ECG of the Month

An 8-year-old 527-kg (1,159-lb) castrated male Appaloosa was evaluated by a referring veterinarian because of colic, of which the horse had a history of repeat episodes. During an episode of colic 6 months previously, the horse responded to treatment with flunixin meglumine. The referring veterinarian evaluated the horse at the farm; because the horse did not respond to treatment with flunixin at this time, it was transported to the veterinarian's clinic for further examination. During transrectal palpation, the referring veterinarian palpated a taut ventral cecal band. A sample of peritoneal fluid was collected via abdominal paracentesis; the protein content (0.4 g/dL; reference range, < 2.5 g/dL) of the fluid sample was considered normal. The results of the abdominal ultrasonography by the referring veterinarian were suggestive of a mass lesion adjacent to the spleen (at the level of the seventh intercostal space). The horse was referred to the Cornell University Hospital for Animals for further evaluation.

On initial evaluation, the horse assumed an abnormal stance with a pronounced forward extension of the forelimbs and rearward extension of the hind limbs with respect to the body. Auscultation revealed tachycardia (heart rate, 60 beats/min) with a normal cardiac rhythm; borborygmi were diminished in all 4 abdominal quadrants. The horse was administered a dose each of xylazine hydrochloride (0.3 mg/kg [0.14 mg/lb], IV) and butorphanol tartrate (0.01 mg/kg [0.005 mg/lb], IV). Transrectal palpation revealed mild gas distention of the large intestine and a taut large colon tenia that was oriented transversely in the abdomen. Abdominal ultrasonography revealed that the small intestine was hypomotile and nondistended. In the area of the left seventh intercostal space, an accumulation of peritoneal fluid surrounded a portion of omentum or small intestine, but the overall amount of peritoneal fluid was not considered abnormal, and no mass lesion was detected. The horse received a bolus of isotonic crystalloid fluids (5 L) IV and then was administered isotonic crystalloid fluids with potassium chloride (20 mEq/L) IV at a rate approximately 1.5 times maintenance requirements. Approximately 1 hour after administration of xylazine and butorphanol, lidocaine hydrochloride (1.3 mg/kg [0.59 mg/lb]) was administered IV over a period of 10 minutes, which was followed by commencement of a constant rate infusion (CRI) of lidocaine (0.05 mg/kg/min [0.023 mg/lb/min], IV) to promote intestinal motility.

Approximately 1.5 hours after the CRI of lidocaine was started, the horse developed generalized skeletal muscle fasciculations and an arrhythmia was detected via cardiac auscultation. An ECG rhythm strip was recorded (**Figure 1**), and the lidocaine administration was then discontinued.

Contributed by Kathleen R. Mullen, MS; Anna R. M. Gelzer, DMV, DACVIM; Marc S. Kraus, DVM, DACVIM; Katharyn Mitchell, BVSc; and Thomas J. Divers, DVM, DACVIM, DACVECC; from the Department of Clinical Sciences, College of Veterinary Medicine, Cornell University, Ithaca, NY 14853.
Address correspondence to Dr. Kraus (msk16@cornell.edu).

ECG Interpretation

The ECG rhythm strip obtained from the horse during the lidocaine CRI revealed supraventricular tachycardia at a rate of 140 beats/min and a ventricular rate of 52 beats/min (Figure 1). The P waves appeared to originate from the sinus node at a regular rate. The

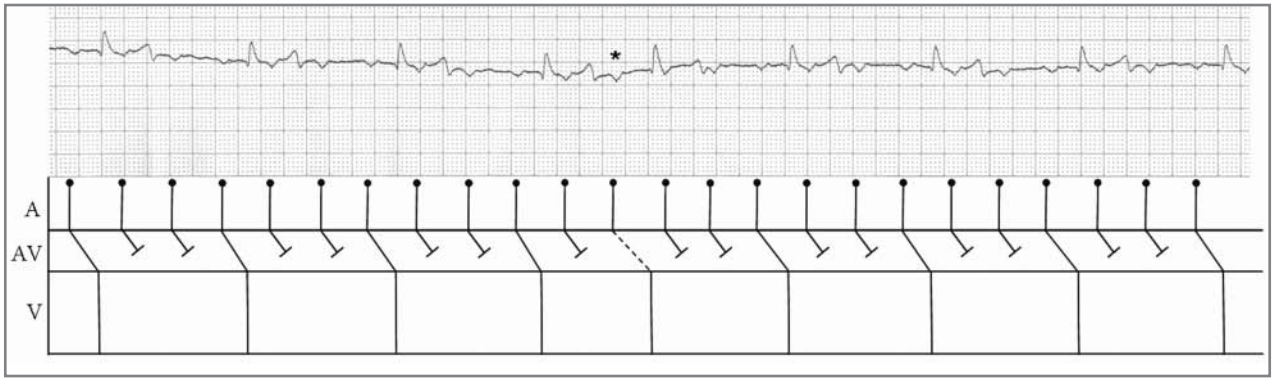

Figure 1—Monitoring lead ECG rhythm strip recorded approximately 1.5 hours after initiation of a constant rate infusion (CRI) of lidocaine to treat an episode of colic in an 8-year-old horse. Mean supraventricular rate is 140 beats/min, and mean ventricular rate is 52 beats/min. In the constructed ladder diagram, the presumed pathway of cardiac conduction is illustrated; vertical lines mark the beginning of P waves (A level) and QRS complexes (V level), and connecting lines between the vertical lines indicate atrioventricular (AV) conduction (AV level). The P waves are depicted as originating from the sinus node at a regular rate. The P wave preceding the ventricular complex is assumed to be that which conducts through the AV node to the ventricles. Notice that there is a 3:1 AV conduction block except at 1 point (asterisk), at which there is a depolarization indicative of a beat conducted after a 2:1 AV conduction block. This conduction occurred with a prolonged PR interval (indicated by the hatched line in the AV segment). The morphologies of the conducting and nonconducting P waves were consistent throughout the recording. The P-P intervals were regular, as were the P-R intervals for conducting P waves except for the complex conducting with the 2:1 block. Paper speed = 25 mm/s; 5 mm = 1 mV.

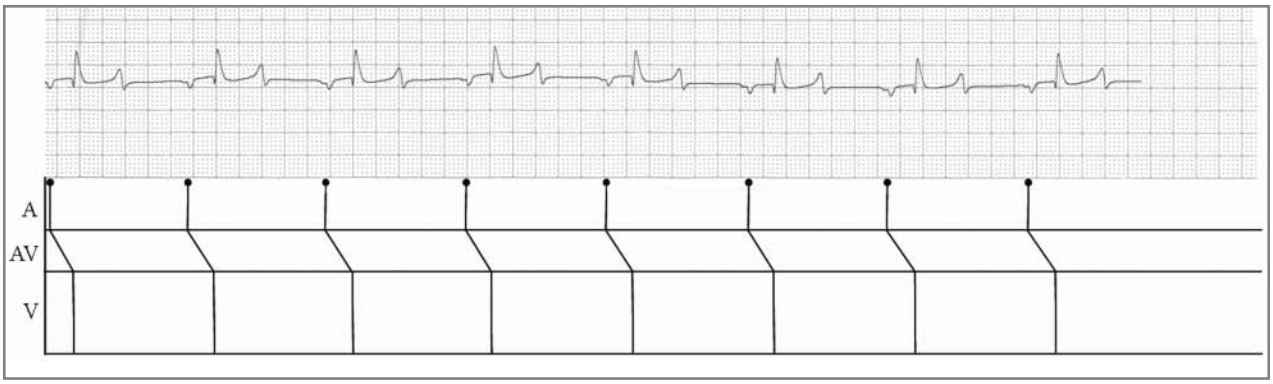

Figure 2—Monitoring lead ECG rhythm strip recorded from the horse in Figure 1 approximately 2 hours after discontinuation of the lidocaine CRI. Conversion to normal sinus rhythm occurred spontaneously; heart rate is 51 beats/min. Paper speed = 25 mm/s; 5 mm = 1 mV.

P wave that preceded the ventricular complex was apparently conducted through the atrioventricular (AV) node to the ventricles. This represented a 3:1 AV conduction block. However, during 1 depolarization, a beat was conducted after a 2:1 block; this conduction occurred with a prolonged P-R interval. This suggested that the P wave that conducted through the AV node after only 1 blocked P wave (2:1 block) did so while the AV node was still partially refractory. For the other conducted P waves, a longer interval (3:1 block) allowed the AV node to fully recover. Alternatively, atrial impulses that are blocked in the AV node and not conducted to the ventricle may slow conduction of subsequent atrial impulses through the AV node, a phenomenon known as concealed conduction, which may have occurred in this horse. The morphology of the conducting and nonconducting P waves was consistent throughout the ECG recording. The P-P intervals were regular, as were the P-R intervals for the conducting P waves except for the 1 complex that was conducted with the 2:1 block. The variable AV conduction pattern resulted in the irregularity of the rhythm detected by auscultation.

Within an hour after detection of the arrhythmia, a CBC and serum biochemical analyses were performed; the results were unremarkable. Approximately 2 hours after discontinuation of the CRI, the ausculted heart rate was considered normal (regular rhythm) and a second ECG rhythm strip was recorded (**Figure 2**). This second ECG rhythm strip revealed a normal sinus rhythm (mean heart rate, 51 beats/min) and complete resolution of the supraventricular tachycardia and conduction block.

Later that day, the horse excreted feces that were considered normal in consistency and appearance, and no abnormal findings were detected via transrectal palpation and abdominal ultrasonography. Echocardiography was not performed because of the transient nature of the arrhythmia and resolution of the clinical signs associated with the colic. The horse was fed small amounts of wet feed, which it ate with an apparently good appetite, and was discharged to the care of its owner later that day. No recurrence of the arrhythmia was detected prior to discharge from the hospital, and no further problems with the horse have been reported by the owner 1 year later.

Discussion

Lidocaine hydrochloride is frequently used in the treatment of ileus associated with colic in horses. Lidocaine also has antiendotoxic, anti-inflammatory, and analgesic properties, making it a potentially beneficial treatment for horses with colic and colitis that do or do not require surgery.[1] Reported adverse effects associated with lidocaine administration at the dosage given to the horse of this report (the recommended dosage for treatment of intestinal ileus in horses) include skeletal muscle fasciculations and ataxia.[2] In a study[3] of 19 horses, the serum lidocaine concentration that resulted in development of skeletal muscle fasciculations did not induce any major changes in cardiovascular variables (ie, heart rate and arterial blood pressure measurements) and respiratory rate.

Despite the relative infrequency of reported cardiovascular adverse effects of lidocaine administration in horses, the rapidity with which supraventricular tachycardia and AV block developed after commencement of lidocaine administration and with which normal sinus rhythm resumed following discontinuation of the drug treatment in the horse of this report supports lidocaine toxicosis as a cause of these cardiac abnormalities. In a study[4] of the effects of lidocaine on cardiac activity in dogs under conditions of vagal stimulation, lidocaine increased the rate of the sinoatrial node discharge and prolonged AV conduction. The mechanism of the differential effects of lidocaine on the sinoatrial and AV nodes is unknown.

The atypical signs that developed following lidocaine administration in the horse of this report were likely a result of mild abdominal distention, which is hypothesized to cause vagal stimulation in horses,[5] in combination with a mechanism (proposed by Lieberman et al[4]) by which lidocaine increases sinoatrial nodal discharge rate.

Potentially, the sedative drugs administered 3 hours prior to detection of the arrhythmia may have contributed to development of the arrhythmia. At admission, the horse received low doses of xylazine, a centrally acting α_2-adrenoreceptor agonist, and butorphanol, an opioid analgesic. It cannot be ruled out that the xylazine may have contributed to the slowed AV conduction and AV block and that the morphine-type sedative butorphanol

may have enhanced vagal tone, which contributed to the proarrhythmic state of the atria. In dogs, a proarrhythmic effect of fentanyl (a different type of synthetic opiate analgesic) has been reported[6]; however, a similar effect of fentanyl in horses has not been reported, to our knowledge, even though the drug is widely used. On the basis of pharmacokinetic study findings,[7,8] it seems probable that xylazine and butorphanol were cleared systemically or ineffective by the time the arrhythmia was detected, making them unlikely causes of the arrhythmia. Regardless, because discontinuation of the lidocaine infusion occurred concurrently with ongoing clearance of the sedative agents, it cannot be definitively determined which drug effect may have caused this arrhythmia or which drug clearance may have led to resolution of the arrhythmia.

If the supraventricular tachycardia were physiologic in nature as a result of pain (that caused increased sympathetic tone) or hypotension (that caused a reflex tachycardia), AV nodal conduction would be expected to accelerate concurrently instead of development of AV block. In addition, the horse had no other overt signs of pain at the time of arrhythmia detection, and lidocaine is not known to have hypotensive effects in horses at the dosage used. The arrhythmia was considered to be sinus tachycardia, as opposed to atrial tachycardia, because the P-wave morphology was compatible with a sinus origin and the heart rate (P-P interval) was consistent with typical findings of sinus tachycardia. The appearance of the P waves in the ECG tracing obtained 1.5 hours after administration of lidocaine was slightly more abnormal than that detected 2 hours after discontinuation of lidocaine administration. This slight change in P-wave morphology that occurred during the interval following cessation of lidocaine treatment might be explained by the fact that the ECG cables were disconnected and reconnected between the 2 recordings. Alternatively, it is possible that the P waves in the first tracing were ectopic P waves that originated at aberrant sites within the atria (ie, a fairly slow atrial tachycardia). However, this cannot be definitively determined from a surface ECG tracing, and definitive elucidation of the origin of the P waves would require further diagnostic evaluation.

Although cardiovascular adverse effects of lidocaine administration are reportedly rare, horses that develop skeletal muscle fasciculations during treatment with lidocaine should be evaluated for cardiac arrhythmias and lidocaine administration should be slowed or discontinued.

References

1. Cook VL, Jones Shults J, McDowell M, et al. Attenuation of ischaemic injury in the equine jejunum by administration of systemic lidocaine. *Equine Vet J* 2008;40:353–357.
2. Malone E, Ensink J, Turner T, et al. Intravenous continuous infusion of lidocaine for treatment of equine ileus. *Vet Surg* 2006;35:60–66.
3. Meyer GA, Lin HC, Hanson RR, et al. Effects of intravenous lidocaine overdose on cardiac electrical activity and blood pressure in the horse. *Equine Vet J* 2001;33:434–437.
4. Lieberman NA, Harris RS, Katz RI, et al. The effects of lidocaine on the electrical and mechanical activity of the heart. *Am J Cardiol* 1968;22:375–380.
5. Abutarbush SM. Use of ultrasonography to diagnose large colon volvulus in horses. *J Am Vet Med Assoc* 2006;228:409–413.
6. Pariaut R, Moïse NS, Koetje BD, et al. Evaluation of atrial fibrillation induced during anesthesia with fentanyl and pentobarbital in German Shepherd Dogs with inherited arrhythmias. *Am J Vet Res* 2008;69:1434–1445.
7. Sellon DC, Monroe VL, Roberts MC, et al. Pharmacokinetics and adverse effects of butorphanol administered by single intravenous injection or continuous intravenous infusion in horses. *Am J Vet Res* 2001;62:183–189.
8. Garcia-Villar R, Toutain PL, Alvinerie M, et al. The pharmacokinetics of xylazine hydrochloride: an interspecific study. *J Vet Pharmacol Ther* 1981;4:87–92.