when 1 mL of 1% methylene blue was added to 500 mL of Jevy
ity enteral formula (Ross Laboratories, Columbus, Ohio). They
concluded that some positive readings observed in our study subjects
may have been due to the presence of blue dye rather than glucose,
even without evidence of blue dye on visual inspection.

To confirm the more limited data we collected in our previous
study (Chest 1993; 103:117-21) suggesting that blue food coloring
added to enteral formulas did not affect their apparent glu-
cose concentrations measured at various dilutions using the
AccuCheck IIM meter, we determined the glucose concentration of
Jevity serially diluted in water, with and without the presence of
methylene blue (1,000 μL of a 1% solution per 500 mL formula)
and blue food coloring at the same and a higher concentration
than used in our earlier study (McCormick & Co, Hunt Valley,
Pa; 300 μL and 1,000 μL per 500 mL of feeding, respectively).
To determine whether methylene blue could influence the
apparent glucose measurement of a glucose-free solution, meth-
ylene blue was also added to plain water to obtain a blue color
similar to the feedings, serially diluted, and apparent glucose concentrations were determined. We measured glucose concentrations in all solutions with an AccuCheck IIM meter (Boehringer Mannheim, Indianapolis). The results are presented in Table 1.

We observed no significant effect from the presence of blue dye or food coloring on the apparent glucose concentrations in any of the dilutions of enteral formula tested (Wilcoxon signed ranks; p value for significance <0.05). Neither did we find any specimen of methylene blue in water to yield a positive AccuCheck reading. Thus, we must disagree with the claim of Dr. Montejo-Gonzalez et al, and instead conclude that the positive glucose readings obtained in our previous study were due to the aspira-

Table 1.—Mean Glucose Concentrations and Visible
Blue Using Methylene Blue and Blue Food Coloring

<table>
<thead>
<tr>
<th>Test</th>
<th>No Dye</th>
<th>MB (200 μL)</th>
<th>AC</th>
<th></th>
<th>AC</th>
</tr>
</thead>
<tbody>
<tr>
<td>FS9</td>
<td>106 2 0</td>
<td>+</td>
<td>106.7 ± 1.8</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>1:2</td>
<td>60.3 ± 3.4</td>
<td>+</td>
<td>56.7 ± 0.7</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>1:4</td>
<td>52.0 ± 2.1</td>
<td>+</td>
<td>56.3 ± 1.9</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>1:8</td>
<td>30.3 ± 1.3</td>
<td>+</td>
<td>26.3 ± 0.3</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>1:16</td>
<td>LLL</td>
<td>+</td>
<td>LLL</td>
<td>LLL</td>
<td></td>
</tr>
<tr>
<td>1:32</td>
<td>LLL</td>
<td>−</td>
<td>LLL</td>
<td>LLL</td>
<td></td>
</tr>
</tbody>
</table>

Jevity, per 100 mL feeding.

<table>
<thead>
<tr>
<th>Test</th>
<th>BD (60 μL)</th>
<th>BD (200 μL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>FS#</td>
<td>106.7 ± 2.2</td>
<td>+</td>
</tr>
<tr>
<td>1:2</td>
<td>55.3 ± 0.9</td>
<td>+</td>
</tr>
<tr>
<td>1:4</td>
<td>52.0 ± 0.2</td>
<td>+</td>
</tr>
<tr>
<td>1:8</td>
<td>31.0 ± 1.2</td>
<td>+</td>
</tr>
<tr>
<td>1:16</td>
<td>LLL</td>
<td>+</td>
</tr>
<tr>
<td>1:32</td>
<td>LLL</td>
<td>−</td>
</tr>
</tbody>
</table>

*MB=methylene blue (200 μL/100 mL feeding).
†BD=blue food coloring (60 μL/100 mL feeding).
‡AC=glucose meter (AccuCheck) measured glucose reading (mg/ dL); represent mean ± SEM of 3 determinations.
<table>
<thead>
<tr>
<th>Color</th>
<th>AC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visible blue; + = present; − = absent.</td>
<td></td>
</tr>
</tbody>
</table>

Adenosine Deaminase in Tuberculous Pleural Effusion

To the Editor:

We have read with interest the article by Valdè et al1 on the utility of biologic parameters in the diagnosis of tuberculous pleural effusion (TPE). Of 405 pleural effusions, all the 91 TPE confirmed by bacteriologic or histologic findings had pleural adenosine deaminase (PADA) levels above 47 IU/L. Although initial studies2 also showed a sensitivity of 1 for values above 43 IU/L, further reports3 described isolated cases of TPE with low PADA. Niwa et al4 reported six cases with low PADA value in a total of 28 TPE, although they used a different technique and cut-off value. In the first large series of 114 patients, Querol et al5 described nine cases of TPE with PADA below 45 IU/L (8%); nevertheless, a second PADA determination performed in seven of these nine cases showed values higher than 43 IU/L in five; the authors thus recommended that the measurement be repeated in cases of high clinical suspicion.

Our experience over the last 10 years was reviewed. Of 87 cases of TPE confirmed by bacteriologic or histologic findings, PADA was lower than 43 IU/L in as many as 19 patients (6 women, 13 men; mean age, 38 years; range, 15 to 76 years; no previous or current antituberculous therapy). A second PADA determination was performed in 14 of these 19 cases, between 5 and 15 days (mean 8 days) after the first; PADA values remained below 43 IU/L in 10 of the 14, ie, values were above 43 IU/L in only 4.
Therefore, despite the utility of PADA determination in the diagnosis of TPE, our data confirm that a value lower than 43 IU/L does not permit this diagnosis to be ruled out. On the other hand, PADA values failed to rise on a second determination in most of our cases.

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1. Valdés L. Diagnosis of tuberculous pleurisy using the biologic parameters adenosine deaminase, lysozyme, and interferon gamma. Chest 1993; 103:458-65

To the Editor:

Orriols et al express doubts as to the sensitivity of adenosine deaminase concentration in pleural fluid (PADA) as a diagnostic marker of tuberculous pleurisy. They point out that since the initial work of Piras et al and Ocaña et al (Chest 1983; 84:51-9), there have been occasional reports of tuberculous pleurisy patients with a low PADA (Eur J Respir Dis 1987; 71:15-8; Chest 1985; 87:351-55, and Eur Respir J 1990; 3:586-87) and add that 19 of their own tuberculous pleurisy patients (87 in the last 10 years) also had PADA levels below 43 IU/L. They conclude that subcutoff PADA levels do not absolutely rule out a diagnosis of tuberculous pleurisy.

In the absence of any information on the methods by Orriols et al, we can best refer the reader, as we did in reply to Dr. Sahoo's recent comments on the specificity of PADA in the metaanalytic study by Ena et al, which we regard as a more reliable evaluation of the value of PADA than any single study. 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 causes, and were not to include series published elsewhere. These conditions were fulfilled by seven studies, including the study by Van Keimpema et al (Eur J Respir Dis 1987; 71:15-8) mentioned by Orriols and colleagues. In these seven studies, the PADA cutoff for diagnosis of tuberculous pleurisy ranged from 33 to 79 IU/L. Of the total of 760 pleural fluids analyzed, 185 were from tuberculous pleurisy patients, 184 of which had PADA above cutoff; there were 40 false-positives. The overall sensitivity of PADA for tuberculous pleurisy in these seven studies was therefore 99% and its specificity 94%, values which are very similar to those found in our own study (sensitivity 100%, specificity 95%) and others (sensitivity 100%, specificity 80.5 to 95%).

We certainly do not wish to claim that subcutoff PADA levels absolutely rule out a diagnosis of tuberculous pleurisy, but we are surprised at the very low sensitivity reported by Orriols et al, 78%, more especially because a study of 1,494 serous exudates carried out in the hospital of Orriols et al using the same cutoff, and presumably in the same laboratory and patients from the same population, found the sensitivity and specificity of PADA for tuberculous pleurisy to be 100% and 92% respectively.

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Biochemical Discrimination of Transudates and Exudates

To the Editor:

We believe we must comment on the recent report by Romero et al in the August 1993 issue of Chest on their interesting evaluation of various criteria for distinguishing between pleural transudates and exudates. Since 1972, the criteria commonly used for making this distinction have been those then established by Light et al, a pleural effusion is diagnosed as an exudate if the pleural fluid to serum protein ratio is >0.5, or the pleural lactate dehydrogenase (LDH) is >200 IU, or the pleura/serum LDH ratio is >0.6; and if diagnosed as a transudate, if none of these conditions are fulfilled. In series by Light et al, the appropriate criteria were met by all but one exudate and by all but one tran-