

ECG of the Month

A 2-year-old 495-kg (1,089-lb) French Standardbred Amare was referred to the Equine Sports Medicine Unit, Centre d'Imagerie et de Recherche sur les Affections Locomotrices Equines, Goustranville, France, because of sudden development of exercise intolerance and cardiac arrhythmia. The horse had been in training for several months and was considered healthy until exercise intolerance developed during a training session at the racetrack. The referring veterinarian detected an arrhythmia and a murmur via cardiac auscultation and referred the horse for further evaluation.

At the initial referral evaluation, the horse was bright, alert, and responsive. Heart rate was 56 beats/min (reference limits for an adult horse at rest, 28 to 44 beats/min); the rhythm appeared regularly irregular but was interrupted by periods of irregularly irregular rhythm. Cardiac auscultation revealed a grade 3/6 right-sided holosystolic murmur with a point of maximal intensity over the tricuspid valve area. Rapid pulsation in the lower portion of the jugular groove was observed. No other abnormalities were detected during physical examination. Initial diagnostic evaluation included a CBC, serum biochemical analysis (including measurement of serum concentration of cardiac troponin I^a), ECG at rest and during exercise, and echocardiography. The CBC revealed slight leukocytosis (9,760 WBCs/mm³; reference range, 8,100 to 9,400 WBCs/mm³); no other abnormalities were evident via the CBC and serum biochemical analysis. Serum cardiac troponin I

concentration was within reference range (0.08 ng/mL; reference range, 0 to 0.11 ng/mL). During echocardiographic examination, discrete tricuspid valve regurgitation was identified (considered inconsequential at this point). Electrocardiography was performed when the horse was at rest and when exercising.

ECG Interpretation

At rest, ECG revealed moderate supraventricular tachycardia with a ventricular rate of approximately 54 QRS complexes/min and an atrial rate of 220 depolarizations/min (Figure 1). On the ECG tracing, several modified P waves (F or flutter waves) were present before each QRS complex. These F waves each had a similar morphology, cycle length (270 milliseconds), and polarity and were regularly spaced. Atrioventricular (AV) conduction was variable with a rate of 3 or 4:1 (ie, 3 or 4 F waves for each QRS complex), resulting in a regular ventricular rhythm and periods during which the conduction rate varied from 3:1 to 12:1; this resulted in an irregularly irregular rhythm (Figure 2). The morphology of QRS complexes appeared normal and did not vary from one complex to another. These features were consistent with a diagnosis of atrial flutter.

During a period of exercise on a high-speed treadmill,^b the horse was monitored via telemetry^c to obtain ECG data. The horse was walked for 5 minutes on the treadmill (at a speed of 1.7 m/s); the speed was then increased to 4.5 m/s, which corresponded to a gentle trot. The horse's heart rate increased rapidly, and severe tachycardia (heart rate, 220 beats/min) developed. The treadmill was stopped to unload the horse and observe it closely. The tachycardia persisted for several minutes before the horse's heart rate returned to the rate at rest. Evaluation of the exercise ECG data revealed 3 ventricular premature depolarizations with modified QRS complex morphology (R-on-T) before the onset of severe supraventricular tachycardia (Figure 3). The tachycardia was the result of sudden change in AV conduction rate to 1:1, during which each atrial depolarization was transmit-

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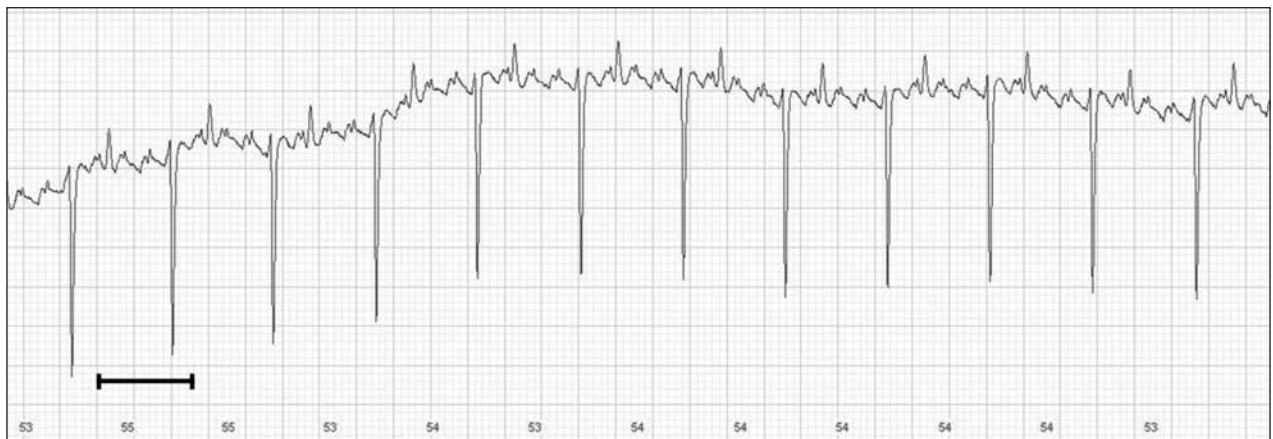


Figure 1—Modified base-apex ECG tracing obtained at rest from a horse that was evaluated because of exercise intolerance and arrhythmia. The heart rate is 54 to 56 beats/min, and the heart rhythm appears regular in this tracing. The QRS complexes are considered normal in terms of morphology and duration. Each QRS complex is preceded by 4 F waves that have a similar morphology, cycle length, and polarity and are regularly spaced. Atrial rate is approximately 220 depolarizations/min. These features are consistent with a diagnosis of atrial flutter. The numbers at the bottom of the image represent the heart rate (beats/min) calculated by the software program. Black bar represents 1 second; 3 cm = 1 mV.

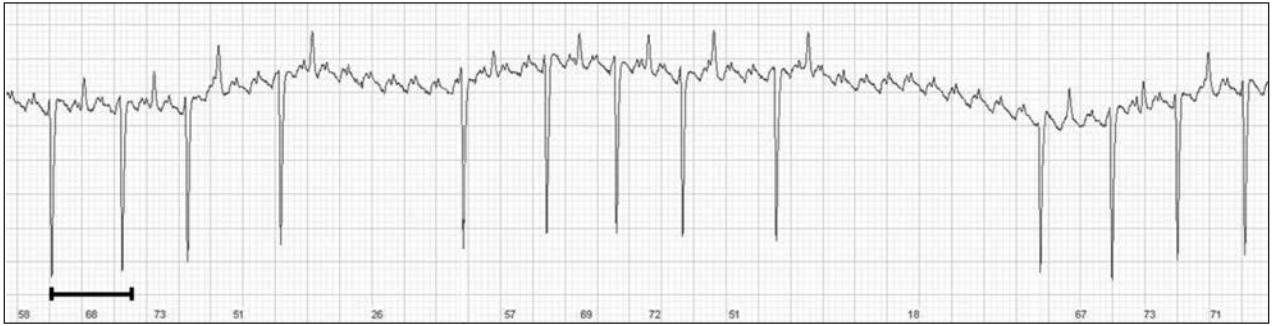


Figure 2—Modified base-apex ECG tracing obtained at rest from the same horse in Figure 1. In this tracing, the atrioventricular conduction rate is variable (from 3:1 to 12:1) leading to an irregularly irregular ventricular response. See Figure 1 for remainder of key.

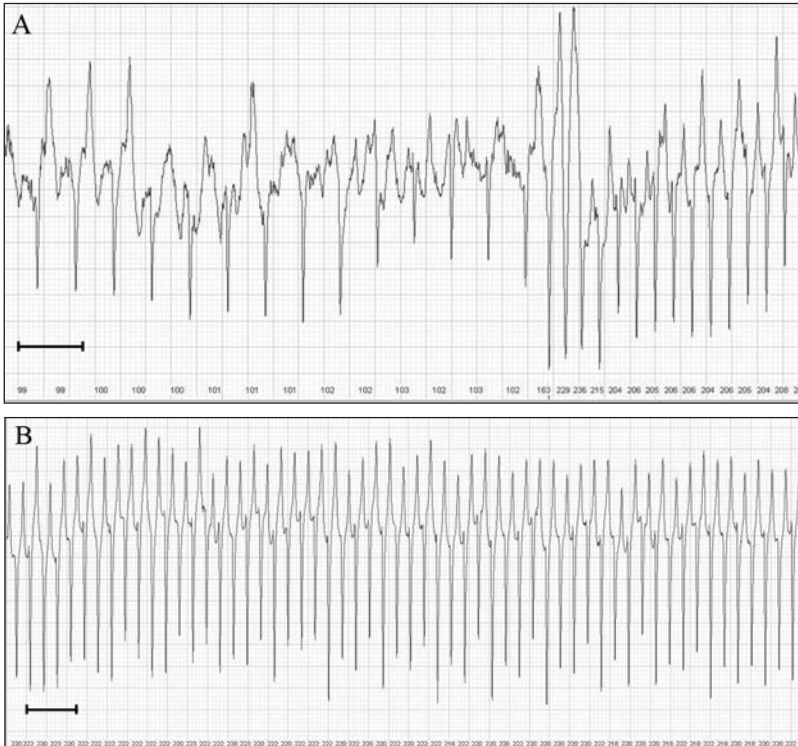


Figure 3—Modified base-apex ECG tracings obtained during exercise from the same horse in Figure 1. Notice the stepwise increase in heart rate with exercise that is interrupted by 3 ventricular premature contractions (A) followed by severe supraventricular tachycardia (B), during which each F wave is conducted to the ventricles (heart rate, 220 beats/min). See Figure 1 for remainder of key.

ted to the ventricles. Atrial and ventricular rhythm was regular and corresponded to a heart rate of 220 beats/min. The ECG pattern at rest subsequently reoccurred and coincided with the decrease in heart rate several minutes after exercise was stopped. These changes in heart rate occurred in a stepwise manner and were typically associated with an AV conduction rate of 1:1 (220 beats/min), 2:1 (110 beats/min), 3:1 (72 beats/min), 4:1 (54 beats/min), or other rate.

On the basis of the ECG and echocardiographic examination findings, diagnoses of atrial flutter, severe supraventricular tachycardia following exercise (a consequence of 1:1 conduction of F waves), and discrete tricuspid valve regurgitation were made. The owner was informed that the horse should be rested and that even light exercise was considered unsafe for horse or driver. Treatment options provided to the owner included medical treatment, atrial burst pacing, or transvenous electric cardioversion.^{1,2} Medical

treatment options included premedication IV or PO with digoxin, followed by administration of quinidine sulfate via nasogastric tube (according to the protocol for treatment of horses with atrial fibrillation), amiodarone hydrochloride (administered IV, although reported conversion rate is low), diltiazem hydrochloride (administered IV; at present, the safety and efficacy are not fully established), or flecainide acetate (administered IV, but associated with risk of fatal ventricular fibrillation).^{1,3} Given that medical treatment may be associated with risk of severe tachycardia in horses with atrial flutter and with risk of other adverse effects, transvenous electric cardioversion was suggested as the treatment of choice. Pharmacological and nonpharmacological treatment options for atrial flutter and atrial fibrillation are similar. Because of the slower and more organized atrial rate associated with atrial flutter, compared with that associated with atrial fibrillation, the success rate for transvenous electric cardioversion is considered greater in cases of atrial flutter.

Discussion

Horses with atrial flutter have clinical signs that are similar to those described for horses with atrial fibrillation. Although some horses with atrial flutter may be clinically unaffected, others may have sudden onset of poor performance or may develop signs of heart failure.⁴ Severity of clinical signs depends on the level of activity of the horse, rhythm disturbance, and presence of underlying disease. Cardiac auscultation in horses with atrial flutter may reveal a variable rhythm that appears fairly regular, regularly irregular, or irregularly irregular, depending on the rate of AV conduction at the time of examination. Because of this characteristic, detection of atrial flutter during routine clinical examination can be challenging. Extended periods of auscultation of a horse during different levels of excitement may be necessary to detect the arrhythmia. Conversely, the rhythm audible in horses with atrial fibrillation is typically irregularly irregular.⁴ Observation of a rapid jugular pulse in the lowest part of the jugular groove of a horse may help in the detection of atrial flutter because the rate of jugular pulsation is faster than the heart

rate, even in a horse for which the auscultated rhythm is perceived as regular. Occasionally, the rapid rate of atrial contractions may be detected on auscultation. Typically, ECG examination is necessary to confirm the diagnosis of atrial flutter.

In humans, several forms of atrial flutter have been characterized depending on the origin, direction, and rate of the flutter waves.⁵⁻⁷ Paroxysmal and chronic flutter in small animals have been reported.^{8,9} Atrial flutter may appear as an unstable arrhythmia in humans and small animals and can deteriorate to atrial fibrillation or even revert to sinus rhythm.⁸ Characteristics of atrial flutter in horses are still poorly understood because this arrhythmia is rarely detected.⁴ Atrial flutter is characterized by a single macro-reentrant wave of electrical activity with regular cycling through the atria (around an anatomic or functional boundary) and a variable transmission rate to the ventricles.^{5,6} As a result, a number of regularly spaced F waves (modified P waves) identical to each other with respect to wave morphology, cycle length, and polarity are seen preceding each QRS complex on the ECG tracing. The atrial rate is usually 180 to 250 depolarizations/min and results in the typical sawtooth ECG appearance of flutter waves indicative of recurrent atrial activation.¹⁰ The ECG appearance may be less characteristic and simply resemble the presence of multiple regularly spaced P waves for each QRS complex in horses in which the atrial rate is less high. In comparison, atrial fibrillation is the most frequent pathological arrhythmia in horses and a well-known cause of poor performance in athletic horses.⁴ Atrial fibrillation is characterized by multiple small reentrant circuits of electrical activity with a rapid and chaotic pattern. An undulating baseline of irregular f waves is observed between QRS complexes on the ECG tracing because no uniform atrial depolarization occurs.

Transmission at the AV node is often variable in animals with atrial flutter.⁵ The ventricular response rate is influenced by atrial input and AV node conduction.¹¹ Atrioventricular node conduction depends on the refractory period of the AV node (ie, a long refractory period decreases AV conduction rate), the level of autonomic tone (ie, a high sympathetic tone increases AV conduction rate), and the degree of concealed conduction within the node (ie, atrial impulses that penetrate the AV node without conduction to the ventricles can slow the conduction of subsequent atrial impulses). Ventricular rate changes (eg, during exercise) appear in a stepwise manner, associated with AV conduction rates of 1:1, 2:1, 3:1, and so forth. Small deviations in ventricular rate occur, probably because of altered sympathetic tone and intra-atrial conduction. Prognosis for animals with atrial flutter is comparable to that for animals with atrial fibrillation and depends on characteristics of the arrhythmia (eg, atrial rate, AV conduction, and changes in heart rate with exercise), the presence or absence of structural heart disease, and the level of activity required from the affected individuals. Exercise or stress often results in severe ventricular tachycardia because of 1:1 AV conduction. In human patients, 1:1 conduction of impulses between atria and ventricles may lead to a sudden, excessively high ventricular rate that induces clinical signs of syncope or collapse. Guidelines for human athletes with atrial flutter state that if no underlying heart disease is present and a ventricular rate that increases and decreases appropriately in relation to exercise is maintained, that person can participate in class IA competitive sports that require only low-intensity

exercise (ie, golf or billiards), with the warning that 1:1 AV conduction may occur.^{12,13} Horses with atrial flutter should be carefully examined to determine prognosis and treatment options. Evaluation should include ECG at rest and during exercise, echocardiography, and assessment of serum cardiac troponin I concentration to determine atrial and ventricular rates, AV conduction rate in response to exercise or stress, presence or absence of other arrhythmias, and presence or absence of underlying heart disease.

The horse of the present report was successfully treated via transvenous electric cardioversion at the University of Ghent, Belgium. A 24-hour ECG recording performed 6 days after cardioversion revealed 37 atrial premature depolarizations and 31 ventricular premature depolarizations. The horse was rested for 6 weeks and treated with decreasing oral doses of prednisolone. After 6 weeks, a 24-hour ECG recording revealed considerable decreases in the frequency of premature contractions (reduction to 2 atrial premature depolarizations and 10 ventricular premature depolarizations). Eight weeks after cardioversion, the horse was returned to training and regular monitoring of the heart rate by use of a heart rate monitor was recommended to assess changes in heart rate with exercise and training.

- a. Access Immunoassay Systems, Beckman Coulter Inc, Chaska, Minn.
- b. Mustang 2200, Graber AG, Fahrwangen, Switzerland.
- c. Televet 100, Rosch & Associates, Frankfurt am Main, Germany.

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