Failure of Adenosine to Terminate Focal Atrial Tachycardia

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SUMMARY. Adenosine has proven effectiveness in the diagnosis and treatment of a variety of tachycardias with both normal and widened QRS complexes in children and adults. Its effectiveness is due to its depressant effects on atrioventricular node conduction. Adenosine might also be effective against automatic tachycardia due to spontaneous activation in partially depolarized cells. This report describes a patient presenting with tachycardia with normal QRS complexes. While the adenosine did not restore sinus rhythm, it did disclose the mechanism of the arrhythmia. Since other investigators have reported successful interruption of automatic atrial tachycardia, this case suggests that automatic atrial tachyarrhythmia may be due to more than one mechanism.

KEY WORDS: Adenosine — Atrial tachycardia

Adenosine has been used in the diagnosis and treatment of a variety of tachycardias including those with normal QRS duration and others with wide QRS complexes in both children and adults [2, 3, 5, 6]. The electrophysiologic basis for the use of adenosine as an antiarrhythmic and diagnostic agent is primarily due to its depressant effect on conduction through the atrioventricular node.

In the present report, we describe a patient with a persistent atrial tachyarrhythmia in whom administration of adenosine was helpful diagnostically but unsuccessful in converting the arrhythmia to sinus rhythm.

Case Report

A 20-year-old white man with Emery-Dreyfuss form of muscular dystrophy was initially evaluated in February 1989 because of tachycardia. The electrocardiogram demonstrated a chaotic atrial rhythm with rapid ventricular response. Administration of a beta-adrenergic blocking agent (nadolol 40 mg, twice daily) resulted in marked suppression of the sinus node and atrial activity with resultant sinus and junctional bradycardia. A ventricular demand pacemaker was subsequently implanted.

During induction of anesthesia for bronchoscopy, the patient developed a regular tachycardia with normal QRS duration at a rate of 200 beats/min (Fig. 1). Eight hours after discontinuing the anesthesia, the tachycardia persisted. Esophageal pacing at rates as fast as 600 beats/min resulted in brief atrioventricular block. However, upon discontinuation of the pacing, the atrial tachycardia was still present and rapid atrioventricular conduction quickly returned.

Adenosine at 12 mg (230 mg/kg) was administered through a peripheral intravenous line and resulted in high-grade atrioventricular block (Fig. 2). The ventricular demand pacemaker responded appropriately to delayed spontaneous ventricular activation. Throughout the 5- to 6-s period of atrioventricular block, the atrial tachycardia was noted to persist. As the effects of adenosine dissipated, an increasing ventricular response to the atrial tachycardia was recorded until the basic underlying rhythm resumed.

The patient was subsequently treated with digoxin (0.125 mg) added to the previous nadolol regimen. This resulted in high-grade atrioventricular block and a ventricular rate of 70–80 beats/min, but the atrial tachycardia persisted.

Discussion

Because of its depressant effect on conduction through the atrioventricular node, adenosine is being used with increasing frequency in the diagnosis and treatment of tachyarrhythmias. The effect of adenosine on arrhythmias confined to the atria remains controversial. Results from cellular electrophysiologic studies on isolated atrial myocytes demonstrate that adenosine depresses intrinsic au-
Automatic activity and inhibits spontaneous depolarization in cells that are already partially depolarized [1]. On this basis, it would be reasonable to expect that adenosine would be successful in suppressing an atrial focal tachycardia provided the underlying mechanism was enhanced automaticity in depolarized cells or microreentry. Most clinical studies, however, have demonstrated failure of adenosine to convert atrial tachyarhythmias to sinus rhythm.

On the other hand, a single report describes transient suppression of a focal atrial tachycardia using adenosine triphosphate [4]. In that report, an electrophysiologic study during tachycardia demonstrated earliest atrial activity from the high anterior right atrium. Administration of adenosine triphosphate resulted in temporary conversion of the atrial tachycardia to normal sinus rhythm. Subsequently, excision of a small atrial aneurysm (presumably in the region of the earliest atrial activation, but not specifically so stated) resulted in restoration of normal sinus rhythm.

Our patient presented with a tachyarrhythmia with normal QRS duration, and inspection of the electrocardiogram did not allow determination of the underlying mechanism. Esophageal pacing at rapid rates resulted in brief atrioventricular block suggesting that the left atrium was successfully stimulated with subsequent conduction through the atria to the atrioventricular node. However, following discontinuation of pacing, the atrial tachyarrhythmia was still present. This finding suggested that the mechanism was a focal tachycardia with entrance block so that the esophageal pacing did not influence the spontaneous electrical activity from the tachycardia focus. Alternatively, the mechanism of the tachycardia could have been intraatrial reentry not affected by rapid atrial pacing, possibly because of the location of the reentry circuit distant from the site of atrial activation from esophageal pacing. Administration of adenosine in a dose adequate to cause complete atrioventricular block had no effect on the presence or rate of the atrial tachycardia.

The difference in response to adenosine between the patient reported here and the patient reported by Perelman and Krikler [4] is not entirely clear. Based on experimental studies it is tempting to speculate that, in the case report from Perelman and Krikler [4], the cells in or near the atrial aneurysm from which the tachycardia apparently arose had unusual electrophysiological characteristics and were partially depolarized. In contrast, the tachycardia in our patient may have arisen from cells that were normally or near-normally polarized and, therefore, unresponsive to adenosine. However, neither report provides data that might confirm the basic underlying mechanism and support such a hypothesis.

Our patient suffers from a form of muscular dystrophy and perhaps there is a diffuse cardiomyopathic process involved. The patient, in whom adenosine was successful in converting the tachyarrhythmia, had a more localized process involving a small atrial aneurysm. It is not clear if this differ-

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**Fig. 1.** Rhythm strip demonstrating tachycardia with narrow QRS complexes, inverted P waves, and a short R-P' pattern.

**Fig. 2.** Lead II rhythm strip during adenosine administration. The tachycardia is interrupted and followed by a three-beat run of a ventricular rhythm, a fusion beat, and a ventricular pacemaker rhythm. The adenosine administration resulted in increased atrioventricular block, but the underlying atrial tachycardia (closed arrows) persists. As the effects of adenosine subside the rapid ventricular response returns.