Gastrointestinal disease is one of the most common problems neonatal foals face during the first weeks of life. The most frequently encountered problem in this age group is contagious gastroenteritis which has been well described. Less well understood and less common is the gastrointestinal (GI) dysfunction which often accompanies neonatal encephalopathy (also referred to as hypoxic ischemic encephalopathy, asphyxia syndrome) and neonatal nephropathy during the first days of a foal’s life. This triad of neurologic, renal and GI syndromes often with signs of sepsis is the most common complaint of foals less than 48 hours old admitted to our Neonatal Intensive Care Unit. The GI component has a wide range of clinical signs and severity form very mild dysmotility to severe necrotizing disease. The necrotizing disease we see has some similarities (and some differences) to the disease well recognized in human infants called Necrotizing Enterocolitis (NEC). This talk will describe our experience with two recent studies which have brought attention to the occurrence of necrotizing gastrointestinal (GI) disease and asymptomatic intussusceptions in neonatal foals.

The relevance and association of clinical and sonographic findings in neonates with gastrointestinal disease will be discussed. During this session we will not attempt to review the technique, normal or abnormal findings in abdominal sonograms in foals. However, we want to point out 2 technical notes. The sonographic examination of the neonate with abdominal disease can be done with the foal standing, in lateral or sternal recumbency. It is important to realize that lesions tend to localize in the most dependent part of the abdomen and that these lesions can be mobile. Therefore it is important to scan carefully the gravity dependent area. This implies to place the probe under the foal to scan the ventral abdomen if the foal is sternal, to scan the left side if the foal is in left lateral recumbency and the right side if the foal is in right lateral recumbency. The author finds microconvex probes to be the most useful probes. These probes typically have a frequency of 5-11MHz. The frequency varies depending on the equipment used. To assess carefully the echogenicity of the wall, the wall layering or to be precise when measuring the thickness high frequency linear probes (usually 8-15 MHz) are required. Soaking the hair with alcohol is, in most foals, enough to obtain adequate images for ‘fluid checks’ or to detect severe abnormalities. To obtain detailed images clipping may be necessary. Clipping and using gel reduces the scanning time although it increases the preparation time.

Abdominal ultrasonography has been used for years as a valuable diagnostic aid and has become routine in the assessment of foals with critical gastrointestinal disease, however information about its usefulness, accuracy and limitations is not available. Abdominal ultrasound examinations are performed commonly by non-radiologists in human and veterinary intensive care units. In human critical care units, the time from presentation to operative care, length of hospitalization, complications and cost decrease when emergency abdominal sonograms are performed, potentially due to a more rapid diagnosis.

### Intestinal wall layering and color doppler

The characterization of the layering of the GI wall has received little attention in equine medicine. There are specific case reports that have shown that this has potential increase the precision of diagnosticians. Sonographic to pathologic correlation has been demonstrated for several disease processes including pediatric colitis and GI emergencies. In these cases the careful assessment of the echogenicity and layering patterns can give information about potential specific diagnoses often helping patient management. Of particular interest to the diagnosis of GI disease in human neonates was the study by Baud et al. In this study different patterns of abnormal intestinal echogenicity, based on the loss/maintenance of stratification and folding helped in obtaining a precise diagnosis. In our opinion the wide use of high quality portable or hospital based ultrasound machines makes possible the assessment of intestinal wall layering. We performed a study to define the normal layering pattern of the GI of normal neonates. Equine neonates showed distinct layering of the stomach and large intestine, and indistinct layering of the small intestine. The reason why the layering of the stomach and large intestine was distinct, but not in the small intestine, is uncertain. It is known that rapid changes in the intestinal wall occur in the first days of life, including marked changes in total and relative thickness of the different histologic layers with the muscularis layer changing quantitatively the most in this period. We believe that the evaluation of the changes in layering patterns could help clinicians be more precise about diagnosis in neonates with GI disease and hopefully help in the management of these cases. Multicenter studies are needed to prove this and to investigate the association of different sonographic patterns with specific diagnoses in foals.

Sonographic evaluation of GI blood supply using color flow Doppler is well correlated with histology and outcome in human neonates with GI disease and allows the differentiation of focal from diffuse necrosis in the setting of NEC. Increased flow is reportedly obvious to the untrained observer. Color Doppler sonography was found to be more accurate than clinical examination and plain abdominal radiography in the prediction of necrosis in human neonates with NEC. The usefulness of this technique in equine medicine had not been evaluated. In our prospective study of normal neonates diagnostic color Doppler signals of mural blood flow could not be obtained. It is possible that different ultrasound equipment, different settings, sedating foals to decrease motion artifacts,
or the use of this technique in foals with abnormal patterns might help in obtaining diagnostic color flow Doppler readings. The authors believe that this modality has potential in the evaluation of GI in horses but further work is needed before it this potential can be defined.

**Necrotizing enterocolitis (NEC) and pneumatosis intestinalis**

The recognition in our hospital population of hyperechoic echoes of gas within the gastrointestinal wall (pneumatosis intestinalis, PI) of foals hospitalized with GI or critical illness led us to retrospectively investigate this phenomenon within the context of all sonographic examinations performed in foals with GI disease. The presence of PI is considered virtually pathognomonic for NEC in human neonates with a compatible clinical picture. NEC is a disease process of the GI tract of human neonates that is marked by inflammation and bacterial invasion of the bowel wall. NEC occurs frequently in human newborn intensive care units, primarily in premature neonates and had been described in foals in isolated case reports. We have the clinical impression, and several texts have also suggested, that this may be an under recognized syndrome. Based on our retrospective study we concluded that: 1- PI was diagnosed frequently in severe cases. 2- Foals with PI have a worse prognosis than foals without PI. 3- Variables associated with the presence of necrotizing GI disease were: prematurity, blood in the feces, gastric reflux, abdominal distension, abnormal colon echogenicity and leukopenia.

Several similarities exist between human NEC and GI disease in equine neonates with a necrotizing component resulting in gas in the wall of the intestinal tract. We chose to describe this group of foals as having a necrotizing component of the GI and avoid call this process NEC as the pathogenesis may differ. This nomenclature has been used in these proceedings and referring to NEC we refer to the GI disease in human babies.

It is important to realize that PI is not a diagnosis or a clinical problem per se, but a pathological or imaging finding that is a consequence of the underlying disease. PI has been attributed to at least 58 causes in man but it is virtually pathognomonic of NEC in human neonates with compatible clinical presentation. Therefore, and as for all imaging findings, PI has to be interpreted in the context of the clinical picture. The process of gas production and accumulation within the intestinal wall is poorly understood. The composition of the gas in human neonates is variable but it is often 30% hydrogen. Three possibilities have been proposed as the source of the gas: (1) intraluminal GI gas that migrates due to injury, (2) bacterial production of gas due to bacterial invasion, and (3) pulmonary gas. The pattern or extent of PI does not correlate with the severity of the signs or the severity of the underlying diseases in human neonates and the presence of intramural gas can even precede the presence of any clinical signs.

**Epidemiology**

An understanding of the epidemiological aspects of NEC may help to place into context our clinical observations of necrotizing gastrointestinal disease in foals. The precise etiology of NEC, which is an ischemic and inflammatory necrosis of bowel, is unknown but three aspects are felt to be major risk factors for NEC to occur in human neonates: ischaemic/hypoxic mucosal injury, feeding and the presence of bacteria. Prematurity is the only independent determinant of human NEC. 90–95% of cases occur in infants born before 36 weeks of gestational age and the incidence varies inversely with birth weight and gestational age. Those most susceptible appear to be infants weighing less than 1000 g at birth and under 28 weeks’ gestation. Less than 10% of NEC cases occur in term infants. This close correlation with prematurity does not occur in foals in our experience as almost half the foals (46.2%) we identified were not premature. However, in our retrospective study, there was a difference (P = 0.03, OR 5.1) in the presence of necrotizing disease between premature foals with GI disease (53.8% of premature foals) and term (18.75%) foals with GI disease. In infants the clinical signs usually start between day 3-10 but NEC can be diagnosed up 90 days of age. In our clinical experience delayed onset beyond the first week does not occur in foals. Approximately 25–33% of all infants diagnoses with NEC die and 27–63% require surgical intervention. Only 33% of our cases with necrotizing disease survived but this figure includes fatalities because of economic considerations as well as humane considerations and so direct comparison might be misleading. 17/85 of foals that had abdominal sonograms due to signs of GI disease were diagnoses as having necrotizing GI disease. This frequency seems subjectively more than what is seen in other veterinary hospitals. This could be a reflection of regional characteristics, the type of cases that present to different hospitals or the specific concern of clinicians about these particular problems. Foals with PI have a worse prognosis than foals without PI. However, it is important to realize that many foals in which PI was diagnosed survived and foals with PI were likely to have more severe disease which introduces the confounders of financial limitations and humane perceptions. The survival rate of foals for which PI was observed sonographically was 37% vs. 72% in foals in which PI was not observed. When comparing the disease seen in foals to that seen in infants, the equine disease may be more similar to NEC seen in full term infants in which general has an earlier onset, shorter course and is less severe (less frequent perforations) and has been speculated to have a different pathogenesis.

**Clinical presentation**

Clinical manifestations of NEC in infants can be specific to the GI tract (such as feeding intolerance, vomit, blood in the feces, abdominal distention, painful or discolored abdominal wall) and others can be vague: apnea, hypoxemia, bradycardia, lethargy, temperature instability, blood pressure instability, erytema or edema. In many cases NEC may be indistinguishable from sepsis.
Laboratory values of babies with NEC are often the reflection of infection, activated coagulation and fluid retention. Leukopenia or leukocytosis, anemia, thrombocytopenia, hypo or hyperglycemia, electrolyte abnormalities and metabolic acidosis are common in this population. Bacteremia is reported in approximately 35% of cases of human NEC.\textsuperscript{16} Variables associated with the presence of necrotizing GI disease in foals are: prematurity, blood in the feces, gastric reflux, abdominal distension, abnormal colon echogenicity and leucopenia showing some parallelism between NEC and necrotizing GI disease in equine neonates. Bacteremia was observed in 55% of foals with necrotizing GI disease. This was not statistically different to the group of foals with GI disease without a necrotizing component in which 49% were bacteremic. In general the clinical picture seen in foals with necrotizing GI disease is consistent with sepsis perhaps reflecting that the final common pathway of intestinal injury is from the activation of the inflammatory cascade as has been speculated in infants.\textsuperscript{33-35}

**Diagnosis**

The diagnosis of NEC can be challenging and it seems that it is often made based on clinical and imaging findings. Classically radiographs showing PI, a thickened intestinal wall, free air in the abdomen (pneumoperitoneum), and portal venous gas were the basis of the diagnosis. The imaging findings (radiography, ultrasonography and computed tomography) have been described.\textsuperscript{28} Radiographic PI has been described in equine neonates.\textsuperscript{18} A recent review in infants described 11 sonographic features of NEC: 1. Increased intestinal echogenicity reflecting wall edema, inflammation or hemorrhage, 2. Intestinal thickening (2.7 mm or more), 3. Intestinal thinning (1 mm or less), 4. Increased intestinal perfusion, 5. Absent bowel perfusion, 6. PI, 7. Portal venous gas, 8. Free gas, 9. Anechoic free intraperitoneal fluid, 10. Free intraperitoneal fluid with echoes, and 11. Focal fluid collections. In our group of foals the presence of PI was part of the definition and apart from this finding only the presence of colonic thickening was different between the group with and the group without necrotizing disease. PI was most frequently imaged as scattered hyperechoic foci within the intestinal wall, although large clusters of hyperechoic foci within the bowel wall were also imaged in some foals. The intramural gas was imaged more frequently in the small intestine (58%) than in the large intestine (32%) and infrequently (11%) in both. The presence of increased peritoneal fluid was somewhat common (25%) but the presence of focal accumulations were rare (5%). The presence of hypo or dysmotility is also frequent in equine neonates with necrotizing GI disease. In our retrospective study only sonography was used. Comparisons with radiography would be interesting. Our clinical impression is that sonograms tend to be more informative when evaluating foals with GI disease. When compared to CT, sonograms have the advantage of being stall side and less expensive. We defined necrotizing disease as the presence of signs of GI disease (colic, diarrhea, gastric reflux or abdominal distension) and sonographic evidence of PI or pathologic presence of necrosis. In foals there are no reliable clinical signs that correlate with the presence of necrotizing GI lesions. Even dramatic signs such as hemorrhagic or coffee ground reflux or hemorrhagic or melanotic diarrhea may occur without any gross or histologic lesions. The major sequela in foals surviving with necrotizing lesions is segmental strictures which can be found in the small intestine, large colon or small colon. These strictures respond well to surgical resection.

**Treatment**

Medical treatment of NEC in infants typically consists of GI rest and decompression, broad spectrum antibiotic therapy, supportive care to manage electrolyte or acid base imbalances and parenteral nutrition.\textsuperscript{15} Although full thickness necrosis and secondary peritonitis may occur in affected foals, unlike infants, perforations are not seen in our experience. We have had anecdotal success treating foals at risk of or recovering from necrotizing disease with trophic feeding initially using fresh or frozen colostrum (even in foals up to 20 days old) or fresh mare’s milk and with judicious gradual volume increases dictated by feeding tolerance. The prevention of NEC is a complex matter. Avoiding preterm birth, judicious use of antenatal steroids in preterm deliveries, breast-milk feedings (vs. formula) and trophic feedings have been suggested as potential strategies to reduce the incidence of NEC in human neonates. The prophylactic use of oral antibiotics or the use of probiotics have received attention but its general use has not been adopted for reasons such as the emergence of antibacterial-resistant organisms or the questionable efficacy of probiotics.\textsuperscript{15} It’s been hypothesized that NEC can be a contagious disease and in some reports clusters of cases have occurred. *Klebsiella, E. coli, Enterobacter, Salmonella, Pseudomonas, Clostridia, Staphylococci*, coronavirus, rotavirus and enterovirus have been implicated as potential causative organisms.\textsuperscript{11,16} There are reports associating *C. difficile*,\textsuperscript{20} *C. perfringens*\textsuperscript{21} and *Rhodococcus equi*\textsuperscript{19} with necrotizing GI disease in foals. We feel that the necrotizing lesions in these reports are a direct result of bacterial pathogenic properties whereas the necrotizing disease we are reporting occurs as a primary problem which may then predispose the foal to secondary pathogenic colonization. In the group of foals that we reported blood cultures, PCRIs for detection of *Salmonella* in feces and fecal testing for the detection of clostridial toxins were performed frequently and a positive result was not associated with a necrotizing component to the gastrointestinal disease. In fact the percentage of foals positive to clostridial toxin detection analysis was lower (not statistically significantly higher) in foals with necrotizing GI disease.

**Asymptomatic intussusceptions**

As a part of our investigations in equine gastrointestinal disease we designed a prospective study in which normal neonatal standardbreds were scanned. Incidentally asymptomatic intussusceptions were found in 10/18 neonates. Finding jejuno-jejunal intussusceptions in these normal foals was unexpected, as asymptomatic intussusceptions had not been previously reported in horses.

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Jejuno-jejunal intussusceptions are the most common type of intussusception in foals with abdominal pain, but intussusceptions are reported to uniformly require surgical correction in horses. Dysrhythmic peristaltic activity has been proposed as the cause of intussusception in horses and people. Asymptomatic small bowel intussusceptions occur in humans and dogs and can resolve spontaneously (up to 20% in people and 8% in dogs) but as stated above this had not previously reported in horses. Sonographic signs that have been associated with transient, nonclinical small intestinal intussusceptions in small animals and infants are the absence of identifiable intestinal lesions, normal wall thickness, length of less than 3.5 cm, normal undilated proximal bowel, normal vascularity on color Doppler, respected layering, and the intussusception being compressible. We did not recognize pathologic lead points, increased wall thickness, or altered echogenicity in any of the asymptomatic intussusceptions we observed. Compressibility, length or mesenteric, and mural blood flow in the area of the intussusceptions were not evaluated. The wall thickness of the intussuscipiens was 0.1–0.2 mm thinner than the lower end of the reference range for normal jejunal wall thickness in 4 cases and the average wall thickness of the intussusciptum was less than the average jejunal wall thickness. Clinical intussusceptions in humans are characterized by an increased thickness of both intussusceptum and intussuscipiens, and the marginal thinning observed in foals with asymptomatic intussusceptions is of uncertain relevance. It is possible that asymptomatic intussusceptions are a normal occurrence in foals of this age range because of initial development of gastrointestinal function and motility. This unexpected finding helped us realize that the finding of intussusceptions in equine neonates should not be considered an absolute indication for surgery and that the clinical, clinicopathologic and rest of imaging findings should be used to decide the course of therapy. Recheck sonograms may be useful in this subset of foals.

References
ECG interpretation
The ‘seven questions of the equine ECG’ is an example of a clinical approach to evaluation of ECGs. The goal of this simplified approach is to get information that will allow you to make clinical decisions in horses. It is an oversimplification in some aspects but helps to diagnose common arrhythmias by understanding the mechanisms that cause the rhythm alteration. You will often be able to obtain a rhythm diagnosis by ‘eyeballing’ the ECG and without answering these questions. I would recommend that you use a protocol. This will build up your reading skills and will decrease the number of misdiagnoses. This is very similar to what you have learnt for ECG interpretation in dogs and cats. Mean electrical axis calculations have little to no utility in equine electrocardiography due to the explosive nature of the conduction system causing cancellation of electrical vectors.

What is the heart rate?
The normal heart rate of a horse is 28-42/min. This breaks down rhythms in the ones that are fast or tachycardic (common tachycardias are sinus tachycardia, ventricular tachycardia or supraventricular tachycardia), slow or bradycardic (the common ones are rhythms with frequent AV block, sinus arrests or pauses) and the ones that have a normal rate (for example normal sinus, sinus arrhythmia or atrial fibrillation). Note that this is the second big difference (after the lack of use of the mean electrical axis) that we have mentioned between ECGs in horses and most other species. Atrial fibrillation occurs, most of the times, at normal heart rates in horses and without the presence of underlying disease (lone atrial fibrillation). If the heart rate during atrial fibrillation is high you should suspect underlying systemic or cardiac disease predisposing to the arrhythmia and the tachycardia.

Sick sinus syndrome is rare in horses but the principles that you learnt for small animals apply to horses. Very long pauses are often followed by escape rhythms or tachycardias. Horses with sick sinus syndrome can collapse and the implications of collapse in horses are very different than in a small animals, particularly from the standpoint of human safety.

If you are using a printed ECG you can calculate the HR by extrapolation from the paper speed. 25mm/sec is the most common paper speed. 60 divided the RR interval (in seconds) will give you the heart rate (ventricular rate) for a particular beat (this is called the instantaneous heart rate). If you do the same using the interval between two consecutive P waves you can calculate the atrial rate. In an ECG trace each of the smallest squares is 1 mm. At 25mm/sec each of the little squares is therefore 0.04sec (1sec/25mm=0.04sec/mm). A large square (5 small squares) is 0.2 secs and 25 small squares (5 large squares) is 1 second. A standard old fashioned pen measures approximately 15 cm. This is 6 secs assuming 25 mm/sec paper speed (6x25mm=150mm=15cm). If you put a pen on a 25mm/sec ECG trace and count the RR intervals that fall under a pen and add a zero after that number this approximates the HR. Remember that if you have scanned copies of an ECG there may be magnified or reduced in size so the ‘pen rule’ may not work. When using digital systems sometimes the size of the squares (especially in old systems) will not be adjusted to the paper speed so you may need to use the electronic calipers in the computer. For most digital systems a screenshot at 25mm/sec is 15 or 16 secs so if count the RR intervals in a computer screen and multiply by 4 you get an approximate heart rate.

Is there a QRS for every P?
If the answer is NO, some or all the P waves are not conducted to the ventricles. Common causes of this in horses are 2nd and 3rd degree AV block, ventricular tachycardia or non-conducted APCs.

In the case of second degree AV block some P waves are conducted and others are not. They are blocked at the level of the AV node. Second degree AV block is very common and most of the times is physiologic in horses due to their intrinsically high parasympathetic tone. The 2nd degree AV block should disappear with exercise or excitement. If it does not, it is concerning. Often trotting the horse up and down or exposing it to some situation that would create excitement is enough. Note that the AV block can come back very quickly after the exercise so in questionable situations you may need to record the rhythm while the horse is exercising. Sinus arrest (or pause) is also caused by high vagal tone, also common, and also should go away with exercise or excitement. In this case it is the SA node activity stops transiently and therefore the next P wave is delayed. If the pause is very long a escape beat can occur but this is rare in horses. If the pause is a multiple of the RR interval it is called sinus block. If it is longer than two RR intervals is called sinus arrest.

Figure: Example of second degree AV block-
This is a base apex ECG and the paper speed is 25mm/s
Ventricular premature complexes (VPCs)

In the case of a ventricular premature complex the QRS will come earlier than expected (has a high instantaneous heart rate). Most people will consider a complex premature if the RR interval is ≥20% earlier than the previous one. The number is somewhat arbitrary but it means that if you calculate all instantaneous hear rates the one from the premature beats will be ≥20% faster than the sinus beats. There is no P for this QRS and often the following P will not have a QRS as the ventricles will 'be caught' in a refractory period. This will cause a prolongation of the R-R interval between the ectopic QRS and the following sinus complex called a ‘compensatory pause’. This is a somewhat confusing nomenclature and almost a misnomer as ‘no one is compensating’, the SA node its doing its job, the ventricles are ‘misfiring’ and they are dissociated. But think about it this way: the word compensatory refers to the fact that the prolonged RR interval (the pause) compensates for the shortened RR interval of the premature beat. In some occasions the P wave after a VPC is conducted and there is not compensatory pause. In this cases the VPC is called interpolated. See the example below.

The QRS is of different morphology than the sinus QRSs and the morphology will be often ‘wide and bizarre’. This is often the case but keep in mind that the closer the ectopic focus is to the normal conduction system the less ‘wide and bizarre’ the ventricular complex will be. In other words, in horses some ventricular complexes are not wide and bizarre and just different to the normal QRS complexes. Sometimes one lead will not clearly reflect the ectopic origin, this means that in a particular lead the ectopic QRS may be similar to the sinus QRS. This is one of the reasons why it useful to have more than one lead. If there is more than one abnormal QRS morphology the rhythm is called multifocal or polymorphic, i.e. there are more than one origins (foci) for the VPCs and therefore they have different shapes (morphologies).

Ventricular tachycardia (VT)

4 or more VPCs in a row is defined as ventricular tachycardia. It is important to remember that there are P waves during ventricular tachycardia. The key is that this P waves are not associated with the QRS complexes. Ventricular tachycardia is a dissociated rhythm and the SA node will keep working ‘at its own pace’ (and generate P waves) while the ventricles work at a different (ectopic) rhythm and generate QRS complexes. The P waves are not conducted to the ventricles because the ventricular rhythm is faster and the ventricles are refractory when the P wave goes through the AV node. Occasionally the P waves go through the AV node when the ventricles are not refractory and will be conducted. This is called a capture beat. Sometimes the capture beat and the ectopic beat happen at the same time and the QRS becomes a combination of the sinus and the ectopic QRS. This is called a fusion beat. It is a very common misunderstanding to think that during VT there are no P waves. It is important to try to identify the P waves. Often the P waves are buried in the QRS complexes or T waves and not visible. Increasing the paper speed and increasing the gain may help you identify P waves. As the sinus rhythm is most of the times regular using calipers can also help you find the hidden or buried P waves. Ventricular tachycardia is one of the most common rhythms requiring antiarrhythmic therapy in horses but not all horses with VT need antiarrhythmic. We will talk later about how to decide.

Two VPCs in in a row is a couplet and three in a row a triplet. Series in which there are one sinus beat followed by an ectopic beat, followed by a sinus beat, followed by an ectopic beat and so on are called bigeminy. If the series are of two sinus beats and one ectopic beat this is trigemini. Couplets, triplets, bigeminy and trigeminy are somewhat more concerning than single VPCs. Ventricular premature complexes/ventricular tachycardia and idioventricular beats/idioventricular rhythms are to names used to characterize for
ventricular ectopy according to their rate. A rhythm is called idioventricular if the rate is similar to the prevailing sinus rate. The definition is somewhat non-specific. Idioventricular rhythms are typically more benign than ventricular tachycardias.

**Torsades the pointes, torsades or torsades-like VT**

This is a particular type of polymorphic ventricular tachycardia that gives the illusion of a twisting (torsades) of the QRS complex around the isoelectric baseline. The QRS peaks first pointing up, then get smaller, then point down… This is a very unstable electrical system that can lead to sudden death. If you detect this rhythm go and get antiarrhythmics fast. Magnesium sulfate is the first choice antiarrhythmic for torsades in horses.

Are all P waves of normal and same morphology?

If the answer is NO there is a supraventricular premature complex (SVPCs), supraventricular tachycardia (SVT) or a wandering pacemaker.

**Supraventricular premature complexes (SVPC)**

In the case of a supraventricular premature complexes the P wave will come earlier than expected (has a high instantaneous heart rate and a short P-P interval). Most people will consider a complex premature if the P-P interval is ≥20% earlier than the previous one at rest (the number is somewhat arbitrary). There is a P for this QRS and often the P is of abnormal morphology although not always (see example above). There are three versions of a SVPCs depending on how premature the complex is but no real difference in their clinical implications.

- **Conducted** = ‘Not very premature’
  
  The P wave will be conducted normally and therefore the QRS will have normal morphology. There is no compensatory pause as the SA node will be reset by the ectopic P wave. In other words the following P-P and R-R interval will be normal. This is the common type.

- **Non-conducted** = ‘Very premature’
  
  The P wave is so early that the ventricles are still refractory. The premature P wave will not be conducted to the ventricles and therefore will not be followed by a QRS complex. This is called a ‘non-conducted SVPC. It is common for electrocardiography students to want to call non-conducted SVPCs 2nd degree AV block. It looks similar to 2nd degree AV block but the difference is that in this case the P wave is premature. They have different implications.

- **Aberrantly conducted** = ‘Somewhat in between’
  
  The ventricles are only partially repolarized and therefore the conduction through the ventricles is somewhat abnormal (aberrant). This causes the QRS complexes to have abnormal morphology. They are called SVPCs with aberrant conduction. They have different QRS morphologies when compared to sinus complexes similarly to VPCs. They are, however associated to P waves and they are not commonly as ‘wide and bizarre’.

  Four or more SVPCs in a row is called supraventricular tachycardia. Two in in a row is a couplet and three in a row a triplet. Series in which there are one sinus beat followed by an ectopic beat, followed by a sinus beat, followed by an ectopic beat and so on are called bigeminy. If the series are of two sinus beats and one ectopic beat this is trigemini. Couplets, triplets, bigeminy and trigemini are somewhat more concerning than single SVPCs.

Is the R-R interval regular?

This will help you break rhythms into regular rhythms, regularly irregular rhythms and irregularly irregular rhythms. The concept of regularly irregular or irregularly irregular if often misunderstood. Let’s break it down: the second word just refers to the presence or absence of an arrhythmia: regular rhythm means no arrhythmia vs. irregular rhythm (regularly irregular or irregularly irregular) means an arrhythmia is present. The first word qualifies the rhythm by describing the underlying rhythm: is there an underlying pattern to the rhythm? Common regular rhythms are normal sinus, sinus tachycardia…. Common regularly irregular rhythms (arrhythmias with an underlying regularity or pattern) are blocks, supraventricular or ventricular tachycardia and common irregularly irregular rhythms are such as atrial fibrillation or sinus arrhythmias. The most common misdiagnosis is equine electrocardiography is to diagnose atrial fibrillation at a fast rate (an irregularly irregular rhythm) as ventricular tachycardia (a regularly irregular rhythm). An irregularly irregular rhythm on a horse is atrial fibrillation until proven otherwise. This is the most common clinically relevant arrhythmia of horses. The other common irregularly irregular rhythm is sinus arrhythmia. Sinus arrhythmia often comes and goes and is vagally mediated. As the other vagally mediated rhythms we talked about before (2nd degree AV block, sinus block or arrest) it will disappear with exercise or excitement and has no clinical consequences. Sinus arrhythmia is not associated with respiration in horses. Atrial fibrillation will not disappear with exercise or excitement. Other characteristics of atrial fibrillation are the lack of an isoelectric line and the presence of ‘f’ waves. Be careful when the rate is fast as the ‘f’ waves may be difficult to see.

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Figure. Example of a base apex ECG showing atrial fibrillation. Note the RR interval changes with each beat. The paper speed is 22mm/sec.
Figure: Base apex ECG showed atrial fibrillation at a higher rate. Note that the RR interval changes with each beat but this is harder to ‘eye ball’. It is easy to notice if you use callipers like the ones in the picture. Note that due to the higher rate the ‘f’ waves are more difficult to see.

Are the PR interval, QRS duration and QT interval of normal duration?
The normal intervals in horses are: P-R= 0.2-0.5s, QRS <0.14s and Q-T ≤0.6s. PR defines the AV conduction. QRS defines ventricular depolarization and QT ventricular repolarization. If abnormal they can help you make sense of the rhythm disturbance. For example, a very short PR interval may be caused by accelerated conduction through an accessory atrioventricular pathway. A very long PR interval may be caused by prolonged conduction associated with AV node/conduction system disease. A prolonged QRS duration can be seen in VPCs or be drug induced (quinidine) etc. Electrolyte abnormalities (hypo or hyperkemia being the most common, but not only) will also change conduction rate and excitability.

It is likely that answering a single question will not give you a rhythm diagnosis but it is unlikely that after answering all 7 you will have one or a very short list of possibilities. Remember this is a set or simplified rules and there are exceptions to all rules.
Interpretation of Equine Murmurs: When Should I Worry?
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This document is written to support the presentation about how to interpret heart murmurs. The objective is to give an easy to follow mental chart useful to field practitioners. To a certain degree the chart is an oversimplification but provides rules that hold true in the majority of cases.

A murmur is a cardiac sound generated by turbulent blood flow. It is useful to think about murmurs from the perspective of the reason for the turbulent flow. There are three main broad categories, these are: regurgitations, stenosis and shunts. Another relevant group of murmurs in horses are the ‘physiologic = ejection = flow murmurs’. Physiopathologically it is useful to place these ones under the category of stenosis. These are not generated by stenosis but mainly caused by ejection of blood through a major vessel and therefore sound similar to pulmonic or aortic stenosis. These are very common murmurs in horses due to the large size of the heart and stroke volume. Physiologic murmurs are particularly common in athletic horses with a large heart and a thin thoracic wall. Physiologic murmurs tend to be more frequent in circumstances that predispose to a turbulent flow during ejection such as anemia, fever, excitement or the immediate postexercise period. Pathologic murmurs vary from asymptomatic and not affecting performance to causing exercise intolerance, heart failure or affecting safety of riders.

The timing of the murmur is the first key. We will pause for a second to make sure the timing of the cardiac cycle in relation to auscultation is clear. There are two loud sounds that are S1-S2. These are traditionally described as ‘lub-dup’. The pause or silence between lub (S1) and dup (S2) is systole. This is the short pause. The pause or silence between dup and the following dub is diastole and is the long pause. In a horse with a high heart rate, or a foal, in can be difficult to differentiate systole vs. diastole as diastole shortens with the increase in rate. In adults the easiest artery to reach is the facial at the level of the medial aspect of the mandible and in recumbent foals the median in the medial aspect of the elbow region. Being able to differentiate systole from diastole is critical to the rest of the explanation. For most experienced clinicians the difference becomes intuitive. If you have questions about systole vs diastole ‘exercising’ your ears at the same time that you think in your head ‘lub-dup’ and tap your foot following the heart rhythm is a good practice.

Murmurs are described based on 7 characteristics. This may sounds superfluous but gives relevant information to diagnosis, prognosis and follow up of these cases. Small animal cardiologists do not usually follow this classification. I feel that the reason is that it is often impossible in a small animal with a high heart rate. Fortunately for us it is much easier in a horse with a normal resting heart rate. Giving 7 adjectives to a heart murmur requires practice and purposeful auscultation but it is very useful.

1. **Grade** - Tells you how loud the murmur is: There are different scales. I am familiar with the following.
   - Grade 1/6 – soft, only audible in a quiet environment
   - Grade 2/6 – heard easily when placing stethoscope over point of maximal intensity.
   - Grade 3/6 – audible immediately when placing stethoscope over chest wall.
   - Grade 4/6 – loud murmur with a faint thrill.
   - Grade 5/6– loud with a strong thrill
   - Grade 6/6 – loud with a strong thrill and heart with the stethoscope away from the chest wall.

2. **Timing**
The most important characteristic. Divides murmurs in systolic, diastolic or continuous. See the description above.

3. **Duration**
   ‘Holo’- between S1-S2 or viceversa (S1-S2 are audible). Pan- extends over S1 and S2 and these are not audible. If shorter than holo can be classified as early, mid, mid to late etc.

4. **Character**
Blowing, coarse or harsh, musical, honking.

5. **Shape**
Crescendo (louder at the end), decrescendo (less loud at the end), band shaped (as loud beginning, middle and end) o crescendo-decrescendo- gets louder and then less loud. The shape is characteristic more difficult to precise.

6. **Location or PMI (point of maximal intensity)**
It refers to the area where the murmur is loudest but not necessarily to the structure that is affected.
   - Mitral valve area: Left fifth intercostal space approximately half way between the shoulder and the sternum. The most common mistake of is to auscultate to low. It is the only PMI in which the head of the stethoscope is visible. For all others the head of the stethoscope is buried under the triceps muscle.
   - Aortic valve area- Left 4th ICS under lower than the point of the shoulder.
- Pulmonic valve area- Left 3rd ICS. This is very cranial. You need to have the horse point the leg forward or push very hard under the triceps. Hard enough that the horse will be bothered by you pushing. Once the horse becomes bothered you need to push harder. I believe many veterinarians have never placed the stethoscope over the pulmonic valve area.
- Tricuspid valve area- Right 3rd and fourth intercostal space midway between the shoulder and the sternum. Also the head of the stethoscope is buried under the triceps.

7. Radiation
Murmurs can radiate cranially caudally or across the chest. It is common that loud, musical or honking murmurs radiate left to right. In the case of murmurs that are less than a grade 3 and not musical if a murmur is heard in both sides of the chest it is more likely that there are two murmurs. A murmur radiates with the same shape and character.

An example of a description of a typical murmur of aortic regurgitation would be:
‘Grade 3/6 holodiastolic decrescendo musical murmur with the point of maximal intensity over the aortic valve and radiating cranially caudally and to the right side of the chest’.

Another important key for murmur interpretation is variation in intensity. There are two types of murmurs that classically vary in intensity: ejection murmurs and mitral valve prolapse. There are easily differentiated as prolapses are mid to late crescendo systolic and ejection murmurs are early to mid crescendo decrescendo systolic. Exercise, excitement, pain or sedation are common circumstances that would make this murmurs louder. Murmurs that vary a lot in intensity are usually benign.

Below are example of a diagnostic algorithms for diagnosis of murmurs in horses.

*CHD= congenital heart disease, AC= aortocardiac, PDA= patent ductus arteriosus.

Diastolic murmur
A diastolic murmur in a horse is aortic regurgitation until proven otherwise. Pulmonic regurgitation is commonly seen echocardiographically but it is rarely audible due to the low pressure in the right side of the heart. Pulmonic regurgitation is rarely clinically relevant. If you hear a diastolic murmur you have to assume aortic regurgitation is the reason and the next immediate step is to feel the peripheral pulses. If peripheral pulses are bounding the aortic regurgitation is likely moderate or severe. The grade of the murmur does not correlate with the severity of the disease but the character of the pulse does. Aortic regurgitation murmurs are almost always decrescendo and often musical or blowing and if musical tend to radiate widely.

A particular type of murmur/physiologic sound is the 2 year old squeak. This is a 1-3/6 early diastolic squeak heard in the left or right side of the chest. It is more commonly heard in young horses with big hearts and more commonly in young TH horses; hence the name ‘2 year old squeak’. It can be heard, however, in horses of any age and breed. It is likely caused by ventricular stretching during rapid ventricular filling and does not have clinical relevance.

Many horses with aortic regurgitation will have successful athletic careers for years and normal life expectancy. Aortic valve degeneration is very common in teenage horses. It is recommended to do an echocardiogram horses with bounding pulses, young horses or horses with other signs of heart disease apart from the murmur. In other horses the decision to make an echocardiogram will vary depending on the circumstances. Horses with moderate or severe aortic regurgitation are predisposed to ventricular arrhythmias and an exercising ECG is indicated in these horses.

Right sided systolic murmur
A right sided systolic murmur (only right sided) is tricuspid regurgitation until proven otherwise. Careful auscultation over the pulmonic valve area needs to be performed to rule out the presence of a ventricular septal defect. The severity of tricuspid regurgitation is correlated with the grade of the murmur. It is very common in Standardbred racehorses. It rarely affects performance or life expectancy. If the right atrium becomes enlarged in can predispose to atrial fibrillation. In horses with a murmur louder than 3/6, pansystolic or musical or in horses with other signs of cardiac disease (poor performance, jugular pulses, edema, exercise intolerance, arrhythmias…) an echocardiogram is recommended. In horses with murmurs 3/6 the recommendation will vary depending on the occupation of the horses are circumstance but significant disease is unlikely.
Bilateral murmurs/Congenital heart disease
An echocardiogram is indicated in all horses with suspected congenital heart disease. Ventricular septal defects (VSD) are, by far, the most common congenital heart defects of horses. The most common combination of murmurs in congenital heart disease is a 4-6/6 pansystolic band shaped and coarse murmur with the point of maximal intensity over the tricuspid valve area and a murmur less loud than the one on the right that is holosystolic or pansystolic crescendo decrescendo and blowing or coarse with point of maximal intensity over the pulmonic valve area. This combination reflect the left to right shunt (right sided murmur) and the left sided murmur of ‘relative pulmonic stenosis’. The second murmur is caused by ejection of an increased amount of blood in the right ventricle due to the shunt. If the left sided murmur is louder than the right sided murmur complex congenital heart disease is suspected. Tetralogy of Fallot is the most common complex congenital heart disease of horses. It is a common misunderstanding to say that in horses with VSD ‘the murmur can be heard bilaterally. These are two murmurs caused by two different processes: shunt + relative pulmonic stenosis.

Horses with small VSDs can have normal life expectancy and sometimes normal athletic careers. The echocardiogram is critical in these horses. In horses with large VSD or complex congenital heart disease the life expectancy may be shortened. They can be exercise intolerant and they may not be safe to exercise.

Left sided systolic murmur
A loud left sided systolic murmur should be considered mitral regurgitation until proven otherwise.

The grade is not correlated with the severity of the disease. The PMI can be the mitral or aortic valve areas.

There are 3 main variations:
- Holo or pansystolic band shaped and coarse or blowing- it is the most common one.
- Musical or honking- raises concerns about a ruptured chorda tendineae often associated with severe disease.
- Mid to late crescendo systolic murmur- suggests a mitral valve prolapse. Mitral valve prolapse in horses is usually associated with mitral valve disease that progresses very slowly, if at all, over the years and good prognosis.

There is one exception to the rule above. If the murmur is \( \leq 3/6 \) early to mid-systolic crescendo-decescendo blowing or coarse over the pulmonic or aortic valve area and variable with hemodynamic changes it is likely a physiologic ejection murmur. Particularly with exercise or excitement an ejection=flow=physiologic murmur is suspected.

If the murmur is \( \geq 3/6 \) holosystolic crescendo-decrescendo blowing or coarse and over the pulmonic valve area pulmonic stenosis or more commonly relative pulmonic stenosis (PS) associated with a VSD is suspected. Isolated PS is rare in horses. Aortic stenosis is almost unheard of in horses.

Mitral regurgitation is both commonly asymptomatic and the most common valvular disease associated with poor performance and shortened life expectancy in horses. In the scenario of a prepurchase examination further diagnostics (echocardiogram) is recommended in horses with left sided systolic murmurs that do not fit the description of an ejection murmur. Particularly if the murmur is 3-6, pansystolic or there are other signs of heart disease. Clinical signs that suggest the MR may have clinical relevance are exercise intolerance, increased resting heart rate, respiratory rate or effort or an irregularly irregular rhythm consistent with atrial fibrillation.

Continuous murmurs
Continuous murmurs are rare in horses. Patent ductus arteriosus are common in new born foals and should close during the first days of life. A continuous murmur in a foal older than 5 days old warrants investigation. Aortocardiac fistula is the most common, and almost only, differential diagnosis in and adult horse with continuous murmurs. Horses with aortocardiac fistulas are not safe to ride as they a predisposed to life threatening arrhythmias and collapse. All adult horses with continuous murmurs should have an echocardiogram.
Prepurchase Examinations and Interpreting Arrhythmias: When Should I Worry?

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Clinical decisions after rhythm diagnosis: black white and grey

There will be times when the decision of how to manage an arrhythmia will be black or white. Other times it will not be that easy are many factors apart from the rhythm will play a role.

- **White**
  
  Vagally mediated arrhythmias: 2nd degree AV block and sinus blocks, pauses or arrests that go away with exercise or excitement and sinus arrhythmias can be considered benign. Sick sinus syndrome or 3rd degree AV block are the pathologic and black version of these physiologic arrhythmias and are very rare but very concerning as can make the horse collapse. They will need therapies similar to the used in small animals and a consultation with a specialist.

- **Light grey**
  
  Rare or occasional SVPCs or unifocal VPCs do not have, in and of themselves, clinical consequences in the resting horse. They would rarely need antiarrhythmic therapy. Electrolyte abnormalities, systemic inflammation, systemic disease or underlying heart disease can predispose to these arrhythmias. So the arrhythmia may be a sign that something else is wrong with the horse that you may need to address. SVPCs are one of the triggers for AF but the risk is difficult to quantify. VPCs are somewhat more concerning than SVPCs but do not panic when you identify VPCs in an ECG strip. It is often said that VPCs should be interpreted considering the ‘company that they keep’. Meaning the rest of the evaluation of the horse, the heart and other ECG abnormalities are critical to make decision about how to manage horses with VPCs. Another layman’s way of explaining the significance of ventricular ectopic beats to an owner is that a few single VCPs are not dangerous but the concern is that they can ‘gang up and try to kill you’.

  Depending on the clinical scenario you will need to decide if you can ignore this arrhythmias or you should investigate them further. Take home message: worry about why the arrhythmia is present and worry about the potential for worsening.

  For example:

  1. **You are treating a horse for acute colitis (or post-operative colic, pneumonia…) and you detect SVPCs. It would be advisable to make sure that electrolytes and acid base status are normal and evaluate the severity of the inflammatory response (do a CBC for example).**
  2. **You are auscultating a high level three day eventer prior to sedating it to float his teeth. You hear a loud left sided systolic murmur and some premature beats. It would be advisable to explain the owner that the loud murmur is likely the cause of mitral regurgitation and that the premature beats could be a sign that there is cardiac remodeling or enlargement due to the mitral valve disease. You could recommend doing an echocardiogram and a holter and exercising ECG to evaluate the underlying heart disease, prognosis and risk for progression.**

- **Grey: Atrial fibrillation (AF)**
  
  The most common clinically relevant arrhythmia in the horse. You definitely need to take this arrhythmia into account for the management of the horse but atrial fibrillation is seldom an emergency. The first step is to assess electrolytes, acid base status and a potential of an inflammatory response similar to the explained above for premature complexes. The next question to decide if the horse should be converted to normal sinus rhythm (cardioverted). There are many considerations that play a role in this decision like: the duration of the arrhythmia, the presence of underlying cardiac disease, the type of exercise the horse does, the potential complications of the cardioversion, the cost of the evaluation and treatment etc. The decision is multifactorial and would be topic for a full instructional video. These are some facts about AF to keep in mind:
  - Not all horses need to be converted to normal sinus rhythm.
  - The success rate for treatment is in general terms high, approximately 85-95%.
  - If you decide to treat the horse you have to main choices: antiarrhythmic drugs (most common choice is Quinidine) or transvenous electrical cardioversion. Both have pros and cons.
  - For any cardioversion attempt the main complication is sudden death. This is very rare but should be mentioned.
  - Paroxysmal atrial fibrillation may occur in young racehorses. It will often spontaneously cardiovert within 48 hours of fibrillation onset.
  - AF causes decreased performance in high intensity athletes (racehorses, eventer, field hunters, elite jumpers) and is often unnoticed in pasture pets or low intensity athletes (dressage, show hunters, conformation…)
  - Treatment may not be necessary in older horses performing up to expectations if exercising ECG is acceptable (rate and rhythm) or in ‘pasture pets’. Horses that exercise at high intensities will not be able to do successfully while on AF.
  - Echocardiography is needed to assess cardiac function and the presence of underlying cardiac disease.
  - Echocardiogram and exercising ECG are mandatory if a horse is going to be exercised while on AF.
Cardioversion is contraindicated if there is heart failure or severe underlying disease.
The recurrence rate varies between 25% and 100% depending on the situation.
The cost of evaluation and therapy is variable but can range from 1000-5000$. 

**Dark grey: Ventricular tachycardia (VT)**

Ventricular tachycardia is a serious arrhythmia. Horses with ventricular tachycardia always need close monitoring, ideally with telemetry and often need antiarrhythmic therapy. However, not all horses with ventricular tachycardia need antiarrhythmic therapy. It is mandatory to assess electrolytes, acid base status and a potential of an inflammatory response similar to the explained above for premature complexes and atrial fibrillation.

There are 5 classic scenarios in which antiarrhythmic treatment is indicated for horses with VT:

1. Clinical signs of cardiovascular collapse (e.g. weakness, syncope…). This applies to all arrhythmias and in general terms to all medical problems. Look at the patient first.
2. Heart rate >100 beats/min. The number is somewhat arbitrary but good place to start.
3. Multifocal rhythms - Implies there is more widespread cardiac involvement. Also has prognostic implications.
4. R on T phenomena- This happens when the premature QRS is so early that ‘comes out’ of the previous T. Creates a very unstable electrical system that predisposes to more malignant arrhythmias (ventricular fibrillation) and sudden death.
   
   Figure: Base apex ECG of a horse with VT. Note that the 6th QRS complex ‘comes out’ of the 5th T. This is R on T phenomenon.
5. **Torsades de Pointes** - This is an emergency. Run and get the emergency drug box (or tell someone to do so) and put an IV catheter ASAP. Start pulling magnesium sulfate (25 g from a 500 kg horse given over 25 minutes) and lidocaine (0.5 mg/kg slow and repeat up to 3 times q 5 minutes). If the horse is standing be aware that it may collapse at any time so have unnecessary or untrained people out of the stall and everybody else ready to get out of the way.

The most common drugs to treat ventricular arrhythmias in horses are lidocaine and magnesium sulfate. Magnesium sulfate is the drug of choice for Torsades. Magnesium sulfate should only be used in horses with low or normal magnesium. Hypermagnesemia is very rare unless it is iatrogenic in origin.
Cardiac auscultation is often the first clue for the detection of valvular, arrhythmic or congenital disease that can affect performance, safety or life expectancy. The most common physical examination findings that alert clinicians about cardiovascular disease are murmurs and irregular rhythms. The importance of careful auscultation and description of the heart rhythm and murmurs cannot be overemphasized. In some occasions diagnostic aids such as electrocardiograms, echocardiograms or measurements or cardiac biomarkers will be useful to the equine clinician.

The electrocardiogram (ECG or EKG) is the recording of the electrical activity of the heart. This can be printed in a paper, displayed on a screen or stored digitally. Units that display the ECG in real time are called telemetry units and the 24-hour continuous ECG recording is called Holter. The base-apex lead is, however, the most commonly used and often gives you the information that you need. To obtain a base-apex ECG place the left arm electrode (LA=Left Arm, positive, usually yellow) behind the left elbow- this is the apex. The right arm electrode (RA=Right Arm, negative, usually red) in front of scapula and the ground electrode (RF=Right Foot, usually black) attached at a site remote from the heart. For example over the right jugular groove. If you have position the leads correctly (and the horse has a normal rhythm) leads one and two will show a positive P wave followed by a QRS complex with formed by a small positive deflection and a large negative deflection (rS morphology) and followed by a T wave that is the typical base apex ECG of horse. If you decide that you want to see additional leads you can easily apply more self-adhesive electrodes and clip the leads to them or use alligator clips.

Currently is common to record ECGs digitally. A system called Televet is the most popular system worldwide for horses. It records ECGs without the need of clips and the rhythm is transmitted to a computer via Bluetooth. It can be used for continuous or exercising recordings.

Another popular option to obtain an ECG is to use a case that can be adapted to a smart phone (Alive core™). This will quickly give you a non-standard lead that you can interpret on the screen, print or email to a colleague. It is a good option for field veterinarians, for short recordings and for owners of animals with recurrent arrhythmias. It is less useful for the hospital setting in which continuous recording are more commonly needed.

When premature beats are auscultated during a prepurchase examination an electrocardiogram is almost always indicated. A continuous electrocardiogram (Holter) may be important as occasional premature contractions (average of less than 1/hour) is consider ‘normal’. Arrhythmias cab be intermittent and often short recordings do not demonstrate all arrhythmias present in one individual. In other species the prognosis and relevance of some arrhythmias is correlated with their frequency over a 24-hour period.

In horses that are to be ridden performing an exercising electrocardiogram is often indicated. Auscultation of an arrhythmia or a diastolic murmur are the most common indications for an exercising electrocardiograms. In general SVPCs are less concerning than VPCS although the increased risk for atrial fibrillation needs to be considered. Premature beats that are overdriven during exercise are considered more benign. Aortic regurgitation and exercise make the perfect arrhythmogenic cocktail; the ventricular enlargement and remodeling, the decreased coronary perfusion caused by the aortic regurgitation, the shortened diastole caused by the increased heart rate, the increased oxygen demand and the increased sympathetic tone caused by the exercise combine to predispose to arrhythmias.

Horses with moderate or severe aortic regurgitation that continue to exercise should have an exercising electrocardiogram to investigate if exercising arrhythmias are present and an echocardiogram to assess the cardiac structure and function. Mitral or tricusdic regurgitation are less commonly associated with exercising arrhythmias.

The interpretation of continuous or exercising electrocardiograms is usually simple. However it requires practice, is often time consuming and in some cases the interpretation is complex. For practitioners that see cardiac cases very frequently or that integrate exercising electrocardiograms in a busy sports medicine practice it may be useful to learn how to interpret exercising rhythms. For many others learning to acquire recordings and seeking advice for interpretation is the highest yield approach. The equipment is currently affordable (2.5-3000USD) and after an initial investment the electrodes and other supplies needed to acquire a continuous or exercising ECG may cost 2-3USD/recording.

Echocardiograms are indicated when significant valvular disease is suspected, to determine if arrhythmias are associated with structural heart disease and in all cases in which congenital heart disease is suspected or continuous murmurs are heard in adult horses. Some general rules follow.

A horse with aortic regurgitation needs an echocardiogram when:
- When the arterial pulses are bounding
- When it is young
- When the horse has ventricular premature beats
When the horse has other signs of heart disease:
  o mitral regurgitation murmur
  o VSD murmur is present
  o atrial fibrillation
  o exercise intolerance

A horse with a right sided murmur needs an echocardiogram when:
When the murmur is grade 4-6/6 and holosystolic or pansystolic
When other signs of heart disease:
  o Poor performance
  o A fib
  o MR or VSD (relative PS) murmurs

A horse with a left sided systolic murmur needs an echocardiogram if:
If the murmur is 3-6/6 and holo or pansystolic
If the murmur is band shaped and holo or pansystolic
If the horse has other signs of heart disease:
  o Increased respiratory rate or effort
  o Atrial fibrillation
  o Exercise intolerance/poor performance

A horse with an irregularly irregular rhythm should be considered to have atrial fibrillation until proven otherwise. **Atrial fibrillation** is the most common clinically relevant arrhythmia in humans and horses. Atrial fibrillation affects performance in horses that practice high intensity exercise but many horses used for pleasure riding or low intensity equestrian sports can do so while in atrial fibrillation. The decision to convert a horse to normal sinus rhythm is multifactorial. Duration of the arrhythmia, presence of previous episodes, presence of underlying heart disease, economic factors, risk aversion of the owners and the use of the horse are some of these factors. There is ongoing debate about the safety/risk for collapse in horses with AF. If a horse is not going to be converted an echocardiogram and an exercising electrocardiogram are needed to determine if it is safe for the horse to exercise.

Current recommendations in horses with sustained atrial fibrillation are that these horses should only be used by informed adult riders and exercise should be limited to a level considered relatively safe based on the exercising ECG.

The evaluation of horses with murmurs or arrhythmias is centered on echocardiograms and electrocardiograms. In the work up a horse with poor performance evaluation of the cardiovascular system is often better done in conjunction with the evaluation of the musculoskeletal and respiratory (upper and lower) systems and the assessment of the fitness status and progression of training. The protocol often includes evaluation of fitness, musculoskeletal system, upper respiratory tract, lower respiratory tract and cardiovascular system by means of: historical questionnaire, general physical examination, lameness examination and gait analysis using gyroscopes, resting and dynamic upper airway endoscopy, bronchoalveolar lavage, echocardiograms, exercising electrocardiograms and measurements of lactate, PCV, heart rate, CK and sweat response before, during and after exercise. I believe that a team approach for the simultaneous evaluation of different body systems and fitness is useful for the evaluation of poor performance and in some cases as a preventative medicine approach for the management of high level athletes.
Cardiac disease is a cause of poor performance in equine athletes. The musculoskeletal and respiratory systems, are the body systems more commonly involved in poor performance in horses participating in any equestrian disciplines, followed by the cardiovascular system [1]. Sudden death (SD) during sports is a rare event but has catastrophic consequences for the horse, the safety of the human partner and the public perception of welfare during equestrian sports.

Cardiac auscultation is often the first clue for the detection of valvular, arrhythmic or congenital disease that can affect performance, safety or life expectancy. The most common physical examination findings that alert clinicians about cardiovascular disease are murmurs and irregular rhythms. Careful description of the cardiac auscultation may seem superfluous but is key to the diagnosis of heart disease in horses.

**Mitral valve disease**
The most common equine valvular disease causing poor performance or shortened life expectancy. A left sided systolic murmur should be considered mitral regurgitation until proven otherwise. The exception to this rule is the physiologic ejection (or flow) murmur. Auscultation is often sufficient for the differentiation. A physiologic ejection murmur is usually 1-2/6 (sometimes 3/6) early to mid-systolic blowing or coarse crescendo-decrescendo murmur with the point of maximal intensity over the pulmonic or aortic valve areas. Ejection murmurs do not radiate and often vary in intensity with exercise or excitement. Physiologic murmurs are common in horses and this leads to many horse owners to the false conclusion that a cardiac murmur in a horse is never a problem. Mitral regurgitation murmurs are 1-6/6 holo- or mid- to late systolic blowing, coarse, musical or honking band shaped or crescendo murmurs with the point of maximal intensity over the mitral or aortic valve areas. Murmurs of mitral regurgitation may or may not radiate and usually do not vary in intensity unless they are caused by a prolapsed valve. Systolic murmurs that are variable in intensity (with excitement, exercise sedation or pain) are frequent. Physiologic ejection murmurs and murmurs of mitral valve prolapse are the common variable murmurs and the clue to their differentiation is their timing and shape: physiologic ejection murmurs are early to mid-systolic crescendo-decrescendo and mitral valve prolapse are mid to late crescendo. When the auscultation is not clear and to determine the severity and prognosis of mitral valve disease an echocardiogram is needed.

**Aortic regurgitation**
A common disease of teenage horses. Aortic valve disease is frequently an incidental finding but some horses with aortic regurgitation can develop exercising arrhythmias, exercise intolerance or heart failure. A diastolic murmur in a horse should be considered to be aortic regurgitation until proven otherwise. The presence of a diastolic murmur, and the consequent suspicion of aortic regurgitation, should prompt the clinician to feel the peripheral pulses. A horse with a diastolic murmur and strong or bounding peripheral pulses likely has moderate or severe aortic regurgitation. Moderate to severe aortic regurgitation predisposes horses to ventricular arrhythmias particularly during exercise. Aortic regurgitation and exercise make the perfect arrhythmogenic cocktail; the ventricular enlargement and remodeling, the decreased coronary perfusion caused by the aortic regurgitation, the shortened diastole caused by the increase in heart rate, the increased oxygen demand and the increased sympathetic tone caused by the exercise create ideal conditions for arrhythmias. Horses with moderate or severe aortic regurgitation that continue to exercise should have an exercising electrocardiogram to investigate if exercising arrhythmias are present and an echocardiogram to assess the cardiac structure and function. **Pulmonic and tricuspid regurgitation** rarely cause performance problems or affect safety unless tricuspid regurgitation causes atrial enlargement that predisposes to atrial fibrillation.

**Ventricular septal defect (VSD)**
The most common congenital heart disease of horses. A VSD can be identified on cardiac auscultation by the presence of a characteristic combination of murmurs. The left to right shunt causes a loud right sided systolic murmur. The second murmur is a systolic crescendo-decrescendo murmur with the point of maximal intensity over the pulmonic valve area that is less loud than the one on the right. This second murmur is caused by the ejection of blood (increased due to the shunt) out of the right ventricle and is called the murmur of ‘relative pulmonic stenosis’. The pulmonary artery is relatively small to the amount of blood ejected by the right ventricle. It is key in horses with right sided systolic murmurs to carefully auscultate the pulmonic valve area to differentiate tricuspid regurgitation (only right sided murmur) from VSDs (right and left sided murmurs). It takes conscious effort to place/push the stethoscope on the third intercostal space well under the left triceps to auscultate the pulmonic valve area. If this is not done the murmur of relative pulmonic stenosis is easily missed. Echocardiograms are needed to determine the prognosis and level of exercise that will be tolerated by a horse with a VSD.
Atrial fibrillation

The most common clinically relevant arrhythmia in humans and horses [2,3]. Atrial fibrillation affects performance in horses that practice high intensity exercise but many horses used for pleasure riding or low intensity equestrian sports can do so while in atrial fibrillation. The decision to convert a horse to normal sinus rhythm is multifactorial. Duration of the arrhythmia, presence of previous episodes, presence of underlying heart disease, economic factors, risk aversion of the owners and the use of the horse are some of these factors. There is ongoing debate about the safety/risk for collapse in horses with AF [4-7]. If a horse is not going to be converted an echocardiogram and an exercising electrocardiogram are needed to determine if it is safe for the horse to exercise. Current recommendations in horses with sustained atrial fibrillation are that these horses should only be used by informed adult riders and exercise should be limited to a level considered relatively safe based on the exercising ECG. Other cardiac diseases that can cause decreased performance are myocarditis, poor myocardial function, aortocardiac fistulas, complex congenital heart disease, 3rd degree AV block, sick sinus syndrome, severe pericarditis etc.

The field of **exercising arrhythmias** deserves special mention. The presence of arrhythmias is common in normally and poorly performing horses [8-13]. The influence of exercising arrhythmias on performance or tolerance to exercise is intuitive and extrapolated from basic physiologic studies documenting the associated cardiovascular changes. However this influence is not proven and in many cases it is not possible to ascertain if arrhythmias are incidental or the cause of poor performance. The fact that a malignant arrhythmia has the potential of causing collapse or sudden cardiac death (SCD) complicates the decision making processes [14]. Collapse and SCD can affect the health of horses, the public perception of welfare during equestrian sports and most importantly the safety of riders. The incidence of malignant arrhythmias causing collapse or SCD is fortunately low [15,16] but likely 50-100 fold higher in horses than in analogous human athletes [17]. Conditions to which equine sudden cardiac death has been attributed include: cardiac failure, arrhythmias, pulmonary hemorrhage or idopathic blood vessel rupture. Idiopathic blood vessel rupture (particularly aortic rupture) is a classically described cause of sudden death in horses, reported in 9–24% of sudden death cases. The presence of abnormalities in the aortic root or signs of pulmonary hypertension should alert clinicians about an increased risk for sudden death. Many of the leading causes of SCD in humans, such as hypertrophic cardiomyopathy, coronary anomalies, arrhythmogenic right ventricular cardiomyopathy (ARVC), channelopathies, Marfan Syndrome, *commotio cordis*. [16-20] or coronary artery [18,21] are not reported or frequent in horses.

The evaluation of horses with murmurs or arrhythmias is centered on echocardiograms and electrocardiograms. In the work-up of a horse with poor performance, evaluation of the cardiovascular system is often better done in conjunction with the evaluation of the musculoskeletal and respiratory (upper and lower) systems and the assessment of the fitness status and progression of training. The protocol often includes evaluation of fitness, musculoskeletal system, upper respiratory tract, lower respiratory tract and cardiovascular system by means of: historical questionnaire, general physical examination, lameness examination and gait analysis using gyroscopes, resting and dynamic upper airway endoscopy, bronchoalveolar lavage, echocardiograms, exercising electrocardiograms and measurements of lactate, PCV, heart rate, CK and sweat response before, during and after exercise. The simultaneous evaluation of different body systems and fitness is useful for the investigation of poor performance and as a preventative medicine approach for the management of high level athletes. We believe that each member of a team composed of a primary care veterinarian, trainer and specialists in internal medicine, surgery and sports medicine and rehabilitation can solve a part of the poor performance or preventative medicine equation.

References

Acute Non-Weight-Bearing Lameness
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Diseases may result in acute non-weight-bearing lameness because of severe pain and/or instability. Causes of pain severe enough to result in non-weight-bearing lameness are often related to restriction of inflammation or infection within a closed space resulting in distension (i.e. synovial structure, facial plane, etc.). In these cases, reducing the inflammation, resolving the infection, and relieving the distension are the keys to improving the comfort of the horse. Horses with instability of their limb resulting in acute non-weight-bearing lameness often have pain associated with the cause of instability, but are additionally distressed due to their inability to bear weight. Providing stabilization, most commonly with external coaptation is important to minimize stress, discomfort, and further damage prior to definitive treatment.

The first step in evaluation of a horse with a non-weight-bearing lameness is performing a triage examination. A triage exam is designed to determine if a patient is in shock and requires immediate treatment or if the remainder of the physical examination and additional diagnostics can be performed prior to treatment safely. The triage examination focuses on rapid evaluation of three organ systems: neurologic, respiratory, and cardiovascular. The clinical signs are primarily related to either poor perfusion (cardiac output) or the body’s compensatory mechanisms to improve perfusion and arterial oxygenation. Although relatively uncommon, shock due to hemorrhage or severe dehydration can occur in horses with non-weight-bearing lameness. The cardiovascular triage examination should include heart rate, mucus membrane color, capillary refill time, jugular refill, pulse quality, and temperature of the extremities. Clinical signs of pain can be difficult to differentiate from signs of inadequate perfusion. For example, both can result in increased heart rate, pale mucus membranes, and cool extremities as a result of increased sympathetic tone. Signs such as poor pulse quality, prolonged capillary refill time, and poor jugular refill should not occur as a result of pain and are likely signs of hypovolemia. Hemorrhage associated with fractures and other injuries resulting in non-weight-bearing lameness can be difficult to identify and quantify. Some of the most severe hemorrhage can occur with fractures of the upper limb, including fractures of the humerus, femur and pelvis. These fractures are in close proximity to major vessels and surrounded by a large amount of musculature making significant blood loss without external evidence of blood possible. Lacerations in the upper limb are also more likely to result in severe hemorrhage. It is also important to remember that the trauma that caused the limb injury may have involved other regions of the horse such as the abdomen or thorax where bleeding could occur without external evidence. Horses that sustain a fracture or injury far from a source of water can become dehydrated. As noted, luckily, most horses will be stable enough to continue examination.

Causes of non-weight-bearing lameness associated with pain can often be identified rapidly on physical examination. As noted, many of the causes are associated with inflammation and/or infection being trapped within a confined space. One of the most common examples is a sub-solar abscess. Diagnosis of sub-solar abscess is based on clinical signs localizing the lameness to the foot including lack of other obvious findings within in the limb (swelling at the coronary band, pastern region can occur uncommonly), increased digital pulses, heat, and sensitivity to hoof testers. While these findings most consistently indicate an abscess, clinical signs will be identical in horses with fractures of P3 and this should be considered as a differential in cases with suggestive history or not responding to therapy. Another important example is infection of a synovial structure. Depending on the structure affected heat, pain, and swelling (effusion and periarticular) can be used to localize the problem. In adult horses, infection is generally secondary to a wound or injection/surgery. Careful palpation for un-noticed wounds and a good history can provide important information for these cases. Definitive diagnosis will require cytology and culture of the affected synovial structure. Other, less common, examples include myositis and cellulitis, which can often be identified with palpation and examination. Causes of non-weight-bearing lameness associated with pain without instability that are not the result of inflammation and/or infection being trapped within a confined space include non-displaced and/or incomplete fractures and lacerations to other soft tissues. Non-displaced and/or incomplete fractures are often difficult to diagnose and can become catastrophic if not suspected. Veterinarians must have a high index of suspicion based on history and physical examination findings and recommend strict stall rest (with or without tying) while awaiting definitive diagnosis or rule out of the fracture. Nuclear scintigraphy is the most accurate imaging modality for these types of fractures. Uptake will be greatest once remodeling is underway (7-10 days). Multiple radiographs in many planes will often miss these fractures until fracture remodeling and periosteal callus formation has started.

Causes of non-weight-bearing lameness associated with instability of the limb are generally not a diagnostic challenge and frequently examination even from some distance will give a diagnosis. Sources of instability that are frequently easily diagnosed on visual examination include complete, displaced fractures, disruption of tendons or ligaments required for weight bearing (ex: suspensory apparatus breakdown), and joint luxation. However, some causes of instability are less obvious including intermittent or sub-luxation of a joint and nerve paralysis/paresis. Fractures may be less obvious in the upper limb where palpation for swelling and
The length of the tube should be the height of an adult horse’s withers or hip to be used for full limb splinting. The splint can be of correct size, inexpensive, and readily available. PVC tubing fulfills these criteria and is frequently recommended for splinting horses.

The application of casts will not be discussed in this presentation. The ideal splinting material is strong but lightweight and available, alternatives include wood boards or multiple broom handles bound together. Casting material can also be used to make bandage casts or splint/cast combinations can be used if the veterinarian is comfortable with casting and has casting material is easily shortened for half limb splinting. The diameter of the tube should be large enough to provide 3-4 splints that are approximately 4-6 inches wide with a gentle curve. If a splint is too curved it creates gaps between the splint and the bandage. If PVC is not available, alternatives include wood boards or multiple broom handles bound together. Casting material can also be used to make splints. It is easily molded to the correct length and shape. Casting material is particularly useful when making splints for foals.

There are some important general principles that can be used to optimize stabilization with splints. They provide guidelines for bandaging, splint length, and placement.

1. A splint should span the joint above and the joint below the fracture. In most cases splints are started at the ground. Ending a splint or bandage at or below the fracture should be avoided at all costs because it results in a lever arm that will increase motion at the fracture.
2. If possible, two splints should be used to stabilize the limb preventing motion in the dorsal to palmar/plantar or cranial to caudal plane and the medial to lateral plane.
3. The bandage beneath a splint must be composed of multiple thin layers to avoid compaction and shifting of excessive padding. Each layer should be composed of cotton padding (sheet cotton, roll cotton, or combine) wrapped with brown gauze to make a smooth, even layer. Layers that are <2 cm thick are recommended. I prefer using roll cotton because I can create the most even bandage that way. A layer of vetwrap or elasticon can be placed over the second layer. A bandage that is 2-3 layers provides adequate padding and results in the leg being a uniform tube to minimize dead space. Stabilization is optimal when the stabilization is as close to the center of gravity of the limb as possible (i.e. internal fixation). Additional layers of padding (as previously recommended) provide little/no additional stabilization and allow more compression, shifting, and/or bunching of the bandage.
4. Motion at the fracture is minimized by preventing movement of the splint.
   a. The shape of the splint should match the shape of the bandaged limb. The bandage should be used to provide a smooth tube so that the splint is in close contact along the entire bandage. Increased padding can be applied in areas of indentation in the contour of the leg (like the palmar/plantar aspect of the pastern).
   b. The material used to affix the splint to the underlying splint should be inelastic. Inelastic materials that can be used include white athletic tape and duct tape. Duct tape is much less expensive, but does not breathe and the limb will sweat more.
c. The splint should be attached to the underlying bandage over as much of the length as possible. The attachment cannot be stopped at the level of the fracture for the same reason that the bandage and splint cannot be stopped at that level.

The above principles provide the basis for understanding how to splint specific regions/injuries in horses. The principles are adapted to the anatomy and mechanics of the region affected.

1. Instability at the level of the first and second phalanx of the forelimb and hindlimb are managed with splints from the ground to the level of the proximal metacarpus or metatarsus. It is vital to align the boney column—the dorsal aspect of the phalangeal bones and the metacarpus or metatarsus should form a straight line. This can be accomplished by placing the horse on its toe and applying two splints (dorsal or palmar/plantar and lateral) to the limb. A Kimsey splint will align the boney column, but only provides stabilization in the dorsal to palmar/plantar plane (which may be all that is necessary with a soft tissue injury). Medial to lateral stabilization can be augmented with the addition of a lateral splint. Kimsey splints are very easy to apply and are available for forelimbs and hindlimbs. Disadvantages include expense and weight of the splint. Additionally, the Velcro loses its stickiness with repeated use and needs to be reinforced with tape along the length of the splint.

2. Disruption of the suspensory apparatus causes instability only in the dorsal to palmar/plantar plane. Breakdown of the suspensory apparatus is the injury that sparked the design of the Kimsey splint.

3. Third metacarpal fractures are stabilized with a caudal and lateral splint from the ground to the level of the elbow. Aligning the boney column will require placing the horse on the toe similar to what is needed for P1 and P2 fractures.

4. Third metatarsal fractures are stabilized with a lateral splint from the ground to the level of the stifle. When a straight splint is used, in order to support the center of weight bearing of the limb, it will start just plantar to the heel and pass cranial to the hock and caudal to the stifle. Due to the anatomy of the hindlimb, it is not possible to place a caudal or cranial splint that will truly immobilize the hock. However, a caudal splint can be placed from the ground to the level of the proximal aspect of the calcaneus. Although it does not immobilize all of the joints of the hock, it does restrict the movement of the distal joints and decrease the motion of the tarsocural joint. Additionally, the caudal splint is helpful to align the boney column of the metatarsus and phalangeal bones. If custom splints are used, they can be created to fit the contour of the limb.

5. Radius fractures are stabilized with a lateral splint from the ground to the shoulder or withers. The bandage and the attachment of the splint to the bandage should go as proximal as possible. It is vital that they are proximal to the location of the fracture. It is not possible to firmly affix the splint to the body above the elbow. Some have recommended figure eight bandaging, but the restriction in motion appears minimal. Although a caudal splint cannot completely immobilize the elbow, a caudal splint is frequently placed to the level of the proximal olecranon to decrease the motion at the elbow joint.

6. Carpal instability can be stabilized in a manner similar to radial fractures.

7. Tibial fractures are stabilized with a lateral splint from the ground to the level of the hip. The splint will follow the center of weight bearing of the limb. The bandage and the attachment of the splint to the bandage should go as proximal as possible. It is vital that they are proximal to the location of the fracture. It is not possible to firmly affix the splint to the body above the stifle. The addition of a customized cranial or caudal splint may be useful.

8. Tarsal instability can be stabilized similar to tibial fractures.

9. Olecranon fractures, some humeral fractures and radial nerve paralysis result in a “dropped elbow appearance”. Olecranon fractures do not require stabilization of the fracture to allow the horse to bear weight. The olecranon is not a weight bearing bone, but it is integral to weight bearing because of its role in the stay apparatus and locking the carpus. Horses with olecranon fractures that have a “dropped elbow” will be able to bear weight if a splint is placed to lock the carpus in extension. A caudal splint from the ground to the elbow is appropriate. It is important to differentiate a horse with a “dropped elbow” due to an olecranon fracture from one due to a distal humeral fracture because splinting a humeral fracture with a splint that ends at or below the elbow will provide a lever arm and increase motion at the humeral fracture.

10. Humerus and femur fractures are not amenable to splinting. Splints do not provide adequate stabilization of the joint above or below these fractures. Splinting may allow the horses to bear more weight, but without stabilization of the fracture this can lead to more damage.
Field Evaluation of Colic
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The goals of evaluating a horse showing signs of colic in the field are to localize and categorize the cause of colic and to use that information to determine whether treatment in the field, referral, or euthanasia are most appropriate. Most frequently, the cause of colic can be localized to the gastrointestinal tract, more specifically to either the small or large intestine. Less commonly, diseases of the stomach, non-gastrointestinal organs, or peritoneum can result in colic. Within the small and large intestine, the majority of causes of colic can be categorized as non-strangulating (simple) obstructive, strangulating obstructive, or inflammatory. History, signalment, and a directed colic examination, including physical examination, nasogastric intubation, and examination per rectum, are the core of a basic colic work-up. In most cases, these basics will provide enough information for you to localize and categorize cause of colic and make treatment recommendations to your client. Advanced diagnostics that can be performed in the field include ultrasound, abdominocentesis, bloodwork, and fecal diagnostics. Knowing how to perform and interpret these tests in a field situation is valuable. However, it is important to realize the limitations of these tests and that not all of the information from some of these diagnostics will be available immediately, i.e. on an emergency basis, in the field. The use of advanced diagnostics should be reserved for cases where the results are required to localize and characterize the lesion or, more importantly, allow you to help you client make the decision to treat, refer, or euthanize their horse.

A complete history should include information about the current episode of colic as well as previous medical history and management and performance history. Information about the current episode of colic should include duration and severity of signs, changes in quantity, frequency, and quality of manure production, and any medications administered. Questions about previous relevant medical history should determine frequency, severity, and cause of previous episodes of colic, breeding, pregnancy and parturition details, deworming and vaccination protocols, ongoing illnesses, and any medications or supplements. Examples of associations between history and lesion localization and characterization include: horses with strangulating obstructions are typically acutely, severely painful whereas horses with inflammatory lesions are typically depressed and mildly painful, stallions with inguinal hernia may have a history of recently breeding, antibiotics can lead to antibiotic associated colitis, and alfalfa hay has been associated with cantharadin toxicity and enterolithiasis.

The signalment of a patient includes age, gender, and breed. Signalment has been associated with the prevalence, cause, and clinical signs displayed during colic. Examples of associations between signalment and lesion localization and characterization include: variations in gastric ulceration in foals of different ages and the tendency for older horses, gaited breeds and draft horses to behave stoically and show less overt signs of colic than expected with the underlying disease.

As with any emergency, the first part of the examination of a horse showing signs of colic is a triage exam. A triage exam is designed to determine if a patient is in shock and requires immediate treatment or if the remainder of the physical examination and additional diagnostics can be performed prior to treatment safely. The triage examination focuses on rapid evaluation of three organ systems: neurologic, respiratory, and cardiovascular. The clinical signs are primarily related to either poor perfusion (cardiac output) or the body’s compensatory mechanisms to improve perfusion and arterial oxygenation. Signs of shock are more likely to occur in addditional diagnostics can be performed prior to treatment safely. The triage examination focuses on rapid evaluation of three organ systems: neurologic, respiratory, and cardiovascular. The clinical signs are primarily related to either poor perfusion (cardiac output) or the body’s compensatory mechanisms to improve perfusion and arterial oxygenation. Signs of shock are more likely to occur in horses showing signs of colic associated with strangulating obstruction and inflammatory lesions and are uncommon in horses with non-strangulating obstructions. In my experience, large intestinal strangulating and inflammatory lesions are particularly likely to result in shock.

Once the triage examination has been completed, a complete physical examination should be performed. Examples of associations between physical examination findings and lesion localization and characterization include: abdominal distension is more common with lesions of the large intestine than the small intestine, horses with inflammatory lesions are the most likely to have a fever, and some changes in mucus membrane color such as cyanosis may occur with non-gastrointestinal causes of colic such as cardiac (CHF) or respiratory (pleurupneumonia) diseases.

The quantity and quality of nasogastric reflux should be determined in all horses showing signs of colic. A nasogastric tube should be passed immediately if the heart rate on initial examination is greater than 60 bpm. Greater than 2L of nasogastric reflux is considered abnormal. Stomach contents should normally be green with some flecks of feed material present and smell of fresh grass. In general, small intestinal diseases are more likely to result in net nasogastric reflux. Within small intestinal disease, the highest quantity of reflux occurs with inflammatory disease (anterior enteritis). Additionally, reflux from horses with anterior enteritis may be bloody and fetid smelling. The amount and time from initial signs of colic until onset of reflux for horses with obstructive small intestinal lesions will depend on the location of the lesion.

Unless there is a contraindication, such as risk to the horse or veterinarian, all horses showing signs of colic should have an examination per rectum. When performing examination per rectum, the first thing to determine is if the examination is normal or abnormal and the second is to determine if the abnormality is distension of the large or small intestine. Identification of a specific

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underlying cause of colic is uncommon. Distension of the large intestine is characterized by the size of the distended structure the presence of tight bands on the distension. Distension can be caused by gas, fluid, or ingesta. Specific causes of large intestinal colic that can be identified on examination per rectum include left dorsal displacement of the large intestine and impactions of the small colon, cecum, or pelvic flexure. Fluidy distension suggests colitis. Other large intestinal diseases generally result in non-specific distension. Small intestinal distension is also characterized by the size of the distended structure and the absence of bands. Most causes of small intestinal disease are not associated with specific findings on examination per rectum. Ileal impactions can be palpated in some cases. Distension with anterior enteritis is generally less turgid and the intestine may feel thickened. Strangulated small intestine can also be thickened.

Portable ultrasound machines have become widely available and increasingly affordable for use in equine practice. While the same machine can be used for transrectal, musculoskeletal, transabdominal, and thoracic imaging, using the most appropriate probes will optimize the quality and types of images you are able to obtain. In horses showing signs of colic, transabdominal ultrasound examination is most commonly performed. In some cases, transrectal ultrasound and thoracic ultrasound are indicated as well. For transabdominal ultrasound, a low frequency (3-5 MHz) curvilinear probe is recommended to provide the maximal depth penetration. Alternatively, higher frequency probes such as a 5-7.5 MHz transrectal linear probe or a 5-8 MHz Microconvex probe can be used for transabdominal image acquisition, but will not be able to reach the same depth as the 3-5 MHz curvilinear probe. Regardless of the probe used, the best resolution of the image will be obtained if the minimal depth required is maintained and the focal distance is appropriately adjusted. Any of the probes discussed related to transabdominal ultrasound will work well for most aspects of transthoracic ultrasound because relatively minimal depth penetration is required with the exception of echocardiography. Transrectal ultrasound is performed with a linear probe designed for that purpose. Appropriate restraint (physical and chemical) and technique must be used when performing transrectal ultrasound just as for any examination per rectum.

Transabdominal ultrasound is non-invasive and safe to perform in most horses showing signs of colic. If possible, ultrasound should be performed in a dark area and seeing the image will be quite difficult in direct sunlight. For the best image quality, the skin should be clipped and cleaned and coupling gel should be used. However, an adequate image can usually be obtained by soaking the hair with alcohol. Horses with thick, long, and/or dirty coats are more likely to require clipping. Image quality is also decreased in horses with excessive fat. Similar preparation considerations should be used when performing transthoracic ultrasound.

As with any examination, each clinician should develop a systematic and consistent method for ultrasound. A complete transabdominal ultrasound involves imaging each rib space (from approximately ICS 7/8 caudally) and the paralumbar fossa as well as the ventral abdomen and inguinal areas on both left and right sides. Depending on the amount of gas within the visceras and several other factors such as age, portions of the gastrointestinal tract (stomach, small intestine, cecum, large colon, and small colon), spleen, liver, kidneys, and bladder as well as peritoneal fluid can be imaged. With regards to the gastrointestinal tract, ultrasound is useful in evaluating wall thickness, motility, distension, and contents. Depending on the information that is being sought, the question being asked, an abbreviated, targeted transabdominal ultrasound may be more useful in cases of acute colic. A complete transthoracic ultrasound involves imaging each rib space on each on both right and left sides. Thoracic ultrasound is useful for evaluating surface of the lung, the pleural space, and the heart.

Transabdominal ultrasound is indicated when it can provide information that a basic colic examination cannot or when it is necessary to improve confidence in a diagnosis and help an owner make a decision. One set of indications is for situations where performing an examination per rectum is dangerous for the patient or veterinarian. It can be safely performed in patients too small for examination per rectum like foals and some miniature horses and ponies. It may also be safely performed in recumbent horses and fractious horses with appropriate sedation/restraint. For gastrointestinal lesions, transabdominal ultrasound is particularly useful for identifying and evaluating the distension, motility and wall thickness of small intestine, intussusceptions, fluid contents within the large intestine, and ruling out left dorsal displacement of the large colon when the spleen and kidney can be imaged adjacent to one another. Transabdominal ultrasound is more sensitive than examination per rectum for identifying small intestine and is indicated to identify small intestine not palpable on examination per rectum if other findings suggest a small intestinal lesion. Distended, amo bile, thickened small intestine is a typical appearance for a strangulated segment of small intestine. Increased wall thickness occurs with large colon volvulus and may also occur with inflammatory lesions. Increased fluid contents of the large intestine are consistent with colitis. When trying to differentiate gastrointestinal lesion location, ultrasound is most useful for small intestinal identification. For non-gastrointestinal causes of colic, transabdominal ultrasound is useful in quantifying and qualifying peritoneal fluid and evaluating the architecture of the liver and spleen. It can be particularly useful identifying the characteristic swirling fluid associated with hemoabdomen and can provide valuable improved confidence and decision making is a horse with ruptured gastrointestinal tract locating a site for abdominocentesis to ensure the sample is not an enterocentesis.

Transthoracic ultrasound has limited indications in horses showing signs of colic. The only cause of true colic that it is particularly useful in identifying is diaphragmatic hernia. However, several thoracic diseases can present as colic—either due to the tissue hypoxia they create or an inability to distinguish thoracic pain from abdominal pain. Examples of thoracic disease that can present as colic are congestive heart failure and pleuropneumonia. Transrectal ultrasound should be considered in two types of cases. The first
is when the reproductive tract (particularly in mares) is the suspected source of colic. The other is any case where it may help characterize an abnormal finding from examination per rectum, such as a mass.

Site selection for abdominocentesis can be empirical or ultrasound guided. The most common locations selected empirically are just behind the xiphoid on midline or a hands breadth to the right of midline at the most ventral aspect of the abdomen. Ultrasound can be used to identify a pocket of fluid or to avoid gastrointestinal structures or spleen to be confident that the fluid is not an enterocentesis or splenic sample. It is important to note, that in a normal abdomen there may be no fluid or only small pockets seen intermittently on ultrasound. Despite this, abdominal fluid can be obtained in most cases. Once the site is selected, it should be clipped and prepared in aseptic fashion.

There are two basic techniques for performing abdominocentesis: using a needle or with a blunt tipped catheter such as a teat cannula or bitch catheter. In most horses, an 18 gauge, 1.5” needle can reach an adequate depth, but a spinal needle may be required in very fat horses. When using a needle, it is not necessary to block the site with local anesthetic. The needle should be advanced slowly until fluid is obtained. In some cases, due to the negative intra-abdominal pressure, it may help to use a second and third needle for sampling. When using a teat cannula or bitch catheter, the site is blocked with local anesthetic and a blade is used to incise the skin, subcutaneous tissue, and nick the external rectus sheath (off midline) or linea. Off midline, the teat cannula or bitch catheter must be pushed through the external, rectus abdominus muscle and internal rectus sheath. At either location, the peritoneum must be penetrated. The peritoneum cannot be blocked and most horses react when the teat cannula or bitch catheter tents it prior to puncture.

The sample should be collected in a purple top (EDTA) tube and a red top (serum tube). If possible, a purple top with liquid EDTA should be used and excess EDTA should be shaken out so that it does not falsely elevate protein readings. Information that can be gained from the peritoneal fluid in the fluid includes gross appearance and possibly total protein and lactate if a refractometer and lactatometer are available. Cytological analysis and bacterial culture and sensitivity will require submission to a laboratory. Important diagnoses that are suggested by gross appearance include peritonitis, hemoabdomen, and strangulating lesions. Fluid from horses with peritonitis will be opaque and white/yellow/pink in color. Fluid from a hemoabomen will have a high PCV (allowing the tube to settle will help estimate this) and will not clot (it will if it is a splenic sample). Fluid from a horse with a strangulating lesion will become serosanguinous. Rarely horses with inflammatory lesions can also have serosanguinous fluid. It can be difficult to differentiate this from blood contamination. Increases in total protein (>2-2.5 g/dl) occur in both strangulating and inflammatory diseases. Minimal or no increase in total protein is seen with non-strangulating lesions. In inflammatory diseases, the increase is more marked than the increase in WBC count, but that number will not be known in field conditions. Increases in peritoneal lactate (>2 mmol/L), especially if they are greater than the increase in lactate in peripheral blood, are most common in strangulating lesions. However, non-strangulating and inflammatory lesions can increase peritoneal lactate in some cases.

Abdominocentesis is indicated if the information gained will alter the client or veterinarian’s decision for referral. If an owner is unable or does not wish to treat a serious condition such as hemoabdomen or strangulating lesion, euthanasia may be recommended over referral based on abdominocentesis. If gastrointestinal rupture can be diagnosed in the field, the horse may not have to endure the trip to the referral center prior to euthanasia. Abdominal fluid analysis is also useful in characterizing small and large intestinal lesions, although typically changes occur more quickly with small intestinal lesions. Strangulating lesions are typified by a serosanguinous appearance, increased total protein, and increased lactate. Inflammatory lesions increase total protein and may rarely result in serosanguinous appearance. Non-strangulating lesions result in little to no change in the abdominal fluid.

Most bloodwork requires submission to a laboratory and is therefore rarely indicated in cases of acute colic in the field. Blood lactate can be measured in the field and may help support a diagnosis of a strangulating lesion. However, in my opinion, decisions should not be based on a single blood lactate measurement. It is nearly impossible to be sure that the increase in blood lactate is due to ischemia of the gastrointestinal tract rather than total body poor perfusion. In cases of horses with more chronic or recurrent colic, complete bloodwork can be useful in identifying inflammation and impairment of renal or hepatic function.

As with bloodwork, most fecal diagnostics require submission to a laboratory and are therefore rarely indicated in cases of acute colic in the field. One test that can be performed in the field is placing feces in a rectal sleeve, adding water, and determining if sand sediments in the fingers. This test has significant limitations in that it is not standardized and does not reliably predict significant sand when it is positive nor reliably predict no sand when it is negative. In cases of horses with more chronic or recurrent colic, fecal diagnostics for infectious diseases can be performed.
Field Treatment of Colic
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Treatment of horses with colic in the field will fall into two main categories—horses that require treatment to allow evaluation and stabilization prior to referral or euthanasia and horses with diseases that can be managed effectively, economically, and efficiently in the field. The determination of which category the horse you are attending falls into can be made based on lesion localization and characterization. Field evaluation of colic is the topic of a separate presentation, but will include a minimum database of history, physical examination, nasogastric tube intubation, and examination per rectum. Most frequently, the cause of colic can be localized to the gastrointestinal tract, more specifically to either the small or large intestine. Less commonly, diseases of the stomach, non-gastrointestinal organs, or peritoneum can result in colic. Within the small and large intestine, the majority of causes of colic can be categorized as non-strangulating (simple) obstructive, strangulating obstructive, or inflammatory.

Horses with strangulating lesions obviously fall into the first category, treatment for evaluation and stabilization prior to referral or euthanasia. Most horses with inflammatory lesions will also fall into the first category for several reasons including: they are at high risk for hypovolemic and/or distributive shock, once stabilized they will likely require intensive medical management, and the risk of infectious etiologies makes biosecurity a priority. However, if the owner is willing to manage the biosecurity risk, some horses with mild inflammatory lesions can be treated effectively in the field. Field treatment of horses with inflammatory lesions should be restricted to horses that are not in shock, relatively comfortable, and able to take in oral fluids (not refluxing). Thus, most of the horses that fall into the second category, with diseases that can be managed effectively, economically, and efficiently in the field, have non-strangulating (simple) obstructions. The decision of referral or field treatment in these cases must be made based on the clinical signs of the horse (pain, response to analgesics, duration) combined with the owner’s level of comfort and financial considerations. Always remember when considering costs that, although in the end it is your choice, you should be charging for your services and time.

Treatments required to allow evaluation and stabilization prior to referral include analgesics, anti-spasmodics, and intravenous fluid therapy. Treatments for horses that can be managed in the field can be divided into non-specific, symptomatic therapies of analgesics, anti-spasmodics, and trocharization and specific therapies for impactions, mild inflammatory lesions, and left dorsal displacement of the large colon. Analgesics used in the field treatment of colic include non-steroidal anti-inflammatories, alpha-2 agonists, and opioids.

Non-steroidal anti-inflammatories inhibit cyclooxygenase and decreased the production of prostaglandins and thromboxane. Flunixin meglumine is the most common non-steroidal anti-inflammatory used in horses with colic. Flunixin meglumine has analgesic, anti-inflammatory, anti-endotoxic, and anti-pyretic effects. Other non-steroidal anti-inflammatory drugs include phenylbutazone and firocoxib. In one study firocoxib was as good as flunixin meglumine at controlling postoperative visceral pain. In addition to their role inflammation, prostaglandins are important for acid regulation in the stomach and blood flow to the gastrointestinal tract and kidney. As a result, toxic effects of non-steroidal anti-inflammatories include stomach ulceration, right dorsal colitis, and renal tubular necrosis.

Alpha-2 agonists activate α2 (and to a lesser extent α1) adrenoreceptors in a variety of tissues. The α2 effects in the central nervous system are responsible for the desired effects of sedation and analgesia. They also produce muscle relaxation and decreased reactivity to painful stimuli and procedures. The α2 agonists that are currently widely available for use in horses and most practitioners should be familiar with are xylazine, romifidine, and detomidine. The main differences between the drugs are in their duration of action and potency (xylazine>romifidine>detomidine). α2 agonists produce an initial period of hypertension associated with vasoconstriction followed by hypotension due to bradycardia (with the potential for AV block) and resultant decreased cardiac output. α2 agonists are respiratory depressants decreasing respiratory rate and tidal volume and increasing upper airway resistance. α2 agonists markedly reduce intestinal motility. α2 agonists also produce hyperglycemia and diuresis (inhibit ADH). In horses with colic, α2 agonists are valuable for facilitating diagnostics and assessing severity of pain. Due to the cardiovascular, respiratory, and gastrointestinal effects of α2 agonists, these systems should be assessed prior to administration with a brief physical examination. Once given, it is important to monitor the duration of sedation and pain relief. I typically expect pain relief for ≥ 20 minutes with xylazine, ≥ 30-40 minutes with romifidine, and ≥ 60 minutes with detomidine. To allow for timely assessment, xylazine is generally chosen for initial sedation.

The most commonly used opioid in equine practice is butorphanol. Butorphanol is an opioid agonist (κ)-antagonist (μ) that produces analgesia through κ opioid receptors primarily located in the brain and spinal cord. Because of it’s greater activity at κ opioid receptors, it is believed to be effective in treating visceral pain. In horses, stimulation of opioid receptors can result in hyperexcitability and increased locomotion. These effects can be minimized by coadministration with α2 agonists. Butorphanol has the potential to induce respiratory depression, but this effect is generally minimal in horses without respiratory compromise that are not heavily sedated or anesthetized. Butorphanol decreases gastrointestinal motility, but the clinical relevance of this side effect in
horses with colic is controversial. Butorphanol is generally used in combination with α2 agonists for a synergistic effect on sedation and analgesia. The combination is more potent and longer acting than α2 agonists alone and minimizes side effects of each. In my experience, butorphanol given without α2 agonists is a fairly weak analgesic and provides little sedation.

In the U.S., N-butylscopolammonium bromide (NBB) is the only commercially available anti-spasmodic. NBB is an acetylcholine receptor antagonist that inhibits muscarinic receptors in the parasympathetic autonomic nervous system. In the gastrointestinal tract, this inhibition results in decreased tone and motility. The parasympatholytic effects of NBB on the cardiovascular system result in tachycardia, hypertension, and rare cardiac arrhythmias. NBB is used to reduce abdominal pain. It is particularly effective in horses with spasmodic, or gas, colic where the pain is associated with hypermotility. It is also effective at reducing rectal pressure and rectal straining during rectal examination and, therefore, can be used to decrease the risk of complications.

Intravenous fluid therapy may be indicated in the field for stabilization of horses showing signs of shock based on triage examination in the field prior to referral. The goal of emergency fluid therapy is rapid restoration of preload, cardiac output, and oxygen delivery. This requires use of the intravenous route of fluid administration. A wide diameter (10-14g), short catheter with large bore fluid administration sets will maximize the speed of delivery. Depending on the type of fluid chosen and packaging, pressurized administration may be possible. The two main alternatives for rapid volume resuscitation are: 1) using a relatively small volume of hypertonic saline (2-4 ml/kg or 1-2 L/adult horse [not generally recommended in foals]) and/or synthetic colloids (5-10 ml/kg or 2.5-5 L/adult horse or 250-500 ml/foal) to borrow/pull fluid from the interstitium into the vascular space followed by administration of larger volumes of isotonic crystalloids (at least 20 ml/kg) or 2) using isotonic crystalloid fluid alone. In either case, the “shock dose” of 80-90 ml/kg can be used as a guideline for the maximum amount of isotonic crystalloids to administer as a bolus. In general, it is rare to need the entire “shock dose”. I recommend starting with a 20 ml/kg bolus (10 L/adult horse or 1 L/foal), reassessing clinical signs/physical examination parameters, and determining if additional boluses are required. In horses with diseases that are associated with protein loss, the use of colloids in the resuscitation plan might have additional benefits. In the case of hemorrhagic shock, whole blood should be considered as part of the fluid resuscitation plan.

Trocharization of the large intestine can be performed in the field in selected cases. Trocharization may be effective in relieving pain and distension of the large intestine in cases of non-strangulating lesions of the large intestine. For cases of severe spasmodic colic, this treatment may be definitive. Whereas in cases of displacement or impaction, trocharization may provide enough relief to allow further treatment and time for resolution. Although complications of trocharization appear to be rare, the potential for severe complications (rupture and abscess formation) is possible. These risks must be clearly explained to the owner prior to performing trocharization particularly in cases where referral (and/or surgery) is an option. The procedure is simple to perform. A site is chosen, generally the right flank, using clinical exam (ping, rectal, distension) and/or ultrasound. The site is clipped, prepared aseptically, and blocked. A 14 gauge catheter (over the needle) with an extension set placed into a cup of water is slowly advanced with the stilette in place for 2-3 cm past where bubbles start exiting. Some clinicians will advance the catheter past the needle or remove the stilette at trocharization particularly in cases where referral (and/or surgery) is an option. The procedure is simple to perform. A site is chosen, generally the right flank, using clinical exam (ping, rectal, distension) and/or ultrasound. The site is clipped, prepared aseptically, and blocked. A 14 gauge catheter (over the needle) with an extension set placed into a cup of water is slowly advanced with the stilette in place for 2-3 cm past where bubbles start exiting. Some clinicians will advance the catheter past the needle or remove the stilette at this time. My preference is to leave the stilette and catheter in the same location. Then, wait… Once the bubbles have stopped the catheter is removed. Some clinicians recommend injecting procaine penicillin G or gentamicin during removal. Some clinicians will also place the horse on a short course of antimicrobials.

Impactions can be effectively treated with oral laxatives. There are several mechanisms of action that cause loosening and/or softening of gastrointestinal contents by laxatives. Laxatives used in horses include bulk laxatives that provide volume and help retain water (psyllium), surfactants that help incorporate water and fat (dioctyl sodium sulfosuccinate [DSS]), lubricants that are slippery and decrease water absorption (mineral oil), and hydrating agents that help attract and retain water (isotonic balanced electrolyte solution or magnesium sulfate). DSS can be irritating and may facilitate the absorption of mineral oil or magnesium sulfate and associated toxicities. Overdoses of magnesium sulfate alone can also result in magnesium toxicity.

An isotonic balanced electrolyte solution has been shown to result in the largest increase in fecal water. Water has also been shown to effectively hydrate ingesta. Psyllium is frequently used for the prevention and treatment of sand impactions. Mineral oil can be used as a marker for intestinal transit in addition to it’s laxative activity. My preference for treating horses with large intestinal impactions is the use of isotonic balanced electrolyte solutions and/or water. When administering oral fluids/laxatives, I never exceed 6-8L of fluids at one administration time and always check for reflux prior to administration.

Specific treatments for horses with mild colitis include anti-endotoxic therapy with flunixin meglumine and di-tri-octahedral smectite. In blocking prostaglandin production, flunixin meglumine minimizes some of the inflammatory effects of endotoxin that contribute to the development of systemic inflammatory response syndrome. Flunixin meglumine can be administered at a low (quarter dose) every 8 hrs. Di-tri-octahedral smectite has been shown to adsorb endo- and exo-toxins. It can be administered via nasogastric tube or as an oral paste formulation and is thought to bind these toxins preventing them from entering the circulation and stimulating a cascade of inflammation.

Non-surgical treatments for left dorsal displacement of the large colon have been described and reported to have high success rates. Physical methods of jogging and rolling attempt to “jostle” the colon off of the nephropleenic ligament. In order to roll a horse, the horse is induced into general anesthesia and rolled from right lateral into dorsal (jostled around) and then into left lateral recumbency.
During jogging and/or rolling, there is the chance of gastrointestinal rupture. The administration of phenylephrine has also been described alone or combined with the physical methods. When administering phenylephrine horses should be monitored for reflex bradycardia and other arrhythmias. Fatal hemorrhage has been reported in horses administered phenylephrine. Most horses with hemorrhage were older, but owners should be informed of this risk.
There are a wide variety of indications for fluid administration in horses. They include volume restoration, rehydration, maintenance, ongoing losses, diuresis, electrolyte and/or acid base imbalance, parenteral nutrition, support of colloid osmotic pressure, immunoglobulin or coagulation protein supplementation, and improving oxygen carrying capacity. In general, fluids can be grouped into crystalloids and colloids based on their composition. When developing a fluid plan, one of the first things to decide is whether the horse requires rapid, emergency fluid therapy or if you are developing a 24-hour fluid plan. The underlying disease and reason for fluid administration will dictate the appropriate options for fluid type, amount, and route of administration.

Crystalloid fluids are categorized based on their tonicity. Tonicity refers to the gradient of osmotic pressure between two solutions across a semipermeable membrane. In the case of fluids, the tonicity is determined by the impermeable solutes in the fluid relative to the impermeable solutes within cells. Hypertonic fluids have more effective solutes, isotonic fluids have a similar effective osmoles, and hypotonic fluids have less effective solutes than within cells. In plasma and in fluids, the main solute that determines osmotic pressure is Na+. Glucose and BUN also contribute to osmotic pressure, but are not significant except during disease states. In fluids, dextrose is used to augment osmotic pressure in fluids containing less Na+. K⁺ can also contribute to osmotic pressure, but because the concentration is so low in comparison to Na⁺ in plasma and in fluids, it is generally ignored. The formula for calculating plasma osmolarity is

\[ \text{Osmolarity (mOsm/L)} = 2 \times [\text{Na}^+ (\text{mmol/L})] + \frac{[\text{Glucose (mg/dL)}]}{18} + \frac{[\text{BUN (mg/dL)}]}{2.8} \]

In addition to being defined by tonicity, crystalloid fluids can also be categorized based on their electrolyte and buffer composition as well as whether they are replacement or maintenance fluids. Most crystalloids are either straight NaCl or balanced electrolyte solutions with a buffer (acetate or lactate). Replacement fluids generally mimic the electrolyte composition of extracellular fluid (high Na⁺ and low K⁺, Ca²⁺, and Mg²⁺) while maintenance fluids generally mimic electrolyte composition of intracellular fluid (low Na⁺ and high K⁺, Ca²⁺, and Mg²⁺). Due to the low Na⁺ concentration, maintenance fluids many contain dextrose to make them isotonic or are hypotonic.

Whether crystalloid fluids are being administered intravenously, subcutaneously, or orally/intragastrically, they are being added predominantly to the extracellular fluid space. The extracellular fluid is composed primarily of plasma/intravascular fluid (1/5) and interstitial fluid (4/5). In some conditions transcellular or third space fluids are also included. Although vessel walls separate the plasma and interstitial fluid, the solutes can move fairly freely within the extracellular fluid space. For this reason, regardless of the route of fluid administration, redistribution within the extracellular space is expected. Most importantly, this results in retention of only 20% of intravascularly administered crystalloids within the intravascular space within approximately 30 minutes.

Colloids contain relatively large insoluble molecules that exert oncotic or colloid osmotic pressure (COP). Colloid solutions are grouped based on whether the molecules are natural or synthetic in origin. Natural colloids are blood products and albumin is the main molecule responsible for their COP. There are a variety of synthetic colloids, but the most widely available products for veterinary use currently are the hydroxyethyl starch solutions (HES). There are several HES commercially available that are differentiated by the molecular weight of the hydroxyethyl starch molecules as well as the carrier solution (isotonic NaCl or balanced polyionic solution).

Starling’s Equation is a simplified, theoretical explanation of the factors that control movement of fluid across the capillary wall. Fluid movement from the capillary into the interstitium is related to capillary wall permeability and area and the balance between hydrostatic and oncotic pressures within the capillary and the interstitium. The main force maintaining fluid within the capillary is plasma oncotic pressure.

Colloids are administered intravenously and the relatively large insoluble molecules remain within vascular space. As a result, administration of colloids contributes to the oncotic pressure. The increase in oncotic pressure not only decreases the movement of fluid from the vascular space into the interstitium, but can also pull fluid from the interstitium into the vascular space.

The most common indications for rapid, emergency fluid therapy are conditions that result in shock associated with decreased stroke volume due to decreased preload. Preload must be restored rapidly in order to prevent progression to decompenated shock and death. Less common indications for emergency fluid therapy include rapid correction of life threatening electrolyte and acid-base abnormalities or hypoglycemia. Most other conditions requiring fluid therapy can be treated less rapidly and frequently are best treated by designing a 24-hour fluid plan.

Shock occurs when the energy needs of cells are greater than the energy being delivered by the blood. Most commonly, there is a deficiency in oxygen delivery. Oxygen delivery is a product of oxygen content and cardiac output. Oxygen content is primarily dependent on [Hb] and SpO₂ and cardiac output is the product of heart rate and stroke volume with stroke volume determined by preload, afterload, and contractility. Although conditions that affect either component can result in shock, in horses it is more

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common for shock to be caused by decreased cardiac output. Specifically horses are affected by conditions that result in decreased preload. The two main types of shock seen in horses that are due to decreased preload are hypovolemic and maldistribution of fluids. Examples of hypovolemic shock are hemorrhagic shock and fluid loss associated with large volume diarrhea or nasogastric reflux. Examples of maldistributional shock are endotoxic and septic shock that result in massive vasodilation and venous pooling of blood.

Clinical signs of hypovolemic and maldistributional shock are similar and associated with poor perfusion and decreased intravascular volume as well as the body’s attempts to improve oxygen delivery. Physical examination findings associated with poor perfusion and decreased intravascular volume are cool extremities, prolonged jugular refill, prolonged capillary refill time, poor pulse quality, and decreased/absent urination. Physical examination findings associated with the body’s attempts to improve oxygen delivery are increased heart rate, increased respiratory rate, decreased/absent urination, and production of concentrated urine. If available, handheld lactatometers and/or blood gas monitors can be used to assess plasma lactate concentrations to support inadequate oxygen delivery.

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The two main alternatives for rapid volume resuscitation are: 1) using a relatively small volume of hypertonic saline (2-4 ml/kg or 1-2 L/adult horse [not generally recommended in foals]) and/or synthetic colloids (5-10 ml/kg or 2.5-5 L/adult horse or 250-500 ml/foal) to borrow/pull fluid from the interstitium into the vascular space followed by administration of larger volumes of isotonic crystalloids (at least 20 ml/kg) or 2) using isotonic crystalloid fluid alone. In either case, the “shock dose” of 80-90 ml/kg can be used as a guideline for the maximum amount of isotonic crystalloids to administer as a bolus. In general, it is rare to need the entire “shock dose”. I recommend starting with a 20 ml/kg bolus (10 L/adult horse or 1L/foal), reassessing clinical signs/physical examination parameters, and determining if additional boluses are required.

In horses with diseases that are associated with protein loss, the use of colloids in the resuscitation plan might have additional benefits. In the case of hemorrhagic shock, whole blood should be considered as part of the fluid resuscitation plan.

There are three main components to consider when calculating the volume of fluid for a 24-hour plan: maintenance, dehydration, and ongoing losses. In adult horses, maintenance requirements are 50 ml/kg/day and in foals, maintenance requirements are 80-100 ml/kg/day. In horses, estimating dehydration is very difficult. Traditional clinical signs of dehydration such as skin tent and dry/tacky mucus membranes have been shown to be completely inaccurate. Ongoing losses may be easy to measure, such as with horses that are producing nasogastric reflux, or difficult to determine, such as with horses with diarrhea or polyuria.

The route of administration for a 24-hour fluid therapy plan can be intravenous or oral/intragastric (as long as the gastrointestinal tract is functional). Oral administration of fluids is as effective for dehydration and more effective for hydration of ingesta compared to intravenous fluids.

Depending on the underlying disease, the electrolyte composition and colloid requirement will be variable. Although isotonic, polyionic crystalloids are generally a safe first choice, clinical pathology is useful for tailoring fluid therapy.
The bifurcation of the trachea divides the upper from the lower respiratory tract. The upper respiratory tract includes the nares, nasal passages, pharynx, larynx, and trachea. The lower respiratory tract is separated into the airways (bronchi, bronchioles, and alveoli), vasculature, interstitium, and pleural space. Differential diagnoses for respiratory distress can also be divided by anatomic location. Within the upper respiratory tract, bilateral nasal passage obstruction, mechanical and functional pharyngeal or laryngeal obstruction, and less commonly tracheal obstructions are possible. Examples of nasal passage obstructions include congenital choanal atresia or masses within the nasal passage or sinus (infectious, neoplastic, sinus cysts). Examples of pharyngeal obstructions include neurologic dysfunction or physical obstruction associated with swelling within the pharyngeal musculature (bleeding with longus capitus rupture), guttural pouches (typany), or retropharyngeal lymphadenopathy. Examples of laryngeal obstruction include bilateral laryngeal paralysis associated with systemic disease or toxins or physical obstructions such as artenoidal chondritis or subepiglottic cysts. Examples of tracheal diseases include intratracheal masses and tracheal collapse. Within the lower respiratory tract, infectious and non-infectious diseases of the airways, vasculature, interstitium and pleural space can result in respiratory distress. Inflammatory diseases of the airway such as RAO and SPAOPD can result in respiratory distress. Infection of the lower respiratory tract in horses affecting the parenchyma and airways alone (pneumonia) or involve the pleural space as well (pleuropneumonia) can also result in respiratory distress. Other diseases of the pleural space that can result in respiratory distress include hemotherax, pneumothorax, or pleural masses. Vascular causes of respiratory distress are very uncommon, but would include persistent fetal circulation and pulmonary hypertension secondary to congestive heart failure.

Observation of clinical signs and physical examination should be performed rapidly. They are generally sufficient to determine which portion of the respiratory tract is affected and often significantly further localize and prioritize differential diagnoses. The most obvious, consistent, and important clinical sign is respiratory noise. Horses that are making a respiratory noise have an upper respiratory cause of distress. Respiratory noise is the result of turbulent airflow. Turbulence, and respiratory noise, is most likely to occur during inspiration in the entire upper respiratory tract except the intra-thoracic trachea. This is because intra-airway pressure is negative, thereby promoting collapse and airflow disruption, during inspiration in the extra-thoracic airway. With more complete, rigid obstructions, respiratory noise may not be limited to the inspiratory phase. Further localization of the upper respiratory obstruction can be achieved with auscultation along the upper respiratory tract with a stethoscope to determine the point of maximal intensity, palpation along the upper respiratory tract for swelling, and dull percussion of the sinuses. Phase of dyspnea and symmetry of airflow and nasal discharge are important physical examination components for both upper and lower respiratory causes of distress. As with noise, dyspnea of the extra-thoracic airway will occur during inspiration and of the intra-thoracic airway will occur during expiration most frequently. Diseases of the pleural space decrease lung capacity and will result in a rapid, shallow breathing pattern. Changes in airflow can be detected by placing your hands or a mirror in front of the horse’s nostrils. Decreased/obstructed airflow and nasal discharge will be unilateral rostral to the nasal septum. Diseases of the pharynx that are related to the guttural pouch may also result in unilateral signs. Diseases caudal to the pharynx will uniformly result in bilateral decreased airflow and/or nasal discharge. Thoracic auscultation is the most important physical examination component for further localization of lower respiratory causes of respiratory distress. In horses in distress, rebreathing examination should only be performed if adequate examination is not possible without increasing respiratory effort and it is does not cause excessive distress to the horse. Abnormalities detected on auscultation may include increased bronchovesicular sounds, crackles, wheezes, or decreased/absent bronchovesicular sounds. Crackles are heard when fluid filled airways snap open and closed. This means that they occur when alveoli are filled with fluid preventing surfactant from minimizing surface tension appropriately. Wheezes occur when the airway is narrowed. Regional increases in bronchovesicular sounds (harsh or tubular lung sounds) can occur in areas of airway inflammation or consolidated lung. Regional decreases or absence of lung sounds can occur when portions of the lung are obliterated by abscesses or masses or, more commonly, secondary to accumulation of fluid (ventral) or air (dorsal) in the pleural space. Thoracic auscultation in horses with upper respiratory causes of distress will result in hearing referred upper airway noise. Once the upper airway obstruction is relieved, full thoracic auscultation should be repeated to ensure there is no lower airway involvement. Percussion of the thorax is an additional component of the physical examination that may be useful for localizing lower respiratory tract problems. It is useful to detect air/fluid or air/tissue interfaces such as detecting the extent of fluid in the pleural space in horses with pleuropneumonia or localizing areas of pulmonary abscesses, masses, or consolidation.

Once the cause of respiratory distress has been localized, emergency treatment can begin. Temporary tracheostomy is a life saving emergency procedure for horses with upper respiratory obstruction of the nares, nasal passages, pharynx, larynx, and trachea proximal to the tracheostomy site. Horses with lesions of the trachea distal to the temporary tracheostomy will not respond unless a tube can be
passed through the tracheostomy site past the obstructed area. In horses with lower respiratory tract causes of respiratory distress, intranasal oxygen therapy, if available, may improve oxygenation and clinical signs of distress. Which other emergency treatment is indicated will depend on the further localization and characterization of the cause.

When done on an “elective” basis, temporary tracheostomy is simple to perform and has a low incidence of complications. When performed in horses with severe respiratory distress, the risks of complications may increase because steps to ensure asepsis are often skipped and careful dissection is forgone in order to establish an airway as quickly as possible. For these reasons, I recommend performing the tracheostomy sooner than later in several situations to avoid difficulty, complications associated with a tracheostomy, and, most importantly, death. First, in horses that are at risk for developing respiratory distress during diagnostic procedures and/or awaiting improvement with treatment. Second, in horses that are at risk for developing respiratory distress and are going to be out of the direct supervision of a person (i.e. veterinarian) qualified to perform a tracheostomy, such as on a trailer or at a farm. Additionally, I strongly recommend that practitioners keep a preassembled kit, including include local anesthetic, gloves, a scalpel handle and blade, scissors, hemostats, 4 × 4’s, and several sizes of tracheostomy tube, for performing an emergency tracheostomy accessible in their truck and clinic. At minimum, it is valuable to have several options of sterilized, commercial tracheostomy tubes. Although a makeshift tracheostomy tube can be made from items a veterinary typically carries, they do require some manufacturing and therefore are not immediately available. Additionally, it is almost impossible to make one with a diameter as large as the commercially available tubes. They are also more difficult to place and maintain.

When performing a temporary tracheostomy, if the horse is excitable and/or distressed, sedation can be used in patients with upper respiratory distress. In fact, decreasing the respiratory effort with sedatives can reduce the degree of obstruction by decreasing the degree of negative intra-airway pressure. However, it should be noted that it also impairs pharyngeal and laryngeal function. If distress is very severe horses are frequently panicked and dangerous. In these cases it may be necessary to wait for the horse to pass out/fall down. This is obviously not a good situation for the horse or the veterinarian. Temporary tracheostomy is most commonly performed at the level of the junction of the cranial 1/3 and caudal 2/3 of the cervical trachea. Anatomically, this is located just cranial to the point where the sternocephalicus splits. In this location, there is less musculature overlying the trachea making tracheostomy easier to perform. The location may need to be modified if the horse has had a tracheostomy in the past or if it is likely that the horse will be having a permanent tracheostomy in the near future. If a permanent tracheostomy is expected, the tracheostomy should be performed as close to the split of the right and left sternocephalicus muscles to avoid interference with the surgical procedure. The selected area should be clipped and prepared aseptically. A line block with local anesthetic should be performed on ventral midline approximately 12-15 cm in length. In horses with severe obstruction, these steps may need to be brief or even omitted. The procedure begins with a 10-12 cm incision through the skin and cutaneous coli muscle on ventral midline. Division of the underlying paired sternothyrohyoideus muscles on midline will minimize bleeding. Pale fibrous tissue separates the left and right sides and can be incised with a scalpel or bluntly divided with scissors. Blunt dissection will minimize bleeding and prevent damage to the underlying trachea, but use of a scalpel is faster. The sternothyrohyoideus muscles are separated for a length of approximately 8 cm. This exposes several tracheal rings and the connecting annular ligaments. There is a loose connective tissue that overlies the trachea. Blunt dissection with scissors or using a sterile 4 × 4 results in better visualization. However, this step should be skipped in animals with severe respiratory distress. A scalpel blade is used to make a transverse stab incision through an annular ligament. It is very important to use a stab incision to enter the trachea to avoid separating the mucosal layer from the outer layers. Air will escape when the mucosa is incised. The incision is then extended with the scalpel blade or scissors. The tracheostomy should not exceed 1/3 to 1/2 of the circumference of the trachea. This is important to avoid injury to the adjacent vessels and nerves as well as stenosis of the trachea after removal. A variety of commercial temporary tracheostomy tubes are available. Tubes can be made of plastic or metal and may be self-retaining or may necessitate suturing to the skin or securing with gauze around the neck. If an inflatable cuff is present on the tube, it SHOULD NOT be inflated because pressure necrosis of the mucosa is possible. The largest tracheostomy tube that will fit should be used. My preference is the metal self-retaining tubes. If softer, plastic tubes are used, those that have a tapered end are generally easier to place. If no commercial tube is available a shortened plastic syringe or syringe case (as large as possible) can be used. A short piece of syringe or syringe case can be used to maintain separation of the rings in combination with a nasogastric tube to allow for a more flexible tube within the trachea. Other materials that have been used include the handle of a gallon milk jug and garden hoses. If tracheal collapse distal to the site of the tracheostomy is the cause of respiratory distress, a long endotracheal tube can be passed through the area of collapse to prevent collapse. Sutures encircling the tracheal rings adjacent to the tracheostomy site can be placed to make placement and replacement of the tube easier. Although they are not generally required, they can be useful when using stiff tubing. Upper respiratory obstruction can result in pulmonary edema due to alterations in intra-thoracic pressure, capillary permeability, and pulmonary vasculature tone. If present, foam may be evident in the trachea, crackles may be heard on thoracic auscultation, and the respiratory distress may not respond as expected to the tracheostomy. Treatment for pulmonary edema secondary to upper respiratory obstruction includes intravenous furosemide (1-2 mg/kg) (with caution in animals that are dehydrated) and oxygen if available. Following periods of respiratory distress and hypoxia, supplemental intranasal oxygen may benefit the patient.
Emergency treatment of horses with respiratory distress associated with inflammatory lower airway disease includes bronchodilators, corticosteroids, and environmental management. Bronchodilators can be administered intravenously, orally, or via inhalation from a metered dose inhaler with commercially available masks for horses. Bronchodilators are important to provide symptomatic relief, but do not treat the underlying cause of the disease. In an emergency situation, oral bronchodilators are the least rapid option and therefore not particularly useful. Intravenous anticholinergic drugs such as n-butylscopolamine and atropine are potent bronchodilators. Inhaled bronchodilators act rapidly, but also have a short duration of action and require repetitive dosing. Clenbuterol and albuterol are β-2 agonists and ipratropium bromide is an anticholinergic available as inhalers. Corticosteroids are the most potent anti-inflammatory drugs available and are a mainstay of treatment for inflammatory lower airway disease. As with bronchodilators, intravenous and/or inhaled administration will be preferred in an emergency situation. Inhaled corticosteroids may avoid some of the complications associated with systemic corticosteroids while providing high local levels of anti-inflammatory therapy. Environmental control is vital in the long-term management of patients with inflammatory airway disease and should begin immediately.

Emergency treatment of horses with infectious airway and interstitial disease (pneumonia) should include appropriate antimicrobials and anti-inflammatory drugs. However, it is important to remember that these will take time to work and that the horse’s level of distress may not improve much immediately beyond what it does with oxygen therapy.

Emergency treatment of respiratory distress associated with diseases of the pleural space may result in rapid improvement of the respiratory distress if the space occupying material in the pleural cavity can be removed (fluid or air, rather than a mass). Due to the effect of gravity, fluid accumulated ventrally and air accumulates dorsally. The site for fluid removal can be chosen empirically in the cranioventral lung field avoiding the region of the heart or with the ultrasound guidance. The site for air removal is generally chosen empirically and placed dorsally around the 13th intercostal space. Removal of fluid or air can be achieved with intermittent thoracocentesis using a teat cannula or continuous or intermittent drainage with an indwelling chest drain (8-10 French catheter). Removal can be active (applying suction to the end of the tubing) or passive. Placement of a teat cannula or indwelling drain is performed in the same manner. Placement at the cranial aspect of a rib avoids the intercostal vessels that run along the caudal edge of each rib. The area selected is clipped and prepared aseptically and local anesthetic is infused in the subcutaneous tissues and intercostal muscles. An incision just long enough to accommodate the teat cannula or drain is made in the skin, subcutaneous tissue and external fascia of the intercostal muscles. The teat cannula or drain is then inserted and advanced into the pleural space bluntly. Care should be taken to assure a slow and controlled advancement into the pleural cavity and precautions should be taken to avoid air being sucked into the pleural space during inspiration (negative pressure within the pleural space). When using a teat cannula, extension tubing should be connected to a three-way stop-cock and the skin should be closed when the teat cannula is removed. When using an indwelling chest drain, hemostats, a one way valve (Heimlich), or active suction can be applied to prevent air entering the pleural space and the tubing should be secured with a purse-string suture and Chinese finger trap to prevent leakage around the tube. Distress associated with bilateral accumulation of fluid or air may or may not respond to unilateral drainage depending on the amount of fluid or air that has accumulated and whether the mediastinum is incomplete or complete (in horses with pleuropneumonia, inflammatory proteins frequently clog fenestrations in the mediastinum). Complications can be associated with draining the pleural cavity, particularly if rapid drainage is performed. “Re-expansion pulmonary edema” is reported secondary to rapid drainage of pneumothorax in people. Evacuation of the air with suction applied at no more than 20 cm H₂O is recommended. Cardiovascular compromise associated with rapid removal of fluid from the third space can occur when large quantities of fluids are removed. Intravenous fluid support and controlled drainage should minimize this effect.

Once emergency treatment has been performed to improve stability and make the horse as comfortable as possible, further diagnostics can be performed to identify the underlying cause and choose the most appropriate therapy. For the upper respiratory tract, the most useful diagnostic for all causes will be upper airway endoscopy because it allows direct visualization of the obstruction. Additional diagnostics that may be indicated include radiographs, ultrasound, histopathology, hematology, and microbiology. Similarly, for the lower respiratory tract, diagnostic imaging, histopathology, hematology, and microbiology may be indicated. Diagnostic imaging of the lower respiratory tract frequently involves the combination of radiographs and ultrasound. Ultrasound can be performed rapidly with portable ultrasound machines whereas radiographs of an adult horse’s chest requires large x-ray units generally only available in referral hospital settings. Ultrasound provides information on the pleural space and the periphery of the lung while radiographs provide information on the lung parenchyma. Samples from the lower respiratory tract may include fluid obtained via transtracheal wash, bronchoalveolar lavage, and thoracocentesis or tissue obtained via biopsy.
The first question to answer is: what is shock? By definition, shock occurs when cells are unable to produce enough energy (adenosine 5′-triphosphate [ATP]) to meet the demands of the cell to function. Cells metabolize nutrients, such as glucose and volatile fatty acids, to generate ATP. To produce the most ATP per unit of nutrient, this process requires oxygen. For this reason, energy production can be limited by decreased availability of either nutrients or oxygen. It is far more common for a reduction in ATP production to be due to decreased oxygen availability than to insufficient nutrients. Specifically, most often shock is the result of inadequate amounts of oxygen reaching the cells (oxygen delivery [DO2]). In rare cases, adequate nutrients and oxygen reach the cells, but the cells are unable to perform aerobic metabolism or the cells use more energy than normal due to a disease process.

Because most shock is caused by decreased DO2, understanding DO2 is the key to understanding how diseases result in shock, how the body compensates for shock, which clinical signs are associated with shock and compensation, and how treatments will improve shock. Oxygen delivery (DO2) is the product of cardiac output (Q) and arterial oxygen content (C\text{a}O2) \( (DO2=Q\times C\text{a}O2) \). Q is the product of stroke volume (SV) and heart rate (HR) \( (Q=SV\times HR) \). SV is affected by filling of the cardiac chambers (i.e., preload), the force against which the heart contracts (i.e., vascular resistance or afterload), and the strength of cardiac contractions (i.e., contractility). SV is directly proportional to preload and contractility and inversely proportional to afterload. \( C\text{a}O2 \) is the product of oxygen carried by hemoglobin and dissolved in the blood (P\text{a}O2). The amount of oxygen carried by hemoglobin is the product of hemoglobin concentration in the blood ([Hb]) and the saturation of that hemoglobin with oxygen (S\text{a}O2). It is very important to recognize that the vast majority of oxygen in the blood is carried by hemoglobin and that very little is dissolved.

\[ C\text{a}O2 = 1.34\times[Hb]+S\text{a}O2+0.003\times P\text{a}O2 \]

There are a variety of ways to classify the causes of shock. One of the more complete and simple methods divides the causes of shock into 5 classifications: hypovolemic, distributive, obstructive, cardiogenic, and metabolic. Hypovolemic, distributive, obstructive, and cardiogenic shock all cause decreased DO2 by decreasing Q. In contrast, metabolic shock causes shock by a variety of mechanisms that can either decrease energy production or increase energy requirements by cells. Keep in mind that this classification scheme is overly simplified and often does not completely explain what is occurring in the patient due to the complexity of the diseases that cause shock with multiple pathophysiological changes occur concurrently.

Diseases that result in a decreased circulating (intravascular) volume are associated with hypovolemic shock. Decreased intravascular volume causes a decrease in preload, leading to a decrease in SV leading to a reduction in Q and a decrease in DO2. The decreased circulating volume can be due to losses of whole blood (hemorrhage) or free water or due to severe and/or prolonged lack of fluid intake. Fluid can be lost outside of the body or sequestered within the body, but outside of the circulating volume. The latter situation is referred to as ‘third space’ loss. In horses, diseases such as middle uterine artery rupture, guttural pouch mycosis, or large artery lacerations secondary to trauma can result in hemorrhagic shock. Examples of diseases in horses that result in fluid loss include gastrointestinal losses in diarrhea in animals with colitis or nasogastric reflux with anterior enteritis or losses in sweat. In horses, fluid can be sequestered within the colon in horses with a large colon volvulus or within the pleural space with pleuropneumonia.

Diseases that result in relative hypovolemia (i.e., a normal amount of intravascular fluid is distributed throughout an expanded intravascular space) are associated with distributive shock. Like true hypovolemia, relative hypovolemia causes a reduction in preload, which leads to a decreased SV leading to reduced Q and a decrease in DO2. The expansion of the intravascular space is most often due to widespread vasodilation associated with the release of cytokines and inflammatory mediators from damaged or inflamed tissue. In horses, infectious and non-infectious triggers can result in a systemic inflammatory response syndrome (SIRS) and severe vasodilation. One trigger of SIRS is endotoxin. Endotoxin, also known as lipopolysaccharide (LPS), is a component of the cell wall of gram-negative bacteria that is released during times of rapid bacterial death or reproduction. These endotoxin molecules gain access to the systemic circulation, where they initiate a strong inflammatory response. Endotoxemia is common in diseases such as colitis and retained placenta due to increased availability of endotoxins combined with compromise in the integrity of the normal mucosal barrier to their absorption. Other causes of SIRS in horses are bacteremia secondary to failure of passive transfer in neonates and ischemic bowel associated with strangulating obstructions.

Diseases that cause physical obstructions of blood flow to or from the heart are associated with obstructive shock. When blood flow returning to the heart is obstructed, preload decreases, whereas obstruction of blood flow from the heart results in an increase in afterload. In either case, this leads to a decrease in SV leading to a decreased Q and decreased DO2. In horses, diseases that obstruct blood flow returning to the heart are more common than those obstructing blood flow from the heart. Severe gastrointestinal tract distention can increase intra-abdominal pressure and compress the caudal vena cava, limiting venous return to the heart and reducing preload. Pericardial limits the heart’s ability to expand, effectively decreasing preload.

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Diseases that affect the heart primarily are associated with cardiogenic shock. Cardiogenic shock can be due to a decrease in HR or SV (due to a reduction in preload, increase in afterload, or decrease in contractility) leading to decreases in Q and DO₂. In horses, reductions in heart rate (i.e., bradyarrhythmias) can be associated with 3rd degree AV block or hyperkalemia secondary to ruptured bladder. Cardiac diseases that can be associated with decreased preload include AV valve regurgitation with or without cordae tendonae rupture, and tachyarrhythmias such as ventricular tachycardia. Cardiac diseases that can be associated with decreased contractility include influenza and *Strep. equi*-related cardiomyopathy and monensin toxicity.

Most diseases in this category of shock reduce [Hb], SₐO₂, or PₐO₂ leading to decreased CₐO₂ leading to decreased DO₂. Less commonly, diseases can cause decreased nutrient availability to the cell, impair utilization of oxygen by the cell, or increase energy requirements of the cell. In horses, diseases that cause decreased [Hb] included red maple leaf toxicosis and immune mediated anemia. Diseases that decrease SₐO₂ and PₐO₂ are related to impaired oxygen uptake due to severe respiratory disease such as pneumonia. Other causes for metabolic shock include insulin resistance, which decreases the nutrients available to the cell, cyanide poisoning, which impairs the ability of the cell to utilize oxygen, and sepsis, which increases the energy requirements of the cells.

There are three stages of shock determined by how well the body is compensating in order to meet the energy demands of the cells. During shock, the body focuses on meeting the needs of the most “vital” structures, such as the heart and the brain, potentially to the detriment of less “vital” structures such as the gastrointestinal tract.

The body’s response to shock is triggered by detection of a variety of abnormalities associated with decreased DO₂ (acidosis, hypercapnea, and hypoxemia), decreased intravascular water (detected by osmoreceptors in the hypothalamus), and decreased arterial blood pressure (detected by baroreceptors in the aortic body and carotid sinus). Responses mediated through the sympathetic nervous system and renin-angiotensin pathway are key to the body’s ability to compensate for these abnormalities. Through a relatively complex series of events, the body is able to improve DO₂ and nutrient delivery and also limit energy requirements by minimizing non-essential functions. Improvement in Q is through increasing preload (shifting interstitial fluid to intravascular space, retaining fluid at the level of the kidney, and vasoconstricting to maintain blood pressure), increasing contractility, and increasing heart rate. Improvement in CₐO₂ occurs by increasing the respiratory rate (thereby maximizing PₐO₂ and SₐO₂) and splenic contraction to increase [Hb]. Nutrient delivery is increased with stimulation of gluconeogenesis and protein synthesis.

The clinical signs of each stage of shock are associated with the compensatory mechanisms and how well they maintain Q and organ function. Stage I is compensated (a.k.a. hyperdynamic) shock. During stage I, blood flow to the heart and brain is maintained through compensatory mechanisms. Due to these mechanisms, clinical signs include tachycardia, tachypnea, hyperemic mucous membranes, decreased capillary refill time, and bounding pulses. During this phase, mentation should remain fairly normal because the brain is receiving enough energy. Stage II is early decompensated shock. During stage II, energy demands are not being met, resulting in increased anaerobic metabolism and lactate production as well as organ dysfunction. The clinical signs associated with the compensatory mechanisms, including tachycardia and tachypnea, are still present. However, during stage II, signs of inadequate Q (increased CRT, cold appendages, decreased blood pressure) and organ dysfunction (decreased urine production and abnormal mentation) become apparent. Stage III is late decompensated (a.k.a. Irreversible) shock. During stage III, anaerobic metabolism continues and sympathetic compensation with vasoconstriction is overwhelmed, resulting in blood pooling in venules, fluid leaking into the interstitium, and activation of inflammation, as evidenced by SIRS. With inflammation, coagulation is also activated throughout the body resulting in disseminated intravascular coagulation (DIC). Microvascular thrombosis associated with DIC combined with dwindling energy delivery leads to organ failure and death. Because compensatory mechanisms have been overwhelmed, clinical signs of compensation are no longer present. Instead, clinical signs are related to cardiovascular collapse (marked hypotension, bradycardia, pale/gray mucous membranes) and organ failure. Once stage III is reached, recovery is rare even with aggressive treatment.

These proceedings are an adaptation of an iBook created for teaching purposes at the University of Georgia.
Accurate and thorough wound evaluation is vital to being able to provide the owner with information about treatment options, possible complications, and prognosis. The single most valuable tool for the veterinarian when evaluating wounds is a solid knowledge of anatomy. Although the appearance, configuration and location of the skin wound are obviously an important component, involvement of underlying structures often is the limiting factor in prognosis. As with all emergencies, the first step will be to evaluate the horse’s systemic status. Although relatively rare, any systemic compromise should be addressed before proceeding. In order to evaluate a wound fully, the horse must be appropriately restrained and the wound must be properly prepared. After these steps are taken, based on anatomic location evaluation of the appropriate underlying soft tissues (vessels, nerves, muscles, tendons, and ligaments), bone, and synovial structures can be performed.

In this presentation, interactive case discussions will be used to illustrate the techniques required to fully evaluate wounds. The details of the cases are not provided to stimulate more discussion.
Wounds:
Emergency Treatment
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Emergency treatment of horses with limb wounds varies dramatically in complexity depending on location and involvement of underlying structures. The single most valuable tool for the veterinarian when treating wounds is a solid knowledge of anatomy. Treatment of the wound can begin only after the horse’s systemic status has been assessed, the horse has been stabilized if necessary (rare), and the extent of the wound has been determined. Considerations for emergency wound treatment will include addressing limb instability, inflammation, contamination or infection, wound repair, dead space management, and bandaging/coaptation.

In this presentation, interactive case discussions will be used to illustrate the techniques required during emergency wound treatment in several locations. The details of the cases are not provided to stimulate more discussion.
The variety of acute wounds presenting to the equine practitioner offers a challenge to select the most appropriate management to facilitate healing. A complete wound history along with anatomic and specific medical considerations for each patient provides the basis of decision making for wound management. It is essential to evaluate the entire animal and apply an evidence-based approach. The practitioner should consider each wound individually in order to create the optimal conditions for wound healing.

Patient assessment
Assessment of any patient with suspected acute traumatic injury should be systematic and thorough. The size of the wound does not necessarily dictate severity and very small wounds are often overlooked when a large traumatic wound is present. Particularly in horses, the small wound can be the real danger and significant often fatal consequences result.

A useful tool for patient assessment following trauma follows the acronym “A CRASH PLAN” and will be used for this series of case evaluations.

- **Airway**
  - Airway and Circulation: Primary Survey
- **Circulation/Cardiovascular**
  - *Respiratory: Secondary Survey
- **Respiratory**
  - *Abdomen-Nerves: Tertiary Survey
- **Abdomen**
  - *Spine
- **Spine**
  - *Pelvis
- **Pelvis**
  - *Limbs
- **Limbs**
  - *Arteries
- **Arteries**
  - *Nerves
- **Nerves**

Trauma types
- Blount trauma: can be very high energy; falls over a jump, vehicular trauma, etc.
- Penetrating wounds: most common; lacerations, puncture wounds, impalements, foreign body
- Combination of Blount and Penetrating: often times deceiving in extent of injury

Assessment of specific wounds
- Mechanism of Injury
- Depth/Size
- Contamination
- Age of wound
- Structures involved (superficial and underlying)
- Tissue vitality
- Tension
- Temperament of patient
- Environment
- Follow-up care

Use all available information to determine the most appropriate treatment plan. Consider referral if hospital conditions are more appropriate for management or advanced diagnostics are needed.
Puncture wounds and lacerations are a common occurrence in equine practice. No matter how pristine a pasture is, horses seem to find some way to injure themselves. This session will discuss particular wound locations that should warrant greater concern.

**Wounds affecting synovial structures**

Wounds of the limb are at risk of entering synovial structures (joints, tendon sheaths, and bursae). If left untreated, these wounds can result in infection within the synovial cavity leading to permanent disability of the horse. Knowledge of the specific anatomy of the limbs is essential to identifying potential synovial involvement early (Figure 1). Synovial structures that are most often penetrated include: digital tendon sheath, metacarpo/metatarsophalangeal joint, and tarsal joints.[1-3]

Lacerations or puncture wounds into synovial structures often directly introduce bacteria and contaminants (e.g., hair, dander, dirt, etc.) into the synovial space. Once bacteria overwhelm the natural defenses of the synovial structure, detrimental effects on cartilage, synovium and other associated synovial structures happen very quickly. The longer the duration of the infection, the greater the likelihood for permanent damage to the synovial structure. Multiple retrospective studies have provided mixed reports regarding the effect of time until treatment on the outcome of horses with infected synovial structures due to wounds.[4-9] Though early treatment is best; cases that have had synovial sepsis longer than 24 hours can still carry a good prognosis with appropriate treatment.

All wound near synovial structures should be cleaned thoroughly, and the hair from the wound edges should be clipped. Once the wound is adequately cleaned the wound should be explored with sterile gloves. Synoviocentesis should be performed for all neighboring synovial structures. The sample should be submitted for synovial analysis in an ethylenediamine tetraacetic acid (EDTA) blood tube. Normal synovial fluid should have nucleated cell counts less than 1000 cells/ L, of which most are mononuclear cells (macrophages and lymphocytes).[10] Total protein concentration is approximately 20-25% of plasma protein of the same animal.[11] Generally this should be less than 2.0 g/dL. A differential cell count is also important for a complete assessment. There should be less than 10% neutrophils.[10] A sample should synovial fluid should also be submitted for culture and sensitivity whenever possible.

Confirmation of communication between a wound and synovial structure may not always be straightforward. Synovial fluid analysis is beneficial but those results may not be available immediately. The most common method of confirming communication is to distend the synovial structure and watch for fluid to flow from the wound. Other methods include radiography and ultrasonography.

Management of wounds that involve synovial structures should be targeted at efficient removal of contamination and inflammation. Lavage of the synovial structure is usually necessary for removal of bacteria from the synovial space. This is often combined with regional and/or systemic antimicrobial therapies. Prognosis can be variable. Factors associated with a poor prognosis are increased intrasynovial total protein concentration at the time of admission,[4] positive culture results from synovial fluid,[2] and evidence of osteolysis.[12]

**Wounds affecting bones, tendons, and ligaments**

If a wound is distant to a synovial structure, you may not be able to take a sigh of relief yet. The force by which many of these wounds are afflicted can cause damage to structural components of the musculoskeletal system. Figure 2A depicts a kick wound to the medial antebrachium further examination reveal incomplete fractures of the radius (Figure 2B). Sharp lacerations, even when small, can also cause significant damage to the underlying soft tissue structures. Imaging is an crucial component of many wounds on the limbs of horses, especially if the degree of lameness is worse than what is expected for the size of the wound.

**Hemorrhage**

Hemorrhage in the horse can be classified based on rate (acute or chronic), severity (controlled or uncontrolled), location and cause. With these classifications in mind, treatment is based on clinical assessment, resolution of cause, prevention of ongoing losses and treatment of resulting circulatory perturbations.

**Diagnosis and estimation of blood loss**

Estimation of blood volume loss can be difficult to determine particularly when the hemorrhage is partially internal or external blood loss was not witnessed. Initially, packed cell volume and plasma protein levels may be normal due to blood components lost in equal volumes. Additionally, the erythrocyte storage capacity of the equine spleen and resulting splenic contraction in acute hemorrhage may mask initial changes in the hemogram. Changes in red blood cell numbers and total protein may remain within the normal reference range for up to 24 hours even in cases where a large volume of blood is lost. However, after vascular equilibration does
occurs a decrease in packed cell volume, hemoglobin, and panhypoproteinemia will be apparent. A neutrophilic leukocytosis may also be present by 3 hours after hemorrhage.

Blood loss can be estimated based on clinical signs and other dynamic indices even in the absence of visualized hemorrhage. Hypovolemic shock resulting from hemorrhage is divided into 4 classes based on percentage loss of total blood volume. The clinical signs (sweating, tachycardia, an anxiety) described in these classes are also consistent with pain and it is important to differentiate the underlying cause. Class I involves blood loss of less than 15% blood volume and can be compensated with little to no change in physical parameters or clinical signs other than decreased urine output. Class II (15-30% blood loss) results in hyperdynamic shock and the inability of the body to completely compensate for the volume of blood lost. Clinical signs include tachycardia, tachypnea, and bounding pulses. The patient may be anxious and sweating. Blood pressure may be normalized but a metabolic acidosis will result with increased blood lactate levels. Class III-IV (>30% or continued uncontrolled blood loss results in compensatory shock with marked tachycardia and tachypnea present. Capillary refill time and jugular filling are prolonged, peripheral vasoconstriction causes cold extremities and urine output is diminished. Metabolic acidosis is severe and blood lactate levels may be profoundly increased. Without intervention cellular hypoxia and acidosis results in complete decompensation, failure of cardiac function, circulatory collapse and death.

**Treatment for acute blood loss**

The first steps in treatment of acute hemorrhage are location determination and reduction or cessation of further blood loss. When hemorrhage is external, treatment involves application of direct pressure and/or ligation of lacerated vessels. Internal hemorrhage may be more difficult to diagnose. Epistaxis warrants airway endoscopy to evaluate upper and lower airway sources of hemorrhage including guttural pouches (mycosis or ventral straight muscle rupture) and pulmonary vessel rupture. Hemothorax and hemobdomen can be diagnosed with radiography, ultrasound and thoraco- or abdominocentesis. In cases of abdominal hemorrhage, an exploratory celiotomy should not be performed unless there is confirmation of the source and a high likelihood of successful ligation.

Autotransfusion from body cavities permits the body to reuse blood components and approximately two thirds of the erythrocytes lost into the abdomen or thorax are resorbed within 24 to 72 hours. The remaining one third are lysed or phagocytized and the iron and protein are reused.

Hemorrhage may be reduced by administration of aminocarproic acid, an anti-fibrinolytic lysine derivative, which may enhance clot maintenance possibly through a reduction in partial thromboplastin time (PTT). Due to its short half-life, this medication has been shown to provide adequate therapeutic levels using a constant rate infusion of 3.5 mg/kg/min for 15 minutes, followed by 0.25 mg/kg/min. In the field, a bolus dose of 30 mg/kg may provide some benefit, if a CRI is not possible.

**Conclusion**

Acute wounds in the equine should be evaluated as quickly as possible and all scenarios with regards to involved structures should be assessed. Very small and seemingly insignificant wounds may be overlooked especially when multiple wounds are present. Additionally, in the equine patient the size of the external wound often has very little effect on the ultimate prognosis. The direct puncture wound into the carpus is much more dangerous than a large pectoral laceration and will often get the attention of the owner more quickly. It should also be noted that horses with severe gastrointestinal disease may present with self-induced trauma. If the overall system presentation does not fit with a seemingly simple wound, reevaluate! It is important to educate clients to these facts so that prompt notification of the equine practitioner occurs and appropriate therapy is initiated. All body systems should be evaluated for potential involvement and the overall status of the patient should not be forgotten. Cardiovascular, respiratory and neurologic status can play a major role in treatment plans.

**References**

Chronic, non-healing wounds can be particularly frustrating for the practitioner. It is often difficult to have access to a complete history with respect to the wound. Regardless, the evaluation of chronic wounds should follow the same systematic process used for all wounds. The way in which a wound is managed in the early stages has a dramatic effect on the quality and speed of healing. Delayed appropriate treatment of wounds limits treatment options and will likely affect the prognosis and quality of healing.

**Exuberant granulation tissue**

The development of exuberant granulation tissue (EGT) or “proud flesh” has long been a thorn in the side of the equine practitioner. Despite extensive evaluation of this phenomenon in the equine, there is no universal treatment option and multi-modal treatment is often required for the chronic wound with excessive granulation tissue formation. Fibroplasia, or the formation of granulation tissue, is a required phase of wound healing but exuberant production is difficult to predict. There are a number of factors which are known contributors to the development of proud flesh. Individual inflammatory response, wound location, presence of infection, and initial treatment are all contributing factors.

Treatment options for EGT include both chemical and physical means. Small to moderate amounts of granulation tissue can be managed with the application of topical corticosteroid cream which inhibit inflammation and fibroplasia. The use of corticosteroids is controversial in that they are also known to inhibit angiogenesis, wound contraction and epithelialization. Therefore, the application should be infrequent and limited to the EGT, sparing surrounding tissues. Alternatively, sharp excision is an excellent means of treating EGT. This is particularly helpful in chronic wounds with large proliferative masses of EGT.

Epithelialization cannot extend “uphill” upon the edge of the EGT and becomes stagnant over time. Quite often chronic wounds will appear larger than they actually are due to the mushroom-like proliferation of tissue extending over wound edges. Removing the excessive tissue may reveal a wound of a smaller size than expected. Removal renews the potential for epithelialization along wound edges. The excess granulation tissue should be trimmed flush with the healing epithelial edge. Granulation tissue is aneural so local anesthesia is not required when the excision is limited to the EGT. Additionally, it is highly vascular so a tourniquet may be used to reduce hemorrhage and preserve visualization. Blood loss is unlikely to be of a dangerous volume but it can be quite unsettling for a client observing the procedure. The wound will likely continue to bleed until a padded bandage is placed. Multiple trimmings of the granulation tissue is necessary in many cases. However, if the wound fails to progress toward healing other causes of delayed healing should be investigated.

**Differential diagnoses**

Exuberant granulation tissue is often confused with tumors in the equine, particularly sarcomas. Contributing to this is the reality that an equine wound can transform into a sarcoma or other tumor. The history of a known wound at the site does not preclude the sarcoma diagnosis. This transformation can occur at any wound site but distal limb wound sarcomas are fibroblastic in nature, mimicking the presence of EGT. Horses with sarcomas present at other sites are more prone to sarcoma transformation of wounds. Sarcoma or other neoplasia should be suspected in any chronic wound which is not healing with what is deemed to be appropriate management. In these cases biopsy and histologic evaluation is warranted.

Habronemiasis and the presence of “summer sores” can occur in open wounds and take on the appearance of EGT, sarcomas or squamous cell carcinoma. With this condition the wound may also have calcified granules contained within and be pruritic. As with any non-healing wound a biopsy is appropriate for diagnosis.

**Excessive motion**

Excessive motion can inhibit healing when all other aspects of the wound are appropriate and healing well. Measures to reduce motion include stall confinement, bandaging and splint or cast application. Often these measures result in a dramatic increase in the speed and quality of healing. Owners are often reluctant to stall confine but the treatment time may be so significantly reduced with a short period of bandaging and stall confinement, it should be encouraged when possible.

**Conclusions**

The management of chronic equine wounds in the field can be challenging and require dedication and commitment on the part of practitioner and owner, alike. The importance of diligent wound care and bandaging especially in the lower limb cannot be over emphasized. Many horribly chronic, seemingly hopeless wounds can be successfully managed with the appropriate tools.
Evaluation of colic in foals is particularly challenging for several reasons. Foals differ from adults in presentation of clinical signs, etiologies and in some diagnostic tools available to the practitioner. The essential elements of the colic exam are still present and additional diagnostics unique to the foal may be helpful. The decision for exploratory celiotomy in foals is a particularly difficult decision to make due to the risks involved in surgery at that age.

**Signalment and history**
The signalment and history can be particularly important in differentiating the cause of colic in foals. Age is an important factor pointing toward certain etiologies. For example, meconium impactions are seen most commonly in the newborn while ruptured bladder or congenital anomalies such as atresia coli usually cause clinical signs in 2-5 day old foals. Small intestinal volvulus, intussusceptions and gastric outflow obstructions are more common in older foals while ulcers and enteritis can be found in foals of any age.

Some causes of colic are more common or exclusively found in particular breeds such as lethal white syndrome in overo Paint horses. The gender of the foal may also indicate risk for certain types of colic. For example, colts are more likely to be diagnosed with ruptured bladders.

**Physical examination**
A complete and thorough physical examination is imperative in the evaluation of foals with colic. Underlying issues such as failure of passive transfer/sepsis, neonatal encephalopathy, fractured ribs, musculoskeletal problems and congenital abnormalities can result in colic signs or make diagnosis more difficult. A foal that is unable to stand due to profound weakness, sepsis or musculoskeletal abnormalities will be more difficult to assess. Colic signs may be more subtle if the foal is weak. Physical examination should be directed at the entire foal with evaluation of all physiologic parameters. Evaluation of the foal should include temperature, pulse, respiratory rate, mucous membrane color and capillary refill time. The adequacy of passive transfer and measurement of immunoglobulins should be evaluated and a complete blood count, serum biochemistry and blood culture should be obtained whenever possible. Low serum IgG concentrations will be an important factor in management of the colicky foal and more than one plasma transfusion may be necessary during the course of treatment.

**Gastrointestinal examination: diagnostic techniques**
Abdominal circumference can be serially measured in order to establish a baseline and appreciate increasing or decreasing abdominal distention through the course of treatment. Abdominal distention may be due to peritoneal fluid or gas or fluid accumulation in the gastrointestinal tract. GI tract distention is more evident in the foal than the adult horse and can result from either small intestinal or large colon distention.

Digital rectal examination is an important diagnostic tool particularly when meconium impaction is suspected in the neonate. The impaction can often be palpated in the pelvic canal. Meconium impaction may result in partial or complete obstruction, the latter causing marked abdominal distention on physical examination. Diarrhea in foals may also be preceded by abdominal distention and signs of colic.

Nasogastric intubation should be performed in any colicky foal. It is more difficult to perform than in the adult and it is helpful to measure the distance to the stomach before beginning intubation. A stallion catheter with stylet in place is the appropriate size for gastric intubation in most neonates. As is true in adults showing colic signs lack of nasogastric reflux does not rule out small intestinal disease in the foal.

Abdominal ultrasonography is particularly helpful in evaluation of the colicky foal and has become routine. It can be performed with the foal standing or recumbent. Clipping the hair may be necessary for good probe to skin contact and visualization. Ultrasound is useful for visualization of distended loops of small intestine including enteritis, intussusceptions, and other obstructions. A large portion of the small intestine can be visualized in neonates but it is more obscured in older foals as the large colon develops at around 4 months of age. Enteritis will appear as hypermotile, fluid-filled small intestinal loops. Intussusceptions may appear as a target lesion or multiple concentric rings on a single cross-section of the GI tract. It is also helpful in the diagnosis of uroabdomen and the bladder defect can occasionally be visualized. Ultrasonographic identification of peritoneal fluid increases the safety of abdominocentesis in foals and is used routinely for this purpose. While a 3-5 MHz probe is most useful for abdominal evaluation, any commonly used probe has the ability to penetrate the abdomen of the foal and can be useful.
Abdominal radiography is much more commonly utilized in the foal than it is in the adult colic but use has declined with the availability of quality ultrasound imaging. Abdominal radiography remains particularly helpful in evaluation of large bowel obstructions such as sand or meconium impaction as well as in the presence of peritoneal effusion.

Abdominocentesis is a useful diagnostic in the foal but should be performed with caution due to the increased risk of bowel laceration and peritoneal contamination in the foal when compared to the adult. Abdominal ultrasonography is useful to determine the presence of peritoneal fluid and aid in sampling success. Abdominocentesis is not always necessary to decide that surgery is warranted especially if diagnostic imaging is available to the practitioner.

**Common causes of colic in the foal**

**Congenital anomalies**

Congenital anomalies must always be considered in the neonate. Atresia coli, atresia recti, and atresia ani have each been described in the foal. Most foals are born asymptomatic and begin to show signs of colic and abdominal distention at 24-48 hours with a history of no fecal production. In some cases abdominal radiography may reveal a blind stump of colon filled with gas. Many are diagnosed at exploratory celiotomy and successful anastomosis of bowel segments is rare.

Overo Lethal White Syndrome or uleocecolic agangliosis is a genetic defect recognized in homozygous, overo, paint foals. Usually these foals are completely white but may also have a few small spots of color.

In this genetic disease an endothelin receptor B mutation results in incomplete agangliosis of the myenteric and submucosal ganglia of the intestinal tract. Affected foals appear normal at birth but rapidly develop signs of colic, profound ileus, and intestinal distention. A diagnosis of lethal white syndrome is based on clinical signs coupled with ultrasonographic evidence of profound ileus. Unfortunately, there is no treatment and euthanasia is strongly recommended. A genetic test is available to identify heterozygous individuals and should be recommended in future breedings.

**Meconium impaction**

Meconium impaction is the most common cause of colic in the neonate. Clinical signs of meconium impaction include decreased nursing, straining to defecate with an arched back, flagging of tail, and lack of meconium production. Particularly early on or with incomplete obstruction foals may show intermittent colic signs that responds well to analgesia. A digital rectal examination may reveal hard fecal material in the rectum. Complete obstruction may cause significant and grossly evident abdominal distension to develop. In severe cases profound abdominal distension can result in respiratory compromise. Radiographs or ultrasound may reveal fecal material in the distal colon or rectum. A barium enema can be performed for further evaluation and to attempt to rule out focal atresia of the intestinal tract. Treatment of meconium impactions includes enemas, analgesics, oral laxatives, IV fluids and supportive care. An enema consisting of 200-500 ml of warm soapy (Ivory or other gentle soap) water is the first line of treatment. This should be performed using a small, well-lubricated catheter and gravity flow. Multiple enemas with a detergent such as dioctyl sodium succinate (DSS) are discouraged due rectal and colonic irritation and resulting edema as well as DSS toxicity. Retention enemas using acetylcysteine can be performed with caution if soapy water enemas prove unsuccessful in reducing the impaction. If the contents becomes strangulated it will be firm on palpation and not reducible. Most of these occur as direct hernias when the vaginal tunic has ruptured and bowel passes through to subcutaneous tissues. The foal should be sedated for this procedure as distention of the rectum may cause discomfort. A Foley catheter is inserted 2-4 inches into the rectum and the balloon is distended, using caution not to overinflate. Infuse 100-200 ml of 4% acetylcysteine solution into the rectum and clamp the Foley catheter. The fluid should remain in place for 15 minutes before deflating and removing the catheter. Manual evacuation of meconium using forceps should never be performed due to the risk of iatrogenic tissue damage. Occasionally, surgical reduction via celiotomy is required.

**Inguinal hernias**

Foals may be born with inguinal hernias or they may develop in the first 2-3 days of life. Foals born with congenital inguinal hernias are often not colicky particularly if the content of the hernia is easily reducible. Many can be successfully treated by repeated manual reduction or application of a truss bandage. If the contents becomes strangulated it will be firm on palpation and not reducible. Most of these occur as direct hernias when the vaginal tunic has ruptured and bowel passes through to subcutaneous tissues. The foal will quickly show signs of discomfort and emergency celiotomy is the required treatment.

**Uroperitoneum**

Uroperitoneum is most commonly diagnosed in foals at 2-3 days of age although the rupture may have occurred at the time of parturition. Uroperitoneum can occur from rupture of the urachus, ureter or bladder with the same resulting clinical presentation. Foals most often present with abdominal distention, low-grade pain and frequently posture to urinate. Symptoms can be similar to those exhibited by foals with meconium impaction and further diagnostics are often required for differentiation between the two. Subcutaneous rupture at the urachus may cause yellowish skin discoloration and subcutaneous swelling. The serum chemistry abnormalities of foals with uroperitoneum classically include hyponatremia, hypochloremia and hyperkalemia. Foals receiving intravenous fluids and supportive care for other abnormalities may not exhibit these electrolyte derangements and diagnosis may be more challenging. A peritoneal fluid/serum creatinine ration of >2:1 is diagnostic. As stated earlier, ultrasound is helpful in obtaining peritoneal fluid and a tear in the bladder can sometimes be visualized. Medical treatment involves stabilization and correction of electrolyte abnormalities. Intravenous fluids should not contain potassium as marked hyperkalemia is common. While uroperitoneum
is an emergency which ultimately necessitates abdominal surgery for correction, surgery should be delayed until the patient is stable, serum potassium levels are below 5.5 mEq/L and thus a better anesthetic candidate.

**Diaphragmatic hernias**

Neonates may present with diaphragmatic hernias and are often associated with birthing trauma. The integrity of the ribs should be evaluated and any foal in with rib fractures could have a resulting diaphragmatic hernia. Congenital diaphragmatic hernias are also possible and the presenting foal may be up to several weeks old. Diagnosis is usually suspected with plain radiographs and thoracic ultrasound and confirmed on exploratory celiotomy.

**Indications for surgical treatment**

The indications for surgical exploration of foals are similar to those in adults. Persistence of pain despite appropriate analgesia, progressive abdominal distention or surgical diagnosis on ultrasound or radiography are all common indications for surgical management.
Dealing with Thoracic and Abdominal Trauma
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Trauma or lacerations of the abdomen and thorax have the capacity to heal remarkably well provided deeper structures are not involved. However, wounds involving the thoracic and abdominal cavity are particularly worrisome due to the potential for significant internal damage with minimal external trauma. Definitive investigation of the depth of these wounds is essential for appropriate management.

Aseptic technique is imperative for evaluation in these areas if thoracic or abdominal penetration is suspected.

Thoracic wounds
Thoracic trauma may involve rib fractures or penetration of the pleural space with pneumothorax or hemothorax. It may be possible to hear a “sucking” sound if the environment is quiet indicating pleural space involvement. Thoracic auscultation may also be helpful but radiography or ultrasonography may be necessary for definitive diagnosis. If the pleural space is involved pneumothorax may cause respiratory distress and removal or air by thoracocentesis may become necessary. These wounds should be explored for foreign bodies and only closed primarily if the wound is deemed clean of contamination and there is adequate tissue to do so. Second intention healing is a viable option in most thoracic wounds not involving deeper structures and allows for an adequate cosmetic outcome.

Abdominal wounds
Abdominal wounds may be challenging to diagnose with regards to depth and thus peritoneal cavity involvement. Tissue planes may shift after wounding and depth may not be immediately apparent. An abdominocentesis may be of benefit in the diagnosis particularly if hemoabdomen is suspected but normal parameters do not rule out peritoneal penetration. Additionally, large wounds with extensive tissue loss are at risk of herniation even if the wound does not penetrate the abdomen. Initial management should involve cleaning the wound aseptically and evaluating for foreign materials and depth. Like thoracic wounds, many non-penetrating abdominal wounds heal by second intention with acceptable cosmesis.

Inguinal and axillary wounds
Axillary wounds in horses have the potential to induced widespread subcutaneous emphysema, due to entrapment of air in the wound. As the horse advances the involved forelimb the wound gaps open filling with air. This air becomes trapped and is then “pumped” into the SQ by further motion of the forelimbs. Depending on the configuration of the wound SQ emphysema can lead to pneumomediastinum which can rarely advance to become a pneumothorax. If pneumomediastinum or pneumothorax develop they must be treated appropriately (see above). The SQ emphysema is usually limited and resolves once the wound is sealed. Packing the wound may decrease the severity of emphysema but the best way to decrease severity is to limit the movement of the horse to only what is absolutely necessary. Once it forms it cannot be removed and must be resorbed over time. Primary closure of these wounds is often difficult due to loss of tissue and may not be sufficient to prevent subcutaneous emphysema form forming. Wound dressing which promoting granulation tissue development should be used.

Inguinal wounds commonly involve fence post or tree branches and should be evaluated extensively for depth of penetration and foreign body material. Due to location it may be necessary to explore these wounds under general anesthesia in dorsal recumbency. The extent of the wound may not be evident with the horse in a standing position.

Head and neck wounds
Wounds involving the head are most frequently the result of horses running into a stationary object, tossing the head into overhanging objects or being kicked. The list of structures that can be involved in head trauma is extensive and involves many body systems. Many of these can be extremely cosmetically or athletically detrimental or even fatal. It is important to inspect for possible fractures and sinus involvement. Additionally, injuries to the head may result in neurologic signs or respiratory distress due to swelling or impaired function. It is important perform a thorough evaluation for neurologic signs as this may be the most significant indicator of prognosis. Respiratory distress may occur at some time after the initial injury due to inflammation and swelling occluding the upper airway. A temporary tracheotomy may be necessary in these cases.

Lacerations of the head are common and are usually presented with less contamination than other areas of the body. The head has an excellent blood supply and many lacerations will heal well given tissue loss is not too extensive. Lacerations of the eyelids, nostrils, lips, tongue and ears present more of a challenge because preservation of function is of paramount importance. This relies heavily on reconstruction and primary or delayed primary closure should be attempted whenever possible.
Wounds involving the facial bones, sinuses and orbit may be more extensive than initial examination indicates particularly when the skin is minimally affected. Fractures may be present even when the skin isn’t open. Radiography is warranted to evaluate the extent of these fractures and determine involvement of sinuses or orbital rim. Overlying soft tissues should be preserved whenever possible to facilitate closure and protection of underlying osseous structures. It is important to remember the anatomically position of sinuses, nasolacrimal duct and facial nerves when evaluating and repairing wounds of the head.

Neck wounds are most commonly caused by barbed or high-tensile wire fencing. They are usually presented in a horizontal orientation and often occur at the base of the neck. Usually only the skin, subcutaneous tissues and underlying muscles are involved. Involvement of the trachea, esophagus or underlying neurovascular structures is possible and may change the prognosis associated with these wounds considerably. Like head trauma, a tracheotomy may need to be performed if there are signs of respiratory distress. If the neck wound involves only skin, subcutaneous tissues and some underlying muscle the prognosis is favorable. Primary closure should be performed wherever possible. A neck cradle may be necessary to reduce tension on the sutures and prevent dehiscence.
The equine upper airway is a high, resistance, low-capacity path between the nares and lungs. Proper function of the equine upper airway requires the coordinated effort of a large number of anatomical structures. Very small changes in diameter of the upper airway are magnified in their effect on the ability of the horse to move air into the lower respiratory system and oxygenate tissue effectively. For example, a 20% decrease in airway radius doubles airway resistance. Disease diagnosis of the equine upper airway can be a challenge due to the dynamic nature of many abnormalities. The airway may appear mildly affected or even completely normal on resting endoscopy and have severe functional abnormalities at times of increased respiratory effort. Additionally, some abnormalities are intermittent in their appearance and only manifested under certain situations such as increased head and neck flexion. (McGorum 2007)

The use of radiography and resting endoscopy has been the mainstay of upper airway diagnosis for several years. Many abnormalities affecting the upper airway can be diagnosed using these modalities. More recently the use of such modalities as dynamic endoscopy, either on a treadmill or over-ground, and ultrasonography has increased the tools in the diagnostician’s arsenal. Resting endoscopy permits the diagnosis of more advanced grades of laryngeal hemiplegia, arytenoid chondritis, persistent dorsal displacement of the soft palate (DDSP), persistent epiglottic entrapment, subepiglottic cysts and fourth branchial arch defects. Radiography is a valuable secondary tool to evaluate epiglottic and palatopharyngeal arch position as well as subepiglottic cysts and laryngeal cartilage mineralization.

Dynamic over-ground and treadmill endoscopy
Dynamic endoscopy has several advantages for diagnosis of upper airway abnormalities. Some abnormalities only occur during exercise particularly at maximum-exertion when negative pressure is highest. Also, the actual effect of pathology on athletic performance can be observed and response to treatment documented. Often multiple, related abnormalities are causing poor performance or noise while only one may be diagnosed on resting endoscopy. Upper airway abnormalities detectable only under dynamic, exercising conditions include some grades of laryngeal hemiplegia, axial deviation of the aryepiglottic folds, intermittent DDSP, intermittent epiglottic entrapment, pharyngeal collapse and epiglottic retroversion. In approximately 30% of horses with upper airway abnormalities, more than one abnormality is actually present. (Lane, Bladon et al. 2006)

Dynamic upper airway endoscopy can be performed either on a treadmill or over-ground using an endoscope fitted to the patient. Both modalities allow for the detection of dynamic abnormalities but there are differences between them. Treadmill upper airway endoscopy allows direct manipulation of the patient’s exercising speed and incline but it is much less flexible in other important variables which can be introduced to the examination. Additionally, a period of training is required for the patient to decrease the risk of injury during examination. Advantages of over-ground dynamic upper airway examination include the ability to exercise the horse in the normal environment where the abnormality occurs. It is a useful tool for both sport horses and race horses. Variables that can be introduced include exercise under saddle, exercise with significant head-neck flexion, or training on the track in the company of other horses. Both modalities represent significant advances in the diagnosis of equine upper airway disorders.

Laryngeal ultrasound
The use of laryngeal ultrasound for the diagnosis and management of upper airway disorders in the equine has become a valuable tool in recent years. It is most useful in evaluation of the laryngeal cartilages and intrinsic musculature. Advantages of this diagnostic modality are expense and non-invasive nature. Unlike MRI or CT the technique does not require general anesthesia but rather mild sedation. In combination with resting upper airway endoscopy, laryngeal ultrasound is useful for diagnosis and follow-up of arytenoid chondritis and diagnosis of left laryngeal hemiplegia. (Garrett, Woodie et al. 2011)

The technique is performed with a 7-10 megahertz microconvex or linear transducer. Both lateral and ventral image windows can be obtained. The lateral image window orienting the probe in a longitudinal and transverse plane is used to assess the echogenicity of the cricoarytenoideus lateralis and cricoarytenoideus dorsalis muscles for identification of laryngeal hemiplegia. Neuromuscular dysfunction results in hyperechogenicity of these muscles evident on ultrasound.

Computed tomography
Computed Tomography (CT) is particularly useful for the evaluation of diseases within the paranasal sinuses particularly masses, tooth root abscesses, fractures and sinusitis of other etiologies. The detail of osseous structures obtained with CT is greater than that for magnetic resonance imaging (MRI) and CT has become a valuable tool for planning surgical intervention in these cases. The acquisition time is short and three dimensional reconstructions can be quickly created.
References
The ability to adequately sedate and offer pain management is an essential part of veterinary care of the equine patient. Unfortunately, these areas do not appear to have advanced as rapidly in equine medicine as they have in the treatment of small animals. Several limitations exist which may explain this dichotomy. High expectations exist for sedation and analgesia in the equine patient. Sedation should keep the horse still with all four feet on the ground but not induce recumbency. There should be no or little effect on motility of the gastrointestinal tract. Likewise, analgesia should not inhibit normal gastrointestinal function. In the case of orthopedic disease, pain relief should be sufficient enough to allow full and even weight distribution, less support-limb laminitis develop. Dosage and route should be practical for the field situation. Finally, these compounds need to be economical for the average size horse. Despite these requirements, certain areas of sedation and analgesia have made recent advances.

Many surgical procedures can be performed in the standing horse in either a field or hospital situation. Regardless of the procedure, meticulous planning and adequate restraint with appropriate analgesia are essential for a successful outcome. Additionally, thorough knowledge of what complications can occur and how to deal with them are just as important as technical skills when any surgery is performed. With these tools in hand, standing surgery in a field situation can be rewarding for the equine practitioner.

Sedation and chemical restraint

Phenothiazine tranquilizers

Phenothiazines are primarily used for the calming effect and are often combined with other drugs. They are not thought to have analgesic properties themselves and are thus quite useful for calming the fractious horse during a lameness exam, training or traveling where analgesia (and ataxia) are not desired. Phenothiazines act by blocking the action of dopamine both centrally and peripherally. This action can lead to arterial hypotension and they should not be used in horses with cardiovascular compromise from hemorrhage or dehydration. There is concern for use with stallions as persistent penile paralysis has been rarely reported. Acepromazine is the most commonly used phenothiazine and is available as a 1% solution (10 mg/ml) which can be delivered by IM, IV or oral route. The onset of action is around 30 minutes and lasts for 4-6 hours.

Alpha-2 agonists

Alpha-2 agonists produce a variety of effects including sedation, muscle relaxation and analgesia by binding to and stimulating alpha-2 adrenergic receptors both peripherally and in the central nervous system. Xylazine, detomidine and romifidine are approved for use in the United States. The level of sedation is pronounced and they are each useful for performing standing procedures although they vary in the length of sedation and degree of ataxia produced. Additionally, the horse will take on a saw-horse stance but still may still become rapidly aroused and kick. For this reason, alpha-2 agonists are often augmented with opioids, particularly butorphanol for more balanced sedation. Romifidine is the most recent alpha-2 agonist to be approved for use in horses and is reported to cause less severe ataxia while maintaining adequate sedation.

Opioids

Opioids are often combined with alpha-2 agonists for a more profound and longer lasting sedation. The use of opioids alone can cause excitement and nervousness and are thus not used for sedation in this manner. The most commonly used opioid is butorphanol, a synthetic agonist/antagonist, which can be combined with detomidine for a variety of standing procedures. Opioids provide significant analgesia but still need to be combined with local anesthesia for standing surgical procedures.

Butorphanol administered at a dose of 0.01-0.1 mg/kg IV or IM is the most commonly used opioid for the equine patient. There is considerable variability in the amount of analgesia produced with this drug but under the best circumstances analgesia can be excellent and last up to 4 hours.

Recently advances have been made in the delivery of opioid analgesics. The use of transdermal fentanyl has been reported in horses although the drug is not approved for use in this species. The most common dose is two 100-microgram/g patches per 450-kg horse which can provide analgesia within 4 hours of application. The patches are applied to a clipped area of the skin and secured with a bandage for good skin contact. Reapplication should occur every 48 hours.

Epidural use of morphine is a reliable, inexpensive route for analgesia to the hindlimbs. Any medication introduced into the epidural space must be done with strict aseptic technique but this is worthwhile when a balanced and significant analgesic plan is required. The recommended dose is 0.1 to 0.2 mg/kg diluted in a volume of 20ml with 0.9% saline. The onset of action is 20-30 minutes and analgesia can last up to 16 hours. This route of administration offers the benefit of morphine analgesia without the side effects of CNS excitement which can occur with systemic administration. Epidural analgesia is particularly helpful in cases where treatment for hindlimb injury is underway but risk of support-limb laminitis is apparent.
Gabapentin

The newest area of interest for analgesia in the equine patient is that of neuropathic pain. Neuropathic pain is pain which is initiated or caused by a primary lesion or dysfunction in the nervous system. It is associated with chronic disease and cannot usually be alleviated with traditional analgesics such as opioids. This area is of particular interest in treating diseases such as lamination where pain appears to be upregulated and is unfortunately difficult to manage with more traditional medication. Gabapentin, a gamma-aminobutyric acid (GABA) analogue, originally developed to treat epilepsy in humans has shown promise as an adjunct therapy in refractory, chronic pain in the equine patient.

Sedation protocols for standing procedures

Alpha-2 agonists are likely an important part of any standing surgical procedure. Xylazine sedation has a duration of 20-30 minutes and, while useful for brief sedation, does not last long enough for most surgical procedures. Detomidine is approximately 100 times as potent and lasts twice as long as xylazine. Detomidine can be used alone or in combination with an opioid such as butorphanol for standing surgery. It can be administered as a single or “as needed” dose of 0.01-0.02 mg/kg IV and provides reliable sedation for approximately 60 minutes. Additionally, it can be administered as an IV variable rate infusion which offers prolonged sedation while decreasing the total dose infused. For IV infusion, an IV catheter is placed and 6 micrograms/kg is administered as a bolus. Butorphanol can be used with this initial bolus at a dose of 0.01-0.03 mg.kg IV. The infusion is prepared by adding 24 drops/sec and serially half the rate every 15 minutes. The rate can be adjusted depending on the sedation level of the individual patient and butorphanol can be administered as needed.

This will provide adequate sedation and analgesia for restraint but local anesthetic will likely be required depending on the procedure to be performed. Local anesthetic plans can include perineural infusion, epidural and regional infusion and will depend on the location and specifics of the surgery. Both lidocaine and mepivacaine are routinely used for perineural and local infusion. Mepivacaine is probably the most widely used of the local anesthetics and has less vasodilatory activity and causes less local edema than lidocaine. Anesthesia can last for 1-2 hours with lidocaine and 2-4 hours with mepivacaine.

Surgeries of the eyelid and ear

Eyelid

The frontal nerve, a branch of the supraorbital nerve, provides sensory innervation to the medial and middle two thirds of the upper lid. This nerve is anesthetized as it emerges from the supraorbital foramen, palpated as a depression 2.5 cm above the supraorbital process, by inserting a 25 gauge needle into the foramen and depositing 1-2 ml of local anesthetic.

Other nerves providing sensory innervations to the skin around the eye include the infraorbital, lacrimal and zygomatic nerves which can be anesthetized as they cross the rim of the orbit or by local infiltration in the skin at the surgical site.

Motor innervation to the periocular cutaneous muscles which are responsible for closure of the lids is provided by the palpebral nerve, a branch of the auriculopalpebral nerve. This nerve can be blocked where it crosses the zygomatic arch dorsolaterally by infusion of 3-6 ml of local anesthetic with a 25 gauge needle.

Ear

Local anesthesia of the ear can be performed by blocking the internal auricular nerve and great auricular nerve. The location of the internal auricular nerve is palpable as a notch or depression on the lateral aspect at the base of the auricular cartilage. At this location, anesthetic is deposited by directing a 25 gauge needle into the notch and depositing 2 ml of local anesthetic. The great auricular nerve is palpable at the caudal aspect of the base of the pinna. Again 2-3 ml can be infused over the nerve using a 25 gauge needle.

Surgeries of the paranasal sinuses

Trephination and lavage of both the frontal and maxillary sinuses can readily be performed in the standing patient with adequate sedation and local anesthetic. After infusion of local anesthetic both in the subcutaneous tissue and periosteum at the location of trephination, a stab incision is made at the location of trephination. A Steinmann pin and hand-held chuck or Michelle trephine are used to create a hole in the sinus adequate in size for the insertion of the infusion port of a standard drip-set. The location of trephination sites are as follows: for the frontal sinus, 60% of the distance in a lateral direction from midline to the medial canthus and 0.5 cm caudal to the medial canthus; for the caudal maxillary sinus, 2 cm rostral and 2 cm ventral to the medial canthus; and for the rostral maxillary sinus, 50% of the distance from the rostral end of the facial crest to the level of the medial canthus and 1 cm ventral to a line joining the infraorbital foramen and the medial canthus. The surgical site can be left open and bandaged for repeat lavage.

Surgeries of the perianal area and vulva

Local infusion of anesthetic or epidural anesthetic will be necessary for any surgery involving the tail, perianal area or vulva. Surgeries such as a Caslick’s procedure or small mass removal can be easily performed with local anesthetic infusion however, the time spent to perform epidural for more invasive or prolonged procedures is time well spent. Injection is performed in the space between the first and second coccygeal vertebrae. The site is located cranial to the tail hairs and is determined by moving the tail up and down. After aseptic preparation of the site, bleb of local anesthetic is placed to facilitate the procedure. The epidural is performed with an 18 gauge spinal needle with stylet. The needle is introduced on midline with the bevel in a cranial direction, perpendicular to...
the skin. A “popping” sensation may be felt when the ligamentum flavum is penetrated and an audible “hiss” may be apparent when the epidural space is entered. A drop of sterile saline placed in the hub of the needle will be aspirated into the space when encountered. A maximum of 10 mls is usually used to prevent paralysis of the lumbosacral nerves. A combination of lidocaine (0.22mg/kg) and xylazine (0.17 mg/kg) expanded to a total volume of 8 mls with sterile saline offers reliable analgesia. Sedation and ataxia may occur with epidural administration. A tail-tie is usually enough to provide support in the face of mild to moderate ataxia. Recumbency is a rare but possible result of epidural anesthesia.

**Surgeries of the distal limb**

Surgery of the distal limbs including wound evaluation, laceration repair, or coffin bone debridement can be performed with use of routine perineural nerve blocks and adequate sedation.

**Conclusion**

A combination of sedation and local anesthetic will allow the performance of many surgical procedures in the standing horse. The individual animal temperament and environmental conditions as well as safety of the patient, veterinarian and assistants must be evaluated when deciding if standing surgery is appropriate. Regardless of the surgical procedure, appropriate antimicrobial and anti-inflammatory medications should be used and tetanus toxoid should always be current. Most importantly, aseptic technique is as important in the standing patient as it is under general anesthesia. Keeping those facts in mind, many procedures can be successfully performed standing negating the risks and expense of general anesthesia.
Many surgical procedures can be performed in the standing horse in either a field or hospital situation. Regardless of the procedure, meticulous planning, appropriate patient selection and adequate restraint with appropriate analgesia are essential for a successful outcome. Additionally, thorough knowledge of what complications can occur and how to deal with them are just as important as technical skills when any surgery is performed. Many surgeries are simple as long as nothing goes wrong. With these tools in hand, standing surgery can be rewarding for the equine practitioner.

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This will provide adequate sedation and analgesia for restraint but local anesthetic will likely be required depending on the procedure to be performed. Local anesthetic plans can include perineural infusion, epidural and regional infusion and will depend on the location and specifics of the surgery. Both lidocaine and mepivacaine are routinely used for perineural and local infusion. Mepivacaine is probably the most widely used of the local anesthetics and has less vasodilatory activity and causes less local edema than lidocaine. Anesthesia can last for 1-2 hours with lidocaine and 2-4 hours with mepivacaine.

Surgery of the eyelid and ear
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Paranasal sinuses
Standing surgery of the paranasal sinuses offers several advantages over performance under general anesthesia. Complications and cost associated with general anesthesia is avoided. Less hemorrhage occurs and there is better visualization of sinus anatomy. Successful standing surgery requires adequate sedation and restraint. The use of stocks is recommended. Adequate steady support for the head is a must and this can be accomplished by a trained assistant or head stand. The practitioner must have a thorough understanding of the anatomic structures involved.

Trephination and lavage of both the frontal and maxillary sinuses can readily be performed in the standing patient with adequate sedation and local anesthetic. After infusion of local anesthetic both in the subcutaneous tissue and periosteum at the location of trephination, a stab incision is made at the location of trephination. A Steinmann pin and hand-held chuck or Michelle trephine are used to create a hole in the sinus adequate in size for the insertion of the infusion port of a standard drip-set. The location of
Subsequent damage to the periosteum and a loss of blood supply leads to necrosis of a superficial segment of bone. The result is sequestrum treated and allowed to heal by second intention. The defect must be soaked gauze and the foot is appropriately bandaged and covered with an impermeable dressing such as duct tape. The contamination. A burr or sterilized hoof knife is used to create a defect at the determined location. The defect is extended to the level of the diseased pedal bone and debrided to remove the sequestrum and abnormal bone. The defect is then packed with betadine preparation of the site, bleb of local anesthetic is placed to facilitate the procedure. The epidural is performed with an 18 gauge spinal needle with stylet. The needle is introduced on midline with the bevel in a cranial direction, perpendicular to the skin. A “popping” sensation may be felt when the ligamentum flavum is penetrated and an audible “hiss” may be apparent when the epidural space is entered. A drop of sterile saline placed in the hub of the needle will be aspirated into the space when encountered. A maximum of 10 mls is usually used to prevent paralysis of the lumbosacral nerves. A combination of lidocaine (0.22mg/kg) and xylazine (0.17 mg/kg) expanded to a total volume of 8 mls with sterile saline offers reliable analgesia. Sedation and ataxia may occur with epidural administration. A tail-tie is usually enough to provide support in the face of mild to moderate ataxia. Recumbency is a rare but possible result of epidural anesthesia.

Surgery of the distal limbs including wound evaluation, laceration repair, or coffin bone debridement can be performed with use of routine perineural nerve blocks and adequate sedation. Once the location of disease is confirmed pedal osteitis and sequestrum formation can be treated using standing debridement with the following technique.

After adequate sedation an abaxial sesamoid block is performed with mepivacaine on the affected foot. The foot is surgically prepared by scrubbing and soaking in a dilute betadine solution. The distal limb proximal to the foot is wrapped to limit contamination. A burr or sterilized hoof knife is used to create a defect at the determined location. The defect is extended to the level of the diseased pedal bone and debrided to remove the sequestrum and abnormal bone. The defect is then packed with betadine soaked gauze and the foot is appropriately bandaged and covered with an impermeable dressing such as duct tape. The defect must be treated and allowed to heal by second intention.

Sequestrum formation is a common sequelae to trauma common to the equine limb such as kicks, lacerations and penetrating wounds. Subsequent damage to the periosteum and a loss of blood supply leads to necrosis of a superficial segment of bone. The result is devitalized bone and sequestrum formation. This may take several days to weeks to occur and the owner may be unaware at that point of the inciting traumatic incident. Reactive bone forms around the necrotic sequestrum and is commonly referred to as the involucrum. Although wounds involving loss of soft tissue coverage, periosteal damage and desiccation of bone are most at risk of sequestrum development the diagnosis is not made until lysis around the bone becomes extensive enough to be evident radiographically. Often the diagnosis is not made until a wound fails to heal appropriately or begins to drain after antimicrobials are discontinued. Diagnosis is frequently delayed when the failure to heal appropriately is blamed on inappropriate antimicrobial choice or length of treatment. Radiographs may be unrewarding until the lysis becomes more apparent. A cloaca or draining tract often develops which leads the practitioner directly to the area of bone to be addressed. A small sequestrum may be treated successfully without surgical intervention given a prolonged period of time with appropriate antimicrobial therapy but treatment may actually be less expensive with definitive surgical removal of the sequestrum.

Standing surgical removal of a sequestrum requires definitive radiograph diagnosis, a patient amenable to standing surgery and knowledge of the underlying anatomy. It is helpful to use per-surgery radiographs to measure the size of the sequestrum before surgical exploration to ensure the entire sequestrated bone has been removed. Post-operative radiographs are also helpful to ensure removal. With proper debridement, intensive local antibiotic therapy and long-term systemic antimicrobials are usually not necessary.
As with any standing surgical procedure, adequate sedation and local anesthesia is crucial for success. In the distal limb a tourniquet is helpful to limit hemorrhage and aid in visualization. However, there is often a lack of direct visualization of the sequestered bone and surgery is more based on feel with appropriate instrumentation.

**Conclusion**
A combination of sedation and local anesthetic will allow the performance of many surgical procedures in the standing horse. The individual animal temperament and environmental conditions as well as safety of the patient, veterinarian and assistants must be evaluated when deciding if standing surgery is appropriate. Regardless of the surgical procedure, appropriate antimicrobial and anti-inflammatory medications should be used and tetanus toxoid should always be current. Most importantly, aseptic technique is as important in the standing patient as it is under general anesthesia. Keeping those facts in mind, many procedures can be successfully performed standing negating the risks and expense of general anesthesia.
The equine upper airway is a high, resistance, low-capacity path between the nares and lungs. Proper function of the equine upper airway requires the coordinated effort of a large number of anatomical structures. Dysfunction of these structures results in profound changes in airway dynamics, decreases in air moving into the lungs and ultimately decreased tissue perfusion. Very small changes in diameter of the upper airway are magnified in their effect on the ability of the horse to move air into the lower respiratory system and oxygenate tissue effectively. For example, a 20% decrease in airway radius doubles airway resistance. For this reason, upper airway abnormalities must be addressed appropriately to maximize the equine athlete’s performance.

Dorsal displacement of the soft palate (DDSP)

DDSP most commonly occurs in racehorses, but can occur in other types of performance horses, particularly those required to over flex at the poll. It is an expiratory obstruction and the presenting complaint is often “choking down” or making a “gurgling” noise. These horses are often observed to be open-mouthed breathing during episodes and once the palate displaces they are unable to breathe sufficiently, which leads to rapid slowing or stopping. At this time, they usually swallow and replace the palate into normal position, causing the gurgling noise to dissipate and the open-mouth breathing to stop. Substantial exercise intolerance occurs during DDSP due to disruption in airflow. While gurgling is relatively common, DDSP cannot be ruled out in a horse that is exercise intolerant, but does not make a noise. Approximately 30% of horses affected with DDSP reportedly do not make a noise. (Parente, Martin et al. 2002)

This causes of this condition are complex not completely understood resulting in several treatment options both conservative and surgical. Conservative management includes rest, tack changes such as a figure-eight noseband or tongue-tie and anti-inflammatory medication. Failure to respond to these treatments may necessitate surgical intervention in the form of thermal palatoplasty, sternohyoideus and/or sternothyroideus myectomy, or laryngeal tie-forward.

The laryngeal tie-forward replaces the thyrohyoideus muscle which is thought to be dysfunctional in many cases of DDSP. The surgery resulted in significant improvement for 80-85% of cases on which it was performed and recovery time was minimal. (Woodie, Ducharme et al. 2005)

Laryngeal hemiplegia

The most appropriate treatment for laryngeal hemiplegia depends on the severity of disease, presenting complaint and the intended use of the horse. The non-racehorse presented for upper respiratory noise without a history of exercise intolerance is a candidate for ventriculocordectomy or ventriculectomy. This results in resolution of clinical signs in most cases but is unlikely to have a significant effect on exercise intolerance. Prosthetic laryngoplasty or “tie-back” is necessary to improve cases of exercise intolerance or poor performance particularly in racehorses. In situations of maximal exertion the increased airway diameter gained from the “tie-back” is required. The prosthetic laryngoplasty technique has been modified recently resulting in a more effective stable situation which may prove to be more effective with less relaxation of the suture and affected arytenoid.

Axial deviation of the aryepiglottic fold (ADAF)

Axial deviation of the aryepiglottic fold is a dynamic condition which can only be evaluated and diagnosed using dynamic endoscopy. The aryepiglottic fold is a membraneous piece of tissue that extends between the corniculate process of the arytenoids and the lateral edge of the epiglottis. On resting endoscopy the upper airway will appear completely normal but the condition can result in a greater than 80% reduction in inspiratory airway. It tends to occur at near maximal exertion when the airway pressures are most negative. ADAF can be seen in combination with other upper airway abnormalities or alone. It can be unilateral or bilateral and most often the right aryepiglottic fold exhibits the most deviation. Most commonly surgical treatment involves resection of a portion of one or both aryepiglottic folds using a laser as a transcendoscopic procedure. Recovery time is minimal and success rates are high.

References