YOU'VE GOT THE BEAT: DIAGNOSIS OF ARRHYTHMIAS BY AUSCULTATION AND ELECTROCARDIOGRAPHY

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INTRODUCTION

The purpose of this talk is to review the basics of the most common rate and rhythm disturbances in horses. The story is not new, but the way in which the material will be presented will hopefully assist you in your auscultation skills. Common arrhythmias were recorded from patients, digitized, and inserted into QuickTime movies. The rhythms were simultaneously captured by an electrocardiogram (ECG). The audience will be able to hear as well as see the sounds files, movies, and ECGs.

NORMAL SINUS RHYTHM

A rate or rhythm disturbance is detectable by auscultation, but definitive diagnosis requires an ECG.

It would be highly unusual for clinically significant cardiac disease to be present without a change in the heart rate, rhythm, or the presence of a murmur. The last session reviewed the normal equine heart sounds and the meaning of a murmur. Like the murmur, auscultation is the first key to detecting a rate or rhythm disturbance. *Probably one of the most common reasons for missing a rate or rhythm disturbance in a horse is insufficient time in auscultation.* **Sustained bradycardia** (heart rate < 24 beats/minute) is uncommon in the horse and usually indicates an underlying pathologic etiology. Likewise, **sustained tachycardia** (heart rate > 50 beats/minute) that cannot be explained by excitement or pain should be further investigated as it may be a sign of underlying cardiac disease. An arrhythmia simply refers to any change in the time between cardiac cycles that disrupts the regular pattern of systole. *Thus the key to detecting an arrhythmia is cardiac auscultation and/or simultaneous palpation of the pulse of sufficient duration to establish a rate as well as the pattern of systolic events (pulse generation or generation of S1/S2). If a rate or rhythm disturbance is detected, the best way to definitively determine the cause is to perform an ECG.*

To understand the arrhythmias, one must first appreciate the electrical events that generate the cardiac cycle.

Most cells in the body have an **electrical potential difference** across their cell membrane. That is, differences in the respective concentrations of ions inside versus outside of the cell create an electrical charge, with the inside of the cell carrying a negative potential charge, relative to the outside. Ion pumps that remove sodium from within the cell and replace it with potassium maintain this negative potential charge. A unique aspect of myocardial cells is that in addition to maintaining an electric potential, they can rapidly change this electric potential in response to signals from neighboring cells. This creates the "**action potential**" that drives myocardial contraction. The **sinoatrial node** (SAN), located in the right atrium, is comprised of cells that have an unstable resting potential that drift toward a positive potential. This automatically driven action potential sets the pace of myocardial contraction. As each atrial myocyte produces an action potential, calcium is delivered to the intracellular contractile

units. As the wave of excitation moves toward the apex, it enters the **atrioventricular node** (AVN), wherein ventricular contraction is controlled. The AVN sends impulses to the extensive network of the Purkinje cells, that themselves do not contain contractile units. Thus, the Purkinje cells serve to disseminate the pace setting wave of excitation almost simultaneously to the contractile ventricular myocytes.

The electrical events of the heart translate to what we see on an ECG.

By setting both positive and negative electrodes on the body surface, strategically around the heart, the general pattern of the sum of the action potentials created by the drive of the relative ionic charge of the cells can be detected. In other words, as the cells depolarize and become relatively positive on the inside, the surface electrodes detect this wave of change in charge movement. The ECG recorder generates an "upswing" when the wave of depolarization moves parallel to electrodes, in the direction from the negative electrode to the positive electrode. Why is this at all meaningful? *It tells us that when trying to optimize the size of the deflections recorded by an ECG machine, the electrodes should be set relatively parallel to the main wave of excitation of the myocardial cells, with the positive electrode set away from the initial site of excitation. This is exactly what the* ***Base-Apex Lead*** *does in a horse.* In fact, because of the size of a horse's heart and the extensive Purkinje cell system, the Base-Apex Lead typically is the only lead that is used to record the electrical activity of a horse's heart. Furthermore, an ECG in a horse will document rate and general rhythm disturbances, but it is less useful in detecting changes in the overall direction of electrical activity (i.e. shifts in the mean electrical axis) that may detect changes in relative size of heart chambers in other species. The bottom line to remember is that the **P wave** represents atrial depolarization. Atrial repolarization occurs during ventricular depolarization and is not seen as a separate deflection on the ECG recording. The **QRS complex** represents ventricular depolarization. Technically, the Q wave is the first negative deflection of the trio, the R wave is the first positive deflection, and the S wave is the next negative deflection generated by ventricular depolarization. The **T wave** represents ventricular repolarization.

There are 4 steps to run a Base Apex Lead recording in the horse.

1) The right arm electrode (frequently color coded white) is clipped to the skin over the right shoulder or right jugular grove.
2) The left arm electrode (black) is placed over the apex of the left heart slightly above the left elbow.
3) The left leg (red) electrode serves as a ground and is usually place on the right side of the neck.
4) The recorder is set to lead I. This will make the left arm electrode positive and the right arm negative.

The large atria in horses often results in slightly asynchronous depolarization, thus the P wave often appears as biphasic positive deflection in the Base-Apex recording (i.e. has 2 small humps). The QRS in horses mostly is depicted as a downward deflection of the ECG recording in the Base Apex mode, and thus can also be referred to as a QS or an S wave. The T wave usually is represented by an upward deflection, but may also be diphasic or negative. A few other helpful facts are that when the ECG recorder is set to a paper speed of 25 mm/sec, each of the smallest boxes on the ECG paper grid is equivalent to 0.04 sec. In general, the duration of

the complexes and the intervals (time between) generation of the complexes on the ECG paper are less meaningful diagnostically than they are in other species and normal values are provided elsewhere.[1] *More helpful is the fact that most recorders place a large "tick" on the top of the paper that is generated every 3 seconds. The heart rate can be determined by counting the number of QRS complexes generated over 6 seconds and multiplying by 10, when the paper speed is 25 mm/sec.*

There are several steps to interpretation of an ECG, but a key step is identification of the QRS complexes.

When interpreting an ECG 1) make sure the horse is standing still and that the leads are securely attached and not disturbed, 2) determine the heart rate, 3) find the largest deflections. These should be the QRS or the QS or the S complexes, 4) if there was a QRS complex i.e. ventricular depolarization, then there **must be** ventricular repolarization. Look immediately behind the QRS complex for the T wave, 5) look in front of the QRS complexes. Is there a P wave for every QRS and is there a QRS for every P wave? 6) study the intervals from QRS to QRS are they regular or irregular? 7) do the QRS complexes come earlier or later than expected? 8) do the configurations of all the QRS complexes and P waves look the same? and 9) are the durations of the complexes normal?

The predominant rhythm should be sinus in origin, but even sinus origin rhythm can be irregular.

In normal sinus rhythm, the SAN fires and sets the pace of systole by sending the wave of depolarization through the atria and to the AVN to the ventricles. *Common nonpathologic variations of normal sinus rhythm are second degree atrioventricular (AV) block, sinus block, and sinus arrhythmia*. **Second degree AV block** occurs with high resting vagal tone that slows conduction of impulses from the SAN to the AVN. Thus, the SAN fires, causing atrial contraction and generation of a P wave, but no conduction through the AVN. Systole, S1, S2 and the QRS complexes do not occur (see Figure 1). Typically, second degree AV block is detected as a fairly rhythmic (regularly irregular) loss of the sounds of S1 and S2. During these periods of asystole, occasionally the sole sound of atrial contraction (S4) is audible. Because second degree AV block is physiologically associated with increased parasympathetic tone, it should dissipate with exercise or excitement. If it does not go away with exercise and the heart rate does not increase with exercise, it should be considered pathologic.

Sinus block is less common. On auscultation it sounds very similar to second degree AV block, as there are periods of asystole, but S4 is never audible during the periods of asystole. Sinus block is also due to high vagal tone that blocks the pacing cells of the SAN. So here, the atrial do not contract (thus no S4) and the ECG shows a period during which neither P waves or QS/T complexes are generated (see Figure 2). Like second degree heart block, sinus block should dissipate with exercise.

Sinus arrhythmia is less common in horses than people, but it is a normal physiologic response to the changes in parasympathetic and sympathetic tone with the respiratory cycle. During inhalation, the sympathetic nervous system is stimulated, thus the heart rate may increase slightly. During exhalation, parasympathetic tone is greater, thus the heart rate slows.

COMMON PATHOLOGIC ARRHYTHMIAS

Atrial fibrillation is a common pathologic arrhythmia that is often confused with second degree AV block.

Atrial fibrillation is caused by inhomogeneity of depolarization of the atrial myocytes. Coordinated atrial contraction does not occur and thus, the sound of S4 is never heard during atrial fibrillation. The conduction of impulses from the atria to the ventricles is not in a directed path, as it is with normal sinus conduction, thus the random event of an atrial impulse firing the AVN accounts for the irregularly irregular rhythm of atrial fibrillation. Another audible characteristic of atrial fibrillation is that the intensity of S1 and S2 can vary from beat to beat. Because horses can have a normal heart rate while in atrial fibrillation, not surprisingly, atrial fibrillation is frequently confused with second degree AV block on auscultation**.** The irregularly irregular rhythm can be hard to detect if the heart rate is faster, thus the ECG is the best way to confirm your diagnosis (see figures 3 and 4). The classic ECG findings of atrial fibrillation are: 1) no definitive P waves, 2) flutter in the baseline, and 3) normal appearing QS complexes at irregular intervals. Atrial fibrillation is not normal and if documented, further evaluation of the heart with an echocardiogram is recommended.

Premature contractions and their tachyarrhythmias represent ectopic myocardial centers of depolarization.

Atrial premature contractions (APC) may be normal in horses if they are rare in occurrence or occur post exercise. An APC occurs when a focus in the atria, other than the SAN, fires an impulse that sets off atrial conduction. In auscultation, systole is heard "sooner than expected," may be louder than beats originating from the SAN, and the premature beat is not followed by a pause. If there are 4 or more premature atrial contractions in a row or it is sustained, it is referred to as **atrial tachycardia.** Atrial tachycardia (not normal) is almost impossible to distinguish from sinus tachycardia by auscultation alone. On the ECG, APCs are identified by: 1) a normal looking QS complex that appears sooner than expected and is preceded by a P wave that may be of different conformation that SAN origin beats (see Figure 5). Frequent APC's or those that result in an increased heart rate are not normal and require further investigation into their cause. Even with an ECG, atrial tachycardia can be difficult to distinguish from sinus tachycardia. Atrial tachycardia should be suspected if tachycardia is sustained and cannot be explained by pain or excitement.

Ventricular premature contractions (VPC) are less common than APCs, but are less worrisome if they are rare in occurrence. A VPC represents an impulse that originates from the ventricles, resulting in ventricular depolarization and contraction without atrial conduction or contraction. On auscultation, S1 and S2 occur "sooner than expected," are often quieter than SAN origin beats, and are followed by a pause, as the SAN resets. **Ventricular tachycardia** is never normal and represents 4 or more VPCs in a row. Because the impulses originate from the ventricles, on the ECG the characteristic findings of VPCs are: 1) a bizarre shaped QRS complex that occurs sooner than normal, 2) the QRS is not preceded by a P wave, and 3) there often is a pause or period of asystole after the VPC, if it is isolated (see Figure 6). Ventricular tachycardia is difficult to distinguish from nonpathologic sinus tachycardia by auscultation alone. However, an ECG will distinguish ventricular tachycardia as a series of rapidly occurring, bizarre shaped QRS complexes.

As for interpreting murmurs, the best way to feel comfortable with detecting arrhythmias is to practice![3]

Figure 1. Second Degree AV block. The period of asystole occurs at the arrowhead, when only a P wave is generated.

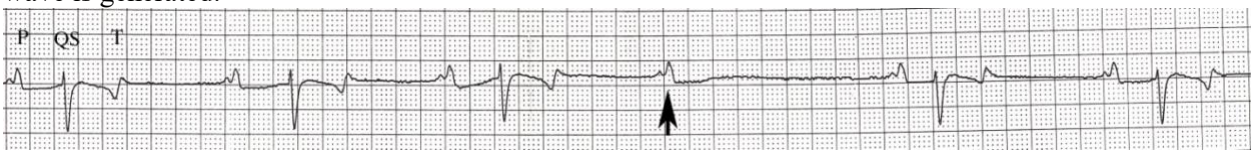


Figure 2. Sinus block. Note the period of asystole between the arrowheads.

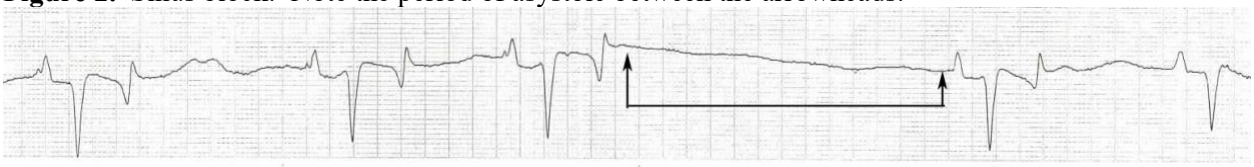


Figure 3. Atrial fibrillation with a normal heart rate.

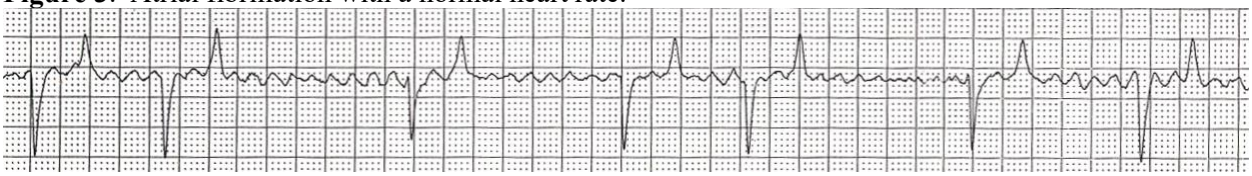


Figure 4. Atrial fibrillation with an increased heart rate. Although less obvious than figure 3, note the differing R-R intervals.

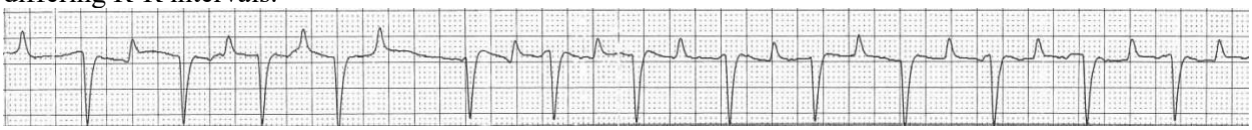


Figure 5. Atrial premature contraction (arrow).

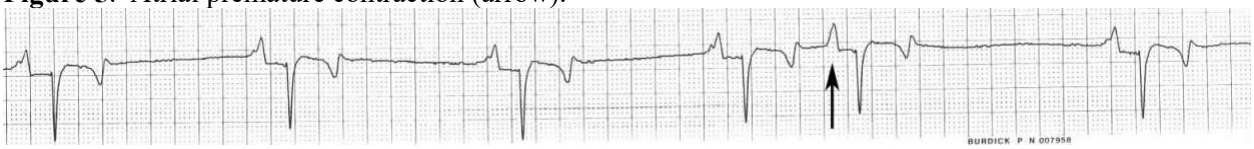
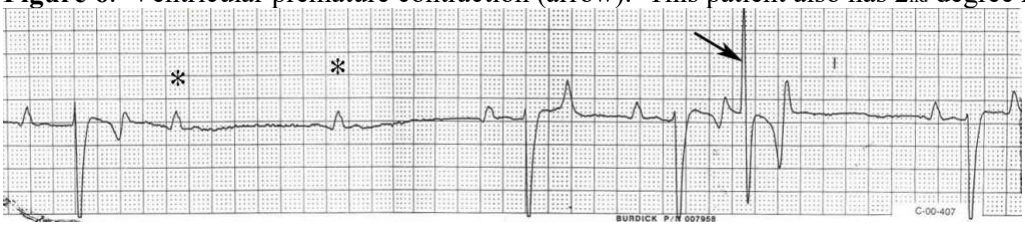


Figure 6. Ventricular premature contraction (arrow). This patient also has 2nd degree AV block (*).



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