ventilatory therapy was administered by a volume-controlled ventilator.

Anaerobic culture of the aspirate from the left upper lobe grew Bacteroides fragilis, which was resistant to therapy with penicillin and sensitive to administration of chloramphenicol and clindamycin. Therapy with penicillin was discontinued, and 600 mg of clindamycin was administered intravenously every eight hours. The patient required ventilatory assistance for 21 days after the bronchoscopic procedure. The clinical course over the next three weeks demonstrated improvement, with abatement of fever, resolution of the abscess, and clearance of the alveolar infiltrates.

**DISCUSSION**

The primary use of a bronchoscopic procedure in pulmonary abscess is to establish drainage in those patients whose condition does not respond to antibiotic therapy and thoracic physiotherapy. Additional indications include the suspicion of endobronchial obstruction by bronchogenic carcinoma or a foreign body and the need to obtain tracheobronchial secretions for bacteriologic examination.

In our patient, therapy with penicillin and postural drainage were unsuccessful in promoting resolution of the pulmonary abscess. The sequence of clinical events and the disappearance of the air-fluid level coincident with the appearance of new alveolar infiltrates on the chest X-ray film suggest massive aspiration of infected material occurring after the bronchoscopic procedure. Culture of bronchial washings was subsequently helpful in directing antibiotic therapy, despite possible contamination by flora from the upper portion of the airway. Wanner and associates reported the avoidance of contamination by utilizing a sterile brush within a protective polyethylene catheter.

The precipitating factors in the catastrophic drainage of the contents of the cavity in this patient would appear to be sedation and depression of the cough reflex and the manipulation of the abscess by introduction of a catheter with a brush. The following precautions may be helpful in preventing this complication: (1) minimal prebronchoscopy sedation; (2) judicious use of topical lidocaine anesthesia in an effort to maintain some degree of the gag-and-cough reflex; (3) avoidance of manipulation of the abscess by the catheter or the forceps for biopsy; (4) observation in an intensive care unit for a period of up to six hours after the bronchoscopic procedure; and (5) placement of the patient in the lateral position (with the affected lung dependent) until complete return of the gag-and-cough reflex, in an effort to prevent intrabronchial aspiration of infected material.

**REFERENCES**


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**Progressive Development of a Left Ventricular Thrombus**

**Detection and Evolution Studied with Echocardiographic Techniques**

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In a patient with ischemic heart disease and terminal left ventricular failure, echocardiographic techniques (single-crystal M-mode echocardiograms and bidimensional, linear, dynamically focussed multislice recordings) allowed the detection and follow-up of a left ventricular thrombus, later documented at autopsy.

Left ventricular clots are frequently found at postmortem examinations in patients with healed infarctions. A mural thrombus is a nonmobile structure; therefore, its diagnosis is difficult by M-mode echocardiographic studies. In the literature, we found two reports describing the echocardiographic findings in patients with a left ventricular thrombus. We have recently observed the occurrence and evolution of a left ventricular thrombus, progressively adhering to the interventricular septum and to the anterior wall of the left ventricle; this observation, based on serial single-beam and cross-sectional echocardiograms, was confirmed at autopsy.

**CASE REPORT**

A 56-year-old man sustained an extensive anterolateral infarction in 1974 and a stroke in 1975, with residual right hemiparesis. In March 1977, he was hospitalized because of heart failure and was successfully treated with diuretics and digoxin.

On May 18, 1977, the patient was admitted again because of increasing fatigue and dyspnea; on May 20, supraventricular tachycardia produced a drop in blood pressure, and the patient was transferred to our department. On physical examination, we found an extremely sick man with weak pulses and distended jugular veins; the systolic blood pressure was 80 mm Hg, and the heart rate was 110 beats per minute.

The heart was enlarged, with a diffuse apical heave, soft heart sounds, and a gallop rhythm with third and fourth heart sounds but no murmur. Moist rales were heard at both bases. The liver was not enlarged, and there was no edema of the ankles.

The electrocardiogram showed sinus tachycardia (110 beats per minute), with left atrial overload and an old anterolateral infarction. A chest X-ray film showed cardiac enlargement and distention of the upper lobe veins. A thermodilution Swan-Ganz catheter was positioned in the pulmonary artery through an antecubital vein; the right atrial pressure was 18 mm Hg, the pulmonary capillary wedge pressure was 26 mm Hg, and the cardiac output was 3 L/min.

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The heart was examined with ultrasound. A single-beam M-mode echocardiogram (Fig 1) showed a reflecting, mobile membrane-like structure in the left ventricle, separated from the interventricular septum by an echo-free space, yet attached to the apex and to the septum at the level of the left ventricular outflow tract. When the heart was examined with a dynamically focussed multiscan system, a band of echoes was seen extending from the outflow tract to the apex of the left ventricle (Fig 2A). Follow-up examinations showed a decrease in motion of this structure; and two weeks after the initial examination, echoes could be seen between the membrane-like structure and the interventricular septum, progressively filling the echo-free space until the image resembled a thickened interventricular septum (Fig 2C and 3).

Treatment was initiated with dopamine, diuretic drugs, and intravenous administration of heparin; and the patient recovered from an episode of hepatic and renal insufficiency. On May 30, he was discharged from the intensive care unit to the cardiologic ward, where he was improving, until he died suddenly on July 2, 1977. At autopsy, a mural thrombus was found in the left ventricle, adhering to the interventricular septum and to the anterior wall of the left ventricle.

**DISCUSSION**

Arteriosclerotic heart disease is the most frequent form of heart disease associated with mural thrombi of the left ventricle. In a group of patients who underwent car-

![Figure 1](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21012/)

**Figure 1.** M-mode sweep from left ventricle (LV) to aorta (Ao). In left ventricular cavity, mobile echo-producing structure is seen (arrow). RV, Right ventricle; IVS, interventricular septum; LVPW, left ventricular posterior wall; MV, mitral valve; and LA, left atrium (May 23, 1977).

![Figure 2](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21012/)

**Figure 2.** Ultrasonic short-axis cross-sectional images recorded at different stages in development of left ventricular thrombus. A (top) corresponds to M-mode scan in Figure 1 (May 23, 1977). Echo-free space can be seen between band of echoes (arrow) and interventricular septum (IVS). B (center) was recorded four days later (May 27, 1977). Echo-free space is markedly reduced. C (bottom), Fourteen days after first examination, mass of echoes is seen attached to interventricular septum (June 7, 1977). By now, space between echo-producing structure and septum is filled with echoes. RV, Right ventricle; MV, mitral valve; and LV, left ventricle.
diac catheterization because of symptomatic coronary arterial disease, the frequency of mural thrombi detected angiographically was 4.8 percent. In the presence of a previous anterior wall myocardial infarction, the frequency of mural thrombi was 20 to 30 percent. At autopsy, mural thrombi have been noted in 24 percent of the patients dying with healed infarctions.

There are two reports concerning the echocardiographic evidence of an intraventricular thrombus. Levin-

Our report illustrates the development of a left ventricular thrombus that was detected and evaluated with serial ultrasonic examinations. In the left ventricular cavity a peculiar pattern of motion of the membrane-like structure was seen on the initial M-mode echocardiogram (Fig 1). This motion seemed to be related to geometric changes of the left ventricle and to the flow of blood through a small communication in this membrane-like structure. An intracardiac thrombus is probably difficult to visualize because most thrombi are layered and adhere to the ventricular or atrial wall; it may, therefore, be impossible to differentiate this mass from echoes reflected by the ventricular or atrial walls. When not carefully sought on echocardiographic examination, a ventricular thrombus can also be missed because it is frequently localized at the apex of the left ventricle.

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