Abdominal Distention in the Adult Horse

DEFINITION
Process by which the abdomen becomes enlarged leading to a change in its normal contour and shape.

PATHOPHYSIOLOGY
The accumulation of fluid, gas, or ingesta in the peritoneal GI tract and/or peritoneal cavity, precursory to abnormalities of abdominal organs, or abdomen wall abnormalities may result in the distention and/or change in shape of the abdominal contour.

SYSTEMS AFFECTED
- GI—Any condition, physical or functional, resulting in the vascular or nonvascular obstruction of the GI tract
- Cardiovascular—Fluid sequestration may lead to a decreased circulating volume and hypovolemic shock. Often compounded by the presence of vascular compromise of the GI tract leading to translocation of bacteria and/or toxins to the systemic circulation. Hemoperitoneum may occur from trauma or rupture of mesenteric vessels due to increased traction or trauma (e.g., during foaling), from any other abdominal visceras (e.g., rupture of the spleen or liver), or as a sequela to serious heart failure.
- Respiratory—Abdominal distention or hernialed abdominal visceras (diaphragmatic hernia) may lead to hypoventilation.
- Musculoskeletal/nervous/ophthalmologic/skin—Due to self-inflicted trauma secondary to abdominal pain.
- Reproductive—Hydrops and rupture of the prepubic tendon is seen in pregnant mares and both conditions will manifest as change in the abdominal contour.

SIGNS
- All horses are susceptible.
- Pregnancy mares—hystops (anytime during pregnancy), uterine torsion (mid-term), ureteroliths (postpartum), rupture of the mesocolon (postpartum) leading to hemoperitoneum and large colon torsion (postpartum).
- Rupture of the prepubic tendon occurs in older, sedentary mares in late pregnancy.
- Miniature horses—prepubic tendon rupture.
- Older horses—prepubic tendon rupture.

HISTORICAL
The clinical presentation should help the clinician differentiate between vascular and nonvascular GI obstructions and other non-GI causes of distention. Also, signalment and geographical location, time of year, and sex should provide clues toward a final diagnosis.

PHYSICAL
- Evaluate progression of clinical signs, historical facts, and cardiovascular system.
- Rectal examination is practical, inexpensive, and quick, but evaluates only the caudal abdomen.
- Abdominal ultrasound examination may help determine the location, nature, and severity of the cause of colic.

CAUSES
Accumulation of Gas
- Functional obstruction—primary ileus due to increased sympathetic drive. Secondary ileus due to pain (visceral or mucosal), ischemic necrosis (e.g., verminous arteritis), electrolyte abnormalities (e.g., hypomagnesaemia), sepsis, inflammation of the bowel (amputation) or abdominal cavity (peritonitis), and drugs (e.g., agents, morphine).
- Physical obstruction—Either vascular (large colon volvulus, mesenteric root volvulus, strangulating lipoma) or nonvascular (impaction, emphysema, nontropical enteritis).
- Cecal tympany from abnormal colic motility patterns.

Signs
- Abdominal pain are usually present.
- “Ping”; depending on the inciting cause and the location of the fluid, few to no GI sounds may be identified on percussion as a hyperresonant abdomen.
- Fluid is heard, and increased gaseous distention may be identified on percussion as a hyperresonant “ping”, depending on the inciting cause and the degree of distention present, various degrees of abdominal pain are usually present.

Accidents from right-sided heart failure—Tissue hypoxia results in findings including heart murmur, exercise intolerance, jaundice, death, and edema of the ventral abdomen, pedal muscles, and distal limbs. Progression of the disease is slower than acute abdominal distention due to GI obstruction.

Accidents from intra-abdominal mesothelioma—Because this tumor originates from the fluid-producing cells of the peritoneum, several liters of peritoneal fluid may be produced
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within a 24-hr period, acines may be more dramatic than is noted with other conditions.
• Body wall defect from prepubic tendon rupture—One of the only causes of unilateral abdominal distention in the horse, also results in cranioventral positioning of the mammary gland, cranial tilting of the pelvis, and severe segmental distention in small segments of the abdomen. Intestinal obstruction may result if the bowel becomes distended through the body wall, thereby decreasing the incidence of direct peritoneal contamination. The incision site should ideally be situated in the mid-lateral region of the most tympanitic wall.

DIAGNOSTIC PROCEDURES
• Laparoscopy permits direct visualization of the abdominal cavity in the standing horse and can be used to provide a definitive diagnosis of the cause of abdominal distention. However, it must be used carefully in cases of abdominal distention to not damage accidentally any abdominal visera upon entrance in the abdominal cavity. In the presence of GI distention, the ability of identifying the nature of the obstruction may be compromised.

Exploratory laparotomy through a flank incision in the standing horse is very limiting and should only be performed in selected cases as a therapeutic intervention if a confirmed diagnosis such as nephrosplenic entrapment or uterine torsion has been made. Exploratory laparotomy through a ventral midline incision in the anesthetized horse should not be delayed unsecuredly as it may be a life-saving diagnostic and therapeutic tool if used appropriately.

TREATMENT
• Treatment is dependent on the cause of abdominal distention.
• Cardiovascular stabilization through rehydration and correction of electrolyte and acid-base abnormalities should be initiated prior to treatment of the primary disease process.
• In horses with severe gaseous distention, trocharization of the ceacs and/or large colon may be necessary to improve ventilation. Any horse that is trocharized should be treated preemptively with broad-spectrum antibiotic therapy to reduce and minimize the inherent risk of peritonitis. The site for trocharization is situation within the paralumbar fossa and can be delineated through auscultation and percussion of the distended vescus. The author prefers the highest point possible and on the right side this may coincide with the costal attachment to the body wall, thereby decreasing the incidence of direct peritoneal contamination. The trocharization site should ideally be situated in the mid-superolateral region of the most tympanitic area. A longer catheter may be used in this procedure to ensure that the viscus is entirely decompressed. Following clipping and aseptic preparation of the site, a small (f1 cm) incision of local anesthetic should be injected into the skin and muscle layers. A 525-in. (13.5-cm) 14-gage, stiff intravenous catheter with stylet should be used for trocharization. The catheter should be inserted through the skin, muscle layers, and distended viscus with a gentle thrust. The stylet could be removed once the viscus has been penetrated and maintained until no further escaping gas is heard or fluid is seen at the hub of the catheter. The audible escape of gas confirms correct placement within the lumen of the distended viscus. In order to prevent laceration of the bowel wall, the needle/catheter should be held carefully during the decompression phase and the hand should follow gently in the direction that GI motility dictates. As the bowel becomes decompressed, the catheter may require further advancement into the lumen of the viscus. In order to prevent leakage of intestinal contents from the tip of the catheter into the peritoneal cavity, the catheter should not be withdrawn until the decompression process is complete. The catheter is then withdrawn while injecting 10 mL of procaine penicillin or gentamicin. The trocharization site should be wiped clean with alcohol.
• Horses with abdominal distention should be confined to a stall and monitored continuously until a diagnosis has been made and appropriate treatment initiated. Feed should be withheld from horses showing any signs of abdominal discomfort. Prompt and adequate referral to a hospital facility may be required in cases requiring surgical intervention or prolonged nursing care.

MEDICATIONS
Drug therapy is dictated by the causing incite.

FOLLOW-UP
Plans for monitoring are based on cause and treatment.

MISCELLANEOUS

PREGNANCY
• Termination of pregnancy may be indicated in mares with hydronephrosis or nonreflexing uterine torsion. In case of mares with hydronephrosis in the future, a different stallion should be selected.
• Induction of parturition may be necessary in mares close to term that have experienced rupture of the prepubic tendon. These mares should be monitored carefully and parruration attended as they may require assistance with delivery due to their inability to perform effective abdominal press for fetal expulsion.

SYNONYMS
Bloat

SEE ALSO
See Causes

ABBREVIATIONS
• Cr = creatinine
• GI = gastrointestinal
• PCV = packed cell volume
• SG = specific gravity
• TP = total protein
• WBC = white blood cell

SUGGESTED READING

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Consulting Editors: Henry Stampfl and Olimpio Oliver-Expósito

EQUINE, SECOND EDITION
Abdominal hernia is an exteriorization of internal organs through a defect or an anatomic opening in the abdominal wall. In adult horses, abdominal herniae include ventral, incisional, and acquired inguinal/scrotal hernia.

**Ventral Hernia**
- Most frequently seen in older, late-term pregnant mares. The draft breeds appear to be predisposed.

**Incisional Hernia**
- It is a complication of ventral celiotomy in 10%–15% of horses. No breed or sex predilection. Incisional herniation can develop up to 3 mo after ventral celiotomy; but acute form develops within 8 days after surgery.

**Acquired Inguinal/Scrotal Hernia**
- Inguinal hernia refers to the passage of intestine and/or omentum through the vaginal ring into the inguinal canal.
- Scrotal hernia describes presence of herniated contents in the scrotum.
- Distal jejunum and ileum are most frequently involved, but omentum or small colon may also herniate.
- Acquired inguinal/scrotal hernia occurs exclusively in the intact male horse but isolated cases of inguinal herniation in geldings and mares have been reported.
- Standardbred, Tennessee Walking Horses, American Saddlebreds, and draft breeds seem to be predisposed.

**Signs**

**Ventral Hernia**
- Mares with ventral hernia walk slowly and often lie down. Often, the herniae are painful and the horses have an increased heart and respiratory rates. A large swelling over the flank or caudal ventral abdomen is present. Orientation of the pelvis and the mammary gland is normal. Signs of colic may be present if the herniated content is compromised.

**Incisional Hernia**
- Brown serosanguineous discharge from the incision and progressive increase in drainage of peritoneal fluid are commonly observed prior to dehiscence. Ventral swelling developing over the abdominal incision site is observed. Gaps in the abdominal wall between sutures may be palpated.

**Acquired Inguinal/Scrotal Hernia**
- Scrotal swelling may be mild in inguinal hernia but marked in horses with scrotal hernia. The testis on the hernia side is usually firmer and cooler compared to the opposite testis.
- Abdominal pain may vary from mild to severe depending on degree of intestinal strangulation.

**Causes and Risk Factors**

**Ventral Hernia**
- In pregnant mares, old broodmares, and twin gestation. Often associated with degenerative changes in the body wall and may also herniate.
- Standardbred, Tennessee Walking Horses, American Saddlebreds, and draft breeds seem to be predisposed.

**Incisional Hernia**
- Incisional infection and swelling, postoperative endometritis and pain, repaired sutures, and use of chronic gut sutures predispose hernia formation after celiotomy.

**Acquired Inguinal/Scrotal Hernia**
- Inguinal/scrotal hernia often follows breeding activity or strenuous athletic exercise. Large vaginal rings may predispose to herniation, but it also occurs in horses with small to normal-size vaginal rings.

**Diagnosis**

**Differential Diagnoses**

**Ventral Hernia**
- Prebubic tendon rupture. Clinical signs are similar; however, the pelvis becomes tilted cranioventrally. Cranioventral displacement of the udder can lead to rupture of blood supply and blood can be observed in the milk of such mares.

**Incisional Hernia**
- Postoperative wound infection, severe peri-incisional edema, seroma, and sinus formation are easily differentiated from incisional herniae with the abdominal wall being intact on palpation and ultrasonographic examination.

**Acquired Inguinal/Scrotal Hernia**
- Torsion of the spermatic cord, infectious epididymitis or orchitis, thrombosis of the testicular artery, hydrocele, hematocele, and testicular neoplasia.

**CBC/Biochemistry/Urinalysis**
- Unremarkable in absence of secondary intestinal obstruction.
Abdominal Hernia in Adult Horses

IMAGING

Abdominal Ultrasonography

Transcutaneous abdominal ultrasonographic examination with a 3.5- or 5-MHz transducer is used to rule in herniation, to evaluate the extent of the abdominal wall defect, and to identify hernia contents. May also reveal presence of herniated intestine in acquired inguinal/scrotal hernia or rule out hydrocele, hematocele, and testicular neoplasia.

OTHER DIAGNOSTIC PROCEDURES

External Palpation

To define the hernia ring and hernia contents but is more difficult with extensive abdominal edema. Mares with ventral hernia resist deep palpation of affected area. Palpation of inguinal regions and scrotum is mandatory in stallions with signs of colic.

Rectal Palpation

Ruling out pubic tendon rupture by rectal palpation can be difficult, depending on the defect’s location and size of the fetus. Palpation of distended loops of intestine associated with abdominal pain warrants immediate exploratory laparotomy. Rectal palpation of stallions with inguinal/scrotal hernia reveals presence of a loop of intestine entering the vaginal ring. Multiple loops of distended intestine are usually palpated with intestinal obstruction.

TREATMENT

Ventral Hernia and Incisional Hernia

Ventral or incisional herniae are treated initially conservatively by supporting the ventral abdominal wall, decreasing the amount of local inflammation and edema, and preventing worsening of the condition. Afflicted horses should be rested, fed with low-bulk feed, and monitored for signs of intestinal obstruction. Abdominal pressure bandage should be applied for 24 hr a day and removed twice daily for cold (initial phase) or warm (chronic phase) hydrotherapy for 20–30 min. Ventral or incisional hernia may resolve with conservative treatment, but for the surgical closure of the abdominal defect 8–12 weeks after its occurrence is usually required. Application of a mesh will be performed based on the size of the wall defect and the surgeon’s preference. Horses with acute severe incisional dehiscence (eventration) are emergency surgical candidates.

Acquired Inguinal/Scrotal Hernia

Treatment of acute inguinal/scrotal hernia is surgical. During early phase, when intestinal strangulation has not yet occurred, it may be possible to reduce the hernia using external inguinal/scrotal massages under general anesthesia in dorsal recumbency.

MEDICATION

Ventral and Incisional Hernia

Pending surgical correction, the use of NSAIDs (phenylbutazone 2.2 mg/kg PO q12 h) is advocated to decrease abdominal edema. Parenteral broad-spectrum antibiotics are also required for incisional hernia. Resolution of incisional infection is mandatory prior to attempting surgical correction.

FOLLOW-UP

The prognosis for ventral hernia is guarded. Incisional and inguinal/scrotal herniations warrant a favorable prognosis. From 3 to 5 mo of rest is required after surgical correction of both ventral and incisional herniae.

Suggested Reading


Author

Ludovic Bouré

Consulting Editors

Henry Stämpfli and Olimpo Oliver-Espinosa
**Abdominocentesis**

**Overview**
- Procedure for sampling peritoneal fluid by collection through the abdominal wall.
- Fluid is collected into EDTA and into a sterile clot tube for bacterial culture or biochemical tests.
- Equine abdominal fluid normally appears clear and colorless to slightly yellow and does not clot.
- Total protein commonly is assessed by refractometer and normally is <2.5 g/dL.
- Nucleated cell count in fluid from normal horses is <10,000 cells/μL, with a predominance of nondegenerate neutrophils (22%–98%) and large mononuclear cells (1%–68%), which include mesothelial cells and macrophages. Small lymphocytes may comprise 0%–36% of the total and eosinophils up to 7%; mast cells and basophils rarely are seen. Normally, few erythrocytes are present.
- Biochemical measurements other than total protein may include lactate as an indicator of intestinal ischemia and creatinine and/or potassium to diagnose unabsorbed.

**Pathophysiology**
- Normal peritoneal fluid is a dialysate of plasma; many of the low-molecular-weight substances in blood are present in the peritoneal fluid at similar concentrations.
- High-molecular-weight molecules (e.g., protein) normally are not present in abdominal fluid.
- Cells in normal peritoneal fluid include mesothelial cells and small numbers of cells from the blood and lymphatics.
- Fluid circulates constantly through the abdominal cavity and is drained via lymphatic vessels. When fluid production exceeds drainage, an effusion develops.
- This may occur with some systemic disorders (e.g., cardiovascular disease) or with local disorders of abdominal organs or mesothelium. Changes in peritoneal fluid protein, cell numbers and types may reflect those disorders.
- In the face of inadequate intestinal perfusion and ischemia, anaerobic glycolysis can result in increased peritoneal fluid lactate concentration.

**Systems Affected**
- GI
- Hepatobiliary
- Hemic/lymphatic/immune
- Renal/urologic
- Cardiovascular
- Reproductive

**Signalment**
- Any breed, age, or sex

**Signs**
- Colic
- Chronic weight loss
- Abdominal distention
- Diarrhea

**Causes and Risk Factors**
- Peritonitis caused by compromised gut wall
- Hemorrhage
- Neoplasia
- Intestinal parasitism and secondary thromboembolism
- Inflammation of abdominal organs
- Breeding and foaling injuries
- Bile or urine leakage
- Postsurgical inflammation
- Abdominal abscess
- Decreased oncotic pressure
- Congestive heart failure

**Diagnosis**

**Differential Diagnosis**

- **Peritonitis**
  - Fluid is an exudate with increased nucleated cell count and a predominance of neutrophils.
  - Total protein usually is >2.5 g/dL because of inflammation.
  - Bacteria are present in septic peritonitis and may be intracellular or extracellular.
  - With gut rupture, cells often are degenerate and mixed bacterial types, ciliated protozoa and plant material may be seen.
  - Postsurgical peritonitis also produces an exudate with increased cell numbers and total protein within 24 hr. Neutrophils generally are not degenerate and no bacteria are seen. Increased RBC numbers may be seen.

- **Hemorrhage**
  - With a splenic tap, PCV is higher in abdominal fluid than in blood, and small lymphocyte numbers may be increased.
  - With hemorrhage into the abdomen, PCV of fluid is lower than that of blood. Platelets are absent, and erythrophagocytosis may be present.
  - With blood contamination at the time of sampling, fluid initially may look clear, with bloody streaks appearing during sampling. Phagocytosis of RBCs is not seen, and platelets may be present.

- **Neoplasia**
  - Diagnosis may be established on finding neoplastic cells in fluid but absence of neoplastic cells does not rule out neoplasia.
because tumor cells may not exfoliate into fluid.

**Parasitism**
Migration of parasitic larvae may be associated with increased eosinophils, but this does not occur often and is not diagnostic for parasitism.

**Uroabdomen**
- Typically, peritoneal fluid creatinine and potassium are increased compared to serum concentrations.
- Hyperkalemia, marked hyponatremia, and hypochloremia are typical but are not present in all cases.

**Ascites**
- A transudate with low cell numbers and low protein content may be present with hypoalbuminemia or lymphatic or vascular obstruction or stasis.
- Serum biochemical profile and history contribute to this diagnosis.

**Congestive Heart Failure**
Increased hydrostatic pressure within vessels may result in a modified transudate with a higher cell count and protein level than a transude, but these values may be normal for equine abdominal fluid.

**CBC/BIOCHEMISTRY/URINALYSIS**
- Inflammatory causes of abdominal effusion may be associated with leukocytosis or hyperfibrinogenemia if disease is systemic.
- Left shift or toxic changes in neutrophils indicate systemic inflammation.
- Serum biochemistries help to assess causes of transudates—hypoproteinemia is consistent with GI protein loss; elevated liver enzymes suggest hepatic disease.
- Serum electrolytes and comparison of serum and fluid creatinine aid in diagnosis of uropertoneum.

**OTHER LABORATORY TESTS**
Bacterial culture is helpful in some cases, such as abdominal abscess.

**IMAGING**
- Ultrasonography: May be used to look for intestinal entrapment, intussusception, masses, adhesions, enlarged liver, and enteroliths.
- Ultrasonographic location of peritoneal fluid sometimes helps in performing abdominocentesis.

**Abdominal Radiography**
In adult horses, may aid in establishing the diagnosis of diaphragmatic hernia, sand, and enteroliths.

**OTHER DIAGNOSTIC PROCEDURES**
- Laparoscopy may be used to establish the diagnosis in cases of chronic colic or weight loss.
- Gastroscopy can be useful in establishing the diagnosis of gastric ulcers, impaction, and neoplasia.
- Exploratory laparotomy is necessary for definitive diagnosis in some cases.

**TREATMENT**
Directed at the underlying cause

**MEDICATIONS**
None

**FOLLOW-UP**

**POSSIBLE COMPLICATIONS**
Accidental enterocentesis (rarely associated with clinical disease) causes increased nucleated cell count in abdominal fluid within 4 hours.

**MISCELLANEOUS**

**AGE-RELATED FACTORS**
Foals normally have protein levels similar to peritoneal fluid cell counts (<1500 cells/μL) but lower than adults.

**PREGNANCY**
No significant differences in fluid from mares that are pregnant or have recently foaled compared with fluid from nonperipartum mares.

**ABBREVIATIONS**
- GI = gastrointestinal
- PCV = packed cell volume

**Suggested Reading**

**Author** Susan J. Torquigiat
**Consulting Editor** Kenneth W. Hinchcliff
DEFINITION/OVERVIEW

Estrus—period of sexual receptivity by the mare for the stallion.
- Abnormal—individual's overt display of sexual behavior for longer or shorter periods than normal.
- Abnormal interestrus intervals result from endocrine, infectious, parasitic, or behavioral causes.

ETIOLOGY/PATHOPHYSIOLOGY

- Mare—seasonally polyestrous in spring and summer months.
- Average estrus cycle—21 days (range: 19–22); period of time between ovulations.
- Estrus coincides with P4 levels < 1 ng/mL. Estrus and estrus cycle lengths repeatable in individual mare from cycle to cycle.

Key Hormonal Events in the Equine Estrus Cycle

- FSH causes ovarian follicular growth.
- Estriol (E2) stimulates increased GnRH.
- Seasonal influence—Individual variation.
- Clitoral size—Enlargement may be indicative of hormonal abnormalities.
- Uterine disease—Uterine inflammation may cause endometrial PGF2α release resulting in prolonged CL activity, affecting fertility.

Physical Examination

- In normal, cycling mares—average 5–7 days; range 2–12 days.
- Diestrus Length—Less variable than estrus in normal, cycling mares, averaging 15 +/- 2 days.

Sexual Behavior

- Abnormal estrus behavior—Individual variation.
- Normal cyclic ovarian activity but minimal or no overt sexual receptivity may be ineffective LH release.

SYSTEMS AFFECTED

- Reproductive
- Behavioral
- Endocrine

SIGNALLMENT

- Any breed • Mares of any age > 20 years tend to have prolonged transition periods; > estrus duration, fewer estrus cycles per year • Mares may have longer estrus cycles than horses (average 25 days).

SIGNS

- Historical
- Chief complaints—infertility, failure to show estrus, prolonged estrus, silent estrus, or frequent estrus behaviors • Racing records—Review methods, frequency, trainer type (pony, horse, gelding), stallion behavior, aggressive/ passive, vocalization, proximity); and handler experience.
- Seasonal influence—Individual variation (oestrus/duration/termination of cyclicity) can be mistaken for estrus irregularity. • Mare’s reproductive history—Can clinical abnormalities be linked to estrus cycle length, mating, foaling, previous injuries, or genital infections.
- Pharmacological—Clinical abnormalities related to current and historical drug administration.

Physical Examination

- Body condition—Poor condition/malnutrition (label dosing) inhibits spontaneous formation and release of endogenous PGF2α.
- Clinical abnormalities related to current and historical drug administration.
- Gonadal dysgenesis due to chromosomal defects (e.g., XO, XXX) may cause atypical endogenous PGF2α release, leading to prolonged CL activity.

CAUSES

Abnormal Estrus Intervals

- Shortened Estrus Duration
- Early embryonic death after maternal recognition of pregnancy.
- Late luteolysis and concurrent decline in P4 levels.

- Shortened Interestrus Interval
- May mimic or be linked to estrous cycle length, teasing, foaling, or previous injuries, or genital infections?

- Lengthened Estrus Duration
- Early embryonic death after maternal recognition of pregnancy.
- Late luteolysis and concurrent decline in P4 levels.

- Lengthened Interestrus Interval
- Late luteolysis and concurrent decline in P4 levels.

DIFFERENTIAL DIAGNOSIS

Differential Conditions with Similar Symptoms

- Frequent urination
- Cystitis/urethritis
- Uterine disease—Uterine inflammation may cause endometrial PGF2α release resulting in prolonged CL activity.

- No evidence that chronic PGF2α treatment (label dosing) inhibits spontaneous formation and release of endogenous PGF2α.
- Gonadal dysgenesis due to chromosomal defects (e.g., XO, XXX) may cause atypical endogenous PGF2α release, leading to prolonged CL activity.

- Diagnosis—TRP minimum 3 per week, may be needed (several weeks) to define her estrous cycle.

- U/S–affected ovary—multilocular.
Abnormal Estrus Intervals

**MEDICATIONS**

**DRUG(S) OF CHOICE**

• PGF2α (10 mg IM) or its analog to lycost CL tissue.

• If follicles ≥ 35 mm—deslorelin 2.1 mg implant SC or hCG (2500 IU IV) can stimulate ovulation.

• Alternogest (0.064 mg/kg PO daily, minimum 15 days) can be used to shorten the duration of normal transition, provided multiple follicles ≥ 20-mm diameter are present and the mare is demonstrating behavioral estrus.

• PGF2α (10 mg IM) on day 15 of the alternogest treatment increases the reliability of this transition management regimen.

**CONTRAINDICATIONS**

PGF2α and its analogs—contraindicated in mares with luteomas, or other bronchocutaneous disease.

**PRECAUTIONS**

• Hormone

• PGF2α causes urethral and colic-like symptoms due to its stimulatory effect on smooth muscle cells. If cramping has not subsided within 1–2 h, symptomatic treatment should be instituted.

• Antibodies to hCG can develop after hCG use in nonpregnant mares. The antibodies cross-react with hCG and its analogs—contraindicated in pregnant mares with heaves, or other bronchocutaneous disease.

**TREATMENT**

• Vary teasing methods—Silent estrus may be a reflection of poor teasing management.

• Monitor the problem mare, including TRP and U/S–endocrine assays.

• If follicles are persistent, or luteomas are identified, then the problem mare should be treated for at least 30 days, then retreated in the next season. The half-life of these antibodies ranges from 30 days to several months; typically do not persist from one breeding season to the next.

• Deslorelin implants—associated with suppressed FSH secretion and decreased follicular development in the diestrus period immediately following use; results in a prolonged interovulatory period in nonpregnant mares. Implant removal post-ovulation is recommended. Injectable product still available in the United States. Alternogest, deslorelin, and PGF2α or its analog should not be used in horses intended for food.

• Immune pooling/aversion—Vaccines and vaginal discharge—Vulvar conformation

**ABBREVIATIONS**

• CL = corpus luteum

• E2 = estradiol

• FSH = follicle-stimulating hormone

• GCT = granulosa cell tumor

• GnRH = gonadotropin-releasing hormone

• GTCT = granulosa theca cell tumor

• hCG = human chorionic gonadotropin

• LH = luteinizing hormone

• PGF2α = prostaglandin F2α

• PMS = pseudopregnancy

• PVE = prostaglandin analog

• PROG = progesterone

• U/S = ultrasonography

**Suggested Reading**


### Abnormal Scrotal Enlargement

#### Basics

**Definition/Overview**
A condition causing the gross appearance of the scrotum to deviate from normal size and texture, e.g., scrotal enlargement and/or asymmetry.

**Etiology/Pathophysiology**
- Equine scrotum and associated contents are positioned on a horizontal axis between the hind limbs of the animal and are relatively well protected from external insult.
- Scrotal skin is thin and pliable, and contents are freely movable within the scrotum.
- Blunt trauma (breeding accidents, jumping) is the most common cause of scrotal abnormality.
- Trauma can result in scrotal hemorrhage, edema, rupture of the tunica albuginea, hematoma, hydrocoele, and inflammation.
- Similar signs can occur with inguinal/scrotal herniation, torsion of the spermatic cord, or neoplasia.

**System Affected**
Reproductive

**Genetics**
N/A

**Incidence/Prevalence**
Dependent on cause—traumatic, vascular, infectious/noninfectious, neoplastic

**Signs**
- Intact male horses
- Any age

**Historical**
- Gross changes in the size of the scrotum (usually acute)
- Pain (generally colic-like symptoms)
- Refusal to bred, jump, or walk
- Extreme environmental temperatures (hot or cold)

**Physical Examination**
- Increased scrotal size (unilateral or bilateral)
- Abnormal testicular position
- Abnormal scrotal temperature (too warm or cold)
- Edema/engorgement of scrotum and/or contents
- Scrotal laceration
- Derangements in systemic parameters (elevated HR, RR, inappetence, CBC abnormalities)
- Any combination of abnormalities may be present and not all signs are present in every animal.

**Diagnosis**

**Differential Diagnosis**
- Duration of problem
- Acute—traumatic injury, torsion of spermatic cord, herniation, infection
- Chronic—neoplasia, temperature-induced hydrocoele, edema, varicocele, infection
- History of recent breeding, semen collection, and/or trauma
- Palpation of the caudal ligament of the epididymis (attaches epididymal tail to caudal tests and aids in the determination of testicular orientation)
- Palpation of the inguinal rings
- US (see Imaging)

**CBC/Biochemistry/Urinalysis**
- Inflammatory or stress leukocyte response
- Increased fibrinogen
- Results of serum biochemistry profile and urinalysis are usually normal

**Other Laboratory Tests**
- EIA
- AGID or ELISA, the Coggins test
- Palpation of the inguinal rings
- History of recent breeding, semen collection, and/or trauma
- Palpation of the caudal ligament of the epididymis
- Results of serum biochemistry profile and urinalysis are usually normal
- Virus isolation from serum and/or seminal plasma

**Treatment**
- Management of inflammation is a primary concern with abnormal scrotal enlargement.
- Chronic scrotal enlargement may or may not warrant hospitalization, etiology dependent

**Inpatient or Outpatient Treatment?**
- Depends on cause—traumatic, vascular, infectious/noninfectious, neoplastic
- Primary scrotal—melanoma, sarcoid
- EVA/EAV
- Orchitis/epididymitis
- Neoplasia
- Primary scrotal—melanoma, sarcoid
- Testicular neoplasia—seminoma, teratoma, interstitial cell tumor, Sertoli cell tumor
- Noninflammatory scrotal edema
- Vasculitis
- See also: Abnormal Testicular Size

**Inflammation**

**Scrotal Enlargement**

**Other Diagnostic Procedures**
- Needle aspirate and cytology—to differentiate hydrocoele from recent hemorrhage
- Neoplasia—diagnosed using fine needle aspirate and/or biopsy

**Pathological Findings**
Dependent on etiology

**Imaging—Scrotal US**
Examination of scrotal contents may reveal:
- Bowel with inguinal/scrotal herniation
- Rupture of the tunica albuginea
- Accumulation of hypoechoic fluid in scrotum with loss of discrete hyperechoic tunica albuginea around testicular parenchyma
- Hypoechoic appearance of contents will gradually contain echogenic densities with the formation of fibrin clots.
- Engagement of the pampiniform plexus and/or testicular congestion with torsion of the spermatic cord
- Doppler can verify loss of blood flow to the testis.
- Hypoechoic dilution of venous plexus of spermatic cord with varicocele
- Hyperechoic accumulation of fluid within the vaginal cavity with hydrocoele
- Loss of homogeneity in testicular parenchyma with neoplasia
- May see areas of increased or decreased echogenicity or be variable throughout

**Appropriate Health Care**

**Inpatient or Outpatient Treatment?**
- Acute scrotal enlargement warrants hospitalization for treatment and care.
- Chronic scrotal enlargement may or may not warrant hospitalization, etiology dependent

**CAUSES**
- Three most common:
  - Trauma, may include testicular hematoma/rupture
  - Inguinal/scrotal hernia
  - Torsion of the spermatic cord, also known as testicular torsion

**Inflammatory/infectious causes:**
- EIA
- EVA/EAV
- Orchiepididymitis
- Neoplasia
- Primary scrotal—melanoma, sarcoid
- Testicular neoplasia—seminoma, teratoma, interstitial cell tumor, Sertoli cell tumor
- Noninflammatory scrotal edema
- Vasculitis
- See also: Abnormal Testicular Size

**Risk Factors**
- Breeding activity
- Reluctance to breed, jump, or walk
- Torsion of the spermatic cord, also known as testicular torsion
- Any age

**Duration of Problem**
- Chronic scrotal enlargement may or may not warrant hospitalization, etiology dependent

**Management of inflammation is a primary concern with abnormal scrotal enlargement.**
- Chronic scrotal enlargement may or may not warrant hospitalization, etiology dependent

**EIA**
- Hypoechoic accumulation of fluid within the vernix cavity with hydrocoele
- Hypoechoic dilution of venous plexus of spermatic cord with varicocele
- Hyperechoic accumulation of fluid within the vaginal cavity with hydrocoele
- Loss of homogeneity in testicular parenchyma with neoplasia
- May see areas of increased or decreased echogenicity or be variable throughout

**Other Diagnostic Procedures**
- Needle aspirate and cytology—to differentiate hydrocoele from recent hemorrhage
- Neoplasia—diagnosed using fine needle aspirate and/or biopsy

**Pathological Findings**
Dependent on etiology

**Imaging—Scrotal US**
Examination of scrotal contents may reveal:
- Bowel with inguinal/scrotal herniation
- Rupture of the tunica albuginea
- Accumulation of hypoechoic fluid in scrotum with loss of discrete hyperechoic tunica albuginea around testicular parenchyma
- Hypoechoic appearance of contents will gradually contain echogenic densities with the formation of fibrin clots.
- Engagement of the pampiniform plexus and/or testicular congestion with torsion of the spermatic cord
- Doppler can verify loss of blood flow to the testis.
- Hypoechoic dilution of venous plexus of spermatic cord with varicocele
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**Other Diagnostic Procedures**
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- Neoplasia—diagnosed using fine needle aspirate and/or biopsy

**Pathological Findings**
Dependent on etiology
Abnormal Scrotal Enlargement

NURSING CARE
- Cold therapy (cold packs, ice water baths, water hose) for acute scrotal trauma is implemented only in the absence of testicular rupture.
- Testicular tunics must be intact.
- Cold therapy sessions should not exceed 20 min and can be repeated every 2 hr.
- Scrotal massage with emollient salve—useful to reduce scrotal edema and ischemic injury
- Fluid removal should be considered with hydrocele.
- Use only an aseptically placed needle or an IV catheter.
- Excess fluid accumulation may cause thermal damage to the testes.
- Administration of IV fluids is dependent on systemic status of the horse.

ACTIVITY
The need to restrict activity depends on etiology of scrotal enlargement.

DIET
Diet modification is necessary only with secondary ileus or as a preoperative consideration.

CLIENT EDUCATION
- Fertility may be irreversibly impaired with acute scrotal trauma.
- Semen evaluation should be performed 90 days after nonsurgical resolution of scrotal enlargement.
- Compensatory semen production may occur in the remaining tests of a horse undergoing hemicastration.
- Following removal of a neoplasia, examine carefully for evidence of metastatic tumor growth (serial examinations).

SURGICAL CONSIDERATIONS
- Hemicastration is the treatment of choice for:
  - Torsion of the spermatic cord, if the duration of vascular compromise has caused irreversible damage and/or gonadal necrosis
  - Unilateral inguinal/scrotal herniation
  - Testicular rupture
- Unilateral neoplasia
- Vaniocerc
- Nonresponsive hydrocele/hematocoele
- Primary repair of scrotal laceration is required to protect scrotal contents.
- Repair generally fails due to extensive scrotal edema associated with traumatic injury.

MEDICATIONS

DRUG(S) OF CHOICE
- Anti-inflammatory therapy (phenylbutazone 2–4 mg/kg PO or IV BID or flunixin meglumine 1 mg/kg IV BID) indicated in all cases
- Diuretics (furosemide 0.5–1 mg/kg IV) may be useful in managing scrotal edema.
- Antibiotic therapy should be considered in cases of scrotal laceration or scrotal hemorhage.
- Tetanus toxoid should be administered for scrotal trauma or prior to surgery.

CONTRAINDICATIONS, PRECAUTIONS, POSSIBLE INTERACTIONS, ALTERNATIVE DRUGS
N/A

FOLLOW-UP

PATIENT MONITORING
Semen collection and evaluation 90 days after complete resolution of cause and/or surgery

PREVENTION/AVOIDANCE
N/A

POSSIBLE COMPLICATIONS
- Infertility
- Endodermia
- Laminitis
- Scrotal adhesions
- Death

EXPECTED COURSE AND PROGNOSIS
N/A

MISCELLANEOUS

ASSOCIATED CONDITIONS, AGE-RELATED FACTORS, ZOONOTIC POTENTIAL, PREGNANCY, SYNONYMS
N/A

SEE ALSO
- Abnormal testicular size

ABBREVIATIONS
- AGID = agar gel immunodiffusion
- CBC = complete blood count
- CF = complement fixation
- EIA = equine infectious anemia
- ELISA = enzyme-linked immunosorbent assay
- EVA = equine viral arteritis
- EAV = equine arteritis virus
- HR = heart rate
- RR = respiratory rate
- SN = serum neutralization
- TRP = transrectal palpation
- U/S = ultrasound, ultrasonography

Suggested Reading

Author Margo L. Macpherson
Consulting Editor Carla L. Carleton
A primary testicular neoplasia may be affected subsequent to metastasis of horse orchitis/epididymitis. Trauma, torsion of the spermatic cord, or activity. Protected from external insult, are at increased within the scrotum. limbs of the horse and are freely movable in a horizontal orientation between the hind.

signalment

incidence/prevalence

manifestations pathogenesis: may be presented by... ...abnormalities). Expected result of neoplasia.

diagnosis

differentiation and... ...found. Suspected causes include genetic... ...be compromised. Elevated FSH and...

other diagnostic procedures

needle aspirate and cytology—diagnose... ...inflammation, testicular degeneration, and/or parasitic infection. Increased fibrinogen in peripheral blood. Serum biochemistry profile and urinalysis are usually normal.

other laboratory tests

cbc/biochemistry/urinalysis

other diagnostic procedures

needle aspirate and cytology—diagnose... ...inflammation, testicular degeneration, and/or parasitic infection. Increased fibrinogen in peripheral blood. Serum biochemistry profile and urinalysis are usually normal.

other diagnostic procedures

needle aspirate and cytology—diagnose... ...inflammation, testicular degeneration, and/or parasitic infection. Increased fibrinogen in peripheral blood. Serum biochemistry profile and urinalysis are usually normal.
**PATHOLOGICAL FINDINGS**

N/A

**TREATMENT**

Treatment is directed at the cause of testicular abnormality.

**APPROPRIATE HEALTH CARE**

Inpatient versus Outpatient

- Most cases of testicular enlargement require hospitalization for treatment/resolution.
- Horses with testicular degeneration that are not systemically ill may be managed on the farm.
- Horses with hypoplastic testes can be managed on an outpatient basis.

**NURSING CARE**

- Cold therapy (cold packs, ice water baths, water hose/hydrotherapy) is indicated for acute orchitis/epididymitis.
- Cold therapy sessions should not exceed 20 min and can be repeated every 2 hr.
- Sexual rest is indicated in most cases until resolution of the problem.
- Administration of IV fluids is dependent on systemic status of the horse.

**ACTIVITY**

Restriction depends on cause of the testicular alteration.

**DIET**

Modification is necessary only with cases of secondary etiology or as a preoperative consideration.

**CLIENT EDUCATION**

- Fertility may be permanently lowered.
- Testicular degeneration and subsequent reduction in semen quality can be transient or permanent, depending on the inciting cause.
- Testicular hypoplasia is a permanent condition.
- Horses with neoplasia should be examined carefully for evidence of metastatic tumor growth.
- Compensatory sperm production may occur in the remaining testis of a horse undergoing hemicastration.
- Serial semen evaluations are beneficial to monitor fertility status of horses following testicular insult and treatment.

- Senes should be evaluated 75–90 days after complete resolution of testicular insult.

**SURGICAL CONSIDERATIONS**

- Hemicastration is the treatment of choice for:
  - Torsion of the spermatic cord, if the duration of vascular compromise has caused irreversible damage and/or gonadal necrosis
  - Testicular rupture
  - Unilateral neoplasia or any condition causing irreparable damage to testis/es

**CONTRAINDICATIONS PRECAUTIONS, ALTERNATIVE DRUGS**

N/A

**MEDICATIONS**

**Drugs of choice**

- Anti-inflamatory therapy (phenylbutazone 2–4 mg/kg PO or IV BID or flunixin meglumine 1 mg/kg IV BID) is indicated in most cases.
- Antibiotic therapy should be considered in cases of orchitis/epididymitis and testicular trauma.
- Tetanus toxoid should be administered after testicular trauma and/or prior to surgery.
- Antiparasitic therapy for Strongylus edentatus is indicated in most cases.
- Antihelminthic therapy for Strongyloides stercoralis infection (ivermectin 0.2 mg/kg PO q30days until resolution of lesions)

**CONTRAINDICATIONS PRECAUTIONS, POSSIBLE INTERACTIONS, ALTERNATIVE DRUGS**

N/A

**FOLLOW-UP**

**PATIENT MONITORING**

Semen collection and evaluation 90 days after complete resolution of testicular problem and/or surgery

**POSSIBLE COMPLICATIONS**

- Infertility/subfertility
- Endotoxemia
- Seroadhesions
- Death

**EXPECTED COURSE AND PROGNOSIS**

Dependent on etiology

**MISCELLANEOUS**

**ASSOCIATED CONDITIONS**

- Cryptorchidism is commonly associated with testicular hypoplasia.
- Male equine hybrids (mules or hinnies) often have hypoplastic testes.

**AGE-RELATED FACTORS**

- Prepubertal testes are small and can be misdiagnosed as pathologically hypoplastic.
- Testicular growth increases rapidly from 12 to 24 mo of age in horses.
- Testes may take 4–5 years to reach full size and maturity.

**ZOOONOTIC POTENTIAL**

N/A

**PREGNANCY**

N/A

**SYNONYMS**

N/A

**SEE ALSO**

- Cryptorchidism
- Abnormal sexual enlargement

**ABBREVIATIONS**

- AGID = aga-gel immunodiffusion
- CBC = complete blood count
- CF = complement fixation
- EAV = equine arteritis virus
- ELISA = enzyme-linked immunosorbent assay
- EIA = equine infectious anemia
- EIAV = equine infectious anemia
- HR = heart rate
- LH = luteinizing hormone
- RR = respiratory rate
- SN = serum neutralization
- US = ultrasound, ultrasonography

Suggested Reading


Author: Margo L. Macpherson
Consulting Editor: Carla L. Caletto
# Definition

Fetal loss >40 days; maternal, placental, or fetal invasion of microorganisms

## Pathophysiology

- Fetal death by microorganisms
- Fetal expulsion after placental infection, insufficiency, or separation
- Premature parturition by microbial toxins, fetal stress, combination mechanisms
- Result: fetal absorption, maceration, autolysis; live fetus incapable of extrauterine survival

## Systems Affected

- Reproductive
- Other organ systems if maternal systemic disease

## Incidence/Prevalence

- 5%–15% infectious abortion
- Abortion storm, especially EHV-1

## Signalment

Nonspecific, associated specific risk factors

## Signs

**General Comments**
- Early pregnancy loss unobserved often termed asymptomatic
- Unless complications occur, abortion may occur rapidly; sole sign is relatively normal, previously pregnant mare later found open
- Signs—none to multisystemic and life-threatening
- May be multiple animals
- Most asymptomatic spontaneous infectious abortions are in second half of gestation

## Causes

### Viruses
- EHV-1 (1P and 1B strains); EHV-4 > 7 mo of gestation; rarely EHV-2
- EVA (> 3 mo of gestation)
- EIA—direct causal relationship not yet established
- Vesivirus—recent correlation between antibodies of vesivirus and equine abortion

### Bacteria
- Placentitis and possible, subsequent fetal infection by *Streptococcus sp.*, *Actionobacillus sp.*, *Escherichia coli*, *Nocardia asteroides* (to include *Amycolatopsis sp.*, *Cellulosimicrobium sp.*, *Crossiella sp.*, and *Rhodococcus sp.*), *Taylorella equigenitalis* (rare, reportable), and *Lepomis serowus*
- Endotoxemia cause release of PGF<sub>2α</sub> (especially < 80 days of gestation [day 60 in many mares]; may be factor later in gestation, if repeated exposure)
- Exposure to ETC setae in conjunction with MRSL theorized associated with microscopic bowel puncture and bacteremic spread to fetus and/or placenta
- Rickettsiae
- *Ehrlichia risticii*—PHF

### Protozoa
- *Sarcocystis neurona* or, possibly, *Neospora sp.* in aborted fetuses from EPM affected mares
- *MRLS*
- Early (≈40–150 days’ gestation) and late (> 269 days of gestation) abortion syndromes
- Association with ETCS
Abortion, Spontaneous, Infectious

RISK FACTORS
- Pregnant mares intermixed with young horses or horses-in-training are susceptible to EHV-1, EVA, or *Ehrlichia risticii*.
- Immunologically naïve mares brought to premises with enzootic EHV-1, EVA, *Ehrlichia risticii*, or *Leptospira* infections
- Pregnant mares traveling to horse shows or competitions
- Poor perineal conformation—predisposes mares to bacterial or fungal placentitis and, possibly, subsequent fetal infection
- Concurrent maternal GI disease or EPM
- Large numbers ETCs in pastures with pregnant mares
- Geographical location with respect to MRLS and nocardioform placentitis

DIAGNOSIS
- Except for placentitis and abortion secondary to endotoxemia, most abortions are asymptomatic; expelled fetus and fetal membranes vary in condition—intact to autolytic
- Definitive causative diagnosis of equine abortion in ∼50%–60% of all cases
- Excluding twins and EHV-1, diagnostic rate may approach only 30% if limited samples are submitted and accompanied by moderate to severe fetal and placental autolysis.

DIFFERENTIAL DIAGNOSIS
Other Causes of Abortion
- Abortion, spontaneous, noninfectious
  - Twinning
  - Fetal abnormalities—teratogenesis
  - Umbilical cord abnormalities—excessive twisting; thrombosis
  - Placental pathology
- Maternal malnutrition, other noninfectious systemic disease
- Old mare, history of EED or abortion
- Old mare, poor endometrial biopsy (inflammation, fibrosis)
- Endophyte-infected tall fescue pasture, exposure to ergotized grasses, small cereal grains during last month of gestation—no mammary development (agalactia, if term is reached); phytoestrogens; xenobiotics

Other Causes—Signs of Labor or Abdominal Discomfort
- Normal parturition
- Dysuria unassociated with abortion
- Periparturient uterine artery rupture
- Colic associated with uterine torsion
- Discomfort associated with hydrops of fetal membranes or prepubic tendon rupture
- Colic unassociated with reproductive disease

Other Causes—Vulvar Discharge
- Normal estrus
- Endometritis
- Metritis or partial RFM
- Mucometra or pyometra

CBC/BIOCHEMISTRY/URINALYSIS
Determine inflammatory or stress leukocyte response, other organ system involvement

OTHER LABORATORY TESTS
Maternal Progesterone
- Indicated if pregnancy outcome is doubtful (prediagnosis of an infectious cause of impending abortion), with suspected endotoxemia
- ELISA or RIA for progesterone may be useful at <30 days of gestation (normal levels vary from >1 to >4 ng/mL, depending on reference lab).

Maternal Uterine Swabs
May aid in establishing diagnosis of abortions caused by placentitis

Other Maternal Hormones
- See Abortions, noninfectious.

Maternal Serology
- Take serum samples in all cases of abortion in which cause is unknown. Paired sample (21 days later), may be indicated.
- Diagnostic for abortions by *Leptospira* serovars
- Confirms EVA abortion

IMAGING
Transrectal and Transabdominal U/S
- Evaluate fetal viability, placentitis, alterations in appearance of amniotic and/or allantoic fluids.

OTHER LABORATORY TESTS
Pathology, Serology, Molecular Techniques, and Culture
- Fresh/chilled fetal thoracic or abdominal fluid, serum from fetal heart or cord blood, if available
- Fetal stomach content
- 10% Formalin-fixed and chilled/frozen samples of fetal membranes (allantochorion; allantoamnion), fetal heart, lungs, thymus, liver, kidney, lymph nodes, thymus, spleen, adrenal, skeletal muscle, and brain

Molecular Techniques
- Specific PCR, other molecular analyses, various samples for selected viral infections

Maternal Uterine Swabs
May aid in establishing diagnosis of abortions caused by placentitis
### Abortion, Spontaneous, Infectious

#### Pathological Findings

**Viruses**
- EHV
  - Gross—pleural effusion, ascites, fetal icterus, pulmonary congestion and edema; 1-mm, yellowish-white spots on enlarged liver; fetus is fresh.
  - Histopath (EHV-1 and -4)—areas of necrotic, prominent, eosinophilic, intranuclear inclusion bodies in lymphoid tissue, liver, adrenal cortex, and lung as well as a hyperplastic, necrotizing bronchitis; FA staining of fetal tissues; virus isolation from aborted fetus
  - EVA
    - Few gross lesions
    - Autolyzed fetus
    - Placental/fetal vascular lesions
- Vesivirus
  - Nonspecific lesions

**Bacteria and Fungi**
- Fetal infection and placentitis
  - Gross—pleural effusion, ascites; enlarged liver; rare plaques of mycotic dermatitis; placental edema and thickening with fibrinonecrotic exudate (chorionic surface), especially at cervical star (especially if fungal)
  - Histopath—inflammatory disease; autolysis may make interpretation difficult
- Leptospirosis
  - Gross—fetal icterus and autolysis
  - Histopath—nonspecific; mild, diffuse placitis

#### Treatment

**Appropriate Health Care**
- Except late-gestational placentitis (>270 days) and endotoxemia, no therapy indicated to preserve fetal viability with spontaneous, infectious abortion
- Aborting mares—only prophylactic therapy for metritis or endometritis. Therapy limited to intrauterine, may include a systemic component
- Preventing GI disease and complications may warrant hospitalization and intensive care

**Nursing Care**
- Most affected horses require limited nursing care, except for endotoxemia and gram-negative septicemia, RFM, metritis, and laminitis.

**Activity**
- Paddock exercise to permit observation

#### Client Education
- Inform owners of possible complications of abortion

#### Medications

**Drug(s) of Choice**
- Altrenogest 0.044–0.088 mg/kg PO daily—start later during gestation, continue longer, or use only short periods of time depending on serum progesterone levels during first 80 days of gestation, clinical circumstances, risk factors, clinician preference. Note—Serum levels reflect only endogenous progesterone, not exogenous/oral product.
  - If near term, altrenogest frequently is discontinued 7–14 days before foaling date unless indicated otherwise by fetal maturity/viability, or actual gestational age is in question.

#### Contraindications
- Altrenogest only used to prevent abortion in cases of endometritis or placentitis (>270 days of gestation) if fetus is viable.

#### Precautions
- Altrenogest—absorbed through skin, wear gloves and wash hands

#### Alternative Drugs
- Injectable progesterone (150 to 500 mg oil base IM)

#### Follow-up

**Patient Monitoring**
- 7–10 days postabortion—TRP and U/S, monitor uterine involution
- Assess genital tract health—vaginal speculum, urethral speculum, uterine culture and cytology, endometrial biopsy
- Base treatment on clinical results. Urine culture <14 days postpartum or postabortion is affected by contaminants at parturition

#### Prevention/Avoidance

**Vaccines**
- A killed-virus EHV-1 vaccine, 5, 7, and 9 mo of gestation; approved for abortion prevention in pregnant mares; 2-mo interval due to short-lived vaccinal immunity
  - EVA vaccine; not specifically labeled for abortion prevention
    - MLV
    - Only use pregnant mares 3 weeks before anticipated exposure to infected semen or in enzootic conditions
    - Isolate first-time vaccinated mares, 3 weeks after exposure to infected semen.
    - Some countries forbid importation of horses with titers to EVA
**Additional Prophylactic Steps**

- Segregate pregnant mares from horses susceptible/exposed to infections.
- Isolate immunologically naive individuals until immunity to enzootic infections is established/enhanced. Depending on infectious agent, protection may only be accomplished postpartum.
- Limit transport of pregnant mares to exhibitions or competitions.
- Isolate aborting mares, proper disposal of contaminated fetal tissues
- Proper diagnostics to ID infectious cause
- Correct poor perineal conformation, prevent placentitis.
- Prevent pregnant mare exposure to ETCs until 7–8 weeks after ETC death.
- Insecticides to control ETCs; consider toxicity of insecticides.

**POSSIBLE COMPLICATIONS**

Future fertility and reproductive value impaired by dystocia, RFM, endometritis, laminitis, septicemia, trauma to genital tract

**EXPECTED COURSE AND PROGNOSIS**

- Most patients recover with appropriate treatment.
- Complications—significant impact on mare’s survivability and future fertility
- Prognosis—guarded for pregnancy maintenance with endotoxemia and placentitis

**ASSOCIATED CONDITIONS**

- Abortion, noninfectious
- Dystocia
- Endometritis
- EPM
- EVA
- Metritis
- Pericarditis, MRLS
- Placental insufficiency
- Placentitis
- PHF
- Premature placental separation
- RFM

**AGE-RELATED FACTORS**

Immunologic status of young mares

**SEE ALSO**

- Abortion, noninfectious
- Dystocia
- Endometrial biopsy
- Endometritis
- Fetal stress/stability
- High-risk pregnancy
- Metritis
- Placental insufficiency
- Placentitis
- Premature placental separation
- RFM

**ABBREVIATIONS**

- EED = early embryonic death
- EHV = equine herpesvirus
- EIA = equine infectious anemia
- ELISA = enzyme-linked immunosorbent assay
- EPM = equine protozoal encephalomyelitis
- ETC = eastern tent caterpillar
- EVA = equine viral arteritis
- FA = fluorescent antibody
- MRLS = mare reproductive loss syndrome
- PCR = polymerase chain reaction
- PHF = Potomac horse fever
- RIA = radioimmunoassay
- RFM = retained fetal membranes/placenta
- TRF = transectal palpation
- U/S = ultrasound, ultrasonography

**Suggested Reading**


**Author** Tim J. Evans

**Consulting Editor** Carla L. Carleton
**BASICS**

**DEFINITION**
Fetal loss >40 days (term, stillbirth may apply >300 days) associated with a variety of noninfectious conditions.

**PATHOPHYSIOLOGY**
- Fetal death/premature parturition from some intrinsic structural or functional defect or exposure to xenobiotics
- Fetal expulsion <80 days of gestation after CL loss as a result of endometritis or other factors
- Fetal death/expulsion by placentomal insufficiency or separation
- Fetal stress, dead twin fetus, maternal stress, or combination
- Fetal resorption, mummification, mummification, autolysis, or live fetus incapable of extrauterine survival

**SYSTEM AFFECTED**
Reproductive

**INCIDENCE/PREVALENCE**
- 5%–15% spontaneous abortion, multiple risk factors
- Bred predisposition for swimming

**SIGNALMENT**
- Non specific
- breeds—Thoroughbred, draft mares, Standardbreds, related breeds (twinning)
- Mares >15 years
- Maiden American Miniature Horse mares—aneutodal placental insufficiency

**SIGNS**

**General Comments**
- Depending on cause, time of fetal death, stage of gestation, duration of condition, and whether pregnancy ended in dystocia or with RFM, dam may show few signs or, in extreme cases, suffer life-threatening multiorgan system disease.
- Most in second half of gestation

**CAUSES**
- Twins
- Twin pregnancies that persist >40 days—5%–15% end in abortion/stillbirth.

**ABORTION, SPONTANEOUS, NONINFECTIONOUS**

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**CAUSES**
- Twins
- Twin pregnancies that persist >40 days—5%–15% end in abortion/stillbirth.
Abortion, Spontaneous, Noninfectious

Iatrogenic Causes
- PGF2α—may require repeated injections if >40 days of gestation
- Procedures mistakenly done on a pregnant mare—AI; intrauterine infusions; samples taken for cytology, culture, or biopsy

RISK FACTORS
- Family history of twinning or noninfectious, spontaneous abortion
- Systemic maternal disease
- Grazing endophyte-infected fescue, ergotized grasses, or plants producing phytoestrogens (anecdotal) late in gestation
- Exposure to xenobiotics

DIAGNOSIS
- Most mares asymptomatic before aborting
- Fetus(es)—variable condition, fresh to autolytic
- Definitive diagnosis possible ≥50%–60% of cases
- Excluding twins and EHV-1, diagnosis is only 30% if few samples are submitted and moderate/severe autolysis of fetal and placental tissues.

DIFFERENTIAL DIAGNOSIS
Other Causes of Abortion
- Infectious, spontaneous abortion
- Placentitis—by physical examination or by lab diagnostics

Other Causes of Signs of Labor or Abdominal Discomfort
- Normal parturition
- Dystocia associated with abortion
- Peripartum uterine artery rupture
- Colic associated with uterine torsion
- Discomfort associated with hydrops of fetal membranes or prepubic tendon rupture
- Colic associated with reproductive disease

Other Causes of Vulvar Discharge
- Normal parturition
- Dystocia unassociated with abortion
- Normal estrus
- Endometritis
- Metritis or RFM
- Mucometra or pyometra

CBC/BIOCHEMISTRY/URINALYSIS
- Determine inflammatory/stress leukocyte response, other organ system involvement

OTHER LABORATORY TESTS
Maternal Progesterone
- Indicated with history of abortion or in an old mare, previous biopsy presence of endometritis or fibrosis
- ELISA or RIA <80 days of gestation; acceptable levels are >1 to >4 ng/mL, depending on reference lab
- >100 days of gestation, RIA detects progesterone (may be very low >150 days) and cross-reacting 5α-pregnanes of uterofetoplacental origin
- Decreased maternal levels of 5α-pregnanes with cases of equine fescue toxicosis

Maternal Estrogens
- Reflect fetal estrogen production and viability, especially conjugated estrogens, e.g., estrone sulfate

Maternal Relaxin
- Decreased maternal relaxin concentration—thought associated with abnormal placental function

Maternal Prolactin
- Decreased prolactin secretion, late gestation, associated with fescue toxicosis and ergotism

Maternal T3/T4
- Anecdotal reports of lower levels in mares with history of conception failure, EED, or abortion
- Significance of low T3 levels is unknown.

Cytogenetic Studies
- If suspect maternal chromosomal abnormalities
- Difficult if fetus autolysed

Maternal and Fetal Assays for Xenobiotics
- Indicated in cases of specific intoxications
- Sample the dam’s whole blood, plasma, or urine samples
- Sample fetal serum from heart blood, thoracic or abdominal fluid, liver, and kidney

Feed Analysis
- Indicated for specific xenobiotics—ergopeptine alkaloids, phytoestrogens, heavy metals, or endophyte (Neotyphodium coenophialum)

IMAGING
- Transrectal/transabdominal U/S to confirm pregnancy, diagnose twins, evaluate fetal viability and development, assess placental health, diagnose other gestational abnormalities, e.g., hydrops of fetal membranes

DIAGNOSTIC PROCEDURES
- If entire fetus and placenta are available, appropriate samples for pathology, histology, culture, and serology
- Fresh/chilled fetal thoracic or abdominal fluid or serum from fetal heart or cord blood (if available); fetal stomach contents; 10% formalin-fixed and chilled/frozen samples of fetal heart, lung, thymus, liver, kidney, lymph nodes, spleen, adrenal gland, skeletal muscle and brain; 10% formalin-fixed and chilled/frozen fetal membranes (i.e., allantochorion and allantoamnion)
- Uterine swabs from dam may be useful to establish placitis diagnosis
- Unless cause is obvious, e.g., twins, iatrogenic, rule out infectious causes of abortion, especially if multiple mares are at risk.
Abortion, Spontaneous, Noninfectious

**Pathological Findings**

- **Twins**
  - Two fetuses, often dissimilar in size, with one mummified or severely autolytic
  - Avillous chorionic membrane at point of contact of two placentae
- **Placental Abnormalities**
  - Umbilical cord torsion—confirm with evidence of vascular compromise
  - Villous atrophy or hypoplasia may suggest endometrial fibrosis
  - Placental edema, gross and histopathological, consistent with equine fescue toxicosis
  - Hydrops allantois and amnion—a gross diagnosis if dam suffers prepartum death

**Fetal Abnormalities**

- Developmental abnormalities—hydrocephalus; anencephaly; gross and histopath confirmation

**Treatment**

**Appropriate Health Care**

- Treatment only if early diagnosis of the pathologic process, before irreversible fetal or placental compromise occurs
- Main therapeutic approach to twinning—early selective reduction
- Late-gestation twin diagnosis—pregnancy may be maintained until term, in some instances, with progestin and antibiotic therapy
- Mares with abortion history—evaluate and treat before rebreeding; progestin supplementation may be appropriate, especially with suspected luteal insufficiency (anecdotal) or early luteal regression, but this therapy is controversial and is contraindicated in some circumstances. ET may be indicated for mares with a history of repeated abortion.
- Villous atrophy or hypoplasia can be treated with D2-dopamine receptor antagonists or progesterone; cases of abortion, i.e., stillbirth, frequently occur before therapy begins.
- Aborting mares generally only require prophylactic therapy for metritis or endometritis.
- Most patients managed on an ambulatory basis
- Systemic maternal disease may need hospitalization and intensive care.

**Nursing Care**

Most noninfectious abortions require limited nursing care, unless systemic disease develops.

**Activity**

Limit to paddock exercise to allow observation.

**Client Education**

Problem mares are likely to have future reproductive problems.

**Medications**

**Drugs of Choice**

- Test specific topics.

**History of Abortion, Endometritis, or Fibrosis**

- Treat with altrenogest 0.044–0.088 mg/kg PO daily.
- Begin 2–3 days after ovulation or at diagnosis of pregnancy, continue to at least 100 days of gestation.
- Taper dose gradually during a 14-day period at end of treatment.
- Mares with abortion history—evaluate and treat before rebreeding; progestin supplementation may be appropriate, especially with suspected luteal insufficiency (anecdotal) or early luteal regression, but this therapy is controversial and is contraindicated in some circumstances. ET may be indicated for mares with a history of repeated abortion.
- Villous atrophy or hypoplasia can be treated with D2-dopamine receptor antagonists or progesterone; cases of abortion, i.e., stillbirth, frequently occur before therapy begins.
- Aborting mares generally only require prophylactic therapy for metritis or endometritis.
- Most patients managed on an ambulatory basis
- Systemic maternal disease may need hospitalization and intensive care.

**Contraindications**

- Uses of altrenogest—prevent abortion of viable fetus, for noninfectious placentitis, and endotoxemia
- Monitor fetal viability at least weekly to avoid retaining a dead fetus in utero or lead to development of pyometra.
- Altrenogest absorbed through skin; wear gloves and wash hands.
- Ancodiotal success of supplemental progestin to maintain equine pregnancy

**Alternative Drugs**

- Progesterone 150–500 mg oil base IM daily
- T4 supplementation—anecdotal success treating subfertile mares; use remains controversial, considered deleterious by some clinicians
Follow-up

Patient Monitoring
- 7–10 days post-abortion—TRP, U/S, or both; evaluate uterine involution.
- Rate of involution depends on therapy used, presence of systemic disease, secondary complications.
- Further examination—vaginal speculum, uterine cytology/culture, endometrial biopsy

Prevention/Avoidance
- Early recognition of at-risk mares
- Records of double ovulations
- Early twin diagnosis (<25 days, as early as day 14 or 15)
- Selective embryonic/fetal reduction
- Managing preexisting endometritis before next breeding
- Remove mares from fescue pasture during last third of gestation (minimum 30 days).
- Domperidone (1.1 mg/kg PO daily) at earliest signs of equine fescue toxicosis or 10–14 days prior to due date, continue until parturition and development of normal mammary gland
- Injection with fluphenazine (25 mg IM in pony mares) on day 320 of gestation has been suggested for prophylaxis of fescue toxicosis.
- Careful use of medications in pregnant mares
- Avoiding exposure to known toxicants

Possible Complications
- Recovery uneventful after many asymptomatic abortions
- Dysuria, RFM, metritis, laminitis, sepsisemia, endometritis, reproductive tract trauma may impact the mare’s future well-being and reproductive value.

Expected Course and Prognosis
Uneventful recovery in most cases with appropriate treatment

Miscellaneous

Age-Related Factors
- Development of chronic endometritis and endometrial fibrosis
- Maiden American Miniature Horse mares

Pregnancy
- Pregnancy associated by definition

See Also
- Abortion, infectious
- Endometritis
- Fetal stress/diabetes/viability
- High-risk pregnancy
- Hydrops allantois/amnion
- Metritis, postpartum
- Multiple ovulations
- Placental insufficiency
- Placentitis
- Premature placental separation
- RFM
- Twin pregnancy

Abbreviations
- AI = artificial insemination
- CL = corpus luteum
- EED = early embryonic death
- EHV = equine herpesvirus
- ELISA = enzyme-linked immunosorbent assay
- EPM = equine protozoal encephalomyelitis
- ET = embryo transfer
- MRLS = mare reproductive loss syndrome
- RIA = radioimmunoassay
- RFM = retained fetal membranes/placenta
- U/S = ultrasound, ultrasonography

Suggested Reading

Author Tim J. Evans
Consulting Editor Carla L. Carleton
**ACER RUBRUM (RED MAPLE) TOXICOSIS**

**OVERVIEW**
- An equine disease that follows ingestion of wilted or dried *Acer rubrum* (red maple) leaves and is characterized by methemoglobinemia, hemolytic anemia, and Heinz-body formation
- Most frequently reported in the eastern half of North America, where trees are more prevalent
- The specific toxin has not been identified, but apparently is found only in wilted or dried leaves, because the disease has not been induced using fresh leaves.
- Clinical findings are consistent with oxidative injury to RBCs, resulting in the formation of methemoglobin (i.e., oxidation of iron in hemoglobin from ferrous to ferric form), Heinz bodies (i.e., precipitated oxidized hemoglobin), and hemolytic anemia.
- Affected organ systems include:
  - Cardiovascular—tachycardia secondary to anemia
  - Hemic—methemoglobinemia, hemolytic anemia, and Heinz bodies (i.e., precipitated oxidized hemoglobin), and hemolytic anemia.

**SIGNS**
- Acute death can result from rapid formation of methemoglobin. Alternatively, hemolytic crisis can develop over several days as the hemolysis and methemoglobinemia progressively worsen.
- Historical findings include lethargy, weakness, anorexia, and perhaps colic or fever.
- Physical examination findings include yellow or brown mucous membranes, red or brown urine, tachycardia, polypnea, and dehydration.

**CAUSES AND RISK FACTORS**
- It usually occurs during the summer and fall months after an event that results in leaf wilting such as tree pruning, fallen branches after a storm, or autumn leaves falling.
- Consider all causes of equine hemolytic anemia, which include oxidant poisons, EIA, immune-mediated hemolytic anemia, piroplasmosis, and liver failure.

**DIAGNOSIS**
- CBC/BIOCHEMISTRY/URINALYSIS
  - Decreased PCV, hemoglobin, and unconjugated bilirubin, is increased because of hemolytic anemia and anorexia.
  - Increased albumin and total protein result from dehydration.
  - Urinalysis results include proteinuria and hemoglobinuria, with few or no intact erythrocytes.
  - Increased liver enzymes and creatine phosphokinase may occur, probably secondary to cell damage caused by anemia-induced hypoxia.
  - Eccentrocytes and ghost cells have been reported.

**SIGNALMENT**
- No breed predilections
- No gender predilections
- No age predilections
- No genetic basis
OTHER LABORATORY TESTS
The percentage of methemoglobin in the blood is elevated.

IMAGING
N/A

OTHER DIAGNOSTIC PROCEDURES
N/A

PATHOLOGICAL FINDINGS
- Gross findings include generalized icterus, enlarged spleen, and discolored kidneys.
- Histopathologic findings include erythropagocytosis by macrophages, renal pigment casts and sloughed epithelial cells, splenic and hepatic hemosiderin, and centrilobular hepatic lipidosis. Pulmonary thrombosis has been reported in one horse.

TREATMENT
- The decision regarding inpatient or outpatient treatment depends on severity of the clinical signs and ability of the owner to care for the animal. Frequently monitor progression of the methemoglobinemia and anemia.
- Give IV fluids to replace fluid deficits and to maintain adequate renal perfusion.
- Blood transfusion may be needed with severe anemia.
- Limit physical activity of anemic animals.
- Continuous nasal oxygen administration may be helpful.
- Offer a high-quality diet, especially because affected horses often lack an appetite.

MEDICATIONS
- Ascorbic acid has been used for its antioxidant effects (30–50 mg/kg q12h added to IV fluids).
- It also can be given orally but may take several doses to achieve adequate tissue levels.

CONTRAINDICATIONS/POSSIBLE INTERACTIONS
- Do not treat methemoglobinemia with methylene blue because of its poor efficacy in horses and reports that it may increase Heinz-body formation.
- NSAIDs may be necessary to control pain but can compromise renal function.

FOLLOW-UP
PATIENT MONITORING
- Monitor methemoglobinemia and anemia, and adjust therapy based on the severity and speed of progression.

PREVENTION/AVOIDANCE
- Instruct owners not to plant red maples.
- Prune or remove existing trees only when no leaves are on the trees.
- Owners should check for fallen branches immediately after storms.

EXPECTED COURSE AND PROGNOSIS
- Prognosis depends on the quantity of leaves ingested and how soon veterinary care is sought after ingestion.
- Death is attributed to severe methemoglobinemia or anemia or to renal failure secondary to pigment nephropathy.

ASSOCIATED CONDITIONS
- Laminitis can occur during or after the course of the disease.
- Anemia and methemoglobinemia can result in fetal hypoxia, followed by abortion.

ABBREVIATIONS
- EIA = equine infectious anemia
- MCH = mean corpuscular hemoglobin
- MCHC = mean corpuscular hemoglobin concentration
- PCV = packed cell volume

Suggested Reading

Author Konnie H. Plumlee
Consulting Editor Robert H. Poppenga
ACIDOSIS, METABOLIC

BASICS

DEFINITION
A disruption of acid-base homoeostasis producing increased H3O+ concentration, which is reflected by acidemia—decreased blood pH and low plasma HCO3–.

• Plasma bicarbonate level is ≤ 24 mEq/L.

• pH of arterial blood ranges from 7.35 to 7.45.

PATHOPHYSIOLOGY
Fixed acid is produced via normal metabolic processes in large quantities daily.

H+ is regulated by intracellular and extracellular buffering, respiratory buffering (i.e., variation of CO2 levels via changes in ventilation), and regulation of HCO3– via renal excretion of H+.

Renal H+ excretion is accomplished by direct secretion of limited amounts of H+, increased generation of ammonium ions, and titration of phosphates and urates (titratable acidity).

Regulation of HCO3– occurs when H+ is secreted—90% in the proximal tubule, the remainder in the distal nephron.

• The minimum pH (4.5) of the tubular fluid limits secretion of H+.

• Titratable acidity increases minimally in acidotic patients.

• In most species, production of ammonia with subsequent excretion of ammonium ions is the major mechanism by which the kidney handles an acid load.

Intracellular and extracellular buffering of H+ occurs immediately or within minutes and is accomplished by proteins (primarily albumin and hemoglobin), phosphates, and bicarbonate.

Carbohydrate storage in bone also is a significant site of intracellular buffering.

• The most important buffer is HCO3–, because it is present in high concentrations and the end product of its activity, CO2, is readily eliminated via ventilation.

• Respiratory compensation responds within minutes and is effective for mild and moderate acidemia.

Defensive regulation of H+ and HCO3– levels is accomplished by the kidney.

• Renal processing of an acid load begins within hours but may take days to normalize pH.

• Inability to excrete H+; loss of HCO3–; increased production of H+ (i.e., lactic acidosis); and accumulation of acids are the major mechanisms producing metabolic acidosis.

• Hyperkalemia (i.e., weak acids) and overhydration (i.e., dilutional acidosis) also produce metabolic acidosis via alteration of the balance between strong cations and anions in body fluids.

SYSTEMS AFFECTED

Respiratory
• Peripheral and central chemoreceptors sense low pH in blood or CSF and stimulate hyperventilation to increase elimination of CO2 and increase pHa.

• Decreased respiratory muscle strength can lead to muscle fatigue and worsening metabolic status, especially in neonates.

Cardiovascular
• Decreased cardiac contractility
• May predispose to arrhythmias
• Vasodilation of arterioles; constriction of veins
• Vascular effects may be offset by catecholamine effects.

Neuroendocrine
• Catecholamine release
• CNS depression
• CSF acidosis in acute situations

Renal
• The kidney responds to low arterial pH by increasing H+ excretion and generating increased levels of HCO3– to bring the systemic pH back to normal.

• This response begins within hours, but it may take days to be effective.

Metabolic
• Inhibition of anaerobic glycolysis
• Insulin resistance
• Increased affinity for oxygen–hemoglobin binding, enhancing release of oxygen to the tissues
• Increased protein catabolism
• Increased ionized calcium concentration

SIGNALMENT
Any species

SIGNS
• Hypertonic/hypoxic physical examination findings vary primarily with the underlying cause.

• Weakness, depression, and tachypnea are clinical signs specific to acidosis.

CAUSES
• Many diseases result in metabolic acidosis via more than one mechanism.

• Loss of bicarbonate most commonly is seen in horses with colic; RIA results in HCO3– loss both directly and indirectly, depending on the type of tubular dysfunction.

• Renal failure results in an inability to excrete H+ and accumulation of uric acids.

• Increased H+ production (i.e., lactic acidosis) is seen with diseases producing decreased effective circulating blood volume—hypovolemia or hypovolemia caused by inadequate intake, hemorrhage, septic or hypotensive fluid loss or sequestration (e.g., ureterolithiasis, peritonitis, pleuritis, ascites, neutrophilic types of colic), strangulating lesions of the GI tract, endo- or exenteral, or cardiac failure.

• Chronic causes of hypoxemia produce lactic acidosis.

• Grain overload produces metabolic acidosis via production of lactic acid, fluid sequestration in the GI tract, secretion into the GI tract, and endo- or exenteral.

• High-intensity anaerobic exercise results in production of lactate, which can affect fluid balance/SID and result in metabolic acidosis, however, this is short-lived.

• Severe exertional rhabdomyolysis associated with anaerobic exercise produces lactic acidosis.

• Acute or end-stage hepatic failure may result in metabolic acidosis due to failure of the detoxification systems of the liver.

• Aspiration or aspiration may cause multiorgan damage or failure, which can result in metabolic acidosis in neonates.

• Accumulation of exogenous acids is uncommon, as this is usually caused by ingestion of toxic substances; it may be seen with xylazine, propylene glycol, ethylene glycol, paraldehyde, and me簿anol.

• Malignant hyperthermia is uncommon but has occurred in anesthetized horses and results in severe lactic acidosis.

• Proteins are weak acids; conditions producing increased levels of proteinuria (i.e., chronic infections, immune-mediated disease, plasma cell myeloma, lymphoma) produce metabolic acidosis.

• Excessive or inappropriate fluid therapy, especially in neonates, produces free-water excess and dilutional acidosis.

• TPN can lead to metabolic acidosis when many diseases result in metabolic acidosis via more than one mechanism.

• Highly anionic diets have been suggested to induce metabolic acidosis in equine.

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS
• Some causes of metabolic acidosis can be identified on physical examination (i.e., diarrhea, dehydration, colic with ischemic lesions).

• Decreased HCO3– levels are also seen in conditions with chronic respiratory alkalosis, (Pco2<40 mm Hg), if hyperventilation is occurring, but pH will be normal or mildly increased.

LABORATORY FINDINGS

Drugs That May Alter Lab Results
With poor peripheral perfusion or cardiovascular surprise, results of blood gas analysis on samples taken from peripheral vessels may differ from those taken elsewhere or not reflect the overall systemic condition.

Valid If Run in Human Lab and if sample submitted properly?

CBC/BIOCHEMISTRY/URINALYSIS
• Measurement of serum electrolytes and protein levels is important to determine the cause and to guide treatment.

• Calculation of the anion gap also may be useful, especially in mixed acid-base disorders.
ACIDOSIS, METABOLIC

• Proportional changes in sodium and chloride levels occur with alterations of fluid balance.
• Normal sodium levels with hyperchloremia or hyperkalemia indicate acid-base imbalance.
• Disproportional changes in Na⁺/Cl⁻ usually are associated with simultaneous acid-base imbalance and hydration abnormalities.
• Anion gap/protein levels are not considered when calculating the anion gap; however, because proteins are weak acids, hyperproteinemia can produce the condition—dehydration, chronic infection, and sepsis.
• Urinalysis and fractional excretion of electrolytes are useful in cases of renal failure and RTA.

HORSAKS AFFECTED BY HYPOCHLOREMIA AND NORMAL AG

• Loss of HCO₃⁻—sketch, type II RTA, and primary respiratory alkalosis; however, severely affected colt patients often are acidic and low in Na⁺, K⁺, Cl⁻, and HCO₃⁻ because of water intake after isotonic fluid loss.
• Addition of Cl⁻—fluid therapy with Cl⁻-containing fluids (i.e., 0.9% NaCl, KCl), salt poisoning, TPN, NH₄Cl, or KCl supplementation
• Cl⁻—renal failure, type I or IV RTA, and acrosidemolone

HORSAKS AFFECTED BY INCREASED AG

• Accumulation of unmeasured anion:
• Lactate—conditions with hypoxemia or hypotension (e.g., shock, sepsis, cardiac failure, ischemic/inflammatory types of colic); conditions with inflammation or fluid sequestration (e.g., pleuritis, peritonitis, uroperitoneum, grain overload); conditions using anaerobic glycolysis (e.g., anaerobic exposure, severe erythematous rhodanymosis, malignant hyperthermia
• Phosphates, sulfates, and organic acids—renal failure, tonic necrosis

OTHER LABORATORY TESTS

• Total CO₂
• Measured by many labs using the same sample submitted for electrolyte
• Close approximation HCO₃⁻, because most CO₂ is carried to the blood as bicarbonate
• Respiratory alkalosis also decreases TCO₂; differentiation can only be made with blood gas analysis.
• Analyze rapidly with minimal room-air exposure within the sample tube as CO₂ will decrease.

IMAGING

• Diagnosis of cardiac, renal, and hepatic failure can be facilitated via ultrasonography.

DIAGNOSTIC PROCEDURES

• Bupyr for suspected organ failure and cytology and microbiology of exudates or effusions may be useful with inflammation or infection.

TREATMENT

• Directed at the primary cause. Alkalizing therapy is described below.
• Replacement of fluid losses with balanced isotonic fluids may be all that is needed to restore acid-base status in mild cases.
• With hypervolemia caused by hemorrhage, hypertonic saline, colloids, or blood transfusion may be necessary to restore effective circulating volume in addition to crystalloid therapy.
• Specific electrolyte losses should be addressed, i.e., K⁺, Ca²⁺, in GI cases. Levels may change with alkalizing therapy.

MEDICATIONS

DRUG(S) OF CHOICE

• Alkalizing therapy is reserved for patients with a pH < 7.2 that persists following rehydration or volume replacement
• Sodium bicarbonate is most frequently utilized.
• The bicarbonate deficit is calculated as follows: Base deficit × body weight (kg) × 0.3 (ECF space [0.3 in foals]) = HCO₃⁻ (mEq)
• A negative BE or 24 < HCO₃⁻ can be used for the base deficit.
• In acute cases, the deficit can be given safely over 30 min, in fluids or as a 5% solution to adults.
• Isotonic bicarbonate (1.3%) is a good choice in neonates or severely affected adults with colitis.
• Correction to a pH ≥ 7.2 and BE ≥ −5 is usually adequate, especially with organic acidoses, because these are metabolized once the primary problem improves.

CONTRAINDICATIONS

• Sodium bicarbonate cannot be mixed with calcium.

PRECAUTIONS

• Use bicarbonate therapy cautiously in patients with respiratory compensatory, because the CO₂ that is generated may not be eliminated, causing a further decrease in pH.
• Hyperosmolar solutions may cause vascular irritation and affect tonicity of the CSF.
• Sodium load may affect blood volume in neonates and patients with compromised renal, neurologic, or cardiac function.
• Rhabdomyolysis or renal or cerebral acidosis is reported from overdose or too-rapid administration of bicarbonate since both CO₂ and HCO₃⁻ cross the blood-brain barrier.

POSSIBLE INTERACTIONS

• Alkalizing therapies (i.e., HCO₃⁻). Lactate can combine with Ca²⁺ in crystallized solutions that form a harmful precipitate.

ALTERNATIVE DRUGS

• Replacement of IV fluid solutions with other alkalizing agents (e.g., lactate, citrate) are effective, because these are metabolized to HCO₃⁻. Adequate hepatic function must be present, so these may not be useful in severely acidic, hypoproteinemic, or septic patients.
• Oral rehydration solutions (i.e., 2–3 liters PO in adults without ileus) have been used as primary therapy or an adjunct to IV fluid therapy in less severe cases.
• THAM, tromethamine, can be used as an alkalinizing agent. Its use does not increase CO₂ or sodium levels, and can be useful in patients with pneumonia or hypernatremia.

SUGGESTED READING


Author Jennifer G. Adams
Consulting Editor Kenneth W. Hinchcliff
ACIDOSIS, RESPIRATORY

BASICS

DEFINITION
- Increase in blood Pco2
- Homeostatic mechanisms maintain normal blood levels within a narrow range.
- Arterial levels range from 35–42 mm Hg.
- Venous levels range from 43–49 mm Hg.

PATHOPHYSIOLOGY
- CO2 is formed in all tissues during metabolic energy production and diffuses passively out of cells and into the blood in gaseous form.
- Most of this CO2, (65–70%) combines with water almost instantaneously to form carbonic acid, which then dissociates into bicarbonate ion and hydrogen.
- Most CO2 is transported in the blood as bicarbonate. Some is bound to proteins, especially deoxygenated hemoglobin, and a small amount is dissolved directly into plasma.
- In the lungs, the reverse occurs, and CO2 passively diffuses out of capillaries into the alveoli.
- The three forms of CO2 exist in equilibrium in the blood, but the Pco2, as measured by blood gas depends on the dissolved portion.
- The chemical components of the carbonic acid equilibrium are:
  
  \[ \text{CO}_2 + \text{H}_2\text{O} \rightleftharpoons \text{H}_2\text{CO}_3 \rightleftharpoons \text{H}^+ + \text{HCO}_3^- \]
- Alveolar CO2 then is removed mechanically by ventilation as air moves in and out of the lungs.
- Hypercapnia is present only when tissue production exceeds the capacity of normal lungs to eliminate CO2, or when components of the respiratory system are abnormal.
- Hypercapnia is uncommon in conscious patients, because the respiratory center responds to changes in partial pressures by increasing minute ventilation.
- Respiratory acidosis results from disease or obstruction of the respiratory center in the medulla and peripheral chemoreceptors that control respiration, the mechanical components (i.e., chest wall, respiratory muscles), or the conducting airways, alveoli, and pulmonary vasculature, which are already involved in gas exchange, by causing hypoventilation, barriers to diffusion, or V/Q mismatching.
- Because CO2 diffuses very readily across the respiratory membrane in direct proportion to ventilation, hyperventilation usually has the most significant effect on blood levels.
- Hypermetabolism, as seen with malignant hyperthermia, may produce CO2 in greater amounts than the lung can eliminate.
- Increased CO2 also develops as a compensatory response of the lungs to metabolic acidosis.

SYSTEM AFFECTED
- Respiratory—See Pathophysiology.

SIGNALMENT
- Any horse
- Almost every anesthetized patient develops some degree of hypercapnia when breathing spontaneously.
- Because of their size, equines are especially predisposed to hyperventilation under anesthesia.

SIGNS
- Respiratory noise may be heard, especially with exercise, in cases of upper airway obstruction.
- Exercise intolerance may be reported with many causes.
- Any horse can develop hypercapnia under anesthesia as well.

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS
- Physiologic hyperventilation processes that present with tachypnea—fever, hyperthermia, excitement, anxiety, painful conditions, hypoxemia, metabolic acidosis, and CNS derangements.
- Under anesthesia, tachypnea also may result from a light plane of anesthesia, hypoxemia, metabolic acidosis, or faulty anesthetic rebreathing systems.
- Diseases resulting in metabolic alkalosis may have a compensatory hyperventilation—upper GI obstruction, early large colon impactions or simple obstructions, supplementation with bicarbonate or other alkalinizing agents.

Measurements of pH in these cases often are still higher than normal, because compensatory hyperventilation is limited once hypoxemia develops.

LABORATORY FINDINGS

Disorders That May Alter Lab Results
- With poor peripheral perfusion or cardiovascular shunt, results of blood gas analysis on samples taken from peripheral vessels may differ from those taken elsewhere or not reflect the overall systemic condition.
- Exposure to room air via air bubbles in the sample may change the Pco2 level, because the sample equilibrates with the air.
ACIDOSIS, RESPIRATORY

Acidosis decreases heart contractility and overall status, especially cardiovascular and neurologic, may improve respiratory function. Hypercarbia leads to air trapping in alveoli, which may result in barotrauma in foals with meconium aspiration, partial obstruction of the small airways may lead to air trapping in alveoli, which may rupture.

Precautions

Monitor ventilated patients continuously for airway obstruction caused by accumulation of secretions, kinking of tubing, tubes, and so on.

Oxygen toxicity can develop with inspired PO₂ >50% or if Paco₂ >100 mm Hg is maintained for prolonged periods (10–12 hr).

Possible Interactions N/A

Alternative Drugs N/A

Follow-up

Patient Monitoring

Decreased respiratory effort should be seen quickly after improvement of ventilation.

Use serial arterial blood gas analysis or capnometry to assess adequacy of ventilation and monitor progress, especially during weaning.

Possible Complications

Respiratory acidosis lowers systemic pH and may affect ionization of protein-bound drugs.

Acidosis decreases heart contractility and may cause or contribute to CNS depression.

Hypercarbia and the resultant acidosis predispose patients to cardiac arrhythmias, especially under anesthesia.

The Paco₂ level greatly affects cerebral blood flow and CSF pressure.

Severe or prolonged hypercapnia may contribute to brain damage or herniation in cases with head trauma.

Miscellaneous

Associated Conditions

Disorders that result in metabolic alkalosis.

Age-Related Factors

Neonates, especially premature foals, may be more prone to hyperventilation because of decreased compliance of the lungs and lack of strength (i.e., immaturity) of the chest wall.

Zoonotic Potential N/A

Pregnancy

See Risk Factors.

Synonyms

Hypercapnia

Hypercarbia

Hyperventilation

See Also

See specific diseases in Causes.

Abbreviations

CNS = central nervous system

COPD = chronic obstructive pulmonary disease

CSF = cerebrospinal fluid

GI = gastrointestinal

V/Q = ventilation/perfusion

Suggested Reading


Author Jennifer G. Adams

Consulting Editor Kenneth W. Hinchcliff
**Actinobacillosis**

**Basics**

**Overview**
- Acute rapidly progressive septicemia due to *Actinobacillus equuli* or *A. suis*-like organisms in neonatal foals.
- *A. equuli* is a gram-negative cocccobacillary to rod-shaped pleomorphic organism that produces gray 1- to 3-mm colonies after 24-hr incubation on blood agar. *A. equuli* is a normal inhabitant of the mucous membranes of the alimentary tract.
- Fetal infection may follow transplacental infection. The kidneys are a frequent site of neonatal infection.
- In adults, infection is frequently endogenous and results from fecal contamination or spread from oral mucous membranes. Adults have soft tissue abscesses, respiratory infections, and rarely conjunctival, urinary tract, joint, guttural pouch, skin, and genital tract infections.

**Signalment**
- Foals: <2 days of age
- Adults of any age and use

**Signs**
- **Foals**
  - Acute onset, depression, diarrhea, recumbency, distended painful joints, sudden death
  - Fever may not be present and foals may be hypothermic. If left untreated, foals may progress rapidly to septic shock.
  - Bone and joint infections in neonates may not be obvious for days to weeks and may be unaccompanied by signs of systemic disease.
- **Adults**
  - Signs are generally referable to the affected organ system.
  - Primary peritonitis due to *Actinobacillus* has been reported in adult horses.

**Causes and Risk Factors**

**Foals**
- Commonly seen associated with failure of passive transfer of immunoglobulins. Perinatal stress, prematurity, and/or unsanitary environmental conditions may predispose the foal.
- Portals of entry include respiratory tract, gastrointestinal tract, placenta, and umbilical remnant.
- *A. equuli* and pleuropneumonia may develop secondary to viral infection or stressful events including but not limited to general anesthesia, athletic events, transport over prolonged distance, and other environmental stressors and concurrent illnesses.
- Trauma may predispose to abscess formation.

**Adults**
- Pneumonia and pleuropneumonia may develop secondary to viral infection or stressful events including but not limited to general anesthesia, athletic events, transport over prolonged distance, and other environmental stressors and concurrent illnesses.
- Trauma may predispose to abscess formation.
- Primary peritonitis due to *Actinobacillus* has been reported in adult horses.

**Diagnosis**

**Differential Diagnosis**

**Foals**
- Any other cause of neonatal sepsis including bacterial, viral, and fungal agents
- *A. equuli* is the most common bacterial agent isolated in cases of neonatal sepsis, although infections with only gram-positive pathogens have been reported.
- Foals with equine herpesvirus type 1 and equine viral arteritis infections may appear identical to those with *A. equuli* infection.
- Foals with perinatal hypoxic-ischemic anoxic or inflammatory insults may present with nearly identical clinical signs, depending on severity.

**Adults**
- Any other cause of fever
- Any other cause of peritonitis
- Any other bacterial, viral, or fungal agent causing pneumonia or pleuropneumonia

**CBC/Biochemistry/Urinalysis**

**Foals**
- Leukocytosis or leukopenia
- Hyperfibrinogenemia at birth is occasionally present with in utero infections. Hyperfibrinogenemia is common in postnatal infections.
- Increased creatinine and/or blood urea nitrogen with renal involvement
- Metabolic acidosis, hyperkalemia, and hypercapnia may be observed with foals in septic shock.
- Hypoglycemia may be present.
- Frequent complete or partial failure of passive transfer (serum IgG < 800 mg/dL)
- Urinalysis may be abnormal with renal involvement.

**Adults**
- Leukocytosis and hyperfibrinogenemia are possible.
- Low PCV in longstanding infection due to anemia of chronic disease
- Other abnormalities, depending on body system involved

**Other Laboratory Tests**

**N/A**

**Imaging**

**Foals**
- Thoracic radiographs may demonstrate pulmonary involvement. Radiographs of affected joints may not show acute changes; bony involvement may take days to become radiographically apparent.
• Ultrasonographic examination of the umbilical remnant may demonstrate focal infection. Ultrasonographic examination of kidneys may be abnormal.

Adults
Radiographic and ultrasonographic evaluation of affected body system may be beneficial.

OTHER DIAGNOSTIC PROCEDURES
Foals
• Blood culture may be diagnostic.
• Bacterial culture of synovial fluid may be diagnostic and should be attempted in affected joints.
• Kidneys frequently have multifocal microabscesses at post-mortem examination.

Adults
• Culture of affected body system may be diagnostic.
• Culture of peritoneal fluid may be diagnostic.
• Culture and cytology of transtracheal aspirates and thoracocentesis fluids may be diagnostic. Because *A. equuli* is a normal inhabitant of equine gastrointestinal mucosa, results should be interpreted cautiously.

TREATMENT
Foals
Affected foals are quite ill and are best managed in a hospital. Administer intranasal oxygen supplementation as needed.

MEDICATIONS

**DRUG(S) OF CHOICE**

Foals
• Administer isotonic polyionic balanced fluids or 0.9% NaCl to maintain adequate hydration and fluid balance. Intravenous plasma as required based on serum or plasma IgG concentrations.
• Intravenous dextrose or parenteral nutrition as needed for nutritional management.
• Broad-spectrum antimicrobial therapy, gentamicin 12 mg/kg IV SID or amikacin 25–30 mg/kg IV SID and potassium penicillin 10,000 IU/kg IV QID or cefuroxime sodium 10 mg/kg IV QID. Monitor plasma creatinine concentration. Therapeutic drug monitoring desirable.

Adults
• Antimicrobial therapy based on culture and sensitivity results

MISCELLANEOUS

Antimicrobial therapy should be modified based on response and culture/sensitivity results. Therapeutic monitoring of aminoglycoside levels should be performed. Continue treatment until clinical signs have resolved and white blood count, differential, and fibrinogen concentration are within normal limits for 48 hours.

*Actinobacillus* spp. were commonly isolated from foals lost to mare reproductive loss syndrome (MRLS) and adult horses affected by pericarditis during the same time period.

**ABBREVIATIONS**

• SIRS = systemic inflammatory response syndrome
• MODS = multiorgan dysfunction syndrome
• MRLS = mare reproductive loss syndrome

**Suggested Reading**
Acute Adult Abdominal Pain—Acute Colic

**DEFINITION**
Clinical condition associated with discomfort originating within the abdominal cavity. May develop acutely or progressively. Considered chronic when persist for >3–4 days.

**PATHOPHYSIOLOGY**
- It originates primarily from the gastrointestinal tract but may also arise from other abdominal structures such as liver, spleen, kidneys, uterus, bladder, or peritoneum.
- Intestinal pain may originate from increased intramural tension, tension on the mesentry, regional or generalized ischemia, mucosal inflammation, smooth muscle spasm associated with hypermotility, or a combination of any of these.
- Nonstrangulated lesions have no compromise to the local blood supply.
- Intramural lesions (impaction, foreign body, concretions), extramural lesions (adhesions, stricture), mural lesion (thickening), as well as spasmotic colic, intestinal displacement, ileus, and inflammatory bowel disease are usually considered nonstrangulated lesions.
- Strangulated lesions, such as torsion and incarceration, are usually associated with compromised local blood supply, intestinal necrosis and cardiovascular shock.

**SYSTEMS AFFECTED**
- Gastrointestinal— anywhere from the stomach to the small colon can be involved. The large colon and the distal part of the small intestine are most commonly involved.
- Cardiovascular system—dehydration and endotoxemia may lead to shock and result in organ failure.
- Other systems can be the source of abdominal pain.

**SIGNALMENT**
- Nonspecific. There may be an age, breed, or sex predisposition. A specific problem (e.g., intussusception of the small intestine is more commonly seen in young horses; polypoidal lipomas are commoner on older horses; lipomas are commoner on older horses; large colon tumors commonly occur around partition in mares; pain from the reproductive tract is seen in pregnant or postpartum mares and in stallions of breeding age).

**SIGNS**
- General Comments
  - Signs of abdominal pain may be subtle initially and are often easily missed and the source of pain may be difficult to identify.
- Historical
  - Signs can appear acutely or following an episode of anorexia, depression, and/or decrease in fecal output. History of change in exercise regimen, diet, or availability of drinking water may also precede the signs of colic, which can be of different intensity.
  - Mild—decrease in appetite and fecal output, mild depression, yawning, increased neck and rolling of the upper lip in Flehmen-like response, teeth grinding
  - Moderate—pawing at the ground, flank watching, grunting, posture for urinating but only a small quantity of urine is passed, leaning against the wall, kicking the abdomen with the hind legs, ears pinned backward, lying down more frequently, may attempt to roll
  - Severe abdominal pain—walking in a tight circle, constantly getting up and down, rolling, traumatizing self and handlers, sweating, labored breathing

**Physical Examination**
- Signs may vary, depending on stage of the disease.
  - General findings—abdominal distention, sweating, increase in respiratory rate, elevated or subnormal body temperature, abnormal quality and quantity of feces
  - Cardiovascular findings—congested mucous membrane, increase in capillary refill time and in heart rate, dehydration and cold extremities are suggestive of a strangulated lesion or to a severe inflammatory process such as colitis or peritonitis
  - Gastrointestinal findings—decrease, decrease or absence in gut motility, gas-filled resonant viscera indicative of a strangulated lesion or to a severe inflammatory process
  - Abdominal pain—abdominal distention, tension of a viscera by gas, liquid, or fluid; displacement of a section of thickening of the intestinal wall; intra-abdominal abnormalities; findings will assist in the differentiation among problems involving the small intestine, large colon, Cecum, small colon, or non-gastrointestinal lesions

**CAUSES**
- Gastrointestinal
  - Gastric—gastric ulcers, gastric distention or impaction, gastric rupture, gastric tumor
  - Small intestine—nonstrangulated obstructive lesions: duodenal ulcer, duodenal/jejunal intussusception, ascariid impaction, ileal impaction, ileo-appendiceal stricture, strangulated obstructive lesion: incarceration of a segment of the small intestine into the epiploic foramen, a space within the mesentery (intussusception), gas distention, mild displacement, nephrosplenic entrapment, enterothel, adhesions, sand impactions
  - Large intestine—nonstrangulated obstructive lesion: ulceration, colitis, impaction, idiopathic gas distention, mild displacement, nephropathic entrapment, enterothel, adhesions, sand impactions
  - Strangulated obstructive lesion: volvulus, herniation, incarceration, thromboembolic infarction
  - Cecum—nonstrangulated obstructive lesion: impaction, adhesions, strangulated obstructive lesion: incarcation, strangulated obstructive lesion: incarceration, strangulated ileum, submucosal hematomas, thromboembolic infarction
  - Retropexy—urether impaction, ureteral distention, ascension, paromea, testicular, peritoneal, hematomas in the large ligament, trauma
- Nongastrointestinal
  - Renal/urinary—renal/ureter/blade/chute, urethral calculi, cystitis, urinary calculi, peritonitis
  - Hepatic—hepatitis, hepato-lobular calcification
  - Others—parasites, hemoproteinum

**RISK FACTORS**
- No access to water
- Sudden change in diet
- Poor systemic parasite control
- Pregnancy
- Previous abdominal surgery
- Congenital abnormalities
- Certain medications

**DIFFERENTIAL DIAGNOSIS**
Other causes of pain that may mimic pain originating from the abdominal cavity include myositis, pleuropneumonia, neurologic diseases such as rabies, and musculoskeletal injuries.

**CBC/BIOCHEMISTRY/URINALYSIS**
- Increase in PCV and TP in face of dehydration.
- Possible hypoproteinemia secondary to protein loss in the intestinal lumen and/or in the abdominal cavity. Lack of protein in acute inflammatory process and endotoxemia or leukocytosis in chronic inflammatory process.
- Possible metabolic acidosis related to cardiovascular shock and release of lactic acid and/or loss of bicarbonate and electrolytes (colitis) or metabolic alkalosis if a large amount of gastric reflux is present, resulting in loss of chloride.
- Hypokalemia and hyponatremia can be present, especially if the horse has been anorectic or is lactating mare. Hypokalemia and hyponatremia may be present in colitis. Alkaline phosphatase may be increased. Anemia is found in horses with severe dehydration or urinary tract disease. The increase in some or all of the following is suggestive of liver disease:
- GLDH, AST, GGT, conjugated bilirubin and bile acid. A selective increase in serum GGT in a horse with colic is suggestive of a displacement of the right colon.

**DIAGNOSTIC TESTS**
- N/A

**CHEMISTRY**
- Various tests may be performed.
Acute Adult Abdominal Pain—Acute Colic

**TREATMENT**

**MEDICATIONS**

- **DRUG(S) OF CHOICE**
  - **Analgesics**—control the abdominal pain
    - NSAIDs—indomethacin 10 mg/kg, flunixin meglumine 0.5–1.1 mg/kg IV, IM q8 h, phenylbutazone 2.2–4.4 mg/kg IV q12–24 h, ketoprofen 1.1–2.2 mg/kg, x–c blockers such as oxatilazine 0.25–0.5 mg/kg IV, IM; detomidine 5–10 μg/kg IV, IM, or remifentanil 0.02–0.05 mg/kg IV. IM can also be given if the pain is not controlled by NSAIDs.
  - The narcotic analgesics such as butorphanol 0.02–0.075 mg/kg IV or meperidine (pethidine 2 mg/kg) can be given alone or in conjunction with xylazine. These two drugs potentiate each other.
  - Any drugs should be used judiciously as they may mask clinical signs and may lead to postponement of surgery, thereby decreasing the chance of survival. Furthermore, most of these drugs have a detrimental effect on gastrointestinal motility.
  - Spasmolytics (indicated in spasmodic colic) — hyoscine 20–50 mL IV and NT-butylooctopamine bromide 0.3 mg/kg.
  - Laxatives—for treatment of impactions
    - Mineral oil—10 mL/kg via nasogastric tube.
    - Enemas—large quantity such as atresia coli.
  - Nuclear scintigraphy
    - Can be used to assess motility, presence of inflammation and infection of the gastrointestinal tract, and the reticuloendothelial function.

**DIAGNOSTIC PROCEDURES**

- Exploratory laparatomy or laparoscopy.

**TREATMENT**

- **Horses** should be taken off feed until diagnosis of the underlying problem.
- **Indication** for an exploratory laparatomy includes: signs of severe abdominal pain, peritoneal signs, or severe impaction. In cases of severe abdominal distention, fever, or progressive increase in gut motility, a severe increase in heart rate or heart rates above 60–70/min, cardiovascular compromise or depression, presence of moderate to severe gas distension or of a displacement of the large colon on rectal examination, gastric reflux, abnormal peritoneal findings, or presence of severe impaction of the large colon or the omentum. Animals presenting with these signs should be referred to a surgical facility.
- Supplemental treatment for medical and surgical cases includes intravenous fluids, gastric decompression if necessary, electrolyte replacement, and control of the abdominal pain.

**MEDIATION**

- Butorphanol 0.02–0.075 mg/kg IV or meperidine (pethidine 2 mg/kg) can be given alone or in conjunction with xylazine. These two drugs potentiate each other.
- Plasma electrolytes should be corrected, especially hypokalemia and hypocalcemia, which are important for intestinal motility. Moderate to severe bicarbonate deficit should be corrected as well as low plasma protein level (<45 g/L).
- Treatment of endotoxemia—inulin (2 mg/kg IV q6 h, IM q6 h, or parenteral crystalloid solution)
- Intravenous fluid therapy
- Antimicrobial therapy if peritonitis is suspected or if surgery is performed.

**CONTRAINdications**

- Acepromazine is contraindicated due to its peripheral vasodilatory effect.

**PRECAUTIONS**

- Repeat use of x–c blockers and butorphanol causes prolonged ileus. Repeat dose of NSAIDs, especially in presence of dehydration, can result in gastric or large colon ulceration as well as renal damage.

**FOLLOW-UP**

**PATIENT MONITORING**

- The patient should be monitored closely for deterioration of clinical signs and cardiovascular status until resolution of the abdominal pain.
- Following resolution of these signs, reintroduction to feed should be done gradually.

**POSSIBLE COMPLICATIONS**

- Endotoxemia
- Laminitis
- Circulatory shock
- Adhesions
- Gastrintestinal rupture
- Peritonitis

**AGE-RELATED FACTORS**

Older horses are more predisposed to large colon torsion; and younger horses are more predisposed to ulcers, mesenteric, and ascarid impactions.

**PREGNANCY**

Mares in late gestation or in the postpartum period are predisposed to large colon torsion.

**SYNONYM**

- Colic

**ABBREVIATIONS**

- **PCV** = packed cell volume
- **TP** = total protein

**Suggested Reading**


**Consulting Editors**

- Henry Stimpfl and Olmo Oliver-Expósito
Acute Epiglottiditis

**BASICS**

**OVERVIEW** Epiglottiditis is a nonspecific inflammatory disease of the epiglottis.

**SIGNMENT**
- Primary: horses (2–10 years) in active training or other horses undergoing repeated, strenuous exercise
- Occasional: in older horses (15–18 years) associated with neoplasia
- No known breed or sex predilection

**CAUSES AND RISK FACTORS**
- Exercise
- Stress
- Repetitive trauma
- Genetic predisposition

**SIGNS**
- Chief complaints—variable amount of abnormal respiratory tract noise and exercise intolerance
- Coughing during exertion is fairly common
- Some horses act mildly pyrexic when swallowing

**DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS**
- The diagnosis is established on the basis of endoscopic examination of the upper respiratory tract.
- Occasionally, the endoscopic appearance is misinterpreted as epiglottic entrapment by the aryepiglottic folds.
- May be associated with epiglottic abscess or chondritis

**CBC/BIOCHEMISTRY/URINALYSIS**
- These tests are not typically performed.

**OTHER LABORATORY TESTS**
- Additional laboratory tests are not typically performed.

**IMAGING**
- Imaging is not usually performed.

**DIAGNOSTIC PROCEDURES**
- During routine endoscopy, the epiglottis may appear swollen and discolored (reddish-purple), partially along the lateral margins and ventral (lingual) mucosal surfaces.
- This swelling may obscure the normal, serrated margins and cause the epiglottis to appear more rounded and bulbous.
- The ventral mucosal surfaces often are ulcerated, and in more chronic, untreated cases, granulation tissue surrounded by fibrous connective tissue is seen.
- The epiglottis looks thicker and may be elevated a variable amount into an abnormal axis above the soft palate.

**TREATMENT**
- Outpatient (out-of-hospital) basis
- Discontinue exercise for a minimum of 7–14 days, depending on the extent of the problem.
- If swallowing is difficult or stimulates coughing, hay may need to be eliminated from the diet or, at least, made wet until the inflammation resolves.
- A complete diet or gruel made from pellets may be easier to swallow.

**MEDICATIONS**

**DRUG(S) OF CHOICE**
- Epiglottiditis usually responds to medical therapy consisting of NSAIDs, parenteral corticosteroids, and topical pharyngeal sprays that contain anti-inflammatory and antimicrobial medication.
- With evidence of infection or a fever, antimicrobial therapy may be indicated—IM procaine penicillin G, PO trimethoprim sulfamethoxazole, or IM or IV ceftiofur at normal recommended dosages for 5–7 days.
- Horses are initially treated with phenylbutazone (4.4 mg/kg IV) or flunixin meglumine (1.1 mg/kg IV) and dexamethasone (0.044 mg/kg IV). Ten to 20 mL of a pharyngeal spray (730 mL of Furacin, 250 mL of DMSO, 1000 mL of glycerin, and 2.0 g of prednisolone) is sprayed slowly, while watching for swallowing, into the pharynx twice daily for 7–14 days through a 10-F catheter introduced into the nasal pharynx via the nasal passages. After the initial IV dose of either phenylbutazone or flunixin meglumine and dexamethasone, oral therapy is continued with phenylbutazone (2.2 mg/kg PO twice daily for 7–14 days) and prednisolone (2.2 mg/kg PO once daily for 7 days). The same dose is then administered orally every other day for three treatments.
- Contraindications/Possible Interactions
  - No contraindications

**FOLLOW-UP**

**PATIENT MONITORING**
- Substantial improvement in the overall appearance of the epiglottis and adjacent tissue and in pharyngeal function usually is seen at follow-up endoscopy after about 1 week of therapy with acute inflammation.
- Complete return and therapy until healing is judged complete based on repeated endoscopy performed at about 1-week intervals.

**PREVENTION/AVOIDANCE**
- Horses with more chronic-appearing inflammation or with associated epiglottic abscess and/or chondritis may require more protracted therapy (2–4 weeks), and complete resolution of thickening and cartilage deformity may not occur.
- Occasionally, epiglottic entrapment may develop, but this usually can be corrected using dorsal midline division or excision with a curved bistoury or a coaxial laser.
- Externally bulbous or fibrinous-appearing entrapping membranes may need to be excised through a laryngotomy.

**POSSIBLE COMPLICATIONS**
- Advises owners that healing may result in fibrosis or scar tissue on the lingual epiglottic surface sufficient to interfere with normal soft-palate function.
- Endoscopy may reveal intermittent or persistent dorsal displacement of the soft palate, which may need surgical treatment—laryngeal tie-forward, soft-palate trim, or excision of fibrous connective tissue on the subepiglottic surface.

**EXPECTED COURSE AND PROGNOSIS**
- Epiglottiditis is a serious, potentially career-limiting or ending problem in racehorses.
- Prognosis depends primarily on severity of the condition during the initial examination and the degree of involvement of the arytenoid cartilage.
- Resolution of acute inflammation results in complete return to normal exercise tolerance and elimination of abnormal respiratory tract noise.
- Horses with more chronic or extensive lesions may experience epiglottic deformity and suffer from intermittent or persistent dorsal displacement of the soft palate despite appropriate medical or surgical therapy.

**SEE ALSO**
- *Dorsal displacement of the soft palate*
- *Inspiratory dyspnea*

**ABBREVIATION**
- DMSO = dimethylsulfoxide

**SUGGESTED READING**
- Author: Norm Ducharme; Eric Tulleners (First Edition)
- Consulting Editor: Daniel Joan
In cases with hepatic encephalopathy, house biopsy is performed on the right side equine viral arteritis. Hematuria, Coagulation profile recommended by some GGT r lactate dehydrogenase r H9253. If the horse is still eating, a high-carbohydrate, Bilirubin—moderate increase in unconjugated frequent yawning has been reported in some alkaline phosphatase r. Most commonly associated with Bilirubinuria. Acute failure. If the animal is not drinking, administer IV urea—normal to low. Reduced production and absorption of toxic WEE, and acute protoscolysis myosin phosphatase. Ureter and serum biochemical changes help in differentiating these problems. Hematuria, hemoglobinuria, myoglobinuria, and bilirubinuria may cause pigmenturial urinanalysis serum and biochemistries aid in differentiation. CBC/BIOCHEMISTRY/URINALYSIS. Bilirubin—moderate increase in unconjugated and conjugated levels. Liver enzymes—increases in SDH (IDH); AST, GGT, and ALP. Some may assay LDH, particularly asparagine 5. GGT—normal to low. Urea—normal to low; Bilirubin. OTHER LABORATORY TESTS. Bromsulfthalidin clearance—2.2 mg/kg IV. Half-life is determined by sampling at 5, 6, and 9 min after injection. normal half-life is 2.8 ±/− 0.5 min. Half-life is prolonged when >50% of liver function is lost. Arthritic test. IMAGING. Ultrasonography may suggest the liver is smaller than normal, with a loss of normal parenchymal structure. DIAGNOSIS. Liver biopsy is performed on the right side, between the 12th and 14th intercostal spaces, where a line drawn from the tuber coxae to the elbow intersects the selected intercostal space. Ultrasonography may ensure accurate placement of the biopsy needle. Ultrasound guidance may ensure accurate placement of the biopsy needle. Histopathology defines the nature and severity of the lesions. PATHOLOGIC FINDINGS. The liver is usually smaller than normal, but it may be enlarged in peracute cases. Generalized atrophy. Histologically, centrilobular to midzonal hepatic necrosis, with mononuclear cell accumulation in the portal triads. Possibly mild bile ductular proliferation in more chronic cases. TREATMENT. Restrict activity, and avoid sunlight. In cases with hepatic encephalopathy, house the horse in a quiet place, preferably padded to avoid injury. If the horse is still eating, a high-carbohydrate, low-protein diet is recommended. The protein should be high in BCAAs—two parts beef pulp with one part cracked corn and added molasses. Out or grass hay is preferred over alfalfa, and the diet should be fed in small amounts five or six times daily. MEDICATIONS. The drugs of choice are: 4. Xylazine (0.5–1.0 mg/kg) or detomidine (0.05–0.4 mg/kg) can be used to control the signs of hepatic encephalopathy. 1. In hypoglycemic animals, 10% glucose solution at 0.2 mL/kg may be given, followed by continuous drip of 5% glucose solution at 2 mL/kg per hour, reducing after 24 hr to half this rate. 2. If the animal is not drinking, administer IV polyionic fluids at maintenance rate. 3. Reduced production and absorption of toxic metabolites can be achieved with mineral oil and neomycin (20–30 mg/kg qID), both via stomach tube.

CONTRAINdications/POSSIBLE INTERACTIONS. Neomycin—usually not given for more than 24–36 hr, because it may induce severe diarrhea. Because the liver metabolizes many drugs, their duration of action may be increased in acute hepatic disease.

FOLLOW-UP. PATIENT MONITORING. Monitor liver enzymes and bilirubin every 2–3 days. PREVENTION/AVOIDANCE. N/A POSSIBLE COMPLICATIONS. N/A EXPECTED COURSE AND PROGNOSIS. Horses with severe hepatic encephalopathy have a poor prognosis, but if the animal remains alert for a week after the onset of clinical signs, recovery is possible. If the SDH (IDH) continues to fall, then the prognosis improves.


ACUTE HEPATITIS IN ADULT HORSES (THEILER’S DISEASE)
Renal failure (ARF) is a consequence of an abrupt, sustained decrease in GFR, resulting in azotemia and disturbances in fluid, electrolyte, and acid-base homeostasis. The pathophysiology of ARF is complex and can be due to a variety of causes. Patients with ARF often present with clinical signs such as lethargy, anorexia, dehydration, edema, and gastrointestinal disturbances.

**CAUSES**
- **Low Flow (Prerenal Failure)**
- **Renal Tissue Injury (Intrinsic Failure)**
- **Obstruction of Urinary Outflow (Postrenal Failure)**
- **Immune-Mediated Injury (Intrarenal Failure)**

**DIFFERENTIAL DIAGNOSIS**
- Prerenal failure—oliguria with increased BUN (50–150 mg/dL) and Cr (2.0–20 mg/dL)
- Renal parenchymal injury—hematuria, proteinuria, azotemia
- Postrenal failure—stranguria, anuria, or hematuria
- Intrarenal failure—renal ulcers, renal calculi

**SIGNS**
- **Renal/Pulmonary—failure**
- **Gastrointestinal—failure**
- **Metabolic—disturbances**
- **Renal/Urologic—failure**
- **Hemic/Thromboplastic—failure**
- **Hypothalamic—failure**
- **Neurologic—failure**

**DIAGNOSIS**

**BASICS**

**DEFINITION**
A consequence of an abrupt, sustained decrease in GFR, resulting in azotemia and disturbances in fluid, electrolyte, and acid-base homeostasis.

**PATHOPHYSIOLOGY**

- **Prerenal**
- **Intrinsic**
- **Postrenal**

**SYSTEMS AFFECTED**
- **Renal/urologic—failure**
- **Gastrointestinal—failure**
- **Metabolic—disturbances**
- **Endocrine—failure**

**GENETICS**

**INCIDENCE/PREVALENCE**

**Breed Predilections**

**LOW**

**GEOGRAPHIC DISTRIBUTION**

**SIGNALS**

**Bred Predictions**

**N/A**

**Mean Age and Range**

**Fawns <30 days of age (especially when receiving nephrotoxic medications)** may be at greater risk, but all ages can be affected.

**GENERAL COMMENTS**

**Diagnosis**

**CAUSATIVE FACTORS**

**MEDICATIONS**

**NURSING CARE**

**TREATMENT**

**AIMS OF TREATMENT**

**APPROPRIATE HEALTH CARE**

**CARE TAKING**

**IMAGING**

**RENAL ULTRASOUND**

**DIAGNOSTIC PROCEDURES**

**PATHOLOGICAL FINDINGS**

**THERAPY**

**FLUID THERAPY**

**FLUID THERAPY**

**EVALUATION**

**SUMMARY**

**REFERENCES**

**AUTHOR INFORMATION**

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**ACKNOWLEDGEMENTS**
Acute Renal Failure (ARF)

Oral Electrolyte Supplementation
- Sodium chloride (30 g) can be administered in concentrate feed or as an oral drench (paste) BID–QID to encourage increased drinking and urine output.
- Potassium chloride can be supplemented in nonhyperkalemic patients with total body potassium depletion—common with asthenia of 2 days.

ACTIVITY
- Stall rest, with limited hand-walking for grasing if appetite is poor

DIET
- Encourage intake by offering a variety of concentrate foods; bran mash, and hay types.
- Hand-walking or short periods of turn-out to graze grass encourage feed intake.

CLIENT EDUCATION
- Prognosis is most dependent on progression of the underlying primary disease process.
- ARF may complicate recovery, prolong hospitalization and treatment, and increase cost.

SURGICAL CONSIDERATIONS

MEDICATIONS

DRUG(S) OF CHOICE
- Judicious fluid therapy is the mainstay of treatment—see Nursing Care.
- Furosemide—For oliguria/anuria (i.e., lack of urination during first 6 hr of fluid therapy), this diuretic may be administered 2 times (1–2 mg/kg IV) at 1- to 2-hr intervals if effective; urination should be observed within 3 hr after the second dose; if ineffective, discontinue treatment.
- Mannitol—has been used in the past as an osmotic diuretic agent for oliguria/anuria unresponsive to furosemide; however, recent evidence suggests that this treatment is not of benefit in critically ill human patients and ROUTINE USE OF MANNITOL IN PATIENTS WITH ARF IS NO LONGER RECOMMENDED.
- Dopamine—has been used in the past as a continuous rate infusion (3–5 μg/kg per minute IV in a 5% dextrose solution) for persistent oliguria/anuria; however, this drug can induce arrhythmias and recent evidence suggests that this treatment is not of benefit in critically ill human patients and ROUTINE USE OF DOPAMINE IN PATIENTS WITH ARF IS NO LONGER RECOMMENDED.

CONTRAINDICATIONS
- Furosemide—or diuretic dosages can result in electrolyte derangements.

PRECAUTIONS
- Monitor response to fluid therapy (i.e., urine output)—at least as 40 mL/kg of IV fluids (20 L per 500 kg horse) may produce increases in CVP and significant pulmonary edema in oliguria/anuric patients.
- Reassess dosage schedule of drugs eliminated by urinary excretion; consider discontinuing all potentially nephrotoxic medications—gentamicin, tetracycline, and NSMIDs.

POSSIBLE INTERACTIONS
- Use of multiple anti-inflammatory drugs (e.g., corticosteroids and one or more NSMIDs) will have additive negative effects on renal blood flow, avoid combined administration in azotemic patients.

ALTERNATIVE DRUGS
- Consider peritoneal dialysis or hemodialysis (foals only) in refractory cases.

FOLLOW-UP

PATIENT MONITORING
- Assess clinical status (emphasizing hydration), urine output, and body weight at least twice daily during the initial 24 hr of treatment and at least daily thereafter.
- Assess magnitude of azotemia and electrolyte and acid–base status at least daily for the initial 5 days of treatment.
- Consider placing a central venous line to maintain central venous pressure < 8 cm H2O in more critical patients and neonates.

PREVENTION/AVOIDANCE
- Anticipate compromised renal function in patients with other disease or undergoing prolonged anesthesia and surgery; institute appropriate treatment to minimize dehydration and potential renal damage.
- Ensure adequate hydration status in patients receiving nephrotoxic medications.
- Avoid concurrent use of multiple anti-inflammatory drugs—NSMIDs.

POSSIBLE COMPLICATIONS
- Pulmonary and peripheral edema; conjunctival edema may be dramatic.
- Severe hypophosphatemia accompanied by cardiac arrhythmia, cardiac arrest, and death.
- Laminin—often refractory to supportive care.
- Signs of neurologic impairments—ataxia, mental obtundation.
- GI ulceration or bleeding.
- Congestive failure.
- Sepsis.

EXPECTED COURSE AND PROGNOSIS
- Prognosis for recovery varies with the underlying primary disease process.
- Prognosis for recovery from prerenal failure and nonischemic intrinsic ARF usually is favorable if azotemia decreases by 25%–50% after the initial 24 hr of treatment; extent of recovery of renal function in patients with intrinsic failure may require 3–6 weeks to fully assess.
- Guarded prognosis for patients with Cr > 10 mg/dL at initial evaluation and when azotemia remains unchanged after the initial 24 hr of treatment.
- Poor prognosis for patients that have persistent anuria, increased magnitude of azotemia after the initial 24 hr of treatment, that rapidly develop edema, or that remain oliguric >72 hr.

MISCELLANEOUS

ASSOCIATED CONDITIONS
- Calcium, phosphorus, magnesium
- Proteinuria, hypoalbuminemia
- Dehydration
- Anemia
- Excessive water administration
- Hypothyroidism

AGE-RELATED FACTORS
- Neonates with hyperoxic-ischemic multiorgan damage or sepsis may have increased risk of intrinsic ARF.
- Neonates, especially premature or dysmature foals, may have markedly elevated Cr concentrations (approaching 25 mg/dL) due to placental insufficiency; this azotemia typically resolves in 2–3 days and should not be confused with intrinsic ARF or sepsis.

ZOONOTIC POTENTIAL

Leprosy is infectious and zoonotic potential; avoid direct contact with infected patient.

PREGNANCY

Puerperal mastitis are at risk of hemolitic shock and renal failure or intrinsic ARF consequent to rupture of a uterine artery.

SYNONYMS
- Acute nephritis
- Acute tubular necrosis
- Vasomotor nephropathy

SEE ALSO
- Anuria/oliguria
- CRF

ABBREVIATIONS
- Cr = creatinine
- CVP = central venous pressure
- GGT = γ-glutamyltransferase
- GFR = glomerular filtration rate
- GI = gastrointestinal
- PUPD = polyuria/polydipsia
- USG = urine specific gravity

Suggested Reading

Author Harold C. Schott II
Consulting Editor Gillian A. Perkins
Acute Respiratory Distress Syndrome in Foals

**DEFINITION**
Respiratory distress syndrome is defined as ventilatory efforts in excess of the metabolic demands. ARDS is defined as acute onset of respiratory distress.

**PATHOPHYSIOLOGY**
- Inflammatory stimuli may initiate events leading to clinical signs of respiratory failure—aspiration pneumonia; viral, bacterial, or fungal infections; thermal injury (e.g., heat stroke); systemic or pulmonary septal/membrane, or inhalation of irritant gases, or smoke may be the initiating insult.
- A manifestation of SIRS, leading to MODS, resulting in artemia, hypotension, hypoxemia, or DIC and bleeding.
- Diffuse injury to pulmonary alveolar epithelium and capillary endothelium, leading to pulmonary edema.
- Immunosuppression may be a factor associated with development of ARDS/interstitial lung disease in foals infected with *Pneumocystis carinii*.

**SYSTEMS AFFECTED**
- Primarily respiratory.
- Often accompanied by dysfunction of the renal, hepatic, and cardiovascular systems and by clotting cascades as disease progresses—MODS.

**INCIDENCE/PREVALENCE**
- Not established, but relatively uncommon.
- Worldwide, in areas with hot summer weather.

**GEOGRAPHIC DISTRIBUTION**
Worldwide.

**SIGNAMENT**
- All ages, but foals 1–8 months of age are predisposed (mean age, 3.5 ± 1.0 months).
- Not sex- or breed-predisposed.

**SIGNS**
- Acute or peracute depression, lethargy, fever, tachypnea, pronounced respiratory effort, neck flaring, increased abdominal and intercostal effort (i.e., “double-cavity” life or “shave line” or paradoxic breathing pattern) and cyanosis.
- Nasal discharge and cough may be frequent but inconsistent findings.
- Thoracic auscultation—loud bronchial sounds over central airways, with either increased or diminished peripheral airway sounds.

**CAUSES**
- Likely the common end result of a variety of different intrapulmonary (inhaled) or systemic insults that initiate SIRS and lead to MODS.
- Heat stress may play a role.
- Foals with subclinical respiratory disease have limited ability to dissipate body heat. Use of erythromycin during hot weather is associated with increased susceptibility to environmental temperatures.
- Viral and bacterial pneumonia can produce respiratory distress in foals with widespread lung involvement.
- Ingestion or inhalation of toxicants (e.g., *P. carinii*).

**OTHER LABORATORY TESTS**
- Arterial blood gas—arterial hypoxemia, hypercapnia, and respiratory acidosis.
- Blood culture may help to identify bacteria.
- Other laboratory abnormalities—dehydration, disseminated intravascular coagulation, and injury to other organs—may be seen.

**DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS**
- Viral pneumonia—equine influenza, equine viral arthritis, equine herpesviruses 1 and 4, equine paramyxovirus, and equine adenovirus.
- Bacterial pneumonia—*P. carinii* infection.
- Pulmonary abscessation or granuloma.
- Upper airway dysfunction, with aspiration of oropharyngeal fluids.
- Ingestion or exposure to toxicosis.

**CBC/BIOCHEMISTRY/URINALYSIS**
- Common abnormalities—neutrophilic leukocytosis, elevated fibrinogen, and anemia.
- Common serum enzymes—lactic dehydrogenase, alkaline phosphatase, and gamma-glutamyl transferase.
- Elevated blood urea nitrogen and creatinine.

**PATHOLOGIC FINDINGS**
- Diffuse injury to other organs—may be seen.
- Diffuse alveolar septal thickening and infiltrates, with superimposed, mixed interstitial and subpleural consolidation.
- Blood filling of alveolar spaces with large numbers of leukocytes.
- Lesions include prominent bronchiolitis; alveolar septal necrosis; and injury to other organs—may be seen.

**TREATMENT**

**AIMS OF TREATMENT**
- Minimize ventilatory and metabolic demands.
- Reduce core body temperature (in hyperthermic foals).
- Reduce lung edema and inflammation.
- Promote adequate oxygenation.
- Discontinue predisposing medications (e.g., erythromycin).
- Eliminate infectious agents with broad-spectrum antimicrobial therapy.
- Support fluid and nutritional needs.

**APPROPRIATE HEALTH CARE**
- Avoid transporting these patients until temperature decreases. Transportation in extreme temperatures may result in their death.
- On-farm examinations during high environmental temperatures should be conducted after moving the mare and foal to a controlled environment on the premises or awaiting stabilization and the cooler period of the day before transportation.

**NURSING CARE**
- These cases are respiratory emergencies and require immediate attention.
- Reduce core body temperature using alcohol baths, fans, and/or misters or by carefully moving the mare and foal to a cooler area protected from direct sunlight or to an air-conditioned stall.
- Cold-water enemas provide significant relief, especially when used in conjunction with the above treatments.
- Judicious use of chilled IV fluids lowers core temperature; however, rapid infusion of large volumes may exacerbate pulmonary edema.
- Balanced electrolytes (e.g., lactated Ringer’s solution) are appropriate initial therapy.
- Insufflation of humidified oxygen (10–15%)
- Transthoracic lung biopsy may be useful, but should not be performed in foals with severe respiratory distress or tendency to bleed.

**PATHOLOGIC FINDINGS**
- Gross Findings
- Lungs are diffusely red, wet, heavy, firm, and fail to collapse when chest is opened.
- In many instances, lungs have a lobulated appearance, with dark, reddened areas interspersed between areas of normal appearing tissue.
- A substantial number of foals also have other lung lesions (e.g., *R. equi* pyogranuloma) representing preexisting pulmonary disease.
- Many foals demonstrate hypoxemia or sepsis-induced lesions in other organs.

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- Insufflation of humidified oxygen (10–15%)
DRUG(S) OF CHOICE

Guided by bacteriologic culture, if possible.

Reduction of discomfort.

Systemic effects of sepsis or endotoxemia, and reducing body temperature, decreasing the sodium succinate (0.5–1.0 mg/kg IV [0.05–0.2 mg/kg IV q 12–24h]), prednisolone, dexamethasone sodium phosphate.

Doses of short-acting corticosteroids (e.g., hydrocortisone).

Address inflammation and hyperthermia.

EVALUATION

Determine patency of the upper airway.

Evaluate thorax for effusion or pneumothorax.

Removal of foals from extremes of heat and environmental temperatures are low) allow the use of fans, misters, or swamp coolers.

Prevention of foals against respiratory pathogens.

Client education regarding prevention and education is aimed at prevention.

Consulting Editor: Daniel Jean

Acute Respiratory Distress Syndrome in Foals

Acute Respiratory Distress Syndrome (ARDS)

Risk factors for ARDS

Expected course and prognosis

Follow-up

Client education regarding potential adverse effects of use of drugs such as erythromycin during hot weather.

Possible complications

Diet

Control—fans, misters, or swamp coolers.

Environment temperature and humidity

Reduce the patient's activity, by confinement (preferably in the morning, when ambient environmental temperatures are low) lower early detection of subclinical cases.

Clients should observe mares and foals carefully, on a daily basis, and consult a veterinarian when foals appear to be unthrifty or depressed.

Removal of foals from extremes of heat and placement in well-maintained stalls providing shade or fans to lower temperature are beneficial.

Minimize exposure to high environmental temperatures, providing cooler stalls for foals treated with antimicrobial agents (especially erythromycin).

Surgical considerations

Evaluate thorax for effusion or pneumothorax.

Evaluate upper airway function to determine patency of the upper airway.

In HYPP with foals, hyperkalemic episodes may result in laryngeal paralysis.

Medications

Drug(s) of choice

The treatment protocol should specifically address any underlying conditions and hyperthermia.

Use of corticosteroids in stressed foals can result in gastrointestinal or hepatic effects. NSAIDs may result in gastrointestinal or hepatic effects. Drugs such as erythromycin/rimantadine or drugs with an effect that may be altered by concurrent therapy with drugs undergoing metabolism by the liver—chelyrexine, amphotericin.

Possible interactions

Drugs such as erythromycin/rimantadine that may change or inhibit hepatic drug metabolizing enzymes may alter the disposition of concurrently used medications (e.g., methotrexate), leading to side effects.

Septic animals are more likely to have multiorgan dysfunction, and hepatic and renal function should be monitored during therapy.

NSAIDs may result in gastrointestinal or renal compromise in anorexic and dehydrated patients.

Alternative drugs

Amphotericin (ambisome, sulfadiazine) used in combination with fl-ketoglutarate and cephalosporins.

Rifampin in combination with erythromycin, clindamycin, or zidovudine may be indicated for R. equi.

Follow-up

Patient monitoring

Reduction in body temperature and respiratory rate and effort and improvement in mucous membrane color typically indicate clinical improvement.

Frequent thoracic auscultation may reveal increased bronchovascular sounds in foals with positive response to therapy.

Arterial blood gas analysis is the most sensitive indicator function.

Repeated thoracic radiography is useful.

However, overall radiographic appearance may lag behind clinical appearance by days or weeks.

Prevention/avoidance

Client education regarding prevention and early recognition of respiratory tract disease in foals is beneficial—minimizing heat stress, control of dust, manure disposal, and plasma therapy on farms with endemic respiratory disease.

Expected course and prognosis

The initial prognosis is guarded to poor in most affected foals.

The mortality rate is high.

Long-term outcomes vary, but cases that are recognized and treated early respond well.

In survivors, the diffuse alveolar pattern tends to resolve quickly, whereas increased interstitial pattern resolves over weeks or months.

Miscellaneous

Age-related factors

Can occur at all ages.

In 1- to 8-month foals.

Suggested reading


Authors: Jeffrey Lakritz and W. David Wilson

Consulting Editor: Daniel Jean
Adenovirus

Basics

Overview
• Causes fatal respiratory disease in Arabian foals with SCID
• May be a severe pathogen in Fell Pony foals affected by "Fell Pony syndrome"
• Other breeds may be affected as foals, but seldom succumb.
• Approximately 25% of affected foals also have diarrhea.
• A rule for adenovirus in the development of respiratory disease in adult horses has been suggested.

Signs
• Signs are essentially identical to other causes of foal pneumonia and include fever, tachypnea, dyspnea, depression, and abnormalities on thoracic auscultation.
• Mild to moderate diarrhea may also be present.

Causes and Risk Factors
Foals with SCID have a defect in lymphoid stem cells that may result from altered pasteurellosis metabolism. The absence of an adaptive immune response causes these foals to be susceptible to even minor pathogens, such as adenovirus. Due to maternally derived immunity reaching a nadir at 1–2 months of age, these foals become unable to mount an appropriate immune response and deteriorate after 2 months of age. Foals that are immunosuppressed for other reasons, such as Fell Pony syndrome, are also susceptible. It has been suggested that adenovirus may predispose foals to bacterial pneumonia and may play a significant role in the pathogenesis of bacterial pneumonia in non-SCID foals. An antigenically distinct adenovirus has been identified in non-SCID foals with diarrhea, usually associated with concurrent rotavirus infection. The role of adenovirus in foal diarrhea is not clear.

Diagnosis

Differential Diagnosis
Other viral and bacterial causes of pneumonia in immunocompromised foals include, but are not limited to, the following:
• Equine herpesvirus type 1
• Equine arteritis virus
• Streptococcus suis var. suisadenumus
• Actinobacillus equuli
• Pasteurella spp.
• Klebsiella pneumoniae
• Salmonella spp.
• Bordetella bronchiseptica
• Rhodococcus equi
• Equine influenza virus
• Pasteurella multocida
• Equine adenovirus
• Equine rotavirus

CBC/Biochemistry/Urinalysis
• Persistent severe lymphopenia (≥500 cells/µL) and the absence of IgM on SRID (see below).

Other Laboratory Tests
• Antibody titers—SCID foals do not demonstrate a 4-fold rise in antibody titer to adenovirus, whereas non–SCID-affected foals develop a rise in antibody titer in 10 days.
• Virus isolation—Adenovirus may be isolated from normal and infected foals.
• Histopathology—Intranuclear inclusions can be detected in tissues. Autopsy examination may demonstrate intranuclear inclusions in conjunctival and nasal epithelial cells. At post-mortem examination there is gross and histologic evidence of lymphoid lymphophthisis of the thymus, spleen, and lymph nodes.
• SRID—Percutaneous testing of SCID foals also demonstrates an absence of IgM, but as IgM is absorbed by the foal from colostrum, foals with adequate transfer of maternal antibody cannot be tested until IgM levels have waned, usually at ≥3 weeks of age.
• Fall Pony syndrome—Measurement of IgM after 4 weeks of age (concentration will be decreased) and demonstration of B-cell lymphopenia will aid in diagnosis.

Imaging
• Radiographs are consistent with pneumonia.
• Ultrasonographic imaging of lymphoid tissues may be suggestive of, but not diagnostic for, SCID.

Treatment
• There currently is no treatment specifically for adenovirus.
• In non–SCID foals, treatment is primarily supportive, with broad-spectrum antimicrobial coverage provided.
• Foals with SCID and Fall Pony syndrome eventually die, and treatment is not productive. There has been some investigation into immunologic reconstitution of SCID patients by transplantation of bone marrow stem cells. This treatment remains experimental as of the time of this writing.

Medications

Drug(s) of Choice
• Non–SCID foals should be treated for concurrent bacterial infection based on culture and sensitivity results.
• Foals with adenovirus associated with rotavirus should be treated with supportive therapy, including intravenous isotonic polyionic fluid replacement of deficits and nutritional support as warranted.

Contraindications/Possible Interactions
N/A

Follow-up

Client Education
• Prevention of SCID requires identification of carriers and removal of them from breeding programs.
• Approximately one of four foals from the mating of two heterozygotes results in an SCID foal.
• Arabian foals should be tested at birth for IgM levels (presuckle) and lymphocyte count. Those foals with an absence of lymphopenia should be closely monitored until 5 months of age. Alternatively, there are genetic tests available now to identify carriers of the genetic defect.
• Recommendations for client education regarding Fell Pony syndrome are not yet established.

Miscellaneous

Abbreviations
• SCID = severe combined immunodeficiency syndrome
• SRID = serological radial immunodiffusion

Suggested Reading

Author: Pamela A. Wilkins
Consulting Editors: Ashley G. Boyle and Corinne R. Sweeney
OVERVIEW
- Synonymous with hypocortisolism and “steroid lixiviation syndrome”
- Characterized by glucocorticoid and mineralocorticoid deficiency caused by adrenal cortex destruction (i.e., primary AI or Addison’s disease) or ACTH deficiency (i.e., secondary AI)
- Primary AI—Both glucocorticoid and mineralocorticoid are deficient.
- Secondary AI—Mineralocorticoid secretion usually is normal.

SYSTEMS AFFECTED
- Endocrine
- Cardiovascular
- Renal
- Musculoskeletal
- GI
- Behavioral

SIGNALMENT
Any age, sex, and breed.

SIGNS
- Acute cases—muscular weakness, hypotension, anorexia, hemoconcentration, hyperthermia, polyuria/polydipsia, mild abdominal pain, colic, and diarrhea.
- Chronic cases—depression, anorexia, hemoconcentration, hyperthermia, polyuria/polydipsia, mild abdominal pain, colic, and diarrhea.

CAUSES AND RISK FACTORS
- Chronic administration of glucocorticoids, exogenous ACTH, or anabolic steroids.
- Primary-adrenal axis immaturity attributable to prematurity in neonatal foals.
- Adrenal hemorrhage and necrosis subsequent to sepsis or severe bouts of endotoxemia.

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS
- Acute cases—endotoxemia, septicemia, renal failure, and colics.
- A normal ACTH stimulation test rules out adrenal insufficiency.

CBC/BIOCHEMISTRY/URINALYSIS
- Acute AI is characterized by hyperkalemia, hypochloremia, metabolic acidosis, and azotemia.
- Chronic cases, including secondary AI—mineralocorticoid secretion (i.e., aldosterone) is generally maintained. Therefore, serum electrolytes are within normal limits.

OTHER LABORATORY TESTS
- With insufficient aldosterone secretion, fractional excretion of sodium (reference range, <1%) is increased despite a normal or low serum sodium concentration.
- Administration of exogenous ACTH (1 U/kg IM) resulting in less than a doubling of the cortisol baseline 6–8 hr later is consistent with AI. Alternatively, pulsatile ACTH (Cosyntropin 100 μg IV for a neonatal foal) may be used. Less than a doubling of the cortisol baseline 1 hr later is consistent with AI. Because acute AI is life-threatening, dexamethasone (0.044 mg/kg IV) should be administered simultaneously with exogenous ACTH. Serum cortisol is measured 2 hr later, and horses with AI exhibit a negligible increase in cortisol. This eliminates any delay in treatment while diagnostic tests are being performed.

IMAGING
N/A

DIAGNOSTIC PROCEDURES
N/A

TREATMENT
- Complete rest and avoidance of stress, particularly surgery, infection, and trauma.
- Treat the underlying primary cause.
- Provide sodium supplementation (e.g., salt) to horses with increased sodium losses.

DRUGS
- Glucocorticoid and, if necessary, mineralocorticoid replacement. The maintenance dose of prednisolone, which is equivalent to daily cortisol secretion in normal adult horses, is approximately 25 mg/day. However, exposure to stress dramatically increases corticosteroid requirements. During periods of stress, increase the dose by 2- to 10-fold and divide into 2–3 daily doses.

- Acute AI—dexamethasone in conjunction with IV crystalloid solutions (i.e., normal saline) and dextrose in cases of hypoglycemia. Although dexamethasone has minimal mineralocorticoid activity, 20 mg administered daily is sufficient to maintain adrenalectomized horses alive.
- Mineralocorticoid replacement with fludrocortisone may be considered.

CONTRAINDICATIONS/POSSIBLE INTERACTIONS
N/A

FOLLOW-UP
- Monitor electrolytes, renal function, acid-base balance, and hydration status.
- Once the animal is stable, adrenal recovery can be documented by repeating ACTH-stimulation tests.

PREVENTION/AVOIDANCE
- Avoid excessive use of exogenous glucocorticoids, ACTH, and anabolic steroids.

POSSIBLE COMPLICATIONS
- Excessive glucocorticoid administration, especially with long-acting forms (e.g., triamcinolone), increases susceptibility to infections and may result in laminitis.

EXPECTED COURSE AND PROGNOSIS
N/A

MISCELLANEOUS

ASSOCIATED CONDITIONS, AGE-RELATED FACTORS, ZOONOTIC POTENTIAL, PREGNANCY
N/A

ABBREVIATIONS
- ACTH = adrenocorticotropic hormone
- AI = adrenal insufficiency
- GI = gastrointestinal

Suggested Reading
- Author: Laurent Couvelard
- Consulting Editor: Michel Levy
AFLATOXICOSIS

BASICS

OVERVIEW
• Aflatoxicosis is the condition of intoxication by the A. flavus fungal metabolite aflatoxin.
• Diffuse liver disease is its hallmark with acute and chronic forms dictated by dose and duration of exposure.
• Aflatoxin-contaminated feed grains, especially corn, are the sources of toxin.
• Aflatoxin is usually produced on grain grown during drought conditions.

SIGNMENT
Younger horses are more susceptible.

SIGNS
• Ponies given single lethal doses of aflatoxin (2 mg/kg) had increased temperatures, elevated heart and respiratory rates, tenemus, bloody feces, and icteric coats.
• Some ponies died within 3 days while others lived for 32 days post-dosing. Ponies administered high oral doses (0.4 mg/kg for 5 days) or the equivalent of several ppm in the feed of aflatoxin were lethargic, anorectic and slightly icteric on the 5th day. Serum liver enzymes were elevated on the 4th day of dosing. Signs of hepatic encephalopathy such as belligerence, somnolence, circling, blindness, and head pressing may occur when serum ammonia levels are sufficiently elevated. Chronic low-level exposure may present as an ill-defined loss of condition.

CAUSES AND RISK FACTORS
The most likely contaminated diets are corn-based, while less likely exposure comes from diets containing peanut and cottonseed meal. Forage is an unproved source of aflatoxin.

DIAGNOSIS
• Signs and lesions of aflatoxicosis reflect liver disease. None are pathognomonic for either acute or chronic aflatoxin poisoning.
• Feed concentrations of several hundred ppb aflatoxin in grain rations, together with appropriate clinical signs, are supportive of a diagnosis.
• Ill-thrift is associated with lower levels of aflatoxin intake.

DIFFERENTIAL DIAGNOSIS
Elevated serum hepatic enzyme levels can occur in association with many multisystemic diseases. Specific causes of hepatic disease include:
• Fumonisin-induced mycotoxicosis—detection in feed
• Alkaloid dose or kleingrass toxicosis—evidence of exposure
• Hepatic neoplasia or abscessation—imaging or biopsy
• Biliary obstruction—serum chemistries and biopsy
• Theiler's disease—history, biopsy
• Parasitoid algaloid-containing plants such as Amsinckia spp., Crepidieus spp., and Senecio spp. cause chronic progressive liver disease—history of consumption, biopsy

CBC/BIOCHEMISTRY/URINALYSIS
• White blood cell counts, especially lymphocytes, are decreased and serum glucose is decreased. Total serum lipid and cholesterole are increased.
• Elevations of prothrombin time and serum AST, ALT, and GGT were consistent with the severe liver necrosis and biliary hyperplasia seen post-mortem.

OTHER LABORATORY TESTS
• Chemical analysis of feed samples is necessary to confirm the presence of aflatoxin. The inability to obtain samples at the time of exposure often precludes detection of aflatoxin levels consistent with acute intoxication.
• Feed concentrations necessary to induce acute intoxication typically approach the ppm range, while chronic exposure to several hundred ppb is sufficient to induce subclinical liver damage and associated ill-thrift.

IMAGING
N/A

OTHER DIAGNOSTIC PROCEDURES
• Necropsy findings include fatty liver, hemorhagic enteritis and pale swollen kidneys.
• Histologic changes in the liver include fatty degeneration, centrilobular necrosis, periportal fibrosis, and bile duct hyperplasia.

TREATMENT
Specific antidotes are unavailable. Horses suffering only moderate liver damage will benefit from supplementation with high-quality protein, fat-soluble vitamins, and selenium. Management for liver failure includes high-carbohydrate, low-protein diets.

MEDICATIONS

DRUG(S) AND FLUIDS
Dextrose 5% should be given slowly IV to hypoglycemic animals. Balanced electrolyte solutions are given for maintenance.

CONTRAINDICATIONS/POSSIBLE INTERACTIONS
Drugs subject to hepatic clearance should be given cautiously.

FOLLOW-UP
PATIENT MONITORING
Liver enzymes should be monitored to evaluate liver function.

PREVENTION/AVOIDANCE
Reliable feed sources are critical when grains are produced during drought conditions. Test grains before feeding.

EXPECTED COURSE AND PROGNOSIS
Survival of acute intoxication does not guarantee complete recovery. Ponies have died from liver failure up to 30 days following a single toxic dose of aflatoxin.

ABBREVIATION
• ppb = parts per billion

Suggested Reading

Author Stan W. Casteel
Consulting Editor Robert H. Poppenga

BLACKWELL'S FIVE-MINUTE VETERINARY CONSULT
African Horse Sickness

OVERVIEW
Infectious disease affecting the cardiovascular and respiratory systems, characterized by fever and edema
Not reported in the United States
Most commonly found on the African continent
with recent outbreaks investigated in South Africa, Zimbabwe, and Mozambique, India, Turkey, Iraq, Syria, Lebanon, Jordan, and Spain have reported outbreaks in the past
Geographic range of the disease is limited by that of its principal vector, Culicoides spp.
There is no apparent breed, age, or sex predilection
Angora goats are also susceptible. Zebras and elephants may serve as natural reservoirs
The incubation period ranges from 7 to 21 days.
AHS does not affect humans.

SIGNS
Fever (but not accompanied by inappetence)
Palpable edema with coughing, frothy nasal discharge, dyspnea
Subcutaneous edema of head and neck, edema of supraorbital fossa
Colic

CAUSES AND RISK FACTORS
Caused by the AHS virus, a viscerotropic RNA virus of the genus Orbicuina
Transmitted by anthropod vectors, primarily Culicoides spp., but also mosquitoes and ticks
Spread of the disease to uninfected countries can occur through travel of infected horses or movement of infected insect vectors in aircraft or heavy wind.
Virus affects vascular endothelium, resulting in the clinical sign of edema that predominates.
Disease occurs seasonally, during warm wet periods.

DIAGNOSIS
DIFFERENTIAL DIAGNOSIS
Equine infectious anemia, equine viral arteritis, purpura hemorrhagica, equine ehrlichiosis, and equine piroplasmosis may have similar clinical presentation as AHS and may require laboratory testing to differentiate it.
Index of suspicion for AHS should be raised when there is a history of travel to countries known to harbor the disease.
Congestive heart failure may result in pulmonary and subcutaneous edema, but heart murmurs and/or venous distention should be present, and fever may not be present.

CBC/BIOCHEMISTRY/URINALYSIS
N/A

OTHER LABORATORY TESTS
Definitive diagnosis depends on isolation of virus from whole blood or tissues, or antibodies to AHS virus in serum.
In the United States, if AHS is suspected, the federal area veterinarian in charge should be notified immediately so that appropriate samples can be forwarded for testing.

IMAGING
Thoracic radiography may reveal evidence of pulmonary edema.
Thoracic ultrasonography may reveal pleural effusion or pericardial effusion.

PATHOLOGIC FINDINGS
Pulmonary edema, with frothy fluid in the bronchi and trachea
Pleural effusion
Pericardial effusion
Yellow gelatinous edema fluid in the musculature of the neck and jugular groove
Pericardial hemorrhages on endocardium, epicardium, and oral mucous membranes and tongue

TREATMENT
There is no specific treatment for AHS.
Supportive nursing care and symptomatic treatment may improve outcome in some cases, but usually the course of the disease is not altered by treatment.

MEDICATIONS
DRUG(S) OF CHOICE
N/A
CONTRAINDICATIONS/POSSIBLE INTERACTIONS
N/A

PREVENTION/AVOIDANCE
• Vaccination is effective. However, 42 antigenic strains of the virus exist, and vaccination with one strain does not result in immunity to heterologous strains, so polyvalent strains of vaccine should be used.
• Vaccination should be combined with other measures aimed at limiting exposure to insect vectors, such as fly-proof stabling, pasturing only during daylight, use of insect repellents, and keeping horses on high ground away from low-lying, swampy, insect-infested areas.
• Countries free of the disease restrict importation of horses from countries known to harbor the disease, or impose quarantine of at least 60 days in insect-proof housing.

EXPECTED COURSE AND PROGNOSIS
Mortality in horses generally is high, up to 90%. In mules and donkeys, mortality may be lower (50%).
The incubation period ranges from 7 to 21 days. Once clinical signs are observed, the clinical progression is rapid. Death usually occurs within 6–5 days after the onset of fever.
Survivors do not harbor the virus.

ZOOONOTIC POTENTIAL
The disease does not affect humans.

ABBREVIATION
AHS = African horse sickness

Suggested Reading
http://www.vet.uga.edu/vprp/gray_book/
FAD/AHS.htm
The African Horse Sickness Website, The African Horse Sickness Trust, 2015,
http://www.africanhorsesickness.co.za/
Author Raymond W. Sweeney
Consulting Editors Ashley G. Boyle and Corinne R. Sweeney


**Agalactia/Hypogalactia**

**BASICS**

**DEFINITION**
- Agalactia—postpartum lactation failure
- Hypogalactia—subnormal milk production

**PATHOPHYSIOLOGY**
- Estrogens (fetoplacental unit) in late gestation induce mammary duct development.
- P4 stimulates lobuloalveolar growth.
- Lactogenesis is triggered by the sharp decrease of P4 and sharp increase of prolactin just prior to parturition.
- The increased production of prolactin by the anterior pituitary gland results from suppression of a prolactin inhibitory factor (likely dopamine) and release of a hypothalamic prolactin-releasing factor (proposed to be serotonin).
- Agalactia/hypogalactia may be caused by alterations of hormonal events (endocrine disease), defects in mammary tissue itself (mammary disease), or as a result of systemic illness or disease.

**SYSTEMS AFFECTED**
- Reproductive
- Endocrine/metabolic

**SIGNMENT**
Mares of any breed or age may be affected.

**SIGNS**

**General Comments**
- Tall fescue predominant in central and southeast United States; fescue syndrome
- Grazing endophyte-infected fescue—most likely cause of lactation failure
- Ingestion of tall fescue grass—infected with *Neotyphodium coenophialum* (formerly *Acremonium coenophialum*) or feedstuffs infected with *Claviceps purpurea* sclerotia.
- Ergot alkaloids depress prolactin secretion (dopamine D_{2} receptor agonists and serotonin antagonists).
- Abortion/premature birth affects normal P4, estrogen, and prolactin fluctuations needed for lactation onset.

**Mammary Gland Disease**
- Inflammation and/or infection
- Abscessation or fibrosis
- Neoplasia
- Trauma

**Systemic Disease**
- Any debilitating systemic disease or stress-producing disorder
- Malnutrition/nutritional deficiency

**CAUSES**

**Endocrinologic Disorders**
- Ingestion of tall fescue grass—infected with *Neotyphodium coenophialum* (formerly *Acremonium coenophialum*) or feedstuffs infected with *Claviceps purpurea* sclerotia.
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- Inflammation and/or infection
- Abscessation or fibrosis
- Neoplasia
- Trauma

**Systemic Disease**
- Any debilitating systemic disease or stress-producing disorder
- Malnutrition/nutritional deficiency

**DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS**
- Differentiate agalactia/hypogalactia from behavioral nursing problems.
- Mare anxiety, pain, udder edema
- Direct examination of udder and secretions
- Observe interaction between mare and foal as its attempts to nurse.
- Failure of milk letdown can occur in mares.
- Oxytocin stimulates milk letdown, NOT milk secretion.

**DIFFERENTIATING CAUSES**

**Endocrinologic Disorders**
- History of fescue ingestion
- Prolonged gestation
- Dystocia
- Retained fetal membranes, thickened fetal membranes
- Weak, dysmature foal, mare with agalactia

**Mammary Gland Disease**
- Full physical examination to differentiate—mastitis, mammary fibrosis, neoplasia, abscessation, traumatic injury, systemic illness

**OTHER LABORATORY TESTS**
- Serum prolactin levels are decreased in fescue-induced agalactia

**OTHER DIAGNOSTIC PROCEDURES**

**Endocrinologic Disorders**
- If history of fescue ingestion suspected
- Direct examination of udder and secretions
- Observe interaction between mare and foal
- Oxytocin stimulates milk letdown, NOT milk secretion

**Mammary Gland Disease**
- Cytology or culture of udder secretion
- Fine-needle aspirate—cytology
- Biopsy—histopathology

**OTHER LABORATORY TESTS**
- If mastitis is suspected
- Cytology or culture of udder secretion
- If neoplasia is suspected
- Fine-needle aspirate—cytology
- Biopsy—histopathology

**OTHER DIAGNOSTIC PROCEDURES**

**Endocrinologic Disorders**
- If history of fescue ingestion suspected
- Direct examination of udder and secretions
- Observe interaction between mare and foal
- Oxytocin stimulates milk letdown, NOT milk secretion

**Mammary Gland Disease**
- Cytology or culture of udder secretion
- Fine-needle aspirate—cytology
- Biopsy—histopathology
**TREATMENT**

- Mastitis
- Lactating cow intramammary treatments
- Systemic antibiotics based on culture/sensitivity
- Frequent stripping of mammary gland
- Hot-packs or hydrotherapy
- Correct nutritional deficiencies.
- FPT foals
- Nutritional supplementation during period of agalactia
- Plasma transfusions

**MEDICATIONS**

**DRUG(S) OF CHOICE FOR FESCUE TOXICITY**

- Domperidone (1.1 mg/kg PO daily)
  - Selective D₂ dopamine receptor antagonist; reverses effects of fescue ingestion.
  - Not approved by FDA, still experimental product.
  - No known side effects associated with treatment of pregnant mares.
  - Treat minimum 15 days prepartum; discontinue when/if lactation is observed at foaling.
  - If agalactic at foaling and not treated prior to parturition, initiate treatment at foaling and continue for 5 days or until lactation ensues.
- TRH—2.0 mg, SQ, BID, 5 days, begin day 1 postpartum
  - Increases serum prolactin, due to its action as a prolactin releasing factor.

**CONTRAINDICATIONS**

- Perphenazine, dopamine receptor antagonist—published, but:
  - Severe side effects in horses preclude its use.
  - Sweating, colic, hyperesthesia, ataxia, posterior paresis.
- Metoclopramide used to treat agalactia of unknown origin
  - Significant risk for developing severe CNS side effects in horses
  - Its use is contraindicated.

**PRECAUTIONS**

- Remove pregnant mares from endophyte-infected fescue pastures hay minimum 30 days, preferably 60–90 days, prepartum.
- If removal is not possible, treat with domperidone during last 2–4 weeks of gestation.

**ALTERNATIVE DRUGS**

- Acepromazine maleate (20 mg IM TID)
  - Some dopamine antagonistic properties, tried as agalactia treatment
  - At least one report of it having no effect on lactation
  - Sedation is its No. 1 primary side effect.
- Reserpine (0.5–2.0 mg IM q48h or 0.01 mg/kg PO q24h)
  - Depletes serotonin, dopamine, and norepinephrine in the brain and other tissues
  - GI motility greatly increased; can cause profuse diarrhea
  - Sedation—common side effect
  - Not Food and Drug Administration approved for agalactia
- Sulpiride (3.3 mg/kg PO daily)
  - Dopamine antagonist to treat agalactia; less effective than domperidone
  - Not FDA approved for agalactia

**FOLLOW-UP**

**PATIENT MONITORING**

- If effective, most treatments stimulate milk production in 2–5 days.
- In absence of other systemic signs, agalactia is not life-threatening.
- Foals need intensive medical and nutritional management with prolonged agalactia.

**MISCELLANEOUS**

**ASSOCIATED CONDITIONS**

- Mare
  - Prolonged gestation, abortion, dystocia, uterine rupture, thickened placental membranes, red bag, retained fetal membranes, infertility, prolonged luteal function, early embryonic death, weak and dysmature foals.
- Neonate
  - FPT
  - Malnutrition
  - Stunting
- SEE ALSO
  - Dystocia
  - Fescue toxicosis
  - Mastitis
  - Prolonged pregnancy
  - Retained fetal membranes
  - FPT

**ABBREVIATIONS**

- FPT = failure of passive transfer
- P4 = progesterone
- TRH = thyrotropin-releasing hormone

**Suggested Reading**


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**Consulting Editor** Carla L. Carleton
A

**BASICS**

**DEFINITION**

- Behavior that is, or attempt to do, injury to another with the apparent motivation of causing harm. Predation is generally not considered aggression.
- Agonistic behaviors include threat, offensive aggressive behaviors, defensive behaviors, and submissive behaviors.
- Occurs in specific contexts or circumstances and is influenced by numerous variables, including internal states (e.g., hormones, hunger, fear), external stimuli (e.g., presence of offspring), learned experiences, etc.
- Can be classified in overlapping categories—according to the target, e.g., people, predators, horses, other animals; according to function and motivation, e.g., establishing dominance status, maternal, defensive, redirected, etc.; whether offensive or defensive.

**GEOGRAPHIC DISTRIBUTION**

- Not necessarily a pathological condition, but usually a normal, species-typical behavior.
- When extreme in frequency or intensity, can be a sign of an underlying pathological condition.

**PATHOPHYSIOLOGY**

- Genetic abnormalities can influence specific behaviors and physiological systems.
- Genetics has been shown to influence specific behaviors.
- Hypertension in mares—ovarian enlargement, cystic ovaries; generally isolated stallions may exhibit aggressive behaviors.
- Anabolic steroids may cause aggression.
- Basal levels of androgens may cause aggression.
- Neurotransmitters are involved in the regulation of aggressive behaviors.
- Neurotransmitters that affect the CNS—

**GENETICS**

- Genes affect the development of behaviors.
- Any pathophysiology that results in pain can affect behavior.
- High levels of aggression may be associated with endocrine abnormalities.
- Hypothalamic-pituitary-cortical continuum.
- Agents that affect the CNS—

**RISK FACTORS**

- Insufficient designs of water and feeding sites can lead to aggression.
- Inappropriate use of punishment.
- Any age or species.

**INCIDENCE/PREVALENCE**

**Unknown**

**GEOGRAPHIC DISTRIBUTION**

**Unusual**

**SIGNALMENT**

**Breed Predictions**

- Mares may exhibit more frequent head-on approach and threat.
- Mares often exhibit aggression more frequently than stallions.
- Mares with elevated testosterone levels may exhibit more frequent head-on approach and threat.

**DIFFERENTIAL DIAGNOSIS**

- Rule-out pathological conditions, especially painful ophthalmic diseases, before establishing a nonmedical behavioral diagnosis.
- Neurological conditions or trauma.
- Inflammatory or infectious diseases.
- Endocrine abnormalities.

**DIAGNOSIS**

- Differential diagnosis.
- Rule-out pathological conditions, especially painful ophthalmic diseases, before establishing a nonmedical behavioral diagnosis.
- Psychoactive drugs.
- Feeding regimens.
- Environment.

**OTHER LABORATORY TESTS**

- Depend on clinical signs.

**BLACKWELL'S FIVE-MINUTE VETERINARY CONSULT**

**AGGRESSION**

**AGE**

- Any age.
- Maternal aggression—more common among mares with unweaned foals.
- Playful aggression—more common in young horses and colts.
- Intermale aggression—more common and intense in mature males.

**Predominant Sex**

- Intact males are more likely to show aggression to other horses and people.

**SIGNS**

**General Comments**

- Aggressive behavior ranges from mild threats to intense injurious acts.
- Mares may exhibit aggression more frequently than stallions.
- Stallions may fight with each other in the presence or absence of mares.

**Causes**

- Pain
- Fear/defense
- Play aggression
- Anthropomorphic behaviors
- Environmental factors
- Learning experiences
- Internal states (e.g., hormones, hunger, fear)
- External stimuli (e.g., presence of offspring, predators)

**Physical Examinations**

- Examine head and neck, ears, eyes, mouth, teeth, coat, skin, and in the area of the reproductive tract.
- Mares that exhibit aggression may have evidence of past injuries or pain.

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**Aggression**

- Aggression in mares, especially when accompanied by stallion-like behaviors, warrants thoroughcutaneous, estrogen, and inhibin assays.
- Karyotyping of mares exhibiting stallion-like behaviors.
- Thyroid panel

**IMAGING**
- Transrectal ultrasonography of reproductive organs.

**OTHER DIAGNOSTIC PROCEDURES**
- Racial palpation
- Ventral examination

**PATHOLOGICAL FINDINGS**
- Dependent on etiology of aggression

**DIET**
- Implemented simultaneously and it is difficult to changes in environment.
- Aggressive interventions (generally increased exercise and aggressiveness). However, numerous reported to reduce activity level and behaviors.

**ACTIVITY**
- Prevent or control access to targets of other animals that may interact with the horse.
- Assess the risks of treating and keeping a horse the animal when considering treatment.
- Adequate space to play and defer to dominant

**TREATMENT**
- Aims of treatment
  - Identify underlying reason for aggression and contributing factors.
  - Correct medical causes.
  - Specific treatments vary with the kind of aggression.
- Most treatments of nonmedical conditions involve changing the physical or social environment and/or using behavior modification techniques to change the motivational state of the animal.
- Behavior modification must be done precisely if it is to be safe and effective. Referral to an experienced and competent veterinary behaviorist, applied animal behaviorist, or trainer usually is necessary to help the client implement the plan.
- Assess the risks of treating and keeping a horse exhibiting aggression. Factors to consider are length of time the behaviors have been occurring, severity of aggression, number of situations in which the behaviors occur, predictability of the aggression, case of stopping or preventing it, number of different targets, environment of the horse, and the people and other animals that may interact with the horse.

**APPROPRIATE HEALTH CARE**
- Nursing care
- Activity
- Prevent or control access to targets of aggression.
- Increase appropriate and safe types of exercise.

**Diet**
- Reduction of energy and protein intake is reported to reduce activity level and aggressiveness. However, numerous interventions (generally increased exercise and changes in environment) are usually also implemented simultaneously and it is difficult to assess the effect of the diet.

**CLIENT EDUCATION**
- Advise owner that even if underlying medical reasons are alleviated, there may be residual behavior patterns that require behavior modification and/or training.
- Castration of stallions and colts usually reduces, but does not always eliminate, aggressive behaviors directed towards other horses and people. Age and experience of horse prior to castration are reported to be unrelated to effectiveness of castration.
- Castration only cures self-mutilation that appears to be self-directed in male aggression by stallions about 30% of the time. Seventy percent remain unaffected by castration.

**CONTRAINdications**
- Benzodiazepines may increase aggressive behavior in horses.
- Anxiolytics or antidepressants may help with fear-motivated aggression.

**Precautions**
- Inform clients that use of psychoactive drugs for aggression problems constitutes off-label and experimental use.
- Inform clients regarding possible benefits, dangers, and side effects.
- Obtain written informed consent before prescribing off-label medication.

**PossIBLE INTERACTIONS**
- No drug is approved by the US Food and Drug Administration for use with aggressive problems in horses.
- Identification of aggressive stallions for aggression problems constitutes off-label and experimental use.
- Treatment of hypothyroidism with levothyroxine can effectively reduce aggressive behavior in horses.

**SuPERVISED DRUGS OF CHOICE**
- No drug is approved by the US Food and Drug Administration for use with aggressive problems in horses.

**MISCELLANEOUS**
- Associated conditions, age-related factors, zoonotic potential, pregnancy, synonyms
- See also
  - Excuse maternal behavior/foal stealing.
  - Fear and phobias.
  - Maternal foal rejection.
  - Fears and phobias.
  - Male sexual behavior problems.
  - Endocrine disorders.
  - Training and learning problems.

**Suggested Reading**

**EXPECTED COURSE AND PROGNOSIS**
- Resolution of aggressive and stallion-like behaviors of mares with ovariectomy is good.
- Removal of normal or retained testicles in males generally results in reduction of aggressive and typically masculine behaviors. Approximately one-third retain some aggressive behaviors to other horses and interested in mares. From 5% to 17% retain some aggressiveness toward people.
- Treatment of hypothyroidism with levothyroxine can effectively reduce aggressive behavior in horses.

**POTENTIAL, PREGNANCY, SYNONYMS**
- N/A

**SURGICAL CONSIDERATIONS**
- Removal of abnormal gonads in mares (ovarian tumors, aberrant testicular tissue) has a good prognosis.

**NON-surgical CONSIDERATIONS**
- Correct medical causes.
- Identify underlying reason for aggression and address such behaviors.
- Castration of stallions and colts usually results in reduction of aggressive behavior in horses.
- Removal of normal or retained testicles in males generally results in reduction of aggressive and typically masculine behaviors. Approximately one-third retain some aggressive behaviors to other horses and interested in mares. From 5% to 17% retain some aggressiveness toward people.
- Treatment of hypothyroidism with levothyroxine can effectively reduce aggressive behavior in horses.

**SUGGESTED DRUGS OF CHOICE**
- No drug is approved by the US Food and Drug Administration for use with aggressive problems in horses.

**ALTERNATIVE DRUGS**
- N/A

**FOLLOW-UP**
- Patient monitoring
- Consult clients on a regular basis to check compliance with recommendations and to provide additional support.
- Behavioral problems generally require intensive follow-up.

**PREVENTION/AVOIDANCE**
- Rear young foals with other horses. Ideally should remain with mother for 6 mo and allowed access to other foals and (appropriate) horses as much as possible and for as long as possible.
- Sufficient exercise.
- Adequate space to play and defer to dominant horses.
- Ground work that results in the horse consistently and quickly yielding to the requests of the handler. Most easily accomplished with native and young horses.
- Avoid inappropriate use of punishment.
- If the aggression is not pathophysiological, at the first indication there might be an aggressive behavior problem; advise client to seek help from a qualified, accomplished professional who addresses such behaviors.
**Alkaline Phosphatase (ALP)**

**Basics**

**Definition**
- Serum ALP is mainly used as a marker for cholestasis.
- Routine chemistry panels report total ALP, but nonhepatic tissue (especially bone) may contribute to total ALP.
- It is infrequently used as a marker for changes in other tissues, requiring isoenzyme separation techniques.
- For routine interpretations, the potential contributions by nonhepatic tissues must be considered to accurately interpret increased ALP as evidence for cholestasis.
- Reference intervals vary depending on the assay substrate employed; thus, comparisons across labs may not be valid.

**Pathophysiology**
- Two genes produce distinct ALP isoenzymes—intestinal ALP and tissue-unspecific ALP.
- Generally, the intestinal ALP gene is expressed only in the intestine and the tissue-unspecific ALP gene elsewhere; however, the equine kidney expresses both.
- Posttranslational modification (especially glycosylation) produces additional tissue-specific isoforms of ALP (e.g., bone, liver) and affects circulating half-life.
- Various ALP forms can be quantified, but only at specialized labs.
- High tissue concentrations occur in kidney, intestine, liver, and bone; lower concentrations occur in placenta and other tissues.
- Although intestine and kidney have much higher tissue concentrations than liver and bone, renal ALP usually is not released into blood, and intestinal ALP has a very short half-life of 38 min. Thus, serum concentrations normally consist mostly of liver and, to a lesser extent, bone ALP.
- Liver ALP activity generally is greatest on the biliary canaliculus (i.e., indurate) or membrane release. Increased blood activity results from increased synthesis (i.e., induction) or membrane release. The mechanism of release into the blood is proposed to involve membrane solubilization by bile salts, release of mitochondrial fragments, or biliary regeneration. A serum phospholipase contributes to cleavage of the enzyme from the membrane.
- Cholestasis leads to increased serum ALP concentrations. Hepatocellular injury alone (e.g., carbon tetrachloride toxicity) has little effect. Bile duct ligation leads to nearly 3-fold elevations within 10 days. Presumably, much higher increases would require considerable cholestasis. Recent work suggests that ALP may cause biliary proliferation, as seen with GGT.
- Because increased ALP involves enzyme induction, serum ALP increases in acute obstructive jaundice are preceded by other markers such as conjugated bilirubin and bile salts.

**Systems Affected**
- Hepatobiliary System
- GI System
- Reproductive System
- Endocrine System
- Musculoskeletal System
- Nervous System
- Dermatologic System

**Diagnosis**

**Differential Diagnosis**
- Increased caused by bone ALP mostly are seen in growing animals. In adults, increased bone ALP likely involves lameness or obvious bony lesions. Increases from bone ALP are relatively mild.
- Highest elevations generally are associated with long-standing conditions involving severe cholestasis—chronic active hepatitis, cirrhosis, cholelithiasis, and lipidosis.
- Concurrent obesity and high enzyme values suggest hyperlipemia/lipidosis, whereas anorexia and weight loss are typical of most other differentials.

**Laboratory Findings**

**Drugs That May Alter Lab Results**

**Hematopoietic System**

**Reproductive System**

**Hematopoietic System**
- Severe anemia (e.g., acute EIA, red maple leaf toxicity, anemia, vitamin A, phosphates, phytates, phytobacteria, etc.) may cause falsely low values.

**RISK FACTORS**

**General Comments**
- Signs do not result directly from increased serum ALP activity but from the underlying disease process.
- Historical Findings
- Owners may report icterus, dark yellow/orange urine, anorexia, weight loss, listlessness, and behavioral changes associated with hepatic failure in conditions associated with cholestasis.
- Abnormal pain (e.g., swelling, rolling) may occur with acute hepatoapathy (i.e., capillary bleeding) or biliary obstruction.

**Physical Examination Findings**
- Icterus is common.
- Increased pulse and respiratory rates, fever, photophobia, weight loss, or obesity vary with the type and severity of the underlying disease process.

**Causes**

**Hepatobiliary System**
- Metabolic—secondary to severe anemia (see Hematopoietic System), hyperlipemia, or fasting (<50% increase within 2-3 days, nonpathological).
- Infectious—chronic active hepatitis, Thelers disease (i.e., serum hepatitis), amyloidosis, endotoxemia, viral (e.g., EIA, FEA, EHV in perimortem foals), bacterial (e.g., Tyzzer's disease, salmonellosis), fungal, parasitic (e.g., leishmania, toxoplasmosis), and parasitic (e.g., liver flukes, strongling larval migration).
- Nutritional—lipidosis, phosphorus, salt, or specific diet.
- Degenerative—cirrhosis, cholelithiasis
- Toxic—pyrolidinic alkaloid-containing plants (e.g., senecio, crotolaria), alake clover, alluvial, trauobacter, chemical toxins (e.g., arsene, chlorothalidone, phenol, paraquat) primarily cause hepatocellular injury; cholestasis secondary to hepatocellular swelling may increase ALP. Some anesthetics (e.g., halothane) are associated with mild, transient increases.
- Aniony—serum creatinine, porcine endocardial growths, neoplastic—primary liver tumors (rare); metastatic neoplasia (uncommon).

**Musculoskeletal System**
- Rapid bone growth—juvenile—Severe bony lesions

**GI System**
- Severe GI disease—diarrhea

**Reproductive System**
- Pregnancy increases placental ALP with mild increase in total serum ALP.

**Hematopoietic System**
- Severe anemia (e.g., acute EIA, red maple leaf toxicity, anemia, vitamin A, phosphates, phytates, phytobacteria, etc.) may cause falsely low values.
- Complexing anticoagulants (e.g., citrate, EDTA, oxalate) inhibit the enzyme and should not be used for sample collection.

**Disorders That May Alter Lab Results**
- Extreme anemia, severe lipemia, and marked hemolysis may affect values.
- Activity tends to increase with storage.

**Valid If Run in Human Lab?**

**Valid, but increases vary with the methodology used.**
- Equine reference intervals should be generated in-house or based on literature values using the same methodology.

**CBC/Biochemistry/Urinalysis**

- No routine laboratory tests provide a causative or specific diagnosis for increases.
- Most suggest a type of injury (e.g., process, shunting, insufficiency) rather than a cause.
- Others, confirming the presence of cholestasis, support a suspected hepatic origin for the increase.

**Erythrocytes**
- Nonregenerative anemia may be seen with liver disease.
- Microcytosis is associated with pernicious anemia.
- Acanthocytes and echinocytes (hepatic microvascular disease) are associated with decreased RBC survival and may contribute to mild hemolytic anemia.
- Severe hemolytic anemia (regardless of mechanism) can cause hepatic injury leading to hepatocellular swelling and secondary cholestasis.
LEUKOCYTES

- Neutrophilia or neutropenia and monocytenosis may occur with inflammatory liver disease—bacterial (bacterial hepatitis). • Evidence of antigen stimulation (e.g., lymphocytes, reactive lymphoid cells) may be seen.

GLUCOSE

- Postprandial hyperglycemia or fasting hyperglycemia may occur with hepatic insufficiency/shunts.
- Hyperglycemia with liver disease carries a guarded prognosis.

ALBUMIN

- Decreased production with hepatic insufficiency may decrease serum levels; usually a late event.
- Albumin is a negative acute-phase reactant—Mild decreases may occur with inflammation.

BUN

- Decreased levels (especially relative to creatinine) occur with hepatic insufficiency/shunts due to decreased conversion of ammonia to urea.

GGT

- Increased with either injury or cholestasis

BILIRUBIN

- Conjugated—increases with cholestasis
- unconjugated—increase with increased RBC destruction (i.e., hemolysis), or decreases hepatic uptake and with fasting

CHOLESTEROL

- May decrease with hepatic insufficiency/shunts
- Sometimes increases with cholestasis and lipid metabolic disorders—hyperlipemia

TRIGLYCERIDES

- Increased with hyperlipemia

URINALYSIS

- Bilirubinuria indicates cholestasis.
- Ammonium urates may be observed with hepatic insufficiency/shunt.

OTHER LABORATORY TESTS

Bile Acids

- Sensitive indicator of hepatic disease but not specific for the type of process—liver injury, cholestasis, or insufficiency.
- Anosus enterohepatic circulation, adequate hepatopulmonary perfusion, and hepatopulmonary function.
- More sensitive than ALP for cholestasis

Ammonia

- Serum concentrations are affected by hepatic uptake and correlate inversely with hepatic functional mass.

Clearance Tests (BSP, ICG)

- Prolonged clearance intervals with decreased functional mass or cholestasis
- Accelerated clearance (possibly masking insufficiency) with hyperammonemia

Serology

- Depends on the degree of suspicion for specific diseases—viral, fungal, and so on

Coagulation Tests

- May be prolonged with hepatic insufficiency/shunting—prothrombin time, activated partial thromboplastin time.

IMAGING

- Ultrasonography—useful for assessing liver size, shape, position, and parenchymal texture; may help to detect focal parenchymal lesions (e.g., abscesses, neoplasms) and abnormalities in the biliary tree (e.g., dilatations, obstructions) or large vessels (e.g., shunts, thromboses).

DIAGNOSTIC PROCEDURES

- Aspiration cytology or biopsy for microbiologic testing, cytologic imprint, and histopathological evaluation may provide specific diagnostic information.

TREATMENT

- Decision regarding outpatient versus inpatient care depends on the severity of disease, intensity of supportive care required, need for isolation of infectious conditions, and so on.
- Fluid and nutritional support may be needed.
- Anemic and hyperglycemic cases may benefit from IV 5% dextrose (2 mL/kg per hr).
- Otherwise, fluid support depends on specific electrolyte and acid-base abnormalities.
- Avoid negative energy balance, especially in ponies and donkeys, to avoid/treat hyperlipemia and hepatic lipidosis.

MEDICATIONS

DRUG(S) OF CHOICE

- On the suspected cause and observed complications

CONTRAINDICATIONS

- On the suspected cause and observed complications

PRECAUTIONS

- On the suspected case
- With suspected hepatic insufficiency, assess coagulation profile before invasive procedures.

ALTERNATIVE DRUGS

- On the underlying cause

FOLLOW-UP

- Serial chemistries can help to establish a prognosis by characterizing disease progression and identifying evidence of improvement—Initial evaluation at 1- to 2-day intervals helps to establish the disease course; subsequent testing can be at increasing intervals, depending on signs and severity.

PREVENTION/AVOIDANCE

- Depends on the underlying cause

POSSIBLE COMPLICATIONS

- Depend on the underlying cause

EXPECTED COURSE AND PROGNOSIS

- Depend on the underlying cause

ASSOCIATED CONDITIONS

- Depend on the underlying cause
- One study showed ALP values >900 IU/L were associated with increased risk of nonsurvival (hazard ratio = 10.66).

AGE-RELATED FACTORS

- See Signalment

ZOOONOTIC POTENTIAL

- N/A

SEE ALSO

- See Signalment

SYNONYMS

- N/A

ABBREVIATIONS

- BSP = bile-salt-staining bile
- EHV = equine herpesvirus
- EIA = equine infectious anemia
- EHV = equine viral arteritis
- GGT = γ-glutamyltransferase
- GI = gastrointestinal
- ICG = indocyanine green

Suggested Reading


The author and editor wish to acknowledge the contributions in this chapter of Armando Fierro-Ruiz, author in the previous edition.

Author: John A. Christian
Consulting Editor: Kenneth W. Hinchcliff
Alkalosis, Metabolic

**BASICS**

**DEFINITION**
- A disruption of acid-base homeostasis producing decreased H⁺ concentration reflected by alkalemia—increased pH and high plasma HCO₃⁻, [HCO₃⁻], or BE.
- Normal plasma bicarbonate level in horses is ≥24 mEq/L.
- Normal pH of arterial blood ranges from 7.35 to 7.45.
- Hyperventilation should increase CO₂ levels to lower pH; however, respiratory compensation is limited once hypoxemia develops.

**PATHOPHYSIOLOGY**
- The kidney normally is extremely capable of responding to a high pH; correcting metabolic alkalosis (MAK) via excretion of HCO₃⁻ into the urine. Even with daily administration of high bicarbonate levels, the alkalosis is short lived in normal horses. Therefore, MAK persists only when an initiating factor develops simultaneously with conditions in which renal excretion of HCO₃⁻ is impaired or respiratory compensation is enhanced.
- Excessive loss of H⁺, retention of HCO₃⁻, and contraction of ECF volume without loss of HCO₃⁻ (i.e., contraction alkalosis) are the common mechanisms thought to initiate MAK.

**SYSTEMS AFFECTED**

**Respiratory**
- Peripheral and central chemoreceptors sense high pH in blood or CSF and depress ventilation to decrease removal of CO₂; hypercapnia and hypoxemia may follow.

**Cardiovascular**
- Cardiac arrhythmias
- Arterial vasodilatation
- Decreased coronary blood flow

**Neuroendocrine**
- Decreased cerebral blood flow caused by vasodilatation
- Neurologic signs (e.g., delirium, seizures, lethargy, stupor) are rare but can be seen with severe alkalosis.
- Neuromuscular excitability and tetany may occur.

**Diagnosis**

**Differential Diagnosis**
- Increased bicarbonate levels also are seen in conditions with respiratory acidosis. Po₂ is high but the pH close to normal or high on blood gas analysis.
- Compensation may be very effective in chronic respiratory acidosis.

**Laboratory Findings**

**Drugs That May Alter Lab Results**
- Excessive anticoagulant may falsely decrease HCO₃⁻ levels.

**Valid If Run In Human Lab?**
- Yes, if properly submitted

**CBC/Biochemistry/Urinalysis**
- Measurements of serum electrolytes, protein levels, and serum chemistries are important to determine the cause and to guide treatment.
- Proportionate changes in sodium and chloride levels occur with alterations of fluid balance. Normal sodium levels with hypochloremia or hyperchloremia indicate acid-base imbalance, whereas disproportionate changes usually are associated with simultaneous acid-base imbalance and hydration abnormalities.
- Potassium and chloride are decreased in horses that sweat excessively. Potassium may be low because of the primary cause or as a response to the extracellular shift of H⁺.
- Ionized calcium is decreased.
- Magnesium may be decreased, especially with stress and exercise.
- Urticaria may reveal decreased urine pH.

**OTHER LABORATORY TESTS**
- Many labs measure TCO₂ using the same sample submitted for electrolytes.

**Alkalosis, Metabolic basics**

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**Other Laboratory Tests**
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Alkalosis, Metabolic

The $T_{\text{CO}_2}$ closely approximates $HCO_3^-$, because most $CO_2$ is carried in the blood as bicarbonate. Like MAK, respiratory alkalosis also results in high $T_{\text{CO}_2}$. These conditions can be differentiated only by complete blood gas analysis. The $T_{\text{CO}_2}$ must be analyzed rapidly and with minimal room-air exposure within the sample tube, because $CO_2$ can dissipate from the sample.

**TREATMENT**
- Treatment of the primary cause is essential.
- Replacement of fluid losses with isotonic fluids may be all that is needed to restore acid-base status in mild cases.
- Address specific electrolyte losses.
- Large volumes may be needed in some endurance athletes with excessive fluid losses from sweating or hyperthermia.

**MEDICATIONS**
- With hypochloremia, give fluids containing chloride, or the alkalosis will not be corrected even if hydration is restored.
- Saline or Ringer's solution with added calcium and KCl is the fluid of choice.
- With excessive potassium loss, PO supplementation is necessary if the horse remains anorexic.
- With hypokalemia, give fluids containing potassium, or the alkalosis will not be corrected even if hydration is restored.
- Saline or Ringer's solution with added calcium and KCl is the fluid of choice.
- With excessive potassium loss, PO supplementation is necessary if the horse remains anorexic.

**CONTRAINDICATIONS**
- Any alkalizing therapy (i.e., LRS) can worsen the alkalosis. Check contents of oral electrolyte therapies closely.

**PRECAUTIONS**
- Give calcium-containing solutions slowly to avoid arrhythmias.
- Monitor cardiac rhythm during administration.

**POSSIBLE INTERACTIONS**
- N/A

**ALTERNATIVE DRUGS**
- Oral rehydration solutions have achieved good results in horses, being very effective in mild cases and an excellent adjunct to IV therapy. From 1–2 gallons can be given PO every few hours to adults without ileus.

**FOLLOW-UP**
- Serial blood gas analysis and measurement of electrolytes and calcium are very important in evaluating efficacy of therapy; repeat within a few hours of initial treatment and thereafter according to patient response.

**MISCELLANEOUS**

**ASSOCIATED CONDITIONS**
- Hypochloremia
- Hypokalemia
- Respiratory acidosis

**SEE ALSO**
- Exertional rhabdomyolysis
- Exhausted horse syndrome
- Heat exhaustion
- Hyperthermia

**ABBREVIATIONS**
- $BE$ = base excess
- $CSF$ = cerebrospinal fluid
- $ECF$ = extracellular fluid
- $GI$ = gastrointestinal
- $LRS$ = lactated Ringer's solution
- $MAK$ = metabolic alkalosis

**Suggested Reading**

**Author** Jennifer G. Adams
**Consulting Editor** Kenneth W. Hinchcliff
Alkalosis, Respiratory Basics

**DEFINITION**
- A decrease in blood P_{CO_2} and pH
- Arterial P_{CO_2} tensions of <35 mm Hg
- Venous P_{CO_2} tension <43 mm Hg

**PATHOPHYSIOLOGY**
- Most (65%–70%) CO_2 combines with water almost instantaneously to form carbonic acid, which then dissociates into bicarbonate ion and hydrogen. Therefore, most CO_2 is transported in the blood as bicarbonate, with some bound to proteins (especially deoxygenated hemoglobin) and a small amount dissolved directly into plasma.
- In the lungs, the reverse occurs, and CO_2 passively diffuses out of capillaries and into the alveoli.
- These three forms of CO_2 exist in equilibrium in the blood, but P_{CO_2} as measured by blood gases depends on the dissolved portion.
- Alveolar CO_2 then is removed mechanically by ventilation as inspired air displaces alveolar gas, which is expired.
- Respiratory alkalosis is present with hyperventilation or when tissue production of CO_2 drops but ventilation remains unchanged.

**SYSTEMS AFFECTED**
- The brain is most affected by CO_2 levels, because hypocapnia decreases cerebral blood flow.
- Low pH affects acid-base balance, protein binding, and electrolyte levels directly in the blood and via effects on the kidney.
- The kidney responds to low pH by generating more H^+ and excreting more HCO_3^-. It also reabsorbs Cl^- to maintain electroneutrality. Alkalosis decreases serum potassium and ionized calcium levels.
- Severe alkalosis can cause vasoconstriction and predispose to arrhythmias, and it may result in hyperexcitability of muscle and nervous tissue.

**SIGNALMENT**
- Any horse

**SIGNS**
- Respiratory rate, volume, or both usually are increased.

**CAUSES**

**Acute**
- Usually is a temporary change in response to a stimulus causing hyperventilation
- Physiologic causes of hyperventilation— exercise, fever, and hyperthermia
- Psychological causes—pain, anxiety, excitement, and fear
- Stimulation of medullary respiratory centers by CNS disorders, early septicemia, acidosis, or endotoxemia may result in hyperventilation.
- Anemia, hypoproteinemia, and hypoxemia of any cause increase respiration in response to tissue hypoxia.

**Chronic**
- May result from chronic respiratory disease (e.g. pleuropneumonia) or chronic, painful conditions (e.g., laminitis, septic arthritis)
- Overventilation with mechanical ventilators produces low P_{CO_2} in anesthetized patients and sick neonates.
- Hypothermia or decreased metabolic rates seen with prolonged general anesthesia may lower tissue CO_2 production and produce respiratory alkalosis in patients ventilated at appropriate settings.
- Also seen as a compensatory response to primary metabolic acidosis

**DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS**
- Physiologic states or disease processes that present with tachypnea—fever, hyperthermia, excitement, anxiety, painful conditions, hypoxemia, metabolic acidosis, and CNS derangements; most of these can be differentiated with history and physical examination findings.
- Most acute problems have low pH and low P_{CO_2}.
- Chronic respiratory alkalosis results in compensatory metabolic acidosis, in which bicarbonate is low and pH should be normal, because compensation is very effective in this circumstance.
- Acute metabolic acidosis has low bicarbonate. Often, pH remains low in severe cases, because respiratory compensation rarely is complete.

**LABORATORY FINDINGS**

**Disorders That May Alter Lab Results**
- With poor peripheral perfusion or cardiovascular shunt, results of blood gas analysis on samples taken from peripheral arteries may differ from those taken elsewhere or may not reflect the patient’s overall systemic condition.
• Prolonged exposure to air may alter CO₂ levels, because RBC metabolism continues and equilibration with room air may occur.

Valid If Run in Human Lab?
Yes, if properly submitted

OTHER LABORATORY TESTS
• Blood gas analysis is the definitive laboratory test.
• Venous samples may be adequate to identify the condition, but arterial samples are necessary to evaluate adequacy of pulmonary function as a cause.
• Handheld analyzers are now available and easy to use. Some require only small amounts of whole blood; otherwise, heparinized syringes before sampling.
• Perform sampling anerobically immediately evacuate any air bubbles present, and cap the needle with a rubber stopper.
• Analysis should be performed within 15–20 min. If not, samples can be stored on ice, and results will be valid for 3–4 hr.

DIAGNOSTIC PROCEDURES
• Capnography is an indirect method of measuring CO₂ levels.
• Samples of ET gases reflect arterial Pco₂, because ET gas is essentially the same as alveolar gas.
• Continuous monitoring can be performed on anesthetized or ventilated patients via a gas-sampling port incorporated into the endotracheal tube or attached to an adapter.
• Because some V/Q mismatch usually is present, ET levels may underestimate arterial tension by 10–15 mm Hg.
• Periodically compare values obtained via capnography with those via blood gas analysis.

TREATMENT
• Most often, treatment of the primary problem resolves the need for hyperventilation, or metabolic rate will increase and normal Pco₂ levels return.
• Alteration of ventilator settings in anesthetized patients is necessary to return CO₂ and pH to normal; however, this may decrease oxygen levels.

MEDICATIONS
PRECAUTIONS
• Monitor ventilated patients continuously for airway obstruction caused by accumulation of secretions, movement of endotracheal tube, kinking of tubing or hoses, and so on.
• Inspiratory pressure should range from 20 to 30 cm H₂O in normal patients.
• Pressures of ≥40 cm H₂O may be utilized in patients with abdominal distention—those anesthetized for colic surgery.
• Pressures of >40 cm H₂O compromise venous return and cardiac output.

FOLLOW-UP
PATIENT MONITORING
• Decreased respiratory effort should be seen quickly after resolution of the primary problem.
• Evaluate repeat blood gases analyses soon after institution of mechanical ventilation to ensure appropriate settings have been selected. Further evaluation thereafter is dictated by the patient’s condition.

POSSIBLE Complications
Severe alkalemia can result in neurologic signs from decreased cerebral blood flow, muscular excitability, and cardiac arrhythmias.

MISCELLANEOUS
ASSOCIATED CONDITIONS
• Hyperchloremia
• Hypokalemia
• Metabolic acidosis
PREGNANCY
Pregnant females often hyperventilate because of decreased lung volume caused by abdominal distention from the gravid uterus.

SYNONYMS
• Hypocapnia
• Hypocarbia

ABBREVIATIONS
• ET = end-tidal, refers to gas expired at the end of expiration, which should be the alveolar gas most recently involved in gas exchange
• V/Q = ventilation-perfusion ratio

Suggested Reading


Author Jennifer G. Adams
Consulting Editor Kenneth W. Hinchcliff
Alopecia is an absolute decrease in the number of hairs per given area of body surface or hairs that are shorter than normal even though their number is within normal limits. It is a loss or lack of the hair from skin areas where it is normally present.

**DEFINITION**

- Alopecia is congenital or acquired.
- **Congenital alopecia** is rare in horses.
- **Aquired alopecia** can be subdivided into infectious and noninfectious causes. Common etiologies of acquired alopecia are adenal destruction or atrophy secondary to infection, physical trauma, immune-mediated reactions, nutritional supplements and deficiencies, toxicities, physiologic stressors, and miscellaneous causes.

**PATHOPHYSIOLOGY**

- **Acquired alopecia** represents a disruption in the growth of the hair follicle with or without damage to the hair bulb, follicular wall, hair shaft, or both. The animal is born with a normal hair coat, has or had normal hair follicles at one time, and is or was capable of producing structurally normal hairs.
- **Congenital alopecia** is the result of abnormal morphogenesis or lack of adnexa (therefore hair) in regions of the body where they normally are expected. Animals with congenital hypotrichosis may be born with varying degrees of hypotrichosis or a complete haircoat; however, if born with a complete haircoat, a rapid onset of progressive permanent alopecia within the first few months of life ensues.

**SYSTEM AFFECTED**

Skin/exocrine

**GENETICS**

Congenital alopecia does not necessarily imply a genetic basis, although in most cases the disease is based on genetic abnormalities and thus is hereditary. The exact mode of inheritance is unknown.

**INCIDENCE/PREVALENCE**

- True incidence is unknown.

**GEOGRAPHIC DISTRIBUTION**

- Presumably worldwide.

**SIGNALMENT**

- **Congenital hypotrichosis** has been documented in certain Arabian lines and a blue roan Percheron.
- **Appaloosas** with foundation bloodlines have hair dystrophy/thinning of the long mane and tail hair.
- **Aquired alopecia** can occur in all breeds.
- **Both sexes** are affected equally.

**SIGNS**

**General Comments**

- May be an acute onset or slowly progressive.
- **Multifocal patches of circular alopecia** are most commonly associated with bacterial folliculitis, dermatophytosis, or dermatophytosis.
- **Large diffuse areas of alopecia** may indicate an immune-mediated etiology or congenital abnormality.
- **Congenital hypotrichosis** may be regional, multifocal, or generalized. It might become clinically apparent only weeks after birth and usually does not continually progress with age.

**CAUSES**

- **Noninfectious**
  - Mild to moderate inflammation of the hair follicle (folliculitis and furunculosis).
  - Defects in the hair shaft.
  - Hair follicle dystrophies.
  - Altered hair follicle function.
  - Trauma (self-induced from pruritus).
  - Cicatricial alopecia (scarring causes).
  - Physical, chemical, or thermal injury.
  - Severe furunculosis.
  - Neoplasia.
  - Severe inflammatory disease such as in cutaneous onchocerciasis.
- **Congenital Causes**
  - **Congenital hypothyroidism** may be a cause of congenital hypotrichosis and alopecia.
  - **Tichothrix nodosa** is a hair shaft defect that may be hereditary or acquired.
- **Congenital hypothyroidism**
  - **Epidermolysis bullosa**
  - **Mane and tail dystrophy**
  - **Follicular dysgenesis**

**Acquired Causes**

- **Infectious**
  - **Bacterial**
    - The most common bacterial infection is dermatophilosis. Folliculitis and furunculosis due to *Staphylococcus* spp. and *Corynebacterium pseudodiphtheritae* are uncommon. Other bacterial causes are abscesses due to *Streptococci* and *Staphylococci* spp.
  - **Fungal**
    - *Dermatophylosis* due to *Microsporum* gypseum, *M. equinum* or *M. canis* or *Trichophyton equinum var. equinum* causes alopecia. Other fungal causes are mycetoma and the subcutaneous mycosis such as phymatolysis and pythiosis.
  - **Parasitic**
    - Follicular parasitic infections of the follicle that result in alopecia are rare and include *Demodex* spp. and *Trombiculidae* spp.
  - **Viral**
    - Follicular viral infections of the follicle that result in alopecia are rare and include *Herpesvirus* spp. and *Pseudovirus* spp.
- **Noninfectious**
  - **Immune mediated**
  - Cell-mediated autoimmune disease directed toward the hair follicle and adnexa.
  - **Alopecia areata**
  - **Hair follicle dystrophy**—possible variant of alopecia areata.
  - **Sarcoidosis**
  - **Systemic lupus erythematosus**
  - **Bacterial**
  - **Fusiformis**
  - **Embryonic**
  - **Pseudohypertrophic**
  - **Systemic lupus erythematosus**
  - **Sarcoidosis**
  - **Physical**
  - **Burns** from chemicals, hot, cold, or ropes.
  - **Scalp** from friction, urine, or feces.
  - **Tail and mane rubbing** as stable vice.
**Alopecia**

**RISK FACTORS**
N/A

**DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS**

- Accurate diagnosis of alopecia requires a careful history and physical examination.
- Key points in the history include recognition of breed predispositions for congenital alopecia, the duration and progression of lesions, the presence or absence of pruritus, or evidence of contagion.
- Progression of lesions, the presence or absence of pruritus, or evidence of contagion.
- The distribution of lesions should be noted (focal, multifocal, generalized, or symmetrical), and the hairs examined to determine if they are being shed from the hair follicle or broken off. Signs of secondary infections or ectoparasites should be noted.
- Key points in the history include recognition of breed predispositions for congenital alopecia, the duration and progression of lesions, the presence or absence of pruritus, or evidence of contagion.
- The distribution of lesions should be noted (focal, multifocal, generalized, or symmetrical), and the hairs examined to determine if they are being shed from the hair follicle or broken off. Signs of secondary infections or ectoparasites should be noted.
- The degree of crusting, scale, and exudate helps prioritize the differentials.
- Patchy, localized to multifocal
- Bacterial folliculitis and furunculosis
- Dermatophilosis
- Dermatomyositis
- Linear alopecia
- Alopecia areata—results from selective and reversible damage to anagen hair follicles. Initial lesions may be focal, multifocal well-circumscribed alopecia that progresses to diffuse alopecia. The mane and tail of horses are often involved and hoof dystrophies can occur. The alopecic skin has minimal or no visible inflammation. Prognosis varies, some cases spontaneously resolve, some respond to immunosuppressive doses of steroids, while others have no hair regrowth.
- Generalized, symmetrical, and large patchy multifocal

**DIAGNOSTIC PROCEDURES**

- Cytology should be obtained from pustules, papules, erosions, or ulcers. Neutrophils predominate with intra- and extracellular cocci and gram-negative rods. Bacterial exudate may be absent or minimal. Culture to identify bacteria, including anaerobes. Culture of skin scrapings or lesions for aerobic and anaerobic bacteria. Culture of crusts or pustules for aerobic and anaerobic bacteria.
- Direct hair examination (microscopy): Hairs will either have anagen or telogen roots. Telogen hairs have uniform shaft diameters and slightly rough-surfaced tapered spear-shaped non-pigmented roots. Anagen hairs have rounded smooth pigmented bulbs that bend. Distal ends of hair shafts may appear fractured from self-induced trauma. No normal animal should have all of its hairs in telogen but rather should have an admixture of anagen and telogen. Anagen defluxion reveals fragmented hair shafts with the absence of roots.
- Perform skin scrapings to rule out ectoparasites.
- Perform bacterial and DTM cultures to determine bacterial species and susceptibility.
- Perform skin biopsies if the tests listed above do not identify or suggest an underlying cause. A biopsy evaluates hair follicles, adnexal structures, inflammation, and anagen/telogen ratios. Biopsies may reveal evidence of bacterial, parasitic or fungal causes of alopecia but should NOT be considered as the definitive test for determination of alopecia caused by infectious agents. If cytocentrifugation reveals evidence of folliculitis, treat the patient with appropriate antimicrobials, parasiticides, or antifungals for a minimum of 3 weeks. If no improvement in the degree of alopecia is noted, obtain a biopsy for histopathology, preferably, while the patient is still receiving treatment. Often biopsies submitted from patients with moderate to severe bacterial folliculitis make it difficult to determine and may mask the primary cause of alopecia. Submit biopsies from affected and non-affected sites.
- Definitive diagnosis of alopecia areata requires histologic confirmation. Multiple biopsies need to be collected as pathognomonic lesions can be sparse. Biopsy from newly developed areas of alopecia, rather than older lesions.

**PATIENT FINDINGS**

- Biopsies of telogen defluxion are misleading, as they will demonstrate most follicles in the active growing (anagen) phase. Often the hair cycle has returned to normal by the time the definitive diagnosis is made.
- Anagen effluvium findings include apoptosis and fragmented cell nuclei in the keratinocytes of the hair matrix of anagen follicles, as well as eosinophilic dysplastic hair shafts within the pilar canals.

**OTHER LABORATORY TESTS**
N/A

**IMAGING**
N/A
Alopecia

**Alopecia areata** has two major histologic features. The first is hair follicle miniaturization and the second feature is lymphocytic bulbitis. The lymphocytic bulbitis involves anagen follicles and is best found in recently developed areas of alopecia. A lymphocytic mural folliculitis affecting the follicular isthmus is possible. The bulbitis may be very difficult to demonstrate especially in chronic lesions where the inflammation may be nonexistent. Chronic lesions only exhibit small telogen follicles lacking hair shafts that may be somewhat atrophic.

Histologic findings of alopecia secondary to infectious organisms are covered in the appropriate dermatology sections.

**TREATMENT**

**AIMS OF TREATMENT**

The clinical approach to alopecia is to identify the cause and, if the etiology is something that may benefit from pharmaceutical treatment, then therapy may resolve the clinical signs.

**APPROPRIATE HEALTH CARE**

Relevance equated to etiology; most require outpatient medical management.

**NURSING CARE**

Relevance equated to etiology.

**ACTIVITY**

Patients with multifocal to generalized hypotrichosis may be more susceptible to hypothermia and solar dermatoses.

**DIET**

Telogen effluvium has been associated with the administration of a feed supplement.

**CLIENT EDUCATION**

Relevance equated to etiology.

**SURGICAL CONSIDERATIONS**

N/A

### MEDICATIONS

**DRUG(S)**

- Varies with cause
- Dermatophytosis—lime sulfur or miconazole, miconazole/chlorhexidine rinses; systemic griseofulvin
- Dermatophilosis—topical antimicrobial therapy
- Bacterial folliculitis—systemic and topical antimicrobial therapy
- Pemphigus foliaceus—immunosuppressive therapy
- There are no hair growth–promoting pharmaceuticals for horses.

**CONTRAINDICATIONS**

N/A

**PRECAUTIONS**

N/A

**POSSIBLE INTERACTIONS**

None

**ALTERNATIVE DRUGS**

N/A

### FOLLOW-UP

**PATIENT MONITORING**

Varies with cause

**PREVENTION/AVOIDANCE**

Varies with cause

- Patients with documented congenital alopecia and their parents should not be used for breeding.

**POSSIBLE COMPLICATIONS**

N/A

### EXPECTED COURSE AND PROGNOSIS

- Prognosis is based on whether the alopecia is classified as noncicatricial or cicatricial.
- Cicatricial alopecia is characterized by permanent destruction of the hair follicles and regrowth of hair will not occur.
- In noncicatricial alopecia, future hair growth will occur if the causative factors are eliminated or corrected.
- Telogen and post anagen effluvium resolve upon identification and elimination of cause.

### MISCELLANEOUS

**ASSOCIATED CONDITIONS**

N/A

**AGE-RELATED FACTORS**

N/A

**ZOONOTIC POTENTIAL**

Dermatophytosis and dermatophilosis are zoonotic.

**PREGNANCY**

- Post-partum telogen effluvium is thought to be due to the physiologic stress of pregnancy and lactation.
- Avoid the use of griseofulvin to treat dermatophytosis in pregnant mares.
- Mares that receive iodine-deficient diets give birth to weak or dead foals with no haircoat.

**SYNONYMS**

- Alopecia = hypotrichosis
- Telogen effluvium = telogen defluxion or defluvium
- Anagen effluvium = anagen defluxion or defluvium

**SEE ALSO**

- Dermatophytosis
- Pemphigus foliaceus
- Bacterial folliculitis
- Dermatophilosis
- Linear alopecia
- Sarcoids

**ABBREVIATION**

DTM = dermatophyte test medium

**Suggested Reading**


Author: Gwendolen Lorch

Consulting Editor: Gwendolen Lorch
Amitraz Toxicosis

BASICS

OVERVIEW
- Amitraz is a formamide acaricide widely used for the control of mites and ticks in veterinary medicine.
- While not approved for use in horses, it is sometimes used intentionally for ectoparasite control and accidental exposures may occur. Amitraz may be deliberately administered intravenously to alter performance in athletic horses.
- Amitraz has complex pharmacological and toxicological effects in animals. It acts on α₂-adrenergic receptors in the central nervous system and both α₁ and α₂-adrenergic receptors in the periphery. It is also believed to inhibit monoamine oxidase, block prostaglandin E₂ synthesis, and cause a local anesthetic effect.
- Amitraz-induced central nervous system stimulation or depression appears to be dose dependent with high doses causing depression while low doses result in hyperactivity to external stimuli, and in some cases, aggressive behavior.
- Amitraz reduces smooth muscle activity in the gastrointestinal tract. Clinically and experimentally, this results in a reproducible and reversible impaction colic syndrome.
- Amitraz depresses respiratory rate centrally, probably by inhibiting respiratory neurons located in the ventral portion of the brain. α₂-Adrenergic agonists can reduce both sensitivity of the breathing center to increased Pco₂ and tidal volume, thus accentuating respiratory depression.
- Amitraz inhibits adrenergic hormone and thus may promote diuresis.
- Amitraz and its active metabolite both induce hyperglycemia and hyperinsulinemia by inhibiting insulin secretion mediated by α₂-adrenergic receptors located within the pancreatic islets.
- Amitraz is more slowly metabolized in horses than sheep, which may explain its toxicity in equines.
- Clinical signs of amitraz toxicity are usually referable to the central nervous or gastrointestinal systems.

SIGNALMENT
N/A

SIGNS
- Affected horses display signs of autonomic dysfunction, anorexia, muscular incoordination, and impaction colic, which may persist for days.
- The impaction colic syndrome is characterized by rapid cessation of gastrointestinal sounds, gastrointestinal stasis, extensive impaction, and tympany throughout the large colon.

CAUSES AND RISK FACTORS
- Amitraz toxicity after topical application is due either to deliberate exposure for parasite control or accidental exposure.
- Because of its known sedative/tranquilizing actions, amitraz may be deliberately administered intravenously to alter performance in athletic horses.
- Amitraz in stored solutions may break down to the highly toxic N-(3,5-dimethylphenyl)-N-methyl-formamide derivative and more easily induce toxicosis.
- In a chronic low-dose toxicity study in horses, there were no demonstrable adverse effects from amitraz.

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS
- Signs of colic can be due to many other disorders.
- Signs of depression and anoxia can be due to viral disease (e.g., rabies, equine encephalomyelitis, West Nile virus), hepatopathophathy, meningitis, and brain abscess or tumor.

CBC/BIOCHEMISTRY/URINALYSIS
- With acute intoxication, total protein and packed cell volume may increase due to dehydration and a mild acidosis may be seen.
- Hyperglycemia and hyperinsulinemia result from inhibition of insulin release.

OTHER LABORATORY TESTS
Drug-testing laboratories have methods for the detection of amitraz and its major metabolite in performance horses.

IMAGING
N/A

OTHER DIAGNOSTIC PROCEDURES
N/A

PATHOLOGICAL FINDINGS
In an experimental model, amitraz-treated horses had fatal obstruction in the proximal small colon about to marked colonic impaction.

MEDICATIONS

DRUG(S)
- The α₂-adrenergic antagonist yohimbine and atipamezole are used for treatment of amitraz intoxication in dogs and cats, but their use has not been documented for amitraz-intoxicated horses.
- Yohimbine is a α₂-adrenergic antagonist with high affinity for the α₁-adrenergic receptors and α₂C and a low affinity for the α₃D receptor. Yohimbine reverses amitraz-induced sedation in horses. A suggested dose for horses is 0.15 mg/kg IV slowly.
- Atipamezole is a potent and selective α₂-adrenergic antagonist approved to reverse sedative and analgesic effects of medetomidine in dogs. It is considered a new generation of α₂-adrenergic antagonists due to its high selectivity for α₂B and α₂C receptors, like α1A, α1B, and α1C receptors, and has a 100-times higher affinity for the α₃D receptor than yohimbine. Atipamezole has a higher affinity for α₂-adrenergic receptors and is more efficacious in reversing amitraz toxicity in cats than yohimbine. A suggested dose for horses is 0.1 mg/kg IV.
- Adverse drug interactions are possible with heterospecific antiparasitics, xylazine, benzodiazepines, and macrocyclic lactones.

CONTRAINDICATIONS/POSSIBLE INTERACTIONS
- While used in humans to treat amitraz-intoxicated bradyarhythmia, atropine is contraindicated in horses due to the known sensitivity of horses to the anticholinergic effects on gastrointestinal motility.
- Adverse drug interactions are possible with heterospecific antiparasitics, xylazine, benzodiazepines, and macrocyclic lactones.

FOLLOW-UP

EXPECTED COURSE AND PROGNOSIS
The impaction colic effects of amitraz toxicity may persist for days, but affected horses usually return to normal with treatment.

SUGGESTED READING

Author Patricia M. Dowling
Consulting Editor Robert H. Poppenga
Ammonia, Hyperammonemia

BASICS

DEFINITION
- Free ammonia (NH₃) is a nonprotein nitrogen compound that can permeate cells and result in hyperammonemia. At physiologic pH, almost all blood ammonia is the ammonium ion (NH₄⁺), which is less permeable for cells. In order to eliminate waste nitrogen as ammonia, the mammalian body converts it to an excretable form, urea.
- Ammonia is also derived from catabolism of glutamine and protein, and skeletal muscle exercise. Ammonia is delivered to the liver via the portal vein or hepatic artery, where functional hepatocytes remove ammonia to form urea by means of the Krebs-Henseleit cycle.
- If functional liver mass is inadequate, ammonia is not converted to urea, and plasma ammonia concentrations increase. Serum urea concentrations also rise when glomerular filtration is inadequate. Acid-base status affects the absorption of ammonia.
- As blood pH increases, free ammonia (NH₃) increases and can permeate cells via nonionic diffusion to produce toxicity. Ammonia is one of the compounds responsible for clinical signs of hepatic encephalopathy.
- Other described neurotoxins in hepatic encephalopathy are alterations in monoamine neurotransmitters due to altered aromatic amino acids, alterations in γ-aminobutyric acid (GABA) and/or glutamate, and increased endogenous benzodiazepine-like substances.
- Nervosa—Ammonia is neurotoxic and the brain is affected by high plasma concentrations.

GENETICS
N/A

INCIDENCE/PREVALENCE
N/A

SYSTEMS AFFECTED
- Neurological—Ammonia neurotoxicity.
- Gastrointestinal—Increased blood ammonia due to dysregulation of the gut.

PATHOPHYSIOLOGY

- Ammonia is derived primarily from dietary nitrogen with the gastrointestinal tract action of bacterial proteases, urases, and amine oxidases resulting in the major source of blood ammonia.
- Ammonia is also derived from catabolism of glutamine and protein, and skeletal muscle exercise. Ammonia is delivered to the liver via the portal vein or hepatic artery, where functional hepatocytes remove ammonia to form urea by means of the Krebs-Henseleit cycle.
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- Nervosa—Ammonia is neurotoxic and the brain is affected by high plasma concentrations.
- Gastrointestinal—Increased blood ammonia due to dysregulation of the gut.

CAUSES
- Liver disease—Hepatic encephalopathy is a prominent clinical feature of hepatic failure in the horse, and is associated with acute hepatitis and hepatic cirrhosis. Abnormalities of the urea cycle, abnormal portal blood flow, or any disorder that results in markedly impaired liver function can cause hyperammonemia. Decreased functional hepatic mass can result from pyrrolizidine alkaloid toxicity, acute hepatitis, chronic active hepatitis, hepatotoxic drugs or chemicals, Tyzzer’s disease in foals, and hyperbilirubinemia in ponies with associated hepatic dysfunction.
- Portal-systemic shunts—acquired or congenital.
- Toxicities—urea toxicity/poisoning, ammonium salt fertilizer toxicity.

RISK FACTORS
- Horses in known areas with hepatotoxic plants would be prone to develop hepatopathies.
- Administration of equine-derived biologics may induce hepatopathies.
- Feedstuffs contaminated with high levels of urea, nitrogen, or ammonium salts.

DIFFERENTIAL DIAGNOSIS
- Hepatic encephalopathy must be differentiated from primary neurologic diseases such as inflammatory, degenerative, infectious, or neoplastic CNS diseases. Rabies should be a differential diagnosis for abnormal behavior in the horse. Behavior-based alterations or problems should be ruled out.
- Differentiation consists of evaluating the history, signalment, and results of serum biochemistry, hematology, urinalysis, and hepatic biopsy.
- Possible intestinal bacterial overgrowth resulting in transient hyperammonemia (proposed).

CBC/BIOCHEMISTRY/URINALYSIS

Findings vary with the nature of the liver disease.
- CBC—microcytosis may occur in animals with portosystemic shunts, but may be difficult to determine in the horse; RBC histograms may be useful.
- Biochemistry—liver enzymes may be normal in animals with portosystemic shunts, but bile acid concentrations as well as ammonia concentrations will be elevated.
- Usually, other biochemical abnormalities are present, indicating hepatic dysfunction if the liver disease is severe enough to produce hepatic encephalopathy. Finding elevated liver enzymes (SDH, GPDH, ALT, GGTP, or AST) and hyperbilirubinemia, hyperglycemia (fast and slow), or elevated liver enzymes and low BUN support a diagnosis of liver disease.

IMAGING
- Ultrasound evaluation of the liver and portal vessels is advised.

OTHER DIAGNOSTIC PROCEDURES
- Hepatic biopsy is often necessary.

OTHER LABORATORY TESTS
- Measurement of serum bile acid concentrations has largely replaced ammonia assays due to convenience of sampling.
- Coagulation factor production may be decreased in liver failure resulting in prolonged PT and PTT.

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Ammonia, Hyperammonemia

PATHOLOGICAL FINDINGS
- Decreased functional hepatic mass; decreased liver size; microhepatica
- Portosystemic shunt
- Degenerative changes of the neurons and supporting cells have been observed in chronically affected animals.

TREATMENT

AIMS OF TREATMENT
Prevent signs and adverse effects of hepatic encephalopathy.

APPROPRIATE HEALTH CARE
Fluid administration is needed to correct dehydration and maintain tissue perfusion. It is important to maintain normal plasma potassium concentrations because low plasma potassium may increase the intracellular movement of ammonia.

NURSING CARE
Given above

ACTIVITY
Restrict activity.

DIET
Feed a very low protein diet, or fast the patient initially, and then institute a protein-restricted diet when the patient is stable.

CLIENT EDUCATION
Discussion of the prognosis with a hepatopathy and related causes

SURGICAL CONSIDERATIONS
Correction of hepatic shunts

MEDICATIONS

DRUG(S) OF CHOICE
- Lactulose is an acidifying agent used to decrease ammonia absorption from the intestine and lower plasma ammonia concentration in equine hyperammonemia. Lactulose acts as a cathartic laxative and maintains ammonia in its nonabsorbable ammonium ion form.

ALTERNATIVE DRUGS
N/A

FOLLOW-UP

PATIENT MONITORING
Repeated assessment of plasma ammonia can be helpful. Monitoring of serum potassium and glucose is advised in critical patients.

PREVENTION/AVOIDANCE
N/A

POSSIBLE COMPLICATIONS
Inaccuracy is the biggest problem because of the labile nature of ammonia in blood samples. Delay in processing results in false readings of high ammonia concentration.

EXPECTED COURSE AND PROGNOSIS
Guarded prognosis for most causes of hyperammonemia.

ASSOCIATED CONDITIONS
N/A

AGE-RELATED FACTORS
Congenital hepatic shunts are found in young animals versus acquired shunts that may occur at various ages.

ZOOONOTIC POTENTIAL
N/A

PREVENTION/AVOIDANCE
N/A

POSSIBLE INTERACTIONS
Because of impaired hepatic metabolism, any drugs that inhibit metabolism by the liver or are metabolized by the liver should be used with caution or the dosage should be adjusted.

ALTERNATIVE DRUGS
N/A

SUGGESTED READING

Author
Claire B. Andreasen
Consulting Editor
Kenneth W. Hinchcliff
Amylase, Lipase, and Trypsin

**Basics**

**Definition**
- Serum amylase or lipase concentrations above laboratory reference interval in horses are suggestive of pancreatic disease.
- Pancreatic disease is rare in horses.
- Reference range for serum activity of amylase and lipase are <35 IU/L and <87 IU/L, respectively.
- Amylase and lipase are rarely measured in routine equine serum biochemical profiles.
- Trypsin is released from damaged pancreatic cells.

**Pathophysiology**
- Amylase in the blood comes from a number of sources, including the intestinal mucosa, liver, and pancreas.
- Amylase is cleared from the blood by the kidneys, so renal dysfunction could lead to higher concentrations remaining in the blood.
- Damage to pancreatic cells can cause leakage of amylase into the blood or peritoneal fluid, but this is not common in the horse.
- Lipase is derived from the pancreas, gastrointestinal mucosa, and other tissues. Clinical serum assays detect all forms of lipase.
- Although uncommon in the horse, damage to pancreatic cells can cause release of lipase into the blood or peritoneal cavity.
- Pancreatitis can result in abnormally high serum activity of amylase and lipase. This disease is often associated with pancreatitis.

**Causes**
- Proximal enteritis
- High intestinal obstructions
- Intestinal mucosal damage
- Hyperlipemia
- Cortical administration
- Hepatic-induced lipoprotein lipase activity
- Obstruction to common bile and pancreatic duct
- Renal disease with renal failure
- Pancreatitis
- Panniculitis

**Risk Factors**
- Unknown other than risk factors for colic

**Increased activity of trypsin in blood is a result of leakage from damaged pancreatic cells, usually in horses with colic.**

**Systems Affected**
- Pancreas, peritoneum, peripheral adipose tissue

**Signalement**
- N/A

**Signs**
- Varies with underlying cause:
  - Pancreatitis—colic, gastric reflux, tachycardia, and signs of hypovolemic shock
  - Hyperlipemia—depression, anorexia, and lipemia serum
- Other intestinal diseases—colic, gastric reflux, and tachycardia
- Panniculitis (inflammation of adipose tissue) sometimes evident as colic

**Causes**
- Proximal enteritis
- High intestinal obstructions
- Intestinal mucosal damage
- Hyperlipemia
- Cortical administration
- Hepatic-induced lipoprotein lipase activity
- Obstruction to common bile and pancreatic duct
- Renal disease with renal failure
- Pancreatitis
- Panniculitis

**Laboratory Findings**

**Drugs That May Alter Lab Results**
- N/A

**Disorders That May Alter Lab Results**
- Hemolysis inhibits lipase activity.
- Lipemia falsely decreases serum lipase activity measured by kinetic assays.

**Valid If Run in Human Lab?**
- Not unless horse reference intervals are available.

**CBC/Biochemistry/Urinalysis**
- Peritoneal fluid amylase and lipase activities are usually less than those in blood except in pancreatitis.
- GGT is concentrated in the pancreas as well as the liver, so increased serum activity could mean pancreatitis as well as hepatitis or cholestasis, or elevations of GGT could be secondary to the proximity of the bile duct to an inflamed pancreatic duct.
OTHER LABORATORY TESTS

- Serum triglycerides above 500 mg/dL would mean hyperlipemia and expected increases in serum lipase or lipoprotein lipase activity.
- Nonesterified fatty acid (NEFA) concentrations > 0.5 mEq/L could mean hyperlipemia due to fat mobilization and expected increases in serum lipase activity.
- Urine GGT:creatinine ratio above 25–50 would indicate renal tubular damage and possible impairment of renal excretion of amylase or lipase.
- Abdominocentesis has been used for cytology to define inflammation and for chemical comparisons of peritoneal amylase and lipase concentrations to serum concentrations. Finding peritoneal fluid concentrations above serum concentrations can be indicative of pancreatitis, but this also can be a nonspecific finding in peritonitis/serositis.

DIAGNOSTIC PROCEDURES

Exploratory celiotomy may be indicated in cases of colic with undiagnosed causes of continued pain or indications of small intestinal obstruction. Abdominal fluid analysis should precede this invasive procedure.

TREATMENT

Treatment varies with the underlying cause. There is no specific treatment for pancreatitis in horses.

MEDICATIONS

As appropriate for underlying disease.

FOLLOW-UP

- Repeat blood and peritoneal fluid activity of amylase, lipase, and trypsin.
- Observe every hour for signs of colic.

POSSIBLE COMPLICATIONS

- Small intestinal obstruction, pancreatitis, and hyperlipemia can cause death.
- Distention of the stomach may cause rupture and death due to peritonitis.
- Leakage of amylase and lipase into the peritoneal cavity can induce nonseptic peritonitis.

SUGGESTED READING


The author and editor wish to acknowledge the contribution to this chapter of Erwin G. Pearson, author in the previous edition.

Author Kenneth W. Hinchcliff
Consulting Editor Kenneth W. Hinchcliff
Anaerobic Bacterial Infections

**Basics**

**Definition**
Anaerobic bacterial infections are caused by organisms that live and grow in the absence of molecular oxygen. Anaerobic bacteria are classified as either facultative or obligate, the former growing with or without oxygen. The anaerobic infections discussed here are caused by obligate anaerobic organisms.

**Pathophysiology**
Dermal and mucosal surfaces serve as protective barriers to infection. Normal flora and commensal bacteria contribute to this protective barrier. A breach in this protective barrier allows normal flora or commensal bacteria to gain access and potentially allows pathogenic infection to become established. There is a delicate balance between normal flora and commensal bacteria. When this balance is upset, commensal bacteria may become pathogenic or allow normally sterile sites to become contaminated. In other cases, contamination of a wound or an injection site by environmental organisms may lead to infection. Infectious challenge is dependent on inoculum size, virulence of the organism, and microbial resistance. Anaerobic organisms establish infection through virulence factors, and release of enzymes and toxins; these result in tissue destruction and provide protection from the host’s defenses. Anaerobic infections develop primarily in body sites where there is low oxygen tension, a low redox potential, or both.

**Systems Affected**
Upper Respiratory Tract
- Apical abscesses
- Sinusitis
- Pharyngeal abscesses

Lower Respiratory Tract
- Pneumonia
- Pulmonary abscesses
- Platinum pneumonia
- Pleuritis

Gastrointestinal System
- Peritonitis
- Abdominal abscesses
- Enteritis
- Colitis

Musculoskeletal System
- Soft-tissue abscesses
- Foot abscesses
- Thrush
- Carcinosis
- Osteomyelitis
- Sequestrums
- Septic arthritis
- Tenosynovitis
- Clostridial myonecrosis

Neuromuscular System
- Botulism
- Tetanus

Vascular System
- Omphalophlebitis/Omphalitis
- Thrombophlebitis

Hematopoietic System
- Septicemia

Reproductive System
- Metritis

Incidence/Prevalence
Dependent on the organism, body system infected, and how easily the infection is noted and treatment is instituted.

**Geographic Distribution**
Worldwide distribution

**Signalment**
Any age, breed, and sex can be affected.

**Signs**
Signs are variable depending on what system is involved and which organism is involved.

Upper Respiratory Tract
- Nasal discharge
- Facial swelling and crepitation
- Malodorous breath, sputum, pleural fluid
- Fever
- Inappetance
- Abnormal lung sounds
- Lethargy

Lower Respiratory Tract
- Cough
- Nasal discharge
- Malodorous breath, sputum, pleural fluid
- Fever
- Inappetance
- Abnormal lung sounds
- Lethargy

**Diagnosis**
Upper Respiratory Tract
- Nasal discharge
- Fever
- Lethargy
- Endoanemia

Upper Respiratory Tract
- Bacteroides spp.
- Peptostreptococcus spp.
- Peptostreptococcus magnus,
- Peptostreptococcus spp.
- Escherichia coli—Clostridium spp. and Bacteroides spp.

Musculoskeletal System
- Soft-tissue/foot abscesses and canker—Bacteroides spp. and Peptostreptococcus spp.
- Septic arthritis—Clostridium and Bacteroides spp.
- Myositis—Clostridium spp.

Gastrointestinal System
- Peritonitis/abdominal abscesses—Bacteroides spp., Peptostreptococcus spp., Peptostreptococcus magnus, E. coli—Clostridium spp. and Bacteroides spp.

Musculoskeletal System
- Soft-tissue/foot abscesses and canker—Bacteroides spp. and Peptostreptococcus spp.
- Septic arthritis—Clostridium and Bacteroides spp.
- Myositis—Clostridium spp.

Neuromuscular System
- Tetanus—Clostridium tetani

Vascular System
- Botulism—Clostridium botulinum
- Tetanus—Clostridium tetani

Hematopoietic System
- Myeloid leukemia
- Chronic myeloid leukemia

Reproductive System
- Metritis
- Ovaritis
- Peritonitis
- Abdominal abscesses
- Staphylococcal abscesses
- Inappetance

RISK FACTORS
Concurrent diseases, corticosteroid therapy, antibiotic therapy, immunosuppression, leukopenia, tissue anoxia, prior or concurrent aerobic infections, or the presence of a foreign body may also predispose the horse to anaerobic infections.

**Diagnosis**
Upper Respiratory Tract
- Nasal discharge
- Fever
- Lethargy
- Endoanemia

Upper Respiratory Tract
- Bacteroides fragilis, Peptostreptococcus magnus, and/or Clostridium spp.

Musculoskeletal System
- Soft-tissue/foot abscesses and canker—Bacteroides spp. and Peptostreptococcus spp.

Gastrointestinal System
- Peritonitis/abdominal abscesses—Bacteroides spp., Peptostreptococcus spp., and/or Clostridium spp.

Neurological System
- Botulism—Clostridium botulinum
- Tetanus—Clostridium tetani

Vascular System
- Botulism—Clostridium botulinum
- Tetanus—Clostridium tetani

Hematopoietic System
- Myeloid leukemia
- Chronic myeloid leukemia

Reproductive System
- Metritis
- Ovaritis
- Peritonitis
- Abdominal abscesses
- Staphylococcal abscesses
- Inappetance

RISK FACTORS
Concurrent diseases, corticosteroid therapy, antibiotic therapy, immunosuppression, leukopenia, tissue anoxia, prior or concurrent aerobic infections, or the presence of a foreign body may also predispose the horse to anaerobic infections.
ANAEROBIC BACTERIAL INFECTIONS

Lower Respiratory Tract
- Anaerobic infection (Streptococcus spp., Staphylococcus spp., Escherichia coli, Klebsiella, Pasteurella, Bifidobacteria spp.)
- Fungal infection (Candida auris, Cryptococcus, Histoplasma, Aspergillus, Candida spp.)
- Mycoplasma infection
- Thoracic trauma
- Esophageal rupture
- Bacteroides spp., E. coli, Actinobacillus

Aerobic infection (spp.)
- Corynebacterium
- Hyperfibrinogenemia
- Sonograms of the affected anatomic region
- Radiographs of the affected anatomical region
- Fecal cultures
- Blood cultures
- Salmonella infection
- Staphylococcus aureus, Proteus spp., Enterococcus spp., Clostridium perfringens
- Pseudomonas aeruginosa, Klebsiella pneumoniae

Bacterial pneumonia
- E. coli, Klebsiella
- Hemorrhagic consolidation
- Elevated total protein
- Atelectasis
- Lymphatic involvement

Pathologic Findings
- Lesions characterized by necrotic, edematous, emphysematous, and hyperemic tissues. Neutrophils, monocytes, and macrophages may accumulate in the tissue architecture, with bacteria interspersed.

Treatment
- Elimination of infection with effective antimicrobial therapy and exposure to oxygen, drainage of purulent exudates, and debridement of necrotic tissue if possible

Aims of Treatment
- Appropriate health care
- Initial hospitalization for intensive therapy; antimicrobials, and debridement/drainage
- Hyperbaric oxygen therapy can be utilized in areas with extensive tissue necrosis. Once stabilized, the patient may return home for continued care.

NURSING CARE
- Dependent on severity/duration of infection, body system affected, and causative organism.
- May include staged debridement, frequent hot compress therapy, and/or bandaging. Intensive care of indwelling tubes for constant drainage of body cavities may be required. Supportive care includes intravenous fluids and/or total/partial parenteral nutrition.

Activity
- Most likely decreased or restricted and will depend on the body system affected.

DIET
- The diet will most likely remain unchanged.

Client Education
- Some cases may be life-threatening depending on the extent of the illness and complications.
- In cases with severe muscle necrosis requiring debridement or fasciotomies, a cosmetic appearance may not be likely.

Surgical Considerations
- Surgery may be necessary to perform fasciotomies, to debride necrotic tissue, or to skin graft large areas that sloughed tissue during active infection. Surgery may also be required for the placement of an indwelling catheter to allow for lavage and flushing.

Medications
- Penicillin
- First line of defense against anaerobic infections. Excellent activity against most anaerobic infections, except beta-lactamase producing Bacteria. Preferred drug for cholelithiasis. Dose: 22,000–44,000 IU/kg, QID IV (aqueous) or BID IM (procaine).
ANAERObic Bacterial Infections

Ampicillin
Comparable to penicillin in its spectrum, but it is expensive in some countries, limiting its use to foals. Dose: 25–100 mg/kg IV QID.

Cephalosporins
Fifth-generation cephalosporins are generally less efficacious for anaerobic infections compared to penicillin. Cefotaxim (second generation) kills Bacteroides fragilis but may be used less due to expense. Other cephalosporins are effective for anaerobic infections but activity is unpredictable.

Trimethoprim-Sulfonamides (TMS)
TMS is effective against some obligate anaerobes but activity is unpredictable. Dose: 15–30 mg/kg BID PO.

Metronidazole
Consistently effective against obligate anaerobes including Bacteroides fragilis, not effective against facultative anaerobes or aerobes. It is rapidly absorbed after oral administration (bioavailability 75%–85%) and distributes well into synovial fluid, peritoneal fluid, cerebrospinal fluid, and urine, but has poor endometrial concentrations. It is used orally in cases of diarrhea caused by Clostridium difficile. It can also be given per rectum to horses that are anorexic or are refluxing; the bioavailability is about 30%. Dose: 15–25 mg/kg PO, IV, or per rectum QID-TID.

Chloramphenicol
All obligate anaerobes are susceptible. It has good tissue penetration into CNS, peritoneal, pleural, and synovial fluids. Absorption decreases with replete oral administration, the result is lower concentrations with subsequent doses. Dose: 45–60 mg/kg PO TID-QID.

Rifampin
Usually not necessary in most anaerobic infections but it may be useful in polymicrobial infections in walled-off abscesses. Most strains of Bacteroides and Clostridium are sensitive to rifampin. Dose: 5 mg/kg PO BID.

Tetracyclines
Can be used for anaerobic infections but penicillin-resistant Bacteroides spp. are demonstrating tetracycline resistance. Dose: 5–7.5 mg/kg IV BID.

Aminoglycosides
Ineffective against anaerobes due to mechanism of action requiring oxygen activity.

CONTRAINDICATIONS
Any drug causing diarrhea or enteritis. Sturic drugs like chloramphenicol are not recommended for immunocompromised patients.

PRECAUTIONS
Sustained high dose systemic penicillin therapy may have complications including secondary immune-mediated anemia, thrombocytopenia, and porcine reactions. Chloramphenicol can cause the development of aplastic anemia rarely in humans. Oral administration of metronidazole may cause anemia but resolves when the drug is discontinued.

POSSIBLE INTERACTIONS
Chloramphenicol may affect the metabolism of other drugs. Concurrent administration of cimetidine with metronidazole may decrease the metabolism of metronidazole and increase the likelihood of dose-related side effects.

FOLLOW-UP

PATIENT MONITORING
Response to therapy can be noted by monitoring changes in clinical signs. Hematologic and sonographic evaluations also help to establish the patient’s response to therapy.

PREVENTION/AVOIDANCE
Intramuscular injections have been reported to cause severe neuroaxial myonecrosis; avoid giving IM injections if possible or monitor injection sites closely after administration. Provide proper and immediate treatment of wounds to help prevent anaerobic infections.

POSSIBLE COMPLICATIONS
The possibility of complications depends on the body system affected and the severity of the disease. Severe infections may result in severe tissue sloughing, laminitis, endotoxemia, or death.

EXPECTED COURSE AND PROGNOSIS
Depends on the body system affected and the severity of the disease.

MISCELLANEOUS

ASSOCIATED CONDITIONS
Depends on the body system affected and the severity of the disease.

PREGNANCY
Infection of the reproductive tract may result in breeding and conception problems.

ABBREVIATIONS
• EPM = equine protozoal myeloencephalitis
• HYPP = hyperkalemic periodic paralysis
• NSAID = nonsteroidal anti-inflammatory drug

SUGGESTED READING

Author Shannon B. Graham
Consulting Editors Ashley Boyle and Conine R. Sweeney
The sensitizing
Exposure to the allergen may occur by
Clinical signs within a few minutes to hours
Less severe reactions may only warrant
Previous exposure to an antigen
A wide range of antigens may induce
An inappropriate dose or route of drug
David Hodgson and
Signs suggestive of anaphylactic shock require
Identify and remove inciting antigen— if
Blood transfusion reactions
Corticosteroids potentiate the effect of
Large-volume fluid therapy is indicated in
Clinical signs usually occur
Widespread petechiation, edema, and
Therapeutic goals include reversal of the effects
Epinephrine is the most effective treatment of
Glucocorticoid administration has been
Epinephrine may cause profound excitement
Continuous blood pressure and cardiac
Eosinophilia and basophilia
Signs are
Alternatively, signs may be transient and
Reactions may occur during initial exposure to
Drugs (especially antibiotics) (e.g., penicillin).
Administration of drugs (especially antibiotics)
May result in acute anaphylactic reactions and death.
Therefore, caution must be used if there is
expected course and prognosis
Variable and will depend on the severity of the
reaction, speed of diagnosis, and administration of treatment.
MISCELLANEOUS
Suggested Reading
Author Jennifer Hodgson
Consulting Editors: David Hodgson and Jennifer Hodgson

**BASICS**
- Exposure to the allergen may occur by ingestion, inhalation, contact with skin, or systemic introduction (e.g., IV injection).
- Clinical signs are related to mast cell degranulation in tissues and organs, including lung, liver, and other sites.
- Signs are attributable to the inflammatory mediators, enzymes, and cytokines released from sensitized mast cells.
- Signs may be localized or systemic. Additional signs include asthmatic episodes, urticaria, pruritus, edema, and diarrhea.
- Systemic signs may include hypotension, shock, and death.
- Epinephrine is the most effective treatment of anaphylactic shock.

**DIFFERENTIAL DIAGNOSIS**
- A variety of conditions may mimic anaphylaxis, including anaphylactoid reactions and systemic vasculitis.
- Histamine release may occur in response to other medications, food, or environmental allergens.

**PATHOLOGICAL FINDINGS**
- Severe lung injury with eosinophils, epithelial necrosis, and vasculitis are common at necropsy.
- Eosinophilia and basophilia are often present.
- Hemorrhage and edema are common findings.

**TREATMENT**
- Therapeutic goals include reversal of the effects of mediators, prevention of their further release, and treatment of the underlying cause.
- Epinephrine is administered immediately, followed by glucocorticoids, antihistamines, and other supportive care.

**MEDICATIONS**
- Epinephrine is the most effective treatment of anaphylactic shock.
- Glucocorticoids, antihistamines, and other supportive care are used in severe cases.

**CONTRAINDICATIONS/POSSIBLE INTERACTIONS**
- Epinephrine may cause profound excitement and cardiac arrhythmias.
- Glucocorticoids may cause increased sensitivity to other medications.
- Administration of drugs may be contraindicated in certain circumstances.

**SEE ALSO**
- Epinephrine and vasopressors
- Blood transfusion reactions

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**OVERVIEW**

**DIAGNOSIS**

**CAUSES AND RISK FACTORS**

**SYMPTOMS**

**PATIENT MONITORING**

**ANAPHYLAXIS**

**INTRODUCTION**

**ANAPHYLACTIC SHOCK**

**PATHOPHYSIOLOGY**

**PATHOLOGY**

**SYMPTOMS**

**DIAGNOSIS**

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**MEDICATIONS**

**CONTRAINDICATIONS/POSSIBLE INTERACTIONS**

**SEE ALSO**

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**OVERVIEW**

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**DIAGNOSIS**

**PATHOLOGICAL FINDINGS**

**TREATMENT**

**MEDICATIONS**

**CONTRAINDICATIONS/POSSIBLE INTERACTIONS**

**SEE ALSO**

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Anemia

BASICS

DEFINITION
A decrease in the erythrocyte content or oxygen-carrying capacity of blood as a consequence of a decrease in PCV, RBC count, and, except in cases of intravascular hemolysis, a decrease in Hb concentration to less than the lower limit of the laboratory reference interval.

PATHOPHYSIOLOGY

• Anemia is not a disease but a hematologic clinical sign that develops when one or more of the following 3 basic pathophysiologic mechanisms is present:
  □ Decreased or ineffective RBC production
  □ Increased RBC destruction (intravascular or extravascular hemolysis)
  □ Decreased circulating RBC mass
• Characterization of anemia in horses
  □ Regenerative (due to hemorrhage or hemolysis) or nonregenerative (due to decreased/ineffective RBC production) is assessed most accurately by examination of bone marrow aspirates. Serial monitoring of PCV and plasma TP concentration also may be helpful. Evaluation of immature RBC and RBC indices in peripheral blood is useful in understanding horses as equine reticulocytes or nucleated RBCs are rarely released into circulation until mature, even during intense erythropoiesis.
  □ The circulating RBC mass is extremely labile due to the effects of breed, age, level of activity, and splenic contraction, which can increase the RBC by ≈50%.
  □ Nonregenerative anemia occurs when the rate of erythropoiesis is insufficient to replace aged RBCs removed by the mononuclear phagocyte system. Nonregenerative anemia usually develops slowly due to the long life span of equine RBCs (≈50 days).

SYSTEMS AFFECTED

• Mechanisms associated with nonregenerative anemia may include:
  □ Diseases that interfere with erythropoiesis (e.g., by shortening erythrocyte life span or decreasing responsiveness to erythropoietin)
  □ Deficiency or alterations in specific substances necessary for RBC production or survival
• Diseases that damage or displace normal bone marrow elements and affect RBC precursors are also of concern.

SIGMENT

There is no breed, sex, or age predilection for anemia, although some specific primary diseases that result in anemia are more likely in some types of horses.

SIGNS

General Comments
Anemia generally occurs secondary to another disease. Clinical signs relate to the compensatory mechanisms activated in response to anemia as well as the primary disease process, which often are more prominent.

Historical
• Very dependent on the primary disease process, although frequently are related to trauma with visible hemorhagia, and exposure to oxidant toxins, medications, parasites, or infectious agents.
• Most common presenting complaints are exercise intolerance, signs of depression, and inappetence.

Physical Examination
• May be subclinical in horses with chronic anemia, although exercise may induce exaggerated tachycardia, weakness and reduced performance.
  □ In acute or severe cases, tachycardia, tachypnea, and low-grade holosystolic heart murmur are present at rest.
  □ Pal pustule membranes
  □ Other signs depend on the primary disease process and may include:
    □ Fever, tachycardia, and pyrexia in cases of hemolysis
    □ Weight loss, pancytopenia, and polydipsia in chronic renal failure
    □ Weight loss, fever, and lethargy in cases caused by chronic infectious, inflammatory, neoplastic, or immune-mediated processes

CAUSES

Hemorrhage
• External hemorrhage due to trauma, surgery, or external parasites
• Ectoparasitism due to gummed pouch mycosis, pulmonary abscess, septicemia, EIPH, cribitis, bacterial endocarditis, sinusitis, osteomyelitis, or trauma
• Hemorrhage due to trauma, ruped pulmonal abscess, rupted vatrial valve, or neumony
• Hematoma due to pyothorax, septicemia, osteomyelitis, rupted or infected abscess, or trauma
• Hemology due to trauma, ovian hemorrhage, metastatic vential ruped resection, or neumony

Coagulopathy
• Deficiency or alterations in specific factors necessary for hemorrhage or hemolysis should be suspected.
• Chronic nonregenerative anemia secondary to infectious, inflammatory, or neoplastic conditions usually is indicated when there is fever, weight loss, and dramatic increase in heart and respiratory rates if the horse is subjected to exercise or stress.
• Laboratory error due to insufficient mixing of samples, delay in analysis of samples, or samples left in hot conditions (hemolysis) may result in falsely low PCV or RBC count and falsely high Hb concentration.
• Iatrogenic; hypotonic or hypertonic solutions administered IV
• Other toxicities—intavenous dimethyl sulfoxide, heavy metal toxicosis, bacterial toxins (Clostridium sp.), smoke inhalation
• Miscellaneous—end-stage hepatic disease, hemolytic uremic syndrome, hemangioma, and disseminated intravascular coagulation

Nonregenerative Anemia
• Anemia of chronic disease associated with infectious, inflammatory, neoplastic, or immune-mediated processes
• Iron deficiency due to chronic hemorhagia (especially GI) and nutritional deficiency (particularly Feals)
• Bone marrow failure—myelophthisis, myeloproliferative disease, bone marrow tumor (e.g., phenylbutazone), radiation, immune-mediated, and idiopathic hyperplasia anemia
• Miscellaneous—chronic renal disease, chronic hepatic disease, and recent hemorhagia or hemolysis

RISK FACTORS
• Depends on risk factors for the primary disease process
• Age (e.g., neoplasia, middle usteus artery rupture) and sex (e.g., idiopathic urethral hemorhagia in geldings)
• Any infectious or inflammatory disease
• Feels consuming incompatible colorum are at risk for NI
• Inadequate preventative anthelminthic use or long-term high-dose phenylbutazone administration
• Geographical location for exposure to infectious agents or toxic plants

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS
• Determination of the primary disease causing anemia should be the focus of investigations.
• Initial investigations should focus on identifying the basic mechanisms (see Causes) involved using historical, clinical, hematologic and biochemical findings.
• When onset of clinical signs is sudden, or if there is a history of trauma, severe external or internal hemorrhage or severe hemolysis should be suspected.
• Chronic nonregenerative anemia secondary to infectious, inflammatory, or neoplastic conditions usually is indicated when there is fever, weight loss, and dramatic increase in heart and respiratory rates if the horse is subjected to exercise or stress.
• Laboratory error due to insufficient mixing of samples, delay in analysis of samples, or samples left in hot conditions (hemolysis) may result in falsely low PCV or RBC count and falsely high Hb concentration.
• Miscellaneous—end-stage hepatic disease, hemolysis below the lower limit of reference intervals.
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CBC/BIOCHEMISTRY/URINALYSIS
• PCV, total RBC count, and (except in cases of intravascular hemolysis) Hb concentrations below the lower limit of reference intervals.
• Miscellaneous—end-stage hepatic disease, hemolysis below the lower limit of reference intervals.
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rarely observed in horses with regenerative anemia and RBC indices are less useful for diagnosis or classification of anemia.

- A moderate increase in MCV (hemolytic anemia) and RDW (hemolytic anemia) may occur 2–3 weeks after onset of regenerative anemia. Additionally, increased MCH values may indicate presence of free Hb and hemolysis and decreased MCH, MCHC, and MCV may indicate iron deficiency anemia.

- Hematocrit values may be observed near the cellular margins of RBCs stained with New Methylene Blue in horses with hemolytic anemia due to oxidative injury.

- Spherocytosis, indicative of immune-mediated hemolytic anemia, may be difficult to detect in equine blood smears due to the small size and lack of central pallor of normal RBCs. In addition, rouleaux formation of normal equine RBCs may complicate identification of autoagglutination in cases of immune-mediated hemolytic anemia.

- Neutrophil cells may be observed in blood smears of horses with myeloproliferative disorders.

- Severe neutropenia and thrombocytopenia may be observed in horses with myeloproliferative disorders.

- Horses with blood loss usually have a concurrent decrease in PCV and plasma TP whereas horses with hemolytic anemia usually have decreased PCV, normal plasma TP and marked increases in serum total direct bilirubin concentration with some hemolytic disorders.

- Horses with nonregenerative anemia due to increased erythropoiesis usually have decreased PCV, normal or increased TP (due to decreased globulin and fibrinogen concentrations), and an inflammatory leukogram.

- Bilirubinuria or hemoglobinuria may occur with renal insufficiency.

- Isothemia in horses with chronic renal failure.

**OTHER LABORATORY TESTS**

- Positive direct Coombs test is evidence for immune-mediated hemolytic anemia.

- Blood diluted with saline (1:4) aids in differentiating erythrophagocytosis from normal rouleaux formation in horses with immune-mediated hemolytic anemia.

- Serum iron concentration usually is increased, total iron-binding capacity usually is decreased and storage iron usually is increased in horses with anemia of chronic disease.

- Serum iron concentration, percentage saturation of transferrin, and storage iron usually are decreased, while total iron-binding capacity usually is increased in iron deficiency anemia.

- Coggins test or C-ELISA test for diagnosis of EIA.

- Serology for Babesia, Theileria or A. phagocytophilum.

- Identification of organisms in blood smears.

**IMAGING**

- As indicated to diagnose underlying disease process.

- Ultrasonography or radiography may assist in diagnosing thoracic or abdominal hemorrhage.

**OTHER DIAGNOSTIC PROCEDURES**

- Bone marrow aspiration or core biopsy may demonstrate increased erythropoiesis and a decreased myeloid-to-erythroid (M:E) ratio in horses with regenerative anemia or may reveal decreased erythropoiesis and an increased M:E ratio with nonregenerative anemia. Infiltration with abnormal cell types may be observed in myeloproliferative or myelodysplastic disorders.

- Abdominocentesis or thoracocentesis to detect internal hemorrhage.

- Fecal occult blood test to detect GI hemorrhage.

- Endoscopy to assist in detecting respiratory or GI hemorrhage.

**TREATMENT**

**AIMS OF TREATMENT**

The major aims of therapy in horses with anemia are to identify and eliminate the primary cause, provide nursing care, ensure adequate tissue perfusion, and administer blood transfusions if indicated.

**APPROPRIATE HEALTH CARE**

- Inpatient medical management may be necessary depending on severity and rapidity of onset of anemia and underlying disease condition.

- Cross-matched whole blood or packed RBC transfusion is recommended if PCV decreases to <0.08–0.12 L/L (<8–12%).

- Large-volume, isotonic (e.g., lactated Ringer’s solution) or small-volume hypertonic saline (7% NaCl) fluid therapy if patient has signs of hemorrhagic shock.

**NURSING CARE**

- Close monitoring of vital signs, serial determination of PCV and TP and adjustment of rate are essential in horses receiving fluid therapy.

- Monitor horses for renal failure induced by hemoglobinuria or heparin and for lactic acidosis.

**DIET**

- Ensure access to ionic protein sources is eliminated.

- Oral iron supplementation in horses with confirmed iron deficiency anemia. However, for horses with external blood loss, iron supplementation is rarely required because most diets are rich in this element.

**SURGICAL CONSIDERATIONS**

May be indicated in horses with significant uncontrolled internal hemorrhage, although these horses have a high anesthetic risk.

**MEDICATIONS**

**DRUG(S) OF CHOICE**

Specific therapy indicated for the primary underlying disease process.

**PRECAUTIONS**

- Severe reactions to blood transfusions may occur and necessitate careful monitoring and prompt therapy (see Blood Transfusion Reactions).

- Hypertonic saline should be used with caution in horses with uncontrolled bleeding as it may cause increased blood loss.

- Concomitant drug should be used with caution in horses with suspected chronic infectious condition.

- Parenteral administration of iron formulations is not recommended because iron deficiency is extremely rare and there is the possibility of serious adverse reactions.

**FOLLOW-UP**

**PATIENT MONITORING**

- Monitor PCV to assess regenerative response. PCV should increase by an average of 0.5%–1% per day within 3–5 days of an acute anemic or hemolytic episode.

**EXPECTED COURSE AND PROGNOSIS**

Highly dependent upon the cause, severity, and rapidity of onset.
**Anemia, Pure Red Cell Aplasia**

**BASICS**

**OVERVIEW**

Pure red cell aplasia is characterized by selective reduction or hypoplasia of erythroid precursors in the bone marrow resulting in development of a nonregenerative anemia. The white cell (granulocytic) and platelet (megakaryocytic) cell precursors are not affected (as they are in aplastic anemia/pancytopenia).

- In horses, pure red cell aplasia has been reported secondary to repeated doses of rhEPO.
- Primary pure red cell aplasia, described in a number of case reports in dogs and cats and considered to be an immune-mediated disorder responsive to treatment with corticosteroids and/or lymphocytotoxic drugs, has not been reported in horses.

**SIGNALMENT**

Most commonly this anemia is reported in performance horses such as racing Standardbreds and Thoroughbreds.

**SIGNS**

- Can occur in the absence of other systemic disease
- Signs depend on the severity and duration of anemia and may consist of poor performance, weight loss, signs of depression, inappetence, weakness, mucous membrane pallor, and tachycardia and polypnea (exaggerated when horses subjected to stress).
- Prolonged or severe nonregenerative anemia may cause tissue hypoxia resulting in cardiac, hepatic, and renal dysfunction and can be life-threatening.

**CAUSES AND RISK FACTORS**

- The strongest risk factor, and likely cause, of this disorder is repeated administration of rhEPO to race horses in order to increase total red cell mass and oxygen-carrying capacity with the aim of enhancing athletic performance.
- Although the mechanism for erythroid hypoplasia is unclear in this syndrome, the recombinant hormone may induce production of anti-rhEPO antibodies that bind endogenous equine erythropoietin, preventing the latter hormone from stimulating RBC differentiation and multiplication in bone marrow.
- Increased frequency of exposure may lead to an exaggerated immune response and more severe clinical signs.

**DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS**

- Anemia of chronic disease associated with infectious, inflammatory, or neoplastic disorders. In general, these disorders also result in leukocytosis and elevated fibrinogen concentrations.
- Folate deficiency after treatment of EPM with antifolate drugs, which, paradoxically, occurs in horses administered oral folic acid while receiving antifolate drugs. Diagnosis of EPM and exposure to these drugs easily distinguishes this from pure red cell aplasia.
- Aplastic anemia. Granulocytic and megakaryocytic stem cell lines in bone marrow also fail to undergo differentiation resulting in generalized marrow hypoplasia and peripheral pancytopenia.

**Other Causes of Inadequate Erythropoiesis**

- Primary myelophthisic disease may cause anemia in the presence of leukopenia or thrombocytopenia. Because the life span of platelets and WBCs is shorter than that of RBCs, clinical signs of thrombocytopenic hemorrhage, infection, and fever typically precede those of anemia.
- Erythropoietin deficiency from chronic renal failure. Signs specifically referable to the renal system will also be present (e.g., polyuria, polydipsia, renal azotemia, reduced urine concentrating ability).

**Diagnosis**

**CBC/BIOCHEMISTRY/URINALYSIS**

- Anemia, with PCV 0.16 L/L (16%) or below
- Normal WBC count, platelet numbers and plasma fibrinogen concentrations
- Normal urinalysis
Anemia, Pure Red Cell Aplasia

OTHER LABORATORY TESTS
- Reported cases have demonstrated increased serum iron and serum ferritin concentrations.
- Negative Coombs test for EIA and negative Coombs test for immune-mediated hemolytic anemia.
- Bone marrow aspiration demonstrates an increased M:E ratio and erythroid hypoplasia, confirming nonregenerative anemia.
- Serum from affected horses may inhibit rhEPO-induced proliferation of erythroid progenitors in vitro.
- Other diagnostic tests appropriate to rule out other disorders on the differential diagnostic list.

TREATMENT
- Avoid further rhEPO administration.
- Blood transfusion from a cross-matched donor is warranted if anemia is severe (<8–12%) and there are clinical signs of tissue hypoxia (e.g., tachypnea, tachycardia, weak pulse pressure, weakness).

MEDICATIONS
DRUG(S) OF CHOICE
Dexamethasone (0.05 mg/kg once daily) has been used to treat horses with pure red cell aplasia, although efficacy is unproved. The dose should be adjusted or discontinued depending on a favorable or negative response, respectively.

CONTRAINDICATIONS/POSSIBLE INTERACTIONS
Avoid iron supplementation, because the iron binding-capacity of the serum may be exceeded, leading to hepatic necrosis.

FOLLOW-UP
PATIENT MONITORING
- Monitor the degree of anemia with serial PCV measurements over several weeks to months.
- Some horses are nonresponsive and die despite multiple transfusions and steroid administration, whereas others recover completely.

SEE ALSO
- Anemia
- Pancytopenia

ABBREVIATIONS
- EIAV = equine infectious anemia virus
- EPM = equine protozoal myeloencephalitis
- M:E = myeloid:erythroid
- PCV = packed cell volume
- RBC = red blood cell
- rhEPO = recombinant human erythropoietin

Suggested Reading

Author
Nicholas Malikides
Consulting Editors Jennifer Hodgson and David Hodgson
Anemia, Heinz Body

DEFINITION
- Acute or chronic hemolytic anemia following exposure to agents that oxidize and denature RBC hemoglobin.
- Hemolysis is caused by various factors, including free radicals, oxygen, drugs, or toxins.

PATHOPHYSIOLOGY
- Exposure of RBCs to oxidant toxins, drugs, or chemicals results in oxidation of sulfhydryl groups and formation of disulfide linkages in the protein component of the hemoglobin molecule.
- The denatured or hemoglobin precipitates to form Heinz bodies.
- A hereditary, familial form of hemoglobin disease is characterized by the formation of Heinz bodies.

RISK FACTORS
- Exposure to toxins (e.g., wilted or dried leaves), drugs, or oxidant toxins.
- Ingestion of wilted or dried red maple leaves or bark.
- Contact with red maple leaves in autumn compared to spring.

SIGNS
- Anemia, jaundice, icterus, weakness, pale or icteric mucous membranes, and pallor of conjunctivae.
- Respiratory rates, a holosystolic heart murmur, tachypnea, tachycardia, or other respiratory signs.

DIFFERENTIAL DIAGNOSIS
- Other causes of anemia must be considered in the differential diagnosis.
- Hemolysis is usually evident from clinical signs.

OTHER LABORATORY TESTS
- Serum biochemistry and urinalysis may reveal hyperbilirubinemia, hyperbilirubinemia, or hemoglobinuria.
- Increased total and indirect bilirubin, increased BUN, and increased serum hepatic enzyme activity are indicative of hemolytic anemia.

IMAGING
- Splenic or hepatic ultrasonography may be used to detect splenic or hepatic enlargement.
- Magnetic resonance imaging (MRI) may be useful in detecting splenic hemoglobin deposits.

INCIDENCE/PREVALENCE
- No incidence or prevalence data currently available for oxidant-induced hemolytic anemia in horses.

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may be some loss of architecture due to increased fluid component.

OTHER DIAGNOSTIC PROCEDURES
• Arthrocentesis diagnostic synovial fluid should be undertaken to rule out other causes of hemolytic anemia.

PATHOLOGICAL FINDINGS
• Pulmonary edema
• Enlarged liver and spleen and severe, diffuse congestion of the kidneys
• If chronic, possible signs of congestive heart failure include pulmonary edema, pulmonary edema, cardiomegaly, or hepatic congestion.
• Histopathologic lesions might include renal tubular necrosis, glomerular degeneration and necrosis, and glomerulonephritis in the spleen and liver.

TREATMENT

AIMS OF TREATMENT
• Treatment of Heinz body hemolytic anemia involves identification and removal of the oxidant source and provision of supportive care.

APPROPRIATE HEALTH CARE
• Even if several days have elapsed since exposure to the oxidant, activated charcoal (8–24 mg/kg PO via nasogastric intubation) should be administered to reduce further absorption of toxins.
• In-hospital medical management may be necessary depending on severity and the capacity of onset of the anemia.
• Balanced IV fluid therapy with isotonic crystalloid solutions to prevent hemoglobin-induced nephropathy and promote diuresis.
• Cross-matched blood transfusion of PCV decreases to < 6-12%, or if there is permanent tachycardia, tachypnea, prolonged CRT, mucous membrane pallor and weak pulse pressure or a poor response to isotonic fluid therapy.
• Oxygen therapy may be useful but often is ineffective if hemoglobin oxygen-carrying capacity is too low.

NURSING CARE
• Close monitoring of cardiovascular status, vital signs, fluid rates (to avoid hemodilution) and blood hematology and clinical chemistry are indicated and likely aid recovery.
• Concomitant monitoring for renal failure induced by hemoglobinuria or hypoxia and for laminitis also is necessary.

ACTIVITY
• Minimize activity and stress.
• No forced exercise.

DIET
• Provide the horse with a balanced diet, including good-quality hay and grain.
• Fresh water should be available ad libitum.

CLIENT EDUCATION
• The hazards of exposure to wilted red maple leaves (including red maple hybrids) should be explained and suggestions given concerning housing and removal of branches blown down in storms or cut down in areas where horses may have access to them.

MEDICATIONS
DRUG(S)
• There is no specific medicinal treatment for Heinz body anemia and treatment is mainly supportive.
• Along with administration of isotonic IV fluids for pigment-induced nephropathy, furosemide or dopamine may be indicated in cases with oliguria or anuria.
• Dexamethasone (0.05–0.1 mg/kg IV q12–24h) may help to stabilize cellular membranes and decrease phagocytosis of damaged RBCs.

CONTRAINDICATIONS
• Use of Methylene Blue or other reductive therapy may be detrimental, because these agents may enhance Heinz body formation.

FOLLOW-UP

PATIENT MONITORING
• Serial determination of PCV should be performed to assess the bone marrow regeneration and response to treatment. The PCV should remain stable or slowly increase over time.
• Renal function should also be reassessed and signs reflective of laminitis monitored, particularly in horses also receiving corticosteroid therapy.

PREVENTION/AVOIDANCE
• Limiting access to excess phenothiazine, onions, or wilted red maple leaves.

POSSIBLE COMPLICATIONS
• Laminitis
• Nephropathy
• General dehydration
• Absorption, weak foals

EXPECTED COURSE AND PROGNOSIS
• Prognosis for recovery depends on the amount of oxidant ingested and whether or not methemoglobinemia also is present. If the ingesting cause can be removed and methemoglobinemia is minimal or absent, the prognosis for recovery is fair to good. Several weeks may be required for full recovery.
• The prognosis is guarded in horses with red maple leaf toxicity when methemoglobinemia is present.
Anemia, Immune-Mediated

**Basics**

**Definition**
- IMHA is an acute or a chronic destruction of RBCs associated with immunoglobulin and/or complement attachment to either RBC antigens or foreign antigens coating the surface of RBCs.
- Affected RBCs are most commonly removed by the RES (also called mononuclear phagocyte system) after immunoglobulin-mediated opsonization (intravascular hemolysis).
- Less commonly, they may undergo intravascular, complement-mediated lysis.

**Pathophysiology**
- IMHA most commonly occurs secondary to agents that:
  - Alter the RBC membrane, exposing antigens to which the host produces antibody (e.g., infectious agents, neoplasia).
  - Form immune complexes that adhore to the RBC and fix complement (e.g., infectious agents).
  - Directly bind to the RBC and act as haptons that bind antibody (e.g., drugs); or
  - Stimulate the immune system resulting in production of antibodies with cross-reactivity to RBCs (e.g., infectious agents, neoplasia).
- Occasionally, the immune system produces specific autoantibodies to normal erythrocyte antigens (e.g., primary or idiopathic autoimmune hemolytic anemia, NI, or transfusion reaction).
- Antibody- and/or complement-coated RBCs are removed from the circulation by extravascular hemolysis (if removed by the RES) and/or intravascular hemolysis (if complement-mediated).

**Systems Affected**
- The humoral/lymphatic immune systems are involved due to intravascular and/or extravascular hemolysis. In cases where extravascular hemolysis predominates, splenomegaly may occur. Pyrexia may result from release of hemoglobin and other end products of red cell breakdown.
- Cardiovascular and respiratory systems may be involved with increased heart and respiratory rates, hypotension heart murmur, and pallor of mucous membranes observed.
- Hepatobiliary system—Hemolytic anemia can result in hyperbilirubinemia and icterus whereas hypotension may result in centrilobular degeneration.
- The renal system may be involved in cases where significant intravascular hemolysis and hemoglobinuria cause pigment nephropathy.
- The gastrointestinal tract may be involved due to hemosiderin damage to intestines resulting in mortality disorders, colic or diarrhea.

**Incidence/Predvalence**
- No incidence or prevalence data are available for immune-mediated hemolytic anemia for specific horse populations. Weak evidence in human and veterinary patients suggests IMHA is a rare consequence of other disease states in adult horses. These forms of IMHA are reported to have a low case fatality rate.

**Signs**
- NI, which is a specific form of IMHA, is reported in foals in most countries, particularly horse study where there are large numbers of breeding mates.

**Signalment**
- Can occur in horses of any breed, age, or sex.

**General Comments**
- IMHA often reflects a primary, underlying disease process such as infection or neoplasia.

**Historical**
- History generally reflects an underlying disease process and may include chronic weight loss (e.g., neoplastic diseases) or signs of depression and inappetence (e.g., infectious disease).
- Exercise intolerance, weakness, and lethargy are common presenting signs.
- There may be a history of exposure to blood transfusion(s) or certain drugs.

**Physical Examination**
- Signs of anemia—Severely proportional to the degree of anemia.
- Exercise intolerance, weakness, pale or icteric mucous membranes, fever, tachypnea, tachycardia, hypotonic heart murmur, abdominal pain, and hemoglobinuria may be observed.
- Rectal examination may reveal an enlarged spleen.
- In foals with NI, there is usually an acute onset of intravascular hemolysis, with weakness and icterus during the first few days of life.
- Severe dehydration and death may occur in severe cases.

**Causes**

**Primary Immune-Mediated**
- NI: Autoimmune hemolytic anemia
- Incomparable blood transfusion

**Secondary Immune-Mediated**
- Infections—e.g., EIA, acute viral infections, infection with Clastobrosis perffigens, injection site abscesses
- Neoplastic—e.g., lymphosarcoma and hemangiosarcoma
- Drug-associated—e.g., penicillins, cephalosporins, and tetracyclines
- Microangiopathic—disseminated intravascular coagulation
- Systemic lupus erythematosus

**Risk Factors**
- Fresh born to multiruminate dams that have previously had a blood transfusion(s), or the mare is known to be RBC antigen Aa or Qa negative, are at increased risk of developing NI.
- Exposure to incompatible blood transfusion and certain drugs

**Diagnosis**

**Differential Diagnosis**
- Other diseases causing anemia must be differentiated from IMHA

**Clinical Features**
- Horses with hemoglobin (acute or chronic) often have a history of external blood loss or exposure to oxidative toxins or endotoxin.
- Horses with Heinz-body anemia (e.g., wilted red maple leaf toxicity, onion toxicosis, phenothiazine toxicosis) may have a history of exposure to oxidative toxins and presence of Heinz bodies or metachromasia on routine blood analysis.
- Horses with purpura hemorrhagica often have a history of exposure to Streptococcus equi or a history of certain infectious or respiratory tract pathogens. In these cases edema of the legs, abdomen, and face and petechial hemorrhages of mucous membranes are common.

**CBC/Biochemistry/UrineALYSIS**
- PCV is often <0.20 L/L (<20%).
- May have neutrophilic leukocytosis.
- RBC agglutination may be observed, but must be distinguished from rouleaux formation or RBC clumping due to other inflammatory disorders. Splenomegaly may be observed in blood smears, but are more difficult to identify in horses due to the lack of central palmar in normal equine RBCs.
- Increased MCH suggests intravascular hemolysis, which may also result in discoided plasma.
- Increased serum total bilirubin concentration (index greater than direct)
- Bilirubinuria and hemoglobinuria may be observed in the rarer cases where intravascular hemolysis occurs.

**Other Laboratory Tests**
- Positive direct antiglobulin (Coomb’s) test, which detects presence of antibody on the surface of RBCs. False-negative results are possible, particularly if there has been prior cross-contamination therapy. False-positive results also can occur, emphasizing the need to use multiple methods to confirm the diagnosis of IMHA.
- Confirmation of true agglutination is performed by diluting EDTA-anticoagulated blood 1:4 with physiologic saline solution. RBCs should remain agglutinated with saline dilution.
- CBC normocytic fragility may be increased in IMHA, although the test can be positive with RBCs damaged by oxidative insults.
- Bone marrow aspiration reveals a diffuse, regenerative erythropoiesis (NEE rate >0.5).
- Infectious causes of IMHA may have positive serology and/or evidence of hematologic parasites on direct or special stained blood smears:
  - Horses with EIA will be seropositive for virus on Coggins or C-ELISA tests.
  - Horses with equine protozoal myeloencephalitis may have organisms observed in Giemsa or New Methylene Blue stained blood smears, be seropositive or seroconvert on their convalescent titer.
  - Horses with equine granulocytic ehrlichiosis may have granular inclusion bodies observed in cytoplasm of neutrophils in Giemsa stained blood smears; be seropositive or seroconvert with acute and convalescent samples.

**Imaging**
- Splenic/hepatic ultrasound determines splenic/hepatic enlargement and highlights
hypochoic or hypochromic areas indicating loss of architecture. • Radiography of the thorax is usually within normal limits unless a primary neoplasia is the underlying cause.

OTHER DIAGNOSTIC PROCEDURES A thorough diagnostic workup should be performed to rule out neoplasia and infectious causes of secondary IMHA.

PATHOLOGICAL FINDINGS • Necropsy findings may include an enlarged liver and spleen and pale or icteric stances. • If chronic, there may be signs of congestive heart failure (with pulmonary edema, reticular edema, cardiomegaly), renal tubular nephrosis with hemoglobin casts, and centrilobular hepatic degeneration and necrosis.

TREATMENT

AIMS OF TREATMENT • Treatment of IMHA involves identification and resolution (if possible) of any underlying infection or disease, reduction of the immune response, and provision of supportive care. • Administration of any drugs should be discontinued as IMHA could be caused by an adverse drug reaction. If antimicrobial therapy is required, a molecularly dissimilar antibiotic should be used.

APPROPRIATE HEALTH CARE • Most cases of IMHA are treated in hospitals, especially if severe. • Balanced polyionic IV fluid therapy may be indicated to expand vascular volume and induce diuresis.

SURGICAL CONSIDERATIONS • Consider splenectomy if the primary cause cannot be identified.

MEDICATIONS

DRUG(S) • In adults, corticosteroids (dexamethasone, 0.05–0.2 mg/kg IV or IM q12–24h) are indicated until PCV ceases to decline. The dose may then be decreased by 0.01 mg/kg/day until the total dose is 20 mg/day (for a 500-kg horse), after which alternate-day oral prednisolone is recommended. Alternatively, oral prednisolone (2.5–5 mg/kg) may be used in place of dexamethasone at any time during therapy. • From 4 to 7 days often are needed for corticosteroids to have a therapeutic effect (with stabilization of PCV) and up to 10 weeks of treatment may be necessary.

CONTRAINDICATIONS • Corticosteroids may exacerbate underlying infections so should be used only in horses that are negative for IFA (Coggins) and free of other infectious diseases.

PRECAUTIONS • Cross-match blood before blood transfusion.

ALTERNATIVE DRUGS The immunosuppressive agent azathioprine (5 mg/kg PO once daily) and cyclophosphamide (<100 mg/m² body surface area/24h) have been used successfully in one horse that was nonresponsive to corticosteroids.

PREGNANCY

Use corticosteroids cautiously in pregnant mares.

EXPECTED COURSE AND PROGNOSIS • If the primary cause can be identified and successfully treated, the prognosis for IMHA is good. • Red cell numbers replenish as the immune-mediated response resolves. This may take several weeks in some horses. • Horses requiring constant corticosteroid treatment (if diagnosed with idiopathic autoimmune hemolytic anemia) may have an incurable underlying disease such as neoplasia (e.g., lymphosarcoma). The prognosis for survival in these horses is poor.

MISCELLANEOUS

ASSOCIATED CONDITIONS • Pigment nephropathy with intravascular hemolysis • Laminitis

SYNONYMS • Autoimmune hemolytic anemia • Immune-mediated hemolytic disease

SEE ALSO Anemia, Neonatal Isoerythrolysis Anemia, Neonatal Hemolytic Anemia Anemia, Hereditary Anemia Anemia, Immune-Mediated Anemia Anemia, Hemolytic Anemia

ABBREVIATIONS • C-ELISA = competitive enzyme-linked immunosorbent assay • IM = intramuscular • IV = intravenous • EIA = equine infectious anemia • IMHA = immune-mediated hemolytic anemia • MCH = mean corpuscular hemoglobin • MCV = mean corpuscular volume • RI = reticuloendothelial index • PCV = packed cell volume • RBC = red blood cell • RES = reticuloendothelial system

Suggested Reading


Author Nicholas Malikides Consulting Editors Jennifer Hodgson and David Hodgson
Anemia, Iron Deficiency

**BASICS**

**OVERVIEW**
- Iron is stored in horses as hemoglobin (65% of total iron stores), ferritin, and hemosiderin.
- Iron deficiency may arise from either chronic external loss of blood (most common in adult horses) or dietary deprivation (in young rapidly growing foals). Unlike adult horses, have inadequate access to soil, pasture, or feed, inadequate iron intake is unlikely.
- Iron deficiency results in delayed hemoglobin synthesis, resulting in arrested and ineffective RBC maturation in bone marrow and anemia. The small hemoglobin deficient RBCs (i.e., hypochromic microcytes) produced have reduced deformability and life span.
- Nonregenerative anemia and reduced blood hemoglobin concentration may lead to compromised oxygen delivery to tissues.
- Nonheme, iron-containing enzymes may also be depleted and result in impairment of cell-mediated immunity and neutrophil killing of ingested bacteria.

**SIGNALMENT**
- No breed or sex predilections
- Rapid growth of foals is associated with high tissue demands for iron. Mare’s milk has low iron concentrations (≈0.88 g/g by 2 weeks and ≈0.6 g/g by 8 weeks postpartum) and therefore deficiency may occur in foals with limited access to pasture, iron-rich soils, or consuming forage or grain.

**SIGNS**
- Initially, clinical signs may be absent or mild due to adequate physiologic compensation for the gradual reduction in oxygenation.
- Lethargy and exercise intolerance may be the first overt clinical signs noted.
- When PCV is <12%, tissue hypoxia can cause tachycardia, tachypnea, pale mucous membranes, systolic heart murmur, and signs of depression.

**CAUSES AND RISK FACTORS**
- Risk factors for chronic hemorrhage may include inadequate preventative anthelmintic use, phenylbutazone administration, and exposure to toxins.

**Chronic, Low Grade Hemorrhage**
- Severe internal parasitism (Strongylus vulgaris, small strongyles) or external parasitism (e.g., heavy infestation of sucking lice—*Hematopinus asini*)
- Bleeding GIT, respiratory, and urinary tract lesions (e.g., gastroduodenal ulcers, NSAID toxicosis, neoplasia [especially gastric squamous cell carcinoma], hemorrhagic or erosive cystitis, guttural pouch mycosis, and ethmoid hematoma)
- Coagulopathies leading to chronic blood loss (e.g., heritable coagulopathies, warfarin toxicosis, moldy sweet clover [dicumarol toxicosis])

**Diet**
- Inadequate dietary intake (foals)

**DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS**
- Causes of low-grade, hemolytic anemia must be ruled out including immune-mediated hemolysis, oxidant-induced hemolysis, and parasite-induced hemolysis. Distinguishing features may include hemoglobinuria, hemosoglobinuria, and a normal serum protein concentration. Serum iron concentrations may be increased.
- Causes of decreased erythrocyte production must be ruled out including anemia of chronic disease and aplastic anemia. Increased serum ferritin concentrations are typical in anemia of chronic disease and bone marrow morphology is diagnostic for aplastic anemia.

**CBC/BIOCHEMISTRY/URINALYSIS**
- Normochromic, normocytic anemia is initially observed, but usually develops into a microcytic, hypochromic, nonregenerative anemia in later stages. Microcytosis often precedes hypochromasia.
- Thrombocytosis may be observed.
- Decreased plasma protein and albumin concentrations.

**OTHER LABORATORY TESTS**

**Initial Stage**
- Decreased stainable iron (Russian Blue stain) in bone marrow macrophages
- Decreased serum ferritin concentrations (reference range 85–155 ng/mL) where serum ferritin <45 ng/mL is highly indicative of iron deficiency.

**Later Stages**
- Decreased SI concentration (reference range, 120–150 µg/dL.)
- Normal or increased TIBC (reference range, 300–400 µg/dL.)
- Decreased percentage transferrin saturation ( ≈ 30%–50% [Arabian horses ≈60%] with values <16% reflecting insufficient iron available for erythropoiesis)
- Presence of microcytes (decreased MCV) with decreased hemoglobin concentration (hypochromia, decreased MCHC)
- SI, serum ferritin, and TIBC may be affected by conditions other than iron
Anemia, Iron Deficiency

**DEFINITION**
Deficiency including acute and chronic inflammation, renal disease, and corticosteroid therapy.

**OTHER DIAGNOSTIC PROCEDURES**
- Cytology of bone marrow aspirate may show a predominance of late rubricytes and metarubricytes, depletion of macrophage iron, and sideroblasts.
- A diagnostic workup of causes of chronic hemorrhage is required.

**TREATMENT**
- Horses with lethargy, intolerance to mild exercise, or a PCV <15% should be restricted to stall rest.
- Blood transfusion is rarely necessary unless PCV drops below 8% (0.08 L/L) or there are clinical and laboratory signs of tissue hypoxia.

**MEDICATIONS**

**DRUG(S)**
- Appropriate treatment of underlying disease process to resolve chronic blood loss
- Oral ferrous sulfate (1.0 g/450 kg body weight) is the safest means to administer iron.
- Iron cysideate (1 g/adult horse) may be given slowly IV, but must be used with caution due to the possibility of an anaphylactic reaction.

**CONTRAINDICATIONS/POSSIBLE INTERACTIONS**
- Do not administer iron dextran due to idiosyncratic reactions (anaphylaxis and sudden death).
- Iatrogenic iron overload has been reported in adult horses given unnecessary oral and/or parenteral iron supplementation.
- Do not give foals iron-containing products during the first 2 days of life as fatal toxic hepatopathies may result.

**MISCELLANEOUS**

**SEE ALSO**
- Anemia
- Anemia, aplastic (pure red cell aplasia)
- Anemia, Heinz body
- Anemia, immune-mediated
- Hemorrhage, chronic equine infectious anemia

**ABBREVIATIONS**
- GI = gastrointestinal
- PCV = packed cell volume
- SI = serum iron
- TIBC = total iron-binding capacity

**Suggested Reading**

The author and editors wish to acknowledge the contributions of Catherine W. Kohn, author of this topic in the previous edition.

**Author** Nicholas Malikides

**Consulting Editors** Jennifer Hodgson and David Hodgson
Anestrus

**Definition/overview**
Postpartum stalls are small and static. Characterized by indifferene behavior of mare to stallion.

**Etiology/pathophysiology**
Seasonally polyestrous, estrous cycles (ovulatory period) in spring and summer; primarily regulated by photoperiod, light begins a cascade:
- Increased day length decreases melatonin secretion (pineal gland).
- Decreasing melatonin allows increased production and release of GnRH.
- GnRH stimulates gonadotropin release (FISH and LH).
- FSH promotes folliculogenesis and ultimately the onset of estrus behavior.
- When sufficient LH is present, ovulation occurs; end of estrous cycle. Key hormonal events of equine estrous cycle:
  - FSH causes follicular growth.
  - Estradiol (follicular) stimulates increased GnRH pulse frequency and secretion of LH.
  - LH surge causes ovulation; estradiol returns to baseline levels 1–2 days post-ovulation.
- Progesterone (CL origin) rises from basal levels (<1 ng/mL) at ovulation to 4 ng/mL by 4–5 days post-ovulation.
- Progesterone causes increased GnRH pulse frequency and increased FSH secretion; it stimulates a new wave of follicular development beginning in diestrus.
- Endogenous PGF2α (endometrial) is released 14–15 days post-ovulation causing luteolysis and consequent decline in progesterone levels.

**Symptoms affected**
Reproductive
- Endocrine

**Signs**
**Historical**
- Chief complaint—failure of mare to accept stallion. Rarely reported—stallion-like behavior.
- Trajectory—Review methods used, results, frequency, tracer type (pony, horse, gelding), stallion behavior (aggressive/passive, vocalization, proximity), and handler experience.
- Seasonal influences—Evaluate normal individual variation of onset, duration, termination of cyclicity.
- Individual reproductive history—estrus cycle length, estrous pooling, feeding data, previous gestation/injuries/infections, and relationship to clinical abnormalities.
- Pharmaceuticals—Current and historical drug history may relate to clinical abnormalities.

**Physical examination**
- Poor body condition/malnutrition may contribute to anestrus.
- Poor perinatal conformation can result in pneumoenteritis, ascending infections and/or uterine pooling, anestrus/infertility.
- Clitoral enlargement may relate to drug history—anabolic steroids, progestational steroids, or immune conditions.
- TRP is essential to evaluate suspected anestrus mare. Assess uterine size and tone, ovarian size, shape and location, and cervical relaxation. Serial TRP 3 times a week 1–3 weeks to completely define status.
- Transrectal US to define normal and abnormal features of uterus and ovaries.
- Vaginal examination (digital and/or speculum) to identify inflammation, uterine pooling, cervical competency, conformational abnormalities.
- Dietary stage of the estrous cycle.

**Causes**

**Normal physiology**
- Winter anestrus—~20% of mares cycle through the winter (Northern Hemisphere—November to January), most enter a period of ovarian quiescence. Failure to cycle is normal during winter anestrus.
- Two transitional phases occur yearly—autumnal transition (ovulatory to anestrus) and vernal transition (anestrus to cyclicity/ovulatory).
- Behavioral patterns vary during transition periods. Individual variation in onset and length of transitional periods is normal.
- Behavioral anestrus (silent heat)—a normal estrous cycle as determined by serial TRP; failures to demonstrate estrus.
- Pregnancy—After recognition of pregnancy, CL progesterone production continues; majority of pregnant mares exhibit anestrus behavior.
- Pseudopregnancy—Embryos die after recognition of pregnancy or formation of endometrial cups, resulting in persistent CL activity and behavioral anestrus.
- Cushing’s disease—Adenomatous hyperplasia of the adrenal glands; can result in anestrus, silent estrus, or show increased aggression.
- Anabolic steroids—Affected mares behave as if in anestrus, silent estrus, or show increased aggression.
- Progesterone/progestin—Continued treatment inhibits estrus behavior.
- NSAIDs—Potential to interfere with endogenous PGF2α release; result is prolonged CL activity. No evidence that exogenous treatment (recommended therapeutic dose) inhibits spontaneous formation and release of endogenous PGF2α.

**Risk factors**
Postpartum anestrus occurs more often in early foaling mares and mares in poor body condition at time of parturition.

**Diagnosis**

**Differential diagnosis**

**Differentiating causes**
- Closely review teasing records, general and reproductive history—feeding data, evidence of infections, injuries, medications that may affect reproductive health. Serial TRP with/without US 1–2 weeks is adequate to differentiate transitional and behavioral anestrus.
- Pregnancy—Serum progesterone concentrations (~<1 mg/mL q7 days for 2 weeks) and pyometra.
- Other laboratory tests—Serum progesterone concentrations (~<1 mg/mL q7 days for 2 weeks).

**Other laboratory tests**
- Serum progesterone—Recommended therapeutic dose
- Active CL—~4 mg/mL
- Serum testosterone and inhibin
  - Mares—~<50–60 pg/mL, inhibin <0.7 ng/mL
- Levels suggestive of GCT/GTCT (in a nonpregnant mare) are—nonfunctional >50–100 pg/mL (if testicles are significant).
human chorionic gonadotropin

DIAGNOSTIC PROCEDURES

TREATMENT

MISCELLANEOUS

Anestrus

KEYWORDS

Anestrus

AGE-RELATED FACTORS

ACCESS TO FULL TEXT

PREGNANCY

POSSIBLE COMPLICATIONS

SUDDEN INFARCTION

 הפער הקטן ביותר בין השתיים של ציון וביון

Suggested Reading


Angular Limb Deformity

BASICS

DEFINITION
ALD is an abnormal rotation from the normal axis of the limb in the frontal plane. Valgus is the lateral deviation of the limb distal to the location of the deformity, while varus is the medial deviation of the limb to the location of the deformity. The deformity is named by the joint around which the deviation is centered (e.g., carpal valgus).

PATHOPHYSIOLOGY
There are two main categories associated with the etiology of ALD—perinatal factors and developmental factors.

Perinatal Factors
- Flexibility of patellar soft tissue structures and perinatal soft tissue trauma can lead to unstable joints, resulting in abnormal loading of the articular surfaces inducing ALD (manually correctible in the early stages).
- Anything to jeopardize the intrauterine environment of the foal (i.e., placenitis, twin foal) and premature birth (<315 days) may result in incomplete ossification (carpus and tarsus) at birth. If the joints are unready loaded while the bones are not yet ossified, the uneven pressure may result in abnormal shape once ossification occurs, leading to permanent ALD.
- Developmental Factors
  - Unbalanced nutrition (i.e., “clib feeding” leading to excessive grain intake, unbalanced trace minerals) can result in disproportionate growth at the level of the physis, causing ALD.
  - Frequently observed in rapidly growing foals. Can occur days to months after birth.
  - Excessive exercise and trauma can result in microfractures and crushing of the growth plate leading to early closure in severe cases (i.e., Salter-Harris type V fracture).

SYSTEM AFFECTED
Musculoskeletal—One or more joints may be involved in the front limbs and/or hindlimbs, including the fetlock, carpus, and tarsus. Most commonly, the angular limb deformity originates at the carpus. Carpal valgus deformity is the most commonly observed ALD, but tarsal valgus and fetlock varus are also seen commonly.

GENETICS
N/A

INCIDENCE/PREVALENCE
Most foals are born with some form of angular limb deformity; however, most cases resolve within 4 weeks without intervention.

GEOGRAPHIC DISTRIBUTION
N/A

SIGNALMENT
Most commonly encountered in neonatal foals.

General Comments
Natural growth of foals can lead to spontaneous correction of the deformity. However, foals with ALD must be monitored appropriately, as if they do not correct their conformation, there is a limited window during which time surgical intervention can occur prior to various physiologic closures. Ideal conformation varies between breeds and type of work desired (i.e., pleasure versus racing).

- Each foal will respond differently to treatment.
- Manipulation/palpation of the limb can cause or exacerbate the deformity.
- Hoof trimming may also be indicated to correct the deformity.

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS
- Laxity of patellar soft tissues
- Incomplete ossification/collapse of the cuboidal bones
- Diaphyseal curvature (MCIII/MTII)

CBC/BIOCHEMISTRY/URINALYSIS
N/A

OTHER LABORATORY TESTS
N/A

IMAGING
Radiographs allow for determination of the location and degree of the deformity, as well as concurrent physiologic strain or physical crushing, or cuboidal bone crushing. Radiographs should be centered over the joint of interest, including the mid-diaphysis of the bones proximal and distal to the deformity (longer cast for easier assessment of the deformity). Only two views are required for ALD (lateral/proximal and dorso/plantar). If there is evidence of joint problems, oblique images should be included.

OTHER DIAGNOSTIC PROCEDURES
- Examination of the limb in both a standing and a flexed position.
- Observation of the foal from several angles.
- Examination from a position perpendicular to a frontal plane through the limb. The toe should point in the same direction as the carpus.
- Observation of the foal at a walk.
- Manipulation/palpation of the limb can help determine whether the deformity was caused by perinatal (manual correction) or developmental factors (permanent).

PATHOLOGICAL FINDINGS
- Asymmetric early closure of either the medial or lateral physis due to injuries or inflammation.
- Delayed ossification.

TREATMENT

AIMS OF TREATMENT
To manage ALD, either conservatively or by providing surgical intervention, if needed, in order to correct growth. A straighter limb will allow for more even load-bearing and should reduce the incidence of athletic injury.

APPROPRIATE HEALTH CARE
N/A

NURSING CARE
Splints and Casts
- The purpose is to maintain the limb in proper alignment and to facilitate adequate weight-bearing without adverse consequences.
- Five foals with incomplete ossification of the cuboidal bones and deviation of the limbs
- Problems with casts and splints in foals include osteopenia and tendon/ligament laxity. Ending the cast/splint at the level of the fetlock can help prevent these problems.
- Splints should be changed every 3–4 days.
- Casts should be changed every 10–14 days.

Corrective Shoeing
- Application of glue-on/composite materials with an extension on the medial aspect (valgus deformities) or the lateral aspect (varus deformities) may assist in correction of the deformity.
- Hoof trimming may also be performed—the outside of the hoof should be lowered for valgus deformity; the inside for varus deformity.

Lameness
It is important to note oversize or create an abnormal hoof shape that will further alter normal weight-bearing.
Angular Limb Deformity

ACTIVITY

Stall Rest
- Effective treatment for newborn foals, specifically for incomplete ossification and straight limbs. The maximum period of rest is 6 months.
- Failure to return the foal to normal activity too soon results in disproportionate growth at the level of the physis (>10 degrees) and diaphyseal deformities should be stall rested for 4–6 weeks.
- Feals with laxity of the periarticular supporting structures require exercise in addition to stall rest. It is important not to prolong stall rest beyond 4–6 weeks.

DIET
Balanced nutrition is very important.

CLIENT EDUCATION
- Early recognition and treatment are important.
- The examination of a foal for ALD should begin shortly after birth, followed by examination once a week for 4 weeks, and once monthly for 6 months. This allows close monitoring to determine if the foal will self-correct or need surgical intervention.

SURGICAL CONSIDERATIONS

Growth Acceleration (Periosteal Transection and Elevation)
- Periosteal transection is performed on the convex aspect of the limb (e.g., lateral aspect of the distal radial physis for a carpal valgus deformity) in order to accelerate growth.
- Studies have indicated an 80% improvement in foals that have undergone a periosteal transection.
- The procedure is relatively inexpensive and easy, with the ability to be performed in the field. Foals should have this surgery at 4 weeks (earlier if the deformity is severe) to 3 months of age (limited growth beyond this time). The timing of surgery also depends on the site of abnormal growth (below).
- The maximum effect is observed within 2 months.

Growth Retardation (Transphyseal Bridging)
- Performed in foals <5 months with severe ALD or foals with significant ALD following the rapid growth phase (MCIII/MTIII and proximal phalanges) with ribia ≥ 4 months, and radius ≥ 6 months.
- The bridge is performed on the convex aspect of the affected limb. The goal is to retard growth on the convex side of the limb, allowing the shorter side of the affected limb to keep growing.
- Screws and cerclage wires are the most commonly used implants.
- Current techniques include two screws, one inserted in the center of the epiphysis and one into the proximal physis, with cerclage wire connecting the two in a figure-eight pattern. A more recent technique includes one transphyseal screw, which can be used across the physis of the distal MCIII/MTIII, the distal radius and the distal ulna. Surgical staple techniques and small bone plates have also been described for use in transphyseal bridging.
- Periosteal transection and elevation are often performed in combination with growth retardation techniques.
- A bandage should be maintained for 10–14 days.
- Stall rest the foal for 2–3 days following surgery.
- Evaluate radiographically every 2 weeks to assess.
- Implants need to be removed as soon as the deformity has been corrected, as overcorrection can occur.

Corrective Osteotomy
- Osteotomies have been performed for correction of significant ALD in foals with closed growth plates.
- Current techniques—closing wedge osteotomy, step osteotomy in the sagittal plane and step osteotomy in the frontal plane.
- Most frequently performed on MCIII/MTIII.
- Maintain a bandage and splint or cast for several weeks following surgery.

PREVENTION/AVOIDANCE
Balanced nutrition is very important.

POSSIBLE COMPLICATIONS
- Non-surgical management—pressure sores, osteopenia, and tendon/ligament laxity from cast/splint application
- Surgical management—hematoma/sequestrum formation at surgery site, incisional infection, wound dehiscence
- Overcorrection is possible if transphyseal bridging implants are not removed as soon as ALD has been corrected.
- Failure of passive transfer may result if foals are unable to nurse due to ALD following birth.

EXPECTED COURSE AND PROGNOSIS
- Studies have indicated an improvement in approximately 80% of foals that have undergone a periosteal transection. It has been reported that an athletic use was pursued for 80% of foals with ALD of the carpus and 27.3% of foals with ALD of the metacarpus/metatarsus after transphyseal bridging.

ASSOCIATED CONDITIONS
N/A

AGE-RELATED FACTORS
Timing of intervention is important, as the greatest effects of surgical manipulation will occur during the rapid growth phases.

ZOONECTIC POTENTIAL
N/A

PREGNANCY
N/A

SYNONYMS
N/A

SEE ALSO
Flexural limb deformity

ABBREVIATIONS
- ALD = angular limb deformity
- MCIII = third metacarpal bone
- MTIII = third metatarsal bone

Suggested Reading

Author: Shannon J. Murray
Consulting Editor: Margaret C. Mudge
Anhidrosis

OVERVIEW

Anhidrosis (also known as dry coat disease or a nonsweater, or as “dry puffers”) is the inability to sweat effectively in response to appropriate stimuli. The current theory is that overstimulation of sweat gland $\beta_2$-receptors causes diminished function or a period of unresponsiveness of the receptors.

SIGNALMENT

No coat color, age, sex, or breed predilections. Up to 20% of horses may be affected when exercising in a hot, humid climate.

SIGNS

- Extended tachypnea after exercise, later combined with a lack or reduction of sweating
- Horses recently introduced into a hot and humid climate may sweat excessively before showing signs of anhidrosis.
- With acute onset, horses may demonstrate partial or complete absence of sweating when exposed to appropriate stimuli.
- Horses with long-standing anhidrosis may exhibit dry and flaky skin with alopecia, lethargy, and decreased water intake. Body areas that may retain the ability to sweat include under the mane, saddle and halter regions, and the axillary, inguinal, and perineal regions.

CAUSES

- Systemic—Heat-stressed horses may have higher-than-normal levels of circulating catecholamines. Anhidrotic horses have significantly higher levels of epinephrine compared with normal horses at rest. These catecholamines act as $\beta_2$-agonists and may overstimulate the sweat gland receptors, which results in either desensitization of the receptor (i.e., the receptor is sequestered away from its normal site to another site within the cell) or down-regulation (i.e., decreased number of receptors). Down-regulation is a long-term mechanism that may involve altered synthesis or degradation of receptor proteins.
- Horses maintained in hot, humid climates are at risk, and exercise magnifies this risk.

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS

Respiratory diseases that cause an increase in the respiratory rate (both obstructive and restrictive diseases)

CBC/BIOCHEMISTRY/URINALYSIS

Dehydration, as evidenced by prerenal azotemia and, possibly, increased urinary specific gravity

DIAGNOSTIC PROCEDURES

Intradermal injections, in the neck area below the mane, of a specific $\beta_2$-agonist (e.g., terbutaline sulfate, salbutamol sulfate), serial dilutions ($10^{-7}$ to $10^{-8}$ [w/v]), and a control injection of sterile saline—read the results at 30 min. Normal horses sweat in response to all dilutions, whereas anhidrotic horses show a diminished response to some or all.

PATHOLOGICAL FINDINGS/HISTOPATHOLOGY

Thickened basal lamina, evidence of poor myoepithelial contraction, dilated connective tissues, and marked reduction of vesicles in the secretory cells. Luminal microvilli often are absent and the lumen of the duct is obstructed with cellular debris.

TREATMENT

- Advise clients that sound environmental management is the only reliable treatment option at present.
- Horses with acute anhidrosis who exhibit signs of heat stress should be immediately taken to a cooler environment, and attempts to reduce the body temperature should be made.
  - Restrict to a stall with adequate air movement (i.e., a fan) during hot periods of the day.
  - If exercise is necessary, do so during the cooler periods of the day. After exercise, make sure the horse is “cooled off” adequately by hosing it down with water.
Anhidrosis

Concentrates should be fed in decreased amounts. Allow access to cool, fresh water as well as water with electrolyte supplementation.

Inform clients that these horses will be prone to poor performance and will only improve once the capability to sweat effectively has returned.

It may not occur again in a horse’s lifetime but is usually a lifelong problem. However, when it does occur attempts to provide a cool, dry environment must be made.

If exogenous β2-agonists such as clenbuterol for concurrent respiratory problems are being administered, consider this as a possible cause and cease administration.

**PRECAUTIONS**

Anaphylaxis has been reported when using injectable vitamin E.

**ALTERNATIVE DRUGS**

Drugs that either reduce down-regulation or decrease sympathetic drive are still in the investigative stages.

**FOLLOW-UP**

**PATIENT MONITORING**

Normal thermoregulatory abilities allow a horse to reduce its body temperature to within normal limits approximately 30 min after exercise. Monitor respiration and rectal temperature post-exercise.

**PREVENTION/AVOIDANCE**

- Do not expose anhidrotic horses, especially when exercising, to extreme ambient temperatures.
- Exercise during the cooler periods of the day and stall the horse in a cooler environment (e.g., an air-conditioned stall) during the hotter periods of the day.
- Relocating the horse to a more temperate climate may lead to resolution of the clinical signs.
- Avoid administration of exogenous β2-agonists such as clenbuterol.

**POSSIBLE COMPLICATIONS**

Heat stroke may occur if horses are exercised during the hotter periods of the day.

**EXPECTED COURSE AND PROGNOSIS**

- Most horses respond to a change in environment and begin to sweat normally after a few weeks.
- Horses that have previously suffered from the disease will usually, but not necessarily, become anhidrotic if exposed to hot, humid conditions again.

**MISCELLANEOUS**

**ASSOCIATED CONDITIONS**

Skin lesions—dry, flaky skin and alopecia, especially around the eyes and shoulders

**SEE ALSO**

Skin diseases

**Suggested Reading**


**Authors**

Jeremy D. Hubert and Ralph E. Beadle

**Consulting Editor**

Michel Levy
Anorexia and Decreased Food Intake

**BASICS**

**DEFINITION**
Anorexia is the loss of appetite or lack of desire for food. Some conditions that cause anorexia may not lead to complete loss of appetite, but merely reduced food intake.

**PATHOPHYSIOLOGY**

**Appetite Suppression**

- A decrease in appetite appears to be the result of a modification of central regulation of feeding behavior in the hypothalamus. *Many factors and substances appear to be involved in regulating food intake.* Anorexia associated with alterations of smell and taste has not been shown in the horse.
- Decreased food intake has been associated with parasitic infections, but the mechanism is unknown.
- Pain and depression appear to cause anorexia, as well as causing dehydration, electrolyte imbalances, acid-base disorders, micronutrient deficiencies, and changes in concentrations of neurotransmitters, hormones, or mediators.
- Serotonin agonists decrease food intake, apparently via central histaminergic activity. The neurotransmitter neuropeptide Y and various cytokines may cause CACS. Cytokines induce anorexia when administered peripherally or directly into the brain. Administration of specific cytokine antagonists mitigates cachexia in experimental animal models.
- Oral lesions may not lead to complete loss of appetite, but merely reduced food intake.

**CAUSES**

Anorexia

- Commonly due to gastrointestinal or abdominal disorders, including colic. May be secondary to one of the following primary disease processes in any organ system:
  - Inflammation
  - Infections (bacterial, viral, fungal, or parasitic)
  - Injury
  - Trauma
  - Immunologic reactions
  - Malnutrition
  - Necrosis
  - Dehydration
  - Electrolyte imbalances
  - Acid-base disorders
  - Severe respiratory distress
  - Neurologic disorders
  - Ulcers or oral sublingual abscesses
  - Cardiac disease
  - Metabolic disorders
  - Side effects of medications
  - Pain

Food consumption problems may be due to:

- Pain in lips, tongue, or mouth (e.g., ulcers, lacerations, dental "points")
- Mechanical obstructions (e.g., severe swelling of the lips)
- Nervous dysfunction of the lips or tongue
- Necrosis of the lips or tongue
- Neurologic dysfunction

Swallowing problems may be due to:

- Pain in pharynx or esophagus
- Mechanical obstructions in pharynx or esophagus
- Neurologic dysfunction (e.g., CN-XI, although questioned kindly)
- Unpalatable food due to contamination or spoilage

**RISK FACTORS**

Clot, which is the layperson’s term for food impaction of the esophagus, occurs more commonly in animals that bolt their food or have defective teeth.

**DEFINITION**
Anorexia

- Colic
- Esophagitis
- Gastrointestinal ill
- Gastric ulcers and pyloric stenosis
- Peritonitis

Secondary to a primary disease process in any organ system:

- Renal failure
- Renal tubular acidosis
- Cardiac amyloidosis
- Severe respiratory distress
- Depression of the nervous system—especially cerebral disorders
- Inflammation or endotoxemia
- Injury
- Trauma (e.g., mounen, lead)
- Immunologic reactions
- Malnutrition
- Necrosis

Secondary to diseases leading to dehydration, electrolyte imbalances, or acid-base disorders

- Hypernatremia
- Side effect of mercurial diuretics or toluene or cyanophenidate

**DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS**

Anorexia

- Gastric ulcers and pyloric stenosis
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**SYMPTOMS**

Inability to swallow

- Hypoesthesia of the face
- Bilateral paralysis of facial muscles (CN-VII)
- Inability to swallow
- Increased salivation (ptyalism) due to:
  - Reduced food intake can also be caused by various factors affecting the lips, mouth, tongue, pharynx, esophagus, or stomach, and may include painful conditions, mechanical obstructions, or nervous or neuromuscular dysfunctions.

**RISK FACTORS**

- Other primary disease conditions, such as infection, inflammation, injury, toxins, immunologic reactions, and necrosis, may cause anorexia via cytokines as well.
- In addition, a proteoglycan has been identified on the cell membranes of animals and has been named anesie. It reduces food intake and may be a source of anorexicogenic substance.
- Reduced food intake can also be caused by various conditions affecting the lips, mouth, tongue, pharynx, esophagus, or stomach, and may include painful conditions, mechanical obstructions, or nervous or neuromuscular dysfunctions.

**SIGNMET**

Any signalment

**SIGNS**

May be a lack of interest in food or an interest only in certain types of food. May note difficulty or inability in swallowing, chewing, or swallowing of food, and food may appear to the mouth. Nasal discharge and cough can occur due to foreign material entering trachea, acquired aspiration pneumonia, or both. Some of the signs seen in horses with anorexia may include the following:

- Increased salivation (pyralism) due to:
  - Inability to swallow
  - Hypoesthesia of the face (CN-V)
  - Neuropathy of the masticatory muscles (CN-V, motor component)
- Bilateral paralysis of facial muscles (CN-VII)
- May expel partially chewed food (“gulping”)
- Oral lesions

**CAUSES**

Anorexia

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  - Inflammation
  - Infections (bacterial, viral, fungal, or parasitic)
  - Injury
  - Trauma
  - Immunologic reactions
  - Malnutrition
  - Necrosis
  - Dehydration
  - Electrolyte imbalances
  - Acid-base disorders
  - Severe respiratory distress
  - Neurologic disorders
  - Ulcers or oral sublingual abscesses
  - Cardiac disease
  - Metabolic disorders
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**RISK FACTORS**

Clot, which is the layperson’s term for food impaction of the esophagus, occurs more commonly in animals that bolt their food or have defective teeth.
Anorexia and Decreased Food Intake

**DIET/ACTIVITY**
Offer highly palatable and varied food in cases of anorexia. Supply feed that is easy to chew and swallow in cases of dysphagia. Force-feeding by nasogastric intubation or parenteral nutrition may be required. Activity should be limited to stall rest or hand-walking in most cases.

**MEDICATIONS**

**DRUG(S) OF CHOICE**
- Depends on primary disease process
- Oral administration of 40 g of KCl once or twice daily in anorectic patients

**CONTRAINDICATIONS**
- KCl administration may be contraindicated in patients suspected of having hyperkalemic periodic paralysis.

**FOLLOW-UP**
**PATIENT MONITORING**
The patient should be monitored for dehydration, electrolyte imbalance, acid-base abnormalities, and weight loss, and, in cases of dysphagia, aspiration pneumonia.

**EXPECTED COURSE AND PROGNOSIS**
- Depends on the underlying cause

**ASSOCIATED CONDITIONS**
- Other primary disease conditions, such as infection, inflammation, injury, toxins, immunologic reactions, and success
- Cancer-related anorexia/cachexia syndrome (CACS), a syndrome of anorexia and weight loss that occurs secondary to malignancy
- Guttural pouch disease can cause neurologic damage to CN-XII, CN-XI, and impair chewing and swallowing as well as causing mechanical obstruction to swallowing.
- Trauma
- Neoplastic maladjustment syndrome may interfere with swallowing or the suckle reflex.
- Moderate jaundice may occur due to increased indirect bilirubin levels in the blood. This is an idiopathic finding in the horse that occurs with fasting or decreased intake of feed.
- Dysphagia, electrolyte imbalances (hyperkalemia, hypoalbuminemia), or acid-base disorders as a result of lack of intake of fluid and electrolytes may exacerbate the anorexia.
- Salivary loss of electrolytes leads to metabolic alkalosis and hypochloremia, primarily.

**SECONDARY OR CONDUCTIONAL**
- Secondary or conditional PCM involves weight loss with prolonged anorexia.
- Aspiration pneumonia occurs secondary to dysphagia.

**AGE-RELATED FACTORS**
- Old age: hyperkalemia, hypoalbuminemia, and nutritional muscular dystrophy (white muscle disease) are noted most commonly in the neonatal period.

**ZOONOTIC POTENTIAL**
- Rabies can cause anorexia or dysphagia. Precautions should be taken while examining and treating the patient.

**SYNONYM**
Decreased appetite

**SEE ALSO**
- Aspiration pneumonia
- Botulism
- Cerebellar disorders of the central nervous system
- Choanal
- Colic
- Dental disease
- Epiglottic cysts
- Esophagitis
- Fractured mandible
- Gastric ulcers
- Gastrointestinal enteritis
- Gastrointestinal ileus
- Lead toxicity
- Monosodium toxicity
- Organophosphate toxicity
- Rabies
- Pharyngeal abscess
- Pharyngitis
- Post traumatic myasthenia
- Ruptured rectus capitis ventralis muscle
- Sinusitis
- Snake bites
- Strangles
- Tetanus
- Tick paralysis
- Yellow star thistle poisoning
- Vascular stenoses
- Vitamin E/alpha-tocopherol deficiency causing swollen masserer muscles

**ABBREVIATIONS**
- CACs = cancer-related anorexia/cachexia syndrome
- CN = cranial nerve
- PCM = protein-calorie malnutrition

**Suggested Reading**

**Author**
Gail Abells Sutton

**Consulting Editors**
- Henry Stimpfl and
- Olga Oses-Espinosa
Anthrax

Overview
Anthrax is a rapidly fatal septicemic disease of animals and human beings caused by Bacillus anthracis, which occurs in localized regions worldwide. In the horse, infection usually results from ingestion of soil, forage, or water contaminated with B. anthracis spores. In the animal, the organism germinates and produces endotoxin that impair phagocytosis and vascular integrity resulting in hemorrhage, edema, renal failure, shock, and almost invariably death. When B. anthracis is exposed to the environment, long-lasting spores are formed that are a potential source of infection for other animals.

Signs
• Fever, depression, and death in <4 days is characteristic of the acute form.
• Severe colic, bloody discharge from body orifices, and painful subcutaneous swellings may be noted.
• A chronic form resulting in pharyngeal edema has been described.
• The paretic form, in which death occurs with few clinical signs, appears to be less common in horses than in ruminants.

Causes and Risk Factors
• The source of infection is usually soil contaminated by exudates from infected animals. B. anthracis forms spores that are very resistant to environmental conditions and most disinfectants, and these spores may persist in the soil for decades. Ingestion of contaminated soil, feed, or water is the most common route of infection, but the organisms may also be inhaled or inoculated by biting insects.
• Anthrax is most common in tropical and subtropical climates but is seen sporadically in temperate regions, usually in the summer. Anthrax usually occurs in regions with alkaline soils and with climatic cycles of heavy rain and drought.
• Overgrazing increases the risk of disease by increasing the ingestion of soil. Coarse forages with few clinical signs, appears to be less common in horses than in ruminants.

Diagnosis

Differential Diagnosis
• Lightning strike can be differentiated on the basis of history of storms and absence of post-mortem findings typical of anthrax.
• Colic and enteritis can be differentiated by finding evidence of gastrointestinal disease at post-mortem.
• Purpura hemorrhagica has similar signs but is not rapidly fatal.
• Toxicity can be differentiated based on history and lack of post-mortem findings typical of anthrax.
• Malignant edema may appear similar, but crepitation of swellings is not found with anthrax.

CBC/Biochemistry/Urine analysis
Routine laboratory findings have not been reported.

Other Laboratory Tests
Bacterial culture of blood or exudate is useful, although results may be negative early in disease or if antibiotics have been administered. Cultures should only be performed in a facility capable of containment to prevent infection of laboratory personnel.

Imaging
N/A

Other Diagnostic Procedures
Organisms may be seen by microscopic examination of blood smear or edema fluid. Bacilli are gram-positive, have blunt ends, are encapsulated, and occur singly or in short chains.

Fluorescent antibody of blood or tissue may be diagnostic.

Pathologic Findings
• Due to human health risk and danger of environmental contamination, necropsy should not be performed if anthrax is strongly suspected. Diagnosis can be made without necropsy.
• Dark, nonclotting blood from orifices; absence of rigor mortis; splenomegaly; and lymphadenopathy are hallmarksof anthrax.
• Serosal and mucosal hemorrhage and edema of many organs are seen.

Treatment
• The high mortality and rapid course of disease usually limit opportunity for treatment. The prognosis is poor even with treatment.
• Isolate affected and in-contact animals.

Medications

Drug(s) of Choice
• Penicillin G (40,000 IU/kg IV q6–8 h) or oxytetracycline (5–11 mg/kg IV q12 h) is traditionally recommended. Enrofloxacin (7.5 mg/kg PO q12h or 5 mg/kg IV q24h) is potentially a good choice in adult horses. Continue treatments for at least 5 days.
• Anthrax antiserum may be useful but is not available in the United States.

Contraindications/Possible Interactions
N/A

Follow-Up
• Regulatory officials should be notified when anthrax is suspected and the premises placed under quarantine.
• Carcasses should not be opened, and may be disposed of by burning or deep (>6 ft) burial with lime. The area can be disinfected with 5% aqueous lye or 10% formaldehyde.
• Susceptible animals should be vaccinated. An antigenive live spore vaccine is administered subcutaneously and provides immunity in 1 week. Some authors recommend a second vaccination in 2–4 weeks. Annual boosters are required to maintain immunity. Severe adverse reactions have been reported; therefore, the vaccine is indicated only in endemic regions. No antibiotics should be administered within 5 days before or after vaccination, or the vaccine organism may be inactivated.

Miscellaneous

Zoonotic Potential
Anthrax is a zoonotic inhalation or ingestion of spores may lead to fatal disease. Gloves and mask should be worn if it is necessary to contact infected material or animals. Canine anthrax is the most common form in human beings, resulting from inoculation of an open wound with spores.

SYNONYMS
• Woolsorters’ disease
• Charbon
• Splenic fever

Consulting Editors
Ashley G. Boyle and Corinne R. Sweeney

Suggested Reading

Author
Laura K. Reilly

Consulting Editors
Ashley G. Boyle and Corinne R. Sweeney
ANTICOAGULANT RODENTICIDE TOXICOSIS

BASICS

OVERVIEW
• Injection of anticoagulant rodenticides interferes with normal blood clotting in horses.
• Anticoagulant rodenticides are the most commonly used class of rodenticides.
• First-generation anticoagulants (i.e., warfarin, gindone, coumarin, coumadin) are short-acting coumarin derivatives requiring multiple feedings to result in toxicosis.
• Intermediate anticoagulants (i.e., brodifacoum, bromadiolone, difethialone) are highly toxic to non-target species after a single feeding.
• Most anticoagulant rodenticides commonly used today are long-acting, second-generation anticoagulants, with activity in the body of 3–1 month.
• Coagulopathy has been reported in horses after a dose of brodifacoum of 0.125 mg/kg (equal to ingestion by an average-size horse of 1 kg of bait containing 0.005% brodifacoum).
• Warfarin has been used therapeutically (30–75 mg per 450 kg) in horses with DIC, and thrombophlebitis.

CAUSES AND RISK FACTORS

• Ingestion of bait packages or as a result of feeding.
• Poisoning, i.e., accidental ingestion of bait packages or as a result of malicious intent.
• Iatrogenic warfarin toxicosis may result from overdosing, dietary vitamin K deficiency, or concurrent use of protein-bound drugs that interfere with normal blood clotting in horses.

SIGNALMENT
• May affect all animals.
• Poisoning can occur after accidental ingestion of bait packages or as a result of malicious intent.
• Poisoning is rare in horses because of the amount of bait needed to be ingested to cause signs.
• Iatrogenic warfarin toxicosis may result from overdosing, dietary vitamin K deficiency, or concurrent use of protein-bound drugs that increase the concentration of active, unbound warfarin.

SIGNS
• Bleeding diathesis ranging from mild to severe.
• Hemorrhage—internal or external
• Signs generally manifest within 3–5 days after ingesting bait.
• Signs are similar to those seen with dicumarol toxicosis.

EXPECTED COURSE AND PROGNOSIS

• Prognosis is based on the severity of blood loss and damage to organ systems affected by hemorrhage.

PREVENTION/AVOIDANCE
• Prevent access to bait packages.
• Keep anticoagulant rodenticides well out of the reach of children and pets.

FOLLOW-UP

PATIENT MONITORING
• Continue monitoring for blood loss.
• Check PT 2–3 days after the last dose of vitamin K₃, to determine if additional treatment is necessary.

CONTRAINDICATIONS/POSSIBLE COMPLICATIONS
• Do not use vitamin K₃ (menadione) in horses. Vitamin K₃ is ineffective against anticoagulant rodenticide toxicosis and is nephrotoxic.
• Drugs generally contraindicated are NSAIDs, phenothiazine tranquilizers, local anesthetics, antimicrobials, sulfonamide antibiotics, anabolic steroids, and epinephrine.

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS
• Moldy sweet clover ingestion—history of ingesting plant, detection of dicumarol in forage or tissue samples
• DIC—reduced plasma concentrations of platelets and coagulant and anticoagulant proteins; increased concentrations of coagulant byproducts; petechial hemorrhages

SIGNS
• Signs are similar to those seen with dicumarol toxicosis.
• Signs generally manifest within 3–5 days after the last dose of brodifacoum.
• Hemorrhage—internal or external
• Hemorrhage ranging from mild to severe
• Coagulopathy

DRUG(S) OF CHOICE
• Vitamin K₁ (phytonadione 2.5 mg/kg q12h SQ initially then PO after 3–5 days and continuing for 3–5 weeks) effectively reverses the clotting defect.
• AC at 1–4 g/kg body weight in water slurry (1 g AC in 5 mL water) PO. One dose of cathartic PO with AC if no diarrhea or dea (70% sorbitol at 3 mL/kg or sodium or magnesium sulfate at 250–500 mg/kg).

MISCELLANEOUS, ASSOCIATED FACTORS, ZOONOTIC POTENTIAL

• See also Dicumarol (moldy sweet clover) toxicosis

ABBREVIATIONS
• AC = activated charcoal
• aPTT = activated partial thromboplastin time
• DIC = disseminated intravascular coagulation
• PT = prothrombin time

Suggested Reading

Author: Anita M. Kore
Consulting Editor: Robert H. Poppenga
Anuria/Oliguria

**BASICS**
- **Anuria**—lack of urine production
- **Oliguria**—decreased urine production (<0.25 mL/kg per hr, or <125 mL/hr in a 500-kg horse)
- Anuria or oliguria may be physiologic or pathological.
- This chapter will focus on intrinsic renal failure causing anuria and oliguria.

**SYSTEM AFFECTED**
Renal/urologic

**CAUSES AND RISK FACTORS**
- **Physiologic oliguria**—hyperosmolality; any disease process leading to renal hypoperfusion (e.g., dehydration, hypotension, low cardiac output).
- **Pathological anuria/oliguria**—intrinsic ARF or birth trauma (e.g., dystocia) would increase the risk of urinary tract disruption and uroperitoneum in neonates and their dams; penile trauma is more common in breeding stallions.

**DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS**
Pathologic Anuria/Oliguria
- Intrinsic ARF, terminal CRF, lower urinary tract disruption resulting in uroperitoneum, and urinary tract obstruction consequent to urolithiasis
- Bladder displacement

**TREATMENT**
- Treat anuria/oliguria as a medical emergency because persistent renal hypoperfusion may lead to ischemic ARF.
- If untreated, metabolic disturbances, most notably hyperkalemia, may lead to cardiac arrhythmias and death.
- Once the patient is stabilized (largely with supportive treatment in the form of IV fluid therapy), pursue further diagnostic evaluation to determine if surgical intervention (for correction of uroperitoneum or relief of obstruction) is needed.
- Proper recognition and treatment of all primary disease processes, usually on an inpatient basis for continuous fluid therapy, is warranted.
- Avoid nephrotoxic medications.

**MEDICATIONS**
**DRUG(S) OF CHOICE**
- Fluid therapy to correct renal hypoperfusion—afore initial measurement of body weight, correct estimated dehydration with normal (0.9%) saline or another potassium-poor electrolyte solution over 6–12 hr; monitor closely for subcutaneous and pulmonary edema (i.e., increased respiratory rate and effort); conjunctival edema may develop rapidly in horses with intrinsic oligemic to anuric ARF; use maintenance fluid therapy judiciously in animals that are not clinically dehydrated; if hemorrhage is contributing to hypovolemia and renal hypoperfusion, initial treatment with hypertonic saline and/or a blood transfusion may have value.
Anuria/Oliguria

Severe hyperkalemia (>7.0 mEq/L) or cardiac arrhythmias—treat with agents that decrease serum potassium concentration (e.g., sodium bicarbonate [1–2 mEq/kg IV over 5–15 minutes]), or counteract the effects of hyperkalemia on cardiac conduction (e.g., calcium gluconate [10.5 mL/kg of a 10% solution by slow IV injection]).

Furosemide—this diuretic may be administered two times (1–2 mg/kg IV) at 1–2-hr intervals; if effective, urination should be observed within 1 hour after administration of the second dose; if ineffective, discontinue.

Based on recent evidences in critically ill human patients the ROUTINE USE OF MANNITOL or DOPAMINE IN EQUINE PATIENTS WITH ARF IS NO LONGER RECOMMENDED.

CONTRAINDICATIONS
Avoid all nephrotoxic medications unless specifically indicated for the underlying disease process, and then modify dosage accordingly.

PRECAUTIONS
• Monitor response to fluid therapy closely—as little as 40 mL/kg of IV fluids (20 L to a 500 kg horse) may produce significant pulmonary edema.
• Reassess dosage schedule of drugs eliminated by urinary excretion; consider discontinuing all nephrotoxic medications (especially gentamicin, tetracycline, and NSAIDs).

POSSIBLE INTERACTIONS
Use of multiple anti-inflammatory drugs (e.g., corticosteroids and one or more NSAIDs) will have additive negative effects on renal blood flow; avoid combined administration in azotemic patients.

FOLLOW-UP

PATIENT MONITORING
• Assess clinical status (emphasizing hydration), urine output, and body weight frequently for the first 3 days.
• Assess magnitude of azotemia and electrolyte and acid–base status at least daily for the first 3 days of treatment.
• Consider placing a central venous line to maintain central venous pressure ≥8 cm H₂O in more critical patients and neonates.

POSSIBLE COMPLICATIONS
• Severe hyperkalemia accompanied by cardiac arrhythmias, cardiac arrest, and death
• Pulmonary and peripheral edema; conjunctival edema may be dramatic.

MISCELLANEOUS

ASSOCIATED CONDITIONS
• Colic; enterocolitis
• Urosepsis; peritonitis; septicemia
• Exhausted horse syndrome—multiorgan failure

AGE-RELATED FACTORS
Neonates afflicted with hypoxic-ischemic multiorgan damage or septicemia may be at increased risk of anuric/oliguric ARF.

ZOONOTIC POTENTIAL
Leptospirosis has infectious and zoonotic potential; avoid direct contact with infective urine.

SEE ALSO
ARF
CRF
Urinary tract obstruction
Uroperitoneum

ABBREVIATIONS
ARF = acute renal failure
CRF = chronic renal failure
GFR = glomerular filtration rate
PCV = packed cell volume
USG = urinary specific gravity
UTI = urinary tract infection

Suggested Reading

Author Harold C. Schott II
Consulting Editor Gillian Perkins
Aortic Regurgitation

**BASICS**

**DEFINITION**
- Occurs when the aortic valve allows blood to leak into the left ventricular outflow tract during diastole, creating a holodiastolic decrescendo murmur with its P2 in the aortic valve area.
- The murmur radiates toward the left cardiac apex and the right side.

**PATHOPHYSIOLOGY**
- The aortic leaflets do not form a complete seal between the aorta and left ventricle.
- During diastole, blood regurgitates into the left ventricular outflow tract, causing a left ventricular volume overload. At this volume overload becomes more severe, stretching of the mitral annulus occurs, and mitral regurgitation often develops. Mitral regurgitation compounds the severe left ventricular volume overload, and these horses often rapidly develop congestive heart failure.
- Severe regurgitation results in decreased coronary artery blood flow and decreased myocardial perfusion. Ventricular arrhythmias may develop secondary to decreased myocardial perfusion.

**SYSTEM AFFECTED**
- Cardiovascular

**GENETICS**
- N/A

**INCIDENCE/PREVAILANCE**
- N/A

**GEOGRAPHIC DISTRIBUTION**
- N/A

**SIGNMENT**
- Usually horses > 10 years

**SIGNS**
- General Comments
  - Often an incidental finding during routine auscultation
  -** Poor performance  •  Positively congestive heart failure**

**Physical Examination**
- Grade 1–6/6, decrescendo or musical holodiastolic murmur at the aortic valve area (left or right fourth intercostal space) radiating to the left apex and right side.
- Other, less common findings—bounding arterial pulses, atrial fibrillation, ventricular premature depolarizations, accentuated third heart sounds, and congestive heart failure

**CAUSES**
- Degenerative changes of the aortic leaflets
- Percutaneous aortic leaflets
- Nonvegetative valvulitis
- Flail aortic leaflet
- Infective endocarditis
- Ventricular septal defect
- Congenital malformation
- Disease of the aortic root

**RISK FACTORS**
- Old age

**DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS**
- Pulmonic regurgitation—rare; murmur usually are soft or not detectable and should have P2 in the pulmonic valve area; bounding arterial pulses are not present; dierentiate echocardiographically.
- CBC/BIOCHEMISTRY/URINALYSIS
  - May have neutrophilic leukocytosis and hyperleucocytosis with bacterial endocarditis

**OTHER LABORATORY TESTS**
- Elevated cardiac isoenzymes may be present (e.g., cardiac troponin I, CK-MB, HBDH, LDH-1 and LDH-2) with concurrent myocardial disease.
- Positive blood culture may be obtained from horses with bacterial endocarditis.

**IMAGING**
- Echocardiography
  - Ventricular premature depolarizations may be present in horses with severe regurgitation and be caused by poor myocardial perfusion.
  - Atrial fibrillation often develops in horses with marked left ventricular volume overload and subsequent left atrial enlargement.
  - Aortic regurgitation
    - Most affected horses have thickened aortic valve leaflets.
    - An echogenic band parallel to and a nodular thickening of the left coronary leaflet free edge are the most common findings.
    - Prolapse of an aortic leaflet (usually the noncoronary or right coronary leaflet) into the left ventricular outflow tract frequently is detected.
    - Fenestration of the aortic leaflet, flail aortic leaflet, vegetation associated with infective endocarditis, or aortic root abnormalities infrequently are detected.
    - Left ventricular—enlarged and dilated, with a rounded apex.
    - Thinning of the left ventricular free wall and interventricular septum.
    - Increased septal-to–E-point separation may be present.
    - Pattern of left ventricular volume overload.
    - Normal or decreased fractional shortening in a horse with left ventricular enlargement is consistent with myocardial dysfunction.
    - Dilatation of the aortic root in horses with longstanding regurgitation.
    - High-frequency vibrations on the mitral valve septal leaflet usually are detected with M-mode echocardiography and are created by turbulence in the left ventricular outflow tract.
    - In some horses, high-frequency vibrations may be visualized on the interventricular septum instead of, or in addition to, vibrations on the mitral valve septal leaflet.
    - High-frequency vibrations on the aortic leaflets usually are visualized in horses with musical holodiastolic murmurs.

**DIAGNOSTIC PROCEDURES**
- Cardiac Catheterization
  - Right-sided catheterization may reveal elevated pulmonary capillary wedge pressures and pulmonary arterial pressures in horses with severe regurgitation and concurrent mitral regurgitation.
  - Right ventricular and atrial pressures may be elevated in affected horses with congestive heart failure.
  - Oxygen saturation of blood obtained from the right atrium, right ventricle, and pulmonary artery should be normal.

**Continuous 24-Hour Holter Monitoring**
- Use in the diagnosis of horses with suspected ventricular premature depolarizations.

**PATHOLOGIC FINDINGS**
- Focal or diffuse thickening or distortion of one or more aortic leaflets may be present.
- Nodules, bands, plaques, and fenestrations have been described on the aortic leaflets at postmortem examination.
- Flail aortic leaflets, infective endocarditis, or congenital malformations of the aortic valve infrequently are detected.
- Aortic root dilation usually is present in horses with severe, long-standing regurgitation.
- Jet lesions are not detected on the ventricular side of the mitral valve septal leaflet and, less frequently, on the interventricular septum.
- Left ventricular enlargement and thinning of the left ventricular free wall and interventricular septum in horses with significant regurgitation.
- Atrial myocardial thinning with atrial dilation has been documented in horses with atrial fibrillation and enlargement.
- Inflammatory cell infiltrate has been detected in horses with myocarditis and aortic regurgitation; however, most affected horses do not have significant underlying myocardial disease.
**TREATMENT**

**AIMS OF TREATMENT**
- Management by intermittent monitoring in horses with aortic regurgitation that is mild or moderate in severity
- Palliative care in horses with severe aortic regurgitation

**APPROPRIATE HEALTH CARE**
- Most affected horses require no treatment and can be monitored on an outpatient basis.
- Horses with moderate to severe regurgitation may benefit from long-term vasodilator therapy, particularly with ACE inhibitors.
- Treat horses with severe regurgitation and congestive heart failure for the congestive heart failure with positive inotropic drugs, vasodilators, and diuretics on an inpatient basis, if possible, and monitor response to therapy.

**NURSING CARE**
- **N/A**

**ACTIVITY**
- Affected horses are safe to continue in full athletic work until the regurgitation becomes severe or ventricular arrhythmias develop.
- Monitor horses with moderate to severe regurgitation by ECG during high-intensity exercise to ensure they are safe for ridden activities. These horses can be used for lower-level athletic activities until they begin to develop congestive heart failure.
- Horses with significant ventricular arrhythmias or pulmonary artery dilatation are no longer safe to ride.

**DIET**
- **N/A**

**CLIENT EDUCATION**
- Regularly palpate the arterial pulses to monitor the progression of left ventricular volume overload. Bounding arterial pulses indicate significant left ventricular volume overload. Moderate to severe regurgitation usually is present in these horses.
- **N/A**
- Carefully monitor for exercise intolerance, respiratory distress, prolonged recovery after exercise, inhaled exerting respiratory rate or heart rate, or cough; if detected, seek a cardiac examination.

**SURGICAL CONSIDERATIONS**
- **N/A**

**MEDICATIONS**

**DRUGS**
- Severe regurgitation—Administer enalapril (0.25–0.5 mg/kg PO q24h or q12h) or another ACE inhibitor
- ACE inhibitors prolong the time to valve replacement in humans with moderate to severe regurgitation.
- The bioavailability of enalapril is poor but horses with moderate to severe regurgitation have experienced a decrease in left ventricular chamber size with ACE inhibitors.
- Treatment of affected horses in heart failure include digoxin, furosemide, and vasodilators.

**CONTRAINDICATIONS**
- ACE inhibitors and other vasodilators must be withdrawn before competition to comply with the medication rules of the various governing bodies of equine sports.

**PRECAUTIONS**
- ACE inhibitors can cause hypotension; thus, do not give a large dose without time to accommodate to this treatment.

**POSSIBLE INTERACTIONS**
- **N/A**

**ALTERNATIVE DRUGS**
- Most other vasodilatory drugs should have some beneficial effect in horses with moderate to severe regurgitation, but they may be less effective than the ACE inhibitors.

**FOLLOW-UP**

**PATIENT MONITORING**
- Regularly monitor arterial pulses and cardiac rhythm.
- Reexamine horses with mild to moderate regurgitation by ECG every year.
- Reexamine horses with severe regurgitation by echocardiography every 6 mo to monitor progression of valvular insufficiency and determine if the horse continues to be safe to ride or drive.

**PREVENTION/AVOIDANCE**
- **N/A**

**POSSIBLE COMPLICATIONS**
- Chronic regurgitation—ventricular arrhythmias; atrial fibrillation; mitral regurgitation; congestive heart failure.

**EXPECTED COURSE AND PROGNOSIS**
- Most affected horses have a normal performance life and life expectancy.
- Progression of regurgitation associated with degenerative valve disease usually is slow. With the typical onset of regurgitation that occurs in old horses, other problems are more likely to end a horse’s performance career or shorten life expectancy.
- Affected horses with congestive heart failure usually have severe underlying valvular heart disease and myocardial disease and a guarded to grave prognosis for life. Most affected horses being treated for congestive heart failure respond to the supportive therapy and improve. This improvement usually is short lived, however, and most are euthanized within 2–6 mo of initiating treatment.
Aortic Root Rupture

BASICS

DEFINITION
A defect in the wall of the aorta at the aortic root, usually in the right sinus of Valsalva.

PATHOPHYSIOLOGY
- Aortic rupture results in the exanguination into the thoracic cavity, cardiac tamponade from hemo-pericardium, or a shunt between the aorta and heart.
- With an aortic rupture confined to the right sinus of Valsalva, an aortieocardiac fistula is created. Blood from the aorta shunts into the right side of the heart, at either the atrial or ventricular level, depending on the site of the rupture.
- Subendocardial dissection of blood into the interventricular septum is common, with subsequent rupture into the right or left ventricle (more commonly, the rupture is into the right ventricle).
- Often associated with a unifocal ventricular tachycardia that may be associated with dissection of blood into the interventricular septum.

SYSTEM AFFECTED
Cardiovascular

INCIDENCE/PREVALENCE
More frequently occurs in old horses, particularly males.

SIGNALMENT
Often occurs during or after breeding or other exercise.

SIGNS
General Comments
Often interpreted by owners as colic, because the horse appears distressed, may be looking at its flanks, and acts uncomfortable.

Historical
- Acute onset of colic or distress, usually after exercise or breeding.
- Less commonly, exercise intolerance; syncope.

Physical Examination
- Tachycardia
- Tachypnea
- Continuous machinery murmur—usually loudest on the right side.
- Bounding arterial pulses.
- Other, less common findings—irregular pulses and dis-tention, ventricular tachycardia (unifocal), and congestive heart failure.

CAUSES
- A congenital aneurysm in the wall of the aortic root, usually in the right sinus of Valsalva, predisposes to aortic root rupture.
- Necrosis and degeneration of the aortic media have been associated, especially in old breeding stallions.
- Absent parasite migration in the ascending aorta is unlikely.

RISK FACTORS
- Aortic anomalous.

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS
Ventricular Septal Defect with Aortic Regurgitation
- Murmurs are systolic (band shaped and pansystolic) and diastolic (holodiastolic and diastolic decrescendo), not continuous.
- Arterial pulses usually are not bounding, unless the associated aortic regurgitation is severe.
- No history of acute colic or distress
- No unifocal ventricular tachycardia
- Differentiate echocardiographically.

Patent Ductus Arteriosus
- No history of acute colic or distress
- No unifocal ventricular tachycardia
- Differentiate echocardiographically.

CVC/BIOCHEMISTRY/URINALYSIS
Elevated serum creatinine, BUN can be elevated with sustained ventricular tachycardia and blood loss.

OTHER LABORATORY TESTS
Serum cardiac troponin I and cardiac isoenzymes of creatine phosphokinase and lactate dehydrogenase can be elevated with significant myocardial cell injury.

IMAGING
ECG
- Uniform ventricular tachycardia with a heart rate of >100 bpm may be present.

Echocardiography
- Two-dimensional echocardiography is diagnostic for a defect in the aortic root at the sinus of Valsalva or for a sinus of Valsalva aneurysm.
- The rupture may be a small, irregular defect in the aortic wall (usually associated with the right aortic leaflet) or be visualized flaking in the right atrium or ventricle.
- Anechoic to echoic fluid may be detected dissecting subendocardially into the interventricular septum, most frequently along the right ventricular side; however, dissection of blood subendocardially along the left side also occurs.
- Right atrial or ventricular enlargement if the aorta has ruptured into one of these chambers.
- Paradoxical arial motion with severe right ventricular volume overload.
- Ruptured tricuspid chordae tendineae or ruptured or flail tricuspid valve leaflet may be detected, particularly with rupture of an aneurysm of the sinus of Valsalva.
- Subendocardial dissection of blood along the left side of the interventricular septum may result in rupture into the left ventricle and left ventricular volume overload.
- Hyperdynamic interventricular septum and left ventricular free wall are associated with left ventricular volume overload, producing increased fractional shortening, until the myocardium starts to fail.
- Rupture of a mitral valve chordal tendineae and a flail mitral valve leaflet may occur, producing acute onset of severe mitral regurgitation.
- Significant left ventricular volume overload can lead to dilatation of the mitral annulus and mitral regurgitation.
- Use color-flow Doppler, pulsed-wave Doppler, or contrast echocardiography to localize the shunt associated with the aortic cardiac fistula.
- Continuous-wave Doppler can be used to determine peak velocity of the shunt.

Thoracic Radiography
- An enlarged cardiac silhouette should be present in horses with a large aortieocardiac shunt.
- Pulmonary overcirculation and edema may be detected.

DIAGNOSTIC PROCEDURES
Cardiac Catheterization
- Elevated right ventricular pressure, pulmonary arterial pressure, pulmonary capillary wedge pressure, and oxygen saturation of the blood are detected in horses with aortieocardiac fistula into the right ventricle.
- With a shunt into the right atrium, right atrial pressures and oxygen saturation also are elevated.
Arterial Blood Pressure
Demonstrates the wide difference between peak systolic pressure and end-diastolic pressure associated with continuous shunting of blood from the aorta into the heart.

PATHOLOGIC FINDINGS
- Post-mortem examination confirms the site and extent of the rupture and the presence of aortic root dilatation.
- Path of the dissection can be traced and the rupture into the right atrium, tricuspid valve, right ventricle, or left ventricle confirmed.
- Dissecting tracts into the interventricular septum usually are lined with immature and mature fibrous tissue, and disruption of the conduction system has been detected.
- Degeneration and necrosis of the aortic media have been reported in some horses with aortic root rupture but not in other affected horses.
- An absence of media in the right sinus of Valsalva was reported in one horse with a sinus of Valsalva (i.e., aortic root) aneurysm.
- Fibrosis and scarring of the rupture site have been reported in old breeding stallions that died of unrelated causes.
- Biatrial and biventricular enlargement usually is detected, and hepatic congestion and pulmonary edema may be present.

AIMS OF TREATMENT
Palliative care

APPROPRIATE HEALTH CARE
- Closely monitor affected horses with ventricular tachycardia if the tachycardia is uniform, the heart rate is > 120 bpm, no R-on-T complexes are detected, and no clinical signs of cardiovascular collapse are observed.
- If ventricular tachycardia is multifocal, R-on-T complexes are detected, heart rate is > 120 bpm, or with clinical signs of cardiovascular collapse, institute antiarrhythmic treatment on an inpatient basis.
- If congestive heart failure also is present, institute treatment for congestive heart failure as well. Consider humane destruction, however, because the horse is no longer safe to use for athletic work.

NURSING CARE
- Perform continuous ECG monitoring during the attempted conversion from ventricular tachycardia to sinus rhythm.
- Keep horses quiet and unmoving during antiarrhythmic treatment.

ACTIVITY
- Stall confinement until conversion to sinus rhythm has been successfully achieved.
- Restrict athletic activity as much as possible once ventricular tachycardia has been converted.

CLIENT EDUCATION
- Affected horses are not safe to ride or use for any type of athletic work because of the risk of sudden death associated with further aortic rupture or development of lethal ventricular arrhythmias.
- If the horse is a breeding stallion and such continued use is desired, warn the stallion and mare handlers (and all other personnel involved) about the risk of sudden death.
- Develop an emergency plan in the event the stallion becomes unsteady or unsafe to handle.

SURGICAL CONSIDERATIONS
N/A

TREATMENT

AORTIC ROOT RUPTURE

MEDICATIONS

DRUG(S) OF CHOICE
Antiarrhythmics
- Indicated with multifocal ventricular tachycardia, R-on-T complexes, heart rate > 120 bpm, or clinical signs of cardiovascular collapse.
- Drug selection depends on severity of ventricular tachycardia and associated clinical signs.
- IV lidocaine is rapidly acting and has a very short duration of action. However, it also has CNS effects in horses and, thus, must be used carefully.
- IV procainamide and quinidine gluconate have been effective in converting sustained, uniform ventricular tachycardia but have a slower onset of action.
- IV magnesium sulfate has been successful in converting sustained ventricular tachycardia and is not arrhythmogenic.

ACE Inhibitors
- May be indicated in stallions to decrease resistance to forward flow once ventricular tachycardia has been converted.
- Enalapril (0.5mg/kg PO BID) has no effect on the stallion’s libido, breeding performance, or fertility.
- Other vasodilators or antihypertensive drugs can be considered, but their effect on breeding stallions is unknown.

CONTRAINDICATIONS
Other vasodilators or antihypertensive drugs have the potential to adversely affect the stallion’s libido, breeding performance, or fertility.

PRECAUTIONS
Affected horses could experience sudden death at any time; thus, everyone working around these horses must be aware of the safety issues involved.

POSSIBLE INTERACTIONS
Any antiarrhythmic drug has the potential to cause development of a more adverse arrhythmia as well as to convert to sinus rhythm.

ALTERNATIVE DRUGS
Propranolol
- The IV form is less likely to be effective but should be considered in affected horses with refractory ventricular tachycardia.
- Lowers systolic blood pressure.

FOLLOW-UP

PATIENT MONITORING
- Routine monitoring of heart rate and of respiratory rate and rhythm after conversion to sinus rhythm.
- Persistent tachypnea, tachycardia, or new arrhythmias indicate deterioration in clinical status.
Aortic Root Rupture

• Return of venous distention and jugular pulsations or development of ventral edema or coughing indicates the onset of congestive heart failure and worsening of ventricular volume overload.

PREVENTION/AVOIDANCE
• With congenital aneurysms of the sinus of Valsalva, control of systemic blood pressure may prolong the time until rupture occurs.
• With degenerative changes in the aortic media, antihypertensive drugs theoretically should have some benefit. However, identification of horses at risk has not yet been accomplished.
• Routine echocardiography of old breeding stallions and high-performance horses potentially at risk may help to identify these horses before development of a tear in the aortic root.

POSSIBLE COMPlications
• Deterioration of uniform ventricular tachycardia into fatal ventricular arrhythmia
• Severe, acute congestive heart failure from massive right atrial or ventricular, left atrial, and left ventricular volume overload
• Tricuspid valve rupture, leading to massive tricuspid regurgitation and congestive heart failure
• Rupture of a chordate tendineae of the tricuspid or mitral valve, leading to massive tricuspid or mitral regurgitation, respectively, and acute, right- or left-sided congestive heart failure
• Sudden death

EXPECTED COURSE AND PROGNOSIS
• Prognosis for life of affected horses is grave, with sudden death expected in those with extracardiac or intrapericardial rupture.
• Onset of congestive heart failure is likely after development of an intracardiac fistula, and the speed of its development depends on the location and size of the shunt.

MISCELLANEOUS

ASSOCIATED CONDITIONS
Aortic root aneurysm

AGE-RELATED FACTORS
Old horses are more likely to be affected, but horses as young as 4 years have been diagnosed.

PREGNANCY
• Rupture of a sinus of Valsalva aneurysm has been seen in one late-gestation pregnant mare. The volume expansion of late pregnancy may predispose pregnant mares to aortic rupture at this time.
• Aortic root rupture has been seen in one mare during early pregnancy. This mare experienced acute onset of ventricular tachycardia and subendocardial dissection of blood into the interventricular septum but survived to have the foal.

SYNONYMS
• Aortic cardiac fistula
• Aortocardiac fistula

SEE ALSO
• Ventricular tachycardia

ABBREVIATIONS
• CNS = central nervous system

Suggested Reading

Author Virginia B. Reef
Consulting Editor Celia M. Marr


ArSENIC TOXICOSIS

BASICS

OVERVIEW

- Repeated exposure to arsenic-containing pesticides, arsenic-contaminated soils, burn piles, and water or feed
- Toxicity depends on the form of arsenic ingested.
- Tertiary inorganic forms (e.g., arsenic trichloride, sodium, potassium, and calcium salts of arsenate) are 10-fold more toxic than inorganic pentavalent forms (e.g., sodium, potassium, and calcium salts of arsenate).
- Toxicity of organic pentavalent forms used as growth promoter in swine (e.g., arsanilic acid, arsine oxide) has not been determined for horses.
- Tertiary inorganic arsenicals inhibit cellular respiration and damage capillaries.

SIGNALMENT

No breed or sex predilections

SIGNS

- Perspiration or acute syndromes are most likely
- Pause—patient often found dead; death caused by cardiovascular collapse
- Acute—intercostal abdominal pain, hypereosinophilia, severe watery diarrhea, decreased diaphragmatic sounds, muscle tremors, weak and rapid pulse with signs of circulatory shock, ataxia, depression, and recumbency; if the animal survives for several days, edema, and proteinuria secondary to renal damage
- Chronic—not described in horses

CAUSES AND RISK FACTORS

Ingestion of arsenic-containing products or arsenic-contaminated soils, water, or feed

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS

- Lead toxicosis—evidence of neurologic dysfunction is likely.
- Mercury toxicosis
- NSAID toxicosis—history of previous use
- Carbamidin toxicosis—evidence of cystitis
- Salmonellosis
- Colitis X
- Acute cyanobacteriosis
- Clostridial colitis

CBC/BIOCHEMISTRY/URINALYSIS

- Reflect circulatory shock and possible liver and kidney damage
- Hemocconcentration—elevated PCV and plasma total protein
- Leukopenia with degenerative changes in PMNs
- Anemia
- Electrolytes—hyperkalemia, hypoproteinemia, hypochloremia
- Hyperglycemia
- Hyperlipidemia
- Elevated LDH and CK

OTHER LABORATORY TESTS

- Ante-mortem—measurement of arsenic in urine, whole blood, or GI contents
- Post-mortem—measurement of arsenic in liver or kidney
- Chronic exposures—arsenic can be measured in hair
- Arsenic is rapidly excreted after exposure

IMAGING

N/A

DIAGNOSTIC PROCEDURES

N/A

PATHOLOGICAL FINDINGS

Gross
- GI hemorrhage, mucosal congestion, edema, and erosion are either localized or throughout the GI tract, which may be filled with watery, dark-green, black, or hemorrhagic ingesta, with necrotic material from mucosal sloughing
- Post-mortem—edema and epicardial and serosal hemorrhage

Histopathologic
- Necrotizing, hemorrhagic thyphlocolitis, with necrotizing vasculitis, renal tubular necrosis, and hepatic fatty degeneration

TREATMENT

- Urge treatment is necessary
- Remove animal from known or potential source of exposure
- GI decontamination
- Treat circulatory shock and acidosis
- Appropriate fluid therapy

MEDICATIONS

DRUG(S)

- Butorphanol (0.02–0.03 mg/kg IV, followed by 2–3 mg/kg q4h for 24 hr and then 1 mg/kg q6h for 2 days)
- Adverse reactions include tremors,

CONTRAINDICATIONS/POSSIBLE INTERACTIONS

- Use NSAIDs cautiously because of possible adverse GI and renal effects.

FOLLOW-UP

- Monitor renal and hepatic function
- Provide a bland diet, containing reduced amounts of high-quality protein
- Identify source of exposure, and properly dispose of source
- Expected course and prognosis depend on the severity of clinical signs
- If the animal survives, recovery should be complete

MISCELLANEOUS

ASSOCIATED CONDITIONS, AGE-RELATED FACTORS, ZOONOtic, POTENTIAL, PREGNANCY

N/A

ABBREVIATIONS

- CCA = chromated copper arsenate
- CK = creatine kinase
- DMSSA = 2,3-dimercapto-1,1 dimethyl xanthine
- GI = gastrointestinal
- LDH = lactate dehydrogenase
- PCV = packed cell volume
- PMN = polymorphonuclear leukocyte

Suggested Reading


Author

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**Artificial Insemination**

**BASICS**

**DEFINITION/OVERVIEW**
- Extended fresh, cooled, frozen semen introduced into the mare's uterus using aseptic technique
- Standard AI—minimum 300–1000 × 10^6 PMS deposited into uterine body
- DHF or low-dose AI—1–25 × 10^6 PMS deposited into 1/2 of uterine horn (ipsilateral to dominant follicle)

**ETIOLOGY/PATHOPHYSIOLOGY**
- AI increases live cover.
- Efficient use of semen.
- Ejaculate divided—several AI doses, greater number of mares bred in a season (120 by AI; 40–80 by live cover).
- Wider use of genetically superior stallions.
- Antibiotics in semen extenders prevent many genital infections.
- Fewer breeding injuries.
- Continue using stallions with problems (musculoskeletal and behavioral).
- Protect mares with genital tract impairments or recent surgical repair from further breeding-related trauma.
- Low-dose AI—stallions with limited availability or costly semen due to:
  - Excessive size of book
  - Low sperm cell production or high percentage of dead sperm
  - Use of sex-sorted sperm
  - Epididymal spermatozoa collected at the time of castration or stallion's death

**SYSTEM AFFECTED**
- Reproductive system

**SIGNALMENT**
- Thoroughbreds allow only live cover.
- All other breed registries allow AI, may impose restrictions

**DIAGNOSIS**

**PROCEDURAL ISSUES**

**Timing and Frequency of Breeding**
- Depends on semen longevity—affected by stallion idiosyncrasy, semen preservation method (fresh, cooled, frozen)
- Equine ova—short viability, 8–18 hr postovulation

**Teasing and Examinations**
- GnRH analog or hCG when preovulatory follicle is ≥35 mm to induce ovulation within 36–42 hr, under semen—overnight shipment
- Inseminate within 48 hr preovulation to achieve acceptable pregnancy rates.

**Frozen Thawed Semen**
- Precise timing of AI post-thaw longevity is reduced to 12–24 hr.
- Mare management—serial, daily teasing, TRP, and U/S.
- Deslorelin or hCG when dominant follicle ≥35 mm
- TRP and U/S—TID–QID, ensure AI as close before ovulation as possible; most important, ≤6 hr postovulation
- New frozen semen strategy for AI if have multiple doses:
  - Deslorelin or hCG when dominant follicle ≥35 mm
  - AI at 24 hr and again at 40 hr after injection; ensures viable sperm are available during ovulatory period
- Freeze mare if intrauterine fluid is present 4–6 hr after first AI.
- Pregnancy rate is equivalent to a one-time AI 6 hr postovulation, but minus the intensive labor and fewer veterinary examinations.
Low-Dose Insemination
- Allows use of a reduced dose of semen (fresh, cooled, frozen)
- Varies with semen quality
  - DHI dose has been decreased to as low as 14 x 10^6 motile, frozen-thawed sperm.
  - Average of 60–150 x 10^6 of PMS for DHI.
- Semen is deposited at the UTJ, tip of the uterine horn ipsilateral to dominant follicle.
- DHI can be either hysteroscopically guided or transrectally guided (with or without U/S).
- Mare management varies according to method of semen preservation.

General Comments
- If ovulation has not occurred within the recommended times for fresh (48–72 hr), cooled (24 hr), or frozen (6–12 hr) semen, rebreed is recommended.
- Stallion’s Disease Status
  - Should be negative for EIA, EVA, CEM, and PPHV.

Semen Analysis
- Minimum parameters—volume, motility, concentration
- Morphology—optional, but of particular use in ruminants
- Small sample of cooled or frozen semen should be saved and warmed (at 37°C) to evaluate immediately after AI.
- Slide, coverslip, and pipet—prewarm, stallion hands.
- Lubricant applied to dorsum of the gloved hand.
- Sterile and disposable equipment. Mares are restrained and the perineal area is thoroughly cleansed with a mild detergent, antiseptic solution or soap; then completely remove any lubricant.
- Presence of uterine fluid, then LRS uterine lavage immediately before AI.

Mare Selection
- Her fertility takes on special significance if using frozen semen or its quality is less optimal.
- Include reproductive history (+/- normal estrous cyclicity, results of uterine culture and cytology, presence of intrauterine fluid during estrus).
- Fertility alters by status—normal maiden > normal pluriparous > older maiden, pluriparous or barren mare

Prebreeding Uterine Culture and Cytology of Mare
- All, except young maiden mares, should have at least one negative uterine culture and cytology prebreeding.
- Avoid transmitting infections to the stallion.
- Early identification of possible mare problems
- Maximize the likelihood of first-cycle conception.
- Pregnancy rates lower and EED higher if ovulation has not occurred within the recommended times.

Prebreeding
- Mare management varies according to status—normal maiden, older maiden, pluriparous, or barren mare.
- Mare’s fertility takes on special significance if using frozen semen and its quality is less optimal.
- Stallion selection.

Frozen Thawed Semen
- A 0.5-mL straw contains 200–800 x 10^6 sperm/mL.
- A 5-mL straw contains 600–1000 x 10^6 sperm/mL.

Cooled Transported Semen
- A semen-to-extender ratio of 1:3 or 1:4 is acceptable; may be as high as 1:9.

Prebreeding
- Mare selection.
- Stallion selection.
- Presence of at least one negative uterine culture and cytology prebreeding.
- Avoid transmitting infections to the stallion.

TREATMENT

Prebreeding
- Presence of ≥2-cm height of prebreeding uterine fluid, then LRS uterine lavage immediately before AI.
- Does not affect fertility

AI Technique
- Sterile and disposable equipment.
- Mesometrium and peritoneal area thoroughly cleansed with a mild detergent, antiseptic solution or soap; then completely remove any lubricant (minimum three times).
- Sterile sleeve on arm and nonspermicidal lubricant applied to dorsum of the gloved hand.
- 250–56 cm (20–22 inch) AI pipet is carried in the gloved hand.

Fresh Extended Semen
- Perform AI immediately after collection.
- Semen can be mixed with an appropriate extender for immediate insemination, with semen-to-extender ratio of 1:1 or 1:2, if the ejaculate volume is small and of high concentration.

Stallion’s Disease Status
- Should be negative for EIA, EVA, CEM, and sepsis-related diseases.

Frozen Thawed Semen
- Frozen semen is packed in 0.5–5 mL straws and stored in liquid N2.
- Thawing protocols vary and are reported ideally to be paired with a particular freezing method. Seek specific information regarding thawing.

Equine, Second Edition
ARTIFICIAL INSEMINATION

If details are not provided with frozen semen received, seek instructions regarding thawing before the day of AI to ensure proper handling.

Post-shaving, semen should be in the mare within 5 min and avoid inducing uterine trauma.

Post-AI uterine treatment is strongly recommended. The high concentration of sperm cells in a thawed straw and absence of seminal plasma (provides a natural protective effect in the uterus) may induce an acute endometritis.

Contraceptive drugs may be used to treat PMIE—endometritis indicated by a shortened cycle due to endogenous prostaglandin release.

U/S examination 4–6 hr after AI for pregnancy.

Serial TRP pregnancy examinations—45, 60, 90, and 120 days.

Follow-up TRP and U/S—24–30 days; confirm heartbeat in the embryo.

Follow-up pregnancy examinations—45, 60, 90, and 120 days.

Possible complications:
- AV preparation, handling, maintenance
- Sperm evaluation at collection—ship semen to the appropriate location
- Shipping methods—Equateiner, reusable box cooling containers, vapor tank
-_operator skill—to manipulate and place semen through the cervix, into the uterine lumen or to the tip of the horn, in a proper and timely manner
- Menstrual cycle is at the mercy of airlines/couriers.

MISCELLANEOUS

Pregnant

Cooled semen

Per cycle pregnancy rates are equivalent to on-farm AI with fresh semen (60%–75%) if semen quality remains good after cooling period of 24 hr at 5–6°C.

Frozen semen

- Pregnancy rates decrease for most stallions.
- Spermatozoa suffer many stresses; anticipate attrition rate of ≥50% with freezing and thawing.

First-cycle pregnancy rates—30%–40% (range—6%–70%); wide range between stallions

Intense breeding management and good quality of semen—positive impact on the pregnancy rate.

Follow-up

Patient monitoring:
- Begin teasing by 11 days post-ovulation.

If present, lavage uterus with sterile saline—introduction of an endoscope into the mare’s uterus:
- Approach and visualize the UTJ ipsilateral to the dominant follicle.
- Small catheter is passed through the endoscope’s channel and semen deposited at/on the UTJ.

- DHI—Pass a flexible Al pipet through the cervix toward the tip of the uterine horn ipsilateral to the dominant follicle:
  - Pipet is guided by either TRP or U/S.
  - Semen is deposited to or onto the UTJ.

- Manual TRP elevation of the tip of the uterine horn may help pass the pipet.

Post-breeding

- U/S examination 4–6 hr after AI for presence of intrauterine fluid.

- If present, lavage uterus with sterile saline or LRS, followed by oxytocin beginning 4–6 hr after AI.

- Repeat oxytocin at 2-hr intervals until 8–10 hr post-AI, and again at 12–24 hr until the inflammation resolves.

Drugs (Choice)

- Ovulation induction most effective if follicle is ≥35 mm
- Within 24–42 hr with hCG (1500–3000 IU IV), response range is 12–72 hr.
- Within 36–42 hr with GnRH analogue (Deslorelin 1.5 mg IM)
- Exsudate drugs may be used to treat PMIE and DUC.
- Prostaglandins—Misoprostol for cervical relaxation or intrauterine PGF2α (0.25 mg)

2 hr before deep AI (only with good-quality semen)

CONTRAINDICATIONS
- See Endometritis.

PRECAUTIONS
- See Endometritis.

Follow-up

Patient monitoring:
- Begin teasing by 11 days post-ovulation.

Early detection of endometritis—indicated by a shortened cycle due to endogenous prostaglandin release.

Follow-up TRP and U/S—24–30 days; confirm heartbeat in the embryo.

Possible complications:
- AV preparation, handling, maintenance
- Sperm evaluation at collection—ship semen to the appropriate location
- Shipping methods—Equateiner, reusable box cooling containers, vapor tank
- Operator skill—to manipulate and place semen through the cervix, into the uterine lumen or to the tip of the horn, in a proper and timely manner
- Menstrual cycle is at the mercy of airlines/couriers.

Follow-up pregnancy examinations—45, 60, 90, and 120 days.

Possible complications:
- AV preparation, handling, maintenance
- Sperm evaluation at collection—ship semen to the appropriate location
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Follow-up pregnancy examinations—45, 60, 90, and 120 days.
**ARYТЕNІD CHONDROPATY**

**BASICS**

**OVERVIEW**
- A spas tic inflammatory process of one or both arytenoid cartilages, resulting in deformation with stiffness.
- This interference with the mobility of the affected arytenoid cartilage to fully abduct during forced inspiration and to seal off its lumen during expiration.

**SIGNALMENT**
- Male and Thoroughbred racehorses are more commonly affected.
- Incidence increases with age.

**SIGNS**
- Upper respiratory noise, exercise intolerance, or both.
- The disease usually worsens gradually, with progressive involvement of one or both arytenoid cartilages.
- The condition leads to valvular incompetence proportionate to the loss of abductory function and the mechanical size of the affected arytenoid cartilage. The more intense the high-intensity exercise occurs, the more severe the dyspnea, as the horse does not “breathe” or breathe well.
- In show horses, loss of points during competition because of upper respiratory noise may be the main concern; this upper airway noise resembles that of horses with laryngeal hemiplegia.

**CAUSES AND RISK FACTORS**
- Physical trauma to the mucosa of the arytenoid cartilage.
- Upper respiratory noise, exercise intolerance, or both. The disease usually worsens gradually, with progressive involvement of one or both arytenoid cartilages.
- The condition leads to valvular incompetence proportionate to the loss of abductory function and the mechanical size of the affected arytenoid cartilage. The more intense the high-intensity exercise occurs, the more severe the dyspnea, as the horse does not “breathe” or breathe well.
- In show horses, loss of points during competition because of upper respiratory noise may be the main concern; this upper airway noise resembles that of horses with laryngeal hemiplegia.
- Upper airway infection leading to cartilage sepsis.
- In many cases, the inciting cause is never found.

**DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS**
- Laryngeal hemiplegia.
- Congenital malformation of the laryngeal cartilages.

**CBC/BIOCHEMISTRY/URINALYSIS**
- Of no value.

**OTHER LABORATORY TESTS**
- Arterial blood gases during exercise.
- Hyperventilation can be evaluated using arterial blood gases—typically at maximal exercise
- 
- 

**IMAGING**
- Lateral radiography of the larynx may reveal enlarged laryngeal cartilages, sometimes with associated osteoarthritic changes.
- Ultrasound examination of the larynx using the mid-ventral and caudal ventral windows to evaluate for abscesses and caudal laryngeal window to assess the presence of disease in the lateral aspect of the arytenoid cartilage.

**OTHER DIAGNOSTIC PROCEDURES**
- The diagnosis is established on the basis of videoendoscopic examination at rest:
  - The body of the arytenoid is irregular and thickened.
  - A mass of granulation tissue may protrude from the axial surface of the arytenoid cartilage into the airway. The size or location of the protruding mass has no correlation with the amount of abduction remaining.
- The corniculate process may be deformed.
- Contact (i.e., “kissing”) lesions may be observed on the contralateral arytenoid cartilage.
- Eventually, the condition leads to decreased or total inability of the affected arytenoid cartilage to abduct during inspiration.

**TREATMENT**
- Medical treatment is indicated only in acute cases with mucosal ulceration and swellings.
- Consider laser-assisted excision of intralaryngeal granulations if the affected arytenoid cartilage retains abductive function.
- Partial arytenoidectomy (excision of the body and corniculate process of affected arytenoid cartilage) is the treatment of choice to restore exercise capacity and to reduce upper airway noise.
- Permanent tracheotomy can be used in countries where athletic competition is allowed with this procedure and to salvage the animal for breeding purposes.

**MEDICATIONS**

**DRUG(S) OF CHOICE**
- Acute case: broad-spectrum antibiotics and NSAI Ds.
- Chronic case: none, other than routine parenteral antimicrobial and anti-inflammatory agents.
- Use of nasopharyngeal spray, consisting of various anti-inflammatory and antimicrobial agents (e.g., 0.25% lidocaine, 500 mL of nitrofurazone, and 50 mL of prednisolone [25 mg/mL] mixed with 250 mL of glycerin) can be applied (20 mL BID) using a soft rubber feeding tube.
- If the airway is significantly compromised, a temporary tracheostomy may be needed until the swelling resolves.

**FOLLOW-UP**

**PATIENT MONITORING**
- Videoendoscopy of the upper airway 6 weeks after surgery to monitor patient response.
- Final response to treatment or continuation of monitoring of affected horses is made on the basis of evaluating exercise tolerance and upper respiratory noise.
- Laser resection of the unsupported ipsilateral aryepiglottic fold might be needed to improve airway patency.

**POSSIBLE COMPLICATIONS**
- Horses undergoing removal of the corniculate and body of the arytenoid cartilage have a slightly increased risk for tracheal aspiration of feed during deglutition. In addition, these procedures do not fully restore the airway diameter, so a mild degree of airway obstruction persists, which may interfere with performance or result in upper airway noise during exercise.
- Bilateral arytenoidectomy increases the risk for tracheal aspiration of feed during deglutition and for glottic stenosis because of webbing at the resection site.

**EXPECTED COURSE AND PROGNOSIS**
- Horses with acute swelling of the arytenoid cartilage may respond favorably toNSAI Ds, topical anti-inflammatory agents, and antibiotics.
- Untreated horses exhibit a progressive increase in exercise intolerance and upper respiratory noise.
- Horses with focal elevated granulations on the axial surface of the arytenoid cartilage that maintain abductory function may respond to simple "lumpectomy." Horses with generalized involvement of an arytenoid cartilage and without surgical treatment often develop contralateral contact or "kissing" lesions.
- Horses with unilateral lesions treated surgically have a fair prognosis (60%) for elimination or significant reduction of exercise intolerance; however, the prognosis is guarded (20%) in horses with bilateral lesions.

**MISCELLANEOUS**

**SEE ALSO**
- Dynamic collapse of the upper airways.
- Laryngeal hemiplegia/hemiple gia.

**ABBREVIATION**
- DM/SO = dimethylsulfoxide.
- DMSO = dimethylsulfoxide.

**Suggested Reading**
Ascarid Infestation

**BASICS**

**OVERVIEW**
- Parasitic roundworm infection caused by *Parascaris equorum*.
- The infection prevalence may be up to 100% in tested farms and up to 80% in foals, with the highest incidence occurring between 100 and 180 days of age.
- The parasite has a direct life cycle that follows the oral-fecal route. Adults, in the small intestine of infected horses, produce large numbers of eggs, which are passed in the feces. The eggs become infective in 10 days to 6 weeks by developing into larvae (L₂). These highly resistant eggs accumulate in the environment, sticking to different surfaces, including the mare’s mammary gland. When ingested, the larvae are released in the small intestine and migrate through the intestinal wall into the bloodstream, reaching the liver via the portal circulation. In the liver, they migrate to a hepatic vein, accessing the caudal vena cava and finally the pulmonary circulation. Molting of the larvae occurs in the lungs, followed by tracheal ascending migration and subsequent deglutition. Arrival to the small intestine completes the life cycle, and a final molting and maturation into the adult form take place.

**SYSTEMS AFFECTED**
- It affects primarily the GI system, causing enteritis, maldigestion, and malabsorption.
- The hepatobiliary and respiratory systems—An exaggerated inflammatory response to migrating larvae, resulting in temporary lung and liver damage in sensitized horses. Varying forms of tracheobronchitis have also been described.

**SIGNALMENT**
- Any ages, but primarily in foals and weanlings up to 9–12 mo of age
- Debilitated and immunocompromised adult horses can also be infected.

**SIGNS**
- Decreased growth rate, generalized weakness, a dull hair coat and dry skin, “pot-bellied” appearance, and decreased appetite
- In severe cases, colic due to obstruction can occur.
- Acute colic signs with peritonitis due to perforation of the intestine.
- Coughing and mucopurulent nasal discharge with or without systemic illness may be seen during periods of larval migration through the lungs.

**CAUSES AND RISK FACTORS**
The disease is caused by *P. equorum*, the roundworm from the family Ascarididae. Animals at risk are susceptible foals and weanlings grazing on infested pastures.

**DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS**
- Any causes of colic, ill-thrift, weakness, malabsorption, and malnutrition
- CBC/BIOCHEMISTRY/URINALYSIS
  - Eosinophilia may be seen during larval migration, 10–40 days postinfection.
  - Leukopenia and mild anemia have been reported.
  - In severe cases, hypoproteinemia can be detected.

**OTHER LABORATORY TESTS**
- Coprology for detection of the eggs (see Other Diagnostic Procedures)

**IMAGING**
- Adult ascaris may be seen on transabdominal ultrasound, within the intestinal lumen or in the peritoneal cavity after intestinal perforation.

**OTHER DIAGNOSTIC PROCEDURES**
- The infestation is confirmed by fecal flotation techniques.

**TREATMENT**
- Treatment is indicated for fecal egg counts greater than 100 eggs per gram. Following sudden and complete paralysis of all ascarids after anthelmintic therapy, small intestinal obstruction or impaction may occur.
- Emergency surgical intervention for removal of dead parasites and correction of secondary complications such as intussusceptions and intestinal volvulus are then required.
- Ascarid impaction should be suspected in colicky foals and weanlings with a recent history (24 hr) of deworming.

**MEDICATIONS**
- The regular use of anthelmintics is the treatment of choice for patent infections with *P. equorum* and should be administered to foals and weanlings every 6–8 weeks, starting at 1.5–2 mo of age.

**PATHOLOGIC FINDINGS**
- Adult forms are found in the intestinal lumen or free in the abdominal cavity following intestinal perforation.
- Hemorrhagic and edematous lesions around necrotic areas in the lungs, liver, and associated lymph nodes are seen during larval migration.
- Microscopy after larval migration reveals multiple foci of white tracts within a fibrotic liver parenchyma.
- Lymphocytic nodules may develop in the lungs after multiple episodes of reinfection in a sensitized host.
Broodmares should be treated at monthly intervals in the last trimester of pregnancy to reduce environmental contamination. Recommended anthelmintics:
- Fenbendazole 10 mg/kg PO given for 5 consecutive days (varies in effectiveness against ascarids)
- Pyrantel pamoate 6.6 mg/kg PO
- Levamisole 8 mg/kg PO
- Ivermectin 0.4 mg/kg PO and ivermectin 0.2 mg/kg PO were advocated to be 100% efficient in eliminating ascarid infection in horses, but resistance to these and other macrocyclic lactone anthelmintics has been identified in Europe, Canada, and the United States in recent years.

CONTRAINDICATIONS/POSSIBLE INTERACTIONS
If severe parasite burdens are suspected, anthelmintics that result in paralysis of the parasites (e.g., pyrantel pamoate, piperazine, organophosphates, ivermectin) should be avoided because it may result in small intestinal obstruction or impaction and can lead to intestinal rupture and peritonitis. Therefore, anthelmintics with a slower action such as benzimidazoles are recommended.

FOLLOW-UP
PATIENT MONITORING
Fecal floatation should be conducted in 10% or more of the foals every 4–6 mo. If 10% of foals or more are positive, failure of the anthelmintic therapy and/or the prevention and control strategies should be suspected.

PREVENTION/AVOIDANCE
- Contaminated facilities should be disinfected with a 5% phenolic compound and sprayed with a high-pressure hose.
- Grazing of broodmares, foals, and weanlings on heavily contaminated pastures should be avoided.
- *P. equorum* eggs can remain viable in the environment for many years.
- Frequent removal of manure from stalls and pastures also reduces transmission between foals and reinfection following treatment.

POSSIBLE COMPLICATIONS
Overdose of anthelmintic can result in toxicity.

EXPECTED COURSE AND PROGNOSIS
- Prognosis is favorable in uncomplicated cases, but a delay in growth and development is common.
- Infection rates start to decline at 6 mo of age, and immunity is long lasting. Patent infections are rarely seen in adults except in immunocompromised animals.

MISCELLANEOUS
ASSOCIATED CONDITIONS
- Gastrointestinal obstruction
- Septic peritonitis

ZOONOTIC POTENTIAL
Human infection, although extremely rare, may occur after ingestion of a viable egg.

PREGNANCY
Transplacental infection with *P. equorum* is not known to occur, nor is the transfer of ascarid larvae in colostrum.

Suggested Reading

Author Carlos Medina-Torres
Consulting Editors Henry Stämpfli and Olimpo Oliver-Espinosa
**Aspartate Aminotransferase (AST)**

**BASICS**

**DEFINITION**
- Catalyzes transamination of L-oxaloacetate and α-ketoglutarate.
- Present in many tissues—liver, striated muscle, myocardium, and others.
- Reported normal AST activity in horses varies from 48 to 455 IU/L.

**PATHOPHYSIOLOGY**
- Increases in AST activity are typically indicative of hepatic cellular and/or striated muscle injury; however, increased AST activity will occur with hemolysis because of the high AST content in erythrocytes.
- Magnitude of the elevation generally is proportional to the number of hepatocytes affected, not so to the severity of a particular insult.
- With skeletal muscle injury; magnitude of AST elevation is not necessarily proportional to the extent of tissue injury.
- Increases above the reference interval occur with intramuscular injections and in doxorubicin animals.
- AST is a sensitive indicator of hepatic cellular and striated muscle injury; however, because it is present in many tissues, AST lacks specificity.
- Other biochemical tests need to be examined concurrently with AST to localize the source of the increase (i.e., SD for liver and CK for muscle).
- After tissue injury AST activity increases more slowly and remains increased longer than SD or CK.
- Increased SD, with normal or increased AST, indicates acute or ongoing hepatocellular injury.
- If serial serum chemistry analyses reveal continuously or progressively increased activities of both enzymes, ongoing hepatocellular injury is likely. During treatment of hepatic disease, the extent of tissue injury.
- Clinical signs due to hepatic failure generally depend on the cause of the increases in AST.
- Causes Depressive conditions—cirrhosis, rhodanemia, and cholelithiasis
- Anomalous, congenital diseases—polycystic liver storage myopathy, biliary atresia
- Metabolic diseases—shock, hypervolemic, hyperkalemia, hyperuricemia, hyperuricemia, hypercalcemia, and vitamin E deficiency
- Infectious and immune-mediated diseases—hepatitis of various causes (e.g., viral, bacterial, protozoal, fungal, parasitic), serum sickness, amyloidosis, endotoxemia, and chronic active hepatitis
- Toxic or trauma—pyrrolidine alkylamide-containing plants, fenvalerate fumurate in newborn foals, tocolome, cancer bean, oak, and alkali clover; fungal toxins, such as aflatoxins, castor bean, oaks, and other similar compounds.
- Cholestasis and lipid disorders, and decreased in inflammation
- Albumin—decreased in end-stage liver disease from decreased production; minimally to mildly decreased in inflammation
- Globulin—generally increased in end-stage liver disease and with chronic antigenic stimulation
- SD—increased with acute and ongoing hepatic insufficiency
- ALF—increased with concurrent cholestatic disease
- GGT—increased with cholestatic disease or hyperlipoproteinemia
- CK—increased with acute or ongoing muscle injury
- Congestive biliary—increased in cholestatic disease
- Unconjugated bilirubin—increased with anemia and prehepatic cholestasis (i.e., massive in vitro hemolysis)
- Cholesterol—may be increased with cholestatics and lipid disorders, and decreased in hepatic insufficiency
- Tryglycerides—increases may be associated with hepatic lipidosis
- Because of high AST activity in erythrocytes, hemolysis falsely elevates serum/plasma AST activity.
- Prolonged in vitro exposure of serum or plasma to erythrocytes falsely increases AST activity even before visible signs of hemolysis are present.
- To avoid this confounding factor, prompt separation of plasma/serum from the cellular components of blood is strongly recommended.
- If laboratory analysis will not occur within 1–2 days, freeze the plasma/serum.

**DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS**
- CBC/BIOCHEMISTRY/URINALYSIS
  - Erythrocytes—liver disease may cause nonregenerative anemia and morphologic changes (e.g., acanthocytes, target cells, non-specific polychromasia, normochromic microcytosis in porcine systemic vascular shunts); severe anemia of any cause may produce cellular injury from tissue hypoxia.
  - Leukocytes—leukocytosis or leukopenia may be seen with inflammatory diseases and leukemia; morphologic changes of the leukocytes (e.g., neutrophilic toxicity in inflammation; neutrophilic cells) also may be seen.
  - Platelets—quantitative decreases and increases may be seen with a variety of systemic diseases that may affect the liver or striated muscle.

**OTHER LABORATORY TESTS**
- SBA
  - Sensitivity test for hepatic insufficiency, but not specific for the type of hepatic insufficiency.
  - May be increased with cell injury, cholestasis, or hepatic insufficiency/decreased functional mass, specificity for the latter condition is greatly
increased when SBA are increased in cases with normal or minimally increased markers for hepatocellular injury (e.g., SD, AST, GGT) and cholestasis (e.g., ALP, GGT, conjugated bilirubin).

- **Main advantage** over plasma ammonia, a more specific test for hepatic insufficiency/decreased functional mass is indicated if fasting or challenge ammonia concentration is increased.
- A sensitive and specific test, because it is not affected by other factors (e.g., cholestasis). However, ammonia measurement requires special handling, which limits its general availability.
- Consult reference laboratory for specific sample submission requirements.

- **Coagulation Tests and Fibrinogen**
  - The liver manufactures many of the coagulation factors; significant decreases in liver function may lead to deficiencies in these factors and to coagulation abnormalities.
  - APTT and PT—Decreased APTT and PT are seen when <30% of the activity of the factors is present.

- **Serologic Tests**
  - Helpful in detecting infectious causes
  - Toxicology
    - Analysis of tissue biopsy material, food, ingesta, serum/plasma, or other body fluids may indicate presence of a toxin.
    - Contact reference laboratory regarding sample selection and submission recommendations.
  - **Bacterial, Fungal, or Viral Culture**
    - May establish a definitive diagnosis regarding the infectious agent involved and help to guide treatment.
    - Request bacterial antibiotic sensitivity to determine appropriate antibiotic therapy.
    - Contact reference laboratory regarding sample selection and submission recommendations.

- **Sulfobromophthalein and Indocyanine Green Dye—Clearance Tests for Evaluation of Hepatic Function**
  - These tests have been replaced by plasma ammonia and SBA.

- **IMAGING**
  - Ultrasoundography for Liver Disease
    - Evaluate size, echogenicity, shape, and position.
    - Useful for guidance when obtaining biopsy material for cytology, histopathology, and microbiology.
    - Helpful in the evaluation of muscle and tendon injuries.
  - Other diagnostic imaging modalities such as radiographic imaging are expensive and available only at select institutions.

- **OTHER DIAGNOSTIC PROCEDURES**
  - Aspiration cytology and histopathology of formalin-fixed tissue (particularly liver)

### ASPARTATE AMINOTRANSFERASE (AST)

- **AIMS OF TREATMENT**
  - Depend on the primary disease process and secondary complications

- **APPROPRIATE HEALTH CARE**
  - Depends on the primary disease process and secondary complications

- **NURSING CARE**
  - Depends on the primary disease process and secondary complications

- **ACTIVITY**
  - Depends on the primary disease process and secondary complications

- **DIET**
  - Depends on the primary disease process and secondary complications

- **SURGICAL CONSIDERATIONS**
  - Depends on the primary disease process and secondary complications

### MEDICATIONS

- **DRUG(S) OF CHOICE**
  - Depends on the primary disease process and secondary complications

### CONTRAINDICATIONS

- With suspected hepatic insufficiency, assess the relative safety/risk of performing invasive procedures (e.g., fine-needle aspiration, tissue biopsy, laparoscopy, surgery) in light of the coagulation panel results.

### PRECAUTIONS

- Depends on the primary disease process and secondary complications

### POSSIBLE INTERACTIONS

- Depends on the primary disease process and secondary complications

### ALTERNATIVE DRUGS

- Depends on the primary disease process and secondary complications

### FOLLOW-UP

- **PATIENT MONITORING**
  - Serial serum biochemical analysis to monitor progression or improvement of the disease process (see Pathophysiology)

- **PREVENTION/AVOIDANCE**
  - Depends on the primary disease process and secondary complications

### POSSIBLE COMPLICATIONS

- Depends on the primary disease process and secondary complications

### MISCELLANEOUS

- **ASSOCIATED CONDITIONS**
  - Depend on the primary disease process and secondary complications

- **AGE-RELATED FACTORS**
  - Depend on the primary disease process and secondary complications

- **ZOONOTIC POTENTIAL**
  - Infectious diseases such as salmonellosis

- **PREGNANCY**
  - See Signalment.

- **SYNONYMS**
  - Previously known as glutamate oxaloacetate transaminase (SGOT)

- **SEE ALSO**
  - See Causes.

### ABBREVIATIONS

- G1 = gastrointestinal
- ID = idiel dehydrogenase
- SBA = serum bile acid

### Suggested Reading

ASPIRATION PNEUMONIA

BASICS

OVERVIEW
- May develop after inhalation of foreign material and bacteria into the lower respiratory tract.
- Causes include:
  - Esophageal disorders.
  - GI reflux and aspiration.
- Clinical signs:
  - Respiratory distress (tachypnea, dyspnea).
  - Sputum production.
  - Fever.

SIGNS
- Physical Examination Findings:
  - Acute:
    - Tachypnea, dyspnea.
    - Tachycardia.
  - Chronic:
    - Coughing.
    - Decreased appetite.

SYMPTOMS
- Dyspnea.
- Tachypnea.
- Coughing.
- Nasal discharge.

CAUSES AND RISK FACTORS
- Dysphagia
- Esophageal disorders
- GI tract disorders
- Esophageal reflex

DIAGNOSIS
- CBC/Biochemistry/Urinalysis
- Thoracic radiography
- Bronchoscopy
- Endoscopy

TREATMENT
- Medical:
  - Analgesics
  - Antibiotics
  - Supportive care
- Surgical:
  - Esophageal transection

PROGNOSIS
- Depends on the underlying cause.

PREVENTION
- Avoid aspiration of foreign material.
- Proper management of GI disorders.

REFERENCES
An epidermal inclusion cyst of the false nostril (nasal diverticulum)

- Also called false nostril cyst
- Present at birth and becomes apparent with age as the cyst enlarges
- Usually a cosmetic issue only
- The term "atheroma" is a misnomer as it implies a sebaceous cyst.
- The false nostril cysts in horses are not sebaceous cysts.

**BASICS**

**OVERVIEW**
- An epidermal inclusion cyst of the false nostril (nasal diverticulum)
- Also called false nostril cyst
- Present at birth and becomes apparent with age as the cyst enlarges
- Usually a cosmetic issue only
- The term "atheroma" is a misnomer as it implies a sebaceous cyst.
- The false nostril cysts in horses are not sebaceous cysts.

**CAUSES AND RISK FACTORS**
- Congenitally aberrant epithelial tissue
- Can slowly enlarge due to progressive exfoliation of keratinized material within the cyst

**DIAGNOSIS**
- Characteristic location and physical features of the swelling

**DIFFERENTIAL DIAGNOSIS**
- An abscess can be ruled out as there is no heat or pain associated with the cyst.
- Cysts could become infected if keratinized material leaks into the surrounding tissue.

**IMAGING**
- Ultrasonographic findings consistent with cystic structure, usually unilocular, mostly homogeneous echogenicity

**OTHER DIAGNOSTIC PROCEDURES**
- Palpation
- Ultrasonographic evaluation
- Centesis
- Histological evaluation

**TREATMENT**
- Do nothing. Usually not removed unless for cosmetic reasons or for airway noise or impairment from large swelling size.
- If removed surgically, it is imperative to remove the entire cyst lining to prevent recurrence.
- Total surgical removal can be done under general anesthesia or standing with sedation and local anesthesia of the infraorbital nerve.
- The cyst can be approached surgically through the skin over the dorsum of the nasomaxillary notch.
- Another option is to open the cyst ventrally and local anesthesia of the infraorbital nerve.
- General anesthesia or standing with sedation and local anesthesia of the infraorbital nerve.
- The cyst then is dissected in its entirety, and the wound is closed.
- Transient swelling if chemical ablation is used.

**MEDICATIONS**
- Antibiotics as appropriate
- Draining and cauterizing or sclerosing the cyst has been done using tincture of iodine, silver nitrate, or both followed by packing;

**MEDICATIONS**
- Aspirated fluid is white to gray, milky to creamy in appearance and odorless.
- Keratinized and nonkeratinized squamous epithelial cells.
- T richrome staining reveals keratinized and nonkeratinized squamous epithelial cells and keratinous debris.
- Histologically, the cyst lining is comprised of varying thickness of stratified squamous epithelium.

**EXPECTED COURSE AND PROGNOSIS**
- Favorable prognosis for both leaving the atheroma untouched and for surgical removal if needed

**CONTRAINDICATIONS/POSSIBLE INTERACTIONS**
- Transient swelling if chemical ablation is used.

**ASSOCIATED CONDITIONS**
- In addition to false nostril cysts, other congenital cutaneous cysts reported in horses are dentigerous cysts and, very rarely, dermoid cysts.

**AGE-RELATED FACTORS**
- May increase in size with age

**ZOOLOGIC POTENTIAL**
- None

**PREGNANCY**
- None

**SEE ALSO**
- N/A

**SUGGESTED READING**

**CONSULTING EDITOR**
- Daniel Jean
Atopic Dermatitis

**BASICS**

**DEFINITION**
Chemically, inflammatory, pruritic skin disease resulting from a predisposition to develop IgE-mediated hypersensitivity to inhalant or commonly absorbed environmental allergens.

**PATHOPHYSIOLOGY**
The complex etiology of equine AD is unknown. Susceptible animal is sensitized to environmental allergens resulting in the production of allergen-specific IgE. Upon further exposure to preconceived absorbed or inhaled allergens, an immediate type I hypersensitivity ensues. The reaction commences by binding of allergen-specific IgE to FcεR1 receptors on mast cells ultimately causing degranulation and liberation of inflammatory mediators such as histamine, cytokines, chemokines, and proteolytic enzymes. The culmination of these inflammatory processes is pruritus and/or urticaria.

**SYSTEMS AFFECTED**
Skin • Respiratory

**GENETICS**
• Genetic predisposition and heritability of AD in horses are unknown. AD must have a genetic component due to the clinical observation that the disease appears more often within certain breeds. • One stallion with AD has five offspring with AD, each from a different mare—suggesting a dominant mode of inheritance.

**INCIDENCE/PREVALENCE**
True incidence is unknown; estimated at 2%–4% of the equine population; within the top 10 most common equine dermatoses, AD is the second most prevalent. One stallion with AD has five offspring with AD, each from a different mare—suggesting a dominant mode of inheritance. Recognized worldwide; local environmental factors (temperature, humidity, and flora) influence the seasonality, severity, and duration of signs.

**GEOGRAPHIC DISTRIBUTION**
Recognized worldwide; local environmental factors (temperature, humidity, and flora) influence the seasonality, severity, and duration of signs.

**SIGNAMENT**
Broad Predictors Arabians, Thoroughbreds, Quarter Horses, and Warmbloods have been reported to be predisposed.

**Mean Age and Range**
Mean 5–6.5 years of age (2–12 years); signs may be mild the first year and usually progress each year.

**Predominant Sex**
• Both sexes affected equally • In a recent small regional study, males ( geldings > stallions) were twice as likely as mares to develop AD

**SIGNS**

**General Comments**
• Difficult to diagnose • Predominantly seasonal and nonseasonal pruritus and/or urticaria (rubbing, itching, bright, pruritus) are exacerbated by cold, wind, and summer heat.
• Primary lesions are wheals representing an urticarial reaction and/or papules. • Secondary lesions reflect self-induced trauma from intense pruritus at the affected body site and consists of alopecia, excoriations, craters to ulcers, scale, lichenification, hyperpigmentation, and mane and tail loss. Lesions may be symmetrical.
• Urticaria in AD may be pruritic or nonpruritic.

**History**
• Most commonly affected sites include face, pinnae, chest, ventral thorax and abdomen, extremity extensor and flexor surfaces • Other common sites include the mane, dorsal rump, neck, and tail base. • Clinical sign may begin in any season and progress from seasonal to nonseasonal. • Symptoms become progressively more severe with time.

**Physical Examination**
• Clinical signs of anaphylactic or recurrent airway obstruction include head shaking, snorting, bilateral mucopurulent nasal discharge, conjunctivitis, dry unproductive cough, labored breathing, stomping, and face rubbing on front legs or objects and exercise intolerance.
• Uncommon clinical signs are head shaking and laminitis.

**CAUSES**
• Allergic pollens (trees, grasses, weeds) • Mold spores (indoors and outdoors) • Animal dander (mouse, cat, cow, poultry, goat) • Possibly storage and house dust mites

**RISK FACTORS**
• Temperate environments with long allergy seasons, high pollen and mold spore levels
• Genetic predisposition, diffuse atopic dermatitis (multiorgan involvement), and development of AD at 3–4 years of age
• Hypersensitivity to inhalant allergens, an immediate type I hypersensitivity in horses. The reaction is given a subjective score (usually 0–6+) based on its size and severity compared to the positive and negative controls. • Reactions are interpreted at 15–30 min for an immediate IgE-mediated type I hypersensitivity and 4 hr for the IgE-mediated late-phase reaction. • Normal horses have one or more positive intradermal reactions. • Interpret results in terms of the horse’s environment, clinical signs, and history to determine allergens that should be avoided and included in ASIT. • Before performing IDT, a withdrawal period of 14 days is observed for oral and topical anthelmintics as well as topical transdermal preparations and 30 days for parenteral corticosteroids. • Cytokine from eosinophils or mast cells shows a neutrophilic exudate with intra- and/or extracellular cocci representative of a secondary folliculitis. • Stress, skin scarrings to rule out ectoparasites. • Stress, bacterial and DTM cultures to determine bacterial species and susceptibility and/or dermatophyte infections.

**DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS**
Insect hypersensitivity—may occur concurrently with AD • Excoriations (pathogens and incidentals) • Cutaneous adenoma • For respiratory disease—respiratory infection (bacterial, fungal, viral), congestive heart failure, and bronchitis

**CBC/BIOCHEMISTRY/URINALYSIS**
Eosinophilia is rare.

**OTHER LABORATORY TESTS**
Serologic Allergy Tests • Detects relative levels of allergen-specific IgE in the serum • Controversy exists as to the usefulness of serum allergy tests in horses; the author does not recommend the use of these tests. • A positive result does not always correlate with clinical manifestation of allergy; therefore, results of these tests must be interpreted cautiously. • The effects of anthelmintics and corticosteroid administration on test results are unknown. • Many false-positive and negative results occur with the currently available assays.

**IMAGING**
N/A

**OTHER DIAGNOSTIC PROCEDURES**

**Intradermal testing**
• Detects levels of allergen-specific IgE in the skin directed to a panel of allergens that are region specific and thought to be clinically-relevant to the patient’s disease. • IDT is the gold standard test for allergen hypersensitivity identification in horses. • Performed for identification of allergens to include in ASIT, possible avoidance or decrease or exposure. • Intradermal injection of allergens results in raised, turgid wheals. The biotic is given a subjective score (usually 0–6+) based on its size and severity compared to the positive and negative controls. • Reactions are interpreted at 15–30 min for an immediate IgE-mediated type I hypersensitivity and 4 hr for the IgE-mediated late-phase reaction. • Normal horses have one or more positive intradermal reactions. • Interpret results in terms of the horse’s environment, clinical signs, and history to determine allergens that should be avoided and included in ASIT. • Before performing IDT, a withdrawal period of 14 days is observed for oral and topical anthelmintics as well as topical transdermal preparations and 30 days for parenteral corticosteroids. • Cytokine from eosinophils or mast cells shows a neutrophilic exudate with intra- and/or extracellular cocci representative of a secondary folliculitis. • Stress, skin scarring to rule out ectoparasites. • Stress, bacterial and DTM cultures to determine bacterial species and susceptibility and/or dermatophyte infections.

**PATHOLOGICAL FINDINGS**
• Skin biopsies—will not rule out other differential diagnosis such as insect or food hypersensitivity • Histopathological changes—epidermal hyperplasia with superficial and deep, perivascular to interstitial dermatitis wherein the epidermis is the predominant inflammatory cell. Concurrent focal eosinophilic infiltrate and/or necrotizing mural folliculitis and/or eosinophilic granulomas are possible.

**TREATMENT**

**AIMS OF TREATMENT**
Reduce pruritus and secondary infections.

**APPROPRIATE HEALTH CARE**
Outpatient medical management

**NURSING CARE**
Frequent bathing using cool water (antimicrobial shampoos, sulfathalidazole, acid- cough cold—oatmeal times or leave-in conditioners) helps to remove allergens, cranes, bacteria, and debris; control secondary infections; hydrate dry skin, and provide antipruritic effects.

**ACTIVITY**
Avoid offending allergens if possible by changing environment.

**DIET**
Essential fatty acid supplementation may be beneficial in some cases.
CLIENT EDUCATION
• Impart important to discuss the progressive nature of the disease • Advise disease is not curable, but rather manageable and life-long therapy may be needed • Advise that commitment to proper management of horses with AD can lead to a horse that has a good quality of life and can continue to work • Discuss that dermatological modifications over the life of the horse are to be expected • Due to the potential hereditary factor, owners should be advised to remove affected individual from breeding stock.

SURGICAL CONSIDERATIONS
NOA

MEDICATIONS

DRUG(s) OF CHOICE
Allergen-Specific Immunotherapy
• Subcutaneous administration of gradually increasing doses of causative allergens in an attempt to reduce sensitivity • Allergens for inclusion are based on correlation with history with positive intradermal tests and knowledge of local flora. A useful nonsteroidal long-term treatment alternative when signs last longer than 2 mo or when nonsteroidal forms of therapy are ineffective • Anticipated improvement may be seen as early as 2 mo; however, minimum treatment duration of 12–14 mo is necessary to determine efficacy • Reports indicate 50%–75% of horses with AD alone or in combination with other medications show improvement in clinical signs with ASIT.

Corticosteroids
• Best selection—prednisolone, greater bioavailability than prednisone, tablets or syrup (compoundd) at 0.5–1 mg/kg q24h until control achieved; then reduce to lowest-dose alternate-day regimen, for example, 0.2–0.5 mg/kg q48h. • For horses that do not respond to prednisolone, try dexamethasone powder or injectable. Initial loading oral or IV dose of 0.02–0.1 mg/kg q8h for 3–5 days; then taper to 0.01–0.02 mg/kg q48–72h for maintenance. • Repository injectable corticosteroids should be avoided as withdrawal upon an adverse reaction is not possible.

Antihistamines—A Nonsteroidal Alternative for Long-term Control
• Not useful when moderate to severe pruritus is present; rather use as a preventative either before the onset of severe pruritus or in a maintenance regimen to suppress pruritus once controlled.
• Pharmacokinetic data for the use of antihistamines in horses is limited. Anecdotal reports suggest that H1-receptor antagonist hydroxyzine hydrochloride/pamcaine (0.5–1 mg/kg q12h), chlorpheniramine (0.25 mg/kg q12h), dexamethasone (0.75–1.0 mg/kg q24h), or pyrimidine malate (1 mg/kg q12h) may decrease pruritus and provide a sedative-sparing effect. • Antihistamines should be given at least 10–14 days before efficacy is determined. If no response, select another class of antihistamine.

Tricyclic Antidepressants
Used to control hypersensitivity with a stress or psychogenic component. Horses may respond to desipramine HCl (0.5–0.75 mg/kg q12h PO) or amitriptyline (1–2 mg/kg q24h PO).

CONTRAINDICATIONS
• Due to the anticholinergic properties of antihistamines and tricylic antidepressants, do not use in patients with a history of cardiac arrhythmia, colic, glaucoma, or urinary retention disorders. Antihistamines may thicken mucus in the respiratory tract. Extra caution should be used in horses with respiratory problems due to excess mucus. • Avoid corticosteroid use during pregnancy and lactation unless the benefit outweighs the risks. Risks are likely low.

PRECAUTIONS
• Corticosteroids—Use judiciously to avoid iatrogenic hyperglycosemia, diabetes mellitus, polyuria and polydypsia, aggravation of bacterial folliculitis, decreased muscle mass, weight loss, poor wound healing, and behavior changes. • Antihistamines—can produce sedation and/or behavior changes, whole body or fine motor or seizures. High dose of antihistamines cause birth defects in laboratory animals. Antihistamines should only be used in pregnant or lactating animals if the benefits outweigh the risks. Do not administer antihistamines intravenously in the horse due to potential CNS stimulation. • Note drug withdrawal times and regulations pertaining to horse show or racing associations.

POSSIBLE INTERACTIONS
• If diuretics such as furosemide are given with corticosteroids, an increased risk of electrolyte imbalances due to calcium and potassium losses exists. • Prednisolone interacts with phenytoin, phenobarbital, rifampin, erythromycin and the anticholinesterase drugs, neostigmine and pyridostigmine. • Antihistamines have an additive effect when combined with other CNS-depressant drugs, such as tranquilizers.

ALTERNATIVE DRUGS
Polyunsaturated omega 3 and 6 fatty acid—variable response in decreasing pruritus; provide support for epidermal barrier function and anti-inflammatory properties. Use as an adjunctive therapy. Response noted within 2–8 weeks after starting therapy. Exact dosing for horse is lacking; the author uses 180 mg of EPA/100 mg EPA/100 mg q12h.

FOLLOW-UP

PATIENT MONITORING
• Examine patient every 2–6 weeks when a new course of therapy is commenced. • Monitor pruritus, self-trauma, secondary bacterial dermatitis, and possible adverse drug reactions.

• Once an acceptable level of pruritus is achieved, examine patient every 4–12 mo.
• CBC, serum biochemical profile, and fibrinogen are recommended within the first week of starting corticosteroid therapy and then every 1–4 mo thereafter if chronic corticosteroid therapy cannot be avoided.

PREVENTION/AVOIDANCE
• Avoidance of allergens is not always possible or practical, especially as many patients have multiple allergens contributing to their disease. • Prevention of the disease may be possible if patient is moved to another region of the country.

POSSIBLE COMPLICATIONS
• Secondary bacterial dermatitis • Secondary lamination, colic, and iatrogenic hyperadrenocorticism due to chronic steroid administration

EXPECTED COURSE AND PROGNOSIS
• Not life-threatening unless intractable pruritus persists • No reports of spontaneous remission exist.

MISCELLANEOUS

ASSOCIATED CONDITIONS
• Insect hypersensitivity • Equine granulomas • Allergic conjunctivitis and rhinitis • Recurrent airway obstruction • Inflammatory airway disease • Summer pasture–associated obstructive pulmonary disease

AGE-RELATED FACTORS
• Security measures with age.

ZOONOTIC POTENTIAL
NOA

PREGNANCY
• Corticosteroids—contraindicated during pregnancy. • Antihistamines—no information on teratogenicity is available for horses; consider this before treating pregnant mares.

SYNONYMS
Equine atopy

SEE ALSO
• Insect hypersensitivity • Urticaria • Bacterial dermatitis • Equine pruritus

ABBREVIATIONS
• AD = atopic dermatitis • AST = alanine specific immunotherapy • ID3 = intradermal test • DTM = dermatophyte test medium

Suggested Reading

Author Gwenelen Lorch
Consulting Editor Gwenelen Lorch
Atrial Fibrillation

**Basics**

**Definition**
- An irregularly irregular cardiac rhythm, with variable-intensity heart sounds and pulses and inconsistent diastolic intervals
- Can be sustained or paroxysmal (resolving spontaneously within 48 hr of onset)

**Pathophysiology**
- A critical atrial mass must be present for the condition to occur.
- Predisposing factors—large atrial mass, high vagal tone, shortened and nonhomogeneous effective refractory period, potassium depletion, atrial premature depolarizations, rapid atrial pacing
- Produces no change in cardiac output at rest without underlying cardiac disease
- During high-intensity exercise, produces a marked increase in the heart rate response and fall in cardiac output and exercise capacity
- Present in many horses with CHF but is not the cause of CHF

**System Affected**
Cardiovascular

**Signalment**
Higher incidence in Standardbreds, Draft, and Warmblood horses

**Signs**

**General Comments**
- Causes exercise intolerance in performance animals, but often an incidental finding in sedentary horses

**Historical**
- Exercise intolerance
- Exercise-induced pulmonary hemorrhage—often profuse
- Weakness or collapse

**Physical Examination**
- Irregularly irregular heart rhythm
- Variable-intensity heart sounds and arterial pulses
- Absent fourth heart sound
- Cardiac murmurs with predisposing cardiac disease

**Causes**
- Normal horses have sufficient atrial mass and high vagal tone to develop AF without evident underlying heart disease.
- Diseases causing atrial enlargement further predispose horses to AF.

**Risk Factors**
- AV valve insufficiency
- CHF
- Electrolyte disturbances

**Diagnosis**

**Differential Diagnosis**
- Second-degree AV block—Regular rhythm is interrupted by pauses containing fourth heart sound.

- Atrial arrhythmia with second-degree AV block—Rhythm usually is regularly irregular; fourth heart sounds are present.
- Sinus rhythm with multifocal ventricular premature depolarizations—Need ECG to differentiate.

**CBC/Biochemistry/Urinalysis**
- Low plasma potassium or urinary fractional excretion of potassium may be present.

**Other Laboratory Tests**
- Elevated cardiac isoenzymes (e.g., CK-MB, HBDH, LDH-1 and LDH-2, cardiac troponin I) may be present but are usually within the normal range.
- RBC potassium concentrations may be decreased.

**Imaging**

**ECG**
- No P waves, replaced by baseline “f” waves
- The “f” waves may be coarse or fine and may occur 300–500 times per minute.
- Irregular R-R interval
- Some variation in the amplitude of QRS and T complexes usually is present, but these complexes are normal in appearance.

**Echocardiography**
- Most have little or no discernible underlying cardiac disease; therefore, the echocardiogram is normal.
- Some have low shortening fraction (24%–32%). This should return to normal within several days of conversion to normal sinus rhythm.
- Mild left atrial enlargement with sustained AF
- Atrial enlargement due to congenital defects or AV valve insufficiency may be present.

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![Base-apex lead, 25 mm/sec, 5 mm = 1 mV](image-url)

**Figure 1.**
Atrial Fibrillation

**DIAGNOSTIC PROCEDURES**

**Continuous 24-Hour Holter Monitoring**
Use in horses with suspected paroxysmal AF to identify underlying arrhythmias.

**Exercise Electrocardiography**
- Useful to detect exercise-induced arrhythmias and to determine exercise limitations if the AF is not or cannot be converted.

**PATHOLOGIC FINDINGS**
- Grossly and histopathologically normal heart in horses with no underlying cardiac disease.
- Focal or diffuse atrial fibrosis may be present in horses with long-standing AF.
- Myocarditis, myocardial necrosis, and fatty infiltration have been documented in affected horses.
- Both atrial and ventricular enlargement in horses with significant AV valvular disease.

**TREATMENT**

**AIMS OF TREATMENT**
- Restoration of sinus rhythm and athletic performance in horses with exercise intolerance but no, or minimal, underlying heart disease.
- Palliative care for horses with AF in conjunction with CHF.

**APPROPRIATE HEALTH CARE**
- Monitor horses for 24-48 hr to determine if the condition will resolve without treatment (i.e., paroxysmal).
- In horses with AF and CHF, institute treatment for congestive heart failure—for example, using digoxin (0.0022 mg/kg IV) and furosemide (1–2 mg/kg IV, not PO).

**MEDICATIONS**

**DRUG(S) OF CHOICE**

- The drug of choice for conversion is quinidine sulfate or gluconate.

**Quinidine Gluconate**
- Indicated with AF of duration ≤2 weeks and no underlying cardiac disease.
- Administered in boluses of 0.5–1 mg/kg every 5–10 min to a total dose of 10 mg/kg.

**Quinidine Sulfate**
- Indicated in horses with sustained AF.
- Administered via nasogastric intubation at 22 mg/kg q2h to a total of four to six treatments, then q6h until the horse shows signs of toxicity or has converted to sinus rhythm.

**CONTRAINDICATIONS**
- Do not administer quinidine sulfate or gluconate to affected horses with CHF.
- Horses with a resting heart rate of >60 bpm and/or grade 3/6 or louder systolic murmurs are likely to have CHF.

**PRECAUTIONS**
- Quinidine is associated with the following complications.

**Cardiovascular**
- Prolonged QRS duration—indicates quinidine toxicity.
- Rapid supraventricular tachycardia—treat aggressively with digoxin to slow heart rate.
- Digoxin is recommended in conjunction with quinidine in horses with myocardial dysfunction or rapid heart rate during quinidine treatment.
Atrial Fibrillation

- If heart rate exceeds 100 bpm, consider digoxin—0.011 mg/kg PO or 0.0022 mg/kg IV.
- If a heart rate exceeds 150 bpm, consider digoxin (0.0022 mg/kg IV) and sodium bicarbonate (1 mEq/kg IV).
- If the heart rate remains high, administer propranolol—0.03 mg/kg IV.
- If a horse receiving quinidine only on day 1 does not convert, consider adding digoxin orally on day 2.
- Base subsequent digoxin administration during quinidine treatment on serum digoxin concentration and need to control heart rate or to improve myocardial contractility.
  - Ventricular arrhythmias require treatment unless ventricular rhythm is slow (<100 bpm), uniform, and no R-on-T is detected.
  - Treat ventricular arrhythmias with magnesium sulfate—2–5 mg/kg bolus IV q5min to 50 mg/kg total or propranolol—0.03 mg/kg IV.
  - Hypotension—monitor and treat, if severe, with intravenous fluids to effect and, if necessary, phenylephrine (0.1–0.2 μg/kg per minute IV to effect).
  - Sudden death—Try to prevent with continuous ECG and treatment of any arrhythmias that occur.

GI
- Flatulence—resolves on return of quinidine plasma concentrations to negligible levels.
- Oral ulcetations—Prevent by not administering quinidine via nasogastric tube.
- Diarrhea—indicates quinidine toxicity; resolves on return of quinidine plasma concentrations to negligible levels.
- Colic—indicates quinidine toxicity; treat with analgesics as needed.

Respiratory
- Upper respiratory tract obstruction—Treat with passage of a nasotracheal tube to relieve the upper airway obstruction; administer corticosteroids and antihistamines; emergency tracheotomy, if necessary.

Dermatologic
- Urticaria—Treat with corticosteroids and antihistamines.

Reproductive
- Paraphimosis—resolves on return of plasma quinidine concentration to negligible levels.

Musculoskeletal
- Laminitis—If the horse is uncomfortable, administer analgesics.

Neurologic
- Indicates quinidine toxicity.
- Ataxia—resolves on return of plasma quinidine concentration to negligible levels.
- Convulsions—Administer anticonvulsants.
- Bizarre behavior—resolves on return of plasma quinidine concentration to negligible levels.

Possible Interactions
Quinidine competes with digoxin for binding to plasma protein, causing potential digoxin toxicity.

Alternative Drugs
Oral, but not intravenous, flecanide and intravenous amiodarone have recently been proposed.

Follow-up
Patient Monitoring
- Perform continuous ECG during treatment, because antiarrhythmic drugs are also arrhythmogenic.
- Measure QRS duration before each dose; discontinue treatment if QRS duration ≥25% of the pretreatment value.
- Discontinue treatment if rapid supraventricular tachycardia, ventricular arrhythmias, diarrhea, colic, ataxia, convulsions, bizarre behavior, urticaria, upper respiratory tract obstruction, or laminitis occurs.
- Following conversion, perform 24-hour Holter monitoring. If atrial ectopy is found, rest and corticosteroid therapy may be indicated.
- Riders should regularly monitor cardiac rhythm; any irregularities or poor performance should prompt reexamination.
**PREVENTION AVOIDANCE**
- Discontinue administration of furosemide and bicarbonate milkshakes.
- Administer potassium or other electrolyte supplementation, if indicated.
- See Supraventricular Arrhythmias.

**POSSIBLE COMPLICATIONS**
- If AF is not or cannot be treated, clinical signs will persist.
- Some horses with AF also have exercise-induced ventricular arrhythmia; this possibility should be explored if AF is not or cannot be treated and the horse is to continue to be used for ridden exercise—see Ventricular Arrhythmias.

**EXPECTED COURSE AND PROGNOSIS**
- Most horses with little or no underlying cardiac disease convert to sinus rhythm with quinidine therapy.
- Recurrences occur in ≈25% of horses with a suspected duration of atrial fibrillation of ≈4 mo.
- Recurrences occur in ≈60% of horses with a duration of atrial fibrillation of >4 mo.
- Recurrence is mostly likely during the first year after conversion but can occur at any time.
- Prognosis for return to the previous level of athletic performance is excellent in converted horses without significant underlying cardiovascular disease.
- Horses with sustained AF that do not convert to sinus rhythm with treatment or that are not candidates for conversion usually have a normal life expectancy and can be safely used for lower-level athletic performance.
- With significant valvular insufficiency, severity of the valvular heart disease and its progression determine the horse’s useful performance life and life expectancy.
- Horses with CHF usually have severe underlying valvular heart or myocardial disease and have a guarded to grave prognosis for life.
- Most affected horses treated for congestive heart failure respond to the supportive therapy and improve for a short time but are euthanized within 2–6 mo of initiating treatments.

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Atrial Septal Defect

**Basics**

**Definition**
- A congenital defect (i.e., hole) in the interatrial septum that creates a communication between the right and left atria.
- Can be located in the atrial septum immediately adjacent to the ventricular septum (i.e., atrium primum defect), in the area of the foramen ovale (i.e., atrium secundum defect), or in the most basilar portion of the interatrial septum (i.e., sinus venosus-type defect).
- ASD can occur in isolation or in conjunction with other cardiac anomalies in complex congenital cardiac disease.
- The atrial septum forms in the fetus from the septum primum and the septum secundum. The slit-like communication between these septa (i.e., the foramen ovale) allows passage of blood from right to the left atrium in the fetus.
- The foramen ovale is functionally closed in normal neonates within 24–48 hr of birth, but anatomic closure may not be complete until 9 weeks.

**Pathophysiology**
- A patent foramen ovale occurs when the foramen ovale fails to close.
- Failed formation of one of the two septa results in the other forms of ASD.
- Blood shunts from the higher-pressure left atrium to the lower-pressure right atrium in foals with ASD, creating a left atrial, right atrial, and right ventricular volume overload.
- Size of the ASD determines severity of the volume overload. In horses with a large ASD, the right and left atrial and right ventricular volume overload is severe.
- Over time, stretching of the tricuspid annulus occurs, and tricuspid regurgitation develops. As the tricuspid regurgitation becomes more severe, increases in right atrial pressure result in increased hepatic venous pressure and development of clinical signs of right-sided congestive heart failure.

**System Affected**
- Cardiovascular

**Genetics**
- Not yet determined in horses
- Although heritable in other species, it is rare in horses

**Incidence/Prevalence**
- These defects are uncommon as isolated congenital defects and more frequently occur in conjunction with complex congenital heart disease, particularly tricuspid and pulmonic atresia.

**Signalment**
- Most frequently diagnosed in neonates, foals, and young horses, but may be diagnosed at any age

**Signs**
- **General**
  - May be detected as an incidental finding, but usually is part of a more complex, congenital cardiac disorder
  - Exercise intolerance—medium-size to large ASDs
  - Congestive heart failure—large ASDs
- **Physical Examination**
  - No murmur may be present, or a course, band- or ejection-shaped, holosystolic murmur with PML in pulmonic valve area may be detected.
  - Premature beats or an irregularly irregular heart rhythm of atrial fibrillation may be present with larger ASDs.
- **Causes**
  - Failure closure of the foramen ovale
  - Congenital malformation of the interatrial septum

**Risk Factors**
- Premature foal
- Neonatal pulmonary hypertension
- Neonatal respiratory distress syndrome

**Diagnosis**

**Differential Diagnosis**
- Physiologic flow murmur—Differentiate echocardiographically.
- Pulmonic stenosis (rare)—murmur usually louder; differentiate echocardiographically.
- Aortic stenosis (rare)—murmur usually louder; weak arterial pulses; differentiate echocardiographically.
- Tricuspid atresia—murmur usually louder; foal is unthrifty, tachycardic, and hypoxicemic; differentiate echocardiographically.
- Atrial septal—murmur usually louder; may have a continuous machinery murmur; foal is unthrifty, tachycardic, and hypoxicemic; differentiate echocardiographically.

**CBC/Biochemistry/Urinalysis**
- N/A

**Other Laboratory Tests**
- N/A

**Imaging**

**Echocardiography**
- Atrial septal defect is detected at the ASD location and should be confirmed by visualization in two mutually perpendicular planes.
- The left and right atria and right ventricle are enlarged, dilated, and have a rounded appearance.
- Paradoxical septal motion is detected with a severe right ventricular volume overload.
- Pulmonary artery dilatation is seen in horses with a large shunt.
- Interventricular septum may be thickened, paradoxically moved, and not able to completely close.
- Contrast or color-flow Doppler reveals the shunt from the left to the right atrium through the ASD.
- A small amount of positive contrast may be seen in the left atrium in horses with normal pulmonary arterial pressures or with the Valsalva maneuver at contrast echocardiography.
- A jet of tricuspid regurgitation may be present in horses with a large ASD and marked right atrial and ventricular volume overload.

**Thoracic Radiography**
- Increased pulmonary vascularity and cardiac enlargement may be detected in horses with large shunts.

**Diagnostic Procedures**

**Cardiac Catheterization**
- Right-sided catheterization can be performed to directly measure right atrial, right ventricular, and pulmonary arterial pressures and to sample blood for oxygen content.
- Elevated right atrial, right ventricular, and pulmonary arterial pressures and increased oxygen saturation of right ventricular and pulmonary arterial blood have been seen in horses with larger ASDs.

**Continuous 24-Hour Holter Monitoring**
- Use in identifying intermittent atrial premature depolarizations.

**Pathologic Findings**
- Defect in the atrial septum
- Jet lesions along the defect margins and on the adjacent right atrial endocardium
- Left atrial, right atrial, and right ventricular enlargement and thinning of the left atrial, right atrial, and right ventricular free wall in horses with a significant shunt
- Pulmonary artery dilatation in horses with a large shunt or that have developed pulmonary hypertension
- With congestive heart failure, ventral and peripheral edema, pleural effusion, pericardial effusion, chronic hepatic congestion, and, occasionally, ascites may be detected.
## Atrial Septal Defect

### Treatment

**AIMS OF TREATMENT**
- Management by intermittent monitoring in horses with small ASDs.
- Palliative care in horses with large ASDs and those with complex congenital cardiac defects.

**APPROPRIATE HEALTH CARE**
- Most affected horses require no treatment and can be monitored on an outpatient basis.
- Monitor horses with large shunts on an annual basis.
- Affected horses with congestive heart failure can be treated for congestive heart failure with positive inotropic drug, vasodilators, and diuretics. Consider humane destruction if congestive heart failure develops, however, because only short-term, symptomatic improvement can be expected.

**Nursing Care**
- Activity: Affected horses are safe to continue in full athletic work until significant tricuspid regurgitation or atrial fibrillation develops.
- Horses with small defects can be in unrestricted activity and may be able to compete reasonably successfully in upper-level athletic competition, although they are unlikely to compete at the upper levels of athletic performance.
- Monitor horses with hemodynamically significant defects echocardiographically on an annual basis to ensure they are safe to ride and compete. These horses can be used for lower-level athletic competition but are unlikely to compete at the upper levels of athletic performance.
- Affected horses that develop atrial fibrillation need a complete cardiovascular examination to determine if they are safe to race or lower-level athletic performance.
- Horses with significant pulmonary artery hypertension cannot be monitored on an outpatient basis.

**Diagnosis**
- N/A

**CLIENT EDUCATION**
- Regularly monitor cardiac rhythm; any irregularities of the rhythm, other than second-degree AV block, should prompt ECG.
- Carefully monitor for exercise intolerance, respiratory distress, prolonged recovery after exercise, increased resting respiratory or heart rate, cough, generalized venous distention, jugular pulses, or ventral edema; if detected, obtain a cardiac examination.

**Surgical Considerations**
- Closure of the ASD would be possible with a transvenous umbrella catheter if the diameter of the umbrella was large enough to close the defect.
- Surgical closure is not financially feasible or practical for obtaining equine athletes at this time.

**Medications**

**Drug(s) of choice, contraindications, precautions, possible interactions, alternative drugs**
- N/A

**Follow-up**

**Patient Monitoring**
- Frequently monitor cardiac rate, rhythm, and respiratory rate and effort.

**Prevention/Avoidance**
- N/A

**Possible Complications**
- Large ASD—atrial fibrillation; congestive heart failure

**Expected Course and Prognosis**
- Horses with small defects should have a normal performance life and life expectancy.
- Horses with moderate defects also have a normal life expectancy. These horses usually perform successfully only at lower levels of athletic competition, and they may develop atrial fibrillation.
- Horses with large defects have a guarded prognosis, because they may have a shortened life expectancy and performance life, even at the lower levels of athletic competition.
- Affected horses with congestive heart failure usually have a guarded to grave prognosis for life. Most such horses being treated for congestive heart failure should respond to the supportive therapy and transiently improve; however, once congestive heart failure develops, euthanasia is recommended.

**Suggested Reading**

**Author**
- Virginia B. Reef

**Consulting Editor**
- Celia M. Marr
AURAL PLAQUES

**BASICS**

**OVERVIEW**
- Aural plaques are whitish plaques on the inner surface of the pinna of horses.
- Likely related to papilloma virus infection
- May or may not be associated with varying degrees of ear sensitivity
- Lesions do not spontaneously regress.
- Treatment has been unsuccessful until recently.

**SIGNALMENT**
- Common in both sexes and all breeds.
- Not frequently observed in horses < 1 year of age.

**SIGNS**
- Depigmented, well-demarcated papules and plaques covered with keratin deposits located on the concave surface of the pinna. Lesions are single, multiple, or coalescing and may affect one or both pinna.
- Horses may be asymptomatic or may resent bridling or handling of the ears.
- Head shaking has been rarely reported.
- Symptoms may be aggravated by biting flies.

**CAUSES AND RISK FACTORS**
- Bovine papilloma virus is suspected.
- Abrasions and insect bites may be involved in transmission.

**DIFFERENTIAL DIAGNOSIS**
- Sarcoids—usually identified on the external surface of the pinna or at the margins of the ear. They may be coexistent with aural plaques.

**DIAGNOSIS**

**Differential Diagnosis**
- Sarcoids—usually identified on the external surface of the pinna or at the margins of the ear. They may be coexistent with aural plaques.

**CBC/BIOCHEMISTRY/URINALYSIS**
- N/A

**OTHER LABORATORY TESTS**
- N/A

**IMAGING**
- N/A

**OTHER DIAGNOSTIC PROCEDURES**
- Diagnosis is based on classic appearance and can be confirmed by biopsy.

**PATHOLOGICAL FINDINGS**
- Histologic features consistent with papilloma virus infection including papillated epidermal hyperplasia, koilocytosis, and increased numbers and size of keratohyalin granules.

**TREATMENT**
- Multiple treatments are advocated but none have been shown consistently effective.
- CO₂ laser ablation, corticosteroids, tretinoin, and Eastern blood root in zinc chloride have all been tried with variable results and recurrence is common.
- Management of affected horses often involves minimizing resistance to ear handling and protecting ears from biting insects.

**DRUG(S) OF CHOICE**
- Imiquimod (Aldara) has recently been evaluated in a clinical trial and is effective at removing the plaques. Recurrence rates are as yet undetermined.
- Imiquimod is applied topically as a thin layer 2–3 × week every other week until resolution (typically 3–6 mo of every other week treatment).

**MEDICATIONS**
CONTRAINDICATIONS/POSSIBLE INTERACTIONS
A strong local inflammatory response is consistently observed with imiquimod due to its mechanism of action. This can make it difficult to clean the ears prior to the subsequent treatment. Sedation is often needed, particularly for the second or third treatments of the treatment weeks. Owners should be warned of the reaction and temporarily increased sensitivity due to local inflammation.

FOLLOW-UP
PATIENT MONITORING
• Monitoring for complete resolution is important. Imiquimod causes enough local reaction that it can be difficult to determine if the plaques are still present. One to 2 weeks without treatment allows better evaluation and recheck evaluation at 1 mo post-treatment is strongly recommended.
• Each lesion must be treated. No effect is observed on untreated lesions.

PREVENTION/AVOIDANCE
• Generally not possible
• Use of fly repellents with permethrin/pyrethrin (for quick insect knockdown) and piperonyl butoxide (as a pesticide synergist) in addition to fly masks that provide ear coverage may help prevent development of additional lesions.

POSSIBLE COMPLICATIONS
• Ear sensitivity and pain on cleaning
• Imiquimod can cause skin erosions or ulcers, particularly if applied in a thick layer. Erosions appear to be more common in the first month of treatment. The amount of reaction seems to decrease as the plaques resolve.

EXPECTED COURSE AND PROGNOSIS
• Aural plaques persist without treatment.
• Post-treatment skin depigmentation may occur.
• Initial results with imiquimod treatment suggest that after resolution of the plaques, horses are less sensitive to ear manipulation than prior to treatment.

MISCELLANEOUS
ASSOCIATED CONDITIONS
None known

AGE-RELATED FACTORS
None known

ZOONOTIC POTENTIAL
None

PREGNANCY
Does not affect disease or treatment

SEE ALSO
• Papillomatosis
• Sarcoid

Suggested Reading

Authors: Erin Malone and Sheila Torres
Consulting Editor: Gwendolen Lorch
Azotemia and Uremia

**BASICS**

**DEFINITION**
- Azotemia: the accumulation of nitrogenous waste (e.g., urea, Cr, other nitrogenous substances) in blood, plasma, or serum.
- Uremia: the clinical manifestation of azotemia; a multisystem disorder resulting from the effects of uremic toxins on cellular metabolism and function. 
  - Cr and Ca levels are measured in serum and used as indices of azotemia.

**PATHOPHYSIOLOGY**
- Serum urea concentration is determined by rate of urea synthesis by hepatoocytes and rate of clearance by the kidneys. 
  - Increased protein catabolism results in elevated SUN.
  - Decreased GFR may result from decreased renal perfusion (i.e., prerenal azotemia), primary renal disease, or other causes, including infection, trauma, myositis, thermal injury, and melena.

**CAUSES**
- **Prerenal Azotemia**
  - Renal hyperperfusion caused by decreased circulating volume or decreased blood pressure.

**SIGNS**
- Weight loss
- Colic
- Respiratory—dyspnea

**DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS**
- **Prerenal azotemia**—diabetes, hypothyroidism, acute blood loss, decreased cardiac output, congestive heart failure, among others.

**RISK FACTORS**
- **Physical Examination**
  - Fever
  - Abnormal urination
  - Poor body condition
  - Poor performance

**TREATMENT**
- **Correction of dehydration deficits and restoring electrolyte balance**

**IMAGING**
- **Ultrasonography**
  - The urinary tract can be examined either transrectally or transabdominally.

**REFERENCES**
• The renal medulla is more colcholic than the renal cortex. The renal pelvis varies in echogenicity.
• With acute renal failure, kidneys may be normal or enlarged, and parenchymal abnormalities often are not detected.
• With chronic renal failure, kidneys are smaller and more echogenic than normal.
• Cystic or mineralized areas more often are associated with chronic renal disease or congenital anomalies.
• Acoustic shadowing represents calcification formation.

Renal Scintigraphy
May be used to document renal function but is not commonly performed.

OTHER DIAGNOSTIC PROCEDURES

Urine GGT/Cr Ratio
• Reflects GGT leakage from damaged renal tubular epithelium containing GGT compared to the constant excretion of Cr.
• Calculated as (Urine GGT/Ur ine Cr) × 100
• A ratio of >25 suggests proximal tubular damage; this elevation may occur before azotemia develops.
• Finding an elevated ratio depends on having enough remaining tubules that can leak GGT—severe renal fibrosis may yield values in the normal range.

Fractional Excretion of Electrolytes
• Measurement of electrolytes in serum and urine can be compared to assess renal damage.
• Calculated as [(Ur ine [electrolyte] × Ur ine Cr)/Ser um [electrolyte] × Ur ine Cr].
• Reported reference intervals for sodium fractional excretion range from 0.01–0.70 in healthy horses.
• Poor indicator of renal function.

Rectal Examination
• Bladder—determine size, wall thickness, and presence of calculi or mural mass.
• Left kidney—determine size and texture.
• Ureter—usually not detectable; enlarged in association with pyelolithiasis or ureterolithiasis.

Ultrasound-Guided Renal Biopsy
Can be used to confirm the diagnosis of primary renal failure, to differentiate acute from chronic renal disease, and to identify a specific cause.

Urethrocystoscopy
• Extremely useful diagnostic aid when evaluating obstructive urinary tract disease, especially in geldings and stallions.
• Early use may be beneficial, especially in geldings and stallions.
• If the urethra is dilated with air (e.g., to aid passage of the endoscope), the mucosa may appear reddened, and a prominent vaginal pattern may appear.
• The ischial arch and colliculi seminales are the most common sites of postural or postvoiding hematuria in geldings and stallions.
• In the dorsal aspect of the trigone, the ureteral openings can be visualized to determine the source of hematuria or pyuria.

Biopsy of a bladder mass or collection of a sterile urine sample can also be obtained.

Renal Scintigraphy
May be used to document renal function but is rarely performed.

TREATMENT

PRERENAL AZOTEMIA
• Correct the underlying cause of renal hypoperfusion and/or correct the dehydration deficit.
• Fluid replacement is primary therapy.
• More aggressive treatment in conditions that can lead to primary renal damage or failure.

PRIMARY RENAL AZOTEMIA
• Measures to stop or reverse the immediate

POSTRENAL AZOTEMIA
• Eliminate the urinary obstruction or correct the cause of urine leakage.
• Surgical intervention often is required, but correction of any metabolic derangements is paramount.
• Solitary stones can follow correction of postrenal azotemia; thus, additional fluid therapy may be required to prevent dehydration.

FLUIDS
• IV fluid therapy is indicated for most azotemic patients.
• Commonly used fluids—0.9% saline, Ringer’s, and lactated Ringer’s solution.
• Base the amount of fluid administered on the dehydration or volume deficit.
• Correction of the fluid deficit may occur during the first 6 hr without untoward effects, except in patients with hyperkalemia/ hypercalcemia and with signs of cardiac disease.

MEDICATIONS

DRUGS OF CHOICE
• Treat any patient exhibiting signs of shock appropriately.

CONTRAINdications
Use nephrotoxic drugs (e.g., aminoglycosides, NSAIDs) with caution in patients with azotemia.

PRECAUTIONS
Use caution when administering fluids to horses with chronic renal failure, because they may develop significant peripheral and pulmonary edema.
Use IV fluids cautiously in oliguric or anuric patients to minimize overhydration.
Use NSAIDs and corticosteroids cautiously.

APPELLATIONS
• GFR = glomerular filtration rate
• GGT = y-glutamyltransferase
• PU/PD = polyuria/polydipsia

Suggested Reading

Author Terry C. Genetics
Consulting Editor Kenneth W. Hinchcliff