Phrenic Nerve Injury

To the Editor:

We read with interest the article by Laub et al 1 on phrenic nerve injury (PNI), which appeared in the August 1991 issue of Chest. We wish to draw the authors' attention to our recent article on the same topic. 2

The advantages of the article by Laub et al are that it is a prospective study (although it is unclear whether the control group is also a prospective one) and it eliminates the variable of multiple surgeons. However, we have several problems with their conclusions.

With regard to the patient descriptions used for comparability of the two groups, we think that Table 1, on prospective variables, does not provide sufficient information. Essential data would have included preoperative clinical characteristics, including cardiac function, diabetes, and smoking history.

With regard to the criteria for diagnosis of PNI, the authors relied primarily on chest radiographic abnormalities, although chest radiographs are neither sensitive nor specific for that. A definitive diagnosis can be made only by measurement of phrenic nerve latency combined with fluoroscopic observation of diaphragmatic movement. Thus, it is difficult to interpret their conclusion about the frequency of PNI.

With regard to outcome, we think that Table 4 is confusing. On the basis of the authors' definition of PNI, it appears that only three patients in the nonintervention group required prolonged airway cannulation. Also, no follow-up information (eg, recovery rate) is provided on any group. The recovery of phrenic nerve function may take months.

In conclusion, PNI following open heart surgery remains a difficult problem. Prospective analysis of all high-risk patients would be ideal.


REFERENCES


To the Editor:

Doctors Abd and Braun bring up some interesting points in their comments about phrenic nerve injury. We wish to emphasize, however, that the primary goal of our study was to prospectively evaluate the effect of one change in surgical technique on an important complication associated with open heart surgery. Because we looked at postoperative alterations in chest radiographic findings and pulmonary function, we felt that these changes were directly attributable to the surgical procedure and were not due to the preexisting medical condition of the patient. In addition, we were not interested in characterizing late postoperative recovery from phrenic nerve injury; but rather the impact of phrenic nerve injury on the early postoperative clinical course. Although a study of the natural history of phrenic nerve injury would make an interesting topic of research, it was not the aim of this study.

Doctors Abd and Braun are concerned that we relied primarily on chest roentgenographic abnormalities for the diagnosis of phrenic nerve injury. The use of chest roentgenographic screening for identifying patients with possible phrenic nerve injury is widely accepted in the literature, as is confirmation of an injury with fluoroscopic observations, which were performed in all patients with clinical evidence of pulmonary dysfunction. As the aim of our article was to determine the impact of a change in technique on the clinical outcome, we believe that this is entirely appropriate. We have no experience with phrenic nerve latency for evaluation of phrenic nerve injury, but we are intrigued that Doctors Abd and Braun recommended it despite their own findings that "normal phrenic latency cannot be equated to normal diaphragmatic motion because latency may be normal in the presence of incomplete phrenic injury."

Doctors Abd and Braun seem to feel that no follow-up information is provided for any group. However, the time on the ventilator, time in the intensive care unit, time to hospital discharge, and mortality rates for all groups are given. Perhaps the authors are missing the point that this is a study of clinical outcomes of patients, not the clinical outcomes of phrenic nerves.

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REFERENCES


Bucillamine-Induced Hypersensitivity Pneumonitis

To the Editor:

Bucillamine is a newly available active drug for rheumatoid arthritis. 3 A 62-year-old woman with rheumatoid arthritis developed dyspnea and interstitial pneumonia after bucillamine therapy for three months. Withdrawal of medication resulted in reversal of the disease process, and an oral challenge with the drug was positive. At bronchoalveolar lavage (BAL) analysis there was alveolar lymphocytosis with a disturbed lymphocyte subset ratio (Table 1). The follow-up BAL analysis showed no change in lymphocyte percentage in two months, but the CD4/CD8 ratio had recovered significantly. The inverted CD4/CD8 ratio was mainly due to an increase in cytotoxic T cells. These findings suggest the usefulness of follow-up