placed with an R-wave inhibited pacemaker (Medtronic 5931). Because the leads had been in place for several years, it was presumed that the current threshold had decreased to a stable level. The initial width of the generated pulses was measured with a pulse-width monitor and was found to be 0.8 msec. During electrocardiographic monitoring, the width of the pulses was reduced to 0.15 msec, at which point capture was lost. The width of the generated pulses was then adjusted to a final value of 0.3 msec. During the adjustment of the width of pulses in either direction, an artifact resembling a ventricular arrhythmia appeared on the electrocardiogram (Fig 1).

**DISCUSSION**

Noninvasive adjustment of the width of pacemaker-generated pulses has been shown to diminish the amount of current delivered with each impulse. The pulse-width controller is a small instrument that is held directly over the implanted pacemaker. It contains two bar magnets which are rotated by a hand crank and are magnetically coupled with two very small bar magnets located in a potentiometer within the implanted pulse generator. Rotation of the crank in one direction shortens the width of generated pulses, and rotation in the opposite direction lengthens the width of pulses.

When the bar magnets are rotated, a magnetic field is generated, which produces an artifact on the ECG. The faster the bar magnets are rotated, the greater the height and frequency of the artifact (Fig 1). Unless the operator is aware of this phenomenon, the wave form of this artifact may be confused with a ventricular arrhythmia. In our case, it was possible to be reasonably certain that the electrocardiographic abnormality was an artifact and not ventricular tachycardia for three reasons: (1) the abnormality occurred only during the turning of the hand crank; (2) the height and frequency of the artifact both increased as the speed of cranking was accelerated; and (3) the morphologic appearance of the artifact (as noted in the bottom strip of Fig 1), with its narrow width and frequency of 660/min, is inconsistent with ventricular tachycardia.

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**Premature Closure of the Mitral Valve**

**Echocardiographic Clue for the Diagnosis of Aortic Dissection**

**To the Editor:**

In chronic aortic regurgitation the dilated left ventricle can accommodate large diastolic volumes without a significant rise in end-diastolic pressure, while in acute aortic regurgitation, there is marked elevation of the left ventricular end-diastolic pressure, often exceeding the left atrial pressure. This hemodynamic pattern results in premature closure of the mitral valve. Recently, we identified premature closure of the mitral valve by echocardiographic studies in two patients with dissection of the ascending aorta and severe aortic regurgitation.

**CASE REPORTS**

**Case 1**

A 38-year-old man with a known murmur of aortic regurgitation for one year was hospitalized after several days of severe pain in the chest and shortness of breath. His blood pressure was 120/40 mm Hg, and the pulse rate was 110 beats per minute. A grade-4/6 systolic ejection murmur and a grade-4/6 decrescendo diastolic murmur were heard over the aortic area, radiating to the rest of the precordium. Cultures of blood were negative.
CASE 1

On a chest X-ray film the left ventricle appeared enlarged; the mediastinum seemed normal. The electrocardiogram showed sinus tachycardia, a P-R interval of 0.16 seconds, and a pattern of left ventricular hypertrophy. The echocardiogram is seen in Figure 1. At cardiac catheterization the left ventricular pressure was 110/34 mm Hg (Fig. 1). An injection into the aortic root showed +4 aortic regurgitation and dissection of the aortic root (without evidence of a false lumen). At surgery, these diagnoses were confirmed, and a Dacron interposition graft was sewn in place of the ascending aorta. A repeat echocardiogram after surgery showed neither fluttering of the anterior mitral leaflet nor early closure of the mitral valve.

CASE 2

A 69-year-old man was hospitalized because of pleuritic pain in the chest for several weeks and the auscultation of a new murmur of aortic regurgitation. The blood pressure was 150/90 mm Hg, and the pulse rate was 80 beats per minute. A grade-3/6 early diastolic blow was heard along the left sternal border.

The ECG showed normal sinus rhythm, a P-R interval of 0.24 second, and nonspecific changes in the S-T segment and T wave. A chest X-ray film showed the heart to be enlarged in its transverse diameter, with a widened mediastinum. The echocardiogram in seen in Figure 2. At cardiac catheteriza-
tion the left ventricular pressure was 140/30 mm Hg. Aortic angiographic studies revealed a dissection of the ascending aorta and +4 aortic regurgitation (Fig 2). At surgery the ascending aorta was resected, and a Dacron graft was interposed, at the same time restoring the competence of the aortic valve.

**DISCUSSION**

Premature closure of the mitral valve is not a usual feature of chronic aortic regurgitation, but its echocardiographic demonstration in the reported cases of bacterial endocarditis with acute aortic regurgitation has been considered an indication of severe hemodynamic derangement, requiring emergency replacement of the valve.\(^2\) In the cases of aortic dissection, emergency surgery would also be indicated, but valvular replacement often will not be necessary, as seen in our patients. A prolongation of the P-R interval of the ECG, the presence of atrioventricular dissociated rhythms, or acute mitral regurgitation may also cause premature closure of the mitral valve.\(^2\) Associated mitral stenosis or the use of vasodilator drugs can prevent or delay premature closure of the mitral valve.

False-positive echocardiographic diagnoses of aortic root dissection have been reported.\(^4\) Our cases would represent false-negative echocardiograms in the presence of significant aortic root dissection. As the echocardiographic characteristics of aortic root dissection\(^6\) were absent in these patients, we believe that in the presence of aortic regurgitation, the echocardiographic demonstration of premature closure of the mitral valve should suggest the diagnosis of aortic root dissection, especially in the face of negative cultures of blood, and even in the absence of the other echocardiographic signs of aortic root dissection.

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Massive Hemothorax due to Metastatic Malignant Melanoma

To the Editor:

Yeung and Bonnet\(^3\) have reported spontaneous pneumothorax as a complication of metastatic malignant melanoma. We wish to record an additional unusual thoracic manifestation of malignant melanoma by reviewing the findings in a patient who had life-threatening hemothorax as the first evidence of metastasis from this aggressive malignant neoplasm. The thorax is a common site for metastases from malignant melanoma,\(^2\) but associated exsanguinating hemothorax has not been reported.

**CASE REPORT**

A 53-year-old white man underwent wide local excision of a level-2 nodular malignant melanoma from the right shoulder in January 1976. A synchronous dissection of the right axillary lymph nodes was also completed as part of the primary therapy and did not demonstrate any nodal involvement. No further therapy was recommended, and the patient remained completely asymptomatic until Oct 28, 1976, when he was admitted for evaluation of progressive shortness of breath, pleuritic pain in the right side of the chest, and fatigue.

On admission, a chest x-ray film revealed fluid obliterating the base of the right hemithorax. Serial thoracocenteses over three days yielded volumes of bloody fluid from 300 to 500 ml, with progressively larger reaccumulations of fluid in the right hemithorax. The findings from cytologic examination were normal, and cultures of the fluid were negative. Measurements of coagulation factors were normal.

Four days following admission, drainage of the right side of the chest via a tube was instituted. On the following day, after administration of six units of blood, multiple units of fresh frozen plasma, and platelets, the patient underwent a right thoracotomy because of persistent bleeding. Surgical exploration revealed blood oozing from multiple pleural, pericardial, and diaphragmatic implants of metastatic malignant melanoma. The bleeding was partially controlled by multiple applications of hemoclips and electrocoagulation. The chest was drained with multiple tubes, and all bleeding was stopped by the third postoperative day. The patient subsequently was treated with chemotherapy and died from disseminated disease on Jan 28, 1977.

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CHEST, 73: 1, JANUARY, 1978

**COMMUNICATIONS TO THE EDITOR** 123