ABOMASAL EMPTYING DEFECT IN SHEEP

BASICS

OVERVIEW
Abomasal emptying defect (AED) is a syndrome of mature Suffolk sheep characterized by chronic, progressive weight loss and abomasal dilatation in the absence of mechanical obstruction.

INCIDENCE/PREVALENCE
Unknown

GEOGRAPHIC DISTRIBUTION
N/A

SYSTEMS AFFECTED
Digestive

PATHOPHYSIOLOGY
• The pathogenic mechanism is unclear. Neurotoxicosis has been suggested.
• The syndrome shares some characteristics with chronic idiopathic intestinal pseudo-obstruction of humans. Affected individuals have clinical signs suggesting partial or complete gastric obstruction, when none is present.
• Morphologic investigations of human patients indicate degenerative changes in the smooth muscle or the tunica muscularis and/or neurons of the enteric plexus.

HISTORICAL FINDINGS
• Condition occurs sporadically, typically affecting a single individual. Flock management is usually excellent despite affected individuals within it.
• Owners may report weight loss in affected animals despite providing extra attention, nutrition, and anthelmintic treatment. Owners may note that the animal appears "bloated" despite inappetence.

SIGNALMENT
• AED affects sheep, with an anecdotal report in a cross-bred dairy type goat.
• Predominantly affects Suffolks; also reported in the Hampshire, Dorset, and Texel breeds.
• Affected animals are typically over 2 years old and of either gender.

PHYSICAL EXAMINATION FINDINGS
• Body temperature is within normal limits unless concurrent disease is present. Heart and respiration rates may be normal to increased. Fecal consistency usually normal, but volume often decreased.
• Abdominal conformation may be normal; bilateral, asymmetrical abdominal distention may occur (distension of the left paralumbar fossa and right ventrolateral abdomen when the animal is viewed from behind); unilateral distension may be present (right ventrolateral aspect of the abdomen).
• Rumen contractions are variable. Rumen hyperactivity can be dramatic in affected sheep and the left paralumbar fossa appears to be in constant motion, reflecting almost constant rumen activity.
• Sheep with AED are in varying stages of cachexia and their abdominal wall feels "thin" due to muscle wasting. Abdominal organs may also lack tone or give the impression of being fluid filled. In some instances, the caudal border of the abomasum may be visible and palpable as it extends beyond the last rib on the ventrolateral aspect of the abdomen. The distended abomasum usually feels fluid filled rather than the doughy or firm consistency often associated with abomasal impaction in cattle.

GENETICS
• Information regarding genetic predisposition is limited. Pedigree analysis of a flock in which 11/92 Suffolks were affected during a 5-month period did not identify a hereditary pattern.

CAUSES AND RISK FACTORS
Unknown

DIAGNOSIS

DIFFERENTIAL DIAGNOSES
• Differential diagnoses for chronic weight loss in adult sheep include caseous lymphadenitis and other chronic infections including scrapie, Johne's disease, malnutrition, dental problems, parasitism, neoplasia.
• Historical and clinical findings are fairly specific to AED.
• Vagal indigestion with resulting ruminal distension is uncommon in sheep.

CBC/BIOCHEMISTRY/URINALYSIS
• Hematology and serum chemistry analysis are usually normal.
• Metabolic alkalosis with hypochloremia and hypokalemia observed with proximal GI obstruction in cattle is not consistently noted with AED.
• Elevations in liver enzymes (AST, SDH, GGT) may be noted.
• Increased intra-abdominal pressure from a distended abomasum may lead to secondary liver congestion and ischemia and can precipitate leakage of hepatic enzymes.
• Urinalysis usually unremarkable.

OTHER LABORATORY TESTS
• Elevated rumen chloride concentration is useful in supporting a diagnosis of AED.
• Normal rumen chloride in sheep is ≤15 mEq/L. Affected sheep will have at least a two-fold increase. Rumen fluid samples are easily obtained by percutaneous aspiration of the rumen from a site in the ventrolateral aspect of the left paralumbar fossa.

IMAGING
• Abdominal radiography may be helpful; however, unless the animal can be positioned for an oblique abdominal radiographic view, results will be difficult to interpret.
• Abdominal ultrasonography may be more useful than radiographic imaging. A 3 to 5 MHz linear or convex array can provide adequate images of the abomasum. When placed on the lower right abdomen, the normal abomasum will not extend beyond the last rib. In animals with AED the abomasum will usually appear 2 to 4 times normal size.

OTHER DIAGNOSTIC PROCEDURES
N/A

PATHOLOGIC FINDINGS
• Gross necropsy reveals a greatly distended abomasum and patent pylorus.
• Abomasal contents are usually liquid but may be dry. Histopathologic changes in the abomasum include smooth muscle degeneration, vacuolation, and varying degrees of necrosis. Degenerative changes have been reported in the celiacomesenteric ganglia.

TREATMENT

THERAPEUTIC APPROACH
• The prognosis for recovery with intensive treatment is variable and dependent upon the duration of abomasal dysfunction and distention. Medical therapy using cathartics and laxatives, and surgical therapy (abomasotomy) have had limited success.
• In animals that are good surgical candidates, abomasotomy followed by metoclopramide and supportive fluid therapy has provided some success.

SURGICAL CONSIDERATIONS AND TECHNIQUES
An abomasotomy is best performed under general anesthesia, although a local line block can be used. The animal is placed in left lateral recumbency and a right paracostal approach provides excellent access to the abomasum. Subsequently the abomasum is opened and its contents removed, and the organ is flushed and closed in a routine manner. Treatment with metoclopramide should be used as an adjunct to the surgery. Concurrent fluid replacement and electrolyte correction therapy is critical to survival and success.

MEDICATIONS

DRUGS OF CHOICE
Metoclopramide (0.1 mg/kg, q8h, SQ) as an adjunct to abomasotomy has been reported to improve abomasal motility. This medication should not be used if GI obstruction is suspected.

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ABOMASAL EMPTYING DEFECT IN SHEEP (CONTINUED)

Erythromycin (8.8 mg/kg, IM) increased abomasal emptying rate in dairy calves; pre-operative administration (10 mg/kg, IM) increased abomasal emptying after surgical correction of left displaced abomasum in dairy cows. Erythromycin may therefore provide some benefit in treatment of AED.

CONTRAINDICATIONS
Neostigmine should not be used in affected animals since it increases frequency rather than strength of rumen contractions.

PRECAUTIONS
Appropriate milk and meat withdrawal times apply to all compounds administered to food-producing animals.

POSSIBLE INTERACTIONS
N/A

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
- The earlier that AED is recognized and treated, the better the prognosis; however, prognosis is guarded for long-term recovery regardless.
- In certain circumstances such as a ram completing a breeding season, or a late gestation ewe completing her pregnancy, a fair to good short-term prognosis may be offered if intensive treatment is provided early.

POSSIBLE COMPLICATIONS
- Complications related to abomasotomy: surgical dehiscence of the abomasal incision (especially if the abomasal wall has undergone degenerative changes) and dehiscence of the abdominal incision may occur (more likely in a debilitated patient).
- Once the condition is recognized, if treatment is declined, euthanasia should be offered as a humane resolution.

CLIENT EDUCATION
Owners of Suffolk sheep should be familiar with the breed predisposition for AED and educated regarding the clinical presentation.

PATIENT CARE
If intensive therapy is undertaken, the animal should be observed for attitude, appetite, fecal production, and abdominal conformation. Signs of improvement following abomasotomy and during metoclopramide therapy may include improvement in attitude and appetite, increased fecal production and decreased abdominal distention.

PREVENTION
Because the underlying cause and heritability of AED is unknown, recommendations cannot be made.

MISCELLANEOUS

ASSOCIATED CONDITIONS
Other concurrent conditions may occur with AED. Pneumonia and other organ failure can be secondary to any chronic debilitating disease.

AGE-RELATED FACTORS
AED usually occurs in mature sheep.

ZOONOTIC POTENTIAL
N/A

PREGNANCY
In spite of treatment, pregnant animals may abort. Pregnant animals (especially mid- to late-term) represent an increased surgical risk.

BIOSECURITY
N/A

PRODUCTION MANAGEMENT
AED is usually observed in a single animal from a well-managed flock.

SYNONYMS
- Abomasal dilatation and emptying defect
- Abomasal impaction
- Acquired dysautonomia
- Functional pyloric stenosis
- Ovine abomasal enlargement

ABBREVIATIONS
- AED = abomasal emptying defect
- AST = aspartate transaminase
- GGT = gamma-glutamyltransferase
- SDH = sorbitol dehydrogenase

SEE ALSO
Abomasal Impaction

Suggested Reading

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ABOMASAL IMPACTION

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BASICS

OVERVIEW
• Abomasal impaction occurs when there is obstruction to the passage of fluid and ingesta from the abomasum through the pylorus by feed, sand, gravel or other foreign bodies, or as a result of neurologic dysfunction from various conditions.
• Pyloric obstruction from improperly placed percutaneous fixation of left-displaced abomasum ("roll and toggle") can also result in abomasal impaction in cattle.
• Clinical signs can be acute or chronic and are characterized by anorexia, decreased or scant feces, abomasal distension, weakness, dehydration, and signs of abdominal pain.
• Abomasal impaction typically occurs in cattle and occasionally in sheep. It is usually sporadic, but morbidity can be associated with feeding of low-quality forages.
• This disorder has a high mortality rate.
• Abomasal emptying defect (AED) is a disorder that primarily affects Suffolk sheep and is characterized by distension and impaction of the abomasum.

INCIDENCE/PREVALENCE
Low morbidity.

GEOGRAPHIC DISTRIBUTION
Worldwide. Occurs more commonly in regions where low quality roughage or low energy diets are fed.

SYSTEMS AFFECTED
Digestive

PATHOPHYSIOLOGY
• Physical obstruction of outflow from the abomasum into the duodenum occurs. This may be due to packing of straw or other poor-quality roughages, or sand or gravel in the abomasum.
• Damage to branches of the vagus nerve as a result of traumatic reticuloperitonitis, lymphoma or other disorders can decrease the emptying ability of the abomasum.
• Failure of fluid to move from the abomasum into the intestines results in dehydration and starvation.
• Sequestration of hydrochloric acid in the abomasum can result in metabolic alkalosis.
• In sheep, no histologic lesion has been consistently associated with AED, and the etiology is unknown. In one study, histologic examination of celiacomesenteric ganglia from affected sheep revealed scattered chromatolytic or necrotic neurons, without inflammation. Chromatolytic neurons were observed more frequently in AED-affected sheep than in healthy Suffolk sheep. Neuronal necrosis was not observed in any of the healthy sheep. Neuronal lesions of AED resemble dysautonomic diseases of humans and other animals.

HISTORICAL FINDINGS
• Abomasal impaction often affects cattle on poor-quality pasture or that are fed chopped, low-quality forages with low dietary energy, especially in cold weather. Beef cattle are more prone due to management characteristics.
• The disorder may also arise in cattle eating from sand or gravel surfaces, or if excessive gravel from the feed storage area contaminates the feed.
• Animals may also develop the condition as a result of pica.

SIGNALMENT
• This disorder typically affects cattle and sometimes sheep.
• Suffolk sheep may be predisposed compared to other ovine breeds due to the higher prevalence of AED in this breed. Abomasal impaction affects juvenile to adult animals; AED affects sheep >2 years old.
• Abomasal impaction may be more prevalent in pregnant females.

PHYSICAL EXAMINATION FINDINGS
• Anorexia, depression, and decreased rumen motility are typical.
• Distension of the abomasum may be identified by visualization, palpation, and ballottement of the lower right flank.
• Affected animals eventually display weight loss, reduced fecal production, and dehydration.

GENETICS
A hereditary pattern has not been demonstrated for abomasal impaction or for AED.

CAUSES AND RISK FACTORS
• Physical blockage of the abomasum in cattle fed low-quality chopped forages, or consuming sand or gravel.
• As a result of "roll and toggle" sutures inadvertently placed in or near the pylorus.
• From conditions that damage the vagus nerve.
• Late pregnancy may predispose.
• Suffolk sheep with AED are predisposed.

DIAGNOSIS

DIFFERENTIAL DIAGNOSES
• Include other disorders that may cause signs of inappetance, scant fecal production, and abdominal distension including abomasal displacement, reticuloperitonitis, lymphoma, and vagal indigestion.

CBC/BIOCHEMISTRY/URINALYSIS
• CBC is usually normal.
• Hypochloremic, hypokalemic metabolic alkalosis may be present in chronic cases.
• Sheep with AED do not typically demonstrate the hypochloremic, hypokalemic metabolic alkalosis commonly found in cattle with outflow obstruction disorders.

OTHER LABORATORY TESTS
Elevated rumen chloride concentrations in sheep (>15 mEq/L) indicate reflux of abomasal contents into the rumen.

IMAGING
• Ultrasonography may be useful to determine distension of the abomasum and to assist detection of potential inciting causes such as traumatic reticuloperitonitis and lymphoma.
• Radiography may be useful to detect traumatic reticuloperitonitis or the presence of excessive sand or gravel within the gastrointestinal tract.

OTHER DIAGNOSTIC PROCEDURES
• Measurement of rumen chloride using fluid samples obtained by orogastric tube or rumenocentesis.
• Laparotomy.
• Abdominocentesis can identify elevated nucleated cell counts, elevated total protein concentration, and/or abnormal cellular morphology in animals with underlying causative disorders that are inflammatory or neoplastic in nature.

PATHOLOGIC FINDINGS
Distension of the abomasum with packing of roughage, gravel or sand is confirmed via laparotomy or necropsy. Concurrent traumatic reticuloperitonitis, lymphoma, and other predisposing causes may also be recognized by these techniques.

TREATMENT

THERAPEUTIC APPROACH
• Surgical therapy likely provides the greatest chance of resolution depending on the underlying cause.
• Medical therapy best accompanies surgical therapy and can consist of administration of cathartics and laxatives daily for 2 to 4 days.
• Correction of metabolic alkalosis may be indicated in severe or chronic disease.

SURGICAL CONSIDERATIONS AND TECHNIQUES
• Impaction may be resolved by abomasotomy with removal of roughage or foreign material. Surgical approaches that allow access to the abomasum include right paracostal, right paramedian and right paralumbar.

MEDICATIONS

DRUGS OF CHOICE
• Cathartics and laxative options include dioctyl sodium sulfosuccinate (50 mg/kg/day), magnesium sulfate (2.5 g/kg/day), mineral oil (8 mL/kg/day), and magnesium hydroxide (1 g/kg/day).

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ABOMASAL IMPACTION (CONTINUED)

• Metabolic alkalosis can be readily corrected via administration of intravenous sodium chloride preparations.

CONTRAINDICATIONS
• Gastrointestinal motility agents should be considered only after surgical or medical correction of impaction to reduce the risk of abomasal rupture.

PRECAUTIONS
• Lactated Ringer's solution should be used cautiously due to the possibility of inducing or exacerbating metabolic alkalosis.
• Abomasal motility agents (neostigmine, metoclopramide, erythromycin, etc.) should be used with great caution to avoid abomasal rupture.
• Appropriate milk and meat withdrawal times must be followed for all compounds administered to food-producing animals.

POSSIBLE INTERACTIONS
N/A

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
Grave prognosis. Death from dehydration, metabolic alkalosis or peritonitis if intervention does not occur.

POSSIBLE COMPLICATIONS
Abomasal rupture and peritonitis.

CLIENT EDUCATION
Feed cattle to meet energy requirements and avoid feeding chopped poor-quality forages with low energy, particularly in cold weather.

PATIENT CARE
Affected animals should be assessed for signs of pain and suffering (lethargy, inappetance, signs of abdominal pain), reduced fecal output, hydration status, and electrolyte balance.

PREVENTION
• Feed good-quality, long fiber-length forage with adequate energy supplementation.
• Avoid feeding on sand or gravel.

MISCELLANEOUS

ASSOCIATED CONDITIONS
Reticuloperitonitis, lymphoma, displaced abomasum.

AGE-RELATED FACTORS
More common in adult and pregnant animals, and mature Suffolk sheep.

ZOONOTIC POTENTIAL
N/A

PREGNANCY
Pregnancy predisposes to abomasal impaction as a result of increased energy requirements, appetite, and the possible effect of size and weight of the gravid uterus on abdominal organs.

BIOSECURITY
N/A

PRODUCTION MANAGEMENT
This disorder is largely preventable through appropriate dietary management.

SYNONYMS
Abomasal emptying defect in sheep

ABBREVIATION
AED = abomasal emptying defect

SEE ALSO
• Abomasal Emptying Defect in Sheep
• Displaced Abomasum
• Lymphosarcoma
• Traumatic Reticuloperitonitis

Suggested Reading

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ABOMASAL ULCERATION

BASICS

OVERVIEW
• Abomasal ulceration represents damage to the abomasal mucosa ranging from mucosal erosion to complete perforation. Often subclinical depending on the severity of disease.
• Abomasal ulceration can be classified as type I (nonperforating ulcers), type II (nonperforating with severe blood loss), type III (perforating with localized peritonitis), and type IV (perforating with diffuse peritonitis).
• When present, clinical signs reflect abdominal pain, blood loss, and peritonitis. The etiology is not clear, but may be associated with stress. There is no demonstrated association with specific bacterial pathogens.

INCIDENCE/PREVALENCE
• Low, likely below 1–2%. May be higher in some types of calf raising systems, and in cattle in the first month postpartum, particularly with concurrent disease.
• Rare occurrence in sheep and goats.

GEOGRAPHIC DISTRIBUTION
N/A

SYSTEMS AFFECTED
Digestive

PATHOPHYSIOLOGY
• Injuries to the protective mucosal layer of the abomasum allow gastric acid and pepsin to diffuse into the mucosa.
• Type I nonperforating ulcers have incomplete penetration, little local reaction, and minimal bleeding.
• Type II bleeding ulcers erode into a major blood vessel in the submucosa. There may be distension of the abomasum and reflux of abomasal contents into the rumen. Melena is typically observed.
• Type III ulcers completely perforate the wall with leakage of fluid and local peritonitis. Adhesions form to viscera, localizing the peritonitis.
• Type IV ulcers also completely perforate the wall; however, the subsequent fluid leakage is not contained by adhesions, resulting in generalized peritonitis.

HISTORICAL FINDINGS
Changes in feeding, such as transition from milk to solid feed in calves or change from a high roughage prepartum diet to high concentrate postpartum diets may be involved.

SIGNALMENT
• This disorder affects cattle, and rarely sheep and goats.
• There are no breed or gender predilections; however, dairy cattle may have a higher prevalence.
• Occurs in calves and adults.

PHYSICAL EXAMINATION FINDINGS
• Melena or occult blood is observed in feces.
• Possible distension of the abomasum detected by ballottement of the ventral right abdomen.
• Pale mucus membranes and tachycardia in cases with severe blood loss or septic shock.
• Anorexia, depression, pyrexia, and abdominal pain (bruxism).
• Peracute death common in adult cattle but not calves.

GENETICS
N/A

CAUSES AND RISK FACTORS
• This disorder has been associated with physical irritation from straw ingestion in veal calves and high grain diets in feedlot cattle.
• May be related to postpartum conditions in dairy cattle.
• Not definitively associated with bacteria such as *Clostridium*, *Salmonella* or *Helicobacter* sp. Recent gene pyrosequencing suggests limited involvement of bacteria in abomasal ulcers.
• No association with hairballs in veal calves.
• Risk factors might include sudden transition from milk diet to dry feed in calves, straw feeding in milk-fed calves, high concentrate diets, recent parturition, and peak milk production.

DIAGNOSIS

DIFFERENTIAL DIAGNOSES
• Lymphoma
• Left displaced abomasum
• Abomasal volvulus or torsion
• Intussusception
• Duodenal ulcers
• Hemorrhagic bowel syndrome

CBC/BIOCHEMISTRY/URINALYSIS
• Acute hemorrhagic anemia in cases of severe gastric hemorrhage, elevated fibrinogen, altered total protein concentration.
• Serum chemistry might reflect chronic inflammation in type III and IV ulceration reflected by high total protein concentrations and possibly neutrophilia on CBC.

OTHER LABORATORY TESTS
• Abdominocentesis may identify peritonitis, with increased leukocyte count, phagocytosed or free bacteria, and possibly feed particles in some cases.
• Testing for occult blood in feces may detect blood in the feces before melena is visible.

IMAGING
Ultrasonography may show free fluid and fibrin in the abdomen.

OTHER DIAGNOSTIC PROCEDURES
• Exploratory surgery.
• Peritoneal emphysema may be detected during per rectum examination.
• Positive withers pinch test may be evident (type III and IV).

PATHOLOGIC FINDINGS
• Ulcers are most commonly found along the greater curvature and usually in the fundic area. Ulcers can be a few millimeters to several centimeters in size. They are often filled with debris or clotted blood. Perforating ulcers are usually adhered to the omentum.
• Cattle with bleeding ulcers have signs of anemia with blood in the distal GI tract.
• Diffuse fibrinous peritonitis may be evident with defects in the serosal surface of the abomasum.

TREATMENT

THERAPEUTIC APPROACH
• Treatment is typically unrewarding but should target correction of management issues (dietary, stress related), correction of concurrent disease, and addressing clinical problems related to abomasal ulceration.
• Medical therapy may include provision of antacids to protect the abomasal mucosa, removal of high energy feedstuffs, and stall confinement.
• Blood transfusion may be beneficial if bleeding ceases or can be controlled, and is indicated if hematocrit declines to ≤14%.
• Broad-spectrum antibiotic therapy is indicated to prevent or address septic peritonitis.

SURGICAL CONSIDERATIONS AND TECHNIQUES
Surgical intervention for perforated ulcers might be attempted for valuable cattle.

MEDICATIONS

DRUGS OF CHOICE
• Magnesium oxide (500 g/400 kg body weight daily for 2–4 days), or a kaolin and pectin mixture (2–3 liters twice daily to mature cattle).
• Treatment options in calves include oral administration of antacids (25–50 mL q8h), ranitidine (50 mg/kg q8h) and omeprazole (4 mg/kg q24h).

CONTRAINDICATIONS
NSAIDs that interfere with the production of prostaglandin E series via the arachidonic acid cascade are not recommended, to avoid compromise of the protective coating of the abomasal mucosa.

PRECAUTIONS
• Avoid NSAIDs and corticosteroid drugs, particularly in susceptible populations.
• Appropriate milk and meat withdrawal times must be followed for all compounds administered to food-producing animals.

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ABOMASAL ULCERATION (CONTINUED)

POSSIBLE INTERACTIONS
N/A

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
Recovery for type I and type III ulcers is considered likely or possible; type II ulcers are fatal if severe hemorrhage occurs; type IV ulcers carry a guarded prognosis.

POSSIBLE COMPLICATIONS
• Septic peritonitis as a result of abomasal leakage or perforation.
• Hepatic lipidosis as a result of anorexia, particularly in cattle in early lactation.

CLIENT EDUCATION
Gradual introduction of dry feed to calves is preferred over abrupt exposure to dry feed during the milk-fed period.

PATIENT CARE
Serial assessments of CBC, anemia, and pain are used to determine recovery over time.

PREVENTION
• Avoid rapid change from liquid to dry feed in calves.
• Avoid excessive concentrate diets in feedlot or dairy cattle.
• Cull animals infected with bovine leukosis virus to eliminate lymphosarcoma as a cause of abomasal ulceration.

MISCELLANEOUS

ASSOCIATED CONDITIONS
Lymphoma

AGE-RELATED FACTORS
Affects all ages

ZOONOTIC POTENTIAL
N/A

PREGNANCY
N/A

BIOSECURITY
N/A

PRODUCTION MANAGEMENT
Avoid sudden dietary changes, rather, gradually introduce dry feed to calves.

SYNONYMS
N/A

ABBREVIATION
NSAIDs = nonsteroidal anti-inflammatory drugs

SEE ALSO
• Displaced Abomasum
• Hemorrhagic Bowel Syndrome
• Lymphosarcoma

Suggested Reading

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ABORTION: BACTERIAL

BASICS

OVERVIEW
Pregnancy loss between 42 days and term, caused by bacterial infection

INCIDENCE/PREVALENCE
• Abortion rates depend on the pathogen and immunologic status of the herd.
• Abortion rates range from <10% (serovar Hardjo of *Leptospira*, *listeriosis*) to 50–70% (serovar Pomona of *Leptospira*, *brucellosis*, epizootic bovine abortion).

GEOGRAPHIC DISTRIBUTION
• Worldwide
• Epizootic bovine abortion: California, Nevada, and Oregon

SYSTEMS AFFECTED
• Reproductive
• Other systems depending on cause

PATHOPHYSIOLOGY
• Infection occurs venereally, orally, via inhalation or across conjunctival mucosa.
• Ticks act as vectors for epizootic bovine abortion, *Anaplasma* spp., and *Coxiella burnetii.*
• Conceptus infection via hematogenous spread, ascending infection through the cervix, or descending infection from the abdomen through the oviducts.
• Bacteria may cause placentitis and fetal septicemia via the umbilical veins or by ingestion of amniotic fluid.
• Fetal death occurs secondary to placental insufficiency, fetal septicemia, lysis of the corpus luteum (CL) or failure of the diseased fetoplacental unit to produce progesterone to support pregnancy (after 200 days).
• A delay in fetal expulsion leads to autolysis or maceration.
• Dams may shed bacteria in urine, milk, feces, oronasal secretions, or uterine discharge. The abortus is an important source of infection.
• Gram-negative bacteria causing maternal systemic disease may also lead to abortion associated with luteolysis secondary to endotoxemia.

HISTORICAL FINDINGS
• Abortions
• Inappropriate vaccination
• Introduction of new animals

SIGNALMENT
• Ruminants, bovine (epizootic bovine abortion)
• Breeding age females
• Epizootic bovine abortion more common in naïve heifers

PHYSICAL EXAMINATION FINDINGS
• Physical examination is usually unremarkable.
• Illness and fever may develop secondary to retained fetal membranes (RFM) or a retained macerated fetus and metritis.
• *Campylobacter fetus venerealis*: Infertility, pregnancy loss between 15 and 80 days' gestation, or abortion at 4 to 6 months.
• *Leptospira* spp.: Last trimester abortion, stillbirth, weak calves, and infertility. Severe acute disease with hemolytic anemia, hemoglobinuria, and mastitis with serovar Pomona.
• *Brucella abortus*: Abortion after 5 months' gestation, weak or premature calves, RFM, metritis, infertility, carpal hygromas, regional lymphadenitis.
• *Listeria monocytogenes* and *L. ivanovii*: Abortion in the last trimester, meningoencephalitis, metritis, weight loss, maternal and neonatal septicemia.
• *Histophilus somni*: Pneumonia, arthritis, myocarditis, meningoencephalitis, sporadic abortions in the second half of gestation.
• *Salmonella enterica* serovar Dublin: Enteritis, dysentery, pneumonia, polyarthritis, pyrexia, abortion during the second half of gestation.
• *Trueperella pyogenes* (formerly *Arcanobacterium pyogenes*): Organ abscessation with varying signs, pyometra, abortion at any time.
• *Ureaplasma diversum*: Embryonic death, last trimester abortion, stillbirth, weak calves, neonatal pneumonia, endometritis, granular vulvovaginitis, salpingitis, endometritis.
• *C. burnetii*: late abortions, stillbirth, weak calves.
• *Mycobacterium bovis*: Organ granulomas with variable signs, late abortion, purulent to caseous vaginal discharge.
• *Anaplasma marginale*: Pale mucus membranes, icterus, fever, weakness, abortion during the acute phase of maternal disease.
• *A. phagocytophilum*: Fever, cough, nasal discharge, abortion during the acute phase of maternal disease.
• Epizootic bovine abortion: Last trimester abortion, premature births.
• *Chlamydophila abortus*: Abortions at 6 to 8 months' gestation, stillbirths, weak calves, RFM, mastitis, infertility.

GENETICS
N/A

CAUSES AND RISK FACTORS
Contagious bacteria
B. abortus, C. fetus (subsp. venerealis or fetus), *C. jejuni, Leptospira* spp., *L. monocytogenes, H. somni, Salmonella* spp., *Y. pseudotuberculosis, M. bovis, C. abortus*

Tick-borne infection
C. burnetii, A. marginale, A. phagocytophilum, epizootic bovine abortion

Mollicute infection
M. bovis, M. bovigenitalium, M. canadense, M. leachii, Ureaplasma diversum

Opportunistic bacteria
Bacillus spp., *E. coli, Pasteurella* spp., *Pseudomonas* spp., *Staphylococcus* spp., *Streptococcus* spp.

Mode of infection
• Inappropriate biosecurity measurements and vaccination schedules
• Nutritional, social or environmental stress
• Seasonal presence of ticks and vectors
• Exposure to wildlife and rodents

DIAGNOSIS

DIFFERENTIAL DIAGNOSES
• Vaginitis, metritis, endometritis
• Other causes of abortion

CBC/BIOCHEMISTRY/URINALYSIS
• Hemolytic anemia and hemoglobinuria (*L. interrogans* serovar Pomona)
• Anemia or leukopenia (*Anaplasma* spp.)

OTHER LABORATORY TESTS
• Bacterial culture from aborted tissues (lung, abomasal contents, placenta) and dam's milk (brucellosis).
• Dam and fetal serology.
• Immunohistochemistry and/or immunofluorescence on fetal tissues (leptospirosis, listeriosis, campylobacter, *C. burnetii*, *C. abortus*).
• PCR on aborted tissues (leptospirosis, *Brucella* spp., *Mycoplasma* spp., *Ureaplasma*, campylobacteriosis, *C. burnetii*, *Anaplasma* spp., *C. abortus*).
• Direct identification on Giemsa-stained blood smears (*Anaplasma* spp.).
• Pathognomonic microscopic lesions in the thymus (epizootic bovine abortion).

IMAGING
N/A

OTHER DIAGNOSTIC PROCEDURES
Fetal necropsy

PATHOLOGIC FINDINGS
• *C. fetus*: Fetal fibrinous pleuritis, pericarditis and peritonitis. Intercotyledonary placentitis, necrotic cotyledons.
• *Leptospira* spp.: Autolyzed and icteric fetus, cholestasis, intercotyledonary edema, diffuse cotyledonary necrosis.
• *B. abortus*: Fetus (autolysis, fibrinous pleuritis, pericarditis, peritonitis, pneumonia); placentitis.
• *Listeria* spp.: Fetus (autolysis, white foci of liver necrosis, abomasal erosions, pneumonia, polyarthritis); multifocal cotyledonary necrosis and intercotyledonary placentitis.
• *H. somni*: Autolytic fetus, necrosis of cotyledons.
• *Salmonella* spp.: Fetus (autolysis, emphysema, liver necrosis); placentitis.
• *T. pyogenes*: Fetus (autolysis), suppurative bronchopneumonia); cotyledonary and intercotyledonary placentitis.

ABORTION: BACTERIAL (CONTINUED)

• *Mycoplasma* spp.: Placentitis, fetal bronchopneumonia, and myocarditis.
• *U. diversum*: Fetus (fresh); thickening and fibrosis of the amnion and chorioallantois, mineralization, necrosis, hemorrhage.
• *C. burnetii*: thick leathery intercotyledonary spaces, multifocal mineralization, and pericotyledonary necrosis.
• Epizootic bovine abortion: Fetus (fresh, ascites, excessive fibrin, enlarged liver, spleen and lymph nodes, petechial hemorrhage on mucosal membranes and thymus, focal necrotizing lesions in lymphatic tissues, depletion of cortical thymocytes, and infiltration with macrophages in the thymus).
• *C. abortus*: Fetus (subcutaneous edema, ascites, thymic and subcuticular petechiae, serofibrinous pleuritis and peritonitis, nodular mottled liver, and enlarged lymph nodes); necrotizing placentitis with leathery, reddish, opaque intercotyledonary patches and multifocal cotyledonary necrosis.

TREATMENT

THERAPEUTIC APPROACH
• Treatment of systemic disease, RFM, metritis or hypocalcemia as needed.
• Vaccination can be curative (*Campylobacter* spp.).
• Supportive as needed.

SURGICAL CONSIDERATIONS AND TECHNIQUES
N/A

MEDICATIONS

DRUGS OF CHOICE
• *H. somnus*: β-lactams, florfenicol, tetracycline, and sulfonamides
• *Leptospira* spp: oxytetracycline, erythromycin, tiamulin, tylosin, tilmicosin, tulathromycin, ceftiofur
• *Listeria* spp.: tetracycline
• *U. diversum* and *Mycoplasma* spp.: tetracycline, tylosin
• *C. abortus*: tetracycline
• Epizootic bovine abortion: chlortetracycline

CONTRAINDICATIONS
N/A

PRECAUTIONS
Milk and meat withdrawal times should be followed.

POSSIBLE INTERACTIONS
N/A

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
Infected or exposed cows may develop natural immunity to some bacteria, with lower rates of abortion in subsequent breeding seasons.

POSSIBLE COMPLICATIONS
• Dystocia
• RFM
• Metritis
• Infertility

CLIENT EDUCATION
• Zoonotic potential. Wear protective gloves and clothes when handling aborted tissues and animals.
• Pregnant women, children, elderly and immunosuppressed people should not handle aborting animals or tissues.
• Keep aborted tissues refrigerated and call a veterinarian.
• Proper disposal of aborted tissues.

PATIENT CARE
• Monitor cow for RFM and metritis.
• Monitor herd for further abortions, stillbirths, or birth of weak calves.
• Change silage source (listeriosis).
• Adjust diet to eliminate ruminal acidosis and bacterial translocation.

PREVENTION
• Appropriate immunization program, nutrition, and environmental management.
• Test and quarantine new animals.
• Test and cull positive animals.
• Use virgin bulls and heifers for replacement.
• Use artificial insemination.
• Reduce contact with wildlife and rodents.
• Cure silage properly and avoid feeding spoiled material.
• Use fall calving season to prevent exposure of pregnant cows to ticks.
• Expose cows to ticks prior to breeding to stimulate natural immunity.

MISCELLANEOUS

ASSOCIATED CONDITIONS
N/A

AGE-RELATED FACTORS
N/A

ZOONOTIC POTENTIAL
Brucellosis, leptospirosis, listeriosis, *Coxiella burnetii, Chlamydophila abortus*, and salmonellosis

PREGNANCY
N/A

BIOSECURITY
• Brucellosis and tuberculosis are reportable diseases.
• See "Prevention."

PRODUCTION MANAGEMENT
See "Prevention."

SYNONYMS
N/A

ABBREVIATIONS
• CL = corpus luteum
• RFM = retained fetal membranes

SEE ALSO
• Abortion: Bovine
• Abortion: Viral, Fungal, and Nutritional
• Brucellosis
• Campylobacter
• Chlamydiosis
• Listeriosis
• Q Fever (Coxiellosis)
• Vaccination Programs: Beef Cattle
• Vaccination Programs: Dairy Cattle

Suggested Reading

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ABORTION: BOVINE

BASICS

OVERVIEW
Abortion in cattle is defined as loss of the fetus from 42 days to term. Prior to 42 days, pregnancy loss is considered embryonic mortality.

INCIDENCE/PREVALENCE
• Should be <5% on a herd basis (<1–2% ideal). • Abortion storms may occur in the case of specific infectious diseases. • In a 10-year study on bovine abortions and stillbirths, bacteria were determined to be the cause of 14.49% of the cases. The five bacteria most commonly associated with bovine abortion in the study were *Trueperella pyogenes*, *Bacillus* spp., *Listeria* spp., *E. coli*, and *L. interrogans*.

GEOGRAPHIC DISTRIBUTION
Worldwide

SYSTEMS AFFECTED
• Reproductive • Other systems depending on cause

PATHOPHYSIOLOGY
• Cattle rely primarily on the corpus luteum (CL) for production of progesterone for the first 180–200 days of gestation, followed by a shift to production of progesterone by the placenta in late gestation. • Abortion may be caused by infectious or noninfectious etiologies. ◦ Infectious causes of abortion include bacteria, viruses, fungi, and protozoa. ▪ Bacteria involved in abortion can be grouped into contagious and opportunistic. ▪ Ability of a pathogen to damage the conceptus is influenced by the dam, stage of fetal development, and virulence of the infectious agent. ▪ The time between fetal death and expulsion may be characteristic for a pathogen. ◦ Noninfectious causes of abortion include nutritional imbalance, exogenous drug administration, malnutrition, stress, environmental toxins, teratogenic compounds, hormone imbalances, and genetic abnormalities. • Abortion may result due to fetal death following: ◦ Invasion by microorganisms through hematogenous spread, ascending infection or presence of organism in the uterus prior to conception. ◦ Placental disease or insufficiency due to hematogenous (umbilical veins) or amniotic fluid (i.e., fungal infections) contamination. Some pathogens may cause severe placentitis leading to fetal hypoxia and death. ◦ Maternal compromise (mastitis, pneumonia, circulatory disorder, hypoxia, endotoxemia, etc.). ◦ Severe congenital malformations in some etiologies.

HISTORICAL FINDINGS
• Introduction of new animals • Return to receptivity after confirmation of pregnancy • Bloody or mucopurulent vaginal discharge in the pregnant cow • Protrusion/expulsion of the placenta or fetus • Premature development of the mammary gland and lactation
A complete history should be taken from each aborting case and include the following information:
• Age, breed, lactation/parity, clinical signs of the aborting female. • Reproductive history (i.e. breeding technique, breeding dates). • Individual case vs herd outbreak ◦ Number of animals ◦ Health problems/body condition ◦ Herd abortion history • Treatments and vaccination administered in the preceding 2 weeks. • Animal movement within the last month. • Previous abortions and any workup performed. • Feeding/nutritional management, quality of pasture (toxic plants). • Layout of the facilities (water sources, proximity to other operations, etc.). • Contact with wildlife or feral cats and dogs.

SIGNALMENT
Females of breeding age

PHYSICAL EXAMINATION FINDINGS
• Physical examination findings and evidence of abortion will depend on the stage of gestation and the cause of abortion. • Clinical examination of the cow(s) should be taken into consideration: ◦ Body condition score. ◦ Thorough physical examination including temperature, pulse, respiration, mucus membrane color, hydration status, presence of vaginal discharge, etc. ◦ Demeanor. ◦ Visual abnormalities.

GENETICS
N/A

CAUSES AND RISK FACTORS
• Causes of abortion can be classified as infectious (viral, bacterial, protozoal, fungal) or noninfectious (iatrogenic, maternal, fetal/placental, nutritional). • Noninfectious causes of abortion may be sporadic or affect several animals in the herd (nutritional deficiencies or administration of certain drugs). Infectious abortions are more likely to affect several animals within the herd simultaneously. • Risk factors include lack of biosecurity measures, presence of vectors or toxins, overcrowding, etc.

DIAGNOSIS

• The etiology of abortion is often difficult to determine and can be frustrating for owners and practitioners. Even when all required samples are submitted, the diagnostic rate is only 30%. • Abortion frequently results from an event that occurred weeks to months prior to the abortion event, making the diagnosis difficult in many cases. • Evaluation should include: ◦ Uterine cytology and culture (indicated in some cases) ◦ Serology
▪ Samples should be taken from the fetus (cardiac blood), from the aborting dam (paired samples) at abortion and 2 to 3 weeks later, and from at-risk pregnant females in the face of an outbreak. ▪ Samples from 10% of the herd will make serological assessment more meaningful. ◦ Bacteriology/Virology ▪ Samples should be taken from the fetus (stomach content, fetal fluids both thoracic and abdominal, kidney, liver, lung, spleen, and thymus), from the dam (vaginal discharge, uterine swab), and from the placenta. ▪ Samples should be collected using aseptic technique into sterile bags, refrigerated, and submitted to the diagnostic laboratory for further evaluation.
◦ Necropsy/Histopathology ▪ Digital photographs are helpful for documenting lesions. ▪ A complete set of tissues should be collected in every case. ▪ Fetal necropsy: Measurement of the crown–rump length and weight. External evaluation of the fetus for developmental abnormalities or lesions and evidence of autolysis, maceration, or mummification. Internal evaluation of the fetus. Either the entire fetus (optimal) or samples from the liver, brain, thymus, heart, spleen, kidney, stomach, lungs, skeletal muscle should be submitted fixed in formalin and fresh chilled to a diagnostic laboratory for further evaluation. Collection of ocular fluid (freeze) for nitrate/nitrite levels. ▪ Placental evaluation: External examination of the chorioallantois (including the cotyledons) and amnion. External examination for signs of placentitis (thickening, degradation, exudate). Examination for developmental abnormalities or lesions of the umbilical cord. Make impression smears of any lesions. Either the entire placenta or samples of both uterine horns and uterine body (cotyledons and intercotyledonary areas), along with any subjective abnormal areas, should be submitted fixed in formalin and fresh chilled for further evaluation.

DIFFERENTIAL DIAGNOSES

Infectious Causes of Abortion
• Viral causes of abortion: ◦ Bovine viral diarrhea virus (BVDV). ◦ Bovine herpesvirus 1 (BHV-1, infectious bovine rhinotracheitis; abortion storms affecting 25–60% in naïve pregnant cattle). ◦ Bovine herpesvirus 4 (BHV-4, often associated with other pathogens). ◦ Bluetongue virus (BTV). ◦ Epizootic hemorrhagic disease virus (EHDV). ◦ Rift Valley fever phlebovirus (RVF; mortality range 10–70% and abortion occurring at any gestational age reaching 80–90%). ◦ Akabane virus. ◦ Schmallenberg virus. • Bacterial causes of abortion: ◦ Brucellosis (*B. abortus*; in susceptible herds, abortion rates may be as high as 70%). ◦ Listeriosis (*L. monocytogenes*, *L. ivanovii*; abortions sporadic and rarely >15%). ◦ Campylobacteriosis (*C. fetus* subsp. *venerealis* (abortions <10% in infected herds), *C. fetus* subsp. *fetus*, *C. jejuni*). ◦ Leptospirosis (*L. interrogans* serovars Hardjo and Pomona most important causes of abortion, losses of

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ABORTION: BOVINE (CONTINUED)

up to 50% may be experienced in some outbreaks). ◦ Tuberculosis (*Mycobacterium bovis*, *M. caprae*). ◦ Epizootic bovine abortion. ◦ *Histophilus somni* (formerly *Haemophilus somnus*). ◦ *Salmonella* spp. ◦ *Trueperella pyogenes* (formerly *Arcanobacterium pyogenes*). ◦ *Mycoplasma bovis*, *Ureaplasma diversum.* ◦ *Chlamydophila abortus* (lesser extent *C. psittaci.* ◦ *Coxiella burnetii.* ◦ Anaplasmosis (*Anaplasma phagocytophilum* has been known to cause abortion storms during first exposure to infected ticks during late pregnancy). ◦ Other opportunistic bacteria: *Escherichia coli*, *Pasteurella* spp., *Pseudomonas* spp., *Staphylococcus* spp., *Streptococcus* spp., *Bacillus* spp. • Protozoal causes of abortion: ◦ *Neospora caninum* (abortion only clinical sign in cattle). ◦ *Tritrichomonas foetus.* • Fungal causes of abortion: ◦ *Aspergillus* spp. (*A. fumigatus*; reported most common cause of mycotic abortion in cattle). ◦ *Candida* spp. ◦ *Zygomycetes.*

Noninfectious Causes of Abortion

• Iatrogenic causes of abortion: Administration of $PGF2_{\alpha}$ or glucocorticoids. • Maternal causes of abortion: ◦ Stress ◦ Systemic disease ◦ Hormonal imbalances, adrenal gland dysfunction (particularly in fiber-producing animals) ◦ Uterine/cervical pathology ◦ Trauma (hemorrhage during pregnancy). • Environmental/nutritional causes of abortion: ◦ Nutritional/trace mineral deficiencies. ◦ Toxic plants: pine needle; broom snakeweed; locoweeds; hairy vetch (sporadic abortions); poison hemlock (cattle surviving acute poisoning generally abort); high plant estrogens; plants that accumulate nitrates. ◦ Aflatoxins° *Fusarium graminearum* (zearalenone) ◦ Organophosphates ◦ Nitrate fertilizer ◦ Vitamin A, selenium deficiency/toxicosis, iodine deficiency. • Fetal/placental causes of abortion: ◦ Twinning ◦ Umbilical cord torsion ◦ Congenital anomalies

CBC/BIOCHEMISTRY/URINALYSIS
May be indicated in some cases

OTHER LABORATORY TESTS
See "Diagnosis"

IMAGING
N/A

OTHER DIAGNOSTIC PROCEDURES
See "Diagnosis"

PATHOLOGIC FINDINGS
See "Diagnosis"

TREATMENT

THERAPEUTIC APPROCH
Depends on systemic involvement and other complications

SURGICAL CONSIDERATIONS
N/A

MEDICATIONS

DRUGS OF CHOICE
Depends on systemic involvement and other complications

CONTRAINDICATIONS
N/A

PRECAUTIONS
N/A

POSSIBLE INTERACTIONS
N/A

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
Dependent on the cause of abortion

POSSIBLE COMPLICATIONS
Dystocia, retained placenta, metritis, mastitis, infertility.

CLIENT EDUCATION
• Establish good preventative programs: biosecurity measures, immunization programs, adequate nutrition, parasite control. • Producers should have an intervention plan in case of abortion (isolation of affected females, collection of aborted materials for submission to veterinarian, examination of female and abortus). • Segregation of animals based on sex, age, and pregnancy status may help reduce transmission of infectious organism.

PATIENT CARE
• Dependent on the cause of abortion. • Isolation of affected cow(s) from remainder of the group. • Adequate nutrition, including balanced trace minerals, for the herd is important for elimination of some of the causes of abortion.

PREVENTION
• General prevention program for abortion (guidelines for biosecurity): ◦ Quarantine new animals (4 to 6 weeks) ◦ Nutrition ◦ Immunization program ◦ Keep feed, pasture, and water sources free from contamination ◦ Control rodent, bird, and feral animal populations. • Observe strict hygiene during parturition, keep good calving facilities. • Reduce stress due to poor nutrition, unsanitary environment, crowded conditions.

MISCELLANEOUS

ASSOCIATED CONDITIONS
N/A

AGE-RELATED FACTORS
N/A

ZOONOTIC POTENTIAL
• Brucellosis (*Brucella abortus*) • Campylobacteriosis (*C. jejuni*) • Chlamydial organisms • Q fever (*Coxiella burnetii*) • *Mycobacterium bovis* • Leptospirosis • Listeriosis

PREGNANCY
N/A

BIOSECURITY
See "Prevention"

PRODUCTION MANAGEMENT
N/A

SYNONYMS
N/A

ABBREVIATIONS
• CL = corpus luteum • $PGF2_{\alpha}$ = prostaglandin $F2_{\alpha}$

SEE ALSO
• Abortion: Bacterial • Abortion: Viral, Fungal, and Nutritional

Suggested Reading

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ABORTION: CAMELID

BASICS

OVERVIEW
Abortion is defined as loss of the fetus from 42 days to term. Prior to 42 days, pregnancy loss is considered embryonic mortality.

INCIDENCE/PREVALENCE
• Pregnancy loss ranges from 2 to 17%.
• Losses of up to 60% may be experienced in some leptospirosis outbreaks (i.e., leptospirosis, brucellosis).
• Loss of 40–50% have been reported in maiden females under some management systems.

GEOGRAPHIC DISTRIBUTION
Worldwide with some regional differences

SYSTEMS AFFECTED
• Reproductive
• Other systems depending on cause and complications

PATHOPHYSIOLOGY
• Camelids rely primarily on the corpus luteum (CL) for production of progesterone and maintenance of pregnancy for the entire gestation.
• Abortion is caused by any factor that causes directly or indirectly luteolysis:
  ◦ Treatment with prostaglandin $F2_{\alpha}$
  ◦ Inflammatory or infectious process
  ◦ Endotoxemia
  ◦ Stress such as heat stress or transport
  ◦ Debilitating diseases
• Abortion can be caused by compromised fetal viability or placental integrity:
  ◦ Placentitis
  ◦ Placental insufficiency (endometrial fibrosis, uterine capacity in maiden females, twinning)
  ◦ Direct insult to the fetus (mechanical or infectious)
  ◦ Fetal malformation/abnormal pregnancy
  ◦ Hormonal insufficiency or imbalance

HISTORICAL FINDINGS
• Presenting complaints may include:
  ◦ Return to receptivity after confirmation of pregnancy
  ◦ Bloody or mucopurulent vaginal discharge in the pregnant female
  ◦ Protrusion/expulsion of the placenta or fetus
  ◦ Premature development of the mammary gland and lactation
• A complete history of the aborting female(s) should include the following information:
  ◦ Age
  ◦ Reproductive history (breeding technique, pregnancy diagnosis, breeding dates)
  ◦ Treatments and vaccination administered in the preceding 2 weeks
  ◦ Animal movement within the last month
  ◦ Possibility of heat stress
  ◦ Feeding management
  ◦ Layout of the facilities (i.e., proximity to stagnant water, run-offs from dairy or swine operations)
  ◦ Contact with wildlife, feral cats

SIGNALMENT
Females of breeding age

PHYSICAL EXAMINATION FINDINGS
• Physical examination findings and evidence of abortion will depend on the stage of gestation and the cause of abortion.
• Anorexia or poor appetite.
• Increased rectal temperature, pulse and respiration are seen in some infectious conditions.
• Signs of colic are often reported prior to abortion particularly in late gestation.
• Emaciated or obese females.
• Mucopurulent or bloody vaginal discharge.

GENETICS
N/A

CAUSES AND RISK FACTORS
• Causes may be infectious or noninfectious (iatrogenic, fetal/placental abnormalities, nutritional, environmental, management).
• Abortion of noninfectious origin is usually sporadic. However, several animals in the herd may be affected in the case of nutritional deficiencies or administration of certain drugs.
• Infectious abortions are more likely to affect several animals within the herd simultaneously.

Infectious Causes of Abortion
• Viral causes:
  ◦ Bovine viral diarrhea virus (most common serotype affecting alpacas and llamas is noncytopathic BVDV-1b)
  ◦ Equine herpes virus-1 (potential)
  ◦ Blue tongue virus (potential)
• Bacterial causes:
  ◦ Brucellosis (*B. meletensis* and *B. abortus*)
  ◦ Listeriosis
  ◦ Chlamydiosis (*Chlamydophila abortus*)
  ◦ Leptospirosis
  ◦ Q fever (*Coxiella burnetii*, well-established cause of abortion in camels)
  ◦ Hemorrhagic disease (*Bacillus cereus*, camels)
• Protozoal causes of abortion:
  ◦ *Neospora caninum*
  ◦ Toxoplasmosis
  ◦ Trypanosomiasis (camels)

Noninfectious Causes of Abortion
• Iatrogenic causes of abortion:
  ◦ Administration of $PGF2_{\alpha}$
  ◦ Administration of corticosteroids (even topical) in the second half of pregnancy
  ◦ Some multivalent vaccines (8-way vaccines against *Clostridium* spp.)
  ◦ Ecbolic drugs
• Maternal causes of abortion:
  ◦ Hypoluteoidism (luteal insufficiency)
  ◦ Stress
  ◦ Systemic disease
  ◦ Hormonal imbalances, adrenal gland dysfunction (particularly in fiber-producing animals)
  ◦ Uterine/cervical pathology
  ◦ Trauma (hemorrhage during pregnancy)
• Nutritional causes of abortion:
  ◦ Nutritional/trace mineral deficiencies (selenium)
  ◦ Toxic plants (limited information available in camelids)
• Fetal/placental causes of abortion:
  ◦ Twinning
  ◦ Umbilical cord torsion
  ◦ Congenital anomalies
  ◦ Placental insufficiency

DIAGNOSIS

DIFFERENTIAL DIAGNOSES
Evaluation of the female should include:
• Transabdominal ultrasonography
• Vaginal speculum examination
• Uterine cytology, culture, and biopsy may be indicated in some cases.
• Serology
  ◦ Samples should be taken from the fetus (cardiac blood), from the aborting dam (paired samples) at abortion and 2 to 3 weeks later, and from at-risk pregnant females in the face of an outbreak.
• Bacteriology
  ◦ Samples should be taken from the fetus (stomach content, fetal fluids both thoracic and abdominal), from the dam (vaginal discharge, uterine swab), and from the placenta.
• Necropsy/Histopathology
  ◦ Fetal necropsy:
    ▪ Measurement of the crown–rump length.
    ▪ External evaluation of the fetus for developmental abnormalities or lesions.
    ▪ Either the entire fetus or samples from the liver, brain, spleen, kidney, stomach, and lungs should be submitted fixed in formalin and fresh chilled to a diagnostic laboratory for further evaluation.
  ◦ Placental evaluation:
    ▪ External examination of the chorionic surface for lack of villi (placental insufficiency) or signs of placentitis (thickening, degradation, exudate).
    ▪ The umbilical cord is examined for abnormalities or inflammatory lesions.
    ▪ The entire placenta or samples of both uterine horns and uterine body, along with any abnormal areas, should be submitted fixed in formalin and fresh chilled for further evaluation.
• Endocrinology
  ◦ In cases of habitual abortion, progesterone determination during pregnancy may be indicative of possible luteal insufficiency. Pregnant females with progesterone levels

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ABORTION: CAMELID (CONTINUED)

<2 ng/mL should be considered suspicious. However, some females may be able to carry a pregnancy to term even if progesterone levels are 1.5–2 ng/mL.

CBC/BIOCHEMISTRY/URINALYSIS
May be indicated depending on disease condition

OTHER LABORATORY TESTS
See "Diagnosis"

IMAGING
N/A

OTHER DIAGNOSTIC PROCEDURES
See "Diagnosis"

PATHOLOGIC FINDINGS
- Studies on pathological findings in camelid abortion are scarce.
- Placentitis is a common feature in bacterial and fungal abortions.
- Placental insufficiency is often suspected if large avillous areas are seen on the placenta.
- Fetal abnormalities are common.

TREATMENT

THERAPEUTIC APPROACH
- Dependent on the cause of abortion
- Abortion due to hypoluteoidism
  - Requires progesterone supplementation (injections of progesterone or hydroxyprogesterone caproate, norgestomet implant). Altrenogest is not active orally in camelids.
  - Hydroxyprogesterone caproate 250 mg, IM, every 3 weeks with treatment discontinued at 300 days to allow normal parturition.
  - Fetal viability should be monitored regularly if progesterone supplementation is implemented.
- Placental insufficiency
  - Early diagnosis and termination of twins.
  - Early diagnosis of uterine fibrosis (uterine biopsy) and sexual rest.

SURGICAL CONSIDERATIONS AND TECHNIQUES
N/A

MEDICATIONS

DRUGS OF CHOICE
N/A

CONTRAINDICATIONS
Appropriate milk and meat withdrawal times must be followed.

PRECAUTIONS
N/A

POSSIBLE INTERACTIONS
N/A

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
See specific disease/condition

POSSIBLE COMPLICATIONS
Dystocia, metritis, endometritis, retained placenta, infertility

CLIENT EDUCATION
- Pregnancy should be monitored in the first 60–90 days of gestation.
- Producers should have a plan if an abortion occurs (isolation of affected females, collection of aborted materials for submission to veterinarian, examination of female and abortus).
- Adequate nutrition, parasite control, and immunization programs for the herd are important for preventing some causes of abortion. Segregation of animals based on sex, age, and pregnancy status may help reduce transmission of infectious organism.
- Adequate nutrition, including balanced trace minerals, for the herd is important for elimination of some of the causes of abortion.

PATIENT CARE
- Depends on the cause of abortion and complications.
- Supportive care is important in debilitated animals.

PREVENTION
- Observe strict hygiene in breeding management.
- Set up guideline for biosecurity: quarantine new animals; during movement of animals between shows and ranch, visiting animals for breeding.
- Isolate any aborting female until diagnosis is established.
- Vaccination for leptospirosis (4 times a year in high-risk situations).

MISCELLANEOUS

ASSOCIATED CONDITIONS
- Infertility, dystocia, poor systemic health
- Fetal abnormalities

AGE-RELATED FACTORS
N/A

ZOONOTIC POTENTIAL
Possible for brucellosis (*B. meletensis, B. abortus*), *Chlamydophila abortus*, leptospirosis, Q fever

PREGNANCY
N/A

BIOSECURITY
See "Prevention"

PRODUCTION MANAGEMENT
N/A

SYNONYMS
N/A

ABBREVIATION
- CL = corpus luteum
- $PGF2_{\alpha}$ = prostaglandin $F2_{\alpha}$

SEE ALSO
- Abortion: Bacterial
- Abortion: Viral, Fungal, and Nutritional
- Camel Diseases
- Congenital Defects: Camelids
- Pregnancy Toxemia: Camelids

Suggested Reading

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ABORTION: FARMED CERVIDAE

BASICS

OVERVIEW
• Definition: Loss of an embryo or fetus
• Causes may be infectious, environmental, congenital or idiopathic

INCIDENCE/PREVALANCE
The incidence of pregnancy loss in farmed deer is relatively low.

GEOGRAPHIC DISTRIBUTION
N/A

SYSTEMS AFFECTED
• Reproductive • Others depending on cause

PATHOPHYSIOLOGY
• Pathophysiology depends on etiology.
• Infectious: organism spreads to gravid uterus and may cause death of the embryo/fetus or placentitis, which leads to placental separation; absorption or expulsion of the conceptus. • Environmental: as in other ruminant species, stress, malnutrition, or toxins can adversely affect oocyte or embryo quality or hormonal regulation in the female.
• Congenital: placental dysfunction or fetal abnormalities usually result, leading to premature expulsion of the fetus.

HISTORICAL FINDINGS
• If fetus is resorbed early in gestation, there may be no findings except missed due date from an expected or confirmed pregnancy. • If pregnancy had been confirmed early in the season, a female may come back into estrus.
• If abortion occurred early in the season, the female may give birth much later than the rest of the herd if re-mating occurred. • If abortion occurred late in the pregnancy, discovery of expulsed fetus. • Female may show signs of imminent parturition (i.e., seeking isolation, restlessness) before expected due date.

SIGNALMENT
• Commonly studied species include those within the genera *Cervus* (red deer, wapiti), *Dama* (fallow deer), *Axis* (chital deer), *Rangifer* (reindeer), *Odocoileus* (white-tailed deer, black-tailed deer, mule deer), *Alces* (moose), *Capreolus* (roe deer), *Ozotoceros* (Pampas deer), *Elaphurus* (Père David's deer), *Rucervus* (swamp deer, Barasingha). • Females of breeding age.

PHYSICAL EXAMINATION FINDINGS
• Likely no visible signs if fetal resorption occurred early in gestation. • If suspected, fetal/embryonic loss can be confirmed by transrectal palpation (in wapiti) or transrectal ultrasonography. • A female with an impending or unobserved late-term abortion may exhibit the following clinical signs:
◦ Bloody or purulent vaginal discharge
◦ Presence of expulsed fetus and/or placenta
◦ Premature udder development and dripping
◦ Nonspecific signs of illness, such as depression, dehydration, pyrexia, anorexia, or weight loss. • An impending late-term abortion may be suspected if the female begins showing signs of imminent parturition before expected due date. • Late-term abortions in cervids may go unobserved if the female eats the placenta and/or a predator consumes the dead fetus. Observation of alternative clinical signs or ultrasound may be able to confirm the loss of a fetus.
• Brucellosis-induced abortion in female reindeer is characterized by a retained placenta and metritis. • Bovine viral diarrhea virus and cervid herpesvirus 2 can both cause ulceration and/or pustules in oral or vulvar mucosa.

GENETICS
N/A

CAUSES AND RISK FACTORS
The major risk factors for abortion are poor herd management ranging from inadequate nutrition to stressful handling to poor biosecurity protocols.

Metabolic Causes
Febrile or severely stressed animals may abort. This may result from factors ranging from environmental (i.e., temperature) to iatrogenic (i.e., excessive handling).

Nutritional Causes
• Inadequate nutrition: Pregnant white-tailed deer females experience a 16% increase in fasting metabolic rates during gestation, 92% of which is in the 3rd trimester in early spring. Females with adequate body condition entering the winter months are more likely to have adequate fat reserves to fulfill gestational requirements in the event of prolonged winter. Females with inadequate fat reserves or that do not receive adequate nutrition in the spring may abort or give birth to nonviable or underweight calves. • Iodine deficiency: Abortion and stillbirth have been reported in cervid species due to iodine deficiency. The deficiency may be caused by insufficient dietary intake, or be secondary to excessive calcium in the diet, ingestion of toxic plants such as *Brassica* spp., gross bacterial contamination of the feed, continuous intake of feeds containing cyanogenetic glucosides (e.g., white clover), or ingestion of canola (rapeseed and canola meal). • Vitamin E and/or selenium deficiency: Congenital white muscle disease has been reported in several deer species and can be fatal to the neonate.

Bacterial Causes
• *Brucella abortus*: Biovars 1 and 4 have been recovered from wild elk in the Yellowstone area of the US. Natural infections have not been reported in axis deer, white-tailed deer, or mule deer, but experimental infections have been established in all three, suggesting potential for interspecies transmission.
• *Brucella suis* biovar 4: Brucellosis is the most common cause of abortion in reindeer and caribou and is endemic in some populations in the Arctic. Natural infection has not been reported in white-tailed deer or mule deer, but experimental infections have been established in both, suggesting possibility for interspecies transmission. • Leptospirosis: Studies in red deer showed reproductive effects, mostly reduced weaning rates, but no reports of abortions. Abortions following experimental inoculation with *L. pomona* have been demonstrated in white-tailed deer, demonstrating its abortigenic potential in cervid species. • Listeriosis: The septicemic form has been reported to cause placentitis and endometritis in farmed red deer, leading to abortion in late-term pregnancy and birth of weak, full-term young. It should be considered as a potential cause of abortion in any cervid species.

Fungal Causes
Incidence of fungal infections in cervid species is very low.

Parasitic Causes
• *Toxoplasma gondii*: Toxoplasmosis has been associated with encephalitis and placentitis in a full-term stillborn reindeer fetus. In red deer, seropositive females experience adverse effects on fetal development. • *Neospora caninum*: Seroprevalence in asymptomatic animals has also been reported in many cervid species; however, there have been reports associated with full-term stillborn Eld's deer and perinatal death in fallow deer and axis deer following suspected vertical transmission.

Viral Causes
• Bluetongue (BTV; orbivirus): Infection of cervid species generally leads to hemorrhagic disease and death. Under experimental inoculation, early embryonic absorption and fetal death were both reported in white-tailed deer. • Epizootic hemorrhagic disease (EHDV; orbivirus): Usually resulting in widespread hemorrhages, dehydration, and sudden death. EHDV has been reported to cause abortion and congenital lesions in cattle and, therefore, cannot be ruled out as a potential cause of fetal death in deer. • Bovine viral diarrhea virus (BVDV; pestivirus): BVDV has been isolated from many cervid species. A wide seroprevalence to BVDV has also been reported in surveys of wild ruminants in North America. Cervid species experimentally inoculated with cattle-derived BVDV experienced mild or no clinical disease. Experimental inoculation with a deer-derived BVDV strain in white-tailed deer resulted in fetal abortion and resorption and establishment of persistently infected (PI) carrier animals, similar to those effects observed in cattle, indicating a potential for natural infection. • Cervid herpesvirus 2 (CvHV-2; Varicellovirus): Endemic in Norway reindeer populations. Reports of vertical transmission and neonatal death in experimentally infected reindeer suggest an abortogenic potential, though this has yet to be reported in naturally occurring abortions.

ABORTION: FARMED CERVIDAE (CONTINUED)

Other Causes

• Other causes of abortion or stillbirth include toxicoses, traumatic injuries, congenital abnormalities, and administration of some drugs. • Locoweed causes abortions in sheep and cattle and may be a problem for cervidae as well. • Congenital abnormalities, fetal oversize, or abnormalities of presentation at time of parturition are rare, but may lead to dystocia and subsequent fetal death.
• Prostaglandins and steroids can induce fetal expulsion.

DIAGNOSIS

DIFFERENTIAL DIAGNOSES

• Vaginal discharge ◦ Normal term parturition ◦ Vaginitis ◦ Pyometra ◦ Metritis ◦ Uterine trauma or hemorrhage ◦ Uterine or vaginal neoplasia • Missed due date ◦ Infertility, either male or female (if pregnancy was not confirmed) ◦ Incorrect due date • Signs of impending parturition before due date ◦ Isolation: may indicate neurologic disease ◦ Abdominal straining: colic due to bloat (frothy vs. free gas) or other GI disease.

CBC/BIOCHEMISTRY/URINALYSIS

Little data is available on routine analyses but may be extrapolated from domesticated ruminants based on cause of abortion.

OTHER LABORATORY TESTS

Pregnancy Diagnosis and Monitoring

Maternal pregnancy-specific protein B (PSPB): Bovine pregnancy-specific protein B antibody cross reacts with caribou, red, fallow, and white-tailed deer PSPB and, therefore, commercial radioimmunoassay developed for cattle can be used for pregnancy diagnosis and monitoring in cervids.

IMAGING

• Transrectal ultrasonography: Used for early pregnancy detection (30–60 days) and fetal monitoring. • Transabdominal ultrasonography: Can be performed in smaller cervid species as early as 35 days and used to count the number of fetuses if performed within the first 2 trimesters.

OTHER DIAGNOSTIC PROCEDURES

Necropsy and sample collection of aborted fetus and placenta should occur as soon as possible to minimize secondary bacterial overgrowth. It may be difficult to obtain the placenta as the female usually ingests it. In herd outbreaks, it may be worthwhile to sacrifice a female for postmortem diagnosis if fetal tissues are inconclusive. Paired serology that shows rising antibody titers in the female may also be beneficial for antemortem diagnosis in some cases.

Bacterial Abortions

• Brucellosis: Four serological tests available – card test, standard plate agglutination test (SPT), complement-fixation (CF) test, and rivanol test. ◦ Positive serology requires that a confirmatory test be performed. This includes culture and isolation of *Brucella* from tissues, secretions, or excretions. ◦ Test results must be reported to state and federal animal officials.
• Leptospirosis: ◦ Paired serology: Microscopic agglutination test (MAT) and enzyme-linked immunosorbent assays (ELISAs); MAT will evaluate response to a selection of serovars.
◦ Immunohistochemistry (IHC) staining or polymerase chain reaction (PCR) on aborted tissue. • Listeriosis: ◦ In captive species, cerebrospinal fluid analysis can show a markedly elevated protein concentration and neutrophilic pleocytosis. These results combined with neurologic manifestations are almost pathognomonic for listeriosis, though no pre-mortem confirmatory test is currently available. ◦ Anti-Listeria IHC from the brainstem in deceased animals is quick and most effective for verifying the diagnosis.

Parasitic Abortions

• Toxoplasmosis or Neospora: ◦ Serology: Indirect or direct hemagglutination, indirect immunofluorescent antibody test, ELISA.
◦ Real-time PCR or IHC on aborted tissue.

Viral Abortions

• BTV or EHDV: Serology (competitive ELISA, agar gel immunodiffusion, microtiter virus neutralization (MVN)); IHC, PCR, virus isolation of aborted tissue. • BVDV: Paired serology (MVN; ELISA); virus isolation from blood or nasal secretions; IHC of aborted tissue. • CvHV-2: Serology (ELISA); PCR from tissues samples, nasal swabs, trigeminal ganglia samples.

PATHOLOGIC FINDINGS

Depends upon etiology.

Bacterial Abortions

• Brucellosis: Necrotizing placentitis characterized by a thickened placenta covered with a purulent exudate. • Leptospirosis: Gross lesions in aborted white-tailed deer fetuses (following experimental inoculation with *L. pomona*) included swollen, hemorrhagic, pulpy kidneys, liver, and lymph nodes. • Listeria: Evidence of fibrinopurulent to necrotizing placentitis on histopathology; fetuses may have either have no gross lesions or suppurative pneumonia and meningitis.

Parasitic Causes

Toxoplasmosis: Necrotizing placentitis and multifocal nonsuppurative encephalitis of the fetus with presence of tissue cysts histologically in sections of brain and tachyzoites in placenta and myocardium that stain positive with *T. gondii* antibodies.

Viral Causes

• BVDV: Abortion patterns are similar to those observed in cattle with fetal death occurring at variable stages of gestation, resulting in variable autolysis or mummification and a variety of dysplastic lesions. • CvHV-2: No specific gross pathologies have been described in aborted fetuses, but severe autolysis has been noted. Affected reindeer cows have demonstrated mild to moderate interstitial verminous pneumonia.

Fungal Causes

Granulomas (gross), fungal hyphae (histopathology) on affected organs.

TREATMENT

THERAPEUTIC APPROACH

N/A

SURGICAL CONSIDERATIONS AND TECHNIQUES

N/A

MEDICATIONS

DRUGS OF CHOICE

• Dependent upon etiology. • Broad-spectrum antibiotics: Indicated for specific bacterial diseases; long-acting antibiotics require less frequent dosing and will help to minimize stress of handling. • Anti-inflammatories: Flunixin meglumine (1.1–2.2 mg/kg IV) or meloxicam (0.5 mg/kg PO) can be administered once daily.

CONTRAINDICATIONS

N/A

PRECAUTIONS

N/A

POSSIBLE INTERACTIONS

N/A

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS

• Dependent upon underlying cause. • BTV and EHDV are most likely to cause rapid, acute death. • CvHV-2 may become latent and recrudesce during periods of stress.
• BVDV may lead to persistently infected animals that can shed the virus throughout their lifetime.

POSSIBLE COMPLICATIONS

• Decreased fertility, increased morbidity in females • Dystocia, uterine infection

CLIENT EDUCATION

See "Biosecurity" and "Production Management"

PATIENT CARE

• In cases of single abortion, important to watch entire herd to ensure no outbreak ensues. • Appropriate health care should be

(CONTINUED) ABORTION: FARMED CERVIDAE

provided based on the underlying etiology. • Supportive care (IV fluids, anti-inflammatories ± antibiotics) for systemic illness. • If suspected nutritional issue, have feed analysis performed to determine underlying deficiency or toxicity. Change feed or supplement nutrients as necessary. • If listeriosis or mycotoxins are suspected, make appropriate changes to silage feeding practices.

PREVENTION

Establish a good nutritional and preventive health program.

Brucellosis

• USDA-APHIS have published minimum program standards and procedures to eradicate and monitor brucellosis in farm or ranch-raised deer. Required cervid surveillance identification tests include: ◦ Interstate movement tests: All sexually intact animals 6 months of age or older must test negative for brucellosis within 30 days prior to interstate movement, with a 90-day post-movement test strongly recommended. ◦ Slaughter establishment tests: All test-eligible animals are blood-sampled at slaughter and tested for brucellosis. • Certified brucellosis-free cervid herds are exempt from testing requirements for interstate movement. • In infected herds, test and removal programs may be practical for intensively managed deer farms but not for game or wildlife parks. • No recommended vaccine is currently available for most cervid species. • *Brucella suis* biovar 4 (killed) vaccine has been shown to be useful for providing protection in reindeer.

Leptospirosis

Extra-label use of cattle vaccine available in the US.

Bluetongue and EHD

• Parasite control should be implemented to decrease the number of arthropod vectors (e.g., *Culicoides* spp. or gnats). • Autogenous vaccines are available through Newport Laboratories.

BVDV

Fencing of adequate height and double fencing are recommended in order to prevent direct contact and disease transmission between captive and wild cervids.

MISCELLANEOUS

ASSOCIATED CONDITIONS

Dystocia, infertility

AGE-RELATED FACTORS

N/A

ZOONOTIC POTENTIAL

Brucellosis, toxoplasmosis, leptospirosis, and listeriosis are all zoonotic diseases; appropriate precautions should be taken when handling fetal or placental tissues.

PREGNANCY

N/A

BIOSECURITY

• Quarantine newly acquired animals for 30 days minimum. • Pre-shipment testing for infectious diseases. • Double fences to minimize direct contact with wildlife species.

PRODUCTION MANAGEMENT

• Monitoring birthweight and growth of young animals and keeping records of previous reproductive performance of females will allow owners to make appropriate herd management decisions. • Habituating animals to gates and chutes makes handling during physical exams and other routine procedures less stressful on the animals. • Adequate nutritional management is crucial. Females should have a body condition score of 3–5 out of 9 to even be considered for breeding. Overweight females may be at increased risk for dystocia whereas underweight females are at an increased risk for abortion. • Close monitoring during gestation will be beneficial in the event of an abortion.

SYNONYMS

N/A

ABBREVIATIONS

• BTV = bluetongue virus • BVDV = bovine viral diarrhea virus • CF = complement fixation • CvHV = cervine herpesvirus • EHDV = epizootic hemorrhagic disease virus • ELISA = enzyme-linked immunosorbent assay • IHC = immunohistochemistry • MAT = microscopic agglutination test • PDG = pregnanediol-3alpha-glucuronide • PI = persistently infected • PSPB = pregnancy-specific protein B • SPT = standard plate agglutination test

SEE ALSO

• Abortion: Bacterial • Abortion: Small Ruminant • Abortion: Viral, Fungal, and Nutritional • Bovine Viral Diarrhea Virus • Brucellosis • Cervidae: Biosecurity • Cervidae: Breeding Soundness Examination • Cervidae Reproduction • Cervidae: Vaccination Programs

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A

ABORTION: SMALL RUMINANT

BASICS

OVERVIEW

• Fetal loss, fetal wastage: conceptus loss at any time during pregnancy. • Most commonly observed in the last 2 months of pregnancy.

INCIDENCE/PREVALENCE

• Flock pregnancy loss should be <5% (<2% ideal). • Abortion storms may occur in the case of specific infectious diseases.

GEOGRAPHIC DISTRIBUTION

• Worldwide • Some diseases processes may be regional (presence of vector)

SYSTEMS AFFECTED

• Reproductive • Other systems depending on etiology

PATHOPHYSIOLOGY

• Abortion results from: ◦ Fetal death from invasion by microorganisms or subsequent to placental disease (placentitis, vasculitis) and placental insufficiency. ◦ Fetal expulsion or premature parturition may result from maternal compromise. ◦ Reabsorption, maceration, mummification, and autolysis may be observed in some cases. ◦ Fetal abnormalities are often a feature of some viral infections. • Causes include a variety of infectious and noninfectious agents.
• Infectious causes of abortion are the most economically significant. • Common bacterial causes are *Chlamydophila abortus*, *Coxiella burnetii*, *Toxoplasma gondii*, and *Campylobacter* spp. • Common viral causes include caprine herpesvirus (CpHV-1), bluetongue virus, BDV, Cache Valley virus.

GENETICS

• Angora goat may be a habitual aborter.
• Some breeds of sheep are more susceptible to viral bluetongue (Merino, British breeds).

HISTORICAL FINDINGS

• Introduction of new animals • Repeat breeding or return to estrus • Premature udder development • Presence of expelled fetuses
• Premature/stillbirths • Increased congenital abnormalities in neonates or fetuses

SIGNALMENT

Nonspecific; females of breeding age that were exposed to the male or inseminated.

PHYSICAL EXAMINATION FINDINGS

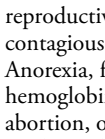
• Mucopurulent or hemorrhagic vaginal discharge containing fetal membranes in early pregnancy loss. • Signs of abortion: vaginal discharge vulvar edema, retained placenta.
• Systemic signs such as fever, anorexia may be present. • Other signs in flock: abnormal fetuses, sick or ill-thrift lambs and kids).
• Clinical signs in the aborting female vary depending on the cause. • Complications depend on cause (deterioration of health, retained placenta, metritis).

Clinical Signs

• Several infectious abortions are subclinical in the dam (Cache Valley, Border disease, caprine herpesvirus, Schmallenberg, Q fever).
• Bluetongue: Febrile, swollen tongue, ear or face, lameness, ulcerative lesions on month.
• Campylobacteriosis: Aborting goats may show diarrhea. • Chlamydiosis: Pneumonia, keratoconjunctivitis, epididymitis, and polyarthritis. Anorexia, fever, bloody vaginal discharge 2 to 3 days before abortion.
• Brucellosis (*B. melitensis*) in goats: weak kids and mastitis. Aborting goats may experience fever, depression, weight loss, mastitis, and lameness. • *B. ovis* in sheep: Rarely a cause of abortion but is responsible for poor reproductive performance and in the ram contagious epididymitis. • Leptospirosis: Anorexia, fever, marked jaundice, hemoglobinuria, anemia, neurological signs, abortion, occasionally may be fatal.
• Salmonellosis: Abortion, retained placenta, metritis, and various systemic signs (fever, depression, diarrhea). Mostly in overcrowded flocks. • Toxoplasmosis: Generally no clinical signs; immunocompromised females may present a neurologic form of the disease.
• Leptospirosis: Septicemia, fever, decreased appetite, reduced milk production, abortion, and meningoencephalitis. • Mycoplasmosis (goats): Mastitis, arthritis, keratoconjunctivitis, vulvovaginitis, and abortion in the last 3rd of pregnancy.

CAUSES AND RISK FACTORS

• Causes of abortion include: ◦ Viruses: bluetongue, BDV, CpHV-1, CVV, RVF, Akabane, Nairobi, Wesselsbron ◦ Bacteria: *Brucella* spp. (*B. ovis*, *B. melitensis*), *C. fetus* subsp. *fetus*, *C. jejuni* subsp. *Jejuni, Chlamydophila abortus, Francisella tularensis, Leptospira* spp., *Listeria monocytogenes* and *L. ivanovii, Salmonella abortus-ovis* ◦ Rickettsia: *Coxiella burnetii* ◦ Protozoa*: Sarcocystis*, *Neospora caninum*, *Toxoplasma gondii* ◦ Toxic plants: annual ryegrass, *Gutierrezia microcephala* (broomweed, snakeweed), locoweed, subterranean clover, *Veratrum californicum* (skunk cabbage) ◦ Mineral deficiencies: copper, iodine, selenium • Risk factors include: ◦ Lack of biosecurity measures ◦ Vector or reservoir population: bluetongue, CVV, Rift Valley fever, Akabane, Nairobi sheep disease, Wesselsbron disease virus, *Coxiella burnetii* (ticks), *Leptospira* spp., *Neospora, T. gondii* ◦ Overcrowding ◦ Pasture type



DIAGNOSIS

DIFFERENTIAL DIAGNOSES

Brucellosis

• Isolation: Best samples are vaginal discharges and milk, stomach contents. • Indirect diagnosis: Complement fixation, agglutination, and precipitation tests may help identify carrier animals.

Chlamydiosis (Enzootic Abortion)

• *C. abortus*, Gram-negative intracellular organism. • Abortion and other clinical signs in neonates. • Aborting females become immune. • Females infected after 100 days of pregnancy may not abort. • Diagnosis:
◦ Generalized placentitis, abortion in the last month of pregnancy, high incidence in newly infected flocks. ◦ Demonstrations of characteristic inclusion bodies on smear from cotyledons, vaginal discharge, fetal stomach content. ◦ Culture from vaginal discharge, placenta and fetal tissue, PCR. ◦ Serology: paired samples from dam and fetal serum. ◦ ELISA or indirect inclusion fluorescence antibody tests (IIFA).

Toxoplasmosis

• Goats more susceptible than sheep
• Diagnosis: ◦ Cotyledons are gray-white to yellow and present small 1–3 mm focal area of necrosis and calcification. Intercotyledonary areas are generally normal. Macroscopic lesions: 2–3 mm necrotic foci on cotyledons, intercotyledonary allantochorion are generally normal. Fetus may be mummified or decomposed. Chalky white necrotic brain lesions. ◦ Samples: placenta, fetal brain, fetal fluids, maternal blood, precolostral blood. ◦ Isolation from cotyledons, brain and fetal fluids, tissues (shipped packed in ice). ◦ Histopathology: fixed cotyledons, fetal brain. ◦ Serology: presence of antibodies in fetal fluids or precolostral serum is the preferred diagnostic technique and indicated transplacental infection.

Q Fever (Coxiella burnetii)

• Placentitis, placental necrosis, thickening of the intercotyledonary areas. Abortion and stillbirth. • Isolation: Placenta, vaginal discharges, fetal stomach content. • PCR techniques are available. • Demonstration of organism by Ziehl-Neelsen staining.
• Complement fixation: Need samples from several animals. • Fluorescent antibody test may be used to identify organism in frozen section of placenta.

Campylobacteriosis

• Campylobacter fetus subsp. fetus, C. jejuni subsp. jejuni, and Campylobacter lari.
• Gram-negative microaerophilic rods.
Symptoms
• Late term abortion, stillbirths and weak lambs, retained fetal membranes. • Placentitis, placental edema. • Fetal lesions: Hepatomegaly, hemorrhagic liver, necrotic foci of 1–3 cm, subcutaneous edema, sero-sanguinous fluid in abdominal and thoracic cavity, bronchopneumonia.
• Histopathology: Necrotic areas of the chorionic villi, arterioles and thrombosis of the hilus of the placentomes.

(CONTINUED) ABORTION: SMALL RUMINANT A

Isolation and Identification
• Samples: Placenta and vaginal discharges, frozen fetal stomach content (–20°C). • Transport medium required. • Isolation from placenta, vaginal discharge, fetal stomach content.

Salmonellosis
• *Salmonella abortus-ovis, S. barndenburg, S. typhimurium, S. dublin, S. montevideo, S. arizona, S. oranienburg.* • Early, mid or late term abortions, septic metritis, peritonitis in aborting females. Fetuses are often autolyzed. Lambs may be born weak or develop bronchopneumonia. Placentitis, placental edema. • Direct diagnosis: Culture from fetal tissues taken aseptically may be preserved at –20°C, placenta and uterine discharges. • Indirect diagnosis: Seroagglutination.

Listeriosis (Listeria monocytogenes)
• Gram-positive, non-acid-fast facultative microaerophilic organisms. *L. monocytogenes* affects sheep and goats; *L. ivanovii* affects sheep only. • Females may show fever, depression, and anorexia prior to abortion mid to late pregnancy. Stillbirths, birth of weak lambs, and retained placenta are common. Fetuses may be mummified. • Direct diagnosis: Placenta, fetal liver and spleen, fetal stomach content, vaginal discharge within 48 hours of abortion. Samples may be refrigerated if not cultured immediately. • Indirect diagnosis: Histopathology on placenta, fetal liver and spleen- microabscesses (white pinpoint spots), necrosis, and infiltration of macrophages and neutrophils. Gram stain reveals numerous Gram-positive rods.

Leptospirosis
• Sheep and goats are generally less susceptible to leptospirosis than other species. Goats are more susceptible than sheep. • Sheep: mostly *L. hardjo,* sometime *L. pomona*, *L. ballum* and *L. bratislava*, late term abortion, stillbirths, and ill-thrifty lambs. • Goat: *L. icterohaemrrhagiae*, *L. pomona*, *L. grippotyphosa.* • Affected flocks are mostly reared indoors. • Clinical signs in case of acute infection include fever and agalactia. Abortion occurs in late pregnancy. • Direct diagnosis: Fetal tissue, fetal fluids, and placenta. ◦ Isolation is difficult. ◦ Demonstration by dark-field microscopy, immunofluorescence, and silver stain. • Indirect diagnosis: ◦ Serology: macroscopic agglutination test.

Border Disease
• Goats are fairly resistant. • Viremic animals may show fever. Fetal death and resorption when infected in the first 2 months of pregnancy. Infection after 60 days results in fetal death, mummification or abortion, and fetal abnormalities. • Virus isolation (buffy coat) and antigen demonstration: Heparinized blood from dam or hairy shaker lambs, fetal tissue (thyroid, kidney, spleen, cerebellum, placenta), hairy shakers (thyroid, kidney, spleen, cerebellum, intestine, lymph nodes). • Histopathology: Cerebellum (white matter necrosis and gliosis), spinal cord. • Serology: Flood from dam and hairy shakers. • Clinical: Small cotyledon with focal necrosis, hairy shakers.

Bluetongue
• Viral isolation: Blood, semen, fetal brain and spleen. Unlikely cause of abortion in goats.

Akabane Disease
• Fetal malformation, positive antibody titer in liveborn and aborted fetuses.

Cache Valley Virus
• Congenital abnormalities. • Detection of antibodies in fetal fluids or precolostral serum.

Mycoplasmosis
• Mycoplasma abortions (*M. mycoides*, *M. agalactiae)* are significant in goats. • Diagnosis: Culture and serotyping of the isolate from milk, fetal fluids, and placenta.

Noninfectious Causes of Abortion
• Genetic (goat) may be a habitual aborter ◦ Angora goats with fine mohair ◦ Abortion at 100 days ◦ Adrenal dysfunction • Energy protein deficiency • Phenothiazine and levamisole in the last 2 months of pregnancy • Corticosteroids in late gestation • Prostaglandin $F2_{\alpha}$ or analogues (goats) • Plants that accumulate nitrates

CBC/BIOCHEMISTRY/URINALYSIS
May be indicated if aborting dams are clinically sick.

OTHER LABORATORY TESTS
• Sampling is critical for the proper diagnosis of abortion. • Placenta ◦ Ideal for the isolation of most abortion-causing agents. ◦ Ideal for identification using specific staining techniques on histological section or impression smears. ◦ For isolation need 5 or 6 cotyledons and section of intercotyledonary spaces both from healthy appearing and diseased areas. ◦ These tissues may be rinsed with sterile saline. ◦ For isolation need a transport medium ▪ Viruses ▪ *Campylobacter* FBP/glycerol ▪ Leptospirosis 100 mL extender with 1% BSA ◦ For histopathology ▪ 0.5 cm section of tissue in 10% formalin 1:10 ◦ For bacteriology ▪ Impression smears • Vaginal discharges ◦ Collect in sterile manner ◦ Vaginal/uterine swabs ◦ Use of specific transport medium is preferred if a specific microorganism is suspected. • Fetal tissues ◦ Tissue samples from all fetal organs (spleen, liver, kidneys, brain, lymph nodes, spinal cord) should be taken in an aseptic manner immediately after abortion or death. ◦ Handle in the same manner as for placenta. • Fetal fluids ◦ If fetus is not autolyzed ◦ Stomach content ◦ Peritoneal/thoracic fluids ◦ Blood from the cardiac cavity • Milk ◦ Samples of milk are taken from both glands using aseptic techniques (clean the mammary gland, disinfect teats, and eliminate the first 2 jets). • Blood ◦ For isolation: immediately after/during abortion. ◦ For serology: paired samples immediately after abortion and 2 to 3 weeks later ◦ In case of an outbreak, blood should be collected from aborting females as well as from lambs/kids before colostral intake.

IMAGING
N/A

PATHOLOGIC FINDINGS
Abortion Associated with Deformities
• Bluetongue: Hydranencephaly. • Akabane disease: Arthrogryposis (dystocia), hydranencephaly, and mummification. • Cache Valley virus: Arthrogryposis, brachygnathia, hydranencephaly, microencephaly, spinal cord hypoplasia, and mummification. • Border disease virus: Cerebellar hypoplasia, hydranencephaly, brachygnathia, arthrogryposis. Dark pigmentation of the fleece, hairy shaker. • Toxic plants: Lupine, skunk cabbage, locoweed, and Sudan grass. • Iodine, copper, manganese deficiency.

TREATMENT
THERAPEUTIC APPROACH
Depends on etiology and complications following abortion

SURGICAL CONSIDERATIONS AND TECHNIQUES
N/A

MEDICATIONS
DRUGS OF CHOICE
• Depends on etiology and complications following abortion. Daily tetracycline treatment of the flock may help with some of the abortion-causing diseases. • Broad-spectrum antibiotics and anti-inflammatory therapy. • *Campylobacter*: Penicillin or streptomycin or tetracycline in feed some strains are resistant. • Chlamydiosis: Tetracycline, Tylosin • Leptospirosis: Tetracycline • Toxoplasmosis: Decoquinate, monensin • Leptospirosis: Tetracycline • Mycoplasma: Tetracycline and tylosin

CONTRAINDICATIONS
Appropriate milk and meat withdrawal times must be followed.

PRECAUTIONS
N/A

POSSIBLE INTERACTIONS
N/A

ABORTION: SMALL RUMINANT (CONTINUED)

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
Dependent upon underlying cause.

POSSIBLE COMPLICATIONS
Dystocia, retained placenta, metritis, mastitis, male infertility (brucellosis, chlamydiosis), female infertility, poor lactation, neonatal losses.

CLIENT EDUCATION
• Establish good preventive program (biosecurity measures, vaccination, good nutritional programs). • Consider every case of abortion as a possible outbreak. • Act quickly and help collect appropriate samples to be examined by a veterinarian. • Zoonotic risk awareness.

PATIENT CARE
• Frequent monitoring of late term females in the entire herd or flock. • Correct nutritional deficiencies if suspected. • Avoid toxic plants and mycotoxins if suspected.

PREVENTION
• Assess disease risk and set up preventive measures. ◦ Toxoplasmosis: cat population. ◦ Leptospirosis: rodent population, humid hot environment, proximity to dairy and swine operation. ◦ Salmonellosis: source of infection: bird, cattle, wildlife, predisposing conditions: overcrowding, shipping, climatic changes ◦ Chlamydiosis ▪ Infection transmission: placenta, fetal fluids ▪ Pigeon/sparrows are reservoirs, ticks or insects may play a role ▪ Vaginal discharge in goat up to 2 weeks before abortion ▪ Reservoir: young maiden females ◦ Listeriosis ▪ Organisms grow in poorly fermented silage ▪ Can survive in soil and feces for extended period of time ◦ Bluetongue: *Culicoides* gnat (cattle may be a reservoir) ◦ Akabane virus diseases: gnats and mosquito population ◦ Cache Valley virus: mosquitoes • General prevention program for abortion ◦ Quarantine new animals (4 to 6 weeks) ◦ Nutrition ◦ Vaccination: *Chlamydia*, *Campylobacter* (2 months and last month of pregnancy) ◦ Feed chlortetracycline (200–400 mg/head/day), monensin (15 mg/head/day) during gestation ◦ Keep feed, pasture and water source free from contamination by run-off, particularly from cattle and hogs. ◦ Control rat, bird, cat population. ◦ Act quickly on any abortion and assume it is an outbreak. ◦ Submit complete samples. ◦ Separate pre-partum from postpartum females. ◦ Keep good lambing/kidding facilities. • Reduce stress due to poor nutrition, unsanitary environment, crowded conditions. • Vaccination ◦ Bluetongue: questionable ◦ Akabane virus: effective ◦ Cache valley: effective ◦ *Campylobacter*: helpful ◦ *Chlamydia*: helpful ◦ Q fever: autogenous vaccines in conjunction with chlortetracycline may help ◦ *B. ovis*: poor efficacy of killed vaccine ◦ *B. melitensis*: live attenuated good when permitted ◦ Salmonellosis: autogenous vaccine may be helpful ◦ Toxoplasmosis: may be helpful

MISCELLANEOUS

ASSOCIATED CONDITIONS
N/A

AGE-RELATED FACTORS
N/A

ZOONOTIC POTENTIAL
• *Campylobacter jejuni* (aborted fetus, stomach content, fetal membranes) • *C. abortus* (fetal membranes, vaginal discharges) • Q fever (influenza-like symptoms, myalgia, endocarditis) • Brucellosis (*B. melitensis*), Malta fever, undulating fever, joint pain • Leptospirosis • Toxoplasmosis (milk, fetal membranes) • Listeriosis: aborted fetuses

PREGNANCY
N/A

BIOSECURITY
See "Prevention"

PRODUCTION MANAGEMENT
• Cull infertile animal • Quarantine measures

SYNONYMS
N/A

ABBREVIATIONS
• ELISA = enzyme linked immunosorbent assay
• IIFA= indirect inclusion fluorescence antibody
• PCR= polymerase chain reaction
• BDV = Border disease virus
• CpHV-1 = caprine herpesvirus-1
• RVF = Rift Valley fever

SEE ALSO
• Abortion: Bacterial
• Abortion: Viral, Fungal, and Nutritional
• Akabane
• Arthrogryposis
• Bluetongue Virus
• Cache Valley Virus
• Campylobacter
• Chlamydiosis
• Congenital Defects: Small Ruminants
• Iodine Deficiency and Toxicity
• Leptospirosis
• Listeriosis
• Neosporosis
• Rift Valley Fever
• Schmallenberg Virus
• Selenium Toxicity
• Toxicology: Herd Outbreaks
• Vitamin E/Selenium Deficiency
• Wesselsbron Disease

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Client Education Handout available online

ABORTION: VIRAL, FUNGAL, AND NUTRITIONAL

BASICS

OVERVIEW
Pregnancy loss during the fetal stage, between 42 days and term, caused by viral or fungal infection of the fetus or placenta, or nutritional problems.

INCIDENCE/PREVALENCE
• BHV-1: sporadic abortions in vaccinated or previously exposed herds, up to 60% in naïve herds. • BVDV: up to 40% in susceptible herds. • Fungal: sporadic, <10% of the herd, can vary from 2 to 20% depending on environment and season. • Selenium deficiency: 4–5% of aborted fetuses in a Canadian study.

GEOGRAPHIC DISTRIBUTION
• Potentially worldwide • Seasonal occurrence of bluetongue virus abortions due to vector cycle (late summer and early autumn in temperate areas) • Seasonal occurrence of fungal abortion (winter and spring)
• Seleniferous areas for selenium toxicosis

SYSTEMS AFFECTED
• Reproductive • Other systems depending on etiology

PATHOPHYSIOLOGY
Viral Abortion
• Maternal infection occurs venereally, orally, via inhalation or across conjunctival mucosa.
• Vector transmission occurs with BTV (*Culicoides* spp.) and Cache Valley virus (multiple mosquitoes). • Viruses replicate in local lymphoid tissue and spread hematogenously to secondary organs.
• Viruses invade the placenta hematogenously from the dam's systemic circulation and cause fetal infection. • Fetal death occurs secondary to fetal infection and direct organ damage, or placental damage. • Fetal expulsion can occur before or immediately after death, with expulsion of a fresh or live fetus. More commonly, a delay in fetal expulsion leads to autolysis. • Fetal mummification may occur with some viral infections. • In the bovine, fetal infection between 100 and 150 days leads to congenital neurologic abnormalities (BVDV, BTV, BPV). • Fetal infection with noncytopathic BVDV strains between 1 and 4 months' gestation leads to birth of persistently infected immunotolerant calves.
• In small ruminants, infection with BTV and Border disease virus during the first 40 to 60 days results in fetal death and resorption. Later infection results in abortion, weak neonates, and congenital abnormalities (hydranencephaly, retinal dysplasia).
• Depending on the pathogen, dams may shed virus in ocular, oral, nasal, or vaginal secretions. • Latency is established with BHV-1 in the trigeminal nerve or the sacral spinal ganglia. Recrudescence and shedding may occur after stress.

Fungal Abortion
Fungi are thought to cause primary maternal respiratory or gastrointestinal disease and spread to placentomes hematogenously. Fetal infection occurs by extension of amniotic fluid infection.

Nutritional Abortion
• Selenium deficiency results from grazing plants growing in low-selenium soils. Development of congenital white muscle disease is thought to lead to fetal cardiac failure and death. • Ingestion of selenium-accumulating plants or contaminated water in areas with high-selenium soils or with environmental contamination, or iatrogenic overdose or oral or injectable selenium causes toxicosis. The toxic dose of selenium is uncertain, ranging from 2.2 to >20 mg/kg in the literature.
• Iodine deficiency has been associated with premature delivery, weak lambs and kids, and congenital goiter.

HISTORICAL FINDINGS
• A herd history of abortions or maternal and neonatal signs (described above)
• Inappropriate vaccination schedules
• Introduction of new animals to the herd
• Ataxic, blind or small calves in the herd (BVDV)

SIGNALMENT
• Ruminants of all breeds • Breeding age females

PHYSICAL EXAMINATION FINDINGS
• Maternal physical examination is usually unremarkable at the time of abortion.
• Maternal illness and fever may develop secondary to retained fetal membranes (RFM). • BHV-1: Abortion between 4 and 8 months' gestation, usually 2 weeks to 3 months after maternal clinical disease, respiratory disease, fever, conjunctivitis, nasal lesions, encephalomyelitis, neonatal disease, severe and painful pustular vaginitis. • BVDV: Variable depending on host and virus characteristics: infertility, embryonic death, abortion, mummification, small calves, persistently infected calves, congenital defects, fever, ocular and nasal discharge, oral ulcers, diarrhea, decreased milk production, epithelial erosions at the interdigital spaces, coronary bands, teats or vulva, hemorrhagic syndrome. • BTV: Maternal ulcers in mouth, tongue, muzzle and coronary bands, sloughing of hooves, abortion, stillbirth, embryonic death, fetal malformations. • BPV: first and second trimester abortions, congenital malformations, infertility.
• Fungal: abortion between 6 to 8 months' gestation, RFM. • Selenium toxicosis: Abortion, infertility, respiratory distress, lethargy, anorexia, diarrhea, fever, teeth grinding, death. In the chronic form: depression, weakness, anorexia, diarrhea, anemia, hair loss, hoof deformities, lameness.

GENETICS
N/A

CAUSES AND RISK FACTORS
Viral Causes
• Bovine herpes virus 1, 4, and 5, bovine viral diarrhea virus, bluetongue virus, bovine parvovirus. • The most common viral causes of abortion in small ruminants are Akabane, bluetongue, Border disease, Cache Valley virus, caprine herpesvirus-1 (goats).

Fungal Causes
Aspergillus spp., *Mucor* spp. *Absidia* spp., *Rhizopus* spp., *Mortierella* spp., *Candida* spp., *Pseudallescheria boydii*.

Nutritional Causes
Selenium toxicity or deficiency, toxic plants

Risk Factors
• Inappropriate biosecurity measurements and vaccination schedules. • Nutritional, social or environmental stress. • Seasonal presence of vectors. • Poorly ventilated moist environment, high animal density, animal confinement, and feeding moldy hay and feedstuffs (fungal). • Selenium-deficient soil.

DIAGNOSIS

DIFFERENTIAL DIAGNOSES
• Additional viral abortions: Bovine enterovirus, pseudorabies virus, parainfluenza virus 3, lumpy skin capripoxvirus, malignant catarrhal fever, bovine leucosis virus, foot and mouth disease virus; emerging or geographically restricted viruses (Kasba virus in Africa, Asia and Australia, Rift Valley fever in sub-Saharan Africa and Madagascar, Akabane virus in Asia, Australia, Middle East and Kenya, Schmallenberg virus in Germany and the Netherlands, Wesselsbron virus in Africa). • Bacterial abortion: Brucellosis, *Campylobacter* spp., *Leptospira* spp., *Listeria monocytogenes*, *Histophilus somni*, *Salmonella* spp., *Trueperella pyogenes*, *Mycobacterium bovis*, *Chlamydophila abortus*, *Coxiella burnetii*, epizootic bovine abortion, *Mycoplasma* spp., *Ureaplasma diversum*.
• Protozoal abortion: *Neospora caninum*, *Tritrichomonas foetus.* • Toxic abortion: Nitrate/nitrite poisoning, Ponderosa pine, broom snakeweed, sumpweed, moldy sweet clover, locoweed, poison hemlock, annual ryegrass infected with *Clavibacter rathayi*, snakeweed, stinkweed, turpentine weed, wild pea, sweet pea, subterranean clover, skunk cabbage mycotoxins, iatrogenic administration of teratogenic or luteolytic drugs. • Vaginal discharge: Vaginitis, metritis, endometritis, pyometra, hemorrhage.
• Ulcers: Foot and mouth disease, bovine papular stomatitis, vesicular stomatitis.
• Respiratory disease: Bovine respiratory disease complex.

ABORTION: VIRAL, FUNGAL, AND NUTRITIONAL (CONTINUED)

CBC/BIOCHEMISTRY/URINALYSIS
N/A

OTHER LABORATORY TESTS
• Virus isolation from aborted tissues (lung, liver, spleen, kidney, adrenal glands, placenta). • Virus isolation from maternal or neonatal buffy coat (BVDV). • Antigen detection (ELISA or IHC) or PCR on maternal or neonatal ear skin biopsies, serum, whole blood, milk, and nasal swabs (BVDV). • Dam, fetal or pre-colostral calf serology (BVDV, BTV, BPV). • Immunohistochemistry and/or immunofluorescence on fetal tissues (BHV-1, BVDV). • PCR on aborted tissues (BHV-1, BVDV) or whole blood (BVT). • Gross lesions in fetal skin and placenta (fungal). • Fungal culture from aborted tissues (placenta, abomasal fluid, lung). • Direct microscopic examination of skin or placental scrapings or histopathology (fungal).

IMAGING
N/A

OTHER DIAGNOSTIC PROCEDURES
• Fetal necropsy (see finding below) • Determination of selenium content in fetal and maternal liver

PATHOLOGIC FINDINGS
• BHV-1: Autolytic fetus, pinpoint white foci of necrosis in liver, pulmonary and renal hemorrhage and necrosis, diffuse placentitis, and yellow/brown amniotic fluid. • BVDV: Calves with congenital abnormalities (hydranencephaly, hydrocephalus, cerebellar hypoplasia, microphthalmia, retinal dysplasia, cataracts, thymic hypoplasia, hypotrichosis, brachygnathism, arthrogryposis, pulmonary or renal hypoplasia or dysplasia), necrotizing myocarditis, hepatic congestion, ascites. Autolyzed fetuses with rarely recognizable lesions, typically necrotizing inflammation with mononuclear infiltrations and lymphoid depletion, and no placental lesions. • BTV: Congenital abnormalities (hydranencephaly, hydrocephalus). • Fungal: Minimal fetal autolysis with numerous epidermal plaques, emaciation, placentitis with severe thickening of cotyledons and intercotyledonary areas with a leathery appearance; cotyledons may contain attached necrotic caruncular tissue. • Selenium deficiency: Fetal ascites, cardiac dilation and nodular liver, myocardial necrosis and mineralization, necrosis of skeletal muscle.

TREATMENT

THERAPEUTIC APPROACH
• Treatment of systemic disease, RFM, or metritis as needed. • Decrease environmental exposure to fungi by decreasing confinement and cow density, and improving ventilation and feed quality.

SURGICAL CONSIDERATIONS AND TECHNIQUES
N/A

MEDICATIONS

DRUG(S) OF CHOICE
N/A

CONTRAINDICATIONS
Use of modified live virus or attenuated vaccines against BVDV and BTV in pregnant cattle is associated with congenital malformations.

PRECAUTIONS
Latency may result after vaccination with modified live virus or attenuated vaccine against BHV-1.

POSSIBLE INTERACTIONS
N/A

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
• Infected or exposed cows may develop natural immunity to some viruses, with lower rates of abortion in subsequent breeding seasons. • 25% of cows with *M. wolfii*-associated abortions may develop post-abortion pneumonia with death within 72 h.

POSSIBLE COMPLICATIONS
• Dystocia • RFM • Metritis • Viral spread to herd mates • Infertility • Secondary bacterial infections

CLIENT EDUCATION
• Wear protective gloves and clothes when handling aborted tissues and animals. • Pregnant women, children, elderly and immunosuppressed people should not handle aborting animals or tissues. • Keep aborted tissues (fetus and placenta) refrigerated and call a veterinarian as soon as abortion was noticed for appropriate diagnostic tests. • Remove additional aborted tissues from the pasture to prevent disease transmission. • Work with a veterinarian to design an appropriate biosecurity and vaccination program. • Follow milk and meat withdrawal times if medications are administered.

PATIENT CARE
• The cow should be monitored to ensure the fetal membranes are expelled and metritis does not develop. • Supportive care as needed. • The rest of the herd should be monitored for further abortions, stillbirths or birth of weak or abnormal calves. • Tests dams of PI calves (BVDV). • Prevent feeding moldy hay or low-quality silage to pregnant animals (fungal).

PREVENTION
• Optimize the herd's health status by providing appropriate nutritional, stress and environmental management. • Test and quarantine new additions to the herd. • Buy replacement animals from BVDV-free herds. • Test and cull positive animals for eradication. • Where vaccines are available, vaccinate breeding stock. • Avoid modified live or attenuated virus vaccines in pregnant cattle.

MISCELLANEOUS

ASSOCIATED CONDITIONS
N/A

AGE-RELATED FACTORS
N/A

ZOONOTIC POTENTIAL
N/A

PREGNANCY
Infection in pregnant animals leads to abortion.

BIOSECURITY
See "Prevention"

PRODUCTION MANAGEMENT
Avoid nutritional, social and environmental stress by using proper management practices.

SYNONYMS
N/A

ABBREVIATIONS
• BHV-1 = bovine herpes virus 1 • BPV = bovine parvovirus • BTV = bluetongue virus • BVDV = bovine viral diarrhea virus • ELISA = enzyme-linked immunosorbent assay • IBR = infectious bovine rhinotracheitis • IHC = immunohistochemistry • PCR = polymerase chain reaction • PI = persistently infected • RFM = retained fetal membranes

SEE ALSO
• Abortion: Bacterial • Abortion: Farmed Cervidae • Abortion: Small Ruminants • Akabane • Bluetongue Virus • Bovine Viral Diarrhea Virus • Infectious Bovine Rhinotracheitis • Rift Valley Fever • Schmallenberg Virus • Selenium Toxicity • Vaccination Programs: Beef Cattle • Vaccination Programs: Dairy Cattle

Suggested Reading

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ACTINOBACILLOSIS: WOODEN TONGUE

BASICS

OVERVIEW
Actinobacillosis is caused by *Actinobacillus ligniersii* infection of the soft tissues, usually in the tongue.

INCIDENCE/PREVALENCE
Seen in up to 3% of cattle tongues at slaughter

GEOGRAPHIC DISTRIBUTION
Worldwide

SYSTEMS AFFECTED
- Digestive
- Musculoskeletal
- Integument
- Hemolymphatic

PATHOPHYSIOLOGY
- *Actinobacillus ligniersii* is a Gram-negative rod, which normally inhabits the alimentary tract of domestic ruminants, and is also found on plant awns.
- Mucosal lesions anywhere on the body, typically in the mouth, can be invaded by these bacteria, causing a localized lesion. Bacteria can also spread to different parts of the body via lymphatic drainage.
- A typical site of bacterial invasion is through small ulcers in the sulcus lingualis at the base of the tongue, leading to hard, painful, diffuse lesions of the tongue interfering with prehension of food, hence the synonym "wooden tongue." The bacteria initially cause an acute diffuse myositis of the muscles of the tongue, followed by development of granules and fibrosis.
- Lesions other than "wooden tongue" are usually uncommon; however, actinobacillosis should be included as a differential diagnosis for cutaneous diseases such granulomatous dermatitis and lymphadenitis.

HISTORICAL FINDINGS
Abrasive feeds and crowded conditions may lead to sporadic herd outbreaks or endemic disease. Lesions outside the oral cavity may be associated with previous wounds or needle punctures.

SIGNALMENT
- Mainly cattle and sheep, occasionally goats
- All ages

PHYSICAL EXAMINATION FINDINGS
Cattle generally present with hypersalivation and tongue may protrude from mouth. Weight loss can be seen in more chronic cases due to inability to prehend feed. Tongue may be diffusely firm and immovable and nodular swellings may be present on the tongue or lips or within the pharyngeal region. If present in atypical sites, signs will vary.

CAUSES AND RISK FACTORS
Caused by infection of soft tissues by *Actinobacillus ligniersii*. Abrasive feeds, crowded conditions, surgical lesions, and other sources of trauma.

DIAGNOSIS

DIFFERENTIAL DIAGNOSES
- Pharyngeal trauma and abscessation
- Retropharyngeal lymphadenitis or lymphosarcoma
- Oral foreign bodies
- Dental disease
- Parasitic or foreign body granuloma
- Exuberant granulation tissue
- Contagious ecthyma and caseous lymphadenitis in sheep and goats

CBC/BIOCHEMISTRY/URINALYSIS
Chronic inflammatory profile

OTHER LABORATORY TESTS
- Acute lesions: Culture and cytology of aspirates.
- Chronic: Biopsy/histopathology and culture of lesions.
- Microscopic examination of pus compressed between two glass slides shows "sulfur granule" or clublike rosette appearance with a central mass of Gram-negative rods.

PATHOLOGIC FINDINGS
Firm, pale, gritty, granulomatous abscesses with multifocal necrotic foci containing mononuclear cells, neutrophils, eosinophils, and plant fibers.

TREATMENT

THERAPEUTIC APPROACH
- Sodium iodide 20%: 70 mg/kg IV, once, repeat at least once at 7–10-day intervals.
- Organic iodides: 1 oz/450 kg PO daily following first IV administration above.
- Antibiotics may be used alone or in conjunction with iodide treatment for severe cases.
- Use of a soft feed will aid prehension during treatment.

SURGICAL CONSIDERATIONS AND TECHNIQUES
May need surgical debulking of lesions in severe cases; however, note that access to the surgical lesion and close proximity to major vessels limit feasibility in some cases.

MEDICATIONS

DRUGS OF CHOICE
- Sodium iodide IV
- Daily organic iodides PO
- Antibiotics: sulfonamides, tetracyclines, ampicillin, streptomycin

CONTRAINDICATIONS
- Use sodium iodide with caution in pregnant cattle; see "Precautions."
- Extra-label use of sulfonamides is restricted in lactating dairy cattle.
- Streptomycin is not labeled for use in food-producing species in some countries.
- Appropriate milk and meat withdrawal times must be followed for all compounds administered to food-producing animals. Consult the Food Animal Residue Avoidance Database (www.farad.org) for current withdrawal times. As of August 2015, suggested Milk and Meat withdrawal times for cattle treated with NaI as above were 96 hours and 1 day, respectively.

PRECAUTIONS
Anecdotal reports of association with abortion in cattle at high doses of sodium iodide.

POSSIBLE INTERACTIONS
N/A

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
- Good prognosis if only the tongue is involved and lesions are acute. Expect dramatic response to therapy in this case.
- Fair to guarded prognosis if atypical sites are involved or lesions are chronic.

POSSIBLE COMPLICATIONS
Anecdotal reports of association with abortion in cattle at high doses of sodium iodide. If signs of iodism seen (dandruff, excessive lacrimation, inappetence, coughing, diarrhea), halt therapy until signs disappear.

CLIENT EDUCATION
Make aware of risk factors.

PREVENTION
Reduce access to abrasive feed and pastures with hard penetrating plant awns or thistles.

MISCELLANEOUS

ZOONOTIC POTENTIAL
Bite wounds from ruminants can contain *Actinobacillus ligniersii*, but rarely result in actinobacillosis.

PREGNANCY
Anecdotal reports of association with abortion in cattle at high doses of sodium iodide.

SYNONYMS
- Wooden tongue
- Woody tongue

ABBREVIATIONS
- IV = intravenous
- NaI = sodium iodide
- PO = per os

ACTINOBACILLOSIS: WOODEN TONGUE (CONTINUED)

SEE ALSO

- Caseous Lymphadenitis
- Oral Disorders
- Orf (Contagious Ecthyma)
- Tongue Trauma

Suggested Reading

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ACTINOMYCOSIS: LUMPY JAW

BASICS

OVERVIEW
Common, sporadic, chronic granulomatous osteomyelitis of cattle caused by non-spore-forming, filamentous, Gram-positive, anaerobic bacterium *Actinomyces bovis.*

INCIDENCE/PREVALENCE
Common, sporadic

GEOGRAPHIC DISTRIBUTION
Worldwide

SYSTEMS AFFECTED
• Musculoskeletal, typically confined to the mandible and maxilla • Potential for hematogenous spread to other organs

PATHOPHYSIOLOGY
• *Actinomyces bovis* is part of the normal flora of the bovine oral cavity. It has low virulence and only causes disease when mucosal barriers are compromised. • Trauma to the oral mucosa from rough feed or foreign objects permits entrance of *Actinomyces* into buccal tissues. The organism may also enter through the dental alveolus. • The organism grows best in anaerobic conditions associated with devitalized tissues where there is a lack of phagocyte delivery. • Disease is typically of the mandible or maxilla, beginning as a painless, hard, immobile swelling. It is possible for teeth to become involved. In the following weeks to months, abscess/granuloma formation with necrotic foci may occur. As the disease progresses lesions may become painful. • Discharge of viscous, sticky, "honey- or whey-like," odorless, yellowish pus from openings may occur. The pus contains sand-like, firm, yellowish granules. The granule contains microcolonies of *Actinomyces* in an eosinophilic, amorphous matrix made of calcium phosphate/antigen-antibody complexes. • Occasional involvement of soft tissues, especially of esophageal groove with spread to lower esophagus, anterior wall of reticulum, may occur. Trauma to the digestive tract could allow for disease in regions distal to the oral cavity. • Hematogenous spread to other organs is rare. • Local lymph nodes are usually not involved. • Severe swelling, particularly of the maxilla, may cause dyspnea.

SIGNALMENT
• Primarily cattle; potentially all ruminant species • Often young with erupting teeth, but may affect any age • No sex or breed predilection

PHYSICAL EXAMINATION FINDINGS
• Hard, immovable swelling of the mandible or maxilla • Draining, fistulous tracts with a yellow, odorless pus • Missing or malaligned teeth with difficulty masticating • Weight loss, intermittent diarrhea, chronic bloat • Dyspnea • Rarely, partial tracheal obstruction, orchitis, brain/lung abscesses

CAUSES AND RISK FACTORS
• Eruption of teeth in young cattle • Rough feeds containing awns, foreign objects • Procedures causing oral lacerations

DIAGNOSIS

DIFFERENTIAL DIAGNOSES
• Tooth root abscess • Osteomyelitis from cause other than *Actinomyces bovis* • Cheek abscess from cause other than *Actinomyces bovis* (movable, located in soft tissue) • Impacted feed/foreign body between cheek and teeth (soft, movable) • Lymphosarcoma/fibrosarcoma (soft)

OTHER LABORATORY TESTS
• Culture: Deep samples should be taken and placed in anaerobic transport media. • Microscopic examination of purulent debris: mix sample with saline and crush granules between slides. Gram stain will reveal Gram-positive organisms that may be branching, filamentous, coccoid or diphtheroid.

IMAGING
Osteomyelitis of the affected area; teeth may be involved. Important to differentiate from tooth root abscess.

PATHOLOGIC FINDINGS
Osteomyelitis with organisms present

TREATMENT

Difficult to treat. Antibiotics and sodium iodide have been historically used. Surgical debridement and drainage may be necessary.

MEDICATIONS

DRUGS OF CHOICE
• Penicillin, streptomycin, sulfonamides, erythromycin, and isoniazid have been used. • Sodium iodide: 70 mg/kg IV every 3 to 5 days until signs of iodism occur (scaling skin, lacrimation, cough, anorexia).

CONTRAINDICATIONS
• Iodine therapy may cause abortion. • Appropriate meat and milk withdrawal times must be followed for all compounds administered to food-producing animals. • Many historically used antibiotics are no longer approved in the United States.

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
• If untreated, disease will progress until the animal is no longer able to appropriately prehend food. • Difficult to treat. Early, aggressive treatment provides the best outcome.

POSSIBLE COMPLICATIONS
• Some animals become distressed with iodine infusion (restlessness, dyspnea, tachycardia). • Subcutaneous iodine causes severe irritation.

PATIENT CARE
• Lesions will slowly remodel after successful treatment. The affected areas may never return to normal. • Recrudescence is possible, even after prolonged periods of time.

PREVENTION
• Avoid feeds or procedures that could cause oral lacerations. • Monitor young cattle for swelling of mandible, especially following tooth eruptions. • Isolate cattle with discharging lesions. • There is no vaccine.

MISCELLANEOUS

AGE-RELATED FACTORS
Young cattle with erupting teeth

PREGNANCY
Iodine treatment during pregnancy may cause abortion.

ABBREVIATION
IV = intravenous

SEE ALSO
• Actinobacillosis: Wooden Tongue • Caseous Lymphadenitis • Oral Disorders • Orf (Contagious Ecthyma)

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A

ACUPUNCTURE

BASICS

OVERVIEW

• Acupuncture is one of the four branches of traditional Chinese veterinary medicine (TCVM) and has been practiced for over 2,000 years.
• The twelve regular channels and the eight extraordinary channels connect acupoints all over the body. These pathways, known as meridians, relate to different organ systems and conduct the acupuncture signal and life energy, known as Qi.
• Techniques for stimulating acupoints include dry needling, aquapuncture, electroacupuncture, hemoacupuncture, and moxibustion.
• In 1997 the NIH (USA) released a consensus statement stating that acupuncture was proven to be effective for treatment of musculoskeletal pain, some gastrointestinal diseases, pulmonary disease, immunomodulation, and reproductive disorders in humans.
• Acupuncture is appropriate for use in organic production systems which are otherwise limited in their choice of treatment options.
• The practice of veterinary acupuncture is restricted to licensed veterinarians, or under the supervision of a licensed veterinarian, in most states and provinces in the US and Canada.

DIAGNOSIS

OTHER DIAGNOSTIC PROCEDURES

A full Western veterinary physical examination should be performed prior to initiating treatment. Both the Western and TCVM examination results are taken together to formulate a diagnosis. Additional TCVM procedures which are not part of the traditional Western examination can include:
• Determination of the patient's temperament type and element association.
• Inspection of the tongue: Tongue color, coating, and degree of moisture.
• Pulse diagnosis: Relative strength of the pulse at different points. In cattle, the pulse is taken at the coccygeal artery; in small ruminants the pulse is taken from the right and left carotid arteries, allowing for comparison between the two.
• Palpation along the meridians to find areas of sensitivity.

TREATMENT

THERAPEUTIC APPROACH

• Dry needle (DN): Insertion of sterile needles into an acupoint.
• Electroacupuncture (EA): Electrical stimulation of an acupoint; electrical leads are connected to the handles of the metal needles and either low or high frequency current sent into the points and along the associated channels. Provides greater, longer-lasting stimulation of a point or channel than dry needling alone.
• Aquapuncture (AA): Injection of sterile saline, vitamin B12, or the patient's own blood into an acupoint. Provides longer-lasting stimulation of a given point than dry needling alone.
• Hemoacupuncture (HA): Release of blood from an acupoint; the point is pricked with a sterile hypodermic needle and allowed to bleed. Used to release excess heat or relieve stagnation.
• Acupressure: Applying pressure to an acupoint or along a meridian without insertion of a needle or other method of stimulation.
• Moxibustion (moxa): Sticks or cones of dried mugwort are burned and held near an acupoint or touched to a dry needle to stimulate the point. Used to break up stagnation or warm the point.

Protocols

Treatment protocols should take into account the Western diagnosis, TCVM pattern diagnosis, patient's temperament, and the owner's primary concern for each individual case. As such there is no true "cookbook" protocol for any particular condition. That said, the most frequently used points for common Western medical diagnoses are given below along with suggested techniques. For explanation of the channel names and point locations, see "Suggested Reading." It is strongly recommended that one completes a formal training program prior to performing any acupuncture treatment.
Please note that for emergencies (e.g., dystocia) acupuncture should not be used as the sole treatment, and the practitioner must adapt his or her approach as the case develops.
• Anestrus: Bai-hui, Yan-chi, GV-1, GV-2, CV-1, BL-23, BL-26, Shen-shu, Shen-peng and Shen-jiao; can use DN, EA, or moxa.
• Retained fetal membranes: Bai-hui, Ba-jiao, GV-1, BL-31/32/34; EAP recommended daily until resolution.
• Infertility/subfertility: Bai-hui, Shen-peng/shu/jiao, Yan-chi, CV-1; DN, EA, or AA.
• Dystocia: SP-6, BL-60/67 to promote normal labor, add GB-21 for dystocia; DN or AA.
• Resuscitation, esp. of neonates: GV-26; DN.
• Heat stress: Er-jian, Wei-jian, Tai-tang (GB-1), GV-14; DN for GV-14, HA for others; EA between GV-14 and Bai-hui.
• Hemorrhage: Duan-xue (GV-6), Tian-ting; DN or AA; can be administered 2 hours prior to surgical procedures (e.g. castration, enucleation) for prevention.
• Diarrhea: GV-1, ST-36, BL-20/21/25; DN or AA.
• Anorexia: Shan-gen (use hypodermic needle), Mi-jiao-gan, ST-36; DN.
• Calving paralysis: Bai-hui, GB-29/30, BL-54, GV-3 and KID-1; EA or AA.

MEDICATIONS

DRUGS OF CHOICE

Chinese herbal medications are sometimes given as a complement to acupuncture. Because of metabolism in the rumen and first compartment, oral doses should be 2 to 3 times greater than those listed for horses. Alternatively, the equine dose can be administered per rectum as a slurry.

PRECAUTIONS

These medications have not been evaluated by the FDA and there are no established withdrawal times for any food-producing species.

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS

• Improvement should be seen by the third treatment. Chronic or severe conditions will often require multiple treatments over an extended period (weeks to months).
• Although acupuncture can be used symptomatically, being able to make a Chinese medical diagnosis allows the practitioner to target the treatment towards the underlying pathologic process and will enhance results.
• Prognosis depends heavily on the severity and chronicity of the disease process, the patient's temperament and demeanor, and the patient's tolerance of acupuncture treatment.

(CONTINUED) ACUPUNCTURE A

POSSIBLE COMPLICATIONS

• Acupuncture should be used with caution during pregnancy. Avoid points around the abdomen, mid to lower back, and hips. Any points which move blood or Qi should be avoided, as well as potent points such as ST-36, LI-4, BL-67, and SP-6. The CV channel (along ventral midline) should not be stimulated during gestation.
• Very old or debilitated patients (See "Age-Related Factors").
• Although extremely rare, needles can break off and become lodged in the muscle. To prevent breakage, use appropriately sized needles for each point, avoid using hypodermic needles except as a guide or for HA, and do not insert the needle completely up to the handle.
• Concurrent use of sedatives or dexamethasone will blunt the response to acupuncture and should be used only when necessary.

PATIENT CARE

For complicated or severe conditions, multiple treatments over an extended period of time should be expected. As the primary condition improves, other symptoms and disease patterns may become more obvious and treatment protocols adjusted accordingly.

MISCELLANEOUS

AGE-RELATED FACTORS

• Neonates and juvenile patients respond readily to acupuncture and may not need extensive treatment.
• Very old patients may not have enough energy or Qi left to tolerate a full acupuncture session. Since acupuncture moves energy around the body, it may deplete what little is left for these patients, leading to death. Short treatments with very few needles should be used.

PREGNANCY

• Acupuncture should be used with caution during pregnancy (see "Possible Complications"). Points which are both safe to use and can promote a healthy pregnancy include Bai-hui, Shen-peng/shu/jiao, and BL-20/21/22/23/24/25/26.

BIOSECURITY

• Acupuncture needles are single use only and should be disposed of properly in a sharps container.
• Use of blood for AA should only be performed with the patient's own blood, injected immediately after being drawn from the jugular or coccygeal vein.

ABBREVIATIONS

• AA – aquapuncture
• DN – dry needle technique
• EA – electroacupuncture
• HA – hemoacupuncture
• Moxa – moxibustion
• TCVM – Traditional Chinese Veterinary Medicine
See "Suggested Reading" for full explanation of acupuncture point abbreviations

SEE ALSO

Alternative Medicine (see www.fiveminutevet.com/ruminant)

Suggested Reading

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Internet Resources

• American Academy of Veterinary Acupuncture: http://www.aava.org/
• American Association of Traditional Chinese Veterinary medicine: http://www.aatcvm.org/index.php/en/
• Association of British Veterinary Acupuncturists (training available): http://www.abva.co.uk/
• Association of Veterinary Acupuncturists of Canada: http://www.avacanada.org/
• Australian College of Veterinary Acupuncture (training available): http://vetacupcollege.com.au/blog/
• Australian Veterinary Acupuncture Group: http://acuvet.ava.com.au/
• Chi Institute (training available): http://www.tcvm.com/
• International Veterinary Acupuncture Society (training available): https://www.ivas.org/
• OneHealth SIM (training available): https://www.onehealthsim.org/
• World Association of Traditional Chinese Veterinary Medicine: http://www.watcvm.org/

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A

Acute Renal Failure

BASICS

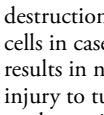
OVERVIEW
Acute renal failure (ARF) is common in ruminants experiencing hemodynamic changes or exposed to nephrotoxins.

PATHOPHYSIOLOGY
• Dehydration, endotoxemia, hemorrhage, and shock could result in ARF due to sustained decrease in renal perfusion (hypoperfusion and ischemia) and release of endogenous inflammatory and pressure mediators. • Infarction of the renal cortex and destruction of the base membrane of tubular cells in cases of decreased renal perfusion results in nephron dysfunction. • Direct injury to tubular cells caused by exposure to nephrotoxins.

SIGNALMENT
There is no predisposition of ruminant species, breed, sex, or age to develop ARF.

PHYSICAL EXMINATION FINDINGS
• Signs of acute renal failure are nonspecific and signs of primary disease may mask renal affection. • Anuria, oliguria, or polyuria may be observed. • Affected animals may present with dehydration, depression, anorexia, and diarrhea. • Oral ulcerations might be observed in uremic animals. • Severe cases develop muscular weakness and recumbency due to electrolyte and acid-base abnormalities. • Rectal palpation in cattle may reveal left renal enlargement.

CAUSES AND RISK FACTORS
• Conditions that result in systemic compromise, dehydration, and hypotension such as diarrhea, septicemia, endotoxemia, disseminated intravascular coagulopathy, and acute blood loss. • Nephrotoxic agents include heavy metals, aminoglycosides and tetracyclines, NSAIDs, toxic plants such as pigweed and oaks, vitamin C and D, hemoglobin, myoglobin, and calcium oxalate among others. • Infectious agents such as *Leptospira* spp. and urolithiasis are less common causes of ARF.



DIAGNOSIS

DIFFERENTIAL DIAGNOSIS
The nonspecific nature of clinical signs in cases of ARF in ruminants makes it difficult to develop a differential diagnosis list. Many primary disease conditions might result in ARF.

CBC/BIOCHEMISTRY/URINALYSIS
• Azotemia (increased BUN and creatinine). • Isosthenuria in the face of azotemia is a strong indicator of ARF. • Proteinuria, glucosuria, and granular casts may be present in urine. • Metabolic alkalosis, hypochloremia, hyponatremia, hypocalcemia, hyperphosphatemia, and hypermagnesemia are common findings with ARF. • Increased liver enzymes (SDH and GGT) values may be observed.

OTHER LABORATORY TESTS
Fractional excretion of sodium may be evaluated but the test should be compared to a normal animal (e.g., herd mate) of similar age, physiologic state, and nutritional status.

IMAGING
Ultrasonographic evaluation may reveal loss of detail of cortico-medullary junction, dilation of renal pelves and perirenal edema.

DIAGNOSTIC PROCEDURES
• Glomerular filtration rate assessment provides precise information of renal function. • Renal biopsy may provide diagnostic as well as prognostic information in cases of ARF.

PATHOLOGIC FINDINGS
Renal tubular degeneration and necrosis is a consistent histopathologic finding.

TREATMENT

THERAPEUTIC APPROACH
• The animal should be removed from the source of the nephrotoxins or exposure discontinued. • Isotonic, sodium-containing IV fluids with added calcium and potassium are indicated to correct acid-base and electrolyte abnormalities, increase renal perfusion, and promote diuresis. IV fluids should be maintained until the serum creatinine has returned to normal (usually 2–3 weeks). • Oral fluid therapy may be used if IV administration is impractical. • IV or oral fluids should be administered at a rate of two times the adult maintenance rate of 60 mL/kg/day. • Hydration and plasma protein should be monitored to avoid overhydration. • Supportive care should include broad-spectrum antibiotics, rumen transfaunations, and nutritional support.

MEDICATIONS

DRUGS OF CHOICE
• Furosemide (1 mg/kg IV or IM, q12h) administered every 2–3 hours to promote diuresis in anuric animals. • A dopamine drip (2 μg/kg/min IV) should be considered if diuresis is not achieved.

CONTRAINDICATIONS
• With repeated use of furosemide, the patient's serum sodium and potassium must be monitored carefully. • Drug withdrawal times need to be determined and maintained in food-producing animals.

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
• ARF due to ischemic episodes generally results in a grave prognosis. • Renal failure due to toxic causes may have a more favorable prognosis. • Failure to produce urine in the face of high volume IV fluids and diuretics carries a grave prognosis.

MISCELLANEOUS

ABBREVIATIONS
• ARF = acute renal failure • BUN = blood urea nitrogen • GGT = gamma glutamyl transferase • IM = intramuscular • IV = intravenous • NSAIDs = nonsteroidal anti-inflammatory drugs • SDH = sorbitol dehydrogenase

SEE ALSO
• Diarrheal Disease: Bovine • Diarrheal Diseases: Camelid • Diarrheal Diseases: Small Ruminants • Oak (*Quercus* spp.) Toxicity • Pyelonephritis

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AGRICULTURAL CHEMICAL TOXICITIES

BASICS

OVERVIEW

• Defined by the 1971 Agricultural Chemicals Regulation Law as "chemical agents such as fungicides and insecticides that are used to control crop-harming organisms (e.g., fungi, nematodes, mites, insects, and rodents) or viruses (hereinafter collectively referred to as 'diseases and pests')." Also included are plant growth regulators and germination inhibitors.
• EPA regulates pesticides in the USA; it must find that a pesticide poses a "reasonable certainty of no harm" before it can be registered for use on food or feeds.
• Residual agricultural chemicals are those remaining in the crops after application. They may become part of livestock feed and end up in meat or milk, harming ruminant animals (fetus and neonates in particular) and human beings.
• Agricultural chemicals are best classified by their specific application target.
  ◦ Insecticides—control pests; includes ovicides and larvicides.
  ◦ Fungicides—control diseases that damage feeds, field crops, and fruit trees.
  ◦ Insecticides/fungicides—act together to control harmful pests and diseases that damage field crops.
  ◦ Herbicides—control weeds; may be selective for a specific plant or group of plants or be totally nonselective.
  ◦ Plant growth regulators—either inhibit or stimulate growth of crops.
  ◦ Attractants—attract insect pests.
  ◦ Repellants—repel birds and small mammals that may damage crops.
  ◦ Spreaders—substances mixed with other chemicals to enhance adherence.
  ◦ Rodenticides—control mice, rats, and other small rodents.
• Ruminants are exposed through ingestion of contaminated feeds, treated seeds, or stored chemicals as well as oral and dermal exposure to recently treated fields or pastures.
• Many banned or cancelled products such as arsenicals and organochlorines may not have been properly disposed of, posing a hazard to ruminants that may ingest them.
• Newer insecticides (i.e., pyrethrins and pyrethroids, fipronil, and neonicotinoids) are safer and have replaced organophosphate and carbamates, but they are not without some harm to animals and the environment.

INCIDENCE/PREVALENCE

Sporadic; may be a single isolated animal or entire herd.

SYSTEMS AFFECTED

• Multisystemic, depending on chemical encountered

PATHOPHYSIOLOGY

• Toxicity:
  ◦ Variable depending on the product.
  ◦ Acute toxicity occurs within a few hours to a day.
  ◦ Chronic toxicity develops over time and is much more difficult to diagnose.
• Systemic absorption may result in accumulation in fat, liver, brain, kidney, and milk; some products result in transplacental transmission.
• Insecticides: Older products
  ◦ Old insecticides such as DDT, most organochlorines, and arsenicals are no longer registered in the USA, but may be used worldwide.
    ■ Highly toxic; many are carcinogens and some have associated reproductive defects.
    ■ Primarily lipid soluble, non-biodegradable, and accumulate in fat resulting in contamination of milk and meat.
    ■ Persist in the environment for very long periods of time.
    ■ High potential for bioaccumulation (absorption occurs more rapidly than excretion) and biomagnification (tissue concentrations of a contaminant increase as it passes up the food chain).
  ◦ Organophosphate (OP) and carbamate compounds
    ■ OPs: diazinon, dichlorvos, malathion, parathion, others.
    ■ Carbamates: aldicarb, carbaryl, carbofuran, methomyl, others.
    ■ Inhibit the enzyme acetylcholinesterase at cholinergic junction.
    ■ Many are highly toxic and no longer registered in the USA; still widely used worldwide.
• Insecticides: Newer products
  ◦ Pyrethrins and pyrethroids
    ■ Cyhalothrin, cypermethrin, deltamethrin, others.
    ■ Slow the opening of sodium channels causing hyperexcitability.
    ■ Most are highly lipophilic.
    ■ Generally safe.
    ■ Salivation, vomiting, tremors, seizures, dyspnea, prostration, death.
  ◦ Phenylpyrazole (fipronil)
    ■ Inhibits $GABA_A$ chloride channels resulting in hyperexcitation and neurotoxicity.
    ■ Generally safe unless exposed to highly concentrated product.
    ■ Buffalo and their calves may have increased risk of toxicosis.
    ■ Anorexia, twitching, tremors, ataxia, seizures.
  ◦ Neonicotinoids
    ■ Acetamiprid, dinotefuran, imidacloprid, nitenpyram, others.
    ■ Imidacloprid widely used worldwide in crop production.
    ■ Act on postsynaptic nicotinic receptors in insects CNS; little effect on mammalian receptors.
    ■ High margin of safety; not carcinogenic, mutagenic, or teratogenic.
    ■ Adult buffalo and their calves may have an increased risk of toxicity.
    ■ Lethargy, tremors, ataxia, hypothermia, death with high concentrations.
• Fungicides
  ◦ Less acute toxicity; may be due to decreased oral absorption.
  ◦ Respiratory and ophthalmic irritation from aerosolized products.
• Herbicides
  ◦ Toxicity varies by compound.
  ◦ Dinitro compounds
    ■ Highly toxic to ruminants.
    ■ Fever, dyspnea, tachycardia, seizures, death.
    ■ Methemoglobinemia, intravascular hemolysis.
    ■ Dinitrophenol compounds may cause yellow staining of the skin, conjunctiva, or hair.
  ◦ Paraquat
    ■ Highly toxic when wet; very low toxicity once dry and bound to vegetation.
    ■ Restricted use, nonspecific herbicide that kills all vegetation.
    ■ Widely used worldwide, less so in USA.
    ■ Dyspnea, anuria, muscle tremors, ataxia, salivation, recumbency, death.
  ◦ Sodium chlorate
    ■ Dyspnea, recumbency, seizures, abortion
    ■ Methemoglobinemia
  ◦ Glyphosate—considered less toxic, but controversial

HISTORICAL FINDINGS

Toxic ingestion may be known or suspected based on thorough history taking.

SIGNALMENT

• Very young and old.
• Neonates without functioning rumen are at a greater risk of toxicity from pyrethroids, OPs, and carbamates.
• Buffalo calves may be more susceptible to fipronil and neonicotinoids (acetamiprid).

PHYSICAL EXAMINATION FINDINGS

• General nonspecific signs include:
  ◦ Anorexia, hypersalivation, decreased rumen motility and bloat, abdominal pain, diarrhea (SLUDGE with OPs and carbamate insecticides)
  ◦ Stimulation or depression; lethargy, ataxia, hyperexcitation, tremors, seizures, recumbency, coma
  ◦ Tachypnea, dyspnea, respiratory arrest (paraquat)
  ◦ Polyuria, anuria (paraquat)
  ◦ Methemoglobinemia (chlorate and nitrate herbicides); cardiac arrest
  ◦ Hypo/hyperthermia
  ◦ Paresthesia, dermal irritation, inflammation (pyrethrins/pyrethroids; paraquat)
  ◦ Ophthalmic irritation (aerosolized products)

AGRICULTURAL CHEMICAL TOXICITIES (CONTINUED)

• See "Pathophysiology" for specific signs.
• Generally occur within 4–24 hours.
• May differ with individual animals, but specific system abnormalities are normally recognized in a herd situation.

CAUSES AND RISK FACTORS

• Improper storage or labeling of chemicals
• Equipment not cleaned well (e.g., ammonium nitrate residue in tank later used to fill water tanks)
• Access to a newly treated pasture or feed batch

DIAGNOSIS

DIFFERENTIAL DIAGNOSES

• Gastrointestinal—bloat, grain overload, coccidiosis
• Nervous– lead poisoning, nervous ketosis, polioencephalomalacia, rabies (single animal)
• Respiratory—bloat, infectious diseases

CBC/BIOCHEMISTRY/URINALYSIS

Often unremarkable

OTHER LABORATORY TESTS

Gas or liquid chromatography on fresh or frozen samples

OTHER DIAGNOSTIC PROCEDURES

• Variable
• Urine or milk analysis helpful for some exposures

PATHOLOGIC FINDINGS

Often unremarkable, especially with sudden death.

TREATMENT

THERAPEUTIC APPROACH

• Remove animals from suspected source.
• Activated charcoal or mineral oil within 10–12 hours of oral exposure.
  ◦ Contraindicated: digestible oils such as corn oil may increase absorption and should not be used as a cathartic.
• Symptomatic and supportive
  ◦ Dermal exposure— bathe with grease cutting dish detergent (wear gloves)
  ◦ Respiratory exposure—fresh air
  ◦ Seizures —barbiturates, diazepam
  ◦ Methemoglobinemia—methylene blue
  ◦ SLUDGE—atropine; pralidoxime not cost effective for larger animals

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS

Specific agent and response to therapy guide prognosis.

POSSIBLE COMPLICATIONS

Chronic poor production

CLIENT EDUCATION

See "Prevention"

PATIENT CARE

Milk and meat testing for clearance time

PREVENTION

• Proper labeling and storage of chemicals
• Disposal of older products
• Effective cleaning of multiple use equipment

MISCELLANEOUS

AGE-RELATED FACTORS

Pre-ruminants are more susceptible to chemicals degraded in the rumen.

ZOONOTIC POTENTIAL

• Meat and milk contamination.
• Movement and/or marketing of animals poisoned with agricultural chemicals varies by state; consult with board of animal health or diagnostic laboratory.

ABBREVIATIONS

• CNS = central nervous system
• DDT = dichloro-diphenyl-trichloroethane
• EPA = Environmental Protection Agency
• OPs = organophosphate insecticides
• SLUDGE = salivation, lacrimation, urination, diarrhea, gastroenteritis

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Internet Resources

• Agricultural Chemicals. Available at: http://toxics.usgs.gov/topics/agchemicals.html.
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AKABANE

BASICS

OVERVIEW

• Akabane virus (AKAV) is an arthropod-borne virus of ruminants.
• The virus is transmitted by small biting midges (or gnats) of *Culicoides.* Some species may carry both AKAV and bluetongue virus.
• AKAV infects a wide range of domesticated and wildlife ruminants. The disease has been reported in cattle, buffalo, sheep, and goats.
• Infection of pregnant animals results in abortion and stillbirth due to variable defects of the fetal nervous system and arthrogryposis, with no clinical signs in the dam.

INCIDENCE/PREVALENCE

• In endemic areas, the disease has a seasonal pattern, with peak vector activity in summer and diagnosis of outbreak made the subsequent winter.
• Most female animals are infected prior to reproductive age.
• Disease is seen in naïve animals which become infected during pregnancy, either due to "spillover" of the vector from its region, or movement of naïve animals into endemic areas.
• Surveys indicate that more than 80% of adult cattle in an endemic area are seropositive for AKAV. However, following years of drought or times of reduced vector populations, native livestock may not be exposed prior to breeding age and therefore become susceptible.
• Data from Japanese and Australian outbreaks suggest that the fetus is infected in 30–40% of pregnant cows which are infected with AKAV.

GEOGRAPHIC DISTRIBUTION

• The virus is widespread throughout Asia, Australia, Africa, and the Middle East.
• The virus is considered a foreign animal disease in the United States.

SYSTEMS AFFECTED

• Reproductive
• Musculoskeletal
• Nervous

PATHOPHYSIOLOGY

• AKAV is a single-stranded negative sense tripartite RNA virus. It is a member of the genus Orthobunyavirus, family Bunyaviridae, and serogroup Simbu. Four genotypes are identified (I, II, III, IV).
• After infection via the *Culicoides* vector, viremia occurs in the host 1–6 days later.
• Antibodies are detectable 14 days after infection.
• The virus crosses the placenta and infects the fetus, leading to the clinical signs.
• A fetus may be infected months prior to abortion, premature birth, or stillbirth.
• Akabane virus is a potent teratogen and affects the limbs (arthrogryposis) and central nervous system (porencephaly, hydranencephaly).
• Type of fetal abnormalities depend on stage of pregnancy. Susceptible periods range from 28 to 56 days in small ruminants and 3 to 6 months in cattle.
• Long-term carriers of the disease are not believed to occur.

HISTORICAL FINDINGS

• Severe epizootics or smaller outbreaks are associated with movement of naïve pregnant animals into an endemic area, or "spillover" of the vector into naïve populations outside the endemic area.
• Abortion outbreak of abnormal fetuses between 4 and 6 months of gestation.

SIGNALMENT

• Disease occurs only in cattle, sheep, and goats but antibodies have been found in several other large animal species.
• Manifestations of infection depend on the gestational age at the time of infection.
• In cattle:
  ◦ Infection between days 79–104 of gestation results in hydranencephaly.
  ◦ Infection between days 103–174 results in arthrogryposis with focal Wallerian-type degeneration of the brain and spinal cord.
  ◦ Infection in late gestation can result in encephalomyelitis.
  ◦ Infection in few postnatal calves and adult cows has been diagnosed in Japan, which manifested as encephalomyelitis.
• In sheep, infection at 32–48 days' gestation resulted in fetal abnormalities.
• In goats, infection at approximately 40 days' gestation resulted in fetal abnormalities.

PHYSICAL EXAMINATION FINDINGS

• Infections in adult ruminants are typically asymptomatic. However, the Iriki strain (Japan and Korea) has been associated with encephalitis.
• The hallmark of AKAV is congenital abnormalities of the neurologic and muscular systems. Effects on the fetus depend on time of infection during gestation.
• One group of investigators divided the gestational effects of AKAV into 5 groups (1 = late gestation infection; 5 = early gestation infection). Group 1 abnormalities included microscopic non-suppurative encephalomyelitis. Group 2 lesions included loss of ventral horn spinal cord neurons and Wallerian-type degeneration of ventral spinal nerves which resulted in ataxia, flaccid paralysis, and mild arthrogryposis. Group 4 lesions included arthrogryposis and hydranencephaly. Groups 3 and 5 were more severe manifestations of group 2 and 4 signs, respectively.
• Dystocia may occur at parturition or abortion due to fetal abnormalities.

GENETICS

N/A

CAUSES AND RISK FACTORS

• Clinical signs of AKAV are caused by exposure of ruminant fetuses to the virus by dam infection via *Culicoides* midges. Outbreaks are related to seasonal factors and vector distribution.
• Exposure of naïve pregnant animals to the virus-borne vector.

DIAGNOSIS

AKAV can be suspected based on clinical appearance of the fetus and knowledge of endemic areas; however, confirmation of the diagnosis by a diagnostic laboratory is required because gross appearance of AKAV is the same as many other vector-borne viruses.

DIFFERENTIAL DIAGNOSIS

• Bluetongue virus
• Bovine viral diarrhea virus
• Border disease virus
• Schmallenberg virus
• Cache Valley virus
• Aino virus
• Toxic, nutritional, or genetic causes of fetal neuromuscular defects

CBC/BIOCHEMISTRY/URINALYSIS

N/A

OTHER LABORATORY TESTS

• Serology can be performed in affected dams and precolostral serum of the offspring.
• Collection of fetoplacental tissues at necropsy can be diagnostic via several molecular techniques, including reverse transcriptase real-time PCR, competitive ELISA, and immunohistochemistry/immunofluorescence.

IMAGING

N/A

OTHER DIAGNOSTIC PROCEDURES

N/A

PATHOLOGIC FINDINGS

• The most common lesions are arthrogryposis and hydranencephaly.
• Other neurologic abnormalities may include porencephaly and microencephaly.
• In the brain, degenerative and necrotic neurons as well as perineuronal and perivascular edema has been described.
• There may be loss of ventral horn spinal cord neurons and Wallerian-type degeneration of ventral spinal nerves.
• Other findings in calves include: gliosis, demyelination, hepatitis, nephritis, and myodegeneration/polymyositis.
• In sheep, there can be a marked loss of the ventral horns of the spinal cord which leads to hypoplastic spinal cord and muscle atrophy (e.g., torticollis). Pulmonary hypoplasia may also be noted.

AKABANE (CONTINUED)

TREATMENT

THERAPEUTIC APPROACH
• There is no treatment for AKAV.
• Most fetuses born alive die or are euthanized due to effects of the virus.
• Subsequent pregnancies of the dam will not be affected.

SURGICAL CONSIDERATIONS AND TECHNIQUES
N/A

MEDICATIONS

DRUGS OF CHOICE
N/A

CONTRAINDICATIONS
N/A

PRECAUTIONS
N/A

POSSIBLE INTERACTIONS
N/A

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
• Most offspring born alive are either euthanized or die shortly after birth.
• Subsequent pregnancies of the dam will not be affected.

POSSIBLE COMPLICATIONS
• Dystocia
• Infertility

CLIENT EDUATION
• In endemic areas, clients should be aware of repercussions of introducing naïve pregnant animals into the herd.
• Vaccination should also be considered in areas where it is available.

PATIENT CARE
• Specific treatment and supportive care if there are any complications following abortion.

PREVENTION
• Prevention of AKAV includes vector control and vaccination.
• Vector control should include elimination of vector breeding sites, and repellents for pregnant animals.
• Naïve pregnant animals should not be introduced during seasons of high vector activity (summer and autumn).
• Naïve animals should be introduced to endemic areas prior to breeding to develop immunity.
• Breeding season may be altered to avoid period of highest risk.
• Live (Japan) and inactivated (Japan, Australia, Korea) vaccines are available for the prevention of AKAV and are to be administered prior to breeding.

MISCELLANEOUS

ASSOCIATED CONDITIONS
Dystocia, retained placenta

AGE-RELATED FACTORS
AKAV affects fetal ruminants.

ZOONOTIC POTENTIAL
There are no indications that AKAV is zoonotic.

PREGNANCY
Effects of AKAV are dependent on the gestational age of the fetus at the time of infection. However, most affected neonates die or are euthanized after birth due to effects of the virus irrespective of time of infection in utero.

BIOSECURITY
Suspected cases or outbreaks of AKAV outside its endemic areas (see "Geographic Distribution") should immediately be reported to the proper governmental veterinary authorities (i.e., state or federal veterinarian).

PRODUCTION MANAGEMENT
• In endemic areas, avoid introduction of naïve pregnant animals in the summer and autumn months.
• Implement vector control programs to reduce potential transmission of AKAV.
• Consider vaccination protocols prior to breeding.

SYNONYM
Arthrogryposis-hydranencephaly syndrome (AH syndrome)

ABBREVIATION
AKAV = Akabane virus

SEE ALSO
• Abortion: Viral, Fungal, and Nutritional
• Arthrogryposis
• Bluetongue Virus
• Border Disease
• Bovine Viral Diarrhea Virus
• Cache Valley Virus
• Congenital Defects: Bovine
• Lupine Toxicity
• Schmallenberg Virus

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ANAPHYLAXIS

BASICS

OVERVIEW
An anaphylactic reaction is a pathologic immune response that occurs following exposure of a sensitized animal to a specific antigen. This exposure results in urticaria, pruritus, and angioedema, followed by vascular collapse, shock and often life-threatening respiratory distress. Anaphylaxis has now been included under type I (immediate) hypersensitivity.

INCIDENCE/PREVALENCE
Sporadic, dependent on exposure to inciting antigen. Tetanus antitoxin is one of the major vaccine antigens associated with anaphylaxis.

SYSTEMS AFFECTED
• Cardiovascular • Respiratory • Urinary
• Digestive • Integument

PATHOPHYSIOLOGY
Anaphylaxis is an acute systemic manifestation of the interaction of an antigen (allergen) binding to IgE antibodies, which are bound to mast cells and basophils. This binding of antigens to cell-bound IgE antibodies triggers the release of chemical substances from the mast cells and basophils. The major biologically active mediators produced by mast cells and basophils include histamine, leukotrienes, the eosinophilic chemotactic factor, platelet-activating factor, kinins, serotonins, and proteolytic enzymes. These chemicals directly affect both the vascular system, causing vasodilatation and increased vascular permeability, and smooth muscles, causing contraction of the bronchi and respiratory distress.

SIGNALMENT
Bovine, ovine, and caprine; also reported in many other species of ruminants.

PHYSICAL EXAMINATION FINDINGS
Sudden, severe dyspnea, muscle tremors, anxiety, occurs within a few to 10–15 minutes following exposure to the antigen; muscle tremor may be severe and temperature may rise to 105°F. History of injection in the previous hour. Occasionally profuse salivation, mild bloat, diarrhea, urticaria, angioneurotic edema, and rhinitis. Laminitis rarely occurs in ruminants. Auscultation of the chest—vesicular murmur, crackling if edema is present, and emphysema in the later stages if dyspnea was severe.

GENETICS
There have been reports of higher incidence in certain lines and breeds of cattle (Holstein-Friesian and Angus) and Saanen goats.

CAUSES AND RISK FACTORS
Common agents causing anaphylaxis include blood transfusions, vaccines, horse sera, insect bites, heterologous enzymes and hormones, and certain drugs, such as penicillin and lidocaine. Milk allergy occurs occasionally in cows. This can happen when there is increased intramammary pressure to a point that normally sequestered milk components, notably casein, gain access to the circulation; these "foreign" proteins induce a type I hypersensitivity. Previous exposure to antigens (i.e., previous treatment with blood or blood products or vaccines).

DIAGNOSIS

DIFFERENTIAL DIAGNOSES
• Acute Bloat • Acute Bronchopneumonia

CBC/BIOCHEMSTRY/URINALYSIS
Increase in PCV, high plasma K^+, neutropenia

PATHOLOGIC FINDINGS
Lungs—severe pulmonary edema in calves and lambs; pulmonary edema and emphysema without blood engorgement.

TREATMENT

THERAPEUTIC APPROACH
• Ancillary support of blood pressure (IV fluids) and respiration may be necessary. • In dairy cattle that have been recently dried off, recovery usually is prompt once the gland is emptied.

MEDICATIONS

DRUGS OF CHOICE
• Anaphylactic shock is treated with an injection of epinephrine. Epinephrine (1/100) subcutaneously or intravenously at a dose of 1 mL per 100 lb. of body weight is the drug of choice and can literally be a lifesaver. A second dose can be given in 15–20 minutes if needed. • In addition, flunixin meglumine (50 mg/mL) can be given at a rate of 1–2 mL per 100 lb. body weight IV or IM as well.
• Corticosteroids potentiate the effects of epinephrine and may be given following the administration of epinephrine.
• Antihistamines have no effect once signs are present.

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
Animals treated promptly usually return to normal within 12–24 h.

POSSIBLE COMPLICATIONS
Emphysema may result from severe dyspnea and violent muscle spasms. Following anaphylaxis, animals may spontaneously abort.

PATIENT CARE
Animals need to have their respiratory system monitored for the next 24 hours to detect any emphysema.

PREVENTION
Discuss the situation associated with the onset with the producer. Certain products may need to be avoided.

MISCELLANEOUS

ABBREVIATIONS
• IgE = immunoglobulin E
• IV = intravenous
• PCV = packed cell volume

SEE ALSO
• Bloat
• Plants Producing Acute Respiratory Distress Syndrome
• Respiratory Disease: Bovine

Suggested Reading

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A

ANAPLASMOSIS

BASICS

OVERVIEW
Anaplasma is a hemoparasite of ruminants.

INCIDENCE/PREVALENCE
Anaplasmosis is the most prevalent tickborne disease of cattle.

GEOGRAPHIC DISTRIBUTION
Worldwide

SYSTEMS AFFECTED
Hemolymphatic

PATHOPHYSIOLOGY
• *Anaplasma marginale* is the most common cause of clinical disease in cattle; *A. centrale* causes mild disease. *A. ovis* causes mild disease in sheep. • *Anaplasma* is transmitted by many ticks including *Dermacentor* (USA) and *Rhipicephalus* spp. (other regions). • Iatrogenic transmission can occur. • Infected erythrocytes are destroyed by the monocyte macrophage system leading to extravascular hemolysis and anemia.

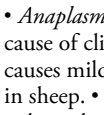
HISTORICAL FINDINGS
N/A

SIGNALMENT
Clinical disease primarily occurs in cattle over 3 years.

PHYSICAL EXAMINATION FINDINGS
• Calves generally display no clinical signs; cattle 6 months to 3 years of age may have mild to moderate signs. • Cattle over 3 years can have severe clinical disease. • Acute disease provokes fever, lethargy and anorexia. • Anemia results in tachypnea, tachycardia, pallor, icterus, exercise intolerance, ataxia, and death. • Hemoglobinemia and hemoglobinuria do not occur. • Survivors generally become subclinical carriers.

GENETICS
Bos indicus cattle may be more resistant to *A. marginale* infection than *Bos taurus* breeds.

CAUSES AND RISK FACTORS
• Disease is spread naturally by ticks and mechanically by *Tabanid flies*, needles, and blood-contaminated instruments. • Moving naïve adults to endemic areas or carrier animals to non-endemic areas may precipitate outbreaks.



DIAGNOSIS

DIFFERENTIAL DIAGNOSES
• Bleeding abomasal ulcer • Babesiosis • Leptospirosis • Bacillary hemoglobinuria • Hepatotoxic plants • Copper toxicity (sheep)

CBC/BIOCHEMISTRY/URINALYSIS
• CBC shows decline in PCV with regeneration indicated by anisocytosis, basophilic stippling, poikilocytosis, polychromasia, and reticulocytosis. On blood smears stained with Wright's, new methylene blue, or Giemsa stain, organisms will appear darkly stained on erythrocytes. • Increased hepatobiliary values. • No hemoglobinemia or hemoglobinuria.

OTHER LABORATORY TESTS
• cELISA is a widely used serologic test for identifying infected cattle. • Rapid card agglutination and complement fixation tests. • PCR more reliable for diagnosing acute infection.

IMAGING
N/A

OTHER DIAGNOSTIC PROCEDURES
N/A

PATHOLOGIC FINDINGS
• Pallor or icterus • Splenomegaly • Hepatomegaly • Prominent erythrophagocytosis in reticuloendothelial organs

TREATMENT

THERAPEUTIC APPROACH
• Minimize stress • Blood transfusion if significant signs of anemia and low PCV

SURGICAL CONSIDERATIONS AND TECHNIQUES
N/A

MEDICATIONS

DRUGS OF CHOICE
• Long acting oxytetracycline 20 mg/kg, IM or SQ, once to twice q 72 h. • Imidocarb efficacious but not approved in Europe or USA.

CONTRAINDICATIONS
N/A

PRECAUTIONS
Appropriate milk and meat withdrawal times must be followed for all pharmaceutical agents administered to food-producing animals.

POSSIBLE INTERACTIONS
N/A

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
• Mildly affected animals may recover and become carriers. • Severely affected animals often die within hours to days.

POSSIBLE COMPLICATIONS
• Old protocols to clear the carrier state are ineffective. • New carrier state protocols violate AMDUCA. • Clearing carrier animals makes them susceptible to reinfection.

CLIENT EDUCATION
N/A

PATIENT CARE
Monitor for clinical indications that transfusion is required.

PREVENTION
• Arthropod control. • Chemoprophylaxis can be achieved with 20 mg/kg of long-acting oxytetracycline every 21–28 days from the start of the vector season until 30–60 days after vector season ends, or with chlortetracycline in feed at 1.1 mg/kg daily during the vector season. • Live and killed vaccines are variably available, and decrease the severity of clinical signs.

MISCELLANEOUS

ASSOCIATED CONDITIONS
N/A

AGE-RELATED FACTORS
See "Signalment"

ZOONOTIC POTENTIAL
N/A

PREGNANCY
Abortion can occur

BIOSECURITY
N/A

PRODUCTION MANAGEMENT
Can cause significant loss in endemic areas.

SYNONYMS
N/A

ABBREVIATIONS
cELISA = competitive ELISA

SEE ALSO
• Babesiosis
• Bacillary Hemoglobinuria
• Leptospirosis

Suggested Reading

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ANEMIA, NONREGENERATIVE

BASICS

OVERVIEW

• Anemia is defined as a decrease in the red blood cell (RBC) count, hemoglobin (Hb) concentration, and/or packed cell volume (PCV)
• Nonregenerative anemia is caused by reduced or defective erythropoiesis.
• Nonregenerative anemia is suspected when signs of bone marrow regeneration (reticulocytosis, polychromasia, and basophilic stippling of RBCs) are minimal to absent.

SYSTEMS AFFECTED

• Multisystemic

PATHOPHYSIOLOGY

• Anemia is characterized by a reduced capacity of the blood to transport oxygen, leading to systemic tissue hypoxia and increased erythropoietin (EPO) production.
• Most clinical signs associated with anemia result from poor tissue oxygen delivery.

Nonregenerative Anemia Caused by Reduced Erythropoiesis

• Chronic inflammation, chronic renal disease, or bone marrow failure can lead to reduced erythropoiesis
• Chronic inflammation is associated with increased liver expression of hepcidin liver, causing alterations in iron metabolism and bone marrow responsiveness to EPO.
• Chronic renal disease can be associated with decreased EPO production by the kidneys.
• Destruction of hematopoietic stem cells due to damage by toxicants, irradiation, immune-mediated mechanisms, or infiltration of the marrow with abnormal cells can lead to bone marrow failure.

Nonregenerative Anemia Caused by Defective Erythropoiesis

• Disorders of Hb or DNA synthesis can lead to defective erythropoiesis.
• Iron (Fe) and copper (Cu) deficiencies can impair Hb synthesis.
• Iron is crucial to Hb synthesis because each Hb molecule is made up of four heme groups, each group being composed of an Fe molecule and a porphyrin.
• Iron deficiency is usually caused by chronic blood loss but can also be secondary to dietary Fe deficiency in young milk-fed animals.
• Copper deficiency can lead to Fe deficiency because several Cu-containing proteins are required for Fe transport.
• Dietary Cu deficiency and/or excessive dietary intake of molybdenum, sulfate, or zinc can cause decreased Cu absorption and lead to Cu deficiency.
• Dietary cobalt (Co) deficiency can cause Co deficiency and lead to vitamin B12 deficiency and defective DNA synthesis

HISTORICAL FINDINGS

• Weakness, lethargy, anorexia, weight loss, exercise intolerance, or syncopes.

SIGNALMENT

Bovine, ovine, caprine, and camelid species.

PHYSICAL EXAMINATION FINDINGS

• Clinical signs are less overt when the anemia progresses slowly.
• Lethargy, weakness, or obtundation.
• Pale mucus membranes.
• Tachycardia and tachypnea.
• Heart murmur (due to reduced blood viscosity).

GENETICS

• Congenital dyserythropoiesis is an autosomal recessive trait in polled Hereford cattle.
• Myelofibrosis is an autosomal recessive trait in pygmy goats

CAUSES AND RISK FACTORS

Nonregenerative Anemia Caused by Reduced Erythropoiesis

• Common causes of chronic inflammation include pneumonia, peritonitis, deep digital sepsis, liver abscesses, paratuberculosis, and lymphoma
• Causes of chronic renal disease include pyelonephritis, urolithiasis, amyloidosis, and glomerulonephritis.
• Causes of bone marrow failure include bracken fern toxicosis, bovine neonatal pancytopenia, irradiation, myelofibrosis, and neoplasia

Nonregenerative Anemia Caused by Defective Erythropoiesis

• Causes of defective erythropoiesis include congenital dyserythropoiesis and deficiencies in Fe, Cu, and Co.
  ◦ Chronic blood loss: GI ulcers, hematuria, hereditary coagulation factor deficiencies, parasitism, dietary Fe deficiency in milk-fed ruminants

DIAGNOSIS

DIFFERENTIAL DIAGNOSES

• Normocytic, normochromic anemia with normal to increased neutrophil and platelet counts can be caused by chronic inflammation or renal disease.
• Normocytic, normochromic anemia with decreased neutrophil and/or platelet counts can be caused by bone marrow failure.
• Microcytic, hypochromic anemia with variable neutrophil and platelet counts can be caused by iron or copper deficiencies.
• Macrocytic, normochromic anemia with variable neutrophil and platelet counts can be caused by cobalt deficiency or congenital dyserythropoiesis.

CBC/BIOCHEMISTRY/URINALYSIS

• The PCV is the easiest and most accurate method to identify anemia.
• The PVC should be interpreted with consideration of the animal's hydration status and any potential cause of splenic contraction (excitement, exercise, handling, or transportation).

Severity of anemia	*PCV (%)*
Mild	20–26
Moderate	14–19
Severe	10–13
Very severe	<10

• Blood should be analyzed within 30–60 minutes of collection or stored at refrigerator temperature (4°C) and analyzed within 24 hours
• Delayed analysis may result in marked cellular swelling and therefore a false increase in MCV
• Hypochromasia and microcytosis are hallmarks of iron and copper deficiencies.
• Hyperfibrinogenemia, hypoalbuminemia, and hyperglobulinemia are often present in with chronic inflammation.

	Chronic inflammation or renal disease	*Bracken fern toxicosis*	*Iron and copper deficiencies*	*Cobalt deficiency*
Mechanism	Increased hepcidin or decreased EPO production	Cytotoxic damage to bone marrow	Defective	Defective DNA synthesis
Plasma protein	N - ↑	N - ↓	N - ↓	N - ↓
PCV	↓ - ↓↓	↓ - ↓↓	↓ - ↓↓↓	↓ - ↓↓
MCV	N	N	N - ↓	N - ↑
MCHC	N	N	N - ↓	N
Neutrophil count	N - ↑	N - ↓	N - ↑	N - ↑
Platelet count	N - ↑	N - ↓	N - ↑	N - ↑

N = normal, ↓ = slightly decreased, ↓↓ = moderately decreased, ↓↓↓ = markedly decreased, and ↑ = slightly increased

ANEMIA, NONREGENERATIVE (CONTINUED)

• Azotemia, hypoalbuminemia, and proteinuria are often present with chronic renal disease.

OTHER LABORATORY TESTS

Chronic Inflammation and Iron Deficiency

	Chronic inflammation	*Iron deficiency*
Serum ferritin	N - ↑	↓
Bone marrow iron content	N - ↑	↓
Total iron binding capacity	N - ↓	N - ↑

N = normal, ↓ = decreased, and ↑ = increased

• Hepatic Fe concentration: <40 ppm on a wet matter basis with Fe deficiency anemia

Copper Deficiency

• Plasma Cu concentration: <0.5 µg/mL
• Hepatic Cu concentration: <35 ppm on a dry matter basis

Cobalt Deficiency

• Serum vitamin B_{12} concentration: <0.2 mg/mL
• Hepatic vitamin B_{12} concentration: <0.2 ppm (dry matter basis)

Parasitism

• Examination of the skin and coat
• Fecal flotation

OTHER DIAGNOSTIC PROCEDURES

• Bone marrow examination is indicated when the cause of a nonregenerative anemia remains undetermined and/or atypical or unexplained immature cells are observed on the peripheral blood smear.

TREATMENT

THERAPEUTIC APPROACH

• Treatment must primarily address the underlying cause(s) of the anemia.
• Blood transfusion is indicated in valuable animals with overt clinical signs of anemia and/or PCV <12%.
• Activity and stress should be minimized.
• Routine care procedures should be delayed (deworming, hoof trimming …).

MEDICATIONS

DRUGS OF CHOICE

• Oral iron supplementation is indicated with iron deficiency anemia.
• Oxygen therapy may be beneficial in hypoxemic animals (PaO_2 <80 mmHg).

CONTRAINDICATIONS

• Iron supplementation is contraindicated in animals with chronic inflammation.
• Appropriate milk and meat withdrawal times must be followed for all compounds administered to food-producing animals.

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS

• Clinical course and prognosis is dependent on the underlying disease process.

PATIENT CARE

• Heart rate, respiratory rate, mucus membrane color, blood lactate, and arterial blood gas analysis can be used to monitor systemic tissue oxygen delivery.
• CBC or PCV with blood smear examination should be repeated every 1–2 days until evidence of bone marrow regeneration is present.
• Reevaluation is indicated after 7–10 days in stabilized animals.

PREVENTION

• Feeding of trace mineral supplements with label claims for the species that are being supplemented is recommended.
• Diet should contain 4–10 ppm of Cu, 0.1–0.2 ppm of Co, and 30–40 ppm of Fe.
• The dietary copper/molybdenum ratio should be maintained between 5:1 and 10:1.

MISCELLANEOUS

ASSOCIATED CONDITIONS

• A congenital syndrome characterized by dyserythropoiesis and progressive alopecia has been described in polled Hereford cattle.

AGE-RELATED FACTORS

N/A

SYNONYMS

• Anemia of inflammatory disease

ABBREVIATIONS

• CBC = complete blood count
• Co = cobalt
• Cu = copper
• DNA = deoxyribonucleic acid
• EPO = erythropoietin
• Fe = iron
• Hb = hemoglobin
• MCHC = mean corpuscular hemoglobin concentration
• MCV = mean corpuscular volume
• PaO_2 = partial pressure of oxygen in arterial blood
• PCV = packed cell volume
• RBC = red blood cell

SEE ALSO

• Anemia, Regenerative
• Bracken Fern Toxicity
• Copper Deficiency and Toxicity
• Molybdenum Toxicity
• Parasite Control Programs

Suggested Reading

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ANEMIA, REGENERATIVE

BASICS

OVERVIEW

• Anemia is defined as a decrease in the red blood cell (RBC) count, hemoglobin (Hb) concentration, and/or packed cell volume (PCV). • Regenerative anemia is caused by blood loss and/or accelerated RBC destruction. • Signs of bone marrow regeneration include reticulocytosis, polychromasia, and basophilic stippling of RBCs.

INCIDENCE/PREVALENCE

• Extravascular hemolysis is more common than intravascular hemolysis. • Chronic copper toxicosis occurs most commonly in sheep. • Neonatal isoerythrolysis (NI) has been described in calves born to cows immunized with *Anaplasma* or *Babesia* vaccines. • NI has been described in lambs and kids following ingestion of bovine colostrum.

SYSTEMS AFFECTED

• Multisystemic

PATHOPHYSIOLOGY

• Anemia is characterized by a reduced capacity of the blood to transport oxygen, leading to systemic tissue hypoxia and increased erythropoietin production. • 2–3 days are necessary for signs of bone marrow regeneration to be evident in the blood. • Reticulocytosis usually peaks about 7–10 days after bone marrow stimulation.

Regenerative Anemia Caused by Blood Loss (Hemorrhagic Anemia)

• Causes of blood loss include trauma, surgery, coagulation factor deficiencies, thrombocytopenia, parasitism, and neoplasia. • Regenerative response is usually higher with internal than external hemorrhage because some RBCs are reabsorbed by lymphatics and iron (Fe) is recycled. • Chronic blood loss is usually associated with mild regenerative response and can lead to Fe deficiency.

Regenerative Anemia Caused by Accelerated RBC Destruction (Hemolytic Anemia)

• Accelerated RBC destruction (hemolysis) can occur within the blood vessels (intravascular) and/or outside of the blood vessels (extravascular). • Extravascular hemolysis is more common than intravascular hemolysis. • Reticulocyte counts are usually higher in hemolytic anemias than in hemorrhagic anemias. • Onset of clinical signs is usually peracute to acute with intravascular hemolysis and more progressive with extravascular hemolysis. • Icterus may develop in animals with hemolytic anemia secondary to increased Hb degradation and bilirubin formation. • Intravascular hemolysis is characterized by hemoglobinemia (free Hb in the plasma) that can lead to red discoloration of plasma and/or increased MCHC. • Hemoglobinuria can develop if the plasma free Hb concentration exceeds the capacity of renal tubular reabsorption. • Causes of intravascular hemolysis include bacterial infections, erythrocytic parasites, oxidative damage to the RBC membrane, primary immune-mediated disorders, osmotic lysis, envenomation, and congenital disorders. • Extravascular hemolysis results from sequestration and phagocytosis of RBCs in spleen or liver due to decreased RBC deformability or immune-mediated mechanisms. • Extravascular hemolysis does not cause hemoglobinemia or hemoglobinuria. • Causes of extravascular hemolytic anemias include erythrocytic or endothelial parasites and congenital disorders.

SIGNALMENT

• Bovine, ovine, caprine, and camelid species. • Neonatal isoerythrolysis (NI) can occur in calves born to cows immunized with Anaplasma or Babesia vaccines and in lambs fed bovine colostrum. • Chronic copper (Cu) toxicosis occurs most commonly in sheep • Hereditary factor VIII and IX deficiencies almost always occur in males. • Anaplasmosis and babesiosis occur most commonly in adults.

PHYSICAL EXAMINATION FINDINGS

• Lethargy, obtundation, weakness, or syncopes • Pale mucus membranes • Tachycardia and tachypnea • Heart murmur (due to reduced blood viscosity) • Icterus • Hemoglobinuria (with intravascular hemolysis)

GENETICS

• Hereditary factor VIII deficiency is an autosomal recessive trait in Hereford and Japanese Brown cattle. • Hereditary factor XI deficiency is an autosomal recessive trait in Holstein and Japanese Black cattle. • Hereditary afibrinogenemia is an incompletely dominant trait in Saanen goats. • Hereditary spherocytosis is an autosomal dominant trait in Japanese Black cattle. • Congenital erythropoietic porphyria is an autosomal recessive trait in Holstein cattle.

CAUSES AND RISK FACTORS

Regenerative Anemia Caused by Acute Blood Loss

• Coagulation factor deficiencies
  ◦ Disseminated intravascular coagulation (DIC) ◦ Hereditary afibrinogenemia and factor VIII or XI deficiencies ◦ Moldy sweet clover and rodenticide toxicosis
• Gastrointestinal ulcers
• Hemorrhagic bowel syndrome
• Thrombocytopenia
  ◦ Bovine viral diarrhea virus infection
  ◦ DIC ◦ Snake evenomation
• Trauma
• Surgery

Regenerative Anemia Caused by Chronic Blood Loss

• Gastrointestinal ulcers
• Chronic hematuria
  ◦ Bracken fern toxicosis ◦ Urolithiasis
  ◦ Pyelonephritis
• Hereditary afibrinogenemia and factor VIII or XI deficiencies
• Parasites
  ◦ Internal (*Haemonchus* spp., *Bunostomum* spp., *Eimeria* spp.) ◦ External (blood sucking lice, fleas, ticks)

Regenerative Anemia Caused by Accelerated RBC Destruction

Causes of Extravascular Hemolysis

• Intraerythrocytic parasites
  ◦ *Anaplasma centrale* and *A. marginale* in cattle ◦ *Theileria annulata*, *T. buffeli*, *T. mutans*, and *T. parva* in cattle ◦ *A. ovis* and *T. lestoquardi* in small ruminants
• Epierythrocytic parasites
  ◦ *Candidatus Mycoplasma haemolamae* in camelids ◦ *Mycoplasma wenyonii* in cattle and *M. ovis* in small ruminants
• Extraerythrocytic parasites
  ◦ *Trypanosoma congolense*, *T. brucei*, and *T. vivax* in cattle
• Endothelial parasites
  ◦ *Sarcocystis* spp. in cattle, and small ruminants
• Hereditary RBC membrane defects
  ◦ Hereditary spherocytosis

Causes of Intravascular Hemolysis

• Bacterial infections
  ◦ *Clostridium haemolyticum* in cattle and sheep ◦ *Clostridium perfringens* type A in cattle and sheep ◦ Leptospirosis
• Osmotic lysis
  ◦ Overuse of hypotonic intravenous fluids
  ◦ Water intoxication
• Intraerythrocytic parasites
  ◦ *Babesia bigemina*, *B. bovis*, *B. divergens*, and *B. major* in cattle ◦ *B. motasi* and *B. ovis* in small ruminants
• Oxidative damage
  ◦ *Brassica* spp. ingestion ◦ Chronic Cu toxicosis ◦ Oak leaves and acorns ingestion ◦ Onion or garlic ingestion ◦ Red maple leaf ingestion ◦ Selenium deficiency ◦ Zinc toxicosis
• Primary immune-mediated disorders
  ◦ Neonatal isoerythrolysis ◦ Incompatible blood transfusion
• RBC membrane alterations due to other mechanisms
  ◦ Congenital erythropoietic porphyria
  ◦ Postparturient hemoglobinuria ◦ Snake envenomation

ANEMIA, REGENERATIVE (CONTINUED)

DIAGNOSIS

CBC/BIOCHEMISTRY/URINALYSIS

• The PCV is the easiest and most accurate method to identify anemia. • The PVC should be interpreted with consideration of the animal's hydration status and any potential cause of splenic contraction (excitement, exercise, handling, or transportation).

Severity of anemia	*PCV (%)*
Mild	20–26
Moderate	14–19
Severe	10–13
Very severe	<10

• Blood should be analyzed within 30–60 minutes of collection or stored at refrigerator temperature (4°C) and analyzed within 24 hours. • Delayed analysis may result in marked cellular swelling and a false increase in MCV.

OTHER LABORATORY TESTS

Hemorrhagic Anemia

• Examination of the skin and coat (external parasites) • Fecal flotation (internal parasites) • Fecal occult blood test (gastrointestinal ulcers) • Coagulation tests (coagulation factor deficiencies)

	aPTT	*PT*	*TT*
Afibrinogenemia	↑	↑	↑
DIC	↑	↑	↑
Factor VIII deficiency	↑	N	N
Factor XI deficiency	↑	N	N
Sweet clover toxicosis	↑	↑	N
Rodenticide toxicosis	↑	↑	N

aPTT = activated partial thromboplastin time, PT = prothrombin time, TT = thrombin time, N = normal, and ↑ = increased

Immune-Mediated Hemolytic Disorders

• Positive autoagglutination and direct antiglobulin (Coombs') test

	Acute blood loss	*Chronic blood loss*	*Intravascular hemolysis*	*Extravascular hemolysis*
Plasma protein	N - ↓	N - ↓	N - ↑	N - ↑
PCV	↓ - ↓↓	↓ - ↓↓↓	↓↓ - ↓↓↓	↓ - ↓↓↓
MCV	N - ↑	N - ↓	N - ↑	N - ↑
MCHC	N - ↓	N - ↓	↑	N - ↓
Reticulocyte count	N - ↑↑	N - ↑	N - ↑↑↑	N - ↑↑↑
Neutrophil count	N - ↑	N - ↑	N - ↑	N - ↑
Platelet count	N - ↑	N - ↑	N - ↑	N

N = normal, ↓ = slightly decreased, ↓↓ = moderately decreased, ↓↓↓ = markedly decreased, ↑ = slightly increased, ↑↑ = moderately increased, and ↑↑↑ = markedly increased

Intravascular versus Extravascular Hemolysis

	Intravascular hemolysis	*Extravascular hemolysis*
Hyperbilirubinemia	can be present	usually present
Hemoglobinemia	Yes	No
Plasma color	pink to red	straw to yellow
Hemoglobinuria	usually present	No
Urine color	pink to red	straw to yellow
Blood reagent strip	positive	usually negative
Bilirubinuria	can be present	usually present
RBCs in urine sediment	No	No

• Abnormal RBC morphology or RBC inclusions may suggest the mechanism of hemolysis. • Heinz bodies suggest oxidative damage. • Spherocytes suggest immune-mediated hemolysis. • Keratocytes and schistocytes DIC.

Chronic Copper Toxicosis

• Serum or plasma Cu concentration: >2 μg/mL • Hepatic Cu concentration >350 ppm (dry matter basis) • Renal Cu concentration >100 ppm (dry matter basis) (most reliable)

Lead Toxicosis

• Lead toxicosis should be suspected when basophilic stippling if RBCs is accompanied by signs of inappropriate bone marrow regeneration (minimal polychromasia with nucleated RBCs).

Selenium (Se) Deficiency

• Blood Se concentration <50 ng/mL • Blood glutathione peroxidase (GSH-Px) activity <15 m/min/mg of Hb

PATHOLOGIC FINDINGS

• Extravascular hemolysis is usually associated with splenomegaly.

TREATMENT

THERAPEUTIC APPROACH

• Treatment must primarily address the underlying cause(s) of the anemia. • Blood transfusion is indicated in valuable animals with overt clinical signs of anemia PCV <12%. • Activity and stress should be minimized. • Routine care procedures should be delayed (deworming, hoof trimming ...).

MEDICATIONS

DRUGS OF CHOICE

• Oral iron supplementation is indicated with chronic blood loss. • Oxygen therapy may be beneficial in hypoxemic animals (PaO_2 <80 mmHg). • Fluid therapy is indicated with hypovolemia to support cardiovascular function and with intravascular hemolysis to prevent renal damage.

CONTRAINDICATIONS

• Appropriate milk and meat withdrawal times must be followed for all compounds administered to food-producing animals.

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS

• Clinical course and prognosis is dependent on the underlying disease process. • Return to reference intervals is expected in 1–2 weeks following a single acute blood loss. • Chronic blood loss should be suspected if reticulocytosis persists >3 weeks.

POSSIBLE COMPLICATIONS

• Iron deficiency anemia loss develops more quickly in young milk-fed animals because they have limited Fe stores.

PATIENT CARE

• Heart rate, respiratory rate, mucus membrane color, blood lactate, and arterial blood gas analysis can be used to monitor systemic tissue oxygen delivery. • CBC or PCV with blood smear examination should

be repeated every 1–2 days until stabilization of the PVC. • Reevaluation is indicated after 7–10 days in stabilized animals.

PREVENTION

• Cross-matching is indicated in animals receiving more than one blood transfusion. • Feeding of trace mineral supplements with label claims for the species that are being supplemented is recommended. • Diet should contain 4–10 ppm of Cu and not less than 0.1 ppm of Se. • The dietary copper/molybdenum ratio should be maintained between 5:1 and 10:1
• Prevention programs of vector-borne diseases should be implemented in endemic areas.

MISCELLANEOUS

ABBREVIATIONS

• aPTT = activated partial thromboplastin time
• CBC = complete blood count
• Cu = copper
• DIC = disseminated intravascular coagulation
• Fe = iron
• GSH-Px = glutathione peroxidase
• Hb = hemoglobin
• MCHC = mean corpuscular hemoglobin concentration
• MCV = mean corpuscular volume
• NI = neonatal isoerythrolysis
• PaO_2 = partial pressure of oxygen in arterial blood
• PCV = packed cell volume
• PT = prothrombin time
• RBC = red blood cell
• Se = selenium
• TT = thrombin time

SEE ALSO

• Anaplasmosis
• Anemia, Nonregenerative
• Babesiosis
• Bracken Fern Toxicity
• *Brassica* spp. Toxicity
• Copper Deficiency and Toxicity
• Haemonchosis
• Parasite Control Programs
• Parasitic Skin Diseases
• Rodenticide Toxicity
• Selenium Deficiency
• Sweet Clover Poisoning
• Trypanosomiasis
• Zinc Deficiency and Toxicity

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A

ANESTHESIA: INHALATION

BASICS

OVERVIEW

• Most ruminants tolerate surgical procedures with appropriate physical restraint, local or regional anesthesia, and sedation (if necessary).
• Inhalational anesthesia requires specialized equipment including: an anesthetic machine, oxygen source, oxygen regulator, oxygen flow meter, agent-specific vaporizer, breathing circuit, and a gas scavenging system.
• Ruminants <60 kg can be anesthetized with a conventional small animal machine. Ruminants 60–250 kg can be anesthetized with a human or small animal anesthetic machine with expanded carbon dioxide absorbent canisters. Ruminants >250 kg should be anesthetized with a conventional large animal anesthetic machine.
• Endotracheal intubation and proper cuff inflation are recommended.
• Inhalational anesthetics do not provide analgesia.
• Inhalant anesthetics are off-label drugs in food-producing animals. These agents are primarily eliminated through the lungs so accumulation within tissues is unlikely.

SYSTEMS AFFECTED

• Nervous
• Musculoskeletal
• Respiratory
• Cardiovascular

PATHOPHYSIOLOGY

• General anesthesia is defined as a state of controlled but reversible depression of the CNS not arousable by noxious stimuli. The sensory, motor, and autonomic functions of the body are attenuated to different degrees based on type of drugs used and the dose administered.
• Inhalant anesthetics are administered and removed primarily through the lungs.
• The mechanism of inhalant anesthetic actions is still largely unknown.

Minimum Alveolar Concentration

• Inhalant anesthetic doses are based on the calculation of MAC in healthy animals anesthetized without other drugs (Table 1).
• MAC is the minimum alveolar concentration of inhalant anesthetic that produces immobility in 50% of patients from responding to a supramaximal stimulus (electrical stimulation of oral mucus membrane). This is equivalent to the ED_{50} and corresponds to a light plane of anesthesia.
• The ED_{95} is equal to 1.2 to 1.4 × MAC_{agent} and corresponds to a moderate plane anesthesia in 95% of patients.
• There is some individual variation in MAC and inhalant dose should be titrated based on evaluation of the patient's monitored anesthetic depth and physiologic parameters.
• If adjunctive anesthetics or analgesics are used, the MAC requirements may be reduced, therefore the inhalant dose should be titrated as stated above.

Table 1

Minimum Alveolar Concentration of Inhalant Anesthetics

Species	*Isoflurane*	*Sevoflurane*	*Halothane*
Cow	1.14	—	0.76
Sheep	1.58	—	0.97
Goat	1.2–1.5	2.33	1.29–1.3

SIGNALMENT

Potentially all ruminant species

PHYSICAL EXAMINATION FINDINGS

• Palpebral reflexes disappear at minimal anesthetic depth in ruminants but corneal reflexes remain intact.
• Cattle demonstrate eye globe rotation at different depths of anesthesia. When awake, the globe is positioned between the eyelids. At induction, the globe rotates ventrally and may be partially hidden below the lower eyelid. As anesthetic depth increases, the globe can be completely hidden under the lower eyelid. At the surgical anesthetic plane, the globe rotates dorsally between the eyelids again.
• Any purposeful movement will indicate an insufficient plane of anesthesia and adjustment should be made as necessary (i.e., administer intravenous anesthetic agent, increase vaporizer setting, administer additional analgesics).

CAUSES AND RISK FACTORS

• Minor procedure or surgery like hernia repair or fracture stabilization.
• Complicated procedures like abdominal exploratory, arytenoidectomy, or bladder repair.

DIAGNOSIS

CBC/BIOCHEMISTRY/URINALYSIS

• May be included as routine preoperative workup.
• Recommended for sick patients with an ASA status of III or higher.

OTHER LABORATORY TESTING

Blood gas analysis (arterial or venous) if suspect respiratory disease or patient is not appropriately fasted.

TREATMENT

THERAPEUTIC APPROACH

Inhalational Anesthetics in Ruminants

• Often not required if adequate restraint plus local or regional anesthesia is provided.
• Fasting is recommended for all ruminants to minimize complications associated with recumbency and general anesthesia (i.e., tympany, regurgitation, aspiration pneumonia, ventilation/perfusion mismatch). The food and water fasting recommendations for calves, sheep, and goats are 12–18 hours and 8–12 hours respectively. The food and water fasting recommendations for adult cattle are 18–48 hours and 12–24 hours respectively. Neonates should not be fasted before anesthesia to avoid hypoglycemia.
• Airway protection with a cuffed endotracheal tube is essential for injectable or inhalational anesthesia. Intubation in adult cattle is usually performed with a blind or digital palpation technique whereas small ruminants and calves can be intubated with a laryngoscope with a 250–300 mm blade.
• The head and neck should be positioned to allow free flow of regurgitation from the mouth.
• Padding and positioning of the patient are critical to prevent complications associated with neuropathy, myopathy, and injuries to the eyes.
• An IV catheter is recommended for administration of analgesic drugs or supplemental injectable drug doses.
• Ruminants usually have smooth, controlled recoveries from inhalational anesthetics as they do not experience emergence delirium like their equine counterparts. Extubation should only take place when swallowing reflexes have returned.

MEDICATIONS

DRUGS OF CHOICE

Isoflurane

• The most common anesthetic agent used today.
• Isoflurane is less arrhythmogenic than halothane and is not dependent on metabolism for elimination.

Sevoflurane

• Currently widely available but not used in food animal anesthesia, often due to cost.
• Sevoflurane is less arrhythmogenic than halothane and is not dependent on metabolism for elimination.

Halothane

• Limited availability but may still be available in some areas.

• Halothane is associated with increased risk of cardiac arrhythmias in patients with high amounts of circulating cathecholamines (i.e., stressed animals, septic shock).
• A preservative, thymol, is added to the halothane to prevent degradation. Thymol can concentrate within a vaporizer over time so frequent cleaning is recommended.

CONTRAINDICATIONS
• Caution in non-fasted animals.
• Caution in patients with compromised airways, respiratory systems, or systemic hypotension.

PRECAUTIONS
Meat and milk withholding: No current published withholding times. Suggested withholding times may be obtained by contacting FARAD.

POSSIBLE INTERACTIONS
N/A

FOLLOW-UP

CLIENT EDUCATION
Discuss the risks associated with general anesthesia with clients prior to performing on the client's animal(s).

PATIENT CARE
• Ruminants tend to hypoventilate under general anesthesia and require mechanical ventilation for procedures >90 minutes or if hypercapnic and/or hypoxemic.
• Other potential complications include: bradycardia, hypotension, hypothermia, hypoxemia, and hypoventilation. Monitoring for these complications is important, especially in compromised patients, and can be assisted by use of EKG, blood pressure monitoring, thermometer, and arterial blood gas analysis when available.

MISCELLANEOUS

AGE-RELATED FACTORS
• Neonatal and geriatric animals will have exaggerated drug effects if standard drugs are used; therefore it is recommended to reduce the drug doses.
• Both have decreased respiratory and cardiac reserves so support of oxygenation, ventilation, and chronotropic or inotropic support should be expected.

PREGNANCY
• There are several physiologic alterations in pregnant animals including an increase in cardiac output, blood volume, oxygen consumption, and minute ventilation.
• In addition, gastrointestinal motility and esophageal sphincter tone can decrease as well as functional residual capacity.
• Therefore the anesthetist should be prepared to intubate with a cuffed endotracheal tube, ventilate, and support the pregnant animal.

ABBREVIATIONS
• ASA = American Society of Anesthesiologists
• CNS = central nervous system
• ED50 = effective dose 50
• ED95 = effective dose 95
• FARAD = Food Animal Residue Avoidance Databank
• IV = intravenous
• MAC = minimum alveolar concentration

SEE ALSO
• Alternative Medicine (see www.fiveminutevet.com/ruminant)
• Anesthesia: Injectable
• Anesthesia: Local and Regional Analgesia
• Pain Management (see www.fiveminutevet.com/ruminant)

Suggested Reading

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A

ANESTHESIA: INJECTABLE

BASICS

OVERVIEW
• General anesthesia produces unconsciousness, analgesia, and muscle relaxation for surgical or diagnostic procedures.
• To prevent complications associated with regurgitation, it is important to protect anesthetized ruminants' airways by placement of a cuffed endotracheal tube and positioning the head to allow regurgitant to flow out of oral cavity during lateral or dorsal recumbency.
• Injectable anesthetics may be administered via intravenous (IV) or intramuscular (IM) injection.
• An IV catheter, placed in jugular or auricular vein, is recommended for anesthesia maintained with continuous IV infusion.
• During recovery, ruminants should be placed in sternal recumbency and the endotracheal tube removed with cuff inflated when the patient regains swallowing and coughing reflexes.

INCIDENCE/PREVALENCE
N/A

GEOGRAPHIC DISTRIBUTION
Worldwide

SYSTEMS AFFECTED
Multisystemic

PATHOPHYSIOLOGY
N/A

SIGNALMENT
All ruminant species

GENETICS
N/A

CAUSES AND RISK FACTORS
N/A

DIAGNOSIS

CBC/BIOCHEMISTRY/URINALYSIS
May be indicated as preoperative workup.

TREATMENT

N/A

MEDICATIONS

DRUGS OF CHOICE

α_2 Agonists (Xylazine, Detomidine, Medetomidine, Romifidine)
• Classified as sedatives/analgesics.
• Produce profound sedation, analgesia and central muscle relaxation by binding to α_2 receptors in the CNS and spinal cord.
• Often use with an injectable anesthetic to produce general anesthesia.
• Ruminants are more sensitive to xylazine's effect and only one tenth of the dose used in horses should be used.
• Doses required for detomidine, medetomidine, and romifidine to produce sedation in ruminants are similar to the doses required for horses.
• At higher recommended doses, α_2 agonists induce analgesia, recumbency, and immobilization suitable for minor procedures.
• Order of breed variation of sensitivity to xylazine in cattle: Brahman > Hereford > Holstein; in small ruminants: goats > sheep; in camelids: llamas > alpacas.
• Recommended dose ranges of xylazine are 0.1–0.2 mg/kg IV for cattle, sheep, and goats, 0.15–0.25 mg/kg IV for llamas and alpacas.
• Recommended dose ranges of medetomidine are 0.02 mg/kg IV for cattle and 0.025–0.05 mg/kg IV for llamas and alpacas.
• A α_2 antagonist (atipamezole, tolazoline, or yohimbine) can be given to reverse an α_2 agonist's pharmacological effect.
• Recommended dose ranges of atipamezole are 0.02–0.06 mg/kg IV for cattle, 0.05–0.2 mg/kg IV for sheep and goats, and 0.125 –0.5 mg/kg IV for llamas and alpacas.
• Recommended dose ranges of tolazoline are 0.2–2 mg/kg IV for cattle and 1–2 mg/kg IV for sheep, goats, llamas, and alpacas.
• Recommended dose ranges of yohimbine are 0.12–0.2 mg/kg IV for cattle and 0.125–0.22 mg/kg IV for sheep, goats, llamas, and alpacas.

CONTRAINDICATIONS
Animals with severely compromised cardiopulmonary functions

PRECAUTIONS
• Side effects: bradycardia, hypotension, decreased respiratory rate, cardiac arrhythmias, decreased gastrointestinal (GI) motility, hypoinsulinemia, hyperglycemia, increased urine output, and oxytocin-like effect.
• Premature parturition has occurred in pregnant ruminants with xylazine during the last trimester.
• Severe hypoxemia and pulmonary edema can occur in sheep.
• In animals with urethral obstruction, rupture of bladder can result from increased urine output.
• Ruminants and camelids are more sensitive to tolazoline's toxicity effect. Low recommended dose and slow IV injection should be used.

POSSIBLE INTERACTIONS
Additive or synergistic effect with other anesthetics.

Benzodiazepines (Diazepam, Midazolam, Zolazepam)
• Primary use for their anxiolytic, anticonvulsant, and central muscle relaxing effects.
• Have little or no analgesic effect.
• Produce minimal cardiovascular depression, used in animals with high anesthetic risk.
• Use with an injectable anesthetic to produce general anesthesia and improve muscle relaxation.
• Diazepam has 40% propylene glycol as solvent in the injectable solution. May precipitate if mix with water soluble solution.
• Midazolam is water soluble with two to three times more potency than diazepam.
• Diazepam or midazolam is used in goats with urethral obstruction, when the effect of increasing urine output is contraindicated.
• Immobilization with good analgesia is produced when diazepam is combined with xylazine in cattle.
• Recommended dose ranges of diazepam are 0.1 mg/kg IV for cattle, 0.23–0.3 mg/kg IV for sheep and goats, and 0.1–0.2 mg/kg IV for llamas and alpacas.
• Recommended dose of midazolam is 0.4 mg/kg IV.

CONTRAINDICATIONS
N/A

PRECAUTIONS
Propylene glycol may cause hypotension if diazepam is administered IV rapidly.

POSSIBLE INTERACTIONS
Additive or synergistic effect with other anesthetics.

Dissociative Anesthetics (Ketamine, Tiletamine)
• Dissociative anesthesia is characterized by unconsciousness while maintaining eye reflexes (palpebral and corneal reflexes) and pharyngeal–laryngeal reflexes (swallowing reflex).
• Dissociative anesthetics cause direct CNS stimulation leading to increased sympathetic outflow and increased heart rate and arterial blood pressures.
• Respiratory pattern is characterized by an apneustic, shallow, and irregular pattern.
• Ketamine is the most popular injectable anesthetic for large animal species.
• Ketamine induces rapid onset and short duration of anesthesia and analgesia. When administered alone, muscle relaxation is inadequate for more painful surgery. Often combine with xylazine or diazepam for their central muscle relaxing effect.
• Subanesthetic doses of ketamine produce profound analgesia by blocking N-methyl-D-aspartate receptors.
• Recommended dose ranges of ketamine are 2.2–4.5 mg/kg IV for cattle, 1–5.5 mg/kg IV for sheep and goats, and 3–5 mg/kg IV for llamas and alpacas.

Table 1

Drugs Used for Injectable Anesthesia in Cattle, Small Ruminants, and Camelids.

	Dosages (mg/kg)			
Drugs	*Cattle*	*Sheep & Goats*	*Alpacas & Llamas*	*Comments*
Diazepam Xylazine	0.1, IV 0.2, IV	—	—	Immobilization with good analgesia for 30 min Total recumbency 60 min
Diazepam Ketamine	0.1, IV 4.5, IV	Mix 1 mL D (5 mg), 1 mL K (100 mg), give 1 mL/18–22 kg, IV	0.1–0.2, IV 4, IV	Anesthesia 10–15 min Total recumbency 30 min
Medetomidine Ketamine	0.02, IV 2.2, IV	0.02, IV 1–2, IV	0.025, 0.035, or 0.05, IM 1–1.5, IM	Anesthesia 70–120 min in calves Light anesthesia for 30–60 min in camelids
Midazolam Ketamine	—	0.4, IM 4, IV	—	Anesthesia 15 min
		Sheep	Alpacas	Anesthesia 30 min
Xylazine Ketamine	0.1–0.2, IV 2, IV	0.1, IV 2.2, IV	0.2, IM 3–5, IM	0.35–0.8, IM 5–8, IM	Preferred combination for adult large ruminants
		Goats	Llamas	
Xylazine Ketamine		0.1, IM 3–5, IM	0.15–0.25, IV 3–5, IV	0.25, IM 5–8, IM	
BKX Butorphanol Ketamine Xylazine	 0.0375, IM 3.75, IM 0.375, IM	1 mL/23 kg, IM; 1 mL/45 kg, IV	Alpacas: 1 mL/18 kg, IM Llamas: 1 mL/23 kg, IM	Anesthesia 20–30 min Mixture: add 1 ml LA X (100 mg), 1 mL B (10 mg) into 10 mL K (1,000 mg), give 1 mL/20 kg IM
Modified BKX Butorphanol Ketamine Xylazine	—	0.02 mL/kg, IV 0.03, IV 1.6, IV 0.03, IV	—	Mixture: add 8 mg of X, 8 mg of B into 400 mg (4 mL) of K For debudding in young animals
Bovine Triple Drip Xylazine (0.1 mg/mL) Ketamine (1–2 mg/mL) into 5% Guaifenesin	 Induction: 0.67–1.1 mL/kg Maintenance: 2.2 mL/kg/hr	 Induction: 0.67–1.1 mL/kg Maintenance: 2.2 mL/kg/hr	 Induction: 0.67–1.1 mL/kg Maintenance: 2.2 mL/kg/hr	Adjustable dose for induction & maintenance Stable plane of anesthesia Smooth recovery
Propofol	4–6, IV	Induction: 3–4, IV or 4–8, IV Maintenance: 18–40 mg/kg/hr	Induction: 2–3.5, IV Maintenance: 24 mg/kg/hr	Anesthesia 5–10 min, single dose Apnea may occur CRI: 24 mg/kg/hr for maintenance of light anesthesia
Telazol	4, IV	2–4, IV	2, IV or 4.4, IM	Anesthesia 45–60 min Smooth but prolonged recovery
Xylazine Telazol	0.1, IM 4, IM	—	0.25, IV 1, IV	Anesthesia 60 min Standing in 130 min
TKX-Ru Telazol Ketamine Xylazine	1.25–1.5 mL/125 kg, IM for small cattle 1 mL/125 kg IM for large, adult cattle	—	1.25–1.5 mL/kg, IM for smaller patients 1 mL/110–115 kg, IM for larger patients	Mixture: Add 2.5 mL (250 mg) of K and 1 mL (100 mg) of X into 500 mg of Telazol Allow 20 min to reach peak effect Effective for capture wild ruminants Deep sedation, recumbency and chemical restraint in camelids; awake and assume sternal recumbency in 40–60 min

• Telazol® is a combination of a tiletamine and zolazepam, in a 1:1 ratio by weight base.
• Telazol® comes as 500-mg powder, and is reconstituted with 5 mL sterile water into 100 mg/mL solution. Telazol® can be reconstituted with a smaller volume of sterile water with higher concentration, like adding 2.5 mL sterile water into 500-mg powder, resulting in a final concentration of 200 mg/mL.
• The pharmacologic effect of Telazol® is predominated by tiletamine. Similar to diazepam and midazolam, zolazepam produces minimal cardiovascular depression. Thus, Telazol® anesthesia is characterized by that of ketamine anesthesia, but better muscle relaxation, more profound analgesia, and longer duration of anesthesia.
• Recovery from Telazol® anesthesia tends to be smooth but prolonged in ruminant as a result of slower metabolism and elimination of zolazepam.
• Recommended dose ranges of Telazol® are 4–6 mg/kg IV for cattle, 2–4 mg/kg IV for sheep and goats, and 1–2 mg/kg IV for llamas and alpacas.

CONTRAINDICATIONS
N/A

PRECAUTIONS
• Ketamine increases salivation and mucus secretion in the respiratory tract, which can be reduced by administration of an anticholinergic, e.g., atropine.
• Prolonged recovery may follow Telazol® anesthesia.

POSSIBLE INTERACTIONS
Additive or synergistic effect with other anesthetics.

Guaifenesin (Glycerol Guaiacolate; GG)
• Administered alone, GG produces muscle relaxation, ataxia, and recumbency.
• Minimal changes on respiratory muscle activity and respiratory function at recommended doses of GG. Respiratory muscle paralysis occurs at doses 3–4 times higher than that required to induce recumbency.
• GG can be reconstituted with 5% dextrose to make up 5% or 10% injectable solution.
• Xylazine, ketamine, and GG combination ("bovine triple drip") for induction and maintenance of anesthesia (see Table 1).

CONTRAINDICATIONS
GG does not produce anesthesia or analgesia. It should always be used with an anesthetic.

PRECAUTIONS
Thrombophlebitis can occur with the administration of 5% solution. Hemolysis, hemoglobinuria, and venous thrombosis may occur with a solution of 10% or higher.

POSSIBLE INTERACTIONS
Additive or synergistic effect with anesthetics.

Propofol
• Short-acting hypnotic/anesthetic.
• Propofol is not water soluble, the injectable solution comes as a white, oil-in-water emulsion. This emulsion contains no preservative, the contents should be used or discarded within 8 hours. New formulation, PropoFlo 28, uses 20 mg/mL of benzyl alcohol as preservative, thus the 28 days of shelf life.
• A single dose of propofol induces 10 minutes of general anesthesia. Complete recovery without residual sedation and ataxia occurs within 20 minutes.
• Side effects include absence of analgesic effect at subanesthetic doses, apnea following bolus injection, and hypotension due to peripheral vasodilation.
• Recommended dose ranges of propofol are 4–6 mg/kg IV for cattle, 3–8 mg/kg IV for sheep and goats, and 2–3.5 mg/kg IV for llamas and alpacas.

CONTRAINDICATIONS
• Do not use in animals with preexisting hypotension.
• Cost is prohibitive for most large animal patients.

PRECAUTIONS
Apnea and hypotension may occur after bolus.

POSSIBLE INTERACTIONS
Additive or synergistic effect with other anesthetics.

FOLLOW-UP

POSSIBLE COMPLICATIONS
Severe CNS and cardiopulmonary depression which may result in death if overdose.

PATIENT CARE
Closely monitor vital signs: eye reflexes, heart rate, rhythm, respiratory rate and depth, and arterial blood pressures.

MISCELLANEOUS

PREGNANCY
• Premature parturition has occurred in pregnant ruminants with xylazine during the last trimester.
• Reduced anesthetic requirement in pregnant animals.

ABBREVIATIONS
• CNS = central nervous system
• GG = guaifenesin (glycerol guaiacolate)
• GI = gastrointestinal

SEE ALSO
• Anesthesia: Inhalation
• Anesthesia: Local and Regional
• Common Pharmacologic Therapies: Adult Dairy Cattle (see www.fiveminutevet.com/ruminant)
• Pain Management (see www.fiveminutevet.com/ruminant)

Suggested Reading

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Internet Resources

• AMDUCA. Animal Medicinal Drug Use Clarification Act of 1994 (AMDUCA). http://www.fda.gov/AnimalVeterinary/GuidanceComplianceEnforcement/ActsRulesRegulations/ucm085377.htm
• FARAD. Food Animal Residue Avoidance Databank, 2015. www.farad.org/amduca/amduca_law.asp

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ANESTHESIA: LOCAL AND REGIONAL

BASICS

OVERVIEW

• Local or regional anesthesia with appropriate patient restraint provides the most cost-effective method of humane anesthesia and analgesia in ruminants. Fractious animals may also require sedation.
• Local or regional analgesia causes a reversible loss of sensation to an area without loss of consciousness. Some techniques (i.e., LS epidural) can affect motor function if motor nerves are targeted.
• These techniques should not be used as the sole source of analgesia but can be used in conjunction with NSAIDs and/or systemic opioids for optimal analgesia.

SYSTEMS AFFECTED

• Nervous
• Musculoskeletal

PATHOPHYSIOLOGY

• All LA will block sodium channels and can be infiltrated near a nerve, epidural space, or in areas near planned surgical sites.
• Signs of LA toxicity include sedation, muscle twitching, convulsions, cardiopulmonary depression, coma, and death.

CAUSES AND RISK FACTORS

Variety of procedures from castration or dehorning to abdominal exploratory procedures.

DIAGNOSIS

CBC/CHEMISTRY/URINALYSIS

Routine blood work may be performed but is not commonplace for routine procedures.

TREATMENT

THERAPEUTIC APPROACH

Anesthesia for Dehorning and Castration

It is well documented that LA should be used in conjunction with NSAIDs for optimal analgesia in these procedures.

Dehorning, Bovine

There are 1 or 2 nerves to block in cattle, depending on horn size.
• *Cornual branch of zygomaticotemporal nerve*: 2.5–3.8 cm, 20G needle with 5–10 mL 2% lidocaine injected SQ ventral to the frontal ridge and halfway between the lateral canthus and the base of the horn.
• *Cutaneous branches of the second cervical nerve:* 2.5 cm, 20G needle with 5–10 mL 2% lidocaine injected SQ along the caudal aspect of the base of the horn. *Note*: this is only necessary in adult cattle with larger horns.

Dehorning, Ovine and Caprine

There are two nerves to block in sheep and goats.
• *Cornual branch of the lacrimal nerve:* 2.5 cm, 22G needle with 2–3 mL 2% lidocaine injected SQ halfway between the lateral canthus and lateral edge of the horn base.
• *Cornual branch of the infratrochlear nerve:* 2.5 cm, 22G needle with 1–2 mL 2% lidocaine inject SQ halfway between the medial canthus and the medial edge of the base of the horn.

Castration, Bovine, Ovine, Caprine

Infiltrate LA into the skin at the proposed incision site to desensitize the skin. Inject LA into the center of the testicle until it feels firm to the touch. The LA will migrate into the spermatic cord.

Anesthesia of the Eyelid and Eye

• *Auriculopalpebral block, bovine, ovine, caprine:* 2.5 cm, 22G needle with 5 mL (2 mL in ovine and caprine) 2% lidocaine injected SQ on the dorsal zygomatic arch about 5–6 cm behind the supraorbital process.
• *Retrobulbar block, bovine:* 9 cm, 18 or 20G curved needle with 5–10 mL 2% lidocaine at each of 4 sites. The needle is passed thru the eyelids at the 12, 3, 6, and 9 o'clock positions around the eye. *Ovine and caprine:* 3.8 cm, 20 or 22G needle curved is inserted as above but only at sites (6 & 12 or 3 & 9 o'clock) with 2–3 mL 2% lidocaine per site.
• *Peterson block, bovine:* 10–12 cm, 20G needle with 15–20 mL of 2% lidocaine. The needle is passed in front of the rostral border of the coronoid process of mandible but caudal to the zygomatic arch and supraorbital process. *Ovine and caprine:* 6.3 cm, 20G needle with 3–4 mL 2% lidocaine using the technique described for cattle.

Anesthesia of the Nasal Passages

• *Infraorbital block, bovine, ovine, caprine:* 3.8 cm, 18G needle with 5–10 mL 2% lidocaine is injected SQ at the infraorbital canal. The foramen is located 5 cm above the second premolar.

Anesthesia of the Flank and Paralumbar Fossa

• *Proximal paravertebral block, bovine:* The area should be prepped from the last rib to the TP of L4 about 4–5 cm from midline. A 3.8 cm, 16G needle is used as a guide for a 8.9 cm, 20G spinal needle. The cannula needle is placed thru the skin at anterior edge of TP of L1, 4–5 cm from dorsal midline. The spinal needle is passed until it contacts the L1 TP and walked off the cranial edge; as it passes thru the transverse ligament inject 6–8 mL for the ventral branch. Then withdraw the needle to 2.5 cm over the fascia and deposit an additional 6–8 mL for the dorsal branch. Continue this at the other sites. *Ovine and caprine:* Same technique with a 2.5–3.8 cm, 20G needle with 0.5–1 mL for the dorsal branch and 2–3 mL for the ventral branch with 2% lidocaine. Do not exceed 5–6 mg/kg total.
• *Distal paravertebral block, bovine:* The area of the block is clipped and prepped from T13 to L3 on the lateral aspect. A 3.5–5.5 cm, 18G needle is inserted ventral to the TP and 10 mL of LA is infused in a fan pattern. The needle is then redirected or reinserted dorsally and LA is infused caudally in a fan pattern as well. Repeat at the other sites.

Anesthesia of the Linea Alba and Paramedian Region

• *Cranial (lumbosacral) epidural block*, bovine: Not recommended as LA infiltration will cause motor blockade of the pelvic limbs preventing ability to stand. *Ovine and caprine*: 3.8 cm or 7.5 cm, 20 or 22G spinal needle is inserted on midline between L6 and S1. Confirmation of correct placement is usually done with the hanging drop technique where saline will be pulled by negative pressure into the epidural space. A dose of 0.4–0.6 mL/kg of 2% lidocaine will provide anesthesia up to the navel region.

Anesthesia of Pelvic Area

• *Caudal (sacrococcygeal or intercoccygeal) epidural, bovine*: The space is located between S5-Co1 or Co1-Co2 and is easily palpable while moving the tail up and down. You will feel this as the space between the last anchored and first movable vertebrae. A 3.8 cm, 20 or 18G needle is passed on midline at a slight cranial angle into the vertebral space. Check placement with hanging drop technique. A dose of 0.01–0.02 mL/kg of 2% lidocaine can be used. *Ovine and caprine:* The same technique as above with 2.5 or 3.8 cm, 20G needle at S4-Co1 or Co1-Co2 with an injection volume of 0.01–0.03 mL/kg. The needle is directed at a 45° angle in small ruminants.

Anesthesia of the Teats and Udder

See "Suggested Reading" for these techniques including a ring block, inverted "V" block, and teat sinus infusion blocks. A proximal or distal paravertebral block extending from T13 to L4 can be used to block a majority of the udder.

SURGICAL CONSIDERATIONS AND TECHNIQUES

The type of block used will largely depend on clinician preference, position and temperament of the animal, and length of procedure.

MEDICATIONS

DRUGS OF CHOICE

Lidocaine

• Only approved LA for use in cattle and sheep in the United States and Canada.
• The maximal effect occurs within 2–5 minutes of injection and lasts 90 minutes.

A

ANESTHESIA: LOCAL AND REGIONAL (CONTINUED)

• The addition of epinephrine (0.01 mg/mL) was found to increase the duration of activity to 304 minutes. However, epinephrine cannot be used in limb blocks or wound edges due to the potential risk of ischemia and tissue necrosis.
• The maximum dose is 6 mg/kg for ruminants.

Bupivacaine and Mepivacaine
• Off-label use
• The maximal effect occurs within 20–30 minutes and lasts 5–8 hours for bupivacaine. Mepivacaine behaves similarly to lidocaine.
• Bupivacaine exhibits the most cardiotoxic effects of all the LA and should be only used for SQ or infiltrative techniques.
• The maximum dose is 2 (bupivacaine) and 5–6 (mepivacaine) mg/kg. No withdrawal times have been established.

CONTRAINDICATIONS

• Regulatory restrictions: See "Suggested Reading" for links to the FDACVM and AMDUCA or recommendations for the use of LA in ruminants.
• Meat and milk withholding: See "Suggested Reading" for the link to FARAD and withdrawal times recommended.

FOLLOW-UP

N/A

MISCELLANEOUS

ABBREVIATIONS

• AMDUCA = Animal Medical Drug Use Classification Act
• FARAD = Food Animal Residue Avoidance Databank
• FDACVM = Food and Drug Administration Center for Veterinary Medicine
• LA = local anesthetics
• LS = lumbosacral
• NSAIDs = nonsteroidal anti-inflammatory drugs
• SQ = subcutaneous
• TP = transverse process

SEE ALSO

• Alternative Medicine (see www.fiveminutevet.com/ruminant)
• Anesthesia: Inhalation
• Anesthesia: Injectable
• Castration/Vasectomy: Bovine
• Castration/Vasectomy: Camelids
• Castration/Vasectomy: Small Ruminants
• Dehorning
• Enucleation/Exenteration (see www.fiveminutevet.com/ruminant)
• Pain Management (see www.fiveminutevet.com/ruminant)

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• FARAD. Food Animal Residue Avoidance Databank, 2015. www.farad.org/amduca/amduca_law.asp

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ANESTRUS

BASICS

OVERVIEW

• Anestrus is the absence of estrus behaviors in female animals.
• Anestrus is physiological before puberty, between estrus periods, for a variable time after parturition or during lactation, during pregnancy, and seasonally in sheep and goats.
• Pathologic anestrus is the absence of estrus during a time when it would normally be expected to occur.
• Pathologic anestrus results from a disruption of the reproductive axis and typically involves the absence of both the behavioral signs of estrus and the underlying normal ovarian events associated with cyclic activity, although follicular waves may still occur.
• The most common clinical manifestation of anestrus is a delayed return to ovular cycles after parturition. Anestrus is also used to describe animals undergoing normal ovular cycles where the signs of estrus are missed, due either to deficiencies in management (unobserved estrus), subtle or absent signs of estrus (silent heat/subestrus), or both. The intensity and duration of estrus behavior may also be reduced or absent altogether at the first postpartum, seasonal or postpubertal ovulation in ruminants.
• Animals experiencing pathologic anestrus are usually anovular though some cases are associated with prolonged luteal function (CL retention).
• Follicular growth may be arrested at any stage of development as reflected in the size of structures found on the ovaries. Growth and persistence of follicular structures beyond the normal ovulatory size and duration is defined as OCD.

INCIDENCE/PREVALENCE

• The incidence of anestrus varies widely with the specific condition on the farm and the species affected.
• In dairy cows, for example, the incidence of delayed return to estrus after parturition in four recent reports ranged from 29 to 54% in primiparous cows and from 15 to 32% in multiparous cows.

GEOGRAPHIC DISTRIBUTION

Worldwide

SYSTEMS AFFECTED

Reproductive

PATHOPHYSIOLOGY

• Pathologic anestrus may be the result of a primary disruption or disease at the level of the hypothalamus, pituitary, or ovary.
• Commonly, anestrus is secondary to disease or derangement in some other body system. Examples include nutritional deficiencies, especially energy; chronic severe illness, pain or stress resulting in a loss of body condition; heat stress; and uterine disease with or without retention of a CL.
• The endocrine basis for profound anestrus associated with very small follicles is unclear. Anestrus associated with follicular growth to the point of deviation and beyond appears to be related to inadequate LH pulses for follicular development and the absence of an LH surge sufficient to cause ovulation.
• Anestrus occurring in association with prolonged luteal function is likely the result of inadequate uterine $PGF2_{\alpha}$ production, secretion, or transport to the ovary.
• Management deficiencies related to estrus detection can lead to a mistaken diagnosis of anestrus in otherwise normally cycling animals.
• In high-producing dairy cattle, increased liver metabolic rates related to high feed intake result in lower circulating levels of gonadal steroids with a subsequent depression of the length and intensity of estrus.
• In heat-stressed cattle, follicular steroidogenic capacity is reduced leading to lower blood estradiol concentration and diminished or absent signs of estrus.
• On rare occasion, anestrus in cattle may be iatrogenic due to inadvertent feeding of a progestational agent (e.g., MGA).

HISTORICAL FINDINGS

• Individual animals: Absence of cyclicity (estrus) beyond 60 days postpartum. Absence of estrus after a negative pregnancy diagnosis.
• Herd or flock: Reduced mating activity, decreased pregnancy rates, increased interval from calving to first insemination, poor heat detection.

SIGNALMENT

• Sexually mature (postpubertal) female animals.
• Specific conditions vary with species and breed depending on nutrition, milk production levels, reproductive management systems, suckling intensity, seasonal (photoperiodic) influences, and the occurrence of systemic or uterine diseases.
• Suckled beef cattle will have a postpartum anestrus period 2 to 3 times as long as beef or dairy cattle that are milked.

PHYSICAL EXAMINATION FINDINGS

• Absence of sexually receptive behavior.
• Poor body condition score or other condition preventing expression of estrus.

GENETICS

• Ovarian agenesis, ovarian hypoplasia, and premature ovarian failure are rare examples of profound anestrus conditions and all probably have a genetic basis.
• The role of inheritance in the more common types of anestrus is uncertain.

CAUSES AND RISK FACTORS

• Poor nutritional management, especially in the transition period.
• High milk production and increased feed intake.
• Heat stress.
• Systemic disease.
• Lameness.
• Loss of body condition.
• Postpartum uterine infections (see chapter, Endometritis) can delay the resumption of normal cycles and increase the incidence of OCD.
• Long dry periods.
• Twin calving/Freemartinism.
• Primary uterine or fetal disease (e.g., pyometra, hydrometra [goats], mummified fetus, prolonged gestation).
• Early postpartum ovulation (<25 days) is associated with a persistent or retained CL.
• Rarely, ovarian tumors, segmental aplasia, ovarian hypoplasia, mycotoxins, and micronutrient deficiencies.

DIAGNOSIS

DIFFERENTIAL DIAGNOSES

• Physiological anestrus (pregnancy, sexual immaturity, lactation, or seasonal).
• Ruling out pregnancy should be the first consideration, otherwise diagnostic and therapeutic procedures could potentially result in iatrogenic abortion.
• Freemartinism.
• Inadequate estrus detection/reproductive management deficiencies.

CBC/BIOCHEMISTRY/URINALYSIS

N/A

OTHER LABORATORY TESTS

Consistently low progesterone concentrations in serum or milk may confirm anovular (lacking ovulation) anestrus.

IMAGING

• Transrectal ultrasonography reveals persistently small inactive ovaries or failure to ovulate when larger follicles or cystic structures are present.
• Repeated examination may be necessary to confirm the diagnosis. A CL will be absent in most cases but, if present, the uterus should be carefully examined for evidence of pyometra or other uterine disease.

OTHER DIAGNOSTIC PROCEDURES

• Records analysis/history.
• Transrectal ultrasonography or palpation per rectum in cattle.
• Serum progesterone analysis.
• Observation for sexual activity as appropriate for each species.

PATHOLOGIC FINDINGS

N/A

A

ANESTRUS (CONTINUED)

TREATMENT

THERAPEUTIC APPROACH
• Correct nutritional deficiencies or other primary problems (e.g., lameness).
• Hormonal induction of estrus and/or ovulation.
• Heat abatement.

SURGICAL CONSIDERATION AND TECHNIQUE
Ovariectomy in case of GTCT.

MEDICATIONS

DRUGS OF CHOICE
• GnRH or hCG
• PG600 (combination of FSH and hCG) in sheep and goats
• $PGF2_{\alpha}$ for retained/persistent CL, pyometra and hydrometra
• Ovulation synchronization (Ovsynch®) protocol with the addition of a CIDR
• Ovulation presynchronization protocols that include an injection of GnRH before the breeding Ovsynch®.

CONTRAINDICATIONS
$PGF2_{\alpha}$ should not be given to pregnant animals.

PRECAUTIONS
N/A

POSSIBLE INTERACTIONS
N/A

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
• Estrous cycles will resume once nutritional, reproductive, and management issues are addressed.
• Delay in initiating treatment increases the risk of culling for reproductive reasons.

POSSIBLE COMPLICATIONS
• Pyometra
• Delayed conception

CLIENT EDUCATION
Use appropriate methods for estrus detection and monitoring of breeding activity.

PATIENT CARE
Monitor body condition score and behavioral activity.

PREVENTION
• Sound nutritional management, especially in the transition period, to avoid excessive loss of body condition.
• Minimize the incidence of dystocia, retained fetal membranes, and metritis/endometritis.
• Heat abatement.
• Decreased dry period length for older cows.

MISCELLANEOUS

ASSOCIATED CONDITIONS
• Pyometra
• Endometritis
• Twinning
• Lameness
• Starvation
• Heat stress

AGE-RELATED FACTORS
In cattle, prolonged postpartum anestrus is more common in primiparous than in multiparous cows.

ZOONOTIC POTENTIAL
N/A

PREGNANCY
• Pregnant animals experience a normal, physiological anestrus.
• $PGF2_{\alpha}$ will invariably (goats and camelids) or frequently (sheep and cattle) cause abortion.

BIOSECURITY
N/A

PRODUCTION MANAGEMENT
• Persistently anestrus females in well-monitored operations with strong management and husbandry protocols should be considered for culling.

ABBREVIATIONS
• CIDR = controlled intravaginal drug release device for delivery of progesterone
• CL = corpus luteum
• FSH = follicle stimulating hormone
• GnRH = gonadotropin-releasing hormone
• GTCT = granulosa-theca cell tumor
• hCG = human chorionic gonadotropin
• LH = luteinizing hormone
• MGA = melengestrol acetate
• OCD = ovarian cystic degeneration
• $PGF2_{\alpha}$ = prostaglandin $F2_{\alpha}$

SEE ALSO
• Artificial Insemination: Bovine
• Artificial Insemination: Small Ruminant
• Body Condition Scoring (see www.fiveminutevet.com/ruminant)
• Endometritis
• Estrus Synchronization: Bovine
• Estrus Synchronization: Small Ruminants
• Freemartinism
• Heat Stress
• Ovarian Cystic Degeneration
• Ovarian Hypoplasia, Bursal Disease, Salpingitis
• Pyometra
• Uterine Anomalies

Suggested Reading

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ANGULAR LIMB DEFORMITY

BASICS

OVERVIEW

• Angular limb deformity (ALD; "bent-leg") is a deviation from the normal axis of a limb (in the frontal plane) and is defined by the joint involved and the direction that the distal aspect of the limb is deviated.
  ◦ **Valgus** deformity: The limb distal to the lesion deviates *laterally.*
  ◦ **Varus** deformity: The limb distal to the lesion deviates *medially.*
  ◦ ALDs are further described by the location of the pivot point (axis of deviation) and by the location of the site of defective growth.
• Some ALDs are caused by asymmetrical lesions involving an active growth plate (e.g., distal radius), but growth plate damage is not always the underlying cause.
• Related conditions include flexural deformities, tendon injuries, joint luxations/joint instability caused by laxity of supporting structures, and rotational/torsional deformities.
• Hereditary chondrodysplasia (HC), or spider lamb syndrome, is a hereditary condition in young lambs characterized by a number of skeletal deformities, including angular limb deformities.

INCIDENCE/PREVALENCE

• Valgus and varus ALDs are common and well documented in horses, but are relatively rare in ruminants. • Congenital limb abnormalities are reported to account for 6.9% of all congenital abnormalities in cattle.

GEOGRAPHIC DISTRIBUTION

Worldwide.

SYSTEMS AFFECTED

Musculoskeletal

PATHOPHYSIOLOGY

• ALDs are considered multifactorial in origin and have congenital/perinatal and developmental predisposing factors.
• Congenital ALDs may arise from environmental factors, genetic factors, or both. These include: toxins, placentitis, laxity of periarticular soft tissues, and intrauterine or perinatal physical factors (e.g., twinning, trauma). • Contributions to the formation of ALDs in immature and mature animals can stem from a low plane of nutrition, trauma, and excessive limb loading. • In llamas, the distal ulnar epiphysis fuses with the distal radial epiphysis. This unique development of the distal portion of the ulna is associated with forelimb valgus deformities in crias. The ulnar epiphysis extends distally, crosses the radial physis, and fuses with the radial epiphysis. This early fusion demands synchronous growth to ensure normal limb development. • Most calves have a mild carpal valgus deformity of approximately 7 degrees, which does not require treatment. Varus deformities in cattle are abnormal and often need treatment.

HISTORICAL FINDINGS

A complete history including current age, birthing details, age at which the deformity was noticed, course and progression of the deformity, and diets of affected animal and dam should be obtained.

SIGNALMENT

Species

• Bovine, ovine, caprine, South American camelids (especially llama crias), cervids—including fallow deer (*Dama dama*), red deer (*Cervus elaphus*), white-tailed deer (*Odocoileus virginianus*)—and a single case report of ALD in a giraffe calf (*Giraffa camelopardalis*).

Breed Predilections

• ALDs have been described in many different beef and dairy cattle breeds. • HC primarily affects black-faced breeds of sheep (Suffolk, Hampshire, Southdown, Shropshire, and Oxford). • Varus/valgus deformities have been described in the distal radial physis of yearling farmed male red and wapiti-red crossbred (elk) deer in New Zealand.

Mean Age and Range

• ALDs primarily affect young growing animals up to 7 months of age, but can be seen in older animals (e.g., trauma-induced ALD). • HC has two distinct clinical entities: lambs are either grossly abnormal at birth or develop the abnormal conformation at 4–6 weeks of age. Radiographic changes at birth are similar for both.

Predominant Sex

No apparent sex predisposition.

PHYSICAL EXAMINATION FINDINGS

• Conformation should be assessed first by having the animal stand in a symmetrical manner on a firm, flat surface and observing it from multiple angles. Affected animals may appear to be knock-kneed or bowlegged. All limbs should be palpated and affected limbs should be manually manipulated. Clinical signs such as abnormal bending of the affected limb, increased laxity, muscle atrophy, swelling, heat, pain on manual pressure, abrasions on lateral or medial side of hoof wall, presence of orthopedic injury, and abnormal gait and locomotion are indicative of ALD. • Compensatory deviation (opposite that of the affected limb) is relatively common in the contralateral limb. • If varus deformity is found unilaterally, the contralateral limb should be examined for a significant orthopedic injury as a cause of excessive weight bearing in the deformed limb/joint.
• Since cattle are considered to have a "normal" degree of medial deviation at the level of the carpus and hock, as well as normal external rotation of the lower limb, ALDs tend to be missed in the early stages of development. • Most bovine ALDs occur at the level of the mid-diaphysis of long bones.
• In sheep affected with HC, various degrees of ALDs of fore- and/or hindlimbs will be noted. Other physical examination findings include severe scoliosis/kyphosis of the thoracic spine, pectus excavatum, retarded growth rates, facial deformities such as angular deviation and/or shortening of maxilla, rounding of the dorsal silhouette, and Roman-shaped noses.

GENETICS

• The questions of heritability in ALDs have not been definitively answered; the details of some syndromes are known. • ALDs in Jersey calves are genetically transmitted as a simple autosomal recessive trait. • HC of Suffolk and Suffolk-cross sheep is inherited as a single, autosomal recessive gene that has been localized to the distal end of chromosome 6. A defect in the gene encoding fibroblast growth factor receptor 3 (FGFR3) is suspected. DNA tests (blood or semen) are available to identify homozygous and heterozygous animals.

CAUSES AND RISK FACTORS

• ALDs are often related to asymmetrical growth of the physis, ligament rupture, or orthopedic injuries.

Congenital Predisposing Factors

• Incomplete cuboidal bone ossification (carpal and/or tarsal). • Physiologic immaturity at birth. • Uterine malpositioning. • In utero bending stress and bone remodeling early in gestation. • Twin (or triplet) pregnancy. • Reduced intrauterine space may limit fetal movement and subsequently cause congenital angular and/or flexural limb deformities. • Laxity of periarticular supporting structures.
• Disproportionate osseous growth of medial and lateral aspects of long bones (e.g., distal radius, tibia, metatarsus). • Nutritional imbalance during gestation. • Genetic causes.

Developmental Predisposing Factors

• Conformational defects (causing abnormal weight distribution across a joint).
• Nutritional disorders (e.g., improper dietary calcium and phosphorus ratios; copper, zinc, manganese, iron, and molybdenum concentrations). • External trauma (e.g., compression of or trauma to growth plate; Salter-Harris fractures, malunion of fractures, inflammation of growth plate). • Iatrogenic (e.g., assisted delivery). • Excessive exercise.
• Hematogenous osteomyelitis involving the physeal region. • Rapid weight gain in heavy breeds (high energy rations and rapid growth).
• Often, no specific cause is identified.

Camelids

• May see ALDs (usually carpal valgus) in growing camelids with hypophosphatemic rickets syndrome. • Ill-thrift syndrome in llamas may be associated with ALDs as well as anemia, low serum iron concentrations, and metabolic disorders (hypothyroidism). Underlying cause not established.

ANGULAR LIMB DEFORMITY (CONTINUED)

DIAGNOSIS

DIFFERENTIAL DIAGNOSES

• Physiologic deformities. • Metabolic bone disease (e.g., rickets). • HC may be confused with arthrogryposis-hydranencephaly syndrome (AHS) in lambs, in which there is characteristic hyperflexion of forelimbs, cranial overextension of hindlimbs, with a corkscrew deviation of the spine. In lambs with AHS, severe deformities result from primary abnormalities of the CNS (including hydranencephaly and others) and not of the skeleton.

Camelids

True ALDs in llamas must be differentiated from valgus deformities of the forelimbs in newborn crias that self-correct without surgical treatment.

CBC/BIOCHEMISTRY/URINALYSIS

• There are usually no associated laboratory abnormalities with ALDs. • HC results in slightly elevated serum alkaline phosphatase activity; insufficient for diagnostic purposes.

Camelids

• Ill-thrift syndrome: Serum vitamin D (25-hydroxycholecalciferol) concentrations are often diagnostic. • May also see hypothyroidism, anemia, erythrocyte dyscrasias, hypophosphatemia, and low serum iron concentrations.

OTHER LABORATORY TESTS

N/A

IMAGING

• Radiographs are critical in diagnosing ALDs—at least two views, 90 degrees apart, should be taken of the affected joint, including joints immediately proximal and distal to the affected joint. • The dorsopalmar/dorsoplantar (DP) view is needed for examination of the anatomical location of the deformity and for measurements. The pivot point is defined as the intersection between lines drawn through the long axis at the center of the proximal and distal long bone using the dorsopalmar view. Location of the pivot point identifies the type of deformity. Measure the angle of deviation with a protractor. • HC: Most consistent lesions include multiple islands of ossification of the anconeal process and malformed, displaced sternebrae. The anconeal lesions of HC are progressive, whereas similar lesions in other skeletal conditions of lambs regress.

OTHER DIAGNOSTIC PROCEDURES

• Bacterial cultures may be indicated in cases of septicemia, arthritis, or osteomyelitis. • Toxicology (heavy metal and mineral analysis) and feed analysis may assist in diagnosis.

PATHOLOGIC FINDINGS

• Histology: Focal or segmental thickening of the physis (expansion of the hypertrophic zone) with extension into the proximal metaphysis; closely resembles physeal manifestations of osteochondrosis. • Histology associated with HC (vertebrae and long bones): Increase in width of the zone of proliferation and hypertrophy and unevenness of growth cartilage; failure to form or maintain orderly columns of chondrocytes.

TREATMENT

THERAPEUTIC APPROACH

• Treatment of neonatal animals with incomplete ossification involves the application of tube casts or splints to the affected limb(s) until ossification is complete (based on repeated radiographs). • Many cases of ALD will resolve without surgery if the underlying cause(s) can be identified and addressed and if the animal does not damage the affected physis or joints with vigorous exercise. • Specific treatment methods are selected on the basis of age, degree of angulation, remaining growth potential of the involved physis, and experience of the veterinarian. • Minor limb deviations may be conservatively treated by manual alignment and external support of the limb (e.g., rigid splinting, bandaging, or casting/tube casts) and/or hoof (claw) trimming. • Hoof manipulations create growth plate response to stress applied opposite the deformity, and self-correction occurs. The hoof tends to turn in the direction of the longer claw or toward the side of the wider wall, resulting in straightening and de-rotation of the limb. • Medial (varus) deformities can be treated by trimming the medial claw shorter than the lateral claw and by placing an acrylic (methyl methacrylate) wing on the weight-bearing surface of the lateral claw (to increase lateral contact with the ground). • Treatment should be directed at the orthopedic injury when varus deformity is present secondary to a contralateral limb injury.

SURGICAL CONSIDERATIONS AND TECHNIQUES

• Surgery is recommended for older animals (near the end of active physeal growth), for those that do not respond to conservative treatments, and for animals with bone malformations that require realignment (via osteotomy). • The choice of surgical technique should take into consideration the economic value and age of the animal, severity of the deformity, and the joint involved.

Treatment Strategies Include the Following:

Growth Acceleration (Periosteal Stripping/ Elevation)

In young calves and lambs with early cases of ALD, surgical growth stimulation via periosteal stripping on the concave aspect (shorter side) of the deformity has been used successfully. Based on the remaining growth potential in the physis, allow the animal to correct the deviation by physeal growth.

Growth Retardation (Transphyseal Bridging)

• Transphyseal bridging is indicated for severe cases of ALD or in animals past the rapid growth phase of the radius and ulna (often recommended for animals older than 5 months of age). • By creating a temporary transphyseal bridge on the convex side of the deformity using staples or screws and wires, limb growth is slowed by restricting growth and allows the other side to continue growing, resulting in limb straightening. • The surgical implants must be removed when the limb achieves normal conformation to prevent overcorrection. This can be used in combination with periosteal stripping to increase the likelihood of full correction in animals with severe deviations.

Corrective Wedge Osteotomy

• This procedure is indicated in mature animals with ALD and in neonates with congenital fracture malunion. If the growth plates are closed or if the growth plate is not involved in an ALD, a corrective osteotomy is recommended. This requires more experience and equipment and often is reserved for valuable animals when response to other therapies has failed. • The site and orientation of the wedge should be determined by clinical and radiographic examination. • The limb needs to be stabilized by internal fixation with a plate and screws for an extended postoperative period.

Camelids

An ulnar osteotomy must be done in conjunction with the periosteal transection because the ulna spans the radial physis.

MEDICATIONS

DRUGS OF CHOICE

Nonsteroidal anti-inflammatory agents (NSAIDs) are recommended to reduce inflammation in some cases of ALD.

CONTRAINDICATIONS

Prolonged NSAID administration has been associated with gastrointestinal (abomasal or C3) ulcers.

PRECAUTIONS

• Many of these affected animals are young; precautions regarding drug choices must take age into consideration. • Appropriate milk

and meat withdrawal times must be followed for all compounds administered to food-producing animals.

POSSIBLE INTERACTIONS
N/A

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
• Prognosis is guarded, yet reasonable for ALDs associated with growth plate imbalances, such as most valgus deformities. • The prognosis for ALDs secondary to contralateral orthopedic injury (such as most varus deformities) is generally poor because it is usually centered over a joint and is dependent on the prognosis of the primary orthopedic injury. • HC-affected individuals rarely survive the neonatal period. • Camelids: The prognosis for ill-thrift syndrome in llamas is poor; etiologic agent often not identified.

POSSIBLE COMPLICATIONS
• In cases of valgus deformity, efforts should be taken to prevent compensatory varus in the contralateral limb. Persistence of hoof distortion is possible if the angular deformity is not corrected. • Surgical: transphyseal bridging—overcorrection of the physis is possible. Owner cooperation is required to determine when limb has regained its normal conformation. • Possible ALD surgical complications include muscle, tendon, and ligament atrophy/laxity, hyperextension of limbs, fibrous scar tissue development, and postoperative infection.

CLIENT EDUCATION
• Examination and intervention of young animals with congenital ALDs should be done as early as possible. • Due to the possible hereditary link with ALDs, breeding of affected animals is not recommended.

PATIENT CARE
• Severely affected animals may be unable to rise to nurse and require additional supportive care. Protect limbs with thick, soft bandages that fit well. • Care should be taken to maintain a soft (padding), clean, and dry environment and to minimize decubital/pressure sores, open arthritis, muscle atrophy, umbilical infections, and septicemia. • Physical confinement (stall) is recommended for the management of ALDs to limit stresses placed on affected limbs.

• Nutritional imbalances have been implicated, such as ill-thrift in llamas. Treatment for this condition includes appropriate vitamin D supplementation. • Other dietary imbalances should be corrected while treating cases of ALD. • Frequent physical monitoring and repeated radiographs should be done to assess the efficacy of corrective measures and to monitor progress. • In cases of transphyseal bridging surgery, owner cooperation is required to determine when the limb has regained its normal conformation, at which time the implants must be removed to prevent overcorrection.

PREVENTION
Avoid breeding affected animals.

MISCELLANEOUS

ASSOCIATED CONDITIONS
• Conditions associated with ALDs include osteochondrosis of the physis, epiphysitis, and incomplete ossification of the cuboidal carpal bones. • Hyena disease (premature physeal closure) has been reported in calves due to overdose of vitamins A, D3, and E. • Congenital lethal chondrodysplasia in Australian Dexter cattle = "Dexter bulldog calves". • Congenital chondrodysplastic dwarfism in Holstein calves. • Complex vertebral malformation is a familial syndrome of Holstein calves. • Syndrome known as "bentleg" or "bowie" associated with ingestion of *Trachymene glaucifolia* (wild parsnip) by pregnant ewes in Australia and New Zealand.

AGE-RELATED FACTORS
The majority of ALDs occur during the active growth phase of the affected bone/joint.

ZOONOTIC POTENTIAL
N/A

PREGNANCY
• Many of the causes of ALDs are congenital diseases in which the in utero environment is somehow disturbed (hormones, vascular supply, teratogens, mechanical factors, or prenatal viral infections). • Cases of ALDs in goats pregnant with triplets have been reported. • Contributing factors likely include stress and in utero malpositioning.

BIOSECURITY
N/A

PRODUCTION MANAGEMENT
N/A

ABBREVIATIONS
• AHS = arthrogryposis-hydranencephaly syndrome
• ALD = angular limb deformity
• CNS = central nervous system
• DP = dorsopalmar/dorsoplantar
• FGFR3 = fibroblast growth factor receptor 3
• HC = ovine hereditary chondrodysplasia
• NSAIDs = nonsteroidal anti-inflammatory drugs

SEE ALSO
• Arthrogryposis
• Congenital Defects: Bovine
• Hereditary Chondrodysplasia: Ovine
• Lameness (by species)

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A

ANTHELMINTIC RESISTANCE

BASICS

OVERVIEW
Gastrointestinal nematodes are common and may cause pathology that impacts health and welfare of infected animals. Infestation with these nematodes decreases production parameters. Control measures have relied predominantly on periodic administration of anthelmintic drugs to the entire herd or flock. Their continual use has led to the selection of populations of drug-resistant worms worldwide. Multi-drug resistance has now been reported in sheep and cattle. Anthelmintic resistance by *Haemonchus contortus* in sheep has been well documented and in cattle recent reports of resistance in *Cooperia* spp. and to a lesser extent in *Ostertagia ostertagi* are now documented.

INCIDENCE/PREVALENCE
Each farm may have different levels of resistant parasite populations. The level of resistance is dependent on how frequently the herd has been treated with anthelmintics and how effective those compounds have been in reducing luminal burdens.

GEOGRAPHIC DISTRIBUTION
Worldwide

SYSTEMS AFFECTED
Gastrointestinal, which may lead to pathology of other organ systems with significant infections.

PATHOPHYSIOLOGY
Individual nematodes that survive an anthelmintic treatment have a reproductive advantage in the absence of competition by susceptible worms in the intestine. This advantage persists until lifecycle features prevail or anthelmintic levels decrease and allow reestablishment of susceptible parasites. Resistant worms transmit their unique, heritable traits to the next generation and by doing so they increase the frequency of their genetic alleles in the general population. Depending upon the parasite, the clinical significance of the infection may be greater than would be present if the treatment was more effective. Resistant worms have no particular advantage until the selection pressure of anthelmintic treatment is applied. Once this happens, a great way to accentuate this advantage is to repeatedly use the same anthelmintic. Individual worms are initially resistant to only one class of anthelmintics, so when "drug A" is used, they survive, but if "drug B" were introduced, the worms would be removed like the rest of the susceptible population. Thus, the reproductive advantage of resistant worms would be favored if "drug A" were used exclusively.

HISTORICAL FINDINGS
Lack of efficacy of anthelmintic treatment evidenced by poor fecal egg count reductions and increased clinical signs of parasitism.

SIGNALMENT
All ruminants may be affected.

PHYSICAL EXAMINATION FINDINGS
The signs of parasitism may be variable depending upon the pathogenicity of the resistant parasites.

GENETICS
All ruminants may be affected, but it is most common to find that 20% of a herd will harbor 80% of the parasites. This fact suggests that a genetic component exists within a population of animals that allows the majority of the herd to have some level of resistance to internal parasites. The issue then becomes identifying those individuals within the herd that are most susceptible to continued infection and providing them with effective treatment.

CAUSES AND RISK FACTORS
Anthelmintic resistance in a nematode population is a phenotypic manifestation of a heritable, genetic trait within that population. The genetic basis and modes of inheritance of resistance are quite complex and differ widely among the various classes of compounds, but positive selection occurs whenever worms carrying resistant alleles are exposed to an anthelmintic to which they have lost their susceptibility.

DIAGNOSIS

DIFFERENTIAL DIAGNOSES
Any cause of weight loss may be considered as a differential for chronic parasitism. In sheep with *Haemonchus contortus* infections anemia and hypoproteinemia are predominate signs.

OTHER LABORATORY TESTS
The standard by which parasite loads are measured is the assessment of fecal egg counts per gram of feces (EPG). Fecal EPG of cattle tend to be less reflective of the adult worm burden when compared to sheep. This fact leads to a great deal of uncertainty in the detection of anthelmintic resistance in cattle. Resistance can be defined as a measurable decrease in efficacy of a compound against parasitic species and its larval stages that were previously susceptible.

OTHER DIAGNOSTIC PROCEDURES
The use of fecal EPGs on a herd basis is important to obtain knowledge of the efficacy of any anthelmintic. It is important to obtain an adequate sample size, although the number of animals with an adequate infection for enrollment can be limited. Recommendations to minimize diagnostic uncertainty include the use of arithmetic means over geometric means to calculate anthelmintic efficacy, the use of individual based group means with pre- and post-treatment individual fecal egg counts, and the preferred use of diagnostic methods with higher analytic sensitivity to minimize inaccuracies in populations with low baseline fecal egg count.

PATHOLOGIC FINDINGS
The pathology associated with internal parasites is variable and dependent on the genus.

TREATMENT

THERAPEUTIC APPROACH
The key to parasite control is either to prevent egg shedding and/or prevent larval uptake. For the last 50 years we have tried to prevent egg shedding by routine anthelmintic treatments. Managing animals to prevent larval uptake can be accomplished by co-grazing different species, removal of feces or clever use of available pasture lands. The assessment of the drug disposition in the host and increased knowledge of the mechanisms of drug interaction in the targeted parasites has increased our understanding of how these anthelmintics actually work. It is clear that we need additional scientific knowledge on how to improve the use of available molecules to avoid or delay development of resistance.

MEDICATIONS

- See "Follow-Up"
- See chapters, Parasite Control Programs

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
This is a life-threatening aspect of small ruminant production and serious consequences will occur if solutions are not forthcoming.

POSSIBLE COMPLICATIONS
Very limited information is available on the potential additive or synergistic effects occurring after co-administration of two (or more) drugs with different modes of action.

CLIENT EDUCATION
A change in treatment philosophy is necessary to combat or delay the development of anthelmintic resistance in ruminant nematodes. Producers have used dewormers in a very cavalier manner over the past 40

(CONTINUED) **ANTHELMINTIC RESISTANCE** A

years and been taught that when deworming, all animals in the herd must be treated. New knowledge and evidence point out that this technique has helped to create the resistance that is now present. Educating producers on how to address parasite problems must be done and this knowledge needs to encompass feed stores and pharmaceutical supply houses as well.

PATIENT CARE

The individual monitoring within a herd will be necessary to allow for identification of those that have the highest parasite loads.

PREVENTION

- Integrated management and pharmacologic intervention.
- Treatment targeted at those individuals that harbor the most parasites and are at risk for the development of adverse clinical signs.
- Different pharmacokinetic-based approaches to enhance parasite exposure.
- Mixed-class anthelmintic treatment.

MISCELLANEOUS

PREGNANCY

Chronic parasitism may significantly affect gestating dams, especially in small ruminants infected with *H. contortus.*

BIOSECURITY

Anytime a new animal is brought on to a property they should be evaluated for their internal parasite load. This is especially critical in small ruminant populations with *Haemonchus contortus*. Purchased additions should be screened and quarantined to evaluate their existing infestation and, if they are infected, their response to treatment.

PRODUCTION MANAGEMENT

Production is imperative to success in ruminants and internal parasites are a major contributor to production losses. Control of these parasites is critical for success in rearing young stock.

ABBREVIATION

- EPG = egg count per gram of feces

SEE ALSO

- Parasite Control Programs: Beef
- Parasite Control Programs: Camelid
- Parasite Control Programs: Dairy
- Parasite Control Programs: Small Ruminant

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Client Education Handout available online

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ANTHRAX

BASICS

OVERVIEW

• Anthrax is a bacterial disease that many animals are susceptible to including humans.
• In ruminants it causes acute bacteremia, septicemia, and usually death. It is caused by *Bacillus anthracis* which is present in the soil.
• It is seen most often in and is highly pathogenic for most wild and domestic herbivores.
• In most areas anthrax is a reportable disease and the regional veterinary authorities should be notified of an outbreak.
• In the USA, *Bacillus anthracis* is listed in the Federal Select Agent Program.

INCIDENCE/PREVALENCE

• Anthrax spores are endemic in parts of the United States, where occurrence of clinical disease is usually sporadic.
• Both sporadic cases and outbreaks are often associated with disruption of the soil.
• Morbidity varies widely but case mortality is 90–95%.

GEOGRAPHIC DISTRIBUTION

• Anthrax is found worldwide but certain regions have higher occurrence of disease.
• Anthrax spores favor areas with neutral to alkaline soils with high levels of calcium and manganese and are organically rich.

SYSTEMS AFFECTED

• Hemolymphatic
• Multisystemic

PATHOPHYSIOLOGY

• Soil-borne spores are the infective form of the bacteria.
• In ruminants the main source of infection is consuming spore-contaminated pasture or feed. However, spores can enter the host via the cutaneous or respiratory systems also.
• Once in the host, the spores germinate and produce the vegetative form of the bacterium which rapidly proliferate.
• These rapidly dividing bacteria produce capsule and toxins. Early in the infection these toxins help the bacteria to evade the immune system resulting in systemic infection. Later in the infection with large numbers of bacteria in the blood, the toxins enter cells of other systems resulting in vascular shock and death.
• Late in the course of the disease and at death all animal excretions contain large numbers of bacteria and sporulate when contacting air. This exposes additional animals and contaminates the environment.

SIGNALMENT

Goats, sheep, cattle, and bison are more susceptible than other species such as horses, pigs, and dogs.

PHYSICAL EXAMINATION FINDINGS

• Commonly animals will be found dead and clinical signs not observed.
• Clinical signs include fever up to 107°F (41.5°C), congested mucus membranes, excitement, depression, stupor, dyspnea, ataxia, muscle tremors, collapse, convulsions, and death. Younger animals will have less severe signs.
• If the animal lives long enough bloody diarrhea, hematuria, and localized swellings maybe seen as well as abortion.

CAUSES AND RISK FACTORS

• Anthrax is caused by *Bacillus anthracis* which is a large (1 μm by 3–5 μm) Gram-positive rod that is rectangular with square ends and commonly forms chains.
• *Bacillus anthracis* exists in two forms: the spore and vegetative cell.
• Once the spore infects the animal it germinates into the vegetative form. The vegetative form in infected tissues of the dead animal only survives up to 1–2 weeks. However, when exposed to air vegetative bacteria will sporulate in several hours. Sporulation is oxygen dependent.
• It is important not to necropsy the carcass to limit the spore formation and therefore keeping contamination of the environment to a minimum.
• Scavengers and biting insects may mechanically disseminate anthrax after feeding on affected carcasses.
• Risk factors include:
  ◦ Animals residing in endemic pastures or range.
  ◦ Exposure to disturbed soil in endemic areas. Disturbances can be due to flooding, excavations, cattle disturbing soil around wet pastures, or shrinking surface water sources.
  ◦ Grazing plants close to the ground, as seen in overstocking range or pasture or drought in endemic areas.

DIAGNOSIS

DIFFERENTIAL DIAGNOSES

Anthrax, because of the nonspecific history and clinical appearance, can be confused with many other causes of acute death in ruminants including clostridial infections (blackleg, malignant edema), lightning strike, acute toxicosis (cyanobacterium), and bloat.

OTHER DIAGNOSTIC PROCEDURES

• Samples must be submitted for a definitive diagnosis.
• The laboratory should be forewarned of a suspected anthrax case submission due to the zoonotic risk.
• Collections of samples are recommended at or soon after death when bacterial numbers are at their highest.
• Blood is the main and best sample for diagnostic submission. Bloody fluids and tissues are also useful; however, opening the carcass is strongly discouraged.
• To prevent contamination of the environment collection of blood sample from a superficial vein using a large gauge needle and syringe is recommended.
• Microscopic examination of blood smears, bacterial cultures, and PCR are a few of the examinations laboratories use to identify *Bacillus anthracis*.

PATHOLOGIC FINDINGS

• If the carcass is inadvertently opened the lesions are those of septicemia.
• Rigor mortis is commonly not present or incomplete.
• Cattle that die of anthrax decompose rapidly and are found gas distended with bloody exudates coming from any of the body orifices.
• Blood is usually thick and dark red with little or no clotting. Any clotted blood found is not well formed.
• Multifocal hemorrhages are common on mucosal and serosal surfaces as well as subcutaneously.
• Gelatinous fluid accumulates in loose connective tissue and serous cavities and could be blood tinged.
• Parenchymatous organs are congested, swollen, and soft. The spleen can be greatly enlarged and bloody.
• The heart has a dull appearance and is flaccid.
• In cattle the lesions at the site of bacterial entry can be more severe, such as ulcerative hemorrhagic enteritis.
• If the bacteria come in through the oropharynx there will be hemorrhage and swelling of local lymph nodes as well as edema in adjacent connective tissue and throat.
• Likewise if there is a case of pulmonary anthrax in cattle, lesions would be most severe in lung and mediastinum.
• Sheep and goats are more susceptible to anthrax than cattle. The course of the disease is more rapid so some of the lesions maybe less prominent or missing.

TREATMENT

THERAPEUTIC APPROACH

Often no therapeutic approach is available as the disease is peracute. Sick animals are treated with antibiotics (see "Drugs of Choice").

MEDICATIONS

DRUGS OF CHOICE

B. anthracis is generally antibiotic-sensitive but some strains can be resistant to penicillin. *B. anthracis* is susceptible to many antibiotics,

including but not limited to penicillin and tetracycline. However, eliminating the bacteria may not increase survival: once the toxins have entered cells in sufficient quantity, they can still manifest their lethal effects.

CONTRAINDICATIONS

Because the Sterne strain vaccine must replicate to effectively stimulate an immune response, antibiotic treatment should not be given in conjunction with or shortly after the administration of vaccine in healthy animals. However, field conditions may dictate a combination of vaccine and antibiotics as a practical course of action.

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS

In ruminants the incubation period is 1–5 days and the course of the disease is from hours to 2 days and the usual outcome is death.

PREVENTION

- Affected premises are quarantined to prevent further spread of the disease.
- Consideration should be given to limiting access to suspected sources of exposure in affected herds. This may include moving livestock to a different pasture, fencing livestock away from low-lying water sources, where spores may recently have been exposed due to drought conditions, providing insect control, and proper disposal of affected carcasses.
- Hygiene and carcass disposal are of paramount importance in halting an outbreak and preventing future disease occurrence.
- Autolysis of the carcass destroys the vegetative forms of *B. anthracis* and, therefore, if carcasses are not opened up, the potential for contamination of the environment is minimized.
- Carcasses must be disposed of in accordance with state or local regulations.
- Livestock in endemic areas should be vaccinated with Sterne-strain spore vaccine at least 2–4 weeks prior to the expected seasonal onset. Booster vaccination is given 4–5 weeks following the initial vaccination, and then annually thereafter.
- The vaccine is a live nonencapsulated organism and therefore antibiotics should not be given within 7–10 days of vaccine administration.
- Following vaccination, there is a 60-day withdrawal time for the carcass, and milk should be discarded for at least 72 hours.

MISCELLANEOUS

AGE-RELATED FACTORS

There are some reports of males being more susceptible than females and older animals being more susceptible than young animals, but these probably reflect differences in grazing behavior rather than inherent differences in susceptibility.

ZOONOTIC POTENTIAL

Anthrax can infect humans and precautions should be taken to avoid contamination with infected animal tissues, contaminated animal products, and anthrax spores.

BIOSECURITY

- Once anthrax is diagnosed, the farm will be quarantined. No movement of animals on or off the premises should occur during the quarantine period.
- Animals surviving an outbreak may be moved to uncontaminated pastures or holding pens in order to reduce the possibility of additional cases.
- Scavenger and insect control is important to prevent the spread of the disease.
- All equipment, vehicles, and working facilities used in an outbreak must be cleaned and disinfected.

PRODUCTION MANAGEMENT

Vaccination of livestock is an important consideration in anthrax endemic areas.

ABBREVIATION

PCR = polymerase chain reaction

Suggested Reading

A History of Anthrax. Centers for Disease Control and Prevention, http://www.cdc.gov/anthrax/resources/history/

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Client Education Handout available online

A

ARSENIC TOXICOSIS

BASICS

OVERVIEW
• Arsenic is the second most common cause of heavy metal intoxication of cattle, after lead.
• Arsenic is still used in herbicides, defoliants, wood preservatives, and other products; however, alternative products with lower toxicity and less environmental persistence are becoming progressively more available.

INCIDENCE/PREVALENCE
Unknown

GEOGRAPHIC DISTRIBUTION
Worldwide

SYSTEMS AFFECTED
• Digestive
• Urinary

PATHOPHYSIOLOGY
• Poisoning most often results from exposure to inorganic arsenic compounds contained in commercial products including herbicides, insecticides, and pressure-treated wood.
• Inorganic arsenic products can be trivalent (arsenite) or pentavalent (arsenate), with the arsenites being more soluble and toxic.
• Organic arsenic compounds are aliphatic or aromatic, and aromatic compounds can also be trivalent or pentavalent in nature.
• Ingestion of soluble arsenic compounds results in distribution of arsenic to many organs, with tissues rich in oxidative enzymes being the most vulnerable to damage, including the lungs, liver, kidneys, endothelium, and gastrointestinal tract.
• Mechanism of toxicity and toxic dose depend upon the formulation, route of exposure, and duration of exposure.
• Arsenates uncouple oxidative phosphorylation and trivalent arsenicals disrupt the citric acid cycle, creating damage to metabolically active tissues.

HISTORICAL FINDINGS
Deliberate or incidental exposure to products containing arsenic.

SIGNALMENT
Any ruminant species of any age, breed, or gender can be affected.

PHYSICAL EXAMINATION FINDINGS
• Signs typically reflect acute toxicosis and can include sudden death.
• Signs of acute or subacute toxicosis occur within 3–12 hours of exposure and reflect shock associated with gastrointestinal and cardiovascular damage.
• Colic, dehydration, weakness, anorexia, and severe watery diarrhea, which may be hemorrhagic, are observed.

GENETICS
N/A

CAUSES AND RISK FACTORS
• Exposure or access of animals to arsenic-containing products or residues such as herbicides, insecticides, and the ashes of pressure-treated wood.
• Grazing animals in areas of high environmental contamination which can result from residue from livestock dip, mining waste, and sources of industrial contamination.
• Ruminants are particularly sensitive to the aliphatic arsenicals MSMA and DSMA which are used as herbicides.

DIAGNOSIS

DIFFERENTIAL DIAGNOSES
• Bovine viral diarrhea virus (BVDV) could take out
• Bacterial enteritis *(Salmonella* spp.)
• Organophosphate insecticide exposure
• Other heavy metal toxicoses (lead)
• Urea toxicosis

CBC/BIOCHEMISTRY/URINALYSIS
Changes are not specific to arsenic toxicosis, but are consistent with dehydration, shock, gastrointestinal damage, and hemorrhage.

OTHER LABORATORY TESTS
• Arsenic concentration can be chemically determined in tissues including the liver and kidney (>3 ppm wet weight associated with toxicity); however, tissue concentrations may be normal in animals suffering peracute death.
• Arsenic can be measured in gastrointestinal content and blood for 24–48 hours after poisoning, and for several days in urine.
• Chemical determination of arsenic in water (>0.25 ppm potentially toxic) or feedstuffs can be performed.
• Skin or hair can be tested in animals exposed for more than 2 weeks.

IMAGING
N/A

OTHER DIAGNOSTIC PROCEDURES
N/A

PATHOLOGIC FINDINGS
• Gross findings include pale, swollen liver and kidneys, and generalized or localized reddening of the gastrointestinal mucosa with hemorrhage, erosions, submucosal edema, and sloughing of the mucosal lining into the gut lumen. In ruminants, the abomasum is often the most severely affected region.
• Peracute cases may have no abnormalities on gross inspection.
• Histopathologic findings include hepatic and renal tubular necrosis; dilation of intestinal capillaries, submucosal congestion and edema, and intestinal epithelial necrosis.

TREATMENT

THERAPEUTIC APPROACH
• Recently exposed animals may benefit from maneuvers to reduce further ingestion and absorption of arsenic from the gastrointestinal tract.
• Effective treatment of clinically affected animals relies on supportive medical care in addition to targeted chelation therapy.
• Fluid support and blood transfusion may be required in severely affected animals.

SURGICAL CONSIDERATIONS AND TECHNIQUES
N/A

MEDICATIONS

DRUGS OF CHOICE
• Absorption from the rumen after recent ingestion may be reduced via administration of mineral oil, or by rumen gavage or rumenotomy.
• Chelation therapy can be attempted using:
  ◦ Thioctic acid (lipoic acid)—50 mg/kg, IM, q8h (20% solution divided into two to three injection sites).
  ◦ Thioctic acid should be used in combination with dimercaprol at 3 mg/kg, IM, q4h for 2 days, q6h on day 3, then q12h for 10 days. Dimercaprol is also known as British anti-lewisite (BAL) and is recommended for treating trivalent inorganic or aliphatic organic arsenic toxicity.
  ◦ Sodium thiosulfate—IV: 30–40 mg/kg; PO: 20–30 g in 300 mL water (cattle) or 5–7.5 g in sheep and goats given q12h or q8h for 3–4 days.
  ◦ d-penicillamine (may be cost prohibitive)—10–50 mg/kg, PO, q8h–q6h for 3–4 days.
  ◦ 2,3-dimercaptosuccinic acid (DMSA, a water-soluble analog of dimercaprol)—10 mg/kg, PO, q8h has been recommended in small animals.

CONTRAINDICATIONS
N/A

PRECAUTIONS
• The source of arsenic exposure should be identified to reduce human risk.
• Carcasses should be disposed of carefully to avoid environmental contamination.
• Appropriate milk and meat withdrawal times must be followed for all compounds administered to food-producing animals.

POSSIBLE INTERACTIONS
N/A

(CONTINUED) ARSENIC TOXICOSIS A

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
Death can occur within the first 3–5 days of clinical signs; surviving animals may demonstrate slow recovery over several weeks.

POSSIBLE COMPLICATIONS
- Bacteremia/septicemia from severe gastrointestinal damage
- Renal failure

CLIENT EDUCATION
Avoid access by ruminants to heavy metals.

PATIENT CARE
Fluid and nutritional support, and nursing care are indicated to optimize survival.

PREVENTION
Avoid access by ruminants to heavy metals.

MISCELLANEOUS

ASSOCIATED CONDITIONS
- Colic
- Renal failure
- Anemia

AGE-RELATED FACTORS
N/A

ZOONOTIC POTENTIAL
N/A

PREGNANCY
Pregnant animals may abort.

BIOSECURITY
N/A

PRODUCTION MANAGEMENT
N/A

SYNONYMS
N/A

ABBREVIATIONS
- BAL = British anti-lewisite
- BVDV = bovine viral diarrhea virus
- DMSA = 2, 3-dimercaptosuccinic acid
- DSMA = disodium methane arsenate
- MSMA = monosodium methane arsenate

SEE ALSO
Toxicology: Herd Outbreaks

Suggested Reading

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ARTHROGRYPOSIS

BASICS

OVERVIEW

• Arthrogryposis is not a specific diagnosis, but rather a clinical finding of congenital contractures; these may be present in numerous disorders.
• Congenital arthrogryposis ("crooked joint") is defined as a syndrome of persistent joint flexure or contracture present at birth and may involve one or multiple limbs (forelimbs and/or hindlimbs).
• The carpal and phalangeal joints are most commonly affected, followed by metacarpophalangeal and metatarsophalangeal joints.
• Arthrogryposis is often associated with cleft palate and primary CNS lesions such as hydranencephaly and syringomyelia. Severely affected animals may also have scoliosis, kyphosis, and torticollis; with rotation, abduction, or curled limbs.
• The arthrogryposis-hydranencephaly syndrome (AHS) is usually associated with flexural contracture of the limbs rather than angular limb deformities (ALDs).
• In contrast with contracted tendons, arthrogryposis involves improper articular alignment or rotational deformity.
• Crooked calf disease (CCD) is a congenital deformity condition widely recognized in western North America, characterized by arthrogryposis, scoliosis, torticollis, and cleft palate. CCD is observed in calves after maternal ingestion of lupines containing the quinolizidine alkaloid anagyrine during gestation days 40–100.
• Congenital arthrogryposis may be associated with denervation muscle atrophy.
• The terms "arthrogryposis multiplex congenita" and "congenital articular rigidity" have been introduced to describe cases in which the rigidity may be due to lack of extensibility of muscles, tendons, ligaments, or other tissues around the joint, or to deformity of articular surfaces, or to fusion between bones at the articular surface.

INCIDENCE/PREVALENCE

• For CCD, there are reports of up to 40% of calves from a single herd being affected. The incidence of disease varies with year, area, and herd.
• Cattle records reveal that the disease usually affects <10% of a herd.

GEOGRAPHIC DISTRIBUTION

• Arthrogryposis is thought to occur worldwide. Depending on the etiologic cause and based on the distribution of vectors, viruses, and plants, the geographic distribution of individual syndromes may vary (e.g., CCD is most common in western North America).
• Calf arthrogryposis has been reported from most parts of the world and in many breeds of cattle.

SYSTEMS AFFECTED

Musculoskeletal

PATHOPHYSIOLOGY

• Congenital arthrogryposis is considered multifactorial in origin and has multiple predisposing factors and etiologies, including inherited defects. The causes are often not clear.
• Can be caused by a number of etiologic agents including: plant teratogens, spinal dysraphism, prenatal viral infections that affect the nervous system, and in utero hormonal and vascular defects.
• May also be attributed to a decrease or lack of motion of the fetus during critical stages of development, such as malpositioning and overcrowding caused by the size of the fetus relative to the dam.
• Ingestion of teratogenic plants such as *Astragalus* or *Oxytropis* spp. (locoweed); *Verratrum californicum* (skunk cabbage); piperidine alkaloid-containing plants such as *Lupinus, Conium,* and *Nicotiana* species.
  ◦ Repeated dosing or continuous low-level ingestion over time may result in cumulative intoxication and/or teratogenesis.
  ◦ Teratogenic plant alkaloids may be transferred to the placenta and induce a sedative or anesthetic effect in the fetus.
• In CCD, there is often a lesion in the CNS that may result in reduced or absence of movement of the affected body parts in the developing fetus, especially during the period of rapid growth. Alpha-motor neurons in the cervical spinal cord are significantly reduced.
• May cause disruption in normal innervation of muscles leading to paresis and instability of the limb, or may result in hypotonic condition of extensor muscle and dysfunction of the radial nerve.

HISTORICAL FINDINGS

A complete history including age, birthing details, age at which the deformity was noticed, course and progression of deformity, diets of affected animal and dam should be obtained. The animal may be normal at birth and develop the flexural deformity within hours or days.

SIGNALMENT

Species

Bovine, ovine, caprine, and camelid species

Breed Predilections

• Certain syndromes are predominantly reported to occur in certain breeds (e.g., congenital arthrogryposis in Charolais cattle). CCD has been observed in most dairy breeds and in all breeds of beef cattle common to western North America.
• No breed predilection or genetic susceptibility in cattle to the lupine-induced condition has been determined.

Mean Age and Range

• Arthrogryposis tends to affect young, growing animals. The incidence of CCD is highest in heifers at first calving, but the disease has been observed in calves from cows of all ages.
• For each species (cattle, sheep, and goats), there are specific periods of gestation when the fetus is susceptible to plant teratogens. The critical gestational period for exposure of cattle to lupines is 40–70 days with susceptible periods extending to 100 days.

Predominant Sex

No apparent sex predisposition

PHYSICAL EXAMINATION FINDINGS

• The animal's conformation should be assessed first by having the animal stand in a symmetrical manner on a firm, flat surface and observing it from multiple angles.
• Arthrogryposis in CCD is characterized by deformities of the limbs (rigid flexion of elbows and carpal joints) and spinal column (scoliosis, lordosis, kyphosis), and rib cage abnormalities. Affected calves occasionally have torticollis and cleft palate.
• The joints are often flexed and cannot be extended even after the flexor tendons are cut—distinguishing the disease from contracted tendons.

GENETICS

• Some genetic patterns have been worked out, for example, the arthrogryposis multiplex anomaly of Angus cattle is thought to be a simple autosomal recessive pattern.
• Syndromes in the Charolais, Friesian, Swedish, and Red Danish breeds of cattle are consistent with a simple recessive or modified recessive characteristic.
• Dominant defect traits are inherited as well and are sometimes selected for.
• Lambs: A congenital arthrogryposis exists in pedigree Suffolk and Australian Merino lambs as an inherited limb deformity.

CAUSES AND RISK FACTORS

A number of etiologic agents such as intrauterine infection with Border disease virus, BVDV, Akabane virus, Cache Valley virus, bluetongue virus, Aino virus, Kasba (Chuzan) virus, Rift Valley Fever virus, Schmallenberg virus, and Wesselsbron virus, as well as teratogenic plant ingestion have been implicated in the pathogenesis of arthrogryposis in ruminants.

Congenital Predisposing Factors

• Uterine malpositioning
• Genetic causes
• Ingestion of teratogenic plants by pregnant dam such as *Astragalus* or *Oxytropis* spp. (locoweed); *Verratrum californicum* (skunk cabbage); piperidine alkaloid-containing plants such as *Lupinus, Conium,* and *Nicotiana* species.
• Conditions associated with arthrogryposis include CCD/congenital arthrogryposis, HC (hereditary chondrodysplasia or spider lamb

(CONTINUED) ARTHROGRYPOSIS A

syndrome), ill-thrift syndrome in llamas, metabolic, and neurovascular disorders.
• Leg deformities in young calves are most commonly associated with congenital contraction of the tendons. Flexural deformities involving contracted tendons and ligaments may be seen in many breeds of cattle and small ruminants.

Risk Factors
Predisposing factors for congenital arthrogryposis include male calves, posterior intrauterine presentation, and double muscling.

DIAGNOSIS

DIFFERENTIAL DIAGNOSES
• Arthrogryposis and CCD differ from contracted tendons; in animals with contracted tendons, the joints are usually properly aligned and the legs are not rotated. In calves with arthrogryposis, the articular and osseous changes are usually permanent and worsen as the calf grows.
• Fracture malunion.
• See "Causes and Risk Factors" (viruses and toxic plants).
• Arthrogryposis-hydranencephaly syndrome (AHS) in lambs may be confused with HC.

CBC/BIOCHEMISTRY/URINALYSIS
N/A

OTHER LABORATORY TESTS
N/A

IMAGING
• Radiographs can be used to diagnose ALDs; at least two views, 90 degrees apart, should be taken of the affected joint.
• The dorsopalmar/dorsoplantar (DP) view is needed for examination of the anatomic location of the deformity and for measurements. Shoot with radiographic beam in line with the claws.

OTHER DIAGNOSTIC PROCEDURES
• Serology and virologic diagnostic assays may aid in ruling out in utero viral infections (e.g., Cache Valley virus).
• Feed analysis and assessment of the availability of potentially toxic plants in the environment (pasture) may assist in diagnosis.

PATHOLOGIC FINDINGS
• No consistent primary lesion in CCD; a number of varied tissue responses are observed. It is likely that these findings are at least in part due to the animal's inability to stand.
• CCD histology: Few lesions, restricted to muscles of the forelimb, external intercostalis muscle, or radial and femoral nerves—myositis, myodegeneration, muscle necrosis and atrophy, cellulitis, and perineuritis.

TREATMENT

THERAPEUTIC APPROACH
• Severely affected animals may be unable to rise to nurse and require additional supportive care.
• Protect limbs with thick, soft bandages that fit well. Minor deformities may be corrected by manual alignment and external support of the limb (e.g., rigid splinting, bandaging, or casting/tube casts).
• Provide good footing and allow for stretching of flexor tendons.
• Maintain a soft (padding), clean, and dry environment to minimize decubital/pressure sores, open arthritis, muscle atrophy, umbilical infections, and septicemia.
• Restrict activity until it is certain that the deformity is improving; however, some degree of exercise allows for stretching and lengthening of affected limb structures. Weight bearing provides the necessary physical exercise to strengthen and lengthen affected tendons and musculature.
• Dietary imbalances should be addressed while treating cases of arthrogryposis.

SURGICAL CONSIDERATIONS AND TECHNIQUES
• Surgery may be required for animals with severe deformities and for animals that do not improve with age or conservative management.
• Treatment of arthrogryposis includes surgery to improve the animal's posture sufficient for it to obtain slaughter weight (a salvage procedure).
• Surgical procedures include transection of flexor tendon and suspensory ligament, joint capsule release, flexor tendon lengthening procedures, and joint arthrodesis.
• May require postoperative splinting or casting for support.

MEDICATIONS

DRUGS OF CHOICE
N/A

CONTRAINDICATIONS
N/A

PRECAUTIONS
N/A

POSSIBLE INTERACTIONS
N/A

ALTERNATIVE DRUGS
N/A

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
• The prognosis is guarded, depending on the severity of the flexural deformity. Severe deformities requiring surgery often have a poor prognosis.
• For arthrogryposis in cattle, approximately 80% of surgically treated animals can be kept until they reach normal slaughter weight.

POSSIBLE COMPLICATIONS
Some severe cases of arthrogryposis cannot be corrected and full extension may not be possible postoperatively.

CLIENT EDUCATION
• Examination of young animals with congenital arthrogryposis should be done as early as possible to assess the degree of manual correction possible.
• Because of a possible hereditary component associated with some forms of arthrogryposis, breeding of affected animals is not recommended.
• Many affected animals are stillborn or die shortly after birth. Others may fail to thrive and euthanasia should be considered.

PATIENT CARE
Frequent physical examinations and assessing the efficacy of corrective measures should be done to monitor progress.

PREVENTION
• CCD/HC: Avoid breeding affected animals.
• Coordinate grazing times and alter breeding dates to minimize exposure. Avoid grazing potentially teratogenic plants when pregnant cows are at the susceptible stage of pregnancy.
• Control teratogenic plant populations with herbicides.

MISCELLANEOUS

ASSOCIATED CONDITIONS
A syndrome known as "bentleg" or "bowie" has been associated with ingestion of *Trachymene glaucifolia* (wild parsnip) by pregnant ewes in Australia and New Zealand.

AGE-RELATED FACTORS
Majority of cases occur during the active growth phase of the affected bone/joint.

ZOONOTIC POTENTIAL
N/A

PREGNANCY
In cases of congenital arthrogryposis, the teratogenic plants are ingested by the pregnant dam and the compounds are passed to the fetus through the placenta.

BIOSECURITY
N/A

A

ARTHROGRYPOSIS (CONTINUED)

PRODUCTION MANAGEMENT

• Producers should be aware of the association between certain toxic plants (e.g., lupines) and angular limb deformities such as CCD.
• To reduce the incidence of CCD, graze lupines during their least hazardous growth period and reduce exposure of pregnant cows. Lupines are most hazardous when they are young or in the mature seed stage.
• Fence off heavily infested pasture areas and use intermittent, short-term grazing of lupine pastures.

SYNONYMS

N/A

ABBREVIATIONS

• AHS = arthrogryposis-hydranencephaly syndrome
• ALD = angular limb deformity
• BVDV = bovine viral diarrhea virus
• CCD = crooked calf disease/syndrome
• CNS = central nervous system
• DP = dorsopalmar/dorsoplantar
• HC = hereditary chondrodysplasia
• IBR = infectious bovine rhinotracheitis virus

SEE ALSO

• Akabane
• Angular Limb Deformities
• Brain Assessment and Dysfunction (see www.fiveminutevet.com/ruminant)
• Cache Valley Virus
• Lameness: Bovine
• Lameness: Camelid
• Lameness: Small Ruminants
• Lupine Toxicity
• Schmallenberg Virus
• Wesselsbron Disease

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ARTIFICIAL INSEMINATION: BOVINE

BASICS

OVERVIEW
Artificial insemination (AI) is the aseptic delivery of viable spermatozoa into the female reproductive tract (uterus).

INCIDENCE/PREVELENCE
AI is more prevalent in dairy cattle than beef cattle.

GEOGRAPHIC DISTRIBUTION
Worldwide

SYSTEMS AFFECTED
Reproductive

PATHOPHYSIOLOGY
• Improvements in semen extenders, implementation of protocols for estrus synchronization and timed insemination, and availability of sex-sorted semen have increased the use and importance of AI in cattle.
• Advantages of AI over natural service:
  ◦ Efficient use and dissemination of valuable bull genetics
  ◦ Eliminate cost associated with bull maintenance
  ◦ Decreased risk of sexually transmitted diseases
  ◦ Fewer breeding-related injuries
  ◦ Enables evaluation of semen before insemination
• AI success is dependent on semen quality and handling, timing, site of deposition, and female fertility.
• Acceptable fertility in an AI program involves excellent management of all phases of the program.

HISTORICAL FINDINGS
• Estrus synchronization and timed AI
• Cows selected based on estrus detection

SIGNALMENT
Females of breeding age

PHYSICAL EXAMINATION FINDINGS
• Estrus behavior and observations:
  ◦ Standing to be mounted (cardinal sign of estrus)
  ◦ Cervical mucus discharge
  ◦ Mounting other cattle
  ◦ Increased locomotion and vocalization
  ◦ Decreased feed intake/milk production

Semen Collection and Cryopreservation
• Semen used for AI is commonly collected using an artificial vagina (AV) (most common in commercial AI bull centers) or by electroejaculation (custom freezing of bulls not trained to an AV, salvage of genetics from terminally ill bulls).
• Epididymal spermatozoa may be collected to salvage genetics from bulls following catastrophic injury or terminal illness.
• Semen collected with an AV presents better post-thaw motility than semen collected with electroejaculation.
• Although fresh/cooled semen may be used in some ranches, most AI in the bovine uses frozen-thawed semen.
• Semen is frozen after dilution in various commercial extenders in the presence of antibiotics, egg yolk, and glycerol.
• Bovine sperm is usually frozen in 0.25 or 0.5 mL straws. The majority of sex-sorted (gender-selected) semen is packaged in 0.25 mL straws.
• In general, the total dose per straw is 10 to 20 million motile spermatozoa. However, this dose is considerably lower for elite bulls and sex-sorted semen. Higher doses are used for bulls with lower fertility or in custom freezing of beef bulls.
• The cryopreservation process varies slightly depending on extender and technique used.

Factors Affecting AI Success
Quality of Semen
• The quality of frozen-thawed semen depends on initial quality at production, handling during storage, thawing procedures, and interval until deposition into the uterus.
• Quality at production is affected by initial quality of the ejaculate, method of collection used, the inherent freezability, and methods of processing used for cryopreservation.
• Repeated exposure to temperature above −80°C may cause changes in liquid/frozen phase and damage to the sperm membrane. This can occur during handling or transfer of straws between tanks or may be due to faulty tank performance and lack of verification of LN level. To prevent these problems, the following points should be observed:
  ◦ LN tank management:
    ▪ Keep in a cool, dry, well-ventilated area away from direct sunlight.
    ▪ Check LN level frequently. Presence of frost on the surface of the LN tank is a sign of leakage.
    ▪ Place LN tanks on a wooden or plastic dolly above concrete or wet floors to prevent corrosion.
    ▪ Keep a precise inventory for quick identification of straws to be used.
    ▪ Attach LN refill date logs to the tank.
  ◦ Removing straws from the tank:
    ▪ Remove straws quickly without exposing other straws to air.
    ▪ Do not raise canisters higher than 10 cm above the neck of the LN tank; do not raise the top of the goblet in the canister above the frost line in the neck of the tank.
    ▪ Ensure that the goblet is always filled with LN.
    ▪ Always use forceps to remove straws from the goblets.
  ◦ Handling at the time of use:
    ▪ Use recommended thawing temperature and time. Recommendation of the NAAB is immersion in the water bath at 30–35°C for at least 40 seconds. Others recommend thawing at 70°C for 6 seconds or 35–37°C for 30 seconds. Thawing at high temperatures may be very difficult to observe in field conditions.
    ▪ Thawing procedures should be strictly observed.
    ▪ Check water baths for temperature control accuracy.
    ▪ Use a stopwatch for the timing of thawing.
    ▪ In cold areas, it is preferable to keep straws in the water bath, at 35°C, until use.
    ▪ Prevent exposure of the straw to low temperatures after thawing: use a pre-warmed insemination gun; protect prepared gun from exposure to low temperature.
    ▪ Thaw out straws and prepare insemination guns in a sheltered area when ready to inseminate.
    ▪ Clean and dry straws before loading in the AI gun.
    ▪ Straws should be cut using straw cutters to prevent backflow of semen during insemination.
    ▪ Time period from removal of straw from the tank to insemination should not exceed 15 minutes.

Timing of Insemination
• Insemination timing is important to maximize conception rates. Ovulation occurs 28–32 hours after the beginning of estrus. Optimum fertility of the oocyte is 6–12 hours after ovulation and the viable lifespan of spermatozoa in the reproductive tract is estimated to be between 24 and 30 hours.
• The optimal time for insemination is 12 hours after first observed estrus. This has led to the AM/PM rule: if a cow is observed in estrus in the morning she is inseminated in the afternoon, and vice versa.
• Several schemes for timed insemination have been developed.
• Double insemination at a 12-hour interval is sometimes performed in cows with decreased fertility, when using semen straws with lower concentration, or in cows that have been superstimulated for embryo collection.

Insemination Technique
• Semen should be deposited at the proper site without excess manipulation.
• People who inseminate cows periodically cannot achieve a high level of expertise.
• Semen is deposited in the body of the uterus by transrectal manipulation of the insemination gun for cervical catheterization.
• Deep horn insemination may reduce conception rates in the hands of untrained people due to irritation of the uterus.
• Deep uterine insemination may be important when low spermatozoa numbers are used (i.e., sexed semen).
• Good inseminators should be able to pass the cervix rapidly and with a high degree of reliability. Cervical catheterization is particularly difficult in some breeds of cattle (e.g., Santa Gertrudis).

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ARTIFICIAL INSEMINATION: BOVINE (CONTINUED)

• Strict hygienic measures should be observed during insemination to prevent vaginal or uterine contamination.
• Use a protective plastic sheath (cannula or chemisette) to help prevent contamination.

GENETICS
Semen is selected based on specific selection criteria.

CAUSES AND RISK FACTORS
N/A

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS
N/A

CBC/BIOCHEMISTRY/URINALYSIS
N/A

OTHER LABORATORY TESTS
• Serum progesterone concentration <2 ng/mL may confirm estrus activity.
• Diagnosis of pregnancy can be performed by detection of serum pregnancy associated glycoproteins.

IMAGING
• Ultrasonography of the reproductive tract in an estrus female should reveal a dominant follicle (>10 mm diameter), regressing or absent CL, and uterine edema.
• Diagnosis of pregnancy as early as 28 days post-ovulation.

DIAGNOSTIC PROCEDURES
N/A

PATHOLOGIC FINDINGS
N/A

TREATMENT

APPROPRIATE HEALTH CARE
N/A

SURGICAL CONSIDERATIONS AND TECHNIQUES
N/A

MEDICATIONS

DRUGS OF CHOICE
N/A

CONTRAINDICATIONS
N/A

PRECAUTIONS
N/A

POSSIBLE INTERACTIONS
N/A

ALTERNATIVE DRUGS
N/A

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
• Pregnancy rates depend on many factors: breed, quality of semen, site of semen deposition, nutrition, milk production (dairy), environmental conditions, parity, type of synchronization program, and human factors.
• Conception rates following artificial insemination range between 40 and 50% in cows and 60 to 70% in heifers. However, in dairy cattle, pregnancy rates 35 to 40 days following AI are generally between 20% and 30% due to a high early embryonic loss.
• In addition to the factors already mentioned, other factors involved in decreased conception rate in dairy cows are:
  ◦ Insemination of non-estrous cows
  ◦ Heat stress prior to artificial insemination
  ◦ Incidence of postparturient diseases
  ◦ Clinical mastitis

POSSIBLE COMPLICATIONS
Perforation of the vagina or uterus by unskilled or inexperienced technicians.

PATIENT CARE
Appropriate estrus detection and handling to reduce stress.

PREVENTION
N/A

MISCELLANEOUS

ASSOCIATED CONDITIONS
N/A

AGE-RELATED FACTORS
Normal conception rates have been achieved with as low as 2 million spermatozoa in heifers.

ZOONOTIC POTENTIAL
N/A

PREGNANCY
See "Expected Course and Prognosis"

BIOSECURITY
• Use semen from bulls routinely tested for brucellosis, IBR, BVD, trichomoniasis, and campylobacteriosis, as well as bluetongue virus (Certified Semen Service).
• Bacteriologic quality of semen is ensured by strict hygiene during collection and use of specific guidelines for antimicrobials in the extenders (Certified Semen Service).
• All straws are properly labeled, identifying the semen center, bull, breed, and date on which semen was collected.

PRODUCTION MANAGEMENT
Use a professional inseminator.

SYNONYMS
N/A

ABBREVIATIONS
• AI = artificial insemination
• AV = artificial vagina
• BVD = bovine viral diarrhea
• CL = corpus luteum
• IBR = infectious bovine rhinotracheitis
• LN = liquid nitrogen
• NAAB = National Association of Animal Breeders

SEE ALSO
• Beef Bull Management (see www.fiveminutevet.com/ruminant)
• Estrus Synchronization: Bovine
• Reproductive Pharmacology
• Reproductive Ultrasonography: Bovine (see www.fiveminutevet.com/ruminant)

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ARTIFICIAL INSEMINATION: SMALL RUMINANT

BASICS

OVERVIEW
• Artificial insemination (AI) is used in sheep and goat herds to improve genetics, improve reproductive efficiency of males, reduce disease transmission, and permit out-of-season breeding.
• Success of an AI program is affected by several factors including timing, semen quality, and semen placement.
• For economic reasons, except in a few cases (dairy goat), most AI is performed at a fixed time following synchronization of estrus.

INCIDENCE/PREVALENCE
• AI using fresh or cooled semen is a common procedure in several countries.
• AI with frozen-thawed semen is more common in dairy goats.

GEOGRAPHIC DISTRIBUTION
Worldwide

SYSTEMS AFFECTED
Reproductive

PATHOPHYSIOLOGY
• Adequate fertilization rates are achieved when sperm form a reservoir at the level of the isthmus of the uterine tube and remain viable until ovulation occurs.
• Fertilization rates are dependent on quality of semen, adequate transport to the fertilization site, and proper timing in relationship to ovulation.
• In small ruminants, semen is deposited onto the cervix after natural mating. Sperm is selectively transported through the cervix into the uterus and only highly motile, morphologically normal sperm is found in the isthmus.
• With AI the number of sperm used is significantly lower than in natural mating. In addition, processing and cryopreservation reduces sperm viability. Therefore, timing and site of deposition of sperm are critical in achieving acceptable fertilization rates.
• Oocyte viability is extremely reduced beyond 12 hours post-ovulation. AI should be performed within 12 hours after onset of estrus.
• The number of viable spermatozoa per insemination required for adequate fertilization rates depends on quality and site of semen deposition. Ideally, preserved semen (chilled or frozen-thawed) should be deposited into the uterine cavity.
• In sheep and small breeds of goats, TCI is extremely difficult due to the tortuous anatomy of the cervical canal. To bypass the cervix, insemination is performed laparoscopically.

HISTORICAL FINDINGS
• Flock or herd desiring to improve genetics
• Out-of-season breeding

SIGNALMENT
Postpubertal female sheep and goats

PHYSICAL EXAMINATION FINDINGS
• Females to be inseminated should be in estrus.
• Estrus detection may be performed with a teaser male (sheep and goat) or by visual observation (goats).
• Females in estrus will show typical receptive behavior. Does express estrus more intensely than ewes (tail fanning, mounting other females, and standing to be mounted).
• Females in estrus show variable degrees of vulvar swelling and mucus discharge. Mucus is clear and runny at the beginning of estrus and becomes thick and whitish near ovulation.
• On vaginoscopy, the cervix is hyperemic and open.

Artificial Insemination Techniques
Semen Source and Processing
• Semen is collected from selected males using an artificial vagina (preferred) or electroejaculation.
• Only high-quality ejaculates are used. Semen is diluted in one of a variety of available commercial extenders (milk-based, egg yolk-based, or new chemically defined soybean lecithin-based).
  ◦ Milk-based extenders are commonly used for fresh, chilled (4°C) or cooled (15°C) semen insemination. Skim-milk or UHT-treated milk is used in order to remove lactenin which is spermicidal.
  ◦ Tris-based egg yolk extenders are used routinely for cryopreservation. However, egg yolk is toxic to buck semen because of the presence of lipase produced by the bulbourethral gland (*BUSgp60*). Buck semen needs to be washed prior to addition of egg yolk-based extender. An alternative is to use a minimal quantity of egg yolk in the extender.
  ◦ Chemically defined media have been shown to be effective for the cryopreservation of small ruminant sperm. They offer the advantage of not containing any additional animal products, which guarantee its biosecurity for international movement of semen.
  ◦ Additional improvement in preservation of semen has been achieved by addition of antioxidants.
• Small ruminant sperm is cryopreserved in either 0.25 mL or 0.5 mL straws and rarely in pellet form. Semen handling and thawing procedures (time and temperature) are critical for viability.

Timing of Insemination
• Natural estrus: AI is performed 12 hours after detection of estrus. A second insemination is often needed 12 hours later if frozen-thawed semen is used.
• Fixed time after synchronization:
  ◦ TCI insemination: Sheep – 55 hours (1 dose) or 50 and 60 hours (2 doses) after progesterone removal. Goats – 45 to 65 hours (1 dose) or 30 and 48 hours (2 doses) after progesterone removal.
  ◦ Laparoscopic AI: 54 to 60 hours after progestogen removal (6 to 8 hours earlier for frozen-thawed semen) in sheep, and 43 to 46 hours after progesterone removal in goats.

Method of Insemination
• Blind vaginal insemination—At least 400 million sperm are blindly deposited around the external cervical os. This technique is useful only when fresh, extended semen is used.
• Intracervical—At least 200 million sperm are deposited into the cervical canal as far as the AI gun can advance.
• TCI—Between 50 and 100 million spermatozoa are required. TCI is used more in goats as the cervix is more easily catheterized. In sheep, TCI is limited by the morphology of the sheep cervix, which is long, tortuous, and narrow, with nonconcentric rings. However, several techniques have been proposed to improve TCI in sheep; these include the use of special cradles for restraint, and the use of flexible AI guns.
• Laparoscopic—Semen is injected directly into the uterus via laparoscopic portals. Between 20 and 40 million sperm are required (15–20 million if sex-sorted). Drawbacks to this technique include the need for sedation or anesthesia of the animal, laparoscopic equipment, increased labor time and costs, and postoperative monitoring.

GENETICS
AI is the best method for rapid genetic improvement of a flock or herd.

CAUSES AND RISK FACTORS
N/A

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS
N/A

CBC/BIOCHEMISTRY/URINALYSIS
N/A

OTHER LABORATORY TESTS
Semen analysis

IMAGING
Transrectal or transabdominal ultrasonography for pregnancy diagnosis following artificial insemination.

OTHER DIAGNOSTIC PROCEDURES
Pregnancy diagnosis using serum concentration of pregnancy associated glycoproteins.

PATHOLOGIC FINDINGS
N/A

A

ARTIFICIAL INSEMINATION: SMALL RUMINANT (CONTINUED)

TREATMENT

THERAPEUTIC APPROACH
• Estrus should be synchronized using approved hormonal methods of synchronization.
• Several treatments (oxytocin, estrogen, ß-adrenergic blocking agents, relaxin, and PGE) have been used in sheep to induce cervical relaxation for TCI but were not very successful.

SURGICAL CONSIDERATIONS AND TECHNIQUE
• Estrus animals displaying systemic illness should not be selected for surgical insemination. Obese and non-fasted animals present surgical challenges.
• Animals should be fasted for 18–24 hours prior to laparoscopic AI (LAI).
• LAI is performed under sedation and local block in sheep. Heavy sedation or general anesthesia is preferred in goats. The animal is placed in dorsal recumbency on a cradle in a Trendelenburg position. The ventral abdomen is clipped and prepared for surgery.
• Two portals are created on each side (about 5 cm) of the linea alba and about 6–10 cm from the cranial border of the mammary gland. The laparoscope is inserted into the abdominal cavity from one portal. The uterus is visualized and the insemination gun is inserted through the other portal using a cannula. The uterine horn is stabbed with the needle of the insemination gun and semen is deposited in the lumen.
• Skin incisions are usually closed with staples or absorbable suture.

MEDICATIONS

DRUGS OF CHOICE
• Sedation and anesthesia: xylazine and ketamine.
• Administration of antibiotics and anti-inflammatories is recommended.

CONTRAINDICATIONS
Appropriate milk and meat withdrawal times must be followed.

PRECAUTIONS
N/A

POSSIBLE INTERACTIONS
N/A

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
• Pregnancy rates depend on many factors: species and breed, type of semen used, extender type, site of deposition of semen, nutrition, season, environmental conditions, parity, the synchronization program used, and human factors.
• Expected conception rates when semen quality and number of spermatozoa are adequate are:
  ◦ Vaginal AI: fresh semen 60–75%; frozen semen 5–30%
  ◦ Intracervical AI: fresh semen 50–80%; frozen semen 35–60%
  ◦ TCI: fresh semen 40–80%; frozen semen 30–70%
  ◦ Laparoscopic AI: fresh semen 40–80%; frozen semen 40–70%.

POSSIBLE COMPLICATIONS
• For surgical complications see "Patient Care."
• TCI in sheep may result in cervical trauma or laceration.

CLIENT EDUATION
• Clients should be aware of factors affecting fertility and the expected pregnancy rates for the type of semen and AI used.

PATIENT CARE
• Animals should be handled with minimal stress.
• After laparoscopic AI, animals should be monitored for complications, including incisional infection or dehiscence, fever, peritonitis, and adhesion formation. Animals should be vaccinated against *Clostridium tetani*. Administration of antibiotics and anti-inflammatories is recommended.
• Pregnancy diagnosis should be performed after an AI program.

PREVENTION
N/A

MISCELLANEOUS

ASSOCIATED CONDITIONS
N/A

AGE-RELATED FACTORS
TCI is more difficult in young maiden females.

ZOONOTIC POTENTIAL
N/A

PREGNANCY
N/A

BIOSECURITY
• All males used for semen collection and AI should have a health screening and infectious disease testing prior to use.
• Hygienic semen collection and processing of semen should be performed according to guidelines described by the Office International des Epizooties.
• Diseases that can be transmitted by semen include: Bluetongue, Border disease, brucellosis, leptospirosis, paratuberculosis, Q fever, contagious caprine pleuropneumonia, ovine enzootic abortion, Lentivirus infection, peste des petit ruminants, *Salmonella* serotype abortus-ovis, sheep and goat pox.

PRODUCTION MANAGEMENT
• AI with fresh semen allows more efficient use of top sires, particularly after synchronization of estrus.
• Use of frozen semen allows genetic improvement.
• AI after a synchronization program can allow predictable parturition dates and allow segregation of animals on the farm by physiologic status.
• AI can allow for accelerated lambing programs or out-of-season breeding.

SYNONYMS
N/A

ABBREVIATIONS
• AI = artificial insemination
• PGE = prostaglandin E
• TCI = transcervical insemination

SEE ALSO
Estrus Synchronization: Small Ruminants

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ASPIRATION PNEUMONIA

BASICS

OVERVIEW
Aspiration pneumonia arises from the inhalation or accidental administration of liquids, pastes, gels or foreign bodies (plant debris, dirt) which can result in inflammatory, gangrenous or granulomatous pneumonia, or in the case of oil aspiration, lipid or lipoid pneumonia. Some substances directly insult the respiratory tissues, while others may induce vigorous inflammatory responses associated with nondegradable foreign material. Secondary bacterial infection is a common sequela.

INCIDENCE/PREVALENCE
Uncommon

GEOGRAPHIC DISTRIBUTION
N/A

SYSTEMS AFFECTED
Respiratory

PATHOPHYSIOLOGY
• The disorder often arises following the accidental or forceful administration of substances and medication by unskilled personnel, or after inappropriate regurgitation/aspiration associated with heavy sedation, anesthesia, or oropharyngeal and esophageal disorders.
• Lung tissue in the cranioventral thorax is most commonly affected after inhalation or instillation of a foreign substance.
• The affected lung tissue is irritated, inflamed, and loses capacity for appropriate clearance, immune function, and oxygenation. Secondary bacterial invasion frequently occurs.

HISTORICAL FINDINGS
Consistent with a primary illness or management activity prompting treatment with oral medications or substances, or preexisting signs of upper gastrointestinal dysfunction.

SIGNALMENT
• Varies with different inciting conditions.
• Young ruminants and crias may suffer aspiration from accidental orotracheal intubation during provision of colostrum or milk; from inhalation of meconium in fetal fluids during difficult parturition; from poorly performed bottle feeding; from congenital disorders such as cleft palate and aortic arch defects; and from acquired disorders such as selenium deficiency and necrotic laryngitis.
• Mature ruminants and camelids may suffer aspiration due to pharyngeal and esophageal dysfunction related to trauma, abscessation, choke, or megaesophagus; infectious diseases (botulism, listeriosis); heavy sedation or anesthesia; toxicities (ingestion of lead, crude oil, fuel oil, natural gas condensate, rhododendron); and from oral medication administration or severe hypocalcemia (lactating cattle).

PHYSICAL EXAMINATION FINDINGS
• Acute-onset depression, tachypnea, coughing, and fever.
• Large volume aspiration can produce dyspnea, tachypnea, tachycardia, nasal discharge, and malodorous breath.
• Thoracic auscultation can reveal adventitious lung sounds, pleural friction rubs, or reduced audibility of ventral lung sounds if pleural fluid accumulates.
• Shock and sudden death may occur.
• Milk or feed material may be seen coming from the nostrils in cases where dysphagia or esophageal disorders are the initiating cause.

GENETICS
N/A

CAUSES AND RISK FACTORS
• Accidental or forceful administration of substances and medication orally or orogastrically by unskilled personnel.
• Heavy sedation or general anesthesia.
• Oropharyngeal and esophageal disorders.
• Submergence dipping of livestock.
• Exposure to poorly cured silage (*Listeria*) or specific toxins in the feed or environment.

DIAGNOSIS

DIFFERENTIAL DIAGNOSES
• Acute bronchopneumonia due to infectious agents or causes other than aspiration.
• Systemic sepsis, particularly in neonates.

CBC/BIOCHEMISTRY/URINALYSIS
• Complete blood count may demonstrate leukocytosis or leukopenia with left shift, elevated total plasma protein, and hyperfibrinogenemia.
• Serum chemistry may demonstrate hyperglobulinemia and azotemia, and/or specific electrolyte abnormalities such as hypocalcemia.

OTHER LABORATORY TESTS
N/A

IMAGING
• Radiographs typically show alveolar-interstitial pattern that is most pronounced in the cranioventral lung fields. Pleural fluid lines or pulmonary abscesses may be evident.
• Ultrasonography reveals cranioventral consolidation with or without pleural effusion.
• Pharyngeal abscessation or trauma, and esophageal dilation, stricture or obstruction may be evident on radiographs (contrast enhancement can be performed if indicated).

OTHER DIAGNOSTIC PROCEDURES
• Endoscopic or transtracheal wash can be performed to obtain samples for cytologic examination and aerobic and anaerobic bacterial culture and sensitivity procedures.
• Endoscopy of the upper gastrointestinal tract is appropriate to investigate pharyngeal and esophageal disorders.

PATHOLOGIC FINDINGS
• Reflect acute bronchopneumonia with cranioventral distribution, with or without accompanying fibrinous pleuropneumonia.
• In early disease the main findings may include pulmonary congestion and edema, and the bronchi may be hyperemic and contain froth and potentially foreign material. As disease progresses affected areas of lung become suppurative, necrotic and soft, and fibrinous pleuritis is evident. Chronic cases may have pulmonary abscesses and fibrous adhesions between the visceral and parietal pleura.

TREATMENT

THERAPEUTIC APPROACH
• Commences with identification of the reason or cause for aspiration to protect the airway from further damage, and to determine the prognosis (e.g., treatment may be futile with large volume oil aspiration).
• Treatment is aimed at neutralizing secondary bacterial infection by Gram-positive and Gram-negative aerobic and anaerobic bacterial agents and should start as soon as possible after the aspiration event is recognized, typically regardless of whether clinical signs exist.
• Steroidal and/or nonsteroidal anti-inflammatory agents can be utilized to control shock and inflammation.

SURGICAL CONSIDERATIONS AND TECHNIQUES
N/A

MEDICATIONS

DRUGS OF CHOICE
• Broad-spectrum antibiotic therapy is indicated to cover Gram-positive and Gram-negative bacterial agents. In severe cases anaerobic bacterial coverage is also indicated.
• Appropriate antibiotic coverage can be achieved using combinations of antibiotics, or one antibiotic depending on the desired spectrum and duration of coverage; drugs should be given at recommended doses and dosing intervals.

CONTRAINDICATIONS
Oral administration of medications during treatment is likely contraindicated if deficits in pharyngeal and esophageal function are present.

A

ASPIRATION PNEUMONIA (CONTINUED)

PRECAUTIONS
Appropriate milk and meat withdrawal times must be followed for all compounds administered to food-producing animals.

POSSIBLE INTERACTIONS
N/A

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
- Animals with relatively minor aspiration events that receive prompt therapy are expected to survive.
- Aspiration events that are large volume, promote gangrenous pneumonia, have delayed recognition and treatment, or are associated with congenital or poorly reversible disorders have a poor prognosis.
- Fibrosing alveolitis (oil aspiration), pulmonary abscessation, and pulmonary granulomas are chronic sequelae that typically reduce prognosis.

POSSIBLE COMPLICATIONS
- Chronic respiratory disease related to fibrosing alveolitis or ongoing pulmonary inflammation and infection.
- Septicemia and multiorgan failure.

CLIENT EDUCATION
Personnel should be thoroughly instructed in the correct techniques for administering medications by mouth or orogastric tube.

PATIENT CARE
- Monitor for worsening of respiratory function, fever and any other associated clinical signs.
- Provide a soft and palatable ration.
- Intermittent orogastric feeding may be indicated for animals with upper gastrointestinal dysfunction.

PREVENTION
- Education regarding medication administration is the most important method of preventing aspiration events.
- Fasting of animals combined with careful head positioning, ± intubation during sedation and general anesthesia reduces the risk of inducing aspiration.

MISCELLANEOUS

ASSOCIATED CONDITIONS
N/A

AGE-RELATED FACTORS
Young animals are prone to aspiration events associated with feeding management, or with congenital disorders and selenium deficiency.

ZOONOTIC POTENTIAL
N/A

PREGNANCY
N/A

BIOSECURITY
N/A

PRODUCTION MANAGEMENT
- Avoid unnecessary oral medications and instruct personnel to deliver oral medications or drenches at a measured rate to allow the animal to swallow, with the head positioned level or down, and with great caution during periods of struggling and vocalization.
- Caustic or inflammatory substances (calcium chloride, mineral oil) are often best given by orogastric tube versus oral drench.

SYNONYMS
N/A

ABBREVIATIONS
N/A

SEE ALSO
- Atypical Interstitial Pneumonia
- Calf Diphtheria/Necrotic Stomatitis
- Enzootic Pneumonia of Calves
- Respiratory Disease: Bovine
- Respiratory Disease: Camelids
- Respiratory Disease: Small Ruminant

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ATYPICAL INTERSTITIAL PNEUMONIA

BASICS

OVERVIEW

• Atypical interstitial pneumonia (AIP) is a loosely defined term that covers a variety of respiratory disorders with different etiologies. These disorders typically present with acute respiratory distress and are characterized by the microscopic findings of pulmonary congestion or edema, hyaline membrane formation, epithelial hyperplasia, and interstitial emphysema.
• Specific causes include intoxication of cattle with 3-methyl-indole (3-MI), 4-ipomeanol (4-IP), and perilla ketone. A form of AIP also affects feedlot cattle, and other potential causes include noxious gases, *Brassica* spp. plants, perennial ryegrass intoxication and lungworm infestation.

INCIDENCE/PREVALENCE

Variable; high morbidity and mortality can occur in cattle herds when AIP is related to toxicity.

GEOGRAPHIC DISTRIBUTION

• Widespread, but specific causes have some degree of regional distribution.
• AIP related to 3-MI toxicity has been reported in the United Kingdom, Canada, and the western United States. Moldy sweet potato toxicity and perilla ketone toxicity occur in southeastern USA. Feedlot cattle in the USA, Canada and other countries can develop AIP of unknown etiology.

SYSTEMS AFFECTED

Respiratory

PATHOPHYSIOLOGY

• 3-MI toxicity results from ruminal conversion of L-tryptophan to 3-MI which is absorbed from the rumen and further metabolized in the lung, resulting in bronchiolar, alveolar epithelial and endothelial damage with pulmonary edema, hyaline membrane formation, alveolar epithelial hyperplasia, and interstitial emphysema resulting in hypoxemia and distress. Most often this arises after movement of cattle from sparse, dry feed, to lush green pastures without significant time for acclimation or feed additives provided to prevent AIP.
• 4-IP toxicity arises with the formation of furanoterpenoid toxins by sweet potatoes infected with the fungus *Fusarium solani*. Ingestion of moldy sweet potatoes by cattle allows these compounds to be absorbed from the rumen and converted in the lungs to toxic metabolites that produce bronchiolar, alveolar epithelial, and endothelial injury by irreversible binding to cellular proteins.
• Perilla ketone toxicity occurs in cattle grazing on sparse pastures in late summer in the southeastern USA (*Perilla frutescens*) and New Zealand (*Perilla maculatta*). The tall, green plants are found along the edge of wooded areas in pastures and grow well in later summer when other plants are dry. Intoxication commonly occurs in drought years when forage is limited. Consumption of volatile oil in these plants, including perilla ketone and two other substituted furans, results in comparable lung injury to 3-MI and 4-IP toxicity.
• AIP in feedlot cattle has not been fully elucidated but typically occurs in the late feeding period when animals have been consuming a high concentrate diet for some time. This is considered a multifactorial condition and specific features of feedlot rations and rumen metabolism may contribute to the formation of 3-MI and other metabolites. Mortality is highest in summer and fall, when on finishing diets. Heifers are far more susceptible than males which may relate to the use of melengestrol acetate to control estrus.

HISTORICAL FINDINGS

• Management factors relevant to these disorders include feeding of moldy sweet potatoes, access to toxic plants, exposure to noxious gases, or finishing stages in a feedlot environment. Pasture-related AIP develops within 2 weeks of sudden movement to a variety of lush pastures.

SIGNALMENT

• Adult cattle are most susceptible; typically brood cows or bulls over two years of age
• Nursing calves are generally not affected and yearlings are less susceptible
• Feedlot cattle, particularly heifers, nearing the end of their feeding period are predisposed

PHYSICAL EXAMINATION FINDINGS

• Sudden onset of severe expiratory dyspnea with open mouth breathing, ptyalism, extension of the head and neck, distress, and anxiety.
• Quiet lung sounds are noted on auscultation; coughing is uncommon.
• Collapse and sudden death can occur with exertion.
• Subcutaneous emphysema may occur.

GENETICS

• No breed predilection.
• Heifers are >3 times as likely to develop AIP in feedlot situations.

CAUSES AND RISK FACTORS

• Movement of cattle from dry summer range onto irrigated or fertilized pastures in fall.
• Feedlot environment and ration.
• Female gender (feedlot AIP).
• Mature age (>2 years).
• Access to toxic plants in times of limited feed availability.
• Consumption of moldy sweet potatoes.

DIAGNOSIS

DIFFERENTIAL DIAGNOSES

• Intoxication with 3-MI, 4-IP, and perilla ketone may appear clinically similar to one another but can usually be readily separated on review of management factors.
• Infectious respiratory diseases (particularly bovine respiratory syncytial virus).
• Hypersensitivity pneumonitis of confined adult cattle (extrinsic allergic alveolitis).
• Verminous pneumonia (*Dictyocaulus viviparus*).

CBC/BIOCHEMISTRY/URINALYSIS

Stress leukogram may be evident; however, restraint and sampling can be fatal and is not usually justified.

OTHER LABORATORY TESTS

N/A

IMAGING

Not typically indicated

OTHER DIAGNOSTIC PROCEDURES

Necropsy

PATHOLOGIC FINDINGS

• Typically lesions are confined to the respiratory system. The lungs are heavy, wet and rubbery, and do not float. The lungs may fail to collapse and maintain rib impressions after the thorax is opened. Petechial hemorrhages may be evident in the upper respiratory tract, with foamy fluid in the large airways. Incriminating feedstuffs (Perilla mint, moldy sweet potatoes) may be present in the rumen.
• Microscopic pulmonary lesions include congestion, edema, hyaline membranes, interstitial emphysema, and proliferation of alveolar epithelial cells.

TREATMENT

THERAPEUTIC APPROACH

Largely limited to preventing further exposure to offending pasture, feed, or toxins, and avoiding stress or exertion that exacerbates respiratory distress.

SURGICAL CONSIDERATIONS AND TECHNIQUES

N/A

MEDICATIONS

DRUGS OF CHOICE

• Treatment should be considered carefully and performed with caution due to associated stress.

A

ATYPICAL INTERSTITIAL PNEUMONIA (CONTINUED)

• Furosemide (1 mg/kg IM or IV, q24h or q12h).
• Flunixin meglumine (1.1–2.2 mg/kg IV, q24h or divided q12h).
• Dexamethasone (0.05 to 0.2 mg/kg IM or IV, once or twice).
• Antibiotics may be indicated to prevent secondary bacterial pneumonia.

CONTRAINDICATIONS
N/A

PRECAUTIONS
• Any stress or exertion, including treatment, can precipitate fatal collapse.
• Appropriate milk and meat withdrawal times must be followed for all compounds administered to food-producing animals.

POSSIBLE INTERACTIONS
N/A

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
• Most fatalities from toxic interstitial pneumonia are likely to occur in the first two days of clinical signs. Animals with severe disease may display chronic emphysema or signs of cardiac failure related to cor pulmonale.
• Moderately to mildly affected animals often improve substantially and spontaneously after 72 hours and continue to recover over 10 days.
• Feedlot AIP typically has a poor prognosis.

POSSIBLE COMPLICATIONS
Secondary bacterial pneumonia

CLIENT EDUCATION
Focus on preventive management factors relevant to the various disorders.

PATIENT CARE
• Exertion should be minimized; it is generally best to leave affected animals in their location and to provide alternative sources of safe feed (hay).
• Monitor progression of disease or improvement, so animals in severe distress can be euthanized before undue suffering occurs.

PREVENTION
• Management practices that prevent abrupt exposure of animals to suspect pasture can include gradually increasing pasture time over 10–12 days (commencing with 2 hours/day); strip grazing; or using other species or young stock to graze pasture down before adult cattle. Delaying use of lush pastures until after a hard frost, or cutting and windrowing pasture before turning cattle out is also preventive.
• Prophylactic administration of monensin or lasalocid (200 mg/head/day) for 1 day or 6 days respectively, prior to placing adult cattle on lush pasture can prevent disease if maintained for at least 10 days after introduction to pasture. These drugs reduce the conversion of L-tryptophan to 3-MI.
• Provide sufficient feed and minerals during late summer to limit consumption of toxic plants.
• Do not feed moldy sweet potatoes to livestock.
• Fence off access to toxic plants.

MISCELLANEOUS

ASSOCIATED CONDITIONS
• Secondary bacterial bronchopneumonia
• Viral respiratory infections

AGE-RELATED FACTORS
Young animals are resistant.

ZOONOTIC POTENTIAL
N/A

PREGNANCY
N/A

BIOSECURITY
N/A

PRODUCTION MANAGEMENT
Management has the greatest influence on the occurrence of these disorders, hence attention should be paid to minimizing all associated causes and risk factors, or where possible, employing specific preventive strategies.

SYNONYMS
• Fog fever
• Acute bovine pulmonary emphysema
• Pulmonary adenomatosis
• Acute respiratory distress syndrome

ABBREVIATIONS
• AIP = atypical interstitial pneumonia
• 3-MI = 3-methyl-indole
• 4-IP = 4-ipomeanol

SEE ALSO
Respiratory Disease: Bovine

Suggested Reading

Breeze R. Respiratory disease in adult cattle. Vet Clin North Am Food Anim Pract 1985, 1: 311–46.
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AVOCADO TOXICOSIS

BASICS

OVERVIEW
• There are approximately 150 species of avocado (*Persea*). Of these, *Persea americana* and its races and cultivars are of toxicologic importance. Races and cultivars most commonly encountered include Guatemalan and its hybrid ("Fuerte"), Mexican, and West Indies.
• The tree or shrub has a dense crown with brown to gray bark.
• Leaves, fruit, and seeds are toxic with leaves being the most toxic.
• Leaves alternate and crowd near the end of the twig. Leaf blades are ovate-elliptical with single primary vein.
• Flowers are perfect, greenish yellow.
• Fruit are ovoid to pyriform with thick glossy green to dark green skin.
• Seeds are large and light brown.

GEOGRAPHIC DISTRIBUTION
Cultivated primarily in Mexico, California and Florida, but can also be found as an ornamental in the Gulf coast areas.

SYSTEMS AFFECTED
• Mammary
• Cardiovascular

PATHOPHYSIOLOGY
• Suspected toxin is a R-enantiomer of persin. However, the mechanism of action is unknown.
• Toxin targets the mammary gland and myocardium depending on the amount of plant consumed.

HISTORICAL FINDINGS
Exposure to avocado groves

SIGNALMENT
Bovine, ovine, caprine (especially). Goats are highly susceptible to the mammary-induced effects of avocado poisoning, although all lactating animals can develop noninfectious mastitis and agalactia. With respect to cardiotoxic effects of avocado, all animal species are considered susceptible.

PHYSICAL EXAMINATION FINDINGS
• Mammary gland effects present as mastitis 24 hours post-ingestion with a 75% decrease in milk production. Milk appears to be watery and curdled.
• Myocardial effects present as edema of the neck and brisket, infrequent cough, depression, reluctance to move, leading to respiratory distress and cardiac arrhythmias.

CAUSES AND RISK FACTORS
• Presumed exposure to R-enantiomer of persin.
• Toxic dose:
  ◦ Mammary effects are seen with ingestion of 20 g of fresh leaves/ bwt (kg) in lactating goats.
  ◦ Myocardial effects were seen with ingestion of:
    ▪ 30 g of fresh leaves/bwt (kg) in lactating goats
    ▪ 25 g fresh leaves/bwt (kg) for 5 days in sheep (severe signs)
    ▪ 5.5 g fresh leaves/bwt (kg) for 21 days in sheep (chronic signs)
    ▪ 2.5 g fresh leaves/bwt (kg) for 32 days in sheep (mild signs)

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS
Other causes of mastitis and cardiac disease

CBC/BIOCHEMISTRY/URINALYSIS
• No characteristic changes on CBC
• Elevation of liver enzymes such as AST and LDH
• Elevated CK

OTHER DIAGNOSTIC PROCEDURES
Finding of plant material within gastric contents on gross pathology

TREATMENT

THERAPEUTIC APPROACH
• Remove from source
• Treatment of mastitis
• Supportive care

MEDICATIONS
N/A

CONTRAINDICATIONS
N/A

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
• Recovery possible
• Death

PREVENTION
Avoid feeding avocado plant material or grazing near avocado trees.

MISCELLANEOUS

ASSOCIATED CONDITIONS
N/A

PRODUCTION MANAGEMENT
Restrict grazing of livestock from avocado groves

ABBREVIATIONS
• AST = aspartate aminotransferase
• CK = creatine kinase
• LDH = lactate dehydrogenase

SEE ALSO
• Mastitis: No Growth
• Toxicology: Herd Outbreaks

Suggested Reading

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