once again started on a regimen of amiodarone. After 10 days of this therapy, her ventricular arrhythmias, although decreased, still persisted, with sustained runs of VT and multiple nonsustained runs. She was started on flecainide with the usual dose of 100 mg/m^2. After two doses of flecainide, her delta wave was abolished and second-degree AV block occurred, necessitating bradycardia pacing. Her flecainide dose was decreased to 50 mg/m^2 and her premature ventricular contractions (PVCs) were reduced from a baseline frequency of 40% to 1%; also she had no sustained runs of VT. EP evaluation was once again performed with amiodarone and flecainide, and showed no inducible sustained VT.

After 65 days in the hospital, this child was discharged home and was followed for 3 months on a regime of amiodarone and flecainide with no symptomatic tachycardias. She still had short runs of nonsustained VT apparent on Holter examination. Four months postdischarge, the child, while being monitored at home, cried out at night, and on evaluation by the parents was found to be pulseless. Cardiopulmonary resuscitation (CPR) was immediately started and the child was transferred to the nearest emergency room. Evaluation of her rhythm at that time showed a slow sinus rhythm with polymorphic ventricular ectopy. Over the ensuing hours, cardiac function decompensated despite vigorous resuscitation attempts. Pacemaker function was evaluated and was found to be normal. Thirteen hours after her arrest the child died of cardiac failure.

This case is illustrative of the malignant, lethal nature of tachyarrhythmias associated with infantile cardiac tumors. Since these tumors are often multiple and are located in both the atrium and the ventricle, theoretically the patient may present with both SVT and VT. In most reported cases of arrhythmias secondary to cardiac rhabdomyomas, the most common clinical arrhythmia is VT. Wolff-Parkinson-White syndrome and SVT also can be caused by these cardiac tumors. Patients who have both clinically significant SVT and VT secondary to cardiac tumors are rare. The patient described initially had clinically significant antidromic SVT, which failed medical treatment. After surgical therapy for this SVT, VT became her most important clinical dysrhythmia. Even with aggressive EP-guided pharmacologic treatment of her VT and reasonable control documented by Holter, the patient's rhythm disturbance was progressive. Her cardiac death was probably related to VT, but may have been secondary to atrial fibrillation/flutter with rapid antegrade conduction down her bypass tract. Some believe that if measures are taken to control the VT associated with cardiac rhabdomyomas in infancy, the prognosis for an arrhythmia-free and drug-free future is good.[6] This child, however, illustrates the fact that even with the most aggressive therapy, there is a proportion of these patients who will succumb to this condition.

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Magnitude of ST segment depression during paroxysmal supraventricular tachycardia

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Paroxysmal supraventricular tachycardia (PSVT) may be associated with ST segment depression, and a recent study[1] demonstrated that the ST segment depression, even when marked, is not a result of myocardial ischemia. However, no prior studies have quantitated the degree of ST segment depression that occurs during PSVT. The purpose of this study was to describe the prevalence and magnitude of ST segment depression during PSVT and to determine whether the ST segment depression is related to the rate or mechanism of PSVT or to the presence of underlying heart disease.

A 12-lead electrocardiogram was recorded during sinus rhythm and during PSVT in 100 patients who underwent an electrophysiology test for evaluation and management of PSVT. Therapy with antiarrhythmic drugs was discontinued at least 48 hours before the electrophysiology test. The mean age of the patients was 45 ± 18 years (± standard deviation). Eighty patients had no evidence of structural heart disease, 15 had coronary artery disease, and five had miscellaneous forms of heart disease. The mechanism of PSVT was determined using previously described techniques[2] and was found to be atrioventricular (AV) nodal reentrant tachycardia in 52 patients, orthodromic tachycardia using an accessory AV connection in 46, and an atrial tachycardia in two. Only patients who had narrow QRS complexes (<0.12 second) during PSVT were included in

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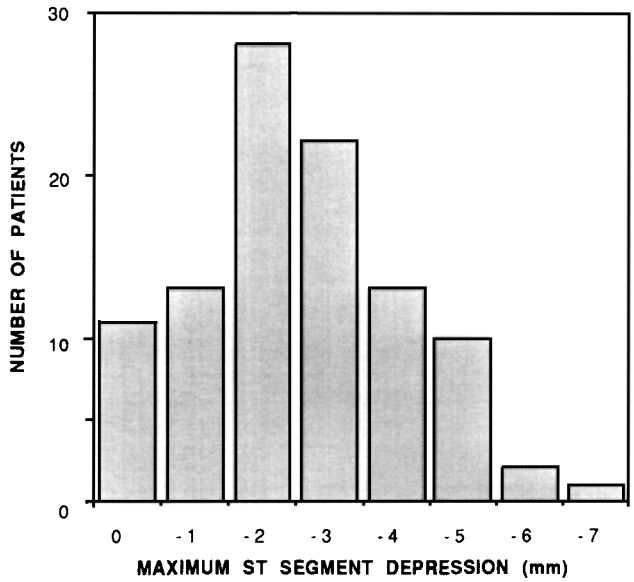

Fig. 1. The maximum amount of ST segment depression present in the 12-lead electrocardiogram during PSVT in 100 patients.

this study. Electrocardiograms were recorded at a paper speed of 25 mm/sec and at a gain setting of 10 mm/mV. The magnitude of ST segment depression was measured manually in every lead to the nearest millimeter, 80 msec after the J point, using the TP segment as a baseline. Intraobserver reproducibility was 98% and interobserver reproducibility between two observers was 97%. The data were analyzed using Student's *t* test, chi square, and linear regression analysis and by analysis of covariance. ST segment depression of 1 mm or more was present during PSVT in at least one lead in 89 of 100 PSVTs. The mean number of leads per electrocardiogram that demonstrated 1 mm or more of ST segment depression was 6.5 ± 2. The maximum amount of ST segment depression in the 100 PSVTs is shown in Fig. 1. ST segment depression of 1 mm or more was present more often in leads II, III, aVF, and V_4 to V_6 (68% to 84%) than in the other leads ($p < 0.01$). The mean magnitude of ST segment depression among the leads that demonstrated ST segment depression was 2.2 ± 1 mm, and the mean maximum magnitude of ST segment depression for each PSVT was 3.0 ± 1.4 mm. There was a significant direct correlation between the rate of PSVT and the number of leads that demonstrated ≥1 mm of ST segment depression ($r = 0.3$, $p < 0.01$) and also with the maximum magnitude of ST segment depression ($r = 0.44$, $p < 0.001$). The mean rate of the 46 orthodromic tachycardias, 190 ± 24 beats/min, was significantly greater than the mean rate of the 52 AV nodal reentrant tachycardias, 173 ± 32 beats/min ($p < 0.01$). The maximum amount of ST segment depression in the orthodromic tachycardias, 3.2 ± 1 mm, was significantly greater than in the AV nodal reentrant tachycardias, 2.6 ± 1 mm ($p < 0.05$). However, after correcting for heart rate, there was not a significant difference in the amount of ST segment depression between the two types of PSVT. Neither the number of leads demonstrating ≥1 mm of ST segment depression nor the mean or maximum magnitude of ST segment depression was related to the age of the patients, the presence of structural heart disease, or the presence of overt preexcitation during sinus rhythm.

The results of this study demonstrate that ST segment depression is quite common during PSVT, with ≥1 mm of ST segment depression being present in approximately 90% of the 100 PSVTs in this series. Marked ST segment depression of 4 mm or more was present in 26% of patients. ST segment depression was found to be a rate-related phenomenon that was independent of patient age and underlying heart disease. This is consistent with the results of a prior study[1] that demonstrated that ST segment depression during PSVT is a physiologic response that is unrelated to myocardial ischemia. Although ST segment depression occurs to a greater degree in orthodromic tachycardia than in AV nodal reentrant tachycardia, this is accounted for by the higher mean rate of orthodromic tachycardia. In conclusion, ST segment depression occurs commonly during PSVT and is a rate-related phenomenon that provides no independent diagnostic information either regarding the mechanism of the PSVT or the presence of underlying coronary artery disease.

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An incessant form of junctional ectopic tachycardia in an adult responsive to a class 1C agent

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An incessant form of junctional ectopic tachycardia (JET) has been well described during early childhood.[1] Also a distinct catecholamine-sensitive paroxysmal JET has been observed in adults. The adult form shares many features with the infant variety but in contrast is almost always paroxysmal, is associated with a distinctly more benign

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