Evidence That Nonsustained Polymorphic Ventricular Tachycardia Causes Syncope (Data From Implantable Cardioverter Defibrillators)

D. Michael Farmer, MD, Craig A. Swygman, BA, Paul J. Wang, MD, N. A. Mark Estes III, MD, and Mark S. Link, MD

Patients with syncope account for ≥6% of hospital admissions and 3% of emergency room visits. Whether and how often nonsustained ventricular fibrillation (VF) or nonsustained polymorphic ventricular tachycardia (NSPMVT) can cause syncope is not entirely clear. In patients with the long QT syndrome, nonsustained Torsades de Pointes causing syncope is well described. Other reports, generally in patients with normal hearts, have occasionally described the association of NSPMVT and syncope. However, it is generally believed that VF is a sustained tachyarrhythmia that results in death and that NSPMVT is unusual as a cause of syncope. The current generation of implantable cardioverter defibrillators (ICDs) with intracardiac electrogram storage offer an opportunity to evaluate the incidence of NSPMVT relative to sustained VF and to document the association of syncope and presyncopal symptoms with these arrhythmias.

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Nonsustained polymorphic ventricular tachycardia (NSPMVT) was defined as a VT of 220 beats/min in which intracardiac morphology varied from beat to beat, was ≥6 beats in duration, and spontaneously terminated. Syncope was defined as loss of consciousness and postural tone. Presyncope was defined as near loss of consciousness. Episodes of VF that resulted from antitachycardia pacing, ICD shock, or VT degeneration were excluded from the study. The correlation of symptoms and time from each episode were documented. Awake hours were defined as from 7 a.m. to 10 p.m.

All continuous variables are reported as mean ± SD. Continuous data were analyzed with the Student’s t test; categoric data were analyzed using the chi-square test. P values of ≤0.05 were considered significant.

From July 1994 to July 2000, 705 patients with third generation ICDs were followed. Ten patients were excluded because of the diagnosis of long QT syndrome, and 10 patients were excluded for possessing ICDs without intracardiac electrogram storage capability. Of the remaining 685 patients, 47 (35 men and 12 women) had ≥1 episodes of spontaneous VF or NSPMVT. Mean age of these patients at the time of implantation was 61.4 ± 14 years, and mean left ventricular ejection fraction was 32.7 ± 14%. Of these 47 patients, 25 (53%) presented with cardiac arrest. Twelve patients (25%) presented with sustained monomorphic VT and syncope or presyncope. Ten patients (21%) presented with syncope and no documented arrhythmia. Of these 10 patients, electrophysiologic testing demonstrated that 2 patients had VT induced, 5 patients had VF induced, and 3 patients had no inducible arrhythmias.

Structural heart disease was found in 46 patients (98%), and it included coronary artery disease in 28 (60%), idiopathic dilated cardiomyopathy in 16 (34%), and arrhythmogenic right ventricular dysplasia in 2 (4%). One patient had no structural heart disease. This patient presented with VF arrest, did not have structural heart disease, Brugada’s syndrome, or long QT syndrome, and was thought to have idiopathic VF.

During the follow-up period, 146 total episodes met the inclusion criteria for the study, including 59 episodes of NSPMVT (40%) and 87 episodes of VF (60%). Thirteen patients had episodes of both NSPMVT (Figure 1) and VF (Figure 2), whereas 12 patients had only NSPMVT and 22 had only VF. All sustained episodes were successfully terminated by the first ICD shock, with 1 episode of VF reinitiating shortly after termination. Amiodarone was prescribed at the time of arrhythmia in 29 episodes (9 patients) and sotalol was prescribed in 3 episodes (3 patients), whereas 114 episodes (35 patients) occurred in the absence of antiarrhythmic agents.

Of the 59 NSPMVT episodes, 6 were associated
with syncope (10%), 7 with presyncope (12%), and 46 were asymptomatic (78%; Figure 3). Of the 59 episodes of NSPMVT, 37 occurred during awake hours (63%). Of the episodes of NSPMVT that occurred during awake hours, 6 were associated with syncope (16%), 6 with presyncope (16%), and 25 were asymptomatic (43%). Mean duration of the NSPMVT episode was 5.3 ± 3.6 seconds.

Of the 87 VF episodes, 28 were associated with syncope (32%), 22 with presyncope (25%), and 37 were asymptomatic (43%). Of the 87 episodes of VF, 65 occurred during awake hours (75%). Of these episodes of VF that occurred during awake hours, 27 were associated with syncope (42%), 17 with presyncope (26%), and 21 were asymptomatic (32%). Mean duration of the VF episodes was 13.3 ± 4.9 seconds.

Gender and type of structural heart disease were not significant predictors of syncope or presyncope. Daytime occurrence of arrhythmia was a predictor of syncope (p < 0.0001), as well as a lower left ventricular ejection fraction (p = 0.002). VF compared with NSPMVT was a predictor of syncope (p < 0.0001). Syncope or presyncope was seen in 57% of the VF episodes compared with 22% in the NSPMVT episodes (68% vs 32% during awake hours). Cycle length of the arrhythmia was not a predictor of syncope. A longer episode duration (p = 0.0008) was a predictor of syncope or presyncope (Figure 3). In symptomatic events, mean episode duration was 12.0 ± 5.5 seconds compared with a mean episode duration in the nonsyncopal events of 8.5 ± 5.8 seconds (p < 0.0001). If NSPMVT or VF was <10 seconds in duration, the incidence of syncope or presyncope was 25% compared with 62% if the arrhythmia was ≥10 seconds.

In patients with current-generation ICDs, 40% of all VF episodes were nonsustained, and of the episodes of NSPMVT that occurred during awake hours, 32% were associated with syncope or presyncope. This incidence of NSPMVT relative to sustained VF was greater than previously believed, and NSPMVT is a distinct tachyarrhythmia that causes syncope without causing death. Furthermore, the incidence of NSPMVT may be higher than found in this report secondary to ICD termination of an arrhythmia that would have spontaneously terminated. Therefore, NSPMVT must be considered in the evaluation of a patient with syncope of unknown etiology and structural heart disease.

There have only been limited previous reports that have documented the association of NSPMVT and syncope, and no reports, to our knowledge, that have compared the incidence of NSPMVT and sustained VF in a large population of patients at risk for ventricular arrhythmias. In contrast with our study population, the previous case reports of documented NSPMVT that caused syncope were primarily in patients without structural heart disease2,4–6 or with long QT syndrome. Our study population excluded patients with long QT syndrome and had only 1 patient with a normal heart. Although 60% of our population had a diagnosis of coronary artery disease, there was no clinical suggestion of acute ischemia causing arrhythmias in this patient group.

Syncope or presyncope was associated with 57% of all episodes of sustained VF and 68% of those episodes of sustained VF that occurred during awake hours. Although the incidence of syncope with VF episodes was lower than expected in our report, other investigators have described similar findings. In a previous report by Grimm et al8, the investigators concluded that most patients with either electrocardiographically documented VT or VF preceding spontaneous ICD shocks had no or mild symptoms preceding the shock. Only 27 of 67 patients (40%) in this study had syncope or severe dizziness associated with electrocardiographically documented ICD shocks for VT or VF. In contrast with our study, which included only episodes of VF, Grimm et al8 did not differentiate rapid VT from VF. Other conditions may have played a role in the large number of asymptomatic VF episodes. Many of the sustained events would likely have caused more symptoms if the arrhythmias continued, but these arrhythmias were terminated by defibrillation. It is also possible that some episodes were asymptomatic because the patient was sleeping or recumbent at the time of the arrhythmia.

These data have important implications for the programming and charge time of current-generation ICDs. There was a clear increase in the incidence of symptoms when the arrhythmia lasted ≥10 seconds. Furthermore, as the length of arrhythmia increased, the relative incidence of syncope compared with presyncope increased (Figure 3). If the time of VF can be decreased to <10 seconds (including detection time, charge time, and therapy), then the risk of syncope can be decreased.

In summary, NSPMVT causing syncope or presyncope in patients with structural heart disease is more common than previously believed. Therefore, in patients with syncope of unknown origin and
underlying cardiac disease, NSPMVT as a cause of syncope must be considered. Limiting the time of VF to <10 seconds using appropriate programming and charge times of ICDs may reduce the risk of syncope.

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Prevalence of Interatrial Block in a General Hospital Population

Navaid Asad, MD, and David H. Spodick, MD, DSC

Interatrial block (IAB) manifests as a prolonged P-wave duration ≥110 ms. It is considered an electric abnormality, principally of the Bachmann bundle, that delays the conduction of atrial depolarization from the right to the left atrium. Not only does IAB correlate with left atrial enlargement and atrial electromechanical dysfunction, but it is also a strong predictor of atrial flutter and fibrillation. A prevalence of 33% of all patients with IAB (41% of patients in sinus rhythm) and its ignorance among physicians was determined in a recent study. Therefore, we decided to determine its prevalence at another general hospital population.

We evaluated 1,000 consecutive electrocardiograms (ECGs). Of these, we measured the maximum P-wave duration in all of the 12 leads in ECGs with sinus rhythm. P-wave duration ≥120 ms in any electrocardiographic lead was regarded as IAB. Patients were later divided into subgroups based on age, and an age-based comparison was made between patients with and without IAB.

Of the 1,000 ECGs, 916 ECGs (92%) showed sinus rhythm, 58 (6%) showed atrial fibrillation, 11 (1%) showed other ectopic atrial rhythm, 5 showed paced rhythm, 4 showed atrial flutter, 3 showed junctional rhythm, and 3 showed supraventricular tachycardia. Four hundred thirty (47%) of the 916 ECGs that had IAB. Eleven percent of patients aged 20 to 29, 21% aged 30 to 39, 32% aged 40 to 49, 50% aged 50 to 59, 59% aged 60 to 69, 59% aged 70 to 79, 58% aged 80 to 89, and 50% aged ≥90 showed P-wave duration ≥120 ms (Table 1). The age and gender of 57 patients in sinus rhythm was not available.

The present investigation showed a very high prevalence of IAB in a hospital population. Above the age of 60 years, more patients in sinus rhythm had IAB than those without it. Considering the consequences of atrial fibrillation, including subsequent stroke in such patients, it is essential that IAB be recognized early because it is associated with a higher incidence of abnormalities in atrial excitability and significant electromechanical dysfunction of the left atrium.


Table 1. Age Distribution of Patients With and Without Interatrial Block (IAB)

<table>
<thead>
<tr>
<th>Age Group (yr)</th>
<th>IAB Present (%)</th>
<th>IAB Absent (%)</th>
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<tbody>
<tr>
<td>All ages</td>
<td>430 (47)</td>
<td>916 (53)</td>
</tr>
<tr>
<td>Teenagers (13–19)</td>
<td>0 (0)</td>
<td>6 (100)</td>
</tr>
<tr>
<td>20–29</td>
<td>4 (11)</td>
<td>37 (89)</td>
</tr>
<tr>
<td>30–39</td>
<td>12 (21)</td>
<td>56 (79)</td>
</tr>
<tr>
<td>40–49</td>
<td>45 (32)</td>
<td>141 (68)</td>
</tr>
<tr>
<td>50–59</td>
<td>80 (50)</td>
<td>160 (50)</td>
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<tr>
<td>60–69</td>
<td>69 (59)</td>
<td>116 (41)</td>
</tr>
<tr>
<td>70–79</td>
<td>92 (59)</td>
<td>157 (41)</td>
</tr>
<tr>
<td>80–89</td>
<td>88 (58)</td>
<td>152 (42)</td>
</tr>
<tr>
<td>≥90</td>
<td>17 (50)</td>
<td>34 (50)</td>
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Values are expressed as number of patients (%).