Evaluation of different criteria for the separation of pleural transudates from exudates. Chest 1989; 105:399-404
3 Peterman TA, Speicher CE. Evaluating pleural effusions: a two-stage laboratory approach. JAMA 1984; 252:1051-53
5 Roth BJ, O’Meara TF, Cragun WH. The serum-effusion albumin gradient in the evaluation of pleural effusions. Chest 1990; 98:546-49

To the Editor:

We appreciate the interesting letter in which Valdés et al analyze some aspects of our article in Chest (1993; 104:399-404) with conclusions rather similar to ours. These are (1) the prevalence in the sample influences the overall correct classification rate when a diagnostic criterion has different sensitivity for transudates than for exudates; and (2) it is important to choose and optimize diagnostic criteria in the light of the characteristics of the local population and the results of each particular laboratory. From these conclusions, it is advisable to obtain a criterion with high and similar sensitivity for transudates and exudates, as we did in our study using Light’s criteria optimized for our own environment.

Nevertheless, we believe that comments by Valdés et al over some “striking” aspects of our study that may bias the results deserve being discussed.

We did not discourage any patient for the convenience of practicing thoracentesis, we just informed them. Of course, we do not share the faith of Dr. Valdés et al in performing thoracentesis on patients with obvious transudates. We do know that transudates may be the first cause of pleural effusion, as some necropsy series show.1 But we strongly believe that any diagnostic exploration, more if invasive, is only indicated when doubt exists regarding the cause of a pleural effusion, as a recent position paper states.2 Because patients with congestive heart failure (CHF) and pleural effusion have a variety of easily discernible features that permit a fairly precise diagnosis without resorting to thoracentesis for confirmation, it must be presumed that in most of them, this diagnostic procedure would not be indicated in routine clinical practice. In fact, some large series as that of Leuallen and Carr3 include only 10% of patients with transudates due to CHF.

Valdés et al suggest that the inclusion of 36 patients (27%) without pleural cytologic or histologic evidence of malignancy in the group of neoplastic effusions may have a distorting effect on our results. As it can be seen in Table 1, the exclusion of these 36 patients does not change significantly the yield of the different diagnostic criteria used. Incidentally, Valdés et al misquote the reference number 10 of his letter, where no mention is found about the percentage of benign pleural effusion in patients with malignancy.3 In a previous paper by the same authors4 a figure of 10% of benign pleural effusions in patients with malignancy is given instead of the 17% stated by Valdés et al. In our study, we excluded 5 of 41 patients (12%) with malignancy without cytologic or histologic evidence of pleural involvement because of the existence of other potential benign cause of pleural effusion, a percentage very close to that found by Rodríguez Panadero et al4 in their study.

Table 1—Number of Correctly Classified Neoplastic Effusions for Every Criteria Studied

<table>
<thead>
<tr>
<th>Criteria*</th>
<th>Total Evidence of Neoplastic Effusions, n=132 (%)</th>
<th>Pleural Involvement, n=96 (%)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PF/S proteins &gt;0.5</td>
<td>115/132 (87)</td>
<td>83/96 (86)</td>
<td>NS</td>
</tr>
<tr>
<td>PF LDH &gt;507 IU/L.</td>
<td>84/132 (64)</td>
<td>64/96 (67)</td>
<td>NS</td>
</tr>
<tr>
<td>PF/S LDH &gt;0.6</td>
<td>121/130 (93)</td>
<td>90/95 (95)</td>
<td>NS</td>
</tr>
<tr>
<td>Criteria of Light et al5</td>
<td>128/130 (99)</td>
<td>95/95 (100)</td>
<td>NS</td>
</tr>
<tr>
<td>PF Chol &gt;60 mg/dL</td>
<td>107/132 (81)</td>
<td>79/96 (82)</td>
<td>NS</td>
</tr>
</tbody>
</table>

*PF=pleural fluid; S=serum; LDH=lactate dehydrogenase; Chol=cholesterol; NS=not significant.

REFERENCES
1 Bede DF, Lovibond JL. Hydrothorax in heart failure. Br Heart J 1941; 3:93-111

Does Vitamin E Precipitate Angina?

To the Editor:

Enormous amounts of vitamin E are being consumed for its alleged value in the prevention or treatment of coronary heart disease. I have challenged such use, as well as the legitimacy of megadosage “antioxidant” therapy in the cardiologist’s armamentarium—namely, 150 IU or more daily.1,3 Others4,5 have reported that hypervitaminosis E may actually