K
aren Yeates and associates,1 in their article on the management of hyponatremia, use the terms “serum osmolality” and “tonicity” interchangeably, a common practice. Although there is not a major difference in meaning, it is important to differentiate these terms in this context. Tonicity is effective serum osmolality and is equal to serum osmolality minus the concentration of ineffective osmolytes (mainly urea), since urea can diffuse in and out of the cell and is not an effective osmole. 

In the algorithm for the management of hyponatremia (Fig. 1 of the paper), Yeates and associates advise assessing extracellular fluid (ECF) volume status after initial treatment of symptomatic acute or chronic hyponatremia, but this should be done before treatment is started. In cases of acute hyponatremia, treatment would not have any ill effects, but if the hyponatremia is chronic and is treated aggressively, the consequences could be fatal, especially in women.2 In addition, aggressive treatment of chronic hyponatremia secondary to syndrome of inappropriate secretion of antidiuretic hormone (SIADH) might lead to a worsening of the hyponatremia,3 as alluded to by Yeates and associates in the text of their article.1 If urine electrolyte levels are determined after treatment (i.e., after volume repletion), the results are often equivocal and thus may not be helpful in patient management.

In the section “The case revisited,” the authors recommend an alternative medication to treat the patient’s systolic hypertension. However, the patient is described as having taken a thiazide diuretic for 5 years with no previous history of hyponatremia. The acute episode of hyponatremia had a clear cause: volume depletion secondary to gastroenteritis and volume replacement with free water. It would be more appropriate to withhold the diuretic until the acute illness had resolved and to reintroduce it with caution, rather than changing the drug entirely.

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References

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References
6. Decaux G, Musch W, Soupart A. Hyponatremia
that aggressive treatment of chronic hyponatremia in the setting of SIADH could lead to worsening of the hyponatremia. We agree and stated this in our article. However, seizures, obtundation and ataxia secondary to hyponatremia are all considered medical emergencies and thus require therapy. In the case of SIADH, infusion of normal (0.9%) saline may not improve sodium level and may in fact worsen it; hence, our recommendation for hypertonic (3%) saline in emergent situations. Unless the patient presents with a clear cause for the SIADH, it is impossible to know how to direct the therapy, especially if there is neurologic deterioration.

Parmar disagrees with our suggestion to use an alternative medication to treat the case patient’s hypertension. We agree that the hyponatremia in this case was a direct result of ECF volume depletion due to gastroenteritis and replacement of that fluid loss with free water. Parmar’s suggestion to reintroduce thiazide as the patient’s diuretic would be a reasonable approach, if combined with appropriate close monitoring for hyponatremia soon after reinitiating the drug. An alternative choice would be an angiotensin II receptor blocker (ARB) or a long-acting dihydropyridine calcium channel blocker, both of which have been suggested as first-line therapy for patients with isolated systolic hypertension. If an ARB were initiated, the patient would have to receive instructions to stop the drug should ECF volume become contracted.

Guy Decaux and colleagues express concern about our suggestion that hyponatremia “be corrected at a rate similar to that over which it developed.” With this recommendation we were attempting to provide a very general rule for correction rates and intended to imply that if hyponatremia has been clearly documented to have developed over a 2- or 3-day period, then in most circumstances it can be corrected over 2 to 3 days, provided the patient does not have life-threatening signs or symptoms. Likewise, if hyponatremia has developed over several weeks, then it can be corrected much more slowly (although not necessarily over a 2- to 3-week period, since it may be adequately treated in a shorter period).

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### References


### Queuing for cardiac surgery

Gerry Hill’s analysis of queuing for cardiac surgery has already been critiqued by David Naylor and associates, but several points deserve further clarification.

Hill’s main finding — that the number of deaths in line per year (D) is independent of queuing strategy — is simply a tautology. By assuming a steady state in which N patients join the queue and S are treated yearly, Hill guarantees that 

\[ D = N - S, \]

which is constant. Hill is incorrect in criticizing the prioritization of high-risk patients on the grounds that this strategy increases the size of the queue without reducing the number of deaths. Suppose that it takes \( n \) years to reach a steady state. At that point, \( nS \) patients have been treated, which means that \( m(N - S) \) patients have entered the queue but have not been treated. Of these, \( Q \) are alive and the rest are dead. That is, the waiting list is longer if high-risk patients are prioritized precisely because fewer patients die before steady state is reached.

Most important, Hill’s model does not consider death from noncardiac causes. Consider a refined model in which the mortality rates of treated patients, low-risk patients and high-risk