Bronchoalveolar Lavage
Let’s Focus on Clinical Utility

Bronchoalveolar lavage (BAL) has been used for over a decade to sample lung cells and proteins. Because this technique can recover material inaccessible by other methods, it has achieved an important place in research into the pathogenesis of lung disease. Several recent articles suggest that BAL analysis in clinical situations has significant utility in patient care. Information about infectious and noninfectious inflammatory diseases can be obtained with sensitivity greater than that possible with noninvasive methods. When BAL data are provided to clinicians, they frequently result in important changes in patient management. Finally, the technique, although invasive, has such low morbidity that it can be safely applied to even the most ill patients.

Once BAL specimens have been obtained, many assays (virtually any serum or cytology assay) can be ordered. The greatest clinical utility thus far has come from assessment of infectious organisms by cytology and culture and cytologic analysis of the recovered cells and cellular inclusions. Staining of the recovered cells for hemosiderin is one type of cytologic assessment. This test has been applied for the last 15 years to cells recovered by lavage, and most authors have found it to be a useful marker of blood in the alveolar space (BAS).

In this issue of Chest (see page 1794), Grebski and colleagues report that measurement of hemosiderin-containing macrophages adds little to the value of BAL analysis in clinical assessment. The presence of hemosiderin in macrophages is related to prior macrophage ingestion of alveolar erythrocytes. Although this finding of an increase in hemosiderin-laden macrophages is probably specific for BAS, the causes of BAS are quite diverse. Processes including aspiration of blood from the upper or large airways, congestive cardiac failure, and diseases associated with severe alveolar space inflammation can cause BAS. Once this occurs, macrophages will degrade the erythrocytes to yield hemosiderin, and this by-product will have a relatively long period of intracellular residence. Thus, although one can assume that the presence of an increased number of hemosiderin-laden macrophages is strongly associated with the prior presence of BAS, the physician must interpret this information in the context of the complex mechanisms available for presentation of erythrocytes to alveolar macrophages.

Therefore, the findings of Grebski et al are not surprising. An increase in hemosiderin-laden macrophages is not specific for primary alveolar hemorrhage. On the other hand, it is not clear the authors have shown that the presence of hemosiderin-laden alveolar macrophages has no import. To show this clearly, the authors should provide the information to the clinician and assess the effect of the new information on clinical decision making.

Where then are we left with the hemosiderin-laden macrophage as a clinically useful test of BAL fluid? For all the reasons listed by these authors, it seems likely that the hemosiderin-laden macrophage is a reasonable marker for the prior presence of BAS. However, because BAS has diverse causes, it seems unlikely that the hemosiderin-laden macrophage will be a diagnostic test specific for primary alveolar hemorrhage. Whether this test is clinically useful is, at present, uncertain. For future investigations, we urge that data be collected so that sensitivity, specificity, and positive and negative predictive value can be calculated. Further, it would be reasonable to provide the data to clinicians to determine whether the data are diagnostically or prognostically useful. It is possible that the true value of this test rests somewhere between the “gold standard” status suggested by Kahn et al and that of being of no clinical value, as suggested by Grebski et al.

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REFERENCES