To the Editor:

Based on Dr. Syabbalo’s study describing the use of pleural alkaline phosphatase (PAP) content in the differentiation of tuberculous effusions from those resulting from congestive heart failure, we demonstrated further evidence suggesting the role of PAP and the role of the pleural fluid to serum alkaline phosphatase ratio (P/SAP) in the differentiation of all exudates (tuberculous and neoplastic effusions) from transudates (p<0.001). The object of that study was to prevent any misunderstanding that increased levels of PAP were not confined to tuberculosis alone; we also showed that measuring PAP and P/SAP was not sufficient to determine tuberculous and neoplastic effusions (p<0.05).

A recent study by Gazquez et al (see previous letter) compared PAP and P/SAP with the criteria of Light et al and concluded, according to their results, that PAP and P/SAP offered no advantages. This was not what we implied in our previous study, nor did we intend to throw suspicion on the reliability of the criteria of Light et al. We support Gazquez et al except for one respect: our statistical data did not yield such poor results as those of Gazquez et al (Table 1). We feel that further studies with larger series are required to test the value of measuring PAP and P/SAP in the differentiation of exudates from transudates.

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Table 1—Comparison of Data From Studies by Tahaoglu et al2 and Gazquez et al

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Accuracy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tahaoglu et al (exudates=56; transudates=21)</td>
<td>89</td>
<td>90</td>
<td>90</td>
</tr>
<tr>
<td>PAP&gt;25 UI/L</td>
<td>92</td>
<td>95</td>
<td>93</td>
</tr>
<tr>
<td>P/SAP&gt;0.24</td>
<td>100</td>
<td>90</td>
<td>90</td>
</tr>
<tr>
<td>When Light’s criteria is used</td>
<td>86</td>
<td>90</td>
<td>90</td>
</tr>
<tr>
<td>Gazquez et al (exudates=76; transudates=21)</td>
<td>89</td>
<td>97</td>
<td>93</td>
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<tr>
<td>PAP&gt;45 UI/L</td>
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<td>79</td>
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<tr>
<td>P/SAP&gt;0.24</td>
<td>97</td>
<td>75</td>
<td>79</td>
</tr>
</tbody>
</table>

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References

More on Myocardial Contusion—With Additional Insight on Myocardial Concusson

To the Editor:

The communication to the editor by Dr. Krista Kaups (October 1996) merits additional clinical commentary. Her letter implies expertise in the knowledge and care of cardiac injury trauma patients as she attempts to apply the coup de grace to the excellent review by Drs. Feghali and Prisant (December 1995). This can be realized from her closing statement “that the authors have a limited familiarity with the current standard of care for cardiac injury in trauma patients and a limited ongoing participation in trauma care.”

In the opening statement of her letter, she portrays correction and clarification “of the information that is both incomplete and incorrect and serves to confuse rather than clarify the management of patients with this injury.” In my opinion, any knowledgeable clinician experienced in cardiology, upon whom the care of the cardiac injury patient generally falls, would find very little confusion in the review by Feghali and Prisant.

Dr. Kaups states correctly that there is a “spectrum of injury” and she therefore prefers the term “blunt cardiac injury.” She notes, “The phrase ‘blunt myocardial injury,’ [as used by Feghali and Prisant] itself is misleading as are similar phrases such as ‘cardiac contusion’ and ‘cardiac concussion’.” This illustrates the continued semantic differences in clinical definition by many individuals and groups worldwide on this subject of increasing incidence and importance. It is my impression, however, that she failed to realize that the review by Feghali and Prisant was a highly condensed discussion, meant to focus predominantly on the entity commonly (and correctly) called (if properly diagnosed) myocardial contusion.

The term “blunt cardiac injury” is actually an all-encompassing topic that includes myocardial contusion, pericardial injury, vascular injury, valvular and associated structural injury, and myocardial rupture.

The term “myocardial contusion” is a correct clinical and pathologic definition. It implies a bruise to a segment of myocardium that reveals subepicardial and intramyocardial hemorrhage, disruption of myocardial fibers, cellular infiltration, and interstitial edema on histologic examination. Myocardial contusion can be, and generally is, a separate and singular entity, but it obviously can occur in the presence of other elements associated with blunt cardiac injury—the term Dr. Kaups prefers—but this use is not exactly correct either. To be more exact, by proper definition, myocardial contusion would be divided into the following three subgroups: subepicardial, transmural, and subendocardial, the latter probably occurring as a result of severe crush injury to the abdomen and legs.

Dr. Kaups’ apparent aversion to the term “myocardial concussion,” although more understandable, also requires concurrent explanation. According to our present state of knowledge, myocardial concussion, sometimes referred to as commotio cordis, is