BMJ 1999;319:514 (21 August)

Letters

Hyponatraemia after orthopaedic surgery

Failsafe system is needed

EDITOR—There are errors and omissions in Lane and Allen's editorial about hyponatraemia[1] and the subsequent responses in the *eBMJ*.[2] The assumption that orthopaedic patients are badly managed has been robustly criticised by those who are insulted about allegations of poor care in this specialty.

What is it about the patients that may put them at risk? Firstly, orthopaedic patients probably represent the largest group of elderly patients undergoing major and emergency surgery. Secondly, many take non-steroidal anti-inflammatory drugs. Thirdly, the use of spinal anaesthesia may result in too much intravenous fluid being given to counteract the effects of sympathetic block. The discussion about the role of the angiotensin-renin system, though interesting, is not relevant to the pathogenesis of hyponatraemia in these circumstances. An old patient who takes non-steroidal anti-inflammatory drugs and receives too much free water while the adrenal axis is affected by a spinal block will probably have problems with sodium balance. The tragedy is that it takes a clinical disaster and an editorial to spell this out.

The adverse outcome that prompted the editorial was an example of a failed system. There are always several steps in a critical incident that lead to adverse outcomes. The solution is to have a failsafe system. We believe that protocols in fluid management have a role, and unless the junior medical staff understand fluid management (and recent changes in training can only make this harder to obtain) the job could be done by specialist nurses acting under the authority of senior medical staff. The issue then is which specialty these senior medical staff should be in: surgical,

anaesthetic, or geriatric medicine?

Frail old women are nursed on busy surgical wards. They have their broken hips mended late in the evening and return to the wards when many of the nurses have gone home and the ones who remain are busy with high dependency nursing interventions on fitter patients having more radical surgery. Signs of hyponatraemia in such patients may be mistaken for postoperative confusion.

Notwithstanding its limitations and the expected criticism, we are pleased that the editorial has alerted the profession to the problem, even though we believe that the proposed solution is inappropriate. A system that fails to prevent progressive symptomatic hyponatraemia should not be further challenged by the use of hypertonic infusions but should be replaced.

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1. Lane N, Allen K. Hyponatraemia after orthopaedic surgery. *BMJ* 1999; 318: 1363-1364 [Free Full Text]. (22 May.)
2. Electronic responses. Hyponatraemia after orthopaedic surgery. *eBMJ* 1999;318. (www.bmj.com/cgi/content/full/318/7195/1363#responses)

General journals must not alienate particular specialties

EDITOR—Lane and Allen report the case of an elderly friend who suffered brain damage after developing hyponatraemia; she had been given (inappropriately) 6 litres of hypotonic dextrose solution over 48 hours after total knee replacement surgery.[1] Solely from the evidence of this anecdote the authors leap to the conclusion that "too many orthopaedic surgeons seem unaware of the dangers of hyponatraemia."

Would it be too much to ask that the authors justify this conclusion with even one reference, or any evidence at all, other than the anecdotal history they describe? None of the references cited supports this conclusion. Only their last reference refers to a series of cases. Arieff collected details of 16 surgical patients over 10 years[2]; just two had undergone orthopaedic surgery.

Given that the management of fluid and electrolyte homoeostasis is core knowledge for every basic surgical trainee, surely it is not within the remit of the Royal College of Surgeons or the British Orthopaedic Association to publish guidelines on such a basic subject. It is surely impossible to pass the FRCS examination without thorough understanding of the principles of intravenous fluid replacement in surgical patients. Do the authors seriously suggest that some surgeons do not realise that giving 6 litres of 5% dextrose saline over 48 hours in a patient with frequent vomiting will lead to severe electrolyte imbalance?

I agree with the authors that iatrogenic hyponatraemia is inexcusable, and it is commendable that

they should highlight the incompetent management in the case described. They should not, though, make assumptions about orthopaedic surgeons in general because of the failings of an individual.

If the *BMJ* is directed towards a general readership (as readers are often told), might I suggest that the editor ensures that it does not alienate a particular specialty. The editorial appeared on the first page of the issue, and on the last page (in Minerva) we read: "Humans—and some orthopaedic surgeons —are characterised by their ability to do more than one thing at once... ."[3]

Such a joke might be acceptable in the give and take of day to day conversation, but seeing it in print is unacceptable. To me, at least, it appears gratuitously insulting.

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1. Lane N, Allen K. Hyponatraemia after orthopaedic surgery. *BMJ* 1999; 318: 1363-1364 [Free Full Text]. (22 May.)
2. Arieff AI. Hyponatremia convulsions, respiratory arrest, and permanent brain damage after elective surgery in healthy women. *N Engl J Med* 1986; 314: 1529-1535[Abstract].
3. Minerva. *BMJ* 1999; 318: 1430[Free Full Text]. (22 May.)

Rigorous audit and introduction of guidelines decreased hospital's figures

EDITOR—We sympathise with the unfortunate and possibly avoidable experience that Lane and Allen's elderly friend suffered.[1] In their editorial the authors warn against the dangers of isotonic dextrose and hypotonic solutions. We think it important to emphasise that dextrose saline is a putatively isotonic but in reality hypotonic solution, which can be harmful in elderly postoperative patients.

The audit by Tolias highlighting the potential problem in orthopaedic patients,[2] cited in the editorial, was carried out at our hospital. After the initial study the audit cycle has so far been completed twice (table). Initially junior orthopaedic staff were given guidelines at the start of their six month job to exercise caution in prescribing any fluids containing dextrose.

View this table: [in this window] [in a new window]	Summary table of audit data on orthopaedic patients who developed hyponatraemia*

After the first audit cycle we found that the use of such fluids had fallen, as had the incidence of iatrogenically caused or exacerbated hyponatraemia. Moreover, the associated mortality in these hyponatraemic patients had fallen. Continued "routine" postoperative prescribing of dextrose saline in theatres before the patients returned to their ward was, however, identified as a factor still

contributing to the problem. It was decided to stop using dextrose saline in our hospital altogether, in an attempt to force clinicians (both anaesthetists and orthopaedic trainees) to justify the indication for prescribing any dextrose. Although this decision met with some resistance, when completing the second audit cycle we found that use of infusions containing dextrose had fallen further, as had the incidence of iatrogenic hyponatraemia and its associated mortality.

We noted that these hyponatraemic patients tended to be frail and elderly—on average in their early 80s. The high mortality was not just related to hyponatraemia but reflected a high prevalence of comorbidity such as heart failure, ischaemic heart disease, and chest infection.

The dangers of hyponatraemia have been well documented, but perhaps the message has not been so well disseminated. At our hospital rigorous audit and the introduction of guidelines for junior medical staff were followed by a reduction in the incidence of and associated mortality from this postoperative complication.

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1. Lane N, Allen K. Hyponatraemia after orthopaedic surgery. *BMJ* 1999; 318: 1364-1365 [Free Full Text]. (22 May.)
2. Tolias CM. Severe hyponatraemia in elderly patients: cause for concern. *Ann R Coll Surg Engl* 1995; 77: 346-348[Medline].

Laboratory must play a part in patients' management

EDITOR—I have read the wounded response of the orthopaedic surgeons, the "I told you so" response of the anaesthetists, the lone voice of a chemical pathologist, and the responses of others[1] to Lane and Allen's reminder of the dangers of postoperative hyponatraemia.[2] It is not surprising that nobody mentioned the role of the hospital laboratory, and only one respondent remembered his clinical pathology teacher.

In my practice as a consultant chemical pathologist any patient with a serum sodium concentration below 125 mmol/l gets a visit from me or one of my resident doctors; at worst, a direct telephone call is made to one of the doctors managing the patient. During the visit or telephone call, possible causes, further investigations, and further management are often discussed. Although the responses of the clinicians vary from outright resentment to cynicism and occasional gratitude (usually from very junior doctors), the advice given is rarely ignored.

Lane and Allen did not elaborate on the role (if any) of the hospital laboratory, but problems of postoperative fluid balance and hyponatraemia are quite common; a multidisciplinary approach to teaching (of medical students and junior doctors) and management of patients is best. Those

"experts" who ignore this approach are the ones mainly responsible for fatal iatrogenic hyponatraemia.

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1. Electronic responses. Hyponatraemia after orthopaedic surgery. *eBMJ* 1999;318. (www.bmj.com/cgi/content/full/318/7195/1363#responses)
2. Lane N, Allen K. Hyponatraemia after orthopaedic surgery. *BMJ* 1999; 318: 1363-1364 [Free Full Text]. (22 May.)

Hypotonic solutions should be used infrequently

EDITOR—Lane and Allen bemoan the scatter of articles on postoperative hyponatraemia and say that it contributes to continuing ignorance about the frequency and seriousness of postoperative hyponatraemia.[1] Colleagues and I published a prospective study of severe hyponatraemia in hospital inpatients 21 years ago.[2] Ten of 44 cases of plasma sodium concentration below 125 mmol/l were due to postoperative administration of intravenous 5% dextrose, with five of these patients also taking diuretics.[2]

Rather than opt for publications for each surgical and anaesthetic specialty, we need to address the two misconceptions that, in my view, drive the seemingly unending epidemic of postoperative hyponatraemia. The first misconception is the long established and overstated fear of inducing fluid overload by using physiological saline. I believe that fluid overload is unlikely to occur when saline is given with care in patients well enough to undergo surgery.

The second misconception is the invocation of the syndrome of inappropriate secretion of antidiuretic hormone; this diagnosis should only be made in euvolaemic patients. Not only are patients likely to have a volume deficit postoperatively but they also may have high levels of antidiuretic hormone as a consequence of nausea, stress, and drugs. It is unhelpful conceptually, as well as almost always inaccurate, to make this diagnosis. In these circumstances the fault lies not in our patient's hypothalamus or posterior pituitary but in ourselves.

I agree with Ayus and Arieff that hypotonic solutions should be used infrequently and reserved for suspected free water deficits (and perhaps carry a government health warning).[3]

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1. Lane N, Allen K. Hyponatraemia after orthopaedic surgery. *BMJ* 1999; 318: 1364-1365 [Free Full Text]. (22 May.)
2. Kennedy PGE, Mitchell DM, Hoffbrand BI. Severe hyponatraemia in hospital inpatients. *BMJ* 1978; ii: 1251-1253.
3. Ayus JC, Arieff AI. Brain damage and postoperative hyponatremia: the role of gender. *Neurology* 1996; 46: 323-328[Medline].

Authors' reply

EDITOR—We are pleased that our editorial has stimulated debate about this important issue. We did not set out to wound orthopaedic surgeons or cause gratuitous offence; but for space limitations, we might easily have addressed other specialties. Nevertheless, as Severn and Dodds point out, orthopaedic patients are the largest group of frail elderly people undergoing surgery and as such are at high risk of hyponatraemia. Regardless of the exact mechanisms, water retention is probable and insensitive perioperative fluid management may have serious consequences.

Most orthopaedic patients do not become hyponatraemic after surgery. Perhaps the low incidence of hyponatraemia persuades Harrington and others to question whether there is a problem. The audit figures reported by Marino et al are revealing. In the first cycle about 1% of their patients developed postoperative hyponatraemia, after infusions of dextrose saline in a majority of cases. A third of these patients died. In other words, the condition is relatively uncommon, but when it does occur the consequences are often catastrophic. The estimate we cited of 10 000-15 000 cases a year is based on very similar figures: a 1-2% risk of postoperative hyponatraemia, with a fifth of symptomatic patients dying or developing permanent brain damage.[1] The large number of people at risk reflects the high throughput of orthopaedic patients.

We agree with Severn and Dodds that fundamentally the system is at fault. Specialist nurses may well be in the best position to supervise perioperative infusions. At present, perioperative fluid management is barely mentioned in medical schools' curriculums. House staff learn on the job, usually without the benefit of formal protocols, before moving on. No specialty claims overall responsibility. We would support an overhaul of this failing system, but we maintain that junior staff would benefit from formal, multidisciplinary training or at least simple guidelines. Mojiminiyi is right to remind us that false pride can prove fatal.

The audits of Marino et al show the utility of guidelines. The figures cited in their second and third audit cycles show substantial reductions in the number of patients developing hyponatraemia and in the associated mortality. The reluctance of clinicians in their hospital to give up infusing dextrose saline is symptomatic of entrenched practices and shows how much needs to be done to change attitudes. Increased awareness and vigilance are a useful start.

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1. Fraser CL, Arieff AI. Epidemiology, pathophysiology and management of hyponatremic encephalopathy. *Am J Med* 1997; 102: 67-77[Medline].

Summary of electronic responses

The editorial by Lane and Allen [1] evoked an emotional response.[2] Of the 24 electronic responses received, nine authors were upset by the attack on orthopaedic surgeons. Six of these were orthopaedic surgeons, but the feeling was shared by other specialists.

1. Lane N, Allen K. Hyponatraemia after orthopaedic surgery. *BMJ* 1999; 318: 1363-1364 [Free Full Text]. (22 May.)
2. Electronic responses. Hyponatraemia after orthopaedic surgery. *eBMJ* 1999;318. (www.bmj.com/cgi/content/full/318/7195/1363#responses) [Accessed 30 July 1999]

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