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STUDY OF POST EXERCISE BLOOD PRESSURE IN THE ENDURANCE HORSE

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STUDY OF POST EXERCISE BLOOD PRESSURE IN THE ENDURANCE HORSE

BY

Margaret Eilidh Wilson

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ABSTRACT

A STUDY OF POST EXERCISE BLOOD PRESSURE IN THE ENDURANCE HORSE

By

Margaret Eilidh Wilson

Endurance horses may develop post exercise hypotension a phenomenon commonly seen in human endurance athletes. Blood pressure may be measured indirectly with oscillometry however no published data exists on the accuracy of this technique in the standing conscious horse.

Thus an initial validation study was performed to compare the accuracy of an automatic indirect oscillometric blood pressure device with direct arterial blood pressure.

Subsequently the same indirect blood pressure device was used to measure pre and post blood pressure in horses competing in endurance races. The effect of exercise on blood pressure was then examined and post exercise hypotension was present in some horses.

TABLE OF CONTENTS

LIST OF TABLES.. iv

LIST OF FIGURES... v

CHAPTER 1
INTRODUCTION.. 1
References.. 14

CHAPTER 2
COMPARISON OF OSCILLOMETRIC AND DIRECT BLOOD PRESSURE
MEASUREMENTS IN CONSCIOUS ADULT HORSES.............................. 18
References.. 32

CHAPTER 3
MEASUREMENT OF POST EXERCISE BLOOD PRESSURE
IN HORSES COMPETING IN A 50 AND 100 MILE CROSS COUNTRY
RIDE AND A 75 MILE DESERT RIDE.. 36
References.. 48

LIST OF TABLES

Table 1.
Direct and Indirect blood pressure measurement of heart rate (beats per minute), systolic, diastolic and mean arterial pressure (mmHg) presented as Mean± SD, Bias (Direct minus indirect) and limits of agreement (Bias ±2SD) 28

Table 2.
Systolic, diastolic and mean arterial blood pressure (mmHg) and heart rate pre and post for each race. Presented as Mean ± SD, Significance was set at p<0.05.. 46

Table 3.
Hemotological changes pre and post race for 50 and 100 mile competitors......... 47

LIST OF FIGURES

Figure 1.
Correlation of direct and indirect blood pressure measurement for systolic (a), mean arterial (b) and diastolic pressure (c)........ 29

Figure 2.
Correlation of indirect and direct heart rate (a). Bland-Altman plot for heart rate (b). Each data point represents the difference between a pair of simultaneously measured direct and indirect measurements. The solid line represents the mean overall difference (direct – indirect) and the dashed line represents the limits of agreement (mean±2SD)........ 30

Figure 3.
Bland-Altman plots. Each data point represents the difference between a pair of simultaneously measured direct and indirect measurements. The solid line represents the mean overall difference (direct – indirect) and the dashed line represents the limits of agreement (mean±2SD). Systolic (a), mean arterial (b) and diastolic pressure (c)........ 31

CHAPTER 1

INTRODUCTION

Exercise-related disorders of endurance athletes

Endurance riding is a popular equine sport, especially in North America and the Middle East. In endurance rides horses race for distances of 80-160 km over varied terrain and in North America typical speeds are 8-12 km/h (Marlin DJ data unpublished). Endurance races are comparable to human long-distance running competitions, especially ultramarathons. Not surprisingly, medical problems observed in both long-distance runners and endurance horses are similar and these can be broadly categorized as musculoskeletal, metabolic or dermatological disorders.[1, 2]

In the horse musculoskeletal problems (i.e. lameness) are the most common reason for both performance limitation and completion failure.[3-5] Although less frequent, metabolic disturbances are often more serious and can, at times, be fatal.[2, 6, 7] The latter include hyperthermia, persistent tachycardia, dehydration, electrolyte and acid-base disturbances, synchronous diaphragmatic flutter, cardiac arrhythmias, altered neurological status, ileus, diarrhea, myopathy, coagulopathies, endotoxemia, renal failure and pulmonary edema.[2, 6-9] "Exhausted horse syndrome" (EHS) is a term that was coined to describe this constellation of medical problems that are considered to develop in horses pushed beyond their physiological capacity and reserve. It is most frequently observed in endurance athletes though it can occur in any horse subjected to high-level competition, especially when competing under adverse environmental conditions of high heat and humidity.[6-8, 10, 11]

Horses can be afflicted by differing degrees of severity and presenting complaints for exhausted horses can range in severity. Some may show vague, non-specific signs (e.g., persistent tachycardia or lack of appetite) where others demonstrate specific organ damage or dysfunction such as rhabdomyolisis or gastrointestinal ileus.[2, 8, 6, 12] Clinical signs are consistent with decreased perfusion of non-essential organs (e.g. kidneys and intestines) during prolonged exercise along with inadequate circulating volume, reflected by persistent tachycardia.[9, 12] Regardless of the presenting complaint of an affected horse the underlying mechanisms that contribute to development of exhaustion are believed to be similar and include prolonged hyperthermia, dehydration and depletion of fuel and electrolyte reserves.

To date, prevalence of the EHS has not been well documented and there has been little systematic research aimed at determining why certain horses develop metabolic disorders characteristic of the syndrome. Available information is mostly limited to individual case reports of affected horses or their inclusion in descriptive studies of actual competitions.[3,6,7,10] In North American rides sanctioned by the American Endurance Ride Conference (AERC), completion rates of 80- and 160-km rides are typically about 85% and 70%, respectively. The rate at which horses are "pulled" or eliminated from competition for development of metabolic problems ranges from 0-20%.[5] Using an average value of 10% for "metabolic pulls" for the nearly 1000 and 8000 horses that compete in 80- and 160-km rides in North America annually, it can be estimated that about 150 horses are eliminated as "metabolic pulls" each year. The AERC also monitors deaths of competition horses and complete necropsies are performed of all horses that die at AERC sanctioned rides. On a yearly basis, 5-10 endurance horses die in association with competition and at least 50% of these deaths appear to be a consequence of exhaustion.[5]

With the increased popularity of the sport in the Middle East over the past decade, however, the level of competition at international endurance races sanctioned by the Federation Equestre International (FEI) has increased dramatically. Speeds of 160-km rides have increased to 16-22 km/h (Marlin DJ personal communication) and, unfortunately, there have been a few highly publicized deaths of exhausted horses in recent World Equestrian Games (FEI.ORG). Thus there is a necessity for further information regarding risk factors for exhausted horse syndrome.

Exercise associated collapse (EAC) is a well-recognized medical problem affecting human endurance athletes. It can be caused by cardiovascular dysfunction, dilutional hyponatremia due to over vigorous water consumption, hyperthermia or hypotension.[1,13,14] Collapse typically occurs either late in the competition or shortly after cessation of vigorous exercise. Until fairly recently, most cases of EAC were unfairly attributed to hyperthermia and dehydration. Holtzhausen et al reported that 85% of athletes that collapse do so after the competition; thus, collapse occurs at a time when metabolic demands and heat production have dramatically decreased. Further, rectal temperatures of collapsed runners were not higher than runners that did not collapse. These observations suggest that persistent hyperthermia (characteristic of heat stroke) was not a common cause of EAC.

Next, nearly all long-distance runners become dehydrated during competition as a consequence of prolonged sweating.[14] The net loss of body fluid is best assessed by body mass (BM) during the event.[15] With long-distance running losses of 0-4% are commonly found with the wide range attributable to varied amounts of fluid replacement during exercise.[16] However, no investigator has been able to demonstrate that athletes that collapse are more dehydrated (greater BM loss) than those that do not.

Some of the most convincing evidence that dehydration is not a cause of EAC comes from studies of military personnel performed in the first half of the last century.[14] Soldiers exercised in desert conditions without fluid ingestion until they were >7% dehydrated. Physiologic changes that accompanied "dehydration exhaustion" included tachycardia and hypotension accompanied by character changes such as low moral, apathy and sometimes aggression. However, clinical signs of fatigue including weakness, nausea and light-headedness resolved within minutes after the recruits lay down and ingested water. A drastic improvement of clinical signs occurred well in advance of complete correction of dehydration. An additional interesting observation that was initially reported over 200 years ago and has since been corroborated in more recent studies is the development of a physiologic phenomenon of post-exercise hypotension (PEH) that is unrelated to dehydration.[14, 17] In particular following prolonged or strenuous exercise the PEH appears to be related to orthostatic intolerance leading to light-headedness and sometimes collapse. PEH is now recognized as the most common cause of EAC.[1, 13, 14] and accordingly treatment of collapsed long-distance runners has evolved over the past decade from rapid cooling and aggressive fluid support to a more conservative approach of having affected athletes lay down and elevated their legs for a short period of time, unless hyperthermia or hyponatremia are documented.[16]

Despite its association with EAC in endurance runners PEH is generally a benign condition. PEH was first described in 1898 by Leonard Hill and but within the past two decades has received considerable attention. PEH appears to be a normal physiological phenomenon that occurs following both aerobic (walking, running, cycling, swimming) and resistive exercise (leg and arm ergometry, weight lifting) at different exercise intensities and durations. The magnitude of hypotension that develops is variable on average being less than 10mmHg for systolic and diastolic blood pressure in normotensive individuals and is asymptomatic and indeed regarded as beneficial for health.[18, 19] However symptomatic hypotension can develop in people following severe physical exhaustion such as marathons races leading to symptoms of lightheadedness, nausea and syncopal episodes.[13, 14, 17]

The exact mechanism and regulation of post exercise hypotension has yet to be elucidated however it is generally accepted that it is caused by a persistent drop in peripheral vascular resistance which is not restricted to exercising muscle. There is evidence for resetting of the baroreflex stimulus response curve to a less sensitive operating set point and reduced muscle sympathetic nerve activity during PEH.[20-,22] However others have reported elevated sympathetic outflow during PEH based on heart rate variability.[23]

Alternatively, the vasculature itself may unresponsive to normal stimuli. Halliwill et al reported decreased transduction of sympathetic activity into vascular resistance during PEH and vasodilation occurs in the face of elevated rennin and catecholamines. [22,24] However in a later study Halliwill reported that the vascular response to phenylephrine (α1 agonist) and clonidine (α2 agonist) post exercise was similar to pre exercise.[25]

Possible vasodilating factors released by the muscle that may over ride the sympathetic nervous system have been investigated including nitric oxide, prostoglandins, histamine, ATP, and adenosine. Pre-treatment with a Histamine (H1) receptor antagonist prevents development of PEH as does pretreatment with caffeine which antagonizes vascular adenosine receptors suggesting possible roles.[26, 27] However treatment with nitric oxide synthase or cyclooxygenase inhibition did not reverse PEH excluding these as important factors.[26, 28]

Hypotension can also be induced following stimulation of the sciatic nerve or hind limb skeletal muscle highlighting the importance of muscle afferent nerves in the etiology of PEH.[29] Although not clearly understood the mechanisms responsible for PEH are likely to be multifactorial.

Hemodynamic responses vary with some studies finding increased cardiac output others reporting reduced cardiac output in the face of decreased peripheral resistance making interpretation difficult.[17, 23, 30]

The metabolic disorders that occur in endurance horses are poorly understood. Why individuals' develop exhausted horse syndrome during a race is unknown. Factors known to increase the risk of development include an unfit or under-conditioned horse, adverse environmental conditions (elevated humidity and temperature) and lameness.[6, 8] Elevated climatic temperatures and humidity may precipitate the development of dehydration and electrolyte imbalances. Horses, like humans, disperse heat through sweating and elevated temperature or humidity reduce the effectiveness of sweating, leading to increased sweating rates, and more severe dehydration and hyperthermia. Hypovolemia is often considered a significant component of EHS and accordingly treatment is often directed at restoration of circulating volume. However, there have been no critical studies that compare and contrast the level of dehydration, electrolytes disturbances and body temperature in both normal horses and horses suffering from EHS.

There is potential that horses may develop PEH which in some cases could contribute to and exacerbate cardiovascular stability leading to the conditions of poor perfusion that are recognized with EHS. Preliminary data supports this notion. Indirect blood pressure was measured in 6 horses before and following a 50 mile endurance race (Schott, unpublished). Post exercise mean arterial blood (MAP) pressure was significantly lower than the resting pre-exercise MAP. This is the first documentation of post exercise hypotension in the endurance horse though the numbers in the study were low and it requires further investigation.

Measurement of Blood Pressure In the Horse

There have been many different methods employed to measure blood pressure in the horse. Indeed the horse was integral to the first demonstration of blood pressure where by the Reverend Stephen Hales inserted a brass pipe connected to a long vertical glass pole into the carotid artery of a restrained horse. As the blood traveled nine feet up the pole he concluded that blood must be pumped by pressure. [31]

Following Hales many great minds contributed to the efforts of indirect blood pressure measurement one of the most famous was the Italian physician, Riva-Rocci who modified earlier designs and developed a mercury sphygomanometer that forms the basic blue print of which is still used today. At that time the blood pressure device consisted of a silk covered rubber band that was placed around the upper arm and fastened with a steel clamp to occlude the brachial artery. A rubber bulb inflated the cuff and a glass manometer filled with mercury measured the cuff pressure. The systolic pressure was registered as the cuff pressure at which the radial pressure was obliterated as determined by palpation.[32] This method of applying an external pressure to the artery occlude blood flow followed by release of pressure allowing the return of flow is the most common frame used to determine indirect blood. In Riva Rocci's sphygomanometer the systolic pressure was estimated by the palpation of the pulse.

Nikolai Korotkoff later realized that distinct noises could be heard in the artery when external pressure was removed allowing return of blood flow through the artery, these are now known as Korotkoff sounds or phases. Thus rather than relying on palpation of the pulse the systolic and diastolic pressure can be determined by auscultation. Despite the propensity for technical error, in human medicine, the auscultation method coupled with a mercury manometer is considered the 'gold standard' of indirect monitoring and is used to validate other electronic devices. However, 1998 the US Environmental Protection Agency identified mercury manometers as a bioaccumulative toxin and a health risk and for this reason many hospitals voluntarily removed them replacing them with aneroid manometers or electronic devices. However, due to concerns of accuracy and validation of electronic alternatives many believe this method should be reinstated.[33, 34]

Indirect doppler ultrasound was developed for use in military air transportation of casualties as the noise prevented use of Koratkoff sounds.[35] It uses the Doppler shift or Doppler effect principle: when a source generating waves moves relative to an observer or the observer moves relative to the source there is scattering of the waves and a change or shift in frequency. A piezoelectric crystal emits ultrasound waves of a fixed frequency into the arm or tail. Without compression there arterial wall does not move enough to produce a signal. However, when compression is applied between systolic and diastolic pressures the artery opens and closes with each pulse. The opening and closing movement have a different effect on the return frequency to the transducer. The signal is converted into an audio signal that can be heard through headphones and interpreted as systolic and diastolic pressure.

In the horse the first account of recording indirect blood pressure was performed by Shilling in 1919 who used an aneroid manometer. He applied an inflatable rubber cuff to the tails of 130 horses. A rubber bulb and aneroid manometer were then attached to the interior of the cuff. The cuff was inflated until the coccygeal artery was compressed such that visible fluctuations of the dial hand on the manometer were observed with each heart beat. The first reading was diastolic pressure and as the cuff was inflated further the systolic pressure was read.[36]

Since Shilling there are many accounts of indirect blood pressure measurement in the horse. All but a few utilize the middle coccygeal artery as described by Schilling as it is superficial and easily accessible in the adult horse. Different methods have been applied with varied success. The auscultation (Korotkoff sounds) method proved difficult and unreliable. Interpretation of sounds proved difficult unless the patient was very still and many of the auscultative events are below audible frequency making detection difficult.[36-38] Even in anesthetized horses the sounds were difficult to hear. Manual palpation of the coccygeal artery distal to the inflatable cuff, the same method that was used by Riva-Rocci in 1886, was deemed to be more reliable than plain auscultation in the horse however the accuracy of the system compared to direct measurement was not evaluated in this study.[37]

An electronic modification was applied to the auscultation technique such that a microphone was applied to the coccygeal artery to improve audibility. Systolic and diastolic pressures were determined in most horses but lacked accuracy and proved unreliable when horses displayed signs of shock.[39]

Doppler shift ultrasound appears to be an accurate means to measure blood pressure in the horse. Garner et al demonstrated it to be a simple technique that produced accurate results when compared to simultaneous direct femoral arterial blood pressure in standing conscious and laterally recumbent anaesthetized horses (correlation coefficients for systolic and diastolic values in standing horses were $r=0.99$ and $r=0.95$ and for anesthetized horses were $r=0.95$ and $r=0.91$ respectively.[34] Similar results were found in other studies in anesthetized horses.[41,42] However, a very poor correlation was detected between direct and indirect blood pressure when Doppler was applied to the tail in dorsally recumbent horses.[43]

Automated oscillometry is now the most common method of indirect blood pressure monitoring in both human and veterinary medicine. Oscillometric devises are technically complex consisting of an occluding cuff, pressure sensor, filters and amplifiers, analogue to digital converters and microprocessors. An inflatable cuff is placed over the artery and a series of pressures are applied to the artery. As different pressures are applied to the artery the arterial wall behaves differently and will oscillate at variable magnitudes. The pressure at which the oscillations have the maximum amplitude corresponds to the mean arterial pressures.

These low amplitude oscillations are superimposed onto the pressure within the occluding cuff. The pressure within the cuff is then applied to the pressure sensor which produces a voltage output proportional to the applied pressure. This information is then channeled through electronic amplifiers and filters. The filters hope to extract underlying occluding pressure from the superimposed pulse oscillations without dampening or erroneously altering the perceived signal. Once filtered and amplified the signal is converted into a digital signal and is communicated to the microprocessor whose task it is to simultaneously store the occluding cuff pressure and the matched pulse amplitude. The maximal amplitude oscillations correspond to mean arterial pressure and complex algorithms are applied to the pattern of oscillations around this center to extrapolate both systolic and diastolic pressures.[31]

The Dinamap [a] was the first commercially available oscillometric monitor and the first model was produced in 1976. Dinamap is an acronym for Device for Indirect Non-invasive Automatic Mean Arterial Pressure. The early models of the Dinamap have been investigated in both conscious[44] and anesthetized horses[45] and laterally recumbent anaesthetized foals.[46] Despite having a reputation for decreased performance under low flow states, Latshaw et al (1979) reported that measure mean arterial pressure could be consistently measured in hemorrhage induced hypotension. However, noted flaws of these early models included the inability to detect large changes in blood pressure.

It is hard to critically compare and contrast these early investigations of indirect blood pressure measurement in the horse. Many studies provide descriptive information without critical comparison with direct blood pressure.[48] Further, there are often differences with cuff placement or cuff size making comparison between studies impossible. In addition, investigations that compared an indirect blood pressure measurement with direct blood pressure often concluded accuracy of the two techniques based on correlation coefficients which does not relate to agreement between methods of indirect and direct blood pressure measurement.

Another important influencing factor is the technology used in the initial investigations of oscillometry in horses. All of these early studies used machines manufactured for humans. Two large differences between adult horses and humans are the lower resting heart rate and a different tissue compliance when comparing the tail and the arm. In 1991 the first automatic blood pressure monitor designed exclusively for veterinarians was designed. This was the Dinamap 8300[a]. The major modification was expansion of the range of heart rate detection from 40-220 beats per minute (human monitors) to a much wider detection range of 19-250 beats per minute making it more suitable for equine. This model was marketed through to 1999 before being discontinued and the manufacturing company Critkon Inc was dissolved. Although it is no longer manufactured it is still used by practitioners and refurbished models are easily acquired. Despite this there are no studies that evaluate this machine in horses.

Despite the predominance of oscillometric devises available there is limited peer reviewed data regarding accuracy of these devises in adult horses. Validation of oscillometric monitors is unregulated and only voluntary guidelines in human medicine exist. The Association for Advancement of Medical Instrumentation and the British Heart Society recommend that validation protocols be performed independently and that results should be published in peer review literature for future reference of clinicians and researchers. In a review of the accuracy of human ambulatory blood pressure monitoring devices only 9 out of 43 devices available on the market had successfully fulfilled the validation protocols.[49] This highlights the inaccuracy of some models and strengthens the need for more validation studies to be performed.

Summary

Thus, PEH occurs in people after endurance exercise and in some individuals results in orthostatic intolerance and syncope. Many of the clinical signs associated with exhaustive horse syndrome can be attributed to compromised perfusion. Though it is reasonable to assume that severe dehydration and consequently hypovolemia could develop as a consequence of prolonged and strenuous exercise especially under adverse climatic conditions this may not be the true for all cases. This is especially apparent in horses that develop EHS despite being well acclimatized and physically prepared for endurance rides. Further EHS can develop following short duration maximal exertion exercise where dehydration is not a factor.[10] Thus it is feasible to suggest that horses also develop PEH and that this could have a role in the development or course of EHS. However, the effect of exercise on blood pressure control in the horse is unestablished. Our goal is to measure blood pressure in normal competing endurance horses (chapter 3). Given a lack of critical data on blood pressure in standing conscious horses our method of blood pressure measurement (Dinamap oscillometric monitor) must first be validated (Chapter 2).

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Footnotes

[a] Critikon Dinamap™ Veterinary Blood Pressure Monitor 8300

CHAPTER 2

COMPARISON OF OSCILLOMETRIC AND DIRECT BLOOD PRESSURE MEASUREMENTS IN CONCIOUS ADULT HORSES

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Abstract

Automated indirect blood pressure monitors are commonly used to measure blood pressure in equine practice. Studies in anesthetized horses and recumbent foals have demonstrated that indirect measurements correlate well with direct blood pressure measurements. However, there are limited data comparing oscillometric indirect blood pressure to direct measurements in conscious, standing horses. Thus, 41 paired measurements of direct blood pressure (arterial canalization of the transverse facial artery) and indirect blood pressure (oscillometry performed over the coccygeal artery) were compared in six conscious, standing horses. Measurements determined by direct and indirect methods were significantly correlated ($r=0.57$, $p<0.001$; $r=0.54$, $p<0.003$; and $r=0.6$, $p<0.003$ for systolic, diastolic, and mean arterial pressures, respectively). However, oscillometry consistently underestimated direct measurements. Mean ± SD bias values (direct minus indirect measurements) of 18 ± 12, 22 ± 11, and 18 ± 10 mmHg were found for systolic, diastolic, and mean pressures, respectively. Although indirect oscillometry can be used to measure blood pressure in conscious, standing horses, values will be lower and somewhat less accurate than direct pressure measurements.

Introduction

Accurate assessment of blood pressure provides important information about the hemodynamic status of a patient. Common scenarios such as sepsis, endotoxemia, hypovolemia, and hemorrhage all produce cardiovascular alterations including hypotension.[1, 2] With hypotension, perfusion of vital organs can be compromised leading to organ damage and failure. Equally, hypertension can also cause organ damage and, in horses, hypertension has been recognized during the developmental stages of laminitis equine metabolic syndrome and insulin resistance.[3-8]

Maintenance of adequate blood pressure in the surgical patient is necessary to limit morbidity and mortality and hypotension in the anesthetized horse has been implicated as an important risk factor for development of post-surgical myopathy-neuropathy syndromes.[9-11] Consequently, monitoring blood pressure of anesthetized horses, either directly or indirectly, has become routine in equine practice.

In contrast, measurement of blood pressure in standing, conscious horses is less commonly performed although it could provide useful information in the evaluation and treatment of sick horses.

Exercising horses may also benefit from blood pressure monitoring. Human athletes experience a transient decrease in blood pressure following exercise, termed post-exercise hypotension (PEH).[12] This mild decrease in arterial blood pressure is attributed to a persistently reduced peripheral resistance.[13-15] Though generally benign, and, indeed, considered a beneficial effect of exercise it can in rare instances be accompanied by clinical signs such as light-headedness, nausea, and syncope.[16]
In particular, these signs may be observed during or after strenuous, exhaustive exercise

or prolonged endurance exercise.[15, 17, 18]

Post exercise hypotension has been documented in other quadripeds though whether it occurs in equids has not been established. If present, PEH could be a contributing factor to medical problems that can accompany exhaustion.[19-23]

Direct blood pressure measurement is straightforward during short-term anesthetic monitoring, however it can be impractical in the conscious, standing horse because arterial catheters can be difficult to place and maintain. Consequently, blood pressure is commonly measured indirectly using either doppler or oscillometric sphygmomanometry.

Oscillometry is now a widely accepted method for measurement of indirect blood pressure in both human and veterinary medicine.[24-26] Variations between different manufacturers and oscillometric models make it necessary to individually evaluate the accuracy of devices by comparison to direct blood pressure measurement before use in an experimental or clinical setting.[4, 26, 27] The Dinamap Veterinary Blood Pressure Monitor 8300™ [a] is an automated oscillometric device that was manufactured from 1993 to 1999 exclusively for veterinary use. This model has been popular for use in large animals due to its sturdy construction, portability, and long hose for connection to a tail cuff. However, there are no published reports of the accuracy of this oscillometric device in standing horses.

Thus, the purpose of this study was to validate the accuracy of the Dinamap 8300[a] in standing, conscious horses.

Materials and Methods

All procedures performed were approved by the Michigan State University Institutional Animal Care and Use Committee.

Horses: Six 2-year-old Arabian geldings performing a 60 km endurance exercise test on a high speed treadmill were studied. The test consisted of four 15 km exercise bouts (including walk, trot, and canter at 1.8, 4, and 8 m/s, respectively) separated by rest periods of 20-60 min.

Instrumentation: To measure direct arterial blood pressure, a catheter[b] was aseptically placed into the left transverse facial artery. The catheter was connected via fluid-filled rigid pressure tubing to an electronic pressure transducer[c] that was secured adjacent to the treadmill at the level of the shoulder joint (approximating level of the right atrium). A continuous flush system was also incorporated. The pressure transducer was connected to a multi channel monitor[d] and calibrated prior to each run using a mercury manometer. Direct blood pressure (systolic, diastolic and mean arterial pressure) and heart rate were continuously displayed. Indirect blood pressure was measured using the Dinamap 8300[a]. All geldings studied were of similar body size (withers height 146-151 cm) weight (376-437 kg) and tail circumference (between 17-25 cm) thus allowing use of a small adult cuff[e] based on manufacturers recommendations. The cuff was placed at the base of the tail over the coccygeal artery by the same operator each time to standardize the position and application and the same cuff was used for all horses. The difference in height between the heart base and mid-portion of the tail cuff was measured and each blood pressure measurement was corrected for hydrostatic difference prior to data analysis (each 1cm height = addition of 0.74mmHg).[29, 30]

Data acquisition: Indirect and direct blood pressures and heart rates (HR) were measured prior to the exercise test, during rest periods in between exercise bouts, and following completion of the 60 km run (with the horses standing quietly on the treadmill in all instances). At each data collection time point the Dinamap was automatically activated and heart rate, systolic, diastolic and mean arterial pressure were displayed upon the monitor at which point a corresponding direct measurement was collected. Measurements were obtained in triplicate or duplicate and an average for systolic, diastolic and mean arterial pressure was used for data analysis. Each horse performed the exercise test twice 2 weeks apart providing 18 opportunities for data acquisition.

Statistical analysis: All data were analyzed using a commercial software program.[f] Mean and standard deviation (SD) values were calculated for all data (systolic, diastolic, and mean arterial pressures and HR) reported by both measuring devices. Results of mean direct and indirect measurements were compared by Pearson correlation analysis. Subsequently, measurement bias (mean of differences of direct minus indirect values) and limits of agreement (± 2 SD) were determined using the method described by Bland and Altman.[31]

Results

Paired data were obtained for 41 time points. Mean ± SD systolic, diastolic, and mean pressures along with HR recorded by both indirect and direct methods are presented in

Table 1. All pressure and HR values recorded from the two devices were significantly correlated ($p<0.01$ for all, Figures 1 and 2).

Bias and 95% limits of agreement are also presented in Table 1. Bias for HR values was <1 beat/min indicating excellent agreement between the devices for HR determination (Figure 2). However, despite use of a correction factor for height of the tail cuff above the right atrium, indirect blood pressure measurements were consistently lower than direct measurements resulting in a positive bias of approximately 20 mmHg for direct measurements (Figure 3). Limits of agreement for systolic (-3 to 43 mmHg), diastolic (-1 to 45 mmHg) and mean (-2 to 38 mmHg) pressures were all similar.

Repeatability of each device was assessed by the mean of the SD of the difference between sequential measurements. Mean SD (mmHg) for sequential indirect blood pressure parameters were similar for both triplicate and duplicate measures; 7, 5, 7 for triplicate and 8, 7, 5 for duplicate for systolic, diastolic, and mean pressures, respectively. Similarly, mean SD for direct blood pressure were similar for both triplicate and duplicate measures; 6, 7, 5 for triplicate and 11, 7, 3 for duplicate measurements for systolic, diastolic, and mean pressures, respectively. Mean SD for repeated measurements of heart rate with indirect was 2 and 3 and for direct was 3 and 4 for duplicate and triplicate measurements respectively.

Discussion

This study illustrates that the Dinamap 8300 does not provide accurate blood pressure measurement in conscious standing horses with large degrees of variation between different methods.

The performance of other Dinamp models (prior to Model 8300 veterinary monitor) have been reported in horses with similar results. Geddes et al compared oscillometry (Dinamap, model not stated) in 12 anaesthetized ponies in lateral recumbancy with simultaneous direct blood pressure achieving a correlation coefficient of r=0.68 for mean arterial pressure.[35]

Muir et al investigated the Dinamap 1255 model in 73 anaesthetized (both lateral and dorsal recumbancy) and standing conscious horses.[37] Data for both groups was collectively reported finding correlation coefficients of >r=0.80 for systolic, diastolic and mean arterial pressures. However, all blood pressure measurements were considerably underestimated and there was a large degree of variability. Furthermore, bradycardia (,25 bpm), 2nd degree AV block, sinus arrhythmia and hypotension prevented oscillometric measurements in 12% of the horses. Fritsch et al also evaluated this device in standing horses but found oscillometry was unacceptably delayed in detecting pressure changes.[38]

These early models were designed for humans and the Dinamp 8300 followed being exclusively manufactured for the veterinary market. The major modification was expansion of the range of heart rate detection from 40-220 beats per minute (human monitors) to a much wider detection range of 19-250 beats per minute making it more suitable for equine.[32] In this study accuracy of heart rate measurement was good reflected by the small bias, although no horses in this study presented with bradycardia.

Nevertheless, accuracy of blood pressure measurement remained poor and similar to previous reports indirect blood pressure generally underestimated direct values by a mean of approximately 20mmHg for systolic, diastolic and mean arterial pressure.

The repeatability of each of the methods was similar which suggests that this is not a major limiting factor limiting the bias.[31]

Lack of accuracy of automatic oscillometric monitors has been highlighted as a problem in human medicine as manufacturers are loosely regulated.[4, 39]Inherent device qualities most likely contribute significantly to the poor accuracy observed in this study. Newer and more sensitive algorithms and step sensitive cuff deflation properties have been created in response to the demand from the medical field. Yet despite these advances in technology inaccuracy remains a problem. In a review of the accuracy of human ambulatory blood pressure monitoring devices only 9 out of 43 devices available on the market demonstrated acceptable accuracy (Bias 5mmHg ± SD ≤8mmHg).[26]

Other factors that may have contributed to our findings include location, motion and bladder width: tail circumference ratio.

Location of the inflatable cuff influenced accuracy in anesthetized foals.[40] However in that study, placement over the middle coccygeal artery improved accuracy of the monitor.

Motion artifact can influence cuff oscillations. Though all measurements were performed in a quiet controlled situation and patients stood still during measurements it is plausible that even small tail or body movements may have hindered the oscillometric technique. In anaesthetized or recumbent patients this is reduced. The most recent evaluation of oscillometry in horses was reported by Banson who investigated a veterinary blood pressure monitor[g] in anaesthetized adult horses.[34] Again oscillometry consistently underestimated direct blood pressure (Bias ± SD; Systolic 18±15, Diastolic

9±8, Mean arterial pressure 11±9) but the slightly improved accuracy may be attributed to the reduced motion.

The width of the cuff bladder relative to circumference of the extremity has been shown to be important and in humans the bladder width should be 40% of the upper arm circumference.[24, 25, 42] Blood pressure cuffs are generally manufactured in different sizes based on this literature. The small adult cuff[e] used in this study is recommended by the manufactures to encompass a human limb circumference of 17-25cm and all horses in this study had tail circumferences within this range. Although cuff size did not appear to affect accuracy in foals a previous study in adult horses revealed that a bladder width: tail girth ratio of between 0.2 and 0.25 improved accuracy using oscillometry.[33, 35, 41, 43] This suggests that use of human guidelines may be inappropriate for horses. There is a marked difference in the rigidity and tissue density of human extremities and foal and adult horse tails and this may play and influential role. Thus further investigation is required.

In this study the indirect blood pressure was measured from the coccygeal artery, an accepted site due to its superficial location, accessibility and the narrow tail which allows human inflatable cuffs to be used. However an unavoidable disadvantage is the effect of gravity requiring blood pressure values to be corrected.[29, 44] The effects of wave amplification on the pulse wave in peripheral arteries are well established. Parry et al detected a significant difference in systolic and diastolic blood pressure when measured from the greater metatarsal and middle coccygeal artery in individual anesthetized horses.[36] However, wave amplification has minimal effect on the mean arterial pressure and thus we consider the influence of wave reflection to be a minor contributor to the poor agreement observed in this study.

Recently, the accuracy of three different oscillometry devices was studied in anesthetized and conscious recumbent foals and good bias was acheived.[40, 41] Thus further research is required to determine if our main limiting factor was aged technology or if it is the inherent difference of the adult horse.

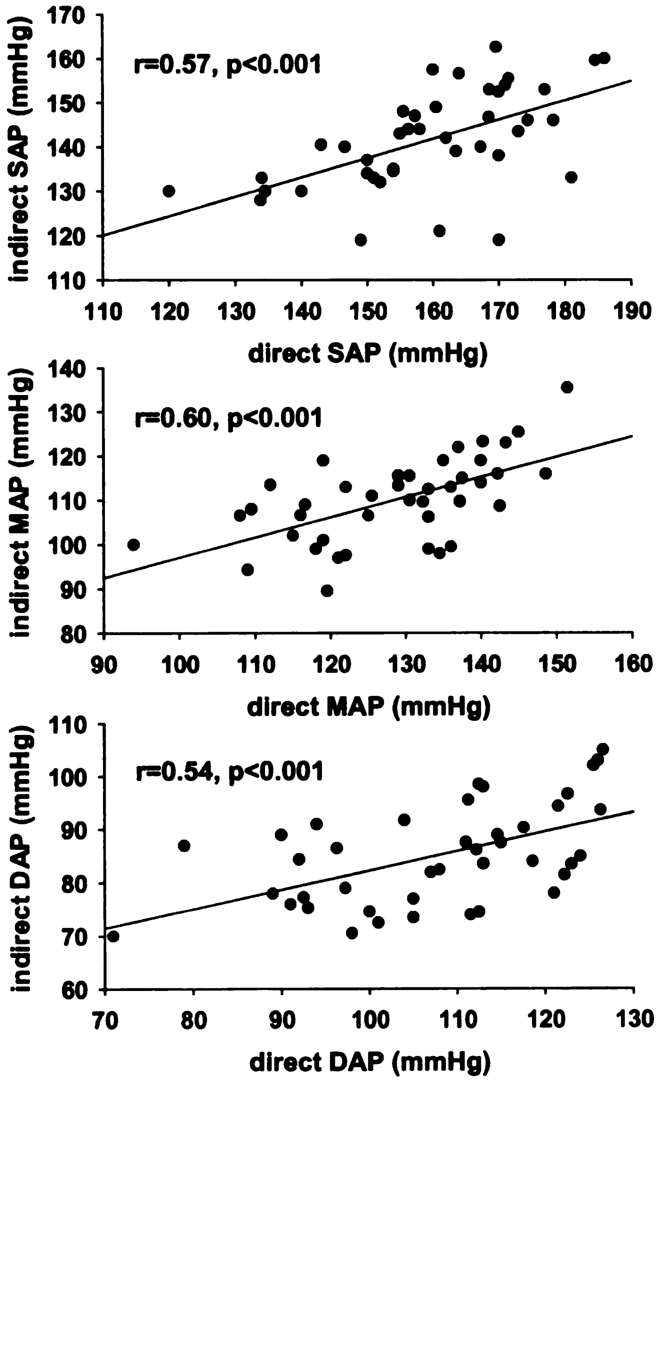
The intended purpose of this validation study was to assess performance of the Dinamap 8300 prior to a study of blood pressure changes in endurance horses. It is evident that the devise tested in this study has potential to provide inaccurate underestimation of blood pressure that could prompt inappropriate therapy. However for the purpose of our intended study a trend in blood pressure fluctuations is adequate. It is anticipated that following endurance exercise horses will develop hypotension. Our validation study model exercised horses over 60 km. Hypotension was not detected in any of these horses following termination of exercise. A limitation of this validation protocol was the absence of assessing agreement of the methods during induced hypotension. It is likely that with hypotension and low blood flow state a greater bias would be detected.

In conclusion, this study illustrates that the Dinamap 8300 does not provide accurate blood pressure measurement in conscious standing horses with large degrees of variation between different methods. However, it is incorrect to predict the same result would be present with modern and more technological data. Thus it is recommended that further studies are required to assess individual devices in the standing horse.

Table 1. Direct and Indirect blood pressure measurement of heart rate (beats per minute), systolic, diastolic and mean arterial pressure (mmHg) presented as Mean± SD, Bias (Direct minus indirect) and limits of agreement (Bias ± 2SD)

	Indirect	Direct	Bias	Limits of Agreement
Heart Rate	51±7	51±5	0.4±5	-10, +10
Systolic	142 ± 11	160 ± 15	18 ± 12	-3, +43
Diastolic	85 ± 9	108 ± 14	22 ± 11	-1, +45
Mean	110 ± 9	128 ± 13	18 ± 10	-2, +38

Figure 1, Correlation of direct and indirect blood pressure measurement for systolic (a), mean arterial (b) and diastolic pressure (c).



(a)

(b)

(c)

Figure 2. Correlation of indirect and direct heart rate (a). Bland-Altman plot for heart rate (b). Each data point represents the difference between a pair of simultaneously measured direct and indirect measurements. The solid line represents the mean overall difference (direct – indirect) and the dashed line represents the limits of agreement (mean±2SD).

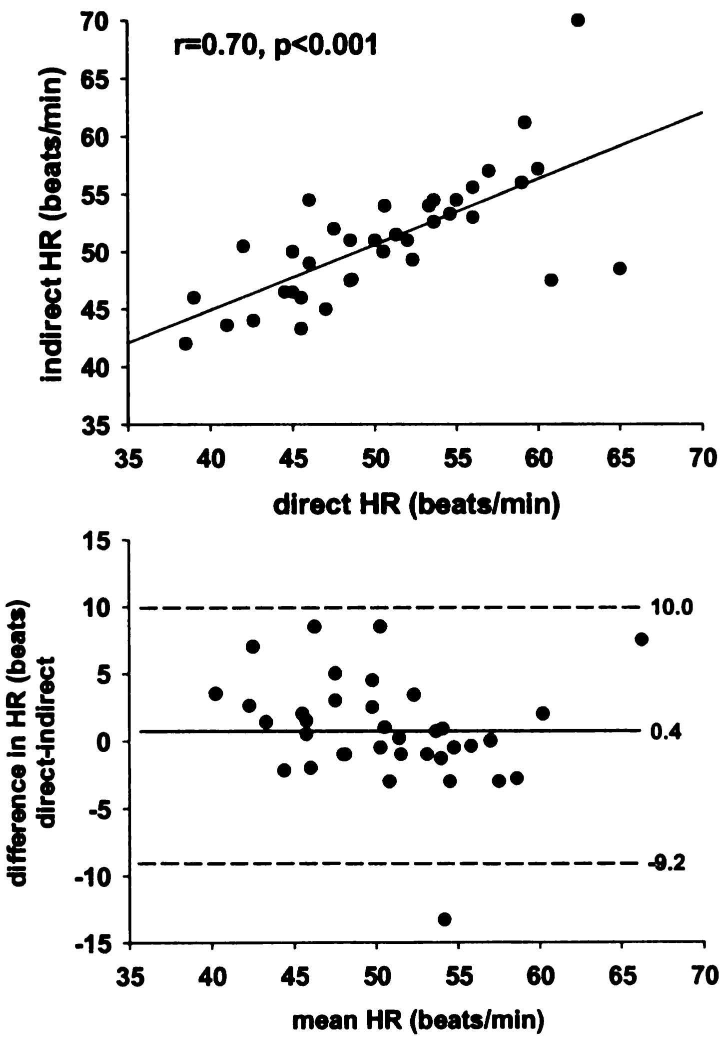
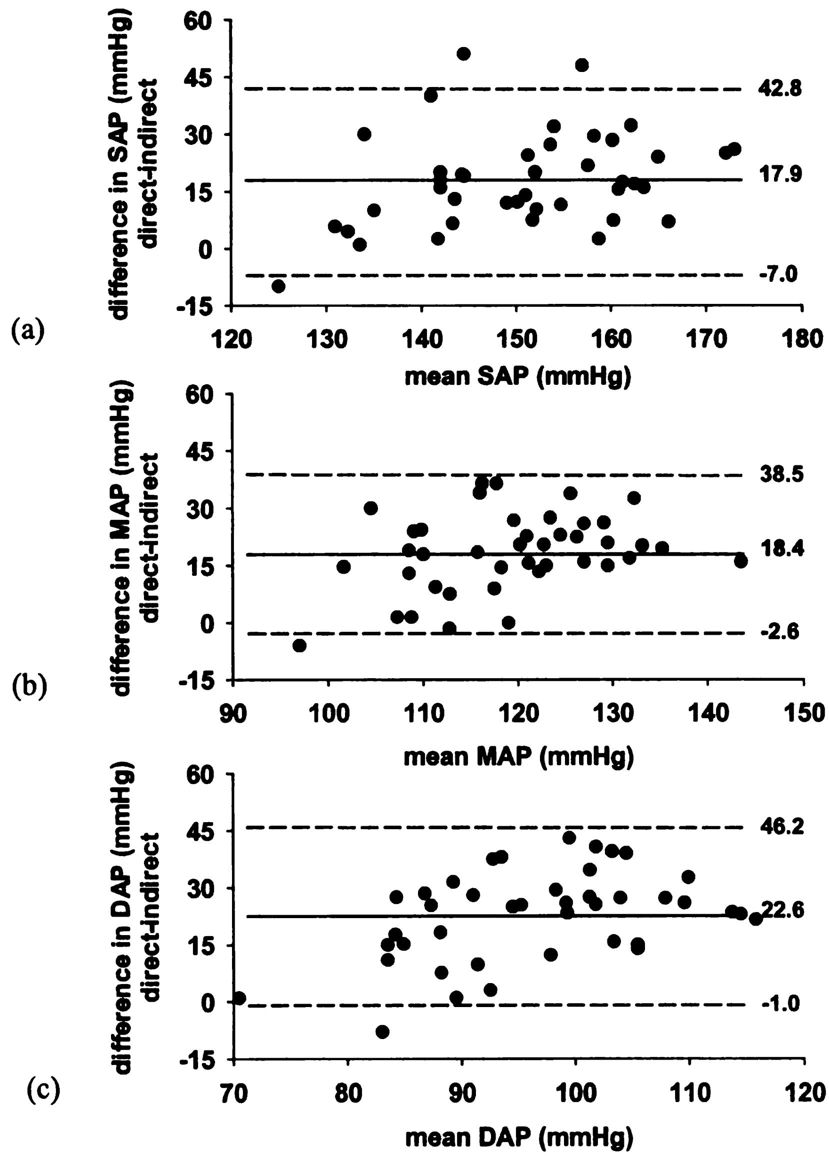


Figure 3. Bland-Altman plots. Each data point represents the difference between a pair of simultaneously measured direct and indirect measurements. The solid line represents the mean overall difference (direct – indirect) and the dashed line represents the limits of agreement (mean±2SD). Systolic (a), mean arterial (b) and diastolic pressure (c).



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Footnotes

[a] Critikon Dinamap™ Veterinary Blood Pressure Monitor 8300

[b] 20 gauge, 6. 25 cm polyurethane catheter Milacath®

[c] Philips Medical System M1000B Pressure module

[d] Datascope Passport 2 Multi Parameter Monitor (Datascope Crop, Paramus, NJ).

[e] Small adult cuff, limb circumference 17-25cm with 12 foot hose, Critikon

[f] 2005 Systat Software Inc

[g] Vet/BP 6000, Sensor Devices, Inc., Waukesha, WI

CHAPTER 3

MEASUREMENT OF POST EXERCISE BLOOD PRESSURE IN HORSES COMPETING IN A 50 AND 100 MILE CROSS COUNTRY RIDE AND A 75 MILE DESERT RIDE

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Data from 50 and 100 mile race presented as a poster at American College of Veterinary Internal Medicine Forum, Seattle, 2007.

Introduction

Post exercise hypotension (PEH) describes a persistent relative hypotension that develops in people following acute dynamic exercise. Although the mechanism behind PEH is unknown there is strong support for persistent vasodilation leading to venous pooling and reduced stroke volume a probable cause.[1-3] PEH can be profound enough to cause clinical signs such as light headedness and syncope. In particular this magnitude of PEH has been observed following severe or exhaustive exercise such as ultramarthons[3-5] PEH has also been observed in some quadrupeds.[6]

Preliminary data (Schott unpublished) demonstrated post exercise hypotension in a group of horses competing in a 50 mile endurance race thus suggesting that horses also develop this distinct phenomenon. It is feasible that PEH could exacerbate or contribute to exhausted horse syndrome (EHS) a condition that is associated with prolonged and exhaustive exercise.[7-10]

Thus the purpose of this study was to investigate the effects of endurance exercise on blood pressure in 3 groups of competing endurance horses. Our hypothesis was that post exercise hypotension would develop after successful completion of the race.

Study Design

For this study blood pressure was measured in horses competing in 3 endurance races. The first two races were of 50 mile and 100 mile duration and both took place at the American Endurance Ride Conference (AERC) National Championship Ride, Fort Valley, West Virginia, on the 26th and 27th October 2006 (respectively). The course was cross country with varied terrain that included forest, steep inclines and declines and rocky footing.

The third race studied horses competing in a Federation Equestre Internationale (FEI) CEI*** 120km (75 mile) flat desert race which was held at Dubai International Endurance City, Dubai, UAE in February 2007. All competitors were informed of the study in advance of each of the rides and volunteered their participation.

Blood pressure (BP), body mass (BM) and blood analysis was collected for the 50 and 100 mile races. Only BP and BM were measured for the 75 mile desert race. All measurements were obtained prior to the start of the race (the day before the race) and within 30 minutes after completion of the race. BP was measured using a portable indirect oscillometric monitor (Dinamap 8300[a]) and measurements were taken in duplicate and averaged. Accuracy of this oscillometric monitor was assessed in a prior study (Chapter 2). BM was measured with a load bar scale validated prior to use using set weights of 200kg and 600kg. Blood was analyzed for blood gases, electrolytes, hematocrit, total solids and osmolality. Heparinized blood samples were placed on ice until analyzed for pH, pO_2, and pCO_2, plasma electrolyte (Na^+, K^+, and Cl^-) concentrations; and plasma glucose and lactate concentrations within 30 min of collection.[b] The remainder of each sample was used for measurement of hematocrit (microhematocrit method) and plasma protein concentration (PP, by refractometry). Serum was used for measurement of Osmolality.

Weather Conditions

Weather conditions for the 75 mile desert race were agreeable. Peak air temperature was 82.4 F and though relative humidity was very high at the beginning of the race (6am) this declined and by mid day this had dropped to 20%. The Wet Bulb Globe Temperature (WBGT) is a composite temperature that is an index of probable heat stress. The WBGT for this race was classified as a category 1 indicating that temperature and humidity were not adverse for acclimatized athletes. All horses were resident to the area.

Statistics

All statistics were analyzed using commercial program (SigmaStat®, 2005 Systat Software Inc). All continuous variables (pre and post systolic, diastolic and mean blood pressures, body weight,) were measured using one-way repeated Measures ANOVA with one fixed factor (time).

Comparison of pre and post race blood gases, electrolytes, hematocrit, total solids and osmolality were measured using a paired t-test or a one-way repeated measures ANOVA. Significance was set at $p<0.05$. Correlation of post race blood pressure and body mass was assessed using a pearson correlation coefficient.

Results

Only horses that successfully completed each race were included in data analysis to eliminate confounding factors such as lameness or medical disorders that may have an additional affect on the blood pressure after exercise. For the 50 miles race, 14 of the 30 volunteers successfully completed the race. For the 100 mile race 8 of the 17 volunteers horses volunteered successfully completed. One horse successfully completed 91 miles before the rider became sick and removed the pair from the race. This horse was included in the data analysis as the horse was fit to continue. For the 75 mile race, 7 of the 22 horses completed successfully.

The average racing speeds were 9.2mph, 7.4mph and 12.4mph for the 50 and 100 and 75 mile races respectively. All horses that raced experienced a loss in BM with mean losses for the successful competitors being 4.2%, 6.5%, 5.7% for horses in 50 mile, 100 mile and 75 mile respectively.

There was a significant increase in heart rate post exercise for all three races (table 2). For horses competing in the 50 mile race the average systolic, diastolic and mean arterial pressures were significantly lower when compared to the starting blood pressure. No significant difference in post race blood pressures compared with pre race values for horses competing in the 100 mile or 75 mile races.

There was no correlation between body mass loss and the development of systolic, diastolic or mean arterial hypotension seen in horses from the 50 mile race and there was no relationship between post exercise blood pressure and body mass for the 100 mile and 75 mile race.

Blood Analysis

For 50 mile race: a significant elevations in hematocrit (pre race 35.4±4.9, post 42.6±3.9, p=0.03), total solids (pre race 6.8±0.2, post 7.1±0.4 p=0.03), lactate (pre race 0.4, post 1.27, p=0.04), BUN (pre race 16.67±2.7, post 22.9±2.1, p=0.001), and the calculated anion gap (pre race 9±1.7, post 12.9±1.5, p=0.001) were detected. A significant reduction in bicarbonate was detected (pre race 33.9±1.2, post 29.9±1.5, p=0.009). No significant changes of pH, glucose, osmolality or measured electrolyes (K, Na, Cl) were detected (table 3).

For 100 mile race: hematology was analyzed from 7 successful competitors. Findings mirrored changes observed in the 50 mile race except that there was no significant elevation in total solids. There was a significant increase in hematocrit (from 35.5% ± 2.2 to 40.7 ± 2.4 p=0.015). Post race elevations in BUN, (pre race 23.2±4.9, post race 25.3±4.9, p=0.001), lactate (pre race 0.55, post race 1.27 p=0.031) and Anion-Gap (pre race 10.3±1.1, post race 12.9±1, p=0.03) were observed, and reductions in iCa (pre race 6.09±0.2, post race 5.88±0.35, p=0.020) and HCO3 (pre race 32.7±0.9, post race 29.8±2.7, p=0.03) were also seen. There were no significant changes of pH, glucose, K, Na, Cl or osmolality when comparing pre and post race findings (table 2).

Discussion

The completion rate for the 50 mile and 100 mile endurance races were similar with 50% and 53% respectively for overall participation, and 47% and 48% respectively for study volunteers. This is slightly less than previous reports and is likely a reflection of increasing competitiveness and increasing racing speeds.[11-13]

The 75mile race had a lower completion rate of only 29% for the overall race and 32% for study volunteers. This is not atypical for endurance races in the Gulf States where races are highly competitive and racing speeds tend to be higher than USA races (Marlin DJ, data unpublished).

The reductions in BM observed in this study were typical compared to previous rides.[11, 14] The primary means of thermoregulation in the horse is evaporation of sweat. Muscular activity during an endurance ride creates a huge thermal load resulting in large volumes of sweat and substantial reductions in body mass loss.[10] Adverse climatic conditions such as increased temperature and humidity render sweating less efficient thus horses tend to sweat more. Though one of the races took place through a dessert where environmental factors would be an anticipated challenge for competitors all horses were resident to the region and were acclimatized to training at high temperatures and on the day of the race the climatic conditions were not severe.[15] In addition, thermoregulation was enhanced by supporters who would run along side horses as they were racing and douse them with chilled bottled water. Thus body mass loss reflects the duration of exercise and consequently greater sweat losses with the greatest body mass loss observed in the longest ride.

A significant reduction in BP (systolic, diastolic and mean) was observed only in horses competing in the 50 mile race indicating post exercise hypotension. Severe dehydration reduces circulating plasma volume and can result in hypotension. However, the degree of BM loss and dehydration was within the expected range and no correlation was found between body mass loss and mean arterial blood pressure making this less likely. This is further supported by the fact that PEH was seen in 50 mile competitors who demonstrated the most modest BM loss.

Although reduced plasma volume has a direct effect on stroke volume there is little evidence in humans that it has an important role in development of PEH.[16] In people hypotension can be elicited after only 10 minutes of moderate exercise and the duration of hypotension often surpasses beyond plasma volume restoration. Though the reduced plasma volume did not contribute to the PEH in the 50 mile competitors it is possible that increasing reductions in plasma volume could have an antagonistic effect on the mechanism of PEH and this could account for the absence of PEH in the more dehydrated horses.

There is no clear consensus on the effects of intensity or duration of exercise on the magnitude of PEH. There is some indication that increasing intensity of either aerobic or resistance exercise does increase the magnitude of PEH that develops however there are conflicting results.[17-20] Recently it has been suggested that total work done (total energy expended) rather than intensity of exercise may mediate PEH response.[21, 22] Jones et al established that both high intensity exercise and low intensity exercise would elicit the same degree of PEH when matched for total work done. Based on the racing speeds as an approximate indicator of exercise intensity the greatest intensity was performed by

75mile competitors and the least by 100 mile competitors however the 50 mile competitors would have expended the least amount of energy thus it is interesting that this was the only group to develop PEH.

The small sample size of 75 mile and 100 mile races could prevent detection of significance although when these results were combined a significant post exercise diastolic hypotension was detected. The implication of this is unknown. Further, studies in people have found that PEH consistently develops in borderline and hypertensive patients but is inconsistent in normotensive populations thus as all competitors had normal resting blood pressure this could be a normal variation.[23] However it is interesting to note that preliminary data (Schott unpublished) also only detected PEH in 50 mile competitors.

The degree of PEH observed in people during controlled studies varies but on average is reduced by 3- 9 mmHg (Systolic and diastolic). In the present study mean decrements of -13mmHg, -10mmHg and -7mmHg were detected for systolic, diastolic and mean arterial pressures respectively for the 50 mile race group. Species differences influence the magnitude of PEH and rats tend to develop greater magnitudes of hypotention.[16] However the accuracy of the blood pressure monitor could also influence the degree of hypotension detected. A previous validation study indicated poor accuracy and the indirect blood pressure monitor consistently underestimated direct values by approximately 20mmHg (Bias mmHg (direct –indirect) 18±12, 22±11, 18±10, systolic, diastolic and mean arterial pressure respectively).

Profound hypotension can develop in people following severe physical exhaustion and in these instances it appears to be associated with orthostatic intolerance.[4,]

[5, 24] Murrel et al demonstrated that the cerebral autoregulation remained normal following a marathon race and rather the altered autonomic function or baroreflex control placed the brain at risk of hypoperfusion.[5, 24]

Endurance horses can develop severe metabolic derangements and exhausted horse syndrome. Risk factors for developing EHS include under conditioning, musculoskeletal defects, adverse weather conditions especially elevated heat and humidity. It is possible that development of PEH and its associated impaired vascular regulation could contribute to or exacerbate this condition.

The hematological variables were similar for both the 50 mile and 100 mile competitors. Elevated hematocrit, BUN, Anion Gap were all mild and are a reflection of dehydration and reduction in plasma volume as a consequence of sweating during racing and are similar to previously reported studies. Lactate is produced during anaerobic respiration and substantial elevations are seen in race horses at the end of a race. The small elevation seen in this study is more likely due to dehydration or reduced hepatic perfusion and thus clearance as apposed to anaerobic respiration associated with high intensity exercise.

Commonly chloride anion deficits are observed during endurance exercise due to its loss in sweat however this was not seen during this event. In addition bicarbonate was actually seen to decrease slightly although remained within reference range. More commonly retention of bicarbonate is observed as a compensation for chloride losses. No information was collected regarding water consumption, appetite or electrolyte administration. Regardless, there were no clinically significant changes in acid base balance observed for competing horses.

Thus in conclusion, endurance horses develop PEH. This develops independent of plasma volume depletion that occurs with dehydration. Further investigations are required to determine if PEH can be repeated in other groups of horses.

Study Limitations

The small sample size of the 100 mile and 75 mile race was a limitation of the study (Power <0.35 and <0.05 for 100 mile and 75 mile respectively) although participation relies on volunteers and this is hard to resolve.

Secondly, the accuracy of the indirect blood pressure monitor used to measure the blood pressure was a limitation to the study and it is possible that subtle deviations in blood pressure could have been missed with this device.

Table 2. Systolic, diastolic and mean arterial blood pressure (mmHg) and heart rate pre and post for each race. Presented as Mean±SD, Significance was set at $p<0.05$

	50 miles (n=14)		75 miles (n=7)		100 miles (n=9)	
	Pre	Post	Pre	Post	Pre	Post
Systolic	116± 10	103±14*	112±13	117±22	105±14	106±9
diastolic	57±8	47±7**	60±16	55±19	48±8	51±9
Mean	84±9	71±9***	78±17	82±18	76±10	72±8
Heart Rate	36±3	55±7 †	34±6	50±6 ‡	38±4	53±7 †‡

*p<0.015, **p<0.02, ***p<0.015 † p<0.001 ‡ p<0.002 †‡p<0.008 compared to pre race values

Table 3. Hemotological changes pre and post race for 50 amd 100 mile competitors

		100	**Mile**		50	**Mile**	
Parameter	Normal	Pre	Post	P Value	Pre	Post	P Value
PCV %		***35.5±2.2***	***40.7±2.4***	***0.015***	**35.4±4.9**	**42.6±3.9**	**0.03**
Total Solids		6.8±0.35	6.7±0.36	0.48	**6.8±0.2**	**7.1±0.4**	**0.03**
pH		7.41	7.41	0.69	7.34±0.01	7.42±0.02	0.16
Sodium		141.4±1.44	141.4±0.68	0.128	143	141	0.57
Potassium		3.57±0.3	3.45±0.45	0.68	4	3.5	0.57
Chloride		103.5±1.6	103.1±2.9	0.8	103.3±1.6	103.1±2.5	0.86
iCalcium		***6.09±0.2***	***5.88±0.35***	***0.02***	6.4±0.3	6.1±0.36	0.08
Glucose		115	122	0.44	110±11.1	113.5±21	0.8
Lactate		***0.55***	***1.27***	***0.013***	***0.4***	***1.27***	***0.004***
BUN		***23.2±4.9***	***25.3±4.9***	***0.001***	***16.67±2.7***	***22.9±2.1***	***0.001***
HCO3		***32.7±0.9***	***29.8±2.7***	***0.03***	***33.9±1.2***	***29.9±2.7***	***0.009***
An Gap		***10.3±1.1***	***12.9±1***	***0.03***	***9±1.7***	***12.9±1.5***	***0.001***
Osmolality		289.7±2.1	288.5±1.6	0.32	286±3.2	289±4.8	0.07

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Footnotes

[a] Critikon Dinamap™ Veterinary Blood Pressure Monitor 8300

[b] (Stat Profile 9 Analyzer, Nova Biomedical, Waltham, MA)