Table 1—Biochemistry of Pleural Effusion in Tuberculous and Congestive Heart Failure Effusions

<table>
<thead>
<tr>
<th></th>
<th>TB Effusion</th>
<th>CHF Effusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>SG</td>
<td>1.4</td>
<td>1.3*</td>
</tr>
<tr>
<td>Protein (g/dl)</td>
<td>4.9 ± 0.7</td>
<td>1.2 ± 0.6*</td>
</tr>
<tr>
<td>Cholesterol (mg/dl)</td>
<td>73.3 ± 19.6</td>
<td>15.0 ± 5.5†</td>
</tr>
<tr>
<td>LDH (IU)</td>
<td>367.7 ± 282.8</td>
<td>53.6 ± 25.4</td>
</tr>
<tr>
<td>Alkaline phosphatase (IU)</td>
<td>118.5 ± 45.7</td>
<td>45.9 ± 33.2*</td>
</tr>
</tbody>
</table>

Values are mean ± standard deviation.
*p<0.001.   †p<0.005 for TB effusion compared with CHF effusion.

from nontuberculous effusions by measuring pleural adenine deaminase (ADA),* lysozyme (LZ),* and interferon-gamma (IFN)*; these biochemical compounds are elevated in tuberculous effusions depending on the size of the effusion, although there is an overlap in the pleural concentrations of ADA and LZ.*

We reasoned that the pleural alkaline phosphatase concentration would be a useful means of differentiating exudative from transudative effusions. We evaluated the specific gravity (SG) and protein, cholesterol, LDH, and alkaline phosphatase concentrations in pleural fluid in 55 patients with histologically proved tuberculous pleurisy, and in ten with effusions from CHF (Table 1).

In addition to having higher pleural protein, cholesterol, and LDH levels, tuberculous exudative effusions also have a high alkaline phosphatase content (approximately >75 IU). Pleural LDH levels in tuberculous effusions do vary a great deal, and estimations of pleural fluid protein, cholesterol, and alkaline phosphatase levels may be the most useful means of differentiating tuberculous effusions from transudative effusions.

There is greater variation in pleural concentrations of protein, cholesterol, LDH, and alkaline phosphatase in CHF; this is due to differences in the degree of decompensation of CHF due to treatment with diuretics.*

The value of estimating the pleural alkaline phosphatase concentration to differentiate tuberculous effusions from nontuberculous exudative effusions needs further investigation.

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4 Ocaña I, Martinez-Vazquez JM, Segura RM, Fernandez de Sevilla T, Capdevila JA. Adenosine deaminase in pleural fluids. Chest 1986; 84:50-54

Nebulized Ipratropium in the Treatment of Acute Asthma

To the Editor:

Summers and Tarala recently reported a study comparing nebulized ipratropium and beta agonist therapy in acute asthma, and concluded that there was no advantage to be gained from combination treatment.1

However, the data show the opposite. This was not apparent in the initial analysis due to type II statistical error, but the results seem to be consistent with those of all other similar trials, as compared by meta-analysis.2

The immediate practical question that should be addressed by the study is: "Should this patient who has just arrived in the emergency room be treated with nebulized beta agonist or with beta agonist and ipratropium bromide together?" Thus, we should compare only groups A and C in the study by Summers and Tarala, lest the data be confounded by treatment crossover. We see that the beta agonist-only group experienced a mean percentage increase in peak flow rate of about 55 percent at 60 min, compared with about 64 percent after combination treatment. The difference was therefore 9 percent, within the 95 percent confidence interval (CI) we calculated from previous trials (mean difference, 12.5 percent; 95 percent CI, +1.2 percent to +23.3 percent). The difference in the study by Summers and Tarala rises to about 14 percent if patients receiving additional intravenous therapy are excluded.

O'Driscoll et al have reported results of a similar trial, differing in that preservative-free ipratropium was used.3 There was a difference between mean percentage change in peak flow of 44 percent (95 percent CI, +8 percent to +84 percent) in favor of combination treatment.

Thus, five published, randomized trials have all produced very similar results indicating that combination treatment is better than beta agonist treatment alone in acute asthma. I am not aware of any unpublished trials with negative results that might affect this conclusion. The degree of homogeneity in the results of these five trials is remarkable, compared to the results of various trials in acute asthma which have addressed other aspects of management.4

The advantage gained from combination treatment does however seem to be modest, and is most apparent in the first hour or two of treatment: whether combination treatment is of benefit after the first four hours, or whether it affects mortality or the duration of hospital stay, has not been determined in these trials.

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1 Summers QA, Tarala RA. Nebulized ipratropium in the treatment of acute asthma. Chest 1990; 97:425-29
2 Higgins RM, Stradling JR, Lane DJ. Should ipratropium bromide be added to beta agonists in treatment of acute severe asthma? Chest 1988; 94:728-22
4 Ward MJ. Clinical trials in acute severe asthma: are type II errors important? Thorax 1986; 41:824-29

To the Editor:

We thank Mr Higgins for his interesting comments. Although we do not dispute the general points raised regarding meta-analysis, we do not agree that a type II error is the only reason for failing to
find a difference between the treatments. Our study had a power of 80 percent to detect approximately a 20 percent difference between the groups, and we feel that any smaller difference may not be clinically important in this group of patients.

As Mr Higgins stated, any advantage from combination treatment is modest. It seems to us that in order to benefit a small number of patients, we would have to treat many unnecessarily. While not wanting to appear to be therapeutic nihilists, we are keen to establish a rational therapeutic plan for acute asthma, and we do not feel that any trial published so far justifies the wholesale treatment of every patient presenting with acute asthma with both drugs in order to provide questionable benefit for a few. Clearly, more work needs to be done, in particular to establish which subgroup of patients are likely to derive benefit from combination treatment of this sort.

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**Metallic Weakness and Breakage of Abrams Pleural Biopsy Needle**

To the Editor:

We read with interest the recent report by Fité et al of one case of pleural effusion in which breakage and detachment in the pleural cavity of the tip of a nearly new Abrams needle occurred during performance of a pleural biopsy. Recently, we cared for a 60-year-old man hospitalized for investigation of left pleural effusion. Chest ultrasound showed loculated pleural effusion and thickening of the parietal pleura. Thoracentesis revealed bloody effusion, samples of which showed predominance of lymphocytes and numerous red blood cells negative for malignancy. Tuberculous bacillus was not found in acid-fast stain. The study of pleural effusion was inconclusive. A pleural biopsy was done by means of an Abrams needle. When the needle was withdrawn, the tip was missing from the midpoint of the window through which we took the biopsy sample (Fig 1). A chest x-ray film showed the tip of the Abrams needle lodged in the left posterior pleural cavity. Computed tomography of the chest showed pleural thickening, pulmonary consolidation, and a metallic tip within the pleural cavity (Fig 2). After four months of observation, the intrapleural foreign body has not caused any complications. A culture of pleural effusion grew *Mycobacterium tuberculosis* organisms six weeks later. Antituberculosis therapy has been started.

We believe that the accident reported above, resulting from detachment of the trocar tip in the pleural cavity, may be attributed to metallic failure at the window through which the biopsy is performed. Another Abrams needle used for approximately the same number of patients (approximately 40) showed angularity and evidence of breakage at the midpoint of the window (Fig 1). This needle is not used for pleural biopsy any more. However, we agree with the opinion of Fité et al that such accidents could be prevented by manufacturing a one-piece trocar and reinforcing at the midpoint of the window. Also, the durability of the Abrams pleural biopsy needle (ie, how many patients or how many years for a new needle could be used) should be determined.

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**Timing of Tracheostomy in Patients with ARDS**

To the Editor:

Deciding to perform a tracheostomy on an intubated patient in the intensive care unit has always been difficult. Heffner and Zamora's article in the February issue (Chest 1990; 97:447-52) suggests that some clinical features of patients after 7 days of ARDS may be helpful in selecting patients for early tracheostomy.

Forty patients were excluded from the study because they died before the 14th day of ARDS. It would appear from the data that 40 of the 64 patients who had ARDS for at least 7 days died within 14 days. The clinical features in this group of patients should be more interesting and important than those of the other two groups since they made up the largest group of patients and were unlikely...