Transbronchial Biopsy of the Lung

To the Editor:

I have read with interest the editorial by James H. Ellis, Jr., M.D.1 entitled “Transbronchial Lung Biopsy: Variations on a Theme,” which appeared in the October 1975 issue of Chest, as well his article,2 the earlier article by Joyner and Scheinhorn,3 and several other articles on this topic.4-6

Having seen a number of patients with diffuse pulmonary parenchymal disease and some of the patients from one of the previous series,1 I have concluded that the transbronchoscopic lung biopsy is of value in sarcoidosis (stages 1, 2, and 3), in pneumonia due to Pneumocystis carinii, in cryptoginal pulmonary eosinophilic pneumonia, and in certain patients with acute farmer’s lung or chronic eosinophilic pneumonitis where biopsy might be indicated. There is no question about the value of transbronchial biopsy of localized lesions under fluorescent guidance. I believe that the transbronchoscopic lung biopsy for diffuse pulmonary parenchymal conditions can be misleading and that open-lung biopsy is by far the procedure of choice. I am particularly referring to certain disease entities, including chronic interstitial pulmonary fibrosis, Wegener’s granulomatosis, and Goodpasture’s syndrome, where it is important to have adequate tissue for pathologic diagnosis and for immunopathologic and anti-basement membrane antibody studies.

We will never be able to classify and understand the diffuse parenchymal pulmonary diseases, the chronic-hypersensitivity pulmonary diseases, the diffuse pulmonary vasculitides, and many of the newer pneumoconioses without open-lung biopsies, where we have an adequate sample of tissue for pathologic, cytologic, microbiologic, and immunologic studies. In certain other cases of the pneumoconioses, we might, indeed, want polarization and diffraction studies where indicated.

I would make a plea that we be more selective with the technique of transbronchoscopic lung biopsy and that we utilize more open-lung biopsies to obtain more information to guide us in the proper diagnosis and treatment.

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REFERENCES


To the Editor:

The thoughtful comments of Dr. Dines are very much appreciated. There is no question that the small pieces of tissue obtained via transbronchial biopsy have their limitations in pathologic diagnosis. They are particularly useless in attempting to diagnose any kind of pulmonary vasculitis, such as Wegener’s granulomatosis, as the technique attempts to avoid biopsy of vessels whenever possible.

Nevertheless, the tissue is quite adequate for immunopathologic studies, such as fluorescent anti-basement membrane antibody studies. While I was at Walter Reed Army Medical Center, we accumulated a series of patients with Goodpasture’s disease (unpublished data), whose linear basement-membrane staining on tissue from transbronchial biopsy was very impressive. I presented an example of this material at the annual scientific assembly of the American College of Chest Physicians in New Orleans (Nov 7, 1975), and Dr. Joyner presented similar material at the American Otologic Society meeting in Atlanta last spring. There is no limitation to cytologic, microbiologic, and, indeed, polarization and diffraction studies on these pieces of tissue. The material obtained by transbronchial biopsy is also excellent for electron-microscopic studies. Certainly in some pneumoconioses, where the lung needs to be ashed and chemical assay performed, larger amounts of tissue may be necessary; however, microtechniques, even in these areas, and in histochemical studies have been progressing, and less and less pulmonary tissue will be required in the future for establishing such diagnoses. Transbronchial biopsy is of value in stage 1 sarcoidosis, as well as in stages 2 and 3.

In conclusion, while I realize that there are limitations to the technique in question, I do not think that they are as extensive as Dr. Dines does. This is a new procedure, and only time will tell how useful it will be. I hope that transbronchial lung biopsy will expand the role of pulmonary pathologic study in clinical medicine and lead to increased understanding of the pathogenesis of disease, not hold back such progress, as Dr. Dines implied it might do.

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To the Editor:

The validity of transbronchial lung biopsy in diagnosing diffuse pulmonary diseases has been established, and that includes immunologic disease and the interstitial pneumonitis-fibrosis entities.1,3 The willingness of the
local pathologist to make such interpretations is crucial, although comparison with specimens from open-lung biopsies has been made and is good. I agree that where and when certain facilities and protocols allow detailed investigation into the etiology and pathogenesis of the "newer entities" and where larger amounts of tissue are required, open-lung biopsy may be indicated.

My present belief remains, as previously stated by Andersen et al., that open-lung biopsy is reserved for patients in whom other methods (particularly transbronchial lung biopsy) have failed. The decision for open-lung biopsy can also be dictated by results of a transbronchial approach, which to date has remained a more benign technique with only a slightly lower yield of definitive diagnoses.

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Endocarditis of Aortic Valvular Prosthesis due to Listeria monocytogenes

To the Editor:

Listeria monocytogenes is a rare, but well-recognized, cause of infectious endocarditis. To the best of our knowledge, this communication presents the first reported case of endocarditis due to Listeria involving a prosthetic heart valve.

CASE REPORT

A 72-year-old white woman with calcific aortic stenosis underwent aortic valve replacement on June 12, 1972. A Starr-Edwards aortic valvular prosthesis (model 2320, size 10) was inserted. Except for mild congestive heart failure, which was controlled with digoxin therapy, the patient recovered without incident and was discharged on the 18th postoperative day.

The patient did quite well for more than two years. On Sept 9, 1974, she was admitted to another hospital with complaints of palpitations and shortness of breath following four days of night sweats and chills. The patient denied any intercurrent illness, or dental, surgical, or genitourinary procedures. Initially, she was found to have atrial tachycardia (160 beats per minute), which spontaneously converted to sinus tachycardia (120 beats per minute). Subsequently, rapid atrial fibrillation (170 beats per minute) developed.

The patient underwent electrical cardioversion to sinus rhythm. On auscultation a grade-2/6 systolic ejection murmur was present over the upper sternum. No rubs, gallops, or diastolic murmurs were heard. The spleen was not palpable. There were no peripheral stigmata of endocarditis. The white blood cell count was normal, with a shift to the left in the differential cell count. The hematocrit reading was 30 percent, the erythrocytic sedimentation rate was 70 mm/hr, the reticulocyte count was 7.2 percent, and the platelet count was 470,000/cc mm. Bone marrow biopsy was normocellular, with mild normoblastic erythroid hyperplasia. Although the patient was afebrile on admission, blood samples for three blood cultures were drawn. The patient became febrile to 39°C (102.2°F) on the next day, and three additional blood cultures were obtained. All six cultures grew Listeria monocytogenes. Therapy was begun with penicillin (20 million units/day intravenously) and kanamycin (500 mg intramuscularly every 12 hours). The patient became afebrile and remained so for 11 days, at which time she was transferred to the Lenox Hill Hospital, New York.

The same antibiotic regimen was maintained until Sept 27, when the kanamycin therapy was discontinued. On Oct 7, 1974, while the patient was receiving penicillin, a diastolic murmur was heard for the first time, suggestive of para-prosthetic aortic insufficiency. Two days later, the patient developed pulmonary edema, which was managed with digoxin and diuretic therapy. On Oct 13, the patient's temperature spiked to 39°C for the first time since antibiotic therapy had begun.

The patient was taken to the operating room on Oct 15, 1974. At surgery the prosthesis was found to be detached for about one third of its circumference along the left coronary sinus. The valve was covered by a thin layer of friable material. The subvalvular area was heavily covered by a large friable vegetation extending diffusely into the ventricle. A Starr-Edwards aortic prosthesis (model 2320, size 10) was replaced in the annulus. The patient suddenly became asystolic about 15 minutes after cardiopulmonary bypass was discontinued. The possibility of a coronary embolism was immediately considered because of the presence of the ventricular vegetations. Bypass was re instituted, and a Fogarty catheter was passed retrograde from the distal left anterior descending coronary artery. A sudden spurt of blood from the vessel was followed by resumption of spontaneous cardiac activity. Multiple attempts to wean the patient from bypass were unsuccessful. All efforts at resuscitation, including intraaortic balloon pumping, were unsuccessful. The patient was pronounced dead in the operating room after five hours of attempted resuscitation.

At autopsy, there was an extensive early myocardial infarction from apex to base. The mitral valvular apparatus was intact. The excised aortic prosthesis was sterile.

DISCUSSION

Infectious endocarditis due to Listeria monocytogenes is extremely rare, but well recognized.1-4 This organism has also been reported as the cause of a bacterial aortic aneurysm.4 Edelstein et al3 reported Listeria infection of an aortotomy site following aortic valvulotomy for congenital aortic stenosis. Until now, there have been no

CHEST, 69: 6, JUNE, 1976