



**A comparison of two treatment protocols in
the management of Exercise-Associated
Postural Hypotension (EAPH): A
randomised clinical trial**

**A dissertation prepared by Cameron Anley
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for the Master of Philosophy degree in Sports
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2 October 2007

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List of abbreviations

[Na ⁺]	Sodium
ACLS	Acute Cardiac Life Support
AVP	Arginine vasopressin
BP	Blood pressure
bpm	beats per minute
CM	Comrades Marathon
CO	Cardiac output
EAC	Exercise-Associated Collapse
EAPH	Exercise-Associated Postural Hypotension
ECG	Electrocardiogram
FEV1	Forced expiratory volume in one second
HR	Heart rate
IMT	Ironman Triathlon
IV	Intravenous
Km	Kilometre
MI	Myocardial Infarction
mmHg	Millimetres Mercury
mmol/l	Millimole per litre
°C	Degrees Centigrade
SNA	Sympathetic Nerve Activity
SV	Stroke Volume
TPR	Total Peripheral Resistance

Abstract

Background: Exercise-Associated Postural Hypotension (EAPH) is one of the most common conditions presenting in athletes following endurance events. However, due to the fact that there is no clear consensus on the pathophysiology of EAPH, there are two treatment protocols for this condition, namely, either intravenous fluids (to restore dehydration), or placing athletes in the Trendelenburg position (to treat venous pooling in the lower limbs). It has been argued that patients with EAPH will respond quicker to intravenous fluids, but no study has compared the time to discharge from the medical facility using these two protocols.

Objective: The aim of this study was to compare which of the two commonly used treatment protocols for Exercise Associated Postural Hypotension (EAPH) (Trendelenburg with oral fluids ad libitum or intravenous fluids) result in quicker recovery and earlier discharge from the medical facility.

Methods: All athletes who collapsed due to Exercise-Associated Postural Hypotension at the South African Ironman 2006, South African Ironman 2007 and the Comrades Marathon 2006, were considered for the trial. Athletes who collapsed underwent clinical assessment and special investigations (using specific inclusion criteria). Thirty-two athletes with EAPH were recruited and 4 were excluded. The remaining 28 athletes were randomised, based on the last digit of their race number. Fourteen athletes were randomised to the Trendelenburg and oral fluid protocol (oral group), and 14 athletes were randomised to the intravenous fluid protocol (IV group). Fluid intake was recorded, and all the athletes were assessed clinically (blood

pressure, heart rate) every 15 minutes until discharge (using the specific discharge criteria). The main measure of outcome was the time to discharge (min).

Results: On admission, groups were similar with respect to age (37.7 ± 7.2 years, 36.5 ± 7.1 ; $P=0.66$), systolic blood pressure (110 ± 11 mmHg; 106 ± 8 mmHg; $P=0.21$), heart rate (81 ± 10 bpm; 90 ± 14 bpm; $P=0.79$) and serum sodium concentration (139 ± 3 mmol/l; 141 ± 4 mmol/l; $P=0.18$). There were no significant differences with regards to systolic blood pressure, diastolic blood pressure and heart rate between groups and over time until discharge. The fluid intake during the treatment period was significantly greater in the IV group, 1045 ± 185 ml (N = 12) compared to the oral group, 204 ± 149 ml (N = 11) ($P<0.001$). The average time to discharge for the oral group (58 ± 23 min) was similar to that of the IV group (52.5 ± 18 min) ($P=0.47$).

Conclusion: In conclusion, athletes with EAPH can be treated effectively using the Trendelenburg position and oral fluids. The administration of IV fluids does not reduce the time to discharge. The findings of this study support the hypothesis that EAPH is as a result of venous pooling due to peripheral vasodilatation, rather than dehydration.

Keywords: exercise, associated, hypotension, treatment, oral, intravenous

Chapter 1

Introduction and scope of the dissertation

The South African Ironman Triathlon and Comrades Marathon are both ultra-endurance events that are held annually in South Africa. The South African Ironman Triathlon consists of a 3.8km swim, 180km cycle and a 42.2km run. The Comrades Marathon is an 89km road run held in KwaZulu-Natal, South Africa. Both of these events attract large fields of athletes.

Athletes who participate in endurance events such as these can place themselves at an increased risk of developing a variety of medical conditions. These medical conditions can range from minor ailments, such as blisters, to life-threatening conditions, such as myocardial infarction and sudden death¹⁻⁷. The medical staff who provide the medical care at endurance events therefore need to have a diagnostic and treatment plan for a wide variety of medical conditions.

Previously published data from the medical facilities at these events have shown that one of the most common clinical presentations at the medical tent is the “collapsed” athlete. It has also been documented that, in the majority of cases, “collapse” in athletes is relatively benign, and has generally been referred to in the literature as Exercise-Associated Collapse⁸⁻¹⁴. However, many other terms describing this condition have also been used, including

postural hypotension ^{15;16}, heat exhaustion ¹⁵, exercise-related collapse ¹⁷, post-exercise postural hypotension ¹⁸, heat syncope ¹⁵, and neurocardiogenic syncope ¹⁹.

From the literature, it appears that various terms have been used to refer to a very similar (or the same) symptom/sign complex that describes the presentation of the “collapsed” athlete on completion of an endurance event. The main focus of this dissertation is on the clinical approach and, in particular, the management of the symptom/sign complex that describes this relatively benign, but very common, cause of the “collapsed” endurance athlete.

In Chapter 2, the terminology and definitions used to describe the “collapsed” athlete will firstly be reviewed. This will be followed by a review of the epidemiology, and pathophysiology of this symptoms/sign complex that results in post-exercise “collapse”. In particular, it will be argued that the most likely cause for this complex is postural hypotension that develops as a result of the pooling of blood in the lower limbs on completion of exercise. This pooling occurs when the muscle pump (also referred to as the second heart) is “removed” when the athlete stops exercising. Therefore, in this dissertation, this symptom/sign complex will then be referred to as Exercise-Associated Postural Hypotension (EAPH). Finally, in Chapter 2, the current clinical approach to the diagnosis and management of EAPH will be reviewed. It will be pointed out in this review that there are two distinct approaches to the management of EAPH, which are largely based on two different hypotheses

to explain the pathophysiology of this condition. These two management approaches are 1) to administer intravenous fluid in order to restore plasma volume (dehydration hypothesis), and 2) to raise the lower limb and hips so that venous return increases (Trendelenburg position) and providing oral fluids (postural hypotension hypothesis). There are no studies that have been conducted to compare clinical outcomes using these two treatment approaches for EAPH.

In Chapter 3 the results of an original randomised clinical trial will be presented. This trial was designed to compare the use of intravenous fluids versus oral fluids with the Trendelenburg position in the treatment of “collapsed” athletes with EAPH. This study was conducted at the South African Ironman Triathlon 2006 and 2007, and Comrades Marathon 2006. Finally, in Chapter 4, a summary of the literature review and the findings of the original research study, together with recommendations, will be presented.

Chapter 2

Post-exercise collapse following ultra-endurance events: A review of the pathophysiology and clinical management

2.1. Introduction

The Comrades Marathon (CM) and the South African Ironman Triathlon (IMT) are both popular ultra-endurance events that are held annually in South Africa. The Comrades Marathon is an 89 kilometre (km) running race that is held between the cities of Durban and Pietermaritzburg in the KwaZulu-Natal province of South Africa. The race, which takes place in June (winter), alternates between the “up” run (from sea level to an altitude of 650m) and the “down” run (from 650m to sea level), and has a cut-off time of 12 hours.

The South African Ironman Triathlon is an ultra-triathlon that is held in the city of Port Elizabeth, which is in the Eastern Cape province of South Africa. This Ironman Triathlon consists of a 3,8km sea swim followed by a 180km road cycle and, finally, a 42,2km run. The race has a cut-off time of 17 hours.

In both these events, the participants represent a spectrum of athletes, and include both professional and recreational athletes of varying ability and

expertise. This means that the athletes who participate may potentially suffer from a range of underlying medical conditions. Currently, there is no specific medical screening to enter the Comrades Marathon or the South African Ironman Triathlon; hence athletes may suffer from any known or sub-clinical medical conditions.

Due to the strenuous nature (physical and mental) of these ultra-endurance events, athletes who participate in them are at risk of developing various medical conditions that are either known, or that can manifest for the first time as a result of participating in endurance events ^{1,4;20-24}. The provision of adequate medical care during and after these events is therefore important. In order to ensure that the athletes receive the appropriate and timely medical care, it is essential that the medical staff at these endurance events are familiar with the common, as well as the possibly life-threatening medical conditions which may present to the staff at the medical facility. Knowledge about possible medical emergencies that may present to the medical facility is important so that the medical director and staff can ensure that the correct facilities and protocols for the provision of medical care are available.

In this chapter, the general epidemiology and the nature of medical problems that may be encountered at ultra-endurance events will be briefly reviewed. This will be followed by a more detailed review of the definition, pathophysiology and current controversies pertaining to the management of post-exercise collapse in ultra-endurance athletes. This condition, which is the focus of this dissertation, is one of the most common reasons for admission to

the medical facility. Yet there is considerable controversy about the appropriate management of athletes presenting with post-exercise collapse.

2.2. Medical conditions encountered at ultra-endurance events

2.2.1. Epidemiology of medical conditions

The incidence of medical admissions following ultra-endurance events can be defined as the percentage (%) of all athletes who started the race and who require medical treatment during or after the race. The incidence of medical admissions has been studied and reported for a number of ultra-endurance events²⁵⁻²⁹. The incidence of medical admissions at standard marathons (42.2km) ranges from 1.9%-12.3%^{30,31}, but this can increase to 13%-25%^{1,28,32} in ultra-endurance events such as the Ironman Triathlon. This large variation and increase in admissions can be explained, in part, by an increased race distance (exercise duration) and an increase in ambient temperature during the event.

The effect of race distance on the incidence of medical admissions during ultra-endurance triathlons is depicted in Table 2.1. It is clear that as exercise duration (race distance) increases, the incidence of medical admissions (% of athletes seeking medical care) increases.

Table 2.1.: The incidence of medical admissions following triathlon events of different distances (Adapted from ³³)

Race	United States Triathlon series	Ironman qualifiers	Hawaii Ironman
Distance (Swim/Cycle/Run)	1.5km/40km/10km	1.9km/90.2km/21.1km	3.9km/180km/42.2km
Finishing time	2–4hrs	4–8hrs	9–17hrs
Incidence of medical admissions (%)	2%	10%	17%

A second important factor that is related to the incidence of medical admissions following ultra-endurance events appears to be ambient temperature. The influence of ambient temperature on the incidence of medical admissions has been studied ^{4,34} and is summarised in Table 2.2. The effect of an increased ambient temperature during a race on the incidence of medical admissions is highlighted by data that were accumulated over twelve years at the Twin Cities Marathon ³⁵. In this study, it was shown that the rate of collapse in athletes increased as the dew point increased.

Table 2.2.: The effect of the length of a race and the weather on the percentage of race starters who collapse and require medical treatment ³⁶

Sun-City-to-Surf, Australia		Boston Marathon, USA		Two Oceans Marathon, RSA		Comrades Marathon, RSA	
12km		42km		56km		89km	
1984	1980	1987	1985	1990	1989	1985	1987
Cool	Warm	Cool	Warm	Cool	Warm	Cool	Warm
0.17%	0.3%	0.5%	1.3%	0.7%	3.7%	1.6%	3.6%

2.2.2. Nature of medical admissions following ultra-endurance events

The common and potentially life-threatening medical conditions which may present to the medical facility following ultra-endurance events are depicted in Table 2.3. It should be noted that various medical conditions may present clinically as “collapse”. There is considerable variation in the definition of what constitutes a “collapsed” athlete, but this will be reviewed in detail in Section 2.3.2.

Table 2.3.: Medical conditions which may present to the medical facility at endurance events ^{1;4;37-41}

Cardiovascular *	Exercise-Associated Postural Hypotension (EAPH) Myocardial Infarction (MI) Angina Cardiac arrest Dysrhythmias
Musculoskeletal	Cramps* Stress or True fractures* Tendonitis Sprains/ Strains
Dermatological	Abrasions (Chafe)/Lacerations Blisters
Respiratory *	Asthma
Metabolic and fluid status *	Hyponatremia Hypoglycaemia Hypernatremia Dehydration
Temperature *	Hypothermia Heatstroke
Neurological *	Cerebral Vascular Accident Epilepsy Intracranial bleeds
Gastro-intestinal	Nausea Vomiting Gastroenteritis
Other	Near drowning * Stings – Sea anemone Eye irritation Anaphylaxis Exhaustion

* Medical conditions that may present as “collapse”

It is also important to emphasise that the triathlon consists of three components, namely, swim, cycle and run. Specific medical conditions that may result from participation in each discipline may therefore occur. Hypothermia and near drowning are more common during the swim and thus the medical facility should be prepared for these conditions during the first 2–3 hours of such an event. Acute traumatic injuries, such as fractures and head injuries, are more common on the cycling leg. These can be serious conditions and may require transfer to an emergency room for further treatment. In most instances, dehydration is reported as the most common medical condition treated during the run, or post-race, in various events. However, in most studies the precise diagnostic criteria that were used to diagnose dehydration are not recorded ^{1;42;43}.

It should be noted that the precise incidence of specific presenting complaints has not been reported in most studies ^{28;44;45}. With the exception of one study ⁴⁶, the reasons for medical admission were grouped into large categories, with little detail on the exact presenting medical complaint. Broad categories of medical conditions, such as dehydration, trauma, exhaustion, cramps, and electrolyte imbalance, are therefore commonly used ^{1;28;47-49}. This is a limitation in most studies where data on medical admissions have been reported.

The frequency (% of all cases admitted to the medical facility) of diagnostic categories of medical problems encountered in the medical facility following ultra-endurance events is depicted in Table 2.4.

Table 2.4.: The frequency (% of all cases admitted) of categories of medical conditions following ultra-endurance events ^{1:28;50-52}

	Race				
	Hawaii Ironman 1988	Twin Cities Marathon 1982-1994	Hawaii Ironman 1984	Hawaii Ironman 1985	Ironman World Championship 1984
Dehydration	52		47	49	47
Exhaustion	20		20	9	19
Trauma	13		22	13	22
Cramps	6	6.1	20	9	22
Electrolyte imbalance	5				
Hyperthermia	0.6				
Other	4	0.74	22	19	20
Exercise-Associated Collapse (EAC)		59.4			
Abrasions/Blisters		21.6			
Sprain/Strain		15.6			
Stress fracture (Suspected)		0.89			
Severe medical conditions		0.14			
Knee pain		0.2			
Fasciitis		0.75			
GIT problems			3	7	7
Hypoglycaemia					9

It is clear from the results presented in Table 2.4. that dehydration is frequently reported as the most common reason for admission to a medical facility at ultra-endurance events. In three studies, it has been reported that dehydration is responsible for 47%-52% of all cases admitted to the medical facility ^{28;53;54}. However, in two of these reports, there is little information as to how the exact diagnosis of dehydration was made ^{28;55}. In the third report, the diagnosis of dehydration was based on a symptom/sign complex of dizziness, tachycardia, syncope, inability to urinate, and hypotension ⁽⁵⁶⁾. However, dehydration is not the only possible cause of this symptom/sign complex, and the exact cause of this sign complex has become the topic of much debate.

The two most common explanations for this symptom/sign complex are 1) dehydration, resulting in hyperthermia, which leads to circulatory collapse ^{57;58} or 2) Exercise-Associated Postural Hypotension (EAPH) due to the pooling of blood in the dilated venous system of the lower limbs on completion of exercise ^{17;59-63}. The possible underlying mechanisms for this symptom/sign complex will be discussed in detail in Section 2.4.4.

2.2.3. Summary: Medical conditions at ultra-endurance events

In summary, there is a lack of well-conducted prospective studies detailing the epidemiology of medical conditions following participation in ultra-endurance events. The value of current information is important as this information is essential to adequately plan the medical facilities at these events. Medical conditions at ultra-endurance events can range from minor non-life-

threatening ailments, such as blisters, to life-threatening conditions, such as collapse due to myocardial infarction. Medical staff need to appreciate that the athletes will present throughout the event, but that sudden increases in admissions can be expected at various stages during the event, often related to medal cut-off times⁶⁴. Furthermore, critically ill athletes may present alongside athletes suffering from relatively minor ailments. It is thus essential that the athletes admitted to a medical facility undergo an appropriate initial screening and triage. Guidelines on how to perform the appropriate triage in medical facilities at endurance events have been published^{4;65;66}. Appropriate triage is essential to prevent a delay in the identification and management of acutely life-threatening conditions.

It is important to note that the incidence of the life-threatening conditions is low^{67;68}. However, medical staff should have a sound clinical, diagnostic and management approach to the more common medical conditions at endurance events. One of the most common clinical scenarios encountered in the medical facility at endurance events is that of the “collapsed” athlete. A discussion of the general clinical approach to the “collapsed” athlete is therefore important.

2.3. General clinical approach to the collapsed ultra-endurance athlete

2.3.1. Introduction

One of the most common clinical presentations during and following an endurance athletic event is that of the “collapsed” athlete. As previously mentioned, there are several postulated causes that can lead to “collapse” in athletes. The causes and management of the “collapsed” athlete have previously been reviewed⁶⁹⁻⁷². The focus of this dissertation is mainly on the management of the athlete suffering from collapse as a result of Exercise-Associated Postural Hypotension (EAPH). However, it is important to define the terminology used for the “collapsed” athlete, and then to briefly review the general clinical approach to “collapsed” athletes. Thereafter, the remainder of this review will focus on the specific condition known as Exercise-Associated Postural Hypotension (EAPH).

2.3.2. Definition of the “collapsed” athlete

In the context of an athlete presenting to a medical facility, the use of the word “collapse” is a non-specific and broad term. A review of the literature has shown that there is no universally accepted definition of the word “collapse.” For the purposes of this dissertation, the term “collapse” will be used for an athlete who is unable to maintain an upright posture, with or without loss of consciousness.

2.3.3. Epidemiology of the “collapsed” endurance athlete

The reported incidence of “collapse” in an ultra-endurance event ranges from 0.2%-3.7% of all race starters⁷³. The incidence of “collapse” is influenced by various factors, and, as previously mentioned, race distance and ambient temperature appear to be important factors. The incidence of “collapse” has been compared in four footraces of different lengths and on cool and warm days (Table 2.2.). The incidence of “collapse” was reported as 0.2% in a short race on a cool day and up to 3.7% in an ultra-marathon on a warm day.

In two extensive descriptive studies conducted during Australia’s Sun-City-to-Surf 12km race⁷⁴ over a twelve-year period in which the rate of collapse was directly proportional to the dew point⁷⁵. An increase in the ambient temperature and humidity was associated with an increase in the number of collapsed athletes. Apart from highlighting the possible effect of environmental conditions on the number of collapses, the results of this study also shows how the rate of collapse can be decreased through education of the participants. In 1978, the incidence of collapse decreased, which coincided with the introduction of an education programme highlighting heat-related illnesses and the importance of appropriate training⁷⁶.

Other factors that have been highlighted as possible risk factors for collapse, and thus medical admission, include inadequate training, poor attention to nutrition before and during the event, and the presence of any pre-race illness

⁷⁷. A detailed discussion of the possible roles of each of these factors is beyond the scope of this dissertation.

2.3.4. General clinical approach to the “collapsed” endurance athlete

The immediate clinical approach to the collapsed athlete should be focused on initial assessment and stabilisation of the athlete, and this should take priority over making an actual diagnosis. The “A B C” (see below) approach is internationally accepted according to the Acute Cardiac Life Support (ACLS) guidelines, and is an accepted approach to an acutely ill patient, including the collapsed athlete ⁷⁸. In these circumstances, a systematic approach should be followed that will provide the medical staff with vital information and allow for the timeous diagnosis and treatment of acute life-threatening conditions. Generally, the following is referred to as the “primary survey”:

A: Airway - athletes who can vocalise have a patent airway.

B: Breathing - Rate and rhythm.

C: Circulation - Pulse: rate and rhythm and vital signs, including BP. Gain an impression of the hydration status.

D: Disability - Documented via the **A V P U** system - **A**lert, responding to **V**ocal stimulus, responding to **P**ain and **U**nresponsive. Additionally, the degree of confusion and orientation should be noted.

E: Exposure - The athlete’s temperature (preferably core temperature) should be taken.

Any abnormalities detected in these assessments should be treated immediately using internationally accepted guidelines, such as the ACLS guidelines. It is important to remember that a collateral history from the stretcher bearers and fellow athletes should also be taken. If the pre-race weight is known, an attempt should be made to obtain an accurate post-race weight. However, in practice, this may be difficult to obtain.

In the case of the “collapsed” athlete, the primary survey should provide information that will differentiate the severe and less-severe “collapsed” athlete (Table 2.5.).

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Table 2.5.: Guidelines for determining the severity of the “collapsed” athlete (Adapted ⁷⁹).

	Less-severe	Severe
Site of Collapse	Finish	On course
Primary Assessment		
Disability	Conscious and Alert	Decreased consciousness or unconscious, confused or disorientated.
Temperature (rectal)	< 40°C	> 40°C
Supine systolic BP	>100mmHg	<100mmHg
Heart rate	>100bpm	<100bpm
Adjunctive tests		
Blood glucose concentration	4 – 10mmol/L	< 4 or >10mmol/L
Serum sodium concentration	135 – 148mmol/L	<135 or >148mmol/L
Pre- post race body weight change	0 – 5% loss	Loss or gain >10%

Once the primary survey has been completed, and the athlete is stable, a specific diagnosis should be made. The possible different causes of “collapse” in endurance athletes have already been listed in Table 2.3. A specific diagnosis of the cause of “collapse” is important, because it will determine the most appropriate treatment. Many of the conditions listed in Table 2.3. that can cause “collapse” in the endurance athlete have specific signs and symptoms which allow medical staff to establish a clear diagnosis and to then treat the condition according to recognised guidelines. However, the majority of athletes who present to the medical facility for “collapse” following exercise, present with a cluster of symptoms/clinical signs that do not fall into a readily recognised specific medical diagnosis. These athletes present with vague

symptoms, such as dizziness or the inability to stand unassisted, which is often associated with nausea and vomiting.

Various terms have been used in the literature to describe this symptom/sign complex, and the terms that are used appear to depend on the author's beliefs or understanding of the cause. This syndrome is frequently referred to as Exercise-Associated Collapse (EAC)^{13,80-85}. Other terms that have been used for this syndrome include postural hypotension^{15,86}, heat exhaustion¹⁵, exercise-related collapse¹⁷, post-exercise postural hypotension⁸⁷, neurocardiogenic syncope⁸⁸, or heat syncope¹⁵. The lack of consistency in the description of these "collapsed" athletes appears to arise from the fact that the definition that is used is based on the author's understanding or experience of the pathophysiology of the condition. For example, some authors feel that the terms heat exhaustion and heat syncope should no longer be used⁸⁹⁻⁹¹, whereas others still use these terms to describe this condition^{92,93}. In some studies, athletes presenting with a similar syndrome have also been classified as dehydrated^{28,94,95}.

In summary, it appears that the most common cause for "collapse" on completion of an endurance event is a symptom/sign complex that up to now is frequently referred to as "Exercise-Associated Collapse" (EAC)^{13,96-101}. However, in the next section (Section 2.4.), the case will be argued that the term "Exercise-Associated Postural Hypotension" (EAPH) is perhaps the preferred term to use for this symptom/sign complex, based on the proposed pathophysiology of this syndrome.

2. 4. Exercise-Associated Postural Hypotension

2.4.1. Introduction and definition of Exercise-Associated Postural Hypotension (EAPH)

As previously mentioned, until now, the term Exercise-Associated Collapse (EAC) is the most commonly used term to describe collapse following exercise. However, it should be noted that there is no universally accepted definition of EAC. Various definitions for EAC have been used in reviews and published studies. EAC has been defined by some authors as “requiring assistance during or after endurance activity for problems that are not orthopaedic or musculoskeletal, and does not include cardiac arrest, hypoglycaemia, asthma, anaphylaxis, or other readily identifiable medical condition ¹⁰²”, while others proposed that EAC can be considered in a matrix that categorises the collapsed athletes into mild, moderate and severe, based on their blood pressure, level of consciousness, rectal temperature and blood parameters ^{13;103;104}.

It is important to note that in none of these definitions the possibility of orthostatic or postural hypotension (defined as “a reduction of systolic blood pressure of at least 20mmHg or diastolic blood pressure of at least 10mmHg within 3 minutes of standing”) is considered. Postural hypotension is a physical sign and not a disease, and there is agreement that in order to use the term postural or orthostatic hypotension, the decrease in systolic blood pressure should be accompanied by appropriate symptoms. These symptoms

include light-headedness, blurring of vision and the inability to stand unassisted. It has been suggested that postural hypotension is important to consider as a diagnosis of the “collapsed” athlete, and that the diagnosis should be made on completion of the race ¹⁰⁵ using the specific clinical signs.

Therefore, recently, it has been suggested the term Exercise-Associated Postural Hypotension (EAPH) ^{17;106} should be used to describe this symptom/sign complex that is associated with the “collapsed” athlete. The advantage of using this term, as opposed to EAC, is that it indicates the cause of the collapse and can thus be considered as a diagnosis. The arguments for the use of this term will be highlighted in the section discussing the pathophysiology of EAPH (Section 2.4.4.).

For the purposes of this review and this dissertation, EAPH will be defined as post-exercise collapse accompanied by the symptoms/sign complex of “the inability to stand unassisted or a feeling of light-headedness with or without blurring of vision, nausea and/or vomiting following strenuous exercises, in the absence of any identifiable medical cause”. This definition can be criticised due to the absence of a measurable change in blood pressure from the supine to the upright position. This is a valid criticism. However, this definition was chosen for practical reasons, because it is very difficult, if not impossible, to measure the upright blood pressure in a collapsed athlete at the time of admission. The terms EAPH and “less severe” EAC could also be used interchangeably, as the symptom/sign complex in the collapsed athlete that defines these two terms is the same (see Table 2.5.).

2.4.3. Incidence of EAPH at ultra-endurance events

EAPH has repeatedly been noted as the most common presenting medical condition at endurance and ultra-endurance events. The precise incidence of EAPH is not known because the criteria to make the diagnosis of EAPH vary considerably. It is frequently stated that EAPH is the most common condition presenting to the medical care facility, without any supportive evidence¹⁰⁷⁻¹⁰⁹. In reports where the frequency of EAPH is documented, the frequency ranges between 27%-59% of all athletes treated in the medical facility^{110;111}.

In one study, the twelve-year experience of the medical tent at the Twin Cities marathon, was reported. In this study, 59% of the patients seen in the medical facility were diagnosed with EAC¹¹², based on a specific classification system¹¹³, and it was noted to be the most common medical condition that was encountered. In this study, 1.1% of the race entrants suffered from EAC¹¹⁴. However, it should be noted that in this study the severity of EAC was classified into mild, moderate and the diagnosis of severe EAC was based on body temperature, blood results and the nature of the symptoms. According to this classification, those athletes suffering from "mild" EAC would fulfil the diagnostic criteria for EAPH. In this study, the frequency of EAPH (less severe EAC) can then be calculated as 55% of the athletes treated in the medical facility^{13;115}.

As previously mentioned, the pathophysiology of the “collapsed” athlete remains contentious. It is therefore difficult to establish the precise incidence of EAPH. In many studies the diagnosis of dehydration is reported as 47%-52% of those athletes treated, but no diagnosis of EAC or the equivalent is mentioned^{28;116;117}. However, in these studies, the diagnostic criteria for “dehydration” were based on symptoms of dizziness, tachycardia, syncope, inability to urinate, and hypotension¹¹⁸. It could be argued that a percentage of these cases with “dehydration” could be classified as EAPH.

It therefore appears that EAPH is arguably the most common medical condition that ultra-endurance athletes suffer from, and it is frequently encountered in the medical facility at ultra-endurance events. It is for this reason, that EAPH is the focus of research presented in this dissertation. As mentioned, the pathophysiology of this condition is not well understood and is controversial. Hence, it requires discussion. Furthermore, differing views regarding the pathophysiology of EAPH have resulted in different clinical approaches to the treatment of this condition. Current treatment protocols for EAPH will therefore also be reviewed.

2.4.4. Pathophysiology of EAC in ultra-endurance athletes

The precise pathophysiology of collapse following exercise, without an obvious, identifiable, medical cause, remains a controversial issue and has been the topic of much debate in the medical literature. Based on a review of the literature the two most commonly proposed theories are collapse due to

dehydration leading to hyperthermia and circulatory collapse¹¹⁹⁻¹²⁴ and collapse due to exercise-associated postural hypotension (EAPH)^{17;125-129}. Before these hypotheses are discussed in detail, a brief review of the physiology related to the control of blood pressure is warranted.

Blood pressure is the product of cardiac output (CO) and total peripheral resistance (TPR). CO is dependent on heart rate (HR) and stroke volume (SV), whereas TPR is affected by dilation (decreasing resistance) or constriction (increasing resistance) of the vascular system. Finally, SV is determined by three factors 1) Preload - the amount of tension in the myocardial muscle before it begins to contract (influenced by the amount of blood returning to the heart), 2) Contractility - the power with which the heart contracts, and 3) Afterload - the pressure the heart must overcome to eject the blood from the left ventricle. Any changes in these factors will therefore increase or decrease the blood pressure.

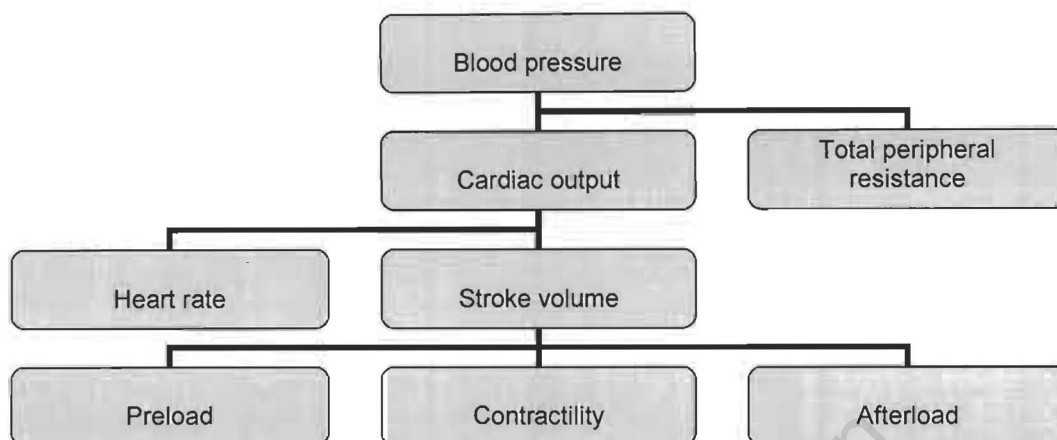


Figure 2.1.: Factors which play a role in the maintenance of blood pressure

For many years it was believed that it was dehydration which caused the collapse of athletes following an event. This is suggested by the fact that in many studies, dehydration is reported as the most common medical condition that requires treatment following an endurance event. Dehydration has been reported to range from 30%-52% of athletes reporting to the medical facility following the event¹³⁰⁻¹³². The hypothesis that dehydration starts a cycle which ultimately results in collapse gained popularity in the late 1960s and early 1970s following two studies which highlighted the dangers of dehydration. In these studies, dehydration was linked to the development of heat stroke. A study of athletes racing over 32km showed that those athletes who lost more than 3% of their body weight had a linear increase in their post-exercise rectal temperature. From these data, the authors concluded that during exercise, the most important determinant of rectal temperature was the level of hydration^{17;125}.

The hypothesis that athletes collapse after exercise as a result of heat exhaustion due to dehydration gained further support from a second study, which was published in 1972⁵⁸. In this study, it was suggested that thermoregulation during exercise relies on a cardiovascular response, and that dehydration impairs this response, resulting in hyperthermia and then circulatory collapse^{17;125}. This hypothesis of circulatory collapse due to dehydration induced hyperthermia has been presented in many studies and reviews^{120;133-137}.

However, the dehydration/hyperthermia leading to circulatory collapse hypothesis has also been challenged^{17;125;126;138-140}. The main reasons for challenging this hypothesis can be summarised as follows:

- The rectal temperature of the collapsed athlete is not significantly elevated in comparison to the non-collapsed runner^{17;28;141;142}
- Most collapsed athletes do not have clinical signs, and biochemical confirmation which suggest that they are more dehydrated than non-collapsed athletes^{17;143;144}
- Most (85%) of the athletes who collapse do so upon completion of the race^{17;145;146}, implying that physiological changes associated with the completion of exercise are causally related to the collapse
- The symptoms of athletes who collapse can be reversed if the athlete is placed in the Trendelenburg position^{17;147;148}.

Researchers have been prompted to re-examine the findings of a study performed in 1947, which first suggested the presence of postural hypotension following exercise¹⁴⁹. Further reviews of the time also highlighted the presence of postural hypotension following exercise,” temporarily, the movements themselves help in some degree to improve the return of blood to the heart. When movements stop, failure is imminent: some persons faint at this point. Lying down promptly relieves the circulation and the symptoms”¹⁵⁰.

On the basis of the above findings and a review of these articles, researchers started to explore the physiology of exercise and thermoregulation in greater detail. By gaining a better understanding of this physiology, researchers have gained better insight into the pathophysiology of Exercise-Associated Postural Hypotension.

During prolonged exercise, in particular in hot environmental conditions, heat is lost from the body in four ways: convection, conduction, evaporation and radiation. During exercise, evaporation becomes an important mechanism to lose heat and this is increased by sweating. The rate of sweating is increased by vasodilatation of the cutaneous venous system. The human body has developed compensatory measures to maintain venous return and hence right atrial pressure. These mechanisms include 1) the contraction of the muscles of the lower limbs which can act as a second heart by pumping blood back to the heart, and 2) one-way valves in the venous system to prevent back flow of blood and 3) a negative intra-thoracic pressure which helps to draw blood back to the heart.

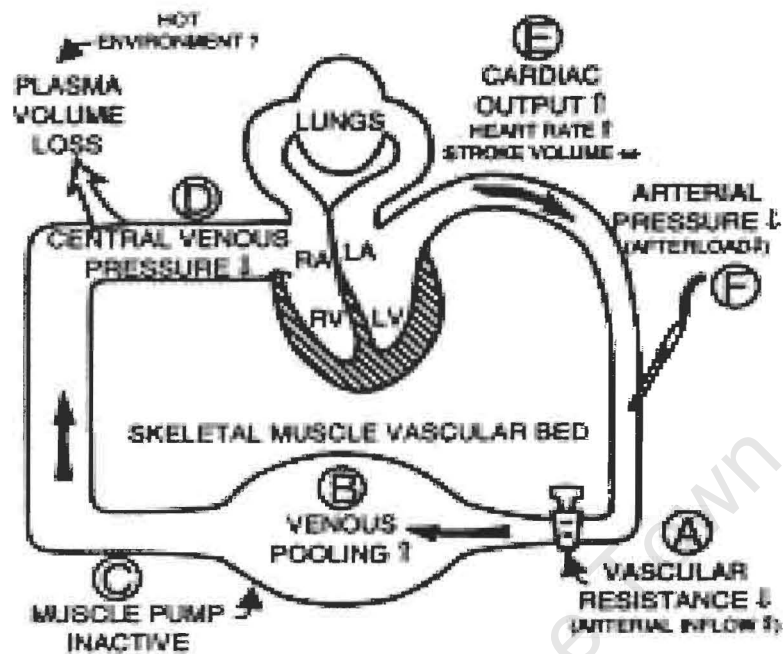


Figure 2.2.: The pathophysiology of Exercise-Associated Postural Hypotension (EAPH) ¹⁵¹

The pathophysiology of EAPH can be explained by considering the changes that occur immediately post-exercise (Figure 2.2.). During exercise, there is an increased metabolic rate resulting in an increased core temperature. The body attempts to decrease the temperature by vasodilation and sweating. Upon cessation of exercise the muscular pump system is removed, and this results in pooling of blood in the lower limbs. The resultant decrease in the venous return (D) may be exacerbated (marginally) by a decrease in plasma volume due to dehydration. Finally, although the cardiac output is increased by a slight increase in heart rate (E) the overall arterial pressure decreases (F), resulting in hypotension and post-exercise “collapse”.

Until recently, this hypothesis could not explain fully why a reflex tachycardia does not develop as would be expected. It has recently been suggested that the explanation for this may be as a result of the Barcroft/Edholm reflex¹⁵². In the original publication from 1944, it was proposed that when the right arterial pressure fell to a certain level or the pressure fell at a certain rate, the result was vasodilation of the venous system and bradycardia. This further decreases the hypotension. This was opposite to the expected response of tachycardia and vasoconstriction which should occur in order to increase the blood pressure. Another possibility for the lack of tachycardia could be neurocardiogenic syncope¹⁵³. In neurocardiogenic syncope, the possible explanation is that with the decreased venous return due to pooling of blood in the lower limbs, the forceful contraction of the ventricle against a decreased left ventricular end diastolic volume results in the stimulation of mechanoreceptors, which form the afferent limb of the vasovagal reflex. This input activates a reflex in the medulla which causes an efferent response of increased vagal tone. This in turn results in a bradycardia, due to the increased parasympathetic output¹⁵⁴.

Apart from the model presented in Figure 2.2., other physiological changes which may play a role in post-exercise postural hypotension have also been suggested^{155;156}. These include, 1) alterations in the sympathetic nervous system, 2) a vascular mechanism in response to the sympathetic nervous system, 3) the action of arginine vasopressin (AVP) on V1 receptors, and 4) the possible role of histamine (H1 and H2) receptors. The possible role of these four mechanisms will be briefly reviewed.

Venous constriction normally occurs due to stimulation of the alpha adrenergic receptors which are under sympathetic control. It has been shown in various studies that immediately post-exercise, there is a decrease in sympathetic nerve activity, resulting in increased vasodilation and reduced vascular resistance^{157),158;159}. However, the precise reason for the decrease in sympathetic activity post-exercise has not been fully explained.

A vascular mechanism, whereby there is a decrease in vascular response to the sympathetic activity, also has to be considered. The most likely site for decreased transmission of the sympathetic outflow appears to be arterial smooth muscle. This could occur either through down regulation of the receptors or via local vasodilator substances, such as nitric oxide. However, these possible mechanisms are not confirmed in humans by scientific evidence^{160,161;162}.

It has been suggested that AVP may contribute to the development of post-exercise hypotension by acting on V1 receptors¹⁶³. The two possible mechanisms by which AVP, which is raised in exercise, contributes to post-exercise hypotension have been identified. Firstly, AVP enhances both cardiopulmonary and arterial baroreflex function, as well as lowering the operation point of the arterial baroreflex, both of which have been shown to mediate post-exercise hypotension¹⁶⁴. Secondly, AVP may enhance arterial baroreflex-induced inhibition of the sympathetic nervous system, which, as

described earlier, results in a decreased ability to vasoconstriction and thus increased susceptibility to postural hypotension ¹⁶⁵.

Finally, recent studies have highlighted the possible role of the histamine receptors, H1 and H2, in post exercise postural hypotension. In one study, it has been shown that after the ingestion of Fexofenidine, a highly selective H1 antagonist, there is a decrease in post exercise postural hypotension ¹⁶⁶. In a second study ¹⁶⁷, administration of the anti-histamine antagonist, Ranitidine (in a dose which made it selective for H2 receptors) has also resulted in a decrease in post exercise postural hypotension. From the results of these two studies, it can be concluded that histamine may play a role in postural hypotension. Furthermore, it appears that H1 receptors play an important role in hypotension in the first 30min post exercise, while H2 receptors have been shown to play a role between 30-60 minutes post exercise.

In summary, there appears to be two primary pathophysiological mechanisms that may explain the development of post exercise collapse in athletes following endurance events. Firstly, there is the hypothesis that dehydration sets in motion a cycle that leads to hyperthermia and eventually circulatory collapse, resulting in the collapse of the athlete. The second hypothesis is postural hypotension due to the pooling of blood in the dilated venous system once the "second heart" (muscle system) is withdrawn on completion of exercise. Various other factors have been studied which contribute to the vasodilatation including, neural mechanism, vascular mechanism, histamine receptors, and the AVP.

2.4.5. Diagnosis of EAPH

The diagnosis of EAPH is essentially a diagnosis by exclusion. As previously noted (Section 2.3.), the priority in an athlete who collapses at the finish of an endurance race is to exclude life-threatening and other identifiable medical causes. Once these have been excluded, the diagnosis of EAPH should be considered. Although there are no specific tests to confirm the diagnosis of EAPH, there are “classic” signs and symptoms which should be obtained from the medical history and the clinical examination. Special investigations are also important to exclude other causes of collapse, and specific tests will be highlighted.

2.4.5.1. Medical history

One of the most important elements in the medical history is to confirm when the athlete collapsed in relation to the race. As previously discussed, an athlete can collapse on completion of a race with the associated “pooling” of blood that occurs when the “muscle pump” stops. Thus the stretcher bearers or assistants should be asked where the patient collapsed. In order to diagnose EAPH, there must be a history of collapse on completion of the race. It could be argued, however, that an athlete may collapse EAPH while in transition, or if the athlete has stopped on the route which has caused the “muscle pump” to cease. The amount of fluid the athlete ingested should also

be obtained, if possible. In reality, this is difficult as the medical staff rely on the athlete's recall which after 8-17 hours of exercise is not always reliable.

Finally, as previously mentioned, many other causes for "collapse" must be considered. Apart from previous medical history, allergies and medications, a summary of the important elements in the history that may assist in the diagnosis of these causes of collapse are presented in Table 2.6.

University of Cambridge

Table 2.6.: Medical history that may be relevant to exclude other causes of collapse in an athlete ¹⁶⁸

System	Condition	Relevant history
Cardiovascular	MI & Angina	Chest pain, including site, nature, radiation, relieving factors, associated symptoms: nausea and vomiting
	Dysrhythmia's	Palpitations and chest pain
Musculoskeletal	Cramps	Muscle cramps
	Stress or True fractures	Sudden onset of pain, Trauma
Respiratory	Asthma	Previous history of asthma, shortness of breath
Metabolic and fluid status	Hyponatremia	Very high fluid intake.
	Hypernatremia	Low fluid intake
	Dehydration	Low fluid intake
Temperature	Hypothermia	Cold water on swim leg, cold weather conditions
	Heatstroke	Hot weather conditions
Neurological	Cerebral Vascular Accident	Acute headache, acute paralysis
	Epilepsy	Previous history of epilepsy
	Intracranial bleeds	Acute headache

2.4.5.2. Clinical examination

The clinical examination of an athlete with EAPH is generally characterised by no specific signs that are diagnostic of this condition. The level of consciousness is an important sign to assess. In an athlete with an altered level of consciousness, a serious cause of collapse should be considered, as this does not occur in athletes with EAPH.

The vital signs should be obtained in the triage area during the “primary survey”. The important vital signs to consider are the temperature, systolic blood pressure and heart rate. As previously discussed (Table 2.5.), the vital signs are used to classify the collapsed athlete as “severe” or “less severe”. The vital signs in an athlete with EAPH will be a core temperature of less than 40°C, a heart rate of less than 100bpm, and a systolic BP of greater than 100mmHg. It should, however, be noted that the cut-off values for heart rate and systolic blood pressure appear to be based on reasonable clinical deductions, but that there is no strong scientific evidence to support the use of these values as specific cut-off values. Furthermore, as previously discussed, the measurement of a decrease in systolic blood pressure (>20mmHg) or a diastolic blood pressure (>10mmHg) from the supine to the upright position is normally considered as diagnostic for postural hypotension. However, in the context of the clinical assessment of the collapsed athlete in the medical facility, it is very difficult to obtain the blood pressure measurement in the upright position in a collapsed athlete. A change in blood pressure from the supine to the upright position has therefore not been included in the

diagnostic criteria that separate “severe” from “less severe” collapsed athletes (see Table 2.5.)¹⁶⁹. Therefore, these diagnostic criteria are not considered essential criteria for the diagnosis of EAPH.

As previously mentioned, many other causes for “collapse” must be considered. A summary of the important elements in the clinical examination that may assist in the diagnosis of these causes of collapse are presented in Table 2.7.

University of Cape Town

Table 2.7.: Clinical examination and relevant examination findings to exclude other causes of collapse in an athlete ¹⁶⁸

System	Condition	Relevant examination findings
Cardiovascular	MI & Angina	Tachycardia, Irregular blood pressures, Murmur, 3 rd /4 th Heart sound.
	Dysrhythmia's	Irregular pulse, Bradycardia, Irregular blood pressure.
Musculoskeletal	Cramps	Palpation of acute spasm
	Stress or True Fractures	Acute pain on palpation.
Respiratory	Asthma	Tachypnoea, Wheezing Severe: Inability to complete sentence, HR >110bpm, Respiratory rate >25/min Life-Threatening: Silent chest, Cyanosis, Bradycardia.
Metabolic and fluid status	Hyponatremia	Confusion, seizure, Oedema
	Hypernatremia	(See dehydration)
	Dehydration	Dry mucous membranes, inability to spit, loss of skin turgor, sunken eyeballs, loss of 10% body weight ⁽¹⁷⁰⁾
Temperature	Hypothermia	Rectal temperature <35°C
	Heatstroke	Rectal temperature >41°C
Neurological	Cerebral Vascular Accident	Confusion, Acute paralysis
	Epilepsy	Confusion, Seizure
	Intracranial bleeds	Confusion, Acute paralysis

2.4.5.3. Special investigations

In a collapsed athlete, special investigations are required to rule out other medical conditions that could mimic EAPH. The standard investigations that should be performed are serum sodium concentration to rule out hyponatremia, and plasma glucose concentration to exclude hypoglycaemia¹⁷¹⁻¹⁷⁴. Although the incidence of hyponatremia is low, this is a potentially fatal condition if not recognised and appropriately treated.

A summary of the important special investigations that may assist in the diagnosis of these causes of collapse are presented in Table 2.8.

University of Cape Town

Table 2.8.: Clinical examination and relevant special investigation findings to exclude other causes of collapse in an athlete ¹⁶⁸

System	Condition	Relevant special investigations
Cardiovascular	MI & Angina	ECG, cardiac markers
	Dysrhythmia's	ECG
Musculoskeletal	Cramps	No special investigations required
	Stress or True Fractures	X-ray (at hospital)
Respiratory	Asthma	FEV1 if possible, Blood gas
Metabolic and fluid status	Hyponatremia	Serum sodium concentration <135mmol/l
	Hypernatremia	Serum sodium concentration >148mmol/l
	Dehydration	Weight loss >10%
Temperature	Hypothermia	Rectal temperature <34°C
	Heatstroke	Rectal temperature >41°C
Neurological	Cerebral Vascular Accident	CT (at hospital)
	Epilepsy	No acute tests required
	Intracranial bleeds	CT (at Hospital)

2.4.6. Management of EAPH

2.4.6.1. Introduction

In the published literature, there are predominantly two, quite differing approaches to the management of EAPH. These management protocols are

based on the two main hypotheses that have been discussed as possible pathophysiological mechanisms of collapse following exercise. As a result, there are predominately two treatment protocols for EAPH that are used in race medical tents. These two management approaches are 1) intravenous (IV) rehydration^{120;175-178}, and 2) placing athletes in Trendelenburg position with oral fluids given ad libitum^{2;14;23;24;31;32}. There is currently an ongoing debate on how best to treat athletes who collapse at the finish of a race and both treatment protocols are currently employed in medical facilities at endurance events in South Africa and the rest of the world.

2.4.6.2. Intravenous fluid administration

In general, medical staff that support the dehydration hypothesis for collapse following exercise, treat these athletes with IV fluid^{18;43}. Although there is a lack of clinical evidence to support IV fluids, the rationale to this treatment seems to be to acutely restore plasma (dehydration) volume⁴⁴ and therefore increase venous return. This “blanket” approach of IV fluid in collapsed athletes may, however, stem from an understanding that EAPH does not occur but that dehydration is the cause of collapse⁴⁴.

The most common IV fluids that are used include normal saline or normal saline with 5% dextrose^{18;27;44}. In one study, the number of IVs used in the Hawaii Ironman Triathlons between 1982 and 1988 was reported. The result of this study showed that the percentage of starters who received IV fluid increased from 3% in 1982 to 15% in 1988. On average, each athlete

received 1.4L of IV fluid. In the Boston Marathon, 9.3%-12.2 % of the athletes treated in the medical facility between 1984 and 1987 received IV fluids ⁵.

IV administration is not without risks and cost implications. Although the risks of IV treatment are generally low, they can include infection, embolism, nerve damage, disease transmission and bleeding ⁴⁴. The costs involved in IV fluid administration may be as high as \$10 000 at an Ironman Triathlon ¹³.

There are certain cases where IV fluid is indicated in a collapsed athlete. These include, 1) a clinically dehydrated athlete, particularly if this is accompanied by severe nausea and vomiting, where the athlete is unable to take fluid orally, 2) an unconscious patient with a serum sodium concentration >130mmol/l, 3) appropriately diagnosed heat stroke, 3) hypoglycaemia, 4) persistent hypotension and 5) tachycardia despite being in the Trendelenburg position for >15minutes ^{13;44;72}.

2.4.6.3. Oral fluid administration

In general, medical staff who support the hypothesis that the collapse is mainly as a result of postural hypotension due to vasodilatation and pooling of blood in the lower limbs on completion of exercise, treat these athletes by elevating their legs and hips (Trendelenburg position), while allowing oral fluids ad libitum ^{2;8;13;16}. The rationale for this treatment is that initially this position will increase the venous return and restore cerebral blood flow. Within a short time (usually less than 60 minutes), vasodilation will decrease and

venous return will increase. A small degree of dehydration (<3% decrease from pre-race body weight) can be corrected by allowing oral fluids. In a recent laboratory-based study, no benefit following IV rehydration was found in athletes with 4% dehydration after exercise when compared to oral rehydration⁴⁵. In fact, the results of this study showed an improved physiological response to a subsequent exercise bout following oral as opposed to IV rehydration⁴⁵.

2.4.6.4. Summary: Management of EAPH

The choice of which management protocol medical staff in medical facilities use to treat EAPH appears to be inconsistent and is influenced by various factors. It appears that the most important factor is the understanding and belief of the pathophysiology of collapse following exercise by the treating physician. Other factors influencing the choice of treatment may include 1) the medical team's experience from previous events and the success or failure of such protocols, 2) the opinions of senior medical staff in guiding the junior staff and 3) in some cases, anecdotal evidence has shown that the medical staff have highlighted time constraints as an important factor. In the case of the speed of recovery following collapse and the use of oral versus IV fluids, there is a perception that athletes with collapse will recover, and are discharged quicker after being given IV fluids when compared with treatment in the Trendelenburg position and oral ad libitum fluids. However, there is no scientific research that has investigated these two treatment protocols for EAPH in a systematic manner. In particular, there are no randomised clinical

trials that have compared these treatment protocols in athletes suffering from EAPH.

2.5. Summary

- Endurance events can result in a variety of medical conditions during and post race
- One of the most common presenting medical complaints is the “collapsed” athlete
- There are many causes for the “collapsed” athlete, the most common being EAPH
- A sound medical treatment approach towards the “collapsed” athlete is essential to the medical team working at endurance events
- The exact pathophysiology of collapse post-race is currently debated with the two most common hypotheses being:
 - Dehydration, leading to hyperthermia and circulatory collapse
 - Postural hypotension due to pooling of blood in the lower limbs when the “muscle pump” is withdrawn on completion of exercise
- The treatment of the “collapsed” athlete with EAPH appears to be based on the medical staff’s understanding of the pathophysiology
- The two most common management approaches to the “collapsed” athlete with EAPH are:
 - Intravenous (IV) fluids
 - Trendelenburg position with oral fluids

- Currently, there no studies where clinical outcomes and recovery time using these two management approaches have been measured and compared.

University of Cape Town

Chapter 3

A comparison of two treatment protocols in the management of Exercise-Associated Postural Hypotension (EAPH): A randomised clinical trial

3.1. Introduction

The Comrades Marathon (CM) and the Ironman Triathlon (IMT) are both popular ultra-endurance events that are held annually in South Africa. The Comrades marathon is an 89-kilometre running race that is held between the cities of Durban and Pietermaritzburg in the KwaZulu-Natal province of South Africa. The Ironman Triathlon is an ultra-triathlon that is held in the city of Port Elizabeth, which is in the Eastern Cape province of South Africa. An Ironman Triathlon consists of a 3,8km sea swim, followed by a 180km road cycle and finally a 42,2km run.

Athletes who participate in these events are at risk of developing various medical conditions, due to the strenuous nature (physical and mental) of these ultra-endurance events¹⁻⁷. One of the most common clinical presentations during and following an endurance athletic event is that of the “collapsed” athlete. There are various causes that can lead to collapse in

athletes, including cardiovascular, metabolic, neurological, musculoskeletal and respiratory causes ¹⁻⁷.

The most common cause for collapse is a benign condition referred to as Exercise-Associated Postural Hypotension (EAPH) ^{3;27}. The exact pathophysiology of this condition is still widely debated in the literature, and currently there are two hypotheses that have been put forward as causes of this condition. The first hypothesis is that a combination of dehydration and hyperthermia causes circulatory failure, which results in the collapse of the athlete ^{3;5;20;28-30}. The second hypothesis is that pooling of blood occurs in the dilated venous system once the “second heart” (“muscle pump” system) is withdrawn on completion of exercise. This results in postural hypotension ^{2;14;23;24;31;32}. The possible mechanisms that cause vasodilation include, neural mechanisms, vascular mechanisms, histamine receptors, and the arginine vasopressin (AVP) ^{22;37-39;41;46}.

As a result of these two hypotheses to explain the pathophysiology of EAPH in athletes, there are predominantly two approaches to the management of EAPH that are used in race medical facilities. These two interventions are 1) intravenous (IV) rehydration ^{5;18;20}, and 2) placing athletes in the Trendelenburg position with oral fluids given ad libitum ^{2;3;6; 8;13;44}. There is, however, an ongoing debate on how best to treat athletes who suffer from EAPH.

There appears to be a perception that athletes with EAPH recover quicker and are discharged earlier if treated with IV fluids, when compared with treating the athlete in the Trendelenburg position and administering oral ad libitum fluids. However, there is no scientific research that has investigated these two treatment protocols for EAPH in a systematic manner. In particular, there are no randomised clinical trials that have compared these two treatment protocols in athletes suffering from EAPH.

Therefore the aim of this study was to compare which of the two commonly used treatment protocols for Exercise-Associated Postural Hypotension (EAPH) (Trendelenburg with oral fluids ad libitum or intravenous fluids) result in quicker recovery and earlier discharge from the medical facility.

3.2. Methods

3.2.1. Type of study

The study was conducted as a randomised clinical field trial.

3.2.2. Subjects

Subjects were recruited for this study from three ultra-endurance events over a two-year period. From these events, all the athletes who collapsed due to Exercise-Associated Postural Hypotension (EAPH) at the South African Ironman Triathlon 2006 (3.8km swim, 180km road cycle, 42.2km run), the South African Ironman Triathlon 2007 and the 2006 Comrades Marathon

(89km road running race), were potential subjects for this clinical field trial.

The methods used to recruit subjects at these three events were similar, and generally consisted of the following: 1) pre-race athlete information, 2) setting up research facilities in the medical tents after the races, which were integrated with the normal medical care facilities, 3) informing all medical staff in the medical tents before the race of the nature of the study and the inclusion criteria for subjects, 4) screening athletes in the triage area, and identifying athletes with EAPH using defined inclusion criteria as potential subjects, and 5) obtaining informed consent from athletes with EAPH.

More specifically, at the South African Ironman Triathlon 2006, information about the study was posted on the race website prior to the event. Informed consent was obtained at registration, which was done in the three days before the race. At the Comrades Marathon 2006 and South African Ironman Triathlon 2007, information about the clinical trial was available at registration, and informed consent was obtained from athletes who agreed to participate in the trial.

Furthermore, recruitment of the athletes took place on admission to the medical tent. This was done with the agreement of the race medical directors of all the events. As both treatment protocols are current medically acceptable protocols for the management of this condition, the race medical directors were satisfied that adequate medical care would still be provided by medical staff, irrespective of the protocol used. All the athletes who collapsed at the South African Ironman Triathlon 2006 and the Comrades Marathon 2006 were

initially brought to the medical tent where they were assessed by members of the race medical team in a triage area. Initially all athletes were placed in the supine position and a baseline assessment of the athlete was completed. If possible a post race body weight was obtained. This assessment was designed to identify any patient who may be classified as severe and who may require urgent medical treatment. Once the athlete was stabilised, a diagnosis of EAPH was confirmed using specific inclusion criteria (Table 3.1.).

Table 3.1.: Inclusion criteria for the diagnosis of EAPH (based on criteria depicted in Table 2.5.)

Clinical criteria:

Collapse # occurred after finishing the race (not during the race)

Normal level of consciousness and oriented *

Systolic blood pressure >100mmHg

Heart rate <100 bpm

Body temperature <40°C

Special investigations:

Serum sodium concentration between 135-148mmol/L

Blood glucose concentration >4mmol/L

#: Collapse was defined as the inability to stand or walk unaided as a result of light-headedness, faintness, dizziness or syncope.

*: The level of consciousness and orientation was assessed based on the following questions. (1) what is your name; (2) how old are you; (3) what is the date; (4) what race have you just completed; and (5) where are you?

Informed consent was obtained. The process of obtaining informed consent did not result in a delay of the treatment of these athletes. Consent was confirmed again on discharge. Prior to the study, the protocol was approved by the Research Ethics Committee of the Faculty of Health Sciences at the University of Cape Town (Ref numbers 425/2005, 185/2005 and 002/2007).

Once athletes were included in the study and informed consent was obtained, members of the medical research team continued the management of these athletes. Thirty-two athletes (26 males and 6 females) were initially included in the trial from the South African Ironman Triathlon 2006 (N=6), South African Ironman Triathlon 2007 (N=16), and the Comrades Marathon 2006 (N=10) respectively.

3.2.3. Experimental procedure

All 32 athletes underwent baseline medical assessments consisting of the following: level of consciousness was assessed, resting heart rate and supine resting blood pressure. A venous blood sample was obtained from the antecubital veins, the blood glucose concentration and serum sodium and chloride concentrations were determined.

All 32 athletes who met the inclusion criteria and who agreed to be part of the trial were then randomly assigned to one of two treatment protocols based on the last digit of their race number. Athletes with an odd race number were assigned to the Trendelenburg and oral fluids protocol, while athletes with an even race number were assigned to the intravenous (IV) fluid protocol.

The details of treatment in each protocol are as follows. The Trendelenburg and oral fluids treatment consisted of the following: (1) insertion of a venflon with the infusion 50ml normal saline over 1 hour; (2) measurement of serum

sodium concentrations; (3) raising the athlete's legs and buttocks to 30°; and (4) advising the athlete that they may drink as much as they like. The IV treatment protocol consisted of the following: (1) insertion of a venflon with the infusion of 1000ml normal saline over 30min; (2) measure serum sodium concentrations; (3) the athletes remained in the supine position; and (4) athletes were advised that they may not drink during treatment.

The initial serum $[Na^+]$ result was obtained, the study continued if the result was between 135-148mmol/l. Reassessments in both groups (level of consciousness, heart rate, blood pressure (supine) were conducted every 15min until discharge. If an athlete's condition changed in any way, a repeat serum sodium concentration was taken. Athletes were removed from the study if their serum $[Na^+]$ dropped below 135mmol/l. The treatment of the athletes who were removed from the trial was taken over by the staff in the race medical tent. The reason for transfer from the trial was documented.

At each 15min interval, athletes were assessed for their eligibility for discharge from the medical facility. The criteria for discharge were: (1) no altered mental state and (2) the athlete was able to stand and walk unassisted for 5m. The time of discharge was recorded, and this was the main measure of outcome. If an athlete requested discharge, they were counselled and if they insisted, this was noted and they were discharged.

Four athletes were removed from the trial: one due to hypoglycemia, one became confused and developed hyponatremia after 15 minutes while on the

IV drip, one of the patients was randomised to the oral trial but requested a drip after thirty minutes, and one did not respond to 1000ml of IV fluids after thirty minutes. Of the final 28 athletes who completed the trial, 14 athletes were randomised into the oral trial group (Trendelenburg and oral fluids) (Appendix 1); 2 athletes at the Comrades Marathon, 4 athletes from the South African Ironman Triathlon 2006 and 8 athletes from the South African Ironman Triathlon 2007; and 14 athletes were randomised into IV trial group (Intravenous fluid) (Appendix 2); 6 at the Comrades Marathon, 1 at the South African Ironman Triathlon 2006, and 7 at the South African Ironman Triathlon 2007.

3.2.4. Measures of outcome

The main outcome measure for this study was time to discharge from the medical tent. Additional measures of outcome were heart rate and blood pressure changes over time.

3.2.5. Statistical analysis of the data

Data were analyzed with the STATISTICA version 7.0 (StatSoft Inc., Tulsa, OK, USA) statistical program. Any significant differences between the protocol groups were determined by a one-way analysis of variance (ANOVA) or, chi-square (χ^2) analysis. When the overall F-value was significant, a Tukey's honest significance *post hoc* test was used to determine specific differences. Statistical significance was accepted when $P < 0.05$.

3.3. Results

3.3.1. Subjects

The average age of the athletes in the oral protocol group was 37.7 ± 7.2 years (N=14) with the average of the IV protocol group being 36.5 ± 7.1 years (N=14). There was no significant difference between the ages of the two groups (P=0.66). There were also no significant differences in the pre-race weights (Oral: 73.6 ± 8.1 kg, (N=7) vs. IV: 76.0 ± 6.2 kg, (N=5), P=0.59) or gender distributions (Oral: 78.6% males vs. IV: 92.9% males, P=0.60) between the two groups.

3.3.2. Weather conditions

The temperature, humidity and wind speeds for the three events are summarised in Table 3.2. As can be seen in the Table, the weather conditions for these three events were similar.

Table 3.2.: Weather conditions for the ultra-endurance events at which the trial was performed

	Ironman 2006	Ironman 2007	Comrades Marathon 2006
Mean Temperature (°C)	20	18	17
Max Temperature (°C)	21	21	22
Min Temperature (°C)	19	14	12
Average Humidity (%)	75	56	75
Maximum Humidity (%)	88	94	94
Minimum Humidity (%)	55	32	47
Wind Speed (km/h)	28	17	4

3.3.3. Admission to medical facility

Table 3.3. shows a comparison of the vital signs and blood results of the athletes in the two protocol groups on admission to the medical tent following EAPH. There were no significant differences between any of the initial parameters between the two groups on admission.

Table 3.3.: Vital signs and blood results of athletes on admission

	Oral Group (N = 14)	IV Group (N = 14)	P Value
Systolic BP (mmHg)	110 ± 11 (91 - 130)	106 ± 8 (89 - 115)	0.21
Diastolic BP (mmHg)	68 ± 9 (48 - 80)	67 ± 10 (48 - 80)	0.86
Heart rate (bpm)	81 ± 10 (60 - 99)	90 ± 14 (42 - 102)	0.79
Serum [Na⁺] (mmol/l)	139 ± 3 (135 - 147)	141 ± 5 (136 - 150)	0.18
Plasma [Glucose] (mmol/l)	5.8 ± 0.9 (4.6 - 8.0)	5.8 ± 1.2 (4.5 - 8.9)	0.92
Axillary Temperature (°C) ¹	36.1 ± 0.9 (34.8 - 37.5)	35.9 ± 0.5 (35.1 - 37.5)	0.62
Pre-post Race Body Weight Changes (%) ²	-3.5 ± 3.0 (-6.6 - 1.7)	-2.2 ± 4.9 (-4.9 - 5.1)	0.62

Values are expressed as averages ± standard deviations with the range in parenthesis.

¹Temperature was only documented in 14 athletes (6 in the oral and 8 in the IV groups).

² Relative weight change was only documented in 10 athletes (6 in the oral and 4 in the IV groups).

There were no significant differences in the absolute (data not shown) and relative pre-post race body weight changes (Table 3.3.) of the athletes between the two groups. Weight changes ranged from a weight loss of 4.9 kg to a weight gain of 3.4 kg in these athletes.

Table 3.4. documents the vital signs for the 28 athletes during their recovery in the medical tent. The number of athletes remaining for every 15 minutes is represented in brackets, thus with time the numbers decrease gradually.

There were no significant differences between the groups or over time in their supine systolic and diastolic blood pressure and heart rate measurements.

Table 3.4.: Vital signs of the athletes during recovery

	Admission	15 minutes	30 minutes	45 minutes	60 minutes	75 minutes	90 minutes
<i>Supine systolic BP (mmHg)</i>							
Oral	110 ± 11 (14) (91 - 130)	111±12 (13) (93 - 130)	109 ± 9 (11) (92 - 121)	105 ± 10 (7) (93 - 120)	110 ± 7 (5) (102 - 120)	107 ± 10 (3) (95 - 115)	123 ± 0 (1) (123 - 123)
IV	106 ± 8 (14) (89 - 115)	104±11 (14) (80 - 120)	107 ± 7 (14) (96 - 121)	105 ± 7 (9) (88 - 110)	104 ± 21 (4) (78 - 129)	97 ± 4 (3) (93 - 101)	120 ± 18 (2) (107 - 132)
<i>Supine diastolic BP (mmHg)</i>							
Oral	68 ± 9 (14) (48 - 80)	69 ± 9 (13) (52 - 85)	66 ± 6 (11) (59 - 80)	64 ± 8 (7) (50 - 73)	68 ± 10 (5) (58 - 82)	65 ± 10 (3) (53 - 71)	78 ± 0 (1) (78 - 78)
IV	67 ± 10 (14) (48 - 80)	64 ± 9 (14) (48 - 80)	68 ± 8 (14) (52 - 80)	68 ± 8 (9) (52 - 83)	65 ± 12 (4) (50 - 78)	63 ± 6 (3) (57 - 69)	65 ± 4 (2) (62 - 67)
<i>Heart rate (bpm)</i>							
Oral	81 ± 10 (14) (60 - 99)	75 ± 10 (13) (62 - 98)	74 ± 11 (11) (62 - 93)	72 ± 10 (7) (55 - 86)	69 ± 12 (5) (55 - 85)	74 ± 6 (3) (70 - 81)	76 ± 0 (1) (76 - 76)
IV	90 ± 14 (14) (42 - 102)	79 ± 13 (14) (63 - 110)	74 ± 9 (14) (60 - 92)	72 ± 7 (9) (65 - 88)	68 ± 3 (4) (65 - 70)	69 ± 6 (3) (65 - 75)	74 ± 8 (2) (68 - 79)

Values are expressed as averages ± standard deviations with the number of subjects (N) and range in parenthesis.

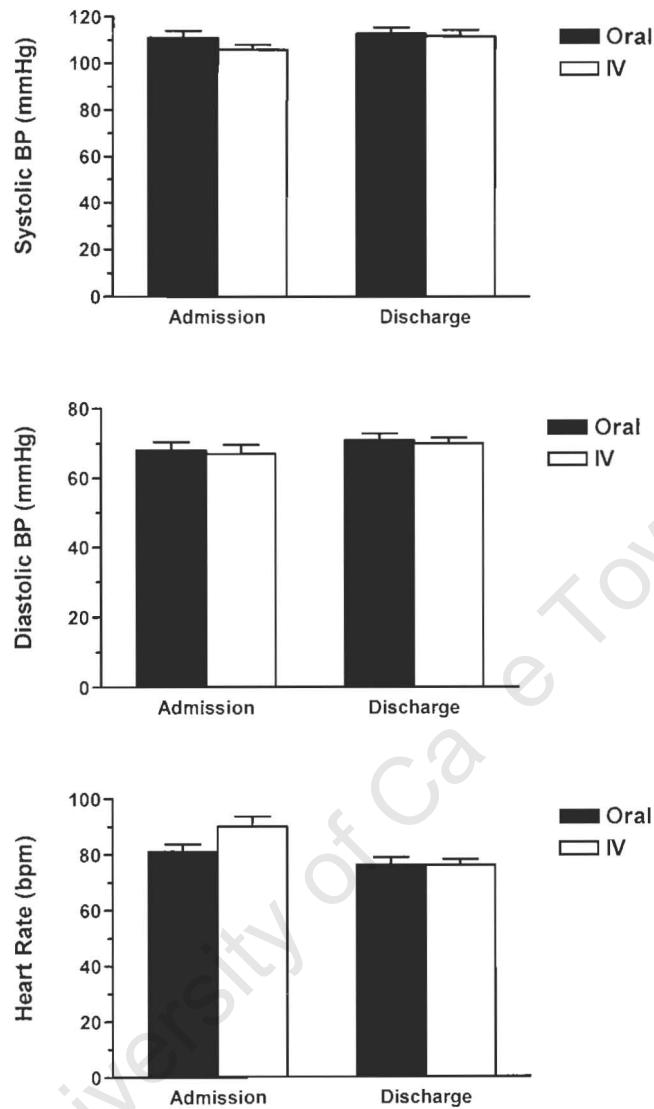


Figure 3.1.: Comparison of supine systolic BP (top panel), diastolic BP (middle panel), and heart rate on admission and discharge between the two groups

Figure 3.1. depicts the admission and final blood pressure and heart rate of the athletes in the oral and IV protocol groups respectively. The discharge values are the last recorded vital signs before discharge and are therefore not recorded at the same time for each athlete. There were no significant

differences in the changes of systolic blood pressure (top panel), diastolic blood pressure (middle panel) and heart rate (bottom panel) from admission to discharge between the two groups.

The amount of fluid received by the subjects in each group during the trial is depicted in Figure 3.2. The total volume of fluid (ml) received by the oral group 204 ± 149 ml (N = 11) was significantly less than that received by the IV group 1045 ± 185 ml (N = 12) ($P < 0.001$).

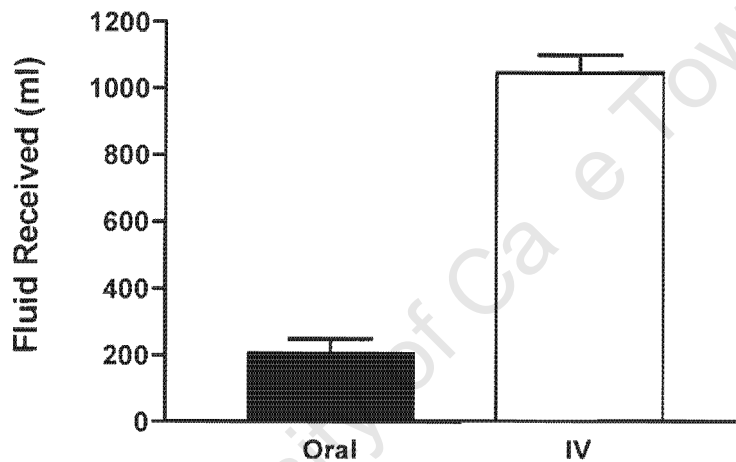


Figure 3.2.: Volume fluid the oral treatment group drank versus the volume intravenous fluid the IV treatment group received while in the medical tent

Finally, Figure 3.2. shows the comparison of time to discharge for the two treatment groups. There was no significant difference in the time to discharge between the athletes who received IV fluids (IV group = 52.5 ± 18 min) versus those who were treated in the Trendelenburg position and given oral fluids ad libitum (Oral group = 58 ± 23 min) ($P = 0.47$).

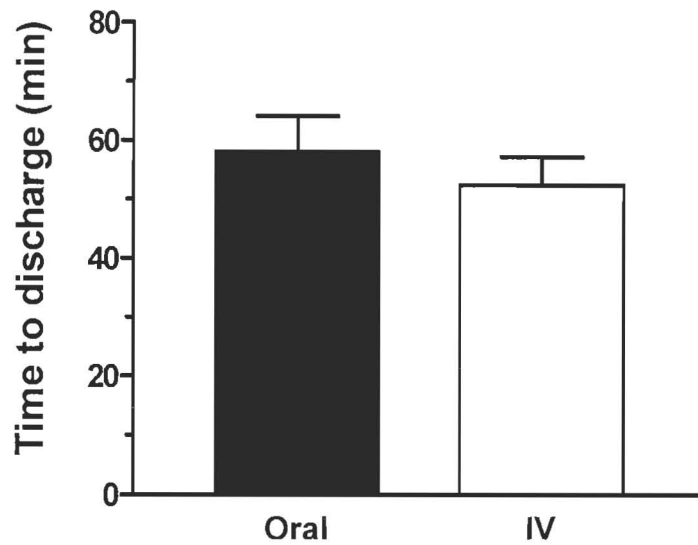


Figure 3.3.: Time to discharge, Oral versus IV treatment groups

3.4. Discussion

The main finding of this study was that there was no significant difference in time to discharge of athletes with Exercise-Associated Postural Hypotension (EAPH) when treated with either intravenous fluids or being placed in the Trendelenburg position and given oral fluids ad libitum. To our knowledge, although the protocol of Trendelenburg and oral fluid is commonly used by medical directors and medical staff to treat athletes with EAPH^{2;4;8;44}, no studies have been done to compare the time to discharge when compared to intravenous fluids.

It is clear that currently the most widely believed hypothesis for the pathophysiology of EAPH is that there is venous pooling of blood following exercise when the “muscle pump” (also referred to as the “second heart”) is removed on completion of exercise^{2;6;8;13;14;16;22;23;31;44}. The alternative

hypothesis for the pathophysiology of EAPH is dehydration, which leads to hyperthermia and circulatory collapse^{3;5;20;28-30;43}.

In the literature, the main findings that support the hypothesis that venous pooling is the cause of EAPH can be summarised as follows:

- The rectal temperature of the collapsed athlete is not significantly elevated in comparison to the non-collapsed runner^{2;8;14;17}
- Most collapsed athletes do not have clinical signs and biochemical confirmation which suggest that they are more dehydrated than non-collapsed athletes^{2;8;14}
- Most (85%) of the athletes who collapse do so upon completing the race^{2;8;14} - implying that physiological changes associated with the completion of exercise is causally related to the collapse
- The symptoms of an athlete who collapses can be reversed if the athlete is placed in the Trendelenburg position^{2;8;14}

Based on the fact that this study showed no difference in the time to recovery between the two protocols, these findings suggest that rapid restoration of plasma volume by IV fluids is not superior to increasing venous return by treating peripheral venous pooling (raising the limbs in the Trendelenburg position). In this trial, athletes who received intravenous fluids were given on average five times as much fluid during their recovery period, yet did not recover quicker than the oral group. The result of this study therefore shows that athletes with EAPH do not require intravenous fluid rehydration, but will respond as rapidly to treatment consisting of minimal oral fluid and raising the

lower limbs (Trendelenburg position) so that venous return is increased.

Athletes with EAPH can adequately replace any fluid orally, provided they do not have any gastrointestinal disturbance, or cannot consume fluid for any other reason.

The results of this study therefore support the hypothesis that EAPH is due to pooling of blood in the lower limbs on completion of exercise rather than dehydration.

The exact reason why some athletes are more predisposed to vasodilatation, which is not matched by an increased cardiac output, is currently the subject of ongoing research. Various theories are currently under investigation.

Firstly, immediately on cessation of exercise, there is a decrease in sympathetic nerve activity (SNA)^{39,46}. However, the exact reason for the decrease in sympathetic activity post exercise has not been fully explained.

As sympathetic stimulation of the alpha adrenergic receptors results in venous constriction, this decrease in (SNA) will result in prolonged vasodilation of the venous system.

In addition, with a decrease in SNA after exercise, it is also possible that smooth muscles have a decreased response to the sympathetic nervous system. Possible mechanisms for this can include down regulation of the receptors or via local vasodilator substances, such as nitric oxide³⁹. However, these possible mechanisms have not been confirmed in humans^{22;35;39}.

A second possible mechanism for the venous pooling relates to arterial baroreflex function. The arterial baroreflex is a negative feedback reflex that regulates arterial pressure around a preset value called a set or operating point. The arterial baroreflex also establishes the prevailing systemic arterial pressure by setting the operating point⁴⁰. On this basis arginine vasopressin (AVP), via its action on V1 receptors³⁷, may contribute to the development of post exercise hypotension. AVP enhances both cardiopulmonary and arterial baroreflex function, as well as lowering the operation point of the arterial baroreflex⁴⁰. AVP may enhance arterial baroreflex-induced inhibition of the sympathetic nervous system, which, as described earlier, results in a decreased ability to vasoconstriction and thus increased susceptibility to postural hypotension³⁷.

Finally, in two recent studies the possible role of the histamine receptors, H1 and H2, in post exercise hypotension has been suggested. In both studies the ingestion of anti-histamines prior to exercise decreased post exercise postural hypotension^{38;41}. From the results of these two studies, it was concluded that histamine, via its stimulation of H1 and H2 receptors, may play a role in postural hypotension. The results of these studies also suggested that H1 receptors may cause hypotension in the first 30 minutes post exercise, while H2 receptors have been shown to cause hypotension between 30-60 minutes post exercise.

The diagnostic criteria for EAPH that were used in this study require some discussion. The criteria used for this study have been documented in the methodology. The two most important criteria when considering the athletes for the trial were 1) a normal level of consciousness, and 2) collapse that occurred after finishing the race. It has been well documented that athletes who are confused or collapse during the race are more likely to suffer from other serious medical conditions^{2;4;8;13;15;16;23}.

The other major criteria for excluding subjects from this study were a body temperature of $>40^{\circ}\text{C}$ (heat stroke), a glucose below 4mmol/l (hypoglycaemia) and a serum sodium concentration below 135mmol/l or greater than 148mmol/L (dysnatraemia). The heart rate $>100\text{bpm}$, and blood pressure $<100\text{mmHg}$ were considered minor exclusion criteria. Although these clinical signs may be present in an athlete with severe collapse, there is no evidence to show that these specific cut-off points distinguish more serious pathology from less serious pathology. In this study, the term EAPH was chosen in place of "less severe" EAC, because it related to the possible mechanism of the symptoms/sign complex. As previously mentioned, the result of this study supports the hypothesis that these athletes have postural hypotension rather than dehydration, and respond to the raising of the lower limbs and moderate amounts of oral fluid ingestion. Furthermore, pre-post body weight changes (as a measure of dehydration) were $<5\%$ and were not significant between the groups at the finish, indicating that both groups were not severely dehydrated.

Unfortunately, due to logistical reasons, data on pre- and post race body weight could not be collected from all the subjects. This is a limitation of this study. Pre- and post race body weights were obtained for ten athletes (Oral N = 6; IV N = 4). One athlete in each group had an increase in body weight after the race (Oral: 1.7% and IV 5.1%). The rest of the athletes lost weight during the race. The greatest loss was 5.2% of pre-race weight, and this athlete, who was randomised to the oral protocol, was discharged after thirty-five minutes.

A further limitation of this study was that it was not possible to document the pre-race blood pressure and heart rates. However, these vital signs, which were measured on admission to the medical tent, show that these athletes generally, had a low blood pressure, and this is in keeping with findings reported from previous studies that have documented post exercise hypotension^{2;15;23;33;47}. The average heart rate of the athletes in both groups was slightly elevated but not different between groups. The serum sodium concentration and blood glucose concentrations of the athletes in both groups were within the normal range throughout the study, as abnormalities in these parameters were exclusion criteria. The final limitation of this study was the small sample size and this trial should be repeated with a larger sample size.

The time to discharge of the athletes is of interest to medical staff providing medical care at these events. In this study, 19/24 athletes were discharged within 60 minutes after admission - 9/12 from the group who received oral fluids, and 10/12 from the group given intravenous fluid. In this study, blood pressure remained stable over time and there was a small (not significant)

decrease in heart rate over time during recovery. The comparison between admission and discharge blood pressure and heart rates showed that there was no significant difference in recovery with regards to the vital signs between the two protocols. The discharge vital signs are the last recorded vital signs before discharge and thus are not recorded at the same time for each patient.

Finally, it is important to point out that there are no scientific-based criteria that justified the selection of discharge criteria for discharge in this study – these were based on experiential evidence from a collective experience of sports physicians treating endurance athletes.

In summary, the result of this study show that time to the discharge following EAPH is the same whether an athlete is given a large volume of intravenous fluid or is treated in the Trendelenburg position and given small volumes of oral fluids. These findings suggest that the cause of EAPH is not due to volume depletion (dehydration) but rather due to venous pooling of blood in the lower limbs upon cessation of exercise.

Chapter 4

Summary and conclusion

Athletes participating ultra-endurance events, such as the Ironman Triathlon (3.8km swim, 180km cycle and 42.2km run) and Comrades Marathon (89km road run) are at risk of developing an array of medical conditions. One of the most common conditions encountered by the medical staff at these events is the athlete who collapses after finishing the race. Although many terms have been used to describe this condition^{2;3;6;8-12;14;16}, the term Exercise-Associated Postural Hypotension (EAPH) is preferred as it gives an insight into the possible pathophysiology of this symptoms/sign complex. .

The pathophysiology of EAPH has been debated in the literature, and currently there are two hypotheses. The first hypothesis emerged following a study in 1967 on runners in South Africa. In this hypothesis, it was suggested that dehydration coupled with hyperthermia eventually results in circulatory collapse. This hypothesis was recently challenged based on the following findings from various studies:

- The rectal temperature of the collapsed athlete is not significantly elevated in comparison to the non-collapsed runner^{2;8;14;17}

- Most collapsed athletes do not have clinical signs and biochemical confirmation which suggest that they are more dehydrated than non-collapsed athletes ^{2;8;14}
- Most (85%) of the athletes who collapse do so upon completing the race ^{2;8;14}, implying that physiological changes associated with the completion of exercise is causally related to the collapse
- The symptoms/clinical signs of athletes who collapse can be reversed if the athlete is placed in the Trendelenburg position ^{2;8;14}.

This lack of scientific support for the “dehydration/hyperthermia” hypothesis has prompted researchers to re-examine the result of earlier studies. This re-assessment revealed that postural hypotension could be a possible trigger for collapse following exercise. This has led to the more recently proposed hypothesis of post exercise postural hypotension as an explanation for the development of EAC. In Chapter 2, it was argued that there is considerable support for this hypothesis, and therefore the term to describe the symptoms/sign complex typical of the “less severe” collapsed athlete that is most appropriate is EAPH.

The proposed pathophysiology of EAPH is that during exercise, vasodilation occurs to increase heat loss through sweating. While exercise continues, the blood in the dilated venous system is returned to the central blood volume through the “muscle pump” system (also referred to as the “second heart”). However, on cessation of exercise, the “muscle pump” is removed, which results in pooling of blood in the venous system, causing a decrease in the

venous return. This vasodilation is not matched by the increase in cardiac output. It has been highlighted that this decreased venous return may be perpetuated by the Barcroft/Edholm reflex which results in further vasodilation and bradycardia.

The exact reason why some athletes are more susceptible to vasodilation is the subject of much research, and areas currently being researched include, alterations in the sympathetic nervous system^{22;35;39;46}, an altered vascular mechanism in response to the sympathetic nervous system^{22;35;39}, the possible role of arginine vasopressin (AVP) on V1 receptors³⁷, and the possible role of histamine (H1 and H2) receptors^{38;41}.

Based on the fact that there are predominantly two hypotheses to explain the pathophysiology of collapse following exercise, it became clear that there were also two main methods of treating athletes who collapse at the finish of a race. The two most commonly employed treatment protocols are intravenous fluids and the Trendelenburg position with oral fluids ad libitum. The aim of this study was to compare the two treatment protocols in the treatment of exercise-associated postural hypotension, namely, the Trendelenburg position and oral fluids compared with intravenous fluids. The results show that there was no significant difference in time to discharge between the two treatment protocols despite the fact that the intravenous group received a five-times greater volume of fluid in the treatment period. The results of this study thus support the hypothesis that EAPH is not due to dehydration, but is in fact due to pooling of blood in the lower limbs when the "muscle pump" is removed.

The clinical implications of this study are as follows:

1. Athletes with EAPH can be treated safely by placing them in the Trendelenburg position and allowing them to drink fluids ad libitum while monitoring their progress
2. Athletes with EAPH that are treated using the Trendelenburg position and oral fluid protocol should not have any contra-indications to oral fluids such as gastrointestinal upsets (vomiting and diarrhoea)
3. Athletes with EAPH who are treated using the Trendelenburg position and oral fluids protocol will result in discharge from the medical facility within 60 minutes, in most cases
4. Athletes with EAPH who are treated with intravenous fluids do not decrease the time to discharge from the medical facility.

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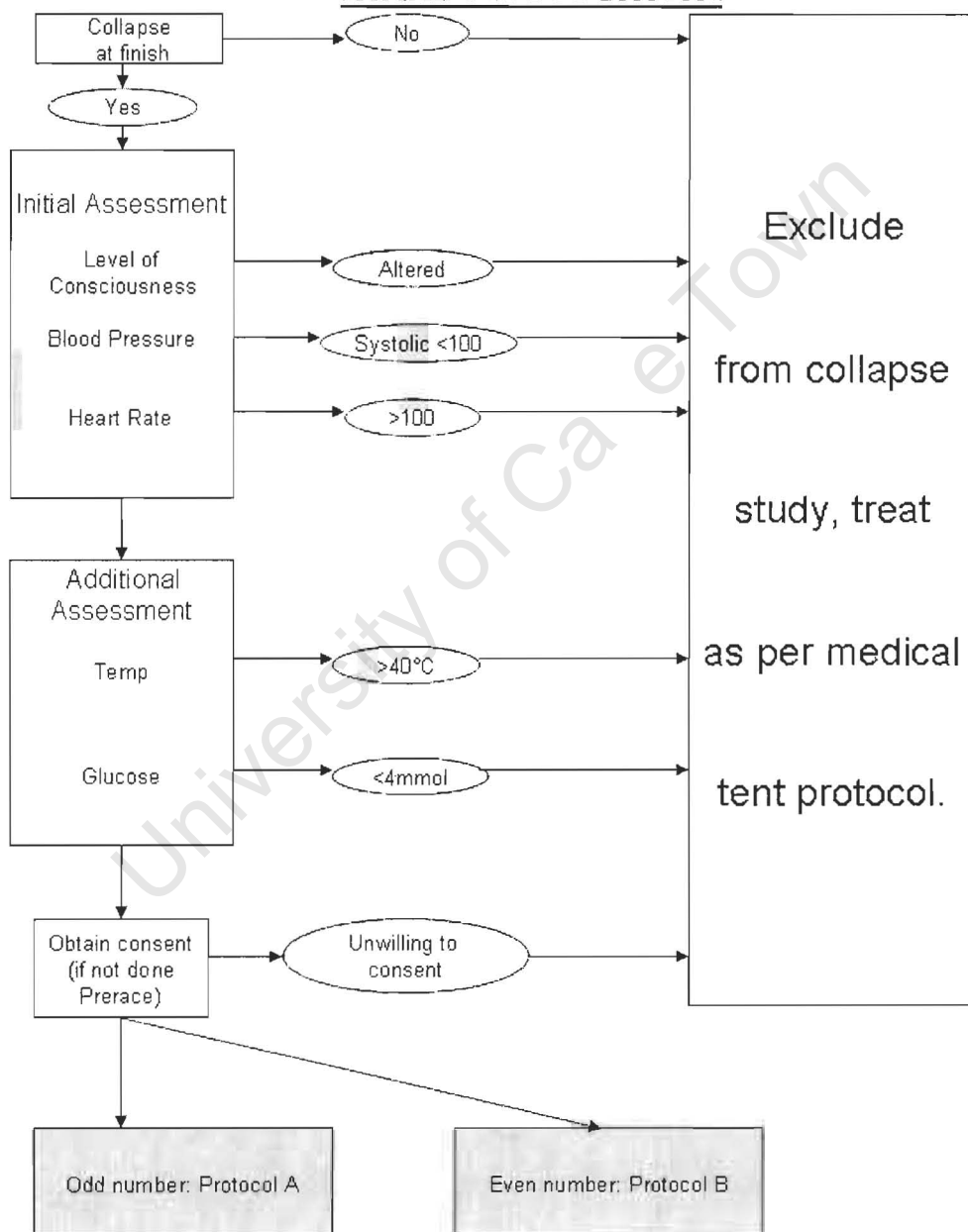
Appendix 1

Collapse Study: Protocol Oral

Race number: _____ Name: _____ Age: _____

Time in: _____ Gender: M / F Bed: _____

Initial Assessment



Protocol A

(Odd race number)

Admission:

Time: _____

Heart rate	
Systolic BP lying	
Diastolic BP Lying	
Systolic BP standing	
Diastolic BP standing	

Mental status: Alert Altered → Remove from study

- What is your name?
- How old are you?
- What is the date?
- What race have you just completed?
- Where are you?

Admission weight: _____

Pre-race weight: _____

Treatment plan:

1. Insert venflon with 50mls Normal Saline over 1 hour. (Take Sodium sample at the same time.)
2. Raise athlete's legs and buttock to 30°.
3. Advice the athlete he/she may drink as much as they like.
4. Reassess every 15min till discharge.
5. Confirm sodium value: If 135 – 148 continue.
If <135 or >148 remove from study.
6. Reassess in 15 minutes

Reassessment 15 min post admission:

Time: _____

Mental status: Alert

Altered → Remove from study

- What is your name?
- How old are you?
- What is the date?
- What race have you just completed?
- Where are you?

Heart rate	
Systolic BP lying	
Diastolic BP Lying	
Systolic BP standing	
Diastolic BP standing	

Additional notes (new symptoms or meds given):

Criteria for discharge:

Athlete requests discharge

No post-exercised postural hypotension
(Change of less than 20 systolic BP)

No altered mental state

Walk 5M unassisted (if above normal)

Repeat sodium taken (if discharged)

Reassessment 30 min post admission:

Time: _____

Mental status: Alert

Altered → Remove from study

- What is your name?
- How old are you?
- What is the date?
- What race have you just completed?
- Where are you?

Heart rate	
Systolic BP lying	
Diastolic BP Lying	
Systolic BP standing	
Diastolic BP standing	
Fluids taken - Volume	
Fluids taken - Type	

Additional notes (new symptoms or meds given):

Criteria for discharge:

- Athlete requests discharge
- No post-exercised postural hypotension
(Change of less than 20 systolic BP)
- No altered mental state
- Walk 5M unassisted (if above normal)
- Repeat sodium taken (if discharged)

Reassessment 45 min post admission:

Time: _____

Mental status: Alert Altered → Remove from study

- What is your name?
- How old are you?
- What is the date?
- What race have you just completed?
- Where are you?

Heart rate	
Systolic BP lying	
Diastolic BP Lying	
Systolic BP standing	
Diastolic BP standing	

Additional notes (new symptoms or meds given):

Criteria for discharge:

- Athlete requests discharge
- No post-exercised postural hypotension
(Change of less than 20 systolic BP)
- No altered mental state
- Walk 5M unassisted (if above normal)
- Repeat sodium taken (if discharged)

Reassessment 60 min post admission:

Time: _____

Mental status: Alert

Altered → Remove from study

- What is your name?
- How old are you?
- What is the date?
- What race have you just completed?
- Where are you?

Heart rate	
Systolic BP lying	
Diastolic BP Lying	
Systolic BP standing	
Diastolic BP standing	
Fluids taken - Volume	
Fluids taken - Type	

Additional notes (new symptoms or meds given):

Criteria for discharge:

- Athlete requests discharge
- No post-exercised postural hypotension
(Change of less than 20 systolic BP)
- No altered mental state
- Walk 5M unassisted (if above normal)
- Repeat sodium taken (if discharged)

Reassessment 75 min post admission:

Time: _____

Mental status: Alert Altered → Remove from study

- What is your name?
- How old are you?
- What is the date?
- What race have you just completed?
- Where are you?

Heart rate	
Systolic BP lying	
Diastolic BP Lying	
Systolic BP standing	
Diastolic BP standing	
Fluids taken - Volume	
Fluids taken - Type	

Additional notes (new symptoms or meds given):

Criteria for discharge:

- Athlete requests discharge
- No post-exercised postural hypotension
(Change of less than 20 systolic BP)
- No altered mental state
- Walk 5M unassisted (if above normal)
- Repeat sodium taken (if discharged)

Reassessment at discharge:

Time: _____

Mental status: Alert Altered → Remove from study

- What is your name?
- How old are you?
- What is the date?
- What race have you just completed?
- Where are you?

Heart rate	
Systolic BP lying	
Diastolic BP Lying	
Systolic BP standing	
Diastolic BP standing	
Fluids taken - Volume	
Fluids taken - Type	

Additional notes (new symptoms or meds given):

Criteria for discharge:

- Athlete requests discharge**
- No post-exercised postural hypotension**
(Change of less than 20 systolic BP)
- No altered mental state**
- Walk 5M unassisted** (if above normal)
- Repeat sodium taken**

Confirm follow-up plan with athlete:

Fax

E-mail

Fax number: _____

E-mail address: _____

Discharge time: _____

Reason if transferred to alternative treatment: _____

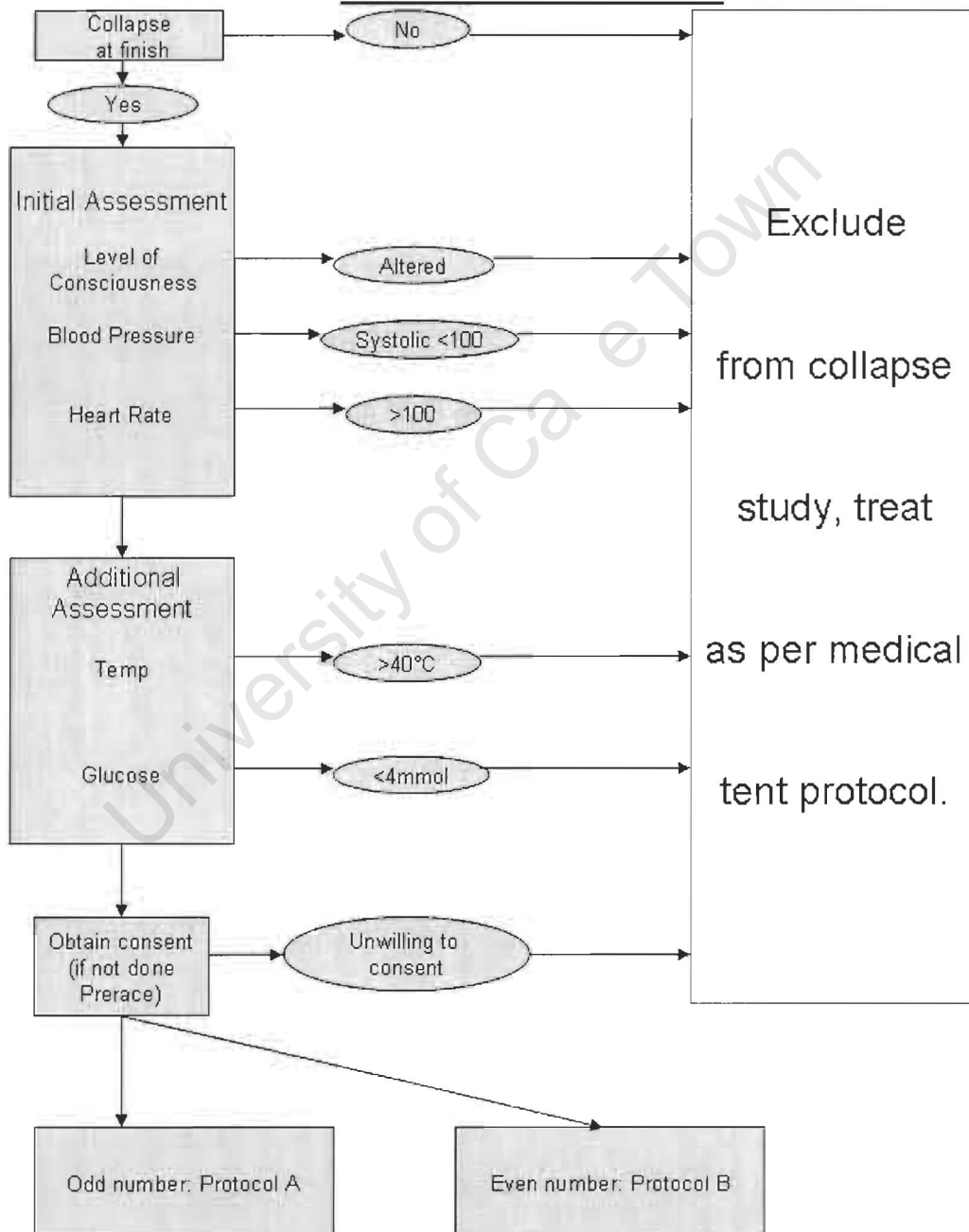
Appendix 2

Collapse Study: Protocol IV

Race number: _____ Name: _____ Age: _____

Time in: _____ Gender: M / F Bed: _____

Initial Assessment



Protocol B

(Even race number)

Admission:

Time: _____

Heart rate	
Systolic BP lying	
Diastolic BP Lying	
Systolic BP standing	
Diastolic BP standing	

Mental status: Alert Altered → Remove from study

- What is your name?
- How old are you?
- What is the date?
- What race have you just completed?
- Where are you?

Admission weight: _____

Pre-race weight: _____

Treatment plan:

6. Insert venflon with 1000 mls Normal Saline over 1/2 hour. (Take Sodium sample at the same time.)
 7. Athlete remains supine
 8. Advice the athlete he/she may NOT drink during treatment.
 9. Reassess every 15min till discharge.
 10. Confirm sodium value: If 135 – 148 continue.
If <135 or >148 remove from study.
6. Reassess in 15 minutes.

Reassessment 15 min post admission:

Time: _____

Mental status: Alert Altered → Remove from study

- What is your name?
- How old are you?
- What is the date?
- What race have you just completed?
- Where are you?

Heart rate	
Systolic BP lying	
Diastolic BP Lying	
Systolic BP standing	
Diastolic BP standing	

Additional notes (new symptoms or meds given):

Criteria for discharge:

- Athlete requests discharge
- No post-exercised postural hypotension
(Change of less than 20 systolic BP)
- No altered mental state
- Walk 5M unassisted (if above normal)
- Repeat sodium taken (if discharged)

Reassessment 30 min post admission:

Time: _____

Mental status: Alert

Altered → Remove from study

- What is your name?
- How old are you?
- What is the date?
- What race have you just completed?
- Where are you?

Heart rate	
Systolic BP lying	
Diastolic BP Lying	
Systolic BP standing	
Diastolic BP standing	
Volume given	

Additional notes (new symptoms or meds given):

Criteria for discharge:

Athlete requests discharge

No post-exercised postural hypotension
(Change of less than 20 systolic BP)

No altered mental state

Walk 5M unassisted (if above normal)

Repeat sodium taken (if discharged)

Reassessment 45 min post admission:

Time: _____

Mental status: Alert

Altered → Remove from study

- What is your name?
- How old are you?
- What is the date?
- What race have you just completed?
- Where are you?

Heart rate	
Systolic BP lying	
Diastolic BP Lying	
Systolic BP standing	
Diastolic BP standing	

Additional notes (new symptoms or meds given):

Criteria for discharge:

Athlete requests discharge

No post-exercised postural hypotension
(Change of less than 20 systolic BP)

No altered mental state

Walk 5M unassisted (if above normal)

Repeat sodium taken (if discharged)

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Reassessment 60 min post admission:

Time: _____

Mental status: Alert Altered → Remove from study

- What is your name?
- How old are you?
- What is the date?
- What race have you just completed?
- Where are you?

Heart rate	
Systolic BP lying	
Diastolic BP Lying	
Systolic BP standing	
Diastolic BP standing	
Volume given	

Additional notes (new symptoms or meds given):

Criteria for discharge:

- Athlete requests discharge**
- No post-exercised postural hypotension**
(Change of less than 20 systolic BP)
- No altered mental state**
- Walk 5M unassisted** (if above normal)
- Repeat sodium taken** (if discharged)

Reassessment 75 min post admission:

Time: _____

Mental status: Alert Altered → Remove from study

- What is your name?
- How old are you?
- What is the date?
- What race have you just completed?
- Where are you?

Heart rate	
Systolic BP lying	
Diastolic BP Lying	
Systolic BP standing	
Diastolic BP standing	
Volume given	

Additional notes (new symptoms or meds given):

Criteria for discharge:

- Athlete requests discharge
- No post-exercised postural hypotension (Change of less than 20 systolic BP)
- No altered mental state
- Walk 5M unassisted (if above normal)
- Repeat sodium taken (if discharged)

Assessment at discharge:

Time: _____

Mental status: Alert

Altered → Remove from study

- What is your name?
- How old are you?
- What is the date?
- What race have you just completed?
- Where are you?

Heart rate	
Systolic BP lying	
Diastolic BP Lying	
Systolic BP standing	
Diastolic BP standing	
Total volume given	

Additional notes (new symptoms or meds given):

Criteria for discharge:

- Athlete requests discharge**
- No post-exercised postural hypotension**
(Change of less than 20 systolic BP)
- No altered mental state**
- Walk 5M unassisted (if above normal)**
- Repeat sodium taken**

Confirm follow-up plan with athlete:

Fax

E-mail

Fax number: _____

E-mail address: _____

Discharge time: _____

Reason if transferred to alternative treatment: _____

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