Syncope in Advanced Heart Failure: High Risk of Sudden Death Regardless of Origin of Syncope

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Objectives. The purpose of this study was to assess the importance of syncope as a warning sign for sudden death in advanced heart failure and to determine the relative importance of cardiac syncope and syncope from other causes.

Background. Despite remarkable advances in the pharmacologic approach to advanced heart failure, 20% to 40% of patients with advanced heart failure will die each year. In such patients, the relation between sudden death and the etiology of syncope has not been evaluated.

Methods. The relation of syncope to sudden death was evaluated in 491 consecutive patients with advanced heart failure (New York Heart Association functional class III or IV), no history of cardiac arrest and a mean left ventricular ejection fraction of 0.20 ± 0.11. Patients were evaluated for the presence and origin of syncope. The severity of heart failure was assessed from serum sodium levels, ejection fraction, functional class and echocardiographic and hemodynamic variables.

Results. Sixty patients (12%) had a history of syncope; the condition had a cardiac origin in 29 (48%) and was due to other causes in 31 (52%). The origin of heart failure was coronary artery disease in 234 patients (48%) and dilated cardiomyopathy in 253 (51%) and its severity was similar in patients with and without syncope. During a mean follow-up interval of 365 ± 419 days, 69 patients (14%) died suddenly and 66 patients (13%) died of progressive heart failure. The actuarial incidence of sudden death by 1 year was significantly greater in patients with (45%) than in those without (12%, p < 0.00001) syncope. In the Cox proportional hazards model, syncope predicted sudden death independent of arterial fibrillation, serum sodium, cardiac index, angiotensin-converting enzyme inhibition and patient age. The actuarial risk of sudden death by 1 year was similarly high in patients with either cardiac syncope or syncope from other causes (48% vs. 39%, p = NS).

Conclusions. Patients with advanced heart failure are at especially high risk for sudden death regardless of the etiology of syncope.

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Despite remarkable advances in the pharmacologic approach to advanced heart failure, 20% to 40% of patients with advanced heart failure will die each year, the majority suddenly and unexpectedly (1–3). Syncope, which is relatively common in patients with heart failure, has many etiologies. In studies of unselected patient groups with syncope, syncope of cardiac origin is associated with an increased risk of sudden death, whereas syncope of noncardiac origin has a more benign outcome (4–7). In many patients the etiology of syncope is not determined despite evaluation, but in the absence of evidence for ventricular tachycardia or other cardiac causes, the risk of sudden death is low (5,7). In patients with advanced heart failure, the relation between the etiology of syncope and sudden death has not been evaluated. The purpose of this study of 491 consecutive patients with advanced heart failure was to assess the incidence of syncope and its importance as a risk factor for sudden death.

Methods

Study group. The study group consisted of 491 consecutive patients with advanced heart failure without a history of cardiac arrest referred to the University of California, Los Angeles Medical Center for optimization of medical therapy, often in conjunction with evaluation for heart transplantation during the period April 1983 to April 1991. The patients had a mean age of 50 ± 12 years and 391 (80%) were male; they had a mean New York Heart Association functional class of 3.4 ± 0.6 on admission, and a mean left ventricular ejection fraction of 0.20 ± 0.07. Heart failure was due to coronary artery disease in 234 patients (48%), idiopathic dilated cardiomyopathy in 224 (46%), valvular heart disease in 25 (5%) and other or undetermined causes in 8 patients (1%). All patients underwent placement of a pulmonary artery catheter to guide therapy with intravenous followed by oral vasodilators and diuretic drugs tailored to meet hemodynamic goals (8,9). Only patients with hemodynamic stabili-
zation permitting hospital discharge were included in this study.

At the time of hospitalization all patients were evaluated by the University of California, Los Angeles Arrhythmia Service and were asked in detail about syncope history. Our general approach to patients with heart failure with recent (≤6 weeks) syncope has included evaluation with history and physical examination, scalar electrocardiogram (ECG), ECG monitoring, echocardiogram and electrophysiologic testing, although use of these procedures is left to the discretion of the attending physician.

Definitions and diagnostic criteria. The following definitions and diagnostic criteria were used.

Sudden death: death occurring within 1 hr of a change of symptoms or during sleep.

Syncope: sudden transient loss of consciousness not requiring electrical cardioversion for recovery.

Causes of syncope were assigned based on the following criteria (4):

Cardiac syncope: syncope due to tachyarrhythmia, bradyarrhythmia, valvular abnormality or acute cardiac ischemia, as defined below.

Noncardiac syncope: syncope due to orthostatic hypotension, situational or neurologic causes as defined later.

Syncope of undetermined origin: no identified etiology after evaluation.

Syncope from other causes: both noncardiac syncope and syncope of undetermined origin.

Ventricular tachyarrhythmia: sustained monomorphic ventricular tachycardia at the time of syncope or inducible at electrophysiologic testing, spontaneous polymorphic ventricular tachycardia associated with prolonged QT interval consistent with torsade de pointes, or symptomatic (presyncope or syncope) nonsustained ventricular tachycardia (spontaneously terminating in <30 s) during monitoring.

Supraventricular tachycardia: spontaneous or inducible supraventricular tachycardia with syncope or hypotension.

Bradycardia: high degree heart block or sinus bradycardia with syncope or sinus node or conduction system abnormalities detected at electrophysiologic testing (defined later).

Acute cardiac ischemia: chest pain at the time of syncope, with subsequent cardiac angiography revealing significant coronary obstruction and no other cause of syncope identified.

Valvular: critical aortic stenosis or mitral stenosis confirmed by echocardiography or cardiac catheterization.

Orthostatic hypotension: syncope related to sudden postural changes not associated with palpitation or chest pain in the absence of findings suggesting an alternative cause.

Situational: micturition or cough syncope.

Neurologic: Syncope associated with neurologic deficit confirmed by examination by a neurology consultant without evidence of a preceding arrhythmia or hypotension. Loss of consciousness due to seizure was not classified as syncope.

Electrophysiologic testing. Patients were taken to the electrophysiology laboratory in a lightly sedated, postabsorptive state. Multipolar electrode catheters were positioned in the high right atrium, His bundle and right ventricular apex. Programmed ventricular stimulation (Bloom Associates) was performed using one, two and three premature extrastimuli at twice diastolic threshold delivered during sinus rhythm and ventricular pacing at two or more basic drive cycle lengths (most commonly 600 and 400 ms) at the right ventricular apex and outflow tract (10). Inducible ventricular tachycardia was defined as sustained monomorphic ventricular tachycardia lasting >30 s or requiring cardioversion because of hemodynamic instability. Neither polymorphic ventricular tachycardia nor ventricular fibrillation was deemed a clinically significant end point. Syncope was attributed to a bradyarrhythmia based on electrophysiologic testing if there was a markedly prolonged sinus node recovery time (>2,000 ms), block distal to the His bundle during rapid atrial pacing, or an HV interval >100 ms (11,12).

Statistics. For discrete variables, chi-square tests were used. For continuous variables, independent Student t tests were used. One-year actuarial sudden death curves, logistic regression analysis and Cox proportional hazards models were constructed with BMDP programs (BMDP Statistical Software). Age, left ventricular ejection fraction, serum sodium, hemodynamic measures and echocardiographic measures were used as continuous variables. Sudden death curves were constructed by censoring all nonsudden deaths from the analysis at the time of death. Patients undergoing heart transplantation were censored from analysis at the time of operation. Actuarial survival curves were compared using the Mantel-Cox test. Statistical significance was defined as p < 0.05 for chi-square and t tests and p < 0.1 for logistic regression analysis, Cox proportional hazards models and the Mantel-Cox test.

Follow-up. Follow-up was considered to start at the time of hospital admission at this institution for optimization of heart failure therapy, not at the time of syncope. Patients were followed up by one of us in the University of California, Los Angeles Cardiomyopathy Clinic. In the event of death, the circumstances at the time of death were obtained from the family, personal physician and autopsy data when available. Telephone contact with all patients and their families or physicians, or both, was attempted in May 1991. Twenty-four patients (4%) were lost to follow-up within 1 yr (5% of patients with prior syncope vs. 3.9% of those without prior syncope, p = NS).

Results

Clinical characteristics. Of 491 consecutive patients with advanced heart failure without a history of cardiac arrest, 60 (12%) had had a prior syncopal episode after the onset of heart failure, the majority within the year preceding referral. Eleven patients (18%) had had syncope within 6 weeks of
Table 1. Clinical Characteristics of Patients With and Without Syncope

<table>
<thead>
<tr>
<th>Coronary artery disease</th>
<th>NYHA functional class</th>
<th>LVEF</th>
<th>Vasodilator</th>
<th>Antiarrhythmic drug</th>
<th>Atrial fibrillation</th>
<th>Pacemaker</th>
<th>Age (yr)</th>
<th>NSVT episodes/24 h</th>
<th>LVDD (mm²/m²)</th>
<th>Serum sodium (mEq/dl)</th>
<th>Hemodynamic variables on therapy</th>
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<tbody>
<tr>
<td>With Syncope (n = 60)</td>
<td>Without Syncope (n = 431)</td>
<td>p Value</td>
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<td>27 (45)</td>
<td>207 (48)</td>
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<td>0.21 ± 0.07</td>
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<td>ACE inhibitor</td>
<td>34 (57)</td>
<td>213 (40)</td>
<td>NS</td>
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<td>Hydralazine</td>
<td>23 (38)</td>
<td>144 (35)</td>
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<td>Other</td>
<td>3 (5)</td>
<td>65 (15)</td>
<td>NS</td>
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<tr>
<td>Antiarrhythmic drug</td>
<td>34 (57)</td>
<td>214 (50)</td>
<td>NS</td>
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<td>Amiodaron</td>
<td>20 (33)</td>
<td>120 (20)</td>
<td>NS</td>
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<td>Class I</td>
<td>10 (27)</td>
<td>94 (22)</td>
<td>NS</td>
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<td>Atrial fibrillation</td>
<td>10 (33)</td>
<td>76 (18)</td>
<td>0.003</td>
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<td>Pacemaker</td>
<td>9 (15)</td>
<td>27 (60)</td>
<td>0.01</td>
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<td>Age (yr)</td>
<td>49 ± 13</td>
<td>50 ± 13</td>
<td>NS</td>
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<td>NSVT episodes/24 h</td>
<td>18 ± 37</td>
<td>65 ± 342</td>
<td>NS</td>
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<td>LVDD (mm²/m²)</td>
<td>41 ± 9</td>
<td>40 ± 6</td>
<td>NS</td>
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<td>Serum sodium (mEq/dl)</td>
<td>134 ± 5</td>
<td>135 ± 5</td>
<td>NS</td>
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<td>Systolic arterial pressure (mm Hg)</td>
<td>101 ± 19</td>
<td>103 ± 42</td>
<td>NS</td>
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<td>HR (beats/min)</td>
<td>89 ± 14</td>
<td>90 ± 16</td>
<td>NS</td>
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<td>RAP (mm Hg)</td>
<td>8 ± 4</td>
<td>7 ± 4</td>
<td>NS</td>
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<td>PAS (mm Hg)</td>
<td>43 ± 13</td>
<td>40 ± 12</td>
<td>0.02</td>
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<td>PAD (mm Hg)</td>
<td>20 ± 6</td>
<td>19 ± 6</td>
<td>NS</td>
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<td>PCWP (mm Hg)</td>
<td>16 ± 6</td>
<td>15 ± 6</td>
<td>NS</td>
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<tr>
<td>CI (liters/min per m²)</td>
<td>2.9 ± 2.2</td>
<td>2.6 ± 0.6</td>
<td>NS</td>
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Values are expressed as mean value ± SD or number (%) of patients in group. ACE = angiotensin-converting enzyme; CI = cardiac index; HR = heart rate; LVDD = left ventricular diastolic index; LVEF = left ventricular ejection fraction; NS = not statistically significant; NSVT = non sustained ventricular tachycardia; NYHA = New York Heart Association; PAD = pulmonary artery diastolic pressure; PAS = pulmonary artery systolic pressure; PCWP = pulmonary capillary wedge pressure; PVCs = premature ventricular complexes; RAP = right atrial pressure; SBP = systolic blood pressure.

Table 2. Multivariate Cox Proportional Hazard Analysis

<table>
<thead>
<tr>
<th>Sudden Death</th>
<th>Univariate p Value</th>
<th>Multivariate p Value</th>
<th>Relative Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Syncope (all cases)</td>
<td>0.0003</td>
<td>&lt;0.0001</td>
<td>13.0</td>
</tr>
<tr>
<td>Serum sodium (mEq/dl)</td>
<td>0.009</td>
<td>0.01</td>
<td>5.8</td>
</tr>
<tr>
<td>Absence of angiotensin-converting enzyme inhibition</td>
<td>0.015</td>
<td>0.02</td>
<td>5.2</td>
</tr>
<tr>
<td>Cardiac index on therapy (liters/min per m²)</td>
<td>0.017</td>
<td>0.05</td>
<td>3.8</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>0.11</td>
<td>0.07</td>
<td>3.1</td>
</tr>
<tr>
<td>Left ventricular ejection fraction</td>
<td>0.05</td>
<td>0.09</td>
<td>2.8</td>
</tr>
<tr>
<td>Pulmonary capillary wedge pressure on therapy (mm Hg)</td>
<td>0.01</td>
<td>0.24</td>
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</table>

Mortality and sudden death. After a mean follow-up period of 365 ± 419 days, 165 patients (35%) were alive and well, 148 (30%) had undertaken heart transplantation, 69 (14%) had died suddenly, 66 (13%) had died of progressive heart failure, 19 (4%) had died of noncardiac or unknown causes and 24 (4%) were lost to follow-up. For the total group the actuarial rate of mortality from all causes at 1 year was 29% and the rate of sudden death was 15%. The actuarial 1-year rate of mortality from all causes was significantly greater in patients with than in those without syncope (65% vs. 25%, p < 0.00001). This increased mortality was predominantly due to the large number of sudden deaths in the patients with syncope. The 1-year actuarial risk of sudden death was significantly greater in patients with than in those without syncope (45% vs. 12%, p < 0.00001) (Fig. 1). In patients with syncope, the risk of sudden death did not differ between patients with recent (~6 weeks) or nonrecent (>6 weeks) syncope (p = 0.68).

Table 2 shows the multivariate Cox proportional hazards model analysis for sudden death. A history of syncope was associated with sudden death independent of atrial fibrillation, serum sodium, absence of angiotensin-converting enzyme inhibition, patient age and cardiac index during therapy.
spy. Similarly, syncope was independently related to mortality from all causes (P = 0.005) but not to deaths due to progressive heart failure (p = 0.66).

Etiology of syncope. Table 3 shows the causes of syncope in the study group. Evaluation included electrophysiologic testing in 10 (91%) of 11 patients presenting within 6 weeks of the syncopal event and in 29 (48%) of all patients with syncope. Of 60 patients, 29 patients (48%) had a cardiac origin of syncope, and 31 patients (52%) had a noncardiac (n = 9) or undetermined (n = 18) origin. Of 29 patients with a cardiac origin of syncope, 21 (72%) had ventricular tachyarrhythmias, including symptomatic nonsustained ventricular tachycardia in 8, sustained ventricular tachycardia recorded during syncope in 5, drug-induced torsade de pointes in 2, and inducible sustained monomorphic ventricular tachycardia in 6. Five patients had syncope due to bradyarrhythmia, including high degree heart block in four and sinus bradycardia due to a beta-adrenergic blocking drug in one. Syncope was due to supraventricular tachycardia in one patient and to critical valvular stenosis in two patients.

Of 31 patients without a cardiac cause of syncope, 9 had orthostatic hypotension, 3 had situational syncope and 1 patient had neurologic syncope due to a transient ischemic attack. The cause of syncope was undetermined in 18 patients despite evaluation, including electrophysiologic testing in 10 patients (56%).

Table 4 compares the clinical characteristics of patients with cardiac syncope and those with syncope from other causes. The two patient groups had a similar severity of heart failure as measured by serum sodium, New York Heart Association functional class, left ventricular ejection fraction and hemodynamic measurements. The time from syncope to referral, cause of heart failure, presence of atrial fibrillation, use of vasodilator therapy and permanent pacemakers did not differ between groups. Patients with cardiac syncope were significantly older than patients with other causes of syncope (54 ± 11 vs. 45 ± 13 years, p = 0.009) and had a lower pulmonary artery systolic pressure during vasodilator therapy (40 ± 10 vs. 47 ± 15 mm Hg, p = 0.04). By logistic regression analysis, coronary artery disease (p = 0.96), time from syncope to referral (p = 0.60) and serum sodium (p = 0.82) were not predictive of a cardiac origin of syncope.

Sudden death and etiology of syncope. Nine (31%) of 29 patients with cardiac syncope and 7 (23%) of 31 patients with syncope from other causes died suddenly. The actuarial risk of sudden death by 1 year did not differ between patients with cardiac syncope and patients with syncope from other causes (49% vs. 39%, p = NS) (Fig. 2). In the 29 patients who underwent electrophysiologic testing, the 1-year actuarial risk of sudden death did not differ statistically between patients with cardiac syncope and those with other causes of syncope (54% vs. 32%, p = NS). Similarly, the risk of sudden death did not differ between patients who did or did not undergo electrophysiologic testing (47% vs. 51%, p = NS). Mortality from all causes did not differ between pa-
tients with cardiac syncope and patients with other causes of syncope (62% vs. 66%, p = NS).

Figure 3 relates the etiology of syncope and type of therapy to sudden death. Of 21 patients with cardiac syncope due to ventricular tachyarrhythmias, 8 received electrophysiologically guided antiarrhythmic therapy that rendered ventricular tachycardia noninducible or well tolerated in 7 and poorly tolerated in 1 patient. Nine patients received empiric antiarrhythmic therapy to suppress ambient ectopic activity (class I agents in six, amiodarone in three). The incidence of sudden death did not differ between patients receiving therapy guided by electrophysiologic testing (one sudden death in eight patients) and those receiving therapy administered empirically (four sudden deaths in nine patients, p = 0.36), although the number of patients in each group was small.

Five patients had cardiac syncope due to bradyarrhythmias. Four of the five underwent permanent pacemaker implantation, one of whom subsequently died suddenly. In the fifth patient, therapy with a beta-adrenergic blocking drug was withdrawn, resulting in resolution of symptoms.

Of 31 patients without a cardiac cause of syncope, 4 were treated with class I drugs, 7 received amiodarone for atrial arrhythmias or frequent ventricular ectopic activity and 20 did not receive an antiarrhythmic drug. Four patients (21%) receiving no antiarrhythmic agents and three patients (27%) receiving antiarrhythmic drugs died suddenly.

**Discussion**

**Syncope of cardiac versus noncardiac causes.** Syncope, whether of cardiac or noncardiac origin, indicates a transient loss of central nervous system perfusion. In large studies of patients with syncope, a cardiac cause of syncope is associated with increased risk of sudden death, presumably because the previously transient abnormality becomes persistent with fatal consequences. For example, nonsustained ventricular tachycardia becomes sustained and degenerates into ventricular fibrillation. In these studies of unselected patients, noncardiac and undetermined causes of syncope have a benign prognosis without long-term consequences (4-7, 13). In striking contrast to patients without heart failure, patients with advanced heart failure who have syncope of noncardiac or undetermined origin have a markedly increased risk for sudden death. Several possible explanations for this discrepancy exist. Patients with heart failure classified as having syncope of noncardiac or undetermined origin may be more likely than patients without heart failure to have a cardiac origin of syncope that has escaped diagnosis. Electrocardiographic monitoring and programmed electrical stimulation are frequently used to determine the etiology of syncope but may be less predictive in patients with advanced heart failure. During ECG monitoring, asymptomatic complex ectopic activity was recorded in 82% of our patients. However, the majority of patients with class III and IV heart failure have complex ventricular ectopic activity during ambulatory monitoring (14-19). Such activity has been associated with an increased risk of sudden death in many, but not all, studies of patients with advanced heart failure (15-19). Yet, unless presyncope or syncope occurs during recordings associated with ventricular ectopic activity, it is difficult to attribute syncope to this highly prevalent arrhythmia. Only 13% of our patients with syncope had nonsustained ventricular tachycardia with symptoms implicating this arrhythmia as the cause of syncope.

In our patients, ventricular tachycardia was initiated with programmed stimulation in only 27% of patients with syncope tested, a finding consistent with prior studies (12) of electrophysiologic testing in patients with syncope who have structural heart disease. But the predictive value of nonin-
ducibility of arrhythmia at programmed electrical stimulation in high risk patients with a severely depressed left ventricular ejection fraction has been questioned (20–23). Programmed stimulation, useful for detecting ventricular tachycardia due to reentry within an infarct scar, may be less useful in detecting arrhythmias associated with hypertrophy and noninfarct-related causes. Ventricular tachycardia due to early afterdepolarizations with triggered activity or abnormal automaticity, modulated by electrolyte abnormalities and changes in autonomic tone, may not be inducible with programmed stimulation (24). In studies of patients with heart failure and idiopathic dilated cardiomyopathy who have not previously had spontaneous sustained ventricular tachycardia, sustained monomorphic ventricular tachycardia is inducible in only 0% to 13% of patients (21–23). However, the risk of sudden death is high despite the absence of inducible ventricular tachycardia, perhaps because of bradyarrhythmias or nonreentrant mechanisms of ventricular tachycardia (25).

Similarly, detection of sporadic bradyarrhythmias by ambulatory ECG recording or programmed stimulation is limited (26,27). Severe, persistent abnormalities can usually be diagnosed, but fluctuating abnormalities influenced by humoral or autonomic triggers may go undetected (26,28). In our study group, no patient had a bradyarrhythmic cause of syncope established at electrophysiologic testing. In summary, the diagnosis of syncope of noncardiac or undetermined origin reflects the limits of available diagnostic tests in advanced heart failure and is not indicative of a more favorable outcome.

In patients with heart failure who have an identified cardiac cause of syncope, the risk of sudden death remains high despite therapy directed to the cause of the syncope. Antiarrhythmic drug therapy may have limited efficacy in preventing sudden death in patients with severely depressed ventricular function (29–31). In fact, in survivors of myocardial infarction and in patients with heart failure, therapy with class I drugs may actually increase the risk of sudden death (32,33). In our study patients, there was no significant difference in the risk of sudden death between patients receiving antiarrhythmic therapy guided by programmed stimulation and those treated empirically, although the number of patients is relatively small.

Possible role of autonomic nervous system. Another potential reason for the adverse prognostic significance of syncope is that it may identify a person with impaired physiologic compensatory reflexes, who is unable to maintain blood pressure in the face of challenges to homeostasis, such as orthostasis or mild arrhythmias. Thus, a simple faint may become sustained, leading to potentially lethal consequences, such as ischemia or arrhythmias. Ferguson and associates (34) studied cardiopulmonary baroreceptors in heart failure and reported paradoxic forearm vasodilation in response to lower body negative pressure in some patients. In a study of the sympathetic nerve response to ventricular tachycardia, Smith et al. (35) recorded sympathetic nerve activity at the peroneal nerve during induced ventricular tachycardia and ventricular pacing in 16 patients, the majority with prior syncope. Hemodynamic stability during ventricular tachycardia was directly related to the increase in sympathetic nerve activity independent of tachycardia rate and left ventricular ejection fraction (35). Abnormally depressed heart rate variability, another measure of autonomic dysfunction, has been reported in patients with heart failure and may be related to sudden death (36,37). Syncope from minor hemodynamic stresses, such as standing or micturition, may identify patients with heart failure who have inadequate sympathetic nerve responses or paradoxic cardiopulmonary or arterial baroreceptor reflexes that under certain circumstances tend to lead to hypotension with a malignant outcome. It is not known whether evaluation of reflex responses mediated by the autonomic nervous system—perhaps by using tilt testing, sympathetic nerve response to ventricular pacing and assessment of heart rate variability—will allow better identification of the highest risk patients.

Limitations of the study. This is a retrospective study. Patients had syncope at various times preceding referral, and therapy was sometimes instituted before our evaluation. Although electrophysiologic testing was not performed in all patients, it was performed in 91% of those patients with recent syncope. In a recent analysis of electrophysiologic testing in patients with syncope, including a subset with markedly decreased left ventricular function, the risk of sudden death was similarly high in patients with diagnostic and nondiagnostic electrophysiologic tests (38). Our evaluation of syncope did not include testing of autonomic nervous system activity or cardiopulmonary and arterial baroreceptor reflexes. Some etiologies of syncope included only small numbers of patients; therefore, conclusions regarding the prognostic significance of a particular subgroup, for example those with torsiade de pointes, cannot be made. Only one patient with syncope received an implantable defibrillator. Our patients have advanced heart failure warranting referral for evaluation for heart transplantation, representing the most severe end of the spectrum of outpatients with heart failure. Therefore, our results may not be applicable to all patients with heart failure.

Conclusions. Patients with advanced heart failure who have syncope are at especially high risk for sudden death regardless of the etiology of syncope. Although evaluation and therapy directed toward the underlying cause of syncope is recommended, the cause often remains undetermined and the rate of sudden death is high even with therapy. Transient abnormalities in autonomic activity or paradoxic baroreceptor reflex responses contributing to syncope could account for the low diagnostic yield of standard testing. Perhaps evaluation of autonomic nervous system responses may improve detection of the cause of syncope and aid in prognostication. Further study is needed to determine whether an aggressive approach to diagnosis and therapy can reduce the incidence of sudden death in these patients.
Earlier consideration for heart transplantation may be warranted in patients with heart failure who have a history of syncope.

References

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