New-Onset Sustained Ventricular Tachycardia After Cardiac Surgery

Jonathan S. Steinberg, MD; Abhishek Gaur, MD; Robert Sciacca, Eng ScD; Edith Tan, RN, MA

Background—The de novo occurrence of sustained ventricular tachycardia (VT) after CABG has been described, but the incidence, mortality rate, long-term follow-up, and mechanism are not well defined.

Methods and Results—This prospective study enrolled consecutive patients undergoing CABG at a single institution. Patients were followed up for the development of sustained VT, and a detailed analysis of clinical, angiographic, and surgical variables associated with the occurrence of VT was performed. A total of 382 patients participated, and 12 patients (3.1%) experienced ≥1 episode of sustained VT 4.1±4.8 days after CABG. In 11 of 12 patients, no postoperative complication explained the VT; 1 patient had a perioperative myocardial infarction. The in-hospital mortality rate was 25%. Patients with VT were more likely to have prior myocardial infarction (92% versus 50%, \( P < 0.01 \)), severe congestive heart failure (56% versus 21%, \( P < 0.01 \)), and ejection fraction \( < 0.40 \) (70% versus 29%, \( P < 0.01 \)). When all 3 factors were present, the risk of VT was 30%, a 14-fold increase. Patients with VT had more noncollateralized totally occluded vessels on angiogram (1.4±0.97 versus 0.54±0.7, \( P < 0.01 \)), a bypass graft across a noncollateralized occluded vessel (1.50±1.0 versus 0.42±0.62, \( P < 0.01 \)), and a bypass graft across a noncollateralized occluded vessel to an infarct zone (1.50±1.0 versus 0.17±0.38, \( P < 0.01 \)). By multivariate analysis, the number of bypass grafts across a noncollateralized total occlusion in a vessel supplying an infarct zone was strongly and independently associated with the development of VT. (Circulation. 1999;99:903-908.)

Conclusions—The first presentation of sustained monomorphic VT in the recovery period after CABG is uncommon, but the incidence is high in specific clinical subsets. Placement of a bypass graft across a noncollateralized total occlusion in a vessel supplying an infarct zone was strongly and independently associated with the development of VT.

Key Words: bypass surgery tachyarrhythmias

Myocardial revascularization performed during CABG surgery for patients with critical coronary artery stenoses can reduce the incidence of sudden cardiac death in selected patients.\(^5\) CABG also contributes independently to a favorable prognosis in survivors of cardiac arrest treated with specific antiarrhythmic interventions.\(^6\) In addition, selected patients who have experienced a cardiac arrest due to critical coronary artery stenosis may have complete therapy without need for adjunctive antiarrhythmic interventions when treated with only CABG.\(^7\)

CABG clearly performs an antiarrhythmic function in selected patients who have experienced previous ventricular tachycardia (VT) and/or ventricular fibrillation (VF). There is, however, a specific subset of patients whose ventricular arrhythmias are aggravated by the surgical procedure. This may take the form of the benign presence of increased ventricular ectopy,\(^8\) but more importantly, the de novo occurrence of ventricular tachyarrhythmia as a consequence of bypass tract surgery has been reported.\(^9-10\) Several case reports and small series\(^5,9,10\) have anecdotally described the development of sustained VT in the recovery period after CABG, but the incidence, mortality rate, long-term follow-up, and mechanism underlying arrhythmia occurrence are poorly defined.

The present prospective study was designed to fully characterize the clinical characteristics and course of patients who develop sustained monomorphic VT in the recovery period after CABG. In particular, a detailed analysis was performed in an attempt to better understand the mechanism underlying VT development in this unique group of patients, with a specific emphasis on the relationship of coronary reperfusion and its potential proarrhythmic effect.

Methods

Patient Selection

The study was performed prospectively. Consecutive patients undergoing nonemergent CABG were enrolled preoperatively. All patients signed an informed consent form approved by the Institutional

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Review Board of the St Luke’s–Roosevelt Hospital Center. Preopera-
tive clinical data and details regarding coronary anatomy (see
below) were collected and recorded. Patients were monitored in the
hospital for the occurrence of sustained VT, which was the primary
description of this investigation. Sustained VT was defined as a
uniform tachyarrhythmia (≥120 bpm) of ventricular origin that had
a duration of ≥30 seconds unless terminated earlier because of
hemodynamic collapse. There was no upper rate cutoff for VT rate.
VT occurring ≥24 hours after surgery during the recovery phase was
included. VT/VF on the day of surgery was excluded because of the
difficulty in differentiating causation in the very early postoperative
setting. VT occurring within 30 days of surgery after patients were
discharged from the hospital also met end-point criteria. Events
occurring beyond 30 days were excluded. Patients who had a
previous history of sustained VT or cardiac arrest before the
performance of CABG were excluded from the study.

With the development of VT, standard advanced cardiac life
support interventions were made to terminate the arrhythmia and to
stabilize the patient. Clinical management of the tachycardia, includ-
ing diagnostic evaluation, electrophysiology investigation, and long-
term treatment, were performed at the discretion of the primary
physician and appropriate consultants and according to generally
accepted clinical principles. The records were reviewed in detail at
the time of each event to assess the presence of any acute reversible
or correctable cause of the arrhythmia, including metabolic alter-
ations, myocardial ischemia/infarction, pericarditis, congestive heart
failure (CHF), and potentially proarrhythmic medications.

Analysis of Coronary Arteriogram and
CABG Procedure
The results of the preoperative coronary arteriogram were reviewed
and recorded for each patient. The presence of significant stenoses,
defined as ≥70% stenosis of a major epicardial vessel, was noted.
The number and location of these lesions were described. In the
presence of a total occlusion, the presence of collateral circulation to
the occluded bed was also recorded. Vessels were defined as a
noncollateralized occluded vessel if there was no visible distal flow
from any source, including collaterals, in a major epicardial vessel
distribution.

Surgical variables were routinely collected. The surgical cross-
clamp duration and details of bypass graft placement and numbers
were noted. Bypass grafts were arterial or venous conduits placed
distal to a stenosed segment of a coronary artery or arteries.
Placement of a graft into a vessel that subtended an infarct zone was
carefully correlated. An infarct zone was defined as the presence of a
severely hypokinetic, akinetic, or dyskinetic myocardial segment.

Follow-Up
The nature of medication use during and after hospitalization was
monitored. In-hospital and out-of-hospital mortality and attention to
the cause were tracked. The development of recurrent ventricular
tachyarrhythmias, both in hospital and after discharge, was followed
and investigated.

Statistics
Continuous data are reported as mean±SD. Categorical data were
compared statistically by χ² test. Continuous data were compared
statistically by Student’s t test. Univariate and multivariate associ-
ations of clinical, angiographic, and surgical variables with the
defined end point were tested with a logistical regression analysis.
Statistical significance was present when the probability value
was P<0.05.

Results
Study Population
A total of 382 patients were enrolled in the study. Details of the
clinical characteristics of the patients are provided in
Table 1. The patients were predominantly male, and two
thirds had a history of prior myocardial infarction (MI). Of
the 196 patients with prior MI, 35 were within 3 weeks before
surgery. A significant minority had severe CHF and reduced
left ventricular (LV) function. In addition to CABG, 5
patients had aortic valve replacement, 7 had mitral valve
replacement, and 1 had LV aneurysmectomy. Patients were
hospitalized a median of 9 days.

Development of VT and Clinical Course
A total of 12 patients (3.1%) developed sustained monomor-
phic VT during the in-hospital postoperative period. In all 12
patients, ≥1 episode of monomorphic VT occurred. In 4 of
these 12 patients, there were additional episodes of poly-
 morphic VT or VF during their postoperative period. The day
of the first episode ranged between day 2 and day 18. None
of these patients with VT had experienced VT/VF on post-
operative day 1. VT occurred 4.1±4.8 days after surgery, and
a histogram displaying the day of onset is shown in the
Figure. No additional events in these 12 patients or new
events in the other patients occurred after hospital discharge
for up to 30 days after surgery.

Of the 12 patients, the review of the medical records and the
events surrounding the initiation of VT revealed no evidence of an electrolyte or metabolic abnormality, myocard-
cial ischemia or infarction, new or worsening CHF, the
presence of potentially proarrhythmic medication (including
intravenous pressors and inotropes and antiarrhythmic
agents), or the presence of any serious postoperative compi-
nication in 11 patients. In 1 patient, perioperative MI occurred
2 days after surgery as documented by ECGs and enzymes,
and VT had its onset 4 days after MI.

The initial VT event had a rate of 199±28 bpm; the
morphology of VT was usually not available. All patients
were successfully resuscitated from the initial episode of VT.

![Histogram of day of onset of VT in 12 patients who met primary end point of study.](image)

Table 1. Clinical Characteristics of Total Group of Study Patients (n=382)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>65±10</td>
</tr>
<tr>
<td>Male sex, %</td>
<td>66</td>
</tr>
<tr>
<td>Prior MI, %</td>
<td>52</td>
</tr>
<tr>
<td>Angina NYHA class III/IV, %</td>
<td>48</td>
</tr>
<tr>
<td>CHF NYHA class III/IV, %</td>
<td>22</td>
</tr>
<tr>
<td>EF&lt;0.40, %</td>
<td>30</td>
</tr>
</tbody>
</table>
Three patients (25%) died during their hospital stay after CABG. One patient died on the third postoperative day of recurrent and refractory VT, despite administration of multiple intravenous antiarrhythmic agents, including amiodarone. The 2 remaining patients had 4 to 6 episodes of sustained VT. One of these patients died of cardiogenic shock on the fourth postoperative day, and the other died later during the hospitalization of respiratory failure. Of the 370 patients without PMI, history of prior MI.

TABLE 3. Comparison of Clinical Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (n=12)</th>
<th>Group 2 (n=370)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>68±6</td>
<td>65±10</td>
<td>NS</td>
</tr>
<tr>
<td>Male sex</td>
<td>8 (67)</td>
<td>244 (66)</td>
<td>NS</td>
</tr>
<tr>
<td>Smoking</td>
<td>5 (42)</td>
<td>200 (54)</td>
<td>NS</td>
</tr>
<tr>
<td>Diabetes</td>
<td>7 (58)</td>
<td>111 (30)</td>
<td>NS</td>
</tr>
<tr>
<td>Hypertension</td>
<td>9 (75)</td>
<td>207 (56)</td>
<td>NS</td>
</tr>
<tr>
<td>Angina class III/IV</td>
<td>10 (83)</td>
<td>170 (46)</td>
<td>NS</td>
</tr>
<tr>
<td>Prior MI</td>
<td>11 (92)</td>
<td>185 (50)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>CHF class III/IV</td>
<td>7 (58)</td>
<td>78 (21)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>EF&lt;0.40</td>
<td>8 (67)</td>
<td>107 (29)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Preoperative β-blockers</td>
<td>10 (83)</td>
<td>278 (75)</td>
<td>NS</td>
</tr>
<tr>
<td>Preoperative Ca²⁺ channel blockers</td>
<td>8 (67)</td>
<td>255 (69)</td>
<td>NS</td>
</tr>
<tr>
<td>Preoperative QRS, ms</td>
<td>99±25</td>
<td>94±38</td>
<td>NS</td>
</tr>
<tr>
<td>Preoperative QT, ms</td>
<td>374±55</td>
<td>394±51</td>
<td>NS</td>
</tr>
</tbody>
</table>

Values are n (%) or as given.

Among the 9 survivors, 5 patients underwent electrophysiological study before hospital discharge, and all had induction of sustained monomorphic VT that was similar to the clinical VT. Three of the 9 surviving patients received an implantable cardioverter-defibrillator (ICD) before hospital discharge. The other 6 patients received chronic therapy with antiarrhythmic drugs. Four patients were treated with β-blocker therapy. All 9 patients are alive, with a mean follow-up of 2.5 years. Two patients, 1 with an ICD and the other on amiodarone, have experienced recurrent VT during follow-up.

Comparison of Clinical Characteristics Between Group 1 and Group 2 Patients

Group 1 comprises those patients who have experienced ≥1 episode of sustained monomorphic VT in the postoperative period, and group 2 patients are those who have not experienced any episodes of VT. Clinical characteristics of these groups of patients are compared in Table 2. There were no significant differences in demographics, medical history, preoperative medications, or preoperative ECG findings between the 2 groups. However, there were substantial differences in these 2 groups when the presence and severity of preexisting myocardial disease were analyzed. Prior MI was present in 92% of group 1 patients (100% if the perioperative MI is included) versus only 50% of the group 2 patients (P<0.01). Group 1 was more likely than group 2 to have recent MI, 17% versus 9%, respectively, but this difference was not statistically significant. A history of severe CHF, ie, NYHA functional class III/IV, was present in 58% of group 1 patients and only 21% of group 2 patients (P<0.01). A reduced LV ejection fraction (EF), <0.40, was present in 67% of group 1 patients but only 29% of group 2 patients (P<0.01). In group 2, 2 patients had experienced VF in the first hours after surgery; none of group 1 had early VT/VF. There was no relationship of the development of VT with preoperative antiarrhythmic drugs, postoperative β-blockers, or β-blocker withdrawal.

The likelihood of developing an episode of VT when no prior MI was present was only 1%. In the setting of prior MI, that incidence increased to 7%. However, in patients whose prior MI was associated with severe LV dysfunction (EF<0.40) as well as a history of severe CHF, the VT risk increased to 30%. The clinical values of these individual clinical risk factors and their combinations are detailed in Table 3. Of note, the individual factor associated with the greatest odds ratio for the development of VT was prior MI, and the presence of either low EF or a history of CHF gave the highest odds ratio, a 16-fold increase in risk.

Comparison of Angiographic Characteristics Between Group 1 and Group 2 Patients

The numbers of native vessels with a significant stenosis were not different between the 2 groups (see Table 4). The

TABLE 3. Predictive Value of Clinical Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Sensitivity, %</th>
<th>Specificity, %</th>
<th>PPA, %</th>
<th>NPA, %</th>
<th>Odds Ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>↓ EF</td>
<td>70</td>
<td>72</td>
<td>9</td>
<td>98</td>
<td>5.9 (1.5, 23.3)</td>
</tr>
<tr>
<td>PMI</td>
<td>92</td>
<td>50</td>
<td>6</td>
<td>99</td>
<td>11.0 (1.4, 86.2)</td>
</tr>
<tr>
<td>CHF</td>
<td>56</td>
<td>79</td>
<td>8</td>
<td>98</td>
<td>4.8 (1, 18.3)</td>
</tr>
<tr>
<td>↓ EF and PMI</td>
<td>60</td>
<td>79</td>
<td>10</td>
<td>98</td>
<td>5.7 (1.5, 20.0)</td>
</tr>
<tr>
<td>CHF and PMI</td>
<td>56</td>
<td>90</td>
<td>17</td>
<td>98</td>
<td>11.8 (3.0, 46.8)</td>
</tr>
<tr>
<td>↓ EF and CHF</td>
<td>33</td>
<td>94</td>
<td>20</td>
<td>97</td>
<td>8.0 (1.8, 36.0)</td>
</tr>
<tr>
<td>↓ EF, CHF, and PMI</td>
<td>33</td>
<td>97</td>
<td>30</td>
<td>97</td>
<td>13.9 (2.9, 67.5)</td>
</tr>
<tr>
<td>↓ EF or PMI</td>
<td>100</td>
<td>45</td>
<td>6</td>
<td>100</td>
<td>...</td>
</tr>
<tr>
<td>CHF or PMI</td>
<td>92</td>
<td>41</td>
<td>5</td>
<td>99</td>
<td>7.8 (1.0, 61.1)</td>
</tr>
<tr>
<td>↓ EF or CHF</td>
<td>90</td>
<td>63</td>
<td>7</td>
<td>99</td>
<td>15.6 (2.0, 124.8)</td>
</tr>
<tr>
<td>↓ EF, CHF, or PMI</td>
<td>100</td>
<td>37</td>
<td>5</td>
<td>100</td>
<td>...</td>
</tr>
</tbody>
</table>

PPA indicates positive predictive accuracy; NPA, negative predictive accuracy; ↓ EF, EF<0.40; and PMI, history of prior MI.
numbers of vessels with a noncollateralized occlusion were different: in the group 1 patients, a noncollateralized occlusion was present in 1.40 vessels, compared with 0.54 vessels in the group 2 patients ($P<0.01$).

Comparison of Surgical and Bypass Graft Characteristics Between Groups 1 and 2. The numbers of bypass grafts placed, both arterial and venous conduits, were similar between groups (see Table 4). Aortic valve replacement, mitral valve replacement, and LV aneurysmectomy did not differ between groups. The amount of cross-clamp time required for bypass graft placement was also similar between the 2 groups. There were, however, substantial differences when the specifics of bypass graft placement across an occluded native vessel were examined.

The placement of $\geq 1$ graft across an occluded vessel was significantly more common in the group with VT, who averaged 1.50 grafts, compared with the group without VT, who averaged 0.42 graft ($P<0.01$). Specifically, a contrast was seen in the graft placement beyond a totally occluded left anterior descending coronary artery; 67% of the patients in group 1 had a graft placed to an occluded LAD, compared with only 11% of group 2 ($P<0.01$). Similar contrasts were seen for grafts to occluded right coronary arteries and left circumflex arteries, although these differences did not reach statistical significance. If this occluded vessel was also supplying an infarct zone, with a graft placed across the occlusion, marked differences were observed: patients in group 1 had 1.50 grafts to an occluded vessel supplying an infarct zone, whereas patients in group 2 had a 10-fold lower average of 0.17 ($P<0.01$).

The relative risk of developing $\geq 1$ episode of postoperative sustained monomorphic VT based on the presence of the significant coronary graft characteristics is outlined in Table 5. Of note, there was a 4-fold increase in risk of VT if a bypass graft was placed to $\geq 1$ occluded vessel. This risk was even higher, 16-fold, if the graft was placed to an occluded LAD. The risk was greatest, a 22-fold increase, if the graft was placed to $\geq 1$ occluded vessel that supplied an infarct zone. In the absence of a bypass graft to an occluded vessel, only 0.8% of patients developed VT. When $\geq 1$ graft was placed to an occluded vessel, that risk increased to 7% and increased further to 14% when the graft was placed to an occluded vessel in an infarct zone.

**Multivariate Analysis**

Multivariate logistic regression analysis of angiographic and CABG variables, alone or in combination, and significant clinical variables yielded the number of bypass grafts to occluded vessels perfusing an infarct zone as the only independent predictor among the 7 significant univariate predictors (see Tables 2 and 4) of new-onset sustained VT after bypass surgery ($\chi^2=4.6, P=0.03$).

**Discussion**

This is the first prospective study to examine the risk, risk factors, and outcome of new-onset sustained VT after CABG surgery. The following observations were made: (1) Although uncommon in the total population, VT in the recovery phase occurred more frequently than previously reported, with an incidence of 3.1%. (2) Patients who developed VT had a high in-hospital mortality rate of 25%. However, long-term outcome was good, possibly related to antiarrhythmic intervention. (3) A clear-cut clinical substrate existed, principally previous MI scar and associated severe LV dysfunction. (4) A strong independent relationship was found between the development of VT and the placement of a bypass graft across a noncollateralized occluded coronary vessel that was subtending a chronic infarct zone. (5) The development of VT was typically not due to a detectable postoperative complication.
Incidence and Hospital Course of New-Onset VT
In this prospective series of consecutively enrolled patients, a more accurate representation of VT incidence can be determined, for the first time. Previous series, all retrospective or based on referral to a consultation service, may be subject to underestimation and in fact predicted an incidence of 0.7% to 1.4%.1,2 The nature of our study design permitted a more accurate representation of risk: 3.1% of the CABG patients in the present series. It is also possible that the true incidence has increased since the publication of the earlier series, possibly because of a greater proportion of patients with prior MI undergoing surgery in the present era.

The VT events in all patients were unstable and required resuscitation. This is not surprising, given the significant LV dysfunction present in all patients and the rapid rate of the tachycardias.11 Although the acute VT event was not fatal in any patient, a substantial mortality rate was nonetheless found; 25% of patients who experienced VT died during the hospitalization of recurrent ventricular tachyarrhythmias or of complications of cardiac arrest. Recurrent VT was not infrequent, observed in 33%, necessitating aggressive efforts at control with antiarrhythmic therapy. Recurrent arrhythmia (which was always multiple) was associated with a high mortality; 3 of 4 patients with these characteristics did not survive to hospital discharge. Previous series have often also commented on the catastrophic outcome when VT interrupts a stable postoperative recovery.7

Identification of High-Risk Patients by Clinical Profile
There were clear-cut distinguishing features between patients who developed VT and those who did not. The prospective nature of this study, with enrollment of a consecutive series of patients, made risk factor analysis possible. In all but 1 patient, MI was present before surgery, with no evidence of a new ischemic event in the perioperative period. In addition, most VT patients had significant LV dysfunction and clinical evidence of severe CHF. These 3 factors were all present more frequently in the group with VT than the group without VT. The absence of prior MI was very useful to exclude risk of VT, because only 0.8% went on to have a postoperative arrhythmic event. Although statistically significant, the risk of VT in those with a prior MI was still relatively low, ≈7%. However, when all 3 risk factors (prior MI, EF < 0.40, and severe CHF) were present simultaneously, the risk of VT was ominously high, ≈30%. Clearly, this preoperative profile, with its risk of VT, would warrant careful and prolonged observation with continuous ECG telemetry to avoid an unmonitored serious, life-threatening ventricular arrhythmia.

Results of Electrophysiological Study and Long-Term Management
Not all patients were able to undergo electrophysiological study. Some patients required aggressive antiarrhythmic therapy to stabilize and were then treated with empirical amiodarone therapy. However, 5 patients were studied, and all exhibited provokable monomorphic VT that resembled the clinical VT, a finding similar to some previous reports,7,8 although others report a lower frequency of inducible VT.9,10

Along with the clinical data described above, the presence of inducible VT supports the premise that a fixed substrate for VT was present that could represent a long-term risk; thus, all were treated with an implantable defibrillator or amiodarone. When inducible VT is absent, some have suggested that long-term therapy is not required, presumably indicating that a transient high-risk process had resolved.9,10 In the patients in our series who received ICDs, 1 of 3 patients subsequently experienced an appropriate therapeutic intervention by the ICD. We cannot be certain as to the long-term need or benefit of either ICDs or amiodarone in these patients, but the presence of sustained VT, LV dysfunction, and prior MI prompted these therapeutic decisions.

Potential Role of Chronic Coronary Occlusion and Bypass Graft Placement
We performed a detailed analysis of factors defined by the preoperative angiographic study and those defined by the surgical placement of grafts. We hypothesized that resumption of blood flow to areas previously poorly perfused could potentially restore electrophysiological function to cells embedded within the borders of myocardial scar and create the possibility of reentrant circuits. In patients who develop VT in the setting of chronic MI, it has been demonstrated previously that a source of coronary blood flow to myocardial scar (and presumed origin of VT) is usually identified.12 When blood flow is interrupted, VT can be rendered noninducible; conversely, when blood flow resumes, VT can recur.12 These findings point to the importance not only of coronary perfusion to maintain the electrical viability of critical arrhythmogenic tissue but also of a reversible state that may chronically exist: in essence, a state of electrical hibernation.

One particular factor was most strongly associated with the development of VT: placement of a bypass graft across ≥1 noncollateralized totally occluded artery that was supplying an infarct zone. This finding strongly supports the hypothesis stated above. When present, this factor imparted a 22-fold increase in risk; the very high negative predictive accuracy (99%) was supportive of the notion that in the absence of resumption of coronary blood flow to an infarct zone, VT was very unlikely to develop in the in-hospital recovery phase. The potential for the critical role played by reperfusion has been raised in the past7,8,13; in addition, a case-control retrospective study13 demonstrated the association between revascularization of an area of previous MI and the development of sustained VT/VF.

Limitations
The data presented in this article can only be applied to the patients defined by our inclusion and exclusion criteria. Specifically, our conclusions reflect observations on patients who experienced VT > 24 hours after surgery and within a 30-day window, with VT having occurred in hospital. Patients with early VT (< 24 hours) or VT at > 30 days may differ from the cohort analyzed in this paper. VT was detected predominantly by rhythm strips rather than full 12-lead ECGs. We cannot be certain that the uniform nature of VT on the 1 or 2 leads available to us at the time of VT would be present on a multilead ECG recording. The diagnosis of new
or worsening CHF in the postoperative period was made on clinical grounds. Hemodynamic data were not routinely recorded in our database and thus were not included in our analysis of postoperative risk factors. Finally, a postoperative coronary angiogram was not performed in the majority of patients with VT because of the absence of clinical evidence of acute coronary ischemia that would reflect graft occlusions or acute coronary occlusion. Nonetheless, we cannot assume that these ischemic events did not occur without definitive proof from coronary angiographic results.

Conclusions
The first presentation of sustained VT in the recovery period after CABG is relatively uncommon in the general population but is much higher in specific subsets of patients. High-risk subsets of patients could be characterized by clinical, angiographic, and surgical data. In particular, placement of a bypass graft across a noncollateralized occlusion in a vessel supplying an infarct zone was strongly and independently associated with development of VT. Reperfusion via CABG placement may restore electrical function to previously quiescent myocardium and thus create reentrant circuits. Because VT risk and fatal outcome are substantial in this subgroup, this hazard should be considered in planning graft placement and postoperative care.

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References