mal range that cardiac output begins to further decline.[7]

I would respectfully suggest that the term "low effective circulating volume" be expunged from the heart failure lexicon. Not only is it physiologically unsound, but it also invites the misperception that high renal function indices in advanced congestive heart failure are a sign of extracellular volume contraction.

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Karen 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 osmoles (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[1] 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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As discussed by Karen Yeates and associates[1] in their review of hyponatremia, evaluation of extracellular volume is sometimes difficult. In a patient with hyponatremia, a trial of saline infusion may be useful in clarifying the diagnosis; however, contrary to information in the review, most patients with SIADH will not experience worsening of hyponatremia after infusion of isotonic saline. We found that only 30% of 33 consecutive patients with SIADH had a decrease in plasma sodium levels after infusion of 2 L of isotonic saline over 24 hours;[2] our observations were similar for patients with urine osmolality above 530 mOsm/L.[3]

Yeates and associates[1] state that "hyponatremia should be corrected at a rate similar to that at which it developed," but this recommendation could be misleading. In the classical model used to induce osmotic demyelination syndrome in hyponatremic rats, initial serum sodium level was 142 mmol/L and decreased to 115 mmol/L after 24 hours, 113 mmol/L after 48 hours and 110 mmol/L after 72 hours.[4] If the sodium level were to be corrected on the first day by 3 mmol/L, on the second day by 2 mmol/L and on the third day by 27 mmol/L, severe brain damage would develop, despite a correction rate similar to the rate of induction of hyponatremia. For the long-term management of SIADH in cases where water restriction is ineffective, we use demeclocycline, urea or furosemide, although immediate introduction of oral vasopressin V2 receptor antagonists may make management easier.[5,6]

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[The authors respond:]

Philip Andrew eloquently explains the problems of referring to circulating plasma volume as "effective," and we concur with his comments. Our reference to low effective circulating volume in heart failure[1] implicitly suggests that cardiac failure and low cardiac output are the underlying problems leading to poor renal perfusion and, as Andrew states, to neurohormonal activation with elevation of natriuretic peptides.

Our article[1] also refers to the other states of ECF volume overload (cirrhosis and nephrotic syndrome). In these situations, low effective circulating volume refers to low oncotic pressure, which is entirely different from the mechanisms seen in a state of low cardiac output. We agree that the term "low effective circulating volume" is used in our article to describe 2 different disease states and might lead to confusion.

While acknowledging the fact that "tonicity" and "osmolality" are frequently used interchangeably, Malvinder Parmar provides the correct definitions.

Parmar has concerns about the suggestion in our management algorithm[1] that ECF volume be assessed after initial treatment of symptomatic acute or chronic hyponatremia and suggests that this assessment should be carried out before treatment is initiated. However, in clinical practice, in an emergent situation, assessment and treatment occur concurrently. Our algorithm is intended to suggest urgent treatment of a constellation of severe signs and symptoms of hyponatremia (confusion, ataxia, headache, seizures, obtundation); such urgent therapy will be essentially the same regardless of the ECF volume. We agree that headache in and of itself should not be an indication to pre-empt appropriate physical assessment before infusion of hypertonic or normal saline. Parmar also mentions 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.[1] 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.[2] 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, than 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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Queuing for cardiac surgery

Gerry Hill's analysis of queuing for cardiac surgery[1] has already been critiqued by David Naylor and associates,[2] 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 $n(N - S)$ 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