



Exercise-associated collapse: an evidence-based review and primer for clinicians

Chad A Asplund,¹ Francis G O'Connor,² Timothy D Noakes^{3,4}

¹Department of Family Medicine, Eisenhower Army Medical Center, Fort Gordon, Georgia, USA

²Military and Emergency Medicine, Uniformed Services University of the Health Sciences, Bethesda, Maryland, USA

³MRC/UCT Research Unit for Exercise Science and Sports Medicine, University of Cape Town, Cape Town, South Africa

⁴Department of Human Biology, Sports Science Institute of South Africa, Cape Town, South Africa

Correspondence to

Chad A Asplund, Department of Family Medicine, Eisenhower Army Medical Center, Fort Gordon, GA 30905, USA; chad.asplund@gmail.com

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ABSTRACT

Exercise-associated collapse (EAC) commonly occurs after the completion of endurance running events. EAC is a collapse in conscious athletes who are unable to stand or walk unaided as a result of light headedness, faintness and dizziness or syncope causing a collapse that occurs after completion of an exertional event. Although EAC is perhaps the most common aetiology confronted by the medical provider attending to collapsed athletes in a finish-line tent, providers must first maintain vigilance for other potential life-threatening aetiologies that cause collapse, such as cardiac arrest, exertional heat stroke or exercise-associated hyponatraemia. Previously, it has been believed that dehydration and hyperthermia were primary causes of EAC. On review of the evidence, EAC is now believed to be principally the result of transient postural hypotension caused by lower extremity pooling of blood once the athlete stops running and the resultant impairment of cardiac baroreflexes. Once life-threatening aetiologies are ruled out, treatment of EAC is symptomatic and involves oral hydration and a Trendelenburg position – total body cooling, intravenous hydration or advanced therapies is generally not needed.

INTRODUCTION

Endurance sports are increasingly popular with over 500 000 participants in marathon running events in 2010.¹ After marathon running events, one study demonstrated that an estimated 25 of every 1000 finishers seek medical attention; however, this number can be highly dependent on environmental conditions.^{2,3} Of those runners seeking medical attention, exercise-associated collapse (EAC) is the most common condition seen in the medical tent, comprising 59–85% of all visits after marathons and ultramarathons.^{2,4} The mechanism of EAC is multifactorial and has previously been attributed to hyperthermia or dehydration.^{5–7} Currently, however, EAC is believed to be principally the result of transient postural hypotension caused by lower extremity pooling of blood once the athlete stops running and the resultant impairment of cardiac baroreflexes.^{8,9} The purpose of this article is to review the available evidence to better elucidate the mechanisms of EAC to ensure the best treatment modalities and to provide clinicians with an evidence-based algorithm to guide race day management.

METHODS

Medline, limited to human subjects and English language, was searched using the following terms: 'exercise-associated collapse', 'exercise-associated postural hypotension', 'postexercise collapse' and 'exercise and orthostatic intolerance', which

resulted in 86 articles, of which 26 were review articles. The abstracts of the articles were reviewed, and the references from the review articles were also reviewed, and a total of 34 studies deemed appropriate. Evidence was graded using the Oxford Centre for Evidence-Based Medicine 2011 Levels of Evidence.¹⁰

DEFINITIONS

Exercise-associated collapse

Collapse in conscious athletes who are unable to stand or walk unaided as a result of light headedness, faintness and dizziness or syncope causing a collapse that occurs after completion of an exertional event or stopping exercise.¹¹

Exercise-associated postural hypotension

Postexercise symptoms caused by a decline in systolic blood pressure by at least 20 mm Hg below supine values on assuming the upright posture.⁴

Orthostatic intolerance

Symptoms caused by orthostatic hypotension, which is a sustained reduction of systolic blood pressure of at least 20 mm Hg or diastolic blood pressure of 10 mm Hg within 3 min of standing.¹²

Although EAC, exercise-associated postural hypotension (EAPH) and orthostatic intolerance (OI) all describe potential causes of syncope or presyncope, EAC and EAPH are specific to exercise. EAPH is differentiated from EAC in that blood pressures have been measured and found to be different in a supine and standing position. However, both EAPH and EAC describe collapse in athletes after exertion.

HEAT STROKE AND HYPONATRAEMIA

Exertional heat stroke (EHS) is characterised by central nervous system dysfunction, which may manifest as collapse or syncope, associated with an increased core body temperature (>40°C), which is induced by exercise.^{13,14} Exercise-associated hyponatraemia (EAH) is a potentially life-threatening condition characterised by a decrease in serum sodium (<135 mmol/L) and mental status changes. Athletes with EAH may have true syncope, confusion or disorientation but will have alteration in serum sodium.¹⁵

Although EHS and EAH can be causes of collapse in endurance sporting activities, they are associated with abnormal vital signs and symptoms and should be considered and ruled out before considering a diagnosis of EAC. The focus of this review is EAC, its mechanism and treatment; therefore, EHS and EAH will not be discussed further in this review.

MECHANISM OF EAC

Endurance training is associated with an increased cardiac output and volume load on the left and right ventricles, causing the endurance-trained heart to a dilatation of the left ventricle combined with a mild-to-moderate increase in left ventricular wall thickness. This training-induced increase in cardiac output allows trained athletes to have a lower resting heart rate compared with the non-trained athletes. Furthermore, during exercise, the active muscles of the lower extremities require increased blood flow, and therefore, peripheral vascular resistance decreases to accommodate this need. To generate this large cardiac output, and to counter the resting decrease in heart rate secondary to training effect, athletes must increase their stroke volume and vascular resistance. Working skeletal muscle functions as a 'second heart', ensuring cardiac return to the heart from the dilated lower extremity vasculature. On cessation of activity, the second heart effect no longer assists venous return, and large volumes of blood may pool in the lower extremities and contribute to EAC. Therefore, the very adaptations that contribute to successful completion of

endurance activities are also a large factor in the increased OI found in endurance athletes.

Evidence supports this increased susceptibility to OI in exercise-trained athletes. Studies support the concept of increase in calf and lower extremity compliance and increased diastolic chamber compliance and distensibility as contributors to OI in athletes.^{16–18} Endurance athletes have larger increases in left ventricular end-diastolic volume compared with non-athletes, which allow them to generate the necessary larger stroke volume.¹⁹ Trained athletes also demonstrated a decreased ventricular untwisting rate compared with non-trained athletes, demonstrating the trained heart's ability to adapt to maintain cardiac output.²⁰ Training-related expansion of vascular volume is associated with decreased heart rate response to baroreceptor stimulation.²¹ In addition, this exercise-induced change in cardiac filling volume and output may lead to a resetting of the cardiopulmonary baroreflex.²² Because of this reset baroreflex, trained individuals may depend more on maintenance of venous return to maintain upright body position after exercise.²³ Finally, a critical review supports the exercise-induced increase

Table 1 Mechanism

Ref	Author/journal	Year	Study type	Patients	Outcome	LOE
16	Morikawa <i>et al</i> <i>Med Sci Sports Exerc</i>	2001	Controlled trial	26 female runners, 23 age-matched controls	Exercise-trained females have a high level of orthostatic intolerance during LBNP. Increase in leg compliance may play an important role	2
17	Levine <i>et al</i> <i>Circulation</i>	1991	Controlled trial	7 athletes and 6 controls	Endurance athletes have greater diastolic chamber compliance and distensibility than non-athletes	2
18	Levine <i>et al</i> <i>J Appl Physiol</i>	1991	Controlled trial	8 high fit, 8 mid fit and 8 low-fit athletes	Calf compliance and carotid baroreflex response contributed to individualised response to LBNP	2
19	Esch <i>et al</i> <i>Am J Physiol Heart Circ Physiol</i>	2007	Controlled trial	8 athletes and 8 controls	Endurance athletes had larger increases in left ventricular end-diastolic volume compared with non-athletes despite similar right ventricular cavity area	2
20	Esch <i>et al</i> <i>J Appl Physiol</i>	2010	Controlled trial	8 athletes and 8 controls	Endurance athletes demonstrated a decreased left ventricular untwisting rate compared with non-athletes	2
21	Convertino <i>Med Sci Sports Exerc</i>	2003	Controlled trial	8 men	Expansion of vascular volume after exercise is associated with reduced heart rate response to baroreceptor stimulation	2
22	Ogoh <i>et al</i> <i>J Appl Physiol</i>	2006	Clinical trial	8 men	Cardiopulmonary baroreflex may be reset during exercise to a new operating point associated with exercise-induced change in cardiac filling volume	2
23	Ogoh <i>et al</i> <i>J Physiol</i>	2003	Clinical trial	14 men	Highly fit individuals depend more on maintenance of venous return to maintain upright body position	2
24	Convertino <i>Med Sci Sports Exerc</i>	1993	Critical review	NA	Increased stroke volume is the main mechanism of compensation in aerobic trained individuals	3

LBNP, lower body negative pressure; LOE, level of evidence.

Table 2 Dehydration/heat

Ref	Author/journal	Year	Study type	Patients	Outcome	LOE
23	Ogoh <i>et al</i> <i>J Physiol</i>	2003	Clinical trial	14 men	Baroreflex response to progressive depletion of central blood volume is attenuated in endurance training	2
26	Crandall <i>Am J Physiol Heart Circ Physiol</i>	2000	Controlled trial	12 subjects	Reduced baroreflex response coupled with a reduction in ability to increase heart rate may contribute to increased susceptibility to OI	2
27	Cheuvront <i>et al</i> <i>J Appl Physiol</i>	2010	Critical review	NA	Dehydration augments hyperthermia and reduced plasma volume, which accentuates cardiovascular strain	3
28	Wilson <i>et al</i> <i>Am J Physiol Regul Integr Com Physiol</i>	2006	Controlled trial	15 men	Heat stress decreases cerebral blood velocity and increases cerebral vascular resistance	2
29	Charkoudian <i>et al</i> <i>J Physiol</i>	2003	Controlled trial	13 healthy subjects	Exercise-induced dehydration leads to changes in baroreflex control of blood pressure	2
30	Holtzhausen <i>et al</i> <i>Med Sci Sports Exerc</i>	1995	Clinical trial	31 runners	All runners were volume depleted (average of 4.6% deficit), 68% developed asymptomatic EAPH, dehydration did not compromise cardiovascular status	2

EAPH, exercise-associated postural hypotension; LOE, level of evidence; OI, orthostatic intolerance.

Table 3 Baroreflex modulation

Ref	Author/journal	Year	Study type	Patients	Outcome	LOE
31	Murrell <i>et al</i> <i>J Appl Physiol</i>	2007	Clinical trial	7 athletes	After exercise, hypotension and postural reduction in autonomic function or baroreflex control place the brain at risk for hypoperfusion	2
32	Halliwill <i>et al</i> <i>J Physiol</i>	1996	Controlled trial	9 men	Baroreflex control of sympathetic activity into vascular resistance is altered after dynamic exercise	2
33	Gratzke <i>et al</i> <i>Eur Heart J</i>	2008	Clinical trial	51 men	Postexercise OI is associated with a high basal sympathetic modulation of vasomotor tone in combination with diminished orthostatic response to resistance vessels	2
34	Privett <i>et al</i> <i>Br J Sports Med</i>	2010	Clinical trial	10 experienced male runners	After prolonged exercise, SBP decreases as a result of inadequately compensated decrease in SV and resultant CO from standing	2
35	Fu <i>et al</i> <i>Am J Physiol Heart Circ Physiol</i>	2004	Controlled trial	10 women and 13 men	Lower orthostatic intolerance in women is associated with decreased cardiac filling rather than decreased vascular resistance compared with men	2

LOE, level of evidence; OI, orthostatic intolerance; SBP, systolic blood pressure.

Table 4 Other

Ref	Author/journal	Year	Study type	Patients	Outcome	LOE
36	Adler <i>et al</i> <i>Diabetes</i>	2009	Clinical trial	20 men	Baroreflex sensitivity and response to hypotensive stress are attenuated by antecedent hypoglycaemia	2
37	Howden <i>et al</i> <i>Exp Physiol</i>	2004	Case-control	9 subjects	Increased arterial CO ₂ may be associated with increased cerebral blood flow	3
38	Lucas <i>et al</i> <i>J Appl Physiol</i>	2008	Crossover	9 men	During prolonged exercise, postural-induced hypotension and hypocapnia exacerbate cerebral hypoperfusion	3
39	Middleton <i>et al</i> <i>Eur J Clin Pharmacol</i>	1987	Case-control	7 women and 7 controls	Significant impairment of cardiovascular reflex responses while taking antidepressant medications	3
40	Lockwood <i>et al</i> <i>J Physiol</i>	2005	Randomized-controlled trial	14 healthy men and women	H1 receptor antagonist reduces vasodilatation after exercise and blunts post exercise hypotension	1
41	McCord <i>et al</i> <i>J Appl Physiol</i>	2006	Randomised-controlled trial	10 healthy men and women	H2 receptor antagonist reduces vasodilatation after exercise and blunts postexercise hypotension	1

LOE, level of evidence.

in stroke volume as a compensatory mechanism against OI²⁴ (table 1).

THE ROLES OF HEAT AND DEHYDRATION

Although dehydration leading to hyperthermia has been postulated as a primary factor for EAC,^{5–7} there is no evidence to support its overall responsibility for OI or EAC in endurance athletes.²⁵ Evidence, however, supports both heat stress and increased skin temperature as contributing factors in OI. Heat stress results in the reduction of baroreflex control in response to an orthostatic challenge.²⁶ Heat stress has also been postulated to impair aerobic exercise performance, primarily through increased cardiovascular strain.²⁷ In addition, increasing body temperature may increase cerebral vascular resistance, reducing the cerebral threshold for neurogenic collapse.²⁸

Two small studies found that laboratory-induced hypovolemia may lead to changes in baroreflex control of blood pressure in certain individuals, which may increase susceptibility to EAC.^{22,29} However, a larger clinical trial following the body composition of 31 runners completing an ultramarathon event found that the collapsed runners did not have a higher body temperature than those who did not collapse, and all the runners were dehydrated, but this level of dehydration was unrelated to the degree of postural hypotension after the event³⁰ (table 2). Therefore, although heat and dehydration have not been found to be true causes of EAC or OI in endurance running events, they may possibly be risk factors for EAC or contribute by impairing peripheral vasoconstriction leading to the orthostatic state.

BAROREFLEX MODULATION

Pooling of blood in the lower extremities at the cessation of exercise has been implicated as a mechanism of EAC; if the systemic vascular resistance, which is reduced during exercise, is not triggered by an intact baroreflex to increase after stopping exercise, a lower body negative pressure (LBNP) situation develops and postural hypotension may occur. LBNP is a widely used technique to study the cardiovascular response to this orthostatic stress. Many studies on the effect of LBNP have shown this altered baroreflex to be a primary mechanism of OI after exercise.

Reduction in baroreflex control has been implicated in the diminished orthostatic response after exercise.³¹ A controlled trial of exercising men found that baroreflex control is altered after dynamic exercise.³² Furthermore, in a clinical trial of 51 finishers of a mountain marathon, it was found that a diminished orthostatic response of resistance vessels was the likely aetiology in the OI in these runners after exercise.³³

In a clinical trial of experienced male runners, systolic blood pressure decreases after exercise secondary to a reduction in peripheral vascular resistance leading to a decreased filling volume.³⁴ Women, however, may respond differently to exercise than do men. A controlled clinical trial of both women and men showed that the mechanism of OI in women is likely caused by reduced cardiac filling rather than impaired baroreflex³⁵ (table 3).

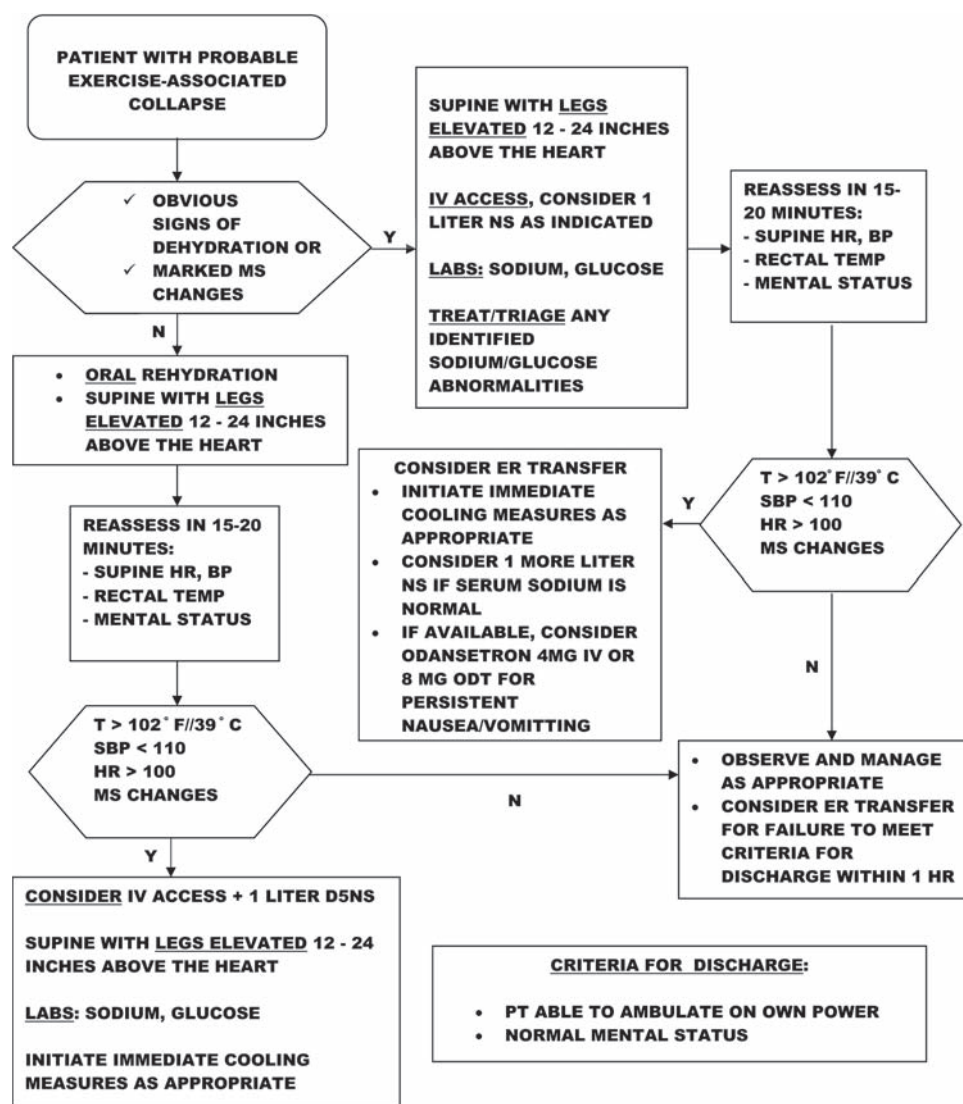
OTHER

There is strong evidence to support an attenuated baroreflex response as a responsible mechanism of OI and EAC. It has been seen that heat may contribute to this response. However,

Table 5 Treatment

Ref	Author/journal	Year	Study type	Patients	Outcome	LOE
42	Journeay <i>et al</i> <i>Aviat Space Environ Med</i>	2004	Randomised controlled trial	6 subjects	Lower body positive pressure promoted restoration of haemodynamics	1
43	Anley <i>et al</i> <i>Br J Sports Med</i>	2010	Randomised controlled trial	28 athletes	EAPH was effectively treated with Trendelenburg positions and oral hydration	1
44	Davis and Fortney <i>Int J Sports Med</i>	1997	Controlled trial	5 men	Fluid ingestion is an effective treatment for orthostatic intolerance	2
45	Durand <i>et al</i> <i>Am J Physiol Regul Integr Comp Physiol</i>	2004	Randomised controlled trial	8 subjects	Skin surface cooling is effective in improving orthostatic tolerance in otherwise normothermic individuals	1
46	Wilson <i>et al</i> <i>J Appl Physiol</i>	2002	Clinical trial	9 subjects	Skin surface cooling may protect against orthostatic intolerance in heat stressed humans	3
47	Cui <i>et al</i> <i>Am J Physiol Heart Circ Physiol</i>	2005	Controlled trial	9 subjects	Skin surface cooling induced an upward shift in central venous pressure	2
48	Privett <i>et al</i> <i>Clin J Sport Med</i>	2010	Controlled trial	6 subjects	Runners who are prone to OI after exercise may reduce this risk by wearing compression stockings while running	2

EAPH, exercise-associated postural hypotension; OI, orthostatic intolerance.

**Figure 1** Exertional collapse algorithm.

there are several other factors that have been studied, which may also exacerbate this response.

Hypoglycaemia has been found to attenuate baroreflex sensitivity, which may be important because serum glucose levels will decrease as length of exercise increases, which may make endurance and ultraendurance athletes more susceptible to EAC.³⁶ Pushing the pace or aiming for a time goal has also been implicated in EAC.³⁰ As the respiratory rate of athletes increases as they try to attain a cutoff or time goal, their level of carbon dioxide will decrease as a result. Studies have shown that hypercarbia may be protective,³⁷ and this resultant hypocapnia may further attenuate the baroreflex response.³⁸

Medications may also affect response to LBNP – antidepressant medications have been shown to lead to a significant impairment in cardiovascular reflex response after exercise, which may implicate neurochemicals as possible factors in EAC.³⁹ In addition, in two separate randomised controlled clinical trials H1 and H2 receptor antagonist medications may blunt the body's postexercise hypotension, suggesting that histamine may also play a role in EAC^{40 41} (table 4).

TREATMENT

The evidence points towards a lower extremity pooling of blood with an attenuated baroreflex response as the primary mechanism of EAC; therefore, treatment options should be directed primarily at correcting these deficits. Because there is no good evidence to support hyperthermia or dehydration as the primary aetiologies of EAC, total body cooling and intravenous fluids should not have a role in the initial treatment of EAC.

In a randomised controlled trial, it has been shown that lower body positive pressure, such as what occurs in the Trendelenburg positions, promoted restoration of normal haemodynamics.^{42 43} Studies also suggest that oral hydration may be preventive against EAC and may also be used as an effective treatment for EAC.^{43 44} A randomised controlled trial and two smaller trials suggest that skin surface cooling may act towards directing peripheral blood flow centrally and decreasing cardiovascular strain, thus treating EAC.^{45–47} Finally, the results of a study of compression stockings in runners suggest that runners who are prone to OI after exercise may benefit from wearing compression hose while running.⁴⁸ Those prone to EAC may also potentially benefit from taking H1 or H2 blocking medications, skin surface cooling along the course and ensuring adequate glucose levels during participation (table 5).

ALGORITHM

Using the evidence for aetiology, mechanism and treatment, we propose an algorithm, which is currently used at the Marine Corps Marathon,^{49 50} as a clinical framework for the treatment of EAC in endurance athletes (figure 1). The key to using the EAC algorithm is to approach a collapsed athlete with a wide differential that includes potential life-threatening causes such as EAH or EHS and ruling those out with a concise physical examination evaluating mental status and body temperature before proceeding down the EAC algorithm.

CONCLUSION

EAC is a common occurrence in medical tents after endurance sporting activities, which is typically characterised by collapse after completion of the event in the absence of neurological, biochemical or thermal abnormalities. Although EAC is perhaps the most common aetiology confronted by the medical provider attending to collapsed athletes in a finish-line tent, the provider needs to be reminded that EAC is a diagnosis of

exclusion and that he or she needs to be vigilant for other aetiologies that cause collapse. There is no evidence to support the previous idea that EAC is caused primarily by dehydration or heat stroke. These factors, however, along with medications, hypocapnia and hypoglycaemia, may be contributory to EAC. Evidence currently supports that postural hypotension caused by pooling of blood in the lower extremities, secondary to decreased vascular resistance in the face of an attenuated baroreflex response, as the principal mechanism of EAC. Women may sustain EAC more from decreased cardiac filling than from altered baroreflex. Treatment of EAC is usually symptomatic and involves oral hydration and a Trendelenburg position – total body cooling, intravenous hydration or advanced therapies are generally not needed.

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