Arrhythmias Beyond AV Block and A Fib: Practical Diagnosis & Management

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In horses, the most commonly identified cardiac arrhythmias are second degree atrioventricular block and atrial fibrillation. Both of these arrhythmias have been discussed extensively in veterinary literature, and are outside the scope of this presentation. Instead, this presentation will focus on some of the less common but still clinically important arrhythmias in the horse. This is not intended to be an exhaustive listing and discussion of every potential arrhythmia--for that, the reader is directed to book chapters and review papers in the literature. The goals of this presentation are to briefly discuss these other pathologic cardiac arrhythmias, specifically their clinical definition, diagnosis, and management, followed by brief case discussions. Special attention will be paid to the new smartphone ECG hardware and Holter monitoring, as a potential practice builders towards the overall conference theme of "Healthy Practices, Healthy Patients."

Diagnosis of Arrhythmias

Auscultation:

Major component of physical examination- important to auscultate on both sides of the chest for 30 seconds. Palpating the pulse concurrently can be helpful to correlate auscultation to palpable pulse timing and quality. The presence of murmurs and abnormal rhythm is always noteworthy, especially at the time of prepurchase examination, and should be documented and described to the best of the practitioner's ability. The volume of the murmur does not correlate to severity of cardiac dysfunction, however the presence of turbulent blood flow in the face of other clinical abnormalities, or in a horse with athletic purpose requires further consideration. For specific guidelines, see the ACVIM Consensus Statement on Management of Equine Athletes with Cardiovascular Abnormalities. Additionally, interpreting auscultation findings is important in the context of the clinical picture in front of you. These findings may mean very different things in a normal, apparently healthy horse at pre-purchase examination, as compared to a horse showing exercise intolerance or signs of systemic illness. Regardless, if there are clinical signs of heart failure, arrythmia present, or heart rate greater than 100 beats per minute, an electrocardiogram (ECG) is an essential component of the diagnostic workup.

Electrocardiography:

Traditional ECG System

Many private practice settings have access to electrocardiography, and it can easily be applied to the horse. Typically, the most useful ECG lead orientation in the horse is the base-apex lead, which assesses electrical vectors from the base of the heart (negative) to the apex of the heart (positive). The correct orientation of leads is: Left Arm (+, usually black)

at left axilla; Right Arm (-, usually white) on right side cranial to scapula, midway down; Right Leg (usually green) at the base of the right jugular vein; and Left Leg (usually red) at the left stifle. This can be recorded on most machines on Lead 1 setting. Paper speed is typically set at 25 mm/sec. Alligator clips should work on skin, with alcohol or gel to improve electrical conduction. Noise can be reduced by minimizing movement of the ECG lead wires as much as possible (holding wires as a bundle can be useful), and restraining the horse within stocks or stall if at all possible. There are other lead orientations that can be used, but this is the most convenient based on the orientation of the equine heart within the thorax and the direction of depolarization vectors within the cardiac tissue. Amplitude of the waveform may be adjusted based on the ECG monitor, and little clinical significance is placed on the amplitude of the waveform as long as it is distinct enough from baseline noise to be able to reliable characterize the waveforms. Waveform morphology cannot be used reliably to characterize chamber enlargement in horses- echocardiography is required to effectively evaluate structural abnormalities of the equine heart. It is important to record the ECG for at least 30 seconds continuously, ensuring appropriate signal quality for sufficient diagnostic accuracy. It is often easier to ensure appropriate ECG signal quality and record without interpretation in real-time, then go back and interpret after the recording is complete.

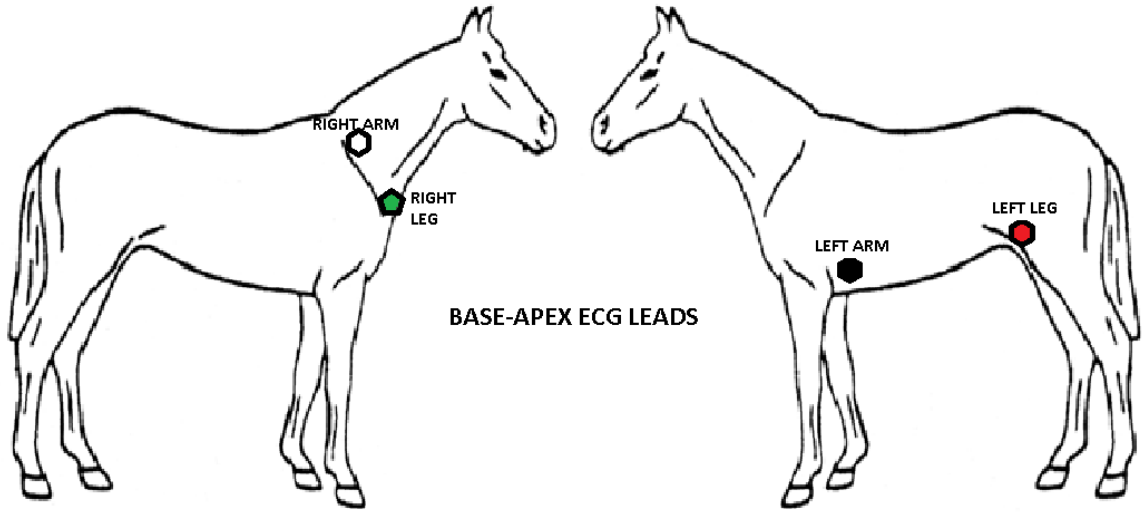

Fig. 1- Placement of electrodes for base apex ECG on the horse: Left Arm (+, usually black) at left axilla; Right Arm (-, usually white) on right side cranial to scapula, midway down; Right Leg (usually green) at the base of the right jugular vein; and Left Leg (usually red) at the left stifle.

Smartphone ECG System

A commercially available, wireless smartphone-based ECG device has been developed for veterinary use, and it has proven to be both accurate and useful for acquisition of an ECG tracing in horses. The AliveCor ECG device is essentially a battery-operated iPhone case with two built-in electrodes that acquire and process the electrical signals and transmit the data to proprietary software via Bluetooth. The AliveCor device has been validated for use in dogs, cats, pigs, and horses (although the company also markets its use in exotics of all varieties). In horses, the two electrodes are applied to the skin of the left axillary region (with alcohol to improve conductivity) at a dorsoventral orientation with 30 degree

cranial inclination (11 o' clock position). Two publications have demonstrated its accuracy for acquiring rhythm and rate in comparison with the traditional base-apex readings. The initial pilot study used 20 horses, and this found 100% agreement in measuring heart rate (instantaneous and average) and intra-observer agreement for rhythm assessment. A larger study of 50 horses found 100% agreement of heart rate and rhythm assessment between the smartphone ECG tracing and base-apex ECG. Several limitations were identified in this study. The presence of baseline artifact was greater in the smartphone ECG tracings, however only 2/50 of tracings were unable to be interpreted by a blinded interpreter. The iPhone app frequently overestimated the instantaneous rate due to misinterpretation of tall T waves, however manual calculation of heart rate was 100% accurate from the tracings. Additionally, there were some variations in polarity of components of the waveforms, but these were clinically insignificant. Finally, both studies utilized normal horses and there are no current studies evaluating the diagnostic utility for detecting arrythmias in horses. However, the use of similar technology in humans (including an identical AliveCor product) has shown great promise and non-inferiority for outpatient diagnosis of several clinically significant arrythmias, including supraventricular tachycardia, atrial fibrillation, and ventricular ectopic beats. Overall, the AliveCor ECG system can be easily implemented into equine practice, and can be useful both in clinics and in ambulatory settings to acquire and interpret equine ECG tracings. An added benefit is the software's ability to store tracings for future reference, as well as automated conversion to pdf files for uploading to medical records or sending for interpretation. Obviously, this is not as high-quality as recordings obtained by Holter monitoring, but can be relatively cost-feasible ($225 for the case + iPhone) and used in multiple species as a practice builder in the mixed animal setting as well.

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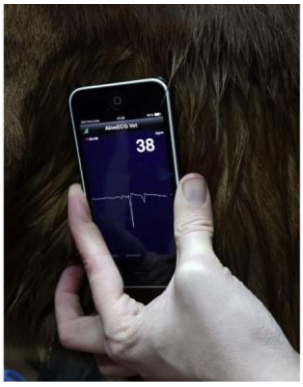


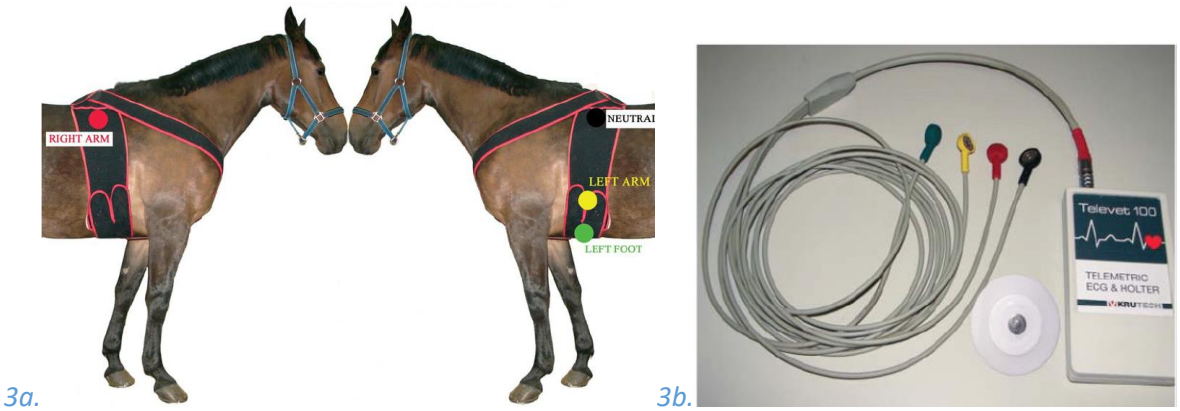
2b.

Fig 2. Smartphone-based ECG system: Fig. 2a Electrodes are built into the iPhone case, sending data to the phone via Bluetooth. Fig. 2b Device is placed at 11 o' clock position in the left axillary region, with the camera ventrally, on skin prepared with alcohol. (Source-Vezzosi, et. al. 2018)

Holter Monitoring

Continuous ambulatory ECG recording, typically over a 24 hour period, in which ECG electrodes are placed in a specific configuration around the girth, with leads running to a small, wearable recording device. This is the best method for accurately assessing the horse's true resting heart rate and frequency of arrythmias over the daily routine in the farm setting, including during exercise. It can be worn under a girth or surcingle, allowing the horse to be worked in its

normal routine and environment. This can be performed remotely by the primary care veterinarian by renting the unit from the AUCVM Cardiology Service, applying and recording data, and returning for interpretation. This can be helpful in the diagnostic work-up of horses with poor performance, behavioral abnormalities, or evaluation of ectopic beats (APC's or VPC's). Through phone consultation with the AUCVM Cardiology Service, this can be a valuable practice builder as it offers a telemedicine-type approach to managing cases in the field, at your home practice. This service is provided for approximately $250, and can also be useful prior to AUCVM cardiology work-up, or as a component of follow-up care. The primary limitation of Holter monitoring is that the recording is not viewed in real-time, so life-threatening arrhythmias cannot be diagnosed at the time they are occurring, and the quality of recording cannot be assessed until retroactive signal analysis by the cardiologist. However, the diagnostic value and convenience of Holter monitoring make it a valuable tool in arrhythmia evaluation in horses.



3a. *3b.*

Fig.3. Holter monitor setup in the horse. Fig. 3a. Electrode orientation, underneath girth to secure electrodes for 24 hour monitoring, including during exercise. Fig. 3b. Holter monitor device Source: Verheyen, et al. 2010

Major Classifications of Arrhythmias

Physiologic Arrhythmias:

Physiologic arrhythmias are considered largely insignificant regarding hemodynamic consequences to the horse.

Sinus Rhythms

Normal PQRST waveform morphology, with the stimulus originating in the sinoatrial node and subsequent depolarization following the appropriate conductive pathway through the heart, but abnormal timing of SA firing. High parasympathetic tone, as mediated by the vagal nerve, drives sinus bradycardia, sinus arrhythmia (waxing and waning of the heart rate), and even sinus arrest. These arrhythmias are typically eliminated with stress or exercise, ceasing that vagal suppression of the SA node and resulting in sinus rhythm or even sinus tachycardia. Sinus bradycardia and tachycardia may be related to stress (sympathetic tone), blood gas and electrolyte derangements, endotoxemia, variations in blood pressure, pharmaceuticals administered, and many other variables as well.

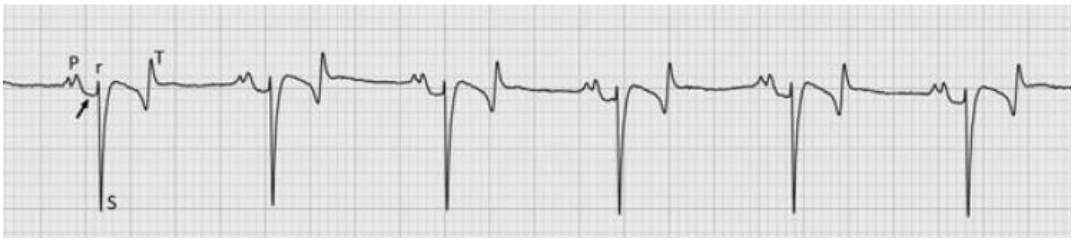


Fig. 4. Regular sinus rhythm: Base-apex lead, recorded at 25 mm/sec. P wave is notched, which is normal in horses. QRS and T waveforms are labelled. Source: Verheyen, et. al. 2010.

Delays in Transmission

Normal P wave (atrial depolarization), but abnormal timing associated with transmission through the atrioventricular conduction system. These are also largely physiologic in origin. High vagal tone drives the majority of first and second degree atrioventricular blocks. They can also occur after exercise and certain medications (most commonly alpha-2 agonists) as well. Second degree AV block should be eliminated by exercise or administration of vagolytic drugs (e.g. atropine). If it persists, more than 2 consecutive beats are dropped (high-grade 2nd degree AV block), or if there is evidence of third degree AV block (complete dissociation of the atria and ventricles), further investigation is warranted into structural and functional cardiac disease.

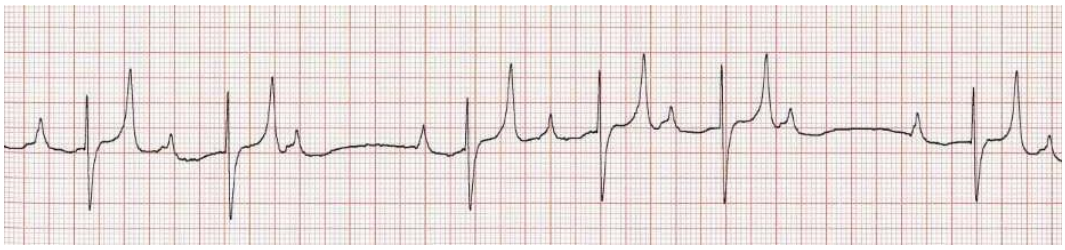


Fig. 5. Second degree AV Block, Type I: Base-apex lead, recorded at 25 mm/sec.
Note the P-R interval increasing with each beat until the dropped beat. This was a vagally driven arrhythmia that was eliminated with exercise.

Pathologic Arrhythmias:

Pathologic arrhythmias are considered more significant regarding hemodynamic consequences to the horse.

Supraventricular Tachycardia

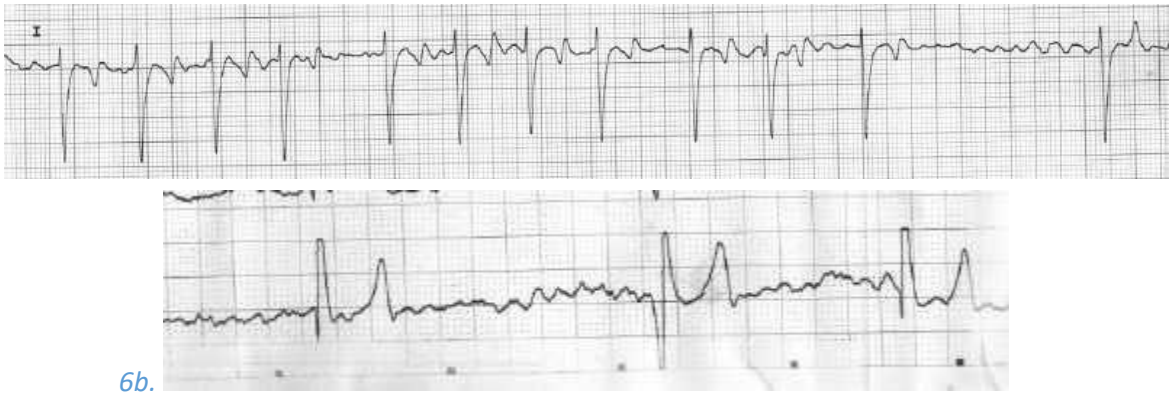
These occur on a spectrum, from atrial tachycardia to atrial flutter, and finally atrial fibrillation. These are non-life threatening, but will always progress to atrial fibrillation and exercise intolerance. These are caused by one or more ectopic foci (excitable region of muscle tissue) within the atrial tissue, causing an elevated firing rate within the atrial muscle.

- *Atrial tachycardia:*

  On auscultation, has a regular rhythm, significantly elevated rate, and acute onset/cessation (often hard to detect). On ECG, it has a P' wave (depolarization of atrial tissue not triggered by SA node) followed by normal QRST waveform. Surface ECG can often not differentiate effectively, and this should be further investigated with Holter monitoring to differentiate sinus tachycardia vs a pathologic arrhythmia caused by ectopic foci within the atria.

- *Atrial Flutter & Fibrillation:*

  On auscultation, there is an irregularly irregular rhythm, due to inconsistent transmission of atrial depolarizations through the AV node. This variable ventricular response rate can be affected by parasympathetic and sympathetic drive, and it can make AF more difficult to diagnose on quick auscultation, especially during times of stress/exercise. On ECG, the classical lack of P wave is seen with either a regularly fluctuating, sawtooth baseline (flutter) or more rapid and fine fluctuations (fibrillation). This can occur as spontaneous and paroxysmal (episodic), or a permanent condition. The presence of atrial fibrillation without any detectable underlying cause is considered lone AF and typically responds well to therapy, whereas the presence of chamber dilation or long-standing arrhythmia causing secondary remodeling decreases long-term success of conversion therapy. Several treatment options are available- quinidine or transvenous electrocardioversion most commonly, and several others under investigation- and should be pursued as early in the disease course as is feasible to optimize the chances of successful conversion.



6a.

6b.

Fig. 6. Atrial Ectopic Rhythms: Base-Apex Lead, recorded at 25 mm/sec.
Fig. 6a. Atrial Flutter: Note the sawtooth appearance to the baseline, and the irregularly irregular timing of QRS complexes. Fig. 6b. Atrial Fibrillation: Note the very irregular baseline.

Premature Complexes

On auscultation, within a regular rhythm, a premature beat is detected, potentially followed by a pause. This premature beat may or may not have a pulse deficit (detectable decrease in pulse quality compared to the rest of the beats) associated with it. The sound quality of the premature beat may be different than the rest of the beats. ECG must be performed to differentiate APC from VPC, because auscultation alone is insensitive and VPC's are potentially life threatening and require immediate further investigation.

- *Atrial Premature Complex (APC):*

Ectopic beats in atrial tissue – the P wave happens too soon, or may overlay previous T wave and be difficult to detect. APC morphology technically has an abnormal P waveform compared to prior sinus beats, but this may or may not be noticeable. The QRST morphology should be normal, just occurring too soon. Typically, there is a refractory period afterwards, as the ectopic beat moves dorsally through the muscle and depolarizes the SA node retroactively. They are considered benign and inconsequential if infrequently detected (normal horses have 1/hr), however detecting the severity (true frequency) of APC's is poor with one-time surface ECG. Holter monitoring is recommended to determine the frequency of APC's over a 24 hour period, and this can be performed as a mail-order service through the AU Cardiology Service. APC's are concerning if occurring too frequently (no consensus on what "too frequent" is, unfortunately), or are occurring in the face of atrial chamber dilation, as this indicates high likelihood of progression into supraventricular tachycardia and atrial fibrillation. Echocardiography is required to assess chamber size, and can be performed on an outpatient basis through the AU Equine Internal Medicine & Cardiology Services. There is minimal risk for anesthesia and exercise if APC's are infrequent and there is no evidence of chamber dilation.

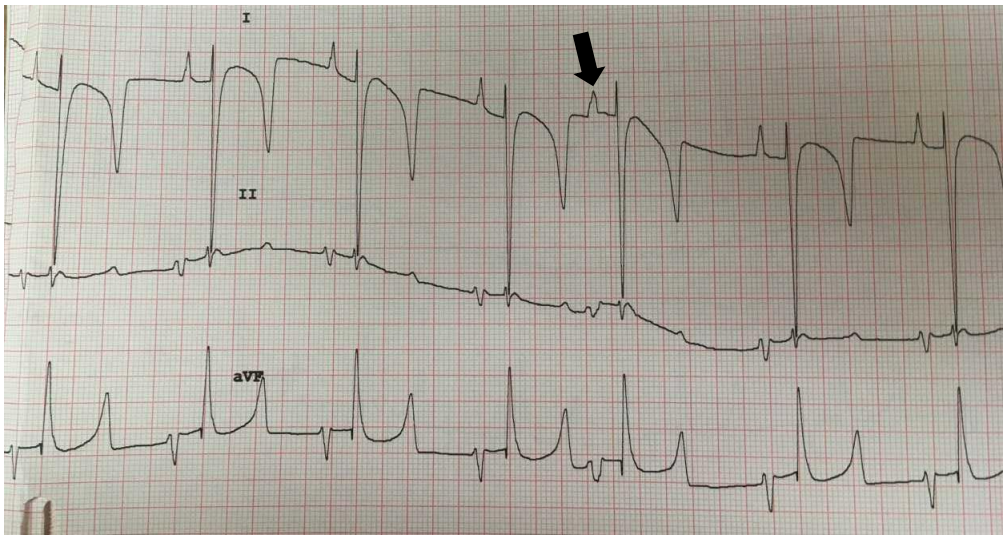


Fig. 7. Atrial *Premature Complex: Base-Apex lead, recorded at 25 mm/sec.*
Note the premature beat (arrow), with different P wave morphology than preceeding sinus beats, normal QRST morphology, and a compensatory pause after the beat.

- *Ventricular Premature Complex (VPC):*

Ectopic beats in ventricular tissue – no P wave associated with the QRST waveform, and QRS is wide and bizarre because it travels through slow/inefficient ventricular myocardial tissue instead of fast/efficient conduction pathways. A single ectopic focus results in uniform VPC morphology, but VPCs originating from multiple ectopic foci are characterized by varied VPC morphologies (multiform VPC), suggesting more significant pathology and greater potential for instability and progression to severe arrhythmias. The presence of VPC's at rest or during exercise is

always a problem and deserves to be further investigated to assess the underlying causes and degree of structural and functional damage in the heart. They are most commonly associated with toxin exposure (e.g. monensin/ionophores, rattlesnake venom), significant myocardial injury, and/or severe heart disease. They can also occur concurrently with significant systemic compromise, such as SIRS, hypotension, strangulating GI lesions (colon torsion), prolonged general anesthesia, and other severe systemic illness. The presence of VPC's always requires further investigation, ideally with echocardiography to fully assess cardiac structure/function, as well as serum cardiac troponin measurement. Holter monitoring can also be useful to better characterize the frequency of VPC's over 24 hours, as this is more indicative of true frequency than a one-time evaluation with surface ECG. Heavily exercised horses may demonstrate VPC's in the immediate recovery period, but this should still prompt further investigation and restricted athletic use until better characterized by Holter monitoring at rest and during exercise.

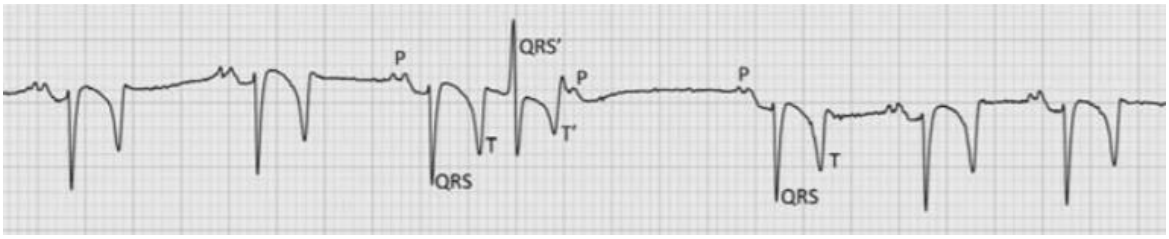


Fig. *8. Ventricular Premature Complex: Base-Apex lead, recorded at 25 mm/sec. Note the premature beat, with no visible P wave, a bizarre QRS morphology, different T wave morphology than sinus beats, and a compensatory pause after the beat. Source: Verheyen, et. al. 2010.*

Ventricular Arrhythmias

On auscultation, ventricular arrhythmias typically cause a rapid heart rate, faster than would be expected based on the clinical presentation (pain, dehydration, etc.). They are caused by ectopic beats firing repeatedly from within the ventricular tissue, and require immediate ECG characterization and medical interventions. They may be paroxysmal (intermittent periods, followed by normal sinus beats), or continuous and ongoing.

- *Accelerated Idioventricular Rhythm (AIVR):*

  Also known as "slow VTach," AIVR is characterized by runs of greater than 3 VPC's consecutively, at a rate greater than sinus rhythm and less than that of VTach. There is no discrete cut-off in horses, however AIVR is generally considered to have a rate between 40–100 beats per minute in horses. Idioventricular rhythm refers to ectopic ventricular escape rhythm with a rate of 20-40 bpm, however when the rate is accelerated and between 40-100 bpm, it is called AIVR. AIVR results from enhanced automaticity of the subordinate pacemaker tissues (His-Purkinje system, or closely associated ventricular myocardial tissue). During AIVR, the passive inward flux of potassium and calcium that drives gradual diastolic depolarization (phase 4) is accelerated by increased sympathetic tone, effects of certain drugs, and electrolyte imbalance (especially potassium, calcium, and

magnesium). This results in a more rapid depolarization at the level of subordinate pacemaker tissues than at the intrinsic rate of the SA node, resulting in dominance by the ectopic rhythm (AIVR). For this reason, there can be intermittent wandering P waves, intermittent VPC's, and fusion of VPC's with sinus beats during the onset and cessation of the AIVR waveform, as the two pacemakers compete for dominance.

AIVR, due to its slower rate, allows for greater ventricular filling compared to VTach and does not generally result in hemodynamic compromise. However, if a horse with AIVR shows clinical signs of poor perfusion or hypotension, antiarrhythmic therapy is warranted. Typically, AIVR develops from extra-cardiac causes, including systemic illness (i.e., SIRS, imbalanced sympathetic or parasympathetic tone, acid-base disturbances, or electrolyte imbalance), or induced by drugs that decrease the sinus discharge rate while increasing sensitivity to catecholamines (i.e., α-2 agonists and halothane). AIVR spontaneously resolves and normal sinus rhythm is restored as the systemic disorder as a cause of AIVR is corrected.

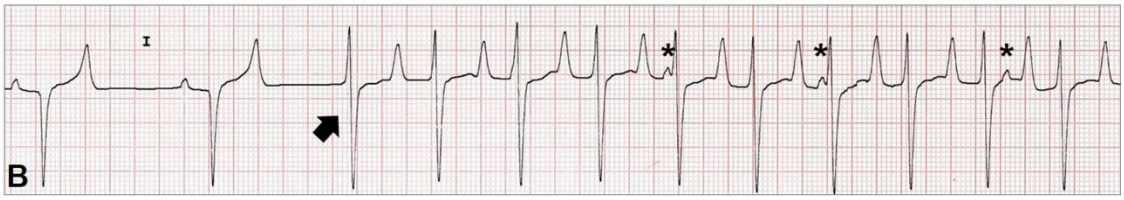


Fig. 9. Accelerated Idioventricular Rhythym: Base-Apex lead, recorded at 25 mm/sec
Note the transition from sinus rhythm (40 beats/min) to continuous run of VPC's (arrow), at a rate of 48-60 beats/min. Asterisks denote P waves overlaid on the AIVR arrhythmia, as there is complete dissociation of the atria and ventricles. This arrhythmia resolved after correction of severe electrolyte derangements (hypokalemia and hypomagnesemia).

- *Ventricular Tachycardia (VTach):*

  Characterized by runs of greater than 3 VPC's consecutively, when the myocardial ectopic focus takes on automaticity and results in a ventricular rate significantly greater than sinus rhythm (typically > 100 bpm in the horse). This results in pulse deficits and decreased cardiac output, as ventricular contraction occurs too rapidly for appropriate filling of the ventricles (lack of preload). Just as with VPC's, in monomorphic VTach, a single ectopic focus results in uniform QRS morphology, and in polymorphic VTach, there are multiple ectopic foci resulting in varied QRS morphology (amplitude, duration, polarity). VTach originating from multiple ectopic foci suggest more significant pathology and greater potential for instability and progression to ventricular fibrillation and sudden death. ECG classification can be easier when the QRS waveforms are clearly wide and bizarre, with complete AV dissociation and wandering P waves overlaying the ECG tracing. However, at times, it can be difficult to distinguish SVT from VTach, either because as the P' wave (from the ectopic atrial beat in SVT) is overlaid on the previous QRST, and/or when the ectopic beat is from the junctional region and QRS morphology

is not as wide and bizarre. These cases may require more advanced diagnostics (intracardiac ECG recording) and/or careful therapeutic trials to differentiate- all things to be attempted at a referral center.

Two important ECG morphologies to note are R-on-T phenomenon and torsades de pointes. When an ectopic beat is overlaid during the T waveform from the previous QRS complex, this is occurring in the vulnerable period for the ventricle. Specifically, the ventricle has not fully repolarized yet (relative refractory period) but is still capable of being depolarized again by a strong enough stimulus (ectopic beat), resulting in disorganized contractions and re-entry circuits, and high likelihood of degenerating into fatal dysrhythmias. Typically, this triggers torsades des pointes, where the QRS complexes fluctuate around the baseline to form a sinusoidal pattern, and if not corrected will progress to ventricular flutter and fibrillation.

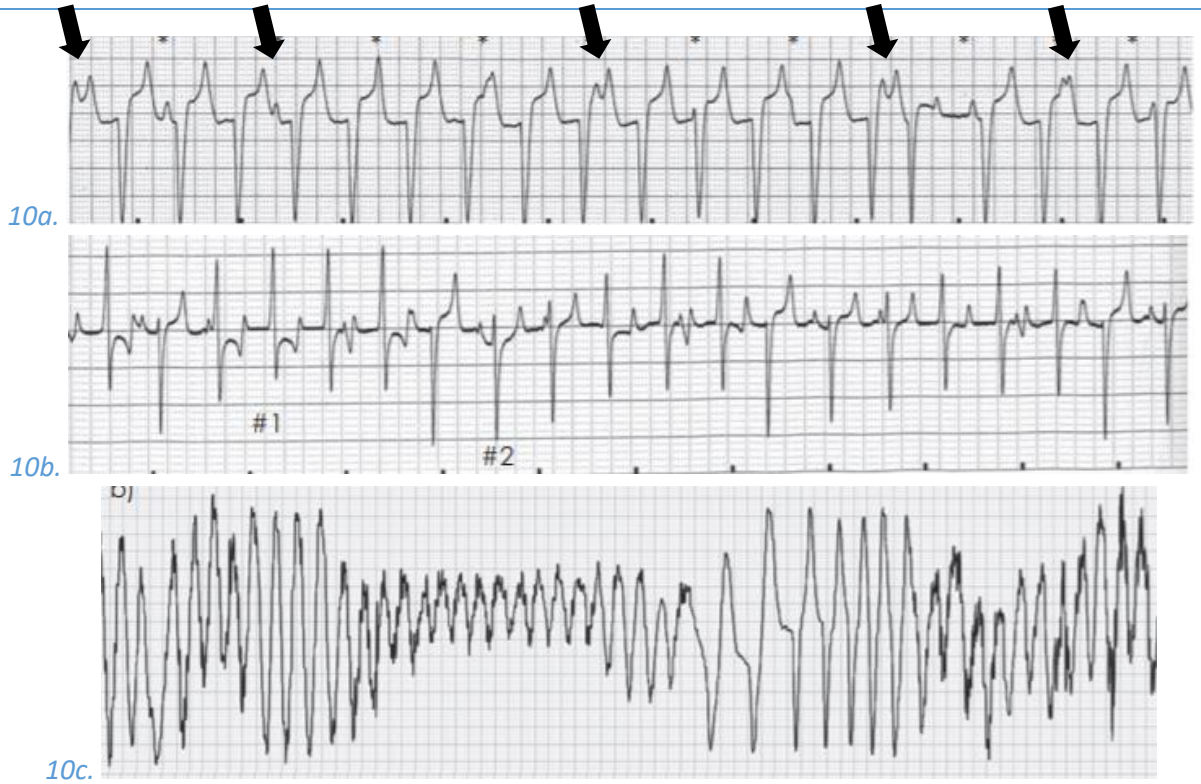


Fig. 10. Ventricular Tachycardia: Base-Apex lead, recorded at 25 mm/sec.
Fig. 10a. Monomorphic VTach, with regular ectopic VPC's of uniform morphology. P waves are intermittently overlaid on rhythm (arrows). Fig. 10b. Polymorphic VTach, with regular, rapid beats from two distinct ectopic foci (#1 + #2) . Fig. 10c. Torsades des pointes, with sinusoidal fluctuation around the baseline. This patient rapidly transitioned to ventricular fibrillation. Source: Mitchell, K., 2017.

VTach can be caused by primary myocardial disease or secondary to severe systemic insult, such as hypoxia, hypotension, or SIRS/MODS, that causes collateral myocardial damage. They often demonstrate signs of mild to moderate heart failure, but these signs may be masked by or concurrent with other clinical signs associated with co-morbidities. Regardless of the cause, VTach results from significant myocardial compromise, and is a true life-

threatening emergency. It necessitates immediate treatment for its potential to progress to ventricular fibrillation, typically with intravenous lidocaine and magnesium sulfate therapy as first line treatment as well as sotalol. Therapeutic lidocaine dosing for VTach consists of intravenous boluses of 0.5 mg/kg every 5 minutes up to 1.3 - 1.5 mg/kg, followed by CRI of 0.05 mg/kg/min. Therapeutic dosing of magnesium sulfate for VTach consists of intravenous administration of 2-6 mg/kg/minute to effect, up to a total dose of 55-100 mg/kg. Without primary anti-arrhythmia treatment, VTach should not resolve spontaneously. After correction of VTach by medical therapy and immediate stabilization of the patient, a complete cardiac evaluation is required to assess structural and functional status of the heart with echocardiography and evaluation of cardiac troponin I concentration.

Management of Arrhythmias

Triage Measures:

Emergent or not?

Most important component is to decide whether the identified arrhythmia is an emergency or can be monitored. It is important to note that if you are not sure or unable to characterize with an ECG, it is better to contact the AUCVM immediately for further instructions.

- *Supraventricular Tachycardia:* Not emergent, but it is certainly not normal and does need to be evaluated and addressed in the short term. Echocardiography is required to differentiate lone AFib from atrial enlargement or other structural/functional changes driving the arrythmia. It is important to pursue treatment as early in the course of disease as is feasible, because correction of ectopic atrial arrythmias is relatively time-sensitive for optimal long term good outcomes. Long-standing atrial ectopic rhythms result in remodeling of the tissues, which negatively impacts the chances for successful permanent conversion to normal sinus rhythm.

- *Premature Complexes:*
  - APC's: Not emergent, unless they are happening very frequently or there is concern regarding concurrent cardiac pathology (concurrent murmur, signs of exercise intolerance, signs of cardiovascular compromise). If benign neglect is elected, it is recommended to recheck auscultation and ECG in 4-6 weeks to reassess frequency of APCs and any changes to cardiac auscultation.
  - VPC's: Emergent- need to evaluate for myocardial damage and address immediately, as VPCs can progress to life threatening arrhythmias (VTach and ventricular fibrillation/sudden death).

- *Ventricular Arrhythmias:*
  - AIVR: Not truly emergent, although the horse may have significant systemic illness that may be emergent or have life threatening consequences. It is critical that you are certain that the runs of VPCs are AIVR before pursuing systemic therapy to correct underlying cause, and not primarily treating the

arrhythmia with anti-arrhythmic medications. Historical cause to suspect concurrent heart disease, loud murmur, or signs of cardiac compromise should be cause for concern and may require more aggressive, emergent treatment and further cardiac diagnostics.

- VTach: Emergent- need to treat immediately as this is a life-threatening emergency. Lidocaine and magnesium sulfate are first line therapy for VTach, and magnesium sulfate is the treatment of choice for torsades de pointes. These drugs can be given concurrently, but lidocaine should be prioritized.
  - LIDOCAINE: IV boluses of 0.5 mg/kg given every 5 minutes up to 1.3 - 1.5 mg/kg, followed by CRI of 0.05 mg/kg/min

    *** For a 500 kg horse, this is a 250 mg IV bolus every 5 minutes up to 3 times ***
  - MgSO4: IV infusion of 2-6 mg/kg/minute to effect, up to a total dose of 55-100 mg/kg

    *** For a 500 kg horse, this is 27 gm diluted in 1 Liter of 0.9% saline over 10 minutes ***
  - For further treatment recommendations, see KJ Mitchell's 2017 EVE article on VTach

Determining the Cause(s) of Pathologic Arrhythmia

Systemic Illness

Complete history and physical examination may reveal signs of systemic illness that are contributing to the presence of an arrythmia. Arrhythmias have been linked with hypovolemia, SIRS, strangulating intestinal lesions, vasculitis, immune mediated disease, intoxications, and many other systemic illnesses. Evaluation of plasma/serum electrolyte concentration is essential, including calcium and magnesium, because derangements in electrolyte status can drive pathologic arrhythmias due to the effects on excitable myocardial tissue. For some ectopic arrhythmias such as APC's or AIVR, correction of these systemic abnormalities will improve or eliminate the arrhythmia.

Cardiogenic Causes

Clinical signs of cardiovascular compromise may be secondary to the arrythmia present, but may also point to a primary cardiac disease that has generated the presenting arrhythmia. These clinical signs may be subtle or overt, and include exercise intolerance, agitation or lethargy, ventral edema, jugular distension or pulses, increased respiratory effort, poor pulse quality, tachypnea, coughing, and fluid overload within the lungs (crackles on auscultation, foamy nasal discharge). Identification of an arrhythmia concurrent with murmur is more suggestive of cardiac origin and worthy of further investigation. Echocardiography is required to assess cardiac structure and function in the horse, and should be the next step for evaluation of the arrhythmia. Cardiac troponin I, an intracellular enzyme specific to cardiac myocytes, is released into the circulation with myocardial damage. Cardiac troponin I activity can be measured in serum to evaluate myocardial damage, and can be a useful adjunct test.

When to Refer for Further Evaluation

First and foremost, referral for cardiology workup is warranted if the clinician is unsure of the diagnosis or concerned about the stability of the patient. However, it is critical to contact the AUCVM immediately and prior to transport, as there may be additional diagnostics to perform and life-saving interventions to administer prior to transport. Additionally, referral is recommended with certain arrhythmias, as in the case of SVT, VPCs, or VTach, to evaluate the structure and function of the heart through echocardiography. Cardiac evaluation is also recommended if seeing clinical signs of heart failure, unexplained exercise intolerance, concerns regarding syncopal episodes, concurrent murmur + arrhythmia of any kind, or if prepurchase examination requires adjunctive diagnostics. The AUCVM Equine Internal Medicine Service & Cardiology Services work together to address the specific needs of each patient in both outpatient and emergent scenarios. They can be reached 24 hours a day, 7 days a week at 334-844-4490.

References

Clinically applicable references for recommended further reading are bolded below:

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