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Cardiac Arrhythmias in Large Animal Species: Is it Worth Worrying About?

By Fernando J. Marqués, DVM, Diplomate ACVIM

Cardiac arrhythmias are often discovered as an incidental finding during regular physical examinations; for example, with prepurchase examinations in the case of sport horses, during investigations of unrelated diseases, or while exploring a suspected primary cardiac problem. The veterinarian must determine whether a cardiac arrhythmia is benign or clinically significant. This issue of *Large Animal Veterinary Rounds* discusses the principles of cardiac examination, diagnosis, and the clinical significance of the most common cardiac arrhythmias in large animals.

Cardiac arrhythmias are abnormalities of the heart rate, rhythm, or conduction pattern due to disturbances of impulse generation, impulse conduction, or a combination of both. Arrhythmias are more common in horses than in other large animal species. Almost 25% of horses with no evidence of heart disease may have cardiac arrhythmias that can be found on regular physical examination or with an electrocardiogram (ECG).¹ At rest, normal horses may have arrhythmias that are considered "benign" or "physiologic" and thought to be caused by an increase in vagal (parasympathetic) tone. Based on 24-hour ECG monitoring of healthy horses, 44% had a second-degree atrioventricular (AV) block, 10% had sinus arrhythmia, 3% had atrial block, 27% had occasional extrasystoles, and 27% had ventricular arrhythmias.¹ In horses with signs of cardiac disease, 40% may present with cardiac arrhythmias.²⁻⁴ Common causes of arrhythmias in horses include excitement, fever, toxemia, colic, electrolyte imbalance, congenital defects, myocarditis, and valvular heart disease.⁵

Cattle do not have physiologic or benign arrhythmias, but seem prone to develop cardiac arrhythmias (eg, atrial premature contractions and atrial fibrillation) in the presence of gastrointestinal (GI) disorders. Other common causes of cardiac arrhythmias in cattle include lymphosarcoma, valvular disease, myocardial damage, cor pulmonale secondary to pulmonary hypertension, pericarditis, foot rot, fever, toxemia, electrolyte abnormalities, and myocarditis.

Cardiac arrhythmias in cattle and horses are seldom a consequence of cardiomyopathy, aortic root rupture, or aortic-cardiac fistula.⁵

Physical and cardiac examination

A good general examination is the key element of the cardiovascular (CV) examination.^{3,6} Findings on physical examination that require attention when observed in combination with cardiac arrhythmias include presence of edema, jugular or milk-vein distension, abnormalities in pulse quality and character, abnormal lung sounds, or altered capillary refill time.^{4,5} Cardiac arrhythmias should always be evaluated in the context of all findings from physical examinations and medical/health history.

Cardiac auscultation

Cardiac auscultation requires an understanding of cardiac conduction, dynamics, and cycles, in addition to valve location relative to external landmarks. Auscultation should be performed on both sides of the thorax over the 3rd to 5th intercostal spaces (under the muscles of the shoulder). Cardiac sounds are caused by changes in blood flow, not valve closure; however, sounds are often described as *associated* with valve closure. Four heart sounds can be heard in normal horses and cattle:³⁻⁷

- S1 ("lub"): associated with closure of the AV valves
- S2 ("dub"): associated with closure of the semilunar valves
- S3: associated with the elastic recoil at end-diastole
- S4: associated with atrial contraction



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Cardiac arrhythmias should be considered when auscultation reveals evidence of:

- tachycardia or bradycardia
- irregular rhythm
- long pauses
- extra sounds

Cardiac electrical activity and heart function

One basic concept is the association between the electrical activity of the myocardium and rhythmic pumping of the heart. The electrical activity of the heart results in a contraction of the heart muscle (myocardium); therefore, through knowing and understanding the normal and abnormal electrical impulse pathway and conduction patterns, it is possible to assess cardiac function. The ECG records electrical activity of the contracting myocardium and provides useful information about cardiac function, heart rate, and rhythm.⁶⁻⁹

During the resting state, the interior of the normal heart muscle cell (myocyte) is negatively charged and the outside cell surface is positively charged. When a wave of depolarization reaches the cell, the interior of the cell becomes positive and the cell contracts. This electrical change is carried out by fast-moving sodium ions. Contraction of the heart can be visualized as an advancing wave of positive charges going through the interior of the cells (depolarization), causing progressive contractions of the myocardium.⁹ Following depolarization, the myocytes re-acquire their resting state due to a controlled outflow of potassium ions (repolarization), causing the interior of the cells to again become negatively charged.⁶⁻⁹

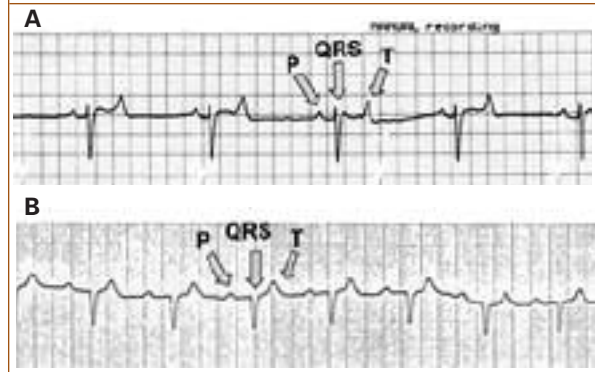
The sinoatrial (SA) node, located in the right atrium, is the dominant pacemaker and generates a spontaneous wave of depolarization (automaticity) that spreads out to the atria and reaches the AV node. The AV node is the conduction pathway between the atria and the ventricles. The impulse conduction through the AV node is slowed, due to slow-moving calcium ions, which allows the blood coming from the atria to flow into the ventricles. The AV node, in turn, conducts the electric impulse through the rapidly conducting Purkinje fibres to both ventricles.⁹ As opposed to humans and small animals, Purkinje fibres in large animals penetrate very deeply; therefore, ventricular activation is explosive and occurs from multiple sites. This sequence of events ultimately results in a normal sinus rhythm.^{4,6-8}

The autonomic nervous system moderates the function of the heart. The sympathetic system activates cardiac β_1 adrenergic receptors to produce excitatory effects; these effects increase the rate of SA-node pacing, the rate of conduction, the force of contraction, and the irritability of foci in the heart. The parasympathetic system activates cholinergic receptors in the heart to produce inhibitory effects, thus decreasing the rate of SA-node pacing, the conduction rate and force of contraction, and the irritability of atrial and junctional foci.^{4,9,10} Any disturbance in impulse generation, impulse conduction, or a combination of both can induce a cardiac arrhythmia.⁵

Clinical electrocardiography

In large animals, ECGs are used primarily to identify and characterize cardiac arrhythmias and conduction disturbances.^{3,5} Electrodes placed on the body surface record changes in membrane potentials and direction (electrical activity) of the heart cells. Whenever a wave of depolarization (positive charges) moves

Figure 1: ECG tracing from a normal adult horse (A) and a normal adult Holstein cow (B) with sinus rhythm. Both are recorded at 25mm/sec. paper speed on a base-apex lead system.



toward a positive electrode placed on the skin of the patient, there is a positive (upward) deflection recorded on the paper of the ECG machine. If the wave of depolarization moves towards a negative electrode, a negative (downward) deflection is recorded on the ECG.⁹ The electrical activity of the heart can be recorded from different angles, based on the location of the electrodes on the body surface. Different views of the electrical activity of the heart can be taken using different leads (up to 12 leads can be used).⁷

The “base-apex” lead system is commonly used in large animals because it records consistent ECG tracings regardless of animal size and breed; in addition, the tracing produces good wave amplitude and is minimally affected by slight movements of the animal or its skin during the recording.^{3,4,6,8} For the base-apex lead system, the positive electrode (LL – lead II) is placed on the left side of the thorax over the heart area at the level of the elbow and over the 5th intercostal space. The negative electrode (RA – lead II) is placed on the right jugular furrow two-thirds of the distance down the neck. The “ground” or neutral electrode (LA) is placed on any location away from the heart, most commonly on the left side of the neck.^{4,6,8} Applying alcohol to wet the skin where the electrodes are attached improves the quality of the ECG recording.

In the base-apex lead, the first positive deflection in the ECG tracing is the P wave that corresponds to atrial depolarization (and subsequent contraction). In horses, the P wave is most frequently notched, whereas, it is not notched in healthy cows. Sometimes a T_a wave (negative deflection) can be seen following the P wave in normal horses and corresponds to atrial repolarization. After the P wave or the T_a wave, the next complex of waves recorded by the ECG is the QRS complex. In the base-apex lead, the QRS is usually registered as a small positive deflection followed by a large negative deflection (“rS”), which corresponds to ventricular depolarization. The T wave is extremely variable in horses and ruminants, and can be either uniphasic or biphasic and either positive or negative. The T wave corresponds to ventricular repolarization (Figure 1).^{3,4,8} ECGs are usually recorded at 25-mm/second (sec) paper speed and 1 mv/cm for the amplitude settings.

The relationship between the cardiac cycle and the ECG waves are:

- P wave represents atrial depolarization and subsequent atrial contraction

- P-R interval is the time between the beginning of atrial depolarization and the beginning of ventricular depolarization
- QRS complex represents ventricular depolarization before contraction
- Q-T interval (from the beginning of the Q or R wave to the end of the T wave) corresponds to ventricular contraction or systole
- T wave represents ventricular repolarization and occurs slightly before the end of ventricular contraction.

Therefore, the P wave and the QRS complex are depolarization waves and the T wave is known as a repolarization wave.^{6,8,9}

In contrast to humans and small animals, an ECG provides little if any information about chamber enlargement in large animals, but it is very useful for determining heart rate, rhythm, and conduction disturbances.^{5,8}

ECG interpretation

Proper interpretation of an ECG and identification of cardiac arrhythmias require a systematic approach that includes a consideration of several aspects.⁸

Heart rate calculation

Calculating the heart rate is the very first step when assessing an ECG tracing. The SA node is responsible for establishing and maintaining this normal and regular heart pacing, ie, the **sinus rhythm**. When the SA node paces the heart at a slower rate, it is termed **sinus bradycardia** and if the heart is paced at a faster rate, it is called **sinus tachycardia**.⁹

Using a “standard” paper speed of 25 mm/sec, each small box represents 0.04 sec and each large box represents 0.2 sec. To determine the heart rate, it is customary to count the number of complexes in 6 seconds and multiply by 10.^{6,8,9} Some ECG recording papers have small marks on the top margin with the space between each mark indicating a 3-sec interval.

Some areas in the heart (eg, atrial foci, junctional foci, and ventricular foci) are capable of taking over the SA node pacemaker function in case of failure, these areas are called **automaticity foci** (potential pacemakers). These automaticity foci have their own inherent pacemaking rate that is slower than the SA node rate. In the normal heart, all automaticity foci are overdrive-suppressed by the SA node. The automaticity centres in the atria have an inherently faster rate than the junctional foci and, in turn, these foci have a faster inherent rate than the ventricular foci.⁹

Recognizing an irregular rhythm

It is important to determine if the P-P and R-R intervals are regular and whether there is an occasional period of irregularity, as in cases of atrial or ventricular premature beats, or if an irregular rhythm is continuously present, as observed in cases of atrial fibrillation. It is also important to verify that the atrial and ventricular rates are identical.⁸ P waves should always be present and precede every QRS complex. For example, in the case of ventricular premature contractions, there is no P wave antecedent the QRS complex.⁸

All P waves should be followed by a QRS complex. In the case of a second-degree AV block, a QRS complex does not follow the P wave because the P wave stimulus is blocked in the AV node.⁸ Assuring that P waves and QRS complexes are normal in appearance, shape, and timing is important to verify when

analyzing the ECG.⁸ P waves, QRS complexes, and the duration of P-R and Q-T intervals should be within normal limits.⁸

Cardiac arrhythmias

The two primary concerns for both the owner of the animal and the clinician are whether a cardiac arrhythmia may have hemodynamic implications and/or its potential for triggering further cardiac complications (eg, ventricular fibrillation) and death.^{3-6,8} Cardiac arrhythmias are classified based on the anatomic portion of the heart in which they originate, the rate, the mechanism of impulse formation, and conduction patterns.⁴ In horses, some arrhythmias are common and considered a normal finding because of the high parasympathetic tone (vagal tone) in this species.^{4,6,8} One important goal for the clinician is to determine whether the arrhythmia is occurring due to a normal variation in autonomic tone (ie, “physiologic arrhythmias”), due to a primary cardiac disease, or secondary to an underlying non-cardiac pathology.³ The following are the most common cardiac arrhythmias in large animal species.

Sinus tachycardia

Tachycardia simply means fast heart rate, whether physiological or pathological.⁹ There is discrepancy in the literature regarding normal values for heart rate in large animal species, but these ranges are widely accepted:^{5,8}

- Cattle: 49–84 beats/minute
- Horses: 26–50 beats/minute and foals: 60–80 beats/minute
- Exercise maximum heart rate in horses: 240 beats/minute

Tachycardia can be induced by an increase in body temperature, either due to hyperthermia or fever, sympathetic stimulation of the heart, or toxic conditions of the heart. In humans, reports suggest that for each degree Celsius increase in body temperature (up to a body temperature of about 40.5° C), the heart rate increases about 18 beats/minute.^{9,11}

Sinus bradycardia

Bradycardia means slow heart rate, whether physiological or pathological.⁹ At rest, normal horses may have a vagal-induced sinus bradycardia that is considered a “physiologic” or “benign” rhythm. Sinus bradycardia may also occur in anesthetized or severely ill patients.^{4,8} In some cases, cardiac output may decrease radically, inducing significant hypotension and requiring treatment with catecholamines or anticholinergic drugs.

Hypoxia, anesthetic drugs, and traction on an abdominal viscus can depress the SA node function and lead to ectopic rhythms.⁴ In cattle, lack of feed intake, specifically holding off feeding for 12–48 hours, is commonly associated with sinus bradycardia.¹²

Sinus arrhythmia

Sinus arrhythmias may occur as a consequence of numerous circulatory conditions altering the input from the sympathetic and parasympathetic system to the SA node.¹⁰ Sinus arrhythmia can originate from a normal physiological mechanism driven by the autonomic nervous system, which causes a slight change in the heart rate related to phases of respiration; ie, there is a minimal decrease in heart rate during expiration and a minimal increase in heart rate during inspiration.^{9,10} Sinus arrhythmia is also commonly observed associated with lack of feed intake in cattle.^{5,12}

Atrial premature contractions

In general, a premature contraction (atrial or ventricular) is also called **premature** or **ectopic beat**. A premature contraction is caused by an irritable automaticity focus in the heart that spontaneously sends an abnormal impulse earlier than expected in the cardiac cycle.⁹ In the case of an atrial premature contraction, the automaticity focus is located in the atria and, depending on its specific location within the atria, it may generate a slightly different shaped P wave (sometimes even buried in the previous T wave) on the ECG.^{4,6,7,9} If the stimulus from the ectopic automaticity focus located in the atria reaches and depolarizes the SA node (the dominant centre of automaticity), it will cause the SA node to reset and it will resume pacing 1-cycle length from the premature stimulus. The SA node will not reset if the stimulus from the atrial automaticity focus does not depolarize the SA node.⁹

When reaching the AV node, the premature stimulus is conducted to the ventricles and a premature QRS complex is recorded on the ECG, unless the atrial premature stimulus finds the AV node in a refractory state (not fully repolarized) and the stimulus is not further conducted to the ventricles. In this case, a premature P wave with no associated QRS complex is recorded on the ECG strip.^{6,8,9}

The clinical significance of atrial premature contractions depends on the frequency of occurrence, the presence of clinical signs related to cardiac disease or poor performance, or associations with atrial tachycardia or atrial flutter. During a 24-hour period, according to one study, occasional atrial premature depolarizations can be detected in otherwise clinically normal horses.¹

Supraventricular premature contractions usually occur in the immediate postexercise period; therefore, an ECG during and after exercise is recommended for exploring this condition in horses.^{4,6,8}

Atrial tachycardia

Atrial tachycardia is a tachyarrhythmia infrequent in horses, but more common in cattle affected with GI disease. It is characterized by a sequence (>4) of premature atrial contractions in a row;⁸ this sequence is caused by an extremely irritable atrial automaticity focus that suddenly begins pacing at a higher rate than the SA node.⁹

Atrial fibrillation

Atrial fibrillation (AF) is associated with poor performance and exercise intolerance and is the most common cardiac arrhythmia in horses.⁶⁻⁸ Horses with AF can be asymptomatic at rest and become symptomatic under exercise conditions. AF is frequently found in cattle with GI disease, but it has also been associated with pneumonia and foot rot. Other common clinical signs associated with AF in cattle are anorexia and decreased milk production.⁸

AF is characterized by a completely erratic rhythm due to the rapid and continuous firing of impulses from multiple, very irritable, automaticity foci within the atria that elicit an irregular ventricular response. These atrial automaticity foci are not vulnerable to overdrive suppression by other foci (entrance block); therefore, all the foci pace at their own rhythm, simultaneously.⁹ Multiple re-entrant

circuits are set and are ultimately responsible for sustaining atrial fibrillation.^{10,11,13} The AV node is randomly and irregularly stimulated by one of these abnormal atrial impulses, leading to an irregular ventricular rhythm.

AF is commonly caused by atrial enlargement due to valvular disease (eg, mitral or tricuspid insufficiency) or due to ventricular failure resulting in a damming of blood in the atria. Atrial enlargement provides an excellent background for slow- and long-conduction pathways that predispose to AF.^{4,6-8}

Many horses with AF have no evidence of underlying cardiac disease. Some consider that otherwise normal horses may have increased parasympathetic (vagal) tone and sufficient atrial mass that could predispose to the development and maintenance of AF.^{6,8} Electrolyte imbalances are also associated with AF; for example, paroxysmal AF is associated with transient potassium depletion in racehorses and treated with furosemide or sodium bicarbonate.^{4,8}

Paroxysmal AF can spontaneously resolve and convert to a normal sinus rhythm in cattle and horses with no atrial enlargement after correction of the underlying disease.⁸ Spontaneous conversions to sinus rhythm usually occur within 24–48 hours after the onset of paroxysmal AF in horses.^{6,8} Cattle usually convert to sinus rhythm spontaneously within 4–5 days after correction of the underlying problem (eg, GI disease and/or electrolyte and acid-base abnormalities).⁸

Animals with AF have an irregular and erratic cardiac rhythm with no underlying regular pattern and no auscultable fourth heart sound.^{4,6-8} The heart rate is variable and, at rest, horses with AF usually have a heart rate within normal limits or slightly increased, unless significant valvular or myocardial disease is present. During exercise, horses with AF usually have a higher heart rate than expected for the level of exercise. The heart rate in cattle with AF generally depends on the underlying problem. During AF, the intensity of the arterial pulse is variable and pulse deficits can occur.⁸

The ECG reveals marked irregular R–R intervals with QRS complexes of normal polarity and morphology, but a slight variation in appearance can occur between complexes (Figure 2). P waves are absent and there is a fine undulated baseline (“F waves”) instead.^{4,6,8}

Before treating AF and converting to a normal sinus rhythm, cardiac disease and other underlying noncardiac pathologies should be diagnosed, addressed, and corrected whenever possible. An ECG and an echocardiogram are indicated to rule out cardiac disease. A complete blood count, biochemistry panel, and blood-gas analysis are useful for detecting possible underlying disease processes and any electrolyte and/or acid-base abnormalities. Those abnormalities should be corrected before attempting medical treatment for AF.⁸ Sustained and untreated AF can lead to cardiac diseases such as atrial dilation, AV valve regurgitation, or myocardial fibrosis.

Conversion of AF to a normal sinus rhythm in horses and cattle is commonly and successfully achieved using quinidine, a Class Ia sodium-channel blocker. Pretreatment with digoxin is indicated in animals with chronic AF, atrial enlargement, or increased ventricular rate. Physical exami-

Figure 2: A) Atrial fibrillation in a 2-week-old calf with diarrhea and hyperkalemia. Note the absence of P waves and the irregular rhythm with no underlying regularity. B) Atrial fibrillation in an adult horse. P waves are absent and there is an undulated baseline or “F waves” (arrow) instead. Note the lack of underlying regularity.



nation, cardiac auscultation, and ECGs should be used to carefully monitor patients undergoing medical treatment with quinidine until conversion to sinus rhythm occurs. For complete information on protocols in treating AF, the reader can refer to these cited sources.^{3,6-8}

Ventricular premature contractions

The pathophysiologic principles previously explained for the genesis of atrial premature contractions also apply to ventricular premature contractions. In ventricular premature contractions, the automaticity focus is located in the ventricle, most commonly generating a bizarre QRS complex on the ECG. This QRS complex occurs early in the cardiac cycle and is not preceded by a P wave. When multiple automaticity foci are present in the ventricle, QRS complexes with different shapes (polymorphic) are seen on the ECG strip.⁹ In a 24-hour period, according to one study, continuous ECG detects occasional ventricular premature contractions in otherwise normal horses.¹ If ventricular premature contractions are frequent, polymorphic, or detected during exercise, they are considered abnormal.⁶

Ventricular tachycardia

A series of four or more premature ventricular contractions is diagnostic of ventricular tachycardia (VT; Figure 3).³ VT is thought to be caused by an abnormally irritable automaticity focus in the ventricle⁹ and the presence of a re-entrant circuit movement pathway (ventricular re-entry) that creates a self-re-excitation conduction pattern.^{10,11,13} Ventricular re-entry is an important mechanism for the development of sustained VT.^{8,11} Alterations in the autonomic input to the heart are also thought to play a role in the genesis of VT. Patients with myocarditis, myocardial fibrosis, bacterial endocarditis, hypoxia, ischemia, sepsis, and

Figure 3: Ventricular tachycardia in a 10-year-old horse.

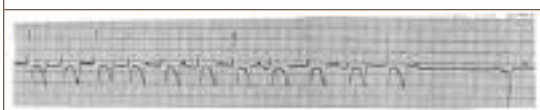
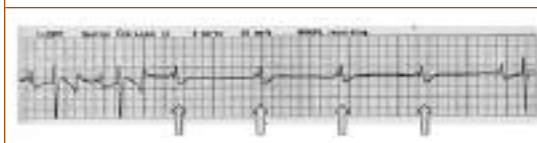


Figure 4: Second degree AV block. Note the presence of non-conducted P waves (block arrows).



electrolyte imbalances may develop ventricular premature depolarizations and eventually VT.^{6,8} Clinical signs depend on the underlying disease and on the characteristics of VT, such as chronicity, uniform versus polymorphic type, and ventricular rate. The clinical presentation of large animals can vary from asymptomatic at rest to signs consistent with congestive heart failure. Common clinical signs in horses include exercise intolerance, weakness, syncope, and respiratory distress. VT in cattle is commonly associated with anorexia, decreased milk production, sepsis, and endotoxemia.⁸

Treatment of VT is indicated when the animal is hemodynamically unstable, reveals sustained VT with heart rates >120 beats/min in horses and >140 beats/min in cattle, or when multiform VT and R on T complexes (a premature T-wave complex) are identified on the ECG. In these clinical scenarios, if VT is left untreated, congestive heart failure may develop in days or weeks. Antiarrhythmic drugs such as lidocaine, quinidine, and bretylium tosylate can be used for treatment.^{6,8}

Ventricular fibrillation

Ventricular fibrillation constitutes a serious emergency; this arrhythmia is ultimately fatal, if uncorrected, since there is no coordinated ventricular contraction. Ventricular fibrillation is caused by a series of very irritable parasystolic ventricular foci that pace very rapidly and suffer from entrance block, as well as the development of re-entrant circuit movement pathway (ventricular re-entry).^{9,10} It is very easy to discern on ECG due to its completely erratic appearance and the lack of a predictable pattern or recognizable normal waves.⁹

Conduction disturbances

First-degree AV block is characterized by a delay in the impulse conduction through the AV node; therefore a prolongation in the PR interval is seen on the ECG.⁹

Second-degree AV block is the most common arrhythmia detected in horses and is even more common in fit horses.^{6,8} It is usually driven by an increased vagal tone (parasympathetic input), which is considered normal in horses; those with second-degree AV block usually have low or normal heart rates. Increasing the sympathetic tone by means of exercise or excitement, or decreasing the parasympathetic tone using vagolytic drugs should resolve this problem. This type of vagal-induced arrhythmia is considered benign or physiologic.^{7,8} Upon cardiac auscultation, a low to normal heart rate is found and there are occasional pauses in the normal cardiac cycle during which only an isolated S₄ (atrial contraction) is auscultated.³ The ECG reveals a slow to normal heart rate with occasional P waves that are not followed by a QRS complex (Figure 4). There are two types of second-degree AV block:

- Wenckebach (formerly called Type I) is the most common and is associated with increased parasympathic tone where the PR interval gradually lengthens until the last P wave of the series fails to conduct to the ventricles and, therefore, no QRS complex is seen.
- Mobitz (formerly called Type II) is characterized by fixed PR intervals and a series of P waves that fail to conduct to the ventricles;⁹ this is potentially indicative of cardiac pathology.

Third-degree AV block is rare in large animals.⁶ This type of block represents a complete AV block of the electric impulse to the ventricles and, therefore, an automaticity focus below the block escapes and becomes the “ventricular pacemaker” with its own inherent rate, which is slower than the atrial rate. This phenomenon can be observed on the ECG as P waves dissociated from the QRS complexes. The rate of ventricular beat is slower than the atrial rhythm.^{9,10} There is no effective antiarrhythmic drug to treat this condition and the implantation of a pacemaker would be the definitive treatment.^{3,6}

Summary

Cardiac arrhythmias are common in large animals and probably even more common in horses than in other species.^{1,8} The crucial steps are to recognize the type of arrhythmia, define its significance, and determine whether it is due to a primary cardiac problem, secondary to an underlying noncardiac disease, or physiologic.³ A thorough and systematic physical examination, including clinical pathologies, electrocardiography, and echocardiography should be considered essential tools in achieving a definitive diagnosis and determining a therapeutic approach.^{3,4,6,7} For horses, an evaluation of the animal during and immediately after exercise is also necessary. Cardiac arrhythmias that warrant medical antiarrhythmic therapy relate to decreased cardiac output and hemodynamic instability, and predispose the animal to developing more severe and life-threatening cardiac arrhythmias.³

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