EDITORIAL

RUNNING, THE KIDNEYS AND DRINKING TOO MUCH — THE HYPONATRAEMIA OF EXERCISE

Dehydration, you will have been taught, is the single greatest danger to the health of the exercising athlete[1-4] — except for the minor intellectual inconvenience that there is no scientific basis whatsoever to support this universally pervasive dogma.[5-8] Rather, the unambiguous evidence is that drinking too much during exercise, leading to self-induced water intoxication (the symptomatic hyponatraemia of exercise), has become the greater threat to the health of modern athletes during prolonged exercise.[9]

In the past 15 years at least 70 cases[10,11] of significant illness including disorientation, grand mal epileptic seizures, coma, respiratory arrest and even deaths[12-14] have been described in ultra-distance triathletes and runners, army recruits and even desert hikers who drank too much during or after prolonged exercise. This includes perhaps as many as four deaths, some of which may have been iatrogenic since the usual history of this condition is spontaneous recovery if the existing fluid overload is not compounded by the administration of large volumes of intravenous fluids. Such deaths are especially sad since they involve physically fit individuals who not only began exercise without significant illness, but who often epitomised consummate 21st-century good health.

In contrast to the hyponatraemia of exercise, other complications of exercise including heatstroke and acute renal failure are reported far less frequently, and then usually in persons with other predisposing medical conditions or traits.[15,16] Nor is there any reputable scientific evidence to even suggest that fluid imbalance, specifically dehydration, contributes meaningfully to the cause of these latter conditions.[5-9,17]

The first reported modern case of the hyponatraemia of exercise occurred in a 46-year-old 49 kg woman competing in the 1981 90 km Comrades Marathon.[18] After running 70 km, she was removed from the race by her husband on the grounds that she had not recognised him! It was the correct decision, for on their return drive to Durban she suffered a grand mal seizure. On hospital admission this lady, who less than 10 hours earlier had been fit enough to start one of the most challenging ultra-distance footraces in the world, a right she had earned because she was appropriately trained and in perfect health, was close to death. She was in a coma, with marked pulmonary oedema, radiographic evidence of cardiomegaly, and a serum sodium concentration of 115 mmol/l. Treated appropriately with fluid restriction, promotion of diuresis and sodium supplementation, she recovered fully and was released from hospital 6 days later.

The earliest reports of the hyponatraemia of exercise speculated that it occurred in athletes who sustained inappropriately high rates of fluid ingestion for many (< 7) hours during prolonged exercise.[18,19] But a contemporary and unfortunately titled article suggested the reverse — that the hyponatraemia of exercise was caused by dehydration[20] and hence occurred in those who drank too little during exercise. This speculation was published in a peer-reviewed journal even though the article did not include measurements of changes in whole-body fluid balance during exercise in athletes who developed the hyponatraemia of exercise. More than a decade later, review articles published even as recently as this year continue to quote that article as evidence that the hyponatraemia of exercise can occur in those who drink too little during prolonged exercise.[21]

This particular controversy should have been laid to rest 10 years ago when another South African study[22] established that eight comatose and desperately ill Comrades Marathon runners with symptomatic hyponatraemia were all suffering from profound fluid overload. In addition, there was no evidence that abnormal losses of sodium in their sweat and urine had contributed significantly to their hyponatraemia. Since then, a series of additional studies[9,10] confirm that patients with the symptomatic hyponatraemia of exercise, and whose serum sodium concentrations are less than 129 mmol/l, all have a whole-body fluid excess of at least 2 - 3 litres and sometimes up to 6 litres. The suspicion exists that the main reason why this now incontrovertible conclusion has yet to be enthusiastically embraced, especially in the USA, is because it conflicts with commercial interests based on the deceptive paradigm that the unrestricted consumption of sports drinks under all circumstances is uniquely beneficial and scientifically proven.[23]

Importantly, these studies of the hyponatraemia of exercise also show that it takes a fluid excess of as little as 2 - 3 litres to produce significant illness, including the risk of mortality, whereas we frequently measure body weight losses of up to 8 kg in top finishers in the South African Ironman Triathlon who are outwardly completely asymptomatic and without any clinical evidence that such high levels of dehydration carry any risks whatsoever (K Sharwood *et al.* — manuscript in preparation).

However, if whole-body fluid content can rise dangerously during prolonged exercise, then the physiological question requiring attention is how this occurs. The logical assumption is that inappropriate secretion of the water-conserving hormone, antidiuretic hormone (ADH), must be involved. But why should the secretion of ADH, the hormone of water lack, be increased when the body has fluid in abundance? In fact there is no evidence that increased serum ADH activity plays

any role in this condition.[10] Yet the clear clinical observation is that the return of adequate diuresis in patients who are profoundly fluid-overloaded occurs only many hours after the termination of exercise, suggesting the presence of a circulating antidiuretic factor other than ADH.

The other possibility is that the healthy kidneys have a limited capacity to excrete fluid that accumulates in the body at a rapid rate. Textbooks of renal physiology state that the maximum daily rate of urine production is about 24 l/day (1 l/h),[24] but this conclusion appears to be rooted in theoretical, albeit probably correct, calculations based on the known maximum rates of glomerular filtration and renal tubular function, and not on directly measured values.

As reported in this issue of the *Journal* (p. 852), we have established a model of fluid overload at rest specifically to determine the most likely mechanism by which such fluid overload induces hyponatraemia. The study establishes that the human capacity for intestinal fluid absorption may exceed a maximal capacity for renal free water clearance that appears to be about 900 ml/h. Hence when fluid is ingested at rates higher than about 900 ml/h, the total body water must expand and tissue osmolality must fall, leading to a generalised whole-body oedema, the most dangerous component of which is a potentially fatal cerebral oedema.

Hence this study provides the physiological explanation as to why it is never safe to consume fluid without restriction or 'the maximum amount that can be tolerated' according to the American College of Sports Medicine fluid replacement guidelines for exercise.[3,4] The simple reason is that the kidneys do not have an infinite capacity to prevent a fluid excess when exogenous fluid enters the body at high rates (in excess of 1 l/h) through either the gut or the veins.

This study will have been even more helpful if it draws attention to the widespread ignorance of the potentially mortal complications of the condition of water intoxication that is neither benign nor uncommon during prolonged exercise.[9,10] It exists because of: (*i*) a fallacious dogma which holds that even mild dehydration during exercise will have potentially catastrophic consequences — what has been termed the 'dehydration myth';[5-8] (*ii*) the resulting hysteria that dehydration must therefore be prevented at all costs;[8] and (*iii*) our confirmation that the kidney, like the gut, has an 'enough but not too much' capacity.[25]

Often it is only when humans adopt bizarre behaviour patterns, in this case aided and abetted by a science gone mad,[7,8] that these apparent limitations in human design become apparent.

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