Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Elsevier hereby grants permission to make all its COVID-19-related research that is available on the COVID-19 resource centre - including this research content - immediately available in PubMed Central and other publicly funded repositories, such as the WHO COVID database with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the COVID-19 resource centre remains active.
Diagnostic Sampling and Gross Pathology of New World Camelids

Robert J. Bildfell, DVM, MSc,a,*, Christiane V. Löhr, Dr Med Vet, PhDb, Susan J. Tornquist, DVM, PhDc

This article is intended to give practitioners an overview of diagnostic testing when submitting camelid-origin samples to a veterinary diagnostic laboratory (VDL). A review of some of the common gross findings and diseases when collecting these samples is included.

The basic principles for proper collection and handling of veterinary diagnostic samples have been reviewed elsewhere and generally apply to sampling of New World camelids (NWC).1,2 Guidelines are often available online for each VDL and, when in

KEYWORDS
• Alpaca • Llama • Camelid • Pathology • Disease • Diagnostics • Sampling

KEY POINTS
• New world camels (NWC) may have up to 1.4% reticulocytes in circulation which makes differentiation of regenerative versus nonregenerative anemia difficult.
• Infection with Candidatus Mycoplasma haemolamae (formerly Eperythrozoon) can be associated with anemia and is best diagnosed by polymerase chain reaction assay using EDTA blood samples.
• Cerebrospinal fluid is a highly rewarding but underutilized diagnostic sample to work-up camelid central nervous system disease.
• Poor body condition due to dental attrition or malocclusion is common in older animals.
• Severe hepatic lipidosis may be associated with serofibrinous effusions and widespread petechia and ecchymoses.
• Cardiovascular anomalies are fairly common and can be easily missed unless the heart and large vessels are carefully examined in situ and in context of the lungs.
• Lymphoma is the one of most commonly diagnosed malignancies of NWC.

This article is intended to give practitioners an overview of diagnostic testing when submitting camelid-origin samples to a veterinary diagnostic laboratory (VDL). A review of some of the common gross findings and diseases when collecting these samples is included.

The basic principles for proper collection and handling of veterinary diagnostic samples have been reviewed elsewhere and generally apply to sampling of New World camels (NWC).1,2 Guidelines are often available online for each VDL and, when in
doubt, the laboratory should be contacted for specific instructions. The following notes regard some of the more common tests and the interpretation of gross observations in various organ systems of NWC.

HEMOLYMPHATIC SYSTEM

Unique anatomic features and common incidental gross findings include

- Spleen: white capsule similar to an equine spleen, roughly triangular shape and a serrated margin
- Lymph nodes small and sometimes difficult to locate
- Hemal nodes present
- Lamellated thrombus in splenic vein: blockage rarely complete, splenic parenchyma usually unremarkable.

Notes on Diagnostic Testing

Blood cellular parameters of NWC are best assessed using EDTA samples and have been reviewed elsewhere. Camelid erythrocytes are elliptical with a high hemoglobin concentration relative to other domestic animal species and some cells may contain diamond-shaped hemoglobin crystals (Fig. 1). Distinguishing regenerative versus nonregenerative anemia is difficult because clear criteria are lacking and nonanemic NWCs may have up to 1.4% reticulocytes in circulation. The normal camelid leukon is characterized by a neutrophil-to-lymphocyte ratio similar to dogs, cats, and horses, although the total white cell count tends to be high (8000–22,000 cells/µl). Platelets are small with azurophilic granules visible. The overall leukocytic responses to various insults or stimuli, as well as the gross character of bone marrow, are similar to those anticipated for other species. However, stress-induced leukocytosis and neutrophilia can be substantial, with white cell counts exceeding 40,000 cells/µl in some cases, including remarkable increases in band neutrophil counts.

Common Diseases and Gross Lesions

Subclinical infection with Candidatus Mycoplasma haemolamae (formerly Eperythrozoon) is common but can also be associated with anemia. Organisms can sometimes be visualized on blood smears as round or ring-shaped structures at the erythrocyte

![Fig. 1. Normal blood smear with elliptical camelid erythrocytes and rectangular hemoglobin crystals (arrows).](image)
 margin, but a more sensitive polymerase chain reaction assay uses an EDTA sample. A more sensitive polymerase chain reaction assay uses an EDTA sample.5 Juvenile llama immunodeficiency syndrome (JLIDS) is characterized by illthrift in young animals and can be diagnosed by flow cytometry methods using whole blood to confirm a B-cell deficit.6 A more commonly requested test is assessment of plasma IgG levels in crias via radial immunodiffusion to check for adequate passive transfer of immunity as well as screen for JLIDS.

Lymphoma is one of the most commonly diagnosed malignancies of NWC in the authors’ region and has been described by others.7–9 The usual cytologic and histopathologic diagnostic procedures apply but it should be noted that, from both gross and microscopic aspects, lymphoma in NWC cannot be distinguished from a primitive round cell tumor neoplasm.10 The latter entity is seen most often in younger alpacas and requires immunohistochemical (IHC) testing to separate it from lymphoma. This study found B-cell lymphomas are more common than those of T-cell origin are, especially if the gastrointestinal tract is affected. Diffuse enlargement of the liver (Fig. 2) and internal lymph nodes is common, but a variety of tissues can be affected.

Fig. 2. Lymphoma in the liver of an adult camelid. The organ is swollen and diffusely infiltrated by neoplastic cells. A fibrinous peritonitis is also present.

INTEGUMENTARY SYSTEM

Unique anatomic features and common incidental gross findings include

- Microanatomy follows basic mammalian plan11
- Marked dermal thickening of cervical skin of intact males.

Notes on Diagnostic Testing

Identical to that applied to other species (eg, cultures, skin scrapings, biopsies). Serum zinc levels do not seem to be reliable for the diagnosis of zinc-responsive dermatosis.

Common Diseases and Gross Lesions

Review articles describe the breadth of conditions that may be encountered.12,13 Hyperkeratosis is a feature of several of the most common entities, including chorioplastic mange (Fig. 3). Table 1 provides information on a few of the most common entities.
Cerebrospinal fluid is a highly rewarding, but underutilized, diagnostic sample during the work-up of camelid central nervous system (CNS) disease. Normal range for CSF nucleated cell counts in some laboratories is as low as 0 to 3 cells/μL with lymphocytes predominating over fewer mononuclear cells and rare neutrophils. Total protein levels may range from 31.2 to 66.8 mg/dL and glucose concentrations are about 40% of the serum levels. This sample may also permit visualization of some agents or be
used for specific immune-based reactions, such as serology or latex agglutination. At necropsy, the collection of appropriate fresh samples of CNS tissue can provide a definitive diagnosis for a variety of infectious agents. It is strongly recommended that half of the brain plus suspected areas of spinal cord damage be fixed in 10% neutral buffered formalin. Histopathology, plus or minus IHC tests, can implicate many agents, provide the diagnosis for conditions such as polioencephalomalacia and cerebrovascular accidents, or confirm spinal cord damage for conditions such as cervical spine trauma or spondylolisthesis.

Disease of the peripheral nervous system seems to be uncommon in camelids, although histopathologic examination of nerves is sometimes rewarding, such as in cases of phrenic nerve neuropathy associated with diaphragmatic paralysis.14

**Common Diseases and Gross Lesions**

An excellent review of camelid neurologic diseases has been published by Whitehead and Bedenice.15 Somewhat unique to camelids is the relatively high prevalence of cerebral edema, usually related to fluctuations in blood glucose or protein. A few of the infectious causes of CNS disease and best strategies to diagnose them are reiterated in Table 2. Occasionally, gross postmortem lesions, such as the cloudy meninges of bacterial meningitis, provide a diagnostic clue to the causes of CNS diseases but ancillary tests are typically required for confirmation.

### Table 2

**Diagnostic sampling for some common infectious diseases of the camelid nervous system**

| Disease Condition                                  | Antemortem Diagnostics | Postmortem Diagnostics— Fresh Tissue | Postmortem Diagnostics— Fixed Tissue |
|---------------------------------------------------|------------------------|--------------------------------------|-------------------------------------|
| Cerebrospinal nematodiasis (eg, *Parelaphostrongylus tenuis*, *Baylisascaris*, *Elaeophora*) | CSF – † protein eosinophilia | None | Histopathology |
|                                                   |                        | Rarely, adult *P tenuis* worms are found in the cerebral vascular sinuses |                                    |
| Equine herpesvirus I and arboviral encephalitis (ie, West Nile virus, eastern equine encephalitis) | CSF – † protein lymphocytosis. Serum – serologic titers | Fresh tissue for virus isolation, PCR | Histopathology IHC |
| Listeriosis                                        | CSF – † protein, elevated cell count May see organisms Culture | Bacterial culture of hindbrain or spinal cord | Histopathology of hindbrain or spinal cord IHC |
| Cryptococcosis                                     | As for listeriosis Also latex agglutination test on CSF and serum | Fungal culture May see malacic cortical areas grossly | Histopathology PCR |
| Bacterial meningitis (eg, *Streptococcus spp*, *E coli*) | CSF – † protein, neutrophilia May see organisms Culture | Bacterial culture May see cloudy meninges | Histopathology |

**Abbreviations:** CSF, cerebrospinal fluid; IHC, immunohistochemistry; PCR, polymerase chain reaction.
URINARY SYSTEM

Unique anatomic features and common incidental gross findings include:

- Kidney unipyramidal similar to sheep and goats
- Renal pallor and swelling common in overconditioned camelids; corresponds to lipid storage in renal tubular epithelium.

Notes on Diagnostic Testing

As in other species, serum urea nitrogen and creatinine are useful indicators of renal function in NWC. Urinalysis is infrequently performed because camelid urine samples tend to be difficult to obtain.

Common Diseases and Gross Lesions

Congenital lesions of the urogenital tract are relatively common in camelids and can include various degrees of hypoplasia or aplasia, aberrant pathways for the ureters or urethra, and renal parenchymal cysts. Significant acquired gross renal lesions often reflect acute or chronic embolic bacterial disease, varying from large cortical infarcts to the firm, shrunken, pitted appearance of chronic interstitial glomerulonephritis. The authors have seen only a few cases of cystitis at necropsy, although urolithiasis with obstruction is occasionally diagnosed in male llamas.

PERITONEUM AND PANCREAS

Unique anatomic features and common incidental gross findings: none. However, of note is

- Serous abdominal effusion with scant fibrin strands secondary to a wide array of disease processes, mostly related to hypoproteinemia and/or metabolic diseases.

Notes on Diagnostic Testing

Data published by Cebra and colleagues suggest that parameters of abdominal fluid obtained by abdominocentesis include nucleated cell counts of less than 3000/μL and protein less than 2.5 g/dL normally; however, there may be healthy individuals with values outside this range. Identifying elevated amylase and lipase levels in peritoneal fluid versus that of serum is useful in the antemortem diagnosis of peripancreatic necrosis.

Common Diseases and Gross Lesions

The gross appearance of peripancreatic necrosis at laparotomy or necropsy is a collection of chalky white foci of fat necrosis along the pancreatic margins. A more common peritoneal gross finding is a fibrinosuppurative exudate, with or without plant material, usually reflecting either compromise of gastrointestinal wall integrity or sepsis. Often this fibrinous peritonitis is due to an ulcer located on distal aspect of the lesser curvature of gastric compartment (C) 3 (Fig. 4). A severe fibrinous peritonitis may also be seen as a component of septicemia due to Streptococcus zooepidemicus (alpaca fever).

ALIMENTARY SYSTEM

Unique anatomic features and common incidental gross findings include:

- Dentition
- Gastric compartments; large C1, highly glandular C2, long tubular C3
- Extensive spiral colon, a common site for obstruction
Slightly pebbled appearance of esophageal mucosa (reflecting mucous glands)
Gastroliths: hard green-brown concretions within glandular diverticula (saccules) of C1
Lack of papillae on the mucosa of gastric compartments
C3: minor cracks or fissures in normally thicker aborad third of C3 (acid-secreting portion)
Proximal duodenum: ampulla and adjacent hair-pin turn are common sites for trichophytobezoars (obstructive, not incidental).

Notes on Diagnostic Testing
As for all species, a broad panel of ancillary tests (ie, bacteriology, virology, and parasitology) is required to adequately investigate infectious causes of weight loss or diarrhea in NWC. Fecal flotation using a double-centrifugation method with a sucrose solution is strongly recommended to detect dense oocysts, such as those of *Eimeria macusaniensis*, *Trichuris spp*, and *Nematodirus spp*. During necropsies, the prompt fixation of histopathological samples from all levels of the gastrointestinal tract is recommended and these samples may be also used for IHC tests.

Common Diseases and Gross Lesions
Poor body condition due to dental attrition or malocclusion is often seen in older animals. In the authors’ region, enteric salmonellosis, paratuberculosis, and bovine virus diarrhea virus infections are rarely seen in NWC, but the approaches to diagnosis of these entities is the same as for bovine patients. Camelids may develop coccidiosis due to several *Eimeria* species but *E. macusaniensis* deserves special mention because it can cause significant clinical disease in any age of camelid during the unusually long prepatent period or in association with low oocyst counts. Some infections may become complicated by *Clostridium perfringens*. Some features of common alimentary diseases are listed in Table 3.

HEPATIC SYSTEM
Unique anatomic features and common incidental gross findings include

- Liver on right side of abdomen, fimbriated appearance along the caudal edges
- Gall bladder absent
- Bile and pancreatic secretions enter duodenum via common opening
- Randomly located tan-white, 1 to 3 mm, mineralized granulomatous or fibrotic lesions, presumably sequelae to migrating parasitic larvae or episodes of bacteremia.
| Disease               | Clinical Features                       | Antemortem Diagnostics                        | Gross Lesions and Diagnostics |
|-----------------------|-----------------------------------------|-----------------------------------------------|------------------------------|
| Clostridiosis         | Hemorrhagic diarrhea in juveniles       | Anaerobic culture and toxin detection from feces | Bloody gut content          |
| *C perfringens*       |                                         | Culture, toxin detection on fresh (<6 h) gut samples. |                             |
| Coccidiosis           | Anorexia, lethargy, diarrhea, colic     | Centrifugal fecal float with sugar solution   | Often normal but may see focal or segmental thickening of small intestine |
| *Eimeria macusaniensis* | Hypoproteinemia Any age                | for large oocysts for large oocysts            | Histopathology from multiple sites |
| Coronavirus           | Diarrhea                                | EM of feces                                    | Fluid filled loops          |
|                       | Any age but especially juveniles       |                                               | Histopathology and IHC      |
| Cryptosporidiosis     | Watery to yellow diarrhea in juveniles  | Fecal sample, flotation                        | Dilated large intestine     |
|                       |                                         | Modified acid-fast stained direct smears       | Variable intestinal congestion |
|                       |                                         | IFAT, ELISA, PCR                               | Histopathology              |
|                       |                                         |                                               | Fecal tests                 |
| Gastric ulcer         | Anorexia                                | Bile acids often found in C3 fluid            | Often located in distal third of C3, lesser curvature |
|                       | Weight loss                             | Abdominocentesis shows fibrinous peritonitis ± bacteria or ingesta in late stages of development | Concurrent fibrinosuppurative peritonitis and hepatic lipidosis common |
|                       | Anemia                                  |                                               |                             |
| Giardiasis            | Watery diarrhea in juveniles            | Fecal sample—IFAT                             | No distinctive gross lesions |
|                       |                                         | ELISA—only significant if large numbers       | Test as per antemortem      |
| Megaesophagus         | Weight loss                             | Contrast studies occasionally helpful          | Ring anomalies seen in a few young alpacas |
|                       | May present with obstruction            | Esophagitis rarely seen if scoped              | Most cases are in adults and idiopathic |
|                       |                                         |                                               |                             |
| Nematodiasis          | Weight loss                             | Centrifugal fecal float with sugar solution   | Abdominal effusion           |
|                       | Hypoproteinemia                         | Lectin test to identify *Haemonchus*           | Loss of muscle mass seen in most fatalities |
|                       | Often anemia (can be severe with hemonchosis) | Small numbers of *Trichuris*                  |                             |
|                       |                                         | *Nematodirus* can be significant               |                             |

**Abbreviations:** ELISA, Enzyme Linked Immunosorbent Assay; EM, electron microscopy; IFAT, indirect immunofluorescent antibody test; PCR, polymerase chain reaction.
Notes on Diagnostic Testing

Serum biochemical assays for camelid hepatic disease parallel those for large animal species with aspartate transaminase (AST) and sorbitol dehydrogenase (SDH) being particularly useful for hepatocellular damage, whereas gamma glutamyl transferase (GGT) best measures cholestasis. Bile acid levels can aid hepatic function assessment (normal range 1–23 \( \mu \text{mol/L} \)). Transabdominal hepatic biopsy can provide critical diagnostic information but, in rare instances of severe hepatic lipidosis, it has been associated with fatal hemorrhage (unpublished observation).

Common Diseases and Gross Lesions

The gross appearance of hepatic lipidosis in camelids mirrors this condition in other species; a pale swollen friable organ that often has a zonal pattern of cream and brown-red areas on closer examination. Cebra\(^{24}\) has reviewed some of the underlying pathophysiology of this condition. Useful serum biochemical changes for this very common problem include elevations in AST, GGT, SDH, bile acids, nonesterified fatty acids, and beta-hydroxybutyrate. Severe hepatic lipidosis may be associated with serofibrinous effusions and widespread petechia and ecchymoses.

\textit{Fasciola hepatica} can cause patent infections in camelids and the gross hepatic appearance of fascioliasis can include tortuous hemorrhagic tracks of acute necrosis, irregularly fissured areas of fibrosis, biliary tree accentuation with black parasitic hematin, and sometimes cyst formation with intraluminal thick brown exudate. Some camelids develop generalized cholangiolar proliferation and fibrosis as a consequence of apparently minor trematodiases, resulting in a diffusely firm organ.\(^{25}\) Another unusual sequel to fluke infection is fibrinosuppurative endocarditis.\(^{26}\)

MUSCULOSKELETAL SYSTEM

Unique anatomic features and common incidental gross findings include

- Esophageal hiatus of diaphragm has cartilaginous ring.

Notes on Diagnostic Testing

Serum biochemistry for AST and creatine kinase are used for assessment of muscle health, recognizing the potential for a hepatic contribution to AST. Except for a slightly higher protein level, normal joint fluid parameters for arthrocentesis samples are not significantly different from those of horses.\(^{27}\) Bacterial culture may yield various opportunistic pathogens in septic crias but septic arthritis is seldom documented in adult camelids at the authors’ facility.

Common Diseases and Gross Lesions

Conformation problems, spondylosis, and degenerative arthropathies are common, but these seldom require sample submission to a VDL. The finding of serum hypophosphatemia in association with typical radiographic changes supports a diagnosis of rickets in young camelids, usually due to hypovitaminosis D, particularly in animals with dark-hair coats born during periods of short daylight.\(^{28-30}\) Another common skeletal problem is swelling and distortion of the mandible. Although a few of these cases are neoplastic processes, most are the result of osteomyelitis, usually initiated by a tooth root abscess.\(^{22}\) If radiographic findings are not diagnostic, aggressive sampling is required to penetrate the outer layer of reactive bone and obtain representative biopsy material plus samples for aerobic and anaerobic bacterial culture.
RESPIRATORY SYSTEM

Unique anatomic features and common incidental gross findings include

- Lungs: lobation and gross appearance similar to horse
- Nasal cavity: long and slender, obligate nasal breathers
- Edematous lungs develop rapidly postmortem, similar to small ruminants
- Alveolar histiocytosis: oval to linear white zones or clusters of small white foci along dorsal aspect of caudal lobes, not associated with textural change, correspond to clusters of alveoli filled by foamy macrophages (endogenous lipid) plus or minus mild alveolar emphysema.

Notes on Diagnostic Testing

Analysis of transtracheal aspirates has been infrequently reported. However, submission of wash fluid in EDTA for cytologic analysis, plus another serum tube aliquot for bacterial or fungal growth, may yield valuable diagnostic information. Culture of samples of pneumonic lungs collected at necropsy is critical in achieving definitive diagnosis of respiratory pathogens, such as Cryptococcus, Bordetella, Actinobacillus spp, and Streptococcus zooepidemicus, and should be submitted in containers separate from samples of digestive tract. Fungi are difficult to recover from samples that have been frozen.

Common Diseases and Gross Lesions

Assessment of the respiratory tract of newborn crias must include checking for choanal atresia, especially if other craniofacial anomalies are present. Crias with choanal atresia often have concurrent anomalies of the optic tract, brain, and/or cardiovascular system; a candidate gene for this complex of congenital defects has been identified. A severe fibrinous pleuritis may be seen as a component of alpaca fever (streptococcal septicemia). At the authors’ facility, aspiration pneumonia is a more common diagnosis than primary respiratory disease in NWC and appropriate histopathology samples assist this diagnosis. Some cases are iatrogenic; others are secondary to megaesophagus or CNS diseases. In most cases of primary pneumonia, common pyogenic bacteria are isolated. Granulomatous pneumonia in NWC in some regions of the United States may be associated with potentially zoonotic fungal infections: cryptococcosis in the Northwest and coccidio-mycosis in the Southwest, so appropriate precautions should be taken when collecting and shipping specimens.

CARDIOVASCULAR SYSTEM

Unique anatomic features and common incidental gross findings include

- Anatomy similar to other mammals
- Heart murmurs common in stressed animals may not have an anatomic basis, especially in older animals
- Arterial sclerosis with mineralization and even ossification in older animals; focal firm to gritty mural plaques in descending aorta and at branching points of large arteries; clinical significance unknown.

Notes on Diagnostic Testing

Cardiovascular anomalies are fairly common in North American NWC and can be easily missed unless the heart and large vessels are carefully examined in situ and
in context of the lungs. When in doubt and when submission of the whole animal is not possible, submission of the entire, unfixed, and chilled pluck provides best results. Samples of heart for histopathology should include sections of ventricular septum and free wall, preferably including papillary muscle.

**Common Diseases and Gross Lesions**

NWC are prone to a wide array of congenital cardiac defects, including ventricular septal defects (most common); complex anomalies, including transpositions of the large vessels; and tetralogy of Fallot.\(^3\) Endocarditis is not a common diagnosis but has been described especially in association with fascioliasis (see liver).\(^2\) Endocarditis seems to be more commonly mural than valvular in NWC and vegetations may fill large portions of the ventricular lumen. Various septicemias, (especially *Streptococcus zooepidemicus*) can result in cardiovascular collapse with concurrent effusion of fibrinous exudates. Both antemortem and postmortem diagnosis of such conditions relies on appropriate culture of effusions or whole blood samples.

**REPRODUCTIVE SYSTEM**

Unique anatomic features and common incidental gross findings include:

- Female tract similar to equine
- Male tract includes paired bulbourethral glands, a very small prostate, sigmoid flexure, and distally located cartilaginous penile process; lacks seminal vesicles
- Fourth membrane (epithelion) clings to fetus (Fig. 5); this delicate membrane is not the amnion
- Hippomanes (allantoic calculi) are often present in the allantoic sac
- Long umbilical cord occasionally results in abortion due to umbilical cord torsion
- Chorionic placenta is diffuse (no cotyledons) with minor variation in the density and/or length of villi (Fig. 5).

![Fig. 5. Normal-term llama fetus with associated placental membranes. Poorly villous area on medial aspect of chorionic surface (long arrow). Amnion and umbilical cord (short arrow). Note that a fourth membrane (epithelion) also covers the fetus.](image-url)
Notes on Diagnostic Testing

Diagnostic samples for infertility problems include uterine swabs and biopsies. The former are not useful unless guarded swabs are used because some of the common pathogenic isolates (Escherichia coli, Streptococcus sp, Pseudomonas sp, Klebsiella sp, Actinomyces pyogenes, and Staphylococcus sp) populate the lower portions of the tract. Powers and colleagues described a grading system for NWC endometrial biopsies that continues to be useful for diagnostic classification and prognosis.

Most cases of abortion or stillbirth of NWC are idiopathic and many of these are presumed to be stress-induced. Placental insufficiency similar to that of equids may occur in NWC. Therefore, it is critical to accurately determine fetal age (crown-rump measurements) and to collect multiple placental samples for histopathologic assessment. The pregnant (almost always left horn), nonpregnant horn, and the body should be sampled, taking care to avoid the medial aspect of the horns, which is normally villus-poor. General principles apply in terms of collecting abortion samples and the panel of samples collected for NWC cases at the authors’ laboratory for an abortion screen is listed below:

- Bacteriology—stomach content, liver, lung, and placenta
- Virology or molecular diagnostics—lung, thymus, kidney
- Serology—fetal thoracic fluid and maternal serum
- Analytic chemistry—liver for mineral analysis
- Histopathology—formalin fixed samples of placenta (multiple), lung, liver, heart, skeletal muscle, kidney, brain, and thyroid, plus or minus other tissues.

Common Diseases and Gross Lesions

Congenital reproductive tract lesions are common causes of infertility in both genders and may be confirmed at necropsy. The urinary tract or, in rare cases, the terminal digestive tract, may also be involved. Foci of placental pallor due to mineralization are common but their significance is uncertain. Abortion and stillbirth are common and often a cause cannot be identified.

ENDOCRINE SYSTEM

Unique anatomic features and common incidental gross findings include

- No unique anatomic features
- Thyroid glands of geriatrics may have incidental cysts filled with yellow to clear fluid.

Notes on Diagnostic Testing

Other than the assessment of reproductive hormones for pregnancy status and abnormalities in gonadal function, antemortem diagnostics are rarely pursued in this system. Serum samples are typically adequate.

Common Diseases and Gross Lesions

Secondary effects of endocrine-related disease include hepatic lipidosis and the skeletal changes of rickets, but significant gross lesions affecting endocrine organs per se are rare.

SUMMARY

The general principles of diagnostic sampling apply well to the diseases of NWC. However, recognition of normal and incidental changes, combined with a focus on
the more common diseases of these species, will maximize the return on diagnostic
effort for these unique animals.

ACKNOWLEDGMENTS

The authors thank Chris Cebra and Pat Long for helpful comments on this article.

REFERENCES

1. Bildfell RJ. Collection and submission of laboratory samples. In: Kahn CM, editor. The Merck veterinary manual. 10th edition. Whitehouse Station (NJ): Merck & Co., Inc; 2010. p. 1463–9.
2. Webb DM. Getting the most from a veterinary diagnostic laboratory—a pathologists’ perspective. Part II. Sampling and testing. Comp Cont Ed Vet 1995;17:1043–71.
3. Hamir AN, Timm KI, Smith BB. Thrombosis of the splenic vein in llamas (Lama glama). Vet Rec 2000;146:226–8.
4. Tornquist S. Clinical pathology of llamas and alpacas. Vet Clin North Am Food Anim Pract 2009;25:311–22.
5. Tornquist SJ, Boeder LJ, Parker JE, et al. Use of a polymerase chain reaction assay to study the carrier state in infection with camelid Mycoplasma haemolama, formerly Eperythrozoon spp. infecting camelids [abstract]. Vet Clin Pathol 2002;31:153–4.
6. Davis WC, Heirman LR, Hamilton MJ, et al. Flow cytometric analysis of an immunodeficiency disorder affecting juvenile llamas. Vet Immunol Immunopathol 2000;74(1–2):103–20.
7. Cebra CK, Garry FB, Powers BE, et al. Lymphosarcoma in 10 New World Camelids. J Vet Intern Med 1995;9(6):381–5.
8. Shapiro JL, Watson P, McEwen B, et al. Highlights of camelid diagnoses from necropsy submissions to the Animal Health Laboratory, University of Guelph, from 1998 to 2004. Can Vet J 2005;46(4):317–8.
9. Valentine BA, Martin JM. Prevalence of neoplasia in llamas and alpacas (Oregon State University, 2001-2006). J Vet Diagn Invest 2007;19(2):202–4.
10. Martin JM, Valentine BA, Cebra CK, et al. Malignant round cell neoplasia in llamas and alpacas. Vet Pathol 2009;46:288–98.
11. Fleis RI, Scott DW. The microanatomy of healthy skin from alpacas (Vicugna pacos). J Vet Diagn Invest 2010;22(5):716–9.
12. Foster A, Jackson A, D’Alterio GL. Skin diseases of South American camelids. In Pract 2007;29:216–23. http://dx.doi.org/10.1136/inpract.29.4.216.
13. Scott DW, Vogel JW, Fleis RI, et al. Skin diseases in the alpaca (Vicugna pacos): a literature review and retrospective analysis of 68 cases (Cornell University 1997-2006). Vet Dermatol 2011;22(1):2–16.
14. Byers S, Barrington G, Nelson D, et al. Neurological causes of diaphragmatic paralysis in 11 alpacas (Vicugna pacos). J Vet Intern Med 2011;25(2):380–5. http://dx.doi.org/10.1111/j.1939-1676.2010.0661.x [Epub 2011 Jan 31].
15. Whitehead CE, Bedenice D. Neurologic diseases in llamas and alpacas. Vet Clin North Am Food Anim Pract 2009;25:385–405.
16. Smith JA. Noninfectious diseases, metabolic diseases, toxicities, and neoplastic diseases of South American camelids. Vet Clin North Am Food Anim Pract 1989;5(1):101–43.
17. Cebra CK, Tornquist SJ, Reed SK. Collection and analysis of peritoneal fluid from healthy llamas and alpacas. J Am Vet Med Assoc 2008;232(9):1357–61.
18. Pearson EG, Snyder SP. Pancreatic necrosis in New World camelids: 11 cases (1990–1998). J Am Vet Med Assoc 2000;217(2):241–4.
19. Cebra CK, Heidel JR, Cebra ML, et al. Pathogenesis of *Streptococcus zooepidemicus* infection after intratracheal inoculation in llamas. Am J Vet Res 2000;61:1525.
20. Fowler ME. Congenital/hereditary conditions. In: Fowler ME, editor. Medicine and surgery of South American camelids: llama, alpaca, vicuña, guanaco. 2nd edition. Ames (IA): Iowa State University Press; 1998. p. 468–97.
21. Cebra CK, Valentine BA, Schlipf JW, et al. *Eimeria macusaniensis* infection in 15 llamas and 34 alpacas. J Am Vet Med Assoc 2007;230:94–100.
22. Niehaus A. Dental disease in llamas and alpacas. Vet Clin North Am Food Anim Pract 2009;25(2):281–93.
23. Rosadio R, London P, Perez D, et al. *Eimeria macusaniensis* associated lesions in neonate alpacas dying from enterotoxemia. Vet Parasitol 2010;168:116–20.
24. Cebra CK. Disorders of carbohydrate or lipid metabolism in camelids. Vet Clin North Am Food Anim Pract 2009;25:339–52.
25. Hamir AN, Smith BB. Severe biliary hyperplasia associated with liver fluke infection in an adult alpaca. Vet Pathol 2002;39:592–4.
26. Firshman AM, Wunschmann A, Cebra CK, et al. Thrombotic endocarditis in 10 alpacas. J Vet Intern Med 2008;22:456–61.
27. Waguespack RS, Belknap EB, Spano JS, et al. Analysis of synovial fluid from clinically normal alpacas and llamas. Am J Vet Res 2002;63:576–8.
28. Van Saun RJ, Smith BB, Watrous BJ. Evaluation of vitamin D status of llamas and alpacas with hypophosphatemic rickets. J Am Vet Med Assoc 1996;209(6):1128–33.
29. Van Saun RJ. Nutritional diseases of llamas and alpacas. Vet Clin North Am Food Anim Pract 2009;25(3):797–810.
30. Van Saun RJ. Nutritional diseases of South American camelids. Small Rumin Res 2006;61:153–64.
31. Gerros TC, Andreasen CB. Analysis of transtracheal aspirates and pleural fluid from clinically healthy llamas (*Lama glama*). Vet Clin Pathol 1999;28:29–32.
32. Whitehead CE. Management of neonatal llamas and alpacas. Vet Clin North Am Food Anim Pract 2009;25(2):353–66.
33. Reed KM, Bauer MM, Mendoza KM, et al. A candidate gene for choanal atresia in alpaca. Genome 2010;53(3):224–30.
34. Boon JA, Knight AP, Moore DH. Llama cardiology. Vet Clin North Am Food Anim Pract 1994;10(2):353–70.
35. Margiocco ML, Scansen BA, Bonagura JD. Camelid cardiology. Vet Clin North Am Food Anim Pract 2009;25(2):423–54.
36. McKenzie EC, Seguin B, Cebra CK, et al. Esophageal dysfunction in four alpaca crias and a llama cria with vascular ring anomalies. J Am Vet Med Assoc 2010;237(3):311–6.
37. Tibary A, File C, Anousassi A, et al. Infectious causes of reproductive loss in camelids. Theriogenology 2006;66:633–47.
38. Powers BE, Johnson LW, Linton LB, et al. Endometrial biopsy technique and uterine pathologic findings in llamas. J Am Vet Med Assoc 1990;197(9):1157–62.
39. Löhrr CV, Bildfell RJ, Heidel JR, et al. Retrospective study of camelid abortions in Oregon. Vet Pathol 2007;44:753.
40. Schaefer DL, Bildfell RJ, Long P, et al. Characterization of the microanatomy and histopathology of placentas from aborted, stillborn and normally delivered alpacas (*Lama pacos*) and llamas (*Lama glama*). Vet Pathol 2011;49(2):313–21.
41. Bravo PW. Reproductive endocrinology of llamas and alpacas. Vet Clin North Am Food Anim Pract 1994;10(2):265–79.
42. Schulman FY, Krafft AE, Janczewski T, et al. Camelid cutaneous fibropapillomas: clinicopathologic findings and association with papillomavirus. Vet Pathol 2003;40:103–7.
43. Cebra CK, Stang BV, Smith CC. Development of a nested polymerase chain reaction assay for the detection of *Eimeria macusaniensis* in camelid feces. Am J Vet Res 2012;73:13–8.
44. Cebra CK, Tornquist SJ, Bildfell RJ, et al. Bile acids in gastric fluids from llamas and alpacas with and without stomach ulcers. J Vet Intern Med 2003;17:567–70.
45. Watrous BJ, Pearson EG, Smith BB, et al. Megaesophagus in 15 llamas: a retrospective study (1985-1993). J Vet Intern Med 1995;9(2):92–9.
46. Jurasek ME, Bishop-Stewart JK, Storey BE, et al. Modification and further evaluation of a fluorescein-labeled peanut agglutinin test for identification of *Haemonchus contortus* eggs. Vet Parasitol 2010;169(1–2):209–13 [Epub 2009 Dec 21].