ECG of the Month

A 6-year-old 430-kg (946-lb) Standardbred gelding with a history of intermittent upper airway noise was examined following completion of a race as part of a track safety initiative. The horse had just paced a mile in 1 minute and 53 seconds and finished in third place (crossing the finishing line 1 second after the winner). After the race, the trainer noticed no abnormalities except that the horse required more time than usual for cooldown. During the scheduled physical examination, the horse had a rapid regular cardiac rhythm with a heart rate of 200 beats/min and visible pulses in the jugular veins. The horse was tachypneic and hyperthermic, consistent with recent exertion. Electrocardiography was performed by use of a handheld clipless digital recording device.[a]

ECG Interpretation

An initial postrace ECG recording (**Figure 1**) was obtained with a handheld device that collects data from a single, nonstandard lead (similar to a base apex lead) when placed over the left heart base just behind the triceps brachii muscle. The first tracing revealed a sustained uniform tachyarrhythmia, with a rapid regular rhythm at a rate of 205 beats/min; uniform QRS complexes (QRS complex duration, 90 milliseconds), an elevated ST segment, and tall, positive T waves. P waves were not visible and were presumed to be buried within the other complexes.

Interpretation of the ECG findings was made in light of the horse's recent exertion. The heart rate was considered inappropriately high given that the recording was obtained 14 minutes after completion of the race when the heart rate would be expected to have decreased to < 120 beats/min. Interpretation of QRS complex width was difficult because of a lack of published information regarding the effects of exercise and heart rate on QRS complex duration in horses. A QRS complex duration of 90 milliseconds is considered normal for a horse at rest. The ST-segment elevation and tall T waves were considered typical for the high heart rate because similar changes are associated with sinus tachycardia at comparable heart rates.[1]

The inability to clearly identify p waves made differentiation between ventricular and supraventricular tachycardia difficult; however, a diagnosis of ventricular tachycardia was supported by the presence of jugular pulsations. Jugular pulsations are produced when there is dysynchrony of the atria and ventricles such as that which develops during ventricular tachycardia. The right atrium contracts against the closed tricuspid valve, causing blood to reflux from the atrium into the jugular veins, resulting in a visible jugular pulsation. With sustained atrial tachycardia, this dysynchrony does not occur and therefore jugular pulsations are not generated.

The horse was treated with lidocaine hydrochloride (0.5 mg/kg [0.23 mg/lb], IV) twice (injections were administered 15 minutes apart). Conversion to a normal sinus rhythm was evident 5 minutes after the second dose of lidocaine (approx 90 minutes after completion of the race; **Figure 2**). At this time, the QRS complex polarity was considered normal for the applied lead system. Following conversion, the horse visibly relaxed and the jugular pulses were no longer visible.

Approximately 90 minutes after completion of the race, a blood sample was collected to obtain plasma for venous blood gas analysis and assessment of electrolyte (sodium, potassium, chloride, and calcium) and cardiac troponin I (cTnI) concentrations. Venous blood gas results and plasma electrolyte concentrations were within reference limits, but plasma cTnI concentration was high (0.38 ng/mL; reference range, ≤ 0.07 ng/mL[b]). The horse was rehydrated PO. Dexamethasone (0.08 mg/kg [0.04 mg/lb]) and flunixin meglumine (1.1 mg/kg [0.5 mg/lb]) were administered IV, and electrolyte paste was administered PO. A Holter monitor[c] was placed to obtain a 24-hour recording, and the horse was referred for monitoring and further investigation of the cause of the tachyarrhythmia.

On arrival at the referral hospital, cardiac auscultation revealed that the cardiac rhythm was regular and heart rate was within reference limits. A grade 2/6 holosystolic, blow-

Contributed by Amy M. Santonastaso, VMD; Laura E. Faulkner, VMD; Kelsey R. Hilton, BS; and JoAnn Slack, DVM, MS, DACVIM; from the Department of Clinical Studies, New Bolton Center, University of Pennsylvania, Kennett Square, PA 19348. Dr. Santonastaso's present address is Department of Large Animal Clinical Sciences, College of Veterinary Medicine & Biomedical Sciences, Texas A&M University, College Station, TX 77843.
Address correspondence to Dr. Slack (slackj@vet.upenn.edu).

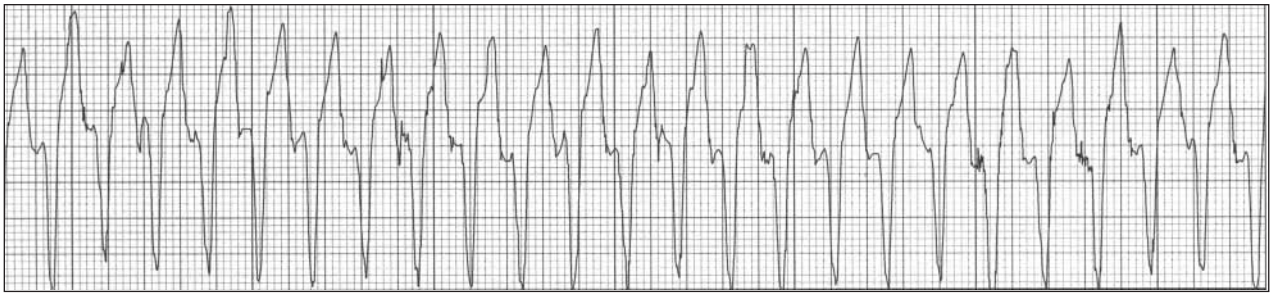

Figure 1—Single, nonstandard lead (similar to a base apex lead) ECG recording obtained with a handheld device from a Standardbred gelding that was examined following completion of race as part of a track safety initiative. Notice the sustained uniform tachyarrhythmia (rapid regular rhythm at a rate of 205 beats/min) with uniform QRS complexes (duration, 90 milliseconds), an elevated ST segment, and tall, positive T waves. P waves are not visible and were presumed to be buried within the other complexes. Paper speed = 25 mm/s; 1 cm = 1 mV.

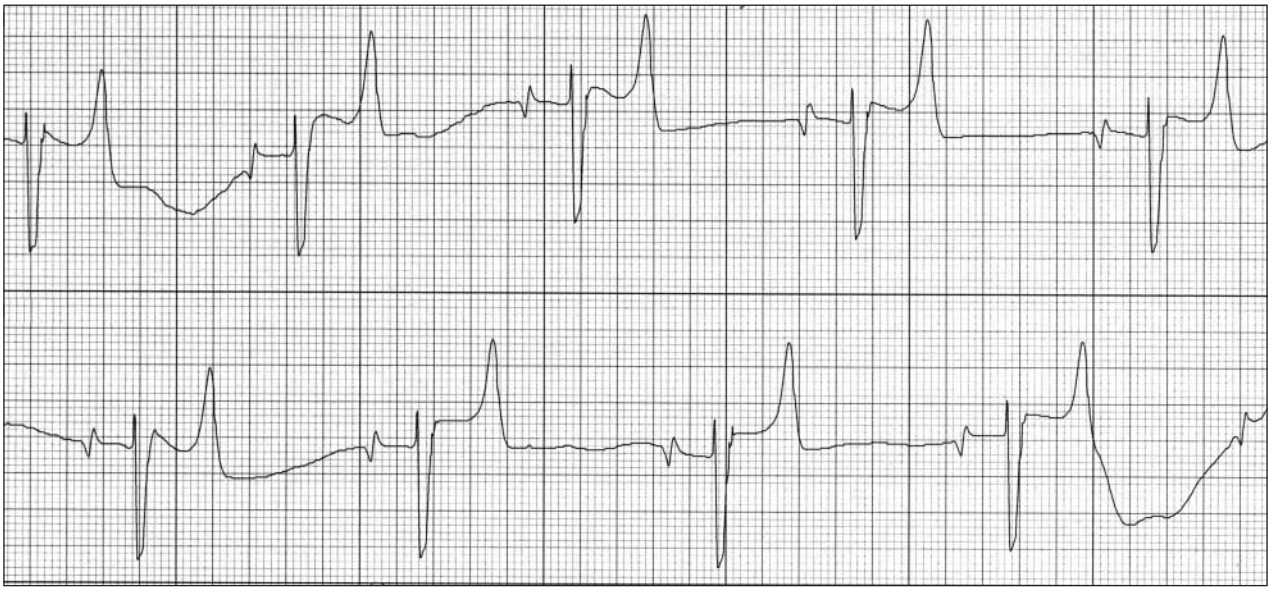

Figure 2—Single, nonstandard lead (similar to a base apex lead) ECG recording obtained with a handheld device from the horse in Figure 1 five minutes after IV administration of a second dose of lidocaine hydrochloride (approx 90 minutes after completion of the race). The upper tracing is continuous with the lower tracing. Notice the normal sinus rhythm (heart rate, 40 beats/min). The p waves are biphasic, which is a normal finding for this lead system. The QRS complexes are uniform (duration, 90 milliseconds), the ST segment appears normal, and the T waves are positive. Paper speed = 25 mm/s; 1 cm = 1 mV.

ing, band-shaped murmur with the point of maximal intensity over the tricuspid valve and a grade 1/6 holosystolic, blowing, band-shaped murmur over the mitral valve were detected. Electrocardiographic evaluation revealed a normal sinus rhythm. On the basis of 2-D, M-mode, and Doppler echocardiographic findings, heart size and myocardial function were considered normal and there were no structural abnormalities of the myocardium. Mild tricuspid and mitral valve regurgitation were evident. The 24-hour Holter monitor recording revealed 2 ventricular premature complexes of differing morphologies and 35 uniform supraventricular premature depolarizations. Results of a CBC and assessment of plasma fibrinogen concentration were within reference limits. Serum biochemical analysis revealed mildly high concentrations of creatinine (1.9 mg/dL; reference range, 0.6 to 1.8 mg/dL) and total protein (7.0 g/dL; reference range, 4.6 to 6.9 g/dL) consistent with mild dehydration. The horse was discharged from the hospital after 2 days; the treatment plan included administration of dexamethasone (tapering dosage), stall rest, and paddock turnout with no forced exercise for 3 weeks. A follow-up, 24-hour continuous ECG performed at the end of the 3-week period revealed 4 supraventricular premature depolarizations but no evidence of ventricular ectopy. It was recommended that the horse undergo an ECG assessment during exercise and a dynamic upper airway evaluation before returning to racing. Unfortunately, those procedures were not performed and the horse was returned to racing, subsequently claimed, and lost to follow-up.

Discussion

Arrhythmias in horses that are associated with racing have been occasionally reported in the veterinary medical literature,[2–4] with atrial fibrillation being the most common. Diagnosis of a tachyarrhythmia in the race recovery period is typically contingent upon the horse developing clinical signs or having a sufficiently poor performance to warrant physical examination. The clinical signs of ventricular tachycardia in horses may include sweating, agitation, colic-like behavior, tachypnea, and jugular pulsations.[5,6] The effects on racing performance may be none, mild, or dramatic depending on the point at which the arrhythmia develops during the race. In the horse of this report, the diagnosis was made on the basis of the ECG findings; the horse's ECG evaluation was prompted by the track's cardiac safety initiative and not by any specific performance problems or physical examination abnormalities. Although the horse's trainer had noticed that it took more time than usual for cooldown, signs of mild anxiety and jugular pulsations were only noted retrospectively after abnormalities in the initial ECG recording were detected. The signs of anxiety were made more evident when the horse visibly relaxed after converting back to a normal sinus rhythm. Tachypnea and sweating are expected postrace findings in horses and did not aid in the identification of a tachyarrhythmia in this case setting. Given the performance of the horse of this report, it is likely that the arrhythmia developed either very near the end of the race or in the race recovery period. In a recent report[7] of arrhythmias in Standardbred racehorses, complex ventricular arrhythmias that occurred in the race recovery period were detected in 55 of 345 race starts. Those arrhythmias resolved quickly and spontaneously and would have likely remained undetected had telemetric monitoring not been in place.

During exercise, there are substantial autonomic and homeostatic changes that could contribute to the development of cardiac arrhythmias. These include autonomic tone changes, marked disturbances in plasma potassium concentration, and alterations in acid-base balance. In physiologically normal mammals, these changes are proposed to occur in a balanced manner, each affording some protection from the proarrhythmic effects of the other.[8] In the postexercise period, this balance of factors may

be more difficult to maintain because rapid alterations in autonomic tone occur with rebound parasympathetic activity and decreasing sympathetic tone. Although these are normal physiologic mechanisms responsible for cardiovascular recovery, they represent a period of autonomic instability that may be further influenced by sympathetic hyperactivity due to stress or excitement or by a reduced rate of parasympathetic reactivation.[7] When combined with other homeostatic disturbances or myocardial abnormalities, ventricular arrhythmias may develop.

Other predisposing factors for ventricular tachycardia include primary myocardial disease or inflammation, hypoxic myocardial injury, myocardial toxins, electrolyte abnormalities, and acid-base or other metabolic disturbances.[5,9,10] Potassium is the electrolyte that receives the most attention as a possible mediator of cardiac arrhythmias because both hyperkalemia and hypokalemia can affect action potentials in cardiac tissue. Hypokalemia increases the resting membrane potential, action potential duration, and refractory period of cardiac tissue, leading to increased automaticity and conduction disturbances.[11] Profuse sweating and the use of furosemide in the treatment of exercise-induced pulmonary hemorrhage may predispose racehorses to development of hypokalemia and arrhythmias. Hypokalemia increases the risk for ventricular arrhythmias and sudden cardiac death in humans.[12] In contrast to hypokalemia, hyperkalemia decreases the resting membrane potential, action potential duration, and effective refractory period of cardiac tissue, which can also lead to increased automaticity and conduction disturbances. During high-intensity exercise, hyperkalemia develops secondary to release of potassium from skeletal muscle.[13] In horses, plasma potassium concentration returns to baseline value usually within 5 to 10 minutes after completing exercise.[14] After exercise in some human athletes, circulating potassium concentrations may decrease from pre-exercise values and remain low for up to 90 minutes.[13] Plasma potassium concentration prior to exercise was not measured in the horse of this report; however, plasma potassium concentration was within reference limits approximately 90 minutes after exercise. Clinically important alterations of the circulating potassium concentration during and immediately after exercise could not be ruled out. The horse's high plasma cTnI concentration may have been an indication of preexisting myocardial inflammation or injury, hypoxic myocardial injury secondary to an upper airway disorder or lower airway inflammation, or poor myocardial perfusion secondary to the sustained tachyarrhythmia. Echocardiographic evaluation did not provide evidence of structural myocardial abnormalities, although small structural abnormalities may have easily been overlooked.

For the horse of this report, antiarrhythmic treatment consisted of IV administration of lidocaine prior to transportation to a referral facility. Delayed treatment of hemodynamically important ventricular arrhythmias (multiform arrhythmias or those with heart rates > 120 beats/min or R-on-T phenomena) is not recommended because affected horses are at risk for sudden death. Lidocaine (without epinephrine) is readily available and easily administered and should be on hand for veterinarians that deliver emergency medical care in racetrack or ambulatory settings. Lidocaine has a narrow therapeutic range in horses; consequently, it is usually administered in small boluses (0.5 mg/kg, IV, q 10 min up to total dose of 1.5 mg/kg [0.7 mg/lb]). Neurologic adverse effects including excitement and seizures may develop with higher dosages.[15]

Ventricular arrhythmias are of particular concern in racehorses because there is considerable danger to the rider or driver as well as other competitors on the track if a horse collapses or dies suddenly. Public perception of the sport may be adversely affected by such an event. Guidelines for human athletes with ventricular arrhythmias indicate that athletic activity should be delayed until a complete cardiac evaluation including echocardiographic evaluation and ECG assessments at rest, during exercise, and for a 24-hour period can be performed.[16] For the horse of this report, ECG monitoring, upper airway evaluation, and blood gas and electrolyte analysis during exercise and collection and analysis of a postexercise bronchoalveolar lavage sample were indicated to identify potential predisposing factors for ventricular tachycardia and determine a prognosis for its safe return to racing.

a. Vet Biolog II, QRS Diagnostic LLC, North Plymouth, Minn.
b. Stratus CS, Siemens Diagnostic Healthcare, Newark, Del.
c. Holter monitor, Impresario, Reynolds Medical Inc, Raleigh, NC.

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