heparin anti-factor Xa levels in the monitoring of unfractionated heparin therapy in the Seventh ACCP Consensus Conference.

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1 Sixth ACCP Consensus Conference on Antithrombotic Therapy. Chest 2001; 119(suppl):1S–370S
3 Fifth ACCP Consensus Conference on Antithrombotic Therapy. Chest 1998; 114(suppl):1S–769S

To the Editor:

The authors of the preceding letter identify another frailty of anticoagulant monitoring. The issue of standardization needs to be addressed. The hope for the future is replacement of therapies that require anticoagulant monitoring with those that do not; low molecular weight heparin therapy offers such an alternative in the majority of patients.

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Outcomes After Bilateral Lung Volume Reduction

To the Editor:

The article by Flaherty and coworkers (May 2001)1 is a much-needed contribution to the slowly accumulating database for lung volume reduction surgery.

The authors have shown that, after lung reduction surgery, patients with localized emphysema do better than those with diffuse disease, although FEV1 values did not correlate well with symptomatology. The findings of Flaherty et al confirm the impression, which many of us have had from our own experiences, that the best results are obtained when severe emphysema is localized, usually in the upper lobe and the apical segment of the lower lobe. Under these circumstances, the remaining portion of the lung is compressed and, if it is relatively healthy, it will function better when allowed to expand.

In general, all of the available published data from surgically treated emphysema patients have the same shortcomings: much time is required to accumulate a satisfactory number of patients for statistical analysis; and, furthermore, there are problems associated with the multiple variables involved, the most obvious being the lack of detailed data on, for instance, pathology, the variable extent of disease, concurrent illnesses, the multiplicity of surgeons, varying criteria, and varying types of perioperative care.

Although the etiology of pulmonary emphysema is incompletely understood, much is already known regarding the pathology. The careful studies of Liebow2 have demonstrated the involvement of both vascular and respiratory elements. Yet, little attention is being focused on the former.

The almost universal involvement of the vasculature would seem to mandate that hemodynamic and angiographic data, in addition to ventilatory and respiratory function data, be accumulated preoperatively and postoperatively. Subjective improvement is notoriously unreliable.

In our experience, ventilatory studies and blood gas analyses have not correlated accurately with pulmonary hemodynamics, which demonstrated a better predictive value for operative mortality.3 Therefore, it would seem expedient to perform concomitant detailed vascular and respiratory assessments on all candidates for surgery.

My experience4 has shown that as much as 70% of a healthy lung can be removed without changing forced expiratory values. Therefore, unless the remaining relatively healthy lung is compressed by the hyperexpanded emphysematous lung, one can expect little or no improvement in this parameter.

Compliance and hemodynamic studies show little change with the amount of reexpansion that one would expect following the excision of localized emphysema.5 I believe that the methodology we used in the latter investigation could be adapted to the study of the effects of surgery on fresh postmortem specimens of emphysematous lungs to provide accurate supplemental informa-
tion to clinical studies. I also believe that inspiratory and expiratory bronchial flow studies could be performed at the same time with proper technical adaptations.

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4 Pecora D. Progressive changes in ventilation following pulmonary resection. Surg Gynecol Obstet 1956; 103:455–458

To the Editor:

We appreciate the opportunity to review the comments provided by Dr. Pecora, which highlight many of the unresolved issues in volume reduction surgery. We agree that the data presented in our recent work have contributed to our understanding of this procedure. We have better identified the short-term and long-term outcomes of volume reduction surgery, but, unfortunately, we also have highlighted the difficulties in adequately predicting long-term outcomes.

One extremely important area that is not addressed in our work revolves around the impact of the pulmonary vasculature. Dr. Pecora is correct in suggesting that the current methodology for evaluating the impact of the pulmonary vasculature has been “notoriously unreliable.” In addition, several reports have suggested the possibility of impairment of pulmonary vasculature function in selected patients after lung volume reduction surgery. This indeed suggests that, as Dr. Pecora points out, it would be important to perform detailed vascular studies in patients being considered for lung volume reduction surgery. The National Emphysema Treatment Trial, a multicenter, prospective, randomized trial, should aid in addressing many of these issues. This important study, which was supported by the National Heart, Lung, and Blood Institute and the Health Care Financing Administration, will prospectively observe >1,000 patients randomized to pulmonary rehabilitation vs the same plus bilateral lung volume reduction surgery. Issues of short-term and long-term outcomes, as well as patient selection criteria, will be more crisply defined with the publication of these data. In addition, several centers have been performing detailed pulmonary vascular studies at baseline and after intervention in both treatment arms. These data should provide better answers to the important questions that are raised by Dr. Pecora in his communication.

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Methimazole-Induced Asthma?

To the Editor:

I read with great interest the case report of Grembiale et al (May 2001).1 I have some questions to the authors about the clinical course of this patient. Although the patient received a diagnosis of bronchial asthma, I haven’t seen any ratio of FEV1 to FVC that is important for obstruction criteria of the airways. Moreover, if a patient has a multinodular goiter, inspiratory flow limitation must also be taken account. I haven’t seen any mention of it in the article. Do the authors suggest that there is no upper-airway obstruction in this patient? The authors also claimed that neither parasites nor other causes of systemic eosinophilic diseases were found. What were they... is, vasculitis or others?

Although the patient has a diagnosis of asthma, I couldn’t understand why treatment with corticosteroids was discontinued instead of increasing the dosage. The benefits of corticosteroid drugs in the treatment of asthma have been documented previously. Knowing that the mortality of asthma is usually due to undertreatment with corticosteroids, osteoporosis or lack of response to treatment should not be a reason for discontinuing treatment with corticosteroids in this patient.

There are >6 weeks from the onset of the symptoms after initial treatment with methimazole. Is it correct to use the phrase, “methimazole-induced asthma”? What were the exclusion criteria of drug-induced eosinophilic lung disease?

Does the increasing eosinophil count in serum and sputum cause asthma, as the authors mentioned? If it is true, why was it resistant to corticosteroid treatment?

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To the Editor:

We are pleased to answer the questions about our report (May 2001)1 asked by Dr. Polatli, who allows us to give additional information about this case of methimazole-induced asthma. The presence of an obstructive ventilatory defect was undoubtable, as shown by the typical aspect of the flow-volume curve, characterized by a progressive reduction of forced expiratory flows: FVC, 56% predicted; FEV1, 48% predicted; FEV1/FVC ratio, 71%; mean expiratory flow (MEF) at 75% of FVC, 42% predicted; MEF at 50% of FVC, 14% predicted; and MEF at 25% of FVC, 10% predicted. The diagnosis of asthma was also confirmed by the prompt reversibility of airway obstruction obtained after the

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