Sustained ventricular tachycardia (VT) or ventricular fibrillation (VF) is an infrequent but life-threatening complication of coronary artery bypass grafting associated with a high mortality. The cause is often difficult to determine. Sustained monomorphic VT observed late after myocardial infarction involves reentry through myocyte bands surviving in the infarct. Interruption of coronary blood flow by transcoronary ablation can abolish reentry, and restoration of blood flow can be followed by VT recurrence. Polymorphic VT/VF is more often due to acute ischemia than to chronic infarct scarring. We hypothesized that (1) restoring perfusion to electrically quiescent myocardium in chronic infarct scars causes some cases of postoperative VT, and (2) polymorphic VT is associated with perioperative myocardial infarction. The present report investigated the clinical and electrophysiologic characteristics of patients with sustained VT/VF to determine the association of sustained monomorphic VT and polymorphic VT with prior myocardial infarction, revascularization of infarct scar, and perioperative myocardial infarction.

The medical records of 110 consecutive patients referred for ventricular arrhythmia who had undergone coronary artery bypass grafting between 1981 and January of 1993 were reviewed. A total of 17 subjects with documented new-onset sustained VT/VF occurring within 30 days after coronary artery bypass grafting were identified and comprise the study population. A control group was obtained from review of 119 consecutive patients discharged from UCLA Medical Center after coronary artery bypass grafting between 1992 and 1993 who did not have postoperative VT. In both groups, cardiac surgery was conducted with moderate hypothermia, cold blood cardioplegia, and warm blood cardioplegia reperfusion. Cardioplegia was administered in both antegrade and retrograde fashion. Patients were excluded from the study if they had any previous cardiac surgery or valvular surgery in addition to bypass grafting. Measurements of left ventricular function were obtained from either preoperative left ventriculograms or 2-dimensional echocardiography. Zones of prior transmural infarction were identified by both the presence of preoperative Q waves on the 12-lead electrocardiogram and by ventriculographic or echocardiographic evidence of segmental dyskinesia or akinesia. Operative reports were reviewed for the number and location of coronary artery bypass grafts.

TABLE I  Clinical Characteristics

<table>
<thead>
<tr>
<th>Subjects with VT/VF (n = 17)</th>
<th>Control Subjects (n = 119)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years ± SE)</td>
<td>62 ± 10</td>
<td>64 ± 11</td>
</tr>
<tr>
<td>Male (%)</td>
<td>14 (82)</td>
<td>83 (70)</td>
</tr>
<tr>
<td>Ejection fraction</td>
<td>0.32 ± 0.09</td>
<td>0.49 ± 0.13</td>
</tr>
<tr>
<td>Bypass grafts</td>
<td>3.1 ± 0.9</td>
<td>3.8 ± 1.1</td>
</tr>
<tr>
<td>Diseased vessels</td>
<td>2.8 ± 0.4</td>
<td>2.8 ± 0.4</td>
</tr>
<tr>
<td>Acute infarction*</td>
<td>8 (47)</td>
<td>9 (8)</td>
</tr>
<tr>
<td>Chronic infarct zone (%)</td>
<td>1.5 (88)</td>
<td>77 (65)</td>
</tr>
<tr>
<td>Bypass graft to infarct zone (%)</td>
<td>14 of 15 (93)</td>
<td>49 of 77 (64)</td>
</tr>
<tr>
<td>Ejection fraction</td>
<td>1.4 (82)</td>
<td>74 (62)</td>
</tr>
</tbody>
</table>

*Infarction within 2 weeks of bypass surgery. Values are expressed as mean ± SE unless otherwise noted. VT = ventricular tachycardia; VF = ventricular fibrillation.

Electrophysiologic studies were performed in 16 patients, and included programmed electrical stimulation with up to 2 or 3 extrastimuli from the right ventricle at ≥2 paced drives. Sustained arrhythmias during electrophysiologic studies were defined as >30 seconds in duration. Ventricular fibrillation was defined as a ventricular rhythm with no discernable QRS complexes requiring defibrillation; ventricular flutter as a ventricular rhythm with a sinusoidal QRS morphology requiring cardioversion; sustained monomorphic ventricular tachycardia as ventricular tachycardia of 1 QRS morphology requiring an intervention for termination; sustained polymorphic ventricular tachycardia as ventricular tachycardia with changing beat-to-beat QRS morphologies requiring cardioversion; and perioperative infarction as myocardial infarction occurring within 14 days of coronary artery bypass surgery assessed by creatine kinase-MB elevation greater than twice normal value and by new Q waves on the 12-lead electrocardiogram.

Continuous data are expressed as mean ± 1 SD. Groups were compared with the Student’s t test and chi-square tests. Fisher’s exact test was used for dichotomous variables when the number of events in any cell was <5.

Patients with postoperative VT/VF are compared with controls in Table I. Patients experiencing new-onset sustained VT had worse preoperative left ventricular function, and 15 of 17 (88%) had a zone of old infarction compared with 65% of controls (p = 0.04). Patients with VT/VF were also more likely to have had an acute myocardial infarction within 2 weeks of surgery. Placement of a bypass graft to a chronically occluded vessel was not performed statistically more often in patients with VT. However, of subjects with a chronic infarct zone, 93% with VT/VF versus 64% of controls had a bypass graft placed to a chronically occluded vessel (p = 0.03).
Sustained monomorphic VT was the presenting VT morphology in 11 of 17 subjects (65%). The majority (64%) had no evidence of perioperative myocardial infarction, but all had a zone of old infarction. Patient 1 presenting with sustained monomorphic VT refused electrophysiologic studies and was discharged on amiodarone. Monomorphic VT was inducible at electrophysiologic study (mean cycle length 396 ± 112 ms) in 8 of the remaining 10 patients (80%) (Table II). VT was noninducible in patient 8 with perioperative infarction taking β-blocker therapy, and in patient 5 without perioperative infarction taking amiodarone.

Polymorphic VT/VF was the presenting morphology in 6 of 17 subjects (35%), and most had had perioperative myocardial infarction (67%). Only 2 of 6 (33%) had inducible monomorphic VT at subsequent electrophysiologic studies (mean cycle length 305 ± 35 ms) (Table III). During a mean follow-up of 35 ± 29 months, there were 4 deaths in patients presenting with sustained monomorphic VT. The cause of death was recurrent sustained VT in 1 subject, congestive heart failure in 2, and unknown in 1. There were no deaths in the group with polymorphic VT/VF.

New-onset monomorphic VT after bypass surgery was associated with old infarct scarring and may, in some cases, be due to revascularization of an area of prior infarction. Polymorphic VT/VF is usually associated with acute ischemia/infarction.

New-onset postoperative sustained VT/VF is a life-threatening and poorly characterized clinical problem. Previous reports of VT/VF associated with cardiac surgery have included patients undergoing valvular surgery, which itself may be an independent risk factor for sustained arrhythmias, and have not fully characterized the arrhythmia morphology and inducibility with electrophysiologic relevant information. Sustained monomorphic VT is associated with old infarct scarring and is inducible at electrophysiologic study, consistent with persistence of an arrhythmogenic substrate. Ours is the first study utilizing a control group which demonstrates an association between revascularization of an area of prior infarction subtended by a chronically occluded vessel and the development of sustained VT/VF. Whereas early restoration of perfusion to an infarct zone promotes acute and chronic electrical stability, the effects of very late restoration of blood flow on electrical properties of infarcted myocardium is unclear. Most patients with sustained VT/VF have some source of blood flow to the arrhythmogenic focus. In one instance, VT that was controlled after transcoronary ablation recurred when new collateral flow to the region developed. It is theoretically possible that providing flow to chronic infarcts may therefore have a proarrhythmic effect in some cases. The frequency with which grafts were placed to infarct vessels in control versus subjects with VT suggests that this risk is small, but further study is needed to determine the potential significance.

Polymorphic VT/VF is often associated with acute myocardial infarction, and electrophysiologic instability may be transient. Polymorphic VT/VF after coronary bypass may represent acute ischemia, late phase dys-
rhythmias occurring in ischemic tissue, or reperfusion arrhythmias. Potential mechanisms of polymorphic VT include alterations in passive and active membrane properties, resulting in early afterdepolarizations or triggered activity as opposed to reentry involving a fixed arrhythmogenic substrate.2,12

The results of this study suggest that polymorphic VT occurring after coronary artery bypass grafting warrants a therapeutic approach targeted at treatment of myocardial ischemia.


Natural History and Management Strategies of Automatic Atrial Tachycardia in Children

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Automatic atrial tachycardia (AAT) can be defined as a supraventricular tachycardia initiated and sustained by an automatic nonsinus atrial pacemaker, characterized by distinctly visible P waves with an abnormal frontal plane axis and atrial activation sequence.1-2 AAT accounts for 4% to 6% of supraventricular tachycardia and is frequently associated with significant cardiac dysfunction.1-6 Reviews of recent reports on management of AAT have varying recommendations, including antiarrhythmic medications7-10 and surgical11-13 ablation, whereas some cases of spontaneous resolution of tachycardia have been described.5,7,10 Several studies of antiarrhythmic drug treatment for AAT have emphasized multiple trials of single drugs used alone in a serial manner except in combination with digoxin.2,5,7,91 Walsh et al18 recently recommended radiofrequency catheter ablation as the preferred first-line therapy for patients with AAT and depressed myocardial function. This study evaluates the response of AAT to combination antiarrhythmic therapy in the event of failure of single-drug therapy, including follow-up without medications.

We collected information on 8 patients (4 boys and 4 girls) with AAT referred to the Children's Memorial Hospital between January 1987 and March 1994. Ages ranged from 3 months to 18 years (median 2.6 years) including 3 infants aged <5 months. The diagnosis of AAT was made on the basis of generally accepted electrocardiographic criteria: (1) distinctly visible P wave with an abnormal axis, (2) P waves at the onset of tachycardia similar to the morphology of subsequent P waves, (3) 2° atrioventricular block without interruption of AAT, and (4) rate variability with a "warm-up" at initiation and a "cool-down" at termination.1,2

Criteria for diagnosis included (1) inability to initiate or terminate tachycardia with programmed atrial stimulation, (2) atrial activation sequence suggesting a nonsinus origin of the pacemaker, and (3) reset behavior similar to the sinus node where the ectopic pacemaker was reset by a premature beat to a degree proportional to the prematurity.2,14

Therapy was initiated with a single drug during continuous electrocardiographic monitoring. If a single oral drug was not successful, a second drug was added, usually within 2 weeks. Success with antiarrhythmic therapy was defined as achievement and maintenance of sinus rhythm for >80% of the monitored duration and restoration of normal left ventricular function.

Periodic electrocardiography, echocardiography, and Holter monitoring were performed after successful drug therapy at 3- to 6-month intervals. Liver and thyroid