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patients who survived, there was a nonsignificant trend toward a higher proportion of patients who had a negative fluid balance > 500 mL on any of the first 3 days (12 of 51 patients), compared with those who died (2 of 29 patients; p = 0.072, two tailed).

These results, in an unselected cohort of critically ill surgical patients, do not confirm the strong prognostic value of a negative fluid balance > 500 mL on any of the first 3 days of hospital admission. The reasons for the conflicting results are unclear, as the mean APACHE II scores in the previous and present study (25 vs 22, respectively) and the percentage of patients who died (56% vs 36%, respectively) were similar.

In the present study, there were approximately twice the number of patients and twice the number of deaths. All the patients were either postoperative or had severe pancreatitis. This would have a major influence on the fluid requirements of the patients. Also, the normal stress response to surgery involves alterations in circulating hormone concentrations, with an anti-

We conclude that a negative fluid balance, achieved by the third day after admission to the ITU, is not a significant predictor of death in the critically ill surgical patient, and therefore, it is not generally applicable to critically ill patients.

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REFERENCE


To the Editor:

We appreciate the opportunity to respond to the interesting letter of Drs. Quasim, McMillan, and Kinsella. We are very pleased that these physicians took the time to interrogate our hypothesis.1 We published our observation because we are convinced that fluid fluxes are common in critically ill patients and that vascular leak and dilatation and the need for refilling are followed by a diuretic phase in those patients with sepsis who recover (it is inevitable). In our original report, we did not imply that these findings were conclusive, nor did we assert that they were applicable to any other than septic patients with sepsis. Nevertheless, a reanalysis of the data provided by Quasim et al.

demonstrates that there was a survival advantage for their postoperative patients who achieved a negative fluid balance in the first 3 days (relative risk = 1.45; 95% confidence interval = 1.08 to 1.94),2 although our studies differ merely in the magnitude of the observed survival advantage. This is to be expected for a number of reasons.

The average amount of fluid administered to our patients with septic shock in the first day was 5.5 L. We have not been given the average amount of fluid administered to their patients on the first day but we doubt that the magnitude was similar. Another possibility is that different surgeries have variable rates of recovery based upon the gravity of the initial injury and factors pertaining to the procedure (eg, duration of anesthesia, size of the wound, site of the operation). Insofar as the dispersion of vascular recovery times is greater in a heterogeneous group of postoperative patients, the dispersion of transition points from positive to negative balance would also be greater. The difference in observed death rate (56% in our study versus 21% in their cohort) may reflect differences in the severity of the systemic inflammatory response in the two patient groups—a factor that may impact the magnitude of vascular dilatation and/or leak and, therefore, outcomes. We are not provided with significant information about the characteristics of their patients. Differences in age, severity of illness, and baseline creatinine levels are bound to effect the magnitude of survival advantage, as demonstrated in Table 3 of our report.1 For instance, whereas patients with sepsis with negative fluid balance and APACHE II score < 20 were 1.3 times more likely to survive, those with negative balance and APACHE II score ≥ 20 were 6 times more likely to survive. Thus, any meaningful differences in the proportional representation of the groups with APACHE II scores < 20 and ≥ 20 would result in significant variations in the reported unadjusted risk ratios.

Accordingly, we are delighted that our findings have been replicated in studies with postoperative patients. We hope that larger cohort studies will further validate these findings and will provide more precise estimates of the true magnitude of survival advantage in various populations of critically ill patients.

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Does Omentoplasty Preclude Cardiac Retransplantation?

To the Editor:

Infectious complications are, with rejection, the main cause of morbidity and mortality in recipients of heart transplantation (HT). Between September 1984 and October 2000, we performed 514 HTs in 305 patients; of these, postoperative mediastinitis developed in 7 patients (1.4%). The mortality rate in this group of patients was 42%.

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Surgery, combined with antibiotic therapy and temporary reduction of immunosuppression, can successfully treat sternal wound infection after HT. Debridement irrigation technique has a low success rate, and usually a more aggressive technique is required. The use of a pedicled omental flap based on the right gastroepiploic artery appears to provide adequate bulk for obliteration of the large dead space that remains after debridement, and for us is the treatment of choice because of its greater rate of success.2,3

Nevertheless, omentoplasty, though effective and useful in treating mediastinitis, is also a relative contraindication for future cardiac reinterventions through median sternotomy. The omental tissue has an excellent blood supply that limits spread of infection but also has perfect adhesive properties that promote strong pericardial adherences and new vascular anastomosis with adjacent vessels,4 which make future repeat sternotomy a real surgical challenge that no cardiac surgeon would like to face. Right or left thoracotomy may be a good alternative approach for these patients if coronary artery bypass grafting or valve surgery is to be performed, but not for other complex surgical procedures in which median sternotomy is mandatory.

We present a case of a 33-year-old man who developed bacterial mediastinitis and sternal dehiscence after orthotopic HT. He underwent prompt sternal debridement, and a transposition of the greater omentum to the thorax was performed. One month later, he was discharged in satisfactory condition. Nine years after HT, he was readmitted to hospital with congestive heart failure and low cardiac output. Cardiac catheterization revealed a left ventricle ejection fraction of 14% and a normal pulmonary artery pressure. Neither angiography nor intracoronary angiographic findings. J Card Surg 1995; 10:46–51


End-of-Life Literature

To the Editor:

The care that physicians provide to patients with pulmonary disease who are at the close of life has received increasing attention in the past year. Recommendations for professional standards on end-of-life care have been made by specialty boards and organizations, including the American College of Chest Physicians and the American Thoracic Society.1–3 Insufficient training of clinicians in this area is an important cause for deficiencies in care at the end of life.4 The textbooks used to train clinicians are partly to blame. Our research has identified major deficiencies in the end-of-life content of 50 best-selling medical textbooks, including 4 top pulmonary textbooks.5 On average, pulmonary textbooks had helpful information for only about one fifth (21.6%) of the expected end-of-life care content. Of the 12 specialties studied, pulmonary textbooks ranked lowest in average percentage of end-of-life care index citations (0.09%). Furthermore, chapters focused on end-of-life care were completely absent from each of these pulmonary textbooks.

In light of such deficiencies, we have undertaken an effort to encourage publishers, editors, and authors to improve the end-of-life content in their textbooks, including book chapters, cross-referencing, and indexing.6 In follow-up to this effort, we recently surveyed textbook publishers and editors to assess their progress in revising their texts.

Unfortunately, only one editor of the four pulmonary textbooks has responded. Overall, however, 23 editors and 19 publishers of 50 top-selling medical textbooks have responded to our follow-up survey. They report planned or completed expansion of end-of-life content in the next editions of 22 textbooks, including 17 textbooks with new end-of-life care chapters, 17 with revised indexes, and 11 with expanded cross-referencing. Thus, of the 50 textbooks, more than one third are planning to expand or have already expanded end-of-life care content in their next editions. Finally, we have received six personal letters from editors and publishers who have been supportive of this project, including a poignant one from a textbook editor who was himself dying of metastatic melanoma at the time he wrote.

Recently, the Robert Wood Johnson Foundation honored the textbook publishers, editors, and authors who have been working to make these important changes. On February 21, 2001, at an awards ceremony at the Last Acts Project National Meeting, the authors presented awards to one medical textbook publisher (Lippincott Williams and Wilkins) and to the editors of three medical textbooks (Emergency Medicine, 5th ed, Judith Tintinalli editor-in-chief; Nelson Textbook of Pediatrics, 16th ed, Richard Behrman, Robert Kliegman, and Hal Jensen, eds; and Textbook of Primary Care Medicine, 3rd ed, John Noble, senior editor).

Naturally, there is yet more progress to be achieved, especially among pulmonary textbooks. Numerous top-selling textbooks have not yet responded to the suggestions of specialty boards, their readers’ needs, or their patients’ and families’ ultimate

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