narrow pathway exhibits a low safety factor and that the narrow pathway cannot conduct with the facility of a broad pathway. Given this narrow pathway, a fractionated irregular wave front might have ineffective depolarizing capabilities, such as decremental conduction, and continually become extinguished. Those atrial wave fronts that do reach the ventricle may be unable to excite a sufficiently large volume of myocardium to produce ventricular depolarization, and they may become "damped off."

The pathophysiologic factors which create dissimilar atrial rhythms are unknown but probably relate to the presence of areas of atrial tissue with functionally different electrophysiologic properties. The clinical importance of dissimilar atrial rhythms is similarly not established. One possible important observation has been our failure to terminate atrial flutter with rapid atrial pacing in the presence of dissimilar atrial rhythms. This factor was an important consideration in the present patient, for whom a permanently implanted, radio frequency-controlled, rapidly firing atrial pacemaker was considered to be of potential therapeutic benefit to help manage the recurrent supraventricular tachycardias.

The question of various artifacts mimicking the presence of atrial fibrillation must be raised. The possibility of the catheter doubling on itself was eliminated by observation of the fluoroscopic image. Catheter whip, possible in the right atrium, was not present in the coronary sinus and would be unlikely to occur only in the middle portion of the latter structure. The presence of electrical artifacts due to 60-Hz interference is eliminated by the relatively "clean" baseline. Thus, obvious causes of artifact were eliminated. Finally, studies in animals using bipolar electrodes fixed to the atrial myocardium have established that dissimilar atrial rhythms can be produced experimentally, and these studies provide support that the basic concept can exist.

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REFERENCES
10 Garrey WE: The nature of fibrillatory contraction of the heart: Its relation to tissue mass and form. Am J Physiol 33:397-414, 1914
14 Kahn A, Citron P: Patient initiated rapid atrial pacing to manage supraventricular tachycardia. Circulation 50 (suppl 3):58, 1974

**Spontaneous Thymic Hemorrhage in an Adult**

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Acute hemorrhage in a normal thymus in neonates and infants has been reported in the literature. These patients were known to have an antecedent cause or defects in coagulation. The case of an adult who developed acute hemorrhage in a normal thymus and who was known not to have any defects in coagulation, hypertension, or other underlying cause is reported.

Widening of the mediastinum secondary to dissection of the thoracic aorta is a well-known phenomenon. Hemorrhage in the mediastinum has been reported in the literature to occur in the presence of parathyroid adenomas, after cardiac catheterization, in hemophilia, and in uremia. In 1974, Woolley et al reported two cases in infants, and Sieger et al reported one case in an infant with acute respiratory distress caused by hemorrhage in the normal thymus. All of these patients were known to have defects in coagulation. Fisher and Reis reported the case of an eight-year-old girl who developed cardiac tamponade due to acute thymic hemorrhage following open-heart surgery for the correction of congenital cardiac anomalies.

The purpose of this report is to present the findings in an adult patient who was admitted with an anterior...
Case Report

The patient was a 43-year-old white man who experienced the sudden onset of substernal chest pain radiating to both shoulders 48 hours prior to admission. The pain lasted approximately one hour and then subsided. Several hours later, the patient experienced a similar attack, which brought him to the hospital. He had no shortness of breath, no palpitation, and no loss of consciousness. There was no history of hypertension.

On physical examination, the patient's pulse rate was noted to be 120 beats per minute and regular. His blood pressure, as recorded in both arms, was 140/80 mm Hg. No abnormality was detected in the respiratory and cardiovascular systems. The results of laboratory examinations were within normal limits. The electrocardiogram showed sinus tachycardia. A chest x-ray film taken on Oct 17, 1975 (Fig 1A) revealed a large mass in the anterior mediastinum, contiguous with the ascending aorta. The patient's previous chest x-ray film, taken on March 1, 1975, was normal. The possibility of a dissecting aneurysm involving the ascending aorta was considered. Aortographic studies were performed, and the findings were normal. Tomograms of the mediastinum demonstrated a large well-defined right anterior mediastinal mass continuous with the ascending aorta. There was no calcification within the mass.

Eight days after admission, the patient experienced another episode of substernal pain. A repeat chest x-ray film taken on Oct 25, 1975 (Fig 1B) showed a definite increase in the size of the mass, and the patient's hematocrit reading dropped to 32.7 percent. An emergency pulmonary angiogram was obtained, which revealed no abnormality. An emergency thoracotomy demonstrated an enlarged hemorrhagic thymus. A total thymectomy was performed. Pathological examination revealed normal thymic tissue with recent hemorrhage (Fig 2). The postoperative period was uneventful, except for an attack of pneumonia in the right middle lobe. The results of hematologic examinations to detect defects in coagulation were normal.

In conclusion, four cases of acute hemorrhage in a normal thymus have been reported in the literature. The patients were under eight years of age, and each had some underlying cause. This report is the first case of an adult patient who initially had an anterior mediastinal mass due to spontaneous thymic hemorrhage associated with no known cause.
REFERENCES

Familial Congenital Bicuspid Aortic Valve*
Secondary Calcific Aortic Stenosis and Aortic Aneurysm

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A patient with an aneurysm of the ascending aorta and calcific stenosis of a congenital bicuspid aortic valve, whose brother also had a stenotic congenital bicuspid aortic valve, is described. Predominant aortic stenosis at cardiac catheterization and the presence of an aneurysm distal to and not including the aortic valvular ring made the initial diagnosis of Marfan's syndrome unlikely. Cystic medial necrosis present in the aneurysmal wall probably arose as a consequence of poststenotic dilatation. Adequate noninvasive evaluation of the ascending aorta requires echocardiographic studies, as well as a chest x-ray film.

Dilatation of the segment of the aorta immediately distal to a valvular stenosis has been described.1,2 Dissecting aneurysm of the poststenotic segment has been reported,3,4 and in some cases, dissection occurred after the aortic valve had been replaced by a prosthesis.5,6

The familial occurrence of calcific stenosis of congenital bicuspid aortic valves is rare; we know of only one such report.7 We now describe a case of congenital bicuspid aortic stenosis and aneurysmal dilatation of the ascending aorta with cystic medial necrosis in a patient whose brother also had congenital bicuspid aortic stenosis and a dilated ascending aorta.

CASE REPORT

A 45-year-old white man was found to have an aortic regurgitant murmur in 1972. Angiographic studies revealed a dilated ascending aorta, and a diagnosis of Marfan's syndrome was made. The patient was treated with propranolol and remained free of symptoms. In October 1975, a chest x-ray film showed an increased transverse mediastinal diameter compared with previous films.

The patient's 43-year-old brother was 194.3 cm (6 ft 4 in) tall and weighed 81.6 kg (180 lb). He had a calcified stenotic bicuspid aortic valve replaced in 1972. His aorta and aortic valvular ring were moderately dilated. There was no other family history of cardiac valvular disease. The father, who was 188.0 cm (6 ft 2 in) tall died at the age of 72 years from a stroke. A son, 185.4 cm (6 ft 1 in) in height, and a daughter, 180.3 cm (5 ft 11 in) tall had mild scoliosis. Another daughter, 166.4 cm (5 ft 5 in) tall, had no apparent physical abnormalities.

The patient was 193.0 cm (6 ft 4 in) tall and weighed 90.7 kg (200 lb); he had an arm span of 77 inches and a ratio of the upper segment to the lower segment of 0.95. There was myopia but no iridodonesis. The palate was high, the fingers were long, and the wrist sign was present on the left, but the thumb sign was absent bilaterally. The feet were narrow, and the great toes were disproportionately long. The cardiac rhythm was regular, and there was a diffuse precordial lift but no thrill. A loud aortic diastolic murmur was maximal down the right sternal edge, and a harsh ejection-type systolic murmur was localized to the base of the heart. The

Figure 1. Right anterior oblique view of ascending aorta, as demonstrated during cardiac catheterization after injection of radiopaque dye into aorta. Gross dilatation of ascending aorta, largely localized to region between sinuses of Valsalva and origin of innominate artery is shown.  

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