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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

patients are m_0, m_1 and m_2, where $m_0 < m_1 < m_2$. With this model, it is not difficult to show that prioritizing high-risk cases leads to fewer cardiac deaths and greater overall survival. The number of deaths on the waiting list in steady state is still independent of queuing strategy, but if high-risk cases are prioritized, then a greater proportion of waiting-list deaths are of noncardiac origin.

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[The author responds:]

In this letter, I comment on both the commentary by David Naylor and associates,[1] published in conjunction with my own commentary[2] and on the subsequent letter from John Neary.

I have had difficulty responding to the "critique" by Naylor and associates[1] because it is clouded in rhetoric. To help readers decide if my analysis was "flawed," I would like to take this opportunity to spell it out in more detail.

The model I used was a deterministic version of the immigration–death process, a continuous-time, discrete-space Markov process.[3] With reasonably large numbers, the result for the deterministic model is the same as the mean of the stochastic model. In the context of the waiting list problem, let $N(t)$ be the instantaneous rate of admission to the list (expressed as patients per year), $S(t)$ the rate of surgery and m the mortality rate of patients on the waiting list (deaths per patient-year). Suppose that before time zero, $S(t) = N(t)$, such that no patients are waiting, but that at time zero, $S(t)$ falls below $N(t)$ and the difference remains constant at, say, $N - S = D$. Then the differential equation for $Q(t)$, the size of the waiting list at time t, is $dQ/dt = D - mQ(t)$. The solution is $Q(t) = [1 - \exp(-mt)]D/m$. Thus, as t increases, $Q(t)$ approaches exponentially the steady-state value $Q = D/m$, and the number of deaths per year in the steady state is $mQ = D$ (constant). If we compare the steady states reached with different values of m, we find that the size of the waiting list will vary inversely with m, but the number of deaths per year will remain the same. For example, if m is reduced by operating preferentially on patients with a greater risk of dying, then *in the steady state* the size of the waiting list is increased but the number of deaths per year is unaltered (emphasis has been added here because Neary counts deaths en route to the steady state and gets a different result). In my commentary, I used the example of 2 risk groups, but the analysis can be extended to any number of groups, and to the situation of progression between groups, with the same conclusion.

The same model underlies the formula used in epidemiology: prevalence = incidence × duration. If the disease is incurable but palliative treatment reduces the mortality rate of those with the disease, then the prevalence of the disease increases but the number of deaths per year remains the same.

Both Naylor and associates[1] and Neary state that my argument is tautological, and I agree. All mathematical models are tautologies, but they sometimes produce results that are not intuitively obvious and that can be especially useful if the conclusions can be tested empirically. This is true in principle in this case, but the data may be difficult to obtain.

Neary's suggestion that cardiac deaths be distinguished from deaths due to other causes adds an interesting dimension to the analysis. If priority is given to patients with increased cardiac risk, then the proportion of cardiac deaths will be reduced, even though the total number of deaths per year is unchanged. In effect, deaths from other causes are substituted for cardiac deaths. The proportion of noncardiac deaths is thus a measure of the effectiveness of the selection process. (For more mathematical detail, see the online appendix to this letter at **www.cmaj.ca**.) Plomp and colleagues[4] have published a breakdown of deaths by cause on Dutch waiting lists, and it would be interesting to compare their results with data from other jurisdictions.

Recently the federal and provincial governments have committed themselves to getting rid of waiting lists. If they attempt to do so by making $S(t) = N(t)$, then the waiting list will remain until patient deaths reduce it to zero. If it took 15 years for the waiting list to develop, then it will take 15 years to eliminate it. To avoid the delay, an oversupply of resources will be needed.

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Clarification

CMAJ wishes to clarify statements made in an editorial "What's wrong with CME?" published on March 16, 2004. Contrary to what was published, mdBriefCase and sponsoring pharmaceutical companies agree on the clinical area to be covered by the case study but do not choose the specific CME topic, design the course content or select the course leaders.

John Hoey
Editor
CMAJ

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