Syncope While Driving
Clinical Characteristics, Causes, and Prognosis
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Background—The risk of syncope occurring while driving has obvious implications for personal and public safety. We aimed to define the clinical characteristics, causes, and prognosis of syncope while driving.

Methods and Results—In this case-control study of consecutive patients evaluated for syncope from 1996 through 1998 at an academic medical center, we documented causes, clinical characteristics, and recurrence of syncope while driving. Of 3877 patients identified, 381 (9.8%) had syncope while driving (driving group). Compared with the 3496 patients (90.2%) who did not have syncope while driving, the driving group was younger ($P < 0.01$) and had higher percentages of male patients ($P < 0.001$) and patients with a history of any cardiovascular disease ($P < 0.01$) and stroke ($P < 0.02$). Syncope while driving was commonly caused by neurally mediated syncope (37.3%) and cardiac arrhythmias (11.8%). Long-term survival in the driving group was comparable to that of an age- and sex-matched cohort from the Minnesota population ($P = 0.15$). Among the driving group, syncope recurred in 72 patients, 35 of whom (48.6%) had recurrence >6 months after the initial evaluation. Recurrences during driving happened in 10 patients in the driving group, 7 of which (70%) were >12 months after the initial evaluation.

Conclusions—In our study, neurally mediated syncope was the most common type of syncope while driving. The causes of syncope, the late recurrences of syncope (during >6 months of follow-up), and the overall low incidence of recurrent syncope while driving provide useful information to supplement current recommendations on driving for these patients. (Circulation. 2009;120:928-934.)

Key Words: prognosis ■ survival ■ syncope

Syncope is the clinical manifestation of a temporary interruption of global cerebral perfusion that causes a relatively sudden onset and transient loss of consciousness and postural tone with spontaneous, complete recovery.1,2 Several studies have shown syncope to be a common event; in the Framingham study, the cumulative incidence of syncope was estimated at 3% to 6% over 10 years, and the recurrence rate was 9% to 22%.3,4 In the healthcare setting, syncope accounts for 3% to 5% of visits to the emergency department and 1% to 6% of hospital medical admissions.5–7 The vast majority of syncopal spells are not a consequence of cardiac arrhythmias or underlying structural heart disease.1,2

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Although syncope has not been shown to be an independent predictor of increased mortality,8–10 the risk of a syncopal episode occurring while driving has obvious implications for personal and public safety.2,11–16 Recommendations for driving with regard to syncope were made by the European Society of Cardiology and in a joint Scientific Statement from the American Heart Association and Heart Rhythm Society; these recommendations focused on patients with primary cardiac arrhythmias and neurocardiogenic conditions associated with syncope.12,17,18 In the general population, much remains unknown about the causes and consequences, clinical characteristics, and predictors of syncope while driving. Few descriptions exist comparing patients experiencing syncope while driving and those experiencing syncope while not driving.

In this case-control study from a large, regional, tertiary medical center, we aimed to achieve several objectives: (1) to identify clinical characteristics of patients with syncope while driving and the causes of these syncopal episodes; (2) to compare patients with syncope while driving and while not driving and to determine predictors of syncope while driving; and (3) to determine outcomes, with focus on recurrent syncope, after the index event of syncope while driving. The ultimate goal of our study is to provide useful information for future considerations on driving to improve public safety for both the patients and the general public.
Methods

Patients
The study group comprised all patients who underwent evaluation for syncope at Mayo Clinic in Rochester, Minn, from January 1, 1996, through December 31, 1998. The patients had been referred from Mayo Clinic outpatient clinics, inpatient services, hospital emergency departments, and other institutions. The inclusion criterion was the occurrence of a documented syncopal event, with syncope being listed as the primary or secondary dismissal diagnosis. Within this group of patients, those who had a syncopal episode while driving were identified as the study group. Patients were excluded if they required resuscitative efforts for cardiorespiratory arrest or had disorders resembling syncope without loss of consciousness (somatization disorders, cataplexy, transient ischemic attacks, or cerebral vascular accidents).18 This study was approved by and followed the guidelines for informed consent of the Mayo Clinic Institutional Review Board.

Study Design
All study patients were identified retrospectively from an ongoing syncope database at Mayo Clinic. Patients who had syncope while driving were designated the study group; all other patients who had syncope that did not occur while driving were designated the control group. The only eligibility criterion that differed between the study and control groups was the presence of syncope while driving. Follow-up was conducted through a prospectively designed written survey.

Characterization of Syncope
Syncope was defined as a clinical manifestation of a temporary interruption of global cerebral perfusion resulting in a sudden and transient loss of consciousness and postural tone with spontaneous recovery without therapeutic intervention.16 “Syncope-mimic” conditions associated with loss of consciousness not caused by cerebral hypoperfusion (seizures, trauma, metabolic conditions, drug or alcohol intoxication) were not excluded from the study because of their relevance to issues related to driving. These conditions were tabulated in the “other” category for causes of syncope (see below). Cases with documented drug or alcohol intoxication with altered state of consciousness were excluded (eg, the drunken-driving population).

The causes of syncope were divided into 10 clinically relevant diagnostic categories modified from our previous report17 based on pathophysiological mechanisms and therapeutic targets. These 10 diagnostic categories were (1) bradyarrhythmia, (2) ventricular tachyarrhythmias, (3) supraventricular tachyarrhythmias, (4) structural cardiac or cardiopulmonary disease, (5) neurally mediated syncope (vasovagal or situational), (6) carotid sinus hypersensitivity, (7) cerebrovascular disease, (8) orthostatic intolerance (including orthostatic hypotension, autonomic conditions, dehydration, or drug induced), (9) others (syncope-mimic conditions described above), and (10) unknown. If a cause was uncertain, the final diagnostic category was assigned by consensus of 2 authors (D.S. and W.-K.S.).

Data Collection
The patient database used for the study contains >2000 fields with 1000 elements categorized as demographics, history, diagnostic and therapeutic intervention, laboratory data, and follow-up. Medical records were reviewed by a trained data abstractor. Patient data abstracted for the study included baseline characteristics; clinical features of syncopal episodes, including prodrome, frequency, body position, and symptoms during recovery; results of diagnostic tests; and follow-up information, including presence, absence, and frequency of recurrent symptoms and mortality information such as cause and time of death if applicable.

Follow-Up
Follow-up information was acquired from medical records and from a prospective survey designed by the study investigators. The survey was mailed to all patients for follow-up. Patients who did not respond to the survey were contacted by telephone by the investigators. Relatives of the patients completed the survey for patients who were unable to complete it for various reasons (such as death or mental illness). Data were obtained on recurrent events, circumstances associated with each recurrence, diagnostic tests, hospitalizations, treatment, and death.

Statistics
The x² test for independence was used to measure differences between the driving and nondriving groups for categorical variables. Continuous variables were compared by use of the Wilcoxon rank-sum test. The cumulative probability of mortality and recurrence of syncope was estimated with the Kaplan-Meier method. The survival rate of the patients with syncope while driving was compared with the expected survival calculated based on age- and sex-specific matched mortality rates from the total Minnesota population. These rates were compared by use of a 1-sample log-rank test. Cox proportional-hazards models were used to predict clinical characteristics associated with decreased survival. Variables were selected for the final model using a stepwise selection process. A value of P=0.05 was considered significant.

The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

Results
During the study period from 1996 through 1998, we evaluated 3877 patients at Mayo Clinic in Rochester, Minn, for syncope. Within this group, 381 patients (9.8%) had an episode of syncope while driving (driving group). Male patients made up the majority (65.1%) of the driving group, whereas only 47.4% of the group of 3496 patients who did not have syncope while driving (nondriving group) were male (P<0.001) (Table 1). The mean±SD age was 55.8±18.4 years in the driving group; the nondriving group was slightly older (mean±SD age, 56.8±23.8 years; P=0.01). Age distributions of the entire group of patients (N=3877) and the driving group (n=381) are shown in Figure 1. A bimodal distribution was present among all syncope patients (Figure 1A) but not among the driving group (Figure 1B).

Significant differences between groups in medical history included history of cardiovascular disease (P=0.01) and stroke (P=0.02), both being more common in the driving group. No other clinical characteristics were significantly different between groups (Table 1). Among both groups, 45% of the driving group and 48% of the nondriving group (P=0.24) were referred for syncope evaluation within 1 month of the index event.

Comparison of Symptoms
Prodromal symptoms of nausea, palpitations, chest pain, dyspnea, and ear ringing were more common in the driving group (all P≤0.05; Table 2). There were no significant differences in the duration, nature, and presence of symptoms during recovery between the 2 groups. In the driving group, 28.6% had an injury, 4.9% more than in the nondriving group (P=0.03), but there was no significant difference in the percentage of patients requiring hospital care (Table 2).

Causes of Syncope
The most common cause of syncope while driving was neurally mediated syncope (vasovagal syncope), occurring in
142 (37.3%) of those patients (Table 3). The next most common identifiable causes were cardiac arrhythmias, including bradyarrhythmias, ventricular tachyarrhythmias, and supraventricular tachyarrhythmias (11.8%; some patients had >1 type of arrhythmia), and orthostatic intolerance (4.7%). No cause was determined in 90 patients (23.6%) in the driving group. In 34 patients (8.9%), potential cause of syncope was present. The presumed causes of syncope had similar frequencies in the nondriving group, except syncope-mimic conditions ("other" cause), which were more common among nondriving patients ($P=0.004$; Table 3).

**Treatment of Syncope**

Among the patients with neurally mediated syncope, drug therapy was used in 39 driving group patients (27.5%) and 333 nondriving group patients (26.7%). The other patients in both groups were treated conservatively (education, fluids, salt, lower-extremity strength training, or drug withdrawal if appropriate). A pacemaker was implanted in only 1 driving group patient with neurocardiogenic syncope because this patient had persistent paroxysms of bradycardia with loss of consciousness noted during head-up tilt testing and documented spontaneously. For patients with bradyarrhythmias, pacemakers were implanted in 9 driving group patients (36.0%) and 94 nondriving group patients (42.5%). Adjustment of medical therapy, drug withdrawal, and observation were implemented for the remaining patients with bradyarrhythmia in both groups. Among patients with ventricular tachyarrhythmias, antiarrhythmic drug therapy was used in 0 driving group patients and 10 nondriving group patients (7.7%). After transient substrates (electrolyte abnormality, acute ischemia or infarction, drug-induced proarrhythmia) were excluded, an implantable cardioverter-defibrillator was implanted in the remaining patients with sustained ventricular arrhythmias in both groups.

**Follow-Up Outcome**

Follow-up, available for 380 driving group patients (99.7%), totaled 1463 patient-years, with a mean duration of 3.85 years; 3473 nondriving group patients (99.3%) had 12,551 total patient-years of follow-up averaging 3.61 years. During follow-up, recurrent syncope was reported by 72 patients in the driving group (8-year probability, 25.1%) and 713 patients in the nondriving group (8-year probability, 28.9%) ($P=0.21$). The actuarial recurrence of syncope at 6 and 12 months was 12.0% and 14.1% for the driving group patients and 12.0% and 17.0% for the nondriving group patients ($P=0.21$) (Figure 2).

Among the 72 driving group patients with recurrence of syncope, 61 (84.7%) had a prior history of syncope. Thirty-seven (51.3%) of these recurrences occurred within 6 months after the initial evaluation, and 44 (61.1%) occurred within 12 months. The recurrences included 10 episodes while driving,
2 of which occurred within 6 months of the initial evaluation, and a total of 3 episodes had occurred by 12 months. The actuarial recurrence of syncope during driving was 0.7% at 6 months and 1.1% at 12 months. The cumulative probability of recurrence while driving was 7% over the 8 years of follow-up. Therapy in this group included 23 pacemaker implantations (31.9%) and 6 implantable cardioverter-defibrillator implantations (8.3%).

There was no increased mortality (P=0.15) when the long-term survival of all driving group patients was compared with expected survival for the age- and sex-matched Minnesota population. Presence of underlying cardiac disease, including ventricular tachycardia, atrioventricular block, coronary artery disease, a history of myocardial infarction, and advanced age, was predictive of decreased survival among patients in the driving group (Table 4). Long-term survival in the nondriving group was lower than in the driving group (Figure 3B).

### Discussion

The age at the index evaluation at Mayo Clinic for all patients with syncope appears to have a bimodal distribution (Figure 1). This bimodal age distribution is similar to that shown in a population-based survey study. A clear peak may not appear in the younger age population among the driving group because most teenagers do not drive independently until 16 years of age, so an early peak is not expected among driving group patients. The peak among the elderly patients in the driving group is intriguing and has potential public health implications because this peak corresponds to an age range with a higher frequency of accidents per driver-year. A more vigorous pursuit of a potential cause-effect relationship between syncope while driving and motor vehicle accidents in older patients is warranted.

### Table 2. Prodrome Symptoms, Recovery Symptoms, and Injury

| Symptom                  | Driving (n=381) | Nondriving (n=3496) | P
|--------------------------|----------------|--------------------|---
| Prodrome symptoms        |                |                    |   
| Any prodrome             | 333 (87.4)     | 2982 (85.3)        | 0.27
| Light-headedness         | 229 (60.1)     | 2058 (58.9)        | 0.64
| Nausea                   | 102 (26.8)     | 770 (22.0)         | 0.04
| Diaphoresis              | 81 (21.3)      | 824 (23.6)         | 0.31
| Palpitations             | 73 (19.2)      | 516 (14.8)         | 0.02
| Chest pain               | 57 (15.0)      | 380 (10.9)         | 0.02
| Dyspnea                  | 50 (13.1)      | 310 (8.9)          | 0.007
| Vertigo                  | 46 (12.1)      | 367 (10.5)         | 0.34
| Fatigue                  | 34 (8.9)       | 232 (6.6)          | 0.09
| Abdominal cramps         | 31 (8.1)       | 230 (6.6)          | 0.25
| Vomiting                 | 23 (6.0)       | 207 (5.9)          | 0.93
| Ear ringing              | 9 (2.4)        | 33 (0.9)           | 0.01
| Recovery symptoms        |                |                    |   
| Confusion                | 35 (9.2)       | 309 (8.8)          | 0.82
| Incontinence             | 22 (5.8)       | 248 (7.1)          | 0.34
| Dyspnea                  | 6 (1.6)        | 35 (1.0)           | 0.30
| Injury                   |                |                    |   
| Any injury               | 109 (28.6)     | 829 (23.7)         | 0.03
| Injury requiring hospital care | 65 (17.1) | 528 (15.1)         | 0.31

### Table 3. Causes and Recurrence of Syncope

| Cause of Syncope                   | Driving (n=381) | Nondriving (n=3496) | P
|------------------------------------|----------------|--------------------|---
| Neurally mediated                  | 142 (37.3)     | 1247 (35.7)        | 0.54
| Bradycardia                        | 25 (6.6)       | 221 (6.3)          | 0.86
| Supraventricular tachycardia       | 8 (2.1)        | 71 (2.0)           | 0.93
| Ventricular tachycardia            | 20 (5.2)       | 130 (3.7)          | 0.14
| Structural cardiopulmonary disease | 1 (0.3)        | 9 (0.3)            | 0.99
| Cerebrovascular disease            | 14 (3.7)       | 100 (2.9)          | 0.37
| Carotid sinus hypersensitivity     | 12 (3.1)       | 100 (2.9)          | 0.75
| Orthostatic intolerance            | 18 (4.7)       | 223 (6.4)          | 0.20
| Others                             | 87 (22.8)      | 1044 (29.9)        | 0.004
| Unknown                            | 90 (23.6)      | 622 (17.8)         | 0.005

<table>
<thead>
<tr>
<th>Recurrence of Syncope, n (%)</th>
<th>Driving (n=72)</th>
<th>Nondriving (n=713)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurally mediated</td>
<td>28 (38.9)</td>
<td>243 (34.1)</td>
</tr>
<tr>
<td>Bradycardia</td>
<td>9 (12.5)</td>
<td>50 (7.0)</td>
</tr>
<tr>
<td>Supraventricular tachycardia</td>
<td>3 (4.2)</td>
<td>15 (2.1)</td>
</tr>
<tr>
<td>Ventricular tachycardia</td>
<td>3 (4.2)</td>
<td>21 (2.9)</td>
</tr>
<tr>
<td>Structural cardiopulmonary disease</td>
<td>0 (0)</td>
<td>3 (0.4)</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>6 (8.3)</td>
<td>21 (2.9)</td>
</tr>
<tr>
<td>Carotid sinus hypersensitivity</td>
<td>1 (1.4)</td>
<td>30 (4.2)</td>
</tr>
<tr>
<td>Orthostatic intolerance</td>
<td>7 (9.7)</td>
<td>53 (7.4)</td>
</tr>
<tr>
<td>Others</td>
<td>11 (15.3)</td>
<td>227 (31.8)</td>
</tr>
<tr>
<td>Unknown</td>
<td>15 (20.8)</td>
<td>111 (15.6)</td>
</tr>
</tbody>
</table>

*Total diagnoses for both the driving and nondriving groups exceed 100% because some patients have >1 presumed diagnosis.
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Factor Hazard Ratio 95% CI

Syncope While Driving

Table 4. Predictors Affecting Mortality Among Patients With the most common cause of syncope.4,23,24 Orthostatic stress consistently demonstrated that neurally mediated syncope is while driving.

Figure 3. Survival comparison. Kaplan-Meier curves showing (A) estimated survival of patients who had syncope while driving (driving; n=381) vs the age- and sex-matched Minnesota population (MN) and (B) estimated survival of patients who did (driving; n=381) and did not (nondriving; n=3473) have syncope while driving.

Studies from selected and unselected populations have consistently demonstrated that neurally mediated syncope is the most common cause of syncope.4,23,24 Orthostatic stress has been suggested as a common prerequisite for the triggering of a neurally mediated response.18,20,25 As a result, neurally mediated syncope theoretically would be uncommon in the seated position; therefore, a cardiac (as opposed to neurocardiogenic) cause of syncope is often suspected when syncope occurs while driving. In a small case series study, Li and colleagues8 reported that among 23 patients undergoing tilt-table testing for syncope while driving, 19 patients had a positive tilt-table response consistent with a neurally mediated origin. In the present study, neurally mediated syncope was the presumed cause of syncope in 37.3% of patients when syncope occurred during driving. Cardiac arrhythmias (bradycardia, ventricular tachycardia, or supraventricular arrhythmias) made up 11.8% of causes of syncope while driving. These observations provide strong evidence challenging our traditional views on the potential causes of syncope during driving. These data could further advance our efforts in establishing a more effective syncope evaluation and patient triage pathway.10,26,27

The high prevalence of neurally mediated syncope occurring in the seated position during driving warrants further evaluation of the triggering mechanism. In the absence of orthostatic stress, several mechanisms are plausible. In the passively seated position without muscle tension, venous pooling, especially in the setting of preexisting dehydration or intravascular depletion, could predispose patients to vasovagal syncope.2 The warm environment of a car interior may cause enough cutaneous vasodilatation to make certain patients more susceptible to a syncopeal episode.28 Strong emotional stimulation while driving is another possibility.29 The observation that neurally mediated syncope is common while driving is hypothesis generating. Future studies could investigate the possibility of driving itself being a trigger for syncope.

Our study identified several factors that were more prevalent in patients who had syncope while driving compared with nondriving patients: male sex, any cardiovascular diseases, stroke, and younger age. To the best of our knowledge, no prior study has reported predictors of syncope while driving. In previous studies, clinical history has been a key part of the assessment for cause and prognosis of syncope.18,23,30–32 In our study, some prodrome symptoms were more common in the driving group (nausea, palpitations, chest pain, dyspnea, and ear ringing). However, the clinical utility of the prodrome symptoms and clinical predictors of syncope while driving is most likely limited in sensitivity and specificity because of the small differences between the 2 study groups.

In this study, we included all consecutive patients meeting the inclusion criteria during the study period. We recognize that patients <16 years of age were excluded from the driving group because of the minimum driving age. To assess any potential significant biases caused by age differences between the 2 groups, additional analyses were performed comparing the 2 groups of patients at cutoff ages of 16 and 21 years (data not shown). Minimal differences were noted from the present data that would not have led to different conclusions.

Although many studies have evaluated the prevalence of recurrent syncope in various patient populations,33–36 our study provides the first long-term follow-up information on recurrence of syncope in a large population of patients who had syncope while driving. Of the driving group patients, 72 had recurrence of syncope during follow-up, with 10 of these episodes occurring while driving. Recurrence rates of syncope were not significantly different between the 2 groups of patients. The syncope recurrence rate in the driving group in our study is lower than that in an earlier prospective study of syncope by Kapoor,33 which showed a 61% recurrence rate within 1 year and 82% within 2 years. The recurrence rate of syncope in our study cohort is closer to data from younger patients with vasovagal syncope from recent randomized trials (>30% to 30% to

Table 4. Predictors Affecting Mortality Among Patients With Syncope While Driving

<table>
<thead>
<tr>
<th>Factor</th>
<th>Hazard Ratio</th>
<th>95% CI</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventricular tachycardia</td>
<td>5.12</td>
<td>2.29–11.49</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Atrioventricular block</td>
<td>4.46</td>
<td>2.00–9.96</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>2.63</td>
<td>1.43–4.84</td>
<td>0.002</td>
</tr>
<tr>
<td>History of myocardial infarction</td>
<td>2.35</td>
<td>1.10–5.00</td>
<td>0.03</td>
</tr>
<tr>
<td>Any cardiovascular disease</td>
<td>2.28</td>
<td>1.31–3.97</td>
<td>0.004</td>
</tr>
<tr>
<td>Increasing age</td>
<td>1.07</td>
<td>1.06–1.07</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

CI indicates confidence interval.
A potential limitation of our study is the referral nature of the study population. Although some referral-related biases may be present, the bimodal age distribution of all patients with syncope suggests that the referral bias for patient selection is likely small; the bimodal age distribution has been well recognized in population-based syncope studies. In this study, we included all patients who presented for syncope evaluation during a consecutive period of 3 years. Another limitation is the retrospective study design and confounders associated with such a study method. However, a prospectively designed survey was used during follow-up, and the follow-up was nearly complete. Some recall bias also was likely present during follow-up; however, the long study period allowed critical assessment of recurrent syncope event rates. We also recognize that drivers’ licensing was not determined in the study groups. Some bias could be introduced if licensing rates were not matched. Finally, consequences such as injury to others and motor vehicle accidents were not recorded. Police reports and other possible sources of information were not investigated. Data from this study showed that the need for hospital care was low among driving group patients. We were unable to determine the risk of injury to others.

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Disclosures

None.

References

In a large case-control study of 3877 patients with syncope, 381 patients (9.8%) reported an episode of syncope while driving. A peak of syncope while driving was observed among elderly patients. This has potential public health implications because this event: implementation of a simplified diagnostic algorithm in a multicentre prospective trial: the OESIL 2 study (Osservatorio Epidemiologicodi Sincope nel Lazio). *Eur Heart J.* 2000;21:935–940.


**CLINICAL PERSPECTIVE**

In a large case-control study of 3877 patients with syncope, 381 patients (9.8%) reported an episode of syncope while driving. A peak of syncope while driving was observed among elderly patients. This has potential public health implications because this peak corresponds to an age group with a higher frequency of accidents per driver-year in the general public. The most common cause of syncope while driving was neurally mediated syncope (37.3%). The next most common identifiable causes were cardiac arrhythmias, including bradyarrhythmias, ventricular tachyarrhythmias, and supraventricular tachyarrhythmias (11.8%). Long-term survival among patients who had syncope while driving (driving group) was comparable to that of an age- and sex-matched cohort from the Minnesota population (P=0.15). Among the driving group, syncope recurred in 72 patients, 35 of whom (48.6%) had recurrence >6 months after the initial evaluation. Syncope while driving recurred in 10 patients in the driving group, 7 episodes of which (70%) occurred >12 months after the initial evaluation. The actuarial recurrence of syncope while driving was 0.7% at 6 months and 1.1% at 12 months during follow-up. The causes of syncope, the late recurrences of syncope (during >6 months of follow-up), and the overall low incidence of recurrent syncope while driving provide useful information to supplement current recommendations on driving for these patients.