can be effectively drained percutaneously, a method of treatment known to and practiced by Hippocrates. However, the treatment is only successful if a pleural symphysis has formed. The importance of a pleural symphysis was recognized by late 19th century and in the 1920s and 30s surgeons were producing a pleural symphysis prior to incision and drainage of the abscess. A short segment of the rib overlying the abscess was excised subperiosteally and an irritant such as a strip of gauze was placed into the incision to produce a pleural symphysis. Four to five days later a tube could be inserted into the underlying abscess without danger of collapse of the surrounding lung.

Incision and drainage remained the standard form of therapy until the advent of anesthesia techniques and equipment which permitted open thoracotomy in the early 1940s. For a brief period of time, the treatment of choice for lung abscess was resection via segmental resection or lobectomy. With the advent of effective antibiotics, however, incision and drainage of a lung abscess or excision of the abscesses became virtually unnecessary. Numerous clinical studies demonstrated that these abscesses heal with appropriate antibiotic therapy in the majority of patients.

An occasional patient with a lung abscess is encountered, however, who remains septic in spite of appropriate antibiotic therapy and radiographic evidence of an abscess cavity persists. If the patient is able to tolerate a thoracotomy and lobectomy or segmental resection, this is the procedure of choice. These procedures (under general anesthesia) should be accomplished with a double lumen endotracheal tube in order to prevent the spillage of purulent material into the opposite lung.

A small percentage of patients with lung abscess will not tolerate an extensive operative procedure. In the past, the majority of these patients have had far advanced pulmonary disease. More recently, the situation is more commonly encountered in patients with malignant disease who are being treated with chemotherapy and/or radiation. In this type of clinical situation, percutaneous incision and drainage is the procedure of choice. If there is firm radiographic evidence of a pleural symphysis, then insertion of a tube can be accomplished primarily under local anesthesia. If there is any question, however, as to the absence of a pleural symphysis, it is necessary to establish such a symphysis as previously described. The majority of these patients will respond to incision and drainage and the tube can be removed over a period of time.

R. Robinson Baker, M.D.
Baltimore

The Persistent Need to Improve our Approach to Sarcoidosis

Our understanding of the basic inflammatory processes involved in the pathogenesis of pulmonary sarcoidosis has greatly increased in the past ten years. This has accompanied the introduction of three new laboratory tests which presumably reflect directly the interstitial inflammatory process without resorting to repetitive pulmonary biopsies, namely, (semi)quantitative gallium 67 lung scanning, measurement of serum angiotensin I-converting enzyme activity, and determination of the proportion of helper T-lymphocytes recovered by bronchoalveolar lavage.1-5 Initially, it was believed that these tests were highly specific for sarcoidosis and would serve as the long-awaited biologic markers of the disease, alone or in combination.1-5,8 Unfortunately, it is clear now that they are not specific and offer much less diagnostic help than was expected.7-9 The diagnosis of sarcoidosis is still one of exclusion and requires supporting histologic evidence of noncaseating granulomas.

These novel laboratory techniques were originally considered as sensitive, objective, quantitative, and reproducible tests for monitoring the kinetics of the inflammatory process in the lungs of patients with sarcoidosis in "real time."9-10 This added insight enabled us to identify early in the course of the disease those patients with the so-called active (high-intensity) alveolitis who were thought to be at greater risk of developing pulmonary fibrosis and death from respiratory failure, cor pulmonale, or aspergillosa.4,5 These new tests were considered simpler, more specific indicators of the activity of the disease than the more traditional methods of clinical assessment, chest roentgenogram, and pulmonary function tests.1-4,10 The conventional examinations are only indirect signs of inflammation and, therefore, imprecise reflections of the activity of the interstitial inflammatory process in the lung.

From currently available data in the literature, it seems that these so-called sarcoid-specific tests did not fulfill those expectations. To date, there is little evidence, based on large-scale prospective studies, that these tests are superior in any practical respect to the traditional and more conventional methods of clinical assessment in pulmonary sarcoidosis.7-8,11-13 Furthermore, we still cannot predict the long-term prognosis of patients with pulmonary sarcoidosis by using these new tests, nor can we differentiate from the entire group of patients those with the more progressive forms of the disease, about 30 percent of all cases, who are at greater risk to develop pulmonary fibrosis.5,8,10,11-13 It also appears that serum angiotensin-converting enzyme, bronchoalveolar lavage, and gallium 67 lung
scanning have not provided more precise indications for corticosteroid therapy in patients with pulmonary sarcoidosis.5,14,15

It is reasonable to assume, therefore, that these three new tests provide only complementary information to that obtained by the older examinations concerning the poorly understood inflammatory process in pulmonary sarcoidosis and the state of activity of this process. We cannot justify their routine use in the day-to-day management of patients with pulmonary sarcoidosis in place of the more customary methods of clinical evaluation. An added consideration is the potential hazard to the patient from two to these new methods: exposure to radiation from gallium 67 lung scanning; and the invasiveness of bronchoalveolar lavage. This is especially pressing if these two tests are to be repeated several times in the course of follow-up, as suggested by some authors.5,4,10

In conclusion, at the time of writing, we believe that the combined conventional clinical, roentgenographic, and spirometric evaluation of patients with pulmonary sarcoidosis gives the information necessary for considering prognosis and determining therapy and that the newer studies of serum angiotensin-converting enzyme, bronchoalveolar lavage, and gallium 67 lung scanning offer little additional information critical for these decisions. We would recommend that treatment of patients with corticosteroids in clinical practice be reserved, at present, for those with symptomatic pulmonary disease in the face of deteriorating chest roentgenograms or pulmonary function tests (or both) rather than for those whose only indication for such treatment is an abnormality of any single new test of the activity of the disease mentioned previously. This conservative approach merely emphasizes our need to find more efficient and accurate indices of the activity, prognosis, and, hopefully, etiology of sarcoidosis. Our approach is not meant to inflate the value of the older methods of evaluation, but rather to express regret that the newer and more sophisticated approaches have not apparently fulfilled their promise.

Israel Rubinstein, M.D.; and Gerald L. Baum, M.D., F.C.C.P.
Tel-Hashomer, Israel

References