“Stable” Ventricular Tachycardia Is Not a Benign Rhythm
Insights From the Antiarrhythmics Versus Implantable Defibrillators (AVID) Registry

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Background—Sustained ventricular tachycardia (VT) can be unstable, can be associated with serious symptoms, or can be stable and relatively free of symptoms. Patients with unstable VT are at high risk for sudden death and are best treated with an implantable defibrillator. The prognosis of patients with stable VT is controversial, and it is unknown whether implantable cardioverter-defibrillator therapy is beneficial.

Methods and Results—Screening for the Antiarrhythmics Versus Implantable Defibrillators (AVID) trial identified patients with both stable and unstable VT. Both groups were included in a registry, and their clinical characteristics and discharge treatments were recorded. Mortality data were obtained through the National Death Index. The mortality in 440 patients with stable VT tended to be greater than that observed in 1029 patients presenting with unstable VT (33.6% versus 27.6% at 3 years; relative risk [RR]=1.22; \( P=0.07 \)). After adjustment for baseline and treatment differences, the RR was little changed (RR=1.25, \( P=0.06 \)).

Conclusions—Sustained VT without serious symptoms or hemodynamic compromise is associated with a high mortality rate and may be a marker for a substrate capable of producing a more malignant arrhythmia. Implantable cardioverter-defibrillator therapy may be indicated in patients presenting with stable VT. (Circulation. 2001;103:244-252.)

Key Words: death, sudden tachycardia cardioversion defibrillation

In the Antiarrhythmics Versus Implantable Defibrillators (AVID) trial, patients presenting with ventricular fibrillation (VF) or unstable ventricular tachycardia (VT) were shown to have significantly reduced mortality when treated with an implantable cardioverter-defibrillator (ICD) compared with amiodarone. To be eligible for randomization in the AVID trial, patients with VT were required to have syncope as a result of VT, a left ventricular ejection fraction (EF) \( \leq 0.40 \), and angina or symptoms of significant hemodynamic compromise during VT. Patients with stable VT that did not cause hemodynamic compromise or angina were not eligible for randomization regardless of their EF. The decision not to enroll stable VT patients was made because the risk of arrhythmic death in this group was thought to be too low to contribute to the assessment of aggressive antiarrhythmic therapy. A review of studies that have examined the risk of sudden death in patients with stable VT reveals conflicting results. Some suggest little risk of sudden death. Others suggest a risk similar to that for patients with more severe symptoms during VT. Although patients with stable VT were not enrolled in AVID, they were included in a registry of patients screened for the study. In this article, the mortality of patients with stable VT enrolled in the AVID Registry is compared with that of patients with unstable VT who were either enrolled in the AVID main trial or included in the registry.

Methods

During the process of screening patients for entry into the AVID trial, the 56 clinical centers in the United States and Canada evaluated all patients presenting to their institutions with sustained VT or VF over the 4-year period from June 1, 1993, to April 7, 1997. Details of the creation and management of the AVID Registry, which includes 4595 patients, have been published. The registry includes patients with rhythms eligible for trial entry, as well as patients who presented with arrhythmias not eligible for entry into the trial, including unstable sustained VT and an EF >0.40, stable sustained VT, VT/VF resulting from a transient or reversible cause, and out-of-hospital unexplained syncope in patients with structural heart disease and sustained VT induced on electrophysiological study. Patients were included in the registry regardless of whether they were eligible for or randomized in the AVID main trial. Patients who had an arrhythmia within 5 days of a myocardial infarction, cardiac

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surgery, or coronary intervention were excluded, as were patients with class IV heart failure or those who were on a heart transplant list, had a prior ICD implant or attempted implant, or had a life expectancy of <1 year. At the time of screening, demographic and baseline data were obtained. At hospital discharge, procedures done after the index event, discharge medications, heart rate, and blood pressure were recorded. Mortality data were obtained from the National Death Index.

Stable VT was defined as electrocardiographically documented sustained VT not associated with significant hemodynamic compromise or angina. Of the patients in the total AVID Registry, 12% (n=536) had stable VT, and 25% (1167) had unstable VT (330 of the 1167 were randomized in the main trial). Patients registered at sites that did not participate in long-term follow-up were excluded (7 with stable VT, 40 with unstable VT), as were early registrants for whom initial hospital survival data were not collected (29 with stable VT, 45 with unstable VT). The population was further restricted by elimination of those patients who did not survive the baseline hospitalization (10 with stable VT, 6 with unstable VT) and who had no identifiable heart disease (50 with stable VT, 47 with unstable VT).

Statistical Analysis
Baseline comparisons were evaluated with the use of the χ² or Student’s t test when appropriate. Because data were obtained both at screening and at hospital discharge, baseline (at the time of the index event) clinical variables were given a first level of entry, and therapies and procedures during the index hospitalization (recorded at the time of discharge) were given a second level. Stepwise logistic regression (P=0.05 to enter and P=0.1 to remove) was used to examine the multivariate relationship of covariate differences between patients presenting with stable VT and unstable VT. Continuous factors were not discretized in the models but were discretized for presentation in tables. Model construction followed the pattern of (1) stepwise selection among baseline factors, (2) stepwise selection from second-order interactions of factors selected in step 1, (3) stepwise selection among discharge factors, and (4) stepwise selection among second-order interactions selected in step 3 with factors selected in step 1 and then with factors chosen in step 3. Survival was measured from index event (even though only those patients discharged alive are included because hospital discharge date was not recorded on early forms) until death or until the National Death Index censor date of December 31, 1997. The univariate effect of baseline factors on mortality in the entire population of patients presenting with either stable or unstable VT was estimated by Kaplan-Meier method and tested with the log-rank statistic. Multivariate relationships were evaluated via a stepwise Cox proportional-hazards model with the same model construction as above. An unadjusted comparison of mortality between unstable and stable VT used Kaplan-Meier estimation and the log-rank statistic. An adjusted analysis was made with a Cox proportional-hazards model, adjusting for significant (multivariate) predictors of death during follow-up and permitting adjustment for any significant interactions between predictors and type of VT on outcome. Finally, group discriminators were allowed to enter the model by including the discriminator and its interaction with type of VT.

Results
Baseline Characteristics
The study population consisted of 440 patients with stable VT and 1029 patients with unstable VT. Of the 1029 patients with unstable VT, 330 had therapy determined by randomization in the AVID trial: 52% received an ICD, 47% amiodarone, and 2% sotalol. Therapy for the remaining 699 patients with unstable VT and the 440 patients with stable VT was determined at the discretion of the attending physician. Table 1 compares the baseline demographic and medical history data for the stable and unstable VT groups. There were significant differences between the 2 groups. Patients with stable VT had a higher mean EF, were less likely to have a history of congestive heart failure and smoking, and were more likely to have a history of VT, to be taking an antiarrhythmic drug at the time of their index arrhythmia, and to have their index event occur in the hospital. In addition, there were significant differences in the type of health insurance between the 2 groups. On multivariate analysis, all the above differences remained significant except for the differences in the history of VT before the index event, history of smoking, and health insurance, whereas the excess of patients with a history of neoplasm in patients presenting with unstable VT became significant. Significant multivariate interactions were observed between a history of neoplasm and EF and between prior use of antiarrhythmic medication and index event location.

Therapy
Table 2 compares the discharge therapies received by the 2 groups. Compared with patients presenting with unstable VT, patients with stable VT were less likely to receive an ICD, were more likely to receive antiarrhythmic drug therapy without an ICD, were more likely to receive no specific antiarrhythmic therapy (no antiarrhythmic drug or ICD), and were more likely to have catheter ablation for VT. In addition, patients with stable VT were less likely to receive digitalis, ACE inhibitors, nitrates, diuretics, and warfarin. There was no difference in the rate of pacemaker implantation, revascularization, or β-blocker therapy between the 2 groups. Stable VT patients had lower heart rates and higher diastolic and systolic blood pressures at discharge. On multivariate analysis, unstable VT patients were more likely to undergo ICD and warfarin therapy, were less likely to undergo arrhythmia surgery or ablation, were less likely to have valve surgery, and had lower systolic blood pressure at discharge.

Mortality
Figure 1 displays the unadjusted mortality in patients with stable and unstable VT. The stable VT patients tended to have a higher mortality (33.6% versus 27.6%) at 3 years, with a relative risk (RR) of death of 1.22 (P=0.07). After adjustment for predictors of mortality (Figure 2), the trend toward increased mortality in stable VT persisted (RR=1.25; 95% CI, 0.99 to 1.59; P=0.06). Tables 3 and 4 show the association between baseline and discharge variables and mortality at 3 years when the stable and unstable VT patients are combined. Multivariate predictors of mortality in the combined VT population included older age, antiarrhythmic drug use before the index event, lower EF, history of congestive heart failure, no history of myocardial infarction, absence of nonischemic dilated cardiomyopathy, index event in the hospital, no CABG surgery during the index hospitalization, no ICD implant, digoxin and/or diuretic therapy at discharge, no β-blocker prescribed at discharge, and higher heart rate and/or lower diastolic blood pressure at discharge. There were significant interactions between prior antiarrhythmic drug use and CABG surgery, location of event and digoxin therapy, ICD therapy and digoxin therapy, and ICD therapy and heart rate. There was also a significant interaction...
between β-blocker therapy and type of VT on mortality (P=0.04), so the increased risk of stable versus unstable VT appears to be restricted to patients discharged on β-blocker therapy. Figure 3 shows that the unadjusted mortality was essentially identical for stable VT and unstable VT patients who were not treated with β-blockers and that treatment with β-blockers was associated with a decreased mortality in unstable but not stable VT patients. To further investigate this finding, the clinical variables that were significant multivariate predictors of mortality were compared as a function of whether patients were taking a β-blocker at the time of discharge. Overall, patients taking β-blockers had a higher EF, were less likely to have a history of congestive heart failure, were more likely have an ICD, and were less likely to be discharged on digoxin or diuretic. Despite all these associations that would suggest lower mortality through other mechanisms, β-blocker use was an independent predictor of lower mortality on multivariate analysis. When patients taking β-blockers were examined, those who had unstable VT as their index arrhythmia and appeared to benefit more from β-blocker therapy as a group were more likely to have had CABG surgery or angioplasty after their index arrhythmia, had a lower EF, were more likely to have a history of congestive heart failure, were more likely to have an ICD, and were more likely to be discharged on a diuretic. Correcting for all these clinical variables did not eliminate the finding of

### TABLE 1. Comparison of Patients Presenting With Unstable VT and Stable VT

<table>
<thead>
<tr>
<th></th>
<th>Unstable VT (n=1029)</th>
<th>Stable VT (n=440)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>65.3±11.4</td>
<td>64.9±11.9</td>
<td>0.53</td>
</tr>
<tr>
<td>Male, %</td>
<td>82</td>
<td>81</td>
<td>0.86</td>
</tr>
<tr>
<td>Nonwhite, %</td>
<td>9</td>
<td>8</td>
<td>0.72</td>
</tr>
<tr>
<td>Antiarrhythmic drug at index arrhythmia, %</td>
<td>19</td>
<td>28</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Index arrhythmia in hospital, %</td>
<td>24</td>
<td>30</td>
<td>0.02*</td>
</tr>
<tr>
<td>Health Insurance, %</td>
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<td></td>
<td>0.02</td>
</tr>
<tr>
<td>None</td>
<td>3</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>VA/military</td>
<td>6</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Private</td>
<td>20</td>
<td>28</td>
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</tr>
<tr>
<td>Medicare/Medicaid</td>
<td>58</td>
<td>55</td>
<td></td>
</tr>
<tr>
<td>HMO</td>
<td>13</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Clinical history, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prior VF</td>
<td>3</td>
<td>4</td>
<td>0.82</td>
</tr>
<tr>
<td>Prior VT</td>
<td>26</td>
<td>33</td>
<td>0.013</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>73</td>
<td>70</td>
<td>0.31</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>45</td>
<td>34</td>
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</tr>
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<td>Atrial fibrillation</td>
<td>23</td>
<td>21</td>
<td>0.34</td>
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<tr>
<td>Bradycardia/heart block</td>
<td>7</td>
<td>7</td>
<td>0.88</td>
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<tr>
<td>Hypertension</td>
<td>47</td>
<td>47</td>
<td>0.85</td>
</tr>
<tr>
<td>Diabetes</td>
<td>20</td>
<td>21</td>
<td>0.77</td>
</tr>
<tr>
<td>Syncope</td>
<td>10</td>
<td>8</td>
<td>0.13</td>
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<tr>
<td>Smoking</td>
<td>21</td>
<td>16</td>
<td>0.04</td>
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<tr>
<td>Neoplasm</td>
<td>9</td>
<td>7</td>
<td>0.12*</td>
</tr>
<tr>
<td>Organic heart disease</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coronary artery disease, %</td>
<td>86</td>
<td>83</td>
<td>0.26</td>
</tr>
<tr>
<td>Hypertrophic cardiomyopathy, %</td>
<td>4</td>
<td>5</td>
<td>0.21</td>
</tr>
<tr>
<td>Dilated cardiomyopathy, %</td>
<td>13</td>
<td>10</td>
<td>0.12</td>
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<tr>
<td>EF</td>
<td>0.31±0.11</td>
<td>0.34±0.13</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Procedures before the index arrhythmia, %</td>
<td>50</td>
<td>50</td>
<td>0.97</td>
</tr>
<tr>
<td>None</td>
<td>39</td>
<td>41</td>
<td>0.50</td>
</tr>
<tr>
<td>Revascularization</td>
<td>2</td>
<td>2</td>
<td>0.82</td>
</tr>
<tr>
<td>Aneurysm surgery</td>
<td>5</td>
<td>4</td>
<td>0.28</td>
</tr>
<tr>
<td>Valve surgery</td>
<td>3</td>
<td>3</td>
<td>0.72</td>
</tr>
<tr>
<td>Thrombolysis</td>
<td>6</td>
<td>6</td>
<td>0.59</td>
</tr>
<tr>
<td>Other</td>
<td>8</td>
<td>6</td>
<td>0.14</td>
</tr>
</tbody>
</table>

*Significant variable on multivariate logistic analysis.
improved survival, with β-blocker use being restricted to patients who presented with unstable VT. In contrast to the result with β-blockers, Figure 4 shows that the improved survival associated with a higher EF occurred in patients who presented with stable and unstable VT.

**Discussion**

This retrospective, nonrandomized, observational analysis suggests that the absence of severe symptoms with sustained VT does not predict a benign prognosis. In contrast, we observed a trend toward increased mortality in patients with underlying heart disease who presented with stable compared with unstable VT. This trend in mortality persisted after adjustment for baseline differences between the 2 populations and the predictors of mortality in the combined population.

It is difficult to accept the proposition that patients with stable VT who present with less severe symptoms and in our study had less severe underlying heart disease have a worse prognosis than patients who present with unstable VT. In fact, the trend toward higher mortality in patients presenting with stable VT did not reach statistical significance. However, these results suggest that stable VT is not a low-risk arrhythmia and that sudden death may be a prominent cause of mortality in this population. It appears that there is a rela-
tively high risk of death associated with having VT (independent of symptoms during VT) that overshadows differences in mortality attributable to factors that determine the severity of symptoms during VT.

In support of the concept that presenting symptoms may not be an important predictor of prognosis is the work of Olson et al. They analyzed the predictors of sudden death in 122 patients at their institution who were treated with amiodarone for sustained VT. Over a median of 19.5 months of follow-up, sudden death mortality was virtually identical in patients with tolerated compared with nontolerated VT (25% versus 24%). The single best predictor of mortality was EF. Our data show that independent of presenting symptoms, measures of left ventricular dysfunction, age, type of underlying heart disease, and other factors correlate with increased mortality. Many of these parameters have been identified as risk factors for death in patient populations accepted to be at high risk for sudden death. Antiarrhythmic drug therapy at the time of presentation was seen more commonly in patients with stable VT. Such a characteristic identifies a patient as resistant to drug therapy and was an independent risk factor for death in our population. It is possible that antiarrhythmic use at the time of presentation may have slowed the VT and caused some patients who would have presented as unstable VT to present as stable VT. This finding and the observed interaction between antiarrhythmic drug use and the index event occurring in the hospital might also have been due to an excess of proarrhythmia in the stable VT population. However, correcting for antiarrhythmic use at the time of the index arrhythmia and for the interaction with the location of the index event did not eliminate the trend toward increased mortality in the patients presenting with stable VT. It is interesting to note that the trend toward increased risk in stable VT was for the most part restricted to patients taking β-blockers. Our results cannot establish a causal relationship between β-blocker therapy and survival or suggest a possible mechanism.

**Figure 1.** Unadjusted mortality for patients with unstable and stable VT.

**Figure 2.** Mortality for patients with unstable VT and stable VT adjusted for significant multivariate differences in baseline characteristics and multivariate predictors of mortality in combined population of stable and unstable VT patients.
If patients with stable VT are in fact at high risk for sudden arrhythmic death, then presumably it is not a recurrence of the presenting stable VT that leads to sudden death but a more malignant arrhythmia. A slow or well-tolerated VT may be a marker for an increased risk of a faster, poorly tolerated

| Table 3. Associations Between Baseline Variables and Risk of Death |
|-------------------|-----------------|-----------------|-----------------|-----------------|
|                   | n               | 3-Year Mortality, (%) | P               |
| **Age, y**        |                 |                 |                 |
| ≥65               | 863            | 36±2             | <0.001†         |
| <65               | 605            | 21±2             |                 |
| **Female**        | 273            | 31±3             | 0.21            |
| Male              | 1196           | 29±2             |                 |
| **Nonwhite**      | 126            | 34±5             | 0.16            |
| **White**         | 1343           | 29±2             |                 |
| **AAD therapy at index event** | | | | |
| No                | 1144           | 27±2             | 0.001*          |
| Yes               | 324            | 38±3             |                 |
| **Index arrhythmia in hospital** | | | | |
| No                | 1084           | 27±2             | <0.001*         |
| Yes               | 385            | 35±3             |                 |
| **Private health insurance** | | | | |
| Yes               | 1138           | 33±2             | <0.001          |
| No                | 331            | 18±3             |                 |
| **Clinical history before index event** | | | | |
| **VF**            | | | | |
| No                | 1418           | 29±1             | 0.35            |
| Yes               | 51             | 36±7             |                 |
| **VT**            | | | | |
| No                | 1057           | 28±2             | 0.47            |
| Yes               | 412            | 33±3             |                 |
| **Myocardial infarction** | | | | |
| No                | 414            | 33±3             | 0.08*           |
| Yes               | 1055           | 28±2             |                 |
| **Congestive heart failure** | | | | |
| No                | 861            | 21±2             | <0.001*         |
| Yes               | 608            | 41±2             |                 |
| **Atrial fibrillation** | | | | |
| No                | 1135           | 27±2             | <0.001          |
| Yes               | 334            | 37±3             |                 |
| **Severe bradycardia or heart block** | | | | |
| No                | 1371           | 28±1             | <0.001          |
| Yes               | 98             | 46±6             |                 |
| **Hypertension**  | | | | |
| No                | 779            | 29±2             | 0.27            |
| Yes               | 690            | 31±2             |                 |
| **Diabetes**      | | | | |
| No                | 1172           | 28±2             | 0.03            |
| Yes               | 297            | 35±3             |                 |
| **Syncope**       | | | | |
| No                | 1333           | 29±2             | 0.17            |
| Yes               | 136            | 32±5             |                 |
| **Smoking**       | | | | |
| No                | 1183           | 30±2             | 0.60            |
| Yes               | 286            | 28±3             |                 |

AAD indicates antiarrhythmic drug.

*Significant predictors of mortality on multivariate analysis.
†Entered into the multivariate Cox regression as a continuous variable.

If patients with stable VT are in fact at high risk for sudden arrhythmic death, then presumably it is not a recurrence of the presenting stable VT that leads to sudden death but a more malignant arrhythmia. A slow or well-tolerated VT may be a marker for an increased risk of a faster, poorly tolerated
TABLE 4. Associations Between Discharge Therapy and Risk of Death

<table>
<thead>
<tr>
<th>Interventions during index hospitalization</th>
<th>n</th>
<th>3-Year Mortality (±SEM), %</th>
<th>P</th>
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<tr>
<td>ICD implant (±AAD)</td>
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<tr>
<td>No</td>
<td>823</td>
<td>34±2</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Yes</td>
<td>646</td>
<td>23±2</td>
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<tr>
<td>AAD only</td>
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<tr>
<td>No</td>
<td>792</td>
<td>23±2</td>
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<tr>
<td>Yes</td>
<td>676</td>
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<tr>
<td>No therapy</td>
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<tr>
<td>No</td>
<td>1322</td>
<td>30±2</td>
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</tr>
<tr>
<td>Yes</td>
<td>146</td>
<td>22±4</td>
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<td>No procedures</td>
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<tr>
<td>No</td>
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<td>Yes</td>
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<tr>
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<tr>
<td>No</td>
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<td>29±1</td>
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<td>Yes</td>
<td>7</td>
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<td>No</td>
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<td>Yes</td>
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<td>0.02</td>
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<td>53</td>
<td>41±9</td>
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<td>β-Blocker</td>
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<td>1081</td>
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<td>&lt;0.001*</td>
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TABLE 4. Continued

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<th>Interventions during index hospitalization</th>
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<th>3-Year Mortality (±SEM), %</th>
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<td>Digitalis</td>
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<td>Discharge vital signs</td>
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<td>Heart rate, bpm</td>
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<tr>
<td>≤72</td>
<td>852</td>
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<td>&gt;72</td>
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<td>Systolic blood pressure, mm Hg</td>
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<td>Diastolic blood pressure, mm Hg</td>
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<tr>
<td>&gt;70</td>
<td>511</td>
<td>23±2</td>
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AAD indicates antiarhythmic drug.

*Significant predictors of mortality on multivariate analysis.
†Entered into the multivariate Cox regression as a continuous variable.

arrhythmia. Multiple VTs (including very rapid, poorly tolerated VTs) are commonly induced during electrophysiological testing in patients with stable VT.9 Bocker et al4 examined ICD therapies in 50 patients with an ICD implanted for stable VT who were followed up for 17±12 months. Of these patients, 22% received ICD therapy for arrhythmias that were judged to be life threatening (heart rate >250 bpm). Thus, the symptoms present on presentation with VT may correlate poorly with the subsequent risk of arrhythmic death. Stable VT may simply be a marker for a substrate capable of producing a more malignant VT or spontaneous VF.

In opposition to the suggestion that sudden death is common in patients presenting with stable VT are the
observations of Sarter et al, who retrospectively analyzed the course of 124 patients with coronary artery disease followed up for 36 months after presenting with stable VT. They found a high total mortality (36%) but a relatively low incidence of sudden death (2.4%/y). There are reasons to believe that the results of Sarter et al do not accurately reflect the natural history of patients with stable VT. First, there are numerous difficulties in determining the true cause of death in retrospective studies. Second, 37% of the patients in the study were treated by endocardial resection with a 20% operative mortality. This aggressive intervention may have improved the outcome of those patients who survived surgery and given an inaccurate estimate of the risk of sudden death.

Study Limitations

Important limitations of this study include (1) the inherent difficulties in correcting for the clinical differences between the patients with stable and unstable VT at baseline, (2) the fact that therapy was not randomly assigned within or between the 2 groups, and (3) a lack of information on additional therapy after discharge from the hospital and on the causes of the observed mortality. Because therapy was not randomly assigned and because we do not know how patient therapy may have changed after discharge, we cannot make any assessment of the impact different therapies may have had on mortality. The trend toward increased mortality in patients presenting with stable VT may be due to important unrecorded differences in therapy after the index hospitalization. It is possible that patients with unstable VT were followed up more closely or were more likely to receive other beneficial interventions, such as a late ICD implant after the initial hospitalization. Such differences, for which we are unable to correct, would not be surprising given the widely held belief that patients presenting with unstable VT have a worse prognosis than those who present with stable VT.

Conclusions

This retrospective, nonrandomized, observational analysis suggests that patients presenting with stable, hemodynamically well-tolerated VT and underlying heart disease have at least as high a total mortality rate as patients presenting with VT and more severe symptoms. Stable VT may be a marker for a cardiac electrophysiological substrate capable of pro-
ducing arrhythmias that are more malignant. ICD therapy has been shown to decrease mortality in patients with unstable VT, and given these results, ICD therapy may decrease mortality in patients presenting with stable VT. Studies of ICD therapy in patients with stable VT are warranted.

Acknowledgment
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for the AVID Investigators

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