Statement of the Second International Exercise-Associated Hyponatremia Consensus Development Conference, New Zealand, 2007

Tamara Hew-Butler, DPM, PhD, J. Carlos Ayus, MD,† Courtney Kipps, BMBS, MSc,‡ Ronald J. Maughan, PhD,§ Samuel Mettler, MSc,¶ Willem H. Meeuwisse, MD, PhD (chair),‖ Anthony J. Page, MBChB, MD,** Stephen A. Reid, MBBS, PhD,†† Nancy J. Rehrer, PhD,‡‡ William O. Roberts, MD, MSc,§§ Ian R. Rogers, MBBS,¶¶ Mitchell H. Rosner, MD,‖‖ Arthur J. Siegel, MD,*** Dale B. Speedy, MBChB, MD,††† Kristin J. Stuempfle, PhD,‡‡‡ Joseph G. Verbalis, MD,§§§ Louise B. Weschler, MAT, PT,¶¶¶ and Paul Wharam, MMedSc‖‖‖‖*

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INTRODUCTION

The Second International Exercise-Associated Hyponatremia (EAH) Consensus Development Conference convened in Queenstown, New Zealand (November 2007) with a panel of eighteen international experts. This conference utilized the United States National Institute of Health (NIH) Consensus Conference protocol and was deliberately free from commercial sponsorship. The delegates represented seven countries and eight medical and scientific sub-specialties pertaining to exercise physiology, sports medicine, water metabolism, and body fluid homeostasis. The primary goal of the panel was to review the existing data on EAH and update the 2005 Consensus Statement.[1] This document serves to replace the 1st International EAH Consensus Development Conference Statement.

The purpose of this update is to summarize the most current information on the prevalence, nature and treatment of EAH for medical personnel, athletes, and the greater public and to curtail the morbidity and mortality associated with the disorder. The panel strived to clearly articulate what we agreed upon, debate issues that we did not agree upon, and describe in detail what we did and did not know, including minority viewpoints that were supported by clinical and experimental data.

The following statement is a concise summary of the data synthesized by the 2007 EAH Consensus Panel and represents an evolution of the most current knowledge on EAH. Further updates will be required as the understanding of EAH advances.

METHODS

The International Exercise-Associated Hyponatremia Consensus Development Conference followed the guidelines set forth by the National Institutes of Health (NIH).[2] The basic principles governing the conduct of a consensus development conference are summarized below:

1. A broad based non-government, non-advocacy panel was assembled to give balanced, objective and knowledgeable attention to the topic. Panel members included researchers in endocrinology (JGV), epidemiology (WHM), nephrology (JCA, MHR), emergency medicine (IRR), family medicine (WOR), internal medicine (AJS), sports medicine (CK, AJP, SAR, DBS, PW) and exercise physiology (TH, RJM, SM, NJR, KJS, LBW).
2. These experts presented data in a public session, followed by inquiry and discussion. The panel then met in a closed session to prepare the consensus statement.
3. A number of specific questions were prepared and posed in advance to define the scope and guide the direction of the conference. The principle task of the panel was to elucidate responses to these questions and incorporate these answers in the updated document.
4. A systematic literature review was prepared and circulated in advance for use by the panel in addressing the conference questions.
5. The consensus statement is intended to serve as the scientific record of the conference.
6. The consensus statement will be widely disseminated to achieve maximum impact on both current health care practice and future medical research.

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From the *University of Cape Town, Cape Town, South Africa; †Renal Consultants of Houston, Houston, Texas, USA; ‡Centre for Sports and Exercise Medicine, Queen Mary, University of London, London, United Kingdom; §Loughborough University, Leicestershire, United Kingdom; ¶ETH Zurich, Department of Agricultural and Food Sciences, Zurich, Switzerland; ‖University of Calgary, Calgary, Alberta, Canada; **Sport and Exercise Medicine Specialists, Christchurch, New Zealand; ††Sports Medicine Practice, Tasmania, Australia; ‡‡University of Otago, Dunedin, New Zealand; §§University of Minnesota Medical School, St. Paul, Minnesota, USA; ¶¶University of Western Australia, Nedlands, Australia; ‖‖University of Virginia, Charlottesville, Virgina, USA; ***Harvard Medical School, Boston, Massachusetts, USA; †††University of Auckland, Auckland, New Zealand; ‡‡‡Gettysburg College, Gettysburg, Pennsylvania, USA; §§§Georgetown University Medical Center, Washington, DC, USA; ¶¶¶Independent Researcher, New Jersey, USA; ‖‖‖‖Sports Physician, Auckland, New Zealand.

Correspondence: Tamara Hew-Butler, DPM, PhD, Systemic Inflammation Laboratory, Trauma Research, St. Joseph's Hospital and Medical Center, 350 W. Thomas Road, Phoenix, AZ 85013 (e-mail: tamara.hew@chw.edu).

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The panel chairperson (WHM) did not identify with any advocacy position or present data on EAH.[2] The chairperson was responsible for directing the plenary session and guiding the panel's deliberations.

The strict criterion for invitation to the EAH Consensus Panel in 2005[1] was waived for the 2007 Conference, as an independent decision by the conference organizer (TH) to encourage a wider range of expertise. An open call for abstracts was placed in the July 2007 issue of the Clinical Journal of Sport Medicine and formal invitations were sent to clinicians and scientists who had published papers pertaining to EAH. This necessary deviation from the NIH guidelines served to enhance the scope of our deliberations and include researchers (WOR, CK, SM) who had data, yet unpublished, that might significantly contribute to our existing knowledge. Hence, the "no exclusion" criteria served to ameliorate previous concerns raised by those who felt that the previous "inclusion" criteria were too stringent to allow for a well-balanced consensus. Several invitees declined the invitation to participate.

The following focus questions were identified by the panel to be of significant priority for the 2007 closed panel deliberations. The answer to these updated focus questions formed the basis for the discussion and were incorporated into the revised 2007 EAH Consensus Statement:

1. What is the role of sodium supplementation in the prevention of EAH?
2. What is the evidence supporting sodium loss in EAH?
3. What are the potential benefits and dangers of excess sodium supplementation before, during and after exercise?
4. Should on-site rapid sodium measurement be required for all marathons?
5. What are the current evidenced-based options for treating EAH in the field and in the hospital settings (ie, hypertonic saline, urea, mannitol, arginine vasopressin (AVP) receptor antagonists, and fluid restriction)?
6. What is the role of concomitant medication use in the pathogenesis of EAH (ie, selective serotonin reuptake inhibitors or non-steroidal anti-inflammatory medications)?
7. Does EAH only occur in the presence of the syndrome of inappropriate anti-diuretic hormone secretion (SIADH), and what is the role of non-osmotic AVP secretion in the development of dilutional EAH during exercise?
8. What role do ambient temperature, training and/or gender play in the incidence, evolution and treatment of EAH?
9. Is full replacement of body weight losses a physiologically appropriate prevention strategy during prolonged endurance exercise?
10. How can we best disseminate educational information to reduce the increasing incidence and morbidity of exercise-associated hyponatremia?

RESULTS AND DISCUSSION

Definition of Exercise Associated Hyponatremia (EAH)

EAH is the occurrence of hyponatremia during or up to 24 hours after prolonged physical activity[3–38] and is defined by a serum or plasma sodium concentration ($[Na^+]$) below the normal reference range of the laboratory performing the test. For most laboratories, this is a $[Na^+]$ less than 135 mmol/L.[39]

Classification of EAH by Clinical and Laboratory Criteria

EAH should be classified using the same clinical criteria as any acute or rapid onset hyponatremia.[40] In any acute onset hyponatremia, the most important factor is determining the presence or absence of clinical signs and symptoms: specifically neurological manifestations. In general, the lower the $[Na^+]$ the more severe the neurological signs and symptoms[41] However, individual variability is great and the numerical value of $[Na^+]$ is not a reliable predictive index of the clinical severity of hyponatremia, including EAH.[40,42]

Early signs and symptoms of EAH can include: bloating, "puffiness", nausea, vomiting, and headache.[4,5,7,11,14,21,34,43] However, many of these signs and symptoms are non-specific and can be present following prolonged exercise in the absence of EAH.[44] As the severity of EAH progresses, more serious signs and symptoms of hyponatremic encephalopathy can develop as a result of cerebral edema (brain swelling), including alteration of mental status (eg, confusion, disorientation, agitation, delirium), seizures, respiratory distress (pulmonary edema), obtundation, coma and death.[6,7,9–11,17,18,21,31,33,34,43,45–49] The presence of any of these signs and symptoms represents an absolute indication to measure $[Na^+]$.

Signs and symptoms of EAH can develop when the $[Na^+]$ falls below 135 mmol/L.[14,15,22,25,28,30] However, in general, hyponatremia ($[Na^+]$ between 130–134 mmol/L) is relatively asymptomatic,[3,15,22,25,26,28,30,32,50] although exceptions have been reported.[42] Symptomatic hyponatremia can occur if the rate of fall of Na approaches a 7–10% decrease within a 24 hour period.[51] For example, a pre-exercise $[Na^+]$ value of 142 mmol/L - that decreases to a post-exercise $[Na^+]$ value of 132 mmol/L - can be associated with significant morbidity from the increased osmotic pressure gradient of change even though the absolute $[Na^+]$ may appear relatively benign.

Etiology and Pathophysiology of EAH

EAH is predominantly a dilutional hyponatremia caused by an increase in total body water relative to the amount of total body exchangeable $[Na^+]$.[3–6,10–13,13,14,17,19–21,26,34,43,46,48,52–54] The primary etiologic factor in cases that have been adequately studied appears to be consumption of fluids (water or sports drinks) in excess of total body fluid losses: insensible (transcutaneous, respiratory and gastrointestinal),[55,56] sweat and renal (urine) fluid losses.[3–6,10–13,13,14,17,19–21,26,34,43,48]

In most reported cases of symptomatic EAH, there is body weight gain suggestive of an absolute increase in total body water.[3,4,6,8,13,19,20,24,26,28,43,48,50,53,54,57] However, it should be recognized that some loss of body weight is expected with prolonged physical activity due to substrate oxidation without a net loss of total body water.[56,58,59] For example, during a typical 42 km marathon a 1–2% decrease in body weight typically occurs without a change in total body water as inferred by the maintenance of plasma osmolality from pre to

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post race.[60] A dilutional hyponatremia may therefore occur despite no change or even a fall in body weight.[53,61]

A positive fluid balance during exercise can occur because of overconsumption of fluid and/or impaired renal water clearance. Hyponatremia caused solely by the overconsumption of fluids has been demonstrated at rest in athletes with and without a history of EAH.[13,57,62] Weight gain in these athletes occurred despite an increase in free water excretion and what appeared to be maximally suppressed AVP levels (as reflected by mean urine osmolalities <100 mOsm/kg H_2O). This is consistent with known maximal urine excretory rates of 800–1,000 ml/h in normal adults under resting conditions.[63] Thus, in these cases water intake simply overwhelms the renal water excretion rate.

Although some cases of EAH may be due to pure water intoxication from overconsumption of fluids, recent data indicate that AVP secretion is an exacerbating factor in most cases. During exercise, plasma AVP levels are not maximally suppressed implicating non-osmotically stimulated AVP secretion.[5,13,49] In the presence of hyponatremia and/or hypervolemia, plasma AVP levels within "normal ranges" are physiologically *inappropriate* because AVP should be maximally suppressed under these two conditions. This pathophysiology characterizes the syndrome of inappropriate anti-diuretic hormone secretion (SIADH).[26–28,49] Small increases in circulating AVP markedly reduce maximal kidney excretory capacity,[40] thus increasing the propensity to retain ingested fluids even if rates of drinking do not exceed 800–1,000 mL/h. Thus, the risk of developing fluid overload with previously "normal" or excessive fluid intakes is enhanced when AVP is secreted inappropriately during prolonged exercise, resulting in increased urine osmolality and decreased urine volume. High urine osmolalities have been measured in athletes hospitalized with critical hyponatremia, and inappropriate plasma AVP levels have been documented in EAH.[5,6,12,19,34,64] A lower rate of urine production correlates significantly with a higher rate of $[Na^+]$ decrease in athletes drinking excessively during exercise.[65] The higher the plasma AVP level, the more reduced will be the maximum urine excretory capacity. This effect can potentially account for the marked inter-individual variability in the development of EAH, as well as individual variability in outcome across different events, despite similar fluid intakes. Multiple potential stimuli to AVP secretion, such as nausea/vomiting, hypoglycemia, hypotension or hypovolemia[40] can exacerbate fluid retention at any time during prolonged exercise.[4,66] Given the short half life (6–8 minutes)[66] of AVP, measurement of suppressed AVP levels at time points after the cessation of physical activity do not eliminate the possibility of inappropriate AVP secretion as a contributory factor to the development of EAH.

Excessive $[Na^+]$ loss has not been demonstrated to be a primary causative factor in the pathogenesis of EAH. Sodium loss is no greater in individuals who develop EAH than in individuals who do not.[4,17,27,28] However, there may be several potential mechanisms whereby sodium losses play a role in the pathogenesis of EAH: 1) hypovolemia produced by sodium losses can act as a stimulus to AVP secretion, producing a secondary retention of water, as is seen medically in some cases of diuretic-induced hyponatremia[67] and/or 2) sodium losses themselves can worsen the degree of hyponatremia, although in most cases not nearly as much as water retention[68] when summed up over time. While mathematical models suggest that EAH may occur from excessive sodium depletion during ultraendurance exercise,[69] this phenomenon has not been documented in any laboratory or field investigation to date. Therefore, further (direct measurement) studies are necessary to fully investigate the role of sodium losses on this small cohort of athletes who develop EAH, particularly those with a weight loss in excess of 3% or with a large volume of sweat over time, in warmer climates and in events lasting over 12 hours.

Risk Factors

The presence of a risk factor implies a correlation with higher rates of EAH, but not necessarily causation. It is likely that these risk factors interact with each other and, in some cases, may not have an independent association with EAH. Recognized risk factors include:

- Athlete-related
  - excessive drinking behavior[3,7,10,14,19,23]
  - weight gain during exercise[3,4,6,8,13,19,20,24,26,28,43,48,50,53,54,57]
  - low body weight[3,27,30,35]
  - female sex[3,5,7,14,26,30,45]
  - slow running or performance pace[3,7,14,19]
  - event inexperience[14,33,38]
  - nonsteroidal anti-inflammatory agents[6,7,31,45,54,70–72]
- Event-related
  - high availability of drinking fluids[14,29,54]
  - >4 hours exercise duration[3,4,6,8–10,13–15,17–20,22,24–32,48,73]
  - unusually hot environmental conditions[4,5,9,10,12,16,23,37,38,74]
  - extreme cold temperature[30]

There are known medical risk factors for the development of hyponatremia in the general population that also may play a role in EAH. These include altered renal water excretory capacity potentially impaired by drugs (eg, selective serotonin re-uptake inhibitors, thiazide diuretics, etc.),[75] intrinsic renal disease,[76] low solute diets,[77–79] or SIADH.[80] Published data on the cystic fibrosis genotype (either homozygous or heterozygous) is inconclusive.[81–84,85,86] Further studies will need to be made before excluding these variables as potential risk factors for EAH.

Prevention

EAH is caused primarily by the consumption of fluid in excess of urinary and sweat losses. Therefore, it follows that any individual participating in endurance exercise, and particularly those at increased risk for EAH, should avoid over consumption of fluids before, during and after exercise.[29,87,88] Blanket universal guidelines are neither possible nor appropriate because there is a wide variability in sweat rates[11,89] and renal water excretory capacity[65,90] during exercise, both among individuals and in the same individual depending on ambient environmental conditions during the time of exercise.

The primary means of preventing EAH is to avoid excess fluid retention, as manifested by weight gain, during or after exercise. It should be recognized that even maintenance of body weight has been associated with the development of

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EAH.[61] Therefore the goal should be to expect to lose up to two percent of body weight and never to gain weight during exercise.[56,58] There are at least two ways to reduce the risk of excess fluid retention: 1) drink only according to thirst (ie, *ad libitum*)[44,60] and 2) monitor body weight so as to avoid weight gain during exercise.[91] Moreover, athletes should be encouraged to weigh themselves pre- and post-training in a variety of conditions to estimate their fluid requirements during competition. Athletes should recognize the potential limitations of this method, however. In situations where AVP is not appropriately suppressed, a drinking schedule that worked well in identical training conditions may lead to weight gain and EAH in competition.

Ingestion of electrolyte-containing sports drinks cannot prevent the development of EAH in athletes who drink to excess.[3,5,16,32,68,92] This is due to two factors: 1) all such drinks have $[Na^+] < 135$ mmol/L and therefore will cause dilution of $[Na^+]$ if excess water is retained in the body during exercise and 2) it is well known that even administration of isotonic saline will not increase $[Na^+]$ in hyponatremic patients with SIADH because in a euvolemic or hypervolemic state the infused sodium will be excreted in the urine rather than retained.[80]

There is conflicting evidence as to the effect of sodium supplementation, either by tablet or drink, on the incidence of EAH and the rate of change of $[Na^+]$ with exercise.[32,65,90,93,94] However, some risks of excessive sodium supplementation in combination with overhydration have been documented.[95–97]

An education program advising athletes on the risks of overdrinking together with limiting fluid availability at a race are factors associated with a reduction in the incidence of EAH without deleterious effects.[29,88,98] Specifically, for an Ironman distance triathlon, cycle aid station placement every 20 km, and run stations every 2.5 km are recommended.[29] In a standard marathon footrace, placement of aid stations every 5 km is associated with an absence of EAH.[87] However, dissemination of appropriate drinking advice alone has proven to minimize the incidence of EAH in a 90 km footrace despite fluid stations placed <1.6 km apart.[44,99]

Treatment Protocols for EAH

Medical facilities at endurance events should include onsite analysis of $[Na^+]$.[100] Any athlete exhibiting signs or symptoms of acute hyponatremia listed above should be screened for EAH by measuring $[Na^+]$. Based on this determination, the following treatment protocols are advised:

Asymptomatic EAH

Asymptomatic hyponatremia is not normally detected unless an athlete has blood electrolyte concentrations tested for some other reason.[3,13,15,22,25,26,28,30,32,50] In athletes with this biochemical diagnosis, oral fluid intake should be restricted until the onset of urination. Athletes should also be advised to seek urgent medical attention if any signs or symptoms of EAH develop. Asymptomatic EAH is a contraindication for the administration of intravenous normal saline or hypotonic fluids, which can worsen the degree of hyponatremia and fluid overload in some cases.

Symptomatic—Onsite

The definitive emergency treatment of EAH encephalopathy (as described above) is immediate onsite administration of intravenous hypertonic saline because of the known rapid progression of life-threatening encephalopathy and its complications.[101] This treatment modality was first established in 2000.[45] In that paper, all the patients who received hypertonic saline survived and the only death occurred in the patient who was treated with normal saline. The subsequent deaths reported in the literature were patients who failed to receive hypertonic saline.[49]

Intravenous access must be established, and high-flow oxygen should be administered.

Any athlete with EAH encephalopathy should be immediately treated with a bolus infusion of 100 mL of 3% NaCl to acutely reduce brain edema. Up to two additional 100ml 3% NaCl bolus infusions should be given at 10 minute intervals if there is no clinical improvement.[101] This regimen should not pose any substantial danger to the patient. There have been no reported cases of osmotic demyelination, or central pontine myelinolysis, in association with the rapid correction of an acute hyponatremia (i.e., <48 hour duration).[102] Therefore, concern regarding osmotic demyelination should not be an impediment to rapidly correcting hyponatremia in symptomatic EAH.[4–7,10,31,34,45,49,64,103] The efficacy of hypertonic 3% NaCl *infusion* as the definitive treatment for EAH encephalopathy has been documented in the hospital setting[45,49,51,76,104–106] and was first used successfully in the field at the 2004 Boston Marathon under a protocol jointly approved by the Boston Athletic Association Medical Team and the Boston Emergency Medical Services.[49,103] The first successful use of a *bolus* of hypertonic saline was documented onsite by JCA in the 2005 Two Oceans Marathon and subsequently by WOR in the Twin Cities Marathon October 2005 (personal communications) and verified by others.[42]

This therapy aims to stabilize the athlete prior to hospital transfer without producing complications. The athlete should be urgently transferred to a definitive medical care facility. Ideally, the athlete should be transported with knowledgeable personnel able to maintain the same level of care en route. The diagnosis of EAH and transfer must be communicated to the emergency room physician upon transfer of care.

Care must be taken to avoid the administration of isotonic or hypotonic fluids to prevent worsening the degree of hyponatremia and fluid overload (with the exception of cases where there is evidence of circulatory insufficiency where standard Advanced Cardiac Life Support protocols apply).[46,49]

Symptomatic—In hospital

Athletes presenting primarily to the hospital or medical facility with signs or symptoms of hyponatremia should have their serum electrolytes measured immediately and be treated as described above without delay if EAH is confirmed.

Athletes being transferred from the race medical facility require both clinical and laboratory reassessment upon admission, with care to avoid treatment delays while awaiting diagnostic tests such as brain imaging.[7,31,45,64] Administration of hypotonic or isotonic intravenous fluids during this

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re-evaluation is again contraindicated because of the potential to exacerbate hyponatremia and fluid overload.

If symptomatic EAH persists or worsens, current treatment guidelines for acute symptomatic hyponatremia should be followed in an intensive or critical care setting including appropriate consultation with a specialist familiar with this condition.

Dissemination of Advice for Prevention and Treatment of EAH

Athletes and Coaches

Educational strategies aimed at coaches and athletes to promote rational fluid replacement, to avoid the over consumption of fluids (water or sports drinks), to recognize the signs and symptoms of EAH, and to understand the critical need to seek immediate medical attention must be effectively communicated. Special attention should be paid to susceptible athletes with any of the risk factors listed above.

Medical Directors and Race Directors

Race medical directors should be involved in all decisions regarding strategies for optimal hydration. These include number and placement of aid stations, distribution of drinking advice to athletes and training of the aid station personnel. Drinking advice distributed to participants by sponsors should be reviewed by, and not conflict with, the information approved by the race medical director.

Pre-race weight (training weight) should be recorded (eg, on a start list or on the participant's bib number), so that it is available for medical personnel should it be required for comparison to post race weight.

Medical directors should ensure the availability of onsite [Na^+] analysis and hypertonic saline.

A record of EAH cases should be kept, including follow up and outcome, to aid in planning for future events.

Medical Staff

All medical personnel must be educated on the signs, symptoms, evaluation and treatment strategies of EAH. Weighing of all participants presenting to the medical area is strongly advised. Hypertonic saline should be readily available in the medical tent.

Emergency Medical Services and Hospitals

Prior to the race or event, the race medical team should establish a relationship with the local emergency response and transport teams, medical facilities and emergency department physicians. This may include specific collaborative education programs aimed at all of these groups.

Suggestions for Future Research

Prospective and controlled clinical trials on fluid replacement during exercise should be performed both in the laboratory and in the field. The following areas were identified by the panel as priorities for further study:

- Investigate alternative treatments for non-life threatening EAH including oral hypertonic NaCl solutions and vasopressin receptor antagonists
- Investigate the maintenance of normonatremia despite overhydration and weight gain via the possible activation of osmotically inactive sodium pools or from other currently unknown factors
- Determine if cases occur where sodium depletion with significant weight loss play a role in the pathogenesis of EAH
- Investigate the role of different forms of sodium supplementation (eg, liquid or solid) in the modification of EAH
- Investigate the etiology/mechanisms for inappropriate (ie, non-suppressed) plasma AVP concentrations during endurance exercise
- Investigate the potential role of endogenous water production or release during endurance exercise, in particular water complexed to muscle and liver glycogen, in the pathogenesis of EAH

All cases of EAH should be recorded and monitored (ie, scope of presentation, treatment and outcomes) on the International EAH Registry at: www.overhydration.info, while anyone interested in discussing EAH are invited to join the EAH Discussion Group List serve at: https://lists.uct.ac.za/mailman/listinfo/eah-discuss-l.

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INTERNATIONAL EXERCISE-ASSOCIATED HYPONATREMIA REGISTRY

Conducted by the Exercise-Associated Hyponatremia Consensus Group

VISION: To establish a worldwide cooperation, collaboration and understanding of exercise-associated hyponatremia (EAH) to facilitate the prevention and eradication of this entity from endurance sport.

PLAN:

1) To create a database to record cases of hyponatremia after completion of each successive endurance event prospectively from January 2008.
2) To facilitate communication between medical personnel and scientists regarding the etiology, management, treatment and prevention of EAH at endurance events.
3) To offer a website-based forum in which to address questions posed by clinicians, scientists and the general public.
4) To publish the results of this prospective database in order to assess our knowledge and progress.

GUIDING PRINCIPLES:

The EAH Consensus Group will obey the strict practices of confidentiality and anonymity. The identity of each subject will only be known to the researchers, and, in order to maintain anonymity, numbers will be allocated in lieu of names. The data generated will be stored in a secure facility and confidentiality will be ensured. The data will be used by the EAH Group for scientific research purposes only.

Thank you for your cooperation and concern. If you have any questions or concerns regarding the Registry, please contact or send forms to:

Tamara Hew-Butler, DPM, PhD
Systemic Inflammation Laboratory, Trauma Research
350 West Thomas Road
Phoenix, Arizona 85013
Tel: (602) 406-4851
Fax: (602) 406-4113
Email: tamara.hew@chw.edu

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EXERCISE-ASSOCIATED HYPONATREMIA SURVEY

Conducted by the Exercise Associated Hyponatremia Consensus Group

EAH is the occurrence of hyponatremia during or up to 24 hours after prolonged physical activity and is defined by a serum or plasma sodium concentration ($[Na^+]$) below the normal reference range of the laboratory performing the test. For most laboratories, this is a $[Na^+]$ less than 135 mmol/L.

Race information:
Endurance event and year: _____
Event distance: _____
Event date: _____
Temperature Range (C°): Starting temp: _____ Peak temp: _____
Relative Humidity: Start Peak temp
WBGT: Start Peak
Number of entrants: _____
Number of starters: _____
Number of finishers: _____
Number of female starters: _____
Number of female finishers: _____
Percentage female finishers: _____
Winning time: _____
Mean finishing time for event: _____
Number of refreshment stations on course: _____
Beverages provided along course: _____
Athletes alerted to the dangers of overhydration prior to race: Y/N
Written hydration advice distributed to participants prior to race: Y/N
(If yes, please forward)

□ NO CASES OF EXERCISE-ASSOCIATED HYPONATREMIA TO REPORT
□ I HAVE THE FOLLOWING CASES OF EXERCISE-ASSOCIATED HYPONATREMIA TO REPORT:

Reporting Doctor's Information:
Name: _____
Position: _____
Email address: _____
Contact number: _____
Electrolyte analyzer available onsite: Y/N
Hypertonic saline available onsite: Y/N
Pre-race weights obtained on competitors: Y/N
Have experience with treating EAH: Y/N
Number of collapsed athletes seen in medical tent: _____
Number of athletes sent to hospital: _____

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SUBJECT INFORMATION

Sex: □ Male □ Female

Age: _____years

Experience: □ First time participant OR □ Repeat participant:
Number started: _____
Number completed: _____

Fluid consumption: □ more than 1cup/sachet every fluid station □ every station
□ every 2nd station □ less than every 2nd station

Urination on course (number of stops): □ 0 □ 1 □ 2 □ 3 □ 4 □ >4
NSAIDs taken immediately prior to or during race: □ Yes □ No
History of EAH in previous events: □ Yes □ No

Finishing Time: (Hours: minutes) _____:_____

Initial Serum Sodium Concentration (mmol/L): _____

Presentation: □ Symptomatic □ Asymptomatic

Site of collapse/diagnosis: □ before finish □ finish area *(brought into medical tent)*
□ <24 hours after finish *(brought into emergency room)*
Time of collapse/presentation (after finish if appropriate): _____

Signs and Symptoms: □ bloating □ puffiness □ headache □ nausea □ vomiting
□ altered mental status □ respiratory distress □ seizures
□ obtundation/stupor/coma □ agitation □ confusion
□ other: _____

Treatment: □ fluid restriction □ observation □ oral sodium: *__tablets__food__liquid*
□ Hypertonic saline: *if yes, amount given:* _____
□ Other IV fluid: *type and amount* _____
□ Other treatment: _____
_____
□ Duration of major symptoms: _____
Hospitalisation required: □ Yes □ No
How long in hospital: _____

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EXERCISE-ASSOCIATED HYPONATREMIA
REPORT FORM

Please use this form to detail cases, particularly if hospitalization required.
Include all laboratory values, results of imaging tests, time of symptom resolution (specifically neurological and pulmonary) in relationship to treatment, time of admission and discharge and follow up sequelae if known (ie, return to training and competition)

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