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# Field Triage of the Neonatal Foal



Elizabeth A. Carr, DVM, PhD

## KEYWORDS

• Neonatal foal • Field triage • Sick • Recumbent • Weak

## KEY POINTS

- When first evaluating a weak, recumbent, or lethargic foal on a farm it is often difficult to make a definitive diagnosis.
- The approach should be to treat what is treatable and prevent what is preventable.
- In many cases, the goal will be to stabilize a foal before referral to a tertiary care facility where more intensive and continuous treatment can be performed.

## INTRODUCTION

The purpose of this article is to provide a quick reference for field triage of the sick neonatal foal. Therefore, information is focused toward diagnostics and treatments that can be performed in the field. When evaluating a weak, recumbent, or lethargic foal on the farm, it is often difficult to make a definitive diagnosis. Therefore, the approach should be to treat what is treatable and prevent what is preventable. In many cases, the goal will be to stabilize a foal before referral to a tertiary care facility where more intensive and continuous treatment can be performed.

## PHYSICAL EXAMINATION OF THE NEWBORN FOAL

The normal foal should attempt to rise into sternal recumbency within seconds to minutes after delivery. On average, foals stand within 1 to 2 hours and nurse within 2 to 3 hours of birth. Mucous membranes and sclera may show the presence of ecchymotic hemorrhages caused by the pressure of passage through the birth canal and be mildly injected compared with adults. The capillary refill time is similar to adults. A normal cardiac sinus rhythm or sinus arrhythmia is ausculted. It is common to hear a systolic murmur (point of maximum intensity at the left heart base) for a few days after birth. Murmurs that persist longer should be evaluated further. The normal foal's respiratory rate and effort should decrease over the course of the first day of life,

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The author has nothing to disclose.

Department of Large Animal Clinical Sciences, College of Veterinary Medicine, Michigan State University, 736 Wilson Road, East Lansing, MI 48824, USA

E-mail address: [carreliz@cvm.msu.edu](mailto:carreliz@cvm.msu.edu)

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and its heart rate should increase after a few minutes (Table 1). Foals should urinate within the first 24 hours of life, and urine should become progressively more dilute as they begin to consume a liquid diet. Newborn colts will occasionally be born with a persistent frenulum preventing them from dropping their penis to urinate. This condition is generally not a concern, as it will resolve over time. Many normal foals are born with a mild degree of carpal and fetlock valgus in their front limbs and slight varus in their hind fetlocks. This condition typically resolves as they grow. Foals should pass meconium, the first feces, within 12 to 24 hours. Meconium is dark brown to tan and may be hard or pasty. Subsequent milk feces are yellow tan and typically softer in consistency.

Neonates lack a menace response, as this is a learned behavior that will develop at a few weeks of life. Stimulation (auditory or visual) often results in exaggerated, jerky head movements. The neonatal foal's primary behavior should be directed toward maintaining close contact with its dam.

Foals should have a strong suckle reflex and nurse for relatively brief periods (minutes) as many as 8 times an hour. If a mare produces a large volume of milk, the foal may be unable to swallow it all and a small volume may be seen at its nostril after nursing. If persistent, this finding should trigger further evaluation to rule out dysphagia or a cleft palate.

Most lightweight foals will gain between 1 and 2 lb (0.5–1.0 kg) of weight per day. Foals that repeatedly return to the udder to nurse may be frustrated because of a lack of adequate milk production. Measurement of urine specific gravity and weight gain are important methods to determine adequate nutritional intake. Foals ingesting normal volumes of milk will have dilute urine (specific gravity 1.004–1.010). If a scale is not available, a string or weight tape can be used to measure change in body girth to assess daily weight gain.

Foals can be bradycardic at birth; the heart rate should increase relatively quickly to normal values. Persistent bradycardia can be caused by hypoxia, hypoglycemia, and hypothermia. Oxygen supplementation should be instituted. A continuous intravenous (IV) infusion of dextrose is recommended (see section on fluid therapy) if glucose monitoring is not available. Bolus therapy with glucose-containing fluids is not recommended, as hyperglycemia has deleterious effects. If bolus therapy is unavoidable, dextrose should be added to an isotonic crystalloid at a low percent (0.5% solution = 10 mL 50% glucose in 1-L crystalloids). If a foal is mildly hypothermic, it is recommended to allow slow, passive warming (cover the foal and keep in a dry, warm area out of the wind), as hypothermia is protective against hypoxic brain injury.<sup>1</sup> With more severe hypothermia, active warming is recommended and is best done by infusion of warmed IV fluids. The use of external heat sources is controversial as the resultant peripheral vasodilation can cause a reflex drop in core temperatures as cold blood flows centrally from the periphery.

The causes of tachycardia include pain, hypovolemia, anemia, fever, and excitement. If pain, fever, anemia, and excitement are ruled out, fluid therapy is indicated to attempt to correct hypovolemia (see section on IV fluid therapy).

Parameter	<10 min	2–4 h	24 h
Heart rate beats per min	40–60	100–200	80–120
Respiratory rate beats per min	40–60	20–40	20–40
Body temperature (F/C)	99–102/37–39	99–102/37–39	99–102/37–39

As the foal clears fluid from its lungs, its respiratory rate and effort should decline. Prominent rib retraction and the presence of an abdominal effort with paradoxical collapse of the chest wall during inspiration are indicators of respiratory distress and suggest respiratory or cardiac dysfunction. The foal's chest wall is extremely compliant compared with the adult animal. Respiratory muscle contraction is needed to maintain thoracic and lung volume and prevent alveolar collapse and atelectasis. Foals that are sick, weak, hypoglycemic, or have underlying respiratory disease may develop respiratory muscle exhaustion, worsening atelectasis, and pulmonary function.

During parturition, mucous membranes may appear gray or cyanotic; this should rapidly resolve once the foal is delivered. Pale mucous membranes can be an indicator of anemia or hypovolemic shock. Icteric mucous membranes can indicate hemolysis (neonatal isoerythrolysis), in utero placental dysfunction, or liver dysfunction. Further evaluation including blood work (complete blood count and serum chemistry analysis) and ultrasound examination looking for evidence of internal bleeding is recommended. Because cyanosis requires between 2 and 5 g of deoxygenated hemoglobin per deciliter of blood, anemic individuals may not be cyanotic even in the presence of severe hypoxia.<sup>2</sup> Petechia on the mucous membranes or ears can be an indicator of septicemia or thrombocytopenia.

Examination of the eye may aid the clinician in determining a diagnosis as hypopyon, or hyphema may be present in septic foals. Retinal hemorrhages may also be present in neonates born with equine herpesvirus type 1 infection. Sick foals may have abnormal blink responses or tear production making them more susceptible to corneal injury.

Dehydration and poor body fat stores can result in the development of entropion. If not recognized quickly, corneal abrasion and ulceration can develop. Treatment should be directed at correcting the abnormal lid position. This correction can be achieved by pulling the lid margin out to its normal position and placing a skin staple or mattress suture below and perpendicular to the lid margin to hold the lid out. Temporary correction can also be achieved by injecting 0.5 mL of procaine penicillin G subcutaneously approximately 5 mm below the lid margin. As the lid distends, the margin is rolled out and returned to its normal position. This technique may need to be repeated as the solution dissipates over time. Entropion usually resolves once the foal is rehydrated or gains weight.

#### Important Points

- The absence of cyanosis does not rule out hypoxia.
- The presence of hypopyon or hemorrhage in the eye is an indicator of sepsis.
- Persistent bradycardia or deterioration in respiratory rate or effort are signs of cardiopulmonary dysfunction and require immediate intervention.

#### ***Dysphagia/Loss of Suckle/Milk Regurgitation***

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The suckle reflex is one of the first things to deteriorate when a foal is sick. Clinical signs may include coughing after nursing, milk reflux at the nose, auscultation of milk in the trachea, and/or clinical signs of pneumonia. Causes of dysphagia or loss of the suckle include

- Perinatal asphyxia syndrome (PAS), Hypoxic ischemic encephalopathy (HIE)
- Sepsis
- Hypoglycemia
- Hypothermia

- Cleft palate
- Muscle dysfunction (selenium deficiency, glycogen branching enzyme deficiency)
- Hyperkalemic periodic paralysis

In some cases, the foal will appear to nurse normally but, lacking a competent suckle, will aspirate each time it drinks. When sick foals are fed via bottle or held up to nurse, they will often struggle and aspirate a portion of the meal. Auscultation of milk in the trachea during nursing is the most accurate way to assess dysphagia and milk aspiration.

The most common cause of dysphagia seems to be a temporary loss of neuromuscular coordination of the suckle reflex in sick or premature foals. In such cases, an endoscopic examination may reveal upper airway collapse and pharyngeal edema. A cleft palate can be identified by digital examination of the oral cavity unless the cleft is in the soft palate beyond the reach of the examiner. In such cases, endoscopy or a speculum examination may be needed to definitively diagnose the condition.

Regardless of the underlying cause, it is paramount to prevent nursing, as continued aspiration will result in pneumonia. If the foal is housed with its mare, it should be muzzled to prevent nursing and an indwelling nasogastric tube placed for feeding. Antimicrobial therapy is indicated if evidence of aspiration or sepsis is present. Most dysphagic foals resolve the dysfunction over time. The time to resolution varies from days to weeks; in rare cases, swallowing never normalizes. To minimize aspiration, foals with persistent dysphagia can be trained to drink out of a bucket placed on the ground.

### **TRIAGE OF THE WEAK NEONATAL FOAL**

Facing a recumbent, weak, neonatal foal can be overwhelming. Teasing out the cause or causes without immediate access to diagnostic tools and laboratory assessment can be difficult. The most common causes of weakness and the inability to rise or nurse are sepsis, PAS, and prematurity/dysmaturity. The assessment and treatment should be directed at both the treatment of problems and the prevention of potential complications. If a foal fails to respond or if other factors impact the ability to treat the foal at the farm, referral is indicated. Treatment recommendations include both therapy for specific conditions and general treatment of the weak, recumbent neonate.

#### ***Sepsis***

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Sepsis is the leading cause of foal mortality. Sepsis may develop in utero or after foaling. Clinical signs will depend on the organ systems affected as well as the duration and severity. In the early hyperdynamic stage, foals may be lethargic, have a poor suckle reflex, and injected hyperemic mucous membranes. Petechial hemorrhages may be present on the membranes or pinnae. A decreased capillary refill time, tachycardia, and tachypnea may be present. A fever may or may not be present. As sepsis progresses, the foal becomes progressively more depressed or obtunded. Poor cardiac output results in cool extremities, tachycardia, prolonged capillary refill time, and poor peripheral pulses. Hypothermia and hypoglycemia may develop. The common clinicopathologic abnormalities seen with sepsis include leucopenia, neutropenia, and hypoglycemia. With profound hypoglycemia or respiratory compromise, hypoxemia, hypercapnia, and a mixed acidosis may be seen.

#### ***PAS***

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The underlying pathologic process in PAS is thought to be the result of prolonged or severe tissue hypoxia. Hypoxia may occur during pregnancy, parturition, or

immediately after foaling. Decreased energy supply results in loss of membrane pumps, loss of ion gradients, calcium influx, activation of calcium-dependent enzymes and cellular damage.<sup>3</sup> Subsequent reperfusion can result in further damage via production of reactive oxygen species and inflammatory changes. Foals affected with PAS may be normal at birth and gradually become disinterested in their dams as cerebral swelling and cellular injury progresses. They may lose their suckle reflex and become excessively sleepy and difficult to rouse. Severe cases may become recumbent, progress to seizures, and develop an abnormal, apneustic pattern of breathing. Gastrointestinal signs may vary from mild feed intolerance to colic, gas distension, and bloody diarrhea. Many affected foals fail to urinate in response to bladder distension and require catheterization of the bladder to prevent rupture. Additional clinical signs will vary depending on organ system dysfunction.

Most foals affected with PAS will recover with supportive care and time. The goals of treatment of PAS are to prevent further cellular injury; control seizures and other effects of organ dysfunction; and prevent secondary problems, such as sepsis.

### ***Prematurity/Dysmaturity***

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Prematurity is defined as a foal born with a gestational age of less than 320 days. Dysmaturity is defined as a foal born after a normal gestation length (320 days or more) that exhibits the physical characteristics of prematurity.

#### **Clinical signs**

- Small body stature, fine, silky hair coat, floppy ears, flaccid lips, domed forehead
- Tendon laxity and incomplete ossification of the cuboidal bones
- May see inverted neutrophil (N) to lymphocyte (L) ratio (normal N/L 2:1, affected foals  $\leq$ 1:1)

Foals may be weak and unable to stand without assistance and have a poor suckle and poor tolerance to enteral feeding. Premature foals are often unable to regulate their body temperature or blood glucose. The premature intestinal tract may lack the specialized enterocytes necessary to absorb colostral antibodies, putting them at risk for failure of passive transfer (FPT) even after colostral ingestion.

### ***Specific Treatment of Sepsis***

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- Broad-spectrum or culture-targeted antimicrobials
  - Gentamicin 12 mg/kg every 36 hours (first 2 weeks of life). Caution: nephrotoxicity
  - Amikacin 25 mg/kg every 24 hours (first 2 weeks of life). Caution: nephrotoxicity
  - Potassium+ penicillin 22,000 IU IV every 6 hours
  - Ceftiofur 2 to 10 mg/kg IV every 6 hours, intramuscular (IM) route every 12 hours
  - Trimethoprim-sulfonamide 30 mg/kg by mouth every 12 hours; avoid with gastrointestinal disease
  - Ticarcillin/clavulanic acid 50 to 100 mg/kg IV every 6 hours
- Antiinflammatory
  - Flunixin meglumine 0.25 to 1.1 mg/kg every 12 to 24 hours

### ***Specific Treatment of PAS***

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- Further deterioration of the central nervous system (CNS) should be prevented, and seizures should be controlled.
- Secondary infection should be prevented with broad-spectrum antimicrobials.

- Perfusion and oxygen delivery should be maintained. Fluid therapy should be carefully calculated to maintain perfusion but not overhydrate, as overhydration can lead to cerebral edema and worsen neurologic dysfunction.
- Cerebral edema should be controlled. Mannitol (0.25–1.0 g/kg as a 20% solution over 20-minute infusion) will often result in neurologic improvement (temporary in individual cases).
- Respiratory compromise
  - Hypoxemia-bilateral nasal oxygen insufflations at 5 L/min
  - Hypoventilation or apneustic breathing pattern: caffeine loading dose 10 mg/kg by mouth followed by 2.5 to 3.0 mg/kg by mouth once a day to twice a day; if unsuccessful, may require mechanical ventilation

### ***Specific Treatment of Prematurity***

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- Incomplete ossification of cuboidal bones: Lateral and anteroposterior radiographs of the carpus and tarsus should be performed to evaluate for incomplete ossification of the cuboidal bones. Weight bearing should be limited in affected cases to prevent the crush of the cartilaginous precursors. The crush of the tarsal and carpal bones can result in juvenile arthritis and have long-term catastrophic effects on the foal's athletic career; therefore, serial radiographs are important to monitor the ossification process.
- FPT should be assessed/treated.

### **GENERAL THERAPY FOR THE WEAK RECUMBENT FOAL**

Broad-spectrum antimicrobial therapy is recommended in any weak recumbent foal to prevent or treat infection or sepsis.

Hypoglycemia is a frequent problem in the sick neonate. Many septic foals will have episodes of both hypoglycemia and hyperglycemia. If unable to tolerate oral feeding, institute IV nutritional support (see section on partial parenteral nutrition).

### ***Nutrition Support***

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The healthy, full-term neonate is born with enough body fat and glycogen reserves to provide an energy supply for 12 to 24 hours. Premature or systemically compromised foals (eg, in utero sepsis) may lack these minimal reserves, making them at an increased risk for hypoglycemia, hypothermia, and organ dysfunction. The enteral route of nutrition is always preferred over parenteral, as enteral feeding has been shown to increase gastrointestinal mass and function compared with the parenteral route.<sup>4</sup> Unfortunately, the sick neonatal foal may be unable to tolerate enteral feeding. Enteral feeding should be avoided in severely hypotensive or hypothermic foals. Foals with enteropathy, whether secondary to oxygen deprivation or infection, may be unable to absorb nutrients and develop colic and diarrhea when fed. A detailed history and careful repeat assessments of the neonate's gastrointestinal tract (including ultrasound examination if possible) is critical to ensure that the foal is able to tolerate enteral feedings. The measurement of abdominal size can be useful to assess gastrointestinal distension. Colic signs in foals are often subtle; foals may appear fussy or agitated and show an increase in heart rate and respiratory rate. If deterioration is noted, enteral feeding should be discontinued or decreased until resolution.

Generally, a foal that is too sick to stand is also unlikely to have an adequate suckle reflex. Small, soft nasogastric tubes are available that may be sutured in place for longer-term use (MILA International Inc, Erlanger, KY). Before feeding, it is important to determine if the tube is in the stomach. If the foal is less than 24 hours of age,

colostrum should be fed to provide both systemic and local passive immunity. It is generally best to start with small-volume feeding; if tolerated, the volume of feedings can be gradually increased every 1 to 2 feedings with an initial target goal of 10% body weight in milk per day. This volume equates to the approximate metabolic requirements of a sick, recumbent foal maintained in a relatively warm, draft-free environment.<sup>5,6</sup> Colic, bloating, increased gastric residuals, diarrhea, and ultrasound findings of ileus suggest intolerance to enteral feeding and should result in decreasing or temporarily discontinuing enteral feeding.

Assessment of the gastrointestinal tract before enteral feeding

- Borborygmi present
- Evidence of passage of gas or meconium
- Gastric residuals  $\leq$ 60 mL
- Ultrasound findings of motility without evidence of intestinal distension

### **Partial parenteral nutrition**

If enteral nutrition is not tolerated, an IV dextrose infusion should be instituted. The placenta supplies approximately 4 to 8 mg/kg/min of glucose, and this is a useful target when providing short-term parenteral support. This rate would equate to 4 to 8 mL of a 5% dextrose solution per minute. Glucose infusion alone is inadequate as a long-term nutritional source, and more complete parenteral nutrition is needed with prolonged intolerance to enteral feeding.

For example, a 50-kg, 1-day-old foal presents recumbent and weak with signs of sepsis. The physical examination reveals the presence of auscultable borborygmi. The ultrasound examination reveals a normal stomach size, normal-sized small intestine with decreased (but present motility), normal intestinal wall thickness, and no evidence of gas in the intestinal walls. A nasogastric tube is placed, and the aspiration to assess gastric residuals is negative. Approximately 60 mL of milk is given via the nasogastric (NG) tube. If the foal tolerates the feeding (no colic signs and <40 mL residual on subsequent NG tube aspiration), an additional 100 mL is fed 1 to 2 hours later. The goal is to reach approximately 400 mL every 2 hours (10% of body weight) over a 24-hour period. If unable to reach this goal within 24 hours, consider the addition of parenteral nutrition.

With appropriate medical care, most sick neonates will begin to gain weight when provided 10% of its body weight in milk daily (Table 2). The healthy term foal ingests approximately 15% of its body weight in milk daily, this increases to 20% to 30% over the first few weeks of life. As a foal becomes stronger and more active, the amount fed should be gradually increased to normal volumes. The failure to gain weight can be a sign of uncontrolled illness, such as a focus of infection, or be the result of insufficient nutritional support.

### **Enteral feeds**

Mare's milk or colostrum is ideal enteral feed. The mare's milk contains approximately 500 to 600 kcal/L, whereas colostrum contains approximately 1000 kcal/L. In contrast

**Table 2**  
Milk intake volumes for a 50-kg foal

Body Weight in Milk/d (%)	L/d	Feedings (mL/2 h)
5	2.5	208
10	5.0	420
15	7.5	630



to milk, mare's milk replacer is high in potassium and low in sodium and chloride. When using a milk replacer, it is important to provide a fresh-water source to avoid the risk of hypernatremia caused by the excess salt ingestion. A milk replacer designed for foals is ideal to ensure adequate protein, fat, and carbohydrates. Most of the ingredients should be milk based rather than plant based.

### **FPT**

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A complete failure is immunoglobulin G (IgG) less than 400.

A partial failure is IgG greater than 400 and less than 800.

Causes of FPT include

- A lack of colostral antibody in the first milk can be caused by loss (dripping) before foaling or inadequate production (primiparous and aged, debilitated mares at greater risk).
- Failure to ingest colostrum may be caused by the foal's inability to stand and nurse (caused by illness or musculoskeletal problems) during the critical 24-hour period.
- Failure to absorb colostrum may be caused by delayed ingestion (loss of specialized enterocytes) or because of prematurity/dysmaturity/in utero illness (lack of development of specialized enterocytes before birth).

Treatment

- At less than 24 hours of age, feed good-quality colostrum (good-quality colostrum [high in IgG] specific gravity  $\geq 1.060$  [using a colostrometer] or 23% refractive index [using a sugar refractometer]).
- At greater than 24 hours of age, perform a plasma transfusion. The rule of thumb is that approximately 1 L of plasma will increase the IgG to 200 mg/dL in a healthy foal and 100 mg/dL in a septic foal.
- If plasma is not an option, treat the foal with broad-spectrum antimicrobials.

### **IV Fluid Support of the Foal**

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Signs of poor perfusion and hypovolemia in the sick neonate may be caused by decreased fluid volume, inadequate cardiac function (poor contractility), or vascular changes (vasoactive shock), which can result from prolonged hypoxia, hypoglycemia, or sepsis. The placement of a jugular catheter and fluid boluses of 10 to 20 mL/kg of an isotonic crystalloid over a 20-minute period are recommended. Glucose-containing fluids should never be used as bolus fluids, as hyperglycemia (and potentially rebound hypoglycemia) will result. The reevaluation of the foal's cardiovascular status should be performed after each bolus. If no improvement is noted, a second (or third) bolus may be given until the maximum central venous pressure (CVP) is reached (8–10 cm of water). It is difficult to measure CVP in a field situation; consequently, a maximum of 3 fluid boluses is usually recommended to avoid fluid overload. Foals that fail to respond are likely to have cardiac or vascular dysfunction and require more intensive medical support including vasopressor therapy. Such cases are best referred to a neonatal intensive care facility.

### **Maintenance fluid therapy**

The neonatal foal cannot be managed as a small version of the adult horse when calculating fluid therapy. The foal's kidneys are less effective in excreting sodium and water, and its vascular permeability is increased compared with the adult. Further, the septic foal or foal with ischemic injury may have abnormal responses to fluid

fluxes, such as the syndrome of inappropriate antidiuretic hormone secretion resulting in further inability to handle excess fluid infusions. Overhydration can result in edema, vascular volume expansion, and excess weight gain. Edema affects oxygen delivery and can exacerbate existing organ dysfunction.

In human and many equine neonatal units, the approach to fluid therapy has shifted to one of fluid and sodium restriction. Fluid therapy is calculated based on body mass and surface area (Table 3). Sodium intake is restricted to approximately 3 mg/kg/d to attempt to avoid sodium excess. Continuous rate infusions of 5% dextrose with supplemental electrolytes are best used. Remember to include medications when calculating sodium intake.

For example, the following is a calculation for a 47-kg foal:

$$10 \text{ kg} \times 100 \text{ mL/kg/d} = 1000 \text{ mL}$$

$$10 \text{ kg} \times 50 \text{ mL/kg/d} = 500 \text{ mL}$$

$$27 \text{ kg} \times 25 \text{ mL/kg/d} = 675 \text{ mL}$$

$$= 2175 \text{ mL/d or } \sim 90 \text{ mL/h}$$

Using this fluid-restricted plan, urine specific gravity should not be diluted as seen with a healthy foal on a milk diet. If the foal is being supplemented with enteral feeding, the fluid calculations should be adjusted. It is important to adjust this rate if increased losses are present.

### **Supportive Care**

Pressure sores may develop if not kept well bedded, cleaned, and repositioned frequently. Corneal ulcers can result from decreased tear production or trauma caused by accumulation of bedding/debris in the eye. Foals lying in lateral recumbency for prolonged periods will develop atelectasis of the dependant lung. Atelectasis will exacerbate the existing lung disease and affect oxygen delivery. The  $\text{PaO}_2$  can decrease by as much as 15 mm Hg when switching a foal from sternal to lateral recumbency; therefore, it is important to try to keep foals in sternal recumbency to maintain lung volume, for ventilation/perfusion matching, and to maximize oxygen delivery to the tissues.

### **Respiratory Support**

Physical examination findings of respiratory distress indicate the need for further assessment of pulmonary function. Oxygen insufflations should be instituted until further monitoring can be performed. A soft, rubber Foley catheter can be slid up the ventral meatus (to the level of the medial canthus) to facilitate oxygen supplementation; 5 L/min of oxygen insufflations is recommended. In foals exhibiting severe hypoxemia, bilateral insufflations may be necessary (5 L in each nostril). Humidification is

**Table 3**  
Maintenance fluid therapy for the neonatal foal

First 10 kg body weight	100 mL/kg/d
Second 10 kg body weight	50 mL/kg/d
Additional kg body weight	25 mL/kg/d

recommended for long-term oxygen insufflations but is not necessary in the short-term while stabilizing a foal for transport.

#### Important points

- Weak, recumbent foals are at risk for hypoglycemia, which may exacerbate the underlying problems.
- Supportive care is critical to the long-term recovery.
- Do not enterally feed a hypothermic hypotensive foal. Carefully monitor the gastrointestinal response when starting enteral nutrition in the critically ill neonatal foal.

### OTHER COMMON DISORDERS OF THE NEONATE

#### *Neonatal Isoerythrolysis*

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Clinical signs of neonatal isoerythrolysis may include lethargy, tachycardia, tachypnea, pale mucous membranes with/without icterus. The onset of clinical signs varies from 1 to 7 days of age and depends on the rapidity and severity of hemolysis. Mule foals are overrepresented.

#### Treatment

- Blood transfusion is recommended in severely anemic foals or for those foals that are so weak that they are unwilling to nurse. The goal of transfusion is to provide oxygen-carrying capacity until a regenerative response occurs. Crossmatching is ideal; if it is unavailable, transfusion from an Aa Qa blood type negative gelding is recommended.
- Dexamethasone 0.08 mg/kg IV or IM in peracute cases may decrease hemolysis.
- Supportive care includes the following:
  - Nasogastric intubation and feeding if foal is too weak to nurse
  - Broad-spectrum antimicrobials

#### *Disorders of the Lungs*

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##### *Pneumonia*

The most common cause of respiratory compromise/distress is bacterial pneumonia. This condition may result from aspiration or bacteremia. Radiographs reveal the presence of consolidation, most commonly in the cranioventral lung fields, though a diffuse pattern may be seen with hematogenous pneumonia. Thoracic ultrasound examination typically reveals the presence of pleural irregularities and consolidating lesions. Pleural effusion is rare with neonatal pneumonia. Foals with viral pneumonia usually present with severe dyspnea and high fevers and have a very characteristic ultrasound pattern consisting of wide-spread, diffuse pleural irregularities or comet tails and small consolidated (fluid density) lesions.

A tracheal wash with bacterial culture and sensitivity is recommended to ensure appropriate antimicrobial therapy. However, a transtracheal wash should not be performed in foals with signs of respiratory distress, as it may lead to further deterioration and collapse.

**Treatment** Broad-spectrum antimicrobial therapy is indicated unless culture and sensitivity is available (see “Specific Treatment of Sepsis”). Foals should be maintained in sternal recumbency to maximize oxygenation and delivery. Foals that are in respiratory distress may be unwilling to nurse; in such cases, placement of an indwelling nasogastric tube is recommended to ensure nutritional support. Nasal oxygen supplementation is recommended if  $\text{PaO}_2$  decreases to less than 65 mm Hg (approximately equal to an arterial oxygen saturation less than 90%). Bilateral nasal insufflations at 5 L/min can

increase inspired oxygen content as high as 49%.<sup>7</sup> A humidifier should be attached for long-term oxygen insufflations. Ventilatory support is recommended if  $P_{aCO_2}$  is 65 mm Hg or greater. Caffeine is not recommended, as ventilatory failure is usually caused by respiratory muscle failure not neurologic dysfunction. Coupage of the thorax multiple times a day may help to break up and clear the debris within the lung. Keeping the foal well hydrated will also help in the clearance of inspissated material.

### ***Apneustic breathing***

The most common cause of apneustic or an irregular breathing pattern is perinatal asphyxia syndrome. Lack of oxygen delivery results in damage and depression of the respiratory center in the medulla. Other potential causes include hypoglycemia, hypothermia, and other forms of CNS disease (eg, meningitis, trauma). Botulism may also result in a weak, abnormal breathing pattern and hypercapnia but is frequently associated with muscle fasciculations and other signs of muscle weakness.

#### **Treatment**

- Treat the underlying disorder (hypoglycemia, hypothermia).
- For caffeine, the recommended loading dose is 10 mg/kg by mouth followed by 2.5 to 3.0 mg/kg once daily. The loading dose may result in significant stimulation in the neonate. An increase in the respiratory rate and effort indicates pulmonary or cardiac dysfunction.
- If hypoventilation persists, ventilatory support is recommended.

### ***Disorders of the Umbilicus and Urinary Tract***

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The umbilical stalk will typically detach once the foal or mare stands. If bleeding occurs, a temporary clamp or umbilical tape may be applied. A 1:4 chlorhexidine solution or a 2% iodine solution is recommended as the umbilical dips. Stronger iodine solutions should be avoided, as they can scald the skin, cause edema, and cause early separation of the external umbilical stalk, which may predispose to the development of a patent urachus.

#### ***Patent urachus***

Patent urachus may be present at birth or develop in foals that are recumbent for prolonged periods.

#### **Treatment**

- Broad-spectrum antimicrobials
- Phenazopyridine if excessive straining or cystitis/urachitis
- If severe or associated with infection, surgical removal may be indicated

#### ***Omphalophlebitis***

Infections of the structures of the umbilical cord are relatively common in sick neonates and can result in secondary dissemination to other organ systems, including the lungs, joints, and bones. An ultrasound examination of the umbilical structures evaluating the size and echogenicity should be performed in any sick neonate.

#### **Treatment**

- Surgical removal
- Medical management with broad-spectrum or culture-directed antimicrobials

#### ***Dysuria***

Foals that are weak and recumbent may not have the normal neurologic trigger to urinate when their bladder is distended. It is important to monitor urination and/or bladder size (via ultrasound exam) in all sick, recumbent foals to prevent this

complication. In suspect cases a urinary catheter and closed collection system can be placed to prevent distension and rupture.

### **Urinary tract rupture and uroabdomen**

Urinary bladder rupture can occur in utero, during foaling, or after birth in the neurologically impaired neonate. Clinical signs become apparent as urine accumulates, azotemia develops, and electrolyte derangements occur.

**Clinical signs** Clinical signs will depend on the location of the rupture, its duration, and the volume of urine accumulated. If the rupture of a ureter occurs, urine may accumulate in the retroperitoneal space but not in the peritoneal space.

Common findings include

- Pendulous, fluid-filled abdomen
- Tachycardia, tachypnea, lethargy, and weakness
- Decreased appetite and decreased suckle
- Respiratory compromise worsens as intra-abdominal pressure increases
- Pleural effusion may develop; urine may accumulate in the scrotum or ventral abdomen
- Straining to urinate
- With/without signs of sepsis

### **Diagnosis**

- Ultrasound examination shows evidence of fluid within the peritoneal space.
- Peritoneal fluid creatinine is 2-fold or greater of blood creatinine.
- Serum chemistry analysis shows azotemia (creatinine usually elevated to a greater degree than blood urea nitrogen) hyperkalemia, +/- hyponatremia, hypochloremia, hypoxemia, and acidosis. (Electrolyte values will vary depending on the severity and duration of rupture and whether the foal has received prior fluid therapy support.)<sup>8</sup>

**Treatment** The initial treatment is aimed at stabilizing patients before referral for surgical correction. Although resolution with medical therapy has been reported, most cases require surgical repair of the tear. Hyperkalemia can be life threatening when it is 5.5 mEq/L or greater and must be addressed before surgical correction; this can best be achieved by the following:

- Drainage of the uroabdomen can be performed using a 14F or 16F Foley catheter, IV catheter, or chest tube. The catheter can be sutured in place to allow continued or repetitive drainage; however, it is important to know that they often become obstructed by fibrin or omentum necessitating their removal.
- Perform IV crystalloid therapy to normalize electrolyte derangements. The replacement volume should be at least equal to or exceed the volume of urine drained to prevent acute hypotension as abdominal pressure decreases.
- Sodium bicarbonate may be added to the saline infusion to lower serum potassium levels, as it results in intracellular movement of potassium. Calcium gluconate added to saline (separately from bicarbonate) will reduce the impact of hyperkalemia on cardiac function. An insulin and dextrose infusion will also help lower potassium levels, as glucose uptake is coupled to potassium uptake.
- Pleural effusion will generally resolve after abdominal drainage, though it may take several hours to days. Because pleural effusion can cause pulmonary atelectasis and affect respiratory function, it is recommended to delay anesthesia and surgical repair until the effusion has resolved.
- Broad-spectrum antimicrobial therapy is recommended postoperatively.

## ***Disorders of the Musculoskeletal System***

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### ***Flexor tendon laxity***

Mild flexor tendon laxity will usually resolve with time and controlled exercise. Foals that are walking on their fetlocks or unable to stand because of severe laxity may require the placement of shoes with heel extensions and controlled, limited exercise until the laxity resolves. A light wrap may be applied to the fetlock to prevent abrasions. However, thicker support wraps should be avoided, as these will exacerbate the tendon laxity.

Flexural deformities (contracted tendons): Flexural deformities are thought to arise because of uterine malposition, ingestion of toxic plants, nutritional deficiencies, infections, and genetic defects.

### **Treatment**

- The goal of therapy is to achieve tendon loading and stretch.
- Mild: The treatment includes physical therapy, support wraps, or splints and controlled exercise.
- Moderate: The treatment includes bandages and splints, with a daily bandage change and physical therapy.
- Severe: Surgical release may be necessary in severe cases
- The stretching of these tendons is painful, and analgesia and gastric ulcer prophylaxis is recommended.
- Oxytetracycline (2 to 3 g IV once) has been shown to result in a temporary increase in joint extension.

Avoid oxytetracycline in sick, dehydrated, hypovolemic foals or in those with signs of renal compromise.

Limit exercise, as foals can get tired and stumble increasing the risk of injury to the extensor tendons.

### ***Septic arthritis/septic osteomyelitis***

**Clinical signs** The earliest sign of septic arthritis or osteomyelitis is often lameness. Warmth, swelling, and pain may be palpated over the affected area, though these signs may be less obvious with osteomyelitis. Complete blood counts typically reveal an elevated white blood cell count (WBC) and fibrinogen. Foals may or may not have a fever. Regular palpation and assessment of gait is important, as early detection and treatment are factors in the prognosis for athleticism.

### **Diagnosis septic arthritis/osteomyelitis**

- Synovial fluid cytology: A WBC greater than 30,000/ $\mu$ L, greater than 90% neutrophils, and total solids greater than 2.5 g/dL are consistent with sepsis. The absence of bacteria does not rule out the septic process.
- The presence of hyperfibrinogenemia and leukocytosis.
- Synovial fluid culture: \*A negative culture does not rule out septic process.
- Radiographs of the affected region are recommended to evaluate for septic physisitis or osteomyelitis. \*Radiographs of the unaffected limb should be taken for comparison, as the normal foal's physes can appear irregular.

### **Treatment septic arthritis**

- Lavage the affected joint with 1 to 2 L of an isotonic crystalloid solution (pH ~ 7.0). Every other day, lavage is recommended until cytology and clinical signs improve. In cases with excess fibrin and purulent debris, arthroscopy or arthrotomy may be necessary to more effectively debride the joint.

- Broad-spectrum antimicrobial therapy: Treatment for 3 to 6 weeks may be necessary. If an arthrotomy is performed, antimicrobial therapy should be maintained until the joint seals.
- Nonsteroidal antiinflammatory therapy: Treatment is recommended until inflammatory process has improved.
- Prophylactic antiulcer therapy is recommended in foals with painful conditions and those receiving nonsteroidal antiinflammatory therapy.

#### **Treatment septic osteomyelitis**

- Medical therapy is similar to that for septic arthritis.
- Aspiration and culture of the infected bone is recommended for more directed therapy.
- Regional limb perfusion with an aminoglycoside is useful to achieve high concentrations of antimicrobials at the site of infection. Use approximately one-third of the total daily dose, and decrease the daily IV dose accordingly to prevent overdosing.
- Surgical debridement may be performed.

#### **Rib fractures**

Rib fractures can occur without any history of dystocia or trauma. The signs of rib fractures may include tachypnea (often shallow rapid breathing), tachycardia, chest wall edema +/- crepitus at the fracture site, and clinical signs of hemorrhage. Auscultation over the fracture site may reveal a click that occurs with the respiratory cycle. Most rib fractures occur near the costochondral junction and are best visualized by ultrasound examination using a linear probe placed parallel with and on top of the rib.

#### **Treatment**

- Limited exercise/stall rest for 3 to 4 weeks
- Surgical stabilization if unstable or displaced fragments

## **DISORDERS OF THE GASTROINTESTINAL TRACT**

### ***Colic***

Colic signs in a foal can be subtle; foals may appear agitated, unable to settle, and have an increased heart and respiratory rate. They may flag their tail and stomp a foot. With severe pain, signs become more obvious. The evaluation of the colicky foal should include a physical examination, careful history, digital rectal examination, passage of a nasogastric tube, and an abdominal ultrasound. The presence of gastric reflux suggests a small intestinal disorder.

#### **Common causes of neonatal colic**

Meconium impaction

Atresia coli

Ileoceocolic aganglionosis

Enterocolitis

Gastric ulcers

Bowel obstruction: intussusceptions, volvulus, strangulation, incarceration

Peritonitis: usually secondary to ruptured gastric ulcers, umbilical abscessation, or uroabdomen

### **Diagnostic aids for colic**

- Nasogastric intubation should be performed to check for reflux and gastric distension secondary to outflow obstruction.
- Abdominal radiographs with or without contrast agents can be helpful to assess the location and type of distension and to evaluate for impaction or atresia.
- Abdominocentesis and cytologic evaluation should be performed to diagnose peritonitis, ruptured bladder, or bowel.

### **Meconium impaction**

Meconium impaction is the most common cause of colic in the neonatal foal. The clinical signs include decreased nursing, straining to defecate with an arched back, flagging of the tail, and a lack of meconium production. A digital examination may reveal hard fecal balls in the rectum. With complete obstruction, abdominal distension may develop. Severe distension can result in respiratory compromise. Radiographs or ultrasound may reveal fecal material in the distal colon or rectum. A barium enema can be performed for further evaluation and to attempt to rule out focal atresia of the intestinal tract.

**Treatment** A warm water enema (using ~ 150 mL of warm water) can be performed in the standing foal using gentle restraint. A polyurethane stallion urinary catheter or an enema tube can be used. The tip of the tube should be lubricated and gently inserted until resistance is felt. Water can be infused by gravity while gently manipulating the tube to try to advance around the impaction. The addition of a small amount of dish soap may help resolve the impaction; however, repeated infusion of soap can cause further irritation of the mucosa and may ultimately worsen the foal's discomfort.

An acetylcysteine retention enema is recommended for meconium impactions that do not resolve with a warm water enema. Sedation is recommended (diazepam 0.1 mg/kg IV) to keep the foal quite while the enema is infused. Once the foal is recumbent, a 30-cm<sup>3</sup> balloon 30F lubricated Foley catheter is placed, and the balloon is distended with saline. Elevating the foal's hind end will aid the gravity flow. After infusion, the catheter is clamped to retain the enema for a minimum of 15 minutes (ideal time is 30–45 minutes).

#### **N-acetyl-L-cysteine retention enema**

If using Mucomyst (20% acetylcysteine): Add 40 mL of solution to 160 mL of warm water

If using powdered N-acetyl-L-cysteine (NALC): Add 8 g of powdered NALC and 1.5 tbsp of baking soda to 200 mL of water.

**Oral laxatives** The use of oral laxatives, such as mineral oil, can be helpful, particularly with proximal impactions. Mineral oil (~ 60 mL) can be given by nasogastric tube. Mineral oil should never be given by syringe feeding, as aspiration will result. Detergents, such as dioctyl sodium sulfosuccinate (DSS), or castor oil are not recommended because they cause irritation to the intestinal mucosa and can result in further colic and diarrhea.

**IV fluids** With protracted impactions, IV fluids may be necessary to maintain hydration and aid in treatment.

### **Analgesics/prokinetics**

- Nonsteroidal antiinflammatory drugs are potent analgesics. If they are used, it is critical to ensure adequate hydration to prevent renal toxicity and to institute gastroprotectant therapy to attempt to minimize the side effects.



- IM butorphanol (0.1 mg/kg) may provide analgesia. Combining it with diazepam (5–10 mg/50 kg foal) may provide sedation, allowing time for a retention enema or other treatment to take effect.
- Neostigmine (0.005–0.01 mg/kg IM or subcutaneous) has been effective in resolving meconium impactions and allowing passage of gas.

### ***Atresia coli***

Atresia can be a difficult disorder to definitively diagnose. The passage of meconium does not rule out an atresia coli. A barium enema followed by serial radiographic images may identify an area that is constricted or atretic. A definitive diagnosis may require surgical exploration. Reports of successful resection and anastomosis of atresia coli exist.<sup>9</sup>

### ***Ileocecolic aganglionosis (overo lethal white syndrome)***

Ileocecolic aganglionosis, or lethal white syndrome, is a genetic defect recognized in homozygous, overo, paint foals. Affected foals are usually completely white or have only small spots of color on their bodies. An endothelin receptor B mutation results in complete aganglionosis of the myenteric and submucosal ganglia of the intestinal tract.<sup>10</sup> Foals appear normal at birth but develop signs of colic, profound ileus, and intestinal distension. The diagnosis is made based on clinical signs coupled with ultrasonographic evidence of profound ileus and distension in white, homozygous, overo, paint foals. There is no treatment of this disorder, and euthanasia is recommended. A genetic test is available to identify heterozygous individuals.

### ***Enterocolitis/diarrhea***

The causes of enterocolitis and diarrhea include bacterial, viral, protozoal, nutritional, and ischemic (necrotizing enterocolitis) (Table 4). Bacterial causes include *Clostridium perfringens* type A and C, *Clostridium difficile*, salmonellosis, and, less commonly, *Escherichia coli*, *Bacteroides fragilis*, and *Aeromonas hydrophila*. Rotavirus is the most common viral cause, though coronavirus, adenovirus, and parvovirus have been isolated from foals. *Cryptosporidium* species have been isolated from foals with diarrhea but can also be isolated from normal foals. Nutritional causes include overfeeding, lactose intolerance, and sudden diet changes.

Because blood flow is shunted to vital organs during hypoxic/ischemic periods, foals with PAS or septic shock often have ischemic damage to their gastrointestinal tract. Clinical signs of ischemic enteropathy can range from mild colic and intolerance of enteral feeding to profound ileus, sepsis, and bloody diarrhea. Affected foals may present with other organ dysfunction. Sepsis may develop secondarily to bacterial translocation across the damaged gastrointestinal mucosal barrier.

**Table 4**

**Fecal characteristics and diagnostic assays for common diarrheal causes in foals**

<b>Cause</b>	<b>Characteristics</b>	<b>Diagnosis</b>
<i>Clostridium perfringens</i>	May be bloody or foul smelling	Culture and toxin assay
<i>Clostridium difficile</i>	May be bloody and foul smelling	Culture and toxin assay
<i>Salmonella</i> spp	May be bloody or foul smelling	Fecal culture
<i>Rotavirus</i>	Watery	ELISA assay for viral antigens
Lactose intolerance	Watery	Response to lactase supplementation
Overfeeding	Watery	Response to change in feeding regimen
Foal heat diarrhea	Transient, no other signs of illness	Timing with mare's heat cycle

When presented with a foal with enterocolitis and diarrhea, it is important to institute symptomatic treatment regardless of the cause. Foals with enterocolitis often do not tolerate oral feeding. Injured enterocytes may be unable to digest nutrients resulting in bacterial overgrowth in the large bowel, gas production, and further colic. Auscultation and ultrasound examination should be performed to assess motility. A nasogastric tube should be placed to check for reflux and gastric distension. Decreasing or temporarily stopping enteral feeding may be necessary, as feeding may exacerbate clinical signs. Symptomatic therapy includes

- IV fluid support (see section on IV fluid therapy)
- Withhold feeding until motility returns; gradual reintroduction to enteral feeding (see nutrition section)
- Broad-spectrum antimicrobial therapy: for diarrhea, consider addition of metronidazole 15 to 25 mg/kg every 6 hours
- Parenteral nutritional support (see section on nutritional support)
- Antiinflammatory therapy
- Gastric ulcer prophylaxis
- Lactase supplementation when reinstating feeding

#### ***Nutritional causes of colic and diarrhea***

**Overfeeding** Foals that quickly ingest large volumes of milk or milk replacer can develop gastric distension, colic, and diarrhea. Foals fed large volumes infrequently will consume the large volume rapidly; incomplete digestion results in bacterial overgrowth in the large intestine, resulting in colic, gas production, and diarrhea. Treatment is to decrease the volume ingested at each feeding. Feeding milk cold may help, as it is less palatable and may slow the foal down.

**Lactose intolerance** The most common cause of lactose intolerance is injury to and loss of enzyme-producing enterocytes. Supplementation with the enzyme lactase in each feeding is helpful in resolving clinical signs. Over time, as the intestinal tract heals, this syndrome typically resolves.

## **SEIZURES**

### ***Causes of Seizures***

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- Perinatal asphyxia syndrome
- Meningitis
- Trauma
- Idiopathic epilepsy of Arabian foals
- Congenital abnormalities: juvenile epilepsy, lavender foal syndrome, and glycogen branching enzyme deficiency

### ***Clinical Signs***

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Seizures can be difficult to detect, as clinical signs may be subtle, including repetitive tremors, abnormal eye movements, hyperesthesia, excessive stretching, or extensor muscle tone when recumbent. Because secondary injury from excess neurotransmitter and calcium release can occur, it is critical to control seizures.

### ***Treatment Options***

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- Diazepam: Administer diazepam 0.1 to 0.44 mg/kg IV bolus to effect (5–20 mg per 50-kg foal). Diazepam has a relatively short half-life, and the effect may be short lived, requiring additional therapy. Monitor for respiratory depression.

- Midazolam: Administer midazolam 0.04 to 0.1 mg/kg IV slowly (2–5 mg per 50-kg foal). Midazolam may be used as a continuous-rate infusion for long-term control at 2 to 5 mg/h. Monitor for respiratory depression.
- Phenobarbital: For persistent seizures uncontrolled by diazepam or midazolam, use phenobarbital 2 to 3 mg/kg IV.
- Propofol: Use propofol in cases that are refractory to the aforementioned medications at 4 mg/kg IV continuous-rate infusion.

For long-term control with refractory seizures

- Potassium bromide: Administer 60 to 90 mg/kg orally once daily.
- Magnesium infusion: Administer a loading dose of 50 mg/kg IV over 1 hour and a maintenance dosage of 25 mg/kg/h.

### **Additional Support for Seizures**

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If cerebral edema is present

- Administer mannitol 0.5 to 1.0 g/kg of 20% solution IV over 15 minutes; the dose may be repeated.
- Bandage the legs and pad the head to prevent injury. If the head protector is not available, wrap a protective bandage over the eyes (after placing lubricant) to avoid eye abrasions and corneal ulceration.
- Keep the head elevated to prevent increased intracranial pressure and exacerbation of cerebral edema.
- Avoid overhydration.

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