Is maintenance fluid therapy in need of maintenance?

David J Gattas and Manoj K Saxena

Acute illness can cause decreased fluid intake and increased fluid and electrolyte loss, and can be associated with alterations in cardiovascular and other vital organ functions. The stress response to acute illness activates inflammatory, endocrine and other pathways that result in the retention of sodium and water. In this context, what are the daily sodium and water needs of a critically ill patient, and how are they different to normal requirements for salt and water intake during recovery and in health? And if these needs are different, how do we recognise the clinical transition from stress to recovery phase?

In health, the average daily sodium intake for adults in Australia and New Zealand is around 150 mmol/day. The National Health and Medical Research Council recommend an intake of between 20 and 40 mmol/day, and up to 100 mmol/day is considered reasonable.[1] Sodium balance is regulated in part by the renin–angiotensin–aldosterone and sympathetic nervous systems. Intrarenal mechanisms also contribute via regulation of intramedullary and regional renal blood flow. Daily sodium intake is balanced by daily excretion through urinary and cutaneous (sweat) losses, although other mineral intakes (potassium and calcium) have effects on urinary loss of sodium.

In this issue of *Critical Care and Resuscitation,* Bihari and colleagues describe the epidemiology of sodium administration among a broad cross-section of intensive care unit patients in Australia and New Zealand.[2] Previously, in the findings of a single-centre exploratory study,[3] two of these authors have reported that sodium and fluid balance are not predictably related in ICU patients and that sodium accumulation may be associated with morbidity. The current report extends our understanding of sodium and fluid intake during the usual clinical care of critically ill patients. It also demonstrates the value of investing in research infrastructure and capacity — in this case, the Australian and New Zealand Intensive Care Society Clinical Trials Group (ANZICS CTG) Point Prevalence Program.[4-6]

This report describes sodium intake in 356 patients from 40 Australian and New Zealand ICUs on a single day and has several key findings. First, the median amount of administered sodium was 224.5 mmol/day (interquartile range, 144.9–367.6 mmol/day), which is well in excess of the recommended daily sodium intake in health. Second, this daily dose appears to persist throughout intensive care admission. Third, although maintenance fluid is the main source of sodium administration (around 35%), a significant amount of sodium is administered with fluid boluses (around 20%), in vehicles for drugs and in flushes (around 30%). Fourth, sodium administration varies markedly among different ICUs, suggesting significant practice variation as part of usual care. Finally, hypernatraemia was more prevalent than hyponatraemia.

We administer fluid and sodium to patients concurrently, but ICU clinicians think and prescribe predominantly in terms of fluid volume only. There is clinical evidence of an association between positive cumulative fluid balance and adverse clinical outcomes in ICU patients,[7-9] but these data from Bihari and coworkers raise the question of whether sodium balance is also important to help understand the association between fluid accumulation and morbidity. Is it possible that maintenance fluid (together with the fluid used as a vehicle for drugs and electrolytes) is an iatrogenic factor that "maintains" sodium and fluid overload? Is maintenance fluid an intervention whose volume and composition can influence the outcomes of patients in hospital?

Bihari et al also observed that bolus fluid resuscitation was the second most significant source of intravenous sodium input, particularly on the second and third days after intensive care admission. The requirement for bolus fluid resuscitation varies during the course of critical illness, and clinicians determine whether bolus fluid is indicated using vital signs and their assessment of perfusion. However, recent clinical investigation is making it less clear what the net benefits of bolus fluid administration are for patients in the ICU. Colloids offer no general superiority over crystalloids.[10] Specific colloids are associated with harm.[11] Bolus fluid resuscitation is an established practice for early management of sepsis[12] (perhaps albumin has an advantage[13]), but there is a case to be made for reviewing established practice in this diagnostic group.[14] How should we apply evidence from a recent high-quality, large-scale clinical trial which showed that bolus fluid resuscitation (albumin or saline) is associated with increased mortality in children with severe febrile illness presenting to resource-limited clinics in Africa?[15] The study by Bihari et al documents that sodium administration is substantial in ICU patients and suggests an association with some adverse outcomes. We cannot be certain of the importance of these findings, but they contribute to an emerging theme of unclear benefits and possible harms associated with liberal bolus fluid use.

An impressive cross-section of patients has been arrayed from across two countries on a single day, but the study is limited by its inability to report sodium balance; some sources

of sodium input have not been captured, and there are no data for sodium output. The ability of these data to associate sodium with patient outcomes is hypothesis-generating at best. There is no reason to think that recommended daily intake for sodium during health should apply in critical illness, but the physiological rationale for reducing high and persistent levels of sodium administration in the recovery phase of critical illness is plausible. We need to ask the question: what are we maintaining with maintenance fluid? The unknown medical wit who said that the dumbest kidney is still smarter than the smartest doctor may be correct, but this is no excuse for us to stop trying to constantly improve fluid administration to our critically ill patients.

Competing interests

None declared.

Author details

David J Gattas, Intensive Care Physician,[1] Clinical Associate Professor,[2] and Honorary Fellow[3]

Manoj K Saxena, Research Fellow,[3] and Intensive Care Physician[4,5]

1 Intensive Care, Royal Prince Alfred Hospital, Sydney, NSW, Australia.

2 Sydney Medical School, University of Sydney, Sydney, NSW, Australia.

3 Critical Care and Trauma Division, The George Institute for Global Health, Sydney, NSW, Australia.

4 Department of Intensive Care Medicine, St George Hospital, Sydney, NSW, Australia.

5 St George Hospital Clinical School, University of New South Wales, Sydney, NSW, Australia.

Correspondence: david.gattas@sydney.edu.au

References

1 Australian Government National Health and Medical Research Council, New Zealand Ministry of Health. Sodium. Nutrient reference values for Australia and New Zealand including recommended dietary intakes. http://www.nrv.gov.au/resources/_files/n35-sodium.pdf (accessed Sep 2013).

2 Bihari S, Peake SL, Seppelt I, et al. Sodium administration in critically ill patients in Australia and New Zealand: a multicentre point-prevalence study. *Crit Care Resusc* 2013; 15: 294-300.

3 Bihari S, Baldwin CE, Bersten AD. Fluid balance does not predict estimated sodium balance in critically ill mechanically ventilated patients. *Crit Care Resusc* 2013; 15: 89-96.

4 The George Institute for Global Health. Point Prevalence Program. http://www.georgeinstitute.org.au/projects/point-prevalence-program (accessed Oct 2013).

5 Elliott D, Aitken LM, Bucknall TK, et al. Patient comfort in the intensive care unit: a multicentre, binational point prevalence study of analgesia, sedation and delirium management. *Crit Care Resusc* 2013; 15: 213-9.

6 Hammond NE, Saxena MK, Taylor C, et al. Temperature management of non-elective intensive care patients without neurological abnormalities: a point prevalence study of practice in Australia and New Zealand. *Crit Care Resusc* 2013; 15: 228-33.

7 Boyd JH, Forbes J, Nakada TA, et al. Fluid resuscitation in septic shock: a positive fluid balance and elevated central venous pressure are associated with increased mortality. *Crit Care Med* 2011; 39: 259-65.

8 Wiedemann HP, Wheeler AP, Bernard GR, et al. Comparison of two fluid-management strategies in acute lung injury. *N Engl J Med* 2006; 354: 2564-75.

9 Murphy CV, Schramm GE, Doherty JA, et al. The importance of fluid management in acute lung injury secondary to septic shock. *Chest* 2009; 136: 102-9.

10 Perel P, Roberts I, Ker K. Colloids versus crystalloids for fluid resuscitation in critically ill patients. *Cochrane Database Syst Rev* 2013; (2): CD000567.

11 Perner A, Haase N, Guttormsen AB, et al. Hydroxyethyl starch 130/0.42 versus Ringer's acetate in severe sepsis. *N Engl J Med* 2012; 367: 124-34.

12 Dellinger RP, Levy MM, Rhodes A, et al. Surviving Sepsis Campaign: International Guidelines for Management of Severe Sepsis and Septic Shock: 2012. *Crit Care Med* 2013; 41: 580-637.

13 Delaney AP, Dan A, McCaffrey J, Finfer S. The role of albumin as a resuscitation fluid for patients with sepsis: a systematic review and meta-analysis. *Crit Care Med* 2011; 39: 386-91.

14 Hilton AK, Bellomo R. A critique of fluid bolus resuscitation in severe sepsis. *Crit Care* 2012; 16: 302.

15 Maitland K, Kiguli S, Opoka RO, et al. Mortality after fluid bolus in African children with severe infection. *N Engl J Med* 2011; 364: 2483-95. ❑