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REFERENCES
3 Van Keimpema AR, Slats EH, Wagenaar JP. Adenosine deaminase activity, not diagnostic for tuberculous pleurisy. Eur J Respir Dis 1987; 71:15-18

To the Editor:

Dr. Sahoo's letter raises two interesting objections to the determination of pleural adenosine deaminase (PADA) for diagnosis of tuberculous pleurisy. First, he believes that the significance of this parameter is questionable in view of the wide range of diagnostic cutoff points used in the various studies that have supported the use of PADA (25 to 50 U/L). Second, he points out that there have been reports of high PADA levels being an insufficiently specific criterion for diagnosis of tuberculous pleurisy.

Evaluation of the first criticism requires consideration of the possible sources of the variation in cutoff points. It seems improbable that the source suggested by Dr. Sahoo, differences in instrumental method (colorimetry vs spectrophotometry), can contribute greatly to the observed variation; colorimetry and spectrophotometry are both well-standardized procedures based on the Beer-Lambert law, the only essential difference between them being the monochromator used to provide radiation of the working wavelength. A much more likely technical source of discrepancy would seem to be differences in regard to which of the chemical species involved in the adenosine deaminase reaction is measured for the purposes of ADA determination (adenosine, inosine, or—in our laboratory—ammonium ions); such differences might well affect within-run and between-run precision and accuracy in each laboratory, and hence interlaboratory homogeneity. More importantly, however, there are two quite obvious sources of discrepancy that have nothing to do with analytical technique: the characteristics of the populations studied in each case and the criterion used to establish the cutoff point.

Of the four cutoffs mentioned by Dr. Sahoo in addition to our own (47 U/L) (Chest 1993; 103:458-65), two are, in fact, quite close to ours (45 U/L and 50 U/L), while the other two are much further removed (25 U/L and 30 U/L). Two comments may be made concerning the value of 25 U/L attributed by Dr. Sahoo to Piras et al. The first is simply that this value seems to be a mis-

understanding on Dr. Sahoo's part, since the relevant figure in the article by Piras et al. appears to show a higher cutoff. The second shows the point made at the end of the previous paragraph: Piras et al. appear to have taken as their cutoff the highest PADA value among the nontuberculous pleurisy cases they considered—a criterion differing from ours. We cannot offer an opinion on the cutoff of 30 U/L by Prasad et al. because we have, unfortunately, been unable to obtain a copy of their paper before writing this reply.

With regard to Dr. Sahoo's second criticism, concerning the performance of PADA as a diagnostic parameter, we refer the reader to the meta-analytic study of the value of PADA for diagnosis of tuberculous pleurisy by Ena et al. Ena et al. reviewed all the relevant publications included in the Index Medicus since 1980 which fulfilled the following conditions: the patient series studied were to consist of more than one case, were to be composed exclusively of pleurisy cases with no restrictions on etiology, and were not to include series published elsewhere. These conditions were fulfilled by seven studies, including the Van Keimpema et al. study. The Ena et al. conclusion was that, when used appropriately, the sensitivity of PADA for tuberculous pleurisy is 99 percent and its specificity 93 percent, values that are very similar to those found in our own work (Chest 1993; 103:658-65).

We remain convinced that PADA is a useful parameter for diagnosis of tuberculous pleurisy when properly used. For the reasons discussed above, proper use includes the establishment by each center of its own cutoff value after accepted criteria.

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1 Van Keimpema AR, Slats EH, Wagenaar JP. Adenosine deaminase activity not diagnostic for tuberculous pleurisy. Eur J Respir Dis 1987; 71:15-18

Alkaline Phosphatase
Distinguishing Between Pleural Exudates and Transudates

1912 Communications to the Editor

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Table 1  Biochemistry of Pleural Effusion in 
Transudates and Exudates

<table>
<thead>
<tr>
<th>Transudates</th>
<th>CHF Effusion</th>
<th>TB Effusion</th>
<th>Neoplastic Effusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>P/SPROT, gr/d</td>
<td>1.82 ± 0.72</td>
<td>5.05 ± 0.77</td>
<td>4.6 ± 0.78</td>
</tr>
<tr>
<td>P/SAP</td>
<td>0.26 ± 0.09</td>
<td>0.69 ± 0.10</td>
<td>0.64 ± 0.09</td>
</tr>
<tr>
<td>PLDH, IU</td>
<td>143 ± 84.6</td>
<td>780 ± 390</td>
<td>881 ± 917</td>
</tr>
<tr>
<td>P/SLDH</td>
<td>0.32 ± 0.22</td>
<td>1.63 ± 1.03</td>
<td>1.29 ± 0.63</td>
</tr>
<tr>
<td>PAP, IU</td>
<td>16.2 ± 7.06</td>
<td>44.1 ± 18.9</td>
<td>53.1 ± 25.4</td>
</tr>
<tr>
<td>P/SAP</td>
<td>0.15 ± 0.06</td>
<td>0.43 ± 0.13</td>
<td>0.52 ± 0.31</td>
</tr>
</tbody>
</table>

To the Editor:

We read with interest the letter by Dr. Syabbalo, which appeared in the February, 1991, issue of Chest, on the use of the pleural alkaline phosphatase content to diagnose tuberculous effusions and regarding the value of the pleural alkaline phosphatase concentration to differentiate tuberculous effusions from effusions due to congestive heart failure (CHF).1

We report the use of pleural alkaline phosphatase (PAP) and the ratio of pleural fluid to serum alkaline phosphatase (P/SAP) to differentiate transudates from exudates and also to differentiate tuberculous (TB) effusions from neoplastic exudates.

We evaluated the pleural protein (P/SPROT), pleural LDH (PLDH), pleural alkaline phosphatase (PAP), pleural protein/serum protein (P/SPROT), pleural LDH/serum LDH (P/SLDH), and pleural alkaline phosphatase/serum alkaline phosphatase (P/SAP) in 56 patients with exudative pleurisy (37 of them histologically proved tuberculous pleurisy, 19 of them with neoplastic exudate), and 21 patients with transudative pleurisy due to CHF. The results are shown in Table 1.

For PAP and P/SAP parameters, the differences between the mean value of the transudate group and those of the two exudate groups were statistically significant (p<0.001). However, the difference between the mean value of the tuberculous pleurisy group and that of the neoplastic exudates was not statistically significant (p>0.05).

We conclude that PAP and P/SAP are both highly effective in distinguishing between pleural exudates and transudates.

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REFERENCE

1 Syabbalo NC. Use of pleural alkaline phosphatase content to diagnose tuberculous effusions [letter]. Chest 1991; 99:522-23

Effectiveness of Inhaled High-dose Corticosteroid Therapy in Chronic Eosinophilic Pneumonia

To the Editor:

We read with great interest the article by Naughton et al,1 which appeared in the January 1993 issue of Chest.1 This report concerned 12 cases of chronic eosinophilic pneumonia (CEP). The authors emphasized the fact that most of the patients will require steroid treatment for many years and some of them for life. They assumed that in some cases, high-dose inhaled corticosteroid therapy could permit a reduction in the maintenance dose of oral steroid therapy, but "it was too early to judge the efficacy of this strategy."

We have successfully experienced such a treatment with a patient with CEP for over 5 years. This patient, a 52-year-old white woman was first seen in May 1983 for dyspnea, fever, and weight loss. She had all the characteristics of CEP described by Carrington et al3 including histologic findings. The transbronchial lung biopsy specimen revealed infiltrations of eosinophils and histiocytes in the alveolar and interstitial compartments. Prednisolone was started at a dose of 50 mg/d and was decreased by 5 mg daily every week. All attempts to decrease treatment to doses of less than 10 mg/d resulted in reappearance of dyspnea, lung opacities, and blood eosinophils. These symptoms and infiltrates responded to increases in her oral steroid medication. Between December 1983 and December 1987, five attempts to reduce steroid therapy produced relapses. In February 1988, while receiving daily treatment with 10 mg of prednisolone, a chest roentgenogram showed reappearance of new opacities. Orals steroids were maintained at the same dosage and high-dose inhaled therapy (beclomethasone dipropionate 1.500 μg/24h) was added. The pulmonary opacities decreased dramatically within a week and the patient was successfully weaned from the oral steroid over the subsequent month, with beclomethasone dipropionate treatment being maintained at the same level. Furthermore, beclomethasone dipropionate has been progressively reduced to 1,000 μg/24 h over the subsequent year without relapse. Later, the patient developed three acute exacerbations of her CEP. These manifestations responded well, once by increase in high-dose inhaled steroid therapy and twice by addition of a 5-day course of oral prednisolone (40 mg/d). The patient is currently (August 1993) free of symptoms and in radiologic remission while receiving inhaled beclomethasone dipropionate, 1,000 μg/24 h.

We believe that this case confirms the efficacy of such a therapeutic strategy. Inhaled high-dose corticosteroids have proved to be efficient for the treatment of asthma and other various diseases and can substitute for oral steroid therapy. This form of treatment is not always sufficient to control CEP activity and sometimes must be completed by addition of oral steroids. In some cases, it can permit a reduction in the dose of oral therapy. Anyway, the amount of either inhaled or oral medication has to be adjusted in the light of results and may fluctuate over a period.

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