10. The answer is 1 [II D]. Mastitis is a relatively infrequent occurrence in mares. When it does occur, it produces glandular swelling and a thin, discolored discharge. Frequent stripping and systemic or intramammary therapy is recommended. Streptococcus equi causes "strangles," whereas S. zooepidemicus is a common isolate in cases of mare mastitis.

11. The answer is 2 [III C]. Pasteurella multocida is most often associated with pneumonia but may act as a cause of mastitis when transmitted from lambs to ewes through teat injuries from sucking. It is less common as a cause of mastitis than Staphylococcus aureus but produces an acute gangrenous mastitis.

*Mycoplasma* organisms produce the mastitis known as contagious agalactiae.

12. The answer is 4 [IV C 21]. The causative agents are most commonly coliforms, which produce signs of endotoxemia similar to that in dairy cattle. Mastitis is a clinically important disease in terms of the health of the sow and her piglets, who do not receive adequate nutrition during the mastitis episode. This disease most frequently occurs during early lactation and requires systemic therapy with antibiotics, corticosteroids, and oxytocin. Although metritis was once thought to be part of the mastitis complex, it is no longer considered to be involved.

Chapter 18

Neonatal Conditions, with Emphasis on the Equine Neonate

Jeanne Lofstedt

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**Introduction**

A. Goals. Until recently, the focus of equine neonatology was on saving the critically ill foal. The emphasis has now shifted to evaluation of foetaloplacental well-being during late gestation, with the goal being early identification and appropriate intervention in the case of an abnormal pregnancy or periparturient event. Owners, farm managers, and veterinarians should be cognizant of the findings that may indicate that the neonatal foal is at risk for future problems. Early recognition and aggressive treatment of such foals generally improves their prognosis for survival and future athletic performance while at the same time decreasing overall treatment costs. If possible, high-risk mares should receive late-term fetal monitoring and be assured of an attended delivery with adequate resuscitation and stabilization of the foal at birth.

B. Recognition of high-risk pregnancies. Maternal conditions associated with a high-risk pregnancy are presented in Table 18-2. Mares experiencing problem pregnancies can usually be assigned to one of three categories:

1. Mares with histories of abnormal pregnancies, deliveries, or newborn foals
2. Mares at risk with the current pregnancy as the result of a systemic illness or a reproductive abnormality
3. Mares with no apparent risk factors that experience an abnormal periparturient event

C. Outcomes for foals admitted to neonatal intensive care units

1. Short-term outcomes have improved dramatically in the last two decades. Before 1980, fewer than 25% of foals presented to referral institutions for treatment were discharged alive. Foal units today quote overall survival rates of greater than 70% for sick neonatal foals.
   a. Diseases associated with particularly favorable short-term survival rates include hypoxemic-ischemic encephalopathy [HIE, neonatal maladjustment syndrome (NMS)], uroperitoneum, infectious and noninfectious diarrhea, and noninfectious musculoskeletal diseases (e.g., angular limb deformities, flexural deformities).
   b. Diseases associated with poor short-term survival rates include established sepsis, sepsis accompanied by bacterial meningitis, septic arthritis, and septic osteomyelitis.

2. Long-term outcome. One of the major problems in conducting long-term follow-up is finding an appropriate population for statistical comparison. Most often, comparisons are made with half or full siblings, or to the patient population at a referral hospital; however, neither of these populations are representative of the general population.
   a. Most of the common foal diseases seem to have little impact on the foal's ability to perform as an adult. Examples of diseases that appear to have little impact on future foal performance are HIE, severe bacterial lung disease, infectious and noninfectious diarrhea, uncomplicated umbilical diseases (e.g., omphalitis, patent urachus) and uroperitoneum.
   b. There are three disease categories that negatively impact the long-term performance of surviving foals:
      1. Noninfectious orthopedic diseases
TABLE 18-1. Abnormalities Warranting Closer Evaluation of the Neonate

<table>
<thead>
<tr>
<th>Abnormalities of Labor and Delivery</th>
<th>Abnormalities in the Neonate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Premature parturition or abnormally long gestation</td>
<td>Meconium-stained neonate</td>
</tr>
<tr>
<td>Prolonged labor</td>
<td>Twins</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>Orphaned foal</td>
</tr>
<tr>
<td>Induced labor</td>
<td>Dysmaturity or prematurity</td>
</tr>
<tr>
<td>Cesarean section</td>
<td>Delay or failure of colostrum ingestion</td>
</tr>
<tr>
<td>Premature placental separation</td>
<td>Trauma (birth, dam, predators)</td>
</tr>
<tr>
<td>Umbilical cord abnormality</td>
<td>Adverse environmental conditions</td>
</tr>
<tr>
<td>Placental abnormality (placentitis, edema, villous atrophy)</td>
<td>Congenital anomalies</td>
</tr>
<tr>
<td></td>
<td>Weakness</td>
</tr>
<tr>
<td></td>
<td>Failure to stand and nurse within 2-3 hours</td>
</tr>
</tbody>
</table>

(2) Prematurity characterized by small stature and noninfectious orthopedic disorders (e.g., angular limb deformities, tarsal bone collapse)

(3) Septic arthritis and osteomyelitis

II. PREMATURITY OR DYSMATURITY

A. Introduction

1. Terminology. The literature abounds with terms for the neonatal foal with physical characteristics of immaturity: premature, immature, dysmature, intrauterine growth-retarded (IUGR), small for gestational age (SGA), ready or unready for birth, and viable or nonviable.

2. Criteria

a. Gestational age. Although most authors define a foal born before 320 days' gestation as premature (the mean gestation length of a Thoroughbred is 340 days), gestational age is only one of many criteria used to assess readiness for birth. Because gestation of mares is extremely variable, a 335-day fetus may be completely unprepared for birth if its normal gestational length was intended to be 365 days.

b. Endocrine maturation

(1) Normal physiology. The fetal pituitary—adrenal axis controls the final maturation of various organ systems, including the pulmonary system.

(a) Cortisol. The fetal adrenal gland is poorly responsive to adrenocorticotropin (ACTH) throughout most of pregnancy, but in the last 3–5 days of gestation, its sensitivity changes, resulting in a significant increase in fetal cortisol 24–48 hours before birth. This cortisol surge causes maturation of the hematopoietic system, resulting in a total white blood cell (WBC) count of more than 5000 cells/µL in the healthy term foal immediately after birth.

(b) Thyroid hormone. The triiodothyronine (T3) concentration in the healthy term foal is 10–20 times that of an adult horse. Thyroid hormone in the neonatal foal is required for thermogenesis, skeletal maturation, and may also synergistically with cortisol to cause normal lung maturation.

(2) "Unready for birth" foals. Foals removed suddenly from the uterus before final endocrine maturation has taken place (e.g., by a poorly timed cesarean section) have low serum concentrations of cortisol and thyroid hormone and, when challenged with exogenous ACTH, do not respond with appropriate increases of cortisol. They have incomplete body system maturation and generally adjust poorly to extrauterine life.

(3) "Immature but ready for birth" foals. Maturation is often hastened in foals exposed to chronic utero stress (e.g., chronic placental insufficiency) during gestation. Despite having characteristics of physical immaturity, foals stressed in utero have adequate pulmonary and hematologic function and cope well with extrauterine life.

B. Clinical finding are summarized in Table 18-3.

C. Complications

1. Respiratory difficulties are attributed to a number of factors, including dependent lung atelectasis caused by lung and chest wall immaturity, in utero–acquired pneumonia, and lack of mature surfactant.

TABLE 18-2. Maternal Conditions Associated With a High-Risk Pregnancy

<table>
<thead>
<tr>
<th>Past History</th>
<th>Systemic Diseases</th>
<th>Reproductive Problems</th>
</tr>
</thead>
<tbody>
<tr>
<td>Foals with N1, NMS, or congenital problems</td>
<td>Severe malnutrition</td>
<td>Severe endometrial fibrosis</td>
</tr>
<tr>
<td>Premature, septic, or hypoxic foals</td>
<td>Fever, endotoxemia, severe systemic infection</td>
<td>Hydrops allantois or hydrands amnii</td>
</tr>
<tr>
<td>Dyspnea or premature placental separation</td>
<td>Severe anemia or hypoproteinemia</td>
<td>Purulent vaginal discharge</td>
</tr>
<tr>
<td>Foal rejection</td>
<td>Gastrointestinal crises</td>
<td>Prepubertal tendon rupture or abdominal hernia</td>
</tr>
<tr>
<td>Exposed to infectious diseases known to cause Chêvre abortion (e.g., EHV-1, EVA)</td>
<td>Laminitis</td>
<td>Poor colostral quality or premature lactation</td>
</tr>
<tr>
<td></td>
<td>Musculoskeletal or CNS problems causing prolonged recumbency</td>
<td>Pelvic injuries</td>
</tr>
</tbody>
</table>

CNS = central nervous system; EHV-1 = equine herpesvirus-1; EVA = equine viral arteritis; N1 = neonatal isoerythrolysis; NMS = neonatal maladaptation syndrome.

TABLE 18-3. Clinical Findings Suggestive of Immaturity

- Gestation length less than 320 days
- Weak suckle reflex, failure to nurse within 3 hours of birth
- Failure to stand within 2 hours of birth
- Low birth weight (less than 45 kg in Thoroughbreds or less than 10% of the dam's weight in other breeds)
- Short, silky haircoat
- Pliant ears and soft lips
- Bloating, prominent forehead and eyes (especially in equine twins and other growth-retarded newborn foals)
- Generalized weakness ("floppiness")
- Increased passive range of motion of limbs
- Marked flexor tendon laxity in the rear limbs
- Hypothermia and difficulty thermoregulating
- Intolerance to enteral feeding (abdominal pain)
- Progressive tachypnea after birth with evidence of respiratory distress
- Poor ossification of cuboidal bones in the carpi and tarsi
Therapeutic plan. Frequent and comprehensive clinical and laboratory evaluation of respiratory function, and renal function of the immature neonatal foal. General supportive care is necessary.

1. Prevention of decubital ulcers and scalding
   - An air mattress or cushions help prevent decubital ulcers. A foal that is unable to maintain sternal recumbency or stand should have an attendant present to keep it in sternal recumbency, turn it from side-to-side every 2 hours, and assist in standing as needed.
   - Scalding by feces and urine can be minimized by covering the bedding with absorbent pads.

2. Prevention of nosocomial infection. Foals should be separated from the adult horse population and high-traffic areas, and strict hygiene and cleanliness should be maintained in the foal ward to decrease the risk of nosocomial infections.

3. Prevention of hypothermia. The ambient temperature can be increased using radiant heat lamps, hot water pads, blankets, and warmed intravenous fluids.

4. Nutritional support. Premature or dysmature foals are often unable to suckle from the mare and have to be fed via a bottle, bucket, or nasogastric tube.
   - Frequency and amount. It is important to remember that the healthy foal nurses body weight in milk, divided into at least 12 feedings over 24 hours; if the foal tolerates this amount, the volume fed can be increased gradually until the foal is grown to adulthood.
   - Methods:
     1. Bottle feeding. A lamb nipple can be used to bottle feed a foal. A foal with a cuboidal bone ossification center to the carpal and tarsal bones occurs in the last 2-3 weeks of gestation, the premature or dysmature foal that ambulates may experience collapse of these bones, which may in turn result in angular limb deformities.
     2. Prevention of nosocomial infection. Foals should be separated from the adult horse population and high-traffic areas, and strict hygiene and cleanliness should be maintained in the foal ward to decrease the risk of nosocomial infections.
     3. Prevention of hypothermia. The ambient temperature can be increased using radiant heat lamps, hot water pads, blankets, and warmed intravenous fluids.
     4. Nutritional support. Premature or dysmature foals are often unable to suckle from the mare and have to be fed via a bottle, bucket, or nasogastric tube.
        - Frequency and amount. It is important to remember that the healthy foal nurses body weight in milk, divided into at least 12 feedings over 24 hours; if the foal tolerates this amount, the volume fed can be increased gradually until the foal is grown to adulthood.
        - Methods:
          1. Bottle feeding. A lamb nipple can be used to bottle feed a foal. A foal with a weak suckle reflex should never be bottle fed because aspiration pneumonia and malnutrition may ensue. Mare's milk is the optimal milk of choice. There are a number of commercially available mare milk replacers and many foals appear to thrive on goat milk. Hypoglycemic foals (i.e., those with a glucose concentration of less than 40 mg/dl) should receive intravenous dextrose (5%–10%) in addition to oral alimentation.
          2. Nasogastric intubation. A small-diameter nasogastric tube is placed in the distal esophagus. The foal can nurse from the mare while the tube is in place.
          3. Bucket feeding. Foals are easily taught to drink milk from a shallow pan or bucket. The foal should be encouraged to suckle a clean finger which is then gradually lowered into the bucket containing the milk.

4. Parenteral nutrition. Essentially, parenteral nutrition involves constant intravenous infusion of a hypertonic solution containing various concentrations of dextrose, amino acids, lipids, electrolytes, and vitamins.
   - Parenteral solutions should be mixed under a hood wearing sterile gloves and a mask to prevent contamination. They are generally administered via a nonthrombogenic jugular catheter and a dedicated line. Fluid administration rates are carefully controlled and fluid lines and hanging bags are changed daily.
   - Patients receiving parenteral nutrition should continue to receive small-volume enteral feeding if at all possible.
   - The patient should be monitored frequently to detect complications. Potential complications include hyperglycemia, hyperlipidemia, metabolic acidosis, hypokalemia, and infection at the catheter site.

5. Immunologic support. Failure of passive transfer (FTP) of colostral immunoglobulins (see IV) is a common occurrence in the premature or dysmature foal. Good-quality colostrum should be fed within 6 hours of birth, and passive transfer status should be assessed when the foal is 18–24 hours old. Plasma should be transfused if the immunoglobulin G (IgG) concentration is less than 800 mg/dl.

6. Antimicrobial treatment. Many premature births are associated with infections acquired in utero. Broad-spectrum bacterioidal antimicrobials should be used prophylactically in all premature foals (see V E 2).

7. Cardiovascular support
   - a. Dehydration and hyperconcentration. Foals should be treated intravenously with an isotonic balanced electrolyte solution (e.g., lactated Ringer's solution, 40–60 ml/kg/day).
   - b. Acidemia is defined as a blood pH of less than 7.3. Foals with acidemia should be adequately ventilated and receive 1.3% (isotonic) sodium bicarbonate intravenously.

8. Respiratory support. Most immature foals have some pulmonary dysfunction.
   - a. Atelectasis (as a result of lung and chest wall immaturity) can often be managed by simply maintaining the foal in sternal recumbency, turning it frequently, and encouraging lung expansion through stimulating deep breathing or coughing.
   - b. Hypoxia is defined as an arterial oxygen tension (Pao2) of less than 70 mm Hg with a normal arterial carbon dioxide tension (Paco2). Hypoxic foals often benefit from humidified oxygen administered via face mask or nasal insufflation at a rate of 5–10 L/min.
   - c. Hypercapnia is defined as a Paco2 of less than 65 mm Hg and a Paco2 of greater than 65 mm Hg. Hypercapnic foals benefit from assisted or controlled ventilation via orotracheal or nasotracheal intubation.
   - d. Lack of surfactant. Surfactant replacement has been utilized in an attempt to increase survivability of the premature foal with immature lung surfactant. There are anecdotal reports that this treatment modality is beneficial, but large-scale controlled studies have not been undertaken.

9. Musculoskeletal support
   - a. Foals with radiographic evidence of incomplete carpal and tarsal bone ossification should be housed in a small stall to restrict exercise.
   - b. Excessive flexion tendon laxity can be managed with physical therapy and the application of heel extensions to the bottom of the foot. Support wraps should be light and used sparingly because they may exacerbate tendon laxity.

10. Treatment of other complications
    - a. Corneal ulcer. Affected foals should be treated with topical antibiotics, mydriatics, and vertical mattress sutures to evert the eyelid.
    - b. Patent urachus. Treatment includes chemical cautery or surgical excision (see VI B 4).
    - c. Adrenal insufficiency. Treatment is controversial and reserved for those foals with laboratory evidence of adrenocortical insufficiency.
1. Periparturient history
   a. Foals delivered early and abruptly by induction of parturition, cesarean section, or because of severe systemic maternal illness in the mare generally have a poor prognosis. They do not experience the endocrinologic events required for normal maturation, are "unready for birth," and the prognosis for survival is poor (survival rate of 20%–25%).
   b. Foals delivered naturally, but prematurely, by a healthy mare usually have a fair prognosis. With appropriate supportive care, survival rates as high as 70% are possible. Close inspection of the placenta from these deliveries may reveal villous atrophy or placental infarction. Placental pathology is thought to interfere with uteroplacental blood flow, imposing chronic hypoxia on the foal and decreasing fetal lipid–adrenal axis and "readiness for birth."

2. Laboratory testing can be used to assess readiness for birth.
   a. Assessment of electrolyte concentrations in prepartum mammary secretions. The electrolyte concentrations in prepartum mammary secretions change dramatically near term. Calcium and potassium concentrations in mammary secretions in late gestation are typically less than 10 mg/dl and 30 mEq/L, respectively, whereas the sodium concentration decreases to less than 30 mEq/L.
   b. Complete blood count (CBC). Foals with a total WBC count of greater than 5000 cells/µL on day 1 of life (or a low WBC count on day 1 that increases to greater than 5000 cells/µL on day 2 or 3) generally have a favorable prognosis.
   c. Persistent leukopenia and neutropenia usually indicate that the foal is endocrinologically immature and will be a nonsurvivor in the postnatal period.
   d. Fibrinogen concentration. A plasma fibrinogen concentration that exceeds 400 mg/dl at birth is generally associated with a favorable prognosis. This suggests that an in utero infection caused the premature delivery. Foals stressed in utero generally adjust better in the early neonatal period.
   e. Blood gas analysis. Nonviable premature foals usually have a metabolic acidosis with a blood pH persistently less than 7.3. (2) ACTH response test. Foals that are endocrinologically mature respond with a two-fold increase in plasma cortisol and a widening of the neutrophil–lymphocyte ratio in response to the administration of short-acting exogenous ACTH (0.125 mg, intramuscularly).

3. Careful assessment of clinical progression over the first 2 days of the immature foal's life can also be used to formulate a prognosis:
   a. In "unready for birth" foals, the first 12–18 hours after resuscitation are deceivingly uneventful. However, progressive deterioration in neurologic function and an inability to maintain homeostasis soon develop. Death certainly awaits these foals.
   b. Early recognition of abnormalities such as hypothermia, hypoglycemia, and metabolic acidosis can increase survival. Other abnormalities that may indicate a poor prognosis include persistent neonatal maladjustment syndrome (NMS), gastrointestinal enteritis leading to respiratory acidosis, and hypoxic–ischemic brain damage.

4. Other factors influencing outcome include the actual birth weight of the foal (generally, the lower the birth weight, the poorer the prognosis), the presence of other complicating factors in the perinatal period (e.g., in utero–acquired infection or peripartum asphyxia), and the resources available for treatment.
Etiology and pathophysiology

1. Etiology. A variety of fetal and maternal conditions are associated with perinatal asphyxia.
   a. Fetal factors include twinning, meconium aspiration, sepsis, prematurity or dysmaturity, and severe anemia.
   b. Maternal factors include conditions that cause hypotension or impaired tissue oxygenation, such as endotoxemia, anemia, hemorrhagic shock, maternal surgery or cesarean section, and placentation abnormalities (e.g., those caused by ingestion of endophyte-infested fescue during pregnancy, placental infection, or premature placental separation).

2. Pathophysiology
   a. Shunting. Initially, the fetus responds to asphyxia by shunting blood away from nonvital organs (e.g., the gut, kidneys, bone, muscle, and skin) to vital organs (e.g., the brain, heart, and adrenal gland).
   (1) Mild asphyxia causes a mild decrease in heart rate and a slight increase in blood pressure, but little change in cardiac output.
   (2) With severe asphyxia, the heart rate, cardiac output, and blood pressure decrease as oxidative phosphorylation fails and energy reserves in cardiac muscle are depleted.
   b. Metabolic derangements. Without sufficient energy, cellular ion pumps in various body tissues eventually fail, resulting in intracellular accumulation of sodium, chloride, water, and calcium and extracellular accumulation of excitatory neurotransmitters in the brain (e.g., glutamate).
   (1) Glutamate accumulation in the brain after an ischemic event apparently causes excessive stimulation of cell surface receptors, eventually resulting in neuronal death.
   (2) Intracellular free calcium accumulation causes cell death in several ways, including activation of enzyme systems that attack the structural integrity of the cell and impairment of mitochondrial function.
   (3) Oxygen free radicals are generated during the reperfusion phase that follows a hypoxic–ischemic insult. Oxygen free radicals cause membrane fragmentation by attacking the lipids in cell membranes and are in part responsible for increased capillary permeability, edema formation, and tissue damage that occur following restoration of blood flow to previously ischemic tissues.
   c. Sequelae
   (1) Neurologic effects. In the brain, asphyxia can lead to HIE accompanied by edema, necrosis, and hemorrhage.
   (2) Cardiac effects. The effects of perinatal asphyxia on myocardial function can be profound. Decreased myocardial contractility and congestive heart failure (CHF) are common sequelae and may be associated with infarcts in myocardial and papillary muscles. The systemic hypotension caused by these lesions further contributes to tissue hypoxia, development of metabolic acidosis, and decreased renal perfusion in the asphyxiated neonate.

(3) Pulmonary effects. The neonatal pulmonary system responds to hypoxemia and acidosis by reflex vasoconstriction, which, in turn, causes an increase in pulmonary vascular resistance, pulmonary hypertension, and increased right atrial pressure.
   a. If pulmonary arterial pressure exceeds systemic pressure, right-to-left flow through the ductus arteriosus and foramen ovale may result in reestablishment of persistent fetal circulation characterized by severe hypoxemia unresponsive to oxygen therapy.
   b. Decreased pulmonary blood flow may cause decreased delivery of lipid precursors to the lung, resulting in decreased surfactant production.
   c. Meconium aspiration, a common sequela of birth asphyxia, usually initiates a chemical pneumonitis that can further compromise pulmonary function.
   d. Perinatal asphyxia may decrease the responsiveness of respiratory centers in the brain, causing periods of apnea or abnormal breathing.
   e. Renal effects. Redistribution of blood flow away from the kidneys causes decreased renal perfusion and, frequently, acute renal tubular necrosis and oliguria.
   f. Gastrointestinal effects. Reduced intestinal blood flow during an asphyxial episode causes variable degrees of bowel ischemia.
   (a) Mild signs of gastrointestinal dysfunction are commonly exhibited by asphyxiated foals, including meconium impactions and intolerance to enteral feeding manifested as abdominal distention, colic, diarrhea, or delayed gastric emptying.
   (b) Severe ischemia causes total loss of bowel integrity, resulting in overwhelming bacteremia and septic shock.
   (6) Other effects. Severe asphyxia may lead to anoxic liver damage, necrosis and dysfunction of endocrine organs, and coagulopathy. To date, these disorders have not been described in asphyxiated equine neonates.

D. Diagnostic plan

1. Physical examination. An in-depth physical examination is indicated for all recumbent foals to:
   a. Provide baseline information against which foal progress can be compared
   b. Aid in the identification of subtle but important abnormalities that may be overshadowed by grossly obvious clinical signs, such as seizures
   2. Baseline clinicopathologic information should be gathered. A CBC and blood culture should be obtained, and passive transfer status should be assessed. A sepsis score (see V D 3) should be calculated.
   3. Specific assessments
   a. Neurologic assessment. Neurologic signs caused by HIE need to be distinguished from those caused by meningitis, hypoglycemia, or congenital anomalies.
   (1) Cerebrospinal fluid analysis allows differentiation of meningitis and HIE (meningitis is characterized by an increased leukocyte count and protein concentration).
   (2) Imaging techniques, such as computed tomography (CT) and magnetic resonance imaging (MRI), are being used with increasing frequency to document location, severity, and progression of brain injury in asphyxiated large animal neonates.
   b. Respiratory assessment
   (1) Arterial blood gas analysis usually reveals hypoxemia, hypercarbia, and acidemia. These are typical findings in a foal with hypoventilation; affected foals usually respond dramatically to nasal oxygen insufflation. In contrast, foals with persistent fetal circulation do not respond to inspired oxygen.
Thoracic radiography is always indicated for foals with birth asphyxia.

(a) In cases of pulmonary hypertension, thoracic radiographs may reveal pulmonary hypertension/clear fields with decreased pulmonary vascularity/marking.

(b) Suggestive deficiency results in diffuse lung atelectasis and a diffuse reticular and/or linear radiographic appearance with bronchograms.

(c) Meconium aspiration results in patchy perihilar infiltrates, focal areas of atelectasis, and hyperaeration.

c. Cardiovascular assessment

(1) Electrocardiography and echocardiography may be indicated in foals with murmurs or cardiac dysrhythmias. Electrocardiography may reveal persistent fetal circulation.

(2) Determination of cardiac isoenzyme activities is necessary to detect myocardial necrosis.

d. Renal assessment. Renal function is assessed by measuring urine output, performing a urinalysis, and assessing the results of a biochemical profile. Typical findings include oliguria (a urine output of less than 2 ml/kg/hr), azotemia, and electrolyte disturbances such as hypocalcemia, hypernatremia, hyperglycemia, and hypokalemia.

e. Gastrointestinal assessment. Gastrointestinal dysfunction is primarily diagnosed based on clinical signs. The most severe form of gastrointestinal dysfunction associated with birth asphyxia, necrotizing enterocolitis (NEC), may be diagnosed radiographically based on the presence of submucosal gas accumulation in the bowel wall (pneumatosis intestinalis).

D. Differential diagnoses. Conditions such as neonatal sepsis, meconium aspiration, prematurity, and hypoglycemia can mimic perinatal asphyxia. Perinatal asphyxia can also be a complicating factor in many of these conditions.

E. Therapeutic plan. Therapeutic goals are numerous and address the multiple organ failure that is present.

1. Treatment of CNS dysfunction includes seizure control, nursing care to prevent injury, fluid therapy, nutritional support, and control of cerebral edema.

   a. Seizure control

      (1) Diazepam (0.1-0.44 mg/kg intravenously) has a rapid onset of action and is used for initial seizure control.

      (2) Pentobarbital (0.1-0.8 mg/kg intravenously every 12 hours) can be used to manage severe or repeated seizures. Serum concentrations of pentobarbital should be monitored.

   b. Xyloclaine and acepromazine should not be used to control seizures. Xyloclaine causes transient hypertension and may exacerbate cerebral hemorrhage, and acepromazine lowers the seizure threshold.

   c. Control of cerebral edema. Cerebral edema occurs in some asphyxiated foals and is treated with dimethylsulfoxide (DMSO; 0.5-1.0 g/kg administered over 1-2 hours as a 10%-25% solution) and/or mannitol, an osmotic diuretic (0.25-1.0 g/kg given as a 20% solution slowly over 1-2 hours). Mannitol may exacerbate CNS herniation; therefore, it should be used with extreme caution in asphyxiated neonates.

   d. Prevention of trauma. Leg wraps, a padded helmet, and a padded stall may be required to protect the asphyxiated neonate from self trauma.

2. Treatment of respiratory dysfunction includes maintenance of oxygenation and ventilation of the patient.

   a. Treatment of hypoxia. Hypoxia is usually corrected by administering humidified oxygen (3-10 L/min) via nasal insufflation and keeping the foal in a stall recumbency.

   b. Treatment of hypercapnia. Respiratory center depression can cause hypercapnia, characterized by a PaO2 of less than 65 mm Hg and a PaCO2 greater than 65 mm Hg.

   c. Treatment of cerebral edema and gas accumulation. Severe, persistent large bowel distention may respond to decompression. If the foal has delayed gastric emptying and decompression can be used to relieve fluid and gas accumulation, severe, persistent large bowel distention may require percutaneous trocarization using sterile technique and a 15- or 16-gauge catheter.

3. Treatment of cardiac dysfunction revolves around judicious use of intravenous fluids and the administration of inotropes.

   a. Dopamine or dobutamine (infused at a rate of 2-10 mcg/kg/min) may be used to increase cardiac output and improve tissue perfusion.

   b. Diuretics (e.g., furosemide) may be used to treat the edema associated with cardiac failure.

   c. Diazepam (0.02-0.035 mg/kg orally every 24 hours) should be used if there is evidence of cardiac failure.

4. Treatment of renal dysfunction involves judicious use of intravenous fluids, diuretics, and administration of low doses of dopamine to stimulate dopaminergic receptors. Fluid input and urine output should be carefully monitored to avoid overhydration.

   a. Dopamine infusions (2-4 mcg/kg/min) have been advocated to improve renal blood flow and urine output.

   b. Diuretics. Seum electrolyte concentrations should be carefully monitored during diuretic therapy.

      (1) Furosemide infusions of 0.25-2.0 mcg/kg/min have been used successfully to treat oliguric renal failure in asphyxiated foals. Furosemide acts synergistically with dopamine to produce renal vasodilation and diuresis.

      (2) Mannitol therapy may be added if oliguria persists after dopamine and furosemide administration.

   c. Dobutamine therapy should be instituted if cardiac dysfunction appears to be caused by systemic hypotension and renal hypoperfusion.

5. Treatment of gastrointestinal dysfunction. Gastrointestinal dysfunction is treated with decompression, the use of prokinetic agents, or both.

   a. Nasogastric decompression. If the foal has delayed gastric emptying and decreased intestinal motility, nasogastric decompression can be used to relieve fluid and gas accumulation. Severe, persistent large bowel distention may respond to percutaneous trocarization using sterile technique and a 15- or 18-gauge catheter.

   b. Prokinetic agents. Such as metoclopramide (0.25-0.3 mg/kg intravenously) or ondansetron (5-10 mg/kg orally or by intravenous infusion) may improve gastric emptying and small intestinal motility.

6. General supportive care

   a. Fluid therapy. Polyionic isotonic fluids should be used to correct dehydration and expand blood volume. To avoid overhydration, asphyxiated foals should be carefully monitored using clinical assessment of hydration and monitoring of body weight changes and urine output. Normal urine output is 6-7 ml/kg/hr; measures should be introduced to improve urine output if it decreases to less than 2 ml/kg/hr.

      (1) Patients with metabolic acidosis should receive supplemental bicarbonate based on results of blood gas analysis. Caution should be exercised when bicarbonate is given to foals with severe respiratory compromise, because in
these foals, bicarbonate can worsen the acidosis because of carbon dioxide retention.

(2) Specific electrolyte-abnormalities should also be corrected with fluid therapy.

b. Immunologic support. The plasma immunoglobulin concentration should be measured and colostrum or plasma administered if the foal's serum IgG levels are less than 800 mg/dl.

c. Nutritional support

(1) Nasogastric tube. Foals that are unable to nurse from the mare or a bottle should be tube fed using the recommendations given in I D 4. (2) Parenteral nutrition. If gut function is questionable, parenteral nutrition should be employed. To minimize the risk of NEC, foals that have suffered severe asphyxia complicated by hypotension or hypothermia should not receive enteral feeds until their vital signs are stable.

d. Enteral feeding should be initiated gradually and mare's colostrum or milk should be fed preferentially.

e. Antibiotic therapy. Broad-spectrum bactericidal antimicrobial therapy should be given to all asphyxiated foals because gastric ulceration is a common complication.

G. Prognosis

1. The prognosis is usually good for a term foal delivered without obvious complications, particularly if the foal was able to stand for period of time after delivery and had a normal immunoglobulin concentration at 18–24 hours of age. Approximately 75% of foals with good prognostic signs recover with intensive nursing care. In survivors, the clinical signs usually stabilize at 48–72 hours and their condition is significantly improved by 72–96 hours of age. Full recovery can take as long as 2 weeks.

2. The following findings are associated with a poor prognosis:

a. Concurrent septicemia

b. Failure to show any improvement in neurologic function by 5 days of life

c. Failure of the neonate to absorb adequate amounts of colostral immunoglobins and other macromolecules (i.e., lactoferrin, transferrin, lysozyme, and complement).

d. Antibiotic therapy. Broad-spectrum bactericidal antimicrobial therapy should be given to all asphyxiated foals because gastric ulceration is a common complication.

Failure of Passive Transfer (FPT)

Introduction

1. Incidence. FPT (i.e., inadequate transfer of colostral immunoglobulin) is widespread in both equine and bovine neonates, with reported prevalences of 2.9%–25% and 15%–68%, respectively. FPT also occurs in lambs, goat kids, and piglets, but the exact prevalences are unknown.

2. Neonatal immunity, colostral production, and absorption of immunoglobulin. Neonates are capable of mounting an immune response at birth, but it is a primary response characterized by a prolonged lag period and production of low concentrations of antibody. Leukocytes of newborn animals exhibit reduced phagocytic and bactericidal activity as a result of fetal glucocorticoid production shortly before birth. Furthermore, the neonate's serum is deficient in some complement components, resulting in poor opsonic activity. Placental transfer of immunoglobulin does not occur to any extent in neonatal ungulates; therefore, they rely on colostral transfer of immunoglobulin to protect them against infectious disease early in the neonate period. Colostrum is composed of immunoglobulins, immunologically active cellular components (lymphocytes, macrophages, polymorphonuclear cells) and nonspecific immune factors (actetolyn, lysozyme, and complement).

IV. Etiology

1. FPT in foals

a. Poor colostral quality or quantity

(1) Maternal age. The foals of dams older than 15 years are at increased risk for FPT as a result of poor colostral quality.

(2) Leakage of colostrum (premature lactation) results in decreased colostral IgG concentration.
2. FPT in calves

a. Poor colostral quality or quantity

(1) First milking. It is generally accepted that heifers in their first lactation produce less total colostrum containing less total immunoglobulin than cows in later lactations.

(2) Holstein breed. Colostral quality may be affected by breed. Beef cattle generally produce colostrum with a high immunoglobulin concentration and Jersey cows have excellent colostral quality. In contrast, Holstein cows have notoriously poor colostral quality.

(3) Colostral volume greater than 85 kg at first milking. In general, the larger the volume of colostrum produced, the lower the immunoglobulin concentration. If the first-milking colostra of dairy cows that produce more than 8.5 kg of colostrum is discarded, more than 77% of the remaining colostra will have sufficient IgG to ensure adequate passive transfer (if sufficient volumes are fed in a timely fashion).

b. Premature calving or induction of parturition. Because colostrum secretion only occurs in the last month of gestation under hormonal influence, premature parturition or a shortened dry period fail to allow for optimum colostrum production. Premature induction of parturition may also decrease colostral quality. Prostaglandin administration reduces the IgG concentration, and glucocorticoids apparently decrease the volume of colostrum that is produced.

(5) Premature feeding. If colostrum from the mammary gland is delayed after calving, the colostrum will be diluted by secretion of milk into the mammary gland and have a low immunoglobulin concentration.

(6) Delay in obtaining first milking. If milking of colostrum from the mammary gland is delayed after calving, the colostrum will be diluted by secretion of milk into the mammary gland and have a low immunoglobulin concentration.

(7) Colostral handling (pooling, repeated freezing and thawing).

(i) Colostrum pools created by mixing colostrum from different dams generally have lower immunoglobulin concentration than fresh colostrum and calves fed from these pools frequently do not achieve satisfactory passive transfer. There are several explanations for this:

(2) Colostrum from cows producing large volumes of inferior quality colostrum are more likely to be added to the pool.

2. Timing

(a) Individual testing. Testing is most commonly performed when the calf or foal is 18-24 hours of age. If FPT is detected at this stage, it can only be rectified by the intravenous administration of plasma or whole blood.

(b) Early testing. If the foal suckled by its dam within 2 hours of age, passive transfer status can be assessed 8-12 hours after birth, prior to gut closure. If FPT is detected at this stage, an alternative source of colostrum can be administered in an attempt to achieve adequate passive transfer. Passive transfer status should be reassessed when the foal reaches 18-24 hours of age.
3. Laboratory tests used to screen for FPT
   a. Immunoglobulin concentration
      (1) Traditional testing
         (a) Calves. The IgG concentration that is generally considered protective in calves raised under average management conditions is 1000 mg/dL; therefore, FPT is diagnosed if the IgG concentration of a calf is less than 1000 mg/dL.
         (b) Foals. There are two cutoff points for FPT: one for foals that are sick at the time of testing and are being admitted to a referral center (less than 800 mg/dL), and one for foals that are healthy at the time of testing and are being screened on the farm (less than 400 mg/dL).
      (2) Early testing. In foals, an IgG concentration of less than 200 mg/dL at 8–12 hours of age is considered presumptive evidence of FPT.
   b. Single radial immunodiffusion (SRID) test. SRID represents the "gold standard" for assessing passive transfer in both horses and cattle and is the gold standard to which all other tests are compared. It is technically difficult to perform and results are not available for 24 hours, therefore, it is generally not used in the field.
   c. Total serum protein (TSP) or Total solids (TS) test. This test is a useful and inexpensive way of assessing passive transfer status in calves, but its utility has been questioned in foals. A TS concentration of less than 5.0 g/dL generally indicates FPT in the calf. TS concentration should be interpreted with caution in the dehydrated calf; a TS greater than 5.0 g/dL in a dehydrated animal may in fact indicate FPT.
   d. Globulin concentration from a biochemistry profile can serve as an indicator of immunoglobulin concentration. Approximately 1.5 g/dL of the globulin measurement represents the nonimmunoglobulin component and the remainder represents the immunoglobulin fraction. Therefore, in a calf with a globulin concentration of 1.8 g/dL, the immunoglobulin fraction is approximately 0.3 g/dL (1.8–1.5), or 300 mg/dL, and likely indicates FPT.
   e. Zinc sulfate turbidity (ZST) test. The ZST test is used in both cattle and horses. A commercial test kit is available for horses or the reagent can be prepared. Zinc ions in the zinc sulfate solution combine with immunoglobulins to form a precipitate in serum; the more the precipitate at a given concentration of zinc sulfate, the higher the immunoglobulin concentration.
   f. Sodium sulfite turbidity (SST) test. This test operates on the same principle as the ZST test, but is used primarily in calves.
   g. Glutaraldehyde coagulation (CCT) test. This test is used in both calves and foals and is based on the ability of glutaraldehyde to react with gamma globulin, forming a solid clot. Serum or whole blood may be used, depending on the test kit. Unfortunately, test sensitivity and specificity are low when this test is performed on whole blood in the bovine species, probably because fibrinogen and other clotting factors interfere with test accuracy. Therefore, the CCT cannot be endorsed as a screening test.
   h. Enzyme-linked immunosorbent assay (ELISA) is designed for the semiquantitative measurement of IgG in foal serum or plasma. The test uses a color spot with calibration standards corresponding to 200, 400, and 800 mg/dL of IgC. The assay takes 15 minutes to perform and results correspond well with those obtained with SRID.
   i. Latex agglutination test. A commercial latex agglutination test is available for horses. The degree of agglutination between IgC in serum or blood and latex beads coated with antibody to equine IgC is used to estimate the IgC concentration.
   j. Y-glutamyl transferase (GKT) activity is used as an indicator of passive transfer status in the calf. Colostromal GGT concentration in the bovine is about 300 times the concentration in colostrum. GGT from colostrum is absorbed along with other macromolecules during the period when the gut is open. GGT activity is at its peak when the calf is 24–48 hours of age; a CGT value of greater than 300 IU/L indicates that the calf has consumed colostrum.
   4. Assessing colostral quality. Colostrometers (modified hydrometers) are available to assess colostral quality in both horses and cattle. There is an excellent correlation between colostral specific gravity and colostral immunoglobulin concentration in horses, but the strength of this association in cattle is weak.
   a. Bovine colostrum. The literature and companies that manufacture colostrometers for commercial purposes state that bovine colostrum with a specific gravity greater than 1.050 is of satisfactory quality (i.e., the IgG concentrations should be greater than 50 mg/mL). Recently this criteria has been shown to be inaccurate; the colostrometer will classify nearly 75% of low-quality colostra as satisfactory using this standard.
   b. Equine colostrum with a specific gravity of 1.060 (using the equine colostrometer) is usually classified as satisfactory, whereas samples with a specific gravity of greater than 1.090 are ideal. It is important to recognize that milk has a specific gravity of 1.040.

D. Therapeutic plan

1. Diagnosis within 12 hours of birth. If FPT is diagnosed or suspected less than 12 hours after birth, oral supplementation of IgC is indicated.
   a. Foals. All foals receiving oral supplementation should be retested at 24 hours of age.

2. Diagnosis 18 or more hours after birth. If FPT is diagnosed or suspected 18 or more hours after birth, intravenous supplementation of IgC is indicated.
   a. Foals. All foals receiving oral supplementation should be retested at 24 hours after birth.

(1) Equine colostrum from a colostrum bank should be used. Banked colostrum should have a minimum specific gravity of 1.060 (and ideally, a specific gravity greater than 1.090) and the foal should receive 1–2 L in 500-mL increments administered 1 hour apart via a nasogastric feeding tube. Administering colostrum in this way should provide at least 1 g/kg of immunoglobulin to the foal.
   (2) Alternative sources of immunoglobulin include bovine colostrum, equine plasma, or serum, and commercially available lyophilized or concentrated IgG products.

(a) Bovine colostrum can be safely administered to foals; usually 4 L is administered in the first 24 hours of life. Bovine immunoglobulins have a very short half-life in the foal and may not protect the foal against pathogens unique to the equine environment (e.g., Actinobacillus equuli). However, most colostrum-deprived foals that receive bovine colostrum do not succumb to sepsis.

(b) Equine plasma and serum have very low immunoglobulin concentrations, so extremely large volumes are required to achieve adequate passive transfer.

(c) Commercial products. If the foal is treated with a concentrated commercial product, it should receive at least 1 g/kg of IgG (i.e., approximately 40 g for the average 40-kg foal).

b. Calves should be fed fresh or frozen first-milking colostrum from a third or greater lactation cow.

(1) Holstein calves. At least 28 L of colostrum should be administered via esophageal feeder in the first feeding.

(2) Other breeds. Calves should receive colostrum in amounts equal to 10% of their body weight in the first 24 hours, with at least 2 L being fed in the first 6 hours of life.

2. Diagnosis 18 or more hours after birth. If FPT is diagnosed 18 or more hours after birth, intravenous supplementation of IgC is indicated.
   a. Foals. Sources of immunoglobulin

(a) Commercial products. Advantages of commercial products are that they are free of alloantibodies and infectious agents and generally provide a known quantity of IgG. Some products originate from hones immunized with endotoxin or with specific pathogens (e.g., Rhodococcus equi). The major disadvantage of commercial products is that they may lack antibodies specific to pathogens in the foal’s environment.

(b) Plasma harvested from a local donor. Harvested plasma provides specific protection to local pathogens, but harvesting is time consuming. Donors
should test negative for equine infectious anemia and should be screened by a blood-typing laboratory to ensure that they are negative for QU/AA/alantibodies and alloantigens.

(2) Administration
(a) The volume of plasma required depends on the magnitude of the immunoglobulin deficiency, the weight of the foal, and the immunoglobulin concentration in the donor plasma. Preexsisting sepsis dramatically alters the distribution and catabolism of antibody in plasma and generally increases the amount required to achieve adequate passive transfer.
(b) A general guideline is to administer 200–400 mg IgG/kg; for plasma of average quality, this translates to 20–40 ml/kg. In a healthy foal of average weight, administration of 20 ml/kg of plasma (1 L) will raise the IgG concentration by approximately 200 mg/dl.
(c) The highest concentrations of IgG are attained 1–3 hours posttransfusion, but it is best to assess the effects of plasma administration 24 hours posttransfusion, after redistribution has occurred to extravascular sites.
(d) Plasma should be administered through an in-line filter. The first 50 ml is administered slowly and the foal is closely monitored for tachypnea, tachycardia, or altered behavior. If no adverse reactions are observed, plasma is administered at a rate of 20 ml/kg/hour (1 L/hour for a 50 kg foal).

(2) Administration
(a) The principles of administration are the same as for foals. The recommended volume of blood to administer is 25 L for the average calf with FPT.
(b) Lambs, piglets, and goat kids. Bovine colostrum is frequently used to supplement immunoglobulin in lambs, piglets, or goat kids if dam colostrum is not available. Anemia is occasionally reported in lambs fed bovine colostrum; the anemia has been attributed to immune complex attachment to the lamb’s erythrocytes, resulting in the removal of the erythrocytes from the circulation.

E. Prevention

1. Foals
   a. Prophylactic treatment. Colostral IgG content should be evaluated using a colostrometer to predict the risk of FPT in the foal. If the colostral specific gravity is less than 1.060, some degree of IgG deficiency, the weight of the foal, and the immunoglobulin concentration in the donor plasma. Preexsisting sepsis dramatically alters the distribution and catabolism of antibody in plasma and generally increases the amount required to achieve adequate passive transfer.
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2. Calves
   a. Force-feeding of colostrum. Producers should be encouraged to observe early nat-
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V. SEPTICEMIA AND FOCAL INFECTION

A. Introduction. Septicemia and focal infection are major causes of morbidity and mortality in neonatal foals and calves. FPT and unsanitary management practices are important factors predisposing the neonatal foal or calf to septicemia. The early signs of neonatal septicemia are subtle and nonspecific and are often missed by the owner of the foal or calf. Consequently, many septicemic neonates are presented with well-established infections involving multiple organ systems; these animals have a poor prognosis.

B. Etiology, pathogenesis, and predisposing factors

1. Etiology. Gram-negative aerobic bacteria are the predominant cause of septicemia in neonatal foals and calves; however, aerobic gram-positive infections and anaerobic infections have been documented.
   a. Escherichia coli is the bacterial agent isolated most frequently from septicemic foals and calves.
   b. Other commonly isolated bacterial agents include Actinobacillus species (foals), Pasteurella species (calves and foals), Klebsiella species (calves and foals), and Salmonella species (calves and foals).
   c. In addition, there are sporadic reports of the following agents being recovered: Pseudomonas species, Listeria monocytogenes, Clostridium perfringens, Streptococcus species, and Staphylococcus aureus. In foals, streptococci are usually recovered in conjunction with gram-negative bacteria, but have been isolated in pure culture from foals with septic physitis, osteomyelitis, and large subcutaneous abscesses. Polymicrobial infections are documented with some frequency in calves.

2. Pathogenesis. Most neonatal infections are caused by opportunistic bacteria residing in the genital tract, on the skin, or in the environment of cattle and horses.
   a. In utero-acquired infections may ascend from the vagina, occur via hematogenous spread, or spread directly from the uterine wall. Clinical signs are evident during the first 24 hours of life.
   b. Infections acquired during delivery usually occur in stressed foals and should be suspected when meconium-contaminated amniotic fluid or a meconium-stained foal is observed. Portals of entry include the respiratory and digestive tract or the umbilicus.
   c. Infections acquired after birth usually manifest themselves when the neonate is 48–96 hours old and are the result of inadequate passive transfer of colostral immunoglobulin, poor husbandry practices, endemic infectious disease on the farm, or predisposing disease conditions.

3. Predisposing factors. There are a number of periparturient factors that increase the risk of septicemia in the neonate.
Clinical findings

1. Septicemia. Clinical signs of septicemia in the neonate vary according to the stage of disease and the site of localized infection.

   a. The early signs of septicemia in neonatal calves and foals are vague and nonspecific and are often indistinguishable from noninfectious diseases or focal infections (e.g., diarrhea). Early clinical signs may include depression, lethargy, poor suckle reflex, weakness, recumbency, diarrhea, and dehydration.

   b. Body temperature abnormalities may include fever or hypothermia; however, a normal rectal temperature should not be used to exclude a diagnosis of septicemia.

   c. Abnormal mucous membranes are usually present in septicemic neonates. Coloration may range from a muddy red-gray, to mottled, pale, or cyanotic. A toxic limb, with the incisors is occasionally observed in foals. The capillary refill time is usually delayed (>2 seconds) and scleral injection is common. Careful inspection may reveal petechiation of the ears, sclera, vulvar, or buccal membranes and suggests presence of disseminated intravascular coagulation (DIC).

2. Localized infection. Localization of infection in various organs of septicemic neonates can cause a variety of clinical signs.

   a. Pneumonia may occur as a complication of septicemia. Cough, nasal discharge, tachypnea, dyspnea, and fever support the diagnosis, but in many septicemic neonates, respiratory rates and lung sounds are normal despite extensive lung pathology. Therefore, chest radiographs are indicated in all septicemic neonates.

   b. Diarrhea may occur secondary to septicemia or enteritis caused by enteropathogens. Enteritis may provide a portal of entry for opportunistic bacteria.

   c. Septic meningitis is a common complication of septicemia in the neonatal animal. Early signs include lethargy, depression, aimless wandering, and abnormal vocalization. Signs usually progress to diffuse cranial nerve deficits; apparent blindness; truncal and limb ataxia; weakness; recumbency; and coma, seizures, or death.

   d. Septic arthritis and osteomyelitis are common, debilitating sequela of neonatal septicemia. Acute lameness, periarticular edema, joint capsule distention, and arthralgia and increased peripheral ulcerative osteomyelitis (e.g., osteomyelitis, septic arthritis) are common.

   e. Ophthalmitis is characterized by heat, pain, swelling, and purulent discharge from the umbilicus (see also VI A). However, the absence of external signs of umbilical infection should not preclude a diagnosis of ophthalmitis (ultrasonographic evaluation of the umbilical structures and deep abdominal palpation will confirm involvement of internal structures).


   a. The early stage of septic shock (hyperdynamic septic shock, septicemia without circulatory collapse) is characterized by injected mucous membranes, a normal capillary refill time and blood pressure, and warm extremities. Localizing signs of infection may or may not be present. Prompt and aggressive intervention at this time can result in a favorable outcome.

   b. The late stage of septic shock (hypodynamic septic shock) is characterized by tissue hypoperfusion. Clinical signs include cold extremities, sluggish capillary refill, hypotension, pale gray mucous membranes, and markedly altered mentation. At this stage, multiorgan failure is present and therapeutic intervention is often futile.

D. Diagnostic plan

1. Routine laboratory testing

   a. Leukogram abnormalities are commonly encountered in septicemic neonates. The white blood cell (WBC) count may be normal early in the course of sepsis, but an increase in the number of band neutrophils or toxic changes in neutrophils (e.g., Donath-Landsteiner bodies, toxic granulation, vacuolization) are usually present. Neonatal farm animals with established septicemia generally have a profound leukopenia and neutropenia accompanied by toxic changes in their neutrophils.

   b. Fibrinogen concentration can be used to determine whether infection was acquired pre- or postnatally. A foal infected in utero may have a fibrinogen concentration as high as 1000 mg/dl at birth. Moderate increases in fibrinogen concentration (to approximately 400–500 mg/dl) are expected in neonates with early postnatal infections, but with chronicity, or well-established local infection, the concentration increases dramatically.

   c. Blood glucose. Hypoglycemia is a common finding in the neonate with septicemia and has been attributed to decreased feed intake, low hepatic glycogen stores, and abnormal glucose metabolism caused by endotoxemia (depressed hepatic gluconeogenesis, hepatic glycogenolysis).

   d. IgG determination should be carried out in all neonatal farm animals suspected of being septicemic because there is a strong association between poor passive transfer of colostral immunoglobulin and septicemia.

   e. Metabolic blood gas analysis is an important component in the evaluation of a septicemic foal or calf. Hypoxemia and a metabolic acidosis are frequently present. Serum biochemical profile. Biochemical abnormalities that are detected with some frequency in the septicemic neonate are azotemia, which is attributed to poor renal perfusion, and hyperbilirubinemia, which is usually ascribed to endotoxin-induced cholestasis. In addition, foals or calves with severe diarrhea may exhibit electrolyte abnormalities (e.g., hypokalemia, hypochloremia, hypocalcemia).

2. Cultures

   a. Blood cultures. A positive blood culture is required for a definitive antemortem diagnosis of sepsis, but it may take as long as 48–72 hours before a culture can be determined to be positive. Blood cultures should be repeated in any hospitalized foal that deteriorates clinically, has a fever spike, or exhibits a dramatic change in its WBC picture.

   (1) Technique. Blood cultures are easy to perform, but attention should be paid to sterile technique. The hair should be clipped and the venipuncture site surgically prepared. Depending on the culture type, a set amount of blood is withdrawn and transferred in an aseptic manner to the culture bottle or vial. A clean needle should be used to transfer the blood into the bottle.

   (a) Aerobic and anaerobic cultures are often performed.

   (b) Some authors recommend that at least two cultures spaced 1 hour apart be performed to maximize the chances of obtaining a positive result. However, in human neonates, a single culture has been shown to detect the presence of bacteria in the blood 91.5% of the time.

   (2) Sensitivity. Although previous antimicrobial use may cause false-negative blood culture results, the sensitivity of blood cultures in foals has been remarkably good, ranging from 61%–80%.
b. Culture from sites of focal infection. Bacterial cultures can also be performed on fluid obtained from sites of focal infection (e.g., cerebrospinal fluid, joint fluid, peritoneal fluid, tracheal fluid). In foals with *utero-acquired* infections, blood cultures are frequently negative because infection occurs via inhalation or ingestion. In such cases, it may be useful to culture the pharynx, trachea, external ear canal, and stomach contents.

1. Culture of the same pathogen from more than two sites of focal infection supports a diagnosis of bacteremia.
2. Recovery of the same pathogen from blood and a site of focal infection lends support to the contention that the pathogen recovered from blood is in fact significant.

3. Sepsis scoring systems. Because no single laboratory test has emerged as being completely reliable for the early diagnosis of septicemia in farm animal neonates, various scoring systems and predictive models using easily obtainable historical, clinical, and clinical laboratory data have been developed for this purpose. In general, the goal of these mathematical models is to identify septicemic neonates early in the course of disease when appropriate therapeutic intervention would most likely result in a favorable outcome.

a. Laboratory parameters incorporated in these models include neutrophil count, band neutrophil count, toxic changes in neutrophils, fibrinogen concentration, blood glucose, and IgG determination.

b. Clinical parameters that appear in most of these models are scleral injection, fever or hypothermia, and evidence of focal infection (e.g., uveitis, diarrhea, respiratory distress, joint effusion).

4. Historic data incorporated in some of the models include history of vaginal discharge, systemically ill mare, general anesthesia in the mare, induction of parturition, and premature birth (i.e., less than 320 days' gestation).

E. Therapeutic plan

1. General supportive care

a. Respiratory support. Hypoxemia must be corrected and respiratory failure treated, if present (see Table D). Fluid resuscitation.

b. Hypovolemic shock and hypoglycemia should be treated with appropriate warmed intravenous fluids.

1. Alternating lactated Ringers solution with 5% dextrose, or administering 2.5% dextrose and 0.45% saline, is often sufficient.

2. If metabolic acidosis is severe or uncorrectable by volume expansion with balanced polyionic solutions, intravenous infusion of isotonic (1.3%) sodium bicarbonate solution may be required.

c. Intravenous plasma should be administered to restore circulating blood volume, osmotic pressure, and immunoglobulin concentrations.

d. Positive inotropic agents. For circulatory embarrassment that persists after rehydration, positive inotropes should be administered. Dobutamine (2-5 μg/kg/min by infusion) is the drug of choice because it increases cardiac output and causes splanchic and renal vasodilation.

2. Nonsteroidal antiinflammatory drugs (NSAIDs) have been shown to counteract a number of the clinical and laboratory changes associated with endotoxemia, including decreased cardiac output and hypotension.

Flunixin meglumine (0.25-1.1 mg/kg, intravenously or intramuscularly every 8 hours) has been recommended.

3. Nutritional support. Foals that are unable to nurse should be fed via a nasogastric tube using the recommendations given in Table D. Total or partial parenteral nutrition is indicated in foals that cannot be fed orally.

2. Control of generalized infection

a. General principles

1. Antibiotic selection. When possible, antibiotic selection is based on the results of blood culture and sensivity testing. However, because blood culture results are not returned for several days, and the offending agent may not be recovered, empiric therapy is usually initiated and modified later if needed. Broad-spectrum bactericidal drugs are indicated in the treatment of septicemic neonates for the following reasons:

a. Cram-negative and polymicrobial infections should be anticipated
b. Septicemia in neonates is rapidly progressive
c. Many septiceamic neonates are neutropenic
d. Immune function in neonates is usually compromised by stress and FPT

2. Antibiotic administration. Initially, the intravenous route is preferred for antibiotic administration because peripheral circulation may be compromised, making absorption from other routes inconsistent.

3. Duration of therapy. The recommended duration of therapy for suspected but undocumented sepsis is 7–10 days. Neonates with positive blood cultures and no evidence of focal infection should be treated for at least 2 weeks, and those with localized infections should be treated for 3–4 weeks.

b. Antibiotic therapy in foals. Selection of antimicrobials should be based on the results of an antimicrobial susceptibility pattern; however, until culture results are returned selection of drugs will be somewhat empirical.

1. A combination of a *β-lactam* antibiotic (e.g., penicillin, ampicillin) and an aminoglycoside (e.g., gentamicin, amikacin) usually provides good broad-spectrum coverage. Many clinicians prefer amikacin because it is less nephrotoxic and is less likely to be associated with development of resistant bacterial infections. If possible, peak and trough serum concentrations of aminoglycosides should be monitored to ensure that the drug dose and dosing interval are appropriate. General doses are as follows:

a. Gentamicin: 22 mg/kg, intramuscularly or intravenously, every 8–12 hours or 3.3 mg/kg, intramuscularly or intravenously, every 12 hours
b. Amikacin: 7 mg/kg, intramuscularly or intravenously every 8–12 hours or 10 mg/kg, intramuscularly or intravenously, every 12 hours

2. Cefalexin or cefotaxim can be used in the empiric treatment of septic neonates. General doses are as follows:

a. Cefalexin: 20–30 mg/kg, intravenously or intramuscularly, every 8 hours
b. Cefotaxim: 2.2-6.6 mg/kg, intramuscularly or intravenously, every 8–12 hours

3. Other drugs. The following drugs are also listed for the treatment of septicemic foals.

a. Trimethoprim-sulfonamide combinations: 15 mg/kg, intravenously or orally every 12 hours (resistance to this drug is widespread)

b. Chloramphenicol: 25–50 mg/kg, intravenously or orally, every 6 hours
c. Ticarcillin–clavulanic acid: 50 mg/kg, intravenously, every 6–8 hours

c. Antibiotic therapy in calves is simplified by the limited number of choices available.

1. Cefoxitin (5 mg/kg, administered intravenously or intramuscularly every 8–12 hours) is widely used to treat calf septicemia, as are the potentiated sulfonamides (15 mg/kg, administered orally, intramuscularly, or intravenously every 12 hours).

2. In Canada, ampicillin sulbactam (6.6 mg/kg administered intramuscularly every 8–12 hours) has been used successfully to treat neonatal calf septicemia.

3. Aminoglycosides are generally avoided because of prolonged tissue residues (18 months in the kidney) and because their use is not endorsed by the National Cattlemen's Association. However, some clinicians still use gentamicin (3–5 mg/kg, administered intravenously or intramuscularly every 12 hours) if they can obtain assurance from the client that the animal in question will not enter the food chain for at least 18 months.

4. Fluoroquinolones are banned for use in cattle in the United States, but enrofloxacin (2.5–5 mg/kg orally every 24 hours) is used widely in other countries to treat septicemia in neonatal calves.
3. Treatment of focal infections
   a. Septic meningitis (see also Chapter 11 E)

   (1) Antibiotic therapy
   (a) Septic meningitis is treated with bactericidal antimicrobials that penetrate the blood–brain barrier (e.g., trimethoprim–sulfonamide combinations or third-generation cephalosporins).
   (b) Combination therapy using a β-lactam antibiotic or trimethoprim–sulfonamide with an aminoglycoside is also beneficial as a result of synergistic interactions, despite poor penetration of aminoglycosides into the CSF.
   (c) Although chloramphenicol readily crosses the blood–brain barrier, it is bacteriostatic against gram-negative enteric bacteria and is not recommended for the treatment of bacterial meningitis.

   (2) Anticonvulsants (e.g., diazepam, phenobarbital) and NSAIDs (e.g., flunixin meglumine) may also be indicated.

   b. Septic arthritis or osteomyelitis. Therapeutic measures include:
      (1) Systemic antibiotic therapy
      (2) Assurance of adequate serum immunoglobulin concentrations
      (3) Analgesic therapy
      (4) Drainage and removal of debris from the joint and adjacent tissues (lavage with sterile polyionic fluids)
      (5) Articular rest
      (6) Exogenous sodium hyaluronate or polysulfated glycosaminoglycan therapy
      (7) Surgical debridement, installation of a sterile drain, and immobilization of the limb with a Robert–Jones splint bandage (in cases of osteomyelitis with evidence of bone sequestration or osteolysis)

   c. Uveitis. Treatment should include systemic antimicrobial therapy and local therapy to prevent permanent ocular damage: A mydriatic (e.g., atropine), topical ophthalmic corticosteroid, systemic NSAID, and broad-spectrum ophthalmic preparation are indicated.

   d. Omphalitis. Treatment is discussed in VI A.

F. Prognosis

1. The overall survival rate for septicemic neonates is less than 60%, but early recognition of sepsis and appropriate and aggressive intervention improve the outcome.
   a. A septicemic large animal neonate with FPT and evidence of multiple organ involvement should always be given a guarded prognosis.
   b. A neonate with a negative blood culture but evidence of focal infection (e.g., pneumonia, diarrhea) has a more favorable prognosis.
   c. Appropriate and early therapeutic intervention in foals with intraabdominal infections often results in a favorable outcome: survival rates greater than 75% have been quoted for this group.

2. The long-term prognosis for future athletic performance is guarded if multifocal bone or joint disease is diagnosed.

G. Prevention.

Management of the mare with placentitis can decrease the risk of premature delivery and postnatal infection. Aspergillus species, β-hemolytic streptococci, and E. coli are the organisms isolated most frequently from mares with placentitis. Treatment of placentitis includes the use of antimicrobial agents, anti-inflammatory drugs, prostaglandins, and stall rest.

1. Antimicrobial agents are usually selected based on results of culture and sensitivity. Trimethoprim–sulfonamide (25–35 mg/kg every 12 hours) is a good antimicrobial to start with because high concentrations are achieved in the placenta, allantoic fluid, amniotic fluid, and fetal serum.

2. NSAIDs, such as phenylbutazone (4 mg/kg orally every 24 hours) or flunixin meglumine (1 mg/kg orally, intravenously, or intramuscularly every 12 hours) may reduce uterine inflammation and uterine production of prostaglandin F2α, thereby decreasing the risk of premature delivery.

3. Supplemental progesterin therapy with altralogen (0.044 mg/kg every 24 hours) has been employed to maintain pregnancy in mares with placentitis. Although this progesterin will maintain pregnancy in ovariohysterectomized mares, the efficacy of this regimen in late-term mares with high-risk pregnancy is unknown.

VI. UMBILICAL ABNORMALITIES

A. Umbilical remnant infections. Infection may involve the urachus, umbilical veins (omphalophlebitis), one or both umbilical arteries (omphaloarteritis), or many structures (omphalitis, umbilical abscess).

1. Clinical findings may include umbilical enlargement, pain on palpation, patent urachus (common in foals, rare in calves), or purulent discharge. The internal or intra-abdominal umbilical structures may be the only structures affected in foals, making infection difficult to detect on physical examination.
   a. As with most neonatal infections, the first signs noted are decreased suckling and depression. Other abnormalities may include fever, dysuria, poliakuria, and terebrum.
   b. Deep abdominal palpation can be used to evaluate the internal umbilical structures, particularly in calves. Enlargement of the umbilical vein may be palpable coursing cranially toward the liver, enlarged umbilical arteries may be palpable coursing caudally toward the bladder. Palpation may elicit a grunt and abdominal splinting may be noted in calves with a septic umbilicus and associated peritonitis.

2. Pathogenesis. Infection may originate following contamination of the external umbilicus after birth or result from seeding from other sites during periods of septicemia. Bacteria may localize in the umbilical vessels, urachus, bladder, or interstitial tissues and the infection may extend into the peritoneal cavity or progress to a generalized septicemia. Urachal abscessation can cause the previously closed urachus to become patent externally or allow urine to leak subcutaneously or into the peritoneal cavity.

3. Diagnostic plan and laboratory tests
   a. Routine laboratory tests. Clinico-pathological alterations usually include neutrophilia with toxic changes in neutrophils and hyperfibrinogenemia.
   b. Blood culture. Because of the association of umbilical remnant infections with septicemia, blood cultures should always be performed.

4. Ultrasonography should be used to evaluate the extent of involvement of internal umbilical structures. An increased diameter, thickened wall, or abscesses may be visible. The procedure is usually carried out with the foal in lateral recumbency and the calf standing.
   (1) In foals, the umbilical arteries and vein can be followed from the external umbilical stump to the cranial aspect of the bladder and liver, respectively. The urachus can usually be visualized along with the umbilical arteries just caudal to the umbilical stump.
   (2) In calves, the umbilical arteries retrace into the abdominal cavity and thus should not be identifiable in the umbilical stalk; they are most easily located along the apex of the urinary bladder. The umbilical vein of calves is scanned from the umbilical stalk to the liver along the right ventral abdomen. Umbilical remnants can usually not be identified in normal calves.

4. Therapeutic plan. The treatment options for umbilical remnant infections are medical management or surgical resection.
   a. Medical therapy consists of prolonged antibiotic administration (see V B 2) and encouragement of drainage. In one study, 50% of foals responded to medical therapy alone.
b. Surgical management is indicated for the following patients:
   (1) Patients with persistent urine dribbling in spite of cauterization and resolution of predisposing factors (e.g., recumbency)
   (2) Patients with involvement of other umbilical structures (demonstrated by ultrasonography)
   (3) Patients with subcutaneous or intra-abdominal urine accumulation resulting from a rent in the urachus

5. Prevention
   a. The umbilicus should be allowed to rupture spontaneously.
   b. Critically ill neonates should be restrained to prevent excessive tension on the ventral abdomen.

Excessive bleeding from the umbilicus. Bleeding from the umbilicus may occur, particularly if it was cut or ligated. Occasionally, hemorrhage is severe enough to necessitate a blood transfusion. Rarely, hernoperitoneum will result from hemorrhage from an intra-abdominal umbilical vessel.
1. Which of the following conditions affecting neonatal foals is usually associated with a favorable long term outcome (i.e., a satisfactory performance animal)?
   (1) Hypoxemic–ischemic encephalopathy (HIE)
   (2) Prematurity with tarsal bone collapse
   (3) Septic arthritis
   (4) Twinning
   (5) Septic physis

2. Which of the following historic or laboratory findings would indicate that a prematurely delivered foal is endocrinologically mature ("ready for birth")?
   (1) Cesarean section at 322 days' of gestation
   (2) Total white blood cell (WBC) count less than 5000 cells/μL on day 3
   (3) A neutrophil–lymphocyte ratio greater than 2 at birth
   (4) A plasma fibrinogen concentration of 200 mg/dl on day 1
   (5) No change in the neutrophil–lymphocyte ratio in response to adrenocorticotropic hormone (ACTH) administration

3. The bacterial agent that is cultured most frequently from neonatal calves and foals with septicemia is:
   (1) Actinobacillus equuli
   (2) Staphylococcus aureus
   (3) Klebsiella pneumoniae
   (4) Listeria monocytogenes
   (5) Escherichia coli

4. Which one of the following antibiotics, or combination of antibiotic drugs, would be a rational first choice for treatment of a foal with clinical and laboratory findings indicating a diagnosis of septicemia?
   (1) Chloramphenicol
   (2) Oxytetracycline
   (3) Penicillin and amikacin
   (4) Lincomycin and spectinomycin
   (5) Ampicillin

5. Which one of the following antibiotics would be a rational first choice for treatment of a calf with clinical and laboratory findings indicating a diagnosis of septicemia?
   (1) Chloramphenicol
   (2) Oxytetracycline
   (3) Enrofloxacin
   (4) Ceftiofur
   (5) Erythromycin

6. In a study conducted in foals, which one of the following disinfectants was shown to cause the greatest decrease in bacterial count at the end of the umbilicus immediately post-dipping?
   (1) Dilute sodium hypochlorite
   (2) 0.5% Chlorhexidine
   (3) 7% Tincture of iodine
   (4) 2% Iodine
   (5) 1% Povidone–iodine

7. How much milk should a healthy 1-week-old foal weighing 60 kg consume in a 24-hour period?
   (1) 12 liters
   (2) 6 liters
   (3) 4 liters
   (4) 2 liters
   (5) 24 liters

8. Which one of the following statements pertaining to the feeding of bovine colostrum to neonatal foals is true?
   (1) Bovine colostrum should never be fed to foals because it causes severe bloating and diarrhea.
   (2) Bovine colostrum should never be used in foals because it often causes an immune-mediated hemolytic anemia.
   (3) Bovine colostrum prevents septicemia caused by Actinobacillus equuli, but not septicemia caused by Escherichia coli.
   (4) Bovine colostrum has a short half-life, but will protect the foal against septicemia most of the time.
   (5) Bovine colostrum cannot be used in foals because unrealistically large volumes are required to provide adequate protection.

9. Which one of the following laboratory findings likely indicates adequate passive transfer on routine screening of a healthy 2-day-old calf?
   (1) Total serum protein (TSP) concentration of 6.2 g/dl
   (2) Immunoglobulin G (IgG) concentration of 400 mg/dl
   (3) γ-Glutamyl transferase (GGT) activity of 23 IU/L
   (4) IgG concentration of 800 mg/dl
   (5) TSP concentration of 45 g/dl

10. Which one of the following drugs is contraindicated in treating a neonatal foal with coma, intermittent seizures, and oliguria following an acute asphyxial episode at birth (premature placental separation)?
    (1) Diazepam
    (2) Dimethylsulfoxide (DMSO)
    (3) Acepromazine
    (4) Furosemide
    (5) Dopamine
1. The answer is 1 [IC 2 a]. With appropriate care foals with hypovemic-ischemic encephalopathy survive, and in the long term, this condition has little impact on performance. In contrast, foals with infectious or noninfectious orthopedic diseases have poor long-term prognoses. Products of a twin pregnancy also have a grim prognosis as performance animals because they are small in stature and often suffer from noninfectious orthopedic conditions (e.g., tarsal collapse).

2. The answer is 3 [II A 2. E 2 b-c]. A neutrophil−lymphocyte ratio that is greater than 2 by 3 days of age indicates maturity of the adrenal−pituitary−hypothalamic axis and readiness for birth. In contrast, a white blood cell (WBC) count less than 5000 cells/μL in the first 3 days of life and no widening of the neutrophil−lymphocyte ratio in response to adrenocorticotropic hormone (ACTH) administration indicates immaturity of the endocrine system and un readiness for birth, as does a cesarean section performed at 322 days’ gestation. A fibrinogen concentration of less than 400 mg/dl at birth suggests that chronic intrauterine stress due to placentitis was probably not the cause of the premature delivery and, therefore, the fetus was probably not ready for birth.

3. The answer is 5 [IV B 1 a]. Escherichia coli is the pathogen cultured most frequently (representing more than 50% of isolates) from septicemic farm animal neonates. Staphylococcus aureus, Klebsiella pneumoniae, and Listeria monocytogenes are isolated sporadically from septic calves and foals. Actinobacillus equuli is usually only found in foals.

4. The answer is 3 [IV E 2 b [1]]. Both penicillin and amikacin are bactericidal drugs, and the combination provides broad-spectrum coverage. Amikacin is preferred over gentamicin by many clinicians because it is less nephrotoxic and also because resistance is less likely. Chloramphenicol provides broad-spectrum coverage, but it is bacteriostatic and therefore not ideal for treating a rapidly progressing infection in a neonatal animal with an immature immune system. Resistance of septicemia pathogens to ampicillin and oxytetracycline is widespread.

5. The answer is 4 [IV E 2 c]. Cefitiofur is a broad-spectrum bactericidal drug and many clinicians use it for the treatment of septicemia in neonatal calves. It is illegal to use chloramphenicol in cattle; therefore, this antibiotic would not be a good choice. Oxytetracycline is bactericidal but resistance is widespread. It is illegal to use enrofloxacin in cattle in the United States and there are some concerns that this antibiotic may cause cartilage abnormalities in rapidly growing young animals. Erythromycin is bacteriostatic and has a gram-positive spectrum of activity, so this drug would not be a good choice in a neonatal calf with a rapidly progressing infection.

6. The answer is 2 [IV A 5 b]. In a study conducted in foals, application of 0.5% chlorhexidine to the umbilicus was shown to cause the lowest bacterial count on the umbilicus immediately post-treatment. The use of 7% tincture of iodine is discouraged; local tissue necrosis associated with the application of 7% iodine may actually increase the prevalence of infection.

7. The answer is 1 [II D 4 a]. A healthy newborn foal usually consumes approximately 20%–25% of its body weight in milk over a 24-hour period. Therefore, a healthy 1-week-old foal weighing 60 kg should consume 12 liters of milk over a 24-hour period (0.20 x 60 = 12).

8. The answer is 4 [IV D 1 a (2) a)]. Although the immunoglobulins in bovine colostrum are rapidly catabolized in foals, bovine colostrum does provide protection against most pathogens capable of causing septicemia, with the exception of Actinobacillus equuli, a pathogen unique to the equine environment. Bovine colostrum is well-tolerated by foals and only occasionally causes diarrhea. There are no reports of hemolytic anemia in foals fed bovine colostrum, although anemia has been reported to occur in lambs. The volume of bovine colostrum required to confer protection in foals is approximately 4 liters, a volume that is easily achieved.

9. The answer is 1 [IV C 3]. A TSP concentration greater than 5.0 g/dl generally indicates adequate passive transfer. A healthy 2-day-old calf with a total serum protein (TSP) concentration of 6.2 g/dl has adequate passive transfer. An immunoglobulin C (IgC) concentration greater than 1000 mg/dl indicates adequate passive transfer, as does γ-glutamyl transferase (GGT) activity greater than 300 IU/L.

10. The answer is 3 [III F 1 a (3)]. Acetpromazine, which may lower the seizure threshold and cause systemic hypotension, further decreasing renal perfusion, is contraindicated in a neonatal foal with neurologic signs following an acute asphyxial episode at birth. Diazepam is frequently used for seizure control and dimethyl sulfoxide (DMSO) is employed by many clinicians to treat cerebral edema. Furosemide is indicated as a diuretic in oliguric asphyxiated foals and dopamine infusion is advocated to increase renal blood flow.