and nonenzymatic toxins. The overall effects are local tissue digestion, anticoagulation, hemolysis, vasculopathy, and hypotension. Eastern diamondbacks have a more hemolytic effect, and the Mojave rattlesnake has a presynaptic paralytic neurotoxin. Neurotoxin also is present sporadically in other species of pit vipers. In addition to the venom, bites are also often contaminated with a variety of microorganisms, including *Pseudomonas aeruginosa*, *Clostridium*, and *Bacteroides fragilis*.

Affected camelids generally have severe local swelling, which, in the case of bites on the face, may cause severe lip, nose, and eyelid edema and occasionally tracks to the laryngeal region and neck. Swollen areas often exude sanguinous fluid, and close inspection may reveal paired fang marks.

Hemorrhagic diathesis appears to be more common in camelids bit in Colorado than in California. Swelling may also occlude air flow, leading to dyspnea, nostril flare, stridor, and tachypnea and cause dysphagia. Systemic signs of envenomation include bruxism, signs of shock, hyperthermia, tachycardia, cardiac arrhythmias, recumbency, lethargy, ileus, and anorexia. Further obtundation and other neurologic signs, respiratory signs, and colic may occur with progression of envenomation. Obviously, if the bite is on another part of the body such as a leg, signs referable to that body part will be present, possibly in the absence of respiratory signs. Local signs often worsen over the first 48 to 96 hours.

Although not yet described, Mojave rattlesnake bites would be likely to cause much less severe local inflammation, but they cause progressive neuromuscular weakness and respiratory paralysis.

Blood examination reveals evidence of stress, inflammation, muscle damage, and possibly cardiopulmonary or renal failure. Neutrophilia and neutropenia are both possible, usually with concurrent left shift and lymphopenia. Anemia, thrombocytopenia, and hypofibrinogenemia may also be seen. As with other camelid diseases, hypoalbuminemia, hyperglycemia, and hypokalemia are common. Increases in muscle enzymes are often dramatic and may be accompanied by increases in liver enzymes. Azotemia and acidosis are common in more severely affected camelids. Coagulation profiles may reveal the hypocoagulable state.

Treatment may be multimodal and extensive. Airway preservation is often the primary goal and may be achieved through nasotracheal intubation or tracheostomy in severely affected camelids. Characteristically, tracheostomy sites or other skin breaks bleed persistently. Because of the progression of lesions, camelids not requiring immediate airway intervention should still be kept under observation, in case some future intervention is necessary. Even in apparently hydrated camelids, some form of fluid therapy is helpful in preventing thrombotic complications. The finding of hepatic lipidosis in one affected camelid suggests some may benefit from specific treatments against this condition. However, the risk of edema is even higher in camelids with snake bite than in the general population, so any fluids must be given at a judicious rate and sometimes accompanied with a colloid, blood, or blood products. Given the tendency toward tissue swelling, any neck wrap must be loose and assessed frequently, lest it should become constricting.

Broad-spectrum antibiotics with good anaerobic coverage are recommended because of the contaminated nature of the bites and the risk of translocation across a compromised bowel. A tetanus toxoid is also recommended.

Antiinflammatory medications may aid in reducing the swelling and some of the effects of the toxins. Generally, when veterinary treatment is initiated, nonsteroidal medications are preferable to corticosteroids or antihistamines. Corticosteroids are often used in a last-ditch effort and may be indicated in some forms of shock; especially when repeated, they also suppress the immune system during a period of possible sepsis. Antihistamines are unlikely to be of value beyond the peracute stage and may worsen hypotension.

Local wound care should be performed on weeping or necrotizing lesions. Fasciotomy may also release internal pressure and prevent secondary lesions. The use of antivenin has not been explored extensively. It appears to improve survival in most species but usually must be given within hours of envenomation. In one report, a camelid appeared to have an anaphylactic reaction to equine-derived antivenin.[16] Use of ovine-derived antivenin has not been reported.

Reported survival of moderate to severely affected camelids is about 50%. Death occurs secondary to cardiopulmonary failure, asphyxiation, or septic shock. Necropsies have revealed extensive local necrosis, hemorrhage, edema, and congestion, laryngeal edema, third-compartment ulceration with possible perforation, intestinal thrombosis, multifocal ecchymoses, pneumonia, endocarditis, pericarditis, myocarditis, peritonitis, hepatic lipidosis, and hemorrhage, edema, or congestion of a variety of internal organs.

The possibility of survival is higher in less severely affected camelids. A proportion of snakebites results in little to no venom injection and hence may result in minor clinical signs or may never be noticed. Prevention revolves around avoiding contact between camelids and venomous snakes. One report infers that bites occur overnight or in the morning, which suggests a preventative role to housing camelids overnight.

Sinusitis

Nonfungal sinusitis in New World camelids usually occurs secondary to a maxillary tooth root abscess. *Oestrus ovis* infestation is another possibility. The airway is affected by discharge from the primary lesion and by remodeling of bone that encroaches on the airway. Characteristically, mucopurulent nasal discharge is unilateral, and nasal air flow is reduced on the affected side. Distortion of facial symmetry and draining tracts on the side of the face may develop. Other findings are reviewed in the section on tooth root abscesses. Radiography is the best way to diagnose the disorder and confirm the infected tooth. Removal of the infected tooth and establishment of drainage usually lead to resolution of the sinus lesions.

Neoplasia

Airway tumors are rare in camelids. Fibrosarcoma was identified in one elderly alpaca, in which the tumor arose from the anterior aspect of the nasal septum and led to near-occlusion of one nostril. Additionally, a variety of tumors arising from the digestive structures of the mouth may affect the airway by their presence. These include squamous cell carcinomas, odontogenic neoplasms, and ossifying fibromas that appear to arise from a tooth.[18,19] If masses encroach on the airway, unilateral mucopurulent nasal discharge, reduced nasal airflow, and gross distortion of the face or nasal passages may be present. Radiography, endoscopy, or advanced imaging techniques may be used to diagnose and observe the extent of the mass. Biopsy is necessary to confirm the tissue type. Treatment usually involves resection, with or without postoperative chemotherapy.

Lymphoma or malignant round cell tumor compressing the trachea has been described in a llama and an alpaca.[3,20] Both were 7 to 8 years old, older than the median age for camelids with lymphoma. In the llama, a cervical node was palpably enlarged, but the mass was entirely intrathoracic in the alpaca. In addition to the typical signs of emaciation and

partial inappetence, affected camelids have tachypnea or dyspnea. If the mass is palpable, diagnosis may be achieved by aspirate or biopsy. If it is intrathoracic, imaging studies are likely to reveal its presence. Local resection and chemotherapy may lead to some resolution of signs and period of remission.

Lower Respiratory tract

Primary diseases confined to the lower respiratory tract are uncommon in New World camelids. Viral, bacterial, fungal, and parasitic pneumonias are rare, and if present, may arise from or lead to systemic infection. Tumors may be found in the lung, heart, or chest cavity but are also usually part of a multifocal process. Aspiration is relatively common, but usually arises from sedation, neuromuscular disorders, and gastroesophageal disorders. Congenital lesions of the lung are less common or less commonly reported than malformations of other organ systems and usually involve vascular structures (see Chapter 36).

More commonly, lower respiratory signs occur secondary to another disease process. The most common causes of tachypnea or dyspnea at our clinic are cardiac disease, anemia, hypoproteinemia and edema, overhydration, stress, hyperthermia, intracarotid injection, metabolic acidosis, abdominal enlargement, pain, and thrombosis. Therefore, even when examining a camelid with overt signs of lower respiratory tract disease, a comprehensive physical and diagnostic evaluation is warranted.

Acute Pneumonia

Viral Pneumonia. Viral lower respiratory disease appears to be less common in camelids than in horses or cattle, and reports of outbreaks are rare. However, seroconversion to most bovine respiratory viruses does occur in camelids under a variety of management circumstances, and disease is occasionally, albeit rarely, attributed to these infections.[21–25] Bovine herpesvirus 1 was isolated from a small number of llamas with coughs of several months duration that eventually led to bacterial bronchopneumonia.[26,27] Bovine viral diarrhea virus (BVDV) has been linked occasionally to acute and short-lived oculonasal discharge in camelids.[27,28] Increases in titers tend to be short lived. Polymerase chain reaction (PCR) on whole blood is the most common diagnostic modality for BVDV. Virus isolation from blood or discharges and fluorescent antibody testing on lesions are less rewarding but may be used to detect either virus.

Coronavirus has been implicated as the possible cause of the recent outbreaks of nasal discharge and dyspnea in the United States, commonly referred to as the "snots." This is a poorly described syndrome of upper or lower respiratory tract disease, often associated with outbreaks after shows. Affected camelids may have anything from mild serous discharge to thick, profuse nasal discharge or dyspnea. Fever is uncommon and often associated with a poor prognosis. One investigation implicated a group 1 coronavirus, which was distinct from the group 2 coronavirus associated with enteritis.[29] Electron microscopy or PCR on exudates and serologic tests may be used to detect infection.

Adenovirus has also been linked to lower respiratory disease.[30] This virus, although relatively ubiquitous among camelids based on serologic studies, may be found by observing intranuclear inclusion bodies on light microscopic examination or finding evidence of virus by PCR reaction on tissues. Cross-reactivity to antiporcine adenovirus antibodies allows immunofluorescent detection as well.

Most camelids affected by adenovirus are juveniles with progressive dyspnea or tachypnea that does not respond to antibiotic treatment. Unless secondary bacterial infection occurs, fever is uncommon and pulmonary auscultation reveals few abnormalities. Radiographic changes are subtle, with some increase in interstitial density. Clinical pathology data are likewise unremarkable, although one report suggests concurrent adenoviral hepatitis may lead to inflammatory and enzymatic changes.[30] ABG analysis reveals marginal to poor arterial oxygenation and progressive inability to exhale carbon dioxide, in spite of tachypnea and the outward appearance of adequate ventilatory effort. Gross necropsy frequently reveals little more than pulmonary edema. Further investigation using one of the aforementioned methods may reveal evidence of adenoviral infection.

The clinical course is relatively short (1 week) and difficult to reverse. Older camelids may be better suited to surviving the initial infection and may also have a more chronic course. Antibiotics, antiinflammatories, and medications to decrease pulmonary edema may all play some role in treatment. Intranasal oxygen treatment and ultimately mechanical ventilation may be necessary for advanced disease.

Bluetongue. For many years, it has been known that camelids seroconvert to bluetongue, but reports of clinical disease have been rare.[21] Single affected camelids have been reported in Colorado, California, and Germany, and a group of affected llamas were seen in France in 2007.[31–33] The European cases occurred during multispecies outbreaks. Bluetongue is caused by an orbivirus in the reovirus family. There are at least 24 serotypes. Cattle are the usual reservoir species because they often suffer prolonged viremia after infection and rarely show signs of acute clinical disease. The disease is spread by biting *Culicoides* flies (midges), occasionally through semen, and occasionally transplacentally. Most transmission is strongly linked to fly season. Although cattle rarely show more than occasional reproductive problems, including abortion and birth defects, some other species, particular sheep and some wild cervids and ruminants, often suffer severe, often lethal disease. The underlying lesion is vasculitis. Common clinical signs include fever, anorexia, facial and lingual edema, oral ulceration, cyanosis, lameness, and dyspnea. Death is usually the result of pulmonary edema or secondary bacterial infections.

Signs reported in camelids include hiccup-like breathing, anorexia, weakness, recumbency, and respiratory stertor. Signs progress within 24 hours with lethal results. Viremia and seroconversion without clinical signs have been reported as well. Postmortem revealed small ulcers throughout the mouth, severe diffuse pulmonary edema, hydrothorax, and fibrinous pericarditis. Bluetongue virus was identified by PCR in blood, lungs, the heart, lymph nodes, and the spleen. Other camelids in the herds remained healthy. It is still not known whether the affected camelids had some cause for particular susceptibility, their immune system was overwhelmed by the amount of viral challenge during the outbreak, or these represent the innate susceptibility of camelids to bluetongue. Lack of

previous reports in spite of evidence of seroconversion makes the last possibility seem less likely. Preventative measures include fly control, protection from flies, and possibly vaccination. Vaccines are available in endemic or threatened areas and should be given at least 3 weeks before the onset of the fly season. The initial vaccination is a two-injection series at 3-week intervals, followed by an annual booster as long as the danger persists.[34] Since *Culicoides* flies are most active after dusk, evening confinement in barns with fly screens may offer the most practical protection.

Bacterial Pneumonia and Pleuropneumonia.

Primary acute bacterial lower respiratory disease appears to be less common in camelids than in horses or cattle. In most cases, bacterial pneumonia is just one component of a systemic bacterial infection. In some of those cases, the lungs may by the first site and origin of the infection, but in others, another source is suspected.

The paucity of reports on this condition may not be the result of any particular innate immunity in camelids or lack of suitable pathogens, but rather the infrequency of close confinement housing, crowded transport, or other practices which promote the spread of respiratory pathogens. Anecdotal reports are available describing bovine pathogens such as *Pasteurella*, *Mannheimia*, and *Haemophilus* spreading from cattle to camelids when the two species are kept close together. *Mycoplasma* has not been reported but seroconversion occurs, and thus infection remains a possibility.[35]

Streptococcus equi ssp. *zooepidemicus* is the most frequent isolate from camelids with suspected primary acute bacterial pneumonia. Although the source is often unknown, many affected camelids have a history of close contact or shared equipment and housing with horses.[36] Bacteremia and the "Alpaca fever" syndrome are common sequelae to respiratory *S. zooepidemicus* infection.

Camelids with acute bacterial pneumonia display fever, tachypnea, dyspnea, anorexia, and some degree of obtundation. Increased time spent in recumbency is common. Abnormal nasal discharge, coughing, and abnormal pulmonary sounds on auscultation are less frequent or subtle findings. Thoracic radiography and tracheal wash for cytologic examination and bacteriologic culture are useful to confirm the diagnosis and assist in choice of antibiotics. Because of the frequency of concurrent bacteremia, culturing the blood or other body fluids may also be useful. Hematologic analysis is frequently abnormal, with either neutropenia or neutrophilia possible. Left shift, toxic changes in neutrophils, and hyperfibrinogenemia all support a diagnosis of an inflammatory response.[37]

Although *Streptococcus* is the most frequent isolate, the possibility of gram-negative pathogens suggests the need for broad-spectrum antibiotic treatment. Most of the agents approved for use against respiratory infections in cattle are suitable for camelids to some degree; however, the caveat is that long-acting, infrequently administered agents may be less desirable than more frequently administered ones in camelids likely to have bacteremia. Antiinflammatory drugs are also advisable, particularly in patients with high fever, obtundation, septic complications, or hyperfibrinogenemia. Prognosis is guarded. Camelids without systemic complications may recover uneventfully if caught early in the course of the disease.

Streptococcal Infections (Alpaca Fever, Preston Disease)

A growing body of evidence suggests that *S. equi* ssp. *zooepidemicus* is an important primary pathogen of camelids in North America.[36,38,39] This agent is known in South America as the cause of Alpaca fever but was not recognized on other continents for many years. With *S. zooepidemicus* very common in most places with sizeable equine populations, and llamas and alpacas becoming more common, transspecies transmission was just a matter of time. Alpaca fever appears to start as a superficial infection of wounds, the prepuce, or the udder that causes local swelling and discomfort, or as a generalized internal infection. Superficial infections may become generalized, and about half the affected camelids die. Generalized infections may be cleared, leaving abscesses, usually in the abdominal cavity or lungs. In North America, the organism has been isolated from blood, milk, uterine discharges, abscesses, wounds, and body cavity exudates (pleural or peritoneal fluid). Affected camelids have shown a broad spectrum of different clinical signs, ranging from infertility, uterine discharges, abnormal milk, and superficial swelling to tachypnea, dyspnea, cough, tenesmus, diarrhea, and colic. Signs of internal infection are accompanied by fever (102.5°F–106°F), depression, and anorexia, but these may not be present with focal disease. Most camelids with the generalized form of this streptococcal disease show signs referable to the abdominal cavity, and a smaller number also or predominately show respiratory signs.[38] This has given rise to the belief that the organism gains access to the body through the gastrointestinal (GI) tract and not the respiratory tract or lymphoreticular organs, as occurs with most other streptococcal infection (in horses, humans, etc.). This is probably untrue. Research has demonstrated the efficiency of respiratory infection, and greater use of ultrasonography in the diagnostic evaluation of sick camelids has identified pleuritis in a high percentage of cases.[37] Alternative forms of transmission are also possible; anecdotally, this infection has been seen in camelids after orogastric intubation and reproductive examinations using equipment that may have been previously used on horses.

Diagnosis of these infections requires a thorough evaluation. Clinical signs resemble those seen with other, more widely known diseases such as gastric ulcers, intestinal impaction, heat stress, acute eperythrozoonosis, and other infections. Streptococcal infections may also induce or promote some of these other diseases by altering thermoregulation and GI function, increasing stress, suppressing the immune system, and damaging the gut wall.

Localized infections may be found by rectal palpation, thoracocentesis, abdominocentesis, exploratory surgery, or imaging studies (Figure 37-8). Generalized infections may be identified through analysis of blood and other body fluids. Complete blood cell count (CBC) typically shows nonspecific evidence of inflammation including neutrophilic leukocytosis, high band cell counts, and hyperfibrinogenemia. Often, body cavity fluids (abdominal, thoracic) are abnormal, with very high nucleated cell counts (40,000 to 120,000 cells/μL), consisting predominately of nondegenerate neutrophils, and high protein concentrations (>4 g/dL). Often, gram-positive cocci are visible free and within phagocytic cells. Definitive diagnosis requires bacteriologic culture of blood or body
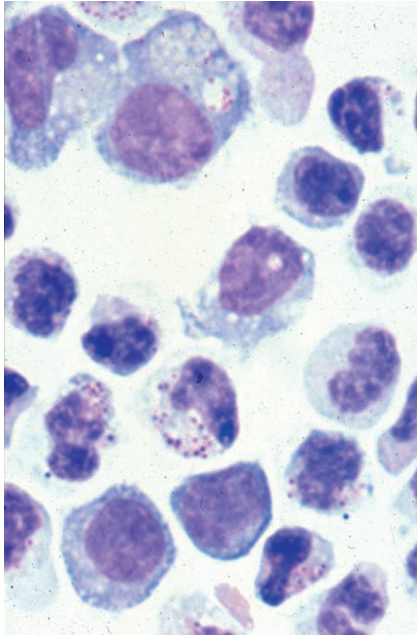
Figure 37-8 Cytologic analysis from an alpaca with *Streptococcus equi* ssp. *zooepidemicus* pleuritis and peritonitis. Note the bacteria within the phagocytic cells.

cavity fluids with generalized infections or exudates with localized infections.

Generalized infections often respond to treatment with antibiotics, including penicillin and ceftiofur (ceftiofur is superior when secondary gram-negative sepsis has occurred). However, this treatment alone occasionally leaves abscesses that may cause problems later (including sudden death). Peritoneal (and theoretically pleural) lavage may be attempted transcutaneously, but the best results are obtained with one-time, thorough surgical lavage of the abdomen in conjunction with medical treatments (antibiotics, fluids, nonsteroidal antiinflammatory drugs [NSAIDs], possibly plasma). The long-term survival rate without surgery is about 60%, whereas the survival rate after surgical lavage is about 80%.

Local lesions appear to be best treated by drainage (if possible) and antibiotics. Some break open spontaneously. Many abscesses recur after treatment. As long as drainage is not internal, and the abscesses are walled off well, camelids may live with them for years.

Prevention efforts are in their infancy and hampered by our limited understanding of the pathogenesis of this disease. Equine strangles vaccines should not be used; the older ones cause severe abscessation in llamas, and the newer ones have not been tested. In South America, the organism is thought to be a widespread oral commensal, so no effort is made to prevent transmission from animal to animal. Instead, preventing stressful management practices and rough handling are emphasized. Several studies on camelids in North America have not supported the theory that the organism is an oral or respiratory tract commensal. Also, herd outbreaks outside of South America are rare.[39] Instead, cases occur sporadically, occasionally in camelids recently exposed to horses. This raises new issues on how transmission occurs. Aerosol transmission of *S. zooepidemicus*, a common pharyngeal commensal of horses, occurs between horses (and humans), and infection is thought to occur through the respiratory tract. It is likely that such transmission occurs to camelids and possibly between camelids and that measures used to prevent the transmission of other respiratory pathogens will limit the spread of this infection as well.

Aspiration Pneumonia

Aspiration pneumonia is one of the most common and most life-threatening lower respiratory disorders in camelids. It occurs following inhalation or administration of feed material into the lungs. Common causes in neonates include tubing or feeding accidents, particularly in crias with generalized weakness or respiratory difficulty caused by choanal atresia, cleft palate, or pulmonary dysmaturity. The condition may also develop spontaneously in crias with any of these conditions, as they frequently try to breathe and drink simultaneously. Common causes in juveniles and adults include esophageal obstruction or megaesophagus, sedation or anesthesia, particularly during recovery, and as part of the stress response to restraint or handling, particularly oral procedures and particularly when the head and neck are restrained in an upright position.

Signs of aspiration pneumonia include acute dyspnea, usually with a productive, retching cough, tachypnea, possible presence of green (plant material) or white (milk) feed material at the nares or coming out of the mouth, and the smell of forestomach contents around the nose and mouth. Pulmonary auscultation may reveal areas of wheezes or dull areas with little evidence of air movement. With chronicity, these dull or abnormal areas may grow, and fever, anorexia, and obtundation may develop in a progressive fashion. The leukogram may show inflammatory changes, including either neutropenia or neutrophilia with a left shift. Hyperfibrinogenemia may develop after 24 hours. ABG analysis may reveal hypoxemia. Thoracic radiography may reveal areas of consolidation, particularly in the cranioventral or midventral lung. Tracheal wash or BAL reveals the presence of feed material and inflammatory cells and, if gastric contents are aspirated, also the presence of bacteria, fungi, and protozoa.

Treatment of aspiration pneumonia is difficult because of the mixed population of microbial contaminants and the possible presence of large fiber particles. On first occurrence, the camelid's head should be maintained lower than the heart base, and the camelid should be allowed to cough freely. In many cases of acute abnormal regurgitation, the feed material does not reach the lung and may be expelled without causing pneumonia. If feed material has reached the lungs, the camelid should be treated aggressively with broad-spectrum antibiotics and antiinflammatory drugs. Many upper airway and forestomach bacteria are facultative anaerobes, so the chosen antimicrobial agents should cover that spectrum. Combinations of an aminoglycoside with a β-lactam agent are common, potentially with the addition of metronidazole. Other basic care, including fluid, oxygen, nutritional support, and environmental support, may be necessary in severe cases. The prognosis relates chiefly to the amount of feed material aspirated and how long it is present in the lungs before initiation of treatment.

All attempts should be made to avoid aspiration pneumonia. Stomach tubes should be checked for patency and

negative pressure or by palpation to confirm that they are within the esophagus. Weak neonates should not be fed by bottle from an elevated position but rather by orogastric intubation or from a pan. Camelids with esophageal obstruction should not be restrained with the head upright. Food and water access should be restricted for a minimum of 12 hours (up to 48 hours for feed, if necessary) before elective procedures that require heavy sedation or anesthesia. Most importantly, if a camelid regurgitates during a procedure, it should be allowed or forced to lower its head until the mouth and upper airway are clear.

Pulmonary Edema and Shock Lung

Camelids are prone to hypoproteinemia and hence are prone to develop edema. This may be worsened by IV fluid treatments. Spontaneous clinical edema appears to be rare. Thus, this condition is most commonly encountered at intensive care facilities, but it may be one of the chief causes of death in camelids at those facilities.

Camelids affected by this condition are usually admitted and examined for another complaint, placed on IV fluids, and later develop dyspnea, tachypnea, and obtundation without fever, possibly losing the ability to remain in sternal recumbency or maintain the head in a normal position. Wheezes may be heard on thoracic auscultation, and pulmonary radiography may reveal a diffuse interstitial pattern or, in severe cases, perihilar consolidation. ABG analysis reveals hypoxemia. Without treatment, the condition is often fatal. Treatment consists of reducing the rate of fluid administration, administering colloids: Start with 8 to 10 mL/kg of 6% Hetastarch or plasma over 30 to 90 minutes, and then reassess; higher doses (up to 40 mL/kg) may be necessary with severe hypoproteinemia. This should be followed by administration of intranasal oxygen and possibly diuretics. Many affected camelids have concurrent upper airway problems such as pharyngeal collapse or dorsal displacement of the soft palate. These camelids have more stridor and often appear to be good candidates for tracheostomy; however, tracheostomy does not result in rapid improvement because of the lower airway disease. As in camelids with upper airway problems, stress and manipulation may worsen the condition, so the clinician should limit treatments to those that are truly beneficial for the patient.

Shock lung is similar except that the stimuli are different and the camelid is more likely to exude blood-tinged froth from the mouth. Many of the stimuli and the pathogenesis are not known. Camelids with septic, toxic, or traumatic conditions, with heat, exertional, or handling stress or other serious diseases frequently develop productive dyspnea and tachypnea. Treatment is the same as for pulmonary edema, with concurrent treatment for the primary condition. The prognosis is decidedly worse, and this condition often culminates in death in a matter of hours.

Pulmonary Dysmaturity

Because of their inconsistent gestation length, judging the maturity of neonatal crias is difficult. Although outward signs may be judged by appearance, lung function can only be judged by performance. Pulmonary dysmaturity results in hypoxemia and, in severe cases, hypercapnia. Because camelids appear to be better adapted to hypoxemia compared with

many other animals, they may appear better than their level of oxygenation warrants. However, that does not mean they thrive under hypoxemic conditions.

Signs of hypoxemia may range from mild decreases in activity to coma, cyanotic membranes, and death. Most camelids with pulmonary dysmaturity appear sluggish at birth. They do not accomplish the tasks of standing, nursing, and passing meconium as quickly or as vigorously as healthy herdmates and may not accomplish them without assistance or at all in some cases. Poor appetite and the tendency to remain recumbent predispose them to neonatal sepsis or poor growth. Owners' attempts to support these crias through tube feeding and provision of warmth and shelter meet limited success.

Whenever a cria appears obtunded since birth or appears to be less vigorous or thrifty than other crias, hypoxemia should be suspected. Thoracic radiography and ABG analysis are the best ways to ascertain the presence of pulmonary dysmaturity. ABG analysis might reveal hypoxemia but does not reveal the specific cause. Dysmature lung appears more radiodense than normal lung and potentially resembles soft tissue in density. Evidence of sepsis may be present or absent.

Crias identified as having pulmonary dysmaturity should immediately be placed on 1 to 3 L/hr supplemental oxygen. With time, that may be enough support to resolve milder cases. Likelihood of success may be estimated by comparing pre- and post-oxygen arterial oxygen content. Crias in which this treatment is likely to work typically achieve PaO_2 of at least 60 mm Hg on oxygen and can usually be removed from supplemental oxygen within 48 hours. Crias that are unable to oxygenate adequately on supplemental oxygen have a poor prognosis.

In extreme cases, surfactant may be administered. This is most successful if done immediately after birth, before tissues have suffered chronic hypoxia and opportunistic infection has set in. Clinical experiences are limited.

Chronic Pneumonia

Chronic infections of the lung are much more frequent findings in camelids compared with acute ones, if one disregards neonatal sepsis. However, the clinical course may be judged to be short because the camelid would have been first noticed to be abnormal only late in the course of the disease. Careful examination and concurrent findings such as emaciation or hypoproteinemia may confirm the chronic nature of the infection.

Chronic infections and infestations may be caused by bacteria, fungi, or parasites. The most common bacteria isolated form abscesses or granulomas. Abscesses may be caused by *Arcanobacter pyogenes*, *S. zooepidemicus*, *Fusobacterium necrophorum*, *C. pseudotuberculosis*, *Rhodococcus equi*, *Burkholderia pseudomallei*, and other bacteria. In some cases, they may have been the original invader; in others, they are likely to be a secondary colonizer. Granulomas are frequently caused by *Mycobacteria*. Several different causes of fungal pneumonia have been identified, including *Aspergillus*, *Histoplasma capsulatum*, *Coccidioides immitis*, and *Cryptococcus neoformans*. Disseminated infections are common with several of the fungi, and nonrespiratory signs may be dominant. Although fungal infections should generally be considered rare, especially outside of the southwestern and northwestern United States, they are one of the more common causes for resting chronic

tachypnea in camelids at our clinic. Parasitic pneumonia may be caused by lungworms or potentially protozoa.

Tuberculosis and other Mycobacterial Infections

Bovine tuberculosis, caused by *M. bovis*, is becoming an increasingly important topic for the camelid industry, particularly in South America, Ireland, and the United Kingdom.[40–44] Infections have been reported in Spain, and the Netherlands, and reactors are reported in New Zealand as well.[45,46] Countries with endemic problems have domestic ruminants or resident wildlife—badgers, bush-tailed possums, or ferrets—that harbor the organism and tend to share living, eating, or drinking spaces with domestic herd animals. In areas without endemic tuberculosis, some outbreaks have been blamed on the importation of infected animals.

Clinically similar disease can be caused by *M. microti* or *M. kansasii*.[47–53] Both are reported most commonly in central and western Europe. *M. microti* is associated with voles; *M. kansasii* is also associated with animal hosts.

If any of these mycobacterial infections are found in an area, local camelids will be at risk, facing the same challenges as other grazing livestock. Transmission is through inhalation or ingestion of materials contaminated by the wildlife or ruminant host. Transmission among camelids and from camelids to humans is suspected as well.[44,54,55] One owner in the United Kingdom was recently diagnosed with the same spoligotype of *M. bovis* that had previously been found in her herd; a common source of infection, rather than direct transmission from a camelid, is also possible. The potential for direct transmission is supported by the presence of oral granulomas and evidence of granulomas communicating with airways in some camelids and also the recent evidence of infection of the udder.[45,56]

Most mycobacteria affect several organ systems but primarily the lung. The basic lesion is the granuloma, and the usual course is slow progression. Camelids covered with fleece and not monitored carefully may develop advanced disease before it is detected, and anecdotal evidence suggests that infection may take years to become apparent in some camelids. Premortem evaluations have been relatively limited. The most common signs are weight loss, weakness, lethargy, recumbency, and anorexia. External lymphadenopathy may be detectable in some animals; internal lymphadenopathy may also be detectable through rectal palpation or imaging studies. The minority show respiratory signs, which may include a shallow breathing pattern with increased expiratory effort, and general harshness of lung sounds. Hematologic abnormalities may include anemia, neutrophilic leukocytosis, and lymphocytosis. Often, marked hyperglobulinemia with concurrent hypoalbuminemia exists. Thoracic radiography and ultrasonographic evaluation of the chest, liver, spleen, and regional lymph nodes may reveal multifocal radiodense or hyperechoic nodular lesions, possibly coalescing into diffuse areas of consolidation. Biopsies or aspirates of these or transtracheal wash samples may reveal acid-fast bacilli, which may be identified by mycobacterial culture and typed by PCR. Typing is important because the different pathogens have different legal aspects and zoonotic potential. Mycobacterial diseases are reportable in many areas.

Because of the advanced nature of the disease upon identification and the zoonotic potential of these organisms, treatment is rarely attempted or successful. Many camelids die shortly after signs are detected. Because of the nonspecific nature of the premortem signs, tuberculosis is not always suspected, and no diagnostic features may be suspected; therefore, it is generally believed that this disease is underreported. Postmortem evaluation reveals the presence of thick-walled masses containing caseous material, particularly in the lung, liver, and regional lymph nodes. Generalized infection is also possible. With *M. microti* an "onion skin like" capsule is frequently described. Some calcification may be evident. Miliary tuberculosis, with widespread serosal nodules may also occur.[45] Histopathologic examination reveals the granulomatous nature of these masses and the presence of mycobacteria. Giant cells are not always evident.[43,52]

Identification of infected camelids has been problematic. Intradermal tuberculin skin testing on the tail is often negative, both in the preclinical and clinical phases of infection, and has also yielded mixed results when done in the axillary region or other alternate sites.[41,43,57–62] False-positives are believed to be rare, but false-negatives are common. Serologic analysis may be complementary.[48,59,63,64] A number of newer, rapid tests, including lateral flow serology, γ-interferon release assay, enzyme-linked immunosorbent assay (ELISA), and dual-path platform testing, are showing promise.[60,61] Skin testing 2 to 3 weeks previously may improve serologic results by evoking an amnestic antibody response.

The most effective means of prevention is decreasing exposure to the host animals. This may be difficult to achieve in some areas. Raising feed and water off the ground (at least 3 feet for badgers) may decrease contamination and also help keep the camelids' heads away from soil-based organisms.

Mycotic Pneumonia and Other Disorders

C. immitis infection (coccidioidomycosis; San Joaquin Valley fever, desert fever) has been reported in California, Arizona, and Texas.[65,66] The causative fungus lives in dry soil, particularly in the Southwestern United States and adjacent regions in Mexico, with the San Joaquin Valley known for high concentrations. Wind may spread the fungus beyond its normal range, but reports of disease far outside of this range are rare. It is postulated that camelids aerosolize and inspire infective arthroconidia during dust baths. Once inspired, arthroconidia grow and mature, and release endospores, which cause local or, much more commonly, disseminated pyogranulomatous disease. Transplacental infection may occur. Many mammalian species are susceptible, although most ruminants suffer only from local disease in the mediastinal lymph nodes. All age groups are susceptible, and in utero transmission has been demonstrated. The most common sites of infection include the lungs, skin, the central nervous system (CNS), bones, and joints.

C. neoformans or *C. gatti* infection has been reported in Oregon, California, British Columbia, and Australia.[67–71] Inhalation of spores is the putative source of infection. The organism is relatively ubiquitous, with some strains associated with bird droppings and others with rotting vegetation; *C. gatti* is potentially associated with the Douglas-fir tree. The disease is usually sporadic in most species and was thought to relate to some degree of immunosuppression, but *C. gatti* appears to affect healthy animals as well. The most common site for infection is the CNS, followed by the lungs and the

kidneys. Meningitis appears to be the most common neurologic form.

H. capsulatum infection has been reported once in Indiana, in a llama imported 7 months previously from Bolivia.[72] In other species, infection has a worldwide distribution. Inhalation of microconidia from the soil is the putative source of infection. Decaying vegetation and bird droppings promote the growth of the fungus. Within the body, the organism multiplies in the yeast form. The primary lesions of the affected llama were in the lungs, with smaller secondary lesions in the reticuloendothelial organs.

Aspergillus infection has been reported in Texas, Wisconsin, Brazil, England, and Australia.[73–77] Aspergillosis is the most common systemic mycosis in domestic animals. The causative fungus is found worldwide on a variety of substrates. Dusty barn air is likely to have high spore counts. Inhalation is the likely cause of some pulmonary infections, but others arise through hematogenous spread after invasion through erosions in the GI tract or skin. The lungs, gut wall, CNS, cornea, and kidney are the most common sites of infection. In some cases, the patient has a preexisting immunosuppressive or erosive condition such as chronic disease, corticosteroid treatment, forestomach acidosis, gastric ulceration, or enteritis. Unlike most other pulmonary fungi, *Aspergillus* grows hyphae within the body.

Rhizopus infection was reported in a single llama in California with dysphagia, bilateral facial paralysis, paralysis of extraocular muscles, and eventually weight loss. On necropsy, fungal rhinitis, nodular pneumonia, and meningoencephalitis were found.

Pneumocystis carinii infection of the lung also appears to be rare.[78] It is seen in a variety of species, often in individuals with reduced immune function. Recently, the organism has been reclassified as a fungus, and the species found to infect animals are different from those that affect humans. Inflammation associated with infection leads to widespread, multifocal, nodular lung disease. Nodules may appear on radiography or other imaging studies or may be too small. Aggregates of cysts also may be fluid in tracheal wash or BAL fluid; *Pneumocystis* cysts are more likely to appear in aggregates than *Cryptococcus* or other fungi affecting the lungs. Cyst also may be identified histopathologically.

Affected camelids may show a variety of signs, depending on where lesions develop. Respiratory signs include tachypnea and exercise intolerance. The most common other signs of the respiratory form or of disseminated (mainly abdominal) infection are anorexia, weight loss, decreased activity, and obtundation. Affected camelids are usually afebrile. CNS infection may occur in the spinal canal or the cranial vault. Signs with spinal infection include increased recumbency, progressive paresis, and hyporeflexia. Intracranial signs may include loss of cranial nerve function or cognition.

Clinical pathology data may similarly be normal or abnormal, depending on the organs affected. Serum increases in nitrogenous waste products, hepatic enzymes, muscle enzymes, and globulins have been seen. Without secondary bacterial infection, hematologic changes are subtle; monocytosis and mild hyperfibrinogenemia may be seen.

Definitive diagnosis requires specialized testing. Thoracic radiography or advanced imaging techniques may reveal granulomatous changes in the lungs, but these can also be caused by *Mycobacteria* or may be confused with bacterial abscesses. Tracheal wash, BAL, cerebrospinal, peritoneal, pleural, or exudative fluid may contain thick-walled, endospore-containing sporangia (*Coccidioides;* 5–50 micrometers [μm]) or "ghost" spherules if the sporangia has burst, or yeast cells (*Histoplasma, Cryptococcus, Aspergillus;* 3–5 μm) either free or within macrophages, or septate hyphae (*Aspergillus* or other, rare fungi). *Cryptococcus* yeast forms are round and surrounded by a mucopolysaccharide capsule, which may increase the diameter to up to 10 μm. Mixing a drop of tracheal wash or cerebrospinal fluid (CSF) with filtered India ink highlights this capsule as a clear halo around the yeast cell. Aggregates of *Pneumocystis* cysts also may be fluid in tracheal wash or BAL fluid; *Pneumocystis* cysts are more likely to appear in aggregates compared with *Cryptococcus* or other fungi affecting the lungs. Cysts also may be identified histopathologically. Samples of most fungi may be left at room temperature for several hours to promote fungal growth of development of hyphae. Caution must be exercised when interpreting the importance of *Aspergillus* hyphae in tracheal wash or exudate samples because the fungus may be found in the upper airway of healthy camelids. Fungal culture and culture characteristics may be used to identify the causative agent. Because of the infectivity of *Coccidioides*, all manipulations should be done on sealed samples or within suitable biohazard hoods. Skin lesions may be aspirated or biopsied. Skin testing and serum complement fixation appear to offer inconclusive results. Qualitative agar gel immunodiffusion for immunoglobulin G (IgG) appears to be relatively sensitive and specific for coccidioidomycosis. A latex agglutination test to detect cryptococcal capsular antigen in body fluids is also available. Most other tests have not been studied sufficiently in camelids.

Specific drug treatments of systemic mycoses have scarcely been investigated in camelids or even ruminants. Historically, most cases have been diagnosed after death. Amphotericin B is the agent that has been most tried for the treatment of systemic mycoses in domestic species and was used unsuccessfully in one affected llama. Other possibilities include imidazole compounds (ketaconazole, fluconazole, miconazole, itraconazole), but their use has not been investigated sufficiently to determine dose or efficacy.

Parasitic Pneumonia

Lungworm infestations of New World camelids appear to be rare and have not been reported in North America. Few reports from South America have been published. Camelids grazed in proximity to infested cattle, sheep, or cervids should be considered at highest risk. Both *Dictyocaulus viviparus* and *D. filari* appear to have the potential to infest camelids.[79] Adult worms live in the bronchi, where they produce eggs that the host coughs up and swallows. Larvae hatch within the intestinal tract and are passed in feces. They mature on pasture to be reingested later. Mature larvae invade through the small intestine and move through blood or lymphatics back to the lung. Most disease relates to the pulmonary larval migration and hence may appear within the 4 to 5 week prepatent period. Affected camelids have productive cough and dyspnea and are typically afebrile. Diagnosis may be made by examining fresh feces using the Baermann technique. Larvae found in fresh feces are likely to be from lungworms or *Strongyloides*.

Pulmonary radiography or computed tomography (CT) may reveal lesions resembling patchy granulomatous disease and tracheal or BAL samples may contain adults, eggs, or larvae. Benzimidazole, avermectin, and levamisole have all shown activity against lungworms in ruminants. Contact animals, especially ruminant hosts, should be treated at the same time.

Neoplasia

The lung is a common site for multicentric and metastatic neoplasms, particularly lymphoma.[3,80–82] At one laboratory, 23% of camelid neoplasms were found in pulmonary tissue. Approximately 50% of camelids with lymphoma have pulmonary involvement.[3] Primary pulmonary or thoracic neoplasms in camelids are rare and include the small percentage of lymphoma cases in which no other site is recognized as well as adenocarcinoma. Respiratory signs rarely predominate and are difficult to distinguish from signs of general weakness. Camelids with severe pulmonary infiltration may show emaciation, inappetence, tachypnea, dyspnea, and exercise intolerance. Normal lung sounds may be difficult to auscultate because of tissue masses or effusions. The major differentials are chronic infection, mycobacterial disease, and fungal or parasitic pneumonia. Finding other sites of neoplastic proliferation often aids in the diagnosis, particularly with lymphoma.

Clinicopathologic changes are usually nonspecific and include anemia, hypoproteinemia, and hypokalemia. Thoracic radiography reveals the infiltrative lesions, and ultrasonography may reveal lesions adjacent to the chest wall or effusions. Cytologic evaluation of effusions is most helpful with lymphoma. Biopsy may be necessary with noneffusive tumors. Currently, experience with chemotherapy in New World camelids is limited.

Reactive Airway Disease

Reactive airway disease has been diagnosed anecdotally in some camelids on the basis of chronic resting tachypnea with or without a cough, lack of fever, and lack of clinicopathologic abnormalities. Although this may be true, a large subset of these camelids are likely to have a more progressive condition such as fungal or mycobacterial pneumonia or thoracic abscesses or neoplasia. A complete evaluation, including thoracic radiography, is recommended before settling on conservative treatment. Bronchodilators or inhaled corticosteroids may lead to some improvement in these cases, especially if the problem is seasonal.

Chylothorax

Chylothorax has been seen at our clinic after thoracic surgery. Traumatic damage to the thoracic duct was suspected. The affected camelid had dyspnea and hypoxemia. Thoracocentesis yielded characteristic milky fluid. No specific treatment was instituted. In other species, surgical repair and various medications have been used.

Hemothorax and Pneumothorax

Hemothorax and pneumothorax are uncommon findings in New World camelids and usually are associated with thoracic trauma. Dyspnea is the major clinical sign, and thoracic radiography or ultrasonography is usually necessary to confirm the diagnosis.

Diaphragmatic Hernia

Diaphragmatic defects account for 1% of all reported birth defects in one study.[9] As clinical reports are rare, most of these are likely to have been found in crias that did not survive the immediate postparturient period or were found incidentally in older camelids.[83,84] Clinical disease is the result of compression of the lungs by herniated abdominal viscera or incarceration of those viscera. Affected crias are likely to be in respiratory distress with tachypnea, dyspnea, and blood gas evidence of hypoxemia and hypoventilation. "Dog sitting" may be an attempt to reduce pressure on the lungs. Depending on which viscera passes through the defect and the tightness of the hernial ring, colic and shock signs may also be present. Diagnosis is accomplished by identifying the herniated viscera on an imaging study; radiography, ultrasonography, or cross-sectional imaging techniques may all be used. Diagnosis may also be achieved during exploratory surgery to identify the cause of the colic. In that case, extreme care must be taken to avoid abrupt, severe pneumothorax on opening the abdomen or retrieving the viscera through the defect. One report described a 7-year-old llama gelding with incarceration of the ileum, cecum, and ascending colon within the thorax through a smooth-edged diaphragmatic defect.[83] Because of the nature of the defect, a congenital lesion was suspected. Herniation recurred 9 months after surgical repair. A second report described a suspected congenital hernia found in an 18-month-old alpaca with recurrent colic episodes.[84] In this case, the heart was found partially within the abdomen after death. Previous exploratory had not revealed the defect but had revealed adhesions involving the jejunum.

Diaphragmatic Paralysis

Phrenic nerve degeneration and diaphragmatic paralysis have been reported in the scientific literature and anecdotally in a variety of locations worldwide.[85,86] The largest study found the disorder only in alpacas less than a year old, but clinical experience suggests that all ages of camelid are susceptible, and many have demonstrated enough normal respiratory function to suggest that this is an acquired lesion. Multiple cases have been identified at specific farms, some sharing a common ancestor.[86] Most clinical cases are bilaterally affected; unilateral cases may exist but may appear normal without extensive examination.

Bilaterally affected camelids usually display an acute onset of progressive dyspnea, with tachypnea, flaring of the nostrils accompanied by expansion of the chest and paradoxic contraction of the abdomen during inspiration, and expansion of the abdomen during expiration. Some appear also to have spinal neurologic signs, including weakness, ataxia, proprioceptive deficits, and knuckling, accompanied by palpable cervical instability in severe cases. Pulmonary sounds are usually normal unless the camelid has aspirated feed or saliva, although the extreme respiratory effort may increase the overall level of noise. Cyanosis and poor body condition may be present but are not consistent findings. Field treatments, usually consisting of antibiotics, steroids or NSAIDs, and mineral and vitamin supplements, yield little to no response.

Blood count and clinical chemistry usually reveal little beyond stress; muscle enzyme activities may be increased in camelids displaying extraordinary respiratory effort or

increased recumbency, and aspiration pneumonia may lead to leukogram changes reflective of inflammation. ABG analysis reveals hypoxemia, usually accompanied by hypercapnea and often by compensated or uncompensated respiratory acidosis. Vitamin, mineral, heavy metal, infectious disease, and toxicologic screenings have revealed no consistent pattern, although vitamin E was judged to be low in one group of alpaca crias.[86]

Radiography may reveal a reduction of lung field through forward bulging of the diaphragm or mild interstitial changes. Fluoroscopy reveals the decreased diaphragmatic function; craniocaudal movement of the diaphragm should be more than one half the length of the adjacent thoracic vertebral body. Plethysmography reveals thoracoabdominal asynchrony, where abdominal volume decreases because of inward collapse of the abdominal musculature during inspiration. Postmortem examination reveals axonal and myelin sheath degeneration of the phrenic nerve and neurogenic atrophy of the diaphragm. Camelids showing other neurologic signs may have evidence of degeneration of other nerves or the spinal cord as well.

Because of the association between spinal neurologic signs and phrenic nerve degeneration, further diagnostic tests, including CSF analysis, cervical radiography, and possibly myelography may be indicated. A variety of neurologic conditions, including vertebral body malformation, abscess, and instability, parasitic migration, tumor, degeneration, and trauma may all be causative and require treatment. Vitamin E deficiency may be a factor in nerve degeneration as well.

Treatment efforts have mainly been supportive or directed at an underlying neurologic disease. Supportive treatments could include administration of nasal oxygen or mechanical ventilation. Treatments of the neurologic disease vary and could include antiinflammatories, antibiotics, antiparasitics, and vitamin E. Prognosis is guarded. Camelids that are in overt respiratory distress or have aspirated are unlikely to thrive. Others appear to tolerate the respiratory compromise and slowly acclimate to it, using the accessory muscles of breathing to drive their respiratory efforts.

Nonseptic Consolidation Associated with Recumbency

Prolonged lateral recumbency leads to nonseptic consolidation of the ventral lung. This is noted outwardly by progressive tachypnea or obtundation. Mucous membranes appear normal unless extreme hypoxemia has occurred. Thoracic radiography reveals an alveolar pattern in the dependent lung. Laboratory evaluation reveals few changes beyond hypoxemia and hypercapnia. Because the changes usually take several days to develop, respiratory acidosis is often compensated by renal retention of bicarbonate. Hypoproteinemia or fluid administration may speed the development of consolidation.

Treatment involves attempts to elevate and reinflate the consolidated side. Mechanical ventilation may help in reinflating a mildly collapsed lung but tends to lead to overinflation of the healthy side when consolidation is too severe. To avoid this condition in recumbent camelids, the position should be changed laterally every 2 hours or kept sternal as much as possible.

Changes of Unknown Significance

Pulmonary Alveolar Histiocytosis or Lipid Pneumonia

In one study, lipid-laden macrophages were found within the alveolar space of camelids, leading to loss of area for air exchange and type II pneumocytosis.[87] All affected camelids were at least 10 years old, and lesions were confined to the dorsal region of the diaphragmatic lung lobes. No specific clinical signs are attributable to the condition, and its importance is unknown.

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