



**Fig. 2.** X-ray film of the right hand shows marked soft tissue calcification and subperiosteal resorption of the phalanges.

over the same period.<sup>1-6</sup> Certain patient characteristics have been found to correlate with development of aortic stenosis over a short period of time including increased age, low physical work capacity at the time of presentation,<sup>1</sup> and a low gradient on the initial study.<sup>3,4</sup> Other studies, however, have failed to identify any risk factors that predict rapid progression of calcific aortic valve stenosis.<sup>5,6</sup>

In adults under the age of 60 years, aortic stenosis occurs most commonly in the setting of congenital abnormalities or following rheumatic fever or endocarditis.<sup>7</sup> Humoral factors such as hypercalcemia may also play an important role in valvular calcification and stenosis but have gained little attention in the literature. This patient lacked any known predisposing valvular abnormalities yet experienced extensive calcification involving both the aortic valve leaflets and mitral valve annulus over the same period that her calcium and phosphate levels were increased. The concomitant severe metastatic calcification of her soft tissues suggests that secondary hyperparathyroidism was the major causative factor in the subacute development of severe aortic stenosis.

Multiorgan soft tissue calcification commonly occurs in patients with chronic renal failure and has been shown to affect the heart in at least one third of dialysis patients.<sup>8</sup> Although several mechanisms may be involved, secondary

hyperparathyroidism is thought to be a major causative factor. In a postmortem study, parathyroid hyperplasia was found in all six patients with chronic renal failure, who were shown to have extensive calcification of the conduction system.<sup>9</sup> This is the first report, to our knowledge, of the rapid development of aortic stenosis from metastatic calcification of a morphologically normal valve.

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### Provocation of ventricular tachycardia by an automatic implantable cardioverter defibrillator

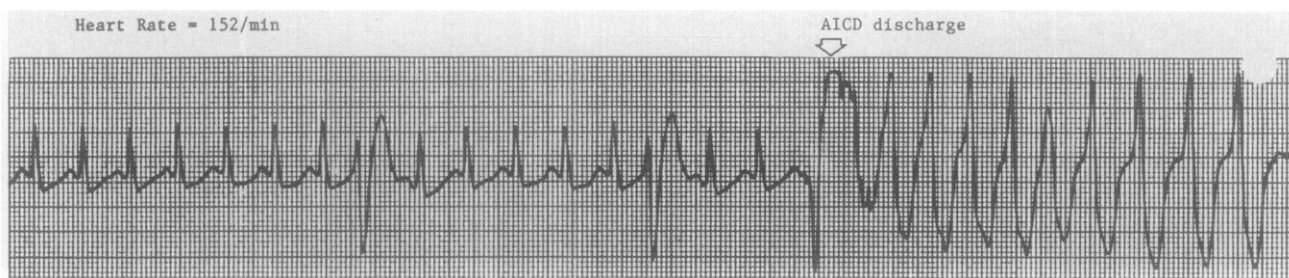
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The automatic implantable cardioverter defibrillator (AICD) has been used to treat patients with life-threatening ventricular arrhythmias, and its efficacy in preventing sudden death has been demonstrated.<sup>1-3</sup> Several possible complications have been recognized as being associated with implantation of the AICD; however, there have been no previous reports of a malignant arrhythmia being induced by an AICD. We describe herein a patient who re-

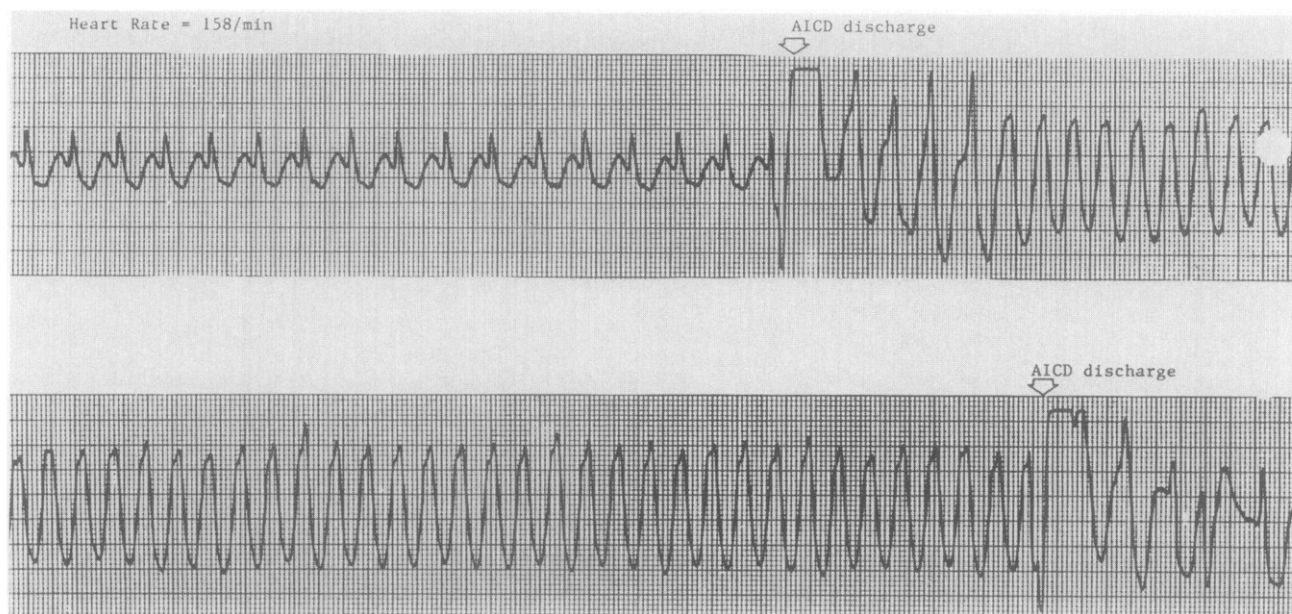
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**Fig. 1.** Nonsustained VT provoked by an AICD shock. The shock was triggered by sinus tachycardia, rate 152 beats/min; there was no VT preceding the AICD shock.



**Fig. 2.** Sustained VT, 250 beats/min, provoked by an AICD shock. The shock was triggered by sinus tachycardia, 158 beats/min. The VT was terminated by a subsequent AICD shock approximately 12 seconds later.

ceived multiple AICD discharges during sinus tachycardia and in whom each AICD discharge provoked ventricular tachycardia (VT).

A 66-year-old man was admitted for treatment of sustained VT. He had a history of myocardial infarction 32 years earlier and had experienced episodes of sustained VT for 5 years. On several occasions, direct-current counter-shocks were required to terminate episodes of VT. Cardiac catheterization revealed high-grade stenoses in the left main coronary artery, the left anterior descending artery, the left circumflex artery and the right coronary artery. The left ventricular ejection fraction was 31%. The patient underwent triple-vessel coronary artery bypass graft surgery. Because of his history of VT, epicardial AICD patches and rate-sensing leads were implanted at the time of bypass surgery for later use with an AICD. Postoperative electrophysiologic testing demonstrated inducible, sustained, monomorphic VT, which had a cycle length of 240 msec. The VT was refractory to quinidine. An AICD pulse gen-

erator was then implanted. The pulse generator was a Ventak model 1520 AICD (Cardiac Pacemaker, Inc., St. Paul, Minn.), which had a rate detection cutoff of 152 beats/min and no probability density function criterion. Testing at the time of implantation of the pulse generator demonstrated that sustained, monomorphic VT was successfully terminated by the first AICD discharge.

After discharge from the hospital, the patient participated in an exercise rehabilitation program. The heart rhythm was monitored during exercise, and the first two sessions of the rehabilitation program were uneventful, with the maximum heart rate during exercise remaining under 150 beats/min. However, during the third session in the recovery phase of a Stairmaster exercise program, the patient experienced three AICD discharges. Each of the shocks was triggered by sinus tachycardia at rates of 152 to 158 beats/min. The first two shocks precipitated nonsustained VT, 9 to 10 beats in duration (Fig. 1). The third shock, which was synchronized to the QRS complex, pro-

voked an episode of sustained VT, rate 250 beats/min, accompanied by palpitations and symptoms of near-syncope (Fig. 2). A subsequent AICD discharge successfully restored sinus rhythm (Fig. 2).

This case report demonstrates that AICD discharges may be proarrhythmic. Although proarrhythmia was never observed during predischarge testing, a synchronized shock triggered by sinus tachycardia reproducibly induced VT immediately after exercise. This case documents a previously unreported complication of the AICD and emphasizes the importance of selecting an appropriate rate cutoff to avoid the triggering of shocks by sinus or supraventricular tachycardia. Although the proarrhythmic potential of the AICD shocks was not apparent during predischarge testing, shocks were never delivered during sinus rhythm. It is possible that ischemia and/or sympathetic activation associated with exercise may have facilitated the induction of VT by the AICD.

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### Low levels of atrial natriuretic factor in patients with atrial standstill

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Atrial standstill is a rare arrhythmia that is caused by the degeneration of atrial myocardium.<sup>1</sup> Echocardiography usually reveals large atria in patients with atrial standstill. Atrial natriuretic factor (ANF) is secreted from atrial myocytes and has potent natriuretic, diuretic, and vasodilator effects. Elevated plasma concentrations of ANF have been reported in patients with various cardiac diseases.<sup>2,3</sup> The predominant stimulus for ANF release appears to be increased atrial transmural pressure with associated atrial stretch.<sup>4</sup> To elucidate whether or not atrial myocardium in

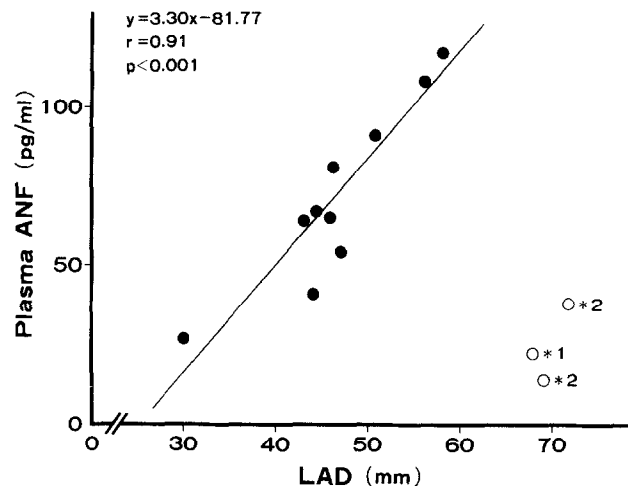


Fig. 1. Correlation between left atrial diameter and plasma concentration of atrial natriuretic factor (ANF). Symbols represent the following patient groups: ●, mitral valve disease; ○, atrial standstill (\*1, patient No. 1; \*2, patient No. 2).

patients with atrial standstill can secrete ANF, we measured plasma concentration of ANF in two patients with atrial standstill, and the data were compared with those in 10 patients with mitral valve disease.

*Patient No. 1.* A 80-year-old man was admitted to the hospital because of hypotension. He had been treated for congestive heart failure for the past 7 years. A grade 3/6 holosystolic murmur was heard at the apex. Electrocardiogram showed junctional rhythm and absence of P wave in all leads. Chest x-ray films showed a remarkable cardiac enlargement. Echocardiography revealed no atrial contraction. Atrial standstill was suspected because of these findings. Right atrial electrogram revealed complete absence of atrial activity. Atrial activity could not be elicited with an electrical stimulus of 10 V. We made a diagnosis of atrial standstill. Mean right atrial and right ventricular pressures (systolic/end-diastolic) were 21 and 50/18 mm Hg, respectively. The patient died suddenly 2 months after the initial measurement of plasma ANF concentration.

*Patient No. 2.* A 69-year-old man was admitted to the hospital for the surgical correction of abdominal aortic aneurysm. He underwent mitral valve replacement at 64 years of age. A grade 4/6 holosystolic murmur was heard at the apex. Electrocardiogram showed junctional rhythm and absence of P wave in all leads. Chest x-ray films showed a remarkable cardiac enlargement and pleural effusion. Furosemide was administered orally for the treatment of heart failure. After treatment, pleural effusion disappeared. Atrial contraction was not proved on echocardiogram. Diagnosis of atrial standstill was confirmed by electrophysiologic study. Mean right atrial and right ventricular pressures (systolic/end-diastolic) were 20 and 88/12 mm Hg, respectively.

*Mitral valve disease.* We studied 10 patients with mitral valve disease (mean age,  $50 \pm 5$  years) as control popula-

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