Syncope Associated With Supraventricular Tachycardia
An Expression of Tachycardia Rate or Vasomotor Response?

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Background. Syncope in patients with supraventricular tachycardia has been suggested to be an ominous finding, predictive of rapid rates during tachycardia.

Methods and Results. To explore the mechanism of syncope during supraventricular tachycardia, tachycardia was induced in the supine position and after passive head-up tilting to 60° in 13 patients with atrioventricular (AV) node reentry, eight patients with AV reentry, and one patient with atrial tachycardia. Tilt testing was also performed in sinus rhythm for 30 minutes (the last 15 minutes with isoproterenol infusion). Mean±SEM age was 38±3 years, and 11 patients had a history of syncope (median number of syncopal episodes, three; range, one to 30). The cycle length of tachycardia when upright was shorter than when supine (297±9 compared with 357±10 msec, p<0.001), and mean blood pressure fell to a greater extent after the onset of tachycardia (fall in mean blood pressure, 53±6 compared with 24±3 mm Hg, p<0.001). Mean blood pressure correlated significantly with tachycardia cycle length when supine (r=0.58, p=0.005) but not when tilted upright (r=0.18, p=0.45). Syncope occurred in seven patients during upright tachycardia. These seven patients had a greater fall in mean blood pressure with upright tachycardia than the 15 patients without syncope (fall in mean blood pressure, 70±4 compared with 45±5 mm Hg, p=0.01), but there was no difference in the tachycardia cycle length (311±10 compared with 290±11 msec, p=0.29). Six of the seven patients with tachycardia-induced syncope also had syncope with tilt testing in sinus rhythm compared with four of the 15 patients without tachycardia-induced syncope (p=0.02).

Conclusions. These data support the view that syncope during supraventricular tachycardia is related to vasomotor factors and does not predict a more rapid tachycardia rate. (Circulation 1992;85:1064–1071)

Key Words • syncope • tachycardia, supraventricular • head-up tilt

Syncope is a relatively frequent symptom accompanying supraventricular tachycardia1–4 and has been suggested to be an important marker of rapid and dangerous tachycardia.3 This hypothesis is not supported by retrospective studies, which did not find an association between induced tachycardia cycle length and a history of syncope.1,2 An alternative mechanism for syncope with supraventricular tachycardia is an abnormal vasomotor response to the hemodynamic stress of tachycardia. This response may be mediated by cardiac mechanoreceptors, activated by diminished left ventricular volume and vigorous ventricular contraction accompanying tachycardia.5–10 Activation of cardiac mechanoreceptors may lead to withdrawal of sympathetic tone, enhanced vagal tone, and hypotension.11 To determine the relative roles of tachycardia rate and vasomotor response in producing syncope with supraventricular tachycardia, 22 patients undergoing electrophysiological study were evaluated. The hemodynamic effects and cycle length of supraventricular tachycardia induced in patients in the supine and upright positions were compared, and the tendency toward neurally mediated syncope was evaluated by passive head-up tilt testing.12,13

Methods

Consecutive patients with supraventricular tachycardia who were undergoing electrophysiological testing were considered for this study. Patients were excluded if they developed atrial fibrillation during the diagnostic study, if sustained regular supraventricular tachycardia could not be induced in the drug-free state, if antiarrhythmic medication was used to terminate tachycardia, if other cardiac disease was present, or if informed consent was withheld.

Electrophysiological Study

Electrophysiological testing was performed after overnight fasting. All patients provided written, informed consent. The electrophysiological protocol has been detailed elsewhere.14 Briefly, one tripolar and two quadripolar catheters were introduced into the right femoral vein and positioned in the high right atrium, right ventricular apex, and His bundle recording posi-
tion, respectively. A quadripolar catheter was introduced into the left subclavian vein and positioned in the coronary sinus. Intracardiac electrograms were recorded simultaneously with surface leads I, II, III, V₁, and V₆ on a Siemn’s mongraph (Sälen, Sweden) at a paper speed of 100 mm/sec. Programmed stimulation was performed at twofold to fourfold diastolic threshold with 2-msec-square wave pulses.

Finger arterial pressure was monitored continuously by the Penaz volume-clamp method¹⁵–¹⁸ using a finger cuff (Ohmeda Monitoring Systems, Englewood, Colo.), except in one patient in whom intra-arterial blood pressure was measured with a femoral artery catheter. The arm was supported with a sling fixed to keep the finger at heart level in the supine and upright positions. Blood pressure was recorded simultaneously with electrograms on the paper recorder. Mean finger arterial pressure measured by the Finapres technique has been shown to differ by 1–10 mmHg from invasively measured brachial artery pressure.¹⁵–¹⁷ Changes in finger arterial pressure closely parallel changes in brachial artery pressure during anesthesia,¹⁵ vasoconstriction,¹⁸ the Valsalva maneuver,¹⁶ and syncope induced by passive tilt testing.¹⁷ Comparison of blood pressure recordings by Finapres and by intra-arterial monitoring (femoral artery catheter) in six patients during five beats of sinus rhythm and the first 50 beats of tachycardia is shown in Figure 1. Mean finger arterial pressure was 4±0.45 mmHg greater than mean femoral artery pressure. The linear regression equation relating mean finger arterial pressure to mean femoral artery pressure was y=0.76x+24 (p<0.001, r=0.95).

**Study Protocol**

After induction and termination of tachycardia in patients in the supine position, patients were tilted upright to 60° on a motorized tilt table with footplate support. Immediately after 60° tilt had been achieved, tachycardia induction was attempted with the same atrial or ventricular stimulation used to induce tachycardia in the supine position. The duration of tilt before initiation of tachycardia was 22±8 seconds (range, 5–120 seconds). During upright tachycardia, blood pressure and heart rate were measured continuously over a 2-minute period. The patient was then returned to the supine position, and tachycardia was terminated. After a 10-minute rest period, the patient was tilted passively to 60° for 15 minutes. If syncope did not occur after 15 minutes, isoproterenol was infused at dosages of from 0.5 to 2 µg/min to decrease the sinus cycle length by 25%. The total duration of the tilt test was 30 minutes. Patients were considered to have syncope when loss of consciousness occurred or when impending loss of consciousness, accompanied by symptoms of presyncope and systolic blood pressure of less than 70 mm Hg for more than 10 seconds, caused the test to be aborted.

**Statistical Analysis**

During tachycardia, sinus cycle length and blood pressure were measured at the nadir of the blood pressure response, unless otherwise stated. Decreases in blood pressure were calculated by subtracting the nadir of blood pressure during tachycardia from blood pressure immediately before tachycardia induction. Continuous variables were compared with paired and unpaired t tests where appropriate. Frequencies were compared with Fisher’s exact test. The relation between tachycardia cycle length and blood pressure was examined by linear regression analysis. Continuous variables were reported as mean±1 SEM.

**Results**

Twenty-five patients were enrolled in the study. Two patients were excluded because tachycardia could not be induced in the upright position, and one patient was excluded because syncope occurred before tachycardia could be induced in the upright position. The study group comprised the remaining 22 patients (mean age, 38±3 years). Eight patients had atrioventricular (AV) reentry, 13 patients had AV node reentry, and one patient had atrial tachycardia inducible at the electrophysiological study. Eleven patients had a history of syncope. In eight patients, syncope occurred in association with palpitations; in two patients, syncope was unassociated with palpitations; and in one patient,
TABLE 1. Clinical Features and Results of Electrophysiological Testing in 22 Study Patients

<table>
<thead>
<tr>
<th>Patient</th>
<th>History of syncope (no. of episodes)</th>
<th>Tachycardia induced</th>
<th>Supine cycle length (msec)</th>
<th>Decrease in mean blood pressure supine (mm Hg)</th>
<th>Upright cycle length (msec)</th>
<th>Decrease in mean blood pressure upright (mm Hg)</th>
<th>Syncope with upright tachycardia (sec)</th>
<th>Syncope with tilt in sinus rhythm (min)</th>
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AVNRT, atrioventricular node reentrant tachycardia; AVRT, atrioventricular reentrant tachycardia; AT, atrial tachycardia.

The median number of syncopal episodes was three (range, one to 30) (Table 1). The estimated frequency of episodes of sustained palpitations per month ranged from 0.1 to 30 (median, four).

In the supine position, tachycardia cycle length was 357±10 msec. Systolic blood pressure decreased by 47±5 mm Hg, and mean blood pressure decreased by 24±3 mm Hg. Mean blood pressure before tachycardia induction was 102±2 mm Hg; at the nadir of the blood pressure response, 78±4 mm Hg; and after 60 seconds of tachycardia, 93±3 mm Hg. Invariably, the lowest pressures were found during the first 10 seconds of tachycardia. By 60 seconds, mean blood pressure had returned to within 10 mm Hg of the value before tachycardia in all except three patients. Tachycardia cycle length was slightly shorter after 60 seconds of tachycardia than at the nadir of the blood pressure response (350±12 compared with 357±10 msec, p=0.06). No patient experienced syncope with tachycardia in the supine position. There was a significant correlation between tachycardia cycle length and the nadir of mean blood pressure (r=0.58, p<0.001; Figure 2). This relation was present whether tachycardia cycle length was measured at the nadir of the blood pressure response or at 60 seconds (r=0.7, p<0.005).

When tachycardia was induced in the upright position, cycle length was shorter (297±9 versus 357±10 msec, p<0.001) and systolic and mean blood pressures declined to a greater extent than during supine tachycardia (decrease in systolic and mean blood pressures, 85±6 and 53±5 mm Hg, respectively; p<0.001 compared with supine tachycardia). Mean blood pressure before tachycardia induction was 110±3 mm Hg; at the nadir of the blood pressure response, 55±4 mm Hg; and at recovery (patients with syncope excluded), 99±3 mm Hg. The cycle length of tachycardia was shorter after 60 seconds than at the time of blood pressure nadir (278±8 compared with 294±9 msec, p=0.002); patients
with syncope within the first 60 seconds of tachycardia were excluded. The lowest blood pressure occurred within the first 10 seconds of tachycardia in all except three patients. These latter three patients had a secondary decline in their blood pressure after initial stabilization with syncope occurring 72–120 seconds after the onset of tachycardia (Figure 3). In these three patients, blood pressure stabilized at a mean pressure of 72±12 mm Hg before decreasing to a nadir of 46±1 mm Hg at the time of syncope. One additional patient experienced syncope 60 seconds after tachycardia induction with a secondary decline in mean blood pressure to 60 mm Hg, but at the time the test was aborted, pressure had not declined below the initial nadir of 55 mm Hg. In an additional three patients, syncope occurred at the onset of tachycardia at the time of the initial decrease in blood pressure (Figure 4). In contrast to supine tachycardia, there was no significant correlation between upright tachycardia cycle length and the nadir of mean blood pressure during tachycardia (r=0.18, p=0.45; Figure 5A). When patients experiencing syncope during upright tachycardia were excluded from this analysis, the correlation between mean blood pressure and tachycardia cycle length improved (r=0.49, p=0.06; Figure 5B).

Comparison of the seven patients with and the 15 patients without syncope during tachycardia is shown in Table 2. The only significant differences occurred in the extent of blood pressure decrease during tachycardia (increase in mean blood pressure, 70±4 compared with 45±5 mm Hg; p=0.01) and in the frequency with which syncope occurred during passive tilt testing in sinus rhythm. The cycle length of tachycardia, in fact, tended to be longer in patients with syncope (311±10 compared with 290±11 msec; p=0.27). Thus, the ratio of mean blood pressure to tachycardia cycle length was considerably lower in patients with syncope (0.14±0.01 compared with 0.21±0.02, p=0.02).

Ten patients experienced syncope with passive tilt testing in sinus rhythm (Figure 5). At the time of syncope, mean blood pressure was 49±3 mm Hg, and mean sinus cycle length was 1,360±521 msec. Syncope occurred in six patients before isoproterenol infusion and in four patients after isoproterenol infusion (all at an infusion rate of 1 μg/min). Six of the seven patients with syncope during tachycardia also experienced syncope with passive tilt testing in sinus rhythm (p=0.02). Five of the 10 patients with a positive tilt test had never experienced clinical syncope. There was no association between the occurrence of syncope with either tachycardia or passive tilt testing and a previous history of syncope (Table 1). This finding was unaltered when only the nine patients in whom syncope occurred in association with palpitations were considered to have a history of syncope.

Discussion

The results of the present study demonstrate that syncope during induced upright supraventricular tachycardia is associated with a propensity toward vasodepressor syncope and is not directly related to tachycardia cycle length. These data support previous retrospective studies that failed to find an association between a history of syncope and induced tachycardia cycle length.1,2 Therefore, syncope with upright supraventricular tachycardia may indicate a particular hemodynamic response to the stress of tachycardia and does not necessarily imply a more malignant or rapid tachycardia.

Vasovagal syncope has been postulated to occur because of inappropriate stimulation of left ventricular stretch receptors by hypovolemia and vigorous ventricular contraction.11,13,19,20 An increase in sympathetic tone may precipitate this reflex and is the theoretical justification for the use of an isoproterenol infusion during passive tilt testing.11,21–23 Supraventricular tachycardia reduces left ventricular filling through abbreviation of the diastolic filling time and alteration in the timing of atrial and ventricular contraction.7,9,24,25 An increase in sympathetic tone occurs at the onset of tachycardia.5,6,8,9 These changes may be magnified when supraventricular tachycardia occurs in the upright position, which in itself reduces left ventricular volume and increases sympathetic tone.26–29 Consequently, there may be activation of cardiac mechanoreceptors during tachycardia as a result of diminished ventricular volume and vigorous ventricular contraction. Activation of these receptors could lead to an inadequate hemodynamic response to tachycardia and syncope by a mechanism similar to that postulated for vasovagal syncope.11,13,20,22

The association between syncope with passive tilt testing and tachycardia-induced syncope found in the present study suggests that some patients may be predisposed to this response, in both sinus rhythm and tachycardia. The tendency toward a longer cycle length in the face of marked hypotension during tachycardia-induced syncope also suggests vagal influence and diminished sympathetic tone at the time of syncope. Vagal stimulation at the time of syncope appeared to be the mechanism of tachycardia termination in one patient (Figure 3). Conceivably, vagal stimulation by this mechanism could facilitate degeneration of reciprocating tachycardia into atrial fibrillation. Onset of atrial fibrillation has been observed in the setting of vasovagal syncope,30 and vagal stimulation is a well-recognized precipitant of atrial fibrillation.31,32

In the present study, syncope during tachycardia occurred either at the onset of tachycardia or 1–2 minutes later, after initial stabilization of blood pressure. In patients in whom a secondary decline in blood pressure led to syncope, activation of cardiac mechanoreceptors may be particularly important in allowing an inadequate hemodynamic response to tachycardia. This mechanism may be less important in patients who experience syncope at the onset of tachycardia. It is important to note, however, that the three patients with syncope at the onset of tachycardia had relatively long tachycardia cycle lengths (330, 340, and 300 msec) and that all experienced syncope with passive tilt testing, emphasizing the role of hemodynamic factors in the occurrence of syncope.

There was no association between a history of syncope and syncope during induced upright tachycardia. There are several possible reasons for this finding. Syncope during clinical tachycardia is an infrequent event for most patients, with syncope usually occurring only two or three times, despite multiple episodes of tachycardia. This suggests that many other factors may influence the occurrence of syncope; chief among these may be the patient’s behavior at the time of tachycardia. Immediate cessation of activity with assumption of the recumbent position may prevent syncope, even in pa-
patients predisposed to tachycardia-induced syncope. On the other hand, continued upright activity during tachycardia could eventually result in syncope in any patient with supraventricular tachycardia. Other factors that may be important include the use of antiarrhythmic medication, intravascular volume, and the autonomic milieu at the time of tachycardia. These variables cannot be exactly reproduced at electrophysiological testing. Finally, the clinical arrhythmia at the time of syncope is usually unknown and may conceivably be different to the arrhythmias induced at electrophysiological study.33

A history of syncope was found more frequently in study patients than in previous reports.1–3 This probably reflects the prospective nature of the present study. Previous retrospective studies may have failed to determine all episodes of syncope because infrequent or remote syncopal episodes were not recorded on the hospital record. In addition, there was a high frequency of tilt-induced syncope (45%). Five of the 10 patients with a positive tilt test had never experienced syncope. These results caution against uncritically extrapolating the results of tilt testing to clinical syncope. Tilting, in the presence of fluid deprivation without active contraction of leg musculature, can result in quite substantial hemodynamic stress that may not be reproduced in normal daily life.12,13,34 The high prevalence of positive results may also reflect the use of tilt testing at the time of electrophysiological testing. Syncope during tilt testing with a parachute harness has been shown to occur more frequently when intravascular instrumentation is performed concurrently.34

This study has limitations. First, the mechanism of spontaneous syncope could not be determined because of technical limitations in monitoring and because of the low frequency of spontaneous syncopal episodes. Second, finger arterial pressure may not always parallel changes in central aortic pressure, particularly at times of marked fluctuation in sympathetic tone. Because the correlation between finger and femoral arterial pressure during tachycardia was excellent, it is unlikely that central aortic pressure varied substantially from the recorded finger arterial pressure. Finally, atrial pressures and atrial natriuretic factor, which were not measured, may have influenced the response to upright tachycardia and passive tilt testing.35,36 Despite these limitations, there was a striking relation between syncope with tilt testing in sinus rhythm and syncope with tachycardia induced in the upright position. These data support the importance of vasomotor factors rather
than tachycardia cycle length in the occurrence of tachycardia-induced syncope.

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