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To the Editor:

We reviewed the letters from Whitlock et al and Dr. Dicicco. Whitlock et al add to our report by stating that, in a symptomatic patient with known HIV infection and sarcoïdosis, bronchoalveolar lavage (BAL) may be helpful in differentiating the nonspecific pneumonitis of HIV infection from active infiltrates of sarcoïdosis. Their report supports this hypothesis in the case of nonspecific pneumonitis.

Dr. Dicicco’s letter lead us to his case report, which confirms our report that sarcoïdosis can develop in the presence of active HIV infection. In their article, Ingram et al postulate that the results of T4/T8 subsets in BAL may differentiate active sarcoïdosis from nonspecific pneumonitis in HIV-infected patients. They did not report BAL findings in their case. The patient in our case study would not allow us to perform BAL, and as yet there have been no reports of BAL from a patient with HIV infection and active sarcoïdosis.

In conclusion, we agree that the T4/T8 ratio in BAL might help to differentiate active sarcoïdosis infiltrates from non-specific pneumonitis in patients with both sarcoïdosis and HIV infection. We wait for the report of BAL results in such a patient. If the T4/T8 ratio is high, then a method of differentiation may have been found. This information will lead to the proper therapy: steroids in conventional doses, or combined with thymostimulin,1 for the patient with active sarcoïdosis; and observation for the patient with nonspecific pneumonitis.

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REFERENCE


Mesothelial Cells and Tuberculous Pleuritis

To the Editor:

We read with great interest the report by Kam-Yung Lau (Chest 1989; 96:2:438-39) of two cases of pleural tuberculosis with significant numbers of mesothelial cells. We wish to make several comments.

As the author points out, at present it is believed that this finding almost excludes a diagnosis of tuberculous pleural effusion. However, various data in the literature and our own experience do not support this concept. Actually, we have recently found numerous mesothelial cells in a patient with tuberculous pleural effusion. Moreover, there are several series—some extensive—in which the presence of significant numbers of mesothelial cells was not a rarity. For instance, in a retrospective study of 88 tuberculous pleural effusions, the authors found more than 5 percent mesothelial cells in 13 patients (15 percent).1 Other studies have shown similar results.2,3

In pleural tuberculosis (as in other chronic pleural effusions), the extensive inflammation of both pleural layers prevents the exfoliation of mesothelial cells into the pleural space. However, in an early phase when the inflammatory process is acute and not extensive, it is possible to find numerous mesothelial cells. In fact, in patients with tuberculous pleuritis the disappearance of mesothelial cells on serial thoracocentesis has been observed as the white cell count changes from neutrophil to lymphocyte predominance.1

Therefore we think, in accordance with Kam-Yung Lau, that pleural tuberculosis remains a diagnostic possibility in the presence of more than 5 percent mesothelial cells. If the thoracocentesis is performed in an early phase of the disease, this finding is not exceptional.

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REFERENCES


To the Editor:

I am grateful to Santos-Sastre et al for their interest in my article. Their experience supports the notion that mesothelial cells in tuberculous pleural effusion may not be as rare as previous studies have suggested.

Dr. Santos-Sastre proposed that, in the early phase of pleural tuberculosis, the inflammatory process is not extensive and exfoliation of mesothelial cells into the pleural fluid is still possible. However, this does not seem to be applicable to the cases that I described. In my article, numerous mesothelial cells were found in both the early and late stages of pleural tuberculosis. In fact, in Case 1 of my report, serial thoracocentesis revealed that mesothelial cells had increased from 3 to 6 percent. The cause for this unusual finding of numerous mesothelial cells in not clear. Further studies would be warranted to clarify the issue of mesothelial cells in tuberculous pleural effusions.

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Variables in Histamine Inhalation Tests

To the Editor:

Cockcroft et al (Chest 1989; 96:505-08) have elegantly and precisely documented and quantified evaporative water loss during standard histamine inhalation tests performed with Wright nebulizers.

The evaporative water loss was associated with a 11°C drop in measured temperature over ten minutes of nebulizer operation. After 2 min, the drop was 7°C. The authors stated they have no knowledge of such a measurement prior to their work.
However, several years ago, Haifer and Plit measured the temperature changes during two minutes of nebulization of histamine from a Wright nebulizer and found it to be exactly the same as in the present report.

They went on to investigate whether this temperature drop may have any clinical effect and compared the response to inhaled histamine (PC_{20}) between nebulizers operating at different but constant temperatures. The nebulizers were designed to prevent any temperature drop during nebulization. Two groups of asthmatic patients were studied: one group inhaled the aerosol at constant 30°C, whereas the other inhaled the aerosol at constant 23°C. In both groups the results were compared to those obtained during the usual test performed at room temperature (23°C) without keeping the temperature constant. The results (PC_{20} in mg/ml) are presented in the table.

Thus, keeping the temperature constant increased PC_{20} (reduced airway reactivity) compared with the standard method. The higher the temperature was kept, the greater PC_{20} (reactivity was further reduced).

Deducing from Cockcroft’s study (Figure 2 and Discussion), one would assume that, compared to the usual method, keeping the temperature constant would prevent the observed reduction in nebulizer output and hence PC_{20} should be smaller. The results of Haifer and Plit’s study (which showed an opposite effect) contradicted this assumption. Moreover, the fact that the effect was enhanced at higher (constant) temperatures indicate a significant role for temperature in the results. Indeed, we have recently demonstrated the importance of temperature in modifying airway response even in normal subjects.

In view of Haifer and Plit’s results, the report of Cockcroft et al is important not only in terms of standardization and methodology but also in clinical terms.

\[ \text{Table} \]

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<th>Usual, Room Temp</th>
<th>Constant 23°C</th>
<th>Constant 30°C</th>
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<tbody>
<tr>
<td>Group 1</td>
<td>0.39</td>
<td>—</td>
<td>3.29</td>
</tr>
<tr>
<td>Group 2</td>
<td>1.75 (2.35)</td>
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To the Editor: We would like to thank Dr. Amirav for bringing the data of Haifer and Plit to our attention. We apologize for having missed their abstract.

The results of constant temperature PC_{20} are interesting and have important clinical relevance. We carefully avoided any clinical predictions based on the results in our paper. Since the reduced output at cooler temperatures represents chiefly a reduction in evaporation, we would have expected warm vs cool nebulization to have produced little difference. Since there may be a tendency for reduced solute output (not certain from our calculations), we might have expected a small change in the opposite direction to that described in the abstract by Haifer and Plit.

The surprise in the data of Haifer and Plit is not the direction but the magnitude of the change; warm nebulization (30°C) produced almost a ten-fold reduction in response to histamine compared to room temperature (23° to 16°C). The mechanism of this difference remains unclear to us. The authors’ speculation that two minutes of tidal breathing of an aerosol between 23° and 16°C might enhance airway responsiveness to histamine based on additive or synergistic effect of cold air seems unlikely, since this represents a very small thermal burden on the airways. It is more likely that the reduced responsiveness with the warm nebulization represents an alteration in the physical characteristics of the aerosol. Alterations in solute output or particle size could possibly explain these changes. The Wright nebulizer generally produces a small particle size (1.0 to 1.5 microns mass median diameter). Previous reports of Wright nebulizers produced from a new manufacturer showed that with similar mass output run at room air temperature, a four-fold reduced response was observed. Further investigations (Hargrave, personal communication) suggested the most likely explanation for this reduced output was an unacceptably low mass median diameter particle size of the order of <0.8 microns. This represents a rather small deviation from the usual Wright nebulizer particle size, and although we are not certain what happens to particle size with temperature, a small reduction in mass median diameter induced by the warmer solution could explain the results documented in the abstract.

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Methemoglobinemia After Lidocaine Administration

To the Editor: In an otherwise complete summary of adverse affects associated with the use of lidocaine topical anesthesia for flexible bronchoscopy, Dr. Kirkpatrick failed to mention an unusual but potentially dangerous complication that can be most confusing if unrecognized. After topical administration to the nasal mucosa, lidocaine occasionally causes severe methemoglobinemia in patients who have the heterozygous form of NADH-Methaemoglobin reductase deficiency. Benzocaine (an ingredient in cetacaine topical anesthetic) can rarely cause methemoglobinemia when applied to the mucous membranes of normal individuals.

The sudden and otherwise unexplained development of severe respiratory distress and cyanosis during or after bronchoscopy should raise the possibility of methemoglobinemia. The diagnosis is confirmed by the chocolate brown color of the blood and by two laboratory findings: 1) arterial oxygen tension may be normal, but oximetry shows a low arterial oxygen saturation, and 2) the quantity of methemoglobin in the blood (measured directly by spectropho-