Racing NSW – Compulsory Equine Welfare Seminar for licenced trainers 2025

Cardiac arrhythmias, Exercise Associated Sudden Death and Exercise Induced Pulmonary Haemorrhage in Thoroughbred Racehorses

Changes in heart function from rest to exercise

Thoroughbred racehorses have an enormous capacity for increasing oxygen delivery from rest to exercise. A table below summarises these changes:

Table 1. Physiological changes from rest to exercise.

VARIABLE	REST	EXERCISE	EXERCISE/REST RATIO
Heart rate (beats/min)	30	210-250	7-8
Cardiac output (L/min)	30	240-450	8-13
Stroke volume (L)	1	1.7	1.7
Aortic pressure (mmHg)	120	180-230	1.7
Pulmonary artery pressure (mmHg)	20-30	90-140	3-5
Packed cell volume (%)	37-47	55-60	1.5
Oxygen uptake (L/min)	1-2	80-100	50-100

Transport of oxygen and carbon dioxide

The heart is made up of four chambers, the left atrium, left ventricle, right atrium and right ventricle. Four valves are present in the heart to keep the blood moving in a forward direction. These are the mitral valve on the left between the atria and ventricle, the tricuspid valve on the right between the atria and ventricle and the aortic valve exiting the left ventricle and the pulmonary valve exiting the right ventricle.

The aorta is the main artery carrying oxygen rich blood from the left ventricle to supply the organs of the body (brain, heart, skin, kidneys, liver, intestines, muscle).

The aorta branches into smaller arteries and then to capillaries. Within the muscle cell, mitochondria are the small organelles that use oxygen to produce energy.

Carbon dioxide is also produced as part of this process. Carbon dioxide then enters the bloodstream at the level of the capillaries and is transported by smaller veins into the major veins and then to the right atrium. This then is carried through the right heart to the lungs via the pulmonary artery where it enters the pulmonary capillaries. The pulmonary capillaries are located adjacent to the alveoli. These alveoli are small air filled sacs at the very ends of the airway. There is an extremely thin membrane between the alveoli and the pulmonary capillary which allows oxygen to enter the capillary and carbon dioxide to pass across into the alveoli. The oxygen rich blood leaving the capillary enters into the pulmonary vein, then the left atrium and ventricle before exiting the aorta and the cycle starts again.

Oxygen and carbon dioxide are transported within the bloodstream by haemoglobin, which is a specialised protein within the red blood cell. The spleen of the horse has an important role in storing extra red blood cells. At the onset of exercise, adrenaline acts on the spleen and causes it to contract, releasing extra red blood cells into the system which increases the packed cell volume to 55-60% and causing a 50% increase in oxygen carrying capacity.

The heart rhythm

The normal impulse for the heart rhythm starts in the right atrium at the sinus node. It then causes a wave of depolarisation to cross the atria which then goes the atrioventricular node. Deep fibres are present in the ventricle which allow for very rapid conduction. The atrial depolarisation produces the P wave and the ventricular depolarisation produces the QRS complex. Ventricular repolarisation produces the T wave.

In normal sinus rhythm there is a regular spacing between each R-R interval. Also, each feature has a similar shape. An arrhythmia is present when there is a difference in the interval or a change to the shape of the complexes. A change to the shape of the complexes generally indicates that the heart rhythm is being conducted in an abnormal direction.

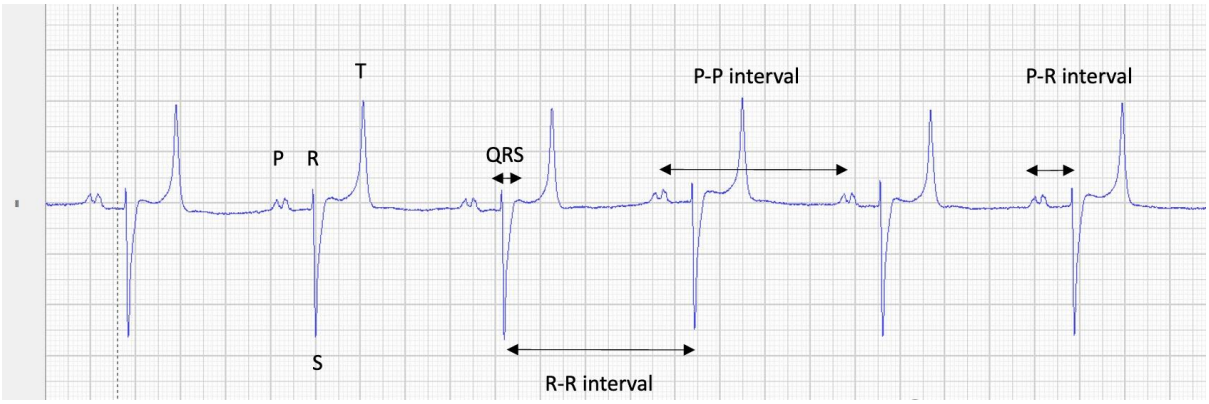

Figure 1. Resting ECG. There is a p wave with a positive deflection, a normal QRS with negative deflection and a positive T wave deflection associated with ventricular repolarisation. Normal sinus rhythm is present, based on regular R-R interval and regular P-R interval. There is a P wave for every QRS and vice versa.

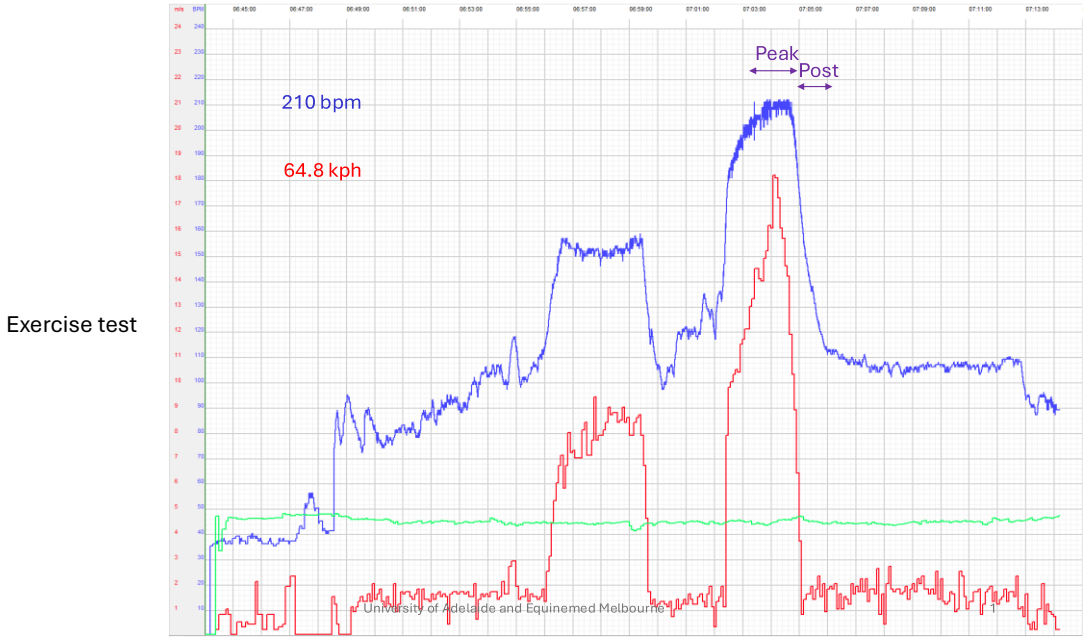

Figure 2. Heart rate overview showing speed in red and heart rate in blue for a standard track gallop. The peak and post exercise periods are labelled.

Cardiac adaptation to training

Thoroughbred racehorses have naturally large hearts, approximately 1-1.5% of bodyweight. In mammals, including humans and horses, exercise training will cause further adaptations to the heart which include an increase in heart size. In racehorses strenuous exercise training causes an increase in the blood volume which then causes increased stretch and pressure in the heart. This results in balanced enlargement of the heart chambers (atria and ventricles) and thickening of the heart muscle wall. This process is called athletic heart syndrome. As a result of the increase in heart size, the stroke volume (amount of blood pumped by each heartbeat) increases. The maximum heart rate for an individual horse is fixed and does not change with exercise training. The cardiac output is the amount of blood pumped by the heart each minute and is proportional to the amount of blood being delivered.

Cardiac output (L/min) = Heart rate (beats/min) x stroke volume (L)

Heart rate is controlled by the balance between the sympathetic (fight or flight) and parasympathetic (rest and digest) systems. The sympathetic system is modulated by adrenaline and increases heart rate. The parasympathetic system is modulated by acetylcholine and decreases heart rate. At peak exercise the system is entirely under the influence of the sympathetic system but the parasympathetic influence returns immediately after exercise and causes a rapid decrease in heart rate. Peak exercise and immediately post-exercise are the most vulnerable times for arrythmia. Pain and

anxiety can also increase heart rate. Imbalance between sympathetic and parasympathetic influence can contribute to arrhythmia.

The role of electrolytes

During strenuous exercise, lactate (also known as lactic acid) is produced because the oxygen supply is insufficient and the muscle cell uses anaerobic metabolism. The increased lactate causes a drop in pH which results in an acidosis. This causes potassium to move from inside cells to the extracellular fluid and cause increased neuromuscular excitability (twitchiness). Importantly, ionised calcium also increases as less calcium is bound to protein in acidic environments. Therefore sufficient potassium stored within the cell and sufficient calcium are very important for reducing neuromuscular excitability. Alkalinising agents have the potential to worsen this. Magnesium also has an important role.

Some of the most common conditions that occur as a result of increased neuromuscular twitchiness are the thumps (synchronous diaphragmatic flutter) and atrial fibrillation.

The thumps is a disease that is caused by stimulation of the phrenic nerve which runs over the heart before innervating the diaphragm. Increased neuromuscular twitchiness can cause the phrenic nerve to be stimulated and the diaphragm to contract with every heartbeat.

Post exercise electrolyte derangements can be limited by feeding adequate dietary electrolytes. The most common electrolyte deficiencies in racehorses are potassium, calcium and magnesium. Electrolyte status is difficult to assess in blood tests because they are tightly controlled within certain ranges. A better indicator of dietary electrolyte balance can be fractional excretion of electrolytes. This involves measurement of electrolytes within a blood sample and urine sample taken at the same time. If the level of a certain electrolyte is higher than the normal range in the urine, this indicates it is being fed at excess. In contrast if the level in the urine is low, this indicates that the body is attempting to hold on to as much of this electrolyte as possible and that the dietary content is insufficient.

Table 2. Approximate normal daily requirements of major dietary minerals. This might be higher in hot weather or in humid climates.

Mineral	Daily requirement in grams
Sodium	30-50
Potassium	40-75
Chloride	50-150
Calcium	35- 40
Phosphorus	25-27
Magnesium	10-15

Abnormalities of cardiac rhythm

Cardiac rhythm abnormalities are very common in Thoroughbred racehorses. This could be due to a number of factors including the large heart, fluctuations in sympathetic and parasympathetic influence on the heart, low blood oxygen, electrolyte derangements, heart muscle inflammation, and inherited rhythm abnormalities. The peak exercise and immediate post exercise period are the most common periods for arrhythmias to be observed with exercising ECGs. Importantly, this is also the most common time for sudden death to occur.

Atrial fibrillation is the most common performance limiting arrhythmia in Thoroughbred racehorses. It should be suspected in horses with poor or erratic performance, a history of exercise induced pulmonary haemorrhage (EIPH), horses with heart murmurs, horses with elevated heart rates detected using heart rate monitors and in horses with a history of weakness or collapse.

It is important to recognise that atrial fibrillation can be very short lived, lasting from 10-30 seconds to minutes, hours or days. Usually, the arrhythmia corrects without specific treatment. If the arrhythmia is present for more than 3 days it is very unlikely to correct by itself and veterinary treatment should be sought. Treatment for atrial fibrillation involves nasogastric administration of quinidine sulphate or electrocardioversion (defibrillation using intra-cardiac catheters), which requires general anaesthesia.

Atrial fibrillation is a specific rhythm abnormality within the atria of the heart where waves of electrical activity are constantly moving around the atria and then randomly passed through the atrioventricular node to the ventricle, resulting in an irregularly irregular rhythm.

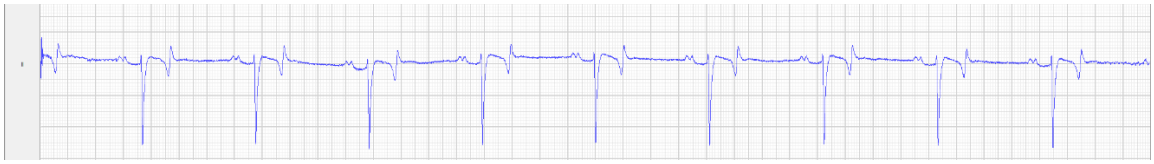

Figure 3a. Normal heart rhythm which is regular.

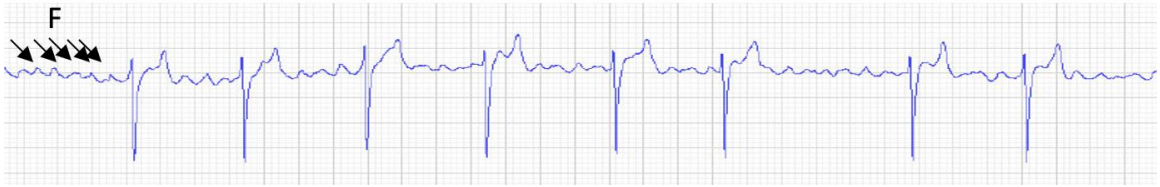

Figure 3b. An example of atrial fibrillation. There are irregular R-R intervals and undulation of the baseline with F (flutter waves).

A study in Hong Kong identified atrial fibrillation in approximately 5% of horses in their career. Of horses diagnosed with atrial fibrillation approximately 25% have a second episode in their career. Horses that have had atrial fibrillation can still have long and successful careers.

A study of horses with post-race atrial fibrillation showed they were more likely to have significant (grade 3 or 4) EIPH (lung bleeding). This is likely because of the

close relationship between cardiac and pulmonary pressures, which means that when an arrhythmia occurs the pressure in the heart increases.

Most racehorses with atrial fibrillation have structurally normal hearts. Interestingly, human athletes have a 2.5 times increased risk of atrial fibrillation compared to non-athletes. This is thought to be due to balanced heart enlargement and stretching of the heart atria during exercise.

Usually atrial fibrillation results in a very poor performance (average 21 lengths behind the winner). There have been occasions when a horse has been wearing an exercising ECG and this has shown atrial fibrillation deteriorating into a ventricular arrhythmia and fatality. Certain features of the ECG for horses with atrial fibrillation are thought to infer a greater risk and this includes heart rates exceeding 150 beats per minute and abnormal ventricular conduction (change in the QRS shape). It is important not to transport horses off the racecourse until their heart rate is below 80-100 beats per minute.

Sudden death

Sudden death accounts for approximately 20% of racing fatalities world wide. Of these fatalities, approximately half are due to cardiopulmonary failure. The cause of a sudden death cannot be determined without post-mortem investigation. In sudden cardiac death, post-mortem changes can be very subtle and this is generally a diagnosis of exclusion. Pulmonary haemorrhage is a common finding at post-mortem for both horses with sudden death and those that are euthanatized for catastrophic orthopaedic injuries.

A study of horses that included both racing fatalities and training fatalities showed that approximately three quarters of fatalities occurred during training and only one quarter during racing. This shows that studies focussing on racing fatalities greatly underestimate overall fatalities from sudden death and also suggests that speed is less important in causing this type of fatality. The same study found that young horses, early in their careers were at highest risk. This finding could indicate that inherited rhythm abnormalities might be involved because inherited causes account for the majority of sudden cardiac deaths in human athletes less than 18 years of age. Another consideration is viral disease. It is known that viral disease is very common in young horses when they enter racing stables. Viruses can cause myocarditis (inflammation of the heart muscle) and this could cause a fatal arrhythmia if horses are exercised whist in the inflammatory phase on the virus. One simple modification to training programs that could prevent this would be taking the temperature of all horses each morning and not exercising horses that have a fever. It is suggested that horses are given at least 48 hours after a fever before they have ridden exercise and gallop exercise shouldn’t occur for 5 days after the fever has resolved.

Use of technology in monitoring

Wearable devices are increasingly being used in racing stables to guide training decisions. Many of these devices also have ECG capability. Smaller, hand held ECGs can also be used at rest to record heart rate and rhythm. These can be particularly useful when the ECG needs to be obtained rapidly such as in the post-race period. The ECG from both the hand-held machines and the wearable devices can be shared and reviewed as needed.

Summary

Horses are phenomenal athletes with an impressive capacity for oxygen delivery and utilisation by the muscle. Very high intracardiac pressures are observed when horses are exercising strenuously and this can predispose to cardiac arrhythmias and pulmonary bleeding. Dietary electrolytes should be optimised and airway disease minimised to reduce the risk of arrhythmias. Wearable heart rate monitors can influence training decisions and are likely to help early identification of arrhythmias.

Other resources

Gerald Leigh memorial lectures Newmarket
https://www.youtube.com/@beaufortcottageeducational8051/playlists

Racing Australia Thoroughbred welfare
https://thoroughbredwelfare.horse

New Zealand Thoroughbred racing
https://nztr.co.nz/thoroughbred-welfare

HISA
https://hisaus.org/resources

DR LAURA NATH
BVSc, MVSc, CertEM, FANZCVS, PhD
Registered Specialist in Equine Medicine