Figure 2. Postmortem injection of barium sulfate showing right coronary artery. Secondary branch is attenuated (arrow) at perimeter of anterior subepicardial infarct.

ly below the apex of the electrode wire loop, and was confined to the subepicardium. Thus, it is difficult to escape the conclusion that it was caused by the pressure of the wire on the epicardial coronary vessels.

Following the pacemaker implantation, the patient had marked pericarditis, as well as congestive heart failure. It is likely that the edema and inflammation of the pericardium, together with cardiac dilatation, put sufficient pressure on the electrode lead to produce indentation of the myocardium and finally ischemic necrosis.

The patient probably died as a result of the old and recent posteroseptal infarcts, rather than the superficial anterior infarct; however, the possibility of indentation and necrosis of the myocardium is quite clear and particularly significant if the electrode wire should cross a major coronary artery.

The second problem relates to penetration of the sutureless helical-coil electrode into the right ventricular cavity. Although a small thrombus was adherent to the electrode, this is not likely to produce a significant complication in the right ventricle. But a potential problem may exist with the pacing and sensing function of the pacemaker.

The original design of the sutureless electrode had a completely exposed platinum-iridium coil, but the resulting large electrode area, fibrosis, low current density, and sensing problems necessitated a change in design. Present models are coated with silicone rubber so that only the distal three-quarters of a turn is exposed. Thus, it is possible for the entire active portion of the electrode to lie within the ventricular cavity.

The present case showed only a few isolated instances of the pacemaker's failure to capture; however, the potential difficulty which this presages is especially relevant because the more popular implantation techniques describe a right ventricular electrode placement.

A revised implantation technique may avoid the first problem by leaving less generous loops of electrode wires outside the pericardium, and the second problem by placing the electrodes into the left ventricle, as originally suggested, carefully dissecting away any pericardial fat to avoid the coronary arteries. This can be accomplished with a subcostal incision if the heart is gently rotated to the left with hand pressure. Also, it is advisable to check the sensing function by measuring the potential between electrodes with a bipolar electrogram at the time of surgery.

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Mycotic Aneurysm of the Thoracic Aorta Caused by Aspergillus fumigatus

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MYCOTIC ANEURYSM OF THORACIC AORTA 81
A 54-year-old diabetic patient had unexplained fever and embolic occlusion of the splenic, right renal, right hypogastric, right superficial femoral, and left popliteal arteries. *Aspergillus fumigatus* was recovered from a femoral clot. An aortogram revealed a mycotic aneurysm of the thoracic aorta to be the source of the infected emboli. Surgical excision of the aneurysm and therapy with amphotericin B were unsuccessful.

The propensity of the *Aspergillus* species to invade blood vessels during the course of deep-tissue infection is well recognized. The widespread hematogenous dissemination that occurs in severely compromised patients is initiated in this manner. Localized endothelial and subendothelial lesions causing endothritis, endoarteritis, and mycotic aneurysm are being reported with increasing frequency and may be amenable to surgical correction combined with antifungal chemotherapy. This report describes a patient with a mycotic aneurysm of the thoracic aorta due to *Aspergillus* and emphasizes the clinical and roentgenographic findings.

**CASE REPORT**

A 54-year-old black man was admitted to the Veterans Administration Center, Wood (Milwaukee), Wis, on Feb 10, 1975 because of the sudden onset of pain in his left leg. The past history was not significant, except for the discovery of diabetes mellitus in 1973.

Physical examination disclosed a well-developed black man in no acute distress. The temperature was 36.9°C (98.4°F). Findings from examination of the abdomen were normal, and the peripheral pulses were palpable.

Laboratory studies disclosed a hemoglobin level of 13.4 gm/100 ml, a hematocrit reading of 40 percent, a white blood cell count of 8,300/cu mm, and a normal differential cell count. A chest roentgenogram was initially interpreted as normal. A retrocardiac density was apparent when all previous roentgenograms were reviewed after the diagnosis was established (Fig 1).

The patient was treated with bed rest and intravenous administration of heparin. On Feb 12, a bilateral venogram of the lower extremities demonstrated no abnormalities, and heparin therapy was discontinued. A low-grade fever appeared, with peak levels at 38.3°C (101°F), and recurred intermittently over the following five weeks.

On March 19, left flank pain developed. A radioisotopic liver and spleen scan disclosed a defect in the superior aspect of the spleen. On the following day the patient experienced the sudden onset of recurrent pain in the left leg. Superior mesenteric, abdominal aortic, and bilateral femoral arteriograms revealed multiple embolic defects. There was complete occlusion of the right renal artery, splenic artery, right hypogastric artery, right superficial femoral artery, left popliteal artery, and a branch of the renal artery supplying the inferior pole of the left kidney (Fig 2).

On March 21 a right femoral embolectomy was performed, and examination of the sections of the embolus revealed branching septate hyphae near the periphery of the clot. *Aspergillus fumigatus* was cultured, and intravenous therapy with amphotericin B was begun on March 27. A tentative diagnosis of endocarditis due to *A fumigatus* was made, despite the absence of a significant cardiac murmur and the failure to isolate the organism from 11 venous and two radial arterial blood specimens. Cardiac catheterization was performed on March 29, and no valvular lesions were demonstrated; however, dye was noted to fill an aneurysm of the descending aorta (Fig 3).

A posterolateral thoracotomy was performed on March 29. The wall of the aneurysm was found to be thin and friable, and a Dacron sleeve graft was sutured into place. After surgery, the patient was paraplegic, required peritoneal dialysis, became hypotensive, and died on April 2, 1975.

**Mycologic and Immunologic Studies**

Sensitivity of *A fumigatus* to amphotericin B was deter...
Mycotic aneurysm of thoracic aorta

Figure 3. Frame of cineaortogram showing aneurysm of thoracic aorta.

dwelling venous catheters, and the intravenous injection of drugs in addicts have been suggested as possible portals of entry for Aspergillus spp causing endocarditis. Vascular inoculation of this type did not appear to play a role in our patient. The source of the organism remains unknown, and diabetes was the only demonstrable predisposing factor.

The most striking feature of the present case was the embolic occlusion of many major vessels. Vascular vegetations in fungal endocarditis are large, friable, and apparently easily dislodged by turbulent blood flow. In a recent review of 40 cases of endocarditis due to Aspergillus spp, Kammer and Utz noted that the diagnosis was made antemortem in only nine patients. In eight of these patients, the diagnosis was established following identification of Aspergillus spp in clotted material removed at the time of embolectomy; however, from our patient, it is clear that this finding is not pathognomonic of endocarditis.

The difficulty in establishing an antemortem diagnosis of left-sided endocardial or endothelial infections due to Aspergillus spp is related, in part, to our inability to culture the fungus from blood specimens. Kammer and Utz have suggested that the hyphal particles may be too large to traverse the systemic capillary bed. When endocarditis due to Aspergillus sp was suspected in this patient, radial arterial specimens were obtained. The subsequent localization of the aneurysm beyond the aortic arch precluded the possibility of isolating the organism from these samples. Therefore, when Aspergillus sp is cultured from a thromboembolus, an infected thoracic aneurysm should be suspected if the following triad is present: (1) multiple emboli to major vessels, but sparing the head and upper extremities; (2) absence of cardiac valvular abnormalities; and (3) fungi cannot be cultured from upper-extremity arterial blood. Cardiac catheterization with a left ventricular dye injection and a thoracic aortogram are necessary to confirm the diagnosis and localize the aneurysm.

Carriozza and associates recently reported a patient who had endocarditis due to Aspergillus fumigatus on a prosthetic valve and who was cured with a combination of surgical replacement of the valve, short-term therapy with amphotericin B, and long-term therapy with flucytosine. Amphotericin B and flucytosine have been shown to have a synergistic fungicidal effect in vitro. Early surgical treatment and chemotherapy with both of these drugs are recommended for the treatment of cardiovascular aspergillosis.

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Cardiac Tamponade Complicating Closure of a Median Sternotomy*

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A case of intraoperative cardiac tamponade manifested during closure of a median sternotomy is presented. We postulate that cardiac tamponade was caused by acute dilatation of the cardiac chambers as a result of intra-aortic balloon pumping in a patient with aortic and mitral regurgitation. It has been shown experimentally that acute rises in ventricular end-diastolic pressure result in increased intrapericardial pressure and that if a certain point on the pericardial pressure-volume curve is reached, cardiac tamponade will occur. Sternotomy closure was accomplished easily as soon as the need for intra-aortic balloon pumping diminished.

Frequent reports of cardiac tamponade following open-heart surgery have appeared in the literature. Cardiac tamponade may appear within hours of operation, necessitating emergency evacuation1,2 of blood and clot, or may be delayed as long as three months, usually appearing in patients who have been on long-term anticoagulation therapy.3-7 This is a report of cardiac tamponade occurring at the time of operation, ostensiby related to attempted closure of a median sternotomy incision following aortocoronary saphenous-vein bypass grafting. We believe this was the result of acute dilatation of the cardiac chambers subsequent to intra-aortic balloon pumping in a patient with aortic and mitral regurgitation. Only one similar case could be found in a review of the literature of the last ten years.8 Though not strictly analogous to ours, it illustrates a difficult clinical situation not commonly encountered.

CASE REPORT

A 64-year-old diabetic man had an 18-month history of progressively worsening exertional pain in the left anterior portion of the chest. Initially the patient was treated with isosorbide dinitrate and nitroglycerin, and subsequently with propranolol hydrochloride, all of which failed to provide relief. At the ages of 11 and 19 years, he had had two episodes of rheumatic fever manifested by joint pains, the second episode followed by the appearance of a murmur.

On physical examination the blood pressure was 116/70 mm Hg, and the pulse was 100 beats per minute. The jugular venous pressure appeared normal. The lungs were clear. The point of maximum impulse was in the fifth intercostal space just beyond the midclavicular line. There was a palpable and audible apical fourth heart sound. There was a grade 2/6 decrescendo diastolic blow along the left sternal border, with a short early-systolic grade 2/6 murmur in the aortic area and at the apex with no radiation. There was no hepatomegaly or peripheral edema.

A chest x-ray film revealed some increase in the transverse diameter of the heart. The pulmonary fields were clear. A cardiac series revealed no specific chamber enlargement. An electrocardiogram revealed a decrease in closure velocity of the mitral valvular leaflets. Left atrial volume was at the upper limits of normal. An electrocardiogram revealed normal sinus rhythm with minor nonspecific ST-T wave abnormalities.

Complete right and left cardiac catheterization revealed the following pressures: central aorta 110/55 mm Hg; left ventricle, 110/12-17 mm Hg (post "a" wave); mean pulmonary capillary wedge, 13 mm Hg; pulmonary artery, 33/13 mm Hg; right ventricle, 32/12 mm Hg; and mean right atrium, 11 mm Hg. The left ventricular end-diastolic volume was 87 ml/sq m, and the end-systolic volume was 30 ml/sq m, with an ejection fraction of 66 percent. Some apical akinesia was noted, as well as mild aortic and mitral regurgitation. There were no valvular gradients. The left anterior descending coronary artery was diffusely diseased, with proximal 70-percent and 95-percent narrowings. The circumflex coronary artery had a 90-percent proximal lesion, and an obtuse marginal branch was 90-percent obstructed. The right coronary artery (a large dominant vessel) had diffuse irregularities throughout.

At operation, only the left anterior descending coronary artery was considered operable. The coronary end of the graft was completed after 12 minutes of aortic cross-clamping at an esophageal temperature of 32°C (89.6°F). Upon removing the cross clamp, the heart spontaneously resumed normal sinus rhythm, and cardiopulmonary bypass was discontinued. Halfway through the anastomosis of the graft to the aorta, the patient abruptly developed pulmonary edema with a marked drop in blood pressure, and cardiopulmonary bypass was resumed. Upon completion of the anastomosis, an excellent pulse was noted in the graft. Subsequent attempts to discontinue bypass with supportive therapy including dopamine hydrochloride and phenylephrine hydrochloride were unsuccessful until diastolic augmentation was begun with the intra-aortic balloon pump. At the time of sternal closure, four wire sutures were placed around the sternum. The pericardium was not closed. With approximation of the sternal edges, the systolic blood pressure fell from 120 to 70 mm Hg. This was accompanied by a rise in the superior vena caval pressure to 25 cm H₂O. Release of the sternal edges or upward traction on the wires with the sternum approximated caused the blood pressure to rise and the caval pressure to fall. This was repeated several times while the patient received intra-aortic balloon support and pressor therapy. After the patient had stable vital signs for one hour, the intraaortic balloon pump was turned down to a rate of

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