Evaluation of heart rate and rhythm during exercise

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Summary

Heart rate and rhythm can readily be monitored during exercise. Although there is considerable variation depending on the athletic discipline, exercise can be considered to be one of the most physiologically demanding times for the cardiovascular system. Assessment of heart rate during exercise typically provides information regarding fitness, the intensity of exercise and on some occasions may provide an early indication of disease. Cardiac causes of poor performance occur relatively infrequently in comparison with disorders of the musculoskeletal and respiratory systems. Nevertheless, exercise induces cardiac hypertrophy, which predisposes athletes to valvular regurgitation and arrhythmias. Consequently cardiac murmurs and arrhythmias are frequently present in equine athletes where upon the clinical significance can be difficult to determine. Undertaking an exercise test to identify exercise-induced arrhythmias is important in the assessment of poor athletic performance and the risk of sudden cardiac death during exercise. This paper describes the assessment of heart rate and rhythm during exercise. Most research has been undertaken on racehorses but where data is available for other disciplines this has been included. Considerations regarding the choice, type and design of exercise test were detailed in the first paper in this series.

Introduction – the athletic heart

The horse is considered a supreme athlete when compared to other species, due its superior aerobic capacity. The maximal oxygen capacity ($\dot{V}O_{2max}$) reached by the Thoroughbred racehorse during intense exercise is more than twice that of most elite human athletes (Jones and Lindsted 1993), with maximal values reported to be over 200 ml/kg/min (Evans

2007; Poole *et al.* 2011). This enhanced aerobic capacity is related to the superior cardiovascular capacity of this species. Cardiac output ($\dot{Q}$) is the major determinant of $\dot{V}O_{2max}$ (Poole 2004; Poole *et al.* 2011) and is in turn influenced by both heart rate (HR) and stroke volume (SV). Horses can experience almost a ten-fold increase in HR from rest to strenuous exercise, with maximal values of HR in the region of 210 to 240 bpm (Vincent *et al.* 2006). Training does not alter the horse's maximal HR (HR_{max}). However, the speed at which HR_{max} is reached does increase with training (Courouce 1999; Kobayashi *et al.* 1999; Vermeulen and Evans, 2006). Stroke volume is related to heart size and horses have large hearts when compared with other species, with values for heart mass of approximately 0.9% of body mass in untrained horses (Poole 2004). Heart size and thus SV also increase further with training (Young 1999; Buhl *et al.* 2005).

It has long been proposed that heart size is associated with athletic performance. Indeed, historically several of the greatest equine athletes have been reported to have extraordinarily large hearts, including Phar Lap, Secretariat, Mill Reef and Eclipse (Poole 2004). More recently, scientific evidence has confirmed associations of left ventricular size with both $\dot{V}O_{2max}$ and race performance (Young *et al.* 2002; Buhl *et al.* 2005; Young *et al.* 2005).

In both human and equine athletes, long-term athletic training is associated with structural and functional adaptations to the cardiovascular system. The cardiac remodeling that occurs with exercise results in eccentric hypertrophy, whereby there is an increase in the size of the cardiac chambers that is matched by an increase in the wall thickness (Young

1999; Cheng 2009). This "benign" physiological remodeling is often termed the 'athletic heart' (Maron and Pelliccia 2006). Differentiating athletic eccentric hypertrophy from pathological left ventricular remodeling arising from cardiac diseases is challenging in all species. In human medicine, advanced imaging techniques such as tissue doppler imaging and 2D speckle tracking show promise in distinguishing these conditions and might ultimately have value in horses where work is ongoing (Decloedt *et al.* 2014). There is increasing evidence of cardiac damage in athletes undertaking extreme exercise and this may explain why elite human athletes (Maron 2003; La Gerche 2013), athletic dogs (Bharati *et al.* 1997) and horses (Kiryu *et al.* 1999; Young 2013) may be more susceptible to arrhythmias and sudden cardiac death (SCD).

Equipment for measuring heart rate during exercise

The gold standard method for measuring heart rate during exercise is through the use of an electrocardiogram (ECG), which permits visualisation of the QRS complexes and enables calculation of the HR by assessment of RR intervals. A number of heart rate monitors (HRM) are also commercially available, which record inter-beat intervals and provide the reader with a calculated HR value. Most devices calculate an average over a 5 second interval of RR collection which can result in errors. Some models allow RR intervals to be exported which allows for more detailed analysis. HRMs are cheaper and very user friendly compared to ECG. Furthermore, modern HRMs can be linked to global positioning systems (GPS), so that speed can be measured concurrently. Most systems have been developed for human use, where they are commonly used to monitor training, and are adapted for use in horses. Compared with other indications of exercise intensity HR is easy to monitor,

relatively cheap and can be used in a variety of settings (Achten and Jeukendrup 2003). However, not all systems have been validated for use in the horse and although some studies have shown high correlation coefficients compared with ECG recordings, other studies have shown measurement related errors (Evans and Rose 1986; Sloet van Oldruitenborgh-Oosterbaan *et al.* 1988; Parker *et al.* 2010). Errors may be related to movement, loss of contact of the electrodes, or due to the high amplitude and variable T wave configuration in the equine ECG.

The HRM electrodes are usually placed such that the one electrode is positioned on the left side of the thorax, under the saddle, and the other electrode is fixed under the girth, over the left side of the heart or on the sternum. The coat should be wetted or electrode gel applied to ensure good electrical contact with the skin. In some cases it may be necessary to clip the area first if the coat is long. Data is transmitted to a watch (usually worn by the rider, or fixed to the horse). With most systems the information can be stored and downloaded on to a computer for later analysis.

Heart rate responses to exercise

Typically heart rate is monitored throughout the exercise session, including the warm-up and recovery periods. Typical heart rates at different levels of exercise are shown in table 1 and heart rates during competition in the different disciplines are shown in table 2.

An anticipatory response to exercise, leading to an increase in heart rate is seen in the equine athlete as in man (Fregin and Thomas 1983). In racehorses high heart rates have

been observed in the starting stalls and pre-exercise in horses undergoing clinical exercise testing on the trainer's own gallops (Krzywanek *et al.* 1970; Allen and Franklin 2010). Pre-exercise and submaximal HR elevations caused by excitement may interfere with the HR interpretation in the clinical setting.

In general there is a linear increase in HR with increasing speed up to HR_{max}. This is the highest value of the HR that can be attained by an individual during an incremental exercise test and is observed as a plateau in the HR despite further increases in exercise intensity. Peak heart rate is the highest heart rate obtained during an exercise test, and this term is used when it is unknown whether HR_{max} was achieved (figure 1). HR_{max} varies greatly between individuals and may be influenced by factors such as age, sex and breed (Vincent *et al.* 2006). When assessing HR and GPS data from racehorses a lag can often be seen whereby the peak HR is achieved after the peak speed (figure 1).

Heart rate recovery decreases in a bi-exponential manner (Rugh 1992) (figure 1). Recovery is usually very rapid in the first minute after cessation of exercise, followed by a slower decline towards resting levels.

Effect of training/ fitness and athletic capacity on HR responses to exercise

In human athletes numerous investigations have revealed an increase in SV as a result of training. This results in a corresponding decrease in HR at rest and during exercise (Warburton *et al.* 2008). Several studies have also demonstrated that the heart rate response to exercise may be influenced by fitness in racehorses (Couroucé 1999; Kobayashi *et al.*

1999; Vermeulen and Evans 2006). Although HR_{max} itself does not change with training, the speed at which a specific heart rate is reached does increase with training. In horses performing intense exercise V_{200} (velocity at a HR of 200bpm) and V_{HRmax} (velocity at maximal HR) may be used; whilst for horses performing less strenuous exercise parameters such as V_{180}, V_{170} or V_{140} (velocity at HR's of 180, 170, 140) may be more appropriate. In racehorses and eventers when the discipline demands that exercise is undertaken close to aerobic capacity, V_{200} and V_{HRmax} are the most useful indicators of fitness and performance. However, for horses engaged in less aerobically demanding sports such as show jumping and dressage these HR's are not attained, therefore V_{180}, $_{170}$ and $_{140}$ are recommended. The disadvantage of these parameters is that these values are far more susceptible to psychological influences on the HR. Repeated measures of any of these V_{HR} parameters in the same horse can be used to demonstrate an increase in fitness during a training program (Vermeulen and Evans 2006).

Individuals with the greatest athletic capacity will reach HR_{max} at the greatest speeds and a correlation has been shown between V_{HRmax} and race earnings (Gramkow and Evans 2006) although other studies have not found V_{HRmax} to change with either fitness or to be a reliable indicator of performance potential in racehorses (Fonseca *et al.* 2010). It is possible that there may be confounding effects of track condition, rider / driver and distance exercised which mask the effect of training and athletic ability on the measured HR parameters and hence it is recommended that these variables also be accounted for in field studies.

In human athletes, heart rate recovery is routinely monitored to assess fitness, with faster recovery rates occurring in highly trained individuals (Hagberg *et al.* 1980; Borreson and Lambert 2007). Although it seems intuitive that the same would be true for the horse, the evidence is conflicting. Seeherman and Morris (1991) found no evidence that HR recovery changes with increasing fitness. However, other studies in racehorses have found that post exercise HR recovery is improved with training (Foreman *et al.* 1990; Hada *et al.* 2006).

Clinical significance of abnormal heart rate responses to exercise

Identification of an abnormal heart rate response during an exercise test is a non-specific finding. Abnormalities should be considered as a 'warning sign' that warrants further investigation and is not diagnostic of cardiac disease per se. Abnormal heart rate responses to exercise have been noted with arrhythmias, in particular atrial fibrillation (Deegen and Buntenkotter 1976; Verheyen *et al.* 2013). When valvular dysfunction or structural cardiac disease results in cardiac decompensation, affected horses show an increased HR during exercise because of failure to maintain forward stroke volume, before clinical signs of heart failure become evident at rest. However, abnormal heart rate responses to exercise are not solely related to cardiac disorders and more often are associated with a variety of factors, including: excitement, lack of fitness, respiratory disease, lameness, pain, anaemia, environmental factors, dehydration, high body condition score or a physiologically inferior horse (Littlejohn *et al.* 1977; King *et al.* 1994; Naylor *et al.* 1993; Couroucé 1999).

There are typically four situations when the heart rate response might be considered abnormal.

1. Abnormally high submaximal heart rates:

   In addition to anticipation of exercise, elevated submaximal heart rates may be due to inappropriate fitness, dehydration, hot conditions, lameness, pain, respiratory or cardiovascular disease.

2. Abnormally high peak or maximal heart rate:

   The most common condition causing supra physiological heart rates (>250bpm) is atrial fibrillation (paroxysmal or sustained).

3. Abnormally low maximal heart rate:

   In human athletes failure of the heart rate to increase appropriately is presumed to be due to ischemic dysfunction of the sinoatrial node (Cooper and Storer 2001). Low maximal heart rates are not well reported in horses and care should be taken to ensure that the exercise was sufficiently strenuous for HR_{max} to be reached.

4. Abnormally high or prolonged heart rate in recovery:

   Lack of fitness may result in prolonged HR recovery. Poorly performing horses have also been reported to show higher post exercise heart rates than good performers, with a prolonged slow phase of recovery (Cardinet *et al.* 1963; Bitschnau *et al.* 2010; Madsen *et al.* 2014). Dehydration appears to be the most likely cause of prolonged HR recovery in endurance horses (Naylor *et al.* 1993). In human patients, HR recovery is used as a prognostic indicator for cardiovascular disease (Cole *et al.* 1999; Smith *et al.* 2005) and in horses, prolonged HR recovery is associated with atrial fibrillation (figure 2).

It is also necessary to consider whether other factors, such as anxiety and excitement are influencing the heart rate particularly if an unfamiliar place or unfamiliar exercise is used. For example, lunging can elicit high HR in otherwise normal horses if they are unfamiliar with being lunged. Furthermore in any situation that an unexpectedly high or low heart rate was obtained it is also important to consider whether the equipment was functioning correctly.

Cardiac arrhythmias

In addition to HR measurements, electrocardiography (ECG) is also commonly performed during exercise in order to assess electrical activity of the heart and diagnose arrhythmias that occur during or immediately after exercise.

There are several indications for undertaking an ECG during exercise:

(i) to evaluate whether an arrhythmia detected at rest goes away with exercise

(ii) in conjunction with echocardiography in horses with valvular regurgitation. For example, horses with aortic regurgitation may develop dilation of the left ventricle. This results in an increased myocardial oxygen demand when there is concurrent reduced coronary perfusion due to reduced diastolic pressures. This can lead to ventricular ischaemia and hence ventricular ectopy during exercise and the predisposition to exercise associated collapse and/or SCD. The exercising ECG provides important information about the safety for continued ridden exercise. Similarly, advanced atrioventricular valvular regurgitation which results in atrial enlargement can predispose the horse to atrial ectopy.

(iii) in horses with sustained atrial fibrillation to ensure that the heart rate response to exercise is appropriate and that horses do not also have concurrent ventricular arrhythmias which may jeopardize rider safety, especially where cardioversion is not pursued (Reef *et al.* 2014)

(iv) as part of a complete investigation of poor performance of unknown origin.

Equipment for recording ECG during exercise

Exercising ECG may be readily performed using portable devices. Electrode placement may vary between clinicians and different devices. In the horse precise location is of limited importance provided that the positive electrode is below and slightly caudal to the heart and the negative above and slightly cranial. Ideally the lead position allows some room for the girth to slip back and isn't interfered with by rider's hands or legs. If the electrodes are placed in a visible position the examiner will be able to see if they have been dislodged.

The authors use a modified base-apex electrode placement during exercise in order to reduce movement artifact. The negative right arm (usually red) electrode and earth electrode (usually black) are placed on the left proximal scapula. The positive left leg (usually green) electrode and left arm (usually yellow) are placed at the cardiac apex (on thorax level with olecranon) (figure 3). This gives identical traces in lead I and II. However if it is possible to separate the left arm and left leg electrodes so they are 10 to 15cm apart this allows two subtly different leads to be recorded which helps recognition of artifact by reducing the risk of both electrodes being simultaneously knocked by the riders leg. During

lunging the absence of a rider results in less interference and the right arm electrode could be placed on the right scapula. Whichever positioning is used it is imperative that the electrodes are placed prior to exercise, as if the coat is moist with sweat they will not adhere.

Interpretation of exercise ECG recordings

Electrocardiography in horses provides information on heart rhythm and not on myocardial function. It is important to note that equine ECG measurements are not indicators of heart size and should not be used for performance prediction. In the horse, depolarization is different to that in humans because of the widespread distribution of the Purkinje network, therefore QRS duration does not accurately reflect cardiac size (Muylle and Oyaert 1977; Verheyen *et al.* 2010).

The electrode configuration described above typically gives a bifid P wave and large QRS complexes. T waves are very labile and change in size and polarity with alterations in HR and sympathetic tone. Consequently analysis of their changes during exercise has no clinical value.

During exercise the intervals between PR and QT shorten but there is little change in the duration of the QRS complex (figure 4). An increase in T wave amplitude is seen and as heart rates progressively increase the P waves disappear into the preceding T waves (figures 5 and 6). Slight changes in the amplitude of the QRS can occur due to respiration.

ECG computer analysis will permit detection of the variation in RR intervals. When HR is very high during intense exercise, definition of a premature complex requires understanding of RR variability (Physick-Sheard and McGurrin 2010). Generally at high heart rates there is only slight variation in RR intervals and the authors typically use a setting on automated software of 8% during exercise.

Identifying arrhythmias on the ECG

Firstly it is important to review the ECG trace to ensure that it is of diagnostic quality. Although computer programs are readily available, it will always be necessary to review the entire trace manually. It is then important to determine the appropriateness of the heart rate and to determine whether the rhythm is regular or irregular.

Normal irregular cardiac rhythms

Classification of arrhythmias is usually based upon the origin of the complex. When the complex originates from the sinus node and passes through the normal conduction pathway it is considered to be normal sinus rhythm. However these sinus rhythms can be influenced, particularly by parasympathetic tone, causing alterations of conduction. Generally speaking the following arrhythmias are physiological and are a reflection of a high vagal tone in an individual horse when they will not be associated with clinical signs. They are most commonly seen before and immediately after exercise. When physiological arrhythmias are detected at rest they will disappear during exercise as sympathetic tone increases. Rarely these arrhythmias can also result from disease in the heart or of the

conduction system where upon they will be associated with dramatic clinical signs and profound bradycardia.

Irregularly irregular rhythms

- Sinus arrhythmia

A phasic increase and decrease in the PP interval is seen (figures 7 and 8).In contrast to other species, sinus arrhythmia is not associated with ventilation in horses. It is most commonly seen in the immediate recovery period after moderate or strenuous exercise particularly if the horse is brought back to halt or walk very quickly. In this context the rhythm probably reflects non-linear return of parasympathetic influence

Regularly irregular rhythms

- Sinus block

The normal sinus impulse is blocked at the sinus node resulting in a regular pause that is twice the preceding PP interval (figure 9).

- Second degree AV block

The ECG shows an isolated P wave that is not associated with a QRS complex because the impulse is blocked at the AV node (figures 10 and 11).

Abnormal irregular cardiac rhythms

Although occasional premature complexes at rest and at certain times during exercise or recovery may be considered clinically acceptable, the following complexes or rhythms do not originate from the sinus node and therefore from an electrophysiologic perspective are abnormal. These arrhythmias may be of clinical significance because they can cause poor performance or may predispose to collapse and/or SCD.

- Atrial fibrillation

Atrial fibrillation can be sustained or paroxysmal. The ECG shows irregularly irregular RR intervals, the absence of P waves and presence of F waves. As the name suggests sustained atrial fibrillation is present continuously and therefore can be detected on clinical examination and confirmed on a resting ECG (figure 12). Whereas the onset of paroxysmal atrial fibrillation (PAF) is typically during or immediately after strenuous exercise (figure 13) and generally most horses revert spontaneously back to sinus rhythm within 24-48 hours. However horses have spontaneously reverted as long as one week after the onset of PAF.

- Supraventricular premature complex (SVPC)

A SVPC (also known as atrial premature complex) is an early beat originating from the atrial myocardium. As the premature complex does not originate from the sinus node a difference in P wave morphology can sometimes be detected. If the complex is very premature the P wave may remain hidden in the preceding QRS or T wave. Furthermore

because of strong vagal influence on the AV node in the horse, it is also not unusual for the premature P waves to be blocked at the AV node.

Typically the morphology of the QRS complex is the same as that of sinus beats (figures 14 and 15), which is useful when trying to differentiate between SVPCs and VPCs. However, there are times when subtle alterations in the QRS or T may be seen with a SVPC and these should not be confused with VPCs. If the P wave of the premature complex coincides with the preceding QRS or T wave, this may cause a slight change in the configuration of the QRS or T. Also for very closely coupled beats the QRS of the premature beat can on occasion be taller than the sinus complexes (Broux *et al.* 2013), however they retain the same basic morphology, which generally aids in differentiation with VPCs.

SVPCs are associated with what is classically termed a non-compensatory pause. This occurs when the premature complex depolarizes the sinus node, following which it is reset and then resumes at its previous rate. The non-compensatory pause is less useful in horses for differentiating SPVCs and VPCs because on many occasions the sinus node resumes at a slower rate.

Supraventricular (atrial) tachycardia is defined as a run of 4 or more SVPCs.

- Ventricular premature complex (VPC)

A VPC is an early beat which originates from the ventricular myocardium or conducting system and the QRS morphology typically differs from that of sinus beats (higher amplitude/longer duration/abnormal morphology) (figure 16). The origin of the VPC dictates the morphology of the QRS complex. When the premature complex originates in the ventricular myocardium the QRS morphology appears most wide and bizarre. A junctional premature complex is a premature beat arising in the atrioventricular junction or His-Purkinje system. If the premature complex originates high in the Purkinje network it will be conducted using the normal pathway, and may not appear wide and bizarre, however typically there is a small change in the R or S wave of the QRS complex.

VPCs are usually followed by a compensatory pause, which is often the easiest thing to spot on the ECG. As the electrical impulse cannot pass retrograde up the AV node the sinus node is isolated from the VPC and consequently its rate of firing is unchanged. As a result a P wave occurring at the normal rate may be visible within the premature QRS or diastolic interval. This P wave is not conducted because the ventricle is refractory (from the VPC) when the impulse arrives at the AV node. Consequently the next sinus impulse should occur at exactly double the normal RR interval.

However, the presence of a compensatory pause is not truly diagnostic for VPCs. A SVPC may also result in a compensatory pause if it depresses the sinus node instead of resetting it. Furthermore in horses, interpolated VPCs can occur. This occurs when the VPC is so closely coupled that the next sinus impulse can be conducted because the ventricles are no longer refractory. In this situation a VPC with no compensatory pause will occur. Therefore

it is advised that more weight is placed on identification of abnormal QRS morphology (Conover 2002) when trying to differentiate between SVPCs and VPCs.

VPCs can occur as isolated beats, couplets or triplets (figure 17). Four or more consecutive VPCs are termed ventricular tachycardia (figure 18).

From an electrophysiological perspective the T wave represents a vulnerable period for myocardial cells during which time ventricular fibrillation can more easily be triggered. Consequently when VPCs are superimposed on the preceding T wave (R on T phenomenon) the rhythm is considered highly unstable because there is a high risk of ventricular fibrillation developing which may lead to SCD.

Clinical significance of arrhythmias occurring during exercise and the immediate recovery period.

Arrhythmias have been reported in both normal horses and poorly performing horses during exercise. Although for some arrhythmias, such as atrial fibrillation, the contribution to poor athletic performance is well understood, for others the clinical significance remains unclear. Interpretation of exercise-induced arrhythmias can therefore pose a significant dilemma to the veterinary surgeon. A recent joint ACVIM / ECEIM Consensus statement (Reef *et al.* 2014) has been released to guide veterinarians in the interpretation of exercising arrhythmias.

Strenuous exercise causes substantial increases in catecholamines, electrolyte disturbances, hypoxaemia, hypercapnia, lactic acidosis, hyperthermia and autonomic disturbances all of which can potentially contribute to cardiac arrhythmias. In both human and equine athletes, the immediate post exercise period appears to be the most vulnerable time for the genesis of cardiac arrhythmias and SCD. At this time there is a rapid decrease in heart rate due to parasympathetic reactivation and sympathetic withdrawal. Abnormal regulation of electrolytes and cardiac sympathovagal balance may increase the incidence of arrhythmias during this time period (Paterson 1996; Beckerman *et al.* 2005; Physick-Sheard and McGurrin 2010). In contrast to human athletes, most racehorses develop hypoxaemia and hypercapnia during strenuous exercise. The extent to which this hypoxaemia and hypercapnia are involved in arrhythmogenesis is unknown. With many forms of upper and lower respiratory disease, hypoxaemia and hypercapnia is further exacerbated, but the precise role of respiratory disease in cardiac arrhythmias is uncertain. Furthermore, hypoxaemia and hypercapnia are more evident in trained horses and horses with a higher $\dot{V}O_{2max}$, it is therefore also unclear whether elite equine athletes with higher $\dot{V}O_{2max}$ and larger hearts are the most at risk of developing arrhythmias.

Questions have certainly been raised in human athletes as to whether the athletic heart becomes more arrhythmogenic. In human athletes, arrhythmias are common and there is some research to suggest that certain arrhythmias are more common amongst trained athletes than their sedentary counterparts (Baggish and Wood 2011). There is also increasing evidence of cardiac damage and fibrosis in athletes undertaking extreme exercise and this may explain why elite human athletes may be more susceptible to

arrhythmias and SCD (La Gerche 2013). It is likely that a similar situation occurs in the equine athlete (Kiryu *et al.* 1999; Ryan *et al.* 2005, Lindholm *et al.* 2008; Young 2013).

Prevalence of arrhythmias during and after exercise

During exercise

Supraventricular premature contractions are the most common arrhythmias detected during exercise. They are more commonly observed during the warm up phase (Ryan *et al.* 2005) but have been reported to occur in 10 – 50% of TB and SB racehorses during strenuous exercise – either in training, racing or on the treadmill (Ryan *et al.* 2005; Jose-Cunnileras *et al.* 2006; Lindholm *et al.* 2008; Buhl *et al.* 2013). Isolated VPCs have also been reported, albeit less commonly, in 3 - 4.5% racehorses during strenuous exercise (Ryan *et al.* 2005; Jose-Cunnileras *et al.* 2006; Lindholm *et al.* 2008; Buhl *et al.* 2013).

In sport horses exercise ECGs are more difficult to interpret as speeds often vary, therefore the HR is more variable and strict definition of prematurity is more difficult. Isolated premature beats have been reported in both dressage horses and show jumpers (Barbesgaard *et al*. 2010; Buhl *et al.* 2010).

After exercise

Arrhythmias have been more commonly identified during the immediate post exercise period than during the exercise period itself. In one study of healthy Thoroughbred racehorses during training 15% had second degree AV block and at least 28% had sinus

arrhythmia in the post exercise period. Approximately 8% had VPCs or SVPCs in the post exercise period, and couplets, triplets and paroxysms were observed (Ryan *et al.* 2005). Similarly, following strenuous treadmill exercise 31% of horses had VPCs and 15% had SVPCs in the immediate recovery period. On average one SVPC (but up to 4) and 3 VPCs (but up to 30) were seen (Jose-Cunnileras *et al.* 2006). In studies of Standardbreds, rhythm disturbances were frequently noted immediately after racing, with 46-54% exhibiting one or more SVPCs (Lindholm *et al.* 2008; Buhl *et al.*, 2013) and 19-28% showing one or more VPCs (Lindholm *et al.* 2008, Buhl *et al.* 2013; Physick-Sheard and McGurrin 2010). In a recent study by Physick-Sheard and McGurrin (2010) complex ventricular arrhythmias were observed in 16% of horses. In about a third of horses, post exercise arrhythmias were associated with a sudden reduction in HR, suggesting autonomic instability and increases in vagal tone were contributing factors (Physick-Sheard and McGurrin 2010). The prevalence of atrial fibrillation (detected post-race) is reported to be 0.03% in Thoroughbred racehorses and 0.14% in Standardbred racehorses (Ohmura *et al.* 2003; Slack *et al.* 2014). Sinus arrhythmia, sinus block, second degree AV block, SVPCs and VPCs were also observed in sports horses during the recovery period.

Effect of arrhythmias on athletic performance

A reduction in cardiac output is the primary mechanism through which a cardiac arrhythmia might affect athletic performance. Atrial fibrillation (persistant or paroxysmal) is the most important arrhythmia affecting performance in athletic horses. In both cases, poor performance may arise because of the reduced cardiac output that occurs due to incomplete ventricular filling. The high heart rates that often occur during atrial fibrillation

500 further reduce the time for ventricular filling compounding the problem. Horses may be
501 able to successfully complete exercise at low intensities but are affected at faster speeds.
502 In cases where AF arises during strenuous exercise, horses may become uncoordinated and
503 "wobbly". This is likely due to the sudden decrease in $\dot{Q}$ and reduced O_2 supply to the
504 exercising muscles and/ or central nervous system.
505
506 The effect of supraventricular and ventricular premature complexes on performance is less
507 clear. Previously it was suggested that arrhythmias occurring during maximal strenuous
508 exercise or immediately after exercise were of clinical importance and a potential cause of
509 reduced performance (Martin *et al*. 2000). However, others have argued that it is unlikely
510 that isolated ventricular or supraventricular premature complexes would have a
511 considerable detrimental effect on cardiac output and hence performance and the
512 prevalence of SVPCs and VPCs in apparently healthy horses further reinforces this. One
513 study reported that the presence of premature depolarisations did not appear to be
514 associated with decrements in athletic performance during a strenuous treadmill exercise
515 test or decrements in race performance (Jose-Cunnileras *et al.* 2006). Similarly, it has been
516 reported that winning time was not associated with the probability of arrhythmias during
517 the post-exercise period in racing Standardbreds (Physick-Sheard and McGurrin 2010).
518 Whether post-exercise arrhythmias are an indicator of increased abnormalities such as
519 hypoxia or electrolyte disturbances which were present during the exercise period remain
520 to be investigated.

521 ***Risk of collapse / Sudden cardiac death***

Ultimately the risk of SCD in the horse during exercise is low; in Thoroughbred racehorses, rates of between 1-3 horse sudden deaths per 10,000 race starts are reported of which approximately 50% can be speculated to have an arrhythmogenic cause (Boden *et al.* 2006; Lyle *et al.* 2011). Irrespective of the relatively low frequency, when SCD occurs during high-profile competitions there may be intense media interest and a negative effect on public perception of horse sports. Sudden cardiac death during competition is thought to be most common in racehorses, but has also been reported in other disciplines including eventing and elite show jumping. Sudden cardiac death can occur during or immediately after strenuous exercise, but it is the immediate post exercise period that is considered to be a particularly high risk for both human and equine athletes (Physick-Sheard and McGurrin 2010). It has been proposed that the concerning exercise-induced arrhythmias may be lessened or avoided by a sustained and gradual warm down after exercise, which may help to moderate autonomic changes, rather than abruptly decreasing to walk (Physick-Sheard and McGurrin 2010).

Ventricular arrhythmias are assumed to be the primary cause of SCD in horses (Kiryu 1999). However, evidence is lacking as longitudinal studies are difficult to perform and post-mortem examinations are often unrewarding (Physick-Sheard and McGurrin 2010). In human athletes sudden cardiac arrest during exercise is most commonly associated with hypertrophic cardiac myopathy, a relatively common genetic disease, with an incidence of 1 in 500 in the general population (Maron 2003). Sudden death in these individuals is most likely a consequence of an electrically unstable and unpredictable myocardial substrate with reentrant ventricular tachyarrhythmias (Maron 2003).

Horses with SVPCs do not appear to be at increased risk of collapse or SCD. However, it is thought that frequent SVPCs during or after exercise are a risk factor for atrial fibrillation (Hiraga and Kubo 1999). In rare cases horses with atrial fibrillation may collapse or die during exercise. This has been reported in horses with both sustained (Deem and Fregin 1982; Lyle *et al.* 2010) and paroxysmal AF (Franklin and Allen 2013) and may be related to ventricular ectopy or aberrant conduction resulting in sudden changes in cardiovascular haemodynamics (Piercy and Marr 2010). Verheyen *et al.* (2012) recently reported that ventricular ectopy occurs frequently in horses with AF. Of 43 horses examined, 69% showed ventricular arrhythmias and 30% had R on T phenomenon. Hence an exercising ECG is warranted in all cases that do not undergo cardioversion in order to determine whether it is safe to continue ridden exercise (Reef *et al.* 2014).

Conclusions

The athletic heart undergoes significant remodeling in response to training. Whilst this cardiac hypertrophy is an important factor for increasing oxygen delivery during exercise, it also predisposes the equine athlete to valvular regurgitation and development of exercise-induced arrhythmias. Although in most cases valvular regurgitation will not significantly affect performance, exercising arrhythmias may result in reduced performance and in some cases may predispose the equine athlete to an increased risk of SCD. Isolated premature complexes have been identified in apparently healthy horses and currently there are no fixed guidelines on what prevalence of premature complexes constitute an increased risk of collapse/ SCD. Therefore clinical judgement becomes important particularly in interpretation of VPCs. In general, ‘judge them according to the company they keep’ is

good advice. For example a few isolated VPCs in the immediate post exercise period of a racehorse after a strenuous exercise test may be judged as not of clinical significance. However the same number of VPCs during walk and trot in a pleasure horse with an aortic regurgitation may be considered highly significant. Further research is warranted to confirm the repeatability of exercising arrhythmias and their relationship with horse / rider safety and SCD.

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Table 1: Typical heart rates at different exercise levels

Speed	Heart rate (bpm)
Walk	60-80
Trot	80-120
Canter	120-180
Gallop	>180 Max 210-240

Table 2: Expected peak heart rates achieved during competition for various disciplines.

Discipline	**Expected approximate peak heart rate (bpm)**
Thoroughbred racing	Flat: 240 National Hunt: 225-230
Standardbred racing	230
Eventing –cross country phase	170-200
Show jumping	Up to 180-190
Polo	Up to 215-225
Dressage	Up to 140-170
References: Krzywanek et al. 1970; Art et al. 1990; Marlin et al. 1995; White et al. 1995; Marlin and Allen 1999; Serrano et al. 2002; Mukai et al. 2007; Barbesgaard et al. 2010; Buhl et al. 2010; Physick-Sheard and McGurrin 2010.	

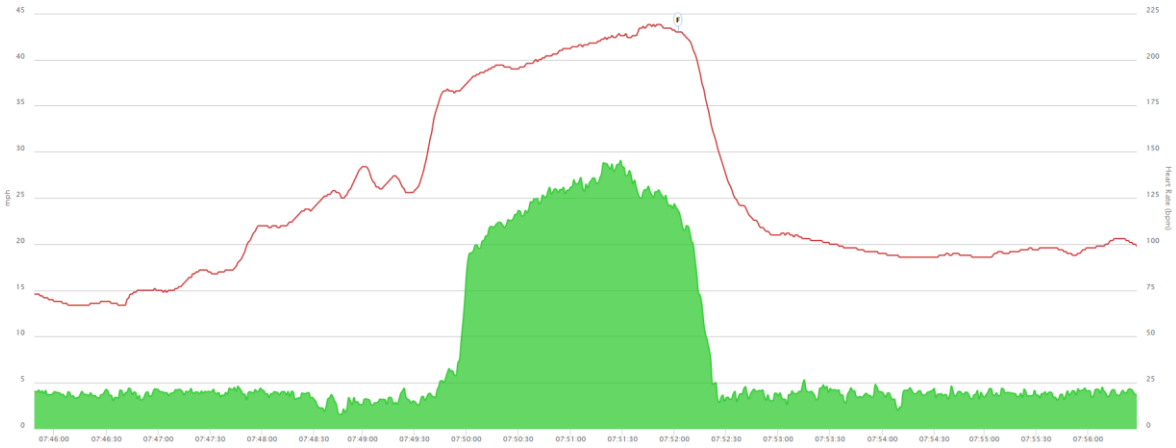

Figure 1: Typical heart rate graph from a Thoroughbred racehorse doing a single gallop interval. The red line shows the heart rate (bpm) and the green shows the speed (mph). An anticipatory HR increase can be seen before the gallop, when the HR reaches values of 100 to 130 whilst the horse is still at walk. The peak heart rate achieved is approximately 220bpm and is achieved after the horse reached peak speed. The graph also shows the initial rapid heart rate recovery.

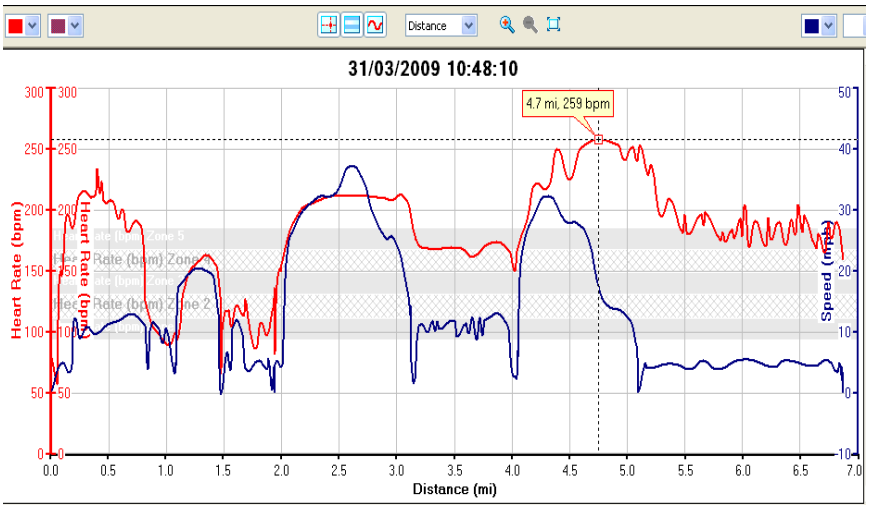

Figure 2: A National Hunt racehorse undergoing clinical exercise testing in the field. The blue line shows speed (mph) and the horse performed two intervals on an inclined gallop. The HR (red line) shows a normal response to the first interval. During the second interval the heart rate becomes abnormally elevated during exercise (peak HR 259 bpm) and shows a characteristic erratic and elevated heart rate during recovery. ECG confirmed paroxysmal atrial fibrillation.

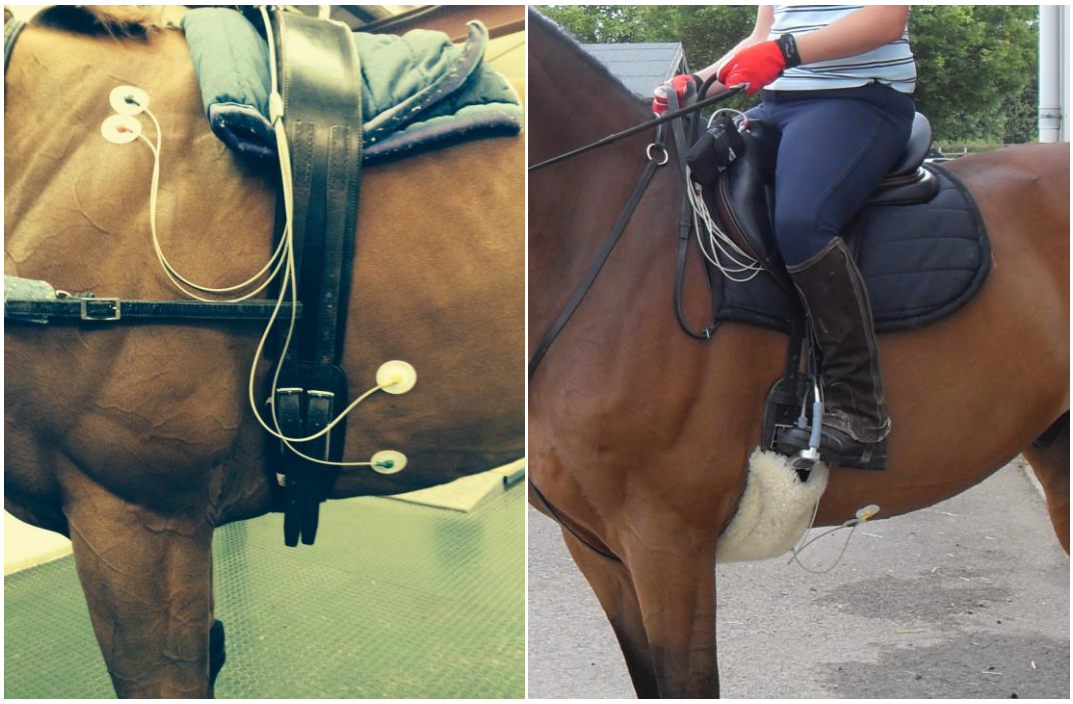

Figure 3 a and b: Placement of electrodes for lunged and ridden exercise. In the ridden horse it can be difficult to separate the yellow and green electrodes because of the rider's leg movement and hence a compromise is often needed. In figure b, both yellow and green electrodes have been placed more ventrally to avoid being disturbed by the rider.

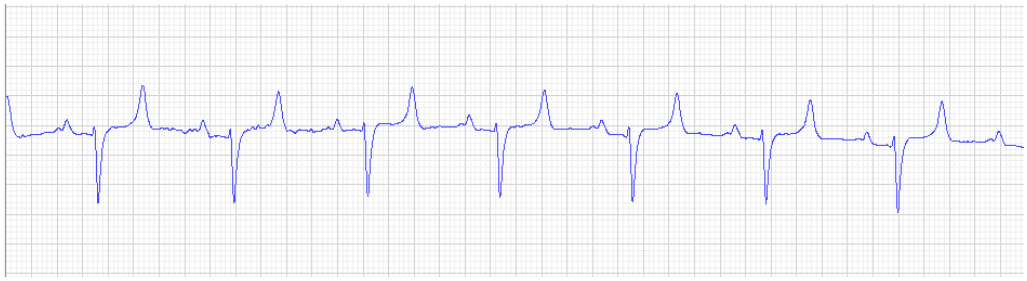

Figure 4: Normal ECG during low level exercise

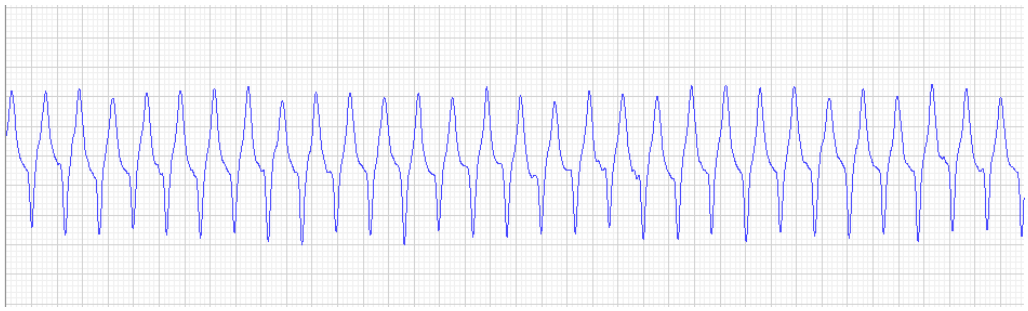

Figure 5: Normal ECG during strenuous exercise. The heart rate is 225bpm. The RR interval is regular. P waves are hidden in the preceding T wave.

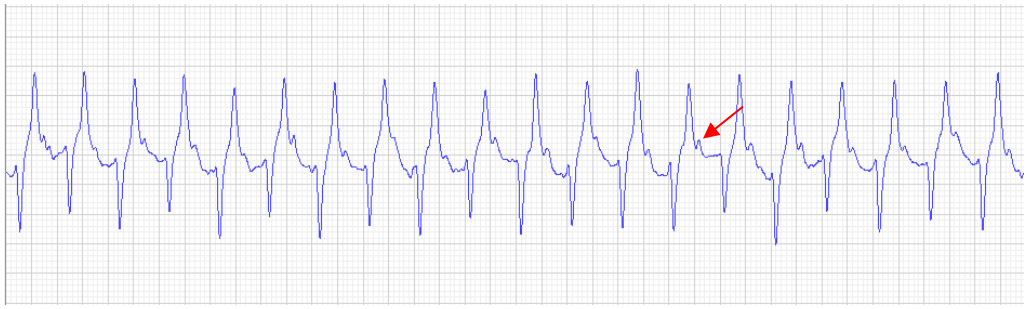

Figure 6: ECG recording in the immediate recovery period. The heart rate is now slowing and P waves become increasingly visible on the edge of the T waves

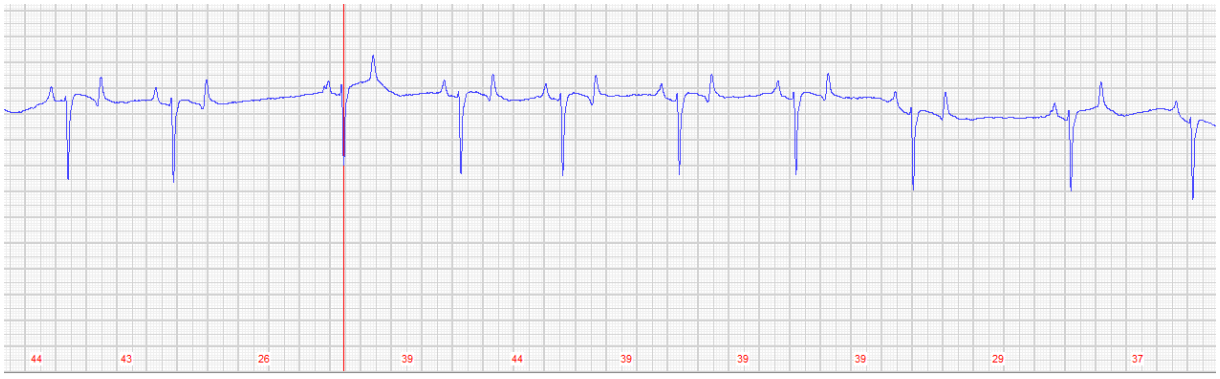

Figure 7: Resting ECG showing sinus arrhythmia

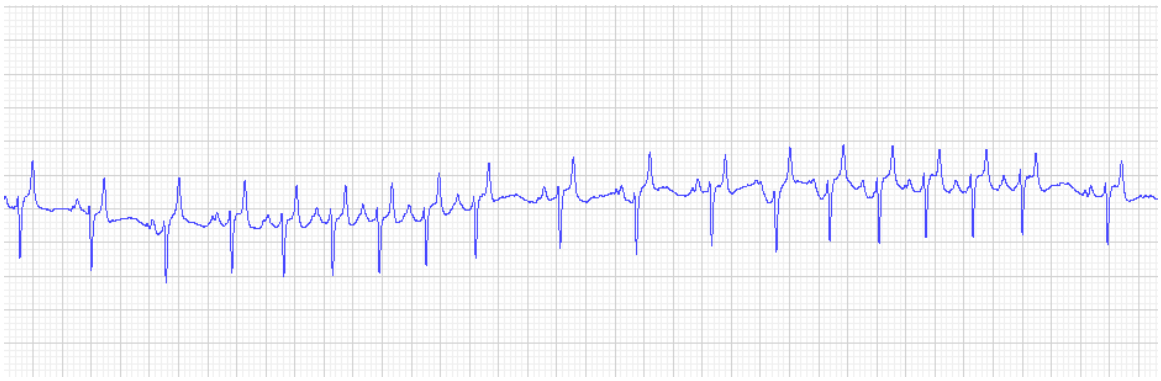

Figure 8: ECG obtained in the immediate post exercise period showing transient sinus arrhythmia. A phasic increase and decrease in RR interval is seen.

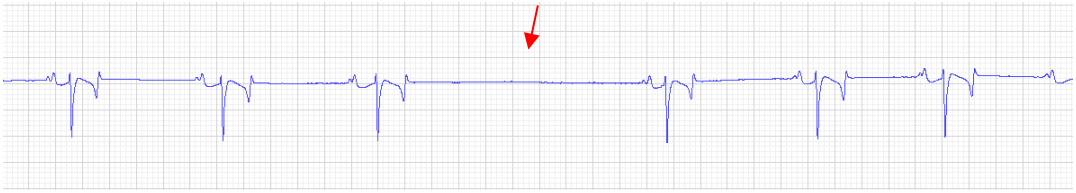

Figure 9: Resting ECG obtained showing a sinus block

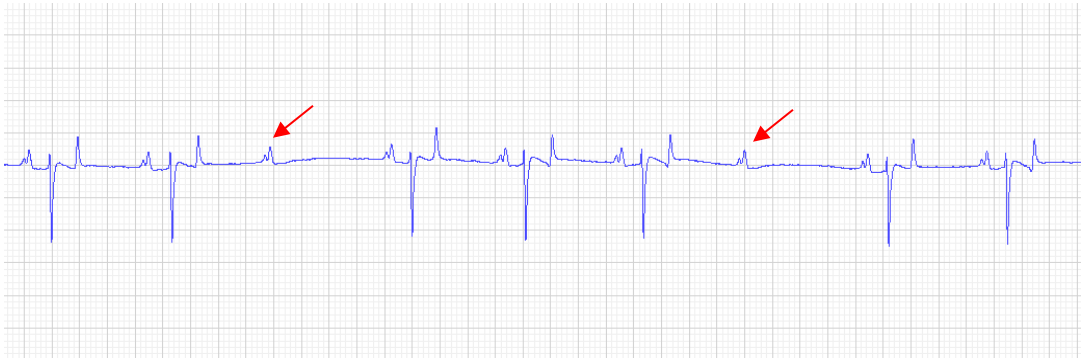

Figure 10: Resting ECG showing second degree AV block

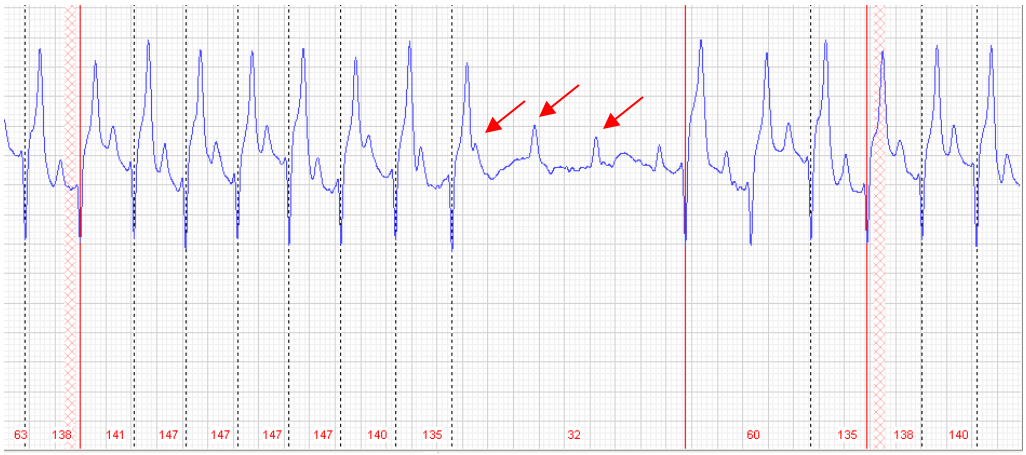

Figure 11: ECG obtained in the immediate post exercise period showing 2nd degree AV block. There are 3 consecutive non-conducted P waves.

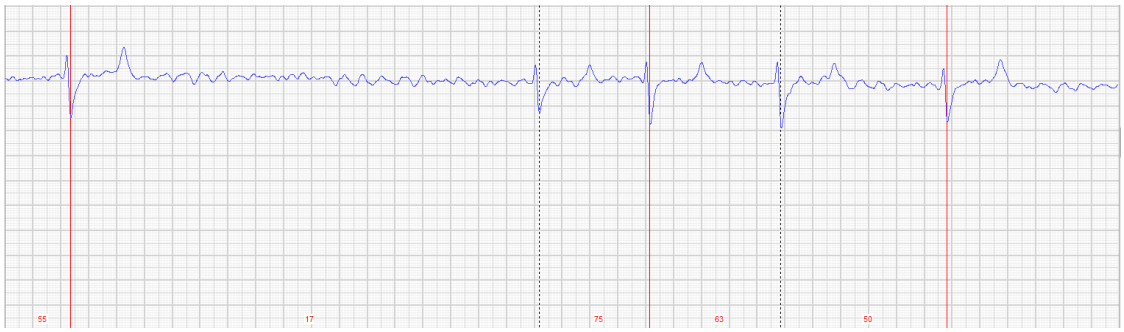

Figure 12: ECG obtained at rest in a horse with sustained atrial fibrillation.

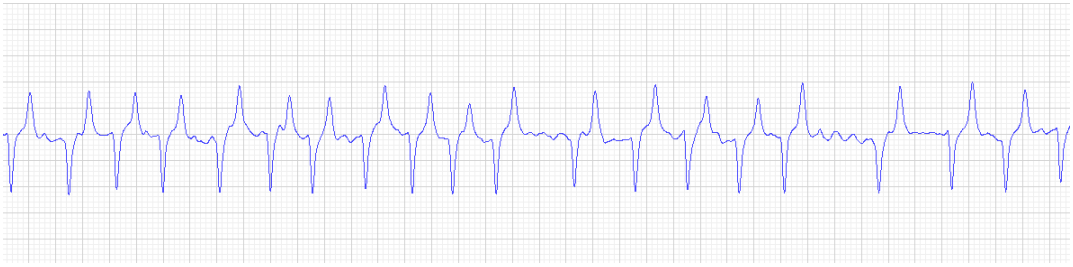

Figure 13: ECG from a collapsing horse which had paroxysmal atrial fibrillation during exercise. The RR interval is irregular, no p waves are visible and f waves can be seen in the longer RR intervals.

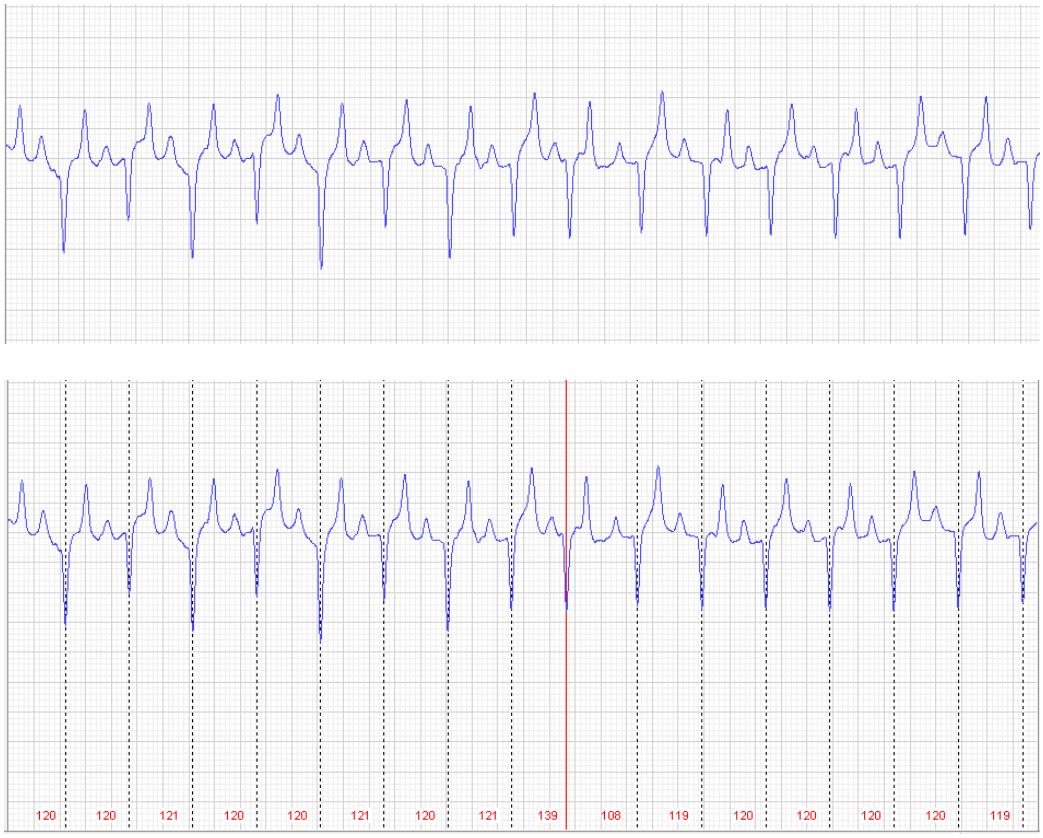

Figure 14: A supraventricular premature complex. The QRS is of similar morphology as the normal complexes. As supraventricular premature complexes are similar to the normal QRS they are more easily missed. The two ECG traces above are identical but the second image also has the computer generated ECG analysis marks. The red line highlights the premature complex. Equipment with this facility aids recognition of supraventricular complexes.

Figure 15: ECG trace of the recovery period showing two supraventricular premature complexes (indicated by red line). Although on the first premature complex the P waves is not identified the QRS morphology is the same indicating that these are most likely supraventricular rather than ventricular in origin.

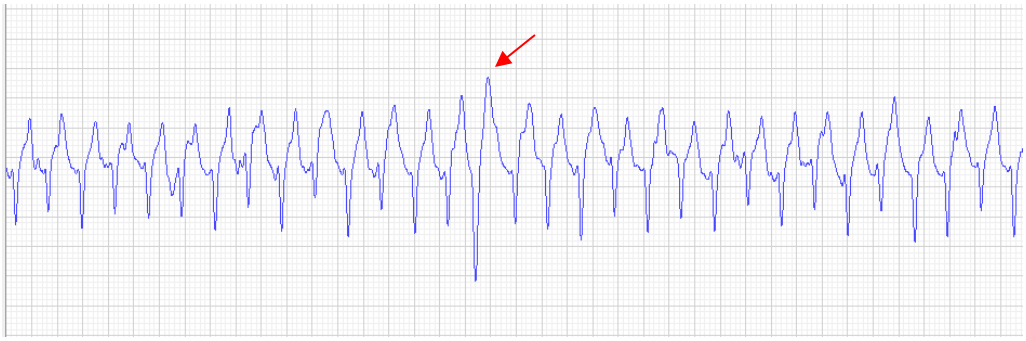

Figure 16: An isolated ventricular premature complex is seen. This complex is premature and of different morphology to other QRS complexes

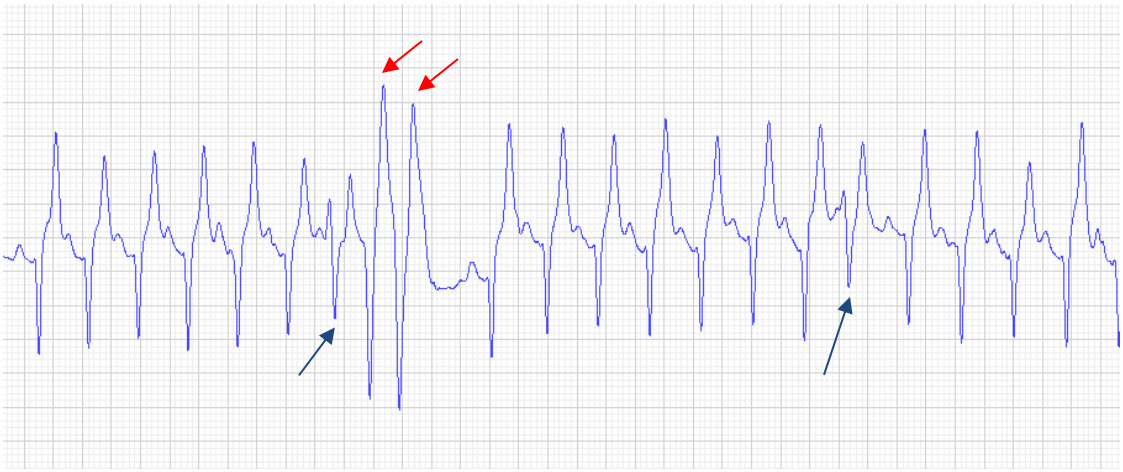

Figure 17: A post exercise ECG showing two obvious VPCs (red arrows) and two less obvious VPCs (blue arrows)

Figure 18: Two runs of ventricular tachycardia were observed in the early recovery period. This rhythm is at risk of deteriorating into ventricular fibrillation