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Life-Threatening Ventricular Arrhythmias Due to Transient or Correctable Causes: High Risk for Death in Follow-Up

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OBJECTIVES This study evaluated the prognosis of patients resuscitated from ventricular tachycardia (VT) or ventricular fibrillation (VF) with a transient or correctable cause suspected as the cause of the VT/VF.

BACKGROUND Patients resuscitated from VT/VF in whom a transient or correctable cause has been identified are thought to be at low risk for recurrence and often receive no primary treatment for their arrhythmias.

METHODS In the Antiarrhythmics Versus Implantable Defibrillators (AVID) trial, patients with a potentially transient or correctable cause of VT/VF were not eligible for randomization. The mortality of these patients was compared with the mortality of patients with a known high risk of recurrence of VT/VF in the AVID registry.

RESULTS Compared with patients having high risk VT/VF, those with a transient or correctable cause for their presenting VT/VF were younger and had a higher left ventricular ejection fraction. These patients were more often treated with revascularization as the primary therapy, more commonly received a beta-blocker, less often required therapy for congestive heart failure and less commonly received either an antiarrhythmic drug or an implantable cardioverter defibrillator. Nevertheless, subsequent mortality of patients with a transient or correctable cause of VT/VF was no different or perhaps even worse than that of the primary VT/VF population.

CONCLUSIONS Patients identified with a transient or correctable cause for their VT/VF remain at high risk for death. Further research is needed to define truly reversible causes of VT/VF. Meanwhile, these patients may require more aggressive evaluation, treatment and follow-up than is currently practiced. (J Am Coll Cardiol 2001;38:1718–24) © 2001 by the American College of Cardiology

The Antiarrhythmics Versus Implantable Defibrillators (AVID) trial was a prospective, randomized comparison of antiarrhythmic drugs versus implantable cardioverter defibrillators in patients with life-threatening sustained ventricular arrhythmias (1–3). Patients qualified for randomization in the AVID trial if they had serious ventricular arrhythmias not due to a transient or correctable cause. These arrhythmias included: 1) ventricular fibrillation (VF); 2) sustained ventricular tachycardia (VT) with syncope; or 3) sustained VT causing angina, near-syncope, hypotension or congestive heart failure (CHF) with a left ventricular ejection fraction ≤0.40.

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Patients with sustained VT or VF who do not have an identifiable transient or potentially correctable cause are at high risk for recurrence of serious ventricular arrhythmias and subsequent death (4–6). Conversely, it is believed that sustained VT or VF due to an identifiable transient or correctable cause has a low subsequent risk of death, as long as the transient cause is corrected. This bit of conventional wisdom has not been critically evaluated. A registry (3) was maintained of all patients screened for the AVID trial. The AVID registry included a category of patients whose presenting ventricular arrhythmias were considered to be due to a transient or potentially correctable cause.

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Abbreviations and Acronyms

AVID = Antiarrhythmics Versus Implantable Defibrillators trial
CAD = coronary artery disease
CHF = congestive heart failure
MI = myocardial infarction
NDIS = National Death Index Service
VF = ventricular fibrillation
VT = ventricular tachycardia

The purpose of this analysis was to examine mortality for patients in the AVID registry with sustained VT or VF due to a putative transient or correctable cause in comparison with mortality for patients with VT/VF known to be at high risk. Our hypothesis was that patients with a transient or correctable cause identified for their VT/VF would have a lower risk of death.

METHODS

Both the AVID trial (1) and the AVID registry (3) have been described previously. Briefly, patients at 56 clinical sites were screened for inclusion in the randomized portion of the AVID trial. All patients had sustained ventricular arrhythmias, although some patients were not eligible for randomization. Patients who were screened for randomization in AVID and who gave informed consent were entered into a registry if they met certain predetermined criteria, and mortality of all such patients was determined whether or not they were randomized. The date of death of patients was determined by routine follow-up in the main randomized portion of the AVID trial or by the National Death Index Service (NDIS) (a nationwide system that records all deaths) in the nonrandomized portion of AVID. Entries into the NDIS are usually accomplished no later than one year after the actual death date, and data are updated near the end of every calendar year. The NDIS database used here was obtained at the end of 1998, which would include all deaths that had occurred through the end of calendar year 1997.

The categories of ventricular arrhythmias compared in this study are outlined in Table 1. The 145 patients with documented sustained VT with an ejection fraction >0.40 (fourth group in Table 1) were not eligible for randomization in AVID but are included in this comparison because there was no ejection fraction limitation imposed on the patients with presumed transient causes for their arrhythmias. Furthermore, a previous AVID registry study showed that these patients, indeed, have a high risk for death (6). Patients whose VT/VF was associated with a transient or correctable cause were further classified by the nature of the reversible factor: new Q-wave myocardial infarction (MI), new non–Q-wave MI, other ischemic event, proarrhythmic drug reaction, electrolyte imbalance (hypokalemia or hypomagnesemia) or other causes. The determination that VT/VF had a transient or correctable cause was made by the AVID principal investigator at each site based on directions provided in the manual of operations. These patients were treated under the direction of their own physicians, and the initial treatment strategy was recorded in the registry. The AVID studies and their consent forms received approval from the institutional review board at each site.

Table 1. Categories of Ventricular Tachyarrhythmias Compared in This Study

Primary out-of-hospital cardiac arrest due to VF*† (n = 992)
Documented out-of-hospital sustained VT with syncope*† (n = 364)
Documented out-of-hospital sustained VT*† with left ventricular ejection fraction ≤0.40 and:
Systolic blood pressure <80 mm Hg or
Near-syncope or
Chest pain or
Congestive heart failure
(n = 512)
Documented out-of-hospital sustained VT* with left ventricular ejection fraction >0.40 and:
Systolic blood pressure <80 mm Hg or
Near-syncope or
Chest pain or
Congestive heart failure
(n = 145)
Out-of-hospital documented sustained VT or cardiac arrest due to VF associated with an identified transient or correctable cause (n = 278)

*No transient or correctable cause identified; †eligible for randomization in the main AVID trial.
AVID = Antiarrhythmics Versus Implantable Defibrillators; VF = ventricular fibrillation; VT = ventricular tachycardia.

Continuous variables are presented as mean ± one SD. Categorical variables are presented as percentages. Comparative analyses included two-tailed *t* test for continuous variables and chi-square test for discrete variables. Given multiple comparisons, we considered $p < 0.01$ significant in these comparisons. Survival estimates for the populations were based on the methods of Kaplan and Meier. Comparisons among groups were made by the global log-rank test. Survival was adjusted using a Cox proportional hazard model, stratifying on potential confounders (variables that distinguished groups and predicted mortality) and including other factors that predicted mortality as covariates. In the last two analyses, $p < 0.05$ was considered statistically significant.

RESULTS

A total of 5,989 patients were screened, and 4,450 patients were entered in the registry. For appropriate comparisons, only the patients whose arrhythmias occurred out-of-hospital and for whom an NDIS search was possible were included in this study (first four arrhythmia groups in Table 1, n = 2,013). A group of 278 patients was identified whose VT/VF was thought to be due to a transient or correctable cause. These last patients form the main focus of this report.

Table 2 classifies the presumed transient or correctable causes for the presenting VT/VF. Most patients had some form of MI or ischemia to explain the VT/VF. Proarrhyth-

Table 2. Putative Transient or Correctable Causes of VT/VF (n = 278)

	n	%
Ischemic events	183	65.8%
New MI	161	57.9%
Non–Q-wave	83	29.9%
Q-wave	78	28.0%
Transient ischemia, no MI	22	7.9%
Other or unknown*	50	17.9%
Electrolyte imbalance	27	9.7%
Antiarrhythmic drug reaction	18	6.5%

*For example, cocaine or illicit drug use, sepsis, hypoxia, electrocution, drowning.
MI = myocardial infarction; VF = ventricular fibrillation; VT = ventricular tachycardia.

mic drug reaction and electrolyte imbalance was less common. The remainder of causes for VT/VF was variable.

Table 3 lists the baseline clinical characteristics of the patients. The 278 patients with a presumed transient cause for their arrhythmias are compared with the 2,013 patients having out-of-hospital events deemed to be at high risk for arrhythmia recurrence (primary VT/VF). Patients with primary VT/VF were older, had a lower left ventricular ejection fraction and were more likely to have had a prior history of VT. They were also more likely to have had a prior MI, CHF and revascularization procedures. Patients whose VT/VF was deemed to have a reversible cause more frequently had a history of coronary artery disease (CAD).

Figure 1 presents survival from the index event of patients with primary VT/VF compared with patients who had a transient or correctable cause identified. The upper panel shows the unadjusted survivals. The lower panel shows survivals after adjustment for five of the most important covariables known to affect survival (age, ejection fraction, CAD, coronary artery bypass grafting and use of aspirin). No significant difference existed between the unadjusted outcomes. However, outcome was actually worse ($p = 0.008$) in the group with VT/VF due to transient or correctable causes after adjustment. A similar analysis using 22 covariables was nearly identical. Analysis of survival by type of transient or correctable cause yielded no group that had a significantly better survival (Fig. 2). However, the number of subjects in each subgroup is small, and there are multiple baseline and treatment differences between subgroups. Ventricular tachycardia or VF associated with non–Q-wave MI or antiarrhythmia drug reaction seemed most likely to presage better survival.

Table 3. Comparison of Baseline Clinical Characteristics of Patients With Primary VT/VF Versus VT/VF Due to Transient or Correctable Causes

	Primary VT/VF	VT/VF Due to Transient or Correctable Cause	p Value
n	2,013	278	
Age (yrs)	63.4 ± 12.3	61.0 ± 12.7	0.004
LVEF	0.35 ± 0.15	0.41 ± 0.15	< 0.001
Men	76.6%	72.3%	0.132
CAD	74.9%	82.0%	0.004
Cardiomyopathy	3.1%	2.9%	0.851
Prior history			
VF	4.3%	2.9%	0.206
VT	15.0%	9.7%	0.007
Atrial fibrillation	22.3%	18.7%	0.148
MI	57.5%	44.2%	< 0.001
CHF	38.4%	21.6%	< 0.001
Diabetes*	17.8%	15.8%	0.406
CABG/PTCA	26.2%	18.7%	0.003
AAD at index event	13.1%	13.7%	0.783

*Treated with insulin or oral hypoglycemics.
AAD = antiarrhythmic drug; CABG = coronary artery bypass graft surgery; CAD = coronary artery disease; CHF = congestive heart failure; EF = left ventricular ejection fraction; MI = myocardial infarction; PTCA = percutaneous transluminal coronary angioplasty; VF = ventricular fibrillation; VT = ventricular tachycardia.

Table 4 lists the procedures and discharge therapies of patients. Patients with primary VT/VF were less likely to undergo revascularization surgery. They were more likely to receive several drug therapies including digitalis, diuretics and an angiotensin-converting enzyme inhibitor, and they were less likely to receive a beta-blocker or aspirin. Patients with VT/VF due to a transient or correctable cause were less likely to receive specific antiarrhythmic therapy. Patients with an ischemic transient or correctable cause for VT/VF were more likely to be treated with revascularization, beta-blockers and aspirin. They were less likely to receive a specific antiarrhythmic therapy and therapy for heart failure.

DISCUSSION

VT/VF risk stratification. Central to the treatment of patients who have life-threatening ventricular arrhythmias is the concept of risk stratification (5,7). Patients at high risk for recurrence need to be aggressively treated. Patients at low risk do not need specific treatment for ventricular arrhythmias. Usually included in assessment of the risk of recurrence is an attempt to identify a transient or correctable cause for the VT/VF. Until now, finding such a reversible cause has been thought to suggest a low risk of recurrence when the cause can be eliminated.

Ischemia and VT/VF reversibility. The most common setting for life-threatening ventricular arrhythmias is myocardial ischemia or scarring (4,5,8–12). The patient with CAD without myocardial scarring who has a cardiac arrest at the onset of ischemia (often induced by exercise) is thought to be at low risk for recurrent arrhythmias when the ischemia is successfully treated, commonly by some type of revascularization procedure. This belief is based on a rather small observational series (13). Many arrhythmia specialists would assess such patients and direct the therapy with programmed electrical stimulation studies after revascularization (14).

MI and VT/VF reversibility. On the other hand, the patient with a myocardial scar from a previous MI who has sustained VT might be at higher risk for recurrent arrhythmias because at least a part of the underlying cause (the myocardial scar) cannot be completely eliminated (15). In the context of this analysis, it should be acknowledged that

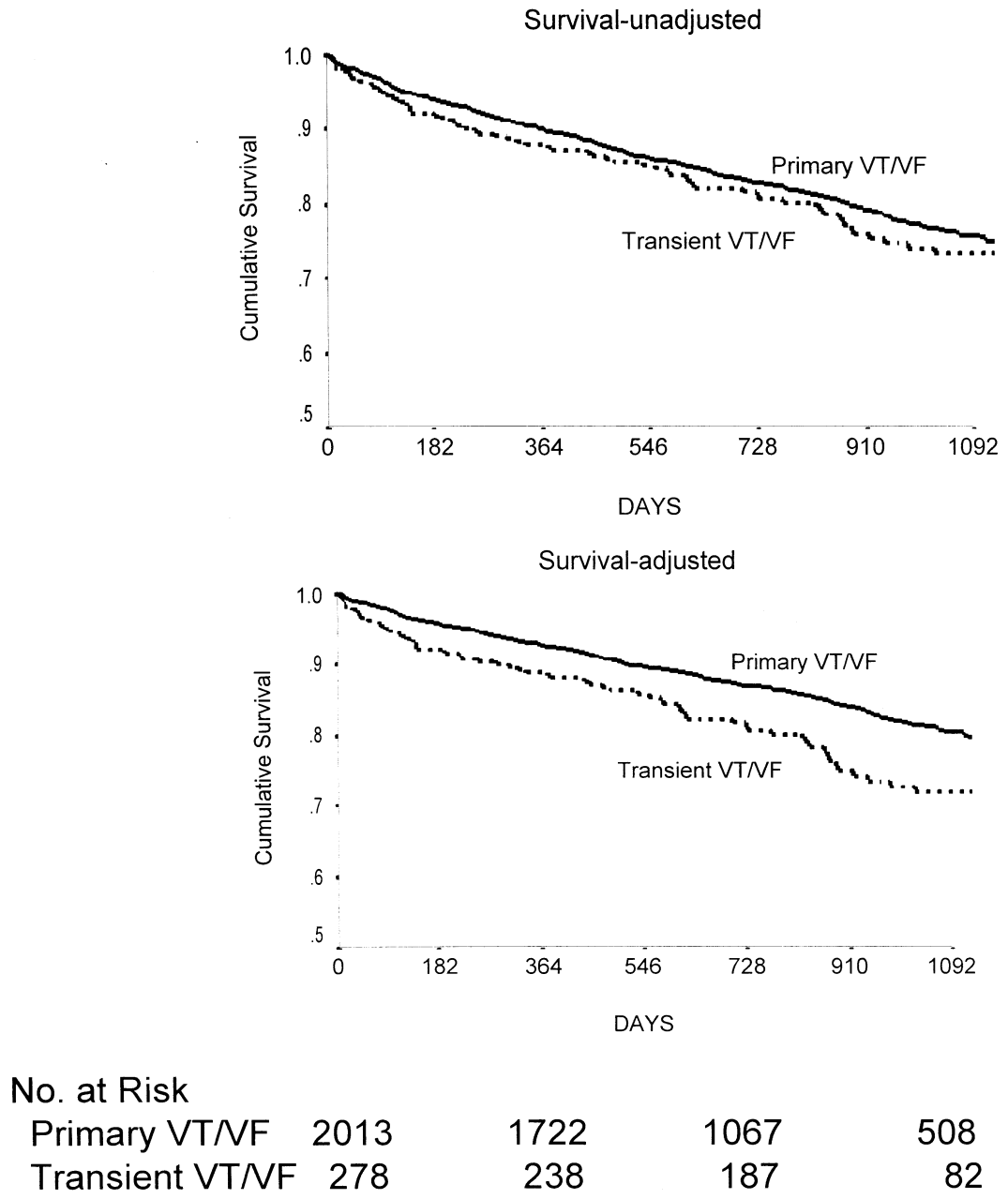

Figure 1. Survival curves comparing patients with high-risk ventricular tachycardia/ventricular fibrillation (Primary VT/VF) versus a transient/correctable cause for the VT/VF (Transient VT/VF). **Upper panel** shows unadjusted data (p = NS), and the **lower panel** depicts results after adjustment for five variables known to affect mortality (p = 0.008, see text).

it is not always possible to know with certainty that a new MI led to VT/VF or vice versa. For example, transthoracic cardioversion in itself can result in transient ST-segment elevation resembling that seen in acute MI (16).

Prognosis of VT/VF in the setting of MI. Previous studies have suggested that the development of VF within 48 h of the onset of a new Q-wave MI (especially out-of-hospital) is likely to identify a patient with a low risk of recurrence after hospital discharge (4,5,8,10). Presumably, the acute MI caused the VF cardiac arrest. If the region of myocardium responsible for VF undergoes death and scar formation, recurrence is unlikely. Ventricular tachycardia or VF developing during the hospital admission, on the other hand, is associated with increased mortality during the initial hospitalization (17,18). This analysis does not distinguish between in-hospital and post-discharge mortality in the patients with VT/VF thought to be due to a new MI.

Other causes and VT/VF reversibility. Other causes of transient events are less common and highly variable. The proarrhythmic effects of antiarrhythmic drugs are well known (19,20). Occasionally patients being treated for relatively minor arrhythmias can develop life-threatening

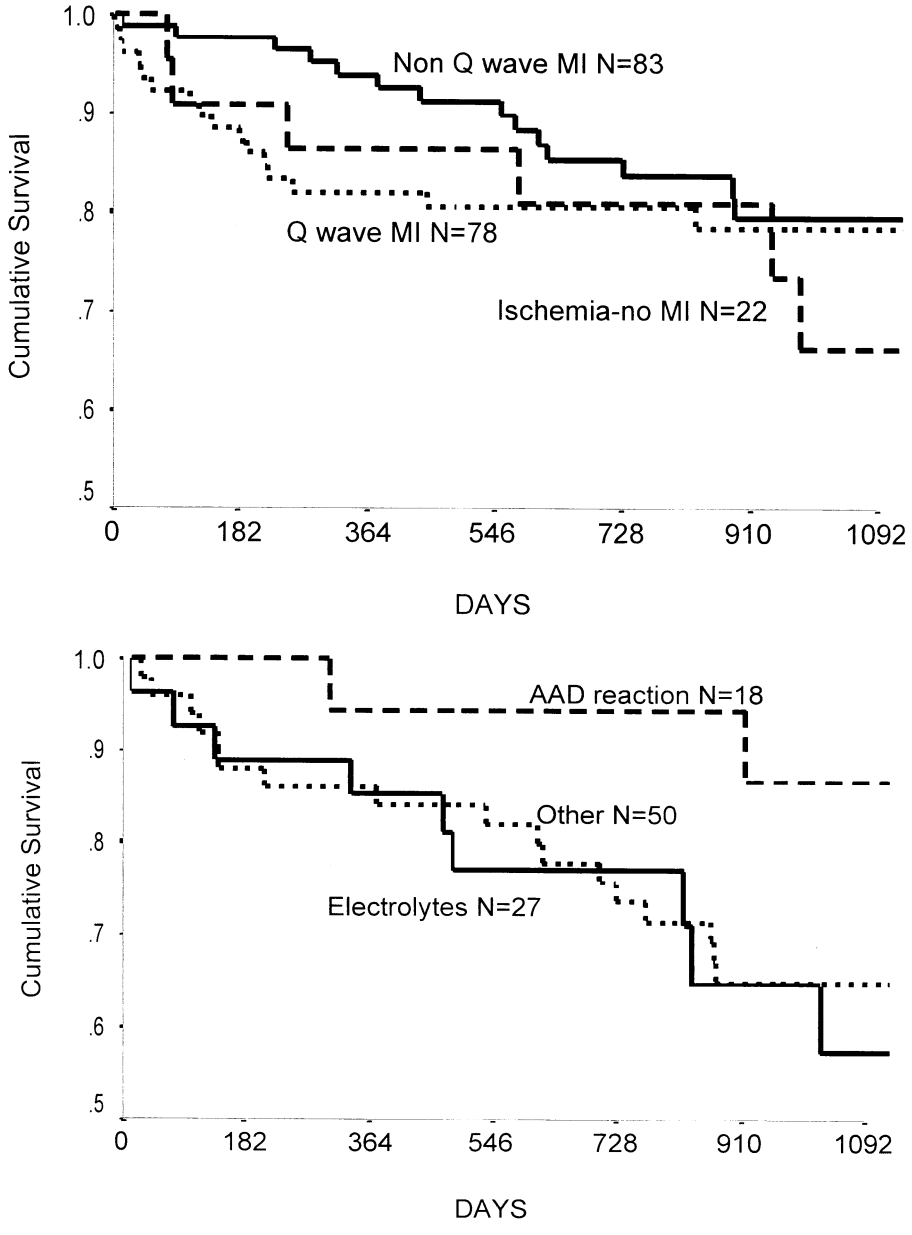

Figure 2. Survival curves for six subgroups of patients with a transient/correctable cause for their presenting ventricular tachycardia/fibrillation. The subgroups are those listed in Table 2: non–Q-wave myocardial infarction (MI) (n = 83), Q-wave MI (n = 78) and ischemia-no MI (n = 22) in the **upper panel**. (**Lower panel**) Electrolyte imbalance (n = 27), antiarrhythmic drug (AAD) reaction (n = 18) and other or unknown (n = 50). P = NS.

arrhythmias because of the antiarrhythmic drug itself. Perhaps these patients might already have underlying abnormalities of sodium or potassium conductance that is unmasked or exacerbated by the antiarrhythmic drug. In these patients, it has been thought that simple elimination of the antiarrhythmic drug will remove the propensity to life-threatening ventricular arrhythmias, though VT can usually be induced at electrophysiologic study in most such patients with structural heart disease (21), even after the drug is stopped.

Patients with cardiac disease who are treated with diuretics occasionally develop serious electrolyte abnormalities that can precipitate ventricular arrhythmias (22,23). However, it is less well known whether correction of the electrolyte abnormalities sufficiently removes the risk for arrhythmia recurrence to forego other treatment. In this study the patients with electrolyte abnormalities had the worst prognosis, perhaps because many of them had severe CHF requiring continued aggressive diuretic therapy, in turn leading to more arrhythmias. Furthermore, it may be difficult to determine whether hypokalemia is the cause or result of VT/VF (24).

Uncertainty in determination of VT/VF reversibility. Our study suggests that identification of low risk patients is indeed difficult. In fact, many of the patients with a transient or correctable cause were given long-term treat-

Table 4. Comparison of Post-Event Procedures and Discharge Therapies

		VT/VF Due to Transient or Correctable Cause				
	Primary VT/VF	All Patients	Ischemic Patients	Other Patients	Primary vs. All p Value	Ischemic vs. Other p Value
CABG/PTCA	13.7%	42.8%	55.7%	13.7%	< 0.001	< 0.001
Beta-blocker	28.7%	45.1%	52.2%	28.7%	< 0.001	< 0.001
ACE inhibitor	58.8%	40.9%	42.5%	58.8%	< 0.001	0.460
ASA	56.7%	66.2%	72.1%	56.7%	< 0.001	0.005
Calcium channel blocker	14.3%	19.2%	18.8%	14.3%	0.049	0.810
Digitalis	38.8%	28.3%	29.3%	38.8%	< 0.001	0.610
Diuretics	44.3%	33.2%	33.0%	44.3%	< 0.001	0.910
Warfarin	23.1%	19.9%	21.4%	23.1%	0.212	0.350
AAD, no ICD	38.6%	26.0%	20.9%	38.6%	< 0.001	0.011
Amiodarone at discharge	38.4%	19.4%	15.3%	38.4%	< 0.001	0.025
ICD, no AAD	42.3%	16.9%	20.8%	42.3%	< 0.001	0.009
AAD and ICD	9.7%	3.6%	2.7%	9.7%	< 0.001	0.333
No AAD or ICD	9.5%	53.6%	55.7%	9.5%	< 0.001	0.320

AAD = antiarrhythmic drug; ACE = angiotensin-converting enzyme; ASA = acetylsalicylic acid (aspirin); CABG = coronary artery bypass graft surgery; ICD = implantable cardioverter defibrillator; PTCA = percutaneous transluminal coronary angiography; VF = ventricular fibrillation; VT = ventricular tachycardia.

ment, suggesting that the physician did not actually believe that the patient was at low risk. Patients thought to be at relatively low risk because their VT/VF was precipitated by presumed transient or correctable causes experienced mortality similar to, or worse than, that of patients with primary VT/VF. Recurrent arrhythmias and subsequent arrhythmic death in these patients are probably more common than previously recognized. This paradox could be the result of misclassification of a transient or correctable cause, failure to fully correct the reversible cause or failure to identify other underlying causes of arrhythmias that might not be reversed by treating the obvious transient or correctable cause. Furthermore, reversible causes may have been transiently corrected but subsequently recurred. Such patients may have severe structural heart disease that, in itself, accounts for the bad prognosis. Thus, it is possible that there is indeed a high intrinsic risk in these patients, even though the transient cause is eliminated.

Clinical implications. These findings should cause the physician to reassess their certainty that identification and reversal of a transient or correctable cause in patients who have an episode of life-threatening VT/VF precludes further definitive treatment. Results from this study suggest that greater diligence is required in treatment and follow-up of these patients. Further evaluation of the relative contribution of these transient or correctable causes, compared with more permanent factors, is warranted. More research is needed to identify truly reversible causes of VT/VF.

Study limitations. Identification of the presumed transient or correctable cause of VT/VF could have been made in error. Physicians attempted to identify all underlying structural heart disease, but the relative contribution between any transient cause and the permanent factors may have been incorrectly assessed. Nevertheless, the assignment to the transient/correctable cause group was made on the best judgment of experienced clinician-investigators and probably reflects actual clinical practice.

The AVID physician did not necessarily perform the long-term follow-up. Transient or correctable factors may have been incompletely treated or could have recurred.

Though therapy at discharge was recorded, it could have been changed during long-term follow-up. Only data from the index hospitalization associated with the episode of VT or VF was recorded.

Nonfatal arrhythmia recurrence in patients with a transient or reversible cause for VT/VF was not followed. Only death was identified. It is possible that causes of death other than ventricular arrhythmias were responsible for our findings. However, it is unlikely that nonarrhythmic causes of death predominated in this population, which was younger and generally healthier.

Conclusions. This study suggests that patients diagnosed with "transient" or "correctable" causes for life-threatening VT/VF have a high mortality risk. Such patients may have a substrate for continued risk for serious arrhythmias. Ideal therapy is unknown, but these patients probably require more aggressive evaluation, treatment and follow-up than is currently practiced. More research is needed to identify truly reversible causes of VT/VF.

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