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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