Proceedings of the 13th International Congress of the World Equine Veterinary Association WEVA

October 3 - 5, 2013
Budapest, Hungary

Reprinted in IVIS with the Permission of the WEVA Organizers
Introduction to Electrocardiography in the Horse and Recognition of Common Abnormalities

Michael Hewetson BSc BVSc Dipl.ECEIM
Department of equine and small animal medicine, Faculty of Veterinary medicine, University of Helsinki, Finland

Summary

Like murmurs, cardiac arrhythmias are common in the horse and a basic understanding of the electrophysiology of the heart will enable the clinician to have a better understanding of how arrhythmias develop and how they can be recognized and treated. Once an arrhythmia has been identified on auscultation, the crucial steps are to identify the type of arrhythmia, interpret its clinical significance, and then determine whether it is due to a primary cardiac problem, or secondary to non-cardiac disease. An arrhythmia may not always be clinical significant, however investigation is always necessary to rule out an underlying disease process. If an arrhythmia is suspected on cardiac auscultation, an electrocardiogram (ECG) should be performed in order to identify the type of arrhythmia. In the horse, an ECG can be obtained using a convenient base apex lead system that is easy to use and well tolerated in most horses. When interpreting the ECG, the cardiac rate and rhythm should be evaluated and the ECG recording should be scrutinized for the presence of abnormal complexes. This can be facilitated by asking five simple questions every time you examine an ECG. Once you have identified the arrhythmia, its significance should be determined by considering its effect on cardiac function. This article describes common abnormalities that may be indentified on a resting ECG in the horse and discusses how these may be interpreted.

I. Introduction

Normal electrical excitation of myocardial cells and propagation of an action potential via the specialized conduction pathways of the heart is essential for coordinated myocardial contraction and relaxation, and forms the basis of normal cardiac function. The depolarization and repolarization of these excitable cells can be detected by placing electrodes at various points on the surface of the body and recording a surface electrocardiogram. The ECG records the potential difference between electrodes and it allows us to detect changes in the electrical field which is built up around the heart during depolarization and repolarization of the atrial and ventricular myocytes. Depolarization and repolarization of the atrial and ventricular myocytes cause characteristic deflections or waveforms on the ECG, and careful examination of the timing of the waves, the relationship between the different waves, and the morphology and duration of the complexes and intervals enables us to deduce the origin and conduction pathway of the impulse. With experience, specific changes in the ECG can be used to identify rhythm disturbances that may be associated with underlying cardiac diseases and secondary non-cardiac diseases that may influence the electrical activity of the heart (e.g. electrolyte disturbances, acidosis and hypoxia).

II. Electrical events that generate the electrocardiogram

Most cells in the body have an electrical potential difference across their cell membranes caused by differences in the concentrations of positively and negatively charged ions on either side of the cell membrane. This potential difference is normally negative and is in the order of -90 mV. Specialized ion pumps that remove sodium from the cell and replace it with potassium maintain this negative potential charge. A unique aspect of myocardial cells is that they are able to rapidly reverse this potential difference in response to signals from neighboring cells (excitable cells), and in doing so, generate an action potential. Contraction of the atrial and ventricular myocytes occurs in response to generation of this action potential via delivery of calcium to intracellular contractile units. In order for coordinated myocardial contraction and relaxation to occur, an orderly sequence of action potential generation and propagation through the atrium and ventricle myocardium is essential. Some specialized myocytes situated in the sinoatrial (SA) node, the atrioventricular (AV) node, and the specialized conduction pathways of the His Purkinje system have an unstable resting potential (-60 mV) that drifts towards a positive potential. When this potential reaches threshold, the cells automatically depolarize (automaticity), giving rise to a spontaneous action potential. These spontaneous action potentials set the pace of myocardial contraction and are referred to as ‘pacemaker’ cells. Once generated, these action potentials are then propagated throughout the atrial and ventricular myocardium, causing coordinated myocardial contraction.

It is important to remember that the change in the potential difference caused by depolarization of the SA node, AV node and His Purkinje system is not strong enough to cause a deflection on the surface ECG, and thus it is only depolarization of the atrial and ventricular myocardium that is recognized as a characteristic waveform on the ECG. Because the SA node has the fastest rate of spontaneous action potential generation, it is the site of impulse formation in the normal heart, and is responsible for generating the sinus rhythm. The SA node is influenced by sympathetic and parasympathetic innervations, which is responsible for regulating the heart rate in response to physiological stimuli such as exercise. From the SA node, the electrical impulse spreads over the atria to the AV node, producing the characteristic P wave on the ECG recording. The impulse is then conducted slowly through the AV node producing a delay that is recognized as the P-R segment on the ECG. The speed of conduction through the AV node is influenced by autonomic tone, with parasympathetic tone decreasing and sympathetic tone increasing the speed of conduction. The impulse is then rapidly conducted through the bundle of His and the Purkinje conduction fibers to the myocardium of the ventricles. Depolarization of the ventricles produces the characteristic QRS complex on the ECG recording. Following a period of refractoriness which is inherent of all myocytes, the myocardial cells of the ventricles begin to repolarize. The ventricular refractory period is represented by the ST segment on the ECG, while subsequent ventricular repolarization is represented by the T wave. This signals the end of one cardiac cycle.

III. Indications for an electrocardiogram

Any horse that has an arrhythmia identified on routine cardiac auscultation should be subjected to an
electrocardiogram in order to identify the cause of the arrhythmia. In simple terms, an arrhythmia refers to a condition in which there is abnormal electrical activity in the heart, and is characterized by an irregular heart rhythm or rate (tachycardia or bradycardia). An arrhythmia can easily be recognized by careful auscultation with a good quality stethoscope. The heart rate should be obtained and the rhythm of the heart should be assessed. The normal horse has a heart rate of 28-44 bpm at rest with a regular (sinus) rhythm. The heart rate of neonatal foals is considerably higher (80-120 bpm), but will also be regular. Sustained bradycardia (< 24 bpm) is uncommon and usually indicates underlying cardiac disease. Likewise, sustained tachycardia (> 50 bpm) that cannot be explained by pain or excitement should be investigated further and may indicate underlying cardiac disease. It is also useful to try and identify all 4 heart sounds, as absence of a heart sound may allude to a specific underlying arrhythmia (e.g. absence of S4 in the case of atrial fibrillation). The arterial pulses should be palpated simultaneously while auscultating the heart to determine if the arterial pulses are synchronous with every heart beat, as ‘pulse deficits’ may signal specific arrhythmias that affect ventricular filling during diastole (e.g. atrial fibrillation or early diasstolic ventricular premature depolarizations).

Other indications for performing an ECG in the horse includes investigation of sudden collapse, exercise intolerance or poor performance; and monitoring of the horse under general anesthesia, during intensive care management, and those undergoing arrhythmic therapy and other specialized procedures (e.g. pericardiocentesis). A surface ECG can also be used to monitor fetal heart beat in the case of the high risk pregnant mare.

IV. Obtaining a resting electrocardiogram

A resting ECG should ideally be obtained in a quiet environment and the horse should be adequately restrained to avoid movement artifacts. When attaching the electrodes to the horse, atraumatic crocodile clips or self adhesive electrodes can be used. There are several lead systems that have been described for use in the horse; however the base apex lead system is used most frequently (Figure 1). It produces large easy to read complexes that are relatively unaffected by movement artifact, and is well tolerated by the horse. To record a base apex electrocardiogram, the ECG recorder is set to lead 1 to record from the right arm (RA) to the left arm (LA) lead. The positive electrode (LA; frequently color coded yellow in Europe and black in the US) is positioned at the cardiac apex in the left axilla; the negative electrode (RA; frequently color coded red in Europe and white in the US) is placed two thirds the way down the right jugular groove and the third electrode, which serves as the ground (LL; frequently color coded as black in the Europe and red in the US) is placed at any site remote to the heart (usually over the withers). The resulting ECG recording will produce a negative QRS complex. Some clinicians will reverse the positive and negative electrodes, and in that case the ECG recording will produce a positive QRS complex. Both systems are appropriate and it is based upon personal preference.

Figure 1: a base-apex lead system in a horse under general anesthesia

Once the electrodes have been attached, the ECG recorder should be set to a paper speed of 25mm/sec and a recording can be obtained (Figure 2). A faster speed (50mm/sec) may be necessary if tachycardia is present. The standard deflection used is 10mm/mV, but 5MM/mV may be preferable in order to fit the large QRS complexes onto the paper.

V. Interpreting the electrocardiogram

A. ECG quality

The ECG should first be assessed in terms of its diagnostic quality. The most common artifacts are due to movement of the horse or the electrode leads; or due to poor electrical contact which results in AC interference. Movement artifacts are seen as sharp deflections of the baseline, which are random and have no pattern (Figure 3). They may however, sometimes resemble a QRS complex and care should be taken not to mistake them as such. AC interference will be recognized as regular ‘saw tooth’ deflection of the baseline at a frequency of 50 Hz and can be avoided by using a filter and ensuring that the ECG machine is adequately grounded.

B. The five question rule

Once you have determined that the ECG recording is of sufficient quality, the heart rate should be determined and then the ECG should be examined for evidence of rhythm disturbances and abnormal complexes. A simple way to do this is to ask yourself 5 questions every time that you examine an ECG recording:

1. What is the heart rate?

When calculating the heart rate, you need to know the paper speed. At a paper speed of 25 mm/second, each small box represents an interval of 0.04 seconds and each large box represents 0.2 seconds. The heart rate can therefore be calculated by dividing 60 by the R-R interval in seconds, or by counting the number of complexes in a known period of time.

2. Is the R-R interval or the P-P interval regular between complexes?

The rhythm should be assessed by determining if the R-R or P-P interval is regular between complexes (Figure 4)
Also check intervals within complexes e.g. P-R interval. This is especially important for atrioventricular (AV) blocks. An ECG caliper is very useful for determining if the R-R or the P-P intervals are regular.

3. Is there a P wave for every QRS-T?

The P wave represents atrial depolarization (from a right to left direction) prior to atrial contraction. A QRS-T complex that is not preceded by a P wave may indicate a ventricular premature depolarization (VPD), atrial fibrillation (AF) and in rare cases, a supraventricular premature depolarization (SVPD). In the case of AF, the P wave is replaced by fibrillation (f) waves. In some cases of SVPD, the P wave may be imbedded in the preceding T wave, and may not be visible.

4. Is there a QRS-T for every P?

The QRS-T complex represents ventricular depolarization and repolarization. A P wave that is not followed by a QRS-T complex indicates failure of conduction through the AV node and is seen in cases of second or third degree AV block.

5. Is there variation in the configuration of any of the complexes?

From a diagnostic point of view, it is important to remember that the ECG represents the electrical activity of the heart, and thus the characteristic waveforms on the ECG precede muscular activity. Thus the P wave represents atrial depolarization (from a right to left direction) PRIOR to atrial contraction. In most mammals the P wave may change configuration because it can originate from different sites within the SA node. This is called a wandering pacemaker and has no clinical significance. In the horse the P wave may be notched (bifid) or have to separate deflections (biphasic). Furthermore, in some horses you may be able to recognize atrial repolarization as a negative deflection in the PQ interval. This is often seen as dip in the baseline after the P wave and before the QRS and is called a Ta wave. The QRS complex represents ventricular depolarization as a whole. The first negative wave deflection is the Q wave, the first positive deflection is the R wave, and the second negative deflection is the S wave. The T wave represents ventricular repolarization and usually positive, but can change directions. The T wave is very variable in size and orientation, which means that T wave morphology, cannot be reliably used as an indicator of cardiac disease or electrolyte disturbances in the horse.

C. Measurement of amplitudes and duration

In the horse, the ECG is most useful to diagnose rate and rhythm disturbances. The duration and amplitude of the complexes and the duration of the intervals on the ECG recording in the horse are less meaningful diagnostically than they are in other species. This is because the ventricular depolarization process is very different from other species. In humans and small animals, the duration of the QRS complex may be prolonged when the left ventricle is enlarged, because the wave front spreads out from the subendocardial myocardium, and thus with enlargement of the ventricle, it takes longer to spread throughout the myocardium. The amplitude may also be increased as a result of the increased muscle mass. In the horse, the Purkinje fibers extend throughout the myocardium and do not terminate at the subendocardial myocardium. Consequently ventricular activation takes place from multiple sites, causing simultaneous depolarization of the entire ventricular myocardium. This means that the amplitude and duration of the QRS complex is not necessarily related to chamber size in the horse.

VI. Recognizing common arrhythmias on the ECG

Cardiac arrhythmias occur very commonly in horses and can be physiological or pathological. By definition, arrhythmias are characterized by uncoordinated electrical activity. This results in uncoordinated myocardial activity, which ultimately leads to decreased cardiac output. Ventricular arrhythmias are most serious as organ perfusion is primarily dependent upon ventricular contraction. Cardiac arrhythmias can be classified according to where they originate from (i.e. supraventricular or ventricular) and their rate (bradyarrhythmias and tachyarrhythmias).

A. Bradyarrhythmias

Most bradyarrhythmias are physiological and are related to the high vagal tone at rest and are therefore abolished by exercise, excitement, or administration of vagolytic or sympathomimetic drugs. Common physiological bradycardias that may be identified on an ECG in resting horses are second degree atrioventricular block, sinoatrial block, sinus bradycardia and sinus arrhythmia. Physiological bradycardias that may be encountered rarely are advanced second-degree AV block and complete (third-degree) AV block.

1. Second degree atrioventricular (AV) block

Second degree AV block is the most commonly detected arrhythmia in the horse and has been reported to occur in 40% of healthy horses at rest. This arrhythmia is physiological, and disappears with exercise or following administration of vagolytic or sympathomimetic drugs (e.g. atropine) in the normal horse. The heart rate is slow to normal (20-40 bpm) and the rhythm regular with intermittent pauses (regularly irregular). The complexes are all normal in appearance; however, the normal rhythm is regularly interrupted by a normal P wave that is NOT followed by a QRS complex (Figure 5). In some cases, two blocks will occur in sequence before the next conducted impulse. This is still regarded as physiological provided that the arrhythmia disappears with exercise. The R-R interval in the case of a second degree AV block is double the normal R-R interval.

2. Advanced second-degree AV block and complete (third-degree) AV block

Advanced second-degree AV block and complete (third-degree) AV block are pathological bradycardias that are associated with a conduction block at the AV node, and are usually caused by underlying electrolyte imbalances, digitalis toxicity or atrionodal disease (inflammatory or degenerative). These types of arrhythmias will cause severe exercise intolerance and syncope, and require treatment.
In advanced second-degree AV block, the heart rate is slow (8-24 bpm), with normally configured QRS complexes preceded by a P wave (i.e. there is still some evidence of AV conduction) interspersed by periods of atioventricular block, where a succession of P waves are not followed a QRS complex. The P-P interval is regular, however the periods of atioventricular block are prolonged (i.e. last for more than two P-P intervals) and do not disappear with exercise.

Complete (third degree) AV block is a more severe form of advanced second-degree AV block in which there is complete dissociation of the atria and ventricles (i.e. there is no evidence of AV conduction). The atrial rate is usually rapid, while the ventricular rate is slow and independent of the atria. The QRS complexes represent the ventricles attempt to ‘escape’ in the absence of AV node conduction, and their configuration will thus depend upon their origin. If they are supraventricular and originate from an idionodal pacemaker, they will have a normal QRS configuration. If however, they are ventricular, and originate from an idioventricular pacemaker, they will be wide and bizarre in appearance. Furthermore, if the QRS complexes all originate from the same pacemaker, they will be similar in appearance and regular in their rhythm. In complete (third degree) AV block, none of the QRS complexes are associated with a P wave and the P-R interval will vary considerably (Figure 6). The independent atria result in a regular P-P interval, but there are many more P waves than QRS complexes.

3. Sinus node (SA) block, sinus bradycardia and sinus arrhythmia

SA block is a physiological arrhythmia which results in a slow to normal heart rate and a regular rhythm with pauses that is equal to two P-P intervals (Figure 7). The QRS complex configuration is normal and each QRS complex is preceded by a P wave. SA block will often occur in combination with second-degree AV block. SA block has no clinical significance unless it is prolonged, associated with ventricular escape rhythms, and does not disappear with exercise or the administration of vagolytic or sympathomimetic drugs. In such cases, SA block is likely to be associated with sinus node disease (inflammatory or degenerative) and will require treatment.

Sinus bradycardia and sinus arrhythmia are also physiological arrhythmias associated with high vagal tone. Sinus arrhythmia is seen as a normal heart rate, with normal QRS complexes preceded by a P wave, however the P-P and R-R intervals are rhythmically irregular. Sinus bradycardia is seen as a slow heart rate with a regular rhythm. In both types of arrhythmia, the P wave, QRS complex and T wave have a normal configuration, and the QRS complex is preceded by a P wave.

B. Tachyarrhythmias

Tachyarrhythmias are defined as either supraventricular or ventricular. Tachyarrhythmias described in the horse are atrial fibrillation (AF); supraventricular premature depolarizations (SVPDs); supraventricular tachycardia; ventricular premature depolarization (VPDs); ventricular tachycardia (VT); and ventricular fibrillation. Persistent tachyarrhythmias are consistently present and hence easy to diagnose. Paroxysmal tachyarrhythmias are transiently present and therefore more difficult to diagnose, especially those that occur only during or immediately after strenuous exercise.

1. Atrial Fibrillation

Atrial fibrillation (AF) is the arrhythmia most likely to cause poor performance in the horse. Diagnosis on physical examination is straightforward with an irregularly irregular rhythm but with a normal number of beats per minute, variable jugular pulse height, variable pulse quality and no S4 audible. Diagnosis should always be confirmed by ECG: no P waves, presence of f waves, irregular R-R intervals and a normal QRS morphology (Figure 8). In approximately 10% of horses however, QRS complexes originating from the ventricle will also be detected, and can be differentiated by their abnormal configuration. During exercise, the increased sympathetic tone will largely abolish the blocking function of the AV node which results in an excessively high ventricular rate during exercise and, in combination with the loss of atrial contractility, cardiac function is significantly reduced. However, clinical signs of poor performance depend on the level of exercise required and so may be an incidental finding in non or low athletic horses. In Thoroughbreds, in particular, paroxysmal AF has been shown to occur relatively frequently during a race. Paroxysmal AF can be suspected in horses that have more than 1 SVPD per hour on a resting ECG with a history of poor performance and an irregularly irregular heart beat on auscultation immediately after exercise. When AF is confirmed (or suspected in the case of paroxysmal AF), echocardiography, haematology, serum biochemistry and cardiac troponin I concentrations should be assessed to try and determine if there is an underlying cause. Whole body potassium and magnesium depletion has been implicated in cases of paroxysmal AF, and should be investigated. The majority of AF cases can be converted to sinus rhythm, provided there is no underlying heart disease, by either medical treatment (quinidine sulphate, amiodarone or flecaïnide) or electrical cardioversion. When significant underlying cardiac disease is present, prognosis for conversion is poor. If cardiac failure is present, supportive therapy (e.g. digoxin or enalapril) or euthanasia is required. Horses that cannot be converted can generally be safely used for non-strenuous exercise.

2. Supraventricular premature depolarizations (SVPDs) and supraventricular tachycardia

SVPDs originate in the atria prior to generation of an impulse from the SA node. On auscultation, early beats are detected. SVPDs are characterized by irregular R-R intervals and premature P waves of different configuration than those from SA node followed by normal QRS complexes (Figure 9). The P wave of an SVPD may often be buried in previous T wave. SVPDs are occasionally found in otherwise healthy horses. Isolated SVPDs are usually overridden by sinus tachycardia during exercise and will thus not affect performance. However, frequent SVPDs have been
associated with reduced performance especially when they result in supraventricular tachycardia during exercise or when they occur frequently and reduce cardiac output. Horses with frequent SVPDs are at increased risk of developing AF and may be seen in horses with paroxysmal AF. The diagnosis should always be confirmed by an ECG.

Supraventricular tachycardia is defined as more than 4 SVPDs occurring in succession and is usually indicative of myocardial disease or atrial enlargement.

3. Ventricular Premature Depolarizations (VPDs) and Ventricular Tachycardia (VT)

VPDs originate in the ventricular myocardium. On auscultation, VPDs can be recognized by a beat that occurs earlier than normal and they may be associated with a pulse deficit if the VPD occurs in early diastole. The intensity of the heart sounds may also vary. On an ECG, VPDs are characterized by irregular R-R intervals and bizarre QRS waves that are not preceded by a P wave (Figure 10). Occasional VPDs at rest are probably insignificant and may be found in otherwise healthy horses with a normal performance history. A thorough cardiac exam should be performed when VPCs occur more frequently however (e.g. more than one VPD per hour on a resting ECG), as they suggest myocardial hypoperfusion, myocarditis or cardiomyopathy. Where VPDs are present at rest but disappear during exercise, they are usually not a cause of poor performance. However, VPDs which increase in number or occur as ventricular tachycardia during exercise, or occur solely at the end of strenuous exercise are likely to be associated with poor performance. The diagnosis should always be confirmed by an ECG.

Ventricular tachycardia (VT) is defined as more than four VPDs occurring in succession and is usually indicative of primary myocardial disease, although non cardiac causes such as hypoxia and electrolyte disturbances have also been implicated. On auscultation, the heart rate is rapid (often > 120 bpm) and may be irregular in the case of monomorphic VT or irregular in the case of polymorphic VT. The heart sounds are often loud and vary in intensity. The ECG reveals a rapid heart rate with numerous wide, bizarre QRS complexes are unrelated to the receding P waves (Figure 11). The P-P interval is regular, but the P waves are often buried in QRS and T complexes (AV disassociation). The QRS complexes may all be similar in configuration (monomorphic), suggesting that they arise from an ectopic focus in the ventricle, or they may be variable in configuration (polymorphic), suggesting multiple ectopic foci of abnormal electrical activity in the ventricle. The QRS complexes Ventricular tachycardia is always pathological and requires treatment, as it can cause syncope and may lead to congestive heart failure or deteriorate into ventricular fibrillation and sudden death.

VII. Conclusion

An ECG can easily be obtained in practice and, in the majority of cases, can be interpreted in order to identify the arrhythmia. Possible underlying causes can then be investigated and the need for therapy determined.

VIII. Additional reading

4. Guglick MA. Cardiac notes. In Colt notes, coltnotes@hotmail.com. 2013

IX. Selected references

X. Electrocardiograms

**Figure 2**: a base apex ECG obtained from a normal horse at rest. Notice predominantly negative QRS complex. Recorded at a paper speed of 25 mm/second and a standard deflection of 10 mm/mV.

**Figure 3**: ECG from a normal horse demonstrating movement artifact and AC interference. AC interference can be recognized as a ‘saw tooth’ jagged baseline and should not be misinterpreted as atrial fibrillation. Recorded at a paper speed of 25 mm/second and a standard deflection of 10 mm/mV.

**Figure 4**: Normal sinus rhythm in a horse. The heart rate is 42 bpm. Notice the regular R-R and P-P intervals. There is a P wave for every QRS; a QRS for every P wave; and the complexes have the same configuration from beat to beat. Recorded at a paper speed of 25 mm/second and a standard deflection of 10 mm/mV.

**Figure 5**: Second degree atrioventricular (AV) block in a horse. Notice the P wave that is not followed by a QRS-T complex and a pause that is approximately equal to two R-R intervals. Recorded at a paper speed of 25 mm/second and a standard deflection of 10 mm/mV.

**Figure 6**: Complete (third degree) AV block in a horse. Notice the non-conducted P waves and the wide and bizarre QRS complexes originating from an idioventricular pacemaker. Recorded at a paper speed of 25 mm/second and a standard deflection of 10 mm/mV. Picture Nicola Menzies-Gow
**Figure 7:** Sinoatrial (SA) block in a horse. Notice the absence of a P, QRS and T wave and a pause that is equal to two P-P intervals. Recorded at a paper speed of 25 mm/second and a standard deflection of 10 mm/mV. Picture Nicola Menzies-Gow

**Figure 8:** Atrial fibrillation in a horse. Notice the absence of P waves, presence of f waves, irregular R-R intervals and a normal QRS morphology. Recorded at a paper speed of 25 mm/second and a standard deflection of 10 mm/mV.

**Figure 9:** Supraventricular premature depolarization (SVPDs) in a horse. Notice the irregular R-R intervals and premature P waves of different conformation than those from SA node followed by normal QRS complexes. Recorded at a paper speed of 25 mm/second and a standard deflection of 10 mm/mV.

**Figure 10:** Ventricular premature depolarizations (VPDs) in a horse. Notice the irregular R-R intervals and bizarre QRS waves that are not preceded by a P wave. Recorded at a paper speed of 25 mm/second and a standard deflection of 10 mm/mV.