Proposed Etiologic Mechanisms: We are not qualified to offer a comment on the first two; we agree entirely with the last and would add, "release of an antigen which is able to trigger the clinicopathologic syndrome of sarcoidosis in susceptible individuals," states our opinion precisely; we differ in not restricting this proposed mechanism to individuals with testicular neoplasms. And, we would add to the proposed linkage criteria, appearance of sarcoidosis closely after the administration of radiotherapy or chemotherapy.

More rigorous evidence of a causal relationship is clearly desirable. A positive "auto-Kveim" or lymphocyte transformation response to autologous tumor antigen might be feasible; identification of autologous tumor markers concentrated within sarcoid granuloma is not practicable (written communication, T. Colby, MD, January 1995). Until more rigorous methods become available, we urge that interested investigators who have large databases for analysis use linkage criteria to expand on our observations and confirm or refute our conclusions.

In summary, we believe that a systemic granulomatous process to tumor antigen, analogous in its pathogenesis to the well-accepted entities of local and regional sarcoid reactions, is a unifying explanation of the published observations on this association, and that it meets Ockham's dictum of explanatory parsimony. In this conceptual framework, neoplastic disorders might be grouped with beryllium inhalation as one of the potential causes of a clinicopathologic syndrome nearly indistinguishable from sarcoidosis.

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Does ICU Care Determine the Outcome of Caring for Patients With Pneumococcal Bacteremia?

To the Editor:

Maybe I’m misreading the article or missing the point but didn’t the study by Marlin et al, which appeared in the February 1995 issue (CHEST 1995; 107:457-62), indirectly refute a widely published tenet of critical care, namely that intensive care has no effect on the mortality of pneumococcal bacteremia? Of the 102 patients in their series, 17 who required mechanical ventilation for respiratory failure survived. It seems reasonable to assume that had those patients not received ventilation and ICU care, the mortality of the cohort would have been 41% instead of the observed 25% and thus it appears as if ICU care reduced the mortality by roughly 35%. It would be interesting to get the authors’ comments on this.

Obviously this study does not provide a final answer to the question of the effect of ICU care in pneumococcal bacteremia. Even if a randomized, controlled study of ICU care could be performed ethically, proving an effect would involve numerous unverifiable assumptions, wide confidence intervals (the published mortality being rather wide at 20 to 45%) and statistical power requiring a greater number of patients than all but a few series ever published. The nature of the problem means that for the foreseeable future, we are probably relegated to inferential reports such as the one by Marlin. Nonetheless, the study suggests that, rather than dismissing the effect of ICU care on pneumococcal bacteremia as out of hand, we should reexamine the sometimes-neglected truth of the adage “absence of proof does not constitute proof of absence.”

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To the Editor:

I agree with Dr. Franklin’s comments. In this series of patients, it appears that the intensive care including utilization of mechanical ventilation did have a beneficial effect on reducing mortality. As I personally care for many of the patients in this series, I believe that use of intensive care resources did result in reducing the mortality in a number of specific cases.

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Confession and Some Lessons
The PISA-PED study

To the Editor:

As a humble pedestrian observing the scientific highway, I have always been fascinated by the acumen of the radiologist/nuclear physician when asked to correctly differentiate between segmental and subsegmental perfusion defects on a ventilation/perfusion (V/Q) lung scan. Their ability to classify these defects as $\pm 75\%$, $\leq 75\%$, $>25\%$, and $\leq 25\%$ of the segment particularly confound me. The Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) trial, criteria were partly based on sizing such defects. Self-education dictated that I follow the published PIOPED criteria. Sadly, I have failed on numerous occasions to achieve any degree of accuracy in the sizing of V/Q defects. Reprieve was at hand when Morrell et al conclusively proved that it was very difficult to size lung perfusion defects. However, it appears that this study was marginalized to academic Siberia. There has been no reference to this work in the continuing analysis, discussion, and implications of the PIOPED study. The recent PISA-PED study (CHEST 1995; 107[suppl];335-38S) was more interesting. I presume that the authors realized that classifying defects in percentages was impossible and therefore defined more appropriate and practical criteria. Again they avoided the difficult issue of size as criteria in the PIOPED study. Heresy was thus avoided.

The second lesson that I have learned from the debate generated by PIOPED is to report the lung scan after integrating the clinical,