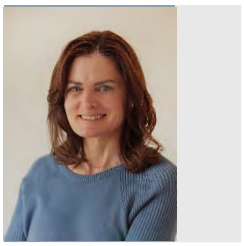


What is normal in the newborn foal?

Siobhan McAuliffe

History
The first part of any neonatal examination is a thorough history. This is often acquired orally at the farm, over the phone or in the form of a foaling record chart.

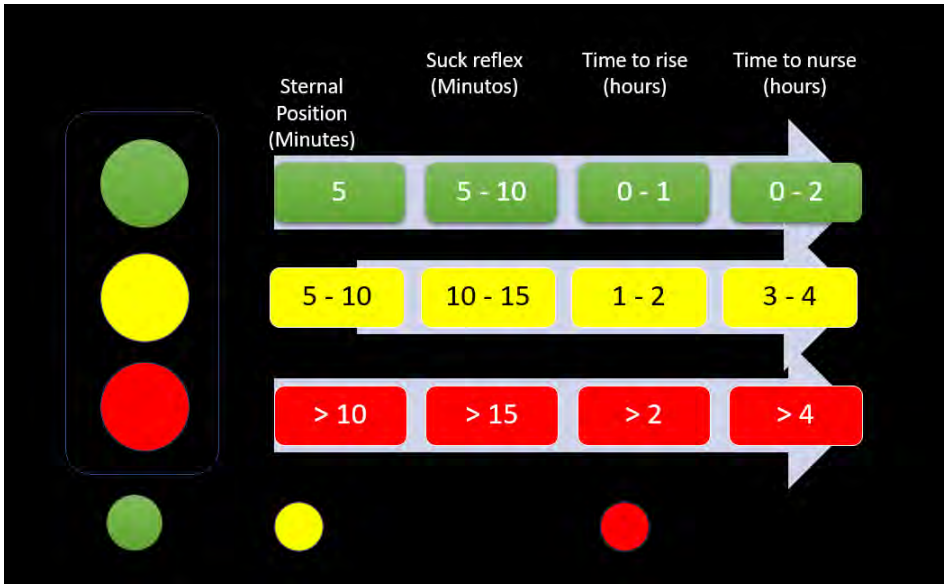
Important details include:

- Time of foaling,
- Duration of foaling,
- Any difficulties during foaling.
- Was the foaling observed, if not where was the foal in relation to the mare when it was found, alone or nearby. Was the foal wet indicating a recent birth or was it dry?
- Has the foal met normal post-delivery milestones?
- Has the foal nursed? Did the mare drip milk before foaling?
- Was the colostrum quality tested?

The general history of the mare is also important:

- Did she carry to term?
- Is she multiparous or is she a maiden foaler?
- Has she had problems with previous pregnancies or foals?
- History or evidence of placentitis (examination of the placenta).
- Vaccination status of the mare.
- Was the placenta passed in an appropriate time frame (<3hours)

Normal developmental milestones and timeframes



Clinical examination of the newborn foal should always begin at a distance, with observation of behaviour, respiratory rate and musculoskeletal abnormalities. Normal foals will nurse for short periods, multiple times

each hour and will follow the mare closely. Changes in bahaviour, including loss of affinity for the dam or abnormal nursing behaviour can be a sign of Hypoxic-Ischaemic encephalopathy (HIE) or sepsis. Respiratory rate and character should be assessed both when the foal is resting and when ambulating around the stall. Signs of prematurity or dysmaturity should be evaluated including small size for breed and gestational age, a domed forehead, floppy ears and a silky haircoat. Limb laxity can also be a sign of prematurity. Many congenital abnormalities are also obvious from a distance while others may be suspected and required a closer examination.

Clinical examination should be systematic and include all body systems.

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 auscultated. 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, and its heart rate should increase after a few minutes. 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.

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 faeces, within 12 to 24 hours. Meconium is dark brown to tan and may be hard or pasty. Subsequent milk faeces are yellow tan and typically softer in consistency.

Neonates lack a menace response, as this is a learned behaviour 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 behaviour should be directed toward maintaining close contact with its dam.

Foals can be bradycardic at birth; the heart rate should increase relatively quickly to normal values. Persistent bradycardia can be caused by hypoxia, hypoglycaemia, 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 hyperglycaemia 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.[1] 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, anaemia, fever, and excitement. If pain, fever, anaemia, and excitement are ruled out, fluid therapy is indicated to attempt to correct hypovolemia (see section on IV fluid therapy).

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 paradoxic 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, hypoglycaemic, or have underlying respiratory disease may develop respiratory muscle exhaustion, worsening atelectasis, and pulmonary function.

During parturition, mucous membranes may appear grey or cyanotic; this should rapidly resolve once the foal is delivered. Pale mucous membranes can be an indicator of anaemia or hypovolemic shock. Icteric

mucous membranes can indicate haemolysis (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 haemoglobin per decilitre of blood, anaemic individuals may not be cyanotic even in the presence of severe hypoxia.[2] Petechia on the mucous membranes or ears can be an indicator of septicaemia 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 haemorrhages 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.

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Ultrasonography of the neonate

Siobhan McAuliffe

Ultrasonography has become a routine and important part of medical examination of the foal. The small size of the foal, lack of muscle mass and body fat facilitate the exam. A variety of ultrasound machines and transducers are available. For the detection of most disorders in neonates, extremely expensive equipment is not required, and a linear rectal probe of variable frequency (5-13MHz) is sufficient for most exams. Although we often separate disorders of the thorax and abdomen into distinct categories, both body cavities are usually evaluated during the same ultrasound examination. The ultrasound examination is performed in a cranial-to-caudal manner—passing the ultrasound probe along each intercostal space, scanning from dorsal to ventral, and then sweeping the caudal and ventral abdomen behind and beneath the ribs.

Thorax
Clipping of the thorax is generally not required for routine examinations but may be needed where additional detail and a clearer image is needed.

Disorders of the thorax in the neonatal foal include rib fractures, pneumonia, and effusions (septic, hemorrhagic, or other). Rib fractures are common in hospitalized neonatal foals, and ultrasound is considered a more accurate method of detection compared with radiography and physical examination. Fractures are most often located with 3cm of the costochondral junction and more commonly involve the first few ribs behind the elbow. Fractures may be non-displaced to severely displaced and are categorised by the number of mm separating the fracture fragments. Fractures may displace over time as the foal becomes more mobile with the distal segment usually moving medially. There may be fluid or hemorrhage in the soft tissues surrounding the fracture ends. Injury to the underlying pulmonary parenchyma can vary from mild bruising with a few echogenic "comet tails" to progressively more involvement with parenchymal consolidation and occasional hemothorax or pneumothorax. Serial evaluation of the degree of displacement is recommended to determine if there is an indication to consider surgical stabilization versus conservative management of restricted mobility in the stall. Ultrasound can also be useful in monitoring the fracture healing process—determining when there is sufficient callus formation and fracture stability to allow more exercise. With fracture of more caudal ribs, there may be injury to the diaphragm and possible diaphragmatic hernia with intestinal structures within the pleural space. In cases where the intestine lies between the lung lobes and cannot be directly visualized there is often and increase in free pleural fluid. This combined with an abnormal respiratory rate of pattern would indicate the additional use of radiography.

Ultrasound has become a routine tool in the evaluation of pneumonia in the foal. Pneumonia can be a primary anatomic site of sepsis in the neonate. Patterns of changes on the ultrasound image can be helpful in predicting the type of lung injury present. Scattered echogenic "comet tails" may be present in the early stages of a variety of bacterial pneumonias, with ventral consolidation being evident with further progression or more serious pneumonia. Broad-based or diffuse echogenic shadowing is more consistent with interstitial lung disease (pulmonary edema or interstitial pneumonia) and suggests a more serious disease process. Serial ultrasonographic examination of the lung is useful in evaluating the progression of disease and can be a component for evaluating response to medical therapy.

Abdominal ultrasound
Ultrasound examination of the abdomen of foals is often used in the evaluation of foals with signs of colic and can be useful in differentiating causes of abdominal distention in foals with and without colic signs. Ultrasound is useful in extremely useful in the evaluation of disorders of the umbilical structures and ab-

normalities of the abdominal wall surrounding the umbilicus and the inguinal area (e.g., traumatic injury acquired during delivery and congenital defects).

The approach to the ultrasound examination of the foal with signs of acute abdominal pain is similar to that used for the adult equine patient. There are some special circumstances and lesions that may be unique to the younger foal that must be evaluated. The use of a high-frequency (5 to 7.5 mHz or higher) probe—whether linear or microconvex—is sufficient for imaging much of the abdominal cavity in the young foal with good resolution of structures. This can be performed with the foal standing or in a recumbent position. The ultrasound examination should proceed as with the adult patient by evaluating the ventral thorax and abdomen by passing the ultrasound probe dorsal to ventral along each intercostal space beginning just caudal to the triceps muscle on each side and progressing in a cranial to caudal fashion to the thigh. The exam is completed by then sweeping the ventral aspect of the abdomen to evaluate the umbilical structures and the urinary bladder.

Gastric distension can be evaluated in the foal in a manner similar to that seen in the adult. Causes of gastric distension may include ileus with or without enteritis or small intestinal strangulation obstruction. Small intestinal obstructive disorders such as volvulus or entrapment in scrotal hernias will appear similar to that seen in the adult patient—with profoundly fluid-distended segments of small intestine occasionally with sedimentation of particulate material to the ventral or dependent aspect. With the hernia, small intestinal segments may also be evident within the vaginal tunic. Small intestinal disorders including enteritis can be easily identified in the foal. The ultrasound finding of fluid distension of the small intestinal lumen along with variable motility and variable thickening (2 to 3 mm) of the small intestinal wall concurrent with fever and leucopenia is supportive of the clinical diagnosis of enteritis. The small intestinal wall may often be less distinct with enteritis due to inflammatory cell infiltrates and variable edema of the wall.

Colic associated with small intestinal obstruction from intussusceptions occur more commonly in young foals, often secondary to enteritis or dysmotility secondary to birth asphyxia. Serial ultrasound examinations may be necessary to identify the intussusception, which is classically described as a "target lesion" with the concentric rings of the intussuscepted intestinal wall. Occasionally, the acute onset of rotaviral enteritis will result in variable signs of colic and inappetence, sometimes before the appearance of diarrhea. Abdominal ultrasound can be useful in identifying liquid contents of both the small and large intestines, which may be indicative of impending diarrhea.

The urogenital system of the foal represents a special system within the abdominal cavity of the foal for ultrasound evaluation. Uroperitoneum secondary to rupture of the urinary bladder is the most common abnormality. Ultrasound imaging usually demonstrates variable volumes of variably echogenic to hypoechoic peritoneal effusion with free-floating intestinal organs. There are some instances in which the peritoneal fluid associated with uroperitoneum will appear quite echogenic and require differentiation from suppurative exudate associated with septic peritonitis by abdominocentesis. In cases of uroperitoneum associated with a ruptured urinary bladder, the bladder is usually collapsed and folded on itself. The actual rupture site is usually located on the dorsal aspect of the bladder, although it may not be easily identified on the ultrasound image. It is important to note that ruptures of the urinary tract may occur at sites other than the urinary bladder, such as the urachus or ureters. Urachal ruptures often will have a periumbilical plaque of edema associated with subcutaneous leakage of urine. In cases of uroperitoneum associated with a ruptured urinary bladder, the bladder is usually collapsed and folded on itself. The actual rupture site is usually located on the dorsal aspect of the bladder, although it may not be easily identified on the ultrasound image. It is important to note that ruptures of the urinary tract may occur at sites other than the urinary bladder, such as the urachus or ureters.

Umbilical ultrasound frequently requires clipping of the area between the umbilicus and udder/prepuce. The thickness and content of both umbilical arteries and the umbilical vein should be determined. Infection of these are common and haematomas can also be identified.

In summary ultrasound imaging is a readily available diagnostic tool that is easily applied to evaluation of the young foal, both in the hospital setting and in the field.

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Field Approach To Treating The Sick Neonatal Foal

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Equine NICUs have greatly improved our knowledge and understanding of the normal and abnormal physiology of the equine neonate, resulting in an improvement in our ability to successfully treat the critically ill equine neonate and save lives. The success rate has increased tremendously since the early 1980s from a little over 50% to 80% or more for most facilities; some of best success has been in the treatment of 'dummy foals' that have a greater than 80% rate of survival to discharge in most hospitals. We have also been able to translate many treatments from the referral hospital to the field with good success for the practitioner. This lecture provides information on how to translate many specific and supportive treatments from the NICU to the field situation.

Neonatal Encephalopathy: The 'Dummy' Foal

Neonatal encephalopathy (NE) is one of the most common diseases of the equine neonate and is also described as dummy foal syndrome and neonatal maladjustment syndrome (NMS). A wide spectrum of clinical signs are associated with NE ranging from mild depression with loss of the suck reflex to grand mal seizure activity. The majority of affected foals are normal at birth but show signs of central nervous system (CNS) abnormalities within a few hours following birth although some will not show signs until 24 hours of age. NE is the most common clinical presentation and management of foals presenting with signs consistent with a diagnosis of NE requires complete examination of other body systems and provision of specific and supportive therapies of all affected body systems. Although PAS is most clinically obvious as NE, the gastrointestinal tract and kidneys are also frequently affected; complications associated with these systems should be anticipated, in addition to cardiovascular, respiratory, and endocrine disorders.

Pathophysiology:

The underlying pathophysiologic details of PAS and NE in the foal are unknown and likely multifactorial and equine neonatologists have long looked to human studies and models of the human disease for understanding of the syndrome in the equine neonate. NE is commonly associated with adverse peripartum events, including dystocia and premature placental separation, but a fair number of foals have no known peripartum period of hypoxia, suggesting that these foals result from unrecognized in utero hypoxia. Severe maternal illness may also result in foals born with PAS. There is increasing evidence that cytokinemia, resulting from placental infection or insult, is a major contributor to NE in infants, and probably foals, with incidence of NE increased with the presence of maternal fever, something veterinarians have suspected for the last decade or so.

Treatment:

Therapy for the various manifestations of PAS involves control of seizures, general cerebral support, correction of metabolic abnormalities, maintenance of normal arterial blood gas values, maintenance of tissue perfusion, maintenance of renal function, treatment of gastrointestinal dysfunction, prevention/recognition/early treatment of secondary infections and general supportive care.

It is important that seizures be controlled as cerebral oxygen consumption increases five-fold during seizures. Diazepam and midazolam can be used for emergency control of seizures. If seizures are not readily stopped with diazepam or midazolam, or more than two seizures are recognized, then diazepam should be replaced with a midazolam constant rate infusion (CRI).

Probably the most important therapeutic interventions are aimed at maintaining cerebral perfusion. Thiamine and vitamin C supplementation in the intravenous fluids can be administered to support metabolic processes. The author rarely uses DMSO, has not used it at for the last decade and has not recognized any change in outcome by discontinuing its use. GABAergic agonists (gabapentin) are being used by some practitioners in the managements of PAS/NE in foals, based on evidence showing neuroprotection when used in ischemia, both alone and in combination with NMDA antagonists like magnesium.

Foals suffering from PAS will also have frequent recurrent bouts of hypoxemia and occasional bouts of hypercapnia. INO_2 is generally needed in these cases both as a preventative therapy and as direct treatment, as the appearance of the abnormalities can be sporadic and unpredictable. Additional respiratory support, particularly in those foals with centrally mediated hypoventilation and periods of apnea or abnormal breathing patterns, include caffeine (per os or per rectum) or doxapram CRI and finally positive pressure ventilation.

Maintaining tissue perfusion and oxygen delivery to tissues is a cornerstone of therapy for PAS in order to avoid additional injury. Oxygen carrying capacity of the blood should be maintained; some foals will require transfusions to maintain a PCV > 20%. Adequate vascular volume is important, but care should be taken to avoid fluid or sodium overloading. Early evidence of fluid overload is subtle accumulation of ventral edema between the front legs and over the distal limbs. Perfusion is maintained by supporting cardiac output and blood pressure by judicious use of intravenous fluid support and inotrope/pressor support. We do not aim for any 'magic' systolic, mean or diastolic pressure. Instead we monitor urine output, mentation, limb perfusion, gastrointestinal function and respiratory function as indicators that perfusion is acceptable.

The Sick Foal

The kidney is a target for injury in these patients and it is not unusual for renal compromise to play a significant role in the demise of these foals. Clinical signs of renal disease are generally referable to disruption of normal control of renal blood flow and tubular edema leading to tubular necrosis and renal failure. These foals present with signs of fluid overload and generalized edema. The urine of normal newborn foals is quite dilute, reflecting the large free water load they incur by their milk diet. Many antimicrobial agents used in the management of these cases, most notably the aminoglycosides, depend on renal clearance. Aminoglycoside toxicity occurs in the equine neonate and will exacerbate or, at the least, complicate the management of renal failure originally due to primary hemodynamic causes.

In general sick foals can suffer from a variety of problems associated with abnormalities within the gastrointestinal tract. Commonly they present with ileus, recurrent excessive gastric reflux and gas distention. These problems are exacerbated by constant feeding in the face of continued dysfunction and continued hypoxia. Frequently, enteral feeding cannot meet their nutritional requirements and partial (PPN) or total parenteral nutrition (TPN) is required. Special attention should be paid to passive transfer of immunity status and glucose homeostasis in these cases. Clinical signs of injury to the gastrointestinal tract can be subtle and lag behind other abnormalities for days to weeks. Low grade colic, decreased gastrointestinal motility, decreased fecal output and low weight gain are amongst the most common clinical signs of gastrointestinal dysfunction in these case, but more severe problems, including necrotizing enterocolitis and intussussception, have been associated with these cases. The return to enteral feeding must be slow in many of these cases.
Foals with PAS are also susceptible to secondary infection. Treatment of recognized infection is required and antimicrobial treatment is almost always required and should be broad-spectrum. Repeat determination of IgG concentration should be made and additional intravenous plasma therapy may be required. Any acute deterioration in the condition of a foal with PAS indicates a need for further evaluation for possible sepsis.

The prognosis for foals with PAS is good to excellent when it is recognized early and aggressively treated in term foals. More than 80% of these neonates survive and go on to lead productive and useful athletic lives. Prognosis decreases with delayed or insufficient treatment and concurrent problems such as prematurity and sepsis with evidence is accumulating that failure resolve hyperlactatemia within 48 hours of presentation in foals with PAS indicates a poorer prognosis for ultimate survival.

Practical Solutions to Common Problems of the Foal
The equine practitioner may be faced with management of fairly ill neonatal foals without access to referral facilities, either due to location or financial restriction placed by the owner. The following are some suggested solutions to problems that may be encountered by the practitioner under these conditions.

Long-term Intravenous Access:
Long-term intravenous access for fluid administration or antimicrobial administration may be necessary in some cases. In these cases, placement of 'over the wire catheters' provides the best solution. The catheters are generally made of non-thrombogenic materials and are more pliable, making them less likely to clot or to break. Although this author uses catheters made by Mila for this purpose, Arrow also has a long-tem catheter that is similar. Placement of these catheters is more challenging than over the needle catheters, but with practice placement becomes straightforward.

Catheterization is performed most readily in recumbent foals but can be performed in standing foals. Having sufficient help available is important, as good restraint of the patient is necessary. If help is not readily available, small doses of diazepam (2-5 mg per foal, IV) can make the process easier. Xylazine should be avoided in very young foals as it can cause transient hypertension. Acepromazine should not be used in foals with potential for seizure activity, such as perinatal asphyxial syndrome foals ('dummy' foals) as it lowers the seizure threshold. Small doses of butorphanol (~3 mg/foal) are tolerated fairly well.

Catheterization should be performed under sterile conditions. The site should be clipped and surgically prepped. Placing a rolled towel under the foal's neck in the mid-cervical region elevates the neck and makes catheter placement easier. Catheters should be sutured in place. This can be made easier by using small blebs of lidocaine over the jugular vein where the catheter is to be places and in the skin where retention sutures are to be placed. All needed materials should be within arms reach once catheter placement has commenced, including syringes for blood sampling, heprinzed saline for flushing and the injection cap. Large volumes of flush are not required for these catheters and the catheter patency can usually be maintained by flushing with 5 ml heparinized saline after each use. These catheters should be flushed at least 4 times daily. There is generally no need to place a bandage or any other type of protection over the catheters.

Tube feeding:
Foals able to tolerate enteral feeding but too weak to suck properly from the mare, or any foal without the ability to suck, may benefit from placement of a long-term enteral feeding tube. The tubes are well tolerated by foals and they can learn to suck from a bottle or from the dam around them. They are small diameter tubes that are placed as nasoesophogeal tubes. They can be purchased with enteral feeding bags that attach to them and they have small, attached caps to prevent air aspiration into the esophagus.

Feeding should always be performed under gravity flow only. It is ideal to pass a large bore nasogastric tube to check for reflux before placing these tubes as a foal with significant gastric fluid accumulation, or blood tinged gastric fluid, should not be fed enterally until this is resolved.

The amount fed should be calculated on a daily basis. Feeding should initially aim at providing milk or milk replaced at about 10% of the foals body weight per day. For examples, a 100 pound foal should have an initial target of 10 pounds of milk. This is divided into 12 feedings given at 2 hour intervals. An approximate feeding schedule would be 14 ounces every 2 hours. Once the feed is administered the tube should be flushed with a small volume of water ad recapped. Foals should be standing or in sternal recumbency during the feeding and should remain in that position for at least 5 minutes once feeding is completed. If the foal tolerates feeding well, the amount fed can be gradually increased to 20% of the body weight per day.

Arterial blood gas analysis:
Arterial blood gas analysis has traditionally been performed primarily at large referral institutions. However, the advent of stable and durable portable blood gas analysis units has expanded this ability t the private practitioner. Units are available as either new units or refurbished units. Two popular units are the IRMA and the I-STAT units. Information can be found on the internet regarding these units.

Fluid Therapy in the Field:
Fluid therapy in foals can be taken can be undertaken in the field, keeping in mind some differences between

the neonate and the adult. The intravenous route should be used for initial resuscitation and administration of glucose and plasma. Oral fluid supplementation is possible, but should only be done under gravity flow. Because renal function and vascular physiology of the equine neonate is substantially different from the adult, fluid therapy in the neonate cannot simply be scaled down from adult therapy. Fluid therapy should be conservative during postpartum resuscitation, as the newborn foal is generally not volume depleted unless excessive bleeding has occurred. Some compromised newborn foals are actually hypervolemic. If intravenous fluids are required for resuscitation administration of 20 ml/kg of a non–glucose-containing polyionic isotonic fluid over 20 minutes (about 1 L for a 50-kg foal), known commonly as a 'shock bolus', can be effective. Non–glucose-containing polyionic intravenous fluids should be used because hyperglycemia, but not hypoglycemia, immediately after fetal or neonatal asphyxia has been shown to interfere with the recovery of brain cell membrane function and energy metabolism in neonatal piglets. These findings suggest that post–hypoxic-ischemic hyperglycemia is not beneficial and might even be harmful in neonatal hypoxic-ischemic encephalopathy. Indications for 'shock bolus' therapy include poor mentation, poorly palpable peripheral pulses, and the development of cold distal extremities, compatible with hemodynamic and/or hemorrhagic shock. The foal should be reassessed after the initial bolus and additional boluses administered as necessary. These same indications should be used when administering 'shock bolus' treatment to neonates with other conditions requiring fluid resuscitation, such as sepsis. For the practitioner, failure to respond well to bolus therapy probably indicates a need for referral to a hospital where continuous fluid therapy and, potentially, inopressor therapy, can be more safely and readily administered. If blood loss appears to be significant, referral should also be considered for administration of blood.

Glucose-containing fluids can be administered as a constant rate infusion (CRI) both after resuscitation and for energy support in the sick neonatal foal at a rate of 4 to 8 mg/kg/min, particularly in the obviously compromised foal. If the foal requires glucose during resuscitation, dextrose can be added to the bolus liters as 0.25-1% dextrose (5-20 ml 50% dextrose) per liter. This will approximate 4-8 mg/kg/min. A rapid method of calculating an appropriate rate in the field for administering 5% dextrose in water at 4 mg/kg/min is as follows:

[(Body weight in pounds) x 2] + [(Body weight in pounds) x 20%] = #mls/hour

For a 100 lb foal, this becomes:
[(100) x 2] + [(100) x 10%] = 200 + 40 = **240 mls 5% dextrose in water/hour**

*** This can be "ballparked" at 250 mls per hour to most average (50 kg) foals!**

If this rate is not sufficient to keep blood glucose concentration within a normal range (60-140 mg/dl) then the rate can be doubled, or the same rate can be used by with a greater dextrose concentration, such as 10% dextrose. A fluid pump is a good idea to use for this and many can be found on the secondhand market for very reasonable cost. Most pumps will require specific administration sets. Alternatively, buretrols can be used to delive a set maximum amount each hour, even if the line is wide open no more than what is in the chamber will be delivered. Hand-held glucometry units are commonly used to monitor glucose concentrations in the blood or plasma, but should be assessed for their accuracy in the specific conditions they are used in, as they can be quite inaccurate.

CRI glucose therapy is indicated to help resolve metabolic acidosis, to support cardiac output because myocardial glycogen stores likely have been depleted, and to prevent postasphyxial or sepsis associated hypoglycemia. Under normal conditions, the fetal-to-maternal blood glucose concentration gradient is 50% to 60% in the horse, and glucose is the predominant source of energy during fetal development. Glucose transport across the placenta is facilitated by carrier receptors (glucose transporter [GLUT] receptors), and a direct relationship exists between maternal and fetal blood glucose concentration when maternal glucose is in the normal range.

At term, the net umbilical uptake of glucose is 4 to 8 mg/kg/min, with most of the glucose being used by the brain and skeletal muscle. The fetus only develops gluconeogenesis under conditions of severe maternal starvation. Fetal uptake of lactate across the placenta is about half that of glucose. The transition to gluconeogenesis, stimulated by increased circulating catecholamine concentration from birth and by stimulation of glucagon release at the time the umbilical cord breaks takes 2 to 4 hours in the normal foal, and glycog-

enolysis supplies needed glucose until feeding and glucose production are accomplished. In the challenged foal, glycogen stores may have been depleted and gluconeogenesis delayed, so provision of glucose at rates similar to what the liver would normally produce during this period is requisite.

The clinician managing critically ill neonates must recognize that intravenous fluid therapy simply cannot be scaled down from adult management approaches. Fluid management of the ill neonate, particularly over the first few days of life, must take into consideration that the neonate is undergoing a large transition from the fetal to the neonatal state and that important physiologic changes are taking place. These transitions include shifts in renal handling of free water and sodium and increased insensible losses because of evaporation from the body surface area and the respiratory tract. The newborn kidney has a limited ability to excrete excess free water and sodium, and the barrier between the vascular and interstitial space is more porous than that of adults. Water and sodium overload, particularly in the first few days of life, can have disastrous long-term consequences for the neonate. In the equine neonate, excess fluid (and sodium) administration frequently manifests as generalized edema formation and excessive weight gain, frequently equivalent to the volume of excess fluid administered intravenously. In cases in which antidiuretic hormone (ADH) secretion is inappropriate, as seen in some foals with PAS, generalized edema may not form and the excess free water is maintained in the vascular space. This 'syndrome of inappropriate anti diuretic hormone secretion' (SIADH) is recognized in the foal that gains excessive weight not manifested as edema generally, with decreased urine output and electrolyte abnormalities such as hyponatremia and hypochloremia. The foal manifests neurologic abnormalities associated with hyponatremia and this should be distinguished from PAS. The serum creatinine concentration varies in these cases, but urine always is concentrated compared with the normally dilute, copious amounts of urine produced by foals more than 24 hours of age on a milk diet. The treatment for this disorder is fluid restriction until weight loss occurs, electrolyte abnormalities normalize, and urine concentration decreases. If the clinician is unaware of this differential diagnosis, the neonate can be assumed mistakenly to be in renal failure, and the condition can be exacerbated by excessive intravenous fluid administration in an attempt to produce diuresis.

The problem of appropriate fluid management in critically ill human neonates has been recognized by medical physicians for years and has resulted in changes in fluid management of these patients. The approach taken has been one of fluid restriction, in particular sodium restriction but also free water restriction, and has resulted in improved outcome and fewer complications, such as patent ductus arteriosus and necrotizing enterocolitis. The calculations used for 'dry' maintenance intravenous fluid support in these patients takes into consideration the ratio of surface area to volume and partially compensates for insensible water losses. The majority of maintenance fluids are then provided as 5% dextrose to limit sodium overload and provide sufficient free water to restore intracellular and interstitial requirements. The calculation for maintenance fluid administration is as follows:

First 10 kg body mass	100 ml/kg/day
Second 10 kg body mass	50 ml/kg/day
All additional kilograms	25 ml/kg/day

As an example, the average 50-kg foal would receive 1000 ml/day for the first 10 kg of body mass, 500 ml/day for the next 10 kg of body mass, and 750 ml/day for the remaining 30 kg of body mass for a total of 2250 ml/day. This translates to an hourly fluid rate of about 94 ml/hr for maintenance. Most foals will generally receive 1.5 to 2 times maintenance but the rate must be adjusted higher if there are large ongoing losses. Potassium is generally supplied to the foal at 20 mEq/L of fluids administered, and then adjusted as needed.

Fluid and sodium requirements can be adjusted for ongoing losses exceeding the maintenance requirements. These losses can take the form of diarrheal losses and excessive urine output, such as those with glucose diuresis and renal damage resulting in an increased fractional excretion of sodium. The normal fractional excretion of sodium in neonatal foals is less than that of adult horses and normal foal urine (foals on a milk diet that are more than 24 hours of age) is generally quite dilute with a specific gravity of 1.005 or so. Remember that the first urine produced by foals is generally quite concentrated, with a specific gravity in some cases of 1.050 or more. In the critically ill foal the sodium requirement can be met with as little as 140 mEq of sodium per day (1-3 mEq per kg/day for growth), about that administered in a single liter of isotonic crystalloid fluids. One liter of commercially available equine plasma will have a sodium concentration of ®170 mEq/L due to aniticoagulation with sodium citrate. One can address sodium deficits by separate

infusion of sodium-containing fluids, although this may not be necessary if one considers the sodium being administered in other forms, including drugs administered as sodium salts and any constant rate infusions (pressors, inotropes, etc.) that are being provided as solutions made with 0.9% sodium chloride. The majority of fluid should be delivered as 5% or greater dextrose in water.

The author has used this approach to fluid therapy in the NICU for many years and believes that the percentage of foals suffering from generalized edema, and related problems, has decreased. When using this approach to fluid therapy, the foal should be weighed at least once daily, although this is frequently impractical in practice, and fluid intake and output should be monitored closely as practical.A normal neonatal foal will gain 1-3 kg/day. Any larger than anticipated weight gains or losses should result in closer evaluation of the foal. Urine output will probably not approach the reported normal of 300 ml/hr for a 50-kg foal on a free choice milk diet because the free water administered is limited, unless the patient is experiencing diuresis (glucosuria, resolution of the syndrome of inappropriate antidiuretic hormone secretion, resolution of previous edematous state, renal disease). If possible, urine specific gravity should be measured several times daily –and should be hyposthenuric after 24 hours, specific gravity < 1.010- and fractional excretion of sodium measured if renal injury or dysfunction is suspected. If the volume of urine produced by the patient is measured accurately, one can determine sodium losses accurately and can obtain creatinine clearance values.

In practice, fluid boluses are frequently used as maintenance of intravenous lines can be challenging, particularly if the foal remains in a stall with the mare. This approach can be used safely, but the amount of fluid need over the day needs to be carefully considered, particularly in foals with diarrhea where losses may be larger than anticipated. The total amount of fluids needed should be calculated for the day and then divided in to bolus administrations ranging from every 2 to every 6 hours, depending on the condition of the foal, ideally bolus administration should be limited to 1-2 liters per bolus. Fluid choices should be dictated by the needs of the foal, but generally the sodium load administered with this route will be greater than that of constant rate infusion as dextrose should not be administered by bolus at large concentration, ie more than 1-2.5%. Potassium should not be added to fluids being administered as a bolus. This approach will result in sodium overload but will work well in a foal that is nursing. Bolus fluids can also be given as 0.45% saline and 2.5% dextrose if calories are needed, although this is less than ideal.

If the oral route is available for hydration, this can be used in foals that are not nursing but are able to tolerate enteral feeding. Placement of an indwelling small feeding tube (Kangaroo tube) will facilitate this, but the foal should be checked for the presence of reflux using a large diameter tube prior to placing the smaller feeding tube. These tubes are generally placed in the espophagus at the mid thorax level and not in the stomach. They also take some practice to learn to place, but once the technique is 'conquered' they are easy to place and maintain. In general a foal with a healthy gastrointestinal tract will tolerate 10% of it body weight in milk or milk replacer divided into every 2 hour feedings (12 times per day). For a 100 pound foal, this would be:

100lb x .10= 10 lbs
10 lb / 12 = 0.83 lb per feeding

There are 16 ounces in a pound, therefore the feeding should be ~13-14 ounces per feeding. If the foal tolerates this well, it can be gradually increased to 15 to 20 percent of its body weight over a few days if the foal continues to not nurse or drink from a bucket. If the foal shows evidence of colic or abdominal distention, feeding should be discontinued for a few hours and reinstated slowly. Water can be substituted for milk for rehydration purposes and milk or milk replacers can be diluted with water if needed for rehydration purposes, but keep in mind that the caloric support of the foal will be less in these circumstances. All enteral fluids should be administered under gravity flow only and the foal should be kept standing or in sternal recumbency at the least, for 5-10 minutes once the feeding is completed to prevent aspiration.

Nursing Care:
Nursing care is one of the most important aspects of treating recumbent foals. Foals should be kept warm and dry. They should be turned at two-hour intervals if they are recumbent. Feeding recumbent and minimally responsive foals can be a challenge if gastrointestinal function is abnormal and total parenteral nutrition may be needed. If at all possible foals should be weighed daily and blood glucose levels monitored

frequently. Some foals become persistently hyperglycemic on small glucose infusion rates. These foals may benefit from constant rate low dose insulin infusions. Recumbent foals must be examined frequently for decubital sore development, the appearance of corneal ulcers and for heat and swelling associated with joints and physis.

Restraint of the Foal
Working quietly and gently with proper restraint will minimize stress and injury to both handler and foal. Foals can be caught by using the mare to corner the foal or by creeping up on the foal with the handler's head height below that of the foals. The foal is caught and subsequently restrained. For longer procedures the foal is "folded" and laid down in lateral recumbency. To stimulate a recumbent foal to stand, scratching along the spine, often vigorously, will help wake the foal up. Ensure the frontlegs are out in front of the foal and, if needed, lift using the sternum /elbows and pelvis; do not use the tail or put pressure on the abdomen.

Care of the Recumbent Foal

Bedding
The recumbent foal should be kept clean and dry to avoid urine and fecal scalding and decubital ulcers. Urinary catheters can be used however there is an increased risk of ascending infections (McDonald et al 1990). The foal is turned every 2 hours and the wet side is dried off with the use of towels and talcum powder.

Respiratory Care
INO2 tubes need to be cleaned at least once a day. The most common complications with INO2 therapy are nasal irritation and airway drying resulting in excessive discharge. Maintaining the foal in sternal recumbency and alternating sides of recumbency every 2 hours assists with improving oxygenation. Coupage, nebulizing with saline and ensuring the foal is hydrated will assist with loosening secretions. When standing the foal can be coupaged with its head lowered.

Umbilical Care
A chlorhexadine or dilute betadine solution is applied to the external stump twice daily for the first 3 days after birth. Recumbent foals are at increased risk of developing patent urachus or umbilical infection.

Eye care
Recumbent foals are at increased risk of developing corneal ulcers. Artificial tears/lubricant/tribple antibiotic ointment should be placed by placing the ointments on a gloved finger and applying to a rolled out lower lid every 6 to 12 hours in foals with decreased eyelid tone and tear production. Corneas should be stained every 24-48 hours in order to evaluate for ulcers. If entropion is present it should be corrected and eyes treated as if ulcerated. If the foal is thrashing, protective foam helmets can be made to encircle and protect the eye socket.

Temperature Control
Premature foals have poor thermoregulation. Hypothermic foals should be gradually warmed with a combination of water bottles, blankets, heated water mats, air blankets, and heat lamps, ensuring scalding and secondary burning of the underlying skin does not occur. Hyperthermic foals can be cooled with cold water and alcohol baths, and fans. NEVER FEED A COLD FOAL!

Care of the Standing Foal
Deep dry bedding should be provided as pressure sores can develop with increased recumbency, muscle weakness and struggling to stand. Straw is preferred over shavings. If the foal is nursing, the mare's udder should be checked frequently to ensure the foal is nursing well. The foal's urine SG is monitored to ensure it is maintaining hydration (SG < 1.008). Foals usually urinate just after standing or nursing. Foals with diarrhea need frequent cleaning and applications of emollients to prevent scalding of the perineum and vulva.

Feeding the foal
Foals which are nursing from the mare should have their nursing observed. Mares whose foals are not nursing are milked out every 2 hours; the milk is stored and frozen for future use. Foals with a functional GIT but no suckle or swallow reflex are fed via an indwelling nasogastric tube. Complications which can occur with

indwelling nasogastric tubes are pharyngeal and esophageal irritation (most common; oral sucralfate helps with alleviating discomfort); aspiration pneumonia or an inflow of air into stomach through an uncapped tube. The position of the tube should be felt prior to each feeding by feeling the tube in the esophagus above the pharynx. The tube is check for reflux by applying gentle suction. Feeding is by gravity flow. Recumbent foals should be sat sternal during and for 10 minutes after feeding (Fig 3). Standing foals are fed near the mares to encourage bonding.

Maintain the Maternal Bond

It is very important to ensure the bond with the mare is maintained. Once the foal is being assisted to stand the mare is brought over to the foal. Vigorously scratching the mare's wither often starts grooming behavior towards the foal. Taking the mare and foal outside on warm sunny days helps improve their (and everyone else's!) attitude. Warm compresses should be applied to the udder if swollen and edematous and to assist with milk let down.

Prognosis:

The prognosis for foals with PAS is good to excellent when it is recognized early and aggressively treated in term foals. Up to 80% of these neonates survive and go on to lead productive and useful athletic lives.Prognosis decreases with delayed or insufficient treatment and concurrent problems such as prematurity and sepsis and evidence is accumulating that failure resolve hyperlactatemia within 48 hours of presentation in foals with PAS indicates a poorer prognosis for ultimate survival.

The Future:

Cooling of the head or whole body, Biomarkers such as early serum S100B, neuron-specific enolase (NSE) and fibrillary acidic protein are associated with neuroradiographic and clinical evidence of brain injury in newborns with encephalopathy and may prove to be good biomarkers of disease severity. Newer drugs, reinvestigations of older drugs and newer imaging modalities are all under current investigation and may reach a veterinarian near you soon

Table 1: Drugs and dosages used in the treatment of perinatal asphyxial syndrome/ neonatal encephalopthy.

Drug	Indication	Dose	Potential complications
Diazepam	Single or short term seizure control	0.1-0.2 mg/kg (5-10 mg to a 50 kg foal IM or IV)	Rapid administration IV may result in respiratory depression
Midazolam	Single or short term seizure control	0.1-0.2 mg/kg (5-10 mg to a 50 kg foal IM or IV) NOT water soluble.	Rapid administration IV may result in respiratory depression
Midazolam CRI	Longer term control for repetitive seizures. Mild sedation for hyper-responsive or 'jittery' foals.	3-6 mg/hr. Midazolam is water soluble and CRI is administered in isotonic crystalloid fluid using a 0.5 mg/ml concentration.	Rapid administration IV may result in respiratory depression Higher doses may be used if necessary. Advantage is ability to titrate to effect and reversibility if needed.
Phenobarbital	Longer term seizure control	2-5 mg/kg IV slowly over 20 minutes. Start with lower dose and monitor to effect. Maximal effect expected after 45 minutes.	Respiratory depression, hypothermia, hypotension and pharyngeal collapse particularly at higher doses or in more severely affected foals.
Thiamine	Metabolic support	1-20 mg/kg q12 hr added to IV fluids (protect from light)	None
Mannitol	Intercellular edema: osmotic diuretic	0.25-1.0 g/kg IV as a 20% solution rapidly over 15-20 minutes	Dehydration. May result in significant hyperosmolarity with repeated administration. May exacerbate cerebral bleeding.
Dimethylsulfoxide	Anti-inflammatory, Intercellular edema: osmotic agent	0.1-1 gm/kg IV as a 10%solution	Odor; OSHA restrictions in some areas, hemolysis; dehydration
Gabapentin	Neuroprotection: GABA receptor agonist.	10-15 mg/kg/day divided equally and given orally 3 to 4 times a day	None described: an uninvestigated drug in equine neonates
Magnesium CRI	Neuroprotection: NMDA receptor antagonist.	*See below. Can precipitate other infusates, test compatibility or discontinue when administering intravenous antimicrobial drugs.	In very high doses (>10x) muscular weakness can occur as can hypotension

CRI: constant rate infusion

*Magnesium CRI: Remove 20 ml from 100 ml bag sterile isotonic saline (0.9%). Add 20 ml 50% MgSO4 for a final volume of 100 ml. Administer loading dose at 25 ml/hr for a 50 kg foal for 1 hour, decrease infusion to 12.5 ml/hr thereafter. This is approximately 50 mg/kg loading dose followed by 25 mg/kg maintenance dose. Infusion can be maintained for 24-48 hours.

Table 2: Commonly used antimicrobials in neonatal foals and their dosages. It is important to recognize that many antimicrobials have different dosages and interval in the foal, especially the neonate, than those used in adults

Drug	Dose, Route, Frequency	Comments
Acycolvir	16 mg/kg PO TID	
Amikacin sulfate	< 1wk old: 25-30 mg/kg IV SID 2-4wk old: 20 – 25 mg/kg IV SID	Nephrotoxic TDM: 30 min peak > 45 ®g/ml 8 hr trough: < 15 ®g/ml 12 hr trough: < 5 ®g/ml
Ampicillin sodium	50-100 mg/kg IV QID	
Azithromycin	10 mg/kg PO SID for 5 days then EOD	Hyperthermia, diarrhea in foal and mare
Cefazolin	25 mg/kg IM TID/QID	1st generation cephalosporin
Cephalexin	30 mg/kg PO TID	1st generation cephalosporin
Cefuroxime	30 mg/kg/day PO BID/QID 50-100 mg/kg/day IV TID/QID	2nd generation cephalosporin
Cefotaxime	50-100 mg/kg IV QID	3rd generation cephalosporin
Ceftazidime	40 mg/kg IV TID/QID	3rd generation cephalosporin
Ceftiofur	5 mg/kg IV BID 10 mg/kg IV QID CRI: 1.5 mg/kg/hr Nebulized: 1 mg/kg as 25 mg/ml solution TID/BID	3rd generation cephalosporin No CNS penetration Ideally given over 20 mins Higher doses: broader gram-ve spectrum
Cefpodoxime	10 mg/kg PO BID/QID	3rd generation cephalosporin
Ceftriaxone	25 mg/kg IV BID	3rd generation cephalosporin
Cefepime	11 mg/kg IV/IM TID	4th generation cephalosporin
Chloramphenicol	50 mg/kg PO QID	Public health/OHS concerns
Clarithromycin	7.5 mg/kg PO BID	Hyperthermia, diarrhea in foal
Doxycycline	10 mg/kg PO BID	
Enrofloxacin	5 mg/kg PO SID	Chondropathy and arthropathy
Erythromycin stearate	25 mg/kg PO TID	Hyperthermia, diarrhea in foal and mare
Fluconazole	8 mg/kg loading then 4 mg/kg PO BID	
Gentamicin sulfate	< 7do: 11-13 mg/kg IV SID Older foals: 6.6 mg/kg IV SID Nebulized: 2.2 mg/kg as 50 mg/ml solution SID	Nephrotoxic TDM: 30 min peak > 25 ®g/ml 8 hr trough: < 5 ®g/ml 12 hr trough: < 2 ®g/ml
Imipenem	10-20 mg/kg IV QID	
Metronidazole	10-15 mg/kg PO or IV TID	10 mg/kg BID if increased GIT absorption may occur.
Oxytetracycline	10 mg/kg IV BID	Nephrotoxic, give slowly
Na or K Penicillin	20,000 - 50,000 IU/kg IV QID	Use upper dose in severe infections
Procaine penicillin	20,000 – 50,000 IU/kg IM BID	
Rifampin	5 mg/kg PO BID	Use with other antimicrobials
Ticarcillin and clavulonic acid	50 – 100 mg/kg IV QID CRI: 2-4 mg/kg/hr	
Trimethoprim -sulfonamide	30 mg/kg PO/IM/IV BID	Dose/kg is combined trimethoprim and sulfonamide

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Dr. Pamela Wilkins Brief Biosketch 2023

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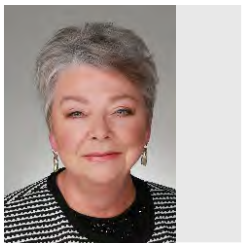

Intensive care management under field conditions; what we really can do?

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Equine NICUs have greatly improved our knowledge and understanding of the normal and abnormal physiology of the equine neonate, resulting in an improvement in our ability to successfully treat the critically ill equine neonate and save lives. We have also been able to translate many treatments from the referral hospital to the field with good success for the practitioner.

The Sick Foal
The kidney is a target for injury in these patients and it is not unusual for renal compromise to play a significant role in the demise of these foals. The urine of normal newborn foals is quite dilute, reflecting the large free water load they incur by their milk diet. Aminoglycoside toxicity occurs in the equine neonate and will exacerbate or, at the least, complicate the management of renal failure originally due to primary hemodynamic causes. In general sick foals can suffer from a variety of problems associated with abnormalities within the gastrointestinal tract. Foals with other ailments, peripartum adverse events or failure of passive transfer of maternal immunity (FPT) are also susceptible to secondary infection. Treatment of recognized infection is necessary and antimicrobial treatment should be broad-spectrum. Any acute deterioration in the condition of a compromised foal indicates a need for further evaluation for possible sepsis.

Practical Solutions to Common Problems of the Foal
The equine practitioner may be faced with management of fairly ill neonatal foals without access to referral facilities, either due to location or financial restriction placed by the owner. The following are some suggested solutions to problems that may be encountered by the practitioner under these conditions.

Long-term Intravenous Access:
In these cases, placement of 'over the wire catheters' provides the best solution. The catheters are generally made of non-thrombogenic materials and are more pliable, making them less likely to clot or to break Placement of these catheters is more challenging than over the needle catheters, but with practice placement becomes straightforward.

Catheterization is performed most readily in recumbent foals but can be performed in standing foals. Having sufficient help available is important, as good restraint of the patient is necessary. If help is not readily available, small doses of diazepam (2-5 mg per foal, IV) can make the process easier. Xylazine should be avoided in very young foals as it can cause transient hypertension. Catheters should be sutured in place. These catheters should be flushed at least 4 times daily. There is generally no need to place a bandage or any other type of protection over the catheters.

Tube feeding:
Foals able to tolerate enteral feeding but too weak to suck properly from the mare, or any foal without the ability to suck, may benefit from placement of a long-term enteral feeding tube. The tubes are well tolerated by foals and they can learn to suck from the dam around themFeeding should always be performed under gravity flow only. It is ideal to pass a large bore nasogastric tube to check for reflux before placing these tubes as a foal with significant gastric fluid accumulation, or blood tinged gastric fluid, should not be fed

enterally until this is resolved. Feeding should initially aim at providing milk or milk replaced at about 10% of the foal's body weight per day. This is divided into 12 feedings given at 2 hour intervals. Once the feed is administered the tube should be flushed with a small volume of water ad recapped. Foals should be standing or in sternal recumbency during the feeding and should remain in that position for at least 5 minutes once feeding is completed.

Fluid Therapy in the Field:
Fluid therapy should be conservative during postpartum resuscitation, as the newborn foal is generally not volume depleted unless excessive bleeding has occurred. If intravenous fluids are required for resuscitation administration of 20 ml/kg of a non–glucose-containing polyionic isotonic fluid over 20 minutes (about 1 L for a 50-kg foal), The foal should be reassessed after the initial bolus and additional boluses, up to 2, administered as necessary. These same indications should be used when administering 'shock bolus' treatment to neonates with other conditions requiring fluid resuscitation, such as sepsis. In practice, fluid boluses are frequently used as maintenance of intravenous lines can be challenging, particularly if the foal remains in a stall with the mare.

Nursing Care:
Nursing care is one of the most important aspects of treating recumbent foals. Foals should be kept warm and dry. They should be turned at two-hour intervals if they are recumbent. Feeding recumbent and minimally responsive foals can be a challenge if gastrointestinal function is abnormal and total parenteral nutrition may be needed. Recumbent foals must be examined frequently for decubital sore development, the appearance of corneal ulcers and for heat and swelling associated with joints and physis.

Umbilical Care
A chlorhexadine or dilute betadine solution is applied to the external stump twice daily for the first 3 days after birth. Recumbent foals are at increased risk of developing patent urachus or umbilical infection.

Eye care
Recumbent foals are at increased risk of developing corneal ulcers. Artificial tears/lubricant/triple antibiotic ointment should be placed by placing the ointments on a gloved finger and applying to a rolled out lower lid every 6 to 12 hours in foals with decreased eyelid tone and tear production.

Temperature Control
Premature foals have poor thermoregulation. Hypothermic foals should be gradually warmed with a combination of water bottles, blankets, heated water mats, air blankets, and heat lamps, ensuring scalding and secondary burning of the underlying skin does not occur. Hyperthermic foals can be cooled with cold water and alcohol baths, and fans. NEVER FEED A COLD FOAL!

Prognosis:
The prognosis for most sick foals is good to excellent when it is recognized early and aggressively treated in term foals.

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Foal pneumonia: *Rhodococcus equi* and more

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Foals develop multiple forms of pneumonia. Differential diagnoses will vary based on clinical signs and age of the foal. As with adult horses, culture and cytology of the tracheal fluid is required to obtain a definitive diagnosis.

Clinical signs

Clinical signs of pneumonia may not be too prominent until disease is severe or may be masked by clinical signs of the primary condition (i.e. sepsis). Fever, lethargy, tachypnea, nostril flaring, increased abdominal respiratory effort, purulent bilateral nasal discharge and cough may be present. Sudden death is not uncommon with severe disease.

Young foals:

Pneumonia associated with sepsis

- The most common organisms isolated are Gram negative enteric bacteria (E. coli, Klebsiella spp, Actinobacillus equuli, etc.) or Streptococcus zooepidemicus.
- Predisposing factors: Placentitis, failure of passive transfer, prematurity, etc.
- Severe disease develops rapidly.
- Route of infection may be vertical (in utero), hematogenous (umbilical infection, ingestion, and absorption of bacteria) or respiratory.
- Treat the underlying disease. Systemic broad-spectrum antibiotics to treat sepsis. Adjust as soon as sensitivity is available.

EHIV-1 pneumonia

- EHV-1 infection of a mare around foaling time may lead to the birth of a weak.
- The time of foal infection remains unknown.
- Foals develop severe, progressive pneumonia that may be complicated by secondary bacterial infection.
- Foals typically die within 2 weeks in spite of critical care; thus prognosis is poor. There might be a history of respiratory clinical signs in the farm or abortions associated with the birth of these affected foals.

Pneumocystis carinii

- Affects immunosuppressed foals (co-infection with other organisms, malnutrition, etc.). Causes interstitial pneumonia.
- Cytology definitive: Macrophage with P. carinii organism inside. Radiographs may show a distinct military pattern.
- Treatment (based on humans) is trimethoprim sulfa (TMS). Correction of underlying cause of immunosuppression if possible.

Adenovirus

- Adenovirus can be present in horses and result in subclinical or mild upper respiratory tract disease that resolves uneventfully.
- It causes progressive, fatal bronchopneumonia in Arabian foals that have severe combined immunodeficiency disease (SCID).
- The clinical sings are of those of pneumonia accompanied by lymphopenia (if SCID) and neutropenia (immunosuppression). The virus replicates in the respiratory epithelium.

Diagnosis
- Auscultation can be misleading: May be normal even if disease is severe.
- If patient is stable: transtracheal wash and submit fluid for culture/sensitivity and cytology.
- Thoracic radiographs: Helps with assessment of disease severity.
- Ultrasound: Widely available, easy to do. Limited to the surface of the lung unless consolidation/ abscessation is present.
- Hematology: Non-specific inflammatory findings

Treatment
- Varies with the condition (see above) but in many cases, treatment is instituted using broad spectrum antibiotics until results from culture-sensitivity are available.
- Intranasal oxygen: For critical patients.
- Anti-inflammatories: Ensure patient is hydrated before using.
- Bronchodilators: Evaluate case by case as it may cause a ventilation-perfusion mismatch.
- Rest

Pneumonia in older foals
Atypical interstitial pneumonia

- Sporadic, rapidly progressive bronchointersitial pneumonia that leads to respiratory failure and death.
- Foals 1-6 months of age.
- The cause is unknown in most cases, but the thought is that is triggered by a pathogen.
- Clinical signs develop fast.
- Diagnosis depends on ruling out other conditions.
- Diagnostics are limited because of the degree of respiratory distress.
- Thoracic radiographs: Caudodorsal interstitial and bronchointerstitial pattern.
- If patient is stable, perform a TTW for culture/cytology.
- Combination of systemic and inhaled corticosteroids and bronchodilators. Broad spectrum antibiotics are recommended until diagnosis is confirmed. Intranasal oxygen is useful until the clinical signs subside.

Rhodococcus equi
- Bacteria must carry the Virulence-associated plasmid gene (vapA) that encodes for the VapA protein to be pathogenic. VapA- strains are not pathogenic.
- Sporadic or endemic in farms.
- Present in normal adult's horse feces (and other mammals).
- Affects young foals: Appear to be susceptible to infection first few weeks of life only.
- Affects adults if immunosuppressed.
- Risk of zoonosis if person immunosuppressed.
- Foals ingest R. equi and it replicates in the intestine: May also cause colitis and diarrhea.
- Inhaled into the lower airways. Bacteria replicates in the alveolar macrophage and leads to granulomas and abscessation.
- Typically cause slow progressive disease but sudden death has been reported. Clinical signs seen 2-4 months of age.
- Definitive diagnosis is made by TTW. Perform only if foal is not in respiratory distress. Fluid sample for cytology and culture. PCR for the vapA gene.
- Thoracic ultrasonography: Abscesses located on the surface of the lung, useful for presumptive diagnosis.
- Radiographs: Pulmonary abscessation. Presumptive diagnosis.
- Hematology: Non-specific chronic infection: Mature neutrophilia, monocytosis, thrombocytosis, mild anemia, increased fibrinogen, and globulins.
- Treatment: Combination of macrolide with rifampin.
    - Rifampin (5mg PO q12h): Urine discoloration (orange) is common, warn the owner.
    - Macrolides: Induce fatal hyperthermia because they prevent sweating: Keep foals inside in cool area.
    - Azithromycin (10mg/kg PO q24h for 5 days, thereafter eod) or clarithromycin (7.5mg/kg PO

q12h) are the most used. Erythromycin old treatment (25mg/kg PO q6h). Clarithromycin reported to be the best.
- A combination of doxycycline (10mg/kg PO q12h) and rifampin can be used in case of diarrhea.
- Intranasal oxygen if needed. Other supportive care and rest

Other bacterial pneumonia: Foals can be affected by the same bacteria than adult horses. Clinical sings are those described above. Definitive diagnosis is made by cytology and culture of TTW sample.

Parasitic pneumonia:
- Parascaris equorum ("ascarids").
- Parasite's life cycle includes a migratory phase in the lung: Weanling and yearlings. Adult horses (> 18m) don't develop pneumonia.
- Clinical sings are mild. Thick, bilateral purulent nasal discharge that improves as they age (and get dewormed). Cough is not uncommon.
- Signs of parasitism, such as poor body condition, rough haircoat may also be present.
- Can affect multiple foals at once because of the timing of the parasite cycle.
- Cytological evaluation of TTW may reveal an eosinophilic reaction or larvae.
- No antibiotics are usually needed.

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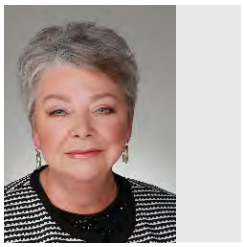
Dr. Sanz *graduated as a veterinarian in La Plata, Argentina. She completed an Equine Internship, a Large Animal Internal Medicine Residency and a Master's of Science degree at Washington State University and she is a Diplomate of the ACVIM College. She also completed a PhD in equine immunology at the Gluck Equine Research Center; her research focuses in equine immunology and infectious diseases. She worked as a Senior Lecturer in Equine Medicine at the Onderstepoort Veterinary School in South Africa for 3 years. Dr. Sanz is an Associate Professor in Equine Medicine at Washington State University in the US.*



Gastrointestinal Disorders of Neonatal Foals

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Colic in the foal can be difficult to diagnose accurately because one cannot perform an examination per rectum. However, many diagnostic aids, most importantly ultrasonography, are available to help differentiate medical from surgical causes of abdominal discomfort in the foal.

Obstruction
Intestinal accidents of all types described in adult horses, with the possible exception of enteroliths, occur in foals. Intussusception, volvulus, displacement, diaphragmatic hernia, and intra- and extraluminal obstruction have been reported in foals.Abdominal ultrasonographic and radiographic evaluation greatly aids diagnosis. Treatment is primarily surgical.

Meconium Retention/Impaction
Meconium retention or impaction is a common cause of abdominal discomfort in newborn foals. Most foals defecate shortly after their first meal. The usual practice for most owners or veterinarians attending the birth of a foal is to administer an enema to aid this process. The best enema is warm soapy water made with a mild soap such as liquid Ivory soap that can be administered through soft rubber tubing using gravity flow. Foals with significant meconium retention become colicky within the first few hours of life as gas accumulates within the bowel. Additional diagnostics can include abdominal ultrasonography and radiography, particularly if one must rule out other, more serious types of colic. One can treat persistent meconium retention resulting in significant abdominal distention by muzzling the foal administering intravenous fluids. Trocharization for the relief of gas can be quite effective and the author uses this approached commonly in severe cases where pain management is difficult. Most cases resolve with medical management and trocharization alone within 12 to 24 hours.

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Congenital Defects
Atresia within the gastrointestinal system of the foal occurs infrequently, but clinical signs are characteristic. Acute colic occurs within the first few hours and is accompanied by abdominal distention similar to meconium retention. Antemortem diagnosis of atresia, short of abdominal exploratory surgery, is aided by the lack of meconium staining of the rectum or any administered enema fluids. Additional diagnostic tests may include administration of a barium enema for a radiographic study, colonoscopy, and abdominal ultrasonography. One can make affected foals more comfortable by muzzling them to prevent further milk intake and by supplying them with fluids and nutrition intravenously. If one attempts surgical correction, one first should initiate broad-spectrum antimicrobial therapy and determine passive transfer status. Frequently, these foals are hypoxemic because of the abdominal distention, and oxygen supplementation is desirable.

Lethal White Syndrome
Solid white foals born to overo-overo mating of American Paint Horses may suffer from congential aganglionosis of the ileum, cecum, and colon. These foals present similarly to foals with meconium impaction or atresia in that colic develops shortly after birth and involves progressive abdominal distention with feeding. The inherited defect is in the endothelin receptor gene. No effective treatment exists, but the clinician should be aware that not all white foals of this mating are affected, and some simply may have meconium retention, so a short period of treatment may be warranted.

Necrotizing Enterocolitis
Necrotizing enterocolitis is considered the most common acquired gastrointestinal emergency of human infants.The clinical spectrum of necrotizing enterocolitis is multifactoral and ranges from temperature instability, apnea, lethargy, abdominal distention, bilious residuals, septic shock, disseminated intravascular coagulation, and death. In the neonatal foal, necrotizing enterocolitis is probably one of the most underrecognized causes of gastrointestinal dysfunction and in the past has been attributed only to infection with anaerobic organisms including Clostridium perfringens type C and C. difficile. Although a specific form of enteritis in the foal is associated with intestinal infection by these organisms, most necrotizing enterocolitis is associated with prematurity or PAS in the infant and the foal. One should suspect necrotizing enterocolitis in any foal that is having difficulty tolerating oral feeding, demonstrating signs of ileus, or having episodes of colic and in any foal with occult blood, digested blood or frank blood in the stool or reflux.

Gastric Ulcers
Gastric ulcer disease has been recognized in foals, and lesions vary in anatomic distribution, severity, and cause. In clinically normal neonatal foals (<30 days of age), gastric ulcers and mucosal desquamation have been documented. Because of these reports and other early reports of death following ruptured clinically silent ulcers in neonatal foals, for years many clinicians felt it necessary to treat critically ill neonates with antiulcer medication prophylactically. Few specific causes have been found for gastric ulcer disease in foals. Excessive administration of nonsteroidal anti-inflammatory drugs can result in ulceration of the glandular and squamous epithelium because of an inhibition of prostaglandin production, which leads to a decrease in mucosal blood flow and an increase in acid production. Nonsteroidal anti-inflammatory drugs also can impair the healing of lesions and rarely are indicated in neonatal equine medicine. In the sick neonatal foal (<7 days of age) a wide variability in the intragastric pH has been documented depending on the type of disease, severity, and milk intake frequency and volume, suggesting that in the critically ill equine neonate, ulcer prophylaxis using histamine antagonists or proton pump inhibitors is not only unnecessary but unlikely to work.

Clinically significant gastric ulcers can occur in the squamous, glandular, or both portions of the stomach as a primary problem or resulting from another problem. Clinical signs include diarrhea, abdominal pain, restlessness, rolling, lying in dorsal recumbency, excessive salivation, and bruxism. In the neonatal foal the only clinical signs present may be depression or partial anorexia until a more catastrophic event, such as perforation, occurs. The presence of a brown gastric reflux fluid may indicate the presence of bleeding ulcers or necrotizing enterocolitis. Blood in the feces of the neonate is more consistent with a diagnosis of necrotizing enterocolitis, which can be associated with gastric ulcers. Traditional therapy for gastric ulceration includes mucosal adherents, histamine type 2 receptor antagonists, proton pump inhibitors, and antacids.

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