

CASE REPORT

Hyponatraemic encephalopathy despite a modest rate of fluid intake during a 109 km cycle race

J P Dugas, T D Noakes

Br J Sports Med 2005;39:e38 (<http://www.bjsportmed.com/cgi/content/full/39/10/e38>). doi: 10.1136/bjism.2005.018820

Objective: To report a case of exertional hyponatraemic encephalopathy that occurred despite a modest rate of fluid intake during a 109 km cycling race.

Methods: Men and women cyclists were weighed before and after the race. All subjects were interviewed and their water bottles measured to quantify fluid ingestion. A blood sample was drawn after the race for the measurement of serum Na^+ concentration.

Results: From the full set of data ($n = 196$), one athlete was found to have hyponatraemic encephalopathy (serum $[\text{Na}^+]$ 129 mmol/l). She was studied subsequently in the laboratory for measurement of sweat $[\text{Na}^+]$ and sweat rate.

Conclusions: Despite a modest rate of fluid intake (735 ml/h) and minimal predicted sweat Na^+ losses, this female athlete developed hyponatraemic encephalopathy. The rate of fluid intake is well below the rate currently prescribed as optimum. Drinking to thirst and not to a set hourly rate would appear to be the more appropriate behaviour.

There is now a near complete international consensus that hyponatraemic encephalopathy develops in subjects who ingest excessive volumes of fluid during exercise and who gain weight, as a result developing “water intoxication”.^{1–6} Accordingly, fluid ingestion guidelines recently accepted by the United States Track and Field⁶ suggest that athletes should drink ad libitum during exercise—that is, according to thirst—as this biologically driven approach, adopted naturally by all creatures other than exercising humans who have been taught otherwise, will probably prevent too little or too much being drunk.

However, the apparent reluctance to accept that a basic physiological drive can produce safe drinking behaviours in humans, as it does in all other creatures, continues to underpin advice that athletes must be provided with specific guidelines for rates of fluid intake during exercise. Therefore the guidelines of the United States Track and Field suggest that rates of fluid intake during exercise should be 400–800 ml/h,⁶ although others advise rates as high as 1000 ml/h.⁷ These values are, however, substantially less than rates of 1200 ml/h advocated by the American College of Sports Medicine⁸ and of up to 1800 ml/h originally proposed by the United States Army.⁹

Yet there are examples of athletes who have developed water intoxication during exercise despite drinking fluid at rates substantially lower than these currently prescribed rates. Speedy *et al*¹⁰ reported the development of hyponatraemia in two Ironman triathletes whose estimated rates of fluid intake were 733 and 764 ml/h during 13.3 and 12 hours of exercise respectively. These rates fall within the range 400–1000 ml/h currently advocated.^{6,7} Glace *et al*¹¹ reported that athletes who ingested fluid at a rate of 700 ml/h developed gastrointestinal symptoms during a 160 km trail race,

whereas Stuempfle *et al*^{12,13} reported a reduction in serum sodium concentrations ($[\text{Na}^+]$) in athletes completing a 100 mile Iditasport ultramarathon completed in cold conditions in Alaska, even though they drank only 200–400 ml/h. Similarly, Twerenbold *et al*¹⁴ reported a mean decrease in serum $[\text{Na}^+]$ of 6.2 mmol/l in women who ingested 1000 ml/h of water for four hours when covering 40 km at a pace equivalent to a 4:15 (hours:minutes) 42.2 km marathon. This rate of fluid ingestion was twice the sweat rate of these athletes; as a result the athletes gained 2 kg during the four hours that they ran.

These data therefore indicate that medical complications can occur in athletes who drink at rates that are less than the maximum “safe” values currently proposed by various authorities.

As part of a cross sectional study of 193 athletes in the 2003 109 km Cape Argus/Pick ‘n’ Pay Cycle Tour in Cape Town, South Africa, which attracts more than 35 000 participants annually, we identified one subject who developed mild hyponatraemic encephalopathy with a serum $[\text{Na}^+]$ of 129 mmol/l after the race, despite a relatively modest rate of fluid intake during the race. Here we report the physiological basis for the development of her condition. This case study adds further to the evidence showing that drinking to a fixed rate in excess of thirst can produce medical complications even when the rate appears quite modest.

CASE REPORT

A 57 year old female cyclist who had previously completed the 109 km cycle tour on 16 occasions (best time 3:57) volunteered to participate in a study of fluid balance and serum $[\text{Na}^+]$ during the race according to previously described methods.^{15–17}

Environmental conditions during the race were the following: at 0600 the ambient temperature was 18.3°C and the relative humidity was 99%; at 0900 the ambient temperature was 19.6°C and the relative humidity was 99%; at 1200 the ambient temperature was 21.7°C and the relative humidity was 77%; at 1600 the ambient temperature was 23.4°C and the relative humidity was 75%.

After completing the race in 5:10, the subject complained of a headache, light headedness, and difficulty concentrating. She had experienced these symptoms previously on numerous occasions after rides of >100 km and in at least one 42.2 km marathon some 20 years earlier.

She started the race with a weight of 66.6 kg. Her total fluid intake during the race was 3800 ml (735 ml/h). Her weight after the race was 69.0 kg, which indicated a total weight gain during the race of 2.4 kg (3.6% of body weight). To account for this weight gain, her estimated sweat rate during the race would have been about 270 ml/h. Her serum $[\text{Na}^+]$ after the race was 129 mmol/l, and she was the only athlete in our trial of 196 cyclists who developed a serum $[\text{Na}^+]$ less than 135 mmol/l (fig 1) and the only athlete in the

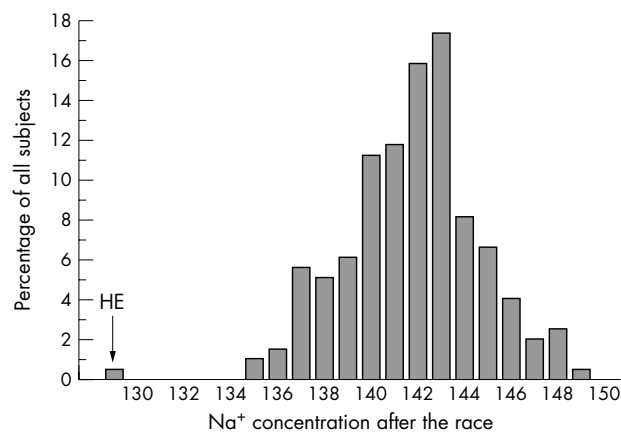


Figure 1 Distribution of serum $[\text{Na}^+]$ after a 109 km race in 196 cyclists (103 men and 93 women). The cyclist described in this study is identified as HE (hyponatraemic encephalopathy) and was the only one who developed hyponatraemia (serum $[\text{Na}^+] < 135$ mmol/l).

study to develop symptoms of hyponatraemic encephalopathy.

As in all our and other previous studies,^{2 4 10 15–19} an inverse linear relationship was found between the change in mass during exercise and serum $[\text{Na}^+]$ after the race, so that those athletes who lost the least weight during the race had the lowest serum $[\text{Na}^+]$ after the race (fig 2). Furthermore, the subject in this case report (“HE” in figs 1 and 2) was the only cyclist to gain a substantial amount of weight.

Two weeks after the race, this athlete performed a series of laboratory experiments for measurement of her sweat rate and the $[\text{Na}^+]$ of the sweat collected from her back, arm, and leg. When cycling in a specially designed environmental chamber²⁰ at a rate of 120 W in ambient conditions of 28.5°C, 55% humidity, and with a facing air velocity of 10 km/h wind speed, her sweat rate was 520 ml/h and the sweat $[\text{Na}^+]$ on her back, arm, and leg were 73, 68, and 64 mmol/l respectively (average of 68 mmol/l). Therefore total Na^+ loss in sweat during the race was probably only 70–105 mmol, similar to values either measured^{3 10 21} or predicted²¹ in other athletes who developed this condition during prolonged exercise.

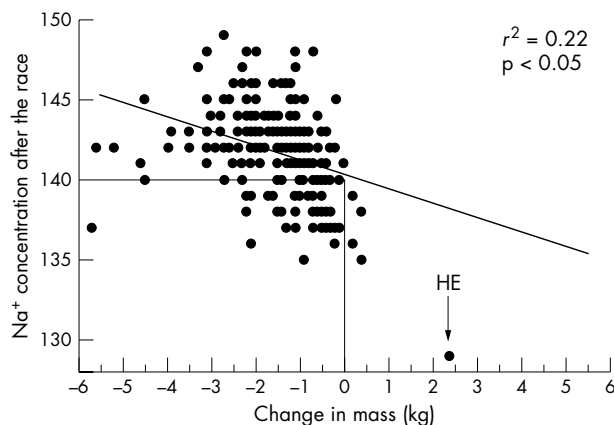


Figure 2 Relationship between change in mass and serum $[\text{Na}^+]$ in 196 cyclists (103 men and 93 women) who completed a 109 km cycle race. The cyclist who gained the most weight is identified as HE and was the only one to develop hyponatraemia.

On the basis of the finding that her condition was due to voluntary overhydration without a substantial Na^+ deficit, she was advised that in future she should always drink ad libitum during exercise.

On follow up after the 2004 Cape Argus/Pick ‘n’ Pay 109 km Cycle Tour, she reported that her ad libitum fluid intake rate had been not more than 500 ml/h. Her race time was 5:38 for the 109 km course, which was similar to her time of 5:10 in the previous year. She reported being symptom-free after the race, as well as in other rides >100 km completed in the 12 months since she had been evaluated in the laboratory. In fact, she enthusiastically stated that her enjoyment of cycling had increased substantially since she began to drink less fluid during prolonged exercise.

DISCUSSION

The only cyclist to develop hyponatraemia in our prospective study of 196 cyclists in this race was also the only one to gain a substantial amount of weight (2.4 kg; 3.6% of body weight; fig 2). This mirrors our finding at the 2001 South African Ironman triathlon in which the only athlete to develop hyponatraemic encephalopathy and who required hospital admission after the race was also the only athlete to gain a significant amount of weight during the race (3.8 kg; 4.7% of body weight).¹⁷

However, more to the point, the athlete in this study developed hyponatraemic encephalopathy within a relatively short time after exercise (5.16 h) despite a rate of fluid intake that complies with some of the current guidelines for fluid replacement during exercise.^{7 8 22} Speedy *et al*¹ have reported the development of asymptomatic hyponatraemia in two female Ironman triathletes who ingested fluids at similar rates to the cyclist in this report. Yet those triathletes developed asymptomatic hyponatraemia only after 13 h compared with 5–6 hours in this case. However, neither had symptoms of central nervous system dysfunction (hyponatraemic encephalopathy).

The aetiology of this condition clearly is the ingestion of free water at rates that exceed the rate of free water clearance by the kidneys, leading to water intoxication as faithfully reproduced in the laboratory.^{18 23} The low sweat rate in this cyclist is probably due to the low exercise intensity (average cycling speed 21.1 km/h) combined with cool conditions in which heat losses due to convection and radiation are high. The latter two avenues of heat loss were not considered when the original studies on which the current drinking guidelines⁸ are based were conducted.^{9 22 24} As a consequence of the low sweat rate of this cyclist and her low rates of free water clearance, fluid retention and cerebral swelling developed, causing hyponatraemic encephalopathy according to the mechanisms we have proposed since 1985,²⁵ and as conclusively proven in 1991,³ even though her rates of fluid ingestion were modest and well within currently accepted guidelines.^{6 7–9 22}

Confirmation that fluid overload caused hyponatraemic encephalopathy in this case comes from the predictions, based on the laboratory measurement of her average sweat $[\text{Na}^+]$, that her total Na^+ losses during the race were small (about 70–105 mmol). Furthermore, simple balance equations show that a positive change in weight of either 3.3% or 4.0% respectively would be enough to lower a female athlete’s serum $[\text{Na}^+]$ to 130 mmol/l, depending on whether the ingested fluid is water (3.3%) or an electrolyte-containing sports drink (4.0%) according to the calculations of Weschler.²⁶ The actual weight change in this athlete was +3.6%, and her serum $[\text{Na}^+]$ after the race was 129 mmol/l, confirming the accuracy of the predictive equations of Weschler.²⁶

The fact that this athlete did not develop symptoms in subsequent exercise when she drank ad libitum and without increasing her Na⁺ intake before, during, or after exercise confirms that this condition is due to fluid overload to which any Na⁺ deficit plays only a minor contributory role²⁶ as it merely moderates the serum [Na⁺] at any given level of fluid overload.

Finally this study suggests that fluid replacement guidelines based on absolute rates of fluid intake are inherently flawed as sweat rates can vary so widely during exercise, ranging from >2 litres/h in American football players²⁷ to about 500 ml/h in ultramarathon runners in cold conditions^{12, 13} to 300 ml/h in this cyclist.

Rather, we continue to argue that no evidence exists to suggest that athletes who drink ad libitum as opposed to at fixed rates are at increased risk of ill health during exercise, or that they will perform less than optimally as a result.^{6, 28} Instead we argue that all the current evidence indicates that athletes perform optimally when they drink ad libitum during exercise.^{29–31}

ACKNOWLEDGEMENTS

We acknowledge the help of Dr Basil Bonner for logistical support. We also acknowledge the assistance of Ian de Witt, for analysing the sweat samples, and Ingeborg Muhsfeldt, for analysing the blood samples. Finally, special thanks go to the Cycle Tour Trust for the opportunity to perform this research.

Authors' affiliations

J P Dugas, T D Noakes, UTC/MRC Research Unit for Exercise Science and Sports Medicine, University of Cape Town, Sports Science Institute of South Africa, Cape Town, South Africa

Competing interests: none declared

Correspondence to: J P Dugas, UTC/MRC Research Unit for Exercise Science and Sports Medicine, University of Cape Town, Sports Science Institute of South Africa, PO Box 115, Newlands 7725, Cape Town, South Africa; jdugas@sports.uct.ac.za

Accepted 30 March 2005

REFERENCES

- 1 Speedy DB, Rogers IR, Noakes TD, et al. Exercise-induced hyponatremia in ultradistance triathletes is caused by inappropriate fluid retention. *Clin J Sport Med* 2000;**10**:272–8.
- 2 Speedy DB, Noakes TD, Rogers IR, et al. Hyponatremia in ultradistance triathletes. *Med Sci Sports Exerc* 1999;**31**:809–15.
- 3 Irving RA, Noakes TD, Buck R, et al. Evaluation of renal-function and fluid homeostasis during recovery from exercise-induced hyponatremia. *J Appl Physiol* 1991;**70**:342–8.
- 4 Hew TD, Chorley JN, Cianca JC, et al. The incidence, risk factors, and clinical manifestations of hyponatremia in marathon runners. *Clin J Sport Med* 2003;**13**:41–7.
- 5 Gardner JW. Death by water intoxication. *Mil Med* 2002;**167**:432–4.
- 6 Noakes TD. Fluid replacement during marathon running. *Clin J Sport Med* 2003;**13**:309–18.
- 7 Coyle EF. Fluid and fuel intake during exercise. *J Sports Sci* 2004;**22**:39–55.
- 8 Convertino VA, Armstrong LE, Coyle EF, et al. American College of Sports Medicine position stand: exercise and fluid replacement. *Med Sci Sports Exerc* 1996;**28**:R1–7.
- 9 Montain SJ, Lutzka WA, Sawka MN. Fluid replacement recommendations for training in hot weather. *Mil Med* 1999;**164**:502–8.
- 10 Speedy DB, Noakes TD, Rogers IR, et al. A prospective study of exercise-associated hyponatremia in two ultradistance triathletes. *Clin J Sport Med* 2000;**10**:136–41.
- 11 Glace B, Murphy C, McHugh M. Food and fluid intake and disturbances in gastrointestinal and mental function during an ultramarathon. *Int J Sport Nutr Exerc Metab* 2002;**12**:414–27.

- 12 Stuemppfle KJ, Lehmann DR, Case HS, et al. Hyponatremia in a cold weather ultraendurance race. *Alaska Med* 2002;**44**:51–5.
- 13 Stuemppfle KJ, Lehmann DR, Case HS, et al. Change in serum sodium concentration during a cold weather ultradistance race. *Clin J Sport Med* 2003;**13**:171–5.
- 14 Twerenbold R, Knechtle B, Kakebeeke TH, et al. Effects of different sodium concentrations in replacement fluids during prolonged exercise in women. *Br J Sports Med* 2003;**37**:300–3.
- 15 Sharwood K, Collins M, Goedecke J, et al. Weight changes, sodium levels, and performance in the South African Ironman Triathlon. *Clin J Sport Med* 2002;**12**:391–9.
- 16 Sharwood KA, Collins M, Goedecke JH, et al. Weight changes, medical complications, and performance during an Ironman triathlon. *Br J Sports Med* 2004;**38**:718–24.
- 17 Noakes TD, Sharwood K, Collins M, et al. The dipsomania of great distance: water intoxication in an Ironman triathlete. *Br J Sports Med* 2004;**38**:E16.
- 18 Speedy DB, Noakes TD, Boswell T, et al. Response to a fluid load in athletes with a history of exercise induced hyponatremia. *Med Sci Sports Exerc* 2001;**33**:1434–42.
- 19 Speedy DB, Faris JG, Hamlin M, et al. Hyponatremia and weight changes in an ultradistance triathlon. *Clin J Sport Med* 1997;**7**:180–4.
- 20 Saunders AG, Dugas JP, Tucker R, et al. The effects of different air velocities on heat storage and body temperature in humans cycling in a hot, humid environment. *Acta Physiol Scand* 2005;**183**:241–55.
- 21 Noakes T. Sodium ingestion and the prevention of hyponatraemia during exercise. *Br J Sports Med* 2004;**38**:790–2.
- 22 Armstrong LE, Epstein Y, Greenleaf JE, et al. Heat and cold illnesses during distance running. *Med Sci Sports Exerc* 1996;**28**:R1–10.
- 23 Noakes TD, Wilson G, Gray DA, et al. Peak rates of diuresis in healthy humans during oral fluid overload. *S Afr Med J* 2001;**91**:852–7.
- 24 Montain SJ, Coyle EF. Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. *J Appl Physiol* 1992;**73**:1340–50.
- 25 Noakes TD, Nathan M, Irving RA, et al. Physiological and biochemical measurements during a 4-day surf-ski marathon. *S Afr Med J* 1985;**67**:212–16.
- 26 Weschler LB. Exercise-associated hyponatremia: a mathematical review. *Sports Med* 2005;**35**:in press.
- 27 Fowkes GS, Godek JJ, Bartolozzi AR. Thermal responses in football and cross-country athletes during their respective practices in a hot environment. *J Athl Train* 2004;**39**:235–40.
- 28 Noakes TD. Can we trust rehydration research? In McNamee M, eds. *Philosophy and the sciences of exercise, health and sport: critical perspectives on research methods*. Oxford: Routledge, 2005:144–68.
- 29 Daries HN, Noakes TD, Dennis SC. Effect of fluid intake volume on 2-h running performances in a 25 degrees C environment. *Med Sci Sports Exerc* 2000;**32**:1783–9.
- 30 Chevront SN, Haymes EM. Ad libitum fluid intakes and thermoregulatory responses of female distance runners in three environments. *J Sports Sci* 2001;**19**:845–54.
- 31 McConell GK, Burge CM, Skinner SL, et al. Influence of ingested fluid volume on physiological responses during prolonged exercise. *Acta Physiol Scand* 1997;**160**:149–56.

COMMENTARY

This is an important paper describing the development of hyponatraemic encephalopathy in a female cyclist. Although similar case studies have been published previously, this one is particularly interesting because the subject developed the disorder over a relatively short time (about five hours) and by ingesting fluids during her event at rates (~700 ml/h) well below those prescribed by the current guidelines of the American College of Sports Medicine (ACSM). The follow up examination is important because it shows that the predicted sweat Na⁺ losses would have been minimal, and lends strong support to the now well established overdrinking theory on why hyponatraemia develops. This calculation is important to silence the doubters of this theory, many of whom sit blindly behind the doors of Gatorade and the ACSM.

P B Laursen

Edith Cowan University, School of Biomedical and Sports Science, Perth, WA, Australia; p.laursen@ecu.edu.au