Abdominal Distention in the Adult Horse

Abdominal ultrasonography may help in determining location, nature, and severity of the
cause of colic.

CAUSES
- Accumulation of Gas
  - Respiratory—Obstructed abdominal wall (e.g., perineum, hernia)
  - Respiratory—Obstructed abdominal wall (e.g., perineum, hernia)
- Accumulation of Fluid
  - Respiratory—Obstructed abdominal wall (e.g., perineum, hernia)
  - Respiratory—Obstructed abdominal wall (e.g., perineum, hernia)
- Body Wall Abnormality
  - Respiratory—Obstructed abdominal wall (e.g., perineum, hernia)
  - Respiratory—Obstructed abdominal wall (e.g., perineum, hernia)

Diagnosis

Differentiating Similar Signs

1. Marked subcutaneous edema along the ventral abdomen and thorax—hypoprothrombinemia,
disturbed regional lymphatic drainage (e.g., pleuritis), cardiac failure postpartum following abdominal celiotomy

- Painful
- "Hay belly"—horses may be diagnosed on history, or by physical examination, laboratory work, recital palpation, and ultrasonography. Evaluation of fluid findings often provide sufficient information to permit a tentative diagnosis. Some conditions are associated with characteristic findings.

- Pregnancy
- "Hay belly"—may be diagnosed on history (multilocal or multifocal painless horses die quickly in poor-quality roughage) and by fecal examination

Differentiating Causes

Signalement, history, physical examination, laboratory work, recital palpation, and ultrasonography. Evaluation of fluid findings often provide sufficient information to permit a tentative diagnosis. Some conditions are associated with characteristic findings.

- GI gas accumulation (blowout)—On auscultation of the abdomen, few to no GI sounds may be heard, and increased gaseous distension may be identified on percussion as a hyperresonant "ping," depending on the cause and the degree of distension present. Patients degrees of abdominal pain are usually present.

- Ascites from right-sided heart failure—
  - Tissue fluid analysis results in findings including heart murmur, exercise intolerance, jaundice, hepatic encephalopathy, and hepatic insufficiency—Because this tumor occurs from the fluid-producing cells of the peritoneum, several lines of peritoneal fluid may be produced.
Abdominal Distention in the Adult Horse

**TREATMENT**

- Treatment is dependent on the cause of abdominal distention.
- Cardiovascular stabilization through fluid therapy and correction of electrolyte and acid-base abnormalities should be initiated prior to treatment of the primary disease process.
- In horses with severe gasous distention, trocharization of the ceccum 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 usually located within the paralumbar fossa or can be delineated through auscultation and percussion of the distended viscus. The incision should preferably be made in the mid-posterior region of the horse’s body wall, thereby decreasing the incidence of direct peritoneal contamination. The trocharization site should ideally be located in the mid-posterior region of the body wall, thereby minimizing the risk of direct peritoneal contamination. The trocharization site should ideally be located in the mid-posterior region of the horse’s body wall, thereby decreasing the incidence of direct peritoneal contamination.

- 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 unnecessarily as it may be a life-saving diagnostic and therapeutic tool if used appropriately.

- Cardiovascular stabilization through fluid therapy and correction of electrolyte and acid-base abnormalities should be initiated prior to treatment of the primary disease process.
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**MEDICATIONS**

Drug therapy is dictated by the causing incite. Plans for monitoring are based on cause and treatment.

**FOLLOW-UP**

- Consulting Editors
- Author

**SYNONYMS**

- Blister

**SEE ALSO**

- See Causes

**ABBREVIATIONS**

- GI = gastrointestinal
- PCV = packed cell volume
- SG = specific gravity
- TP = total protein
- WBC = white blood cell

**SUGGESTED READING**


**Author**

Antonio M. Cour

**Consulting Editors**

Henry Stampfl and Olimpo Oliver-Espinosa

- within a 24-hr period; ascites may be more dramatic than is noted with other conditions.
- Body wall defect from peptic ulcer or 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 inaudible sounds and posteri. Enteroliths or sand impactions may be evident in adult horses in the mid-to ventral abdomen on the lateral view. Standing radiographs of these regions in a 500-kg horse require 450 mA and 100 kVp and therefore are mostly available at referral centers.
- Ultrasonography of the abdomen can be used to provide a definitive diagnosis of the nature of the 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.

- Sepsis
- Treatment should be initiated prior to the development of gas gangrene.
- In horses with severe gasous distention, trocharization of the ceccum 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 usually located within the paralumbar fossa or can be delineated through auscultation and percussion of the distended viscus. The incision should preferably be made in the mid-posterior region of the body wall, thereby decreasing the incidence of direct peritoneal contamination. The trocharization site should ideally be located in the mid-posterior region of the horse’s body wall, thereby decreasing the incidence of direct peritoneal contamination. The trocharization site should ideally be located in the mid-posterior region of the horse’s body wall, thereby decreasing the incidence of direct peritoneal contamination. The trocharization site should ideally be located in the mid-posterior region of the horse’s body wall, thereby decreasing the incidence of direct peritoneal contamination.

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### Overview
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.

#### Signalement

**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 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. It can also be associated with trauma and hydralantois.
- 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.

**Incisional Hernia**
- Incisional infection and swelling, postoperative endotoxemia and pain, repeated surgeries, and use of chromic gut suture predispose hernia formation after celiotomy.

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

#### Diagnosis

**Differential Diagnoses**

**Ventral Hernia**
- Prepubic 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**
- Tension of the spermatic cord, infectious epididymitis or orchitis, thrombosis of the testicular artery, hydrocele, hematocele, and testicular neoplasia.

**CBC/Biochemistry/Urine Analysis**
- 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 prepubic 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 herniae 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. Affected 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**

**Drug(s)**

**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 Bourée

**Consulting Editors**
Henry Stampfl and Olimpo Oliver-Espinosa
**Abdominocentesis**

**BASICS**

**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 nondegenerative 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 uroabdomen.

**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
- Bleed or urine leakage
- Postsurgical inflammation
- Abdominal abscess

**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.
Abdominocentesis

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.
• 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.

Ascites
• Congestive Heart Failure
  Increased hydrostatic pressure within vessels may result in a modified transudate with a higher cell count and protein level than a transudate, 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—hyponatremia is consistent with GI protein loss; elevated liver enzymes suggest hepatic disease.

OTHER LABORATORY TESTS
• Serum electrolytes and comparison of serum and fluid creatinine aid in diagnosis of uropetoneum.

IMAGING

Ultrasongraphy
• May be used to look for intestinal entrapment, intussusception, masses, adhesions, enlarged liver, and enteroliths.
• Ultrasoundographic 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 neoplasms.
• 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.

ABDOMINOCESTESIS

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. Torquint
Consulting Editor Kenneth W. Hinchcliff
Abnormal Estrus Intervals

**BASICS**

**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 interovulatory intervals result from endocrine or environmental factors.

**ETIOLOGY/PATHOPHYSIOLOGY**

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

**Key Hormonal Events in the Equine Estrous Cycle**

* FSH causes ovarian follicular growth.
* Estriol (E2) stimulates increased GnRH pulse frequency to decreased LH secretion. * LH surge causes ovulation; E2 returns to basal levels. * 1–2 days post-ovulation. * Progesterone (P4) rises from basal levels (< 1 ng/mL) at ovulation to > 4 ng/mL by 4–7 days post-ovulation. * P4 causes decreased GnRH pulse frequency, allowing increased FSH secretion to stimulate a new wave of ovarian follicles to develop during diestrus. * PGF2α (endometrial origin) is released 14–15 days post-ovulation, causing luteolysis and concurrent decline in P4 levels.

**Estrus Length**

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**

* Absence of estrus behavior even if E2 is present in small quantities.
* Conditions that eliminate P4 or levels > E2 concentrations are likely to induce estrus behavior. Persistence of these conditions results in abnormal estrous periods or interestrus intervals. The converse is also true.

**SYSTEMS AFFECTED**

* Reproductive * Behavioral * Endocrine

**SIGNALMENT**

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

**SIGNS**

**Historical**

* Chief complaints—infertility, failure to show estrus, prolonged estrus, silent estrus, or frequent estrus behavior.
* Breeding records—review methods, frequency, teaser type (pony, horse, gelding), stallion behavior (aggressive, passive, vocalization, proximity), and handler experience.

* Seasonal influence—individual variation (onset/duration/termination of cyclicity) can be mistaken for estrus irregularity.
* Mare's reproductive history—can clinical abnormalities be linked to estrous cycle length, timing, finding, previous injuries, or genital infections?
* Pharmacological—Clinical abnormalities related to current and historical drug administration?

**Physical Examination**

* Body condition—Poor condition/malnutrition may add to cause abnormal estrous cycles.
* Prolactin concentration—Poor prolactin concentration can result in proestrus, ascending infection, uterine pooling and may result in symptoms consistent with behavioral estrus. * Clinical signs—Enlargement may be related to prior treatment with anabolic or progestational steroids or intersex conditions. * TRF—Essential to evaluate abnormal cycles; uterine size and tone; ovarian size, shape and location; cervical relaxation. Serial examinations, minimum 3 per week, may be needed (several weeks) to define her estrous cycle.
* U/S—Define uterine and ovarian features, normal and abnormal.
* Vaginal examination—To identify inflammation, uterine pooling, cervical competency or abnormal conformation. Also identify stage of estrous cycle (appearance, degree of internal cervical relaxation).

**CAUSES**

**Shortened Estrus Duration**

* Seasonality—Estrus duration decreases in height of breeding season; more efficient folliculogenesis. * Slow estrus—Normal cycle ovarian activity but minimal or no overt sexual receptivity. Often behavior-based problem—nervousness, foul-at-side, maiden mare; possibly previous anabolic steroid use.

**Lengthened Estrus Duration**

* Seasonality—Estrus behavior with transition periods is common. Vernal transition period in the Northern Hemisphere extends from February to April; mare begins to ovulate. * Mare's reproductive history—Can clinical abnormalities be linked to estrous cycle length, timing, finding, previous injuries, or genital infections?
* Pharmacological—Clinical abnormalities related to current and historical drug administration?

**Shortened Interestrus Interval**

* A normal diestrus extends from February to April; mare begins to develop follicles but not regular estrous cycles. * Characterized by persistent estrus behavior, irregular estrous cycles, or irregular diestrous intervals.

**Lengthened Interestrus Interval**

* Diestrous—Season, combined with serial TRF and US, confirms normal estrus behavior, uterine inflammation, or abnormal response to anabolic agents.

**DIFFERENTIAL DIAGNOSIS**

**Differentiating Conditions with Similar Symptoms**

* Frequent urination:
  * Cystitis/vesicovaginitis
  * Bladder anastomotic defect
  * Urinary pooling
  * Vaginitis or pneumovagina

* May mimic submissive urination and be confused with anestrus—review or alter teasing methods.

**Differential Causes**

* Minimum database—Neural full medical and reproductive history, teasing, physical examination, TRF US, vaginal examination.
* May be useful—uterine cytology, culture, and biopsy

* Silent estrus—when due to port detection
* Diagnosis—TRF minimum 3 per week with frequent serum P4 assay to detect a short or inapparent estrus period.

* Transition period in the Northern Hemisphere extends from February to April; mare begins to develop follicles but not regular estrous cycles.

* Characterized by persistent estrus behavior, irregular estrous cycles, or irregular diestrous intervals.
* Diagnosis—Season, combined with serial TRF and US.

* U/S—Define uterine and ovarian features, normal and abnormal.
* Vaginal examination—To identify inflammation, uterine pooling, cervical competency or abnormal conformation. Also identify stage of estrous cycle (appearance, degree of internal cervical relaxation).

* No evidence that chronic PGF2α treatment (label-dosing) inhibits spontaneous formation and release of endogenous PGF2α
* GnRH agonist (deslorelin) implants—stimulates ovulation, associated with prolonged interovulatory intervals; effect more profound if PGF2α is used during the diestrus period to shorten cycle time.
Abnormal Estrus Intervals

MEDICATIONS

DRUG(s) OF CHOICE

• PGF₂α (10 mg IM) or its analog to luteal tissue

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

• Altegun (0.044 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 anestrus treatment increases the reliability of this transition management regimen.

CONTRAINDICATIONS

PGF₂α and its analogs—contraindicated in mares with foetuses, or other bromocriptine-like disease.

PRECAUTIONS

• Humans

• PGF₂α causes sweating 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 initiated.

• Antibodies to hCG can develop after exposure to skin—wash off immediately.

• In patients with histories of asthma, bronchial disease. Accidental exposure to skin—wash off immediately.

• Progesterone but fewer side effects

While not currently approved for use in horses, it is in broad use in the absence of an alternative.

FOLLOW-UP

PATIENT MONITORING

Until normal cyclicity is established or pregnancy confirmed, regular TRP examinations are recommended.

POSSIBLE COMPLICATIONS

Uterine corrected, abnormalities in estrus behavior frequently result in infertility.

#ABNORMAL ESTRUS INTERVALS

OCURRENCE

• Varying estrus intervals—silent estrus may be a reflection of poor teasing management.

• The mare enters the breeding herd and fails to have normal estrus cycles

• Abnormal estrus intervals—occur in mares with normal reproductive tracts

• Vary teasing methods—Silent estrus may be a problem

• Mare testosterone values typically <50–60 pg/mL and inhibin values <50–70 pg/mL

• Progesterone levels of 50–100 pg/mL (if thecal cells are present), inhibin <0.7 ng/mL, with P4 <1 mg/mL

• Transrectal US breeding herd and fail to have normal estrus cycles

• Diagnosis—TRP and US—endocrine assays

• Gonadal dysgenesis—usually ID when the mare transitions US breeding herd and fail to have normal estrus cycles

• Diagnosis—TRP and US confirm absence of normal ovarian tissue and a normal reproductive tract. Karotyping for a definitive diagnosis

• Ovulation in diestrous—Corpus luteum formed from a diestrous ovulation may be insufficiently mature to be lysed by endogenous PGF₂α at the end of diestrus. Ovulations after day 10 of the estrous cycle result in persistent CL activity.

• Diagnosis—demonstration of a normal reproductive tract with failure of clinical estrus for ≥2 weeks post-ovulation; P4 levels of ≥4 ng/mL for ≥2 weeks

OTHER LABORATORY TESTS

• Serum P₄ concentrations

• Basal levels of estradiol

• Basal levels of progesterone

• Uterine cytology, culture, and biopsy

IMAGING

Transrectal US—routine to evaluate equine ovarian reproductive tract.

OTHER DIAGNOSTIC PROCEDURES

• Ultrasound endoscopy can help identify intrauterine adhesions, glandular or lymphatic cysts, and polyps.

• Vestine cytology, culture, and biopsy

TREATMENT

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

• Monitor the problem mare, including TRP and US, 3 times weekly to best define the reproductive cycle.

• Poor verbal communication—Control progestogens by palpation, a portion of the dorsal uterine commissure is closed surgically.

• GCT/GTCT—ovariectomy

• Ultrasound, transrectal ultrasound and cervical smear—surgical correction

• Artificial lighting—management tool to institute earlier ovarian activity

• Mares bred earlier in the season, fail earlier the next year, accommodate breed registries

• Photostimulation does not eliminate ventral transition; merely shifts it to an earlier time of onset

• Photostimulation should begin ≥90 days prior to the onset of early season breeding.

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

• Altegun (0.044 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 anestrus treatment increases the reliability of this transition management regimen.

• Altegun—associated with suppression of 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. Inseparable product still available in the United States

• Altegun, deslorelin, and PGF₂α should not be used in horses intended for food.

• Humans

• PGF₂α or its analog should not be handled by pregnant women or persons with asthma or bronchial disease. Accidental exposure to skin—wash off immediately.

• Altegun should not be handled by pregnant women or persons with thromboembolitis and/or thromboembolic disorders, cerebrovascular disease, coronary artery disease, breast cancer, or other bromocriptine-like disease.

• Altegun should not be handled by pregnant women or persons with thromboembolitis and/or thromboembolic disorders, cerebrovascular disease, coronary artery disease, breast cancer, omega-dependent neoplasia, undiagnosed vaginal bleeding, or tumors that developed during the use of oral contraceptives or estrogen-containing products. Accidental exposure to skin—wash off immediately.

• Possible interactions

• N/A

• Altegun is a prostaglandin analog: similar to natural prostaglandin but fewer side effects

• While not currently approved for use in horses, it is in broad use in the absence of an alternative.

• Photostimulation does not eliminate ventral transition; merely shifts it to an earlier time of onset

• Photostimulation should begin ≥90 days prior to the onset of early season breeding.

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• Dr. C. Miller

• Consulting Editor: Carla L. Carleton

• Equine, Second Edition

• Equine, Second Edition

• Equine, Second Edition

• Equine, Second Edition
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, hematocoele, 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

**SIGNALMENT**
- Intact male horses
- Any age

**HISTORICAL**
- Gross changes in the size of the scrotum (usually acute)
- Pain (generally colic-like symptoms)
- Resistance to breading, 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.

**CAUSES**
- Three most common:
  - Trauma, may include testicular hematoma/rupture
  - Inguinal/scrotal herniation
  - Torsion of the spermatic cord, also known as testicular torsion
- Inflammatory/infectious causes:
  - EIA
  - EVA/EAV
  - Orchitis/epididymitis
- Neoplasia
  - Primary scrotal—melanoma, sarcomatoid
  - Testicular neoplasia—seminoma, teratoma, interstitial cell tumor, Sertoli cell tumor
- Noninflammatory scrotal edema
  - Hydrocele/hematocoele
  - Varicocele
- See also: Abnormal Testicular Size

**RISK FACTORS**
- Breeding activity
- Duration of problem
- Testicular orientation
- Testicular neoplasia—seminoma, teratoma, interstitial cell tumor, Sertoli cell tumor

**DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS**

**Differentiating Causes**
- Duration of problem
  - Acute—traumatic injury, torsion of spermatic cord, herniation, infection
  - Chronic—neoplasia, testicular neoplasia—neoplasma, interstitial cell tumor, Sertoli cell tumor
  - 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
- U/S (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**
- EVA
- AN or CF
- Acute and convoluted serum samples
- If stallion is sterile, carrier state is determined with virus isolation.
- Virus isolation from serum and/or seminal plasma

**TREATMENT**
Treatment is directed at the cause of scrotal enlargement.
- Management of inflammation is a primary concern with abnormal scrotal enlargement.
- Sexual rest is indicated for all causes of scrotal enlargement.

**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.
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 testis 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
  - Variocele
  - Nonreproductive 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 hemorrhage.
- 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
- Endometritis
- Laminitis
- Scrotal adhesions
- Death

EXPECTED COURSE AND PROGNOSIS
N/A

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

SEE ALSO
- Abnormal testicular size

SUGGESTED READING

Author Margo L. Macpherson
Consulting Editor Carla L. Carlson
A primary testicular neoplasia may be affected subsequent to metastasis of horse.

ETIOLOGY/PATHOPHYSIOLOGY
• The testes and epididymides are positioned in a horizontal orientation between the hind limbs of the horse and are freely movable within the scrotum.
• The scrotum and contents, while relatively protected from external insults, are at increased risk for injury during breeding or athletic activity.
• Acute enlargement of a testis occurs after trauma, torsion of the spermatic cord, or sepsis/epididymitis.
  • May be of bacterial, viral, autoimmune, or parasitic origin
• Testicular neoplasia is uncommon in the horse.
  • Seminoma, teratoma, Sertoli cell tumor, interstitial cell tumor
  • Of these, seminoma is the most frequently reported testicular tumor of the stallion.
  • Most equine testicular tumors arise from germ cells, including seminomas and teratomas.
  • The effect of neoplasia on testicular size (increase or decrease) may be insidious.
  • Hypoplastic and degenerative testes are smaller than normal.
• Testicular degeneration can be transient or permanent.
  • An acquired condition, degeneration may arise from thermal injury, infection, vascular insults, hormonal disturbances, toxins, and age.
• Testicular hypoplasia is an irreversible condition.
  • Hypoplastic testes are incompletely developed.
  • Condition is usually congenital.
  • Suspected causes include genetic aberrations, teratogens, cryptorchidism, and postnatal insults.

SYSTEMS AFFECTED
• Reproductive
• Other systems (respiratory, GI, lymphatic) may be affected subsequent to metastasis of primary testicular neoplasia.

GENETICS
Cryptorchid and testicular hypoplasia are subjected to having genetic components.

INCIDENCE/PREVALENCE
Dependent on etiology

SIGNALMENT
• Intact male horses
• Any age

SIGNS
Historical
• Recent history of breeding or semen collection
• Gross changes in the size of a testis
• Reduced fertility
• Pain (generally colic-like symptoms)
• Relevance to breed, jump, or walk

Physical Examination
• Increased or decreased scrotal size
• Increased or decreased testicular size
• Abnormal testicular texture (too soft or firm)
• Abnormal testicular position
• Abnormal scrotal temperature (too warm or cold)
• Edema/engorged scrotum and/or contents
• Deformities in systemic parameters (elevated HR, RR, inappetence, CBC abnormalities)

CAUSES
• Three most common
  • Testes
  • Cryptorchidism
  • Torsion of the spermatic cord
• Torsional degeneration
• Testicular hypoplasia
• Testicular hematomas/rupture
• Neoplasia
  • Seminoma
  • Teratoma
  • Interstitial cell tumor
  • Sertoli cell tumor
• Ovarian/epididymal
  • Bacterial infection
• EIA
• EAV
• Strongyloides edentatus infection
• Endocrine profile (LH, FSH, testosterone, estrogen)
• Reproductive abnormalities

RISK FACTORS
• Breeding activity
• Systemic illness
• Temperature extremes
• Anabolic steroid use

DIAGNOSIS
DIFFERENTIAL DIAGNOSIS
Differentiating Similar Signs
• Serosal enlargement due to scrotal hydrocol/elastocoele and scrotal or inguinal hernia may be confused with testicular enlargement.
• US examination and measurement of the testes is the best means of differentiating the pathologies.

Differentiating Causes
• Duration of problem:
  • Acute: traumatic injury, torsion of spermatic cord, infection
  • Chronic: cryptorchidism, neoplasia, infection, testicular degeneration/hypoplasia
• History of recent breeding and/or trauma
**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 testicular trauma.
- Antiparasitic therapy for testicular trauma and/or prior to surgery.
- 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.
- Laminitis
- Endotoxemia
- Respiratory rate
- Heart rate
- GI gastointestinal
- FSH follicle-stimulating hormone
- LH luteinizing hormone
- EIA equine infectious anemia
- AGID agar-gel immunodiffusion
- EAV equine arteritis virus
- EIA equine arteritis virus
- EVA equine viral arteritis
- FSH follicle-stimulating hormone
- GI gastrointestinal
- HR heart rate
- LH luteinizing hormone
- RR respiratory rate
- SN serum neutralization
- US ultrasonography

**DIET**

Modification is necessary only with cases of secondary etiologies or as a preparative 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 hypoplastic testes 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.

**FOLLOW-UP**

Semen collection and evaluation 90 days after complete resolution of testicular insult.

**Surgical Considerations**

- Hemihemicastrations is the treatment of choice for:
  - Tension 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 testes/es

**PATHOLOGICAL FINDINGS**

N/A

**MISCELLANEOUS**

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

**Abnormal Testicular Size**

<table>
<thead>
<tr>
<th>Age-Related Factors</th>
<th>Possible Complications</th>
<th>Expected Course and Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preadult testes are small and can be misdiagnosed as pathologically hypoplastic.</td>
<td>Inflammatory/infertility</td>
<td>Dependent on etiology</td>
</tr>
<tr>
<td>Testicular growth increases rapidly from 12 to 24 mo of age in horses.</td>
<td>Endotoxemia</td>
<td></td>
</tr>
<tr>
<td>Testes may take 4–5 y to reach full size and maturity.</td>
<td>Laminitis</td>
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<tr>
<td></td>
<td>Respiratory rate</td>
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<td>Heart rate</td>
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<td></td>
<td>Serum neutralization</td>
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<tr>
<td></td>
<td>Ultrasound, ultrasonography</td>
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</tbody>
</table>

**Suggested Reading**


**Author** Margo L. Macpherson

**Consulting Editor** Carla L. Calhoun

**ABBREVIATIONS**

- AGID = agar-gel immunodiffusion
- CBC = complete blood count
- CF = complement fixation
- EAV = equine arteritis virus
- EIA = equine infectious anemia
- ELISA = enzyme-linked immunosorbent assay
- EVA = equine viral arteritis
- FSH = follicle-stimulating hormone
- GI = gastrointestinal
- HR = heart rate
- LH = luteinizing hormone
- RR = respiratory rate
- SN = serum neutralization
- US = ultrasound, ultrasonography

**ASSOCIATED CONDITIONS**

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

**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) is indicated in most cases.
- Antibiotic therapy should be considered in cases of orchitis/epididymitis and testicular trauma.
- Administration of IV fluids is dependent on systemic status of the horse.
- Most causes of testicular enlargement require hospitalization for treatment/resolution.
- In cases of orchitis/epididymitis and testicular trauma.

**CONTRAINDICATIONS PREGNANCIES**

N/A

**POSSIBLE INTERACTIONS**, **ALTERNATIVE DRUGS**

N/A

**SYNONYMS**

N/A

**SEE ALSO**

- Cryptorchidism
- Abnormal testicular enlargement

**PATHOLOGICAL FINDINGS**

N/A

**PREGNANCY**

N/A

**PRIORITY TREATMENTS**

- Premature release of spermatids
- Azoospermia
- Oligospermia

**DIAGNOSTIC TESTS**

- Semen evaluation is useful in the diagnosis of testicular degeneration or hypoplasia.
- Ultrasound, Ultrasonography
- Serum neutralization
- Complement fixation
- Enzyme-linked immunosorbent assay
- EIA = equine infectious anemia
- AGID = agar-gel immunodiffusion
- FSH = follicle-stimulating hormone
- GI = gastrointestinal
- HR = heart rate
- LH = luteinizing hormone
- RR = respiratory rate
- SN = serum neutralization
- US = ultrasound, ultrasonography

**PREFERRED TESTS**

- Ultrasound, Ultrasonography
- Serum neutralization
- Complement fixation
- Enzyme-linked immunosorbent assay
- EIA = equine infectious anemia
- AGID = agar-gel immunodiffusion
- FSH = follicle-stimulating hormone
- GI = gastrointestinal
- 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. Calhoun

**ASSOCIATED CONDITIONS**

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

**BASICS**

**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

**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

- **Bacteria**
  - Placentitis and possible, subsequent fetal infection by *Streptococcus* sp., *Actionibacillus* sp., *Escherichia coli*, *Pseudomonas* sp., *Klebsiella* sp., *Staphylococcus* sp., *Leptospira* serovars
  - History of placental injury with moderate/severe endometritis or fibrosis

- **Rickettsiae**
  - *Ehrlichia risticii*—PHF

- **Fungi**—placentitis caused by *Aspergillus* sp., *Candida* sp., or *Histoplasma capsulatum*

- **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
  - **Exposure to ETC setae in conjunction with MRLS theorized associated with microscopic bowel puncture and bacteremic spread to fetus and/or placenta

**Historical**
- One or more:
  - Vaginal discharge—mucoid, hemorrhagic, serosanguinous
  - Premature udder development; dripping milk
  - Anorexia or colic; GI disease
  - Failure to deliver on expected due date
  - Recent (1–16 weeks before presentation) systemic infectious disease
  - Other mares, recent abortions
  - Inadequate EHV-1 prophylaxis
  - History of placental injury with moderate/severe endometritis or fibrosis
  - None/excessive abdominal distention consistent with gestation length
  - Behavioral estrus, pregnant mare—may be normal depending on gestation length, time of year, gestation length at time of loss
  - Climatic and environmental conditions favor increased ETC (*Malacosoma americanum*) populations and development of MRLS in early and late pregnant mares
  - Possibly geographical location if MRLS and nocardioform placentitis

**Physical Examination**
- Fetal parts/placental structures protruding through vulvar lips; abdominal straining or discomfort
- Vulvar discharge (variable appearance); premature udder development, dripping milk
- A previously documented pregnancy is inapparent at next examination; evidence of fetal death by palpation, transrectal or transabdominal U/S
- Anorexia, fever, signs of concurrent systemic disease, especially with endotoxemia, dystocia, RFM
- Evidence of placental separation with transrectal or transabdominal U/S
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 is rare 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 placentary 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, fibrin)
  - Endophyte-infected tall fescue pasture; exposure to ergotized grains, small cereals; no mammary development (agalactia, if term is reached); phytoestrogens; xenobiotics

- Other Causes—Signs of Labor or Abdominal Discomfort
  - Normal parturition
  - Dysuria unassociated with abortion
  - Post partum uterine artery rupture
  - Colic associated with uterine torsion
  - Discomfort associated with hydronephrosis or pruritic tendon rupture
  - Colic unassociated with reproductive disease

- Other Causes—Vulvar Discharge
  - Normal parturition
  - Dystocia unassociated with abortion
  - 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).

- At >100 days, RIA detects both progesterone (very low >day 150) and cross-reacting 5α-pregnanes of uterofetoplacental origin. Acceptable levels of 5α-pregnanes vary with stage of gestation and laboratory used.

- 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 gestational abnormalities

- DIAGNOSTIC PROCEDURES
  - Pathology, Serology, Molecular Techniques, and Culture
    - If fetus and membranes are available, sample:
      - 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, lung, 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
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 necrosis, 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 fibronecrotic 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

**Endotoxemia**
- Fetus minimally autolyzed

**Rickettsiae**
- Ehrlichia risticii
  - Gross—placentitis
  - Histopath—typical fetal lesions include colitis, periportal hepatitis, lymphoid hyperplasia, and necrosis

**Protozoa**
- Sarcocystis neurona—aneudotal reports on histopath, aborted fetuses from EPM plus mares

**MRSL**
- Path findings—similar to bacterial infections

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 endotoxemia 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, uterine culture and cytology, endometrial biopsy
- Base treatment on clinical results. Uterine 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 open 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.
- 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.
- Pregnancy—significant impact on mare’s survivability and future fertility.
- Complications—guarded for pregnancy maintenance with endotoxemia and placentitis.

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.

MISSCELLANEOUS

ASSOCIATED CONDITIONS
- Abortion, noninfectious
- Dyspnea
- Endometritis
- EPM
- EVA
- Menstris
- Pericarditis, MRLS
- Placental insufficiency
- Placitiris
- PHF
- Premature placental separation
- RFM

AGE-RELATED FACTORS
Immunologic status of young mares

SEE ALSO
- Abortion, noninfectious
- Dyspnea
- Endometrial biopsy
- Endometritis
- Fetal stress/stability
- High-risk pregnancy
- Menstris
- 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
- TRP = transrectal palpation
- U/S = ultrasound, ultrasonography

Suggested Reading

Author Tim J Evans
Consulting Editor Carla L. Carleton
**Abortion, Spontaneous, Noninfectious**

**BASICS**

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

**PATHOPHYSIOLOGY**
- Fetal death/pregnancy termination from some intrinsic structural or functional defect or exposure to xenobiotics
- Fetal expulsion < 60 days of gestation after CL loss as a result of endometritis or other factors
- Fetal death/exploration by placental insufficiency or separation
- Fetal stress, dead twin fetuses, maternal stress, or combination
- Fetal reabsorption, mummification, marnification, abortion, or live fetus incapable of extrauterine survival

**SYSTEM AFFECTED**
Reproductive

**INCIDENCE/PREVALENCE**
- 5%–15% spontaneous abortion, multiple pregnancies
- Breed predisposition for twinning

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

**Historical**
- Signs consistent with labor at unexpected stage of gestation
- Dystocia, birth of nonviable foal
- Vaginal discharge—mucoid, hemorrhagic, or serosanguinous
- Premature udder development; dripping milk
- Anorexia or colic
- Recent systemic disease
- Moderate/severe endometritis or fibrosis
- Failure to deliver on expected due date
- None/Severe abdominal distention consistent with stage of gestation
- Behavioral estrus in pregnant mare—normal for stage of gestation; dependent on time of year and stage of pregnancy when lost
- Geographical location—endophyte-infected fescue pastures/hay and/or ergotized grasses or grains

**Physical Examination**
- Fetal/placental structures protruding through vulvar lips; abdominal straining or discomfort
- Vulvar discharge (variable appearance); premature udder development, dripping milk
- Previously diagnosed pregnancy absent at next examination; fetal death determined by palpation or transrectal/transabdominal U/S
- Twin fetuses identified by transrectal/transabdominal U/S
- Evidence of placental separation or hydrops of fetal membranes during transrectal/transabdominal U/S
- Signs of concurrent, systemic disease, dystocia, or RFM
- Note—Signs variable. Mares pregnant at < 80 days of gestation may apply

**CAUSES**
- Twins
- Twin pregnancies that persist > 40 days = 70% end in abortion/stillbirth

**Luteal Insufficiency/Early CL Regression**
- Anecdotal, somewhat controversial
- Caused by increased levels of luteal progesterone at < 30 days of gestation

**Placental Abnormalities**
- Umbilical cord nomenclature—cord twins are normal, must be evidence of vascular compromise, e.g., cord thrombus, to confirm diagnosis
- Long-umbilical cord/cervical pole ischemia disorder
- Confirmed body pregnancy
- Placental separation
- Villous atrophy or hypoplasia
- Hydrops

**Fetal Abnormalities**
- Developmental abnormalities—hydrocephalus, anencephaly
- Fetal trauma
- Chromosomal abnormalities

**Maternal Abnormalities**
- Concurrent maternal disease, maternal stress
- Trauma
- Malnutrition—starvation; selenium deficiency
- Severe maternal anemia—aneuploidal
- Maternal stress, mild malnutrition, abnormal

**Maternal chromosomal abnormalities
- Chromosome abnormalities
- Maternal chromosomal abnormalities

**Xenobiotics**
- Ergotamine alkaloids associated with fescue toxicosis or ergotism (prolonged gestation is more common)
- Phytoestrogens—aneuploidal
- Xenobiotics causing maternal disease—cardiovascular disease, milk, and RFM
- Xenobiotics causing maternal disease—cardiovascular disease, milk, and RFM
- Xenobiotics causing maternal disease—cardiovascular disease, milk, and RFM
- Xenobiotics causing maternal and/or fetal disease—originally suspected with respect to MRLS, considered less likely at present time
- Possible deleterious effects of medications on pregnancy—EFM therapies (anecdotal)
- Repeated large doses of corticosteroids during late gestation
Equine, Second Edition

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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
  - Dysatonia 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
  - Dysatonia unassociated with abortion
  - Normal estrus
  - Endometritis
  - Menstrus or RFM
  - Mutocerma 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 ng/mL, depending on reference lab
  - >100 days of gestation, RIA detects progesterone (may be very low >150 days)
  - 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 T4 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 placentitis 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 progesterin and antibiotic therapy.
- Mares with abortion history—evaluate and treat before rebreeding; progesterin 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.
- Signs of fescue toxicosis or endometritis can be treated with D2-dopamine receptor antagonists; 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**

See 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.
- **Altrenogest**
  - Start later in gestation, continue longer, or use for only short periods of time depending on serum progesterone levels during the first 80 days of gestation (>1 to >4 ng/mL), clinical circumstances, risk factors, and clinician preference.
  - If used near term, altrenogest often discontinued 7–14 days before expected foaling date, unless otherwise indicated by assessment of fetal maturity/viability or questions arise regarding accurate gestational age.

**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.
- Anecdotal success of supplemental progesterone 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, septicemia, 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/distress/viability
- High-risk pregnancy
- Hydrops allantochorion
- Metritis, postpartum
- Multiple pregnancies
- 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
- MERS = 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**

**BASICS**

**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.

**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.

**DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS**
- Consider all causes of equine hemolytic anemia, which include oxidant poisons, EIA, immune-mediated hemolytic anemia, piroplasmosis, and liver failure.
- Hemolytic anemia accompanied by Heinz bodies and/or methemoglobinemia indicates oxidant toxicosis. The most common causes in horses are onions, red maple, and phenothiazine anthelmintics, which can only be differentiated by a history of ingestion.

**CBC/BIOCHEMISTRY/URINALYSIS**
- Interpretation of laboratory findings may be difficult because of the hemolysis, with resultant discoloration of the serum and urine.
- Decreased PCV, hemoglobin, and erythrocyte count are consistent with intravascular hemolysis with hemoglobinuria.
- Heinz bodies are not present in all cases. They may be seen on routinely-stained blood smears but are more apparent in new methylene blue-stained smears.
- Serum bilirubin, especially unconjugated bilirubin, is increased because of hemolytic anemia and inappetence.
- Urinalysis results include proteinuria and hemoglobinuria, with few or no intact erythrocytes.
- Serum albumin and total protein result from dehydration.
- BUN and creatinine increase if a pigment nephropathy develops and causes acute renal failure.
- Elevated 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

**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.
- Clinical 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.

**DIFFERENTIAL DIAGNOSIS**
- Consider all causes of equine hemolytic anemia, which include oxidant poisons, EIA, immune-mediated hemolytic anemia, piroplasmosis, and liver failure.
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- BUN and creatinine increase if a pigment nephropathy develops and causes acute renal failure.
- Elevated liver enzymes and creatine phosphokinase may occur, probably secondary to cell damage caused by anemia-induced hypoxia.
- Eccentrocytes and ghost cells have been reported.
OTHER LABORATORY TESTS
The percentage of methemoglobin in the blood often is elevated.

IMAGING
N/A

OTHER DIAGNOSTIC PROCEDURES
N/A

PATHOLOGICAL FINDINGS
- Gross findings include generalized icterus, enlarged spleen, and discolored kidneys. Petechiae and ecchymoses may be present on serosal surfaces.
- Histopathologic findings include erythrophagocytosis 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

DRUG(S) OF CHOICE
- 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 tree.
- 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.

PREGNANCY
- 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 homeostasis producing increased H⁺ 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 excretion of phosphates and uric acid (irritable acidity).
- Resorption of HCO₃⁻ occurs when H⁺ is secreted—90% in the proximal tubule, the remainder in the distal nephron.
- The minimum pH (4.3) of the tubular fluid limits secretion of H⁺.
- Titratable acidity increases minimally in acidic patients.
- In most species, production of ammonia with accumulation of acids are the major mechanisms 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.
- Carbonate 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 by hyperventilation.
- Respiratory compensation responds within minutes and is effective for mild and moderate acidemia.
- Definitive 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 decrease H⁺.
- Decreased respiratory muscle strength can lead to muscle fatigue and worsening metabolic stress, especially in neonates.

**Cardiovascular**
- Decreased cardiac contractility
- May predispose to arrhythmias
- Vasodilation of arterioles; constriction of veins is regulated by intracellular and extracellular cations.
- Plasma bicarbonate level is important to determine the cause and to guide treatment.
- Cardiac function may be impaired by hypoxemia, hepatic damage, etc.

**Neuroendocrine**
- Catecholamine release
- CNS depression
- Vascular effects may be offset by catecholamine effects.
- Excessive sodium heparin may alter HCO₃⁻ levels via changes in ventilation.

**Respiratory**
- 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
- Decreased affinity for oxygen-hemoglobin binding, enhancing release of oxygen to the tissues
- Increased protein catabolism
- Increased ionized calcium concentration

**SIGNALMENT**
- Any species

**SIGNS**
- Historical and 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 obstructive RFA results in HCO₃⁻ loss both directly and indirectly, depending on the type of tubular dysfunction.
- Respiratory 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 hypoxemia caused by inadequate intake, hemorrhage, septicemic or hypovolemic fluid loss or sequestration (e.g., ureteroliths, peritonitis, pleuritis, ascites, nonneutrophilic types of colic), strangulating lesions of the GI tract, endotoxemia, 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 endotoxemia.
- 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.
- Acetate or end-stage hepatic failure may result in metabolic acidosis due to failure of the detoxification systems of the liver.
- Asphyxia or asphyxiation may cause multiorgan damage or failure, which can result in metabolic acidosis in neonates.

**ACUMULATION OF EXOGENOUS ACIDS IS UNCOMMON, AS THIS IS USUALLY CAUSED BY INGESTION OF TOXIC SUBSTANCES; IT MAY BE SEEN WITH SALTCACTUS, PROPYLENE OR ETHYLENE GLYCOL, PARALDEHYDE, AND METHANOL.**

**DIARRHEA**
- Inability to excrete H⁺ develops acidosis more readily, as acetazolamide is a carbonic anhydrase inhibitor that causes increased HCO₃⁻ excretion.
- Highly anionic diets have been suggested to induce metabolic acidosis in equine.

**DIFFERENTIAL DIAGNOSIS**
- Some causes of metabolic acidosis can be identified on physical examination (i.e., diaphoresis, dehydration, colic with ischemic lesions).
- Decreased HCO₃⁻ levels are also seen in conditions with chronic respiratory alkalosis.
- Kidney failure is also a source of acidosis; however, the pH will be normal or mildly increased.

**LABORATORY FINDINGS**

**Drugs That May Alter Lab Results**
- Excessive anticoagulant may falsely decrease levels via changes in ventilation.
- Excessive sodium heparin may alter HCO₃⁻ levels via changes in ventilation.
- Excessive or inappropriate fluid therapy, especially in neonates, produces free-water excess and dilutional acidosis.

**ENDOTOXEMIA OR CARDIOVASCULAR DISEASE MAY PRECLUDE THE USE OF AN NEUTRON AMOUNT OF LIQUID**
- TPN can lead to metabolic acidosis when cationic (i.e., lysine, arginine) or sulfur-containing amino acids are metabolized, as H⁺ is formed.
- Endotoxemia produces acidosis via several mechanisms—hypotension, decreased cardiac contractility, tissue ischemia, fluid shifts, hypoxemia, hepatic damage, etc.

**RISK FACTORS**
- Patients with chronic renal failure or chronic hypoxemia (i.e., COPD) may be at greater risk for acidosis with progression of their primary problem or if acid load develops for other reasons.
- Excess sodium heparin may alter HCO₃⁻ levels, because it is an acidic compound.

**Disorders That May Alter Lab Results**
- With poor peripheral perfusion or cardiovascular shock, levels 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/UROLYSIS**
- 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.
• Proportionate changes in sodium and chloride levels occur with alterations of fluid balance.
• Normal sodium levels with hyperkalemic or hypokalemic hyperchloremia indicate acid-base imbalance.
• Disproportionate changes in Na⁺/Cl⁻ usually are associated with simultaneous acid-base imbalance and hydration abnormalities.
• Alkalosis/acidosis levels are not considered when calculating the anion gap; however, because proteins are weak acids, hyperproteinaemias can produce the condition—dehydration, chronic infection, and nephrosis.

Urinalysis and fractional excretion of electrolytes are useful in cases of renal failure and RTA.

Horses Affected with Hyperchloremia and Normal AG

- Loss of HCO₃⁻—diabetes, type II RTA, and primary respiratory alkalosis; however, severely affected colic 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
- GI—renal failure, type I or IV RTA, and acetoacetamide acidemia

Horses Affected with Increased AG

Accumulation of unmeasured anions:
- Lactate—conditions with hyperkalaemia or hypokalaemia or exercise, severe exertional rhabdomyolysis, malignant hyperthermia
- Phosphates, sulfates, and organic acids—renal tubular acidosis
- Rebound alkalosis or cerebral acidosis is possible with CL⁻ administration.

OTHER LABORATORY TESTS
Total CO₂
- Measured by many labs using the same sample submitted for electrolytes.
- Cosmologically approximate HCO₃⁻, because most CO₂ in the blood as bicarbonate
- Respiratory alkalosis also decreases TCO₂; differentiation can only be made with blood gas analysis.
- Anuric rapidly with minimal room-air exposure within the sample tubes as CO₂ will decrease.

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

DIAGNOSTIC PROCEDURES
Bupivacaine for suspected organ failure and cytology and microbiology of caudal 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 hyperkalaemia 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

DRUGS OF CHOICE
- Alkalizing therapy is reserved for patients with a pH < 7.2 that persists following fluid therapy or volume replacement.
- Sodium bicarbonate is most frequently used.
- The bicarbonate deficit is calculated as follows: Base deficit × body weight (kg) × 0.3 (ECF space) ÷ 0.5 (mEq) = HCO₃⁻ deficit (mEq)
- A negative BE or 24 – (total CO₂ or 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 horses.
- Correction to a pH of 7.2 and BE ≥ −5 is usually adequate, especially with organic acids, 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 compromise, because the CO₂ that is generated may not be eliminated, causing a further decrease in pH.
- Hyperventilatory solutions may cause vascular symptoms and affect toxicity of the CSF.
- Sodium load may affect blood volume in neonates and patients with compromised renal, neurologic, or cardiac function.
- Renal alkalosis or carbonic acidosis may be reversed over too-rapid administration of bicarbonate since both CO₂ and HCO₃⁻ can cause the blood-brain buffer.

POSSIBLE INTERACTIONS
Alkalining therapies (i.e., HCO₃⁻). Lactate can combine with Ca²⁺ in crystalloid solutions that form a harmful precipitate.

ALTERNATIVE DRUGS
- Replacement IV fluid solutions with other alkalinizing 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 (1–2 g NaHCO₃ per kg in adults without ileus) have been used as primary therapy or as 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.
**Acidosis, Respiratory**

### Basics

**Definition**
- Increase in blood PaO₂
- 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 (65–79%) combines with water almost instantaneously to form bicarbonate ion and hydrogen.
- Most CO₂ is transported in the blood as bicarbonate. Some is bound to proteins, especially desaturated 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 PaCO₂ as measured by blood gases depends on the dissolved portion.
- The chemical components of the carbonic acid equilibrium are:
  \[ \text{CO}_2 + \text{H}_2\text{O} = \text{H}_2\text{CO}_3 = \text{H}^+ + \text{HCO}_3^- \]
- 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 even small disturbances by increasing minute ventilation.
- Respiratory acidosis results from disease or alteration 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 directly involved in gas exchange, by causing hyperventilation, 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**

**Historical Findings**
- Respiratory noise may be heard, especially with exercise, in cases of upper airway obstruction.
- Exercise intolerance may be reported with many causes.

**Physical Examination Findings**
- None if minute ventilation is increased via increased tidal volume; if not, tachypnea may be present.
- Anesthetized animals with very high levels of CO₂ may have increased rate or depth of respiration.
- Decrease or absence of airway sounds may be found at auscultation in cases with damage or disease of the chest wall or thorax.
- Abnormal sounds may be present with pulmonary disease.

**Causes**
- Nasal edema, cysts, mass lesions, or infection of the paranasal sinuses, laryngeal or pharyngeal paralysis, soft-palate displacement, or palate masses or collapse all cause upper airway obstruction and impede airflow into the lungs.
- Injury or disease of the thorax, diaphragm, or pleura may restrict movement of the chest wall or respiratory muscles or lead to atelectasis because of fluid, blood, air, or intestinal organs in the pleural space.
- Ureteroliths, diseases producing portal hypertension (e.g., cardiac or liver failure), or pleural fluid may restrict movement of the chest wall or respiratory muscles or lead to atelectasis because of fluid, blood, air, or intestinal organs in the pleural space.

**Differential Diagnosis**
- Physiologic and pathologic 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 acidosis may have a compensatory hypercapnia—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**

**Drugs That May Alter Lab Results**
- Without 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 PaO₂ level, because the sample equilibrates with the air.

**Risk Factors**
- General anesthesia, heavy sedation
- Pregnancy, which increases the volume of abdominal contents and may predispose to hypercapnia under anesthesia.
- Prolonged recumbency
- History of malignant hyperthermia in related individuals.
- Prematurity, dystocia, asphyxia or sepsis, persistent fetal circulation, or pulmonary hypertension in neonates.

**Dogs That May Alter Lab Results**
- Spontaneously
- Persistently high levels of CO₂ (above 50 mm Hg) upon awakening may result from a light plane of anesthesia, hypoxemia, metabolic acidosis, or faulty anesthetic rebreathing systems.

**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 PaO₂ level, because the sample equilibrates with the air.
Emergency therapy occasionally may be necessary when hypoventilation is severe (i.e., PaCO₂ > 50 mm Hg). Controlled ventilation is most effective.

With severe lung disease, treat hypercapnia with controlled ventilation. This generally is not feasible in adults, but neonates respond well. Heavy sedation or muscle relaxant therapy may be necessary in some individuals; however, most relapse once respiratory function improves.

Cellular metabolism of RBCs continues when hypoventilation is severe (i.e., PaCO₂ > 60 mm Hg). This may lead to air trapping in alveoli, which may rupture.

Acidosis, Respiratory

**MEDICATIONS**

**DRUGS OF CHOICE**

- **Doxapram**
  - A respiratory stimulant that may be a useful adjunct (0.5–1 mg/kg IV or an infusion of 0.02–0.05 mg/kg per min) in emergency resuscitation and some patients, especially foals with neurologic or muscular weakness.
  - Anesthetized patients who are breathing poorly may respond temporarily to its effects, but controlled ventilation, decreasing depth, and anesthetic reversal are more specific and appropriate therapies.
  - Not indicated for healthy patients being weaned from controlled ventilation

**Other Drugs**

- Anti-inflammatory therapy with corticosteroids or bronchodilator therapy with β₂-agonists or xanthine derivatives may be useful in patients with allergic airway disease and COPD once environmental factors are controlled.

**CONTRAINDICATIONS**

- Controlled ventilation may cause 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, hoses, and so on.
- Oxygen toxicity can develop with inspired PaO₂ > 50% or if PaCO₂ > 100 mm Hg is maintained for prolonged periods (10–12 hr).

**POSSIBLE INTERACTIONS**

- Used in emergency situations.

**FOLLOW-UP**

- 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.
- Hypercapnia 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.

**ASSOCIATED CONDITIONS**

Disorders that result in metabolic alkalosis

**AGE-RELATED FACTORS**

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

**ZOOLECTIC POTENTIAL**

- N/A

**PREGNANCY**

- See Risk Factors.

**SYNONYMS**

- Hypercapnia
- Hypercarbia
- Hypercapnemia

**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

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 flat 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.

DIFFERENTIAL DIAGNOSIS
Foals
- Any other cause of neonatal sepsis including bacterial, viral, and fungal agents
- Pneumonia and pleuroneumonia 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 insults and concurrent illnesses.
- Trauma may predispose to abscess formation.

Adults
- Any other cause of fever
- Any other cause of peritonitis
- Any other bacterial, viral, or fungal agent causing pneumonia or pleuroneumonia
- Other causes of respiratory distress, fever, coughing, and nasal discharge should be considered, including:
  - Sinusitis
  - Guttural pouch empyema
  - Heaves (recurrent airway obstruction)
  - Inflammatory airway disease
  - Interstitial pneumonia
  - Mycoplasma infections
  - Neoplasia
  - Dysphagia

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, hyponatremia, 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)
- Uralysis 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 celisfor sodium 10 mg/kg IV QID. Monitor plasma creatinine concentration. Therapeutic drug monitoring desirable.
• Foals with systemic inflammatory response syndrome (SIRS) or multiple organ dysfunction syndrome (MODS) may require more intensive fluid management and inotropic therapy.
• Foals with severe respiratory distress may require assisted ventilation.
• Regional limb perfusion and/or direct instillation with antimicrobials of choice for septic joints.

**Adults**
Antimicrobial therapy based on culture and sensitivity results

**MISCELLANEOUS**

Antimicrobial therapy should be modified based on response and cultur/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**

**Author** Pamela A. Wilkins
**Consulting Editor** Ashley G. Boyle and Corinne R. Sweeney
ACUTE ADULT ABDOMINAL PAIN—ACUTE COLIC

BASICS

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 mesentery, 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.
• Intraluminal lesions (impaction, foreign body, concretions), extraluminal lesions (adhesions, stricture), mural lesions (thickening), as well as spasmatic 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 to specific problem (e.g., intussusception of the small intestine is more commonly seen in young horses; polycyclic lipomas are commoner on older horses; large colon torsion commonly seen around parturition in mares; pain from the reproductive tract is seen in pregnant or postpartum mares and in stallions in 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, extended neck and rolling of the upper lip in Flhmen-like response, teeth grinding
• Moderate—pawing at the ground, flank watching, groaning, pursue for urinating but only a small quantity of urine is passed, leaping 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—increase, decrease or absence in gut motility, gas-filled resonant viscus on percussion. Gastric reflux on passage of the nasogastric tube is most commonly associated with lesions located at the level of the stomach or small intestine.
• Abdominocentesis on rectal examination—detection of a viscus by gas, liquid, or fluid displacement of a viscus, thickening of the intestinal wall; uterine or renal abnormalities; findings will assist in the differentiation among problems involving the small intestine, large colon, cecum, small colon, or nongastrointestinal lesions

CAUSES
Gastrointestinal
• Gastric—gastric ulcers, gastric dilation or impaction, gastric rupture, gastric tumors
• Small intestines—nonstrangulated obstructive lesion: duodenal ulcer, duodenal/proximal jejunal ulceration, jejunoileal ulceration, ileal ulceration, jejunoileal stricture. Strangulated obstructive lesion: incarceration of a segment of the small intestine into the mesenteric foramen, a space/rent in the mesentry/mesocolic ring/gastric portal ligament, strangulation by a lipoma, volvulus, adhesions, etc.
• Large intestines—nonstrangulated obstructive lesion: ulceration, colitis, impaction, idiopathic gas distention, mild displacement, nephropathic emaciation, enterolith, adhesions, sand impactions. Strangulated obstructive lesion: volvulus, hematomas, incarceration, thromboembolic infarction
• Cecum—nonstrangulated obstructive lesion: impaction, adhesions. Strangulated obstructive lesion: incarceration, strangulation, lipoma, submucosal hematoma, thromboembolic infarction
• Rectum—rectal impaction, teat prolapse

Other causes of abdominal pain that might mimic pain that originates from the abdominal cavity include musculoskeletal injuries, renal/urethral/bladder urinary calculi, cystitis, renal tuberculosis, hepatic abnormalities—hepatitis, hepatocholangitis, pyometra—peritonitis, hemoperitoneum

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

DIAGNOSIS

N/A

DIFFERENTIAL DIAGNOSIS
Other causes of pain that might 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. Leukopenia 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 hypocalcemia can be present, especially if the horse has been anorectic or is a lactating mare. Hypochloremia and hypoproteinemia 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.

OTHER LABORATORY TESTS
Abdominal Paracentesis
• Normal fluid has a pale, clear yellow color.
• Turbidity of the sample indicates an elevation of WBCs, RBCs, or contamination with intestinal contents.
• Increase of the protein level and WBCs is indicative of primary peritonitis or secondary to morphologic change of the viscus.
• A sugary substance is probably indicative of intra-abdominal bleeding or a strangulated obstructive lesion.
• A foul-smelling solidish-brown fluid with an increase in the RBC, WBC, and protein is indicative of presence of necrotic bowel.
• Presence of plant materials in the absence of an enterocentesis suggests intestinal rupture.
• Few leukocytes or cells should be present if an enterocentesis was performed.
Acute Adult Abdominal Pain—Acute Colic

**TREATMENT**

- Horses should be taken off feed until diagnosis of the underlying problem.
- Indication for exploratory laparotomy includes: signs of severe abdominal pain, peritonitis, strangulated lipoma, or strangulation; moderate to severe abdominal distention, fever or progressive reduction in gut motility, progressive increase in heart rate or heart rate above 60–70/min, cardiovascular compromise or dehydration, presence of moderate to severe gas distention or of a displacement of the large colon on rectal examination, gastric reflux, abnormal parasitic findings, or presence of severe impaction of the large colon or the caecum. Animals presenting with these signs should be referred to a surgical facility.
- Supportive treatment for medical and surgical cases includes intravenous fluids, gastric decompression if necessary, electronic repletion, and control of the abdominal pain.

**CONTRAINDICATIONS**

Acetaminophen is contraindicated due to its peripheral vasodilatory effect.

**PRECAUTIONS**

Repeat use of α1-blockers and bumetanide 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 estranged lipoma and epiploic foramen entrapment; pregnant mares are more predisposed to large colon torsion; and younger horses are more predisposed to ulcer problems, submucosus, and ascral impactions.

**PREGNANCY**

Mares in late gestation or in the postpartum period are predisposed to large colon torsion. Parturition can present clinical signs similar to a gastrointestinal accident.

**SYNONYM**

Colic

**ABBREVIATIONS**

- PCV = packed cell volume
- TP = total protein

**Suggested Reading**


**Author**

Olimpo Oliver-Espinosa

**Consulting Editors**

Henry Stimpfli and Olimpo Oliver-Espinosa
Acute Epiglottitis

**BASICS**

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

**SIGNMENT**
- Primarily affects horses (2–10 years) in active race training or other horses undergoing repeated, strenuous exercise
- Occasionally seen in older horses (15–18 years) associated with neoplasia
- No known breed or sex predilection

**CAUSES AND RISK FACTORS**
- Exercise intolerance
- Repeated, strenuous exercise may induce epiglottitis
- Swallowing is difficult or stimulates coughing during eating
- Infections is unknown.

**IMAGING**
- Imaging is not usually performed.
- Additional laboratory tests are not typically performed.

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

**DIAGNOSIS**
- The diagnosis is established based on upper respiratory tract noise and exercise intolerance.
- Endoscopy may reveal intermittent or persistent dorsal displacement of the soft palate, which may need surgical treatment—laryngeal cleft, soft-palate trim, or excision of fibrous connective tissue on the subepiglottic surface.

**EXPECTED COURSE AND PROGNOSIS**
- 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
- Exercise intolerance

**ABBREVIATION**
- DMSO = dimethylsulfoxide

**SUGGESTED READING**
ACUTE HEPATITIS IN ADULT HORSES (THEILER’S DISEASE)

OVERVIEW
Many conditions can potentially lead to acute liver failure in adult horses, with the most common being a syndrome occurring 4–10 weeks after animals have received an equine biologic. This is usually tetanus toxoid, but other agents have been implicated, e.g., Clostridium novyi and bacterial cholangiohepatitis, which can manifest in various ways that may change during the course of the disease. Originally, there may be subtle changes in behavior, progressing to excitement or depression with head pressing. Some may wander aimlessly around the stall or paddock. Frequent yawning has been reported in some cases. If animals live long enough and are outside in the sun, they may develop photosensitive dermatitis on white parts of the body. Possible humoral diathesis or hemolysis may be present. 

CAUSES AND RISK FACTORS
Most commonly associated with administration of an equine biologic 4–6 weeks before the onset of signs; however, not all cases have been reported to an equine biologic. Some epidemiologic evidence suggests that some cases may result from an infectious agent, probably a virus. None has been isolated as yet; however, attempts to reproduce the disease with material from affected horses have failed. Occasional cases caused by Clostridium novyi type B have been described.

DIAGNOSIS
DIFFERENTIAL DIAGNOSIS
Acute onset of icterus in adult horses has multiple causes—phosphatic, hepatic, or postphagic; serum biochemistries and CBC assist in differentiating these causes.
• Phosphatic—red maple leaf toxicity, wild onion toxicity, phenothiazine toxicity, and satyrine poisoning
• Hepatic—anoxia, Theiler’s disease, C. novyi, bacterial cholangihepatitis, EIA, EVA, Strepos sp. migration, ascan toxicosis, and halogenated hydrocarbons.
• Postphagic—cholethiasis and other causes of biliary obstruction. Signs of hepatic encephalopathy can be very similar to those of several acute neurologic diseases—rabies, EEE, WEE, and acute protural myonecrophagitis.

SIGNS
• Usually sudden in onset and rapidly progressive, with death occurring 2–6 days after onset of signs in some cases. • Horses are often very weak and pass dark urine caused by the presence of bilirubin.
• Many have signs of hepatic encephalopathy, which can manifest in various ways that may change during the course of the disease. Initially, there may be subtle changes in behavior, progressing to excitement or depression with head pressing. Some may wander aimlessly around the stall or paddock. Frequent yawning has been reported in some cases. If animals live long enough and are outside in the sun, they may develop photosensitive dermatitis on white parts of the body.
• Possible humoral diathesis or hemolysis may be present.

CAUSES AND RISK FACTORS
Most commonly associated with administration of an equine biologic 4–6 weeks before the onset of signs; however, not all cases have been reported to an equine biologic. Some epidemiologic evidence suggests that some cases may result from an infectious agent, probably a virus. None has been isolated as yet; however, attempts to reproduce the disease with material from affected horses have failed. Occasional cases caused by Clostridium novyi type B have been described.

DIAGNOSIS
DIFFERENTIAL DIAGNOSIS
Acute onset of icterus in adult horses has multiple causes—phosphatic, hepatic, or postphagic; serum biochemistries and CBC assist in differentiating these causes.
• Phosphatic—red maple leaf toxicity, wild onion toxicity, phenothiazine toxicity, and satyrine poisoning
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ACUTE RENAL FAILURE (ARF)

BASICS

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

PATHOPHYSIOLOGY
- Usually prerenal or renal; most commonly due to hemodynamic or nephrotic insults.
- Except for necrotic bladder rupture and urolithiasis, postrenal failure is uncommon in horses (see Ureterolithiasis and Urolithiasis).
- Prolonged by decreased GFR in damaged glomeruli and tubular obstruction with desquamated tubular epithelial cells and debris.

SYSTEMS AFFECTED
- Renal/urologic—failure.
- Endocrine/metabolic—disturbances in electrolyte and acid-base homeostasis.
- GI—insufficiency, possible diarrhea, and increased risk of ulcers.
- Nervous/neuromuscular—occasional ataxia or tremors.
- Musculoskeletal—acute laminitis in severe cases; often refractory to treatment.

GENETICS
N/A

INCIDENCE/PREVALENCE
Low

GEOGRAPHIC DISTRIBUTION
NA

SIGNALMENT
Breed Predictions
N/A

Mean Age and Range
- Foals <30 days of age (especially when receiving nephrotoxic medications) may be at greater risk, but all ages can be affected.

Predominant Sex
None

SIGNS
General Comments
Clinical signs of ARF are vague and nonspecific.

Historical
- Often secondary to other problems leading to hypovolemia and renal ischemia—colic, diarrhea, prolonged exertion, rhabdomyolysis, or sepsis/endotoxemia.
- Previous administration of nephrotoxic drugs.

Physical Examination
- Leukopenia, anemia, dehydration, edema, ulcers, or urologic disease.
- Severity of lethargy and anorexia often are greater than would be expected for the primary disease process.
- Renal examination may reveal an enlarged, painful left kidney.
- Laminitis, often rapidly progressive.
- Markedly azotemic patients may have neurologic deficits—ataxia, hyperesthesia, and mental obtundation.

CAUSES
Prerenal Failure
- Hemorrhagic, hypovolemic, or endotoxic shock.
- Prolonged, exhaustive exercise.
- Severe rhabdomyolysis, vasculitis, or hemolytic diseases.
- Disseminated intravascular coagulation.

Intrinsic Renal Failure
- Prolonged duration of above disorders, lack of adequate fluid support, or concurrent use of normal doses of nephrotoxic medications—aminoglycosides, NSAIDs.
- Excessive doses of NSAIDs or prolonged use of gentamicin, particularly in dehydrated horses.
- Other nephrotoxins include heavy metals (e.g., mercury [in counterirritants or blisters], lead, cadmium), endogenous pigments (e.g., hemoglobin, myoglobin), vitamins D and K3, and high doses of oxytetracycline, especially when administered to neonates with fecal decontamination.
- In occasional cases, infectious agents—Escherichia coli in neonates; Leptospira spp. in all age groups.

RISK FACTORS
- Renal hypertension—Exposure to nephrotoxins, particularly in patients with dehydrated or primary renal disease.

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS
- All conditions leading to hemolytic, hypovolemic, or endotoxic shock; severe rhabdomyolysis, vasculitis and hemolytic diseases; or disseminated intravascular coagulation.
- Prerenal failure—oliguria with concentrating urine (specific gravity >1.035) and rapid correction of azotemia with rehydration.
- Postrenal failure—weakness, anemia, or ureterolithiasis.
- Chronic renal failure—weight loss, poor body condition, ventral edema, PUPPD, hyperalgesia, and limited improvement of azotemia with fluid therapy.

CPC/BIOCHEMISTRY/URINALYSIS
- Normal to high PCV, variable leukogram.
- CBC changes reflect underlying primary disease processes, usually on an inorganic basis.
- Urea nitrogen, creatinine, and electrolyte abnormalities (i.e., hyperphosphatemia more common with intrinsic ARF and urolithiasis).
- Serum creatinine and blood urea nitrogen require specific correction.
- USG—high (>1.035) with prerenal failure, low (<1.020) with intrinsic ARF.
- Urea nitrogen more common with azotemia caused by sepsis.

DIAGNOSTIC PROCEDURES

IMAGING
- Transabdominal/Transrectal Ultrasonography
  - Kidneys may be enlarged (diameter >8 cm; length >15 cm), with increased echogenicity of renal cortices.
- Leptospira spp. may be found in horses with ARF attributable to leptospirosis.

PATHOLOGICAL FINDINGS
- Gross—enlargement of kidneys due to hemodynamic and cellular changes; or disseminated intravascular coagulation.
- Histopathological—Glomeruli may be congested and have a cellular infiltrate; tubules have denuded or flattened epithelium and varying amounts of accumulated debris.

TREATMENT

AIMS OF TREATMENT
- Address underlying primary condition.
- Improve GFR with fluid therapy and medications aimed at restoring normal renal function.

APPROPRIATE HEALTH CARE
- Properly recognize and treat all underlying primary disease processes, usually on an inorganic basis for continuous fluid therapy.
- Remove dosage schedule of, and possibly discontinue, potentially nephrotoxic medications.

NURSING CARE

Fluid Therapy
- After initial measurement of body weight, correct estimated dehydration with normal (0.9%) saline or another potassium—poor electrolyte solution over 6–12 hr.
- Fluids may be supplemented with calcium gluconate or sodium bicarbonate if hypokalemia or acidosis requires specific correction.
- Monitor patients for potential respiratory and circulatory failure as evidence of overhydration.
- Monitor urine output. CVP may be helpful in determining fluid replacement plan.
- Use maintenance fluid therapy judiciously in animals not clinically dehydrated.
Oral Electrolyte Supplementation
- Sodium chloride (30 g) can be administered in concentrated 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 azotemia of 2 days.

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

DIET
- Encourage intake by offering a variety of concentrate feeds, 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 N/A

MEDICATIONS

DRUG(S) OF CHOICE
- Judicious fluid therapy is the mainstay of treatment—see Nursing Care
- Furosemide—For oliguria/anuria (i.e., lack of urine output), body weight at least twice daily during the initial 24 hr of treatment and at least daily thereafter
- Mannitol—has been used in the past as an osmotic diuretic agent for oliguria/anuria
- Dopamine—has been used in the past as a vasomotor nephropathy

PRECAUTIONS
- Monitor response to fluid therapy (i.e., urine output)—as little as 40 mL/kg of IV fluids (20 L per 500-kg horse) may produce increases in CVP and significant pulmonary edema in oliguric/anuric patients
- Administer drugs eliminated by renal route—avoid combined administration in anuric patients
- Potassium chloride can be administered to patients with hyperkalemia

CONTRAINDICATIONS
- Anuric drugs—e.g., oxyurans (2–4 mg/kg PO q24 h or cimetidine 5–10 mg/kg IV q8 h in anemic horses) may be useful in decreasing the anemic risk of gastrointestinal ulcer disease

PRECONCAUTIONS
- Monitor electrolyte concentrations (approaching 25 mg/dL) due to consequential to rupture of a uterine artery

DIURETICS
- Judicious use of furosemide and/or mannitol in more critical patients and neonates

ASSOCIATED CONDITIONS
- Neutrons with hypoxic-ischemic multiorgan failure

POSSIBLE INTERACTIONS
- Use of multiple anti-inflammatory drugs (e.g., corticosteroids and one or more NSAIDs) may have additive negative effects on renal blood flow, avoid combined administration in anuric 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 recovering nephrotic medications
- Avoid concurrent use of multiple anti-inflammatory drugs—NSAIDs

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

EXPECTED COURSE AND PROGNOSIS
- Prognosis for recovery varies with the underlying primary disease process
- Prognosis for recovery from prerenal failure and nonoliguric 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 accomplish
- 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 persistently 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
- Acute nephritis
- Acute tubular necrosis
- Vasomotor nephropathy

SUGGESTED READING

AUTHOR
- Harold C. Schott II

CONSULTING EDITOR
- Gillian A. Perkins
Acute Respiratory Distress Syndrome in Foals

DEFINITION
Respiratory distress 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 distress—apnea, cyanosis, tachypnea, increased abdominal and intercostal flaring, increased abdominal and intercostal effort (i.e., “heave” stroke), systemic or pulmonary sepsis/endotheliitis, or inhalation of irritant gases, or smoke may be the initiating insult.
- A manifestation of SIRS, leading to MODS, resulting in anemia, hypotension, disorientation, 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/mortality of 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, 3.3 ± 2.0 months).
- No sex or breed predilections

SIGNS

- Acute or protracted depression, lethargy, fever, tachypnea, pronounced respiratory effort, nasal flaring, increased abdominal and intercostal effort (i.e., “double eyepatch” or “heave” line or parabronchial breathing pattern), and cyanosis.
- Nasal discharge and cough are 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 (intrinsic) 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 involvement of the lungs.
- *Bordetella equi* is cultured from approximately 25% of foals with respiratory distress, and opportunistic pathogens (e.g., *β*-hemolytic strepococci spp., enteric bacteria, *Actinobacillus spp.*, *Pasteurella aeruginosa*, or *P. carinii*) may be involved in ARDS.
- Lesions in affected foals are similar to those in ruminants with atypical intracerebral pneumonia, suggesting that inhalation may contribute to the syndrome.

RISK FACTORS

- Unknown
- Risk factors—precing subclinical to clinical respiratory tract disease; treatment with antimicrobial agents (particularly tetracyclines) or bronchodilators, producing significant interactions in some patients; viral, bacterial, or fungal respiratory tract or systemic infection; heat stress; infected irritant gases and pneumotoxicants; and immunosuppression

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS

- Viral pneumonia—equine influenza, equine viral arteritis, equine herpesvirus 1 and 4, equine parvovirus, and equine adenovirus
- Bacterial pneumonia—*P. carinii* infection
- Pulmonary abscession or granuloma
- Upper airway dysfunction, with aspiration of oropharyngeal fluids
- Ingestion or exposure to xenobiotics

CBC/BIOCHEMISTRY/URINALYSIS

- Common abnormalities—neutrophilic leukocytosis, elevated lactic acid, and azotemia

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.

IMAGING

Thoracic Radiography

- Findings vary, depending on the stage of injury.
- Lesions include prominent interstitial patterns, coalescing to alveolar infiltrates, with superimposed, mixed bronchial patterns of varying severity throughout all lung fields. A prominent miliary reticulonodular pattern is observed commonly in foals with *P. carinii* infection.
- Other changes sometimes include consolidating anteroposterior pneumonia or diffusely distributed pneumonitis in foals with concurrent *R. equi* infection.

Thoracic Ultrasonography

- Consolidation, abscesses, or other lesions in the lungs

OTHER DIAGNOSTIC PROCEDURES

- Because many foals are near death, ante-mortem culture of respiratory secretions before initiation of treatment may not be practical.
- Routine culture of lower airway secretions should be accompanied by cytologic evaluation.
- Examination of tracheal wash or bronchoalveolar fluid reveals acute inflammation, with large numbers of neutrophils some with phagocytosed bacteria.
- Recognition of *P. carinii* is difficult.

APPROPRIATE HEALTH CARE

- Minimize respiratory 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

AIMS OF TREATMENT

- 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.
- Fluids should be selected to correct acid-base status and blood electrolyte abnormalities.
- Balanced electrolyte (e.g., lactated Ringer’s solution) is appropriate initial therapy.
- Insufflation of humidified oxygen (10–15% of inspired gas) is appropriate initial therapy.
L/min) is facilitated by placement of a nasal or transtracheal catheter.

**ACTIVITY**
Reduce the patient’s activity, by confinement to a clean, cool stall with appropriate environmental temperature and humidity control—fans, misters, or swamp coolers.

**DIET**
- Lowering body temperature may improve feed intake.
- Allow nursing foals adequate time with the mare, and provide high-quality feed.

**PREVENTION**
- Education is aimed at prevention.
- Proper management of the mare is imperative, because early handling and training of foals to accept physical examination and daily rectal temperature (preferably in the morning, when ambient environmental temperatures are low) allow early detection of subtle clinical signs.
- Clients should observe mares and foals carefully, on a daily basis, and consult a veterinarian when foals appear to be anorexic 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 thoracic auscultation.
- Evaluate upper airway function to determine patency of the upper airway.
- In HYPP, plus foals, hyperkalemic episodes may result in laryngeal paralysis.

**MEDICATIONS**

**DRUGS OF CHOICE**
- The treatment protocol should specifically address inflammation and hyperthermia.
- Use of corticosteroids in stressed foals demonstrating clinical signs of sepsis in contrast to nonseptic (e.g., P. aeruginosa or E. coli) or traumatic causes should be considered. Low-dose corticosteroids (e.g., dexamethasone sodium phosphate [0.25–1.0 mg/kg q12–24h]) provide potent, short-duration relief. High-dose corticosteroids (e.g., dexamethasone sodium phosphate [2.2–5 mg/kg IV, or SQ q24h]) may be substituted for antimicrobial agents used prior to onset of respiratory distress.

**CONTRAINDICATIONS**
- Discontinue any medications (especially erythromycin/rifampin) and drugs with an effect that may be altered by concurrent therapy with drugs undergoing metabolism by the liver—chryophilene, aminophylline.

**PRECAUTIONS**
- Because sepsis may represent the underlying cause in some foals, overdose of corticosteroids is discouraged.
- Use NSAIDs with caution, due to gastrointestinal and renal effects.

**POSSIBLE INTERACTIONS**
- Drugs such as erythromycin/rifampin that induce or inhibit hepatic drug metabolizing enzymes may alter the disposition of concurrently used medications (e.g., methylenehimeses), 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**
- Aminoglycosides (amikacin sulfate, gentamicin sulfate) used in combination with fl-k, or cefpodoxime and cephalosporins.
- Rifampin in combination with erythromycin, clindamycin, or coamoxiclav may be indicated for R. equi.

**FOLLOW-UP**

**PATIENT MONITORING**
- Reducing body temperature is important as is evaluation of respiratory rate and effort and improvement in mucous membrane color typically indicate clinical improvement.
- Frequent thoracic auscultation may reveal increased bronchovesicular 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 dispersal, and plasma therapy on farms with endemic R. equi.
- Client education regarding use of anthelmintics and vaccination of mares and foals against respiratory pathogens.
- Client education regarding potential adverse effects of use of drugs such as erythromycin during hot weather.

**POSSIBLE COMPLICATIONS**
- Chronic interstitial pneumonia

**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 gradually, whereas increased interstitial pattern resolves over weeks or months.

**MISCELLANEOUS**

**AGE-RELATED FACTORS**
- Can occur at all ages
- In 1- to 8-month-old foals

**SYNONYMS**
- Bronchiointerstitial pneumonia
- ALI
- Interstitial pneumonia
- Respiratory distress

**SEE ALSO**
- Inspiratory dyspnea
- Expiratory dyspnea

**ABBREVIATIONS**
- AII = acute lung injury
- ARDS = acute respiratory distress syndrome
- DIC = disseminated intravascular coagulation
- HYPP = hyperkalemic periodic paralysis
- MHDS = multiorgan dysfunction syndrome
- SIRS = systemic inflammatory response syndrome

Suggested Reading

Authors: Jeffrey Lakritz and W. David Wilson

Consulting Editor: Daniel Jean
**ADENOVIRUS**

**OVERVIEW**
- Causes fatal respiratory disease in Arabian foals with SCID.
- May be a severe pathogen in Fell Pony foals affected by “Fell Pony syndrome.”

**CAUSES AND RISK FACTORS**
- Foals are usually older than 8–10 weeks when clinical signs become present.
- Adenovirus affects primarily Arabians, although other breeds are affected sporadically, in particular, Fell Ponies.
- SCID-affected foals are frequently clinically immunosuppressed for other reasons, such as metabolic disease, congenital heart disease, hypothyroidism, endocrinopathies, genetic, or congenital defects.

**SIGNS**
- Signs are essentially identical to other causes of foal pneumonia and include fever, tachypnea, dyspnea, depression, and diarrhea.
- Mild to moderate diarrhea may also be present.

**DIFFERENTIAL DIAGNOSIS**
- Other viral and bacterial causes of pneumonia 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 zooepidemicus
  - Actinobacillus equuli
  - Pasteurella spp.
  - Klebsiella pneumoniae
  - Salmonella spp.
  - Bordetella bronchiseptica
  - Rhodococcus equi

**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**
- 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 on SRID (see below).

**DIAGNOSTIC TESTING**

**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 zooepidemicus
  - Actinobacillus equuli
  - Pasteurella spp.
  - Klebsiella pneumoniae
  - Salmonella spp.
  - Bordetella bronchiseptica
  - Rhodococcus equi

Other causes of diarrhea in foals include, but are not limited to, bacterial, viral, and parasitic causes.

**CBC/BIOCHEMISTRY/URINALYSIS**
- Ante-mortem diagnosis of SCID is supported by finding appropriate clinical signs in an Arabian foal of the appropriate age with persistent severe lymphopenia (≤500 cells/μL) and the absence of IgM on SRID (see below).

**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 Fell 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.

**ABBREVIATIONS**
- SCID = severe combined immunodeficiency syndrome
- SRID = serial radial immunodiffusion

**SUGGESTED READING**
## Adrenal Insufficiency

### Basics

**Overview**
- Synonymous with hyperadrenocorticism and “steroid let-down 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 deficiency are present.
- Secondary AI—Mineralocorticoid deficiency usually is normal.

**Systems Affected**
- Endocrine
- Cardiovascular
- Renal
- Musculoskeletal
- GI
- Behavioral

**Signalment**
Any age, sex, and breed

**Signs**
- Acute cases—muscular weakness, hypotension, anorexia, lethargy, hypoglycemia, polyuria, polydipsia, mild abdominal pain, colic, and diarrhea
- Chronic cases—depression, anorexia, weight loss, poor hair coat, exercise intolerance, polyuria/polydipsia, mild weight loss, poor hair coat, exercise intolerance, and laminitis

**Causes and Risk Factors**
- Chronic administration of glucocorticoids, mineralocorticoids; 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 colitis
- A normal ACTH stimulation test rules out adrenal insufficiency.

**CBC/Biochemistry/Urineysis**
- Acute AI is characterized by hyperglycemia, hypercalcemia, hyperphosphatemia, decreased sodium-potassium ratio (reference range, > 2:7), and hypoglycemia.
- Additional abnormalities—metabolic acidosis and anemia
- Chronic cases, including secondary AI—mineralocorticoid secretion (i.e., aldosterone) is generally maintained. Therefore, serum electrolytes are within normal limits.

**Other Laboratory Tests**
- With insulin 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, synthetic 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.

**Medications**
- Glucocorticoid and, if necessary, mineralocorticoid replacement. The maintenance dose of prednisolone, which is equivalent to daily corticosteroid 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 adrenocortically altered horses alive.
- Mineralocorticoid replacement with Fludrocortisone may be considered.

**Contraindications/Possible Interactions**
N/A

**Follow-Up**

**Patient Monitoring**
- 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 Courtois
- Consulting Editor Michel Levy
**AFLATOXICOSIS**

**BASICS**

**OVERVIEW**
- Aflatoxicosis is the condition of intoxication by the *Aspergillus* 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.

**SIGNALMENT**
Younger horses are more susceptible.

**SIGNS**
- Ponies given single lethal doses of aflatoxin (2 mg/kg) had increased temperatures, elevated heart and respiratory rates, tenesmus, bloody feces, and tetanic convulsions.
- Some ponies died within 3 days while others lived for 32 days post-dosing. Ponies administered high oral doses (0.4 mg/kg) had increased temperatures, 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.
- Liver damage and associated ill-thrift.
- Liver enzymes should be monitored to evaluate liver function.
- Drugs subject to hepatic clearance should be given cautiously.

**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.
- The inability to obtain samples at the time of disease—history of consumption, biopsy or biopsy to toxicoses—evidence of exposure.
- Diffuse liver disease—history of consumption, biopsy or biopsy to toxicoses—evidence of exposure.
- Senecio spp. cause chronic progressive liver disease—history of consumption, biopsy.
- Theiler’s disease—history, biopsy.
- Alsike clover or kleingrass—imaging and biopsy.
- Pyrrolizidine alkaloid-containing plants—imaging and biopsy.
- Fumonisin-induced mycotoxicosis—detection in feed.

**DIFFERENTIAL DIAGNOSIS**
- Elevated serum hepatic enzyme levels can occur in association with many multi-systemic diseases. Specific causes of hepatic disease include:
  - Fumonisin-induced mycotoxicosis—detection in feed.
  - Aflatoxin—evidence of exposure.
  - Hepatic neoplasia or abscession—imaging or biopsy.
  - Biliary obstruction—serum chemistries and biopsy.
- Necropsy findings include fatty liver, periportal fibrosis, and bile duct hyperplasia.

**OTHER DIAGNOSTIC PROCEDURES**
- Necropsy findings include fatty liver, hemorhagric enteritis and pale swollen kidneys.
- Histologic changes in the liver include fatty degeneration, centrilobular necrosis, portal fibrosis, and bile duct hyperplasia.

**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.

**IMAGING**

**FOLLOW-UP**

**MEDICATIONS**

**DRUG(S) AND FLUIDS**

**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.

**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
African Horse Sickness

BASICS

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 and Mozambique. India, Turkey, Iraq, Syria, Lebanon, Jordan, and Spain have reported outbreaks in the past.
- Geographic range of the disease is limited to moist, warm areas.

SIGNALMENT
- All breeds of horses as well as other equids, such as donkeys and mules, are susceptible.
- There is no apparent breed, age, or sex predilection.
- Angora goats are also susceptible. Zebras and elephants may serve as natural reservoirs of the virus that causes AHS. Dogs fed uncooked infected horse meat have developed AHS.

SIGNS
- Fever (but not accompanied by inappetence)
- Pulmonary 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 Orbivirus
- Transmitted by arthropod 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 anaplasmosis and equine piroplasmosis may have similar clinical presentation as AHS and may require laboratory testing to differentiate.
- 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 ultrasound 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
- N/A

DRUG(S) OF CHOICE
- N/A

CONTRAINDICATIONS/POSSIBLE INTERACTIONS
- N/A

FOLLOW-UP

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.

MISCELLANEOUS

ZOOONOTIC POTENTIAL
- The disease does not affect humans.

ABBREVIATION
- AHS = African horse sickness

Suggested Reading

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.

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

**CAUSES**

**Endocrinologic Disorders**
- 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 D2 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

**DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS**

**Differentiating Typical Signs**
- 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**
- Indicators of fescue syndrome
- History of fescue ingestion
- Prolonged gestation
- Dystocia
- Retained fetal membranes, thickened fetal membranes
- Weak, dysmature foal, mare with agalactia
- 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**
- If mastitis is suspected
- Cytology or culture of udder secretion
- If neoplasia is suspected
- Fine-needle aspirate—cytology
- Biopsy—histopathology
AGALACTIA/HYPOGALACTIA

TREATMENT

- Mastitis
- Lack of colostrum 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.
  - Severe side effects in horses preclude its use.
  - Sweating, colic, hyperesthesia, ataxia, posterior paresis

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.

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
  - Starvation

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


Author Carole C. Miller Consulting Editor Carla L. Carleton
Pathophysicsiology

- Not necessarily a pathological condition, but usually a normal, species-specific behavior.
- When extreme in frequency or intensity, can be a sign of an underlying pathological condition.
- Any pathophysiology that results in pain.
- Hypothyroidism and acute liver failure have been associated with aggressiveness.
- Hypertension in mares—ovarian tumors such as arthrosalactoma and granulosa-theca cell; resting/filmmation syndrome in mares.
- Retained testicles in males that may produce testosterone or e.g., estrogen.
- Agents that affect the CNS—encephalopathies, rabies.
- Obsessive-compulsive self-mutilation; generally isolated stallions.
- Low blood serotonin levels have been associated with aggressive behavior.
- Anabolic steroids may increase aggression.

Causes

- Increased aggression in response to disturbances, threats, fear, or frustration.
- Presence of offspring, predators, people, or other animals.
- Hormonal imbalances—e.g., estrus.
- Stressful events—e.g., training, competition.
- Medical conditions—e.g., endocrine abnormalities, altered immune function.
- Neuronal plasticity—alterations in neural pathways.

Signs General Comments

- Offensive behaviors range from mild threats to intense injurious acts.
- Mold forms include lassosing, kicking, and striking with head, tail, and legs.
- Defensive aggression adds threats to bite, strike, or kick.
- Submissive behaviors include shifting of hindquarters toward another, tail switching, and other behaviors.
- Offensive behavior usually involves head-on or flight.
- Defensive behaviors involve movement away from and/or shifting of head.
- Reactive behavior usually involves head-on approach and threat.
- Offensive behavior involves head-on approach and threat.
- Defensive behaviors involve movement away from and/or shifting of head.

Abnormalities

- Increased aggression in response to disturbances, threats, fear, or frustration.
- Presence of offspring, predators, people, or other animals.
- Hormonal imbalances—e.g., estrus.
- Stressful events—e.g., training, competition.
- Medical conditions—e.g., endocrine abnormalities, altered immune function.
- Neuronal plasticity—alterations in neural pathways.

Risk Factors

- Inappropriate use of punishment.
- Horses reared in isolation from other horses may not develop adequate social skills.
- Horses reared in isolation from other horses may not develop adequate social skills.
- Horses reared in isolation from other horses may not develop adequate social skills.
- Small enclosures and poorly designed stalls may lead to redirected aggression.
- Sharp edges or protruding sharp wires can cause injury.
- Barriers between horses that engage in aggressive play can prevent injuries.

Diagnosis

- Differential diagnosis includes various medical conditions, e.g., endocrine abnormalities, altered immune function.
- CBC/biochemistry/urinalysis can help rule out medical conditions.
- Nonsurgical examination is essential for diagnosis.

Other Laboratory Tests

- Nonsurgical examination is essential for diagnosis.
- CBC/biochemistry/urinalysis can help rule out medical conditions.
- Endocrine abnormalities are common in mares with unweaned foals.

Prevention

- Early socialization.
- Adequate exercise.
- Appropriate diet.
- Regular veterinary care.

Management

- Environmental modifications—e.g., stalls, separation, feeding.
- Behavioral modifications—e.g., clicker training.
- Medical management—e.g., hormone therapy.

Prognosis

- Generally good with appropriate management.
- Prognosis may be guarded if underlying medical conditions are present.

References


Footnotes

• Aggression in mares, especially when accompanied by stallion-like behaviors, warrants thorough examination, estrogen, and inhibin assays.
• Karyotyping of mares exhibiting stallion-like behaviors
• Thoracic panel

IMAGING
Transrectal ultrasonography of reproductive organs
OTHER DIAGNOSTIC PROCEDURES
• Racial palpation
• Vaginal examination

PATHOLOGICAL FINDINGS
Dependent on etiology of aggression

TREATMENT
AIMS OF TREATMENT
• Identity 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 N/A

ACTIVITY
• Prevent or control access to targets of aggression.
• Increase appropriate and safe type 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 owners of risks involved with keeping such a horse and considering treatment.
• Aggressive animals can deliver serious injury or cause death, and keeping an aggressive animal may place the client at risk of criminal and civil legal actions.

• Advise owner that even if underlying medical reasons are alleviated, there may be residual behavioral patterns that require behavior modification and/or training.

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

MEDICATIONS

DRUGS OF CHOICE
• No drug is approved by the US Food and Drug Administration for use with aggressive problems in horses.
• Pain medications may help to reduce or eliminate pain-elicited aggression.
• Anxiolytics or antidepressants may help with fear-motivated aggression.

CONTRAINDICATIONS
Benzodiazepines may increase aggressive behaviors to other horses and interested in mares. From 5% to 17% retain some aggressive behaviors towards people.

PRECAUTIONS
• Inform clients regarding possible benefits, drawbacks, and side effects.
• Obtain written informed consent before prescribing off-label medication.

POSSIBLE INTERACTIONS

ALTERNATIVE DRUGS
N/A

FOLLOW-UP

PATIENT MONITORING
• Contact clients on a regular basis to check compliance with recommendations and to provide additional support.

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 pathophysiology, 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.

POSSIBLE COMPLICATIONS
See Client Education.

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 aggressive behaviors towards people.
• Treatment of hypothyroidism with levothyroxine can effectively reduce aggressive behavior in horses.

• Successful treatment of nonmedical causes of aggression is dependent on many variables (see above).

MISCELLANEOUS

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

SEE ALSO
• Excessive maternal behavior/foal stealing
• Fear and phobias
• Maternal foal rejection
• Male sexual behavior problems
• Endocrine disorders
• Training and learning problems

Suggested Reading

Authors Victoria L. Voith and Daniel Q. Estep
Consulting Editors Victoria L. Voith and Daniel Q. Estep
The image contains a page from a veterinary textbook. It discusses the Alkaline Phosphatase (ALP) enzyme, its definition, basics, differential diagnosis, and laboratory findings. The text is focused on the pathophysiology and interpretation of ALP levels in various conditions, including hepatobiliary disease, bone metabolism, and reproductive conditions. It includes information on reference intervals, potential causes of increased ALP levels, and how to interpret these levels in different clinical scenarios. The page is part of a larger veterinary consult guide, and the text is professionally formatted, with subheadings and bullet points for clarity and organization.
One study showed ALP values may occur with inflammatory liver disease—bacterial cholangitis/hepatitis. • Evidence of antigenic stimulation (e.g., lymphocytes, reactive lymphoid cells) may be seen.

Glucose • Postprandial hyperglycemia or fasting hyperglycemia may occur with hepatic insufficiency/shunts.

Albumin is a negative acute-phase protein.

Hypoglycemia with liver disease carries a guarded prognosis.

ALP • 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 • Increases with either injury or cholestasis.

Bilirubin • Conjugated—increases with cholestasis.

• Unconjugated—increases with increased RBC destruction (i.e., hemolysis), or decreased hepatic uptake and with fasting.

Cholesterol • May decrease with hepatic insufficiency/shunts.

• Sometimes increases with cholestasis and lipid metabolic disorders—hyperlipidemia.

Triglycerides • Increased with hyperlipemia.

Urinalysis • Bilirubinuria indicates cholestasis.

• Ammonia urates may be observed with hepatic insufficiency/shunts.

OTHER LABORATORY TESTS

Bile Acids • Sensitive indicator of hepatic disease but not specific for the type of process—injury, cholestasis, or insufficiency.

• Anxous enterohepatic circulation, adequate hepatocellular perfusion, and hepato-biliary 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 hyperbilirubinemia.

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

• Ultrasoundography—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., dilations, strictures) or large vessels (e.g., shunts, thrombosis).

DIAGNOSTIC PROCEDURES

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

TREATMENT

• Decision regarding outpatient versus inpatient treatment 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.

• Anorexic 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 or minimize hyperlipemia and hepatic lipodosis.

• Toxicities or hepatic insufficiency may warrant efforts to reduce production/absorption of toxins.

• Mineral oil by nasogastric tube helps to reduce toxin absorption.

• Lactulose (0.5 mL/kg q6h) by nasogastric tube is suggested to combat GI ammonia production/absorption but also causes diarrhea.

• A high-carbohydrate, low-protein diet reduces ammonia production.

• Specific therapy, including surgery, depends on the specific underlying cause.

MEDICATIONS

DRUG(S) OF CHOICE

Depends on the suspected cause and observed complications.

CONTRAINDICATIONS

Depends on the suspected cause and observed complications.

PRECAUTIONS

• With suspected hepatic insufficiency, assess coagulation profile before invasive procedures.

ALTERNATIVE DRUGS

Depends on the underlying cause.

FOLLOW-UP

PATIENT MONITORING

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

Depends on the underlying cause.

EXPECTED COURSE AND PROGNOSIS

Depends on the underlying cause.

MISCELLANEOUS

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.

ZOONOTIC POTENTIAL

Depends on the underlying cause.

PREGNANCY

See Signalment.

SYNONYMS

N/A.

SEE ALSO

• See Signalment.

ABBREVIATIONS

• BSP = sulfobromophthalein.

• EHV = equine herpesvirus.

• ELA = equine infectious anemia.

• EVA = equine viral arteritis.

• GGT = γ-glutamyltransferase.

• GI = gastrointestinal.

• ICG = indocyanine green.

Suggested Reading


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

Author John A. Christian

Consulting Editor Kenneth W. Hinchcliff
Hypoventilation should increase CO₂ levels to lower pH; however, respiratory compensation is limited once hypoxemia develops.

**PATOLOGY**

- The kidney normally is extremely capable of response to the extracellular shift of H⁺ and the urine. Even with daily administration of 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 reabsorption 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.

**LABORATORY FINDINGS**

- CBC/BIOCHEMISTRY/URINALYSIS
  - Yes, if properly submitted

**DIFFERENTIAL DIAGNOSIS**

- Increased bicarbonate levels also are seen in conditions with respiratory acidosis. PaO₂ is high but the pH close to normal or high on blood gas analysis.

**LABORATORY FINDINGS**

- Drugs That May Alter Lab Results
  - Excessive anticoagulant may falsely decrease results via dilution.
  - Excessive sodium heparin may alter electrolytes via sweating.

**OTHER LABORATORY TESTS**

- Many labs measure TCO₂ using the same sample submitted for electrolytes.
**Alkalosis, Metabolic**

**The TCO₂ closely approximates HCO₃⁻, because most CO₂ is carried in the blood as bicarbonate.**

**Like MAK, respiratory alkalosis also results in high TCO₂. These conditions can be differentiated only by complete blood gas analysis.**

**The TCO₂ must be analyzed rapidly and with minimal room-air exposure within the sample tube, because CO₂ 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**

**DRUGS OF CHOICE**

- 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.

**CONTRAINDICATIONS**

- Any alkalinizing 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**

**PATIENT MONITORING**

- 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.

**POSSIBLE COMPLICATIONS**

- Hypokalemia
- Hypocalcemia
- Other, rare complications—cardiac arrhythmias, colic, syncope, diaphragmatic flutter, tetany, and neurologic signs

**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 PaCO\(_2\), and pH
- Arterial PaCO\(_2\) tensions of <35 mm Hg
- Venous PaCO\(_2\) 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 PaCO\(_2\) 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 hyperreactivity of muscle and nervous tissue.

**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 PaCO\(_2\).
- 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, heparinize syringes before sampling.
- Perform sampling anaerobically 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 tensions 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**

- 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**

- 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.

**POSSIBLE COMPLICATIONS**

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

**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.

**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 a complete hair coat, has or had normal hair follicles 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.
- Acquired alopecia can occur in all breeds.
- Both sexes are affected equally.

**SIGNS**
- General Comments
  - May be an acute onset or slowly progressive
- Multifocal or focal
- 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 alopecia (nonscarring causes)
  - Mild to moderate inflammation of the hair follicle (folliculitis and furunculosis) hair dystrophy—possible variant of alopecia.
  - Defects in the hair shaft
  - Hair follicle dystrophies
  - Altered hair follicle function
  - Trauma (self-induced from pruritus)
- Cicatricial alopecia (scarring causes)
  - Cell-mediated autoimmune disease directed toward the hair follicle and adnexa
  - Alopecia areata
  - Hair follicle dystrophy—possible variant of alopecia areata
  - Severe furunculosis
  - Neoplasia
  - Severe inflammatory disease such as in cutaneous onchodermatitis
- 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 hypotrichosis
  - 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 pseudodiphtheriae are uncommon. Other bacterial causes are abscesses due to Fusiformis and Streptococcus spp.
  - Fungal
    - Dermatophytois due to Microsporum gypseum, M. equinum or M. canis or Trichophyton equinum var. equinum causes alopecia. Other fungal causes are mycetomas and the subcutaneous mycosis such as phycomycosis and pythiosis.
  - Parasitic
    - Follicular parasitic infections of the follicle that result in alopecia are rare and include Demodex spp. and Pediculidae, strenglalides. Other more common parasitic infections that cause alopecia are Cylindrosporum, onchodermatitis, lice, ticks, onychotriksis, and mites (Sarcoptes spp., Cheiropteryx spp., and Demodex spp.).
- Viral
  - Viral pappillomata—congenital, cutaneous, or period
- Noninfectious
  - Immune-mediated
  - Cell-mediated autoimmune disease directed toward the hair follicle and adnexa
- Alopecia areata
- Hair follicle dystrophy—possible variant of alopecia areata
- Sebaceous adenitis—rare
- Neoplasia
- Physical
  - Burns from chemicals, hot, cold, or ropes
  - Scalding from exudate, urine, or feces
  - Tail and mane rubbing as stable vice
Alopecia

- Neoplasia
- Sarcoiids
- Squamous cell carcinoma
- Miscellaneous:
  - Symmetrical atrophy of hair follicles secondary to endocrine disorders is extremely rare to nonexistent.
  - Anagen and telogen effluvium
  - Anhidrosis
- Selenium, mimosine, or mercury toxicities
- Copper deficiency

**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.
- The degree of crusts, scale, and exudate helps prioritize the differentials.
- Follicles, as well as eosinophilic dysplastic hair keratinocytes of the hair matrix of anagen hairs grow.
- Biopsies of telogen effluvium 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 decision to biopsy has been 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 canal.
- Normal shed—“physiologic telogen effluvium”
- Telogen effluvium—a reaction pattern characterized by widespread alopecia in response to severe metabolic stress. Serious illness, high fever, pregnancy, and adverse reaction to supplements are all potential inducers of telogen effluvium.
- Rapid premature cessation of anagen growth leads to abrupt synchronization of the follicular cycle such that hair follicles proceed in unison through catagen and telogen. This leads to hair loss of variable severity when old telogen hairs are forced out by new, synchronous anagen hairs. Hair loss usually occurs within 3–4 weeks after the insult but may occur up to 2 mo later. Alopecia resolves spontaneously if the initiating factor is no longer present, and new anagen hairs grow.
- Anagen effluvium—a reaction pattern characterized by shedding during anagen arrest. Severe stresses such as high-dose cytotoxic therapy, infection, or metabolic disease halts anagen hair growth and results in hair loss within days to weeks of the insult. The hairs are lost due to structural weakness or dystrophic changes damaging the hair shaft.

**CBC/BIOCHEMISTRY/URINALYSIS**
Useful to rule out metabolic causes

**OTHER LABORATORY TESTS**
N/A

**IMAGING**
N/A

**OTHER DIAGNOSTIC PROCEDURES**
- Cytology should be obtained from pustules, papules, erosions, or ulcers. Neutrophilic exudate with intra- and extracellular cocci is easily identified if cytology is sampled from ruptured pustules or impression smears made from the underside of crabs or a fresh erosion or ulcer. Impression smears from the surface of lesions often do NOT show bacteria, but rather numbers of shed keratinocytes.
- Direct hair examination (trichography). Hairs will either have anagen or telogen roots. Telogen hairs have uniform shaft diameters and slightly rough-surfaced tapered sparsely stratified 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 effluvium reveals fragmented hair shafts with the absence of roots.
- Perform skin scrapings to rule out ectoparasites.
- Perform bacterial and EIM cultures to determine bacterial species and susceptibility and/or dermatophyte infections.
- 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 for 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 cytologic identification reveals evidence of folliculitis, treat the patient with appropriate antimicrobials, parasiticide, 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 areae 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.

**PATHOLOGICAL FINDINGS**
- Biopsies of telogen effluvium 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 decision to biopsy has been 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 canal.
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/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**
None

**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

**OVERVIEW**

- Amitraz is a formamide acid 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 α2- and α1-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 hyperreactivity to external stimuli, and in some cases, to 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 antidiuretic hormone and thus may promote diuresis.
- Amitraz and its active metabolite both induce hyperglycemia and hypoinsulinemia by inhibiting insulin secretion mediated by α2-adrenergic receptors located within the pancreatic islets.
- Amitraz is more slowly metabolized in ponies than sheep, which may explain its toxicity in equines.
- Clinical signs of amitraz toxicity are usually referable to the central nervous or gastrointestinal systems.

**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-methylformamide derivative and more easily induce toxicity.
- 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 ataxia can be due to viral disease (e.g., rabies, equine encephalomyelitis, West Nile virus), hepatoencephalopathy, 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 hypoinsulinemia 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 fecalith obstruction in the proximal small colon about to market colon impaction.

**TREATMENT**

- If dental exposure to amitraz occurs, horses should be immediately bathed with soap and water to reduce absorption.
- If ingested, activated charcoal (1–4 g/kg PO in water slurry [1 g of AC in 5 mL of water]) can be administered via nasogastric tube to reduce absorption.
- Laxatives and/or a laxative diet may be used to manage the gastrointestinal effects.
- Oxygen and mechanical ventilation may be necessary if respiratory depression is severe.
- Fluid therapy may be beneficial.

**CONTRAINDICATIONS/POSSIBLE INTERACTIONS**

- While used in humans to treat amitraz-induced bradyarrhythmia, 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 heterocyclic antiparasitic agents, 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 results 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.

**Pathophysiology**
- Blood ammonia is derived primarily from dietary nitrogen with the gastrointestinal tract action of bacterial proteases, ureases, 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 urea 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.

**Signs**
- Physical Examination
  - Stunted growth, loss of body condition, poor hair coat, mentation changes and aberrant behavior. Similar findings as discussed in liver disease.
  - Signs may be sporadic and progressive and worsen after feeding.
- Historical
  - Pyralism, behavior changes, visual deficits (blindness), compulsive circling, pacing, anxiety, head pressing, stupor, coma, unusual positions/posture, sudden falling to the ground, violent thrashing.

**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 pyroludine alkaloid toxicity, acute hepatitis, chronic active hepatitis, hepatotoxic drugs or chemicals, Tyzzer’s disease in foals, and hyperplasia in ponies with associated hepatic dysplasia. Deceased functional hepatic mass can result from hyperammonemia resulting in transient hyperammonemia (proposed).

**Diagnosis**

**Biochemistry/Hematology/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.

**Other Laboratory Tests**
- Measurements of serum bile acid concentrations has largely replaced ammonia assays due to convenience of sampling.

**Other Diagnostic Procedures**
- Hepatic biopsy is often necessary.
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 maintain ammonia in its nonabsorbable ammonium ion form.

CONTRAINDICATIONS
Any drugs that affect the CNS must be used with caution because of the common association of hyperammonemia with hepatic encephalopathy and possibly impaired hepatic metabolism. Barbiturates and benzodiazepine-like drugs are of particular concern.

PRECAUTIONS
Sodium bicarbonate in fluids should be administered slowly, because rapid correction of acidosis may favor intracellular ammonia movement.

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

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.

ZOONOTIC POTENTIAL
N/A

PREGNANCY
N/A

SYNONYMS
N/A

SEE ALSO
• Hepatic encephalopathy
• Liver/hepatic diseases
• Hepatic enzyme
• Bile acids

ABBREVIATIONS
• ALP = alkaline phosphatase
• BUN = blood urea nitrogen
• CNS = central nervous system
• GDH = glutamate dehydrogenase
• GGT = /-glutamyltransferase
• PT = prothrombin time
• PTT = partial thromboplastin time
• SDH = sorbitol dehydrogenase

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.
- Panniculitis can result in abnormally high serum activity of amylase and lipase. This disease is often associated with pancreatitis.

**SYSTEMS AFFECTED**
- Pancreas, peritoneum, peripheral adipose tissue

**SIGNALMENT**
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
- Cortisol 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

**DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS**
- Colic with small bowel distention should lead a clinician to suspect inflammation or obstruction of the small intestine rather than pancreatic inflammation, although colic and ileus can be caused by peritonitis secondary to pancreatitis.
- In a pony or miniature horse, hyperlipemia should be considered.

**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.
- Abdominocecostomy 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 wishes 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 allow 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 pathogentic 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 invasion 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 a disruption in the 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 invasion 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
- Fluroneumonia
- Pleuritis

**Gastrointestinal System**

- Peritonitis
- Abdominal abscesses
- Enteritis
- Colitis

**Musculoskeletal System**

- Soft-tissue abscesses
- Foot abscesses
- Thrush
- Canker
- Osteomyelitis
- Septic arthritis
- Tenosynovitis
- Osteoarthritis
- Osteosarcoma
- Clostridial myonecrosis

**Neuromuscular System**

- Botulism
- Tetanus

**Vascular System**

- Ophthalmophlebitis/Oralphalitis
- Thrombophlebitis

**Hematopoietic System**

- Septicemia

**Reproductive System**

- Menitis

**INCIDENCE/PREVALENCE**

Dependent on the organism, body system involved, and how early the infection is noted and treatment is instituted.

**GEOGRAPHIC DISTRIBUTION**

Worldwide distribution.

**SIGNAMENT**

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 exudates

**Lower Respiratory Tract**

- Cough
- Nasal discharge
- Malodorous breath, sputum, pleural fluid
- Fever
- Inappetence
- Abnormal lung sounds
- Lethargy

**Gastrointestinal System**

- Abdominal discomfort
- Fever
- Diarrhea
- Inappetence
- Reflux

**Musculoskeletal System**

- Swollen and painful muscles or joints
- Lameness
- Fever
- Crepitation over swollen muscles

**Neuromuscular System**

- Sclerosis and rigid posture
- Flushing, third eyelid
- Trismus (lockjaw)
- Convulsions
- Dysphagia
- Loss of muscle tone leading to recumbency
- Araxia

**Vascular System**

- Swollen and painful umbilicus
- Fever
- Lethargy
- Inappetence
- Swollen, hard, painful veins

**Hematopoietic System**

- Fever
- Depression
- Tachycardia, +/− arrhythmias
- Tachypnea, +/− dyspnea
- Muscous membrane alterations
- Laminits
- Abdominal discomfort

**Reproductive System**

- Vaginal discharge
- Fever
- Lethargy
- Endoneuropaenia

**CAUSES** (most common)

**Upper Respiratory Tract**

- Bacteriodes spp.
- Fusobacterium spp.
- Peptostreptococcus spp.

**Lower Respiratory Tract**

- Bacteriodes spp.
- Clostridium spp.
- Escherichia coli
- Peptostreptococcus spp.

**Gastrointestinal System**

- Peritonitis/abdominal abscesses—Bacteriodes spp., Fusobacterium spp., Peptostreptococcus spp., E. coli—Clostridium spp. and Bacteriodes spp.

**Musculoskeletal System**

- Soft-tissue/foot abscesses and canker—Bacteriodes spp. and Fusobacterium necrophorum
- Osteomyelitis/sequestrum—Clostridium spp
- Septic arthritis/tenosynovitis—Clostridium and Bacteriodes spp.
- Myonecrosis—Clostridium spp.

**Neuromuscular System**

- Botulism—Clostridium botulinum
- Tetanus—Clostridium tetani

**Vascular System**

- Bacteriodes fragilis, Propionibacterium acnes, Peptostreptococcus magnus, and/or Clostridium spycicum

**Hematopoietic System**

- Clostridium spycicum

**Reproductive System**

- Bacteriodes fragilis, Peptococcus, Fusobacterium, and Bacteriodes spycicum

**RISK FACTORS**

Concurrent diseases, corticosteroid therapy, antiinfective 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

**DIFFERENTIAL DIAGNOSIS**

**Upper Respiratory Tract**

- Aerobic infection (Streptococci spp., Staphylococci spp.)
- Fungal infection (Cryptococcus neoforans, Coccioides immitis)
- Gramanemia
- Neoplasia (anaerobes may proliferate in necrotic neoplastic tissues)
Lower Respiratory Tract
- Aerobic infection (Streptococcus spp., Staphylococcus spp., Escherichia coli, Klebsiella, Pseudomonas, Bacteroides spp.)
- Fungal infection (Candida albicans, Cryptococcus, Histoplasma, Aspergillus, Canidae spp.)
- Mycoplasma infection
- Thoracic trauma
- Esophageal rupture
- Neoplasia—Primary (rare), metastatic (more common)

Gastrointestinal System
- Peritonitis—Aerobic infection (Streptococcus spp., E. coli), neoplasia
- Abdominal abscesses—Aerobic infection (E. coli), Neoplasia
- Myxedema infection
- Thoracic trauma
- Esophageal rupture
- Neoplasia—Primary (rare), metastatic (more common)

Musculoskeletal System
- Aerobic infection (Staphylococcus aureus, Corynebacterium pseudotuberculosis)
- Fungal infection
- Neoplasia

Neuromuscular System
- Botulism—Laminitis, myositis/myopathy, exertional rhabdomyolysis, EPM, tick paralysis
- Tetanus—Acute laminitis, hypocalcemic tetany, rabies, HYPP

Vascular System
- Aerobic infection (Streptococcus spp., E. coli, Proteus spp.)

Reproductive System
- Aerobic infection (Streptococcus equisimilis, E. coli, Klebsiella spp., Staphylococcus spp., Proteus spp., Pseudomonas spp., Corynebacterium spp.)
- Fungal infection (Canidae spp.)
- Neoplasia

CBC/Chemistry/Urine Analysis
- Inflammatory leukogram
- Hyperfibrinogenemia
- Elevated total protein
- +/- Anemia of chronic disease

Other Laboratory Tests
- Direct cytology—All aspirated fluids should be gram stained.
- Anaerobic culture—Aspirates/tissue specimens must be placed in the appropriate anaerobic bacterial transport medium and stored at room temperature with minimal exposure to oxygen.
- Sonograms of the affected anatomic region

Other Diagnostic Procedures
- Fecal cultures
- Bone biopsies
- Identification of toxins or spores in feed

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

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

Appropriate Health Care
Initial hospitalization for intensive therapy; antimicrobial, 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. Care 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

Diets
The diet will most likely remain unchanged.

Client Education
Some cases may be life-threatening depending on the extent of the illness and complications may arise. In cases with severe muscle necrosis requiring debridement or fasciotomy, 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
Drug(s) of Choice
Penicillin
First line of defense against anaerobic infections. Excellent activity against most anaerobic infections, except beta-lactamase producing Bacteroides. Preferred drug for clostridial infections. Dose: 22,000–44,000 IU/kg, QID IV (aqueous) or BID IM (procaine).

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

Cephalosporins
First-generation cephalosporins are generally less efficacious for anaerobic infections compared to penicillin. Cefoxitin (second generation) kills Bacteroides fragilis but may be used less due to expense. Other cephalosporin activity for anaerobic infections 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 repeated 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 procarine reactions. Chloramphenicol can cause the development of aplastic anemia rarely in humans. Oral administration of metronidazole may cause anorexia 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 neutrocytary 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 Coninner R. Sweeney
OVERVIEW

- Basics
- Diagnosis
- Treatment
- Medications
- Contraindications/possible interactions
- Follow-up
- See also
- Miscellaneous

BASICS

- Anaphylaxis is a type I hypersensitivity reaction where antigen-antibody reactions involve mast cells and basophils. IgE, which is commonly involved, attaches to the mast cell and basophil. Upon reexposure, the IgE antibodies bind to the antigen (allergen) and activate the mast cells and basophils.

- Clinical signs usually occur within seconds and can be grouped into four categories: mucosal signs, cardiovascular signs, respiratory signs, and cutaneous signs.

- Diagnosis involves identifying the allergen and confirming the diagnosis through testing.

- Treatment is focused on reversing the effects of mediator release and preventing further release of mediators.

- Medications used in anaphylaxis treatment include epinephrine, glucocorticoids, and antihistamines.

DIFFERENTIAL DIAGNOSIS

- Asthma: Persistent airflow obstruction with exaggerated response to stimuli.
- Acute pneumonia: Inflammation of the lung tissue.
- Acute toxemia: Systemic injury caused by toxins.
- Cardiogenic shock: Reduction in cardiac output resulting in hypoperfusion.
- Neurogenic shock: Hypotension caused by the central nervous system.

PATHOLOGICAL FINDINGS

- Severe, diffuse pulmonary edema and peribronchial edema are common.
- Intense pulmonary congestion and edema are present.
- Kidney, spleen, and liver exhibit congestion and edema.
- Intense hyperemia and congestion of the skin and extremities.

TREATMENT

- Therapy is aimed at reversing the effects of mediator release and preventing further release.
- Supportive care, including fluids, oxygen, and vasopressors, is essential.
- Glucocorticoids are used to reduce inflammation and mediator release.
- Antihistamines are used to block the effects of histamine.

MEDICATIONS

- Drugs used in anaphylaxis treatment include epinephrine, glucocorticoids, antihistamines, and vasopressors.
- Epinephrine is the most effective treatment for anaphylaxis/shock.

CONTRAINdications/possible interactions

- Hypersensitive reaction to epinephrine should be avoided.
- Glucocorticoids potentiate the effect of epinephrine, and their use should be considered in severe reactions.

PREVENTION/Avoidance

- Avoidance of the allergen is the best prevention strategy.
- Education about the signs and symptoms of anaphylaxis is crucial.

EXPECTED COURSE AND PROGNOSIS

- The outcome depends on the severity of the reaction and the effectiveness of treatment.
- Mortality rates range from 0% to 10% depending on the severity of the reaction.

SEE ALSO

- Anaphylaxis
- Hypersensitivity disorders

Miscellaneous

- Jennifer Hodgson
- David Hodgson and Jennifer Hodgson

AUTHOR: Jennifer Hodgson
CONSULTING EDITORS: David Hodgson and Jennifer Hodgson
ANEMIA

BASICS

DEFINITION
A decrease in erythrocyte content or oxygen-carrying capacity of blood as a consequence of a decrease in PCV, RBC counts, and/or 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 pathophysologic mechanisms is present:
  □ Decreased RBC production (due to hemorrhage or hemolysis) or nonregenerative (due to decreased/ineffective marrow production) is assessed most accurately by examination of bone marrow aspirate. Serial monitoring of PCV and plasma TP concentration also may be helpful. Evaluation of immature RBC and RBC indices in peripheral blood is unwarranted in 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 (≈350 days).
• 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 function
  □ Diseases that damage or replace normal bone marrow elements and affect RBC precursors alone, or affect all marrow precursors (WBCs, RBCs, platelets)

SYSTEMS AFFECTED
• Although dependent on the severity and rate of development of anemia, the decreased oxygen-carrying capacity, the decreased circulating RBC mass, and reduced blood viscosity are the main consequences of anemia.
• Hemic/lymphatic/immune systems
• Cardiovascular and respiratory systems
• Hepatobiliary, renal, musculoskeletal, and GI systems

SIGNAMENT
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 hemorraghy, 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.
• Pale mucous membranes
• Other signs depend on the primary disease process and may include:
  □ Fever, icterus, and pigmenturia in cases of hemolysis
  □ Weight loss, polyuria, 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
• Erysipelas due to gummed pouch mycotic, pulmonary abscess, septicemia, EIPH, endotoxic hemolysis, fungal rhinitis, sinusitis, neutrophilic, or trauma
• Hemorrhage due to trauma, ruptured pulmonary abscess, ruptured ventral rectal, or aneurysm
• Hematoma due to pyostomatitis, abscess, septicemia, septicaemia, pulmonary abscess, or trauma
• Hemoptysis due to trauma, ovarian hemorrhage, mesenteric vessel rupture, aneurysm, or abdominal abscess
• GI hemorrhage due to ulceration (e.g., gastric or duodenal ulcers in foals, NSAAI toxicosis), parasites (particularly large strongyle), granulomatous inflammatory disease, neoplasia (e.g., gastric squamous cell carcinoma), or foreign bodies
• Coagulopathy

Hemolysis
• Immune-mediated disease—secondary immune-mediated anemia (e.g., bacterial, viral, or parasitic anicteric hepatitis, or dogs), autoimmune hemolytic anemia, and NI
• Infectious disease—granulomas (Rickettsia rickettsii and Theileria spp., ehrlichiosis (Anaplasma phagocytophilum), and ELA
• Oxidant-induced—wilted red maple leaf, phenothiazine anthelmintics, wild onions and acidic plants
• Lactogenic—hypertonic or hypodermic solutions administered IV
• Other toxicities—intravenous dimethyl sulfoxide, heavy metal toxicosis, bacterial toxins (Clostridium sp.), snake envenomation
• Miscellaneous—end-stage hepatic disease, hemolytic urticarial syndrome, hemangiosarcoma, and disseminated intravascular coagulopathy

Nonregenerative Anemia
• Anemia of chronic disease associated with infection, inflammation, or neoplastic or immune-mediated processes
• Anemia associated with decreased RBC production (e.g., due to decreased marrow production, marrow toxins, or infection)
• Iron deficiency due to chronic hemorrhage (especially GI) and nutritional deficiency (particularly folic acid)
• Bone marrow failure—myelophthisis, myelodysplasia, or aplastic anemia
• Miscellaneous—chronic renal disease, chronic hepatic disease, and recent hemorrhage or hemolysis

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

DIAGNOSIS
DIFFERENTIAL DIAGNOSIS
• Differentiation 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 a falsely low PCV or RBC count and falsely high MCH and MCHC, or erroneous, or false low MCV and MCHC values.

CBC/BIOCHEMISTRY/URINALYSIS
• PCV, total RBC count, and (except in cases of intervascular hemolysis) Hb concentrations below the lower limit of reference intervals
• Reticulocytes, nucleated RBCs, Howell-Jolly bodies, polychromatemia, and anisocytosis are
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.
- Heme bodies may be observed near the cellular margins of RBCs stained with New Methylene Blue in horses with hemolytic anemia due to oxidative injury.
- Spherocytes, 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.
- Neutrophilic cells may be observed in blood smears of horses with myeloproliferative disorders.
- Severe neutropenia and thrombocytopenia may be observed in horses with myeloproliferative disorders.

OTHER LABORATORY TESTS

- Serum iron concentration usually is increased, total iron-binding capacity usually decreased, and transferrin saturation of transferrin usually is decreased in 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 uncontrollable internal hemorrhage, although these horses have a high anesthetic risk.

Suggested Reading


Author: Nicholas Malkides

Consulting Editors: Jennifer Hodgson and David Hodgson

FOLLOW-UP

PATIENT MONITORING

Monitor PCV to assess regenerative response. PCV should increase by an average of 0.3%–1% per day within 5–7 days of an acute hemorrhagic or hemolytic episode.

EXPECTED COURSE AND PROGNOSIS

Highly dependent upon the cause, severity, and rapidity of onset.

MISCELLANEOUS

ABREVIATIONS

- EIA = equine infectious anemia
- EPH = exue-induced pulmonary hemorrhage
- GI = gastrointestinal
- Hb = hemoglobin
- IV = intravenous
- MCH = mean cell hemoglobin
- MCHC = mean cell hemoglobin concentration
- MCV = mean cell volume
- NSAID = non-steroidal anti-inflammatory drug
- PCV = packed cell volume
- RBC = red blood cell
- TP = total protein

Suggested Reading


Author: Nicholas Malkides

Consulting Editors: Jennifer Hodgson and David Hodgson
Blackwell’s Five-Minute Veterinary Consult

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**

Other Causes of Inadequate Erythropoiesis
- 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.
- 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, decreased urine concentrating ability).

**Other Causes of Anemia**
- Chronic EIA may also cause significant bone marrow suppression. These horses are sero- or virus-positive for EHV.
- Regenerative anemia caused by external or internal hemorrhage and infectious (e.g., low-grade equine piroplasmosis, ehrlichiosis), immune-mediated (e.g., immune-mediated hemolytic anemia and its various causes), or toxic (e.g., oxidant-induced) hemolysis may be differentiated from pure red cell aplasia by the presence of icterus, increased bilirubin concentrations, bilirubinuria, and decreased bone marrow M:E ratios.
- Iron deficiency anemia secondary to chronic hemorrhage. Measurement of decreased serum ferritin concentrations can be used to distinguish this condition from pure red cell aplasia.

**CBC/Biochemistry/UrineAnalysis**
- Anemia, with PCV 0.16 L/L (16%) or below
- Normal WBC count, platelet numbers and plasma fibrinogen concentrations
- Normal urinalysis
OTHER LABORATORY TESTS
- Reported cases have demonstrated increased serum iron and serum ferritin concentrations.
- Negative Coggins 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 unproven. 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
- 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.

MISCELLANEOUS
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

**BASICS**

**DEFINITION**
- Acute or chronic hemolytic anemia following exposure to agents that oxidize and denature RBC hemoglobin.
- Hemolysis causes hemoglobin to aggregate onto the RBC membrane to form Heinz bodies, resulting in cell fragility and subsequent premature RBC removal by the spleen.

**PATHOPHYSIOLOGY**
- Exposure to RBC-damaging 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 hemolyzed hemoglobin precipitates to form Heinz bodies, which attach to the RBC membrane and cause increased cell fragility.

**CAUSES**
- Ingestion of wilted or dried red maple leaves or bark of red maple (Acer rubrum) is a common cause of Heinz body hemolytic anemia.
- Hemolysis can also occur following ingestion of phenothiazine, wild onions.
- Access to wilted or dried leaves or bark of red maple (Acer rubrum) is a common tree in the eastern United States.

**SIGNS**
- Anemia, Heinz body anemia will have a positive Coombs test.

**RISK FACTORS**
- Poorly conditioned horses may be at greater risk for phenothiazine toxicosis.
- Severely affected horses may become debilitated and (sudden) death may occur.

**DIFFERENTIAL DIAGNOSIS**
- Other equine diseases causing anemia must be differentiated from Heinz body hemolytic anemia.
- Hemorraghia is usually evident from history.

**DIAGNOSIS**
- Physical examination findings may indicate thoracic or abdominal disease.
- Horses with EIA will have a positive Coggins or C-ELISA test.

**IMAGING**
- Rectal examination may reveal an enlarged spleen.
- Poorly conditioned horses may have abdominal pain.
- Hepatic or splenic ultrasonography may be used to differentiate hemolytic anemia from other causes.

**HEINZ BODIES**
- Heinz bodies are red blood cell inclusions consisting of denatured, aggregated, and precipitated protein component of the hemoglobin molecule.
- Oxidant toxins convert hemoglobin to methemoglobin.
- Methemoglobin cannot transport oxygen, resulting in tissue hypoxia.

**SIGNS**
- Physical examination findings may indicate thoracic or abdominal disease.
- Physical examination findings may indicate thoracic or abdominal disease.

**COUMARINS**
- COUMARIN-induced hemolysis occurs due to ingestion of clover, peanuts, pea sprouts.

**COUMARIN-INDUCED HEMOLYSIS**
- COUMARIN-induced hemolysis occurs due to ingestion of clover, peanuts, pea sprouts.

**HEMATOLOGIC ABNORMALITIES**
- Anemia, Heinz body anemia will have a positive Coombs test.
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**IMAGING**
- Rectal examination may reveal an enlarged spleen.
- Poorly conditioned horses may have abdominal pain.
- Hepatic or splenic ultrasonography may be used to differentiate hemolytic anemia from other causes.
may be some loss of architecture due to increased fluid component.

OTHER DIAGNOSTIC PROCEDURES
A thorough diagnostic workup should be undertaken to rule out other causes of hemolytic anemia.

PATHOLOGICAL FINDINGS

- Pale or icteric tissues
- 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 epithelial neoplasms and hemosiderin 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 toxin.
- In-hospital medical management may be necessary depending on severity and 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 < 8–12%, or if there is persistent 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 cattle septic, 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.
- The prognosis is guarded in horses with red maple leaf toxicosis when methemoglobinemia is present.

MISCELLANEOUS

ASSOCIATED CONDITIONS

- Methemoglobinemia
- Pigment nephrosis

PREGNANCY

- Horses severely affected, and with general debilitation, may abort or deliver a weak foal.

SYNONYMS

- Methemoglobinemia
- Oxidative hemoglobinemia
- Oxidant-induced hemolysis

SEE ALSO

- Anemia
- Anemia, immune-mediated
- EIA
- Methemoglobinemia

ABBREVIATIONS

- BUN = blood urea nitrogen
- C-ELISA = competitive enzyme-linked immunosorbent assay
- CRT = capillary refill time
- EIA = equine infectious anemia
- GIT = gastrointestinal tract
- IV = intravenous
- MCH = mean corpuscular hemoglobin
- M.E. = myeloid-erythroid
- PCV = packed cell volume
- PO = per os
- RBC = red blood cell
- RES = reticuloendothelial system

Suggested Reading


Author: Nicholas M. Kékesi
Consulting Editors: Jennifer Hodgson and David Hodgson
ANEMIA, IMMUNE-MEDIATED

BASICS

DEFINITION
• IMHA is an acute or a chronic destruction of RBCs associated with immunoglobin 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-phagocytes system) after immunoglobin-mediated opsonization (extravascular hemolysis).
• Less commonly, they may undergo intravascular, complement-mediated lysis.

PATHOPHYSIOLOGY
• IMHA most commonly occurs secondary to agents that:
  o Alter the RBC membrane, exposing antigens to which the host produces antibody (e.g., infectious agents, neoplasia).
  o Form immune complexes that abduct to the RBC and fix complement (e.g., infectious agents).
  o Directly bind to the RBC and act as hapten that bind antibody (e.g., drugs); or
  o 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 hematopoietic/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, holotonic heart murmur, and pallor of mucous membranes observed.
• Hepatobiliary system—Hemolytic anemia can result in hyperbilirubinemia and icterus while products of red cell breakdown.
• 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 hemolytic damage to intestines resulting in motility disorders, colic or diarrhea.

INCIDENCE/PREVALENCE
• No incidence or prevalence data are available for immune-mediated hemolytic anemia for specific horse populations. Weak evidence from in-practice experience 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.

CAUSES
• Primary Immune-Mediated
  o Neoplasia
  o Autoimmune hemolytic anemia
• Secondary Immune-Mediated
  o Infection—eg, EIA, acute viral infections, infection with Clastoderm perfringens, injection site abscess
  o Neoplasia—eg, lymphosarcoma and hemangiosarcoma
  o Drug-associated—eg, penicillin, cephaloporins, and tetracyclines.
  o Microangiopathic—disseminated intravascular coagulation
  o Systemic lupus erythematosus

RISK FACTORS
• Fresh born to multumus 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.
• Horses with hemoglobin (acute or chronic) often have a history of external blood loss or signs referable to thoracic or abdominal disease.
• Horses with Heinz-body anemia (eg, willed red maple leaf toxiocin, or outer granulocytic, phagocytic anemia) may have a history of exposure to oxidant toxins and presence of Heinz bodies or methemoglobinemia on routine blood analysis.
• Horses with purpura hemorrhagica often have a history of exposure to Staphylococcus aureus or other respiratory tract pathogens. In these cases edema of the legs, abdomen, and face and petechial hemorrhages of mucous membranes may be observed.

IMAGING
• Splenic/hepatic ultrasound determines splenic/hepatic enlargement and highlights
hypothecic or hypotonic 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 striae.
- If chronic, there may be signs of congestive heart failure (with pulmonary emphysema, 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 immunosuppressive therapy is required, a molecularly dissimilar antibiotic should be used.

**APPROPRIATE HEALTH CARE**
- Most cases of IMHA are treated in hospitals, especially if acute.
- Balanced polyionic IV fluid therapy may be indicated to expand vascular volume and induce diuresis.
- Emergency medical therapy with cross-matched Blood transfusion is indicated if there is evidence of tissue hypoxia (PCV <8%-12%). In foals, washed RBCs from the dam or appropriate blood-type blood is optimal.

**NURSING CARE**
- Serial analysis of PCV in order to monitor response to therapy should be performed.
- Close monitoring of vital signs and adjustment of fluid rate is essential in horses receiving fluid therapy.
- In foals with N1, provide adequate warmth and hydration, avoid stress and confine mare and foal to restrict activity.

**ACTIVITY**
Minimal or eliminate activity, but allow the animal access to fresh air and sunshine if possible.

**DIET**
- Make efforts to keep the horse eating a balanced diet, with good-quality hay and grain.
- Fresh water should be available ad libitum.

**CLIENT EDUCATION**
- Clients should be made aware that horses with primary (or autoimmune) IMHA often require long-term corticosteroid therapy and often are found to have incurable neoplastic disease.
- Additionally, long-term administration of corticosteroids may increase the risk of laminitis, tendon laxity, and immunosuppression leading to secondary infections.
- Clients should be educated in preventative measures for NI.

**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 (2–3 mg/kg) may be used in place of dexamethasone at any time during therapy, although, anecdotally, dexamethasone is more efficacious.
- 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**
- Cross-matched blood before blood transfusion.
- Corticosteroids may predispose horses to laminitis and exacerbate an undiagnosed infectious process. They also should be used with caution in pregnant mares.

**PRECAUTIONS**
- Cross-match blood before blood transfusion.
- Corticosteroid therapy may predispose horses to laminitis and exacerbate an undiagnosed infectious process. They also should be used with caution in pregnant mares.

**ALTERNATIVE DRUGS**
- The immunosuppressive agents azathioprine (50 mg/kg PO once daily) and cyclophosphamide (300 mg/m^2 body surface area) have been used successfully in one horse that was nonresponsive to corticosteroids.

**FOLLOW-UP**

**PATIENT MONITORING**
The PCV should be carefully monitored during dexamethasone treatment. The frequency of dexamethasone administration can be increased to twice daily in horses initially commenced on once/day treatment if the PCV does not stabilize within 24–48h.

**PREVENTION/AVOIDANCE**
Avoid drugs known to have caused secondary IMHA.

**POSSIBLE COMPLICATIONS**
- Pigment nephropathy may occur secondary to intravascular hemolysis.
- Laminitis

**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 disphoric autoimmune hemolytic anemia) may have an incurable underlying disease such as neoplasia (e.g., lymphosarcoma). The prognosis for survival in these horses is poor.

**ASSOCIATED CONDITIONS**
- Pigment nephropathy with intravascular hemolysis.
- Laminitis.

**PREGNANCY**
- Use corticosteroids cautiously in pregnant mares.

**SYNONYMS**
- Autoimmune hemolytic anemia.
- Immune-mediated hemolytic disease.

**SEE ALSO**
- Anemia.
- Anemia, Heinz-body.
- Babesiosis.
- Equine infectious anemia.
- Hemorrhage, acute.

**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.
- MCHC = mean corpuscular hemoglobin concentration.
- NI = neonatal isoerythropoiesis.
- PCV = packed cell volume.
- PO = by mouth.
- RBC = red blood cell.
- RES = reticuloendothelial system.

**SUGGESTED READING**

**Author** Nicholas Malikides

**Consulting Editors** Jennifer Hodgson and David Hodgson
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). Unless 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 mg/g by 2 weeks and ≈0.6 mg/g by 8 weeks postpartum) and therefore deficiency may occur in foals with limited access to pasture, iron-rich soils, or not consuming forage or grain.

SIGNS
Initially, clinical signs may be absent or mild due to adequate physiologic compensation for the gradual reduction in oxygenation.

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, hemospirocytosis, and a normal serum protein concentration. Serum iron concentrations may be increased.

Causes of decreased erythropoiesis 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.

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 infection of sucking lice—Haematopinus asini)

Bleeding GI, 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)

OTHER LABORATORY TESTS

Initial Stage
- Decreased stainable iron (Prussian 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 ( = 100 × SI/TIBC). Reference range is 30%–50% (Arabian horses ≈68%) 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

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.

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Anestrus

BASICS

DEFINITION/OVERVIEW
Poor body condition/malnutrition may contribute to anestrus.

ETIOLOGY/PATHOPHYSIOLOGY
Primary hypothalamic-pituitary-ovarian axis
Postpartum anestrus occurs more often in early

SYSTEMS AFFECTED

Reproductive
Endocrine

SIGNs

Historical
Chief complaint—failure of mare to accept

PHYSICAL EXAMINATION

Poor body condition/malnutrition may contribute to anestrus.

Asthmatics

Endocrine Disorders

Cushing’s disease—Adenomatous hyperplasia of

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS

Differentiating Causes
Corticotropin-releasing factor, general and

THESE TESTS

Serum testosterone and inhibin
Mares—50–60 pg/mL, inhibin ≤0.7 ng/mL
Levels suggestive of GCT/GCTG (in a
Non pregnant mare) are—Nonreactive
>50–100 pg/mL (if thalidomide is significant

OTHER LABORATORY TESTS
tumor component), inhibin > 0.7 ng/mL, progesterone < 1 ng/mL.

- Scrotal CG—measured by ELISA
- GnRH stimulation test—toward primary hypothalamic or pituitary dysfunction

- Karyotype—if suspect gonadal dysgenesis or intersex conditions

IMAGING

Transrectal US of reproductive tract. See related topics.

DIAGNOSTIC PROCEDURES

Urinary cytology, culture and biopsy—diagnosis, select treatment/monitor progress of pyometra and endometritis

TREATMENT

- Combination—shaving treatment techniques, vary teasing methods to elicit a response from a mare and/or base timing of AI on TRP and US.
- Artificial lighting—hasten onset of vernal

Deslorelin implants not currently sold in the US. Treating a mare with progesterone while she is pregnant can lyse CL, causing abortion, especially if < 40 days pregnant. Rule out pregnancy before injecting this drug or its analogs.

- Serum eCG—measured by ELISA

With removal of a GCT/GTCT, suppression of corpus luteum = PGF in analogs—contraindicated in mares with heaves or other bronchodiastolic disease

CONTRAINDICATIONS

PGF2α and, in analogs—contraindicated in mares with heaves or other bronchodiastolic disease

PRECAUTIONS

- Hormones

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

- Antibodies to hCG can develop. Desirable to limit its use to no more than 2–3 times during one breeding season. Half-life of antibodies ranges from 30 days to several months, typically do not persist from one breeding season to the next.

- Deslorelin implants not currently sold in the United States; injectable product is available. Implants were associated with suppression of FSH and decreased follicular development in the diestrous period immediately following implant use; led to prolonged interovulatory period in nonpregnant mares. Implant removal post-ovulation helped some. Follicle stimulation—potential to decrease uterine clearance; its use may be contraindicated in mares with a history of uterine infection.

- Altrhengen, deodrin, and PGF2α should not be used in horses intended for food. Humans—With either product below, accidental skin exposure should be washed off immediately.

- PGF2α or its analogs should not be handled by pregnant women or persons with asthma or bronchial disease.

- Altrhengen should not be handled by pregnant women or persons with thombophlebitis, thrombembolic disorders, cerebrovascular or coronary artery disease, breast cancer, estrogen-dependent neoplasia, undiagnosed vaginal bleeding, or tumors that developed with use of oral contraceptives or estrogen-containing products.

ALTERNATIVE DRUGS

- Cloprostenol sodium (Estrumate [Schering-Plough Animal Health]) 250 µg/mL IM, a prostaglandin analog. Used in similar fashion as natural prostaglandin, but fewer side effects. Not currently approved for use in horses, but is an analog in widespread use in absence of an alternative.

DRUG(S) OF CHOICE

- PGF2α (Lutalyse) [Pfizer] 10 mg/IM or analog—lyse persistent CL. Multiple injections may be needed with pseudopregnancy.

- Deslorelin injectable GnRH analog to induce ovulation within 48 h if follicles ≥ 30 mm. CG 250 IU IV to induce ovulation in mares with follicles ≥ 35 mm. Alternogest (Regu-Mate [Intervet]) 0.644 mg/PO daily minimum 15 days to shorten vernal transition, providing follicles ≥ 20-mm diameter are present at onset of treatment and mare is exhibiting behavioral estrus. Is used in seasonally deep anestrous mares is not recommended. PGF2α (Lutalyse) on day 15 of altrhengen treatment increases the reliability of this transition management regimen.

POSSIBLE COMPLICATIONS

Infertility may result from intractable persistent anestrus.

AGE-RELATED FACTORS

Postpartum anestrus occurs more often in old mares.

MISCELLANEOUS

PREGNANCY

PGF2α to pregnant mares can lyse CL, causing abortion, especially if < 40 days pregnant. Rule out pregnancy before injecting this drug or its analogs.

SYNONYMS

- Gonadal dysgenesis = Gonadal hypoplasia
- Lactational anestrus = Postpartum anestrus

SEE ALSO

- Abnormal estrus intervals = Aggration
- Cushing’s syndrome = Disorders of sexual development = EED = Endometritis
- Large ovary syndrome = Ovarian hypertrophy
- Ovulation failure = Prolonged diestrous
- Pyometra

ABBREVIATIONS

- AI = artificial insemination
- CL = corpus luteum
- eCG = equine chorionic gonadotropin
- EED = early embryonic death
- FSH = follicle-stimulating hormone
- GnRH = gonadotropin-releasing hormone
- hCG = human chorionic gonadotropin
- LH = luteinizing hormone
- Ovx = ovarectomy
- PGF2α = prostaglandin F2α
- TRP = transrectal palpation
- U/S = ultrasonography

Suggested Reading


McCarroll PM, Fungarol VJ, Carnevale EM, Squires EL. Removal of deodrin (OvuPacle<sup>TM</sup>) implant 48 h after administration results in normal interovulatory intervals in mares. Theriogenology 2002;58:805–870.


Author Carole C. Miller

Consulting Editor Carla L. Carleton
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 periartricular soft tissue structures and perinatal soft tissue trauma can lead to unstable joints, resulting in abnormal loading of the articualr surfaces inducing ALD (manually correctible in the early stages).
- Anything to jeopardize the intrauterine environment of the foal (i.e., placentitis, twin foal) and premature birth (<315 days) may result in incomplete ossification (carpus and tarsus) at birth. If the joints are uneventily 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., “crib feeding” leading to excessive grain intake, unbalanced trace minerals) can result in disproportionate growth at the level of the physes, 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 and fetlock varus and valgus 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.

**Breed Predilections**
Observed in all breeds, most commonly Thoroughbreds, Quarter Horses, and Miniature Horses.

**Age and Range**
May either be present at birth or develop days to months following birth.

**Predominant Sex**
N/A

**SIGNS**
General Comments
- Natural growth of foals can lead to spontaneous correction of the ALD. However, foals with ALD must be monitored appropriately, as if they do not correct their conformation, there is a limited window during which surgical intervention can occur prior to severe physis closure. Ideal conformation varies between breeds and type of work desired (i.e., pleasure versus racing).
- Each foal will respond differently to treatment.

**Historical**
- Prematurity/dysmaturity
- Placentitis or twin foal
- Unbalanced nutrition (i.e., “crib feeding”)

**Physical Examination**
- Valgus deformity results in what is termed “flip foot” due to lateral deviation; outward rotation of the entire limb (“soused out”) should not be mistaken for deviation of the limb at the level of the carpus or fetlock.

**Radiographic**
- A valgus deformity results in what is termed “pigeon toed” due to medial axial deviation.

**CAUSES**

**Perinatal Factors**
- Prematurity
- Dysmaturity
- Hypothyroidism
- Twin foal
- Placentitis

**Developmental Factors**
- Nutritional imbalance
- External trauma to the physis
- Overload of a limb

**RISK FACTORS**
N/A

**DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS**
- Laxity of periartricular soft tissues
- Incomplete ossification/collapse of the cuboidal bones
- Developmental factors
- Perinatal factors

**IMAGING**
Radiography allows for determination of the location and the degree of the deformity, as well as concurrent physitis 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 (long cassettes for easier assessment of the deformity). Only two views are required for ALD (lateral and/or dorsopalmar/dorsoplantar). 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—target 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 physes 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.
- For foals with incomplete ossification of the cuboidal bones and deformation 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.
- It is important to not overtight or create an abnormal hoof shape that will further alter normal weight-bearing.
Margaret C. Mudge

Maintain a bandage and splint or cast for 2 months, tibia osteotomies have been performed for periosteal transection is performed on the proximal phalanx, radius and the distal tibia. 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.

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 of the distal radial physis for a carpal valgus deformity. 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.
- Overcorrection of the deformity has not been observed. A bandage should be maintained for 10–14 days following surgery. Keep the foal on stall rest for 2–3 weeks after surgery.

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 phalanx > 2 months, tibia > 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 physeal, 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 physes of the distal MCIII/MTIII, the distal radius and the distal tibia. 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.

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MEDICATIONS

- For surgical cases, NSAIDs (flunixin meglumine 3.3 mg/kg IV daily or q12h) and antibiotics (i.e., gentamicin 6.6 mg/kg IV daily or Amikacin 25–30 mg/kg IV daily and potassium penicillin 22,000 IU/kg IV q12h) can be given as needed perioperatively.

PREVENTION/AVOIDANCE

Balanced nutrition is very important.

POSSIBLE COMPLICATIONS

- Non-surgical treatment-pressure sores, osteopenia, and tendon/ligament laxity from cast/splint application. Surgical management—hematomas/seoma 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 perisosteal 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

AGG-RELATED FACTORS

Timing of intervention is important, as the greatest effects of surgical manipulation will occur during the rapid growth phases.

ZOOLECTIC POTENTIAL

N/A

FERTILITY MANAGEMENT

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 β₂-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 β₂-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 β₂-agonist (e.g., terbutaline sulfate, salbutamol sulfate), serial dilutions (10⁻³ 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 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.

MEDICATIONS

DRUG(S) OF CHOICE
• Supplemental electrolytes, especially potassium salts, can be added to the feed or water.
• Some anecdotal reports of success with iodinated casein (10–15 g/day for 4–8 days) and with 1000–3000 IU PO of vitamin E (i.e., natural γ-tocopherol) daily for 1 mo.
• Amino acid supplements, especially those with tyrosine, are commercially available. (Tyrosine is necessary for the resensitization of sequestered β₂-receptors.)

POTENTIAL MEDICATIONS

• 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.

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**

CAUSES include the following: acquired aspiration pneumonia, or both. Some due to foreign material entering trachea, swallowing of food, and food may appear at the only in certain types of food. May note difficulty May be a lack of interest in food or an interest

**SIGNS**

**DEFINITION**
Anorexia is the loss of appetite or lack of desire to eat. May be secondary to diseases leading to 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 neurotransmitter 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. Other primary disease conditions, such as infection, inflammation, injury, toxins, immunologic reactions, and neurexia, may cause anorexia via cytokines as well. In addition, a proteoglycan has been identified on the cell membranes of animals and has been named anorexigen. It reduces food intake and may be a satiety or anorexigenic substance.

**RISK FACTORS**

Anorexia in general appears to be the result of many factors. It reduces food intake and may be a satiety or anorexigenic 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.

**DIFFERENTIAL DIAGNOSIS**

**Anorexia**

- Cole
- Euphagia
- Gastrointestinal ileus
- Gastric ulcers and pyloric stenosis
- Peritonitis
- Secondary to a primary disease process in any organ system
- Renal failure (nephrosis)
- Renal tubular acidosis
- Cardiac amyloidosis
- Severe respiratory distress
- Depression of the nervous system—especially cerebral disorders
- Inflammation or endotension
- Injury
- Toxicosis (e.g., monein, lead)
- Immunologic reactions
- Malnutrition
- Necrosis
- Secondary to diseases leading to dehydration, electrolyte imbalances, or acid-base disorders
- Hypersalivation
- Side effect of morolamide or toltrazuril or cyproheptadine

**Dysphagia**

Food prehension problems may be due to:

- Inability to swallow
- Hypoesthesia of the face (CN-VI)
- Neurogenic atrophy of the masticatory muscles (CN-V, motor component)
- Bilateral paralysis of facial muscles (CN-VII)
- May experience partial food swollen ("gulping")
- Oral lesions

**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
- Tumors
- Immunologic reactions
- Malnutrition
- Necrosis
- Dehydration
- Electrolyte imbalances
- Acid-base disorders
- Severe respiratory distress
- Neurologic disorders
- Urinary or renal tubular acidosis
- Cardiac disease
- Metabolic disorders
- Side effects of medications
- Pain

Food prehension 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
- Mastication problems may be due to:
- Pain in teeth, mandibles, maxilla, sinuses, muscles, or temporomandibular joint
- Neurologic dysfunction
- Swallowing problems may be due to:
- Pain in pharynx or esophagus
- Mechanical obstructions in pharynx or esophagus
- Neurologic dysfunction (e.g., CN-X, although questionable likely)
- Unpalatable food due to contamination or spoilage

**DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS**

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**RISK FACTORS**

Anorexia and Decreased Food Intake

**SIGNS**

**DEFINITION**
Anorexia is the loss of appetite or lack of desire to eat. May be secondary to diseases leading to 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 neurotransmitter 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. Other primary disease conditions, such as infection, inflammation, injury, toxins, immunologic reactions, and neurexia, may cause anorexia via cytokines as well. In addition, a proteoglycan has been identified on the cell membranes of animals and has been named anorexigen. It reduces food intake and may be a satiety or anorexigenic 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.

**SIGNALMENT**

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 to prehend, chew, or swallowing of food, and food may appear at the mouth. Nasal discharge and cough may 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 (salivation) due to:
  - Inability to swallow
  - Hypoesthesia of the face (CN-VI)
  - Neurogenic atrophy of the masticatory muscles (CN-V, motor component)
  - Bilateral paralysis of facial muscles (CN-VII)
- May experience partially food swollen ("gulping")
- Oral lesions

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- Neurologic dysfunction (e.g., CN-X, although questionable likely)
- Unpalatable food due to contamination or spoilage
**IMAGING**

- Based on primary disease processes

**CBC/BIOCHEMISTRY/URINALYSIS**

- For swallowing problems

**DRUG(S) OF CHOICE**

- Depends on primary disease process

**CONTRAINDICATIONS**

- Oral administration of 40 g of KCl once or twice daily in anorectic patients

**FOLLOW-UP**

- Patient monitoring

**MISCELLANEOUS**

- Depends on the underlying cause

**DIET/ACTIVITY**

- Offer highly palatable and varied food in cases of anorexia. Supply food that is easy to chew and swallow in case of dysphagia. Forage-feeding by nasogastric intubation or parenteral nutrition may be required. Activity should be limited to stall rest or hand-walking in most cases.

**MEDICATIONS**

- Strategy depends on primary disease process

**EXPECTED COURSE AND PROGNOSIS**

- Depends on the underlying cause

**ASSOCIATED CONDITIONS**

- Other primary disease conditions, such as infection, inflammation, injury, toxins, immunologic reactions, and sepsis

**SYMPTOMS**

- Oral examination for painful chewing

**TREATMENT**

- Depends on the primary problem

- Secondary or conditional PCM involves weight loss with prolonged anorexia

- Aspiration pneumonia occurs secondary to dysphagia.
anthrax

**Basics**

**Overview**
Anthrax is a rapidly fatal septicaemic 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 exotoxin 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 4–8 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 purpura 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, forage, 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 are more common than in horses than in ruminants.

**Diagnosis**

**Differential Diagnosis**
- Lightning strikes can be differentiated on the basis of history of storms and absence of post-mortem findings typical of anthrax.
- Toxemia can be differentiated based on history and lack of post-mortem findings typical of anthrax.
- Malignant edema may appear similar, but ulceration of the skin is not found with anthrax.

**Pathologic Findings**
- 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.

**Other Diagnostic Procedures**
- Organisms may be seen by microscopic examination of blood smears 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.

**Other Laboratory Tests**
- Bacteriological culture may be diagnostic.
- Anthrax antiserum may be useful but is not available in the United States.
- Histological examination of tissues is not practical.

**Imaging**
- Routine laboratory findings have not been reported.

**Other Diagnostic Procedures**
- Necropsy should not be performed if anthrax is strongly suspected. Diagnosis can be made without necropsy.
- Dark, nonclotting blood from orifices; fever; edema; 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.

**Treatment**
- Treatment is usually limited 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 q6–12 h) or oxytetracycline (5–11 mg/kg IV q12 h) is traditionally recommended. Enrofloxacin (7.5 mg/kg PO q24h or 5 mg/kg IV q24h) is potentially a good choice in adult horses. Continue treatment 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 authorities 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 arthritic live 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 infection 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.
- Carcinogenic anthrax is the most common form in human beings, resulting from inoculation of an open wound with spores.

**Synonyms**
- Woolsorters’ disease
- Charbon
- Splenic fever

**Suggested Reading**
- Author: Laura K. Reilly
- Consulting Editors: Ashley G. Boyle and Corinne R. Sweeney
Anticoagulant Rodenticide Toxicosis

BASICS

OVERVIEW
• Ingestion of anticoagulant rodenticides interferes with normal blood clotting in horses.
• Anticoagulant rodenticides are the most commonly used class of rodenticides.
• First-generation anticoagulants (e.g., warfarin, pindone, coumafuryl, coumachlor) are short-acting coumarin derivatives requiring multiple feedings to result in toxicosis.
• Intermediate anticoagulants (e.g., chlorophacinone, diphacinone) require fewer feedings than first-generation chemicals and, thus, are more toxic to nontarget species.
• Second-generation anticoagulants (i.e., brodifacoum, bromadialone, difethialone) are highly toxic to nontarget species after a single feeding.
• Most anticoagulant rodenticides commonly used today are long-acting, second-generation anticoagulants, with activity in the body of 9–13 months.
• 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 hemorrhage.

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 interfere with normal blood clotting in the body.

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.

CAUSES AND RISK FACTORS
The mechanism of anticoagulant rodenticide toxicosis is the same as that for dicumarol toxicosis.

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.
• Severe liver disease—clinical pathology, liver biopsy.

SIGNS
• Signs generally manifest within 3–5 days after ingesting bait.
• Hemorrhage—internal or external.
• Signs generally manifest within 3–5 days after ingesting bait.

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.

PREVENTION/AVOIDANCE
• Prevent access to bait packages.

POSSIBLE COMPLICATIONS
N/A

EXPECTED COURSE AND PROGNOSIS
Prognosis is based on the severity of blood loss and damage to organ systems affected by hemorrhage.

MISCELLANEOUS CONDITIONS

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

PREGNANCY
• Lactating mares may excrete anticoagulant rodenticides in their milk.
• Monitor foals for any coagulopathies, and treat with vitamin K₃ if PT rises.

SEE ALSO
• Dicumarol (moldy sweet clover) toxicosis

DIAGNOSTIC PROCEDURES
N/A

IMAGING
N/A

DIAGNOSIS

N/A

CONTRIBUTED BY
Ayala I, Rodriguez MJ, Martos N, McConnico RS, Copedge K, Bischoff KL.

SUGGESTED READING

AUTHOR
Anita M. Kore

Consulting Editor
Robert H. Popperne

MEDICATIONS

DRUG(S) OF CHOICE
• Vitamin K₃ (phytonadione 2.5 mg/kg q12h SIQ 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 diarrhea (70% sorbitol at 3 mL/kg or sodium or magnesium sulfate at 250–500 mg/kg).

CONTRAINDICATIONS/POSSIBLE INTERACTIONS
• Do not use vitamin K₃ (menadione) in horses. Vitamin K₃ is ineffective against anticoagulant rodenticide toxicosis and is nephrotoxic.
• Medications that are highly plasma protein bound may exacerbate toxicosis.
• Drugs generally contraindicated are NSAIDs, phenothiazine tranquilizers, local anesthetics, antimicrobials, nitrofurans, anticoagulants, anabolic steroids, and epinephrine.

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.

PREVENTION/AVOIDANCE
• Prevent access to bait packages.

POSSIBLE COMPLICATIONS
N/A

EXPECTED COURSE AND PROGNOSIS
Prognosis is based on the severity of blood loss and damage to organ systems affected by hemorrhage.


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

SIGNALMENT

**Breed Predilections**
No age, sex, or breed predisposition documented

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

- Progressive abdominal distention should increase suspicion of uroperitoneum.
- Repeated posturing to urinate, with little urine passed, supports urinary tract obstruction.

CBC/BIOCHEMISTRY/URINALYSIS

- Normal to high PCV in most cases; mild to moderate anemia possible with terminal CRF.
- Moderate to severe increases in BUN (50–150 mg/dL) and Cr (2.0–20 mg/dL).
- Variable hypernatremia, hypochloremia, hyperkalemia, hypocalcemia, and hyperphosphatemia—hyperkalemia and hyperphosphatemia most common with intrinsic ARF; hyperkalemia most apparent with urinary tract disruption and development of uroperitoneum.
- Mild to moderate metabolic acidosis—depending on the underlying disease process.
- Mild to moderate hyperglycemia—attributed to stress.
- USG—high (>1.035) with physiologic oliguria, low (<1.020) with oliguria due to intrinsic ARF; specific gravity best assessed in urine collected during initial patient evaluation (before rehydration) or while the horse is not receiving fluids.
- Oliguria with intrinsic ARF may be accompanied by mild to moderate proteinuria, glucosuria, pigmenturia, and increased numbers of RBCs and casts on sediment examination.
- Urine pH—normal to acidic, especially with concurrent depletion of body potassium stores.

IMAGING

Transabdominal and Ultrasonography
- Kidneys may be enlarged, with loss of detail of corticomedullary junction, in intrinsic ARF.
- Kidneys typically are reduced in size, with increased parenchymal echogenicity, in CRF.

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—after 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 oliguric 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

**FOLLOW-UP**

**PATIENT MONITORING**
- Assess clinical status (emphasizing hydration), urine output, and body weight frequently for the first 3 days.
- Assess magnitude of anemia 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.

**ZOOntIC POTENTIAL**
Leptospirosis has infectious and zoonotic potential; avoid direct contact with infective urine.

**SEE ALSO**
- ARF
- CRF
- Urolithiasis
- 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**

**DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS**
- Pulmonic regurgitation
- Mitral regurgitation
- Aortic regurgitation
- Ventricular septal defect

**DIAGNOSTIC PROCEDURES**
- Thoracic Radiography
- Cardiac Catheterization
- Echocardiography
- Electrocardiography
- Continuous 24-Hour Holter Monitoring

**PATHOLOGIC FINDINGS**
- Aortic stenosis
- Mitral valve prolapse
- Aortic regurgitation
- Ventricular septal defect

**RISK FACTORS**
- Old age
- Hypertension
- Left atrial enlargement

**DEFINITION**
Disease characterized by the flow of blood from the aorta back into the left ventricle during diastole, resulting in decreased arterial pressure and increased left ventricular volume.

**SYMPTOMS**
- Weakens in arterial pulses
- Atrial fibrillation
- Ventricular premature depolarizations
- Cardiac enlargement
- Peripheral edema

**CAUSES**
- Degenerative changes of the aortic leaflets
- Inflammatory cell infiltrate
- Aortic root dilatation
- Innominate aortic stenosis
- Aortic regurgitation
- Mitral valve prolapse

**DIAGNOSTIC PROCEDURES**
- Cardiac Catheterization
- Thoracic Radiography
- Echocardiography
- Electrocardiography
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- Hypertension
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**AORTIC REGURGITATION**

**DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS**
- Pulmonic regurgitation
- Mitral regurgitation
- Aortic regurgitation
- Ventricular septal defect

**DIAGNOSTIC PROCEDURES**
- Cardiac Catheterization
- Thoracic Radiography
- Echocardiography
- Electrocardiography
- Continuous 24-Hour Holter Monitoring

**PATHOLOGIC FINDINGS**
- Aortic stenosis
- Mitral valve prolapse
- Aortic regurgitation
- Ventricular septal defect

**RISK FACTORS**
- Old age
- Hypertension
- Left atrial enlargement
- Mitral valve prolapse
- Aortic regurgitation
- Ventricular septal defect
**Aortic Regurgitation**

**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.
- Regularly monitor cardiac rhythm; any irregularities other than second-degree AV block should prompt ECG.
- Carefully monitor for exercise intolerance, respiratory distress, prolonged recovery after exercise, inconstant resting 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 sport.

**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 mos 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**
- 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 of 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 mos of initiating treatment.

**MISCELLANEOUS**

**ASSOCIATED CONDITIONS**
N/A

**AGE-RELATED FACTORS**
- Old horses are more likely to be affected.

**ZONOTIC POTENTIAL**
N/A

**PREGNANCY**
- Affected mares should not experience any problems with pregnancy unless the regurgitation is severe.
- Treat pregnant affected mares with congestive heart failure for the underlying cardiac disease with positive inotropic drugs and diuretics; ACE inhibitors are contraindicated because of potential adverse effects on the fetus.

**SYNONYMS**
- Aortic insufficiency

**SEE ALSO**
- Infective endocarditis
- Ventricular septal defect

**ABBREVIATIONS**
- ACE = angiotensin-converting enzyme
- AV = atriovenous
- CK-MB = MB isoenzyme of creatine kinase
- B-HBDH = α-hydroxybutyrate dehydrogenase
- LDH = lactate dehydrogenase
- PMI = point of maximal intensity

**Suggested Reading**

**Author** Virginia B. Reef

**Consulting Editor** Cola M. Marr

**ABBREVIATIONS**
- Aortic insufficiency
- Ventricular septal defect

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**Suggested Reading**

**Author** Virginia B. Reef

**Consulting Editor** Cola M. Marr
**Aortic Root Rupture**

**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 exsanguination into the thoracic cavity, cardiac tamponade from hemopericardium, or a shunt between the aorta and heart.
- **With an aortic rupture confined to the right sinus of Valsalva,** an aortic aneurysm 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.

**SIGNMENT**
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—jugal pulses and dilatation, ventricular tachycardia (unasifical), 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.
- Abnormal parasite migration in the ascending aorta is unlikely.

**RISK FACTORS**
- Aortic aneurysm
- Aortitis

**DIAGNOSIS**

**Differential Diagnosis**

**Ventricular Septal Defect with Aortic Regurgitation**
- Murmurs are systolic (band shaped and pansystolic) and diastolic (holodiastolic and decrescendo), not continuous.
- Aortic regurgitation is severe.
- No history of acute colic or distress
- Differentiate echocardiographically.

**Patent Ductus Arteriosus**
- No history of acute colic or distress
- Differentiate echocardiographically.

**CBC/Biochemistry/Urinalysis**
Elevated serum creatinine and BUN may occur because of impaired renal perfusion, which is associated 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 flaring in the right atrium or ventricle.

**RISK FACTORS**
- Anechic to echocic fluid may be detected dissecting subendocardically into the interventricular septum, most frequently along the right ventricular side; however, dissection of blood subendocardically along the left side also occurs.
- Right atrial or ventricular enlargement if the aorta has ruptured into one of these chambers.
- Paradoxical septal 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.
- Hypodynamic interventricular septum and left ventricular free wall are associated with left ventricular volume overload, producing increased fractional shortening, until the myoscardium 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 flow.

**Thoracic Radiography**
- An enlarged cardiac silhouette should be present in horses with a large aortiecardiac 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 aortiecardiac fistula into the right ventricle.
- With a shunt into the right atrium, right atrial pressures and oxygen saturation also are elevated.
Aortic Root Rupture

**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 aorto-cardiac fistula.
- Path of the dissection can be traced and the rupture into the right atrium, tricuspid valve, right ventricle, or left ventricle confirmed.
- Dissociation 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 (i.e., aortic root) aneurysm.

**Anterolateral Dissection**
- An absence of media in the left sinus of Valsalva and aortic root aneurysm.
- Fibrosis and scarring of the rupture site have been reported in old breeding stallions that died of unrelated causes.
- Bacterial and inflammatory enlargement usually is detected, and hepatic congestion and pulmonary edema may be present.

**TREATMENT**

**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 multif orm, 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 for any type of athletic work because of the risk of sudden death associated with further aortic rupture or development of fatal 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

**MEDICATIONS**

**DRUG(S) OF CHOICE**

**Antiarhythmics**
- Indicated with multif orm 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 are very effective in converting refractory ventricular tachycardia.

**Drug Selection**
States (but is available abroad); only an oral form is available in this country.
- May have a synergistic effect with propranolol in horses with refractory ventricular tachycardia

**FOLLOW-UP**

**PATIENT MONITORING**
- Routine monitoring of heart rate and of respiratory rate and rhythm after conversion to sinus rhythm.
- Persistent tachycardia, tachycardia, or new arrhythmias indicate deterioration in clinical status.

**ACE Inhibitors**
- May be indicated in stallions to decrease maintenance 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.

**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 systemic blood pressure
- Propafenone
  - Very effective in converting refractory ventricular tachycardia
  - The IV form is not available in the United States (but is available abroad); only an oral form is available in this country.
  - May have a synergistic effect with propranolol in horses with refractory ventricular tachycardia

**CONTRAINDICATIONS**
- Other vasodilators or antihypertensive drugs have the potential to adversely affect the stallion’s libido, breeding performance, or fertility.

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**FOLLOW-UP**

**PATIENT MONITORING**
- Routine monitoring of heart rate and of respiratory rate and rhythm after conversion to sinus rhythm.
- Persistent tachycardia, 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 chordae 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
- Aorticocardiac fistula

SEE ALSO
- CNS = central nervous system

Suggested Reading

Author Virginia B. Reef
Consulting Editor Celia M. Marr
Arsenic Toxicosis

**OVERVIEW**
- Exposure to excessive amounts of arsenic results in auto-intoxication.
- Toxicity depends on the form of arsenic ingested.
- Tetravalent inorganic arsenicals cause cardiovascular collapse and renal damage, whereas trivalent inorganic arsenicals primarily affect the liver.
- Arsenic is rapidly excreted after exposure.
- Urine, whole blood, or GI contents can be measured to assess arsenic levels.

**CAUSES AND RISK FACTORS**
- Ingestion of arsenic-containing products or arsenic-contaminated soils, water, or feed.
- Adverse GI and renal effects.
- Use NSAIDs cautiously because of possible adverse GI and renal effects.

**SIGNS**
- As mentioned in previous use.
- Suggestive diagnosis.
- Expected course and prognosis depend on the severity of clinical signs.
- If the animal survives, recovery should be complete.

**DIAGNOSIS**
- Hematologic changes—elevated PCV and plasma total protein.
- Leukopenia with degenerative changes in PMNs.
- Anemia
- Elevation of serum creatinine and blood urea nitrogen.
- Hyperkalemia
- Hyperglycemia
- Hyperphosphatemia
- Elevated LDH and CK

**INVESTIGATION**
- Serum electrolytes
- Serum calcium and magnesium
- BUN and creatinine
- Leukocyte count and differential
- Hemoglobin
- Hematocrit
- Platelet count
- Serum albumin and globulin
- Urinalysis

**MEDICATIONS**
- Butorphanol (0.02–0.03 mg/kg IV).
- Rectal sedatives—mineral oil or kaolin-pectin.
- The use of activated charcoal is controversial.

**TREATMENT**
- Immediate activated charcoal administration with fluids.
- Administer multivitamins.

**RECOMMENDATIONS**
- Avoidance of exposure to arsenic is the best practice.
- Consultation with a veterinarian is recommended in cases of suspected arsenic toxicity.

**SUGGESTED READING**
Artificial Insemination

**BASICS**

**DEFINITION/OVERVIEW**
- Extended, fresh, cooled, frozen semen introduced into the mare's uterus using aseptic technique
- Standard AI—minimum 300–1000 × 10⁶ PMS deposited into uterine body
- TDI or low-dose AI—1–25 × 10⁶ PMS deposited into tip 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 impulses or recent surgical repair from further breeding-related trauma.
- Semen quality assessed before AI.
- Low-dose AI—stallions with limited availability or costly semen due to:
  - Exercise 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

**SIGNAMENT**
- 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; order semen—overnight shipment
- Mare management—serial, daily teasing, Teasing and Examinations
- New frozen semen strategy for AI if have multiple doses:
  - Deslorelin or hCG when dominant follicle ≥35 mm
  - TRP and US—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
    - Teratogenic 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.

**Advantages**
- Reduce cost and risk of transport, mares rebred next day.
- Mare's uterus is best incubator for sperm, not a chilled shipper.

**Frozen Thawed Semen**
- Precise timing of AI post-thaw longevity is reduced to ≤12–24 hr.
- Mare management—serial, daily teasing, TRP, and US
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  - 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 few as 14 × 10^6 motile, frozen-thawed sperm.
  - Average of 60–150 × 10^6 of PMS for DHI.
- Semen is deposited at the UTJ, tip of 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 hr), cooled (24 hr), or frozen (6–12 hr) semen, rebreed the mare.
- Older ov or semen—due to poor timing, percentage of EED increases.

**OTHER LABORATORY TESTS**
- Progesterone level of >1 ng/mL confirms conception.
- Number of straws needed depends on post-thaw motility and method of AI.
- Thawing protocols vary and are reported ideally to be paired with a particular freezing method. Seek specific information regarding thawing. In the absence of a recommended protocol, 37°C for 30–60 seconds may provide an acceptable alternative.

**TREATMENT**
- 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.
- Early identification of possible mare problems
- Maximize the likelihood of first-cycle conception.
- Pregnancy rates are lower and EED higher if ovulation has not occurred within the recommended times (fresh, cooled, frozen).

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**DIAGNOSTIC PROCEDURES**

**Semen Analysis**
- Minimum parameters—volume, motility, concentration
- Morphology—optional, but of particular use if stallion has fertility problems
- Small sample of cooled or frozen semen should be saved and warmed (at 37°C) to evaluate immediately after AI
- Slide, coverslip, and pip—prewarm, stallion hand.
- Lubricant applied to dorsum of the gloved hand.
- Sterile sleeve on arm and nonspermicidal plastic plunger (e.g., Air-tite) containing the semen is attached to the pipet, and the semen is slowly deposited into the uterus. The remaining semen in the pipet is delivered by using a small bolus of air (1 mL) in the syringe.

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- Mare management varies according to method of semen preservation.
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-thawing, 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 PMIE.

Low-Dose Insemination Procedures (LDI)
- Sedation of the mare is recommended. Procedure should be performed quickly (≤10 min) and avoid inducing uterine trauma.
- 

Ovulation induction most effective if follicle is ≥35 mm
- 

Small catheter is passed through the endoscope’s channel and semen deposited at/on the UTJ.
- 

Endoscopic AI—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 AI pipet through the cervix toward the tip of the uterine horn ipsilateral to the dominant follicle.
- 

Endoscope’s channel and semen deposited at/on the UTJ.
- 

Manual TRP elevation of the tip of the horn, in a proper and timely manner
- 

Semen is deposited close to or onto the uterus lumen or to the tip of the horn, in a proper and timely manner
- 

Misidentification of stallions/mares
- 

Semen collection and artificial insemination.
- 

Possible complications
- 

AV preparation, handling, maintenance
- 

Semen evaluation at collection—ship semen, normal
- 

Semen evaluation, abnormal
- 

Early embryonic death
- 

Conception failure
- 

Due to poor timing; pregnancy rate decreased by 30 days; increased EED

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
- 

US for pregnancy 14–15 days post-ovulation: rule out potential twins vs. lymphocytic cyst.
- 

Follow-up TRP and US—24–30 days, confirm heartbeat in the embryo.
- 

Serial TRP pregnancy examinations—45, 60, 90, and 120 days

MISCELLANEOUS

PREGNANCY

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
- 

Candidate selection for frozen semen breeding
- 

Most fertile—young, maiden and normal pluriparous mares
- 

Least fertile—old, maiden or barren and abnormal pluriparous mares
- 

Older eggs or semen
- 

Due to poor timing; pregnancy rate decreased by 30 days; increased EED

PRECAUTIONS

See Endometritis.

CONTRAINDICATIONS

See Endometritis.

SYNONYMS

Artificial breeding

SEE ALSO

- 

Conception failure
- 

Delayed uterine clearance
- 

Early embryonic death
- 

Endometritis
- 

Semen evaluation, abnormal
- 

Semen evaluation, normal
- 

Vaginal diseases

ABBREVIATIONS

- 

AI = artificial insemination
- 

AV = artificial vagina
- 

CEM = contagious equine metritis
- 

CH = corpus hemorrhagicum
- 

CL = corpus luteum
- 

DHI = deep horn insemination
- 

DUC = delayed uterine clearance
- 

EAA = equine ataxic anemia
- 

EED = early embryonic death
- 

EVA = equine vaginal arteritis
- 

GnRH = gonadotropin-releasing hormone
- 

hCG = human chorionic gonadotropin
- 

LRS = lactated Ringer’s solution
- 

OVD = ovulation depression
- 

PMIE = persistent mating-induced endometritis
- 

PMS = progressively motile sperm
- 

TRP = transrectal palpation
- 

US = ultrasonography, ultrasonography
- 

UTJ = utero-tubal junction

Suggested Reading


Author Maria E. Cadario

Consulting Editor Carla L. Carleton
bilateral arytenoid cartilage to fully abduct during forced inspiration and/or to cause collapsing airway pressure during inspiration.

**Diagnostic Procedures**

- The diagnosis is established on the basis of evaluating exercise intolerance, or both arytenoid cartilages.
- With arytenoid cartilage into the airway. The size or location of the protruding mass has no correlation with the amount of ablation 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 eddies.
- Consider laser-assisted excision of intralaryngeal granulations if the affected arytenoid cartilage remains abductory 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 tracheostomy 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 NSAISS. Chronic case: none, other than routine preoperative antimicrobial and anti-inflammatory agents. Use of nasopharyngeal spray, consisting of various anti-inflammatory and antimicrobial agents (e.g., 250 mL of 90% DMSO, 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**
  - Videodenscopy 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 swellings of the arytenoid cartilage may respond favorably to NSAIDS, topical anti-inflammatory agents, and antibiotics. Ununited 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 “kempjection.” 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.

**Suggested Reading**


Authors Norm Ducharme and Richard P. Hacken

Consulting Editor Daniel Jean
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.

**DRUG(S) OF CHOICE**
- Anthelmintics available at present do not eliminate migrating larvae. Therefore, preventative therapy should be given until 1 year 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
  - Daily prophylactic administration of pyrantel tartrate (2.64 mg/kg) in the feed also prevents penetration of the intestinal wall by ascarid larvae.
  - Moxidectin 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-aspartate and L-α-amino glutamate to glutamate and α-ketoglutarate
- Present in many tissues—liver, striated muscle, cardiovascular (myocardium), Hemic (erythrocytes), hepatobiliary, and others
- Reported normal AST activity in horses varies from 48 to 456 IU/L

**PATHOPHYSIOLOGY**
- Increases in AST activity are typically indicative of hepatocellular 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 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 dozer animals.
- AST is a sensitive indicator of hepatocellular 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 increased AST and progressively decreasing or normal SD activity, cessation of the original insult, and the activity may remain increased for weeks.
- A similar interpretative approach is used when determining if muscle injury is present. Muscle and hepatocellular injury can occur concurrently, and increases in AST, CK, and SD may be seen together.

**SYSTEMS AFFECTED**
- Musculoskeletal
- Hepatobiliary
- Cardiovascular (myocardium)
- Hematologic (erythrocytes)

**GENETICS**
N/A

**INCIDENCE/PREVALENCE**
N/A

**GEOGRAPHIC DISTRIBUTION**
N/A

**SIGNALMENT**
- Depends on the primary disease process and secondary complications

**HISTORICAL**
- Depend on the cause of the increase in AST activity
- Sudden exercise or overtraining

**Physical Examination**
- Depends on the cause of the increases in AST activity
- Muscle disorders—fatigue or inability to move, stiffness, and recumbency
- Liver disorders—jaundice, neurologic deficits, dilated retinal, anorexia, abdominal pain, weight loss, and fever
- Clinical signs due to hepatic failure generally do not appear until 75% of the hepatic functional mass is lost.

**CAUSES**
- Degenerative conditions—cirrhosis, rhabdomyolysis, and cholelithiasis
- Anomalous, congenital diseases—polychromatophilic storage myopathy, biliary atresia
- Metabolic diseases—shock, hypovolemia, hepatoencephalopathy, hypovolemia, hypoglycemia, and vitamin E/selenium deficiency
- Infectious and immune-mediated diseases—hepatitis of various causes (e.g., viral, bacterial, protozoal, fungal, parasitic); serum sickness, amylloidosis, endotoxemia, and chronic active hepatitis
- Tonic or trauma—pyridoxaline, alkaloid-containing plants, ferox fumarate in newborn foals, corticosteroid, cancer bean, oak, and alkylic clover; fungal toxins, such as aflatoxins, cyclosporin acid, fumonisin, phaloidin (i.e., mushrooms), rubratoxins blue-green algae; and chemical compounds/elements, such as ethanol, chlorinated hydrocarbons, carbon tetrachloride, monosodium, copper, iron, and petroleum and its products

**RISK FACTORS**
- Risk factors vary according to the specific disease
- Familial disease, exposure to infected animals, overweight and miniature ponies, post nutrition, excessive exercise, exposure to toxic compounds or plants, or excessive exercise
- Halothane anaphylaxis, particularly of prolonged duration, may result in hepatic injury in horses

**DIAGNOSIS**

**DIFFERENTIAL DIAGNOSIS**
See Causes.

**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 leukemias; morphologic changes of the leukocytes (e.g., neutrophil toxicity in inflammation; neutrophil cells) also may be seen.
- Platelets—quantitative decrease and increases may be seen with a variety of systemic diseases that may affect the liver or striated muscle.
- Serum/Plasma Biochemistry Profile
  - Glucose—increased in diabetes mellitus, glucocorticoid influence (e.g., exogenous, endogenous), decreased in end-stage liver disease, sepsis/endotoxemia
  - BUN—increased in severe rhabdomyolysis from secondary renal injury; decreased in liver insufficiency and end-stage liver disease from decreased conversion of ammonia to urea
  - Albumin—decreased in end-stage liver disease from decreased production; minimally to mildly decreased in inflammation
  - Globulins—generally increased in end-stage liver disease and with chronic antigenic stimulation
  - SD—increased with acute and ongoing hepatocellular injury
  - ALP—increased with concurrent cholestatic disease
  - GGT—increased with cholestatic disease or hepatocellular injury
  - CK—increased with acute or ongoing muscle injury
- Conjugated bilirubin—increased in cholestatic disease
- Unconjugated bilirubin—increased with anemia and prehepatic cholestasis (i.e., massive in vitro hemolysis)
- Cholesterol—may be increased with cholestasis and lipid disorders, and decreased in hepatic insufficiency
- Triglycerides—increases may be associated with hepatic lipoprotein.
- Because of high AST activity in erythrocytes, hemolysis falsely decreases serum/plasma AST activity
- Prolonging 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.

**Urinalysis**
- Bilirubinuria—Conjugated bilirubin, desired by the commonly used dipstick and dye tablet method, indicates cholestatic disease and should not be increased if only hepatocellular injury is present.

**OTHER LABORATORY TESTS**
- SBA
  - Serum test for hepatobiliary disease, but not specific for the type of hepatobiliary disease
  - May be increased with cell injury, cholestasis, or hepatic insufficiency/decreased functional mass, specificity for the latter condition is greatly varied.

**BLACKWELL’S FIVE-MINUTE VETERINARY CONSULT**
Aspartate Aminotransferase (AST)

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/dysfunction. However, ammonia measurement requires special handling, which limits its general availability.

Plasma Ammonia

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/dysfunction. However, ammonia measurement requires special handling, which limits its general availability.

Other diagnostic imaging modalities such as ultrasound or computed tomography may be helpful in detecting infectious causes.

Other diagnostic procedures such as aspartate aminotransferase and alkaline phosphatase can be useful in detecting infectious causes.

TREATMENT

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 analyses 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.

EXPECTED COURSE AND PROGNOSIS

Depends on the primary disease process and secondary complications.

MISCELLANEOUS

ASSOCIATED CONDITIONS

Depends on the primary disease process and secondary complications.

AGE-RELATED FACTORS

See Signalment.

ZOONOTIC POTENTIAL

Infectious diseases such as salmonellosis.

PREGNANCY

See Signalment.

SYNONYMS

Previously known as glutamate oxaloacetate transaminase (SGOT).

SEE ALSO

See Causes.

ABBREVIATIONS

GI = gastrointestinal

ID = idiosyncratic liver injury

SBA = serum bilirubin

Suggested Reading


Aspiration Pneumonia

Blackwell’s Five-Minute Veterinary Consult
Daniel Jean

OVERVIEW
Microaspiration after ingestion of foreign material and bacteria into the lower respiratory tract. Causes include dysphagia, obstructive esophageal disorders, GI reflux, and accidental inhalation of foreign material (e.g., administration of medications into the lung via a nasogastric tube). Characterized by ventral consolidation of the lungs. Other organ systems may be involved depending on the primary cause.

SIGNALMENT
No sex or breed predisposition has been observed. Fresh appear more prone to GI reflux and subsequent AP.

SIGNALS

Historical Findings
- Dysphagia, pyrosis, or discharge of food, water, or milk from the nostrils may have been observed before the onset of respiratory signs. Recent history of drooling or tachypnea should be investigated.

Physical Examination Findings
- Clinical signs—apnea, anorexia, fever, tachypnea, dyspnea, tachycardia, and coughing.
- Food-sticking breath or nasal discharge suggests aeromastic infection.
- Alveolar lung sounds are often heard on auscultation.

CAUSES AND RISK FACTORS
Dysphagia
- Neurologic diseases affecting cranial nerves IX and X—gastric pouch disease, bovine head trauma, and viral encephalitis
- Primary myopathies of pharynx and larynx—muscle atrophy
- Vocal cord paralysis—white muscle disease
- Congenital abnormalities—cleft palate and hypoplasia of the soft palate
- Foreign body—pharyngeal and laryngeal surgery

Esophageal Disorders
- Esophageal obstruction—foreign body, food impaction, atresia, compression (e.g., abscess, neoplasia)
- Megaeosophagus
- Esophageal diverticula
- Esophageal reflux

GI Reflux
Gastric reflux obstruction is usually secondary to ulcer disease in foals. Accidental Inhalation of a Foreign Body Administration of fluids by drosering or nasogastric tubes

DIAGNOSIS
Differential Diagnosis
- Acute bronchopneumonia often follows viral infection or stressful events (e.g., anesthesia, transportation, overexertion, trauma).
- Pharyngitis—possible complication of AP, bronchopneumonia, pharyngeal abscess, or secondary to thoracic trauma or esophageal rupture; aspiration, penetration, ultrasonography, radiography, or esophagoscopy helps confirm partial effusion.
- Intestinal pneumonia—radiographic most common cause results mainly increase in rostral lung activity
- Respiratory distress syndrome—extremely low-dose neonatal 28–48 hours after birth caused by surfactant deficiency; thoracic radiography typically shows diffuse, ground-glass appearance of the lungs with air bronchograms.

CBC/Biochemistry/Urinalysis
- Increased WBC count with absolute neutrophilia is diagnostic for lead toxicity.
- Decreased whole-blood selenium concentration and gluthione peroxidase activity with increased serum creatinine kinase (CR) and aspartate amino-transferase (AST) are consistent with white muscle disease.
- Hyperkalemic periodic paralysis may be diagnosed by genetic testing or by finding hyperkalemic during clinical episodes.

Imaging
- Thoracic radiography commonly reveals ventral lung opacity often obscuring the cardiac silhouette.
- Contrast radiography may help to diagnose causes of esophageal obstructions.
- Thoracic ultrasonography is a sensitive means of detecting pleural effusion.

Other Diagnostic Procedures
- Thoracic ultrasonography can achieve drainage; a one-way valve attached to the tube prevents pneumothorax.
- Thoracic radiography commonly reveals marked increase in overall lung consolidation of the ventral region of the lungs.
- Increased blood and tissue concentrations of lead are diagnostic for lead toxicity.
- Sulfur-stained radiographs may help to identify the primary cause.

Pathologic Findings
- Consequences of the ventral region of the lungs
- Acute cause—recently affected areas are hemorrhagic and edematous
- Chronic cause—affected lung may be mummified and filled with purulent material
- Fluid space involvement—thoracic exudate and inflammatory response

Treatment
- Treat more drosering according to the cause— restoring airway patency; drain pleural effusion, etc.
- Nasal oxygen (to 10 L/min) if severe hypoxemia (PaO2 < 60 mm Hg). The primary disease must be treated.
- Antibiotic treatment is imperative.
- Thoracic ultrasonography may be used via an indwelling nasogastric tube.
- With pleural effusion, thoracentesis or placement of indwelling chest tubes can achieve drainage; a one-way valve attached to the tube prevents pneumothorax.
- Administer fluid therapy as needed.

Medications

Drug(s) of Choice
- Antibiotics: systemic administration of broad-spectrum antibiotics while waiting for culture results. Depending combination includes

SUGGESTED READING

Author: Laurence Couplin Consulting Editor: Daniel Joan.

ABBREVIATIONS
AP = aspiration pneumonia
GI = gastrointestinal

MISCELLANEOUS

SEE ALSO
- Chemotherapeutic agents
- Pharyngitis
- Acute respiratory distress syndrome
The cyst can be approached surgically.

**Palpation**

Aspirated fluid is white to gray, milky to creamy in appearance and odorless. A technique has been reported using intrallesional injection of neutral-buffered 10% formalin until leakage around the needle is seen (2–4.5 mL). There is transient swelling within 24 hours of injection of formalin. Desiccation of the cyst occurs after a few weeks.

**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. The cyst then is dissected in its entirety, and the wound is closed. Alternatively, the wound is left open to heal by second intention.
- Another option is to open the cyst ventrally through the skin over the dorsum of the nasomaxillary notch. Drain the contents, and remove the lining using a burr instrument. In this technique, the wound is left open to heal by second intention.
- A technique has been reported using intrallesional injection of neutral-buffered 10% formalin for treatment of epidermal inclusion cysts in five horses. J Am Vet Med Assoc. 2003;223:221–222.

**Medications**

Draining and cauterizing or sclerosing the cyst has been done using tincture of iodine, silver nitrate, or both followed by packing; this requires daily treatment and carries a high risk of recurrence.

**Contraindications/ Possible Interactions**

- Transient swelling if chemical ablation is used.
- Usual precautions for tetanus prophylaxis and asepsis of the surgical site.

**Possible Complications**

- A cyst may become abscessed if infection is introduced during centesis.
- Transient swelling after surgery.
- Recurrence if lining not removed.
- Infection at surgery site.
- Scar formation.
- White hair at surgery site.

**Expected Course and Prognosis**

Favorable prognosis for both leaving the atheroma untouched and for surgical removal if needed.

**Follow-up**

- Usual precautions for tetanus prophylaxis and asepsis of the surgical site.

**Associated Conditions**

In addition to false nostril cysts, other congenital cutaneous cysts reported in horses are dermoid cysts and, very rarely, dermoid cysts.

**Age-related factors**

May increase in size with age.

**Zoonotic Potential**

None.

**Pregnancy**

N/A

**See Also**

N/A

**Suggested Reading**


**Author**

Wendy Duckett

**Consulting Editor**

Daniel Jean
Atopic Dermatitis

BASICS

DEFINITION
Chronic, itchy, inflammatory, pruritic skin disease resulting from a predisposition to develop IgE-mediated hypersensitivities to induce cutaneous or systemically absorbed environmental allergens.

PATHOPHYSIOLOGY
The complex pathogenesis of equine AD is unknown. Susceptible animal is sensitized to environmental allergens resulting in the production of allergen-specific IgE. Upon further exposure to percutaneously absorbed or inhaled allergens, an immediate type I hypersensitivity ensues. The reaction commences by binding of allergen-specific IgE to FcεRI 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 from different mares—suggesting a dominant mode of inheritance.

INCIDENCE/PREVALENCE
Recognized worldwide; local environmental factors such as climate, seasons, high pollen and mold spore levels influence the seasonality, severity, and duration of signs.

SIGNALMENT
Breed Prepotency
Arabians, Thoroughbreds, Quarter Horses, and Warmbloods have been reported to be predisposed.

Mean Age and Range
Mean 3–6.5 years of age (2–12 years); 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
• Hallmark sign—chronic relapsing seasonal or nonseasonal pruritus and/or urticaria (rubbing, itching, biting themselves, stomping, tail flicking, rarely head shaking, and agitation) • 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 consist 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.

Historical
• Most commonly affected sites include face, pinnae, chest, ventral thorax and abdomen, extremity extensor and flexor surfaces • Other common sites include the mane, dorsolateral neck, crest, 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 atopy-associated recurrent airway obstruction include head shaking, sneezing, bilateral mucopurulent nasal discharge, conjunctivitis, dry unproductive coughs, labored breathing, stomping, and face rubbing on front legs or objects and excessive intolerance. • Uncommon clinical signs are head shaking and lassitude.

CAUSES
• Allergens (trees, grasses, weeds) • Molds (indoors and outdoors) • Animal danders (mouse, cat, cow, poultry, grain) • Possibly storage and house dust mites

RISK FACTORS
• Temperate environments with long allergy seasons, high pollen and mold spore levels • Concurrent pruritic dermatoses, such as insect hypersensitivity or cotnropazitap disease (summation effect)

DIAGNOSIS
DIFFERENTIAL DIAGNOSIS
• Insect hypersensitivity—may occur concurrently with AD • Exoparasites (pathogens and incidentals) • Cutaneous adverse food reaction—rare • Contact hypersensitivity • For respiratory disease—respiratory infection (bacterial, fungal, viral), congestive heart failure, and bronchitis

CBC/BIOCHEMISTRY/URINALYSIS
Eosinophilia is rare.

OTHER LABORATORY TESTS
Sericologic 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 antimicrobial and corticosteroid administration on test results are unknown. • Many false-positive and -negative results occur with the currently available assays. Lack of reproducibility of test results and sensitivity are common. • Do not use to diagnose cutaneous adverse reaction to food or supplements.

IMAGING
N/A

OTHER DIAGNOSTIC PROCEDURES

INTRANASAL TESTING
• Detects levels of allergen-specific IgE in the skin directed to a panel of allergens that are region specific. • 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, possibly avoidance or decrease in exposure. • Intradermal injection of allergens results in raised turgid wheals. The reaction is given a subjective score (usually 0–4+) based on its size and niggledy compared to the positive and negative controls. • Reactions are interpreted at 15–30 min for an immediate IgE-mediated reaction. 6 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 ophthalmic preparations and 30 days for parenteral corticosteroids. • Cytology from lesions or ulcers shows a neutrophilic exudate with intra- and/or extracellular cocci representative of a secondary folliculitis. • Perform skin scrapings to rule out ectoparasites. • Perform bacterial and DTM cultures to determine bacterial species and susceptibility and/or dermatophyte infections.

PATHOLOGICAL FINDINGS
• Skin biopsies—will rule out other differential diagnostic such as insect or food hypersensitivity • Histopathological changes—epidermal hyperplasia with superficial and deep, perivascular to interstitial dermatitis wherein the eosinophil 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
On-patient medical management

NURSING CARE
Frequent bathing using cool water (antimicrobial shampoos, sulfonated/alkylaryl acid, −/−/− colloid/ oatmeal times or leave-on conditionals) helps to remove allergens, craters, 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

- 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 dietary 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

- Non.

MEDICATIONS

DRUG(S) OF CHOICE

- Allergen-Specific Immunotherapy

SURGICAL CONSIDERATIONS

- Non.

CLIENT EDUCATION

- 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 atrophic hyperadrenocorticism due to chronic steroid administration

EXPECTED COURSE AND PROGNOSIS

- Not life-threatening unless intractable pruritus persists
- No reports of spontaneous remission exist

ASSOCIATED CONDITIONS

- Summer pasture–associated obstructive pulmonary disease

AGE-RELATED FACTORS

- None

ZOONOTIC POTENTIAL

- None

PREGNANCY

- Non.

SEE ALSO

- Allergen-specific immunotherapy
- Atopic dermatitis
- Bronchial hyperreactivity
- Eosinophilic granulomas
- Insect hypersensitivity
- Insect hypersensitivity
- Inflammatory bowel disease
- Summer pasture–associated obstructive pulmonary disease

SYNONYMS

- Equine atopy

ABBREVIATIONS

- AD: atopic dermatitis
- ASIT: allergen-specific immunotherapy
- DTH: delayed type hypersensitivity
- IDT: intradermal test
- ITM: intradermal test medium

Suggested Reading


- Author: Gwendolen Lorch

- Consulting Editor: Gwendolen Lorch

Atopic Dermatitis

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 amitriptyline and tricyclic antidepressants, do not use in patients with a history of cardiac arrhythmia, colic, glaucoma, or urinary retention disorders. Amitriptyline 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 corticosteroid-induced obesity, diabetes mellitus, polydipsia and polyuria, aggregation of bacterial folliculitis, decreased muscle mass, weight loss, poor wound healing, and behavior changes. If amitriptyline—can produce sedation and/or behavior changes, whole body or fine tremors or seizures. High doses of amitriptyline cause birth defects in laboratory animals. Amimhamitriptyline should only be used in pregnant or lactating animals if the benefit outweighs the risks. Do not administer amitriptyline intravenously in the horse due to potential CNS stimulation.
- Note drug withdrawal times and regulations pertaining to horse show or racing eligibility.

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. Amimhamitriptyline have an additive effect when combined with other CNS-depressant drugs, such as tranquillizers.

ALTERNATIVE DRUGS

Pulmonary neumoniae omega 3 and 6 fatty acids—variable response in decreasing pruritus; provide support for epidermal barrier function and anti-inflammatory properties. Use up to 2 wk of starting therapy. Exact dosing for home is lacking; the author uses 180 mg of EPA/10 lb q24h.

FOLLOW-UP

- 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.

SYNONYMS

- Equine atopy

ABBREVIATIONS

- AD: atopic dermatitis
- ASIT: allergen-specific immunotherapy
- DTH: delayed type hypersensitivity
- ITM: intradermal test medium

Suggested Reading


- Author: Gwendolen Lorch

- Consulting Editor: Gwendolen 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.

**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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**Figure 1.**
Base-apex lead, 25 mm/sec, 5 mm = 1 mV
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

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).

NURSING CARE
• Perform continuous ECG throughout attempted conversion to sinus rhythm.
• Keep horses quiet and unmoving during quinidine treatment.

ACTIVITY
• AF cases should not perform high-intensity exercise.
• AF cases usually can perform successfully as pleasure horses, in lower-level athletic competition, as broodmares, and as breeding stallions.

DIET
• Oral potassium supplementation may be indicated with low plasma potassium, low RBC potassium, or low urinary fractional excretion of potassium or with excessive sweating.
• Potassium chloride salt can be added to the feed (1 tbsp BID, gradually increasing to 1 oz BID).

CLIENT EDUCATION
• Discuss treatment-associated risks with owners—see Possible Complications.
• Discuss predisposing factors with owners to minimize the likelihood of future episodes.

SURGICAL CONSIDERATIONS
• Successful transvenous electrical conversion of horses under general anesthesia has been described.
• This utilizes a biphasic current delivered between electrodes placed in the right atrium and left pulmonary artery using pressure waveforms, echocardiography, and radiography to guide and confirm electrode placement.

• Initial reports of success rates from one center are very encouraging.

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 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.
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Atrial Fibrillation

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 arrhythmias; 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.

MISSING TEXT

ASSOCIATED CONDITIONS
Any cardiac disease resulting in atrial enlargement predisposes to atrial fibrillation.

AGE-RELATED FACTORS
- Old horses are more likely to have significant underlying cardiac disease with valvular insufficiency and atrial enlargement.
- These horses usually are not candidates for conversion because of significant underlying cardiac disease.

PREGNANCY
- Affected pregnant mares without underlying cardiac disease and congestive heart failure should not experience any problems.
- Affected pregnant mares with CHF can be treated for the underlying cardiac disease with positive inotropic drugs (e.g., digoxin) and diuretics (e.g., furosemide).

SYNONYMS
- Afi

SEE ALSO
- Congestive heart failure
- Mitral regurgitation

ABBREVIATIONS
- AF = atrial fibrillation
- AV = atrioventricular
- CK-MB = MB isoenzyme of creatine kinase
- CHF = congestive heart failure
- GI = gastrointestinal
- HBDH = 3-hydroxybutyrate dehydrogenase
- LDH = lactate dehydrogenase
- RBC = red blood cell

Suggested Reading

Author Virginia B. Reef
Consulting Editor Celia M. Marr
Atrial Septal Defect

**Basics**

**Definition**
- 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 seconundum defect), or in the most basal 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
- Historical
  - Exercise intolerance—medium to large ASDs
  - Congestive heart failure—large ASDs
- Physical Examination
  - No murmur may be present, or a coarse, band- or ejection-shaped, holosystolic murmur with P2 in pulmonic valve area may be detected.
  - Premature beats or an irregularly irregular rhythm of atrial fibrillation may be present with larger ASDs.
- Causes
  - Failed 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 hypostemic; differentiate echocardiographically.
- Pulmonary atresia—murmur usually louder; may have a continuous machinery murmur; foal is unthrifty, tachycardic, and hypostemic; differentiate echocardiographically.

**CBC/Biochemistry/Urinealysis**
- N/A

**Other Laboratory Tests**
- N/A

**Imaging**

**Electrocardiography**
- Atrial premature depolarizations or atrial fibrillation may be present in horses with right and left atrial enlargement.
- Persistent atrial fibrillation has been reported in some affected foals and horses.

**Echocardiography**
- Can determine location of the ASD
- Atrial septal dropout 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 with pulsed-wave or color-flow Doppler with suspected ASD.
- 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

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 drugs, vasodilators, and diuretics. Consider humane destruction if congestive heart failure develops, however, because only short-term symptomatic improvement can be expected.

NURSING CARE
N/A

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 unsanctioned activity and may be able to compete reasonably successfully in upper-level athletic competition.
- 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 ride or lower-level athletic competition.
- Horses with significant pulmonary artery hypertension can be monitored on an outpatient basis.

DIET
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 rates, cough, generalized venous distention, jaundice, or central edema; if detected, obtain a cardiac examination.

SURGICAL CONSIDERATIONS
- Closure of the ASD would be possible with a transaortic 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
DRUGS OF CHOICE
- Positive inotropic drugs, vasodilators, and diuretics. Consider humane destruction if congestive heart failure develops, however, because only short-term symptomatic improvement can be expected.

CONTRAINDICATIONS, PRECAUTIONS, POSSIBLE INTERACTIONS
N/A

ALTERNATIVE DRUGS

FOLLOW-UP

PATIENT MONITORING
- Frequently monitor cardiac rate, rhythm, and respiratory rate and effort.

PREVENTION/AVOIDANCE
N/A

POSSIBLE COMPLICATIONS

EXPECTED COURSE AND PROGNOSIS
- Large ASD—atrial fibrillation, congestive heart failure

PHYSICAL EXAMINATION

TREATMENT

SURGICAL CONSIDERATION

ALTERNATIVE TREATMENTS

PALLIATIVE CARE

MISCELLANEOUS

ASSOCIATED CONDITIONS
- Complex congenital cardiac disease, particularly tricuspid and pulmonic atresia, is likely.
- Tricuspid regurgitation can develop in horses with significant left atrial, right atrial, and right ventricular volume overload secondary to stretching of the tricuspid annulus.
- Pulmonic regurgitation can develop in horses with isolated defects.
- Pulmonic valve leaflets may no longer coapt with stretching of the pulmonary artery from the volume overload.

AGE-RELATED FACTORS
- Young horses are more likely to be diagnosed.

ZOO NTOIC POTENTIAL
N/A

PREGNANCY
- Breeding affected horses is discouraged. The condition is rare, however, and the heritable nature of this defect in horses is not known.

SYNONYMS
N/A

SEE ALSO
- Atrial fibrillation
- Supraventricular arrhythmias

ABBREVIATIONS
- ASD = atrial septal defect
- AV = atrioventricular
- PMI = point of maximal intensity

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 can 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

**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

**MEDICATIONS**

**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 3–4 mo of every other week treatment)
Aural Plaques

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

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AZOTEMIA AND UREMIA

BASICS

DEFINITION
- Azotemia—accumulation of nitrogenous waste (e.g., urea, uric acid, 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 SUN (serum urea nitrogen) typically are measured in serum and used as indices of azotemia.

PATHOPHYSIOLOGY
- Serum urea concentration is determined by rate of urea synthesis by hepatocytes 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 inefficiencies or failure (i.e., renal azotemia) or urinary obstruction (i.e., postrenal azotemia).
- Azotemia results from excretion of urine when urinary tract rupture (i.e., postrenal azotemia) results in accumulation of urine in the body cavity (abdominal or subcutaneous).
- Creatinine is a result of muscle creatine metabolism; serum levels reflect the rate of synthesis and rate of excretion. Rate of synthesis is relatively constant except in the face of rhabdomyolysis. Renal excretion is dependent on GFR. Creatinine is not resorbed by renal tubules. Low serum urea levels may result after prolonged diuresis as a result of impaired liver function. There is no clinical significance to decreased creatinine levels.

SYSTEMS AFFECTED
- Generalized or systemic effects—depression, weakness, weight loss, edema, and dehydration
- Cardiorespiratory—anaemia, uremic stanza, uricemic breath, cyanotic breath, gingivitis, oral/gingival ulceration, mild protein-losing enteropathy, ascites, and melena
- Neuromuscular—dysphoria, lethargy, gait imbalance, tremors, behavioural changes, seizures, and stupor
- Endocrine—secondary hyperparathyroidism, inadequate production of erythropoietin and 1,25-dihydroxycholecalciferol, decreased hormone clearance that prolongs plasma half-life (e.g., parathyroid, gastrin), decreased tissue sensitivity (e.g., insulin, parathormone), decreased hormone production (i.e., testosterone), and hyperconcentration to exorbitant homosism (i.e., parathormone)
- Cardiovascular—elevated blood pressure, heart murmur, and cardiac dysrhythmia
- Respiratory—dyspnoea, tachypnoea, anemia and impaired immune function

GENETICS
No genetic predisposition

GEOGRAPHIC DISTRIBUTION
Nonregenerative anemia caused by decreased renal hematopoietic production occurs with chronic renal failure.

SIGNS
General Comments
- Azotemia does not always equate to clinical signs of disease described here. Unless the animal is uremic, clinical findings are limited to the process causing azotemia—dysuria, urinary outflow tract obstruction, or rupture.

Historical
- Weight loss
- Anorexia
- Abnormal urination
- Depression
- Lethargy
- Dental tartar
- Enuresis
- Vocal pain
- Diarrhea
- Dystocia
- Abdominal distension
- Poor hair coat
- Prolonged purring to urination
- Polyuria/Polydipsia

Physical Examination
- Fever
- Anorexia
- Depression
- Oral pallor
- Poor body condition
- Ventral edema
- Oral ulceration
- Excessive dental tartar
- Seizure
- Colic
- Distended abdomen
- Urine scaling
- Dysuria
- Hematuria
- Halitosis

CAUSES
Prerenal Azotemia
- Renal hypoperfusion caused by decreased circulating volume or decreased blood pressure
- Protein catabolism associated with fever, infection, trauma, myositis, thermal injury, and corticosteroid therapy
- General anesthesia
- Prolonged exercise

Renal Azotemia
- Acute or chronic renal failure—primary renal dysfunction affecting glomeruli, renal tubules, renal interstitium or renal vascularization and impairing 60%–75% of renal function

Postrenal Azotemia
- Obstruction of the urinary tract
- Rupture of the urinary outflow tract

RISK FACTORS
Medical Conditions
- Renal disease
- Diabetes
- Endotoxemia
- Acute blood loss
- Septic shock
- Prolonged exercise
- Urolithiasis
- Exposure to nephrotoxic chemicals or plants
- Diuretics
- Acetaminophen
- Hepatic disease
- Neoplasia

Drugs
- Aminoglycosides
- NSAIDs
- Diuretics

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS
- Prerenal azotemia—dehydration, hypovolemia, acute blood loss, decreased cardiac output, exsanguineous disease syndrome, some colic cases
- Renal azotemia
  - Acute renal failure with increased or decreased urine output is suggestive of vitamin K or taurine, red maple leaf toxicosis, aminoglycoside toxicities, other nephrotoxic chemicals, vasomotor nephropathy, abnormal kidney size, and rarely leptospirosis.
  - Chronic renal failure with progressive weight loss, PU/PD, pitting edema, other signs over several weeks or months suggests chronic glomerulonephritis or pyelonephritis, amyloidosis, polycystic kidney disease, renal hypoplasia, and nephrolithiasis.
- Postrenal azotemia—abrupt decrease in urine output and acute signs of uremia, abdominal distension, stranguria and signs of colic may suggest ruptured uterus, ruptured bladder, or obstructive urolithiasis.

CBC/BIOCHEMISTRY/URINALYSIS
CBC
Nonregenerative anemia caused by decreased erythropoietic production occurs with chronic renal failure.

Biochemistry
- Consider hydration status, presenting complaint and physical exam findings when interpreting SUN/Cr levels.
- In horses, the SUN/Cr ratio is unstable in differentiating acute from chronic renal failure.
- Correlating dehydration deficits and restoring renal perfusion dramatically reduces SUN and Cr in patients with prerenal azotemia.
- Relieving outflow obstruction or correcting the rent in the excretory pathway rapidly decreases the degree of azotemia in patients with postrenal azotemia.
- Hyperkalemia and hyponatremia are common in horses with renal disease and can occur with third-compartment sequestration of fluid as in urethrolithiasis.
- Hyperkalemia is a common finding in urinary tract disruption and ureterosigmoidostomy.
- Calcium and phosphorus levels vary in renal disease.
- Hyperkalemia and hyperphosphatemia often are found with chronic renal failure; hyperkalemia and hyperphosphatemia are seen with acute renal failure.
- Hyperkalemia in renal failure depends on dietary content and intake of calcium.

Urinalysis
- Urine specific gravity >1.020 and urine osmolality >500 mOsm/kg are consistent with prerenal azotemia.
- Fluid therapy and some medications (e.g., furosemide, α-receptor agonists, steroids) may render the urine specific gravity value inconclusive.
- Diurethionated horses with primary renal disease usually lose the ability to concentrate urine—specific gravity and osmolality are <1.020 and <500 mOsm/kg, respectively.
- Urine specific gravity does not differentiate postrenal, prerenal or primary renal azotemia.

IMAGING
Radiography
- Rarely used to evaluate the urinary tract in adult horses but is useful in foals and miniature horses

Ultrasonography
- The urinary tract can be examined either transrectally or transabdominally.
- Bladder ultrasonography is best performed transrectally using a 3-MHz probe.
- Transrectal or transabdominal ultrasonography of the right or left kidney is best performed with a 2.5- or 3.5-MHz probe.
- Note the size and shape of both kidneys and the architecture and echogenicity of the parenchyma.
The renal medulla is more echolucent 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 calcific formation.

Renal Scintigraphy
May be used to document renal function but commonly is not performed.

OTHER DIAGNOSTIC PROCEDURES

Urine GGT/Cre Ratio
• Reflects GGT leakage from damaged renal tubular epithelium containing GGT compared to the constant excretion of Cre.
• Calculated as (Urine GGT/Urinary Cre) x 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 (Urine [electrolyte]) x Serum GFR)/(Serum [electrolyte] x Urine GFR).
• Reported reference intervals for sodium fractional excretion range from 0.01–0.70 in healthy horses.
• Post indicator of renal function.

Rectal Examination
• Bladder—determine size, wall thickness, and presence of calculus 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 obstructed urination, especially in geldings and stallions.
• In adult male horses, a flexible endoscope with an outside diameter of ≤12 mm and a length of ≥3 m is adequate to evaluate the urethra and urinary bladder.
• Normal urethral mucosa is pale pink, with longitudinal folds.
• If the urethra is dilated with air (e.g., to aid passage of the endoscope), the mucosa may appear reddened, and a prominent vascular pattern may appear.
• The ischial arch and colliculus seminalis are the most common sites of postinflammatory or postobstructive hemorrhage in geldings and stallions.
• In the dorsal aspect of the trigone, the urethral openings can be visualized to determine the source of hematuria or pyuria.

Pharmacology

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. Although they can limit intrarenal inflammation, they also nonspecifically block vasodilatory mediators of renal blood flow under conditions of renal hypoperfusion and are not recommended for chronic renal failure.
• Use caution with drugs requiring renal excretion. Horses should be well hydrated when using aminoglycosides and NSAIDs.
• Be aware of adverse reactions and toxic effects that may require altering dosage schedules.

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 diuresis 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 can occur during the first 6 hr without untoward effects, except in patients with hyperproteinemia/ hypalbuminemia and with signs of cardiac disease.

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