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# Ultrasonographic Examination of the Equine Neonate: Thorax and Abdomen



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## KEYWORDS

- Neonatal diagnostic imaging • Thoracic ultrasound • Abdominal ultrasound

## KEY POINTS

- Diagnostic imaging can substantially augment physical examination findings in neonatal foals.
- Used either in combination with radiography or as a stand-alone imaging modality, ultrasound evaluation of the thoracic and abdominal body cavities is often a high-yield diagnostic undertaking.
- Many of the conditions that afflict neonatal foals are highly amenable to sonographic interrogation, including pneumonia and other changes in the lungs associated with sepsis, systemic inflammatory response syndrome, multiple organ dysfunction, and prematurity; colic arising from both medical and surgical causes; and urinary tract disorders.
- Sonographic imaging is not impaired by intracavitary fluid accumulation, and it reveals abnormalities of both soft tissue and bony origin.
- Adding imaging findings to physical examination and laboratory results aids the veterinarian in detection of intracavitary disease early in evaluation, and this translates into an improved level of both patient care and client care.

## IMAGING IS AN IMPORTANT COMPONENT OF A COMPREHENSIVE EXAMINATION

Among the most common owner complaints that prompt presentation of neonatal foals to a veterinarian for evaluation and care are lethargy, failure to nurse, colic, respiratory signs, urinary tract abnormalities, and various manifestations of sepsis and the systemic inflammatory response syndrome. Because foals lack the tolerance for pain or the physiologic compensatory reserves that adult horses can draw on, adopting a passive, wait-and-see approach with a compromised neonate is often

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an unrewarding strategy and is considered by some to fall below the present standard of care. The practitioner should conduct a comprehensive examination to return the highest yield possible of relevant information. Combining the visual input of diagnostic imaging with physical examination findings and laboratory results adds thoroughness to the evaluation, not only at the time of initial presentation but also during serial monitoring in follow-up examinations.

Of the diagnostic imaging modalities available for use, ultrasonography, radiography, and, increasingly, computed tomography (CT), can provide important and specific input into medical problems of the neonate. Although the preponderance of cases in which CT was used in equine practice has involved orthopedic or intracranial applications,<sup>1</sup> tomography has recently been used to evaluate the lungs in foals<sup>2-5</sup> and holds promise for investigation of intracavitary diseases in this subset of equine patients in the future. At present, though, its availability is limited; it necessitates use of trained personnel and is generally unavailable after hours, and it requires a foal to be heavily sedated or anesthetized. Radiography and ultrasonography yield complementary information and have specific strengths and weak points in their capabilities, but both types of equipment are portable and can be used as point-of-care testing in the stall.

Diagnostic ultrasound equipment is light and easy to set up; the imaging involves no exposure to ionizing radiation, and owners or other stakeholders present find the real-time images of ultrasound especially compelling and illustrative of explanatory points being made by the veterinarian. From a purely imaging standpoint, ultrasound confers certain advantages that are useful in emergent scenarios: it is not necessary to take multiple views to determine the laterality of a lesion; there are no issues with magnification and loss of resolution with varying film-focal point distance; it is not necessary to obtain the images at peak inspiration; some points inaccessible to radiography (eg, the cranial mediastinum, pleural space, and lung fields lying ventral to the diaphragmatic crura) are easily and sensitively examined with ultrasound; and accumulation of fluid in body cavities or in the lung does not add a general opacity or loss of detail to the images but rather increases the acuity of the imaging. Accumulation of fluid in the pleural or peritoneal cavity, for instance, does not necessitate draining of the fluid and then reimaging so that the lung or visceral organs can be better evaluated, but reveals details of tissue form and function through the fluid—whether lung is atelectic or consolidated, or whether a given segment of intestine surrounded by peritoneal fluid is distended but contractile or distended and adynamic, for example—and also reveals clinically relevant characteristics of the fluid itself—whether it occupies one or both hemothoraces, or whether it is acellular and consistent with a transudate or cell-dense and likely to be hemothorax or exudate, to site 2 examples. In short, gaining skill with sonographic imaging can yield an impressive body of information to what which is obtained through physical examination and laboratory testing.

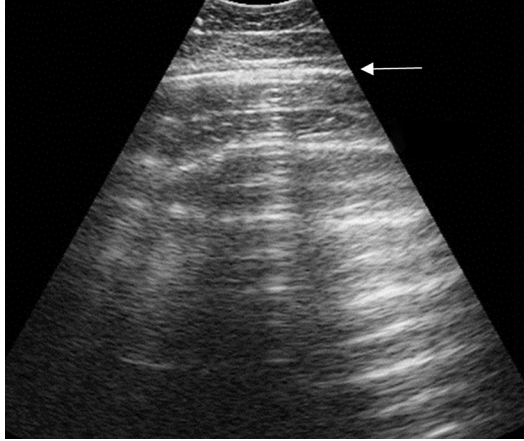
Sonographic imaging does have some shortcomings, and these will be discussed at relevant points in the following sections detailing specific pathologic conditions. Nevertheless, keeping an ultrasound machine in the practice vehicle or plugged in near the examination room is a convenient, safe, and rapid way to add significantly to the body of information derived from the physical examination. At initial evaluation, and later, during serial monitoring as the foal responds to treatment or morbidity progresses, the value of sonographic imaging to the examining veterinarian is hard to overstate and is the focus of this article. The reader is directed to other sources for details of treatment.

## REVIEW OF ACOUSTIC-WAVE BEHAVIOR

Acoustic waves are generated by high-frequency vibration of small crystals that have the quality of being piezoelectric. The word piezoelectric is a derivation of the Greek *piezein*, for press or squeeze, and applies here because a piezoelectric material is one in which charge develops across the surface of the crystal if the crystals are deformed (compressed) by the application of mechanical force. This effect is reversible, and the converse is also true: if electrical charge is applied to the crystal, it will deform slightly the surface configuration of the crystal. When electric current passes into a piezoelectric crystal, these surface deformations give rise to vibrations, and the vibrations generate high-frequency sound waves. Succinctly stated, a piezoelectric material takes electrical energy and converts it to wave energy and takes wave energy and converts it back to electrical energy. In ultrasound transducers, piezoelectric crystals (usually quartz) receive electrical current, vibrate at frequencies unique to their thickness and configuration, and direct pulses of the wave energy into the body. The acoustic waves travel longitudinally away from the transducer and penetrate tissues to depths determined by the wave frequency and tissue impedance. Some of the energy is scattered, and some is reflected back toward the transducer. The time it takes for an echoed pulse to arrive back at the transducer after being emitted is translated by the machine into depth. The ultrasound machine's hardware creates images on the basis of these returning wave echoes in the same way that bats' brains process returning echoes into an image of nearby insect prey. High-frequency sounds are emitted by the bat through its mouth or nose and travel through the air in waves, bouncing off any structures they encounter. After emitting the sound, the bat listens to the returning echoes. The bat's brain processes these echo patterns to construct an image in the same way that the brains of other animals create images from visual information. The processing is unconscious, but the bat perceives echoes that take a longer time to return as distance. If returning echoes reach the bat's left ear before its right ear, it perceives the insect to be to the bat's left. The echoes enable the animal to perceive an insect's vertical position, its size, and in which direction it is traveling, plus distinguish prey echoes from those it receives as background clutter. The return patterns of a pulse of acoustic echoes to the piezoelectric crystals likewise sensitively convey information about organs' depth, location, position, and density.<sup>6,7</sup>

Ultrasound waves are not qualitatively different from the sound waves that can be detected by animals and humans, but are simply higher frequencies than can be detected by the ossicles and other sound detection mechanisms in the middle and inner ear. Healthy young adult humans can hear sound frequencies in the range of 20 Hz to 20,000 Hz, or 20 kHz. Most equine diagnostic ultrasound is conducted using frequencies in the range of 2 to 12 MHz (1 MHz = 1 million vibrations/s).

One of the physical properties of acoustic waves is that they are reflected at any interface where the acoustic impedance of 2 neighboring tissues differs significantly. This principle is relevant for areas in the body where a soft tissue (lower impedance) lies next to a tissue containing gas or to a bony structure (high impedance). Because the generating of a sonographic image depends on the acoustic waves entering and passing through a tissue before returning to the transducer, reflection of the waves at the surface of an organ prevents imaging of any part of that structure below the surface. An acoustic beam aimed at the lungs will traverse the chest wall, parietal pleura, and visceral pleura, and then encounter inflated alveoli. At that interface, the beam is bounced back to the transducer and yields no information about tissue deep to the first millimeter of alveolar tissue. The image yielded by this is a series of parallel white lines called reverberation artifact (**Fig. 1**). In foals with pulmonary disease, lesions



**Fig. 1.** Sonogram of healthy lung. Near-100% reflection of the acoustic beam at the surface of air-filled alveoli just below the visceral pleura (*arrow*) results in generation of the artificial white lines, which yield no information about the tissue other than that there is a gas interface at the pleura. This image was obtained with a convex transducer operating at 5 MHz with a display depth of 12 cm.

deep to a layer of aerated alveoli will be obscured from detection for this reason. Lesions that do extend to the periphery of the lung cause no reverberation artifact but instead yield detailed images. Similarly, in the intestinal tract, waves contacting gas at the bowel wall are reflected without providing an image of anything except the wall itself. Areas of subcutaneous emphysema preclude the imaging of any structures deep to the skin by the same concept.

#### **EQUIPMENT USED FOR IMAGING FOALS**

Transducers are classified according to the internal arrangement of the crystal elements and according to their frequency. Transducers come in linear, curved, or curvilinear, and phased-array sector configurations. In linear array transducers, the crystals are arranged one next to the other, in a line. These probes create a rectangle-shaped image of high detail but shallow depth. Curved or curvilinear transducers also contain crystals placed next to each other in a linear fashion, but they have a curved footprint; these probes generate a wedge-shaped beam and image. Phased-array scanners contain a small group of crystals emitting a beam that is steered into a sector-shaped slice of tissue in front of the probe. These probes scan an area much wider than the probe's footprint, which is useful when tissues, like the heart, must be imaged through a restricted acoustic window such as the narrow intercostal space (ICS). In general, linear array transducers are used most to image tendons and internal reproductive organs, because the structures of interest are close to the probe surface and the probe shape is appropriate for insertion into a tubular space such as the rectum. Curvilinear transducers are most useful for the types of survey imaging done in neonatal foals and effectively penetrate the thoracic and abdominal body cavities. The phased-array sector scanners have a petite, square footprint that enables rotation of the probe so that 90° views can be obtained from the same acoustic window; these transducers are used in imaging of the heart, referred to as echocardiography.

The frequency of acoustic waves used in veterinary diagnostic ultrasound falls in the range of 2 MHz to 12 MHz. The depth to which acoustic waves travel into the body is

inversely proportional to frequency, such that the lowest frequencies travel the farthest, and the higher frequencies penetrate only a few centimeters below the skin surface. There is an inherent tradeoff in selecting a frequency at which to image a body part. The short wavelength of high-frequency sound waves means that there are many returning echoes and a resultant high-detail, high-resolution image, but penetration depth into the body is shallow. The long wavelength of low-frequency waves results in lower-resolution images, but these beams will travel over 30 cm into the body from the skin surface. Multiple-frequency probes are now available, and these emit at a range of frequencies, enabling the examiner to interrogate tissues lying at different depths without having to change probes.

Using sound wave energy to create visual images of organs that are hidden from view inside a body cavity is an amazing but operator-dependent technique, unlike CT, MRI, and radiography, in which the equipment controls the imaging once a button is pushed. The images created by the ultrasound machine are dynamic and change with the examiner's hand position. Scanning structures that are less than perpendicular to the incident beam may introduce changes that look like lesions but are actually positioning artifacts. Acoustic beams that contact tissues at a 90° angle have less scattering and more reflection of energy back to the transducer for processing.

The examiner is well served in investing time to review anatomy with regard to normal locations and spatial relationships among the soft tissue organs occupying the body cavities so that interpreting the images can be done with confidence and accuracy. Diagrams from anatomy texts depicting spatial relationships among organs on both the right and the left sides are helpful in this regard.<sup>8</sup>

## PREPARING TO ULTRASOUND

The acoustic beam emitted from the ultrasound transducer will be halted by the air trapped in the hair coat, so the body area to be scanned should first be either clipped with electric clippers or generously wetted with warmed rubbing alcohol. Alcohol displaces the air between and beneath hairs and creates a necessary interface for transfer of an incident beam into the body in the presence of hair. If the hair is clipped, ultrasound coupling gel is applied to the skin. Clipping the hair coat is generally recommended and facilitates higher image resolution, but the author prefers not to clip hair in most instances for examining structures in the body cavities, especially in cold climates, making application of generous volumes of rubbing alcohol necessary at times. Application of water to the hair will not yield the same result and is unhelpful for ultrasound imaging. The alcohol should be applied in a unidirectional fashion, in the direction of hair growth; rubbing against the nap will introduce air and defeats the purpose of applying the alcohol. Before applying it to the foal's skin, the rubbing alcohol should be warmed by preplacing 1 to 2 plastic bottles in a bucket of hot water (not by heating in a microwave oven) for a few minutes. Sloshing cold alcohol onto a foal's skin induces justifiable reaction and struggling and will also chill the foal. Allowing the alcohol to warm before applying it to the foal's thorax or abdomen makes the procedure more pleasant for patient and examiner alike. Clipping of the hair is recommended for ultrasound of superficial structures, such as the umbilicus. After the ultrasound examination, the foal should be gently towed dry.

## SONOGRAPHIC SURVEY OF THE THORACIC CAVITY

Once the hair coat has been wetted, the thorax should be visually inspected for plaques of edema or swelling in the chest wall, especially near the costochondral junctions. In a neonatal foal, this should raise suspicion of a rib fracture, and the area

should be gently palpated to check for a pain response from the foal or crepitus from fractured rib ends. Foals in which rib fractures are suspected should not be manipulated or handled in any manner that will compress the ribs or sternum. Attendants working with these foals should assist them to stand by lifting from the foal's stifles and elbows rather than by passing an arm under the chest or belly. Rib fractures are a significant source of morbidity and fatality in some practices<sup>9</sup> and appear to be most common in thoroughbred foals, foals born to a primiparous mare, and foals delivered with human assistance, with or without dystocia.<sup>10-12</sup>

The soft tissue structures inside the thorax are viewed in the spaces between the ribs. In neonates, thoracic contents are sensitively surveyed with a microconvex linear transducer operating at frequencies of 4 MHz to 8 MHz. Linear transducers such as those used for transrectal imaging can also yield good images, although some find these images less intuitive to interpret. The author prefers to use a transducer with a microconvex footprint for surveying the organs inside the thorax and a linear transducer operating at 6 MHz to 10 MHz for imaging the ribs and other superficial structures. When a fracture is found, a higher frequency (8–12 MHz) can be used to view the site with maximal detail. Because of the tradeoff between detail and depth, the overarching principle of sonographic imaging is to use the highest available frequency that will penetrate to the depth necessary for any given tissue.

With the foal lying in lateral recumbence and the thoracic skin surface wetted with alcohol, the examiner begins at the cranial-most section of the chest, the thoracic inlet. This area lies deep to the triceps musculature, and the examination commences with the transducer placed on the lateral aspect of the shoulder and parallel to the long axis of the ribs. The transducer should be held perpendicular to the skin surface. The thymus lying in the cranial mediastinal space can be seen by gently pulling the foal's forelimbs forward and aiming the transducer cranially from the third ICS, from either side. Ribs 1 through 4 form struts around the thoracic inlet and cranial mediastinum and lie deep to the triceps musculature. Rib 5 is usually the first rib that can be seen or palpated just caudal to the lateral head of the triceps when the forelimb is in a neutral position. Beginning with the transducer positioned dorsally in each rib interspace, as the examination proceeds caudally rib by rib, the examiner moves it distally slowly and smoothly, keeping the eyes on the ultrasound monitor. The pleural surface can be evaluated for thickness, and the pleural space is evaluated to ensure there is no fluid. The inner limit of the chest wall is delineated by the linear white echo that slides back and forth in the frame (actually dorsally and ventrally with inhalation and exhalation, respectively) with the foal's respiratory excursions. This movement may be subtle in weak, obtunded foals. The moving white line represents the visceral pleura, and the lung is the tissue lying immediately deep to the line. In healthy lung, the series of multiple parallel white lines representing reverberation artifact is seen just below the moving pleural line, yielding no real information about the pulmonary parenchyma. A lesion lying just deep or axial to a zone of inflated lung will be obscured from view.

Unlike normal lung, diseased pulmonary tissue is highly amenable to sonographic interrogation because it does not yield reverberation artifact. Inflammation, fluid accumulation, consolidation of the air spaces and interstitium, and abscess formation all form a favorable medium for transmission of acoustic waves. Pneumonia is discussed further in a later section.

To image the ribs themselves, the transducer is placed directly on top of the rib and the fingers are used to stabilize it there as the probe is drawn distally along the rib arch. Fractures are recognized by a site of discontinuity in the periosteal and endosteal surfaces, and a surrounding area of hypoechoic fluid will be seen adjacent to the site,

representing a hematoma (Fig. 2). If the fracture involves ribs 4 to 6, the beating myocardial wall may be seen beating against the shard of fractured bone, giving a visually sobering appreciation for the potential lethality of these injuries.

### SONOGRAPHIC SURVEY OF THE ABDOMEN

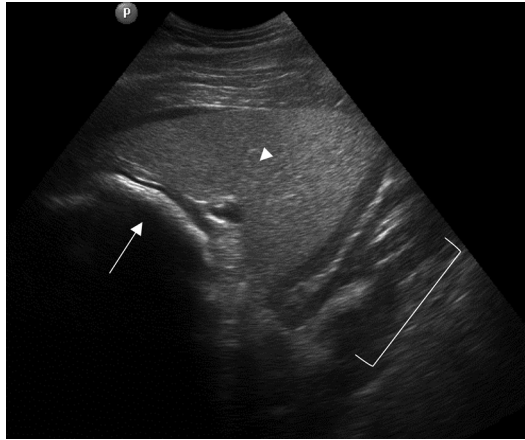
Beginning in the cranial-most aspect of the abdomen, beginning in ICS space 7 on the left side, the first structure to be encountered, lying in contact with the diaphragm, is the liver. Just caudal to that, in ICS 6 to 12, depending on the volume of the gastric contents, lies a curvilinear arc that represents the greater curvature of the stomach (Fig. 3). The liquid contents of the neonatal stomach enables viewing of the luminal contents; by about 7 days of age,<sup>13</sup> gas echoes will fill the stomach, limiting evaluation to the mural structures. Immediate caudal to and in contact with the stomach lies the cranial pole of the spleen, which has a solid, stromal architecture and soft tissue echogenicity. The spleen should be more echogenic than the liver; the difference is easily appreciated where the caudal edge of the left liver lobe lies in contact with the stomach and cranial pole of the spleen, in ICS 8 to 10 on the left side. The spleen can be viewed from ICS 7 back to the paralumbar fossa area, depending on the state of splenic contraction or engorgement at the time of examination. The left kidney is a hypoechoic structure lying axial to the spleen from ICS 15 to the caudal border of the paralumbar fossa and can be recognized by the typical oblong shape and the corticomedullary anatomic pattern with the echogenic cortex, hypoechoic medulla, and hyperechoic renal pelvis. In the groin area ventral to the paralumbar fossa and in the area caudal to the fossa, segments of jejunum can be seen. Large colon segments occupy most of the ventral abdomen in all quadrants and, unlike in older foals and adult horses, will be filled with fluid rather than with a sonoreflective gas layer. The urinary bladder is viewed in the caudoventral aspect of the abdomen.

On the right side of the body, again proceeding in a cranial-to-caudal direction, the first structure seen on the abdominal side of the diaphragm, in ICS 7 to 14, is again the liver. The liver lies in the cranioventral and middle regions of the abdomen in these rib spaces and is also seen in the dorsal abdomen at its caudal-most extent in ICS 14. Bile ducts and hepatic blood vessels can be seen in the liver parenchyma, and the



**Fig. 2.** Sonogram of fractured rib in a neonatal foal. The hematoma formed around the rib ends is organizing, and only anechoic serum (*arrowhead*) is left under the capsule. A patch of fibrin can be seen adherent to the end of the distal fragment (*arrow*). This fracture is characterized by a moderate degree of distraction of the rib ends. Dorsal is to the left. Image obtained with a linear array transducer operating at 6 MHz and with a display depth of 4 cm.





**Fig. 3.** Sonogram of the cranial part of the left side of the abdomen in a 1-week-old foal. Structures seen are the spleen (*arrowhead*) and the white curvilinear echo of an empty stomach (*arrow*). Also seen in this image are several lengthwise jejunal segments thickened with mural edema (*bracket*). Image obtained with a macroconvex transducer operating at 5 MHz with a display depth of 14 cm.

sharpness of the lobe edges and echogenicity of the parenchyma can be appreciated. Caudal and ventral to the liver in ICS 7 to 12, the examiner will see the long white curvilinear echo corresponding with the right dorsal colon. Beginning in about ICS 10, the duodenum can be viewed in transverse section lying in the dorsal surface of the right dorsal colon and the ventral and caudal margin of the liver. To appreciate the structure, it may be necessary to keep the transducer still and observe for several moments until the duodenum goes through a relaxation-contraction cycle, as duodenal motility is typically intermittent. The duodenum can be seen as far caudally as the cranial margin of the paralumbar fossa, lying ventral to the right kidney and dorsal to the base of the cecum. Moving the transducer ventrally from the right kidney, the examiner can view portions of the cecal body and ventral large colon segments.

## DISEASES OF THE THORAX

### *Pulmonary Abnormalities*

Many compromised neonatal foals have pneumonia, whether or not it causes the clinical signs most noticeable to the client. Improper technique for bottle feeding the foal or attempts to nurse by a weak or premature or hypothermic foal can result in aspiration pneumonia. Severe aspiration pneumonitis may be seen in foals that inhaled meconium-contaminated amniotic fluid while gasping during a dystocic birth. Pneumonia is also common in septic foals,<sup>14–16</sup> and in these patients, it should be assumed to be present and treated empirically with a broad-spectrum antimicrobial regimen that has activity against gram-negative bacteria.<sup>17</sup> In hospitalized foals, protracted recumbence can predispose to or exacerbate microbial lung colonization.

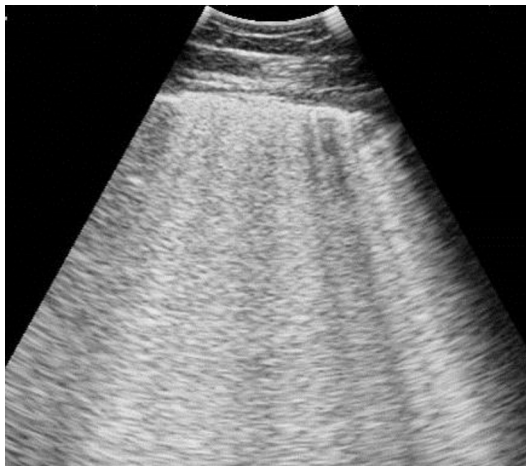
The routes of exposure for pneumonia in a newborn foal are well described and include as a partial list in utero bacteremia or fungemia secondary to placentitis in the dam<sup>18</sup>; in utero infection with equine herpesvirus; aspiration of infectious amniotic fluid during gestation in a mare with placentitis; postpartum viral infection; or postpartum bacteremia from pathogen invasion at the portals of the respiratory tract itself, the umbilicus, or the gastrointestinal tract. After a bacteremic event and multisystemic

showering, pneumonia may persist as a focal infection and the chief clinical problem. The systemic inflammatory response to microbial infection in the lungs or elsewhere involves some combination of fever, tachypnea/hyperventilation, tachycardia, and changes in immature or mature neutrophil numbers on the hemogram. These adaptive responses are familiar to veterinarians and denote immune recognition of microbial invasion and activation of containment and clearance mechanisms.

In contrast, in some foals, inflammation in the lung itself or at some other site in the body elicits an acute syndrome of devastating, global, self-amplifying, and uncontrolled activation of proinflammatory pathways that manifests predominantly in the lungs. These pathways result in injury of both endothelial and epithelial cells in the lung such that the compartments these cells enclose, the pulmonary capillaries and alveoli, respectively, leak. The lower airspaces and lung interstitium flood with cells, proteinaceous fluid, necrotic material, and fibrin, causing rapidly progressive respiratory distress and hypoxemia, with attendant increases in morbidity and mortality. Acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) are clinical stages in the continuum of severe pulmonary dysfunction that develops with this flooding of fluid and cellular debris into the alveolar spaces and interstitium.<sup>19,20</sup>

Radiographic signs of the pulmonary edema of ALI and ARDS are a generalized opacity and loss of detail along with the appearance of a diffuse alveolar pattern with air bronchograms. Air bronchograms develop when gas-filled airways that have escaped the flooding affecting the alveolar and interstitial spaces stand out against the white, more radiodense areas of infiltration that they course through.<sup>21,22</sup> Radiographic patterns reported in foals with ALI or ARDS include alveolar pattern and mixed alveolar + bronchointerstitial pattern.<sup>23</sup> The sonographic appearance of ALI- or ARDS-associated pulmonary edema on ultrasound is that of a diffuse, unbounded sheet of echogenic infiltration seen especially in the dorsocaudal lung fields.<sup>24</sup> This sonographic infiltrative pattern is highly recognizable but does not permit appreciation of tissue details (Fig. 4).

Of the several definition points that must be met in the diagnosis of ALI or ARDS, one involves radiographic detection of infiltrative change in both lungs, and one involves



**Fig. 4.** Sonogram of lung from a foal with NARDS. The lower air spaces and interstitium become flooded with cells and fluid secondary to epithelial and endothelial damage in the wake of severe systemic inflammation. Image obtained with a macroconvex transducer operating at 5 MHz with a display depth of 10 cm.

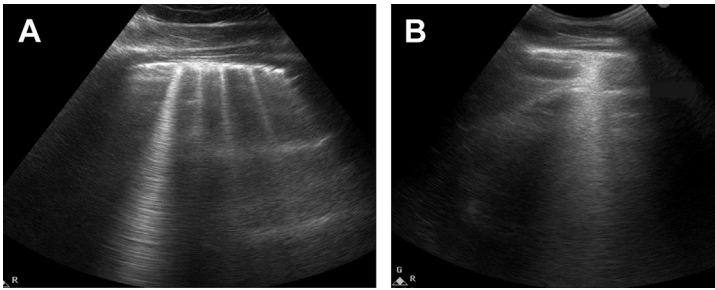
determination of the  $\text{PaO}_2:\text{FiO}_2$  ratio as an index of the degree of oxygenation impairment. By consensus among a panel of veterinary clinicians and criticalists<sup>25</sup> who met for the purpose of revising for animal use the guidelines established for diagnosing ALI and ARDs in humans,<sup>26</sup> ALI in veterinary patients was defined as a  $\text{PaO}_2:\text{FiO}_2$  ratio less than or equal to 300 and ARDS was defined as a  $\text{PaO}_2:\text{FiO}_2$  ratio less than or equal to 200. To underscore the severity of pulmonary injury necessary to result in these values, it should be appreciated that a healthy adult horse breathing air near sea level has a ratio of approximately 476, as calculated from a  $\text{PaO}_2$  of 100 mm Hg and inspired oxygen fraction in atmospheric air of 21%:  $100 \text{ mm Hg} \div 0.21 \text{ mm Hg} = 476$ .

The panel further determined that, given the lower range of arterial oxygen tension that prevails for the first week of postnatal life in healthy foals, compared with adult horses, and the fact that arterial blood sampling is performed with foals restrained in lateral recumbency, different reference ranges for  $\text{PaO}_2$  and hence for the defining of hypoxemia should be used. The syndromes of ALI and ARDS in neonatal foals were thus given the separate designations of NALI and NARDS.<sup>25</sup> The age-adapted cutoff value for neonatal acute lung injury (NALI) is 175 and for equine neonatal acute respiratory distress syndrome (NARDS) is 115. Ratios in healthy foals are greater than 300.

Although the discussion in this article is weighted toward infectious causes of pneumonia and NARDS, it bears repeating that it is an inflammatory response, not just infection, that triggers the life-threatening escalation in immune response that results in NALI and NARDS. Conditions causing direct injury to pulmonary tissue (eg, traumatic chest injury; pneumonia of bacterial, viral, or fungal origin<sup>27,28</sup>; and aspiration of gastrointestinal contents) as well as diseases that injure the lung indirectly by inciting a massive inflammatory response elsewhere in the body (sepsis, disseminated intravascular coagulation (DIC), traumatic injury to body regions other than the lung, and repeated blood transfusion<sup>23</sup>) have all incited ALI and ARDS.

The lung's responses to infection tend to be similar. These changes will be detected with ultrasound if they extend to the lung surface, but because of the qualitative commonalities in response, sonographic changes do not replace microbial culture and other clinicopathologic testing in the diagnostic workup. In addition, radiography may be needed to reveal pathologic changes in the more axial planes of the chest, which are hidden from sonographic detection by aerated tissue in the periphery. However, sonographic imaging of the chest can reveal pulmonary change well before there are any accompanying clinical signs, giving the veterinarian an earlier intervention window.

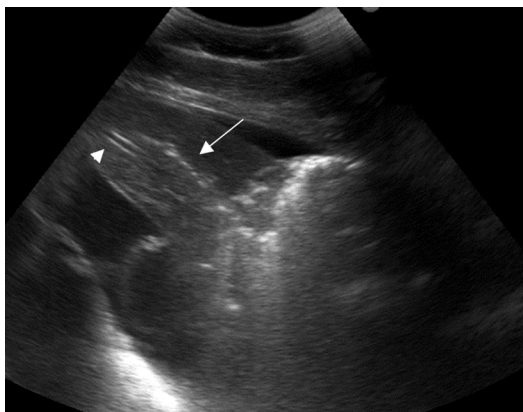
In foals that have a typical response to pulmonary infection (ie, one uncomplicated by NALI or NARDS), the earliest evidence of pneumonia may be thickening of the visceral pleura and the appearance of echogenic (white) projections arrayed vertically, or perpendicular to, the surface of the lung (Fig. 5). These lesions are called comet-tail lesions and are created by focal thickening, with cells and fluid, in the visceral pleura. In more established cases of pneumonia, the influx of cells and exudative fluid into the infected tissue causes consolidation, meaning that the lower airspaces take on soft tissue density rather than being filled with air. In this typical type of immune response and cell infiltration, neutrophils and other immune cells navigate toward the infection site by chemotaxis and exit the vascular space via the process of diapedesis, but the alveolar walls and pulmonary endothelium remain intact. Because there is no air to generate reverberation artifact at the lung surface, ultrasound can yield a detailed image of the normally unseen vascular and bronchial structures in an area of atelectic or consolidated lung (Fig. 6). Although air bronchograms are not sonographically appreciable with pulmonary edema, as they are radiographically, air-filled lower bronchi and bronchioles can be seen with ultrasound in consolidated lung, appearing



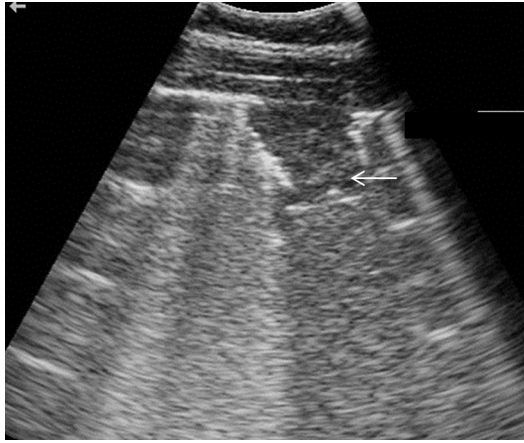
**Fig. 5.** Sonograms of the left lung of a foal with septicemia and pneumonia. (A) Earliest changes noted during serial ultrasound monitoring: pleural thickening and comet-tail lesions extending into the pulmonary parenchyma are seen. (B) Same lung 8 hours later. The discrete comet tails have begun broadening and coalescing, indicating progression of infection and pulmonary change. A similar change will also be seen in the dependent lung in foals left in lateral recumbence without frequent turning. Images obtained with a macroconvex transducer at 5 MHz with a display depth of 14 cm.

as linear or branching hyperechoic structures. The lobar shape and edged borders of the lungs can also be appreciated in lung that is consolidated or atelectatic. Containment and organization of infection into an abscess are also sensitively detected sonographically, as long as the abscess borders extend to the pleural surface (**Fig. 7**). Bright white gas echoes seen in areas of consolidated lung may represent the border between consolidated and aerated tissue or may represent islands of aerated alveoli or foci of gas-producing anaerobic bacteria. Pulmonary edema, as develops with left heart failure or from noncardiogenic causes such as NALI or NARDS, appears on ultrasound as a diffuse sheet of homogeneous echogenic material extending to the pleural surface, without boundaries or localizing characteristics (see **Fig. 4**).

A foal being treated and managed for sepsis that has an acute deterioration in clinical status and in which arterial blood gas reveals hypoxemia should be scanned for



**Fig. 6.** Sonogram of lung near the ventral lung margin. This lung is atelectatic, which can appear similar to consolidated lung. Easily appreciated are the lung's lobar configuration and details of interior pulmonary parenchyma, made possible by the absence of aerated alveoli. The lung is surrounded by anechoic pleural fluid. An air-filled bronchiole can be seen (*arrow*) along with the parallel walls of a pulmonary blood vessel (*arrowhead*). Dorsal is to the right of this image.



**Fig. 7.** Sonogram of lung in a foal with pneumonia that is progressing in severity. This view of lung tissue contains areas of normally aerated parenchyma (where there is reverberation artifact), areas of infiltration with cells or edema (diffuse to broad comet tails), and an irregularly shaped focus of developing pulmonary abscess or consolidation (*arrow*). This lesion is appreciable because it extends to the visceral pleura.

lung changes, and appearance of this diffuse pattern of interstitial infiltration that denotes interstitial edema should prompt revisiting of the treatment regimen to include measures aimed at contravening the development of NALI. Detection of these sonographic changes is a good indication for additional imaging with radiography to provide additional information about the distribution and severity of interstitial flooding.

### ***Pleural Space Abnormalities***

The pleural space merits evaluation as its own compartment inside the thorax. Because in health the parietal and visceral pleurae maintain contact with each other during both the inflation and the deflation phases of lung movement, the pleural space is really only a potential space where the pressure is maintained at a subatmospheric level, and it should be empty save for a small volume of anechoic fluid that may or may not be detected sonographically. When seen, this small volume of fluid is often detected ventrally and caudal to the heart.<sup>29</sup> Imaging of the chest should reveal the glide sign in all areas, confirming that the dorsal part of the pleural space is devoid of gas and the ventral part is devoid of free fluid. This movement is called the glide sign. The glide sign will not be evident in the ventral aspect of the pleural cavity if there is accumulation of fluid and will be missing from the dorsal aspect of the pleural cavity if there is free gas, or pneumothorax. The presence of effusion or blood in the pleural cavity is easy to detect. With practice and repetition, free gas in the dorsal part of the pleural cavity can also be confirmed sonographically. The ultrasound probe should be placed in the dorsal-most extent of an ICS and held motionless while the lung excursions are observed; as it does inside alveoli, gas free in the pleural cavity will yield reverberation artifact on the monitor. With pneumothorax, however, the parallel white lines of artifact do not move back and forth with the lung, but rather are stationary. Moving the probe ventrally in the same ICS very slowly should reveal a point at which the gliding lung surface enters the ventral side of the frame. That point denotes the extent of the cap of free air in the pleural space. Pneumothorax does not always necessitate intervention; diagnosis of the site of escaping air and very close

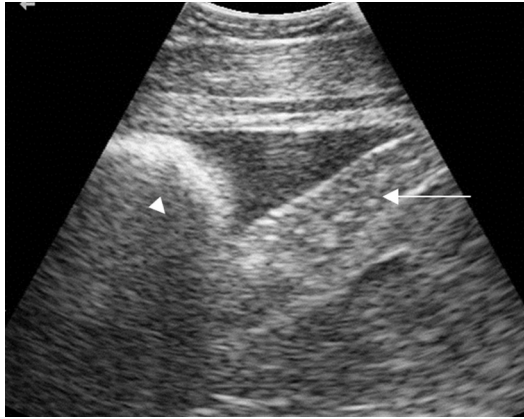
monitoring of the foal are obviously indicated, but clinical signs, anxiety level (the foal's), and blood gas values should be used to determine at what point or whether the air should be removed.

Rib fractures often lacerate small or large intercostal blood vessels along with the parietal pleura and can cause mild or life-threatening hemothorax. The clinical signs associated with internal hemorrhage may actually be the first indication of broken ribs. Active bleeding in either body cavity has a signature sonographic appearance, deemed the smoke sign (Fig. 8). The term refers to the flow pattern of blood exiting an artery or vein: the examiner can usually appreciate constant movement, with the echogenic free fluid swirling into circles and curliques around the lungs and heart. Once a thrombus has formed at the site of bleeding, stationary blood inside a body cavity quickly compartmentalizes, with the heavy, heme-laden erythrocytes settling and being reabsorbed for recycling by the body, and the anechoic serum remaining evident. Scanning at this point reveals only an accumulation of anechoic fluid. It should be mentioned that any fluids with a high cell content have a similar appearance on ultrasound: it is not always possible to differentiate blood from exudate. However, exudate inside a body has no mechanism for self-propagated movement, and the swirling, dynamic appearance of a moving cell-dense fluid inside the chest is strongly suggestive of, if not pathognomonic for, hemothorax.

Laceration of the diaphragm and bowel herniation is another complication of rib fractures. In the author's experience, hemothorax, hemoabdomen, or both should arouse suspicion of diaphragmatic laceration or rupture, and the cavitory hemorrhage may be appreciated as the sole finding, before abdominal organs actually move into the pleural cavity (Fig. 9). Therefore, foals with hemothorax should be scanned carefully for rib fractures at initial evaluation, and the finding also warrants reexamination at least daily for several days afterward to ensure no such additional complications have arisen. The ends of the fractured ribs can be very sharp, and the lung surface is lacerated on these bony projections while sliding dorsally and ventrally during ventilation, leading to bleeding in the lung parenchyma, or pulmonary contusion (Figs. 10 and 11). The most dangerous complication of rib fractures arises when the involved ribs are ribs 4 through 7, those lying adjacent to the heart. Because of the arch of the proximal segments of the ribs, a complete fracture near the costochondral junction results

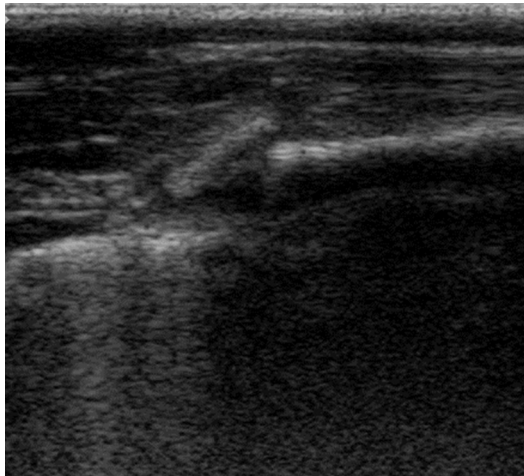


**Fig. 8.** Sonogram of hemorrhage in a foal with rib fractures and diaphragmatic laceration. Notice the swirl and curlique pattern in the echogenic, cell-dense fluid. Movement of the blood giving rise to the swirling in real time is called the smoke sign.



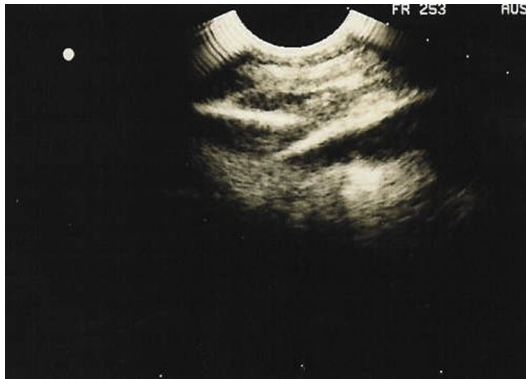
**Fig. 9.** Sonogram from a foal with a diaphragmatic laceration from rib fractures and herniation of large intestine into the pleural space. The curved white echo (*arrowhead*) is that of a viscus lying between the diaphragm (*arrow*) and chest wall. In this view, the lung is displaced to the left and out of view by the viscus. The liver is to the right of the diaphragm, and the pleural cavity is to the left.

in the distal fragment displacing axially, where its tip becomes an instrument of potentially lethal cardiac injury. Cardiac contusion or laceration is usually a fatal and terminal event; sonographic viewing of the heart beating against a sharp bone shard should prompt discussion of surgery with the owners about the option of immobilizing the fractures that put the foal at risk for cardiac puncture. Treatment recommendations for conservative management of fractured ribs involve enforced exercise restriction for varying lengths of time, often 14 to 21 days. Ultrasound is useful in following the organization and maturation of the hematomas that develop over the fractured rib



**Fig. 10.** Sonogram of a rib fracture in a neonatal foal. This rib has fractured at 2 sites, leaving a small floating fragment that is minimally displaced. Even with the modest degree of bony displacement, the comet-tail lesions below the site denote focal hemorrhage in the underlying lung. Dorsal is to the left. Image obtained with a linear array transducer operating at 6 MHz with a display depth of 6 cm.





**Fig. 11.** Sonogram of a fractured rib end piercing and lacerating underlying lung. The heart is protected from similar injury by the lung everywhere but in ribs 3 through 7, where the cardiac notch in the lung lobes leaves the myocardium beating against the parietal pleura and underlying ribs. A fractured rib with this configuration and angle of displacement lying next to the heart would carry a significant risk of fatality.

ends, an important structure in the healing process that cannot be seen in useful detail radiographically; when the hematomas are well-organized and stable, they create a smooth rounded knob that lies between the bone and any neighboring soft tissues, protecting the latter (**Fig. 12**). Formation of a fibrous union between the bone ends can also be verified and followed sonographically (**Fig. 13**).

### ***The Cranial Mediastinum***

The mediastinum is the midline space in the thorax lying between the 2 pleural cavities and created by the medial portions of the left and right parietal pleurae coming into proximity. In the midline space created by these structures lie multiple unpaired structures, including the esophagus, lower trachea, heart, thymus, caudal vena cava, azygous vein, and lymph nodes, among a few other structures. Ultrasonography of the heart, or echocardiography, is a complex undertaking that merits its own treatment and is outside the focus of this article, but basic 4-chamber views of the heart can easily be imaged in the window provided by the cardiac notch, in ICS 3 to 5, in the right lung.

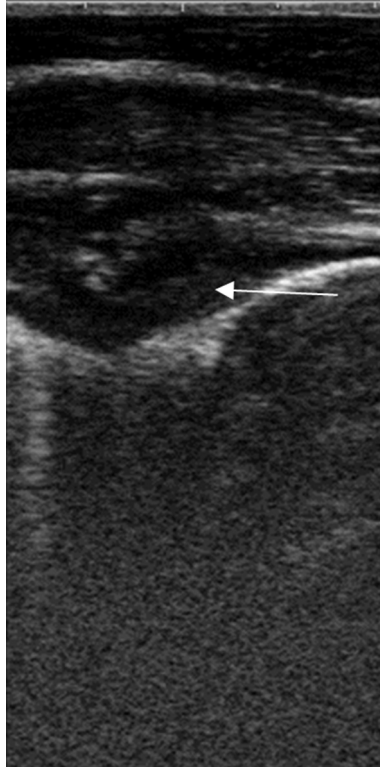
The most common reason for viewing structures in the cranial mediastinal space is to investigate whether there is fluid in the pleural space. In horses with pleuropneumonia, the cranial mediastinum is a site where exudate and infective material can organize into an abscess. In foals and in animals up to about 2 years of age, the thymus can be seen in this site. It is useful to practice imaging this space in both recumbent and standing animals. For a compromised neonatal foal, the cranial mediastinal space can be viewed by lying the foal in left lateral recumbence and gently pulling the right (upper) forelimb forward. The transducer is placed in the right third ICS and aimed toward the opposite elbow and cranially. Effusion in that location will appear as an accumulation of anechoic fluid, while the thymus is a loosely organized hypoechoic soft tissue structure.

## **DISEASES OF THE ABDOMEN**

### ***Gastrointestinal Tract Disease***

The gastrointestinal tract is susceptible to injury resulting from any of the major morbid conditions affecting more seriously compromised newborn foals: prematurity,

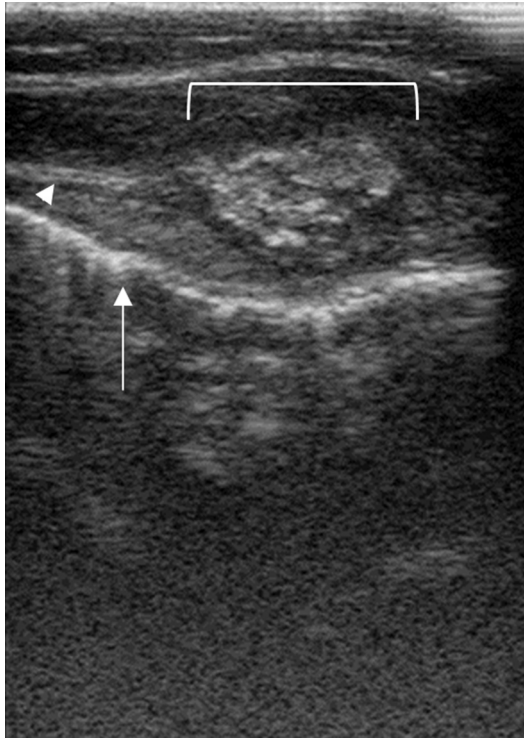




**Fig. 12.** Sonogram of the distracted ends of a fractured rib with a surrounding hematoma in a neonatal foal. The underlying lung is lacerated but is protected from further injury by the moving rib ends by the thick wall of the organizing hematoma (*arrow*). Dorsal is to the left. Image obtained with a linear array transducer operating at 5 MHz with a display depth of 6 cm.

asphyxia-related injury, and septicemia can all induce injury and dysfunction, including diarrhea, in the gastrointestinal tract.<sup>30,31</sup> All can lead to functional failure of the blood-mucosal barrier as well as to motility failure, and these 2 problems are responsible for many of the clinical signs and abnormalities affecting the seriously compromised neonate. The gastrointestinal tract constitutes both a significant potential portal of entry for microbes and a vulnerable target for bacteremic showering by pathogens that entered the body by another route.<sup>31</sup> It is common to find sonographic abnormalities in the abdomen of compromised foals, with or without colic.

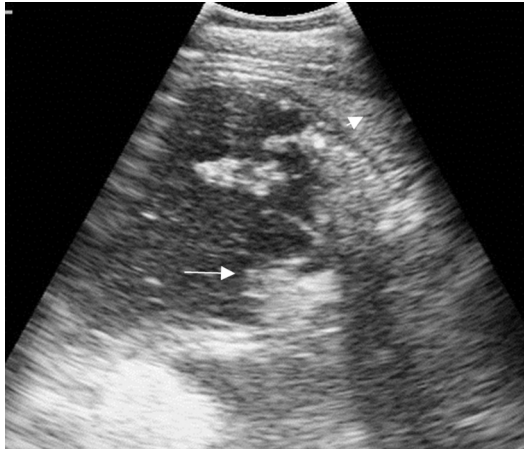
The author approaches sonographic survey of the foal abdomen by imaging all the structures mentioned in the Abdominal Survey section while making sure to record several primary aspects of visceral anatomy and function: the mural width of the small and large intestine walls; the sonographic character of the mural layers, whatever their measured thickness; the nature of the luminal contents; and the contractility patterns, in any discrete segment of interest and in the context of overall tract motility. Particularly with gastrointestinal problems, which can be fulminant and rapidly changing, imaging is most helpful when the foal can be scanned serially over a certain time interval so that change can be detected. Also, the visual observations must be interpreted in light of concurrent changes in physical status, laboratory test values, and response to treatment. That said, foals with colic or other signs referable to the abdomen often do



**Fig. 13.** Sonogram from a foal with several fractured ribs. Shown is the right sixth rib at the fracture site. The hematoma is resorbing, and the fracture ends have formed an early fibrous union (*bracket*). The proximal fragment enters the image from the left (*arrowhead*). The underlying lung still has evidence of contusion injury, with a thickened visceral pleura (*arrow*) and underlying pattern consistent with extravasated blood. Dorsal is to the left. Image obtained with a linear array transducer operating at 5 MHz and with a display depth of 6 cm.

have abnormalities involving the gastrointestinal tract and other abdominal viscera on ultrasound. Surgical and medical lesions in the intestinal tract can manifest very similarly.

The most common indicator of gastrointestinal tract injury from sepsis or hypoxic-ischemic insult in a newborn foal is motility failure. Gastroduodenal ileus results in a distended, hypomotile stomach and duodenum filled with milk or residual fluid that is not proceeding down the tract (**Fig. 14**). When the stomach is distended and atonic, the duodenum will usually be found in a similar state. Normal small intestinal mural thickness in foals is less than or equal to 3 mm,<sup>13,32,33</sup> but any time mural width is scrutinized, the qualitative appearance of the viscus wall should also be noted, even if the mural width is normal. Scanning a bowel segment with a higher-frequency probe is needed to distinguish the layers of the bowel wall, but if available, this should be performed to confirm the following normal echo patterns: hyperechoic mucosal surface–hypoechoic mucosa–hyperechoic submucosa–hypoechoic muscularis propria–hyperechoic serosa.<sup>29,32</sup> Appearance of hypoechoic edema in the bowel wall even before there is thickening of the overall dimensions may be seen with strangulating lesions, and the appearance of hyperechoic gas echoes within the wall layers is diagnostic for pneumatosis intestinalis and necrotizing enterocolitis.<sup>34</sup>



**Fig. 14.** Sonogram from a neonatal foal with enterocolitis and delayed gastric emptying. An interface between curdled milk (*arrow*) and gas-flecked hypoechoic fluid can be seen causing moderate gastric distension. The spleen (*arrowhead*) is to the right of the stomach in this image. Image obtained with a macroconvex transducer operating at 5 MHz with a display depth of 14 cm.

Detection of distension and delayed emptying of the stomach and duodenum help the veterinarian make several management decisions. First, the distension and atony necessitate placement of an indwelling feeding tube to facilitate decompression every few hours, and enteral feeding to be restricted or withheld, until contractility is restored. Persistent gastric engorgement and absence of propulsive contractions would also prompt addition of partial or total parenteral nutrition, prokinetic agents, gastroprotectants, and antiulcer medications to the regimen, in addition to the antimicrobials, intravenous fluids, and other interventions the foal was receiving. Incorporating imaging as part of morning and evening physical examinations can visually confirm the return of contractility, and the foal's management is modified accordingly.

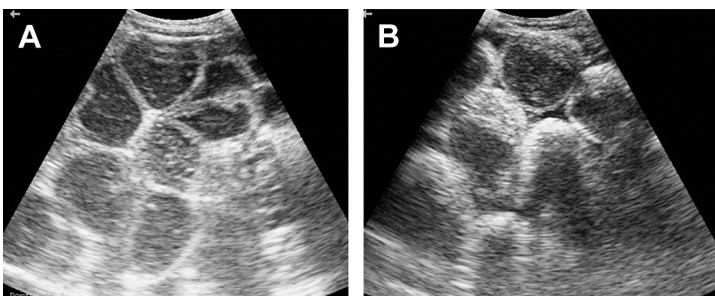
Gastritis and ulcer formation in neonatal foals are a multifactorial and complex clinical entity, discussion of which is outside the scope of this article, but visceral hypoperfusion secondary to sepsis-associated hypotension or from failure to keep up with the foal's fluid needs, to name 2 examples, likely constitute risk factors for this complication that are unrelated to acidity alone, as ulcers can develop and progress to the point of gastric perforation even while foals are receiving proton pump inhibitors. Reflux of duodenal fluids into the stomach may also injure the mucosa by contact with bile acids. Ulcer formation proceeding to gastric perforation can be clinically silent, especially in recumbent, obtunded foals. Gastric endoscopy is the only definitive method for diagnosing gastric ulcers, but sonographic imaging of the area in the left cranial abdomen where the liver, stomach, and spleen lie in contact can reveal gastric wall edema and serositis as local effects of severe ulcerative gastritis and incipient perforation (**Fig. 15**).

Bacterial and viral pathogens can cause enteritis or enterocolitis in foals beginning on the first day of postnatal life. The sonographic hallmark of enteritis of any cause is the combination of distension, hypomotility (although hypermotility may be seen in the prodromal stages), intestinal walls of normal or thickened width, and hypoechoic-to-echogenic gassy luminal contents (**Fig. 16**). With severe enteritis, transudative-to-exudative fluid may also accumulate in the peritoneal fluid and can set the foal



**Fig. 15.** Sonogram from the cranial region of the left side of a foal's abdomen, in rib spaces 9 and 10. This view features the area where the stomach and spleen lie in proximity and reveals local peritonitis with fine fibrinous adhesions forming between the stomach and spleen. The gastric wall (*bracket*) is thickened with edema and serositis from an ulcer in the process of perforating. This image was obtained with a macroconvex transducer operating at 5 MHz with a display depth of 14 cm.

up for fibrous adhesions in the weeks to months following clinical resolution of the enteritis. The distended small intestine segments are easy to find and form the dominant feature in the affected foal's abdomen. In the prodromal phase of infection, hypermotility rather than hypomotility may be appreciated. The intestinal walls are often of normal width, but may also have an inflamed appearance and be thicker than normal. Enteritis and enterocolitis in newborn foals are severe systemic diseases, attended by dehydration, pain, and marked acid-base and metabolic alterations. The most common causes in newborn foals include bacterial infection (*Clostridium*

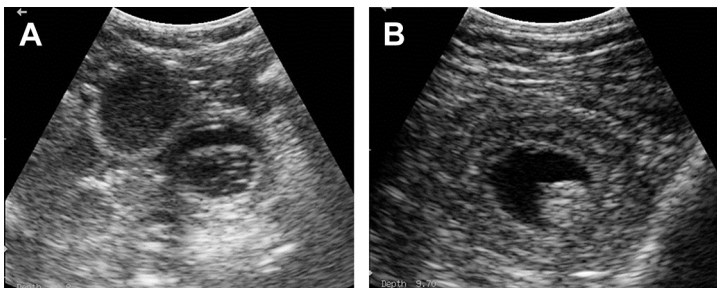


**Fig. 16.** Sonograms from neonatal foals with small intestinal disease. (A) Jejunal segments in a 2-day-old foal with enterocolitis caused by *C. perfringens* type A. Although views of jejunum are not always easily seen in healthy neonates, small intestine distension displaces other viscera and makes the affected segments easy to detect. (B) Jejunal segments from a 1-week-old foal with small intestine volvulus. In this foal, the intestine walls have not become severely thickened from venous congestion, but the effects of profound ileus can be appreciated in the settling out of the solids in the luminal contents. The top portion of the image represents the abdominal floor. Both images were obtained with a convex transducer operating at 5 MHz with a display depth of 15 cm.

*perfringens*, *Clostridium difficile*, *Enterococcus durans*, and *Salmonella* spp) and viral infection (rotavirus, coronavirus). Less common microbial pathogens that have been reported in diarrheic foals include *Aeromonas hydrophila*, *Bacteroides fragilis*, *Cryptosporidium parvum*, and adenovirus.<sup>30</sup> Blood work in foals with enteritis usually features leukopenia with neutropenia, a feature of the mucosal barrier compromise, and fever is often also found, although fever in neonatal foals is not a reliable clinical finding, and its absence should not be relied on to rule out serious illness.

The sonographic appearance of strangulated small intestine is similar to that of enteritis or enterocolitis,<sup>35</sup> but venous congestion in the strangulated bowel wall will lead to a progressive increase in mural width and engorgement of mesenteric vasculature over time (see Fig. 16). Causes of intestinal strangulation in neonatal foals include volvulus; incarceration through a mesenteric rent or by a congenital anomaly such as Meckel diverticulum; and herniation through the inguinal ring or a rupture in the diaphragm. Because the initial appearance of strangulated small intestine may closely resemble that of enteritis, it is necessary to incorporate additional information into the evaluation in determining whether the foal requires surgical intervention. Analysis of peritoneal fluid, blood work, physical status, and response to analgesics is necessary for making this determination.

Intestinal intussusception has been well described as a cause of colic in foals, but this disorder is more common in foals several months of age and older than in neonatal foals. Development of intussusception arises from differences in motility between neighboring segments of bowel and may be associated with many causes, including enteritis, administration of the prokinetic neostigmine, and parasitism. Intussuscepted intestine has a typical and easily recognizable sonographic appearance formed from the concentric layering of one bowel segment (the intussusceptum) inside an outer segment (the intussusciens; Fig. 17).<sup>36,37</sup> Intussusceptions can be jejunojejunal, ileal-ileal, ileocecal, cecocolic, or cecocecal.<sup>37</sup> Clinical signs and severity of colic resulting from intussusception depend on length of involved bowel segment and whether the mesenteric blood vessels attached to the intussusceptum are obstructed. It is generally accepted that diagnosis of an intussusception automatically warrants surgical correction, and in older foals and horses, this is likely true. However, a recent report<sup>33</sup> detailing the existence of intussusceptions as an incidental finding in healthy, asymptomatic standardbred foals confirms that in neonates this is not always true. The findings in that study corroborate observations by the author, who has observed



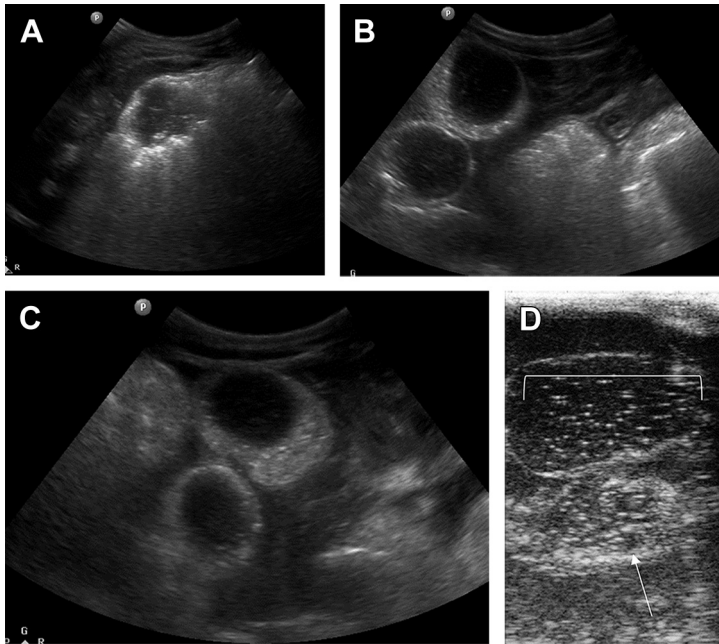
**Fig. 17.** Sonograms of 2 types of intussusception. (A) Jejunojejunal intussusception in a hospitalized neonate. This image was obtained in the caudal abdomen of the foal while it was positioned in right lateral recumbence. (B) Cecocolic intussusception, with the tip of the cecum inverted into the cecal body. This image was obtained in a yearling colt but is included here for example. Image obtained near ventral midline with horse standing.

jejunojejunal intussusceptions arising in multiples in hospitalized foals and resolving spontaneously (unpublished data, 2012).

Most causes of colic in neonatal foals are medical problems of the gastrointestinal tract. In a recent retrospective study<sup>35</sup> of colic in 137 neonatal foals (defined as <30 days of age; median age, 2 days) at a university referral hospital, 89% of the foals had conditions that were managed medically. For all 137 foals, enterocolitis, meconium-related colic, and transient colic for which a cause was not determined but which was managed medically were the 3 most common diagnoses. In the 11% of foals that underwent surgery for colic, small intestine strangulating obstruction was the most frequent diagnosis, with volvulus, intussusception, and mesenteric rent comprising most of these cases.

Meconium, a sterile concretion of intestinal cells and secretions, accumulates in the intestine during gestation and becomes sufficiently tenacious or inspissated in some foals that it becomes impacted in the large intestine or rectum and causes obstruction. In meconium impaction or retention, ultrasound is sensitive at detecting the impacted colon segment and the length or extent of the retained luminal material (Fig. 18). Change in the appearance of the mass can be monitored sonographically after administration of enteral fluids or an enema. The affected colon segments and retained pellets or logs are usually detected in the caudal part of the abdomen. In the 2013 retrospective mentioned previously,<sup>35</sup> ultrasound was helpful in distinguishing large intestinal from small intestinal disease, and, within a portion of the intestine, types of disease. Distended, hypodynamic small intestine was seen more frequently with strangulating lesions, enterocolitis, and necrotizing enterocolitis than in those with meconium-associated colic. Within the large intestine, fluid distension was seen more frequently in foals with enterocolitis and necrotizing enterocolitis than in those with meconium problems. Thickening of the bowel wall was also sensitively detected: mural thickening in the small intestine was seen with strangulating obstructions, enterocolitis, and necrotizing enterocolitis, but not with meconium-associated, transient medical colic, or forms of colic deemed "other". Thickening of the large intestine wall was seen only in foals with necrotizing enterocolitis. The fact that mural thickening was seen in foals with strangulating small intestinal lesions and those with inflammatory bowel lesions underscores how sonography is useful at detecting the site of abnormality but must be supported with physical examination and clinicopathologic data in the process of making a definitive diagnosis in these types of diseases. In the scenario of a colicky neonatal foal with thickened small intestine walls, for example, fever, a low white cell count, abnormal serum electrolyte values, and fluid accumulating in both the small and the large intestine would point toward enterocolitis. Severity of pain and response to pain controlling medications are also clinically useful. In this study,<sup>35</sup> neonates with a strangulating small intestine lesion (usually a volvulus) were significantly more likely to have severe pain and continuous pain than foals with medical causes of colic, and fewer foals with strangulating lesions responded to analgesics, compared with those that had medical colic.

Recently, a protocol of focused abdominal scanning designed for use in emergent settings was assessed in horses with colic.<sup>38</sup> In this protocol, abdominal imaging is limited to assessing visceral structures and free peritoneal fluid volume at 7 topographic locations. The technique is deemed FLASH (fast localized abdominal sonography in horses) and has conceptual precedent in human<sup>39</sup> and small animal<sup>40</sup> emergency medicine. The 7 designated imaging windows were selected on the basis of earlier reports of frequent locations for detection of sonographic abnormalities in horses with colic. In this 2011 report of horses with colic,<sup>38</sup> use of the focused technique was quickly learned by individuals who did not have extensive ultrasound



**Fig. 18.** Sonograms of large intestine in a neonatal foal with meconium-associated colic. (A) Longitudinal view of the bowel segment obstructed by the meconium, which is the hypoechoic material with the oblong shape surrounded by intestinal wall that is contracted around the material. Meconium is sonographically hypoechoic to anechoic, despite its solid consistency. (B) Same area as in (A). The thin layer of echogenic material surrounding the anechoic meconium balls is luminal fluid beginning to accumulate after enteral administration of water and mineral oil and administration of a retention enema. (C) The impacted accumulations of meconium are smaller, and the volume of fluid surrounding them in the colonic lumen has increased in the hour since the image in (B) was taken. This indicates response to treatment and the process of resolution of the meconium retention. (D) The meconium (*bracket*) in this image has a more speckled, echogenic appearance on ultrasound. This image was obtained with a linear array transducer in a foal with hemoabdomen resulting from rib fractures and diaphragm laceration. A thrombus is represented by the echogenic clot lying against the deep surface of this meconium ball (*arrow*).

experience and enabled quick (mean examination time, 10.7 minutes) evaluation and acceptable sensitivity, specificity, and predictive values in horses whose clinical condition made performing a complete abdominal survey impossible. Use of this focused protocol in neonatal foals has not been reported, but will likely be investigated in these patients in the future.

### **Urinary Tract Abnormalities**

Considerable overlap can exist among the clinical signs of gastrointestinal and urinary tract disease. Colic and diarrhea can be seen in diseases of both systems, and ultrasound can help distinguish between gastrointestinal and urinary tract disease. Transitioning of the urogenital system from fetal to postnatal life at the time of birth involves abrupt changes in the umbilical remnant structures, as blood flow ceases entering and exiting the body through the umbilical cord and the roles of urine formation and



handling are transferred from the placenta to the foal's organs. The umbilical cord should rupture cleanly a short distance from the foal's abdominal wall, leaving a 1" to 2" stump,<sup>41</sup> at which time the vascular structures inside the cord immediately contract and begin occluding further blood flow. Inside the umbilical cord are 4 clinically important structures that can be assessed sonographically: 2 umbilical arteries, 1 umbilical vein, and the urachus. In gestation, these structures are anatomically independent, but are bundled into a cord by an outer adventitial covering.

In thoroughbred foals, the maximum normal cord length has been determined to be less than 84 cm (range, 36–83 cm; mean, 55 cm), with up to 4 twists along its length.<sup>42</sup> Cord length is of clinical relevance because an abnormally long cord can become excessively torsed or wrapped around the fetus' trunk or limbs in utero, strangulating or impinging the structures within it. Upstream dilatation and pressure in the urachus and bladder created by cord impingement can predispose to higher-than-normal intravesicular volume, and bladder rupture, during parturition. A short umbilical cord increases traction on the placenta during parturition and may result in premature placental separation and death for the foal if a human attendant is not present. Short cord length can also create tension on the foal's umbilical area and urachus during and after transit through the birth canal, increasing the risk for tearing and urine leakage postpartum. A subjective appreciation for excessive umbilical cord length can be obtained during sonographic monitoring of the gestating mare. Umbilical cord abnormalities in utero thus can have an important influence on events involving the urogenital system in the neonate.

In the fetal vasculature, the umbilical arteries arise as branches of the pudendal arteries, which are branches of the internal iliac arteries. From their origins on the pudendal arteries in the dorsocaudal aspect of the abdomen, the umbilical arteries flow cranially and ventrally toward the bladder, where each travels on its respective side along the lateral aspect of the bladder. From its entry site at the umbilical stalk, the umbilical vein courses cranially along abdominal midline and into the liver. In the weeks following birth, the umbilical vein atrophies and becomes the cordlike round ligament of the liver, running through the fatty connective tissue of the falciform ligament. The umbilical arteries also undergo involution and atrophic change and become the round ligaments of the bladder. Unlike these vascular remnants, the urachus is not a walled tubular structure, but is merely the potential space running between and around the umbilical blood vessels. In the fetus, the urachus is the conduit for urine flow from fetal bladder to the allantoic cavity. At the time of birth, it should involute and close, and the foal should begin voiding urine through the urethra.

Even though the remnant umbilical arteries course caudally and dorsally toward the bladder, because the bladder lies so close to the ventral abdominal wall in neonates, these structures are still quite superficial with regard to imaging depth. High-resolution images are needed for determining the dimensions of small structures such as arteries and vein, and this is one study in which the author routinely clips the hair. Clipping should include the area around the base of the umbilicus, between the base and the groin, and between the base and the liver. Foals can be scanned standing, in lateral recumbence, or in a semi-dorsal recumbent position. Imaging frequencies in the range of 6 MHz to 10 MHz will yield useful images for this study.

The umbilical arteries and vein should be imaged along their full length. The umbilical vein is observed in the transverse plane and longitudinal plane running from its origin at the umbilical base cranially toward the liver. It should be less than 1 cm in diameter throughout its length, with some narrowing about halfway between the base and the point where the vein merges into the hepatic tissue.<sup>32,43</sup> The arteries are observed by placing the probe at the umbilical base on its caudal surface. At



this juncture, the arteries and urachus are seen together in an oblong grouping, and the long-axis diameter there should be less than 2.5 cm. Moving the probe caudally along midline, the urachus will disappear, and the arteries will diverge laterally along the bladder walls. When imaged separately, each umbilical artery remnant in transverse section should be less than 1 cm. It is easier to discern the arteries when the bladder is partially full. Of the structures comprising the umbilical remnant, the urachus is the most commonly infected.<sup>44</sup> Infection appears as soft tissue or echogenic material in the urachal lumen or occupying the tissue spaces around the arteries or urachus. Well-established umbilical infections come to notice by gross thickening, edema, or cellulitis surrounding the umbilical base. Exudate may be dripping from the stalk, or infection may have caused the urachus to become patent again and drip urine. Infection that tracks internally can cause cellulitis of the subcutaneous tissues surrounding the stalk and peritonitis. Internal structures can be infected and have significant sonographic abnormalities even if the external portion of the stalk appears normal.

Sonographic imaging is of great benefit in detecting sites of rupture in the urinary tract and infection of the internal umbilical remnants, the most common causes of urinary tract disease affecting neonates. Leaking of urine into the peritoneal cavity can result from rupture of the ureters, bladder, urethra, or urachus. Underlying conditions that can cause leak are parturition-associated trauma, congenital anomalies, postpartum traumatic injury, strenuous exercise, focal necrosis of the bladder wall, and urachal infection.<sup>45,46</sup>

Foals with uroperitoneum present with one or more of the following well-documented clinical signs: colic, tenesmus secondary to constipation, tachycardia, altered mucous membrane appearance, dehydration, diarrhea, failure to nurse, lethargy, abdominal distension, frequent urinary posturing, and stranguria. Affected foals may also have rapid, shallow breathing secondary to hampering of diaphragmatic contraction by the volume of fluid in the abdomen.<sup>47</sup> Foals usually continue to pass some urine through the urethra as well as through the bladder rent into the abdomen, and it often takes several days after birth for the abdominal distension to become marked enough to prompt veterinary evaluation. When the rupture is in a ureter, the uroperitoneum develops more slowly and takes longer to manifest clinically. The hallmark sonographic appearance of uroperitoneum is voluminous anechoic to hypoechoic fluid in which viscera and mesentery are floating. Some foals with uroperitoneum also develop pleural effusion.<sup>48</sup> If the uroperitoneum is chronic or is associated with rupture of an infected or necrotic urachus, the peritoneal fluid will become more cellular in appearance with the influx of leukocytes. It is not always possible to make out the tear site in the bladder wall, but it can be appreciated in some instances.

The gender predilection toward male foals traditionally reported<sup>49</sup> was not found in 2 more recent retrospective studies on uroperitoneum in foals.<sup>50,51</sup> Moreover, the clinical presentation and serum biochemical abnormalities associated with uroperitoneum may be different in foals hospitalized for sepsis or hypoxic-ischemic injury, in which uroperitoneum arises as one of multiple systemic abnormalities, than they are in foals referred to a hospital with uroperitoneum as the primary complaint.<sup>50</sup> In hospitalized foals, clinical signs can be masked by a neurologically depressed state, and administration of intravenous fluids prevents or blunts the hyponatremia, hypochloremia, and hyperkalemia that have traditionally been strongly associated with uroperitoneum.<sup>49,52</sup> Administration of fluids does not impact the development of high serum creatinine concentration or the usefulness of determining the peritoneal fluid:serum creatinine ratio, with a ratio 2 or greater being diagnostic for uroperitoneum. It is likely that

more prevalent use of diagnostic ultrasound has resulted in diagnosis and intervention before the full gamut of biochemical changes can develop. The bladder is the most common site of rupture in the tract, followed by the urachus. In the 2005 retrospective study<sup>51</sup> cited in these paragraphs, the rent in the bladder wall was ventral at about the same frequency as it was dorsal.

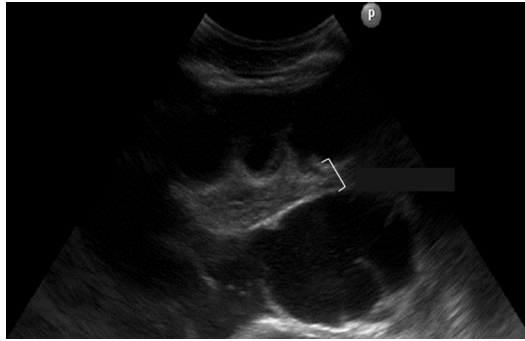
Sepsis and focal infections, in the urogenital tract<sup>53</sup> and elsewhere in the body, are important risk factors for uroperitoneum, irrespective of whether a foal is hospitalized.<sup>50,51</sup> In these foals, rupture of the bladder or urachal remnant is related to infection or necrosis rather than to traumatic rupture during parturition. It is not uncommon to detect uroperitoneum in a septic or bedridden foal one or more days into hospitalization when it was not present at admission. It is advisable to place and properly maintain an indwelling urinary catheter in nonambulatory foals to prevent urine stasis and pressure in the bladder, and broad-spectrum antimicrobial coverage would be an expected part of the treatment regimen of any foal in this state. Daily to twice-daily sonographic imaging of the abdomen during the course of hospitalization enables detection of even a modest volume of free peritoneal fluid, days before abdominal distension, colic, or some of the other clinical signs become evident. The bladder is the most common site of rupture in the tract, followed by the urachus. In the 2005 retrospective study<sup>51</sup> cited in these paragraphs, the rent in the bladder wall was ventral at about the same frequency as it was dorsal. Excellent practical guides on scanning technique for the umbilical remnants are available.<sup>54</sup>

The term navel ill has traditionally been used in reference to septic polyarthritis thought to result from pathogens' gaining entrance to the body through the umbilicus. Although the umbilicus can indeed serve as an access point for pathogens, it is now recognized that the gastrointestinal tract and other portals are often the chief entry point, and the umbilical structures can become infected by bacteremic showering along with other tissues. The most common reported bacterial isolates from omphalitis or omphalophlebitis specimens are *Escherichia coli* and *Streptococcus zooepidemicus*.<sup>44,55,56</sup> Infection with *C. perfringens* has also been reported, as a result of either direct invasion from the environment or bacterial translocation from the intestinal tract.<sup>55</sup> A 2007 report<sup>57</sup> revealed *Clostridium sordelli* as a cause of fulminant omphalitis, peritonitis, and death in 8 foals.

Bladder rupture is nearly always managed by surgery and primary closure of the rent. Dehiscence and rerupture in the few days following surgery are not uncommon, and routine placement of an indwelling urinary catheter for 3 to 5 days after surgery may keep the bladder wall free of tension and challenge to the suture line. Conservative management of bladder ruptures also involves placement of an indwelling catheter and a long-term regimen of antimicrobials and has yielded favorable results.<sup>58</sup> It is tempting to surmise that this approach is most likely to yield success when the rent is not large and when it involves the dorsal aspect of the bladder. This option may be feasible when the owners are not able to finance abdominal surgery, and the bladder rent involves the dorsal wall and is modest in size. Nonsurgical management of bladder rupture in 4 adult horses was recently reported.<sup>59</sup>

Ultrasound imaging can distinguish between other problems affecting the lower urinary tract and resulting in clinical signs that overlap with those of uroperitoneum. Hematoma formation within the bladder, ascribed to tearing of the intra-abdominal portion of the umbilicus, has been reported and is not an uncommon finding.<sup>60</sup> In some instances, however, the resulting thrombus can be large and obstruct urine flow, leading to signs of stranguria in the foal.

Megavesica is another cause of abdominal enlargement and stranguria, but not uroperitoneum, although it is speculated that some cases of bladder rupture occur



**Fig. 19.** Sonogram of the right kidney of a foal with renal agenesis. A central band of stroma representing the renal pelvis can be seen (*bracket*), but most of the renal tissue is replaced by anechoic fluid. The foal's left kidney was sonographically normal.

secondary to undetected megavesica.<sup>61,62</sup> Foals with this syndrome of bladder atony and enlargement have been reported, but the cause is not understood. Urine dribbles from the urethra either passively or during straining, and ultrasound reveals a grossly dilated but intact bladder distended with urine.

The kidneys are easily imaged in foals. Renal dysfunction is not unusual in foals, but is usually caused by shock, hypoperfusion, septic injury, and toxicosis from administration of nephrotoxic medications. Congenital renal conditions are uncommon in horses, but polycystic kidney disease and renal agenesis (**Fig. 19**) and renal/glomerular hypoplasia<sup>63,64</sup> are occasionally reported. A recent case report<sup>61</sup> detailed a diffuse, bilateral alteration of renal parenchyma that was histologically characterized as diffuse cystic renal dysplasia in a 9-day-old thoroughbred foal. The morphologic change in the renal parenchyma was detected with ultrasonography and confirmed at necropsy. Kidneys can have an unremarkable sonographic appearance while being in functional failure; clinicopathologic testing should be done as the primary means of determining renal function.

## SUMMARY

Sonographic imaging brings a visual component to the information database for a compromised neonatal foal that can be invaluable for confirming or ruling out many of the differential diagnoses that could be causing the observed clinical or clinicopathologic alterations in the foal. Ultrasound equipment has grown more affordable and more portable in the past decade, and equine practitioners can significantly augment the level of patient care they provide by gaining facility with this safe, fast, noninvasive, and highly informative imaging modality.

## REFERENCES

1. Barba M, Lepage OM. Diagnostic utility of computed tomography imaging in foals: 10 cases (2008–2010). *Equine Vet Educ* 2013;25:29–38.
2. Lascola KM, O'Brien RT, Wilkins PA, et al. Qualitative and quantitative interpretation of computed tomography of the lungs in healthy neonatal foals. *Am J Vet Res* 2013;74:1239–46.
3. Schliwert EC, Lascola KM, O'Brien RT, et al. Comparison of radiographic and computed tomographic images of the lungs in healthy neonatal foals. *Am J Vet Res* 2015;76:42–52.

4. Pion L, Perkins G, Ainsworth SM, et al. Use of computed tomography to diagnose a *Rhodococcus equi* mediastinal abscess causing severe respiratory distress in a foal. *Equine Vet J* 2001;33:523–6.
5. Lascola KM, Joslyn S. Diagnostic Imaging of the Lower Respiratory Tract in Neonatal Foals: Radiography and Computed Tomography. *Vet Clin Equine* 2015, in press.
6. Fenton MB. The world through a bat's ear. *Science* 2011;333:528–9.
7. Bates ME, Simmons JA, Zorikov TV. Bats use echo harmonic structure to distinguish their targets from background clutter. *Science* 2011;333:627–30.
8. Nickel R, Schummer A, Seiferle E. The viscera of the domestic mammals. 2nd rev edition. Berlin: Springer Verlag; 2013. p. 3–6.
9. Schambourg MA, Laverty S, Mullim S, et al. Thoracic trauma in foals: post mortem findings. *Equine Vet J* 2003;35:78–81.
10. Jean D, Laverty S, Halley J, et al. Thoracic trauma in newborn foals. *Equine Vet J* 1999;31:149–52.
11. Sprayberry KA, Bain FT, Seahorn TL, et al. 56 Cases of rib fractures in newborn foals hospitalized in a referral center intensive care unit from 1997 to 2001. *Proc Am Assoc Equine Pract* 2001;47:395–9.
12. Jean D, Picandet V, Macieira S, et al. Detection of rib trauma in newborn foals in an equine critical care unit: a comparison of ultrasonography, radiography, and physical examination. *Equine Vet J* 2007;39:158–63.
13. Aleman M, Gillis CL, Nieto JE, et al. Ultrasonographic anatomy and biometric analysis of the thoracic and abdominal organs in healthy foals from birth to age 6 months. *Equine Vet J* 2002;34:649–55.
14. Sanchez LC, Giguere S, Lester GD. Factors associated with survival of neonatal foals with bacteremia and racing performance of surviving thoroughbreds: 423 cases (1982–2007). *J Am Vet Med Assoc* 2008;233:1446–52.
15. Stewart AJ, Hinchcliff KW, Saville WJ, et al. *Actinobacillus* sp bacteremia in foals: clinical signs and prognosis. *J Vet Intern Med* 2002;16:464–71.
16. Freeman L, Paradis MR. Evaluating the effectiveness of equine neonatal care. *Vet Med* 1992;87:921–6.
17. Marsh PS, Palmer JE. Bacterial isolates from blood and their susceptibility patterns in critically ill foals: 543 cases (1991–1998). *J Am Vet Med Assoc* 2001; 218:1608–10.
18. Hong CB, Donahue JM, Giles RC, et al. Etiology and pathology of equine placentitis. *J Vet Diagn Invest* 1993;5:56–63.
19. Ware LB. Pathophysiology of acute lung injury and the acute respiratory distress syndrome. *Sem Respir Crit Care Med* 2006;27:337–46.
20. Wheeler AP, Bernard GR. Acute lung injury and the acute respiratory distress syndrome: a clinical review. *Lancet* 2007;369:1553–65.
21. Sande RD, Tucker RL. Radiology of the equine lungs and thorax. In: Rantanen NW, Hauser ML, editors. The diagnosis and treatment of respiratory disease. *Proc Dubai Int equine symp.* San Diego (CA): Neyenesch Printers Inc; 1997. p. 139–57.
22. Nykamp SG. The equine thorax. In: Thrall DE, editor. *Textbook of veterinary diagnostic radiology*. 6th edition. St Louis (MO): Elsevier Saunders; 2013. p. 632–48.
23. Dunkel B, Dolente B, Boston RC. Acute lung injury/acute respiratory distress syndrome in 15 foals. *Equine Vet J* 2005;37:435–40.
24. Dunkel B. Acute lung injury and acute respiratory distress syndrome in foals. *Clin Tech Equine Pract* 2006;5:127–33.
25. Wilkins PA, Otto CM, Baumgardner MD, et al. Acute lung injury and acute respiratory distress syndromes in veterinary medicine: consensus definitions: The

- Dorothy Russell Havemeyer Working Group on ALI and ARDS in Veterinary Medicine. *J Vet Emerg Crit Care* 2007;17:333–9.
26. Bernard GR, Artigas A, Brigham KL, et al. The American-European consensus conference on ARDS, definitions, mechanisms, relevant outcomes and clinical trial coordination. *Am J Respir Crit Care Med* 1994;149:818–24.
  27. Peek S, Landolt G, Karasin AI, et al. Acute respiratory distress syndrome and fatal interstitial pneumonia associated with equine influenza in a neonatal foal. *J Vet Intern Med* 2004;18:132–4.
  28. Patterson-Kane JC, Carrick JB, Axon JE, et al. The pathology of bronchointerstitial pneumonia in young foals associated with the first outbreak of equine influenza in Australia. *Equine Vet J* 2008;40:199–203.
  29. Porter M, Ramirez P. Equine neonatal thoracic and abdominal ultrasonography. *Vet Clin North Am Equine Pract* 2005;21:407–29.
  30. Magdesian KG. Neonatal foal diarrhea. *Vet Clin North Am Equine Pract* 2005;21:295–312.
  31. Hollis AR, Wilkins PA, Palmer JE, et al. Bacteremia in equine neonatal diarrhea: a retrospective study (1990–2007). *J Vet Intern Med* 2008;22:1203–9.
  32. Reef VB. Pediatric abdominal ultrasonography. In: Reef BV, editor. *Equine diagnostic ultrasound*. Philadelphia: WB Saunders Company; 1998. p. 364–403.
  33. Abraham M, Reef VB, Sweeney RW, et al. Gastrointestinal ultrasonography of normal Standardbred neonates and frequency of asymptomatic intussusceptions. *J Vet Intern Med* 2014;28:1580–6.
  34. de Solis Navas C, Palmer JE, Boston RC, et al. The importance of ultrasonographic pneumatosis intestinalis in equine neonatal gastrointestinal disease. *Equine Vet J* 2012;44(Suppl):64–8.
  35. MacKinnon MC, Southwood LL, Burke MJ, et al. Colic in equine neonates: 137 cases (2000–2010). *J Am Vet Med Assoc* 2013;243:1586–90.
  36. Bernard WV, Reef VB, Reimer JM, et al. Ultrasonographic diagnosis of small-intestinal intussusception in three foals. *J Am Vet Med Assoc* 1989;194:395–7.
  37. McGladdery AJ. Ultrasonographic diagnosis of intussusception in foals and yearlings. *Proc Am Assoc Equine Pract* 1996;40:239–40.
  38. Busoni V, De Busscher V, Lopez D, et al. Evaluation of a protocol for fast localized abdominal sonography of horses (FLASH) admitted for colic. *Vet J* 2011;188:77–82.
  39. Soundappan SVS, Holland AJA, Cass DT, et al. Diagnostic accuracy of surgeon-performed focused abdominal sonography (FAST) in blunt paediatric trauma. *Injury* 2005;36:970–5.
  40. Boysen SP, Rozanski EA, Tidwell AS, et al. Evaluation of a focused assessment with sonography for trauma protocol to detect free abdominal fluid in dogs involved in motor vehicle accidents. *J Am Vet Med Assoc* 2004;225:1198–204.
  41. Morresey PR. Umbilical problems. *Proc Am Assoc Equine Pract* 2014;60:18–21.
  42. Whitwell KE. Morphology and pathology of the equine umbilical cord. *J Reprod Fertil Suppl* 1975;(23):599–603.
  43. Reef VB, Collatos C. Ultrasonography of umbilical structures in clinically normal foals. *Am J Vet Res* 1988;49:2143–6.
  44. Reef VB, Collatos C, Spencer PA, et al. Clinical, ultrasonographic, and surgical findings in foals with umbilical remnant infections. *J Am Vet Med Assoc* 1989;195:69–72.
  45. Hackett RP. Rupture of the urinary bladder in neonatal foals. *Compend Cont Educ Pract Vet* 1984;6:S488–91.
  46. Robertson JT, Embertson RM. Surgical management of congenital and perinatal abnormalities of the urogenital tract. *Vet Clin North Am Equine Pract* 1988;4:359–79.

47. Wilkins PA. Respiratory distress in foals with uroperitoneum: possible mechanisms. *Equine Vet Educ* 2004;16:293–5.
48. Wong DM, Leger LC, Scarratt WK, et al. Uroperitoneum and pleural effusion in an American Paint filly. *Equine Vet Educ* 2004;16:290–3.
49. Richardson DW, Kohn CW. Uroperitoneum in the foal. *J Am Vet Med Assoc* 1983;182:267–71.
50. Kablack KA, Embertson RM, Bernard WV, et al. Uroperitoneum in the hospitalised equine neonate: retrospective study of 31 cases, 1988–1997. *Equine Vet J* 2000;32:505–8.
51. Dunkel B, Palmer JE, Olson KN, et al. Uroperitoneum in 32 foals: influence of intravenous fluid therapy, infection, and sepsis. *J Vet Intern Med* 2005;19:889–93.
52. Behr MJ, Hackett RP, Bentinck-Smith J, et al. Metabolic abnormalities associated with rupture of the urinary bladder in neonatal foals. *J Am Vet Med Assoc* 1981;178:263–6.
53. Lores M, Lofstedt J, Martinson S, et al. Septic peritonitis and uroperitoneum secondary to subclinical omphalitis and concurrent necrotizing cystitis in a colt. *Can Vet J* 2011;52:888–92.
54. Franklin RP, Ferrell EA. How to perform umbilical sonograms in the neonate. *Proc Am Assoc Equine Pract* 2002;48:261–5.
55. Hyman SS, Wilkins PA, Palmer JE, et al. *Clostridium perfringens* urachitis and uroperitoneum in 2 neonatal foals. *J Vet Intern Med* 2002;16:489–93.
56. Adams SB, Fessler JF. Umbilical cord remnant infections in foals: 16 cases (1975–1985). *J Am Vet Med Assoc* 1987;190:316–8.
57. Ortega J, Daft B, Assis RA, et al. Infection of the intestinal umbilical remnant in foals by *Clostridium sordelli*. *Vet Pathol* 2007;44:269–75.
58. Lavoie JP, Harnagel SH. Nonsurgical management of ruptured urinary bladder in a critically ill foal. *J Am Vet Med Assoc* 1998;192:1577–80.
59. Peitzmeier MD, McNally TP, Slone DE, et al. Conservative management of cystorrhoeis in four adult horses. *Equine Vet Educ* 2015. <http://dx.doi.org/10.1111/eve.12321>.
60. Arnold CE, Chaffin KM, Rush BR. Hematuria associated with cystic hematomas in three neonatal foals. *J Am Vet Med Assoc* 2005;227:778–80.
61. Rijkenhuizen A. Megavesica and bladder rupture in foals. *Equine Vet Educ* 2012;24:404–7.
62. Toth T, Liman J, Larsdotter S, et al. Megavesica in a neonatal foal. *Equine Vet Educ* 2012;24:396–403.
63. Brown CM, Parks AH, Mullaney TP, et al. Bilateral renal dysplasia and hypoplasia in a foal with an imperforate anus. *Vet Rec* 1988;122:91–2.
64. Medina-Torres CE, Hewson J, Stampfli S, et al. Bilateral diffuse cystic renal dysplasia in a 9-day-old Thoroughbred filly. *Can Vet J* 2014;55:141–6.