A total of 13 (4.5%) of 290 patients with aborted sudden death had either documented (7; 54%) or strong presumptive evidence of supraventricular tachycardia that deteriorated into ventricular fibrillation. Six (46%) of the 13 had an accessory conduction pathway and either atrial fibrillation (5 patients) or paroxysmal atrioventricular (AV) reentrant tachycardia (1 patient) that deteriorated into ventricular fibrillation. Three patients with AV node reentrant tachycardia and four with atrial fibrillation and enhanced AV node conduction presented with supraventricular arrhythmias that deteriorated into ventricular fibrillation.

Patients were treated with medical, surgical or catheter ablative procedures designed to prevent recurrences of supraventricular arrhythmias. Four patients received an implanted automatic defibrillator, but none had an appropriate device discharge. Over a follow-up period of 41.6 ± 33.6 months, 12 patients are alive without symptomatic arrhythmias. One patient died because of severe chronic lung disease and heart failure.

Supraventricular tachycardia was the cause of aborted sudden death in approximately 5% of patients referred for evaluation of sudden cardiac death. Treatment directed at prevention of supraventricular tachycardia was associated with an excellent prognosis. Current treatment techniques appear to obviate the need for automatic defibrillator therapy in these patients.

That ventricular arrhythmias are responsible for the vast majority of sudden cardiac deaths has been substantiated by epidemiologic field surveys (1–3), ambulatory electrocardiographic (ECG) monitoring (4,5) and electrophysiologic studies (6–13). Less well documented are the incidence of supraventricular arrhythmias responsible for sudden cardiac death and the results of long-term follow-up of surviving patients.

In this report we review our experience with patients with aborted sudden death who had either documented supraventricular arrhythmia before cardiac arrest or strong presumptive evidence of the same, as determined by hemodynamically unstable supraventricular arrhythmias induced in the laboratory. We describe the incidence, mechanism of the arrhythmias, treatment and long-term follow-up of these patients.

Methods

Study patients. The study group of 13 patients was derived from 290 consecutive survivors of out-of-hospital cardiac arrest who were referred to the Moffitt-Long Hospital between April 1979 and September 1990 for invasive cardiac electrophysiologic studies. All patients had at least one episode of cardiac arrest with documented ventricular fibrillation requiring direct current countershock. An effort was made to retrieve all ECGs obtained before the cardiac arrest to discern whether supraventricular arrhythmia was present before ventricular fibrillation was recorded. All patients underwent a complete history and physical examination. Electrocardiograms and echocardiograms were obtained in all patients and cardiac catheterization and coronary angiography were performed as clinically indicated.

Electrophysiologic study. Administration of all antiarrhythmic drugs was discontinued for a period of at least four to five half-lives for participants of this study. After written informed consent was obtained from the patient, multipolar electrode catheters were positioned in the right atrium, right ventricular apex and across the tricuspid valve to record the His bundle potential. An electrode catheter was inserted into the coronary sinus as clinically indicated. A total of three to four surface ECG leads were recorded simultaneously. All patients underwent overdrive right atrial pacing to a paced atrial cycle length of ≥280 ms and overdrive pacing with induction of one or two atrial extrastimuli to atrial refractoriness. Ventricular overdrive pacing was initiated from at least two right ventricular sites and the study included delivery of a minimum of two premature extrastimuli from two right ventricular sites at two basic drive cycle lengths with use of a 2-ms rectangular pulse width at twice diastolic threshold. All patients in the present study underwent a protocol using triple extrastimuli at two sites.
In patients without severe coronary artery disease or cardiomyopathy, the study protocol was repeated after infusion of isoproterenol, tailored to produce a 20% to 30% increase in the heart rate. The end point of the study was either completion of the protocol or induction of a sustained arrhythmia.

**Patient characteristics.** On the basis of the clinical presentation or electrophysiologic study, 40 patients were classified into two groups with respect to the possibility that a supraventricular arrhythmia was responsible for the cardiac arrest. Group 1 consisted of 13 patients who had either or both documented spontaneous supraventricular tachycardia that preceded ventricular fibrillation (7 patients) or supraventricular tachycardia induced in the laboratory that required prompt termination because of hemodynamic instability (10 patients). Hemodynamic instability was defined as systolic blood pressure <80 mm Hg associated with severe symptoms of presyncope, syncope, dyspnea or chest pain. Group 2 consisted of 27 patients in whom both supraventricular and ventricular tachycardia were induced. Because supraventricular tachycardia was not and ventricular tachycardia was associated with hemodynamic instability, these patients were excluded from the study. Thus, only data from group 1 patients are discussed here.

**Follow-up.** All patients in group 1 underwent drug therapy monitored by invasive electrophysiologic study and were discharged either taking drugs that prevented arrhythmia induction or after catheter- or surgical ablative procedures. All patients were followed up in our cardiac arrhythmia clinic or by their private physician. Follow-up data were obtained by interview with the patient or the patient’s family or from the patient’s private physician.

### Table 1. Clinical Characteristics, Therapy and Follow-up in 13 Patients

<table>
<thead>
<tr>
<th>Pt No</th>
<th>Age (yr)</th>
<th>Gender</th>
<th>Symptom Duration (yr)</th>
<th>Cardiac Diagnosis</th>
<th>Arrhythmia*</th>
<th>EF (%)</th>
<th>Therapy</th>
<th>F/U (mo)</th>
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</tr>
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<td>VF</td>
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<td>Surgery</td>
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<td>VF</td>
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<td>VF</td>
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<td>AVN mod, AICD</td>
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<td>VF</td>
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<td>Catheter ablation, AICD</td>
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<tr>
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<td>AF-VF</td>
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<td>Catheter ablation, verapamil</td>
<td>52</td>
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<tr>
<td>12</td>
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<td>1</td>
<td>CAD</td>
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<td>AF-VF</td>
<td>25</td>
<td>Catheter ablation</td>
<td>36</td>
<td></td>
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</table>

*Recorded spontaneous arrhythmia. AF = atrial fibrillation; AICD = automatic implantable cardioverter-defibrillator; AVN mod = atrioventricular node modification; AVRT = orthodromic atrioventricular reentrant tachycardia; CABG = coronary artery bypass grafting; CAD = coronary artery disease; CMP = idiopathic cardiomyopathy; COPD = chronic obstructive pulmonary disease; EF = ejection fraction; F = female; F/U = follow-up; IHSS = hypertrophic cardiomyopathy; M = male; Pt = patient; VF = ventricular fibrillation.

### Results

**Study group.** A total of 13 patients (4.5%) among the 290 patients with aborted sudden cardiac death had documented or strong presumptive evidence that episodes of supraventricular tachycardia triggered malignant ventricular arrhythmias. Spontaneous supraventricular tachycardia that deteriorated into ventricular fibrillation was documented in seven patients from the ECGs recorded by paramedics (one patient) or at emergency facilities (six patients).

**Wolff-Parkinson-White syndrome.** Six (46%) of the 13 patients (Cases 1 to 6) had orthodromic atrioventricular (AV) reentrant tachycardia (Table 1). The mean age of this group was 29.7 ± 13.5 years and the mean duration of symptomatic palpitation before cardiac arrest was 7 ± 6.1 years. Only one patient (Case 6) had cardiac arrest as the first manifestation of the Wolff-Parkinson-White syndrome. Hemodynamically unstable atrial fibrillation occurring spontaneously (Fig. 1) or induced in the catheterization laboratory was present in five patients. Five of the six patients had no evidence of organic cardiac disease (all had normal findings on cardiac catheterization or echocardiographic studies) and one had nonobstructive hypertrophic cardiomyopathy. The latter patient (Case 5) had documented rapid orthodromic AV reciprocating tachycardia that deteriorated into ventricular fibrillation.

In the six patients with the Wolff-Parkinson-White syndrome, the mean accessory pathway antrantode effective refractory period was <242 ± 32.7 ms. The determination of accessory pathway refractoriness was limited by atrial refractoriness in three patients (Cases 1, 2 and 4). The mean shortest pre-excited RR interval during induced atrial fibrillation was 207 ± 24.9 ms (range 180 to 245) (Table 2). The mean pre-excited RR interval during atrial fibrillation was 256 ± 37 ms. Atrial fibrillation could not be induced in one
Figure 1. Patient 3. Baseline 12-lead electrocardiogram showing pre-excitation over a posteroseptal accessory bypass tract. This patient presented with atrial fibrillation with rapid ventricular response (lead II) that deteriorated into ventricular fibrillation. F, L, R = leads aVF, aVL and aVR, respectively.

patient (Case 5), but during the study, this patient had hemodynamically unstable rapid paroxysmal supraventricular tachycardia that required emergency direct current shocks for termination. During the same period, a total of 304 patients with the Wolff-Parkinson-White syndrome underwent electrophysiologic study in our laboratory and 8 (3%) presented with aborted sudden death. Two of the eight patients with Wolff-Parkinson-White syndrome and sudden cardiac death were excluded from this study because the sudden cardiac death was thought to be related to malignant ventricular arrhythmia in older patients with severe coronary artery disease.

AV node reentrant tachycardia. Three patients (Cases 7 to 9) with AV node reentrant tachycardia were included in the present study: their mean age was 46.3 ± 13.6 years (Table 1). One (Case 7) had a 50% lesion of the right coronary artery. This patient had a 4-year history of recurrent palpitation and chest pain and right coronary artery spasm was induced after ergonovine administration. Induced typical AV node reentrant tachycardia was associated with marked ST depression in the inferior ECG leads and a pattern of symptoms (palpitation and chest pain) identical to that of the patient's clinical episodes. The two other patients with AV node reentrant tachycardia had no evidence of coronary artery or cardiac disease. Aborted sudden cardiac death was the initial presentation for two of the three patients.

Induced AV node reentrant tachycardia in all patients was associated with severe hypotension that necessitated prompt termination of the arrhythmia by cardiac pacing (Fig. 2). All patients had the typical form of AV node reentrant tachycardia and the mean tachycardia cycle length was 293.3 ± 20.8 ms (range 270 to 310). The mean AV node effective refractory period was 260 ± 8.2 ms (range 250 to 270) (Table 2). During this same interval, a total of 116 patients had AV node reentrant tachycardia diagnosed by electrophysiologic study in our laboratory and 12 presented with aborted sudden cardiac death. In 5 of the 12 patients, ventricular tachycardia or fibrillation was readily inducible and 4 other patients had no significant hemodynamic compromise; hence, these 9 patients were excluded from the study. For purposes of this study, 3 (3%) of the 116 patients with AV node reentrant tachycardia were thought to have sudden cardiac death due to this arrhythmia.

Table 2. Electrophysiologic Data From Nine Patients With Atrioventricular (AV) Reentrant (n = 6) or AV Node Reentrant (n = 3) Tachycardia

<table>
<thead>
<tr>
<th>Pt No</th>
<th>Induced Arrhythmia</th>
<th>SVT-CL (ms)</th>
<th>AFRR (ms)</th>
<th>ERP (ms)</th>
<th>Location</th>
<th>AVN-ERP (ms)</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>AVRT/AF</td>
<td>300</td>
<td>210*</td>
<td>&lt;230</td>
<td>LP and PS</td>
<td></td>
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<tr>
<td>2</td>
<td>AVRT/AF</td>
<td>269</td>
<td>190*</td>
<td>&lt;290</td>
<td>LL</td>
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</tr>
<tr>
<td>3</td>
<td>AVRT/AF</td>
<td>250</td>
<td>180</td>
<td>240</td>
<td>PS</td>
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<tr>
<td>4</td>
<td>AVRT/AF</td>
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<td>242*</td>
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<td>AVRT</td>
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</tr>
<tr>
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<td>AVNRT</td>
<td>300</td>
<td></td>
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</table>

*Observed after isoproterenol. †Observed after procainamide. ‡Atrioventricular node (AVN) effective refractory period (ERP) not determined because of atrial fibrillation. AFRR = shortest pre-excited RR interval during atrial fibrillation. AP = accessory pathway; AVNRT = atrioventricular node reentrant tachycardia; LL = left lateral pathway; LP = left posterior pathway; PS = posterior septal pathway; SVT-CL = supraventricular tachycardia cycle length; other abbreviations as in Table 1.
Patients with enhanced AV node conduction (atrial fibrillation/flutter). Four (31%) of the 13 study patients (Cases 10 to 13) had atrial fibrillation associated with rapid ventricular responses (Tables 1 and 3). Documented atrial fibrillation or flutter preceded ventricular fibrillation in three patients and was induced in one. These four patients had a mean age of 65.8 ± 3.6 years and all had structural heart disease. Patient 10 had severe hypertensive cardiomyopathy with a prior history of congestive heart failure and an ejection fraction of 30%. Patient 12 had a history of prior myocardial infarction; coronary angiography showed complete occlusion of the left anterior descending artery and a critical lesion in the right coronary artery. She was treated with flecainide for control of atrial fibrillation and premature ventricular complexes and was referred to our hospital after she had a cardiac arrest during an exercise treadmill test (Fig. 3). Patient 13 had severe pulmonary disease, chronic alcoholism and a history of atrial flutter with ventricular response of 250 beats/min (Fig. 4).

All four patients showed enhanced AV node conduction. The mean shortest RR interval during atrial fibrillation was 223 ± 25 ms. The mean AV node effective refractory period was <210 ± 18.3 ms and the mean AV node block cycle length was 241.3 ± 31.2 ms. For Patient 10, the baseline AH interval was 70 ms, but during right atrial overdrive pacing, the His deflection merged with the end of the septal atrial electrogram (Fig. 5A). The maximal AH interval occurred during 1:1 conduction at a paced cycle length of 250 ms (Fig. 5B). These findings were consistent with an atrio-Hisian bypass tract. During induced atrial fibrillation, the shortest RR interval was 220 ms and the patient developed chest pain associated with marked anterior ST segment elevation and a reduction of systemic pressure to 60 mm Hg.

Patient 11 had atrial fibrillation that resulted in ventricular fibrillation (Fig. 6A) and findings consistent with rapid AV node conduction and an atriofascicular bypass tract. Rapid overdrive pacing was associated with shortening of the HV interval and evidence of minor ventricular pre-excitation (Fig. 6B). The AH interval was 45 ms and increased by only 30 ms with atrial overdrive pacing at a cycle length of

<table>
<thead>
<tr>
<th>Pt No</th>
<th>Induced Arrhythmia</th>
<th>A/FRR (ms)</th>
<th>AVN-ERP (ms)</th>
<th>AVN-BCL (ms)</th>
<th>AH (ms)</th>
<th>ΔAH (ms)</th>
</tr>
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<tbody>
<tr>
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<td>&lt;250</td>
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<td>AF†</td>
<td>270/250†</td>
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<td>200</td>
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</tr>
<tr>
<td>12</td>
<td>AF</td>
<td>250</td>
<td>&lt;230</td>
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<td>60</td>
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<td>120</td>
<td>&lt;200</td>
<td>&lt;275</td>
<td>60</td>
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</tr>
</tbody>
</table>

*Atro-His pathway; †Atriofascicular pathway; ‡faster isoproterenol administration; ΔAH = maximal changes in AH interval at maximal right atrial overdrive pacing rates associated with 1:1 atrioventricular conduction. AVN-BCL = AV node block cycle length; other abbreviations as in Tables 1 and 2.
Figure 3. Patient 12. Electrocardiographic (ECG) strips taken from a treadmill exercise test. Surface ECG leads I, II and III are shown during various stages of a modified Bruce protocol. Sinus rhythm (first column) is replaced by atrial fibrillation after 1 min 45 s of exercise (second column), at which time exercise was terminated. Approximately 1 min into recovery (third column), a wide irregular rapid tachycardia emerged, then deteriorated into ventricular fibrillation (fourth column) requiring emergency transthoracic shocks. HR = heart rate.

250 ms. Patient 12 also had evidence of enhanced AV node conduction and induced atrial tachycardia was associated with a rate of 250 beats/min; the shortest RR interval during atrial fibrillation was 250 ms. Patient 13 had atrial flutter with 1:1 AV conduction and a ventricular rate up to 250 beats/min; subsequent atrial fibrillation degenerated into ventricular fibrillation (Fig. 4).

Among the entire group of 290 patients with aborted sudden death, persistent atrial fibrillation was induced in an additional 13 patients, 4 of whom also had inducible sustained ventricular arrhythmias and were excluded from study. Nine patients had inducible sustained atrial fibrillation, but without demonstrated hemodynamic instability during supraventricular tachycardia. Enhanced AV node function was found in two of these nine patients. These two patients were excluded from the study because induced atrial fibrillation did not mimic the spontaneous clinical event. Another two of these nine patients were excluded because the possibility of drug-related proarrhythmia could not be eliminated. Atrial tachycardia could be consistently

Figure 4. Patient 13. Representative surface electrocardiographic recordings showing atrial flutter with 2:1 atrioventricular (AV) conduction (upper right panel). Atrial flutter with 1:1 AV conduction is documented in the middle two strips. The bottom strip shows atrial fibrillation with rapid ventricular response that preceded ventricular fibrillation (recordings are not continuous).
induced in four patients; two of the four had inducible ventricular arrhythmias and none of the remaining patients showed hemodynamic instability during atrial tachycardia.

Treatment and follow-up. The 13 study patients were followed up for a mean of 41.6 ± 33.6 months (range 2 to 132). Patients 1 to 12 are alive and have had no symptomatic arrhythmias or syncope after appropriate treatment of their tachycardia. Patient 13 died of severe chronic pulmonary disease and progressive heart failure. Five of the six patients with the Wolff-Parkinson-White syndrome underwent catheter or surgical ablation of the accessory pathway and one (Patient 1) received drug therapy. Patient 5, with the Wolff-Parkinson-White syndrome and hypertrophic cardiomyopathy, underwent attempted surgical resection of the bypass tract and insertion of an implantable cardioverter-defibrillator because his basic disease process placed him at risk for ventricular arrhythmias. Recurrent device discharges were documented to be due to return of AV reen-
Figure 6. Patient II. A, Rhythm strip showing atrial fibrillation with rapid ventricular response that later developed into ventricular fibrillation. The strips are not continuous.

B, Right atrial overdrive pacing (ROD) produces a pattern of intermittent pre-excitation. The first three complexes show merging of the His deflection with the septal ventricular electrogram. The fourth and sixth complexes are conducted over the atrioventricular node-His axis and are conducted with a short AH interval (50 ms) and a normal HV interval. Note the minor changes in the surface QRS complex (especially in lead II) between pre-excited and normally conducted complexes. We interpret the decreased AH and HV intervals during pre-excited complexes to be due to anterograde conduction over a right atriofascicular bypass tract with retrograde activation of the His bundle. Note the consistent change in His bundle contour comparing normally conducted (beats four and six) with pre-excited complexes. CL = cycle length; other abbreviations as in Figure 2.

trant tachycardia and he was subsequently treated successfully with catheter ablation of the posterior free wall pathway.

Two of the three patients who presented with AV node reentrant tachycardia before availability of the implantable cardioverter-defibrillator (Patients 7 and 9) were successfully treated with drug therapy. Patient 8 was initially treated with flecainide and an automatic defibrillator. She had recurrent defibrillator shocks as a result of recurrence of her AV node reentrant tachycardia (Fig. 7) and insisted on removal of the unit. She subsequently underwent a catheter AV node modification procedure and remains asymptomatic without drug therapy for approximately 1 year.

Three (Patients 10, 11, 13) of the four patients with atrial fibrillation and enhanced AV node conduction did not respond to multiple trials of antiarrhythmic agents including beta-adrenergic blockers and calcium channel blockers and were treated with catheter ablation of the AV junction and pacemaker insertion. The fourth patient (Patient 12) underwent coronary bypass surgery and insertion of an implantable cardioverter-defibrillator and was treated with quinidine and nadolol (Corfrad). After a follow-up period of 26 months, she has not received a single automatic internal cardiac defibrillator discharge.

Discussion

Incidence and Mechanism

Several case reports have documented that patients with supraventricular arrhythmias may present with ventricular fibrillation. However, there is little information concerning the incidence and follow-up of these patients. In a large cohort of patients accumulated over 10 years, we found that the minimal incidence of this association was 4.5%.

Wolff-Parkinson-White syndrome. Although published data (14–16) stress the relation between atrial fibrillation in patients with the Wolff-Parkinson-White syndrome with a short accessory pathway refractory period and sudden death, we found this mechanism in <50% (n = 5) of our 13 patients. In general, our observations confirm the conclusion of a much larger series of patients with Wolff-Parkinson-White syndrome and sudden death reported by Klein et al. (14). All except one of our patients had repeated
episodes of arrhythmias and all had a very short accessory pathway refractory period. One of our patients had dual accessory pathways. Two additional patients with the Wolff-Parkinson-White syndrome and aborted sudden death were excluded because the mechanism of sudden death was most likely related to ventricular arrhythmias, an association previously reported by Lloyd et al. (17).

Atrioventricular (AV) node reentrant tachycardia. We report the largest series of patients with AV node reentrant tachycardia who present with aborted sudden death. Patients in this subgroup were older than those with the Wolff-Parkinson-White syndrome, but only one had associated coronary artery disease. In this patient, coronary spasm was readily inducible with ergonovine and induction of the tachycardia produced chest pain and ST changes identical to those of her clinical episode and to those provoked by ergonovine. We are not aware of prior reports of coronary artery spasm initiated by tachycardia. Atrioventricular node reentrant tachycardia was previously reported by Hays et al. (18) as a cause of sudden death in 1 of 100 patients with aborted sudden death. Benditt et al. (19) also reported a patient with ventricular fibrillation who had AV node reentrant tachycardia and ventricular tachycardia. Although the diagnosis is presumptive in our patients with AV node reentrant tachycardia, therapy directed at tachycardia control prevented recurrence of aborted sudden death.

Atrial fibrillation/flutter with rapid AV node conduction. In four patients atrial fibrillation accompanied by a very rapid ventricular response was associated with aborted sudden death. An atrio-Hisian pathway was found in one patient and an atriofascicular pathway in another. Three of these four patients had significant organic cardiac disease and were older than those with the Wolff-Parkinson-White syndrome. In one patient (Case 12), the conversion of atrial fibrillation to ventricular fibrillation occurred during an exercise stress test while the patient was receiving flecainide therapy. Similar cases have been reported by others (20,21).

Prior reports have documented the association of rapid AV node conduction and sudden death. In 1952, Lown et al. (22) reported the association of a short PR interval and narrow QRS complexes with recurrent atrial fibrillation or atrial flutter, and two of their patients died suddenly.

In our Patient 11, a similar finding occurred, except that minor changes in the QRS complex were identified on the surface ECG recording.

Limitations

Several important limitations of this study should be emphasized. First, clear spontaneous documentation of the supraventricular arrhythmia that deteriorated into ventricular fibrillation was available in only 7 of the 13 patients. The relation between the two arrhythmias in the remaining patients remains inferential. Nevertheless, the inference is plausible for two reasons: 1) induction of supraventricular tachycardia precipitated a hemodynamically unstable situation; and 2) treatment directed at the supraventricular tachycardia appears to have been associated with long-term successful arrhythmia control. More likely, the incidence reported in our study represented a minimal incidence of supraventricular tachycardia as a cause of ventricular fibrillation because aggressive pacing techniques for induction of atrial fibrillation were not routinely applied and isoproterenol provocation was not used in all patients. Although isoproterenol was used in all patients without coronary artery disease and without inducible ventricular tachycardia, it was not applied in those with significant coronary artery disease or cardiomyopathy. A significant number of additional patients had the substrate for supraventricular arrhythmias, including inducible AV node reentrant tachycardia, atrial tachycardia and enhanced AV node conduction, but our study did not conclusively prove that supraventricular tachycardia led to hemodynamic collapse or ventricular fibrillation.

Other limitations include inclusion of patients with hypertrophic or dilated cardiomyopathy in whom inducibility of ventricular arrhythmias may be variable. In addition, hemodynamic instability during tachycardia may have resulted from the infusion of isoproterenol. One of the important arguments supporting a link between supraventricular tachycardia and sudden cardiac death is the patient response to therapy for tachycardia. This conclusion has to be evaluated in light of the relatively small sample size and the expected superior prognosis for these patients.

One patient (Case 9) with AV node reentry experienced hemodynamic collapse with a tachycardia cycle length of 300 ms in the absence of obvious organic cardiac disease. The reason for this response is uncertain but may be related to reflex effects on systemic resistance due to atrial contraction against a closed mitral (and tricuspid) valve. Recurrent cardiac arrest in young persons is much less frequent in those without than in those with obvious organic cardiac disease. Thus, it is difficult to assume that the excellent prognosis was, in fact, related to specific treatment of the AV node reentrant tachycardia in these patients with no structural heart disease.

Clinical Implications

Our observations suggest that patients with aborted sudden death should be carefully screened for possible supraventricular tachycardia, which may have triggered the cardiac arrest. Such an evaluation should include judicious use of
isoproterenol and induction of atrial fibrillation, particularly in patients who do not have inducible ventricular arrhythmias. Patients with the Wolff-Parkinson-White syndrome tend to be younger and to have no associated cardiac disease. Our patients with AV node reentrant tachycardia or atrial fibrillation and enhanced AV node conduction were older and had a higher incidence of associated cardiac disease. The latter consideration is important because arrhythmias that may be well tolerated in the absence of cardiac disease may prove to be lethal when cardiac disease supervenes.

The implantable cardioverter-defibrillator. This device was not available during initial presentation for the majority of our patients. Because the precise documentation linking supraventricular tachycardia with ventricular fibrillation will be absent in many clinical instances, defibrillator insertion may be selected (as in four patients in our series) as a fail-safe procedure. It should be emphasized that the presence of a drug-refractory supraventricular arrhythmia greatly complicates the use of the implantable cardioverter-defibrillator. For example, if shocks are triggered by supraventricular tachycardia, it is likely that the defibrillator will not be effective for this arrhythmia and may conceivably induce ventricular fibrillation. Of the four patients who underwent device insertion, one insisted that the unit be removed after she received multiple discharges documented to be due to supraventricular tachycardia; the other three patients remain asymptomatic on drug therapy and have not received defibrillator discharges. Our observations suggest that appropriate and aggressive management of supraventricular tachycardia may make the insertion of an implantable cardioverter-defibrillator unnecessary for these patients.

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References