Ischemically Mediated Sustained Monomorphic Ventricular Tachycardia* Resolution With Anti-ischemic Therapy

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We describe a patient with ischemically mediated sustained monomorphic ventricular tachycardia occurring at rest and resistant to treatment with intravenously administered procainamide. Percutaneous transluminal coronary angioplasty was initially successful, but rest angina and ventricular tachycardia, resistant to procainamide therapy, recurred 2 weeks later and responded to aggressive anti-ischemic medical therapy. We suggest that anti-ischemic medication may be of benefit in patients with malignant ventricular arrhythmias precipitated by spontaneous myocardial ischemia. (Chest 1993; 104:1613-14)

Myocardial ischemia is an important trigger factor in the genesis of ventricular arrhythmias and sudden cardiac death. However, nonfatal ischemically mediated sustained ventricular tachycardia is relatively unusual. Several studies have examined the effects of myocardial revascularization and anti-ischemic pharmacologic therapy upon exercise-induced ventricular arrhythmias. Relatively little is known, however, about the effects of these interventions upon ischemically mediated ventricular arrhythmias unassociated with exercise.

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Case Report

A 51-year-old man with atherosclerotic coronary artery disease was hospitalized for crescendo angina. Five months prior to admission, he underwent 2-vessel coronary artery bypass surgery. He began receiving metoprolol and isosorbide dinitrate and had done well until 3 weeks prior to admission. After admission to the coronary care unit, he experienced chest discomfort and then developed sustained ventricular tachycardia (Fig 1). He converted to sinus rhythm when procainamide was administered intravenously. A maintenance infusion was then begun. Coronary arteriography revealed a patent left internal mammary artery graft to the left anterior descending coronary artery, with poor distal runoff. The left circumflex coronary artery was occluded, and the obtuse marginal branches were diffusely diseased. The graft to the distal right coronary artery was occluded, and there was a filling defect, presumably thrombus, occluding the origin of the large posterior descending artery. Left ventriculography revealed overall normal left ventricular function. Subsequently, the patient experienced intermittent chest discomfort followed by long runs of self-terminating ventricular tachycardia. There was no clear relationship between resting heart rate and the onset of the arrhythmia. Percutaneous coronary artery angioplasty at the origin of the posterior descending coronary artery resulted in some resolution of flow. The arrhythmia gradually subsided, and procainamide therapy was discontinued. Electrophysiologic study, in the drug-free state, revealed no inducible arrhythmias during programmed stimulation using up to three extrastimuli at two right ventricular sites and two paced cycle lengths. Thallium exercise stress test showed small fixed defects inferiorly and apically, but no reversible defects.

Ten days later, the patient's rest angina recurred, followed by sustained ventricular tachycardia with morphologic findings identical to the original tachycardia. Therapy with intravenously administered procainamide was reinstituted, and because he was a poor interventional candidate, the dosage of metoprolol was increased to 150 mg twice a day and therapy with diltiazem to 360 mg daily, was added. After resolution of the arrhythmia, treatment was switched to the oral form of procainamide, and a repeat electro-

Figure 1. An ECG showing the six limb leads during an episode of ventricular tachycardia at a rate of 180 beats per minute. The paper speed is 100 mm/s.
physiologic study again showed no inducible arrhythmias. The exercise stress test was unchanged, and the patient was discharged from the hospital.

The patient was readmitted 6 weeks later because of presumed procainamide-induced lupus. Procainamide therapy was discontinued, but he developed a life-threatening lower gastrointestinal hemorrhage, necessitating emergency transverse colostomy. He experienced no anginal symptoms during this period, and no ventricular arrhythmias were observed. After uneventful recovery, he has remained asymptomatic with no further ventricular tachycardia over a 9-month period while receiving treatment with metoprolol, diltiazem, and isosorbide dinitrate.

**Discussion**

There are several factors suggesting that ventricular tachycardia seen in our patient was ischemically mediated. First, it was always preceded by typical anginal symptoms. Although ischemic symptoms were not preceded by obvious tachycardia, we cannot exclude minor fluctuations in heart rate and blood pressure as trigger factors. Alternatively, spontaneous alterations in coronary vasomotor tone may have been responsible. Second, sustained ventricular tachycardia in patients with ischemic heart disease is inducible with programmed stimulation in greater than 90 percent of cases, though in our patient it was not. Third, the arrhythmia was relatively resistant to anti-arrhythmic drugs but responded dramatically to anti-ischemic therapy, even in the setting of life-threatening gastrointestinal hemorrhage. Although ischemically mediated ventricular tachycardia occurring at rest is unusual, ischemic symptoms are common antecedents to sudden cardiac death, suggesting that this entity frequently may be fatal. Perhaps our patient's well-preserved left ventricular function played a role in his favorable outcome. While the experience with antiarrhythmic drugs in the prevention of sudden cardiac death has been disappointing, the beneficial role of beta blockers has been well established. It has become a common practice to insert automatic implantable cardioverter-defibrillators in patients with sustained ventricular arrhythmias that are not inducible in the electrophysiology laboratory. Our experience suggests that aggressive anti-ischemic therapy may be of benefit in some of these individuals.

**References**


**Unmasking Accessory Pathway Conduction with Adenosine-induced Atrioventricular Nodal Block After Radiofrequency Catheter Ablation**


Radiofrequency catheter ablation is very effective in eliminating conduction over accessory pathways in patients with Wolff-Parkinson-White syndrome. However, accessory pathway conduction recurs in approximately 5 to 9 percent of patients in the weeks to months following ablation. We describe two cases in which intravenous adenosine revealed persistent accessory pathway conduction after apparently successful ablation, thus providing an indication for the delivery of further ablative therapy. Adenosine may improve the long-term efficacy of radiofrequency catheter ablation of accessory pathways by manifesting latent accessory pathway conduction.

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**AV = atrioventricular; ERP = effective refractory period**

Radiofrequency catheter ablation of accessory pathways has been highly effective in the treatment of patients with Wolff-Parkinson-White syndrome. Recent series demonstrate its efficacy in eliminating accessory pathways and thereby sparing patients the need for life-long drug therapy or cardiac surgery. Unfortunately, accessory pathway conduction may recur days to months after apparently successful ablation. Many of these failures may be due to modification of accessory pathway conduction that leaves the tissue viable but transiently unable to conduct. We present two cases in which intravenous adenosine administration revealed persistent accessory pathway conduction following presumed successful radiofrequency catheter ablation.

**Case Reports**

**Case 1**

A 49-year-old man had atrial fibrillation and cardiac arrest. An ECG showed ventricular preexcitation. The pathway was mapped to the lateral mitral annulus. The antegradre effective refractory period (ERP) was 200 ms and the retrograde ERP was less than 220 ms.

Radiofrequency energy (55 V) was delivered for a total of 27 s with preexcitation disappearing after the first 2 s. There was no evidence for accessory pathway conduction during ventricular or atrial pacing after 30 min. Adenosine (12 mg) intravenously demonstrated transient ventricular preexcitation (Fig 1, top). Further radiofrequency energy was delivered to the site and again preexcitation disappeared. After 30 min, adenosine was repeated, producing transient complete atrioventricular (AV) block. The patient's ECG remains free of preexcitation 5 months after the procedure.

**Case 2**

A 41-year-old man had Wolff-Parkinson-White syndrome and a history of atrial fibrillation with rapid accessory pathway conduction.

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