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REVIEW

Exercise-Associated Hyponatremia: Serum Sodium, Symptomatology, Severity, and Sport Specificity

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Abstract: Exercise-associated hyponatremia (EAH) is an important cause of preventable morbidity and mortality. EAH refers to a low blood sodium concentration [Na^+] of <135 mmol·L^{-1}, during or within 24 h of sustained endurance exercise. The current EAH literature contains ambiguities among field studies and unresolved clinical issues. Seeking clarity and resolution, we conducted manual searches of two large electronic databases using pre-defined inclusion criteria and discovered 1516 article titles and abstracts. Subsequent reviews of 345 full-length articles identified 56 eligible field research studies that reported 220 EAH cases during seven outdoor endurance activities (5–29.5 h duration). Our evaluations of these EAH cases generated the following seven findings. First, a greater percentage, not the absolute number, of women experienced EAH than men. Second, event specificity may account for much of the wide range of EAH symptoms and signs (SAS) reported among different outdoor activities. Third, out of 220 reported cases of EAH, none were asymptomatic when [Na^+] was <130 mmol·L^{-1}. Fourth, the absolute value of [Na^+] is not a reliable predictive index of EAH clinical severity or presentation. Fifth, running or hiking resulted in far more EAH cases and published epidemiological studies than cycling, swimming, and triathlon events. Sixth, the most common mild EAH complaints were nausea, weakness or lethargy, dizziness, headache, and extremity swelling. Seventh, the most common SAS of moderate-to-severe EAH (ie, suggesting hyponatremic encephalopathy) included altered mental status, vomiting, seizure, agitation/restlessness, collapse, and loss of consciousness. In conclusion, these findings should inform pre-event medical planning, on-site medical staff briefings, as well as the diagnosis of EAH severity in field settings. We also propose that our inventory of position statements and consensus documents will meet the needs of athletes and coaches who seek dependable information regarding risk factors and prevention.

Keywords: encephalopathy, cyclist, runner, swimmer, triathlete, hiker

Introduction

Musculoskeletal and dermatologic injuries are relatively common at outdoor endurance events. Critical, life-threatening medical complications, including exercise-associated hyponatremia (EAH), exertional heatstroke, and cardiac arrest occur less frequently[1] but require rapid recognition and appropriate treatment to increase the likelihood of a positive outcome.[2–4] To avoid inappropriate treatment and adverse outcomes, care providers in race medical tents and in hospital settings must differentiate EAH from other causes that present with similar signs and symptoms (SAS) such as dehydration, heat exhaustion and exertional heatstroke.[4–9] This is essential because advanced cases of EAH may involve non-cardiogenic pulmonary edema, cerebral edema, or death.[7,10–13]

With few exceptions,[14] the decisive diagnostic criterion for EAH in most clinical laboratories is a blood sodium concentration ([Na^+]; serum or plasma) below 135 mmol·L^{-1} which occurs during or up to 24 h after prolonged exercise, regardless of the presence or absence of SAS.[3,7,12,15,16] Thus, EAH may appear in either asymptomatic or symptomatic

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forms; the former can rapidly progress to the latter within a single athletic or recreational activity.[11,17] Asymptomatic athletes with [Na^+] <135 mmol·L^{-1} usually are identified while participating in research studies or are detected in blood samples obtained for reasons other than suspicion of EAH. These individuals typically present with nonspecific and transient complaints which are commonly experienced by other participants who do not seek medical care following exercise, and whose [Na^+] is rarely analyzed.[18–21] In contrast, athletes with symptomatic EAH typically report to race medical facilities or to hospital emergency departments after finishing an event, seeking treatment for a variety of symptoms. Observations of neurologic impairment, seizures, or vomiting should raise the index of suspicion for moderate-to-severe EAH[3,6,7,17,19,21] and prompt the measurement of serum [Na^+] to confirm or exclude EAH diagnosis.[6,7,19,22] However, symptomatology may not reveal the severity of EAH in all athletes. For example, runners who required on-site medical treatment for various complaints (eg, dizziness, headache, vomiting, seizure), were observed by Hsieh et al.[14] They reported that no unique sign or symptom distinguished marathon runners with a serum [Na^+] <130 mmol·L^{-1} from those who were normonatremic (>135 mmol·L^{-1}). A similar conclusion was published in a field study of ultramarathoners by Hoffman et.al.[23] No SAS distinguished runners who developed EAH along a 161-km course from those not developing EAH. Additional supportive evidence has been reported in the sport of road cycling. The total symptoms score of ultraendurance cyclists, evaluated with a validated symptoms questionnaire, was not statistically correlated to the change of serum [Na^+] (range, +6 to −11 mmol·L^{-1}) during a 164-km summer mass participation event.[15] These ambiguities indicate that additional focused studies are warranted to clarify EAH SAS and their potential relationships to serum [Na^+].

Other important issues regarding EAH remain unresolved. In response, the present narrative review focuses on five clinically relevant but unanswered questions. First, are women at greater risk of experiencing asymptomatic and symptomatic EAH than men? Although field studies have observed that women are at greater risk of experiencing EAH,[6,10,24–27] it is important to note that other publications confirm no effect of sex on EAH development.[28–34] Second, are EAH signs, symptoms, and serum [Na^+] similar during outdoor running, cycling, swimming, and hiking activities? A few previous publications have considered aspects of this question but the scope of those articles was limited.[35–38] A corollary of this question is: at which outdoor athletic and recreational activities have no cases of EAH been observed? Third, what is the relationship between serum [Na^+] and the SAS of EAH? Fourth, do specific SAS distinguish mild EAH from moderate-to-severe EAH? Fifth, which organizational position statements and expert consensus reports provide readily available information about EAH? Considering the aforementioned discordant findings, ambiguities, and topics that require further investigation, we propose that these five clinically relevant questions will reveal previously unrecognized relationships among symptomatology, EAH severity, [Na^+], male-female differences, and the types of outdoor sport activities. These associations can be integrated into a medical support strategy that optimizes decision-making and the treatment of athletes during competitive sports, and recreational enthusiasts at mass participation events.

Terminology

Clarifications of terminology utilized in the present review are necessary. To illustrate, the term "exercise-associated hyponatremia" is preferred; the terms "exercise-induced hyponatremia" and "exertional hyponatremia" do not appear herein. This procedure follows the convention of Wolfson,[39] who observed in 1995 that evidence per se does not support the concept that exercise induces this disorder. Also, the lexicon associated with EAH is not universally established. To standardize key terms, we have adopted the following definitions developed in 2014 and 2019 by the Wilderness Medical Society,[7,40] and in 2015 by the Third International Exercise-Associated Hyponatremia Consensus Development Conference.[19] Thus, we define "exercise-associated hyponatremia" by a serum, plasma, or [Na^+] below the normal reference range of 135 mmol·L^{-1} that occurs during or up to 24 h after prolonged physical activity. "Asymptomatic EAH" relies only on a biochemical determination ([Na^+] <135 mmol·L^{-1}), whereas "symptomatic EAH" refers to a biochemical diagnosis combined with clinical SAS.[7,40] Further, our manual search of two large electronic databases revealed minor differences among the classification of EAH severity. Hew et al[31] delineated three EAH categories in 2003: "mild" (130–135 mmol·L^{-1}), "moderate" (121–129 mmol·L^{-1}), and "critical" (≤120 mmol·L^{-1}). In contrast, three EAH categories included "mild" (130–135 mmol·L^{-1}), "moderate" (125–129 mmol·L^{-1}), and "severe" (<125 mmol·L^{-1}), as compiled from five sources.[24,41–44] In the present review, rather than classifying EAH severity on the

basis of a concentration range or specific [Na^+] values, we aggregate SAS from four sources[3,7,19,45] to classify "mild EAH" and "moderate-to-severe EAH" in tables and figures.

Search Methods

Utilizing the vast range of information stored on internet digital media, manual searches for relevant articles were performed in the PubMed and Google Scholar electronic databases. Combinations of the following key words during literature searches emphasized the clinical diagnosis of exercise-associated hyponatremia (EAH) during large group, recreational, or competitive sport disciplines: "exercise-associated hyponatremia", "exertional hyponatremia", "exercise-induced hyponatremia", "cyclist", "cycling", "biker", "biking", "runner", "running", "triathlon", "triathlete", "swimmer", "swimming", "open water swimming", "hiker", "hiking", "trekker", "trekking", "sport" "athlete", "athletic". All manual searches included "exercise-associated hyponatremia", "exertional hyponatremia", and "exercise-induced hyponatremia" as key words. After removing duplicate records generated by PubMed and Google Scholar, the abstracts and titles of all records were evaluated to determine which studies should be further assessed for eligibility. We then evaluated all articles in portable document format (.pdf) to ascertain if they met the following inclusion criteria: full-text, peer-reviewed, English language, without publication year limits and available through April 24, 2025. Finally, the remaining articles were included in the present review only if (a) data originated at outdoor sport competitions, mass participation events, or recreational activities; (b) each diagnosis of EAH included a measurement of serum or plasma Na^+ concentration <135 mmol·L^{-1};[3,7,12,15,16] and (c) clinical SAS (or absence thereof) and the athlete's sex were described for each case of EAH.

Results

Using the keywords and inclusion criteria described above, the manual screenings of 1516 article titles and abstracts, plus the subsequent reviews of 345 full-length articles, identified the 56 field research studies that were accepted for inclusion in tables and figures. Figure 1 illustrates the steps in this selection process.

The 220 EAH patients described in Table 1 were diagnosed at 66 different outdoor sport competitions, mass participation events, or recreational activities; column 1 specifies the duration of exercise (h), distance completed (km), and year of these events. Each entry in columns 2–5 describes the serum [Na^+] (4 categories), symptomatic or

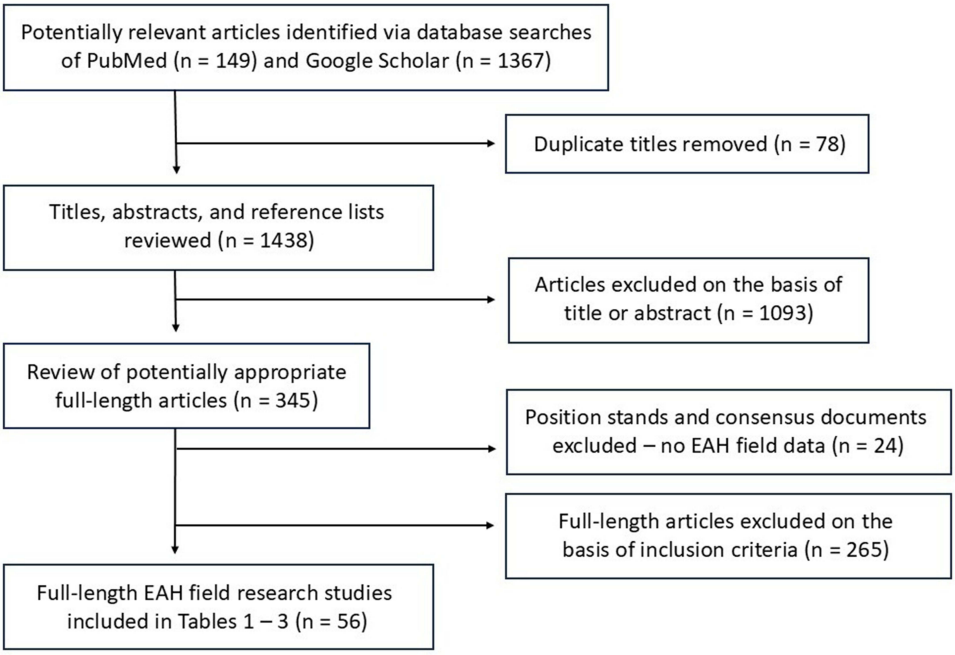

Figure 1 The selection process for the field research studies which appear in tables and figures.

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Table 1 EAH Cases Associated with Outdoor Sport Competitions, Mass Participation Events, or Recreational Activities. Each Data Point (Columns 2–5) Describes the Blood Sodium Concentration, Number of EAH Patients, Symptomatic or Asymptomatic Clinical Presentation, and Sex of Patients

Athletes and Exercise Enthusiasts (Duration of Exercise, Distance Completed, Year)	**Blood [Na+] ($mmol \cdot L^{-1}$)**				**References**
	<120	**120 – 124**	**125 – 129**	**130 – 134**	
Ultraendurance mountain bikers					
(24-h and 4-day races, 441 and 156 km, 2012)			1SM	1SM	[35]
(24-h continuous, 332 km, 2013)				1AF	[46]
(11.9 h, 164 km mountainous, 2013)	1SM				[47]
Ultraendurance road cyclists					
(5.6 h, 109 km, 2003)			1SF		[48]
(27.7 h average, >600 km, 2007)				3AM	[38]
(9.5–12.0 h, 210–250 km, 2010)			1SM	2AM, 1AF	[49]
(8.9 and 10.6 h, 164 km, 2016)				2AM	[15]
(16 h, 300 km, 2017)	1SM[a]				[50]
Marathon runners					
(5.5 h, 42.2 km, 1986)		1SM			[51]
(5.5 h, 42.2 km, 1988)		1SM			[52]
(NR, 42.2 km, 1998–1999)	1SM, 1SF	3SF	1SM, 1SF		[10]
(5.4 h average, 42.2 km, 1998–1999)	4SF	1SM, 10SF[a]	1SM, 2SF	1SM, 7SF[a]	[24]
(4.1–5.3 h, 42.2 km, 2000)		1SM	2SM		[14]
(4.9 h average, 42.2 km, 2000–2004)				6AM, 13AF	[30]
(3.0–5.6 h, 42.2 km, 2003)	1SF	1SM, 5SF	5SM, 1SF	1SM	[53]
(>4.5 h, 42.2 km, 2004)		2SF			[22]
(4 h, 42.2 km, 2007)				1SM	[20]
(5 h, 30 km of a 42.2 km event, 2008)	1SM[a]				[54]
(4.1 h, 42.2 km, 2009)				1SM	[55]
(5.3 h average, 42.2 km, 2009)				1SF, 3SM	[56]
(4.2 h, 42.2 km, 2013)	1SM				[57]
(5.0 h, 42.2 km, 2018)	1SF				[58]
Ultraendurance runners (> 50 km distance)					
(9.0–10.2 h, 70–88 km, 1981–1983)	1SM,1SF[a]	1SM	1SF		[59]
(10.6 and 8.6 h, 80 and 100 km, 1983)	1SM	1SM			[60]
(NR, 61–88 km, 1988)	2SM	2SM, 1SF	3SF		[43]
(NR, 88.5 km, 1993)	1SM[a]				[61]
(NR, 88.5 km, 1994)	1SM[a]				[62]
(12.4 h, 226 km, 2001)			1SM		[63]
(5.7–9.3 h, 60 km, 2003)				3AM, 2AF	[64]
(20–27 h, 121–153 km, 2005–2007)		1SM[a]	1SM, 1SF	1SM	[18]
(10.6 h, 100 km, 2007–2010)				7AM	[65]
(11.5 h, 100 km, 2008)				5AM	[38]
(7-stage mountain event, 350 km, 2008)				2AM	[38]
(12.1 h, 84-km, 2009)	1SM			1SM	[56]
(14.5 h, 100 km, 2009)	1SM		2SM		[56]
(8.1 and 11.3 h, 84 km, 2009)				1SM	[55]
(NR, 100 km, 2011)				4AM	[66]
(11.4 h, 82 km, 2011)				1AM	[67]
(24 h, 221 km, 2012)				1SM	[35]
(24-h continuous, 220 km, 2012)	1SM			1SF	[46]
(27.6 h, 145 km, 2013)					[68]

(*Continued*)

Table 1 (Continued).

Athletes and Exercise Enthusiasts (Duration of Exercise, Distance Completed, Year)	Blood [Na+] ($mmol \cdot L^{-1}$)				References
	<120	120 – 124	125 – 129	130 – 134	
(24-h continuous, 116 km, 2014)				1AF	[46]
(17 h, 72 km, 2014)		1SM			[69]
(29.5 h, 121 km, 2015)				1AM	[70]
(NR, 50 km, 2016)		1SF			[17]
Ultraendurance open water swimmers					
(9.2 h average, 26.4 km, 2008)				2AM	[38]
(6–12 h, 26.4 km, 2009 and 2010)			1SF	2AM, 3AF	[71]
(8.3 h, 20 km, 2013)	1SM				[72]
Triathletes (3.8 km swim, 180 km bike, and 42.2 km run)					
(10.5–13.9 h, Ironman, 1996)		1SM	1SM, 1SF	3SM[a], 2SF	[73]
(12 and 13.3 h, Ironman, 1997)				2SF	[44]
(14.1 h, Ironman, 2000)	1SM				[74]
(11.8 h average, Ironman, 2009)		1SM			[75]
(NR, Ironman, 2012)		1SM			[76]
(NR, Ironman, 2014)	1SF				[77]
(12.5 h average, Ironman, 2014 and 2015)			1SM, 1SF	12SM, 5SF	[78]
Hikers, trekkers					
(1 or 2 d, NR, 1990–1992)	2SF	1SM	1SM		[79]
(1 or 2 d, NR, 1993)	3SF	1SM, 1SF	2SF		[6]
("several hours", NR, 1993)	1SM				[80]
(NR, "a few km", 1996)	3SM, 1SF	3 SF			[81]
(backpacking 2 d, NR, 1997)	1SF				[82]
(hiking 1 d, NR, 2000)	1SF				[83]
(NR, 3 d trek on trails, 2006)	1SM				[84]
(5 h, approx. 10 km, 2008)			1SF		[11]
(9 h, 9 km, 2011)		1SM			[85]
(6 h, 13 km, 2014)		1SF			[86]
Column totals (n = 220)	21 SM 0 AM 17 SF 0 AF	17 SM 0 AM 27 SF 0 AF	17 SM 0 AM 15 SF 0 AF	27 SM 40 AM 18 SF 21 AF	
TOTAL number of male patients	21	17	17	67	122
TOTAL number of female patients	17	27	15	39	98
Ratio of symptomatic to asymptomatic cases (♂ + ♀ combined)	38S:0A	44S:0A	32S:0A	45S:61A	159S:61A
TOTAL (asymptomatic + symptomatic, ♂ + ♀ combined)	38	44	32	106	220

Notes: [a]One athlete did not finish this event. Two exclusionary criteria were used to screen articles in this table: (1) [Na^+] was not reported (or not reported for a specific female or male athlete), and (2) symptomatology and gender were not matched with [Na^+].
Abbreviations: S, symptomatic; A, asymptomatic; F, female; M, male; NR, not reported.

asymptomatic clinical presentation, and the number of female and male patients. Tables 1 and 2 describe the same cyclists, runners, swimmers, triathletes, and hikers.

Table 2 presents (a) two categories of symptoms, signs, and diagnoses: mild EAH and moderate-to-severe EAH; and (b) the number of times each clinical characteristic was reported. The dual classification of SAS as either mild EAH or moderate-to-severe EAH (Table 2, column 1) was created after articles were screened for inclusion and was based on four publications.[3,7,19,45]

Table 2 Number of Symptoms, Signs and Diagnoses Reported Among Cyclists, Runners, Swimmers, Triathletes, and Hikers Who Were Diagnosed with EAH

Symptoms, Signs, and Concurrent Diagnoses Associated with Mild or Moderate-to-Severe EAH	Athletes and Exercise Enthusiasts						
	Ultra-Endurance Mountain Bikers	Ultra-Endurance Road Cyclists	Marathon Runners (42.2 km)	Ultra-Endurance Runners (>50 km)	Ultra-Endurance Open Water Swimmers	Ironman Triathletes	Hikers, Trekkers
Associated with mild EAH[a]							
Diarrhea			1				6
Muscle pain, aches, or cramps	2			5			3
Body weight gain during exercise		2	1	5		3	
Extremity swelling, bloating, or puffiness	1		6	12		2	
Headache		1	7	4	1	1	8
Dizziness	1		10	3		2	7
Weakness or lethargy	1		15	7	1	1	11
Nausea	1		31	14	1	2	13
Associated with moderate-to-severe EAH[a]							
Chest tightness, shortness of breath, or dyspnea				2		1	
Fixed, dilated pupils			1			1	1
Aggressive or combative behavior			1	2			1
Ataxia		1					4
Slurred speech			1	4			1
Frothy sputum			9			1	
Coma or loss of consciousness			3	3		1	4
Collapse		1	6	2		1	2
Pulmonary edema			12	2		1	1
Agitation, restlessness			3	3	1	1	9
Encephalopathy		2	11	4	1		3
Seizure		1	3	6	1	4	12
Vomiting			35	11	1	1	18
Altered mental status (disorientation, confusion)	1	1	29	21	2	2	13
Total symptoms, signs, and diagnoses per column	7	9	185	110	9	25	117
Number of articles represented in each column	3	5	14	24	3	7	10

Notes: To be included, each clinical entity in column 1 had to be reported by ≥ 3 publications from Table 1, column 6. The following signs, symptoms, and diagnoses appeared in less than three of the articles in Table 1 and thus were not included in Table 2: decorticate posture (1), delirium (1), incoordination (1), lightheadedness (1), acute renal failure (2), cough (2), drowsiness (2), irritability (2), malaise (2). [a]The classification of SAS as either mild EAH or moderate-to-severe EAH (column 1) is based on aggregated diagnoses from four publications.[3,7,19,45]

The associations between [Na^+] and specific symptoms, signs, and concurrent diagnoses are shown in Table 3. In a few articles, the [Na^+], EAH SAS, or the sex of each athlete could not be matched with each other (ie, not reported); this resulted in some column totals or row totals to be different in Tables 1–3.

Among the field studies delineated in Tables 1–3, the average number of EAH cases was 3.9. Seventy-nine percent of these studies reported 1–4 EAH cases, and descriptive statistics (ie, height, body mass, exercise time to completion)

Table 3 The Associations of Blood Sodium Concentration with the Symptoms, Signs, and Concurrent Diagnoses of EAH Patients (Seven Sport Disciplines Combined)

Symptoms, Signs, and Diagnoses	Blood [Na^+] ($mmol \cdot L^{-1}$)				References
	<120	120 – 124	125 – 129	130 – 134	
Associated with mild EAH					
Diarrhea	4	2	1	0	[6,58,79,81]
Muscle pain, aches, or cramps	3	1	1	5	[35,46,47,59,69,79,84]
Body weight gain during exercise	4	0	3	4	[30,44,48,49,55,61,63,66,68,74]
Extremity swelling, bloating, or puffiness	1	0	3	16	[35,44,46,55,56,59,66]
Headache	5	7	5	5	[10,11,14,20,24,35,46,48,53,58,69,71,75,76,79,81–83,86]
Dizziness	9	6	2	6	[6,14,20,24,35,44,47,54,62,79,82,83]
Weakness or lethargy	8	15	6	7	[6,35,43,58,71,77,79,84,86]
Nausea	16	20	8	13	[6,10,14,17,20,24,43,47,55,56,58,61,69,72,76,77,79–81,86]
Associated with moderate-to-severe EAH					
Chest tightness, shortness of breath, or dyspnea	0	1	1	1	[46,59,75]
Fixed, dilated pupils	0	1	1	1	[11,20,76]
Aggressive or combative behavior	2	1	0	0	[6,62,82]
Ataxia	2	2	1	0	[49,79,84]
Slurred speech	5	1	0	0	[57,60–62,69]
Frothy sputum	2	6	2	0	[10,11,52]
Coma or loss of consciousness	5	5	1	0	[11,22,43,52,76,84]
Collapse	5	4	1	2	[10,11,20,22,43,50,53,62,68,74,79,84]
Pulmonary edema	4	9	3	0	[10,11,22,43,52,57,59,75,79,85]
Agitation, restlessness	9	7	1	0	[11,18,52,53,57,62,72,74,79–81,85]
Encephalopathy	8	7	4	2	[10,11,17,20,22,48,50,52,55,57,60,61,72,79,84,85]
Seizure	15	9	2	0	[6,17,24,50,53,59–61,72,74–77,79–82,84–86]
Vomiting	18	19	12	16	[6,10,11,14,17,20,24,53,55,56,58,68,69,72,77,79–82,85]
Altered mental status (disorientation, confusion)	20	24	13	10	[6,11,22,24,35,43,47,50,52,53,55–57,59–63,71,72,76,77,79–81,83,85,86]

predominated. Ninety-five percent of these field studies (Tables 1–3) performed no significance testing (eg, *t*-test or analysis of variance) of EAH patients versus athletes without EAH. Two figures allow an assessment of EAH symptomatology. First, Figure 2 offers a visual comparison of the most common SAS of EAH (≥10 reports minimum, females and males combined) observed in seven outdoor events. This figure visually summarizes the data in Table 2. SAS are depicted in two categories: mild and moderate-to-severe. Second, Figure 3 depicts the number of asymptomatic and symptomatic EAH cases reported (females and males combined, seven outdoor events combined). This figure visually summarizes the data in Table 1. The numeric values above each bar indicate the ratio of asymptomatic-to-symptomatic cases. Females comprised the following percentages of EAH cases (asymptomatic and symptomatic combined) in Figure 3: <120 $mmol \cdot L^{-1}$, 44.7%; 120–124 $mmol \cdot L^{-1}$, 61.4%; 125–129 $mmol \cdot L^{-1}$, 46.9%; 130–134 $mmol \cdot L^{-1}$, 36.8%; and total EAH cases (all [Na^+] categories, n = 220), 44.5%.

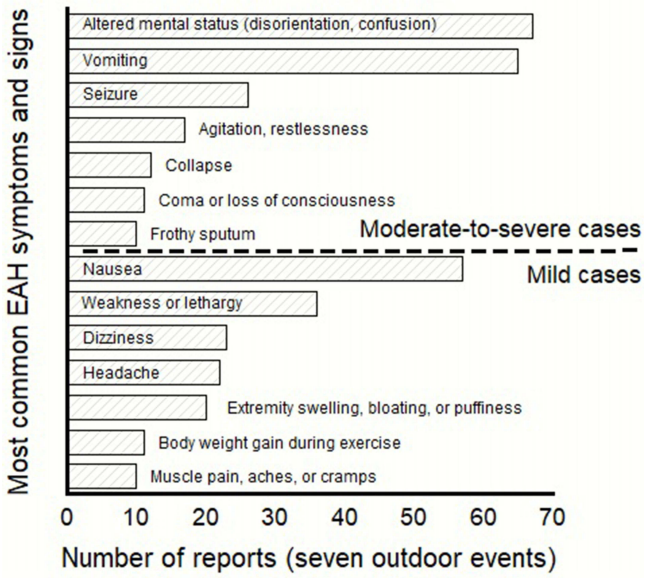

Figure 2 Most common SAS associated with EAH.

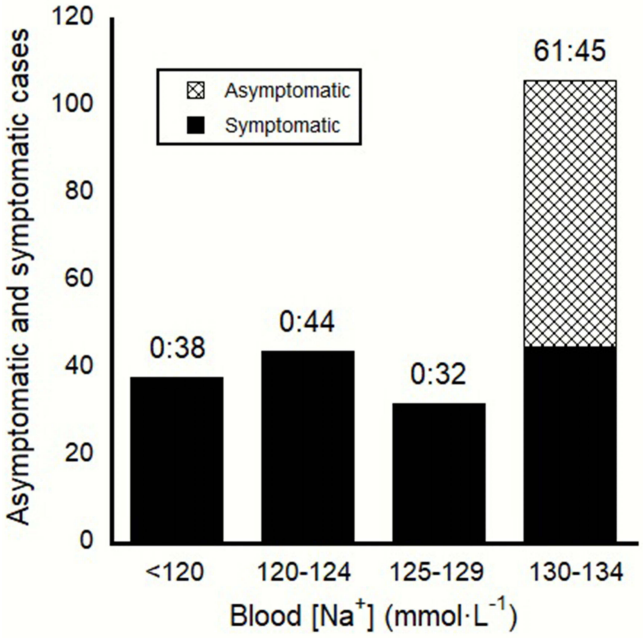

Figure 3 Number of asymptomatic and symptomatic EAH cases at different blood [Na^+].

Table 4 provides a compilation of 24 outdoor events at which no case of asymptomatic or symptomatic EAH was observed between the years 1970 and 2025. Although Table 1 demonstrates that EAH was diagnosed in 220 patients at 66 different outdoor events, Table 4 demonstrates that EAH is not diagnosed at all athletic competitions and mass participation events.

Table 5 presents a guide to six aspects of EAH (columns 3–8). These topics were published by professional sports medicine organizations and consensus writing groups (2006–2021). The information in these 11 articles provides readily available online information for clinicians and physiologists, as well as athletes and coaches.

Table 4 Twenty Four Outdoor Sport Competitions and Mass Participation Events Where No Case of Asymptomatic or Symptomatic EAH Was Observed (1970–2025)

Competitions, Events, Activities	Sports Medicine Publications in Which No Cases of EAH Were Observed	References
Ultraendurance mountain biking	2009, 2010, 2011	[87–89]
Ultraendurance road cycling	2012, 2015	[90,91]
Marathon (42.2 km) running	1970, 1978, 1984, 1989, 2025	[92–96]
Ultraendurance running	1976, 1991, 1994, 1999, 2003, 2007, 2010, 2011, 2025, 2025	[29,97–105]
Open water swimming	2010, 2015, 2023	[106–108]
Ironman Triathlon	2007	[109]

Table 5 Position Statements of Professional Sports Medicine Organizations and Consensus Documents of Expert Writing Groups That Describe Relevant Clinical Aspects of EAH

Professional Organization or Consensus Writing Group, Primary Topic (Year of Publication)	Outdoor Sports or Recreational Activities Which are Emphasized	Clinical Features of EAH Which are Described[a]						Sources
		Etiology, Pathogenesis	Assessment, Diagnosis	Treatment	Risk Factors	Prevention	Incidence	
International Marathon Medical Directors Association position statement, fluid intake recommendations (2006)	Cross country skiing, running, cycling, triathlons[b]	+	+		+	+		[110]
American College of Sports Medicine position stand, exercise and fluid replacement (2007)	Training and competition in ultraendurance sports, team sports, individual sports[b]	+	+		+	+		[111]
Expert panel recommendations, EAH treatment guidelines (2007)	Endurance events, EAH among hospitalized non-athletes	+	+	+	+	+		[112]
International Marathon Medical Directors Association advisory statement, EAH treatment with hypertonic saline (2009)	Marathon running	+	+	+	+		+	[113]
National Athletic Trainers Association position statement, sudden death in sports (2012)	Individual and team sports, running, American football	+	+	+		+	+	[2]
Wilderness Medical Society Practice Guidelines, treatment of EAH (2014)	Ultramarathons, hiking, triathlons, trekking, climbing	+	+	+	+	+	+	[40]
International Exercise-associated Hyponatremia Consensus Development Conference, EAH (2015)	Running, hiking, triathlons, rowing, swimming, American football	+	+	+	+	+	+	[19]
National Athletic Trainers Association position statement, fluid replacement for the physically active (2017)	High school, professional, collegiate and competitive sports; recreational activities[b]	+	+	+	+	+	+	[114]
Hyponatremia consensus writing group, EAH (2017)	Endurance events, team sports, hiking, canoeing, distance swimming, triathlons	+	+	+	+	+	+	[3]
Wilderness Medical Society Clinical Practice Guidelines, EAH management (2019)	Endurance events, ultramarathons, cycling, backpacking, hiking, climbing	+	+	+	+	+	+	[7]
Wilderness Medical Society practice guidelines, EAH brief summary (2021)	Back-country endurance activities	+	+	+		+		[45]

Notes: 1. All articles above consider (one or more of) running, cycling, swimming, hiking, or triathlon events. 2. The + symbol in columns 3–8 indicates that this clinical aspect of EAH is discussed, whereas open (blank) cells indicate that this aspect of EAH was not discussed.

Discussion

The mechanisms by which EAH develops, progresses, and either persists or is resolved are complex, and involve whole-body water and sodium imbalances.[3,115] Although multiple overlapping EAH etiologies have been identified,[3,15,115,116] their differences have impeded universal agreement regarding the mechanisms and symptoms involved.[3,7,15,117–119] Nevertheless, our extensive manual search of electronic databases (Figure 1) indicates that the following two concepts are widely accepted by clinicians and physiologists. First, an increased ratio of total body water volume relative to the amount of solute in the extracellular compartment (ie, total body exchangeable sodium) is the primary pathogenic cause of asymptomatic and symptomatic EAH.[6,28,31,41,81,112,116,120] This complex, dynamic clinical condition may result from excessive fluid intake (ie, exceeding the volume of sweat secreted), a large sodium loss in sweat, inappropriate mobilization of internal sodium stores, and/or inappropriately elevated blood arginine vasopressin concentration (ie, renal water retention which lowers $[Na^+]$).[7,19,112,116,121] Second, asymptomatic or mild cases of EAH can progress rapidly to severe and life-threatening encephalopathy. Thus, symptomatic EAH requires prompt recognition, initial resuscitation, and rapid treatment to minimize morbidity.[2,7,19,22,112] This necessitates that the on-site medical team be aware of common EAH SAS and recognize those which characterize both mild and severe cases. Unlike these two widely accepted concepts, our online search of electronic databases revealed important clinical issues regarding EAH that have not been adequately resolved or investigated.[115] In response, the following paragraphs examine five questions which are relevant to EAH diagnosis and the care of athletes and recreational enthusiasts in field settings.

Are Women at Greater Risk of Experiencing Asymptomatic and Symptomatic EAH Than Men?

Although numerous risk factors for EAH have been proposed,[7,26,30,115,122] it is important to acknowledge that the identification of risk factors implies a correlation with higher rates of EAH, but not necessarily causation or an independent association with EAH.[122] Regarding the influence of biological sex, behavioral characteristics, and physiological differences, multiple field research teams have reported that women are at greater risk of experiencing EAH than men,[6,10,24–27] whereas other investigators observed no effect of sex on EAH development in athletes.[28–34] The fact that the existing EAH literature consists primarily of studies with limited sample sizes (ie, case reports) impedes the clarification of differences between the sexes.[26] This current state of the existing literature is addressed in Table 1. The 220 EAH cases in this table were diagnosed at 66 different outdoor sport competitions, mass participation events, or recreational activities. These cases included 98 females and 122 males, initially suggesting that men have a greater risk of experiencing EAH than women. In terms of clinical assessment, women comprised 48.4% of all symptomatic cases and 34.4% of asymptomatic cases in Table 1. However, in the majority of endurance and ultraendurance events, the number of female competitors is less than male competitors. In 2013, for example, Chlibková et al[29] noted that the proportion of females among ultrarunners was 10–20%.[123] Subsequently, the worldwide participation trends of ultraendurance competitors were reviewed by Scheer.[124] He reported the following proportion of females among the total number of finishers, at various running distances: 50 km, 29.2%; 100 km, 15.3%; 100 mi, 19.1%; and 24-h races, 23.1%. Scheer also reported that women accounted for approximately 32% of all finishers in ultradistance open water swimming—a high percentage when compared to other endurance events. In addition, a field study of Ironman triathlon competitors published in 2023 by Johnson et al prospectively examined three decades of medical records (1989–2019; 53,355 total entrants).[26] They discovered that 9,482 athletes sought medical attention for a variety of medical complaints. The athletes who were diagnosed with EAH (n = 740) represented 29.7% of all women and 21.2% of all men who had serum $[Na^+]$ analyzed (n = 3,138). These data suggested that a greater percentage, not the absolute number, of women experienced EAH than men.[26,28] Unfortunately, because few of the articles in Table 1 reported the total number of male and female race participants, we recommend that future field studies examine this matter.

The column totals shown in the bottom rows of Table 1 demonstrate that, when all levels of $[Na^+]$ are considered, symptomatic EAH was diagnosed in 77 women and 82 men, whereas asymptomatic EAH was experienced by 21 women and 40 men. Clearly, the majority of cases in Table 1 involve symptomatic EAH, for both men and women. This finding (a) agrees with the previously published guidelines of the Wilderness Medical Society,[40] and (b) likely occurs because

athletes with asymptomatic EAH typically present with nonspecific and transient complaints which are commonly experienced by others who do not seek medical care.[18–21] However, this finding opposes an observation published by an expert consensus development team.[19] That group concluded that symptomatic EAH is rare and occurs with considerably less frequency than asymptomatic EAH. We interpret this disparity to be the result of an analysis that involved a smaller sample of EAH cases (ie, often from a single sport discipline) than the 220 EAH cases described in Table 1.

To clarify the aforementioned uncertainties, a more comprehensive understanding of the influence of biological sex can be derived from a longitudinal study design versus a cross-sectional design (ie, which is utilized in most EAH studies). The former would involve making several observations of the same athletes across several years. The latter would involve comparing different groups of athletes at a single point in time such as an outdoor endurance event. Specifically, we recommend prospective cohort studies as an alternative to retrospective case reports, as a means of overcoming biases in subsequent research. These prospective cohort studies would involve collecting data over time to compare the occurrence of EAH in women and men. However, we acknowledge that prospective cohort studies (a) require access to a large pool of research subjects who agree to be followed longitudinally (ie, across many endurance events), (b) can be very costly/time consuming, and (c) may result in bias due to test subject attrition.

Are EAH Signs, Symptoms, and [Na^+] Similar During Outdoor Running, Cycling, Swimming, Triathlon, and Hiking Activities?

Our manual search of electronic databases (Figure 1) determined that three epidemiological studies and one narrative review previously considered some aspects of this question, but the scope of those articles was limited. In total, these three field studies[35,36,38] described 28 EAH cases that occurred during competitive ultra running, road cycling, ultra mountain biking, and ultra swimming. The single narrative review article focused on EAH prevalence during swimming, cycling, running, triathlons, and multi-day stage events but did not compare similarities and differences of signs, symptoms, or [Na^+] among these sports. In contrast, Tables 1 and 2 offer a comprehensive view of symptomatic EAH, asymptomatic EAH, [Na^+], SAS, and concurrent diagnoses that were observed during seven different outdoor events. This comprehensive approach has value because 91% of the previously published articles in Table 1 involved medical encounters during a single sport or recreational activity.

Due to the distinctive fluid-electrolyte perturbations (ie, deficits or excesses) and stressors (ie, venue, terrain, ambient temperature) which athletes experience in various sports, it is possible that EAH SAS and the [Na^+] in blood are unique to some or all athletic disciplines. If true, event specificity could account for much of the wide range of EAH SAS or prevalences reported among sport and recreational activities.[3,7,28,37] Indeed, Tables 1 and 2 provide evidence for event specificity in four ways. First, all 33 cases of EAH in triathletes, and all 25 cases of EAH in hikers/trekkers, were symptomatic (Table 1). Of the remaining five sports, none exhibited a similar symptomatic-to-asymptomatic ratio. Second, the number of EAH cases (columns 2–5) and publications (column 6) in Table 1 was greatest in marathons and ultramarathons. This likely was influenced by the large number of participants and running events worldwide. Third, few cases of EAH in Table 1 occurred among mountain bikers and road cyclists. This observation supports a previous review of cyclists[37] in which a low prevalence of EAH was reported in 10 field studies. Those authors and others[121,125] have observed that cycling is unique in that, unlike runners, cyclists carry nutrition on their bicycle frame and in their jersey pockets, thereby modulating their individual fluid needs. Supporting the concept of event specificity in a parallel clinical domain, the incidence of hyperthermia and exertional heatstroke are considerably lower in cyclists and open water swimmers than among endurance runners and triathletes,[126] due to differences of heat dissipation from skin to air or water. Fourth, no case of mild EAH (ie, [Na^+] of 130–134 $mmol \cdot L^{-1}$) in Table 1 occurred among hikers and trekkers. We interpret this to be an example of individuals who experienced nonspecific and transient complaints which are commonly experienced by others who do not seek medical care after endurance exercise. As a result, their [Na^+] was not analyzed and their asymptomatic EAH went unrecognized.

The following corollary question provides additional relevant information: at which outdoor athletic and recreational activities have no cases of EAH been observed? Table 1 summarizes 220 EAH patient diagnoses at 66 different outdoor

events, between the years 1981–2018. However, Table 4 establishes that EAH was not reported at 24 outdoor events which spanned more than a half century (1970–2025). These competitive and mass participation events included mountain biking, road cycling, open water swimming, Ironman triathlons, marathon and ultraendurance running.

What is the Relationship Between Serum [Na^+] and the SAS of EAH?

The reduced extracellular [Na^+] and total osmolality that is characteristic of EAH may result in an abnormally large shift of water into the confined space of the skull. In advanced cases, this can develop into hyponatremic encephalopathy, herniation, depression of the respiratory center, or death.[7,19,20] However, previously published clinical studies have not clarified the SAS of EAH at different levels of [Na^+], likely because multiple EAH mechanisms have been identified[116] and because idiosyncratic etiologies have been reported among endurance athletes with similar post-exercise [Na^+].[15] Further complicating this complexity, the number of EAH cases at any single event typically is small (Table 1), making it difficult to statistically evaluate the relationship between serum [Na^+] and EAH SAS. As evidence of this, Table 1 shows that (a) an average of 3.3 EAH cases were diagnosed at each of the 66 outdoor sport competitions, mass participation events, or recreational activities; and (b) more than half (53.0%) of the studies in column 6 reported only 1 case of EAH.

Figure 3 depicts 220 asymptomatic and symptomatic EAH cases (representing the seven endurance activities in Table 1), at different levels of [Na^+]. This figure demonstrates that 61 asymptomatic patients had a [Na^+] of 130–134 $mmol \cdot L^{-1}$, and that 45 symptomatic patients also were within this range. The fact that no asymptomatic EAH case, in seven different activities, involved a verified [Na^+] of <130 $mmol \cdot L^{-1}$ suggests that this concentration represents the lower boundary of asymptomatic cases. This observation supports the 2014 Wilderness Medical Society practice guidelines for the treatment of EAH[40] and two narrative review articles,[27,127] which indicated that patients with a [Na^+] ≥130 $mmol \cdot L^{-1}$ generally are either minimally symptomatic or asymptomatic. In fact, the information in Figure 3 closely resembles a figure published by Speedy et al in 1999,[127] depicting 40 hyponatremic ultradistance triathletes.

Two additional considerations are relevant. First, the aforementioned Wilderness Medical Society guidelines[40] state that symptomatic EAH (ie, a biochemical analysis of [Na^+] combined with clinical SAS) has a much lower incidence than asymptomatic EAH.[40] Combining seven different outdoor activities, our Figure 3 refutes this conclusion in that the number of symptomatic EAH cases is 2.6 times greater than the number of asymptomatic cases (159:61 ratio). Second, the number of symptomatic cases in Figure 3 did not differ greatly across the four [Na^+] categories depicted. This finding supports multiple authorities[19,45,55,128,129] who concluded that the absolute value of [Na^+] is not a reliable predictive index of EAH clinical severity.

Table 3 suggests that only a portion of EAH SAS is observed more frequently as [Na+] progressively decreases. Our visual inspection of the right-to-left trends in columns 2–5 of Table 3 (ie, representing decreasing [Na^+]) suggests that the following EAH SAS (ie, total number of reports in parentheses) trend toward supporting this paradigm: diarrhea (7), aggressive or combative behavior (3), ataxia (5), slurred speech (6), coma or loss of consciousness (11), collapse (12), agitation or restlessness (17), seizure (26), and altered mental status (67). Regarding mild EAH, 87.5% (7/8) of the SAS (upper eight rows of Table 3) were not observed more frequently as [Na+] progressively decreased. Regarding moderate-to-severe EAH (lower 14 rows of Table 3), 57.1% (8/14) of the SAS increased when [Na^+] progressively decreased; all of these were associated with cognitive function and central nervous system control of bodily functions. Therefore, we recommend that medical teams covering outdoor endurance activities employ selected SAS from Table 3 to create an a priori medical staff plan that optimizes field diagnosis and treatment of EAH. This plan should consider (a) the sport discipline involved (Table 2) and event specificity (described above), plus (b) a review of athlete medical records (ie, common SAS associated with EAH) from previous years.

Do Specific SAS Distinguish Mild EAH from Moderate-to-Severe EAH?

The SAS associated with EAH depend on both the magnitude of the serum sodium decrease as well as the rate at which this decrease occurs,[19,40,128,129] because both phenomena alter intracellular-extracellular homeostasis.[130–133] The complex interactions of these two deviations (ie, magnitude and rate of change), plus inter-individual differences among athletes and sport-specific stressors, make it difficult to predict the clinical severity of EAH solely on the basis of

[Na^+].[19,45,55,127,128] As a result, clinicians often evaluate neurological SAS when considering a diagnosis of encephalopathy or severe EAH.[10,22,24,40,60,134] Therefore, in the present review, we have aggregated SAS from four harmonized sources[3,7,19,45] to distinguish "moderate-to-severe EAH" from "mild EAH" cases. Figure 2, Tables 2, and 3 follow this convention and provide novel information to assist the interpretation of EAH severity. Regarding these data, we present three clarifications. First, the classification of SAS as "mild" and "moderate-to-severe" represents a clinical paradigm. These two classes of SAS are not mutually exclusive; they may exist concurrently. Second, it is likely that the order/timing of SAS appearance will vary from one patient to another. This means that some moderate-to-severe SAS may appear before mild SAS. Third, the observations of SAS in Figure 2, Tables 2, and 3 were not guided by the same clinical survey or SAS checklist. This suggests that (a) considerable diagnostic variability existed among event medical teams, and (b) recognition of SAS relied on the various past encounters of medical team members with EAH patients. Thus, we recommend that EAH be considered as a differential diagnosis when common EAH SAS are recognized.

Figure 2 allows evaluation of the most common EAH SAS (women and men combined) from Table 2 that were observed at seven different outdoor events. The mild EAH cases (lower half of Figure 2) often involved nonspecific and transient complaints which are commonly experienced by others who do not seek medical care following strenuous endurance exercise (eg, fatigue, presyncope, or dizziness).[18–21] The most common "mild EAH" complaints in Figure 2 were nausea, weakness or lethargy, dizziness, and headache. The most common SAS of "moderate-to-severe EAH" included altered mental status, vomiting, seizure, agitation/restlessness, collapse, and loss of consciousness. Because these neurological SAS suggest possible hyponatremic encephalopathy,[7,19,20] we recommend that future field studies focus on the interactive associations among observed SAS (Figure 2) and long-term outcomes, especially for athletes who exhibit severe SAS (Tables 2 and 3). In future longitudinal studies (ie, involving months and years of observations) it will be important to recognize that neurologic impairment, seizures, or vomiting raise the index of suspicion for moderate-to-severe EAH.[3,6,7,17,19,21]

Table 2 is structured so that the SAS associated with both mild and moderate-to-severe EAH can be compared across each of the seven outdoor events. The column totals (bottom two rows) in Table 2 indicate that upright exercise resulted in far more EAH cases and published epidemiological studies than cycling, swimming, and triathlon events. Specifically, marathons, ultramarathons, and hiking (ie, all weight-bearing activities) accounted for 89.2% (412/462) of all EAH cases in Table 2. To our knowledge, no previous publication has recognized or provided a hypothetical explanation for this phenomenon. Thus, we propose that the association between EAH prevalence and upright exercise warrants future epidemiological research to determine the underlying mechanisms and influential factors. Considering non-weight-bearing exercise, the fewest diagnoses (ie, and the fewest publications) of EAH occurred among mountain bikers, road cyclists, and open water swimmers. This observation supports a previous review involving cyclists,[37] in which a low prevalence of EAH was reported in 10 field studies. Our manual search of online databases discovered no comparable review of ultraendurance open water swimming.

Which Organizational Position Statements and Expert Consensus Reports Provide Readily Available Information About EAH?

Table 5 presents a guide to various aspects of EAH (columns 3–8) that are available in four position statements prepared by professional sports medicine organizations, and in seven expert reports produced by consensus writing groups (2006–2021). This table serves as a resource for physicians, physician assistants, and nurses who require detailed information regarding the etiology, diagnosis, and treatment of EAH, as indicated in columns 3–5. Athletes and coaches may seek information regarding EAH risk factors and the prevention of EAH, as summarized in columns 6 and 7. By consulting the articles in Table 5, readers will discover that clinicians at outdoor endurance events must differentiate EAH from other causes that present with similar SAS (ie, dehydration, heat exhaustion, exertional heatstroke).[4–9] This is vital because appropriate treatment varies among these disorders and because asymptomatic or mild cases of EAH can progress rapidly to severe and life-threatening encephalopathy.[7,10–13] Also, athletes and coaches can benefit from the documents in Table 5 by reviewing (a) the expert recommendations regarding fluid intake during endurance exercise, and

(b) preventive strategies (eg, pre-race education, body weight scales at check points, nutrition and fluid types at aid stations). These guidelines often are tailored to meet the unique characteristics of specific sports.

Limitations

The findings of the present review may be subject to the methodological limitations of the 56 field research studies that were selected for inclusion (Figure 1). We acknowledge the following potential limitations. First, no case of mild EAH (ie, serum [Na^+] of 130–134 mmol·L^{-1}) in Table 1 occurred among hikers and trekkers. This may have resulted from their nonspecific and transient complaints, which are commonly experienced by others who do not seek medical care after endurance exercise. As a result, their serum [Na+] may not have been measured, and their asymptomatic EAH may have gone unrecognized. The influence of this factor on the other six sport disciplines in Table 1 is unknown. Second, due to the vast distances between the course starting line and finish line in numerous point-to-point ultraendurance events, those field studies did not report the data of individuals who dropped out along the course or bypassed patient care at event medical facilities. Third, few of the field studies in Table 1 reported the EAH cases of athletes who became ill up to 24 h after leaving the event venue. As a result, the number of EAH cases may be greater than reported, at numerous outdoor endurance events. Fourth, the number of EAH cases in Figure 3 and Tables 2–4 may have been biased by either unpublished EAH cases, or the recognition of a high prevalence of EAH cases, in specific sport disciplines. For example, the medical teams covering mountain biking, road cycling, and open water swimming events may have been less inclined to publish EAH field studies, when compared to the medical teams covering marathon and ultramarathon events. Fifth, preventive strategies (eg, pre-race education, body weight scales at check points, nutrition and fluid types at aid stations) often vary and may have contributed to the varying incidence among outdoor events. Sixth, we did not examine actual case outcomes or predicted outcomes (eg, based on SAS or presentation severity) because the requisite information was not described. Therefore, we recommend that future epidemiological studies act to counteract these potential limitations by (a) discriminating nonspecific and transient complaints from neurological SAS that suggest a diagnosis of EAH, (b) utilizing a standardized SAS survey or checklist that is specific to the sport discipline involved, (c) providing on-site instrumentation that measures serum [Na^+] when EAH is suspected, (d) identifying competitors who drop out or bypass patient care, and (e) publishing the relevant medical information of all EAH diagnoses. Seventh, although a considerable number of risk factors for EAH have been proposed,[7,26,30,115,122] the identification of additional risk factors could benefit athletes, medical teams, and race directors at outdoor events. These factors could include hydration practices (eg, individualized planned drinking), environmental conditions, and athlete characteristics (eg, age, fitness level, body mass index). We recommend that future field studies evaluate these and other potential risk factors.

Conclusion

EAH symptom recognition is challenging due to the interactions of multiple complex factors, especially the dynamic relationship between serum [Na^+] and symptomatology. Ambiguities and unresolved clinical issues also exist regarding athlete sex as a risk factor, undefined inter-individual differences among athletes, and event-specific stressors. Our review of previously published EAH epidemiological studies prompted us to focus on five clinically relevant questions. Our responses to these questions are unique because they represent 220 EAH patients at 66 different outdoor events, whereas most previous EAH reports involved a small number of athletes who participated in a single outdoor event. Because these patients participated in seven different endurance activities, the present review provides a unique, wide-ranging overview of EAH. It is therefore relevant that severe EAH, verified by a [Na^+] <120 mmol·L^{-1}, occurred in all of the seven sport and recreational activities. We propose that the information provided in this review will inform future pre-event medical planning, on-site medical staff briefings, as well as the diagnosis and treatment of EAH in field settings. We also propose that our compilation of readily available position statements and consensus documents will inform athletes and coaches who seek detailed information regarding EAH risk factors and the prevention of EAH.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising, or critically

reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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