Acute decompensated heart failure

Although Larry Allen and Christopher O'Connor's review of the management of acute decompensated heart failure[1] was generally well written, I have reservations about their interpretation of the evidence concerning the role of loop diuretics, specifically furosemide, and their recommendations regarding furosemide's place in the treatment of acute decompensated heart failure.

First, I disagree with their implication that congestion indicates volume overload and their suggestion that clinicians consequently "rely heavily on diuretic therapy." Such statements help to perpetuate the misuse of furosemide in acute decompensated heart failure. Up to 50% of patients with acute cardiogenic pulmonary edema are euvolemic, and treatment should emphasize fluid redistribution rather than fluid removal. Second, although diuretics have been weakly shown to decrease mortality,[2] if they are relied upon exclusively in acute care in hospitals they have the opposite effect. Third, in acute decompensated heart failure caused by high afterload (e.g., hypertensive emergencies), renal perfusion can drop by as much as 80% and furosemide will produce a delayed diuretic effect 30–120 minutes after administration. Finally, there is very little evidence for any beneficial hemodynamic effect of furosemide. In fact, many studies have shown that furosemide is responsible for adverse hemodynamic effects in patients with acute decompensated heart failure, because it causes an initial release of catecholamines and activates the renin–angiotensin system.[3–5]

In conclusion, I disagree with the authors' summary that "therapy with a loop diuretic currently forms the foundation" of treatment of acute decompensated heart failure despite good evidence that loop diuretics should be reserved for use as third-line agents behind therapies to reduce preload and afterload (e.g., nitroglycerin and angiotensin-converting-enzyme inhibitors) in acute decompensated heart failure in the hospital setting.

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I read with interest the review article on the management of acute decompensated heart failure by Larry Allen and Christopher O'Connor.[1] The authors commented that nitroglycerin is probably underused in patients presenting with acute decompensated heart failure. However, in Table 1, in which they recommend switching from sublingual to intravenous delivery of nitroglycerin, the dose they suggest for initiating intravenous delivery seems low. A sublingual regimen of 0.4 mg every 5 minutes (the typical dose at my institution) is mathematically equivalent to 80 μg/min.

The bioavailability of sublingual nitroglycerin is cited in a 1998 study as 38%,[2] but it can be highly variable.[3] In many patients the tablets appear to be absorbed quickly and completely, but not infrequently one finds a tablet that remains undissolved after 5 minutes. In the latter circumstance, a lower dose of intravenous nitroglycerin, such as that recommended by the authors, would be suitable. In patients with marked hypertension not responding to completely dissolved sublingual nitroglycerin, a higher starting dose may be more appropriate.

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[The authors respond:]

In our review, in particular in the section on loop diuretics, we attempted to outline many of the controversies associated with the treatment of volume overload in acute decompensated heart failure.[1] We provided 3 references to support the statement that use of loop diuretics in the acute setting is associated with increased mortality. We highlighted the potentially detrimental vasoconstrictive properties of furosemide. We also recognized the importance of alternative approaches, including therapy with vasodilators and positive airway pressure.

Joe Nemeth is correct that elevation