

# ECG of the Month

A 7-year-old Spanish Sport Horse was referred to the University of Cordoba for evaluation of an irregular heart beat that was identified during a prepurchase examination. At rest during the initial examination, heart rate was 35 beats/min and cardiac sounds were rhythmic; no cardiac murmurs were auscultated. Cardiac auscultation performed after exercise revealed a clearly audible arrhythmia during the recovery period. No other abnormalities were detected during the physical examination.

Diagnostic procedures were undertaken and included echocardiography and clinicopathologic analyses (hematologic and serum biochemical analyses). No abnormalities were detected during the ECG examination. Serum potassium concentration (3.4 mmol/L; reference range, 3.3 to 4.5 mmol/L) and creatine kinase activity (131 U/L; reference range, 60 to 350 U/L) were within reference ranges. The WBC count and serum fibrinogen concentration (markers of inflammation) were not high (5,900 cells/ $\mu$ L [reference range, 5,400 to 14,000 WBCs/ $\mu$ L] and 200 mg/dL [reference range, 130 to 400 mg/dL], respectively). Electrocardiographic recordings were obtained before and after exercise (Figure 1).

## ECG Interpretation

Initially, an ECG recording (base-apex lead) obtained at rest revealed a sinus arrhythmia with multiple uncondensed atrial premature complexes (APCs) super-

imposed on the ST segment of the previous depolarization (Figure 1). These APCs occurred every other beat (atrial bigeminy) with a fixed interval of 4.4 seconds at a ventricular rate of 30 beats/min and an atrial rate of 45 beats/min. None of the APCs resulted in ventricular depolarization. The timing and positive polarity of the P waves seemed to indicate that the origin of the premature complexes was atrial and probably ectopic. Although it was difficult to evaluate the shape of the premature P wave because of its superposition on the ST segment, it seemed to be narrower than the waves that appeared to be normal. A second ECG recording obtained later on the same day revealed a normal sinus rhythm with sporadic APCs that had the same characteristics.

An ECG recording was also obtained during recovery from exercise (mean heart rate, 70 beats/min). Atrial premature complexes were also evident in that recording, but in contrast to what was observed at rest, some of the APCs caused ventricular depolarization. The QRS complexes after APCs were slightly taller than the QRS complexes that were recorded after sinus complexes, indicative of aberrant atrioventricular (AV) conduction of the APCs. The abnormal QRS complexes evident after exercise could also be interpreted as ventricular premature complexes, which are frequently detected in exercising horses; however, the fact that they always appeared after an APC suggested conduction of the APCs. The P waves of the conducted and nonconducted APCs were dissimilar, but both were superimposed on the preceding ST segment. The APCs that were not conducted were followed by a long TP segment, which progressively became shorter, and by P waves with negative-positive morphology, unlike the normal bifid P wave (Figure 1).

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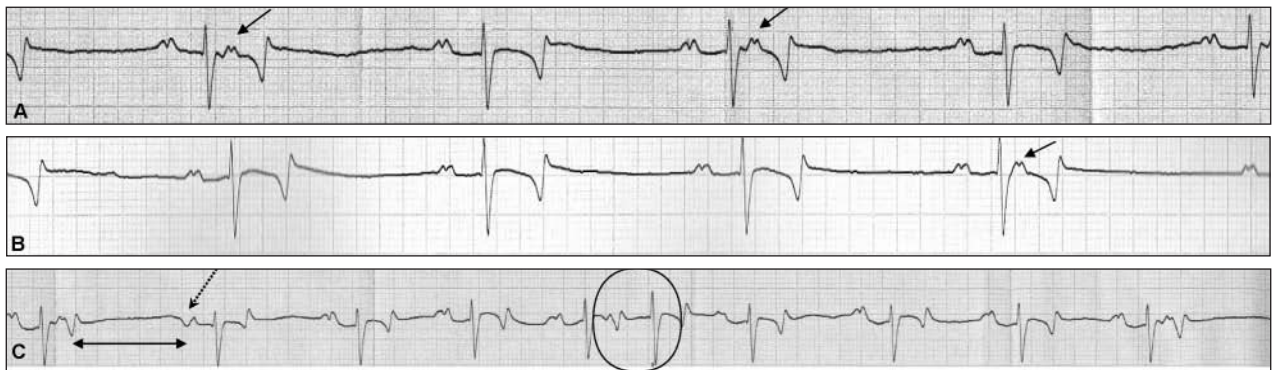


Figure 1—Base-apex ECG tracings obtained from a horse that was evaluated because of an irregular heart beat that was identified during a prepurchase examination. A—Initial tracing recorded during rest. Notice the uncondensed atrial premature complexes (APCs) superimposed on the ST segment of the previous depolarization (arrows) that appear in a bigeminal pattern. B—Tracing obtained during rest later the same day. Notice the sporadic APC superimposed on the ST segment of the previous depolarization (arrow). C—Tracing obtained after exercise. Notice the longer TP segment that follows the uncondensed APC (double-headed arrow) and the aberrant QRS complex following a conducted APC (circle). The P wave after the uncondensed APC has a negative-positive morphology (dashed arrow). For all ECG tracings, the left arm (+) electrode was positioned over the apex of the heart (sixth intercostal space on the left side, just above the point of the elbow) and the right arm (–) electrode was placed on the right jugular furrow at a position two-thirds along the length of the neck. The ground electrode was placed near the withers (highest point of the thoracic vertebrae). Paper speed = 25 mm/s; 1 cm = 1 mV.

## Discussion

Atrial arrhythmias are the most common rhythm disturbance in horses. The occurrence of APCs is considered a benign atrial arrhythmia. Although APCs can be clinically insignificant, they can also be associated with exercise intolerance or other signs of cardiac disease. Moreover, APCs may precede the development of atrial tachycardia, atrial flutter, or atrial fibrillation.

In horses, APCs may be overdiagnosed because of the fact that sinus bradycardia often results in variations in the P-P intervals, and it is not rare to detect a so-called wandering atrial pacemaker that gradually alters the P-wave morphology.

Exercise or excitement abolishes such physiologic rhythms.<sup>1</sup> However, in the horse of this report, exercise did not abolish the arrhythmia but appeared to exacerbate the occurrence of APCs. Atrial premature complexes may originate in the sinoatrial node or in ectopic locations. In the horse of this report, all APCs recorded at rest had a bifid morphology, which suggested an origin close to the sinoatrial node. However, after exercise, some negative-positive P waves were observed, which indicated the existence of a second focus of depolarization in the caudal right atrium near the coronary sinus.<sup>2</sup>

Atrial arrhythmias often develop as functional disorders without the presence of a structural cardiac lesion. Specifically in horses, APCs can be caused by autonomic imbalance (including high sympathetic activity), hypokalemia,  $\beta$ -adrenoreceptor agonists, infections, anemia, and colic. In addition, atrial rhythm disturbances are common in horses with structural lesions of the valves, myocardium, or pericardium. The horse of this report had no echocardiographic evidence of organic heart disease. No abnormalities in serum electrolyte concentrations that could cause changes in myocardial membrane potential and refractory period were identified. Although more specific tests (eg, measurement of plasma cardiac troponin I concentration) were not performed, no signs of inflammatory myocardial disease were detected via routine clinicopathologic analyses. Thus, the underlying cause of the APCs was not determined.

The case reported here had some unique characteristics that emphasize the importance of evaluating arrhythmias under different situations. It is interesting to note that the prevalence of APCs during the day of evaluation was quite variable, ranging from atrial bigemy to almost complete absence of APCs. Given the variability in the ECG findings during the day, the use of a 24-hour ECG (Holter) monitor in the horse of this report might have allowed better characterization of the arrhythmia. Because none of the APCs were conducted at rest and the P waves were superimposed on the ST segment, the arrhythmia was not detectable during auscultation at rest; however, after exercise, it was clearly audible. During auscultation of APCs, a regular sinus rhythm that is interrupted by an obvious premature beat is typically detected.

After exercise, some of the APCs were conducted to the ventricles in what seemed to be an aberrant manner. Aberrant ventricular conduction was identified by a transient increase in height of the QRS complex. Aberrancy may be physiologic. Premature atrial depolarizations that occur early are conducted with aberrancy or are blocked, even if the specialized conduction system is functionally normal. Thus, supraventricular tachycardias may result in degrees of AV

block in the absence of conduction system disease.<sup>3</sup> In the horse of this report, all of the APCs were blocked at rest, probably because they occurred during the refractory period of the AV node. This occurs when there are markedly premature APCs and the P waves become superimposed on the preceding T wave (and are thus masked), giving the appearance of sinoatrial block.<sup>4</sup> Aberrant conduction sometimes occurs because the ventricles are not completely repolarized and the premature APC encounters the AV node when it is still partially refractory from the preceding sinus beat. Alternatively, aberrant conduction may occur if depolarization is triggered by a supraventricular beat that enters the AV node at a different point than that targeted by a beat of sinus origin.

In the horse of this report, the main objective of the evaluation was not only to diagnose and treat the arrhythmia but also to attempt to evaluate its impact on activities that the horse was expected to perform in the future. Generally, if there is no evidence of organic heart disease, as determined in the horse of this report, then treatment is not considered necessary unless the APCs trigger episodes of supraventricular tachycardia.<sup>4</sup> Atrial premature complexes are more likely to be clinically important if they occur frequently at rest. Supraventricular premature contractions are considered benign if the frequency of occurrence is < 10% because the contraction of the atria is thought to have little impact on the cardiac output, except at maximal cardiac performance.<sup>5</sup> Other circumstances in which APCs may have greater clinical importance include their association with nonsustained or sustained runs of atrial tachycardia, if they are related to poor performance (other causes excluded), when they precipitate paroxysmal atrial flutter or fibrillation, or if they develop along with other signs of cardiac disease.

Only the frequency of the APCs was of concern for the horse of this report with regard to possible development of atrial fibrillation. However, the fact that the arrhythmia was not associated with poor performance or other signs of cardiac disease did not support the need for treatment. Thus, no treatment was recommended. Consultation with the owner 6 months later revealed that the horse was not sold and had continued to be used successfully in 3-day events.

Although the ECG abnormality detected in the horse did not seem to have clinical relevance, the presence of APCs should be interpreted as an abnormal and pathological finding, which may progress to more severe arrhythmias (eg, atrial fibrillation). A horse with an ECG abnormality may nevertheless perform well, and predictions regarding its future athletic career are difficult to make; this is a particular problem when such ECG abnormalities are detected during prepurchase examinations.

## References

1. Physick-Sheard P. Pathophysiology and principles of therapy of the cardiovascular system. In: Colahan PT, Merritt AM, Moore JN, et al, eds. *Equine medicine and surgery*. 5th ed. St Louis: Mosby Inc, 1999;337–380.
2. Bonagura JD, Reef V. Disorders of the cardiovascular system. In: Reed SM, Bayly WM, Sellon DC, eds. *Equine internal medicine*. 2nd ed. St Louis: Saunders, 2004;355–459.
3. Achen SE, Miller MW. ECG of the month. Atrial premature complex. *J Am Vet Med Assoc* 2008;233:561–562.
4. Abbott JA. ECG of the month. Sinus arrhythmia with occasional premature atrial complexes. *J Am Vet Med Assoc* 1997;210:900–901.
5. Risberg AI, Slack J, Semrad SD, et al. ECG of the month. Sinus rhythm with supraventricular premature complexes. *J Am Vet Med Assoc* 2005;226:527–529.