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Cardiogenic Shock

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To the Editor:

Drs. Wilkes and Navickis are incorrect that it is the institutional policy of our hospital to encourage the use of hetastarch over albumin. The clinical algorithm our division prepared on this topic was revised in light of the bleeding risk we reported in our publication.[1] We do not disagree with the contention that hetastarch probably increases the risk of postoperative bleeding; this was, after all, the main conclusion of our article. Our main point was not to criticize their metaanalysis on this subject, nor their other work supported by the manufacturers of albumin products; the methodologic concerns we cited were primarily raised by others.[2,3] However, we do not agree with their conclusion that it is time to routinely replace hetastarch with plasma protein products. Clinical policy about such an important matter should be based on large, impeccably conducted randomized controlled clinical trials that compare the relevant choices head-to-head for safety and efficacy. That is particularly necessary in this case because of the important countervailing risks that may be associated with albumin use. A systematic review of randomized controlled trials published in the *British Medical Journal* by the Cochrane Collaborative found increased mortality rates in critically ill patients randomized to receive albumin.[4] That review was recently updated to include a total of 31 randomized controlled trials; this more recent analysis reported a relative risk for death of 1.52 (95% confidence interval, 1.17 to 1.99) in patients randomized to receive albumin vs no albumin, or albumin vs crystalloid. The authors of that report concluded that albumin use would produce one extra death for every 20 critically ill patients receiving albumin.[5]

As the experience with hormone replacement therapy amply demonstrates,[6] even carefully conducted epidemiologic studies such as our own cannot exclude the possibility that patient selection and other confounders might produce incorrect conclusions about the efficacy or safety of commonly used medications. Given the frequency of use of colloids and the importance of the risks being considered, it is time for a randomized trial comparing hetastarch with albumin to resolve these issues once and for all. Studies of this kind have begun to appear in the literature.[7] It is a major failing of our current system for the evaluation of drugs and biological agents that no mechanism exists to ensure that such comparative studies are done promptly, thoroughly, and even-handedly as a matter of routine policy.[8]

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Cardiogenic Shock

To the Editor:

We wish to clarify a few points in the data presented by Lim and colleagues (November 2003).[1] Eighteen patients presented with acute myocardial infarction that developed into cardiogenic shock (CS). No mention is made about the method of reperfusion

or revascularization, information that is absolutely essential in light of the findings of the Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock (SHOCK) trial.[2] An institutional protocol that uses intra-aortic balloon pumps (IABPs) only in those patients with refractory CS, despite the use of therapy with vasopressors, is not dispensing state-of-the art care to patients with CS. While the benefit of therapy with vasopressors on outcome in patients with CS is doubtful, a survival benefit is evident in patients who are supported with an IABP, and this is included in the current American College of Cardiology/American Heart Association guidelines. Data from the SHOCK trial and Registry demonstrated that the reversal of systemic hypoperfusion following IABP therapy is associated with improved 30-day survival, independent of early revascularization.[3]

Hochman also has proposed alterations to the current model and has suggested expanding the paradigm in a recent review.[4] Large myocardial infarctions that are complicated by CS may be accompanied by a substantial inflammatory response with the release of various mediators, including cytokines, leading to high levels of nitric oxide and peroxynitrite with deleterious effects. A nitric oxide synthase inhibitor will be utilized in the SHOCK-2 trial to test this hypothesis.

A recent concept introduced into the lexicon of shock terminology is the cardiac power output. This parameter is calculated by multiplying the mean arterial pressure by the cardiac output. This parameter was found on multivariate analysis to be the single hemodynamic factor associated with in-hospital mortality among patients in the SHOCK Registry.[5] It would be interesting to know whether Lim and colleagues will confirm this finding in their cohort of patients as they have data on the mean arterial pressure and CO for each patient.

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To the Editor:

We thank Drs. Munoz and Thomas for their comments on our article. Indeed, the 18 patients with acute myocardial infarction whose conditions evolved into cardiogenic shock were treated according to the guidelines that were in effect at the time of the study. The patients were admitted to the hospital in one of the following three ways: (1) most were revascularized using percutaneous angioplasty unless coronary angiography disclosed diffuse and/or distal lesions; (2) several patients developed shock a few days after apparently successful thrombolysis; and (3) a small groups of patients did not receive any revascularization intervention due to the long delay between the onset of symptoms and hospital admission. All patients were treated with antiplatelet agents and heparin. Intra-aortic balloon counterpulsation was used when possible, but this procedure was contraindicated in some patients (in more nonsurvivors than survivors). These factors do not invalidate our findings, as the aim of the study was to describe the hemodynamic evolution of nonsurvivors, regardless of the results of any revascularization procedure they may have received. Nine of the 23 nonsurviving patients developed hyperdynamic shock, suggesting that pump failure was not the primary cause of death. As both BP and cardiac output were relatively preserved, cardiac power was not significantly decreased in these patients.

Finally, we agree, and indeed discussed briefly in our article, that nitric oxide and peroxynitrite may be involved in this process. It will be interesting to see the results of the SHOCK-2 trial.

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P Wave in Pulmonary Impairment

To the Editor:

We read with great interest the article by Asad et al in *CHEST* (August 2003)[1] on acute right atrial strain, and P-wave amplitude and axis in the treatment of obstructive airways disease in patients experiencing exacerbation. Their key message was the rapid reversal of characteristic ECG changes with treatment from the emergency department presentation to hospital ward admission. Similarly, in 1973, Carilli et al[2] demonstrated in a retrospective study the predictive value of P-wave amplitude and axis in estimating the severity of nonasthmatic airway obstructive disease in the quiescent state. A good correlation of P-wave amplitude and axis with FEV_1/FVC and residual volume/total lung capacity was seen, also demonstrating a continuum in regression equations. We agree with Yue et al,[3] in their accompanying editorial, that the study was well-designed but lacking in clinical and functional data. Patients with clinical phenotypes of diffuse obstructive airways disease (*ie*, chronic bronchitis/bronchiolitis, emphysema and bronchial asthma) are a clinically and pathophysiologically heterogeneous population. These various phenotypes most often coexist, and the proportion of each is difficult to quantitate clinically by pulmonary function testing and chest-imaging techniques.

The variability of airways obstruction is a defining criteria for the asthmatic type. Although these data are lacking in the