Sodium ingestion and the prevention of hyponatraemia during exercise

The study of Twenbolde et al1 is important for a number of reasons, not all of which may have been emphasised sufficiently by the authors.

Firstly, it confirms that a rate of fluid intake of 1000 ml/h is too high for a group of female runners running at ~ 10 km/h and who would therefore complete a 42 km marathon in about 4.25 hours. As the athletes drank 4 litres and gained 2 kg during the trial, their average rate of weight loss (as opposed to sweat rate) was about 500 ml/h. As not all of the weight lost during exercise is sweat and as much as 1–3 kg of this weight loss may result from fuel and water losses that do not contribute to dehydration,4 the absolute maximum rate at which these athletes should have ingested fluid during exercise was probably even less than 500 ml/h. This is substantially less than the drinking guidelines of the American College of Sports Medicine5 and the Gatorade Sports Science Institute6 and the currently established ‘best practice’.

As the study design should, in retrospect, not have been sanctioned. Rather, the control group in the study should have ingested fluid according to guidelines based on the strongest body of current information. It is, for obvious reasons, my biased opinion that the guidelines that come closest to a defensible evidence base are those that have been recently accepted by the United States Track and Field and the International Marathon Medical Interest Group7.

Fortunately the data of Twenbolde et al do allow some calculations to estimate the likely value of the extra sodium that was ingested by two of their groups. Thus, the athletes in their study had an initial TBW of 1.9 litres. According to the formula of Montain et al,8 their predicted extracellular fluid (ECF) volume would be about 14.3 litres (25% of body weight). As the starting serum (and ECF) sodium concentration ([Na+]i) in the three groups of runners was ~137 mmol/l (table 3 of their article), the average total ECF Na+ content of the three experimental groups was 1909–1993 mmol at the start of the race. As weights increased by 1.8–2.1 kg in the three groups during exercise (table 3 of their article), the increases in ECF volume would have been 450–525 ml in the respective groups, assuming that the ECF increased in proportion to the increase in total body water (TBW). Multiplying this new ECF volume by the serum [Na+] gives the new total ECF Na+ content after the race. As shown in table 1, the total ECF Na+ content increased by 34 mmol in the group that ingested the high salt drink (H) during the race, but fell by 23 mmol in the group drinking water (W). As all groups ran for about four hours, according to these calculations, the hourly rate of Na+ loss would have varied from 6 to 21 mmol/h, giving a sweat [Na+] of 12–42 mmol/l in the W and H groups respectively (as their total sweat losses were ~2 litres in each case, not 3 litres as reported).

The clear paradox identified by the calculations in table 1 is that (a) the total Na+ loss apparently increases with increased Na+ intake and (b) the estimated Na+ loss in the group who ingested normal drink during the race (W) is less than one third of that in the group who ingested the most Na+ (H) during the race.

As these calculations are based on two real measurements (body weight changes and changes in plasma [Na+]), this apparently ludicrous conclusion can only be explained if (a) Na+ ingestion during exercise increases whole body Na+ losses in sweat and urine or (b) the estimated ECF volume in the W group after exercise is less than the value calculated. That is, specifically in the W group, the ECF volume contracted despite an increase in TBW of 1.9 litres. Indeed, this response is to be expected. There is consistent evidence that the response of the ECF and the intracellular fluid (ICF) volumes to fluid ingestion during exercise is influenced by the Na+ content of the ingested fluid9, so that the greater the Na+ content, the more the ECF and ICF volumes are likely to expand if a concentrated (50–100 mmol/l) Na+ drink is ingested at the same rate that body weight is lost during exercise.10 In the latter case, any reduction in the TBW appears to come from a reduction in the ICF.11

For example, if each group did indeed lose 84 mmol Na+ as did group H (table 1), a value that seems eminently reasonable as it equates to a quite reasonable sweat [Na+] of ~40 mmol/l,12 then the true ECF volume in the W group after the race would have been 14.5 litres—that is, it is unchanged from the pre-race TBW value. This value is calculated as: (pre-race ECF Na+ content (~84) in mmol divided by post-race serum [Na+] in mmol).

Indeed, if subjects in the W group did lose 84 mmol Na+ during the race but also had a post-race ECF volume expanded to 14.95 litres, then their post-race [Na+] would have been even lower (128 mmol/l) than that actually measured after the race (132 mmol/l; table 1). It is probable that, if the sweat [Na+] was 34 mmol/l, they would have exhibited the early symptoms of hyponatraemic encephalopathy.8 That they did not have such low serum [Na+] indicates the importance of small changes in ECF volume (in this case only 450 ml or ~3% of the total ECF volume) in determining the extent to which the serum [Na+] changes during prolonged exercise in which subjects both sweat and ingest fluid to excess.13

Unfortunately, the vital importance of these small changes in ECF volume in determining whether hyponatraemic encephalopathy will develop in those who over-drink during exercise is still to be ignored by those14–16 who argue incorrectly that it is the Na+ deficit that determines the extent to which the serum [Na+] falls in those who develop hyponatraemia during exercise. This calculation elegantly shows why small changes in ECF volume determine whether or not hyponatraemic...
encephalopathy will develop in those who overdrink, regardless of whether or not they also incur a Na⁺ deficit during exercise. A recent paper confirms these predictions by showing that mathematical modelling supports the argument that changes in TBW exert a much greater effect on serum [Na⁺] than does whole body Na⁺ content in those who overdrink and hence gain weight during exercise. Perhaps the point of these calculations is to show that it is not possible to calculate the state of Na⁺ balance in athletes during exercise and so to determine whether or not athletes have developed a Na⁺ "deficit", simply by measuring serum [Na⁺]. This is because the ECF volume will not be the same before, during, and after exercise and will change depending on the nature of the fluid ingested and the extent of any fluid deficit or excess that develops during exercise. 

But more importantly, these calculations clearly show why the regulation of the TBW and the ECF volume will have a much greater influence on serum [Na⁺] than will either the expected Na⁺ losses in sweat or the amount of Na⁺ ingested from sodium-containing sports drinks.

For example, a 1 litre (7%) reduction in the ECF volume would "release" 140 mmol Na⁺ into the contracted ECF volume. This means that it is possible to lose 140 mmol Na⁺ in sweat and urine without any change in serum [Na⁺] provided that the ECF volume were to contract by only 7%. If sweat [Na⁺] is about 40 mmol/l, as appears to have been the case in this study of Twerenbold et al. (table 1), then this 140 mmol is the equivalent of the Na⁺ content of about 3.5 litres of sweat. As athletes in this study sweated at a maximum rate of only 500 ml/h when running at 10 km/h, this means that simply by measuring serum [Na⁺] while running for seven hours, one can determine whether the presence of subtle symptoms is related to the degree of fluid overload, and hence the increase in the ECF, than to the level to which the serum [Na⁺] has been reduced.

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References


BOOK REVIEWS

Tennis


It is widely recognised that each sport has its own unique demands and injuries. Therefore the IOC, ITF, ATP, WTA, and Society for Tennis Medicine and Science should be congratulated on publishing, in this production, a comprehensive overview of tennis sports medicine. Together they have assembled an impressive array of experts in this field to write succinct and relevant chapters.

Every aspect of tennis is covered to cater for a broad range of readers, including players themselves. Some areas are covered in a high level of technical detail to please the biomechanists, in particular. However, some of the sports medicine is basic in concept and lacking significant evidence based validity.

Nevertheless, I would highly recommend this book to any health professional who treats a large number of tennis players. Most chapters provide a link between common sports medicine problems and their occurrence in tennis, including conditions that are unique to this sport. At times, some authors are somewhat optimistic with their view of recovery time from surgery—for example, three weeks for arthroscopic debridement of the infrapatellar fat pad.

Overall it is well presented with relevant and useful photographs and diagrams to aid the reader, and each chapter gives a list of further recommended reading. Unfortunately the book does not provide an answer to where 14 million tennis balls go, imported each year into Australia, as discussed by the editor recently!

Rating

- Presentation: 16/20
- Comprehensiveness: 15/20
- Readability: 15/20
- Relevance: 16/20
- Evidence basis: 13/20
- Total: 75/100

T Wood

Dying to win


Dying to win gives an eye opening account of the extent to which drugs play a major role in sport. Doping is not new and has been used in sport since ancient Olympic times; it is just that drug use in modern times is at such a level of sophistication, it is now an industry in its own right. The book describes the privileged position sport holds in society, having appeal for both the participant and the spectator. This has led to the massive media interest, commercialism, professionalism, and governmental regulation and manipulation. Economic pressure in the industrialised world and governmental propaganda in the former East Germany, and more recently China, has led to increasing pharmaceutical intervention in sport. With the fall of the GDR, the world saw for the first time what it had long suspected, the extent of systematic doping on a State run basis, and the most interesting fact is that the East German doping records! Further, the book takes a look at the next big issue surrounding drugs in sport—genetic engineering.

Dying to win does not just describe the evolution of doping. It explains the complex relationship between anti-doping policy, implementation of those policies, and the role of governments, the IOC, and international and national sporting organisations. With the ever increasing involvement of the legal profession, a vicious circle occurs: it becomes too costly for sporting organisations to fight court battles, with their reliance on Government funding depending on results and punishments set in accordance with what will stand up in courts. This all leads to the relative inertia of the governing bodies to be pro-active in the anti-drugs campaign.

The inception of the World Anti-Doping Agency (WADA) after the 1998 Winter Olympics has provided a way forward to standardise and implement anti-doping policy across the world by an independent body.

Problems and solutions to anti-doping policy are addressed. The major problem is inadequate definition of doping—to quote Arthur Gold “The definition lies not in words but in integrity of character.” It is interesting to note that those behind the athlete, namely the coach, administrators, medical profession, and scientists, all seem to lose perspective along with their ethics and “integrity of character” when the race for “gold” is on. Dying to win suggests that these people should be held just as accountable for their athletes themselves. Another unfortunate aspect of anti-doping policy is the difficulty in detecting some abused drugs and the fact that these strategies often lag behind the ability of the pharmaceutical industry to develop new drugs, often for genuine medical reasons but with the unfortunate ability to enhance performance. Education is proposed as a key aspect to anti-doping policy, and parallels with its success in the use of recreational drugs are made. Governments also play a role in limiting supply, decreasing demand for drugs, and the implementation of independent bodies to carry out drug testing. The success of anti-doping policy is also hard to measure. Fewer positive tests may simply reflect a move to less detectable methods rather than a decrease in use, and success may be measured in terms of fewer world records.

Dying to win gives an accurate account of the problem of doping in sport and the difficulties and complexities in finding solutions to the problems. It makes interesting and provocative reading for all who are interested in sport, from the athlete and coach to the sport administrator, the medical profession, and governments.

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CORRECTIONS

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Sran M M. To treat or not to treat: new evidence for the effectiveness of manual therapy (Br J Sports Med 2004;38:388–94). The multiple regression equation within the Abstract section of this paper was published incorrectly. The correct equation is:

\[ HSR = 37.79 - (0.33SHT + 10.05SSP + 2.24STE) \pm 2.34 \]

We apologise for this error.