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# Exercise-Associated Collapse: Is Hyponatremia in Our Head?

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**Abstract**

Exercise-associated hyponatremia (EAH) is one of the most common causes of exercise-associated collapse. The primary pathogenesis of EAH is largely the result of excessive fluid intake but is influenced by other factors, including hormonal abnormalities (ie, inappropriate arginine vasopressin secretion), renal abnormalities, and mobilization of sodium stores. Early recognition of EAH is crucial to appropriate treatment, because symptoms are varied and may be confused with other causes of exercise-associated collapse. Onsite testing of [Na+] will confirm the diagnosis but is not always available. Rapid treatment of EAH will depend upon the type and severity of symptoms. Treatment protocols range from fluid restriction or oral hypertonic fluids for mild symptoms to intravenous hypertonic fluids for more severe symptoms. Preventative strategies should emphasize fluid consumption based on thirst and athlete/coach/staff education regarding proper hydration practices.

## Introduction

Exercise-associated collapse (EAC) is a dramatic event that impacts athletes in all sports. It is defined as the inability to stand or walk unaided during or at the completion of a sports event [1]. EAC is most common in endurance events, such as marathons, ultramarathons, and triathlons. The incidence of EAC appears to increase as the race distance, temperature, and humidity increase [1]. The vast majority of cases involving EAC are benign in nature without any long-term deleterious effects. However, potential life-threatening causes include cardiovascular compromise, heat-related illness, and hyponatremia.

Exercise-associated hyponatremia (EAH) is one of the most common causes of serious EAC. EAH is defined as a serum, plasma, or blood sodium concentration level ([Na+]) below the normal reference range, (usually 135 mmol/L), or a serum sodium decrease of 7%-10% [2]. Delayed recognition and treatment of EAH can lead to severe and potentially life-threatening complications. The pathogenesis of EAH is largely attributable to excessive water intake but is not completely understood. Other etiologies relating to hormonal abnormalities (ie, arginine vasopressin [AVP] secretion, also known as antidiuretic hormone), renal abnormalities, and mobilization of sodium stores influence the development of severe hyponatremia. All of these factors impact our body's ability to maintain a certain level of hydration. The goal of this article is to review the epidemiology, pathophysiology, evaluation, and treatment of EAH.

## Epidemiology

EAH was first described in the literature related to an ultraendurance event in Durban, South Africa, in 1981 [3]. Before 1981, athletes were advised to limit drinking fluids during exercise. However, recommendations changed, and athletes were advised to consume fluids at rates of 400-500 mL/h during activity involving prolonged exercise. It is believed these recommendations led to an increase in the episodes of EAH [4]. Since that time, complications from EAH and EAH encephalopathy (EAHE) have been observed across a wide variety of sporting events, with at least 14 confirmed deaths since 1981 [2].

Since EAH initially was described, there have been a variety of case reports, case series, and cross-sectional observational studies evaluating its influence and relevance. A majority of the studies reveal that it is common for endurance athletes to develop asymptomatic hyponatremia, with a reported incidence of asymptomatic EAH, ranging from 0% to 51% [2-4]. The incidence of symptomatic EAH ranges from 0% to 38% of athletes, although the vast majority of endurance

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events report no cases of symptomatic EAH. Fortunately, severe complications from EAH often are quite rare and represent less than 1% of all cases of EAH [2,4]. These studies suggest the overall incidence of EAH will vary by the sporting event and appears to be multifactorial depending on the length of the endurance event, experience of the athlete, and environmental conditions [5,6]. Risk factors for the development of EAH are listed in Table 1.

A number of observational studies have involved marathon runners. The reported incidence of EAH in marathon runners ranges from 0% to 13% of finishers [7-12]. A prospective study of 766 runners competing in the 2002 Boston Marathon reported an incidence of hyponatremia of 13%. Only 0.6% of runners experienced critical hyponatremia, defined as a serum [Na+] <120 mmol/L [7]. In an observational and retrospective case-controlled series involving 5082 finisher from the Houston Marathon, the reported incidence rate of hyponatremia was 0.4%, with only 0.04% of the runners having critical hyponatremia [8].

Studies of EAH in single-stage and multistage ultramarathons reveal a greater incidence of EAH compared with marathons. In a study of 669 runners competing in a 161-km single-stage ultramarathon in Northern California, the average incidence of EAH during a 5-year period was 15.1% with a range between 4.6% and 51% when each year was evaluated separately [5]. The incidence of symptomatic EAH was approximately 0.06%. Additional studies of single-stage ultramarathon runners performed in Switzerland and New Zealand reported EAH incidences of 0% and 4%, respectively [13,14]. For multistage ultramarathons, the incidence has been found to range between 1% and 12% [15,16].

In addition to running events, there have been a number of studies focusing on the incidence of EAH during other endurance events including triathlons, cycling, mountain biking, and swimming. In a study by Noakes, of 2135 athletes from 8 endurance events (ranging from 42.2 to 161 km), 123 of the athletes (6% of the total) were found to have a biochemical hyponatremia (serum [Na+] between 135 and 129 mmol/L) [11]. A total of 31 of the athletes (1% of the total) were found to have a clinically significant hyponatremia (serum [Na+] <129 mmol/L), with 24 of these athletes having symptoms of EAHE. During an Ironman triathlon race, Speedy et al [17] reported 18% of the athletes had biochemical EAH (serum [Na+] levels <135 mmol/L), with 3% of the athletes having severe hyponatremia (serum [Na+] levels <130 mmol/L). In an observational study done in the Czech Republic, 3.7% ultramountain bikers, 8.3% ultrarunners, and 7.1% multistage mountain bikers developed biochemical hyponatremia (serum [Na+] between 134 and 129 mmol/L) [18]. However, no cases of EAH were found in 65 ultraendurance road cyclists competing in a 720-km ultracycling marathon in Switzerland [19]. In a 26.4-km swim, Wagner et al [20] found that 17% of the participating swimmers developed asymptomatic hyponatremia. In another study, Knechtle et al [16] evaluated 200 athletes competing in different disciplines and found that 12 of the finishers (6% of the total) developed EAH with the prevalence of 13% in the swimmers, 10.7% in the road cyclists, 8% in the ultramarathoners, 8% in the mountain ultramarathoners, and none of the mountain bikers.

Despite increasing reports of EAH and EAHE in a variety of endurance sporting events, there continues to be limited research outside of endurance sports. In a study by Jones et al [21], 10 professional rugby players were evaluated during a 4-week period during routine practice, competitions, and gym training. There was no evidence of clinically significant hyponatremia, but asymptomatic hyponatremia was observed in 33% of the athletes after an 80-minute competition. In another recent study by Mayer et al [22], elite-level rowers were evaluated during a 4-week period, and 70% of the athletes were found to have developed asymptomatic EAH at least once during the 4 weeks of training.

Besides the aforementioned 2 studies, most of the literature of EAH in nonendurance sports comes from case reports. There have been case reports published of a half marathon runner [23], a recreational tennis player [24], an open-water swimmer [25], an American college football player [26], and an American professional football player [27] who all developed EAHE. There have also been at least 3 reports of high school football players who have died from complications of EAHE [2,28,29]. In the literature, there were no case reports of EAH or EAHE described for athletes playing other sports such as soccer (international football), basketball, baseball, or softball.

**Table 1**
Risk factors contributing to exercise-associated hyponatremia

| |
|---|
| Sustained, overconsumption of water or hypotonic fluids |
| Weight gain during exercise |
| Exercise duration >4 hours (slowed pace) |
| Easy access to fluids |
| High and low body mass index |
| Inadequate training |
| Inexperience |

## Pathophysiology of EAH

The etiology of EAH is multifactorial and primarily involves 2 multifaceted models: (1) dilution of sodium and (2) sodium depletion (Figure 1). Within these 2 models are several principles surrounding the physiology of sodium mobilization, activation, and loss. These models are in part based on studies that have plotted changes in weight against serum sodium during an exercise event. In these studies, hyponatremia associated with weight gain supports the theory that excessive water intake decreases the ratio of sodium in a larger volume of total body water (dilution), whereas