An 11-year-old 531-kg (1,168-lb) Quarter Horse gelding was found to have an irregular heart rhythm and tachycardia (heart rate, 79 beats/min) during a prepurchase examination. The current owner reported that the horse appeared normal and was in regular work as a hunter. An ECG rhythm strip revealed an irregularly irregular rhythm with periods of no discernible P waves and variable atrioventricular (AV) conduction (Figure 1). Atrial fibrillation (AF) was suspected, prompting referral of the horse to the Cornell University Hospital for Animals for further diagnostic testing.

On initial evaluation, the horse was bright and alert with no detectable abnormalities on physical examination other than mild tachycardia (heart rate, 48 beats/min) and an irregular cardiac rhythm. Results of a CBC and serum biochemical analysis were unremarkable, with the exception of hyperfibrinogenemia (500 mg/dL; reference range, 0 to 200 mg/dL). Another ECG rhythm strip recording was obtained, and AF was confirmed. Echocardiography revealed mild tricuspid valve prolapse with no regurgitation; cardiac dimensions, wall thickness, and systolic function were considered normal. The horse was deemed to be a good candidate for cardioversion, and transvenous electrical cardioversion was recommended.

ECG Interpretation

Following placement of transvenous cardioversion catheters in the horse’s left pulmonary artery and the right atrium aided by echocardiography and thoracic radiography, anesthesia was induced and the horse was positioned in right lateral recumbency. The horse received 6 defibrillation shocks at incremental doses between 50 and 100 J each, starting at 100 J and increasing to 360 J, at which time presumptive atrial tachycardia with AV nodal block was observed (Figure 1). The horse recovered uneventfully from anesthesia. Following recovery, an ECG examination revealed atrial tachycardia with 1:1 to 5:1 AV conduction, mean ventricular rate of 60 beats/min, and mean atrial rate of approximately 170 beats/min (Figure 2). The horse was prophylactically administered dexamethasone (0.08 mg/kg [0.036 mg/lb]) IV in case underlying myocarditis was the cause of the arrhythmia. Serum cardiac troponin I concentration could not be measured owing to equipment failure. Pharmacologic correction of the atrial tachycardia was attempted that evening with administration of quinidine sulfate (22 mg/kg [10 mg/lb]) via nasogastric tube every 6 hours. However, following the second dose, the horse became profoundly tachycardic (heart rate, 130 beats/min) and treatment was discontinued. The next morning, atrial tachycardia persisted with a heart rate of 52 beats/min, and oral administration of sotalol hydrochloride (2.5 mg/kg [1.14 mg/lb]) every 12 hours was implemented. Electrocardiographic monitoring was continued, and over the next 3 days, the sotalol dosage was incrementally increased to 4 mg/kg (1.8 mg/lb) every 12 hours. However, the horse developed focal sweating over the left maxilla and cranial neck region; therefore, the dosage was decreased to 3.5 mg/kg.

Figure 1—Electrocardiographic rhythm strips recorded during a prepurchase examination of an 11-year-old Quarter Horse gelding (A) and when the horse was later anesthetized for electrocardioversion of atrial fibrillation 7 days later (B). The heart was defibrillated a total of 6 times at doses increasing in increments between 50 and 100 J each, starting at 100 J. The 360-J shock (thick arrow) is preceded by atrial fibrillatory waves. Following the shock, presumptive atrial tachycardia with atrioventricular nodal block is observed. Some P waves are presumed hidden within the QRS complexes or ST segments (thin dashed arrows) following conversion; other P waves are clearly identifiable (solid arrows). The black triangles indicate the defibrillator synchronizing with the R wave. In panel A, paper speed = 12.5 mm/s; 10 mm = 1 mV. In panel B, paper speed = 25 mm/s; 5 mm = 1 mV.
hours following a single IV dose of procainamide hydrochloride (20 mg/kg [9.1 mg/lb]). Normal sinus rhythm is shown; heart rate is 40 beats/min. [Paper speed = 50 mm/s; 5 mm = 1 mV.]

Figure 3—Electrocardiographic rhythm strip obtained from the horse in Figure 1 8 days after initiation of sotalol administration and 24 hours following a single IV dose of procainamide hydrochloride (20 mg/kg [9.1 mg/lb]). Normal sinus rhythm is shown; heart rate is 40 beats/min. Notice that the P waves are biphasic, a normal waveform variation in horses. [Paper speed = 50 mm/s; 5 mm = 1 mV.]

(1.59 mg/lb). Despite treatment, atrial tachycardia persisted. Results of repeated CBCs and serum biochemical analyses remained within reference ranges throughout hospitalization. Serum cardiac troponin I concentration was determined on day 7 of hospitalization and was also within reference limits (0.02 ng/mL; reference range, 0.00 to 0.06 ng/mL).

Seven days after electrical cardioversion, while the horse was maintained on sotalol administered every 12 hours, a single IV dose of procainamide hydrochloride (20 mg/kg [9.1 mg/lb]) was administered slowly over a period of 10 minutes. An ECG recording obtained during and immediately following procainamide administration indicated that the horse had continued atrial tachycardia; however, 24 hours later, an ECG recording revealed normal sinus rhythm (Figure 3). The horse was discharged from the hospital 2 days later, and the owner was instructed to administer sotalol orally (3.5 mg/kg) every 12 hours and restrict the horse to stall rest with hand-walking only. The referring veterinarian performed a recheck ECG examination 6 weeks after hospital discharge and just prior to discontinuation of treatment with sotalol; at this time, the normal sinus rhythm persisted, although the horse was tachycardic (heart rate, 74 beats/min). Three weeks later (9 weeks after hospital discharge), a final ECG recording was obtained, which indicated that the horse had a normal sinus rhythm and heart rate (39 beats/min).

Discussion

In people, it is not uncommon for other atrial tachyarhythmias to develop after undergoing ablation or electrical cardioversion procedures for treatment of AF. Following radiofrequency ablation for AF in humans, atrial arrhythmias have been reported to develop in up to 52% of cases.1 The exact mechanisms involved in development of atrial tachyarrhythmias after ablation or electrical cardioversion procedures are unknown. Some postulated mechanisms include focal scarring and inflammatory effects of ablation that result in cellular dysfunction and changes in the atrial structure, thereby creating a substrate for reentry.1 Atrial stunning or transient dysfunction of the left atrium, which causes atrial stretch, may contribute to arrhythmias after electrical conversion.1 Atrial arrhythmias observed immediately following internal electrical cardioversion are common in people with chronic AF, and the presence of atrial premature beats has been shown to predict early AF recurrence.1 Development of arrhythmias, particularly premature atrial contractions and complete AV block, in horses following electrical cardioversion have been reported.2,3 In the horse of the present report, ongoing myocardial necrosis as a cause for the persistent atrial tachycardia was less likely given that the serum cardiac troponin I concentration determined on day 7 of hospitalization was within reference limits. For this horse, it is unknown whether cardioversion unmasked preexisting atrial tachycardia or whether the atrial tachycardia was a consequence of the procedure. There is a reported case6 of a horse in which an accessory bypass tract was unmasked by electrocardioversion of AF to sinus rhythm. For the horse of the present report, results of electrophysiological mapping to identify the origin of the atrial tachycardia would have been interesting. However, such a procedure requires biplane fluoroscopy to place the mapping electrodes and considerable technical expertise; thus, electrophysiological mapping is not performed routinely in equine practice.

Initially, correction of the atrial tachycardia by administration of quinidine was attempted, whereas repeated electrocardioversion has been used successfully in some humans.7 Although quinidine did not appear to cause gastrointestinal adverse effects in the horse of this report, we were concerned that the vagolytic effects of quinidine would induce accelerated AV conduction (up to 1:1), which could result in very fast ventricular rates (≥ 200 beats/min). Thus, when the heart rate began to accelerate after the second dose of quinidine, this drug treatment was discontinued. Sotalol, a class III antiarrhythmic, has both nonspecific β-adrenoreceptor blocking as well as potassium channel blocking effects. It is effective for slowing of the ventricular response rate in patients with atrial tachyarrhythmias. Although pharmacokinetic studies of sotalol in horses are lacking, extrapolated dosages from humans and dogs have been used with success at various facilities in the experience of one of the authors (MSK). Sotalol was chosen instead of amiodarone, a common first-line choice in human medicine for recurrent atrial tachycardia, because drug-related adverse effects are reportedly less common following administration of sotalol. However, focal sweating (an adverse effect of sotalol described for people8) was observed in the horse of the present report when sotalol was administered orally at a dosage of 4 mg/kg every 12 hours. Procainamide,
a class IA antiarrhythmic drug, has similar electrophysiologi-cal actions as quinidine and, in humans, is indicated for the treatment of supraventricular and ventricular arrhythmias. In horses, pharmacokinetics for procainamide indicates a mean half-life of 3.49 hours when administered IV at 20 mg/kg. The acetylated metabolite of procainamide, N-acetylprocainamide, has a longer mean half-life (6.31 hours) with peak serum concentration at 5.2 hours after administration. It has been suggested that N-acetylprocainamide may accumulate to active concentrations, resulting in a delayed onset of maximal drug effect. Furthermore, pharmacokinetic and pharmacodynamic testing of procainamide was performed in healthy horses without arrhythmias, and the drug and its metabolite may behave differently in horses with arrhythmias. Nevertheless, the horse of the present report had AF and subsequently persistent atrial tachycardia after electrical cardioversion, but was then treated successfully with rest and administration of sotalol and procainamide. To our knowledge, this clinical scenario in horses has not been previously reported.

References