bacilli. Drug susceptibility studies showed a consistently greater degree of susceptibility to certain drugs than most scotochromogens, which was reflected in the good treatment results with antituberculosis drug regimens. Two patients died with active disease. One had been successfully treated originally and died following a relapse.

On the basis of drug susceptibility studies and treatment experience to date, the best treatment regimen for *M. szulgai* infection appears to be a three-drug regimen consisting of rifampin, isoniazid, and ethambutol. The parenteral drugs (streptomycin, capreomycin, or viomycin) should be useful as a fourth or substitute drug.

Response to chemotherapy was prompt, with sputum cultures converting to negative from three weeks to four months after therapy was started. Follow-up information has indicated a continuing good response. One patient (case 6 of reference 2) relapsed after approximately five years and died with active infection.

The length of therapy was not clearly stated in the earlier cases. The present case was treated for a period of time consistent with our present practice of treating drug-resistant mycobacterial infections until the sputum cultures have been negative for two years. (A summary of the clinical data in all 13 patients is available from the author.)

From a patient with lung disease, the isolation of a scotochromogenic Mycobacterium which shows an unexpected degree of drug susceptibility and biochemical characteristics, as mentioned earlier, should alert the laboratory personnel and the clinician that they may be dealing with *M. szulgai*. It is important to recognize disease caused by this microorganism because, unlike disease caused by other scotochromogenic mycobacteria, the infection will respond well to antituberculosis chemotherapy, with complete recovery of the patient.

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REFERENCES
1 Jenkins PA, Marks J, Schaefer WB: Thin-layer chromatography of mycobacterial lipids as an aid to classification: The scotochromogenic mycobacteria including *Mycobacterium scrofulaceum*, *M. xenopi*, *M. aquae*, *M. gordonea*, and *M. smegmatis*. Tuber. Cerebr. 53:118, 1972

Serial Echocardiographic Abnormalities in Nonbacterial Thrombotic Endocarditis of the Mitral Valve

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This report describes the clinical course and serial echocardiographic abnormalities in a patient with nonbacterial thrombotic endocarditis and repeated embolic episodes. Serial echocardiograms revealed a mass of abnormal echoes on the anterior mitral leaflet and progressive restriction of mitral valvular motion. Cineangiographic studies demonstrated a large filling defect in the area of the mitral valve. Surgical intervention confirmed the presence of a large vegetation on the mitral valve. The surgical specimen consisted of a sterile, partially organized fibrin thrombus. The echocardiographic abnormalities are described and discussed in relation to the clinical and pathologic findings.

Nonbacterial thrombotic endocarditis usually occurs in patients who have another major illness, such as a malignant neoplasm,1-5 heart failure,1 or infection,1 but it may also occur in apparently normal individuals.4,5 The process is thought to begin with formation of a thrombus on either an obviously abnormal valve or on one with minor irregularities.1,4 While it is unclear how associated illnesses contribute to the valvular lesions, a variety of nonspecific stresses have been shown to produce valvular changes in experimental studies.7-9 Intravascular and mural thrombi are frequently found on postmortem examinations of such patients,1,2,5,10 and it is possible that abnormalities of coagulation may be present in some cases.

The lesions of thrombotic endocarditis are clinically important because the vegetations frequently embolize and cause arterial obstruction and tissue infarction.1,2,10,11 Autopsy studies have indicated that through organization and fibrosis, this type of endocarditis can produce severe valvular deformities and can also become the focus of subsequent bacterial endocarditis.9 Diagnosis of this illness in living patients is difficult but should be considered in patients with emboli of unknown origin. Physical examination of the heart often reveals no abnormalities, and there are no useful laboratory tests comparable to blood cultures in bacterial endocarditis.

The purpose of this report is to present the clinical

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course, including serial echocardiograms, of a patient with nonbacterial thrombotic endocarditis who suffered multiple embolic episodes before his mitral valve was replaced with a prosthesis. The echocardiographic findings, not previously reported in association with nonbacterial thrombotic endocarditis, contributed significantly to the management of this patient.

CASE REPORT

First Admission

A 34-year-old white man was admitted with the sudden onset of pain and coldness in his left leg. Two weeks prior to admission, a transient episode of impaired speech and weakness in the left leg had occurred. Three months prior to admission, a normal appendix was removed when emergency surgery was performed for right lower quadrant pain. The past medical history was unremarkable.

On physical examination, the patient's oral temperature was 38.8°C (101.8°F), and his pulse was 114 beats per minute. Cardiac examination disclosed no abnormalities. The leg was cold, pale, and pulseless. An emergency embolec- tomy resulted in a return of pulses to the leg. The surgical specimen consisted of a fibrin thrombus which was free of bacteria, fungi, and tumor cells.

On the fourth postoperative day, left hemiplegia suddenly occurred. The patient's temperature was 39.4°C (103°F), and a grade-2/6 systolic ejection murmur was heard along the left sternal border at this time. Numerous blood cultures were obtained prior to initiating therapy for suspected bacterial endocarditis. The blood cultures were subsequently reported as negative for fungus or bacteria.

The echocardiogram showed decreased motion of the anterior mitral valvular leaflet in early diastole. The posterior mitral valvular leaflet moved normally (Fig 1A). Slight changes in the direction of the ultrasonic beam within the area of the mitral valve persistently disclosed an abnormal mass of echoes in the region of the anterior mitral leaflet (Fig 1B). This mass of echoes disappeared when directing the ultrasonic beam toward either the left atrium or the left ventricular apex. Right cardiac catheterization showed normal pressures and no evidence of a filling defect on angio-

The patient eventually became afebrile and was discharged with a residual left hemiparesis after a six-week hospitalization.

Second Admission

Eight months later, the patient developed the sudden onset of pain and numbness in the right leg. A grade 1/6 presystolic murmur was heard by some examiners. The right foot was cold, pale, and without pulses. The left dorsalis pedis and posterior tibial pulses were absent. Bilateral femoral embolec- tomies were performed with partial restoration of blood flow to the legs. Microscopic examination of the embolic material again showed a thrombus without evidence of bacteria, fungi, or tumor cells.

The echocardiogram then (Fig 2) showed thickening of the anterior leaflet of the mitral valve with markedly decreased motion in early diastole. A dense mass of echoes was again noted in the same area of the mitral valve and was thought to be more prominent than on the previous examination. Motion of the posterior leaflet was obscured by this mass. The left atrial cavity remained free of abnormal echoes.

Cardiac catheterization revealed a large filling defect in the region of the mitral valve. Cardiac exploration was advised, and a large vegetative mass was found attached to the atrial surface of the anterior mitral leaflet (Fig 3). Because the mass could not be completely removed from the leaflet, the valve was replaced with a No. 3 Starr-Edwards ball prosthesis. Microscopically, the vegetation was a partially organized fibrin thrombus without evidence of bacteria, fungi, or tumor cells. The valve appeared fibrotic and moder-ately vascularized. No Aschoff's bodies were noted. The postoperative course was unremarkable, and no embolic attacks have occurred during the ten succeeding months.

DISCUSSION

The clinical and pathologic features of this particular case are consistent with the diagnosis of nonbacterial thrombotic endocarditis and demonstrate how difficult it is to make this diagnosis clinically.

During the patient's first admission, the presence of
fear, multiple emboli, a heart murmur, and abnormal echoes on the mitral valvular leaflet (Fig 1) resulted in therapy for bacterial endocarditis, even though blood cultures were negative and the embolic material removed from the ileofemoral artery was sterile. A diagnosis of atrial myxoma was considered but could not be supported by angiographic studies. In addition, the fact that the abnormal echoes were seen only on the mitral valve and never within the left atrium was additional evidence against a left atrial myxoma.12,13

Eight months later, an etiologic diagnosis was still obscure when the patient was readmitted with another embolic episode. The echocardiogram at that time (Fig 2) revealed thickening and abnormal motion of the mitral valve, which suggested a progressive lesion. In addition, the mass of abnormal echoes within the mitral valve was more prominent than on the previous admission. The angiocardiogram revealed a filling defect in the mitral valvular region, and surgical exploration was undertaken for a definitive diagnosis.

It has been shown that vegetations associated with infectious endocarditis may be detected echocardiographically.14-19 Thus, it is not surprising that a nonbacterial vegetation, as seen in this patient, can also produce similar abnormal echoes on cardiac valves. The fact that the echocardiogram was abnormal at a time when catheterization and cineangiographic studies revealed no abnormalities contributed to the patient’s management in that a second catheterization became obligatory. The abnormal mitral valvular echoes suggestive of vegetations are per se nonspecific findings. An echo-producing mass, regardless of etiology, may produce such abnormal findings. The progressive thickening of the mitral valve seen in this case is consistent with nonbacterial thrombotic endocarditis, but the possibility of either a coexisting healed bacterial endocarditis or an underlying chronic rheumatic valvulitis could not be excluded as a cause of the valvular deformity.

This case study illustrates that echocardiography may play a role in detecting nonbacterial thrombotic endocarditis earlier than other diagnostic procedures in the course of this potentially devastating illness and, thus, is indicated for patients with systemic emboli of unknown etiology.

REFERENCES

10 Greany J, Kiely H, McCaughey WTE: Infective and nonbacterial thrombotic endocarditis: Experience in recent cases.
ANNOUNCEMENTS

Third Annual Seminar, Topics in Pulmonary Disease

The Third Annual Seminar, Topics in Pulmonary Disease, will be held August 22-26 at Colby College/Mid-Maine Medical Center, Waterville, Maine. The faculty will include Drs. Barry Fanburg, John Bartlett, S. Gorbach, John Urbanetti, and others. For information, please contact Mr. R. H. Kany, Director, Special Programs, Colby College, Waterville, Maine 04901.

Postgraduate Course: Chest Disease, 1976

Harvard Medical School Department of Radiology, will present the course Chest Disease, 1976: An Interdisciplinary Approach with Emphasis on Radiology, at the Hyatt Regency Hotel, Cambridge, October 18-22. For further information, contact the Associate Dean, Department of Continuing Education, Harvard Medical School, 25 Shattuck Street, Boston 02115.

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15 Hirschfeld D, Schiller NB: Echocardiographic localiz-