Sustained Symptomatic Sinus Node Reentrant Tachycardia: Incidence, Clinical Significance, Electrophysiologic Observations and the Effects of Antiarrhythmic Agents

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The clinical, electrocardiographic and electrophysiologic determinants and effects of antiarrhythmic agents on sustained sinus node reentrant tachycardia remain poorly defined. Of 65 consecutive men undergoing electrophysiologic studies for symptomatic paroxysmal supraventricular tachycardia over a 4 year period, 11 (16.9%), who ranged in age from 39 to 76 years, demonstrated sustained sinus node reentrant tachycardia. On the surface electrocardiogram, before electrophysiologic studies, the following diagnoses were considered in the 11 patients: 1) sinus node reentrant tachycardia on the basis of an RP'/P'R ratio of greater than 1 and P wave configuration similar to that of sinus P waves (7 patients); 2) atrioventricular (AV) nodal reentrant tachycardia on the basis of an RP'/P'R ratio of less than 1 (3 patients); and 3) paroxysmal atrial tachycardia with AV block (1 patient). All 11 patients had a history of recurrent palpitation, 4 had syncope, 2 had dizzy spells and 9 had organic heart disease.

Sustained sinus node reentrant tachycardia could be reproducibly induced in all 11 patients during atrial pacing or premature atrial stimulation, or both, over a wide echo zone. The tachycardia could be terminated by carotid sinus massage, atrial pacing and premature atrial stimulation. Characteristics of tachycardia included: 1) high-low activation sequence; 2) cycle lengths of 250 to 590 ms with wide fluctuations of 20 to 180 ms in individual patients; 3) RP'/P'R ratio of greater than 1 in 8 (73%) of the 11 patients and a ratio of less than 1 in 3 (27%). Induction of sustained sinus node reentrant tachycardia was prevented by intravenous ouabain (0.01 mg/kg body weight) in two of two patients, by intravenous verapamil (10 mg) in two of two patients and by intravenous amiodarone (5 mg/kg body weight) in four of four patients. In contrast, intravenous propranolol (0.1 mg/kg body weight) did not affect induction of sustained sinus node reentrant tachycardia in two of two patients.

It is concluded that 1) sustained sinus node reentrant tachycardia, seen in 16.9% of the study patients with paroxysmal supraventricular tachycardia, is not as benign as previously believed; 2) it is frequently associated with organic heart disease; 3) it demonstrates wide variations in cycle length, unlike other forms of paroxysmal supraventricular tachycardia; 4) it can masquerade as AV nodal reentrant tachycardia and paroxysmal atrial tachycardia with AV block on the surface electrocardiogram in 36% of patients; and 5) it is responsive to intravenous administration of ouabain, verapamil or amiodarone.

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Electrophysiologic evidence of sustained sinus node reentrant tachycardia. The purpose of this paper is to discuss 1) the clinical, electrocardiographic and electrophysiologic observations in patients with sustained sinus node reentrant tachycardia, and 2) the effects of various antiarrhythmic agents on sustained sinus node reentrant tachycardia.

Methods

Study patients. Electrophysiologic studies were performed in 65 men with paroxysmal supraventricular tachycardia in the postabsorptive nonsedated state after explaining the nature of the procedure and obtaining an informed signed consent. All cardioactive medications were withheld for at least 4 days before the study. None of the patients had acute myocardial infarction or ischemia and none had electrolyte or metabolic disturbances.

Electrophysiologic studies. With the use of the percutaneous Seldinger technique, three quadripolar catheters (USCI, no. 6) with 10 mm interelectrode distance were introduced percutaneously through femoral veins and positioned in the high right atrium, at the level of the tricuspid valve for recording His bundle activity and in the right ventricular apex. When necessary, one of the catheters was positioned in the mid right atrium, the low right atrium or coronary sinus to determine atrial activation sequence. The proximal poles of the catheters in the high right atrium and right ventricular apex were utilized for recording electrograms; the distal poles were used for stimulation. However, when sinus node reentry was induced, the distal pole of the catheter positioned vertically in the high right atrium (through the femoral vein) was utilized for recording the electrogram from the region of the sinus node and the distal pole of the second catheter was utilized for pacing the region of the high right atrium.

All patients underwent complete anterograde and retrograde conduction studies that included: 1) atrial pacing up to cycle lengths between 300 and 280 ms; 2) programmed atrial stimulation (S1S2 and S1S2S3) at one or more cycle lengths; 3) incremental ventricular pacing up to cycle lengths of 250 ms; and 4) programmed premature ventricular stimulation at one or more cycle lengths. Atrial and ventricular stimulation studies were performed at twice diastolic threshold with stimuli 2 ms in duration. Two to three electrocardiographic leads (usually I, II and V1), intracardiac electrograms at filter frequencies of 30 to 500 Hz and time lines generated at 40,200 and 1,000 ms were displayed on a multichannel oscilloscope (Varian Associates, VR-12 multichannel recorder) and recorded on thermal paper at paper speeds of 50 to 100 mm/s.

Definition of terms. Sinus node reentrant tachycardia was defined if it satisfied the following criteria (2,6). 1) The sequence of atrial activation was from high right atrium to low right atrium in the region of the atioventricular (AV) junction and similar to that of sinus beats; 2) the polarity and configuration of the P waves were similar to if not identical to the configuration of sinus P waves; 3) the tachycardia could be induced over a zone of S1S2 (that is, atrial pacing cycle length), S2S3, or S3S4 intervals; 4) the tachycardia could be terminated by appropriately timed atrial premature stimuli; 5) the induction of tachycardia or echo beats, or both, was independent of AV nodal conduction delays.

Sustained sinus node reentrant tachycardia. The tachycardia was considered to be sustained if it persisted for at least 1 minute. Patients in whom only sinus echo beats were induced are not the subject of this report.

Echo zone. This was defined as the range of coupling intervals (S1S2 or S2S3) that induced sinus node echo beats or sustained node reentrant tachycardia, or both.

Statistical analysis was performed by utilizing the Student's t test. All values represent the mean ± standard deviation.

Drug studies. In 8 of 11 patients in whom sustained sinus node reentrant tachycardia was induced, a total of 10 patients underwent complete anterograde and retrograde conduction studies that included: 1) atrial pacing up to cycle lengths between 300 and 280 ms; 2) programmed atrial stimulation (S1S2 and S1S2S3) at one or more cycle lengths; 3) incremental ventricular pacing up to cycle lengths of 250 ms; and 4) programmed premature ventricular stimulation at one or more cycle lengths. Atrial and ventricular stimulation studies were performed at twice diastolic threshold with stimuli 2 ms in duration. Two to three electrocardiographic leads (usually I, II and V1), intracardiac electrograms at filter frequencies of 30 to 500 Hz and time lines generated at 40,200 and 1,000 ms were displayed on a multichannel oscilloscope (Varian Associates, VR-12 multichannel recorder) and recorded on thermal paper at paper speeds of 50 to 100 mm/s.

Table 1. Clinical Features of 11 Patients

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (yr)</th>
<th>Symptoms</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>47</td>
<td>Palpitation</td>
<td>ASHD</td>
</tr>
<tr>
<td>2</td>
<td>77</td>
<td>Palpitation, syncope</td>
<td>ASHD, hypertension</td>
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<tr>
<td>3</td>
<td>60</td>
<td>Syncope</td>
<td>None</td>
</tr>
<tr>
<td>4</td>
<td>56</td>
<td>Palpitation</td>
<td>ASHD</td>
</tr>
<tr>
<td>5</td>
<td>55</td>
<td>Palpitation, dizzy spells</td>
<td>ASHD</td>
</tr>
<tr>
<td>6</td>
<td>61</td>
<td>Palpitation, dizzy spells</td>
<td>None</td>
</tr>
<tr>
<td>7</td>
<td>59</td>
<td>Palpitation</td>
<td>HCVD</td>
</tr>
<tr>
<td>8</td>
<td>76</td>
<td>Palpitation</td>
<td>ASHD</td>
</tr>
<tr>
<td>9</td>
<td>39</td>
<td>Palpitation</td>
<td>ASHD</td>
</tr>
<tr>
<td>10</td>
<td>54</td>
<td>Palpitation, syncope/dizzy spells</td>
<td>ASHD</td>
</tr>
<tr>
<td>11</td>
<td>67</td>
<td>Palpitation, syncope</td>
<td>ASHD</td>
</tr>
</tbody>
</table>

ASHD = atherosclerotic heart disease; HCVD = hypertensive cardiovascular disease.
drug studies were performed after the administration of the following drugs: 1) intravenous propranolol, 0.1 mg/kg body weight (2 patients); 2) intravenous ouabain, 0.01 mg/kg body weight (2 patients); 3) intravenous verapamil, 10 mg (2 patients); and 4) intravenous amiodarone, 5 mg/kg body weight as a slow continuous infusion over 20 minutes (4 patients).

Results

Clinical Observations

Of the 65 patients with symptomatic paroxysmal supraventricular tachycardia, AV nodal reentrant tachycardia was induced in 38 (58%), reentrant supraventricular tachycardia associated with the Wolff-Parkinson-White syndrome in 8 patients (12%), reentrant supraventricular tachycardia utilizing a concealed bypass tract in 7 patients (11%), intraatrial reentry in 1 patient (1.5%) and sustained sinus node reentrant tachycardia in 11 patients (17%).

Clinical features (Table 1). The 11 patients were all male and ranged in age from 39 to 76 years (mean 60 ± 11). All 11 patients had a history of recurrent palpitation and 6 (55%) had dizzy spells or syncope, or both. Nine of the 11 patients (82%) had atherosclerotic heart disease or hypertensive heart disease, or both: 7 (64%) had a prior myocardial infarction of whom 5 had prior inferior wall infarction. The site of infarction could not be determined in one patient who had left bundle branch block. All 11 patients had spontaneous supraventricular tachycardia demonstrated on a 12 lead electrocardiogram or 24 hour ambulatory Holter recording. None of the patients had sick sinus syndrome.

Electrocardiographic diagnosis. On the basis of the surface electrocardiogram or Holter recording, seven patients (65%) were diagnosed as having sinus node reentrant tachycardia, three (27%) as having AV nodal reentrant tachycardia and one (9%) as having atrial tachycardia with AV block (serum digoxin level 0.5 ng/ml). Electrocardiograms from Patients 7 and 8 during one of the episodes of spontaneous tachycardia are shown in Figures 1 and 2, respectively. In both tracings during tachycardia, the P waves were not clearly discernible. It was argued by some observers that there were P waves after the QRS complex (Fig. 1, bottom strip), but the polarity and shape of the P waves were not clearly evident. Thus, the diagnosis of AV nodal reentrant tachycardia was entertained in these patients.

Electrophysiologic Observations

Induction of tachycardia (Table 2). Sustained sinus node reentrant tachycardia could be induced in all 11 patients during atrial pacing or premature atrial stimulation, or both, over a wide echo zone that ranged from 20 to 280 ms (mean 63 ± 78). The cycle length of tachycardia ranged from 250 to 590 ms in individual patients and approximated the observed rate of tachycardia as recorded on the surface electrocardiogram or 24 hour ambulatory Holter recording. During induced tachycardia, there was a marked variation in
cycle length of 20 to 180 ms in individual patients. The shortest cycle length of tachycardia was significantly lower (that is, the rate was significantly faster) in patients with dizzy spells or syncope, or both, than in patients without these symptoms (368 ± 61 versus 458 ± 60 ms, respectively, p < 0.05). In eight patients, the RP'/P'R ratio was greater than 1, whereas in 3 patients it was less than 1.

Figure 3 is an example from Patient 7 showing induction of sustained sinus node reentrant tachycardia during atrial pacing (panel A) and premature atrial stimulation with a cycle length of 340 to 480 ms after atrial pacing at a cycle length of 340 ms. Panel B shows induction of sustained sinus node reentrant tachycardia during atrial premature stimulation at an S1S2 interval of 320 ms. During tachycardia: 1) a variation in cycle length, and 2) a spontaneous and progressive increase in Ae-H interval (panels B and C) with the sinus echo beat (Ae) occurring later and later in relation to the next QRS complex simulating AV nodal reentrant tachycardia.
Figure 4. Patient II. Sinus node reentrant tachycardia simulating atrial tachycardia with AV block. Abbreviations and format as in Figure 3. Panel A shows a spontaneous tachycardia with AV block. Note the upright P waves in leads I and II and the high-low atrial activation sequence (vertical arrows). Note that the 2:1 and 3:1 AV block during sinus node reentrant tachycardia is in the AV node (dotted lines). Panel B shows induction of the same tachycardia during programmed premature stimulation at an \( S_1S_2 \) interval of 500 ms similar to the coupling interval of the spontaneous first echo beat in panel A. Panel C shows the termination of tachycardia by carotid sinus massage (CSM) after lengthening of the tachycardia cycle.
Sinus Nodereentrant Tachycardia

Cardia, there was a spontaneous and progressive increase in Ae-H interval (panels B and C) with the sinus echo beat (Ae) occurring later and later in relation to the next QRS complex until it merged in the previous QRS complex with a change in RP'/P'R ratio to less than 1, simulating AV nodal reentrant tachycardia as occurred during a spontaneous episode of tachycardia (Fig. 1). However, intracardiac recordings during induced tachycardia show a high-low activation sequence.

Figure 4 shows an example of spontaneously occurring tachycardia in Patient 11 which was diagnosed as atrial tachycardia with AV block on the surface electrocardiogram, but which was established to be sustained sinus node reentrant tachycardia with 2:1 and 3:1 AV block. Figure 4B shows induction of the corresponding tachycardia during premature atrial stimulation which was promptly terminated by carotid sinus massage (panel C).

In all 11 patients, sustained sinus node reentrant tachycardia could be terminated by carotid sinus massage (Fig. 4C), atrial pacing (Fig. 5) and premature atrial stimulation. None of the patients had increased vagal tone or AV nodal refractoriness and none had intraatrial conduction delays.

Induction of other tachyarrhythmias. Other forms of sustained tachyarrhythmias were not induced in any of the 11 patients. However, single AV nodal echo beats were induced in two patients, single bundle branch reentrant beats in three patients and one to three repetitive ventricular responses due to intraventricular reentry in three patients, all in response to programmed ventricular stimulation. Sustained or nonsustained AV nodal reentrant tachycardia or ventricular tachycardia was not induced in any of the patients. Similarly, atrial flutter or atrial fibrillation, or both, was not induced during programmed atrial stimulation.

Effects of drugs on sustained sinus node reentrant tachycardia (Table 3). Propranolol. Intravenous propranolol was administered to two patients (Cases 1 and 7). After the injection, sustained tachycardia could be induced in both patients. In Patient 1, intravenous propranolol increased the echo zone, whereas in Patient 7, it did not appreciably affect the echo zone. Intravenous propranolol did not appreciably affect the cycle length of tachycardia in either patient, although in Patient 7, there was a 2:1 AV block resulting in slowing of the ventricular response.

Ouabain. In these two patients (Cases 1 and 7), one catheter was left in the high right atrium and studies were repeated the following day. After control electrophysiologic studies, intravenous ouabain (0.01 mg/kg) was administered and stimulation studies were repeated to reinstitute the tachycardia. After the injection, sustained tachycardia could no longer be induced; at most, single echo beats were initiated. In both patients, ouabain increased the echo zone (Table 3).

Figure 6 is a representative example from Patient 1 demonstrating induction of sustained sinus node reentrant tachy-
Table 3. Effect of Antiarrhythmic Agents in 10 Drug Studies

<table>
<thead>
<tr>
<th>Case</th>
<th>Drug Study</th>
<th>Sust-T</th>
<th>Echoes</th>
<th>Echo Zone (ms)</th>
<th>ERP of Atrium (ms)</th>
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<tbody>
<tr>
<td>1</td>
<td>1 (prop)</td>
<td>C</td>
<td>+</td>
<td>80</td>
<td>300</td>
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<tr>
<td>7</td>
<td>2 (prop)</td>
<td>C</td>
<td>+</td>
<td>150</td>
<td>260</td>
</tr>
<tr>
<td>3</td>
<td>3 (ouab)</td>
<td>C</td>
<td>+</td>
<td>20</td>
<td>290</td>
</tr>
<tr>
<td>1</td>
<td>3 (ouab)</td>
<td>D</td>
<td>+</td>
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<td>280</td>
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<tr>
<td>7</td>
<td>4 (ouab)</td>
<td>C</td>
<td>+</td>
<td>60</td>
<td>290</td>
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<tr>
<td>4</td>
<td>7 (amio)</td>
<td>C</td>
<td>+</td>
<td>25</td>
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<td>+</td>
<td>190</td>
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<td>+</td>
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<td>+</td>
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<td>+</td>
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<td></td>
<td></td>
<td>D</td>
<td>+</td>
<td>50</td>
<td>300</td>
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</table>

amio = amiodarone; C = control; D = drug; ERP = effective refractory period; ouab = ouabain; prop = propranolol; Sust-T = sustained tachycardia; verap = verapamil; + = present; - = absent.

cardia during the control study (panel A), after atrial pacing at a cycle length of 440 ms after the administration of propranolol (panel B) and after ouabain (panel C). Sustained sinus node reentrant tachycardia could be induced after propranolol, but not after ouabain.

Verapamil. This drug was administered to two patients (Cases 9 and 10). In both patients, the drug prevented the induction of sustained sinus node reentrant tachycardia or sinus echo beats, or both. Figure 7 is a representative example from Patient 9. During control studies, sustained sinus node reentrant tachycardia is induced during premature atrial stimulation ($S_1S_2$) at $S_1S_2$ and $S_1S_3$ intervals of 400 ms, respectively (panel A). The cycle length of tachycardia ranged from 470 to 490 ms. After verapamil (panel B), tachycardia could not be initiated at similar coupling interval.

Amiodarone. This drug resulted in the inability to initiate sustained tachycardia in all four patients (Cases 4, 5, 6 and 11), although in all four patients, two to eight echo beats could be induced. In two of the four patients, the echo zone decreased (Table 3). Figure 8 is an example from Patient 6. Panel A shows the induction of sustained sinus node reentrant tachycardia after premature atrial stimulation at an $S_1S_2$ interval of 580 ms, and panel B shows induction of sustained sinus node reentrant tachycardia at a coupling interval of 340 ms during control studies. Thus, during control studies, the echo zone in this patient was 240 ms. Note also that at a coupling interval of 340 ms (panel B), sustained sinus node reentrant tachycardia was initiated when $A_2$ blocked within the AV node. Panel C shows the inability to initiate tachycardia after intravenous amiodarone at an $S_1S_2$ interval of 340 ms, similar to the control studies (panel B).

Discussion

Previous reports. Since the original suggestion of Barker et al. (12) in 1943 of the possibility of reentry in the sinus node, Han et al. (13) in 1968 carried out experiments in the isolated rabbit heart to prove the concept of sinus node reentry. Utilizing a single microelectrode technique, these investigators attempted to follow the pathway of the activation wave front within and around the sinus node after induction of an atrial premature beat. They demonstrated functional dissociation within the sinus node and strongly suggested the occurrence of a reentrant phenomenon in the sinus node. However, it was Allessie and Bonke (14), utilizing multiple microelectrode recordings of sinus cells in isolated rabbit hearts, who demonstrated that the mechanism of the sinus echo after stimulation was reentry.

In the last several years, a large number of clinical supraventricular tachycardias with a high-low atrial activation...
Figure 6. Patient 1. Induction of sinus node reentrant tachycardia during the control period and after propranolol (Inderal) and ouabain. Abbreviations and format as in Figure 3. In panel A, the intracardiac recordings are obtained at filter settings of 30 to 500 Hz and in panels B and C at 0.1 to 50 Hz. In panel A, after atrial pacing (arrows) at a cycle length of 400 ms, sustained sinus node reentrant tachycardia is induced at a cycle length of 480 to 500 ms. Note the high-low atrial activation sequence (dotted line). After propranolol (panel B), tachycardia can still be induced after atrial pacing at a cycle length of 400 ms. Note that the cycle length of tachycardia ranges from 480 to 540 ms and the atrial activation sequence is from high to low. In panel C (after ouabain), tachycardia can no longer be initiated. LRA = low right atrium.

sequence were attributed to reentry within the sinus node. Despite those reports, little is known of the incidence and clinical significance of sustained symptomatic sinus node reentrant tachycardia in patients with paroxysmal supraventricular tachycardia. In fact, it is believed that sustained sinus node reentrant tachycardia is a rare entity and that it plays an insignificant role in the genesis of paroxysmal reentrant supraventricular tachycardia in human beings. Thus, Wellens (10) reported an incidence rate of 1.8% in 379 patients studied by programmed electrical stimulation. However, his findings are not comparable with those in our group of patients since he included a large number of patients with atrial flutter and ventricular tachycardia. Wu et al. (15) reported an incidence rate of 10% in 72 patients with paroxysmal supraventricular tachycardia in whom the mechanism of tachycardia was delineated by programmed stimulation.

Path of sinus node reentry. Reentry within the sinus node is the most plausible mechanism of the spontaneous and induced tachycardia in our 11 patients. As in all previous clinical studies of sinus node reentry, the exact site of reentry, whether in the sinus node itself, the perinodal zone or the atrial tissue adjacent to the sinus node, cannot be stated for certain because current techniques do not permit the delineation of the components of the reentrant circuit. Furthermore, atrial electrical events in the region of the sinus node may not reflect sinus node events. Recently, this was clearly demonstrated by Gomes et al. (16,17) by recording sinus node electrograms in human patients. What may be inferred, however, from this and previous clinical studies, is that the region of reentry is in the area of the sinus node. The latter is suggested by the observation of a normal high to low atrial activation sequence and P waves similar if not identical to sinus P waves. Slight variation in the configuration of P waves during tachycardia may be related to different exit sites from within the sinus node, which may not be the same as those during normal sinus rhythm.

Symptoms. Of significant clinical importance is our observation that sustained sinus node reentrant tachycardia is not as rare or as benign as previously believed. This is suggested by the observation that 1) sustained sinus node
Figure 7. Patient 9. Induction of sinus node reentrant tachycardia during the control period and after verapamil. Format as in Figure 4. Panel A shows induction of sinus node reentrant tachycardia after premature atrial stimulation at an S₁S₂ and S₂S₃ interval of 400 ms (arrows). Note the high-low atrial activation sequence. Panel B shows inability to initiate the tachycardia after verapamil at coupling intervals similar to those in panel A. A₁A₂ = basic atrial drive; A₃A₄ = premature atrial depolarization; S₁ = stimulus artifact, basic drive; S₂ and S₃ = premature stimuli.
Figure 8. Patient 8. Induction of sinus node reentrant tachycardia during the control studies (panels A and B) and after amiodarone (panel C). Format as in Figure 4. Panel A shows induction of sinus node reentrant tachycardia with a cycle length of 580 ms. Note the high-low atrial activation sequence. In panel B, the tachycardia is induced when A₂ blocks in the AV node at an S₁S₂ interval of 340 ms. In panel C, after amiodarone tachycardia cannot be induced at an S₁S₂ interval of 340 ms similar to that in panel B. A₁ = basic atrial drive; A₂ = premature atrial depolarization; Ae = sinus echo beat; HRA₂ = distal poles of the high right atrial recording; HRA₁ = proximal poles of the high right atrial recording; S₁ = stimulus artifact, basic drive; S₂ = premature stimulus.

Reentrant tachycardia was the mechanism of reentrant supraventricular tachycardia in 11 (16.9%) of our 65 consecutive male patients studied by programmed electrical stimulation, and 2) all 11 patients were symptomatic with a history of palpitation, dizzy spells or syncope. Although it is unclear why our incidence of sustained sinus node reentrant tachycardia was appreciably higher than that reported previously, it may in part be related to the solely male group of patients of higher age and with a high incidence of atherosclerotic heart disease seen in our institution. Also the higher incidence of dizzy spells or syncope, or both, in our group of patients, in contrast to that reported by previous investigators, is probably related to the faster heart rates during tachycardia in 6 of the 11 patients and the presence of organic heart disease.

Underlying heart disease. The findings in this study also indicate that the majority of patients with sustained sinus node reentrant tachycardia have organic heart disease and variable tachycardia rates unlike patients with AV nodal reentrant tachycardia and those utilizing concealed bypass tracts in the reentrant process. These findings are in agreement with the observations of previous investigators (9,10,15). Of additional interest are our observations that the majority of the patients with previous myocardial infarction had had inferior wall infarction. It is possible that in these patients, ischemia of the sinus node or atrial myocardium adjacent to the sinus node may have provided the anatomic substrate for sinus node reentrant tachycardia. Because coronary arteriography was not performed in these patients, it is not possible to speculate on the location of the coronary artery lesions in relation to the sinus node artery.

Mechanism of tachycardia and diagnosis. The relation of the P' wave to the QRS complex on the surface electrocardiogram has been utilized in the diagnosis of the mechanism of paroxysmal reentrant tachycardia. Thus, in the majority of patients with AV nodal reentrant tachycardia, the P' wave is negative in leads II, III and aVF and occurs within the QRS complex or the early part of the ST segment. In patients with a concealed bypass tract, the P' wave follows the QRS complex, and in sinus node reentrant tachycardia, the P' wave is upright in leads II, III and aVF and precedes the QRS complex. The findings in this study indicate that in the majority of patients with sinus node reentrant tachycardia, the P' wave precedes the QRS complex, resulting in an RP'/P'R ratio of greater than 1. However, in 27% of the patients, the P' wave occurred in the ST segment of the QRS complex, resulting in an RP'/P'R ratio of less than 1. The occurrence of P' waves within the QRS complex in sinus node reentrant tachycardia can masquerade as AV nodal reentrant tachycardia on the surface electrocardiogram. Furthermore, when P' waves occur in the early part of the ST segment, it may be difficult to decipher their exact configuration and polarity. This observation in three of our patients was related to prolongation of AV nodal conduction time during tachycardia. Thus, in sinus node reentrant tachycardia, the longer the AV nodal conduction time during tachycardia, the greater the possibility of the P' wave occurring within the QRS complex or the ST segment.

Unlike AV nodal reentrant tachycardia and tachycardia incorporating bypass tracts in the reentrant process, the timing of the P' wave to the QRS complex bears no relation to the length or conduction velocity of the tachycardia circuit, but is dependent on the rate of tachycardia and AV nodal conduction during tachycardia. It is noteworthy that in one patient, sinus node reentrant tachycardia manifested as atrial tachycardia with AV block masquerading digitalis toxicity. Thus, in patients with sinus node reentrant tachycardia in whom P' waves are not clearly discernible before the QRS complex and in whom the configuration and polarity of the P' waves are not clearly evident, the diagnosis of sinus node reentrant tachycardia may not be evident on the surface electrocardiogram, requiring intracardiac studies for documentation of the mechanism of tachycardia.

Effects of antiarrhythmic agents. Unlike other forms of reentrant paroxysmal supraventricular tachycardias, very little is known of the effects of conventional and experimental antiarrhythmic agents on sustained sinus node reentrant tachycardia in human patients. Furthermore, since it is currently impossible to delineate the pathways of reentry within the sinus node, the mechanism of the action of antiarrhythmic agents will remain speculative at best. Nonetheless, a drug will be effective in preventing induction of sustained sinus node reentrant tachycardia if: 1) it alters refractoriness of reentrant pathways in the sinus node so as to make nonuniform alteration of refractory periods similar so that a premature beat is either blocked in both sinus node pathways or is conducted through both pathways assuming functional dissociation within the sinus node; and 2) it results in further slowing of conduction in one of the pathways to the point of block.

Our findings suggest that intravenously administered propranolol has no appreciable effect in preventing induction of sustained sinus node reentrant tachycardia. In contrast, ouabain was effective in preventing its induction in two of two patients. These observations are in agreement with the
findings of Wellens (10). It is unclear from our study whether the effects of ouabain were direct or vagally mediated. Previous studies in the canine heart by Pauley and Damato (18) showed that digoxin did not appreciably affect sinus node echo beats in 18 of 20 dogs; of which 10 were anesthetized with alpha-chloralose and with extrinsic cardiac denervation, 5 were anesthetized with alpha-chloralose but without extrinsic cardiac denervation and 5 were premedicated with morphine and then anesthetized with alpha-chloralose without cardiac denervation. The relative insensitivity of sinus echo beats to digoxin was probably related to minimal or absent vagotonic effect of digoxin in open chest dogs.

In our study intravenously administered verapamil prevented the induction of sustained sinus node reentrant tachycardia in two of two patients. These findings are in agreement with the observations of Curry et al. (9). The effects of verapamil on sinus node reentrant tachycardia are probably related to blockade of the slow inward calcium current in sinus node cells. In contrast to these findings, Rinkenberger et al. (19) reported that although intravenous verapamil prevented the induction of AV nodal reentrant tachycardia in two patients, it did not prevent induction of sinus node reentrant tachycardia in the same two patients while facilitating sustained sinus node reentrant tachycardia in one of the patients. They postulated that the hemodynamic changes during verapamil infusion would be expected to result in withdrawal of vagal tone and an increase in adrenergic tone which may have masked the direct effects of verapamil on the sinus node. However, they also suggested that the inefficacy of verapamil in two patients may have been related to the lower doses used. Thus, verapamil may have resulted in slowing of conduction, but not enough to induce block in sinus node cells.

Amiodarone administered intravenously as a slow infusion resulted in the inability to induce sustained sinus node reentrant tachycardia in all four patients. However, in all four patients two or more echo beats could still be induced. To our knowledge, the effects of intravenous amiodarone on sustained sinus node reentrant tachycardia have not been previously reported. These effects of intravenous amiodarone on sustained sinus node reentrant tachycardia are in agreement with those previously reported (20,21) in AV nodal reentrant tachycardia. The mechanism of the effect of amiodarone on sinus node reentrant tachycardia is probably related to induction of block in sinus node pathways. Previous studies in anesthetized dogs (22) demonstrated slowing of sinus node discharge rate after intravenous amiodarone after pretreatment with propranolol and atropine. In rabbit sinus node preparations, amiodarone has been shown to prolong action potential duration and reduce the slope of diastolic depolarization (23). In addition, Castillo-Fenoy et al. (24) suggested that amiodarone might induce sinoatrial block.

Clinical implications. Our findings suggest that sustained sinus node reentrant tachycardia 1) is an important clinical entity in the genesis of paroxysmal reentrant supraventricular tachycardia; 2) is not as benign as previously thought, since like other forms of supraventricular tachycardia it can result in dizziness and syncope; 3) can masquerade as AV nodal reentrant tachycardia and atrial tachycardia with AV block; 4) usually occurs in the presence of organic heart disease; and 5) can be prevented by the intravenous administration of digitalis glycosides, verapamil and amiodarone.

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References


