

XXIII



TAGUNG ÜBER PFERDEKRANKHEITEN

mit begleitender Fachausstellung /
with associated trade exhibition

im Rahmen der EQUITANA
Essen, 15. und 16. März 2019

Arrhythmie
Kolonobstipation
Arthroskopie
Stammzelltherapie
Antibiotikatherapie
Halswirbelsäule

Investoren in der Pferdemedizin: „zum Wohle der Tiere“?



FREITAG, 15. MÄRZ 2019*
FRIDAY, 15 MARCH 2019

* Sprache: Englisch/Deutsch mit Simultanübersetzung
Language: English/German with simultaneous translation

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* Simultanübersetzung der englischen Vorträge ins Deutsche

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CLINICAL RELEVANCE OF CARDIAC ARRHYTHMIAS

van Loon, G.

Cardiac arrhythmias are commonly found in horses. Their clinical relevance depends on the haemodynamic effect (blood pressure and cardiac output) and the risk of deterioration into electrically unstable rhythms. An ECG is needed for accurate diagnosis.

The first step should be to identify physiological and therefore clinically insignificant arrhythmias. These are usually related to vagal tone and should disappear immediately upon a change from a vagal to a more sympathetic predominance. Second degree atrioventricular block is a typical example, whereby at regular time intervals a P wave is not followed by a QRS complex, resulting in a regularly irregular rhythm on auscultation. First degree atrioventricular block indicates a slower atrioventricular conduction with a prolonged PQ interval. Finally, sinus block or arrest or sinus arrhythmia may be present, whereby the sinus node depolarization is irregular or occasionally fails to conduct to the atria. All these arrhythmias should immediately disappear upon a change to a more sympathetic predominance (stress or exercise). Besides at rest, sinus arrhythmia in horses is most commonly found during recovery from exercise, upon a change from sympathetic toward parasympathetic tone. This arrhythmia should disappear as soon as the heart rate returns to normal. All of the above-mentioned arrhythmias are generally physiological and of no clinical importance.

Pathological arrhythmias may affect performance or may even carry a risk for collapse or sudden death due to their effect on haemodynamics (due to bradycardia or tachycardia) and electrical instability. Ventricular arrhythmias have a larger impact on cardiac function compared to atrial arrhythmias. Cardiac troponin I (or T) should be measured and ultrasound should be performed in order to identify myocardial disease.

Clinically, the most important arrhythmia that affects performance is atrial fibrillation (AF). Typically, an irregularly irregular rhythm is present on auscultation. The ECG characteristics include replacement of P waves by fibrillation waves, irregular RR intervals and QRS complexes that generally have a normal morphology. Once initiated, AF usually becomes permanent very quickly, which means that it will not spontaneously terminate. In the absence of underlying cardiac disease, the arrhythmia is well tolerated at rest, but may affect performance. In some horses, during exercise, AF may result in more severe clinical signs such as collapse. Premature atrial beats are visible on the ECG as an early P' wave that, depending on its prematurity, may or may not be followed by a QRS complex (Fig. 1). Premature atrial contractions are generally not associated with poor performance and do not carry a risk during exercise. Frequent occurrence of premature atrial beats, especially in large horses, inducing AF and therefore affecting performance, should be taken into account.

Most importantly, ventricular arrhythmias include occasional isolated ventricular depolarizations (VPDs) and ventricular tachycardia (VT) (more than 3 VPDs in a row). VPDs are visible on the ECG as QRS complexes with abnormal morphology and duration, which occur too early and have no relation to a preceding P wave (Fig. 2). Isolated VPDs are usually followed by a compensatory pause. Frequent occurrence of VPDs increases the risk for initiating fast VT that could further destabilize into ventricular fibrillation, which is a fatal event. Occasional isolated VPDs in absence of underlying structural disease probably carry a relatively low risk. Furthermore, the presence of isolated VPDs at the end of strenuous exercise or during recovery can be regarded as 'normal'. However, presence of underlying myocardial disease or clinically significant aortic regurgitation, or VPCs that increase in number during exercise, may carry a significant risk. Slow VT, also called idioventricular rhythm, is recognized by regularly occurring QRS complexes with an abnormal morphology which are not related to a previous P wave, usually with a heart rate of 60-80 bpm. Slow VT is regarded as benign as long as no underlying cardiac disease is present and the arrhythmia disappears during exercise. Rapid VT, however, often at rates of 150 bpm or more, has a large impact on cardiac output and will result in clinical signs of exercise intolerance, weakness or even collapse. The rhythm may deteriorate into electrically unstable rhythms such as torsades de pointes and ventricular fibrillation. Compared to monomorphic VT (Fig. 3), polymorphic VT indicates more widespread myocardial disease and an even higher risk to become unstable. Affected horses should be rested, any underlying disorder such as electrolyte or acid-base disorders, systemic disease, etc. should be treated and anti-arrhythmic therapy should be given.

Finally, bradyarrhythmias such as advanced second degree or third-degree atrioventricular block are associated with exercise intolerance and repeated collapse. During advanced second-degree atrioventricular block, more than two successive atrioventricular blocks are present (several P waves without QRS complex on the ECG) and often there is an impaired heart rate response during exercise. During third degree atrioventricular block there is a total dissociation between the atrial and ventricular rhythm whereby the ventricles rely on their slow intrinsic rhythm. Heart rate response during exercise is usually severely impaired. The severe bradycardia usually leads to multiple episodes of collapse. In the absence of infection, steroid administration might be attempted in order to reduce peri-nodal inflammation and improve atrioventricular nodal conduction. Sympathomimetic or parasympatholytic drugs may be attempted, but they are often unrewarding. Permanent pacemaker implantation is usually indicated.

In conclusion, after diagnosis of an arrhythmia, clear differentiation between a physiological and pathological arrhythmia should be made and ECG recording is needed for reaching a diagnosis. Subsequently, the effect on haemodynamics and performance should be assessed. It is very important to realize that certain arrhythmias may become electrically unstable and as such carry a potential risk for horse and rider. This risk is difficult to quantify but presence of underlying structural disease should be regarded as an important risk factor, especially for ventricular arrhythmias.

Figure legends

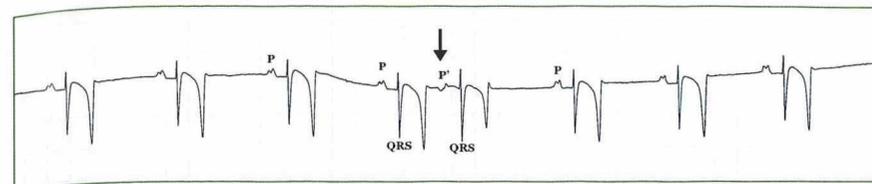


Figure 1: an atrial premature depolarization (arrow) shows on the ECG as an early P' wave which may have a different morphology and which may be followed by a QRS complex, depending on its prematurity.



Figure 2: a ventricular premature depolarization (arrow) occurs as an early QRS complex with abnormal morphology and duration followed by a compensatory pause. The premature depolarization has no relation to a preceding P wave.

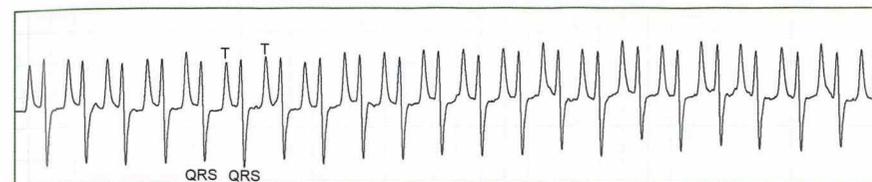


Figure 3: monomorphic ventricular tachycardia at a ventricular rate of 180 bpm is characterized by rapidly and regularly occurring QRS-T complexes that show abnormal QRS duration and presence of one abnormal QRS morphology. P waves are partially superimposed on QRS and T and therefore difficult to identify.