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/m². 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/m² and her prema-
ture 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 sus-
tained VT.

After 65 days in the hospital, this child was discharged
home and was followed for 3 months on a regime of amio-
darone 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. Car-
diopulmonary resuscitation (CPR) was immediately
started and the child was transferred to the nearest emer-
gency 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 func-
tion 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 car-
diac tumors. Patients who have both clinically significant
SVT and VT secondary to cardiac tumors are rare. The
patient described initially had clinically significant an-
tidromic SVT, which failed medical treatment. After sur-
gical 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. 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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New approaches to treatment of incessant ventricular tachy-

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 study1
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 mecha-

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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 V4 to V5 (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 study1 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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