EFFECTS OF CUMULATIVE CHUCKWAGON RACING WITH THOROUGHBRED HORSES ON BLOOD ELECTROLYTES, LACTATE, AND CONTINUOUS ELECTROCARDIOGRAMS

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Abstract

Reasons for performing the study: In chuckwagon racing teams of four Thoroughbred horses start from a standstill, pull a 600 kg wagon through a figure-8 pattern, and then turn onto a track for an 800 meter race. The horses run much more frequently than in harness, steeplechase, or flat racing as they often race multiple days in a row. Horses are in peak physical condition, however rare cases of sudden death occur and these are often attributed to cardiovascular failure. Concerns have been raised regarding the contribution of cumulative racing to electrolyte imbalances, arrhythmias, and sudden death, and its impact on recovery.

Objectives: The objective of this study was to investigate the effects of (cumulative) chuckwagon racing on plasma electrolytes, lactate and the prevalence of cardiac arrhythmias.

Hypothesis: Chuckwagon horses will show plasma hyperkaliemia, hypernatremia, hypochloremia, hypocalcaemia, and lactatemia at the end of the races that will be significantly greater after cumulating consecutive races. The electrolytes changes will be correlated with the number of cardiac arrhythmias observed during exercise.

Methods: 148 Thoroughbred horses aged 9 $\pm$ 2.7 years from 13 chuckwagon teams were monitored during racing and training exercise. The horses were equipped with four electrodes and a TeleVet ECG device. ECG monitors sent information to cell phones that were in the wagon and the data were wirelessly transmitted to a nearby computer. Electrocardiograms (ECGs) were analyzed and the timing and type of arrhythmias were recorded. A sample of 31 horses that performed consecutive racing (day 1 and 2) were selected for analysis. Plasma was frozen until further analysis. Pre- and post-race electrolytes, pH and lactate were compared using the paired T-test. Associations between racing days 1 and 2, and electrolytes were analyzed, as well as the relationship between electrolytes and arrhythmias using the Pearson Correlation.

Results: 377 ECGs were obtained, 82 of them were incomplete due to a loss of one or more electrodes. The ECGs of 31 horses that performed a race on two consecutive days were suitable to be used for analysis. Arrhythmias were predominately second degree atrioventricular (AV) blocks during rest (49.4 % of the horses) and supraventricular premature contractions (SVPCs) during warm-up, race and recovery (54.1 %, 90.1 % and 83.8 % respectively). Ventricular premature contractions (VPCs) were greatest during recovery (16.2 % of the horses). Arrhythmic events did not differ between day 1 and 2 and no fatal arrhythmias were observed. Plasma lactate was significantly higher post-exercise but pre-exercise lactate did not significantly differ on day 1 and day 2. Plasma sodium, potassium and chloride were significantly higher pre-exercise on day 2 than on day 1, while plasma calcium was significantly lower post-exercise on day 2. Potassium and sodium levels increased during exercise, whereas chloride and calcium showed a decrease after exercise.

Conclusions: Good quality ECGs can be obtained from chuckwagon racing horses and high percentages of arrhythmic events were found. Plasma lactate and electrolytes levels were as expected but electrolytes were significantly different pre-exercise on day 2 compared to day 1 which might implicate pathology. Lactate post-race was high but not more extremely than other intense sports. More thorough veterinary checks are needed.

Background Information and Introduction

Chuckwagon racing is a popular sport in Western Canada. The horses used are Thoroughbreds, usually retired race horses. The chuckwagon races are held on typical Stampedes or Heritage Days, outdoor shows in different towns along with rodeos and other entertainment. This means that the horses travel all summer long from event to event. The races are called "heats" and are usually held in the evening. In every heat there are four teams of four horses pulling a wagon and racing against each other, plus two outriders (rider on horseback) per team. In total six horses per team and 24 horses per "heat", for ten heats per evening. The outriders have to follow the wagons and finish within a certain distance of their respective wagon while avoiding any penalties. The race starts with a figure-8 pattern around two barrels, and continues on a circle track with an approximate length of 800 meters. The driver who wins the race has the best time, after adding eventual time penalties. The races on the half mile track take approximately one minute and fifteen seconds.

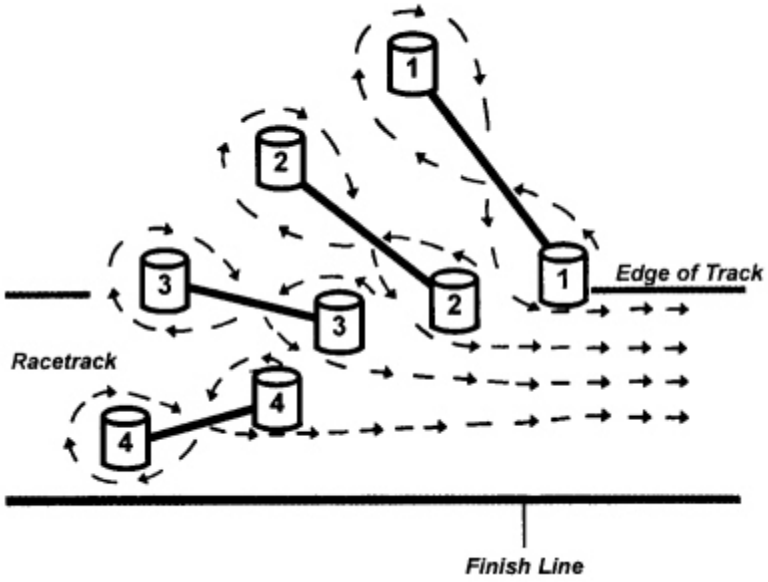

Figure 1: Pattern of the barrels

The barrels are staggered as can be seen in figure 1, so two tracks are called the "short/inside barrels", and the other two are called the "long/outside barrels". The barrel tracks are decided by a draw, and because the drivers usually have a special horse-team for each barrel, it is possible that the horses race several days in a row. For example, the Calgary Stampede takes ten days, and even though there are rules against cumulative racing days, it is still possible for a horse to race four to six times during the ten days of the Stampede. Recently, there have been some public concerns about chuckwagon racing because sudden death or breakdown injuries of horses during the races unfortunately happen every year. Since some sudden deaths were attributed to cardiovascular problems, one of the proposed aims of this research was to monitor the cardiac function of the chuckwagon horses during the race. Other studies have shown that sudden deaths in racehorses can be due to cardiovascular failure.[28] There is also, to the authors knowledge, no other study about the workload effort for chuckwagon horses, so it was postulated that it would be interesting to see how these horses manage this heavy work. [1] Chuckwagon racing is not comparable to other horse sports due to the workload of both racing and pulling as a set of four. The aim of the study is to assess the effect of cumulative racing on electrolytes and cardiac arrhythmias of chuckwagon horses.

The hypotheses are that chuckwagon horses show plasma hyperkaliemia, hyernatremia, hypochloremia, hypocalcaemia, and lactatemia at the end of the races that will be significantly greater after cumulating consecutive racing days. The frequency of cardiac arrhythmias during exercise and recovery will be correlated to the severity of changes in plasma electrolytes at the end of the races.

Materials and methods

Horses

148 horses aged 9 ±2.7 years were tested. All chuckwagon horses were eligible to take part in the study, participation dependent on the willingness of the drivers. Testing was done during racing as well as during training exercise. All testing was done in Alberta, Canada. The drivers were first approached in High River, during the Guy Weadick Heritage Days in mid-June 2012. A lot of testing was done at High River, continuing at the Ponoka Stampede in Ponoka at the end of July, the Calgary Stampede 6-15 July and at last Strathmore at the beginning of August 2012. As many outfits as possible were tested, the limiting factor being the conflicting starting times. The workers were able to test about four teams every night or morning. Only geldings are used in the chuckwagon sport, so the study contains only geldings as well. All testing was done during summer, so the weather was generally warm (around 25 °C) and sunny.

Owners were asked for information about the management of the horses, including supplementation with salts and the provision of fluids. Unfortunately, the information obtained was not consistent and therefore not possible to document.

At every event, an equine veterinarian was there in case of emergency and to check the horses for fitness. This check was mostly done visually without a thoroughly clinical examination of all horses.

Electrocardiograms (ECGs)

Horses were equipped with four electrodes and a TeleVet ECG monitor that was attached to their harness. The electrodes were held in place with circular stickers and a girt.[2] [3] The electrodes were attached before harnessing the horses, and after harnessing the electrodes were connected to the ECG device on the harness via wires. The ECG device was connected by Bluetooth to a smartphone that was attached on the wagon. Each device had a separate smart phone. The smart phone sent the information to a nearby laptop where the heart rate, speed and electrical activity could be seen in real time.

Later, all ECGs were flagged for R-R interval and the arrhythmias were recorded on an excel sheet. The ECGs were seen by final year veterinary students under supervision of Dr Leguillette*.

SVPCs were noted as extra beats outside of the sinus rhythm with a normally shaped QRS-complex. VPCs were noted as a widely shaped QRS-complex. Sinus arrhythmia and AV-blocks were recorded as well. Examples of the arrhythmias in the ECGs are shown in figure 2-4. Type and timing of the arrhythmias was noted. The results of 31 horses that performed a race on two consecutive days were used for the statistics. These data were collected during different races, depending on whether the workers were able to collect samples from horses that raced on cumulative days.

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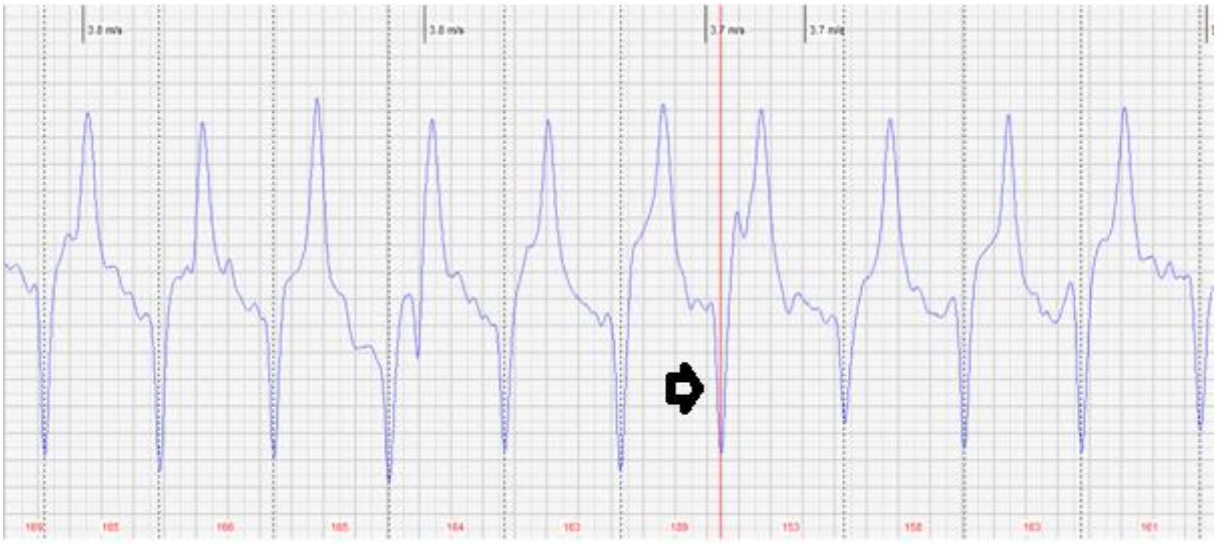


Figure 2: Example of a SVPC

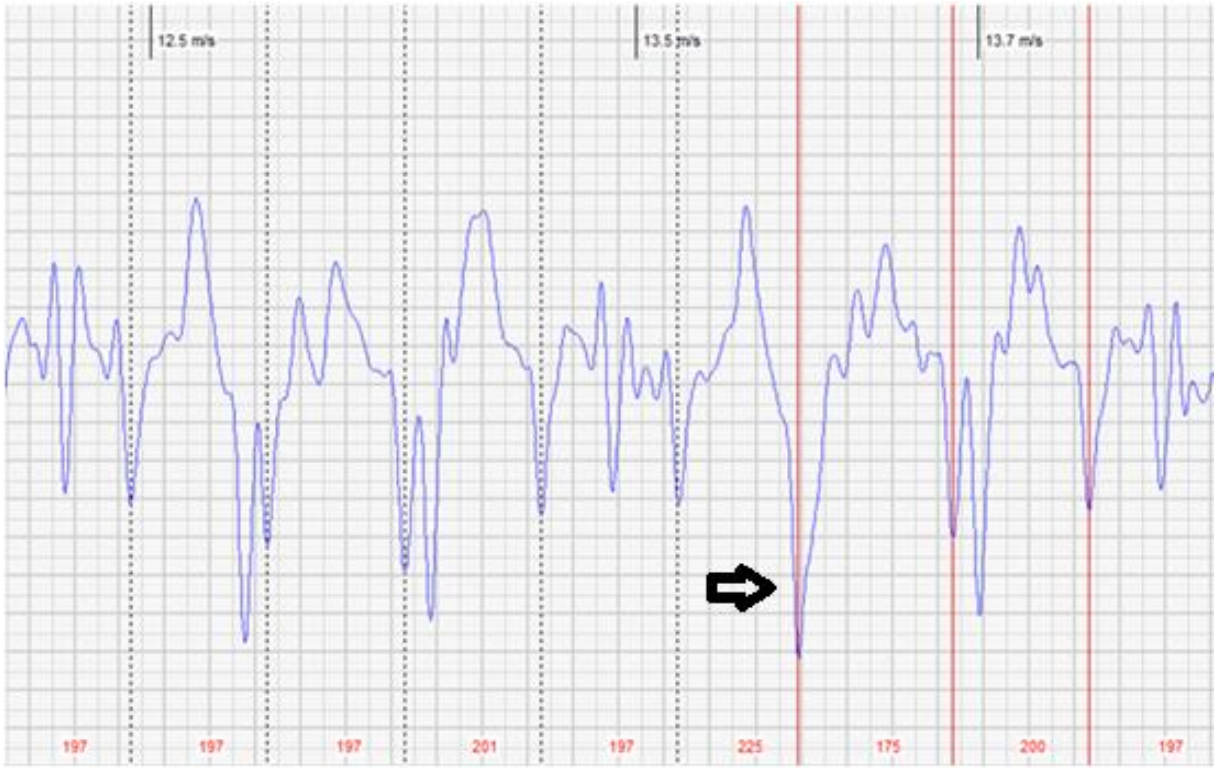


Figure 3: Example of a VPC

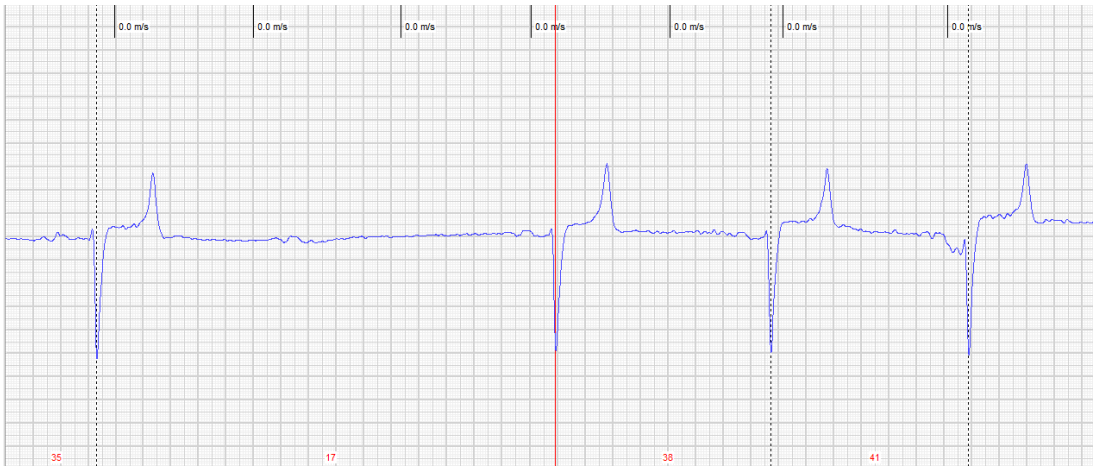


Figure 4: Example of a second degree AV-block

Blood sampling

Blood samples were taken before and after each race from the jugular vein in lithium heparin tubes for electrolytes analysis and sodium fluoride tubes for lactate analysis. Pre-race samples were taken anywhere from an hour down to ten minutes before the horses were hooked up to the wagon. Post-race samples were collected immediately after the horses stopped outside the track. However, due to the regulations and racing protocols, the drivers must parade at a trot or slow canter in front of the grandstand after racing before returning to their barns to unhook. The time elapsed since the horses slowed down was recorded and noted for sample analysis. Blood was sampled between two and ten minutes after the horses went from a racing gallop to a trot or slow canter and the mean time was 5:28 minutes. A sample of 31 horses who performed consecutive racing (day 1 and 2) were selected for analysis for lactate and electrolyte levels. The blood samples were centrifuged on-site and plasma was immediately transferred to plasma tubes and frozen at the race location, then frozen at -80 degrees Celsius until further analysis with the GEM® Premier™ 4000.

Statistics

Statistics were calculated using IBMs SPSS software. Pre- and post-race electrolytes, pH and lactate were compared using the paired T-test. Associations between racing days 1 and 2, and electrolytes were analyzed, as well as the relationship between electrolytes and arrhythmias with the Pearson Correlation.

Results

ECGs

The results of 31 horses that performed a race on two consecutive days were used for the statistics and analysis. Arrhythmic events did not differ between day 1 and 2 and no fatal arrhythmias were observed. Arrhythmias were predominately AV blocks at rest (56.3 % of the horses) and supraventricular premature contractures during warm-up, race and recovery (54.0 %, 90.2 % and 84.0 % respectively). Ventricular premature contractions were greatest during recovery (16.0 %). All results are displayed in table 1.

	Rest		Warm-up		Race		Recovery		TOTAL	
Type	**Day 1**	**Day 2**	**Day 1**	**Day 2**	**Day 1**	**Day 2**	**Day 1**	**Day 2**	**D1**	**D2**
# ECGs	**31**	**30**	**31**	**29**	**23**	**22**	**21**	**22**	**31**	**31**
SVPC	16.2%	14.8%	58.8%	49.1%	87.8%	92.6%	94.1%	73.9%	68.1%	61.3%
VPC	14.7%	21.0%	6.8%	6.6%	4.4%	3.7%	5.9%	26.1%	10.8%	12.1%
SA	5.9%	14.8%	34.4%	44.3%	7.8%	3.7%	--	--	11.7%	16.4%
AV	63.2%	49.4%	--	--	--	--	--	--	9.4%	10.2%

Table 1: Descriptive analysis of cardiac arrhythmias during rest, warm-up, race and recovery. SVPC= supraventricular premature contraction, VPC= ventricular premature contraction, SA= sino-atrial block, AV= atrioventricular block

Lactate

124 Plasma samples, pre- and post-exercise for both day 1 and day 2 were collected from 31 horses performing consecutive racing. Blood was drawn at a mean of 5:28 minutes post exercise. A significant difference ($p<0.001$) between mean plasma lactate pre-exercise (0.69mmol/L±0.02) and post-exercise (28.6mmol/L±0.53) was found. Maximum lactate was 37.8mmol/L. Lactate pre-exercise was not significantly different for day 1 and day 2 and lactate post-exercise was not significantly different for day 1 and day 2 as well.

Pre-exercise day 1 & 2	**Post-exercise day 1 & 2**
0.70 mmol/l	28.60 mmol/l
P= 0.000	
Pre-exercise day 1	**Pre-exercise day 2**
0.66 mmol/l	0.73 mmol/l
P= 0.0699	
Post-exercise day 1	**Post-exercise day 2**
28.84 mmol/l	28.36 mmol/l
P= 0.406	

Table 2: Lactate results

Electrolytes

Pre and post-exercise concentrations in sodium, potassium, calcium and chloride were significantly different ($p<0.001$). Pre-exercise sodium, potassium and chloride were significantly greater than on day 2, p=0.019, 0.020 and 0.016, respectively (Figure 2). Calcium was not significantly different pre-exercise on day 2 (p=0.112). Post-exercise chloride, sodium, and potassium were not significantly different on day 2 compared to day 1 (p= 0.460, p= 0.363, p= 0.096 respectively), whereas calcium was significantly lower post-exercise on day 2 (p= 0.006).

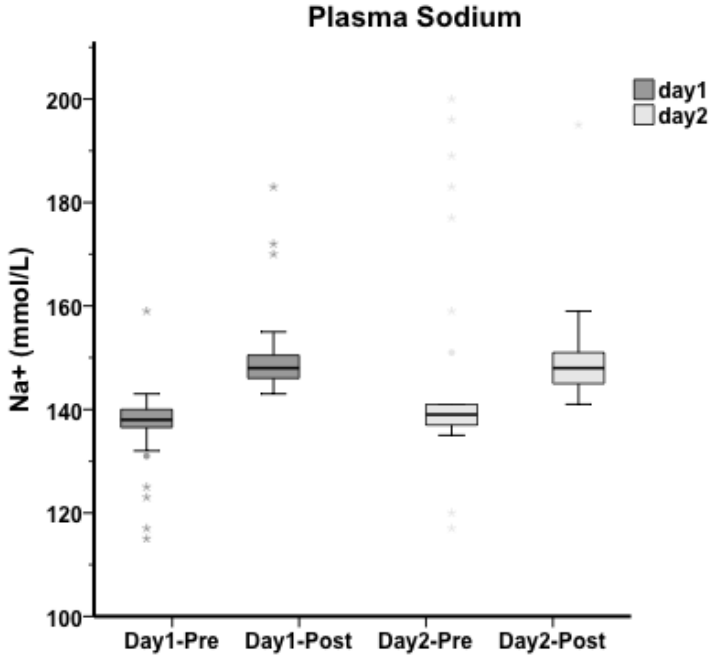


Figure 5: Plasma sodium was post-exercise significantly greater than pre-exercise. Pre-exercise sodium was significantly greater on day 2.

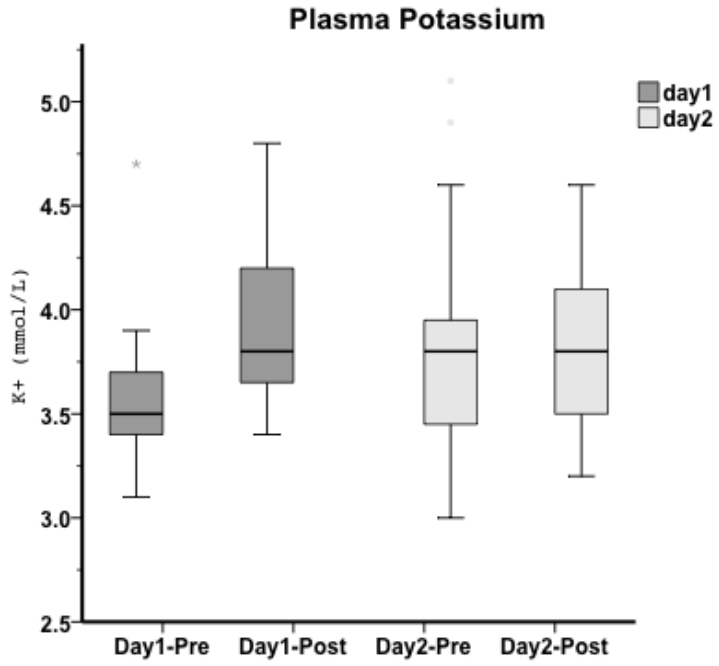


Figure 6: Plasma potassium was post-exercise significantly greater than pre-exercise. Pre-exercise potassium was significantly greater on day 2.

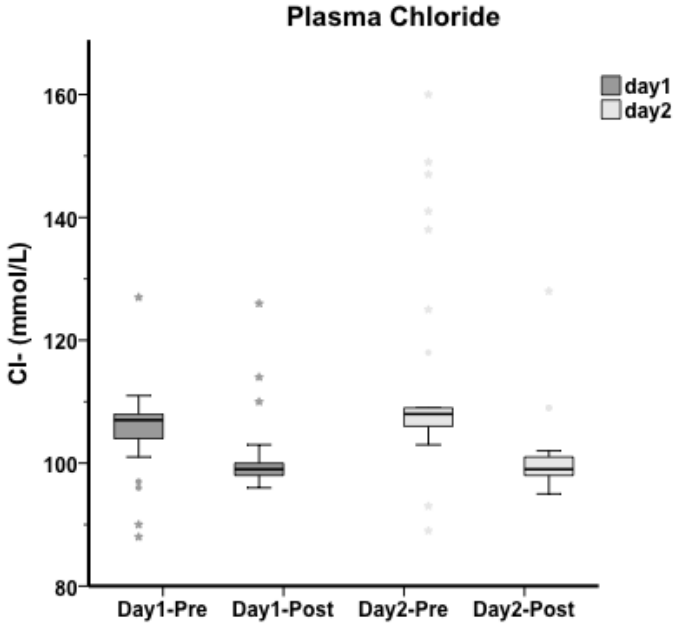


Figure 7: Plasma chloride was post-exercise significantly lower than pre-exercise. Pre-exercise chloride was significantly greater on day 2.

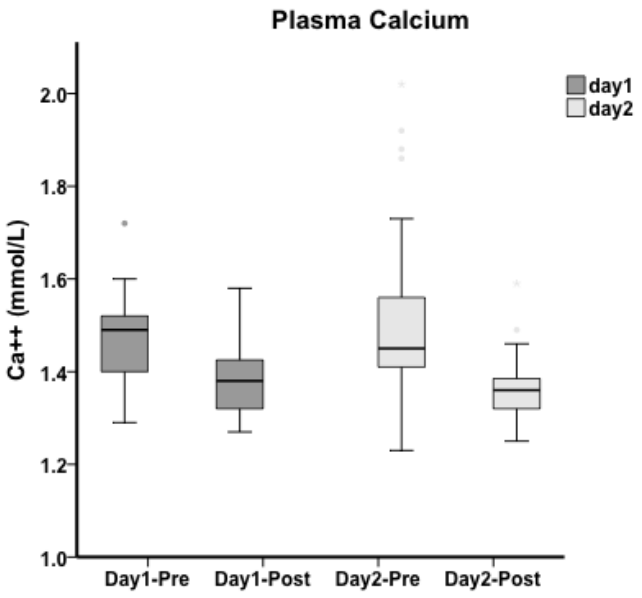


Figure 8: Plasma calcium was post-exercise significantly lower than pre-exercise. Post-exercise calcium was significantly lower on day 2.

Electrolytes in correlation to arrhythmias

A moderate positive correlation was found between post-exercise chloride (p=0.015, r=0.36) and sodium (p=0.035, r=0.32) and the presence of arrhythmias during racing (days 1 and 2 combined). Post-exercise chloride (p=0.005, r=0.495) and sodium (p= 0.029, r=0.393) are strongly correlated with arrhythmias on day 1 however not on day 2.

Discussion

Electrolytes

Water balance is of great importance for the correct function of all organs. Body water is characterized by a specific electrolyte concentration, which is critical for several physiological processes. Their constant concentration is essential for the regulation of osmotic pressure and to maintain multiple processes in a correct way. Especially sodium, chloride, calcium and potassium are important for the correct conduction of electrical impulse through the nervous system and muscles and for muscle contraction. The correct contraction of the heart muscle is especially of high importance. Sodium and potassium are most important in keeping the ionic balance in the intracellular and extracellular fluid. Electrolyte concentrations can change as a result of intensive exercise by working muscle cells and sweat losses. Sweat losses are inevitable since they are outside the control of any regulating influence except the demand for thermoregulation. Horses can lose up to 10-15 l/h of sweat in hot weather.[4] Sodium, potassium and chloride are a major constituent of sweat, calcium is a minor one. To cope with these electrolyte losses, the horses' body can increase the uptake from the gut, decrease the loss via the kidneys and liberate electrolytes from other tissues. The gut forms a temporary reservoir for recently ingested sodium and chloride.[5]

Sodium

45 % of the body's store of sodium is found in the extracellular fluid, 45 % in bone and the remainder is located intracellularly. Sodium is kept at a constant concentration by control of the ingestion and the urinary excretion to maintain the salt balance.[6] Sodium is an important cation in regulating the acid base balance in the body. In this study an increase in sodium was seen, that is not congruent with the literature on equine sport medicine. Sodium is a component of sweat so should decrease after exercise by loss in sweat.[7] The increase in sodium in this study is probably due to the short period between the end of the race and the sampling of the blood. Right after the onset of exercise a movement of water out of the vascular compartment is seen, which increases the concentration of sodium in the blood volume.[44] These fluid shifts are caused by significant increases in mean arterial pressure and consequentially capillary hydrostatic pressure that cause water, electrolytes, and a small amount of protein to be extruded from the vascular compartment. In the interstitial space this water can be used to form sweat. After fluid shifts, plasma volume will still be decreased by losses of water in sweat.[7] Increased sodium concentrations were found in greyhounds after short intensive exercise as well.[42] A treadmill exercise test with thoroughbreds reported elevated sodium levels after exercise too.[24]

Chloride

Chloride is important in water balance. During exercise the blood-pH reduces, resulting in plasma chloride ions moving into the red blood cells in exchange for bicarbonate. The elimination of CO_2 by expiration depends on the Cl^-/HCO_3^--exchanger, so it should be clear that chloride is very important for the acid-base status. A large amount of chloride is lost percutaneously by sweating. A decrease in the chloride concentration of the blood after exercise is thus not surprising.[5 7]

Potassium

89 % of the total body amount of potassium is located within the cells. There are two main transport processes. An active transport involves the Na^+-K^+-ATPase pump, insulin, beta-adrenergic agents, and mineral corticosteroids; and passive transport results from alterations in the pH and extracellular fluid osmolality.[5] The main potassium intake is by the feed. Excretion is exercised by the kidneys but

this is not as strongly regulated as with sodium. During exercise, potassium is disposed by the muscle cells; there is a movement of potassium from the intracellular towards the extracellular space. This means that after exercise an increase in plasma potassium is seen.[5] This study supported these findings.

Calcium

Calcium is stored in skeletal bones providing the body with an almost inexhaustible reservoir of calcium. About half of the calcium in the plasma is protein bound, primarily to albumin. A small fraction is complexed with phosphate, bicarbonate citrate and lactate, and the remaining part is ionized free calcium. Extracellular calcium concentrations are controlled through a complex mechanism mediated primarily by parathyroid hormone (PTH) and modulated through effector cells in kidney, bone, and intestine. The gradient between intracellular and extracellular calcium is 1000- to 10000-fold, making very rapid transmembrane shifts through gated channels possible.[8] Maintenance of low myoplasmic calcium is fundamental for muscle excitation contraction coupling.[9] Therefore calcium plays a crucial role in muscle contraction. Action potentials are conducted into the interior of the skeletal muscle fibers through the T-tubules and there activate a voltage-gated channel known as the dihydropyridine receptor (DHPR). A mechanical link between DHPR and the sarcoplasmatic reticulum (SR) calcium release channel results in the release of calsequestrin-bound calcium from the SRs' interior. A positive feedback loop, the calcium-induced calcium release, takes care of further activation of the channel with the result that the calcium concentration within the cytoplasm increases about 100-fold from a resting concentration. Relaxation happens when the calcium is re-sequestered within the sarcoplasmatic reticulum via the action of the Ca^{++}-ATPase pumps.[10] [11] Calcium plays an important role in the contraction of the heart as well, since it is crucial for the depolarization of pacemaker cells.

An increase or decrease in calcium after exercise depends strongly on the intensity and duration of the exercise. After short intense exercise as in chuckwagon racing a decrease in calcium concentration is likely since it is lost in sweat. Previous studies found a lower calcium concentration after exercise as well, and thought this was because of the decreased production of PTH during exercise.[12]

It can be concluded that the differences between pre- and post-exercise electrolytes were as expected.[14] Elevated pre-exercise sodium, chloride, and potassium on day 2 suggests that horses may be receiving a greater amount of electrolyte supplementation than is necessary. Because of a lack of consistent information from the drivers about the supplementation of the horses it is not possible to investigate this. Another explanation is that the horses may be dehydrated on day 2, resulting in a higher concentration of electrolytes in their plasma.[15] However this is unlikely, since the horses did not show any signs of dehydration like early exhaustion and an unfit appearance, but the researchers did not include a physical examination of every horse that was tested.

Lactate

During maximal exercise, glycogen is the main energy source and is then broken down through glycolysis to produce energy. When the oxygen supply to the (muscle) cell is insufficient, pyruvate and hydrogen ions form lactic acid. Since the lungs capacity and the blood volume to provide the horses' body with a sufficient amount of oxygen is the limiting factor in the horses' ability to performance, this is sure to happen in chuckwagon racing because of the maximal effort of exercise. Lactic acid is a sufficient source of energy, but, if the intensity of exercise requires a maximal effort for a period of 20–120 seconds as it does in chuckwagon racing, it reaches anaerobic threshold and blood lactate concentration tends to increase. Lactate moves out of the muscle cells by diffusion and active transport by the monocarboxylate transporter. Rapid transport of lactate across the plasma membrane is essential for muscle function and for maintaining muscle homeostasis, including the pH, despite the high H^+-load during heavy exercise. The H^+-removal capacity via the lactate/H^+ co-transporter is higher than via the Na^+/H^+-exchanger.

Lactate is metabolized to carbon dioxide and water in well-oxygenated metabolically active tissues, or is recycled to glucose and glycogen in the liver, kidney and inactive muscle cells through the Krebs cycle. The speed whereby the animal disposes lactate to replenish glycogen stores can increase when the animal is trained.[10] [16]

In the bloodstream lactate is buffered mainly by bicarbonate. Despite this buffering, intense exercise induces an acidosis which decreases arterial and mixed venous pH and base excess, and decreases bicarbonate concentration and increases carbon dioxide tension. The acidosis associated with maximal exercise can be severe and is tolerable only for a short time.[17] Generally, when the plasma lactate concentration reaches about 4 mmol/l, there is a sudden exponential rise in intracellular lactate accumulation known as the anaerobic threshold. This increase in intracellular lactate, together with the free H^+ ions, results in a significant decrease in cytoplasmic pH that has been suggested to be the major cause of fatigue during anaerobic exercise. Muscle pH may drop to as low as 6.25-6.50 and lead to impairment of both structure and function of the muscles. Significant disturbance to both mitochondrial and SR structures has been documented in horses that exhibit fatigue during maximal exercise. Low pH leads to dysfunction of the excitation-contraction coupling mechanism due to impairment of the SR calcium release channel, together with decreased reuptake of calcium into SR during relaxation. Low pH also inhibits the glycolytic enzyme phosphofructokinase, thereby diminishing ATP production. A decline in cytoplasmic pH is partially overcome by a buffering system within the myofibers. Buffering of H^+ occurs during the hydrolysis of phosphocreatine reactions catalyzed by creatine kinase and by glutamine synthetase; physiochemical buffering is provided by various proteins, bicarbonate, inorganic phosphate, and carnosine.[18] Race horses have been shown to have a higher muscle buffering capacity, thought to be associated with their myofibers having a high carnosine concentration.[19] [20]

Elevated lactate levels post-exercise were indeed expected since the horses are racing and working intensively[21] [22]. Lactate levels this high might be influencing the wellbeing of the horses. In a comparable study in show jumpers lactate levels were around 6 mmol/l after exercise, which is a lot lower.[16] But show jumping is not considered to be the same effort as chuckwagon racing so it is questionable if one could compare the two. In speed and endurance tests on an international level very high lactate levels were found as well (8.5-38.5 mmol/l) and these horses were fit to compete the next day.[23] Lactate levels above 20 mmol/l were also found during the cross-country phase of a combined training event and other events that needed maximal exercise.[14] [24] [25] In humans lactate levels around 25 mmol/l were found after exercise carried out up to voluntary exhaustion.

A factor which influences the horses' ability to cope with this kind of exercise is the level of training. When athletes are fit, the cardiac output will be higher which means that a higher amount of oxygen will reach the muscle cells, what will decrease lactate levels after exercise. Fitter horses will be able to process lactate quicker, and this will lead to a less severe acidosis.[26] A higher emphasis should be put on the fitness of the animal during training.

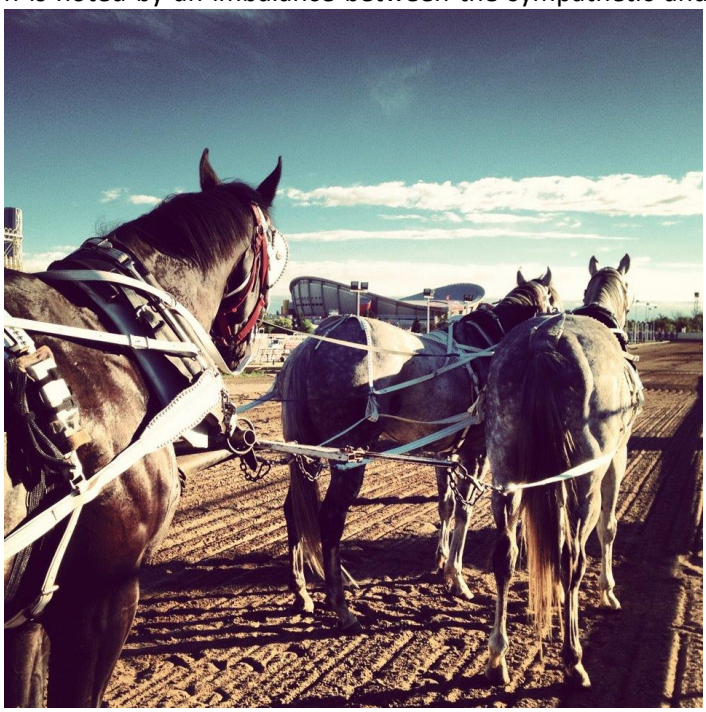
The severeness of the high lactate levels should also be measured considering the clinical appearance of the horses. During the vet checks these horses were considered fit to compete. As well, they showed no clinical signs of exercise intolerance like refusing to work or being slow and they appeared fit.

Day 1 and day 2 were not significantly different which might be a sign that the horses can manage the workload well, because after 1 day they can recover and return to baseline levels.

An influencing factor may be the post-blood pull time, which fluctuates for the different outfits. However this should be negligible, since all post-blood was pulled within 10 minutes after the race.

Arrhythmias

SVPCs and VPCs were noted frequently in this study. SVPCs are extra beats out of the regular sinus rhythm that arise from tissue above the ventricle, thus from the atria, and will be seen on the ECG as an extra QRS-complex. Prior to this QRS-complex a bizarre P wave might be visible or the P wave can be hidden by the preceding T wave. The QRS-complex is shaped normally but is not congruent with the normal heart rhythm. The ventricular rate is not affected by the SVPC. Causes for SVPCs are known to be myocardial inflammation or severe cardiac disease that has resulted in atrial dilation like atrioventricular valve disease, large ventricular septal defects or dilated cardiomyopathy. It can also be caused by systemic disease or any condition that alters autonomic tone and electrolyte and acid-base status. A lot of healthy horses show these arrhythmias without any clinical problems. Usually during exercise when sinus rate exceeds the firing rate of the single ectopic focus, there is overdrive suppression of the ectopic focus and cardiac rhythm is normal.[27] SVPCs are usually seen during recovery after intensive exercise and are then considered physological. The period immediately after exercise while the heartrate is going down is noted by an imbalance between the sympathetic and parasympathetic nervous system.



VPCs are extra beats outside of the sinus rhythm that arise electrically from the ventricles. They are seen in the ECG as an extra QRS-complex that is not consistently in the normal sinus rhythm. The QRS-complex is not preceded by a P wave. Also, the QRS-complex has a widened and larger shape than the other complexes. Five or more consecutive ventricular premature beats constitute ventricular tachycardia, a rapid and potentially life threatening rhythm that can lead to ventricular fibrillation and death. This event was not seen in this study. The underlying etiology of VPCs is not well understood, although increased excitability of equine ventricular myocardium is associated with ventricular dilatation and myocardial hypoxia, inflammation or necrosis. Myocardial ischemia might play a role in the genesis of the arrhythmias and can be a cause of sudden death.[28] Areas of the myocardium with cell damage caused by cell death, fibrosis, ischemia or hypoxia, might act as a focus of abnormal depolarization. These cells are not capable of maintaining the normal electrolyte balance across the cell membrane, resulting in a reduced negative resting potential and contractions out of the normal heart rhythm. The re-entry mechanism can occur as well, meaning that the signal resulting in extra beats keeps circling around of affected repolarization. VPCs are also associated with disease of other

body systems, electrolyte and acid-base derangement. Severe cardiac disease that has resulted in ventricular dilatation like aortic valve disease or dilated cardiomyopathy can cause VPCs. Myocardial inflammation and disruption of the normal intracardiac conduction system as well as extreme exertion and any other conditions that modify autonomic tone and induce hypoxia can also cause VPCs. If VPCs occur just in the early cardiac slowing period after fast exercise in the athletic horse, they rarely indicate cardiac pathology.[27] These events can be attributed to autonomic instability and increases in vagal tone in the horse.[29] [30] Infrequent VPCs can also occur during rest, exercise and recovery in normal horses.[43]

The present study shows that VPCs and SVPCs are common in chuckwagon horses during recovery, which is as frequently described in literature.[27] VPCs and SVPCs were expected during the first 2 minutes of recovery, as these have been reported in a large field study in harness racers[31] and on a small number of jumping warmblood horses.[33] However, what is remarkable in the results is that there are also quite a high amount of VPCs and SVPCs during the race. It is intriguing because these arrhythmias can be pathological during exercise.[27] [28] VPCs and SVPCs during exercise were also reported in other studies but not to this extent.[32] [33] However, VPCs and SVPCs have been detected in high frequency during exercise in show jumpers.[31] An overview of different studies in cardiac arrhythmias during exercise is presented in table 3. Race conditions and in particular chuckwagon racing could be more intense for the horses and thus can cause more arrhythmic events. The workers do not think that chuckwagon racing can be fairly compared to dressage or show jumping. It seems that the arrhythmias may be more forgiving during exercise than at rest since no signs of cardiac failure were seen during this study and that the events could even be considered physiological.[34] In this study it was found that the VPCs during racing were present in 4.1 % of the horses. That may imply that VPCs and SVPCs have a limited impact on the horses' performance, because none of them showed clinical signs of heart failure as exercise intolerance during this project.[35] However, damage to the myocardium might be possible and the author believes that there is a risk for the horses because of the possible event of ventricular tachycardia. This event can lead to the death of the horse. To prevent further cardiac damage and to guarantee the well-being of the horses to the public regular clinical check-ups of the horses are necessary for the acceptance and continuing of the chuckwagon racing sport.

Study	**SVPCs/VPCs during rest**	**SVPCs/VPCs during exercise**	**SVPCs/VPCs during recovery**
29		Occasional SVPC, no VPC	27.8 % VPC
30		SVPC 50 % VPC 3.8 %	SVPC 46.2 % VPC 19.2 %
31	SVPC 32 %	SVPC 89 % VPC 18 %	SVPC 54 % VPC 7 %
33	SVPC rarely	SVPC 28.6 % 2 VPCs in 1 horse	SVPC 61.9 %
34		SVPC 0 VPC 3 %	SVPC 7.1 % VPC 8.1%
Present study	SVPC 16.2 % VPC 14.7 %	SVPC 87.8 % VPC 4.1 %	SVPC 94.1 % VPC 5.9 %

Table 3: Comparison of cardiac arrhythmic events between different studies

Unfortunately, 82 ECGs were incomplete due to lost electrodes and would have probably revealed more arrhythmias if they would have been complete. The electrodes lost contact mainly on very hot days when the horses were sweating profusely.

Electrolyte disturbances resulting in arrhythmias

Myocyte depolarization and repolarization depend on intra- and extracellular shifts in ion gradients, meaning that abnormal serum electrolyte levels can have effects on cardiac conduction and the outcome of the ECG. Most common electrolyte abnormalities affecting the ECG are shifts in plasma potassium, calcium and magnesium. An increase in extracellular potassium decreases the amplitude of the action potential and the voltage of the plateau, shortens the plateau duration and accelerates the phase of rapid repolarization. There is alteration of the transmembrane potential gradient, a decrease in magnitude of the resting potential, and a decrease in velocity of phase 0 of the action potential. The potassium influx causes a shortening of the action potential and results in delayed conduction between the myocytes. Ultimately, these changes produce a slowing of conduction and with higher levels of serum potassium the conduction between myocytes is suppressed. Reduction in atrial and ventricular transmembrane potential causes an inactivation of the sodium channel, decreasing the cellular action potential. Atrial tissue is more sensitive to these changes. There can be suppression of sinoatrial and atrioventricular conduction, resulting in sinoatrial and atrioventricular blocks, often with escape beats.[36–40]

These changes were not found in this study during exercise, suggesting that the potassium levels did not reach high enough to induce these blocks. The minimum plasma potassium concentration required to induce electrocardiographic changes is 6.2 mmol/l and severe cardiotoxic effects were observed at levels of 8.0 to 10.1 mmol/l in an experimental situation.[41] Since potassium did not reach nearly as high in this study (maximal was 4.7 mmol/l), disturbances in potassium levels are not likely to be the cause of the arrhythmia that were found.

Hypocalcemia can trigger torsades de pointes in humans, but less common than with hypokalemia or hypomagnesemia. Serious hypocalcemia-induced dysrhythmias such as heart block and ventricular dysrhythmias are infrequent.[36] Disturbances in sodium and chloride levels are not common to cause arrhythmic events.[37]

An association exists between post-exercise sodium and chloride and the presence of arrhythmias during racing on day 1 as well as day 1 and 2 combined, however its clinical significance is unknown.

Conclusion

It can be concluded that it is possible to obtain good quality ECGs from racing chuckwagon horses.
Electrolyte changes pre- and post-exercise are as expected, and do not influence the arrhythmias that were found. The fact that the electrolytes did not return to baseline levels on day 2 before racing might implicate excessive exertion for the horses.
Post-exercise lactate levels are considered to be very high but not higher than found for other extreme sports. Lactate levels pre-exercise and post-exercise were not significantly different on day 2 compared to day 1 which might implicate that the horses recover well.
Arrhythmias were found with discrepancies in incident to what is confirmed in literature. Mostly SVPCs and VPCs were found in much higher percentages during rest and exercise. However, during this project they seem not to implicate pathology but more thorough examinations of the horses is necessary to ensure their wellbeing. It must be noted that the horses did not get a thorough clinical examination meaning that veterinary checks were minimal. If there were any checks, they mostly focused on lameness and major injuries like large wounds. The horses appeared fit, however it would be recommended that a full clinical examination be included in the events schedule for every horse. The author would suggest a full clinical examination, with a thorough examination of the cardiovasculary system including auscultation of the heart and lungs. By this one would at least be able to expel the horses with heart murmurs, and so delete the hazard of development of arrhythmias from valvular disease. Horses with irregularities would also be detected. In this way horses with exercise intolerance will be found at an earlier stage and hopefully the incidences of sudden death will decrease.

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Photo credits to Shannon Massie.

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