The Sinus Node Electrogram in Patients With and Without Sick Sinus Syndrome: Techniques and Correlation Between Directly Measured and Indirectly Estimated Sinoatrial Conduction Time

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SUMMARY Sinus node electrograms (SNEs) were recorded in 44 patients using a standard quadripolar electrode catheter (USCI #6) with 10-mm interelectrode distance. In 23 patients, the catheter was positioned at the junction of the superior vena cava (SVC) and right atrial (RA) wall so that the concave curve of the catheter was facing the concave surface of the RA wall. The distal poles of the catheter were close to but not in direct contact with the RA endocardium underlying the anatomic location of the sinus node (method 1). In 21 patients, the catheter was looped in the RA and advanced to the junction of the SVC-RA wall so that the distal poles of the catheter were in direct contact with the RA endocardium underlying the area of the sinus node (method 2). Stable SNEs could be obtained in 10 of 23 patients (43%) by method 1 and in 18 of 21 (86%) by method 2. SNEs by both methods showed two negatively directed deflections (diastolic and upstroke slope of the sinus node) of low frequency and amplitude occurring after the deflections of the T and U waves but before the P wave and intratral electrograms from various atrial sites. Unlike method 1, atrial activation by method 2 was characterized by an atrial injury potential. SNEs by method 2 were more stable and less prone to baseline drifts.

Carotid sinus massage performed in 10 patients resulted in prolongation of the onset of the upstroke slope to atrial activation. In one patient, it resulted in marked first-degree sinoatrial block. SNEs were recorded in one patient during and after a run of spontaneous atrial flutter. After termination of atrial flutter, sinoatrial conduction time (SACT) prolonged for the first postflutter sinus beat and normalized by the third sinus beat.

Direct SACT was assessed on the SNE in all 28 patients, 12 of whom had sick sinus syndrome (SSS). SACT was also estimated indirectly by continuous pacing in 28 patients and by premature stimulation in 20 patients. Direct SACT was significantly ($p < 0.001$) longer in patients with SSS (135 ± 30 msec, mean ± SD) than in patients without SSS (87 ± 12 msec).

There was a good correlation between direct SACT and SACT by continuous pacing ($r = 0.843$, $n = 28$) and direct SACT and SACT by premature stimulation ($r = 0.778$, $n = 18$). However, in certain patients, there were appreciable differences and direct SACT was longer than estimated SACT by continuous pacing or premature stimulation. We conclude that (1) the success rate of stable SNEs with no appreciable baseline drift is increased with direct contact of the catheter with atrial endocardium underlying the sinus node area (method 2); (2) carotid sinus massage results in depression of sinus node automaticity and prolongation of SACT; (3) direct SACT is significantly longer in patients with SSS and can separate most patients with from those without SSS; and (4) there is a good correlation between direct SACT and indirectly estimated SACT, although the latter underestimates the actual SACT.

UNTIL RECENTLY, direct sinus node recordings were not possible in man. Thus, sinus node function was assessed indirectly by measuring sinus node recovery time and sinoatrial conduction times (SACTs).1-17 Although the former is taken as a reflection of sinus node automaticity and the latter a reflection of atriosinus and sinoatrial conduction, this differentiation remains far from clear.

Cramer et al.18 studied isolated rabbit right atrium in an attempt to identify extracellular sinus node electrograms simultaneously with transmembrane action potential of the sinus node pacemaker. In 1978, they reported successful recordings of sinus node electrograms in a canine right atrial preparation and in the beating dog heart using handheld probes and conventional bipolar electrode catheters.19,20 Hariman et al.21 were the first to describe a technique for recording sinus node electrograms in the intact human heart using conventional electrode catheters. The technique enabled them to assess direct SACT. However, Hariman et al.21 did not correlate direct SACT with indirectly obtained values. Later, Reiffel et al.22 reported a somewhat different technique to record sinus node electrogams in the intact human heart. They reported direct SACTs and compared them to those obtained by premature stimulation. However, they did not correlate direct values of SACT with those determined by continuous pacing. In addition, although Hariman et al.21 reported direct sinoatrial conduction in a patient with sick sinus syndrome and Reiffel et al.22 in three patients with sick sinus syndrome, it remained unclear whether direct assessment of SACT could distinguish between patients with and those without sick sinus syndrome. These investigators21,22 reported two major drawbacks to their techniques: (1) Hariman et al.21 reported a success rate of only 50% and (2) Reiffel et al.22 noted frequent occurrence of baseline drifts.

The purpose of the current study was to assess the
feasibility of improving the success rate and to obtain more stable sinus node electrograms in man, to determine whether direct values of SACT could differentiate between patients with and without sick sinus syndrome and to correlate direct values of SACT with estimated values by both the premature stimulation and the continuous pacing methods.

Materials and Methods

Forty-four patients, ages 53–76 years old, were studied. Each patient gave informed, signed consent. The patients were undergoing electrophysiologic studies for a variety of reasons. Sixteen of 44 patients had sick sinus syndrome, suggested by the presence of sinus bradycardia, sinus arrest or sinoatrial block and the bradyarrhythmia-tachyarrhythmia syndrome. All 16 had symptoms of light-headedness or syncope and palpitations documented on an ECG or 24-hour ambulatory Holter tape recording. Successful sinus node recordings were obtained in 16 patients (nos. 1–16) without sick sinus syndrome and 12 patients (nos. 17–28) with sick sinus syndrome. The pertinent clinical features of these patients are provided in table 1. The lowest spontaneous sinus rates of the 12 patients with sick sinus syndrome ranged from 32 to 54 beats/min. Corrected sinus node recovery times were assessed in nine of these 12 patients (nos. 19, 20, 22–28) with sick sinus syndrome. Seven of nine patients (nos. 19, 20, 23, 24, 26–28) had abnormal (> 450 msec) corrected sinus node recovery times (range 480–1580 msec).

All patients were studied in the electrophysiology laboratory in the postabsorptive, nonsedated state between 9 a.m. and 12 noon. Two quadripolar catheters (USCI #6) with 10-mm interelectrode distances were introduced percutaneously under local anesthesia and positioned in the superior vena cava, in the high, mid- or low right atrium and at the level of the tricuspid valve for recording His bundle activity. Two or more electrocardiographic leads and intracardiac electrograms at filter frequencies of 0.1–50 Hz and 30–500 Hz and time lines generated at 40, 200 and 1000 msec were displayed on a multichannel oscilloscope (VR-12, E for M) and recorded on thermal paper at paper speeds of 50–100 and 150 mm/sec. Atrial stimulation was performed using a programmable stimulator (Bloom and Associates) or a Medtronic 5325 programmable stimulator utilizing stimuli at twice diastolic threshold and 2 m sec in duration.

Method 1

The catheter in the superior vena cava was positioned at about the junction of the superior vena cava and the right atrial wall such that the concave curve of the catheter was facing the concave surface of the right atrial wall. The distal poles of the electrode catheter were close to but not in direct contact with the right atrial wall underlying the anatomic location of the sinus node (fig. 1A) as determined by fluoroscopic observation of the catheter position in relation to the lateral right atrial wall. The electrogram obtained from the distal two poles of the catheter was displayed on a multichannel oscilloscope at low-pass filter frequencies of 0.1–50 Hz19, 20, 22 and high-gain amplification of 50–100,000 µ V/cm. The proximal two poles were used to obtain intraatrial electrograms at filter frequency settings of 30–500 Hz. The catheter was moved around the superior vena caval–right atrial junction until a sinus node electrogram was obtained. The bipolar recording of the sinus node electrogram was displayed so that the upstroke slope of the sinus node electrogram

<table>
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<th>Diagnosis</th>
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<td>ASHD/VPCs</td>
<td>90 80 90</td>
</tr>
<tr>
<td>2</td>
<td>76</td>
<td>ASHD/LBBB</td>
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</tr>
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<td>70 86 90</td>
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<td>60</td>
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<td>95 100 105</td>
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<td>48</td>
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<td>62</td>
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<td>145 155 170</td>
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<tr>
<td>28</td>
<td>69</td>
<td>SSS</td>
<td>150 146 NO</td>
</tr>
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</table>

Abbreviations: ASHD = atherosclerotic heart disease; VPC = ventricular premature complex; LBBB = left bundle branch block; PSVT = paroxysmal supraventricular tachycardia; AF = atrial fibrillation; WPW = Wolff-Parkinson-White syndrome; SSS = sick sinus syndrome; RBBB = right bundle branch block; LAD = left-axis deviation; SACT = sinoatrial conduction time; CP = SACT measured by continuous pacing; PS = SACT measured by premature stimulation; NO = not obtainable.
was upright.\textsuperscript{18,22} The sinus node electrogram so obtained was characterized by an upstroke slope that preceded the P wave on the ECG and intraatrial electrograms recorded from other atrial sites. The upstroke slope was usually preceded by a diastolic slope (fig. 2A) that was identified in relation to the post T-wave baseline. Method 1 was used in the first 23 patients studied. However, stable sinus node electrograms could be recorded in 10 of 23 patients (43%). Localization of the sinus node electrograms by method 1 took approximately 15–25 minutes. Six to 9 minutes of fluoroscopy were usually required to achieve a satisfactory position of the catheters.

Method 2

Because of the relatively low success rate in recording stable sinus node electrograms by method 1, in the remaining 21 patients the catheter was looped in the right atrium and advanced to the junction of the superior vena caval–right atrial wall such that the distal pole of the catheter was in direct contact with the atrial endocardium (fig. 1B).

The electrogram recorded from the distal two poles of the catheter was displayed on a multichannel oscilloscope at low-pass filter frequencies of 0.1–50 Hz and high-gain amplification of 50–100 μV/cm. Sinus node electrograms obtained by method 2 were characterized by the presence of an upstroke slope, followed by an atrial injury potential.\textsuperscript{23} The upstroke slope was usually preceded by a diastolic slope (fig. 2B). The diastolic and upstroke slope had the same characteristics as the sinus node electrograms recorded in previous human and animal studies.\textsuperscript{18–22} Stable sinus node electrograms could be recorded in 18 of 21 patients (86%) by method 2. Slight manipulation of the catheter away from the atrial endocardium was sometimes associated with loss of atrial injury potential and preservation of the upstroke slope of the sinus node; however, manipulation of the catheter either by withdrawing it by 5–10 mm or by rotating it medially was associated with loss of both upstroke slope and atrial injury potential. The time required to obtain a stable sinus node electrogram by method 2 was more or less similar to method 1. However, sinus node electrograms obtained by method 2 were more stable and less prone to baseline drift.

**Direct SACT**

Direct SACT was measured from the onset of the upstroke slope to atrial activation on the sinus node recording (fig. 2). Direct SACT was measured at paper speeds of 150 mm/sec for five consecutive beats in all patients and the mean was taken as the representative SACT. Direct SACT is a reflection of conduction time from the sinus node to the atrial endocardium.

**Indirect SACT**

We assessed SACT in all 28 patients by the continuous pacing method and in 20 of 28 patients by the premature stimulation method. SACT by all three methods were assessed in 20 patients.

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**Figure 1.** Catheter position used for recording sinus node electrograms by (A) method 1 and (B) method 2. SVC = superior vena cava; IVC = inferior vena cava; EL. CA. = electrode catheter; RA = right atrium.

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**Figure 2.** Schematic of sinus node electrograms (SNEs) obtained by (A) method 1 and (B) method 2. SNEs by both methods are characterized by a diastolic and an upstroke slope that precede the P wave and the intraatrial electrogram. After the upstroke slope, atrial activation by method 2 is characterized by an atrial injury potential (Atr. in.) at the onset of the intraatrial electrogram. Sinoatrial conduction time (SACT) by both methods is measured (as shown by the dotted lines) from the onset of the upstroke slope (straight arrow) to the atrial activation. ATR = bipolar recording of intraatrial electrogram.
Continuous Pacing Method

SACT by the continuous pacing method was estimated as proposed by Narula et al. The high right atrium was paced for eight beats at cycle lengths slightly shorter than the basic cycle length, provided that consistent capture of the atrium was achieved. Pacing was abruptly stopped to allow spontaneous sinus rhythm to return and recordings were continued for subsequent eight spontaneous cycles. The protocol was repeated twice at the same cycle length and at three more cycle lengths of 5, 10 and 15 msec shorter than the first cycle length. SACT was obtained by subtracting the mean sinus cycle from the interval between the last paced atrial electrogram and the atrial electrogram of the first escape sinus cycle. The average of the SACT obtained at all four cycle lengths was taken as the representative SACT. After atrial stimulation, the postspacing sinus cycles were longer than the mean sinus cycles (range 1–9%, mean 2.4%). This finding is in accordance with our previous observations. The SACT obtained by continuous pacing represents the sum total of the conduction time into and out of the sinus node. For comparison with direct SACT values, half of the SACT measured by continuous pacing was taken as the representative SACT. SACT by the continuous pacing method could be estimated in all 28 patients and usually took less than 5 minutes.

Premature Stimulation Method

Premature atrial stimuli were delivered after every eighth spontaneous sinus cycle, beginning late in diastole. The coupling interval of the premature atrial stimuli was decreased in decrements of 10–20 msec until the effective refractory period of the right atrium was obtained. For every premature atrial stimulus delivered, the spontaneous sinus cycles (A1A1), the test cycle (A1A2), and the return cycle (A1A3) were measured. The response of the premature stimuli in the zone of sinus node reset (i.e., the latter third of zone 11) were used to calculate mean values of A1A1, A1A2, and A1A3 intervals. The A1A1, A1A2, and A1A3 cycles were normalized by dividing them with the A1A1 cycles. SACT was calculated according to the formula: SACT = (A1A2) – (A1A1). For comparison with direct SACT, the latter value was divided by 2. SACT could be estimated by premature stimulation in 18 of 20 patients. In the remaining two patients, the absence of a clear zone of sinus node reset precluded the determination of SACT. After premature stimulation, the postreturn sinus cycles (A1, A2) were slightly longer than the mean sinus cycle (range 1–7.5%, mean 2.1%). This finding is in accordance with our previous findings. The premature stimulation protocol usually took less than 10 minutes.

Statistical analysis was performed by using the t test for paired and unpaired data. Values are mean ± SD.

Results

Sinus Node Electrograms

Stable sinus node electrograms were recorded in 28 patients (64%): in 10 of 23 (43%) by method 1 and in 18 of 21 (86%) by method 2. Although sinus node electrograms could be recorded in an additional two patients, because of the difficulties associated with the exact identification of the onset of the upstroke slope, these two patients were excluded.

The sinus node electrograms recorded by either method usually showed two negatively directed deflections of low frequency and amplitude occurring after the deflections of the T and U waves but before the P wave on the surface ECG and intraatrial recordings from various atrial sites. The two negatively directed deflections that were displayed so that they gave positive deflections correspond to the diastolic slope and the upstroke slope of the sinoatrial node. The diastolic and the upstroke slopes were recorded from a discrete area which approximates the anatomic location of the human sinus node. A small change (5–10 mm) in catheter position (upwards or downwards or medially away from the right atrial wall) resulted in loss of the sinus node potential. In addition, recordings at other atrial sites (i.e., the high, mid- and low right atrium) did not reveal the presence of a diastolic or upstroke slope that characterized sinus nodal potentials. Occasionally, the configuration and duration of the upstroke slope varied from beat to beat. The latter was seen primarily in patients with sinus arrhythmia. Figure 3 is an example of a sinus node electrogram in patient 5, who did not have sick sinus syndrome, using method 1. The diastolic and the upstroke slopes follow the T wave on the surface ECG but precede the P wave and the intraatrial electrogram recorded in the low right

![Figure 3](http://circ.ahajournals.org/)

**Figure 3.** Sinus node electrogram (SNE) recorded by method 1. L1, L2 and V1 = surface ECG leads; AT = intraatrial electrogram recorded at the low atrium. The arrows point to the onset of the SNE and the upstroke slope of the SNE. The average direct sinoatrial conduction time (SACT) measured 95 msec.
atrium. Figure 4 is an example of a sinus node electrogram recorded in patient 23, who had sick sinus syndrome, using method 2. The diastolic and the upstroke slopes follow the T- and the U-wave deflections but precede the P wave on the surface ECG and the intraatrial electrogram recorded in the high and mid-right atrium. After the upstroke slope, atrial activation is recorded that is characterized by an atrial injury potential. One of the problems associated with method 1 was baseline drift. Baseline drift occurred less frequently with method 2, suggesting that it is primarily related to free-floating catheter motion. The occurrence of the upstroke slope immediately after the T wave precluded the identification of the diastolic slope, making the onset of the upstroke slope and the measurement of SACT difficult to decipher.

Validation of Sinus Node Electrograms

To determine if the diastolic and upstroke slopes were restricted only to the sinus node region, after recording a sinus node electrogram, the catheter was moved sequentially to the mid-, low and septal right atrium and recordings were obtained at low-frequency settings of 0.1–50 Hz. Recordings from these atrial regions did not reveal diastolic and upstroke slopes (fig. 5).

To determine whether atrial injury potentials preceded by a diastolic and upstroke slope were restricted solely to the sinus node region, atrial injury potentials were recorded also at other atrial sites at similar filter frequencies; these did not reveal the presence of a diastolic and upstroke slope preceding the atrial injury potential (fig. 6).

To determine the effect of atrial pacing on the sinus node electrogram, atrial pacing at rates slightly and considerably faster than the sinus rate was performed. Figure 7 is an electrogram from a patient whose low right atrium was paced at cycle lengths 100 msec shorter than the spontaneous sinus cycle. Atrial pacing at a cycle length of 700 msec results in capture of the
values for SACT in patients with and without sick sinus syndrome are listed in table 2.

Directly measured SACT was significantly longer \( (p < 0.001) \) in patients with sick sinus syndrome (mean 135 ± 30 msec, range 90–200 msec) than in patients without sick sinus syndrome (mean 87 ± 12 msec, range 60–112 msec). SACTs measured by continuous pacing and premature stimulation were significantly longer \( (p < 0.001) \) in patients with than in those without sick sinus syndrome. Figure 4 is an example from patient 23, who had sick sinus syndrome, demonstrating a prolonged direct SACT of 180 msec.

Carotid sinus massage, which was performed in 10 patients, resulted in lengthening of sinoatrial conduction and slowing of the sinus rate. In patient 16, carotid sinus massage resulted in progressive prolongation of sinoatrial conduction, depression of sinus node automaticity, and marked first-degree sinoatrial block (fig. 8). During carotid sinus massage, the AA intervals (beats 4, 5 and 6) were more prolonged on the intraatrial electrograms than the sinus cycle on the sinus node electrogram. The longer AA cycle for these beats was related to progressive prolongation of sinoatrial conduction, with resultant marked first-degree sinoatrial block (beats 7 and 8). Beats 7 and 8 reveal sinus depolarization (clear diastolic and upstroke slopes and repolarization) and atrial activation occurring 600 msec after sinus depolarization. Atrial activation after the seventh and eighth sinus node deflections demonstrate alteration of the P-wave configuration most obvious in lead II, suggesting a change in atrial activation for these beats. Normalization of sinoatrial conduction after carotid massage resulted in normalization of atrial activation and P-wave configuration.

In one patient, sinus node electrograms were recorded during and after a run of spontaneous atrial flutter (fig. 9). After the run of atrial flutter, the first sinus beat revealed first-degree sinoatrial block. Sinoatrial conduction normalized for the third sinus beat.

**Correlation of Direct and Indirect SACT**

There was a good correlation between direct and indirect SACTs estimated by continuous pacing \( (r = 0.843, n = 28) \). Similarly, there was a good correlation between the direct SACT and that estimated by the premature stimulation method \( (r = 0.778, n = 18) \). However, the direct SACT was 7.67 ± 18 msec longer (range i.e., −19 to +60 msec) than that estimated

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<th>Indirect SACT (msec)</th>
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<tbody>
<tr>
<td>No SSS ((n = 16))</td>
<td>87 ± 12</td>
<td>80 ± 17</td>
<td>86 ± 13</td>
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<tr>
<td>SSS ((n = 12))</td>
<td>135 ± 30</td>
<td>129 ± 24</td>
<td>125 ± 23</td>
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\( p < 0.001 \)  

Abbreviations: SACT = sinoatrial conduction time; SSS = sick sinus syndrome; CP = continuous pacing; PS = premature stimulation.
by the continuous pacing method and 6 ± 19 msec (range −25 to +57 msec) longer than estimated by the premature stimulation method.

Discussion

Hariman et al.21 described a simple method for recording sinus node electrograms in the intact human heart. Using standard electrode catheters, they recorded a diastolic and an upstroke slope in 15 of 30 patients (50%), children and adults with various forms of congenital and acquired heart disease. Their method is similar to the method we used in our first 23 patients. The inability to record sinus node electrograms in 50% of their patients was probably related to the inability to obtain accurate catheter positioning on the relatively small and restricted sinus nodal pacemaking site and the lack of obtaining close catheter contact with the atrial endocardium in the region of the sinus node. Reiffel et al.22 recorded sinus node potentials in 23 patients using a tripolar or a quadripolar electrode catheter. Their method was at variance with our recording techniques and those of Hariman et al. in that the convex surface of the catheter was facing the concave surface of the right atrium. Nonetheless, using the latter technique, they recorded sinus node electrograms in 19 of 23 patients (83%). In their studies, the sinus node electrograms were characterized as a smooth, low-frequency upstroke slope before depolarization of the atrium and the onset of the former was defined by the change in slope from the post–T-wave baseline plateau. Apparently, only in some of their patients did the post–T-wave plateau have a mild upsloping character representing the sinus node diastolic slope. The other major difficulty with their technique was frequent baseline drift.

We recorded sinus node electrograms using method 1 in 10 of 23 patients (43%). These results are comparable to those obtained by Hariman et al.21 using a similar method. Inability to record stable and reproducible recordings in the remaining patients was related to lack of close contact with the atrial endocardium in the region of the sinus node and the occurrence of baseline drift. The latter is likely related to free-floating catheter motion induced by spontaneous respirations and atrial contractions. The occurrence of baseline drifts makes the measurements of SACT unreliable and the inference of possible changing sites of focal automaticity within the sinus node24 highly debatable. We increased our success rate and recorded stable sinus node electrograms relatively free of baseline drifts in the remaining 18 of 21 patients (86%) by a small change in the technique that assured greater proximity to or contact with the atrial endocardium underlying the area of the sinus node. Reiffel et al. reported a similar success rate. However, they noted frequent baseline drifts.

That the distal pole of the electrode catheter was in direct contact with the atrial endocardium is suggested by the observation of an atrial injury potential after the upstroke slope of the sinus node. Slight manipulation
of the catheter away from the atrial endocardium was associated with loss of atrial injury potential with preservation of the upstroke slope in some patients; in others, it was associated with loss of both. This suggests that close contact or proximity of the catheter poles to the atrial endocardium underlying the sinus node region is of paramount importance in obtaining successful and stable recordings of sinus node potentials. The inability to obtain sinus node electrograms in three of 21 patients was related to the inability to achieve close contact with the atrial endocardium due to the presence of a large right atrium in these three patients. Although we believe that the higher success rate of stable sinus node recordings with method 2 is related to the different approach relative to method 1, we cannot exclude the possibility that it was not, in part, related to the greater experience acquired during the course of the study.

That the upstroke slope seen before the atrial injury potential or atrial activation was due to sinus node depolarization and was not a part of atrial depolarization is suggested by the following: (1) Recordings at other atrial sites did not reveal an upstroke slope before the atrial injury potential or atrial activation; the upstroke slope was localized to a small area corresponding to the anatomic location of the sinus node. (2) The atrial injury potential or atrial activation occurred simultaneously with the intraatrial electrogram recorded at multiple atrial sites and the P wave on the surface ECG, whereas the upstroke slope preceded both the intraatrial electrogram and the P wave. (3) Carotid sinus massage resulted in prolongation of the onset of the upstroke slope to atrial activation, and in one patient it resulted in marked first-degree sinoatrial block. In another patient with paroxysmal atrial flutter, the sinus beat after a run of atrial flutter revealed first-degree sinoatrial block between the upstroke slope and atrial activation. (4) Atrial pacing studies resulted in abolition of the upstroke with rapid depolarization occurring immediately after the pacing stimulus. (5) Previous studies25, 26 in man of monophasic action potential recorded from the atrial endocardium (as sites remote from the sinus node region) using suction electrodes have observed a rapid depolarization (phase 0) without a preceding upstroke slope.

It may be argued that close contact with the atrial endocardium could result in injury to the sinus node, thus changing its automaticity, or result in prolongation of sinoatrial conduction. This seems unlikely because (1) the sinus node is a subepicardial structure,27, 28 so neither the sinus node nor the perinodal tissue would be expected to be injured; (2) no change in sinus rate or atrial premature contractions occurred, the former suggesting lack of alteration in automaticity and the latter suggesting lack of irritability of the atrial endocardium; (3) in six patients in whom sinus node electrograms were recorded by both methods, there was no appreciable difference in SACT calculated by either method; (4) direct and indirect SACT measured by the two techniques were normal in patients without sick sinus syndrome.3, 7

Shifts in sinus pacemaker location due to an increase in vagal tone were originally reported by Meek and Eyster29 and later by Bowman et al.30, 31 Hariman et al.32 studied extracellular electrograms recorded from the sinus node region in conscious dogs using a sinus node electrode containing 48 silver terminals and implanted over the sinus node. They observed shifts in sinus pacemaker location, which they attributed to an increase in vagal tone, a decrease in vagal tone or an increase in sympathetic tone. These shifts in sinus pacemaker location were associated with various degrees of change in P-wave configuration. In this regard, our observations in patient 16 after carotid sinus
massage (fig. 8) are of considerable interest. The observation after marked first-degree sinoatrial block of a change in P-wave configuration best evident in lead II without a change in P-wave vector suggests a shift in atrial activation due to an increase in vagal tone. The absence of clear diastolic and upstroke slope preceding atrial activation excluded the possibility of a shift in sinus pacemaker.

**Sinoatrial Conduction**

The recording of sinus node electrograms permitted assessment of SACT free of assumptions that undermine the SACTs measured indirectly. In patients without sick sinus syndrome, our values of SACT were 60–112 msec (average 87 ± 12 msec). These findings are in agreement with the observations of Reiffel et al., who reported directly measured SACTs of 46–116 msec (average 90 ± 18 msec) in 16 patients with normal sinus node function. In contrast, our values of direct SACT and those obtained by Reiffel et al., in patients without sick sinus syndrome differ appreciably from those reported by Hariman et al. Hariman et al. reported values of 34.9 ± 2.1 msec in 14 patients without sick sinus syndrome. The reason for discrepancy between our findings and those of Hariman et al. is unclear. Although Hariman et al. pointed to the discrepancy between their direct values of SACT and those obtained by previous investigators using indirect methods of assessment, they did not assess SACT by indirect methods in any of their patients.

It remains unclear whether sinus node recordings can separate patients with sick sinus syndrome from those without sick sinus syndrome. Hariman et al. recorded sinus node electrograms in a single patient with sick sinus syndrome and found a prolonged direct SACT of 210–280 msec. Reiffel et al. recorded sinus node electrograms in three patients with sick sinus syndrome and obtained direct SACTs of 110–126 msec. Our observations that direct SACTs were significantly longer in 12 patients with sick sinus syndrome compared with 16 patients without sick sinus syndrome suggest that direct measurements of SACT by recording a sinus node electrogram can separate patients with sick sinus syndrome from those with normal sinus node function. Furthermore, our observation in two patients with normal direct SACT suggest that first-degree sinoatrial block can develop after a paroxysm of transient atrial flutter and carotid sinus massage.

**Correlation Between Direct and Indirect Sinoatrial Conduction**

Until the development of direct recordings of sinus node potential in man, SACT was estimated indirectly using the premature stimulation method of Strauss et al. and the recently proposed continuous pacing method. However, the indirect methods of estimating SACT require several assumptions: no change in sinus node automaticity as a result of premature stimulation or atrial pacing; atrioventricular and sinoatrial conduction are equal; and all premature impulses are capable of resetting the sinus node in the zone of reset. The validity of these indirect techniques has been questioned because the latter assumptions may not be true. Furthermore, the occasional presence of a chaotic pattern of return cycles and of sinus node reentrant beats and the occasional absence of a zone of reset can make the measurement of SACT by premature stimulation impossible in some patients. Despite these limitations, we found a good correlation between direct SACT and that measured by premature stimulation and the continuous pacing methods. However, in individual patients, appreciable differences were observed and the direct SACT was longer than the estimated SACT by the two indirect methods. Of the methods available to assess sinoatrial conduction, the indirect methods had the highest success rate (96%) and the direct method of assessing SACT was the least successful (43–86% of patients), by either of the two direct methods. We believe that the direct method of measuring SACT is most useful in (1) confirming SACTs obtained by the indirect methods (e.g., in patient 17, with sick sinus syndrome in whom indirect methods of estimating sinoatrial conduction revealed normal values, direct measurement of SACT revealed an abnormal value); (2) assessing patients in whom the atrial premature stimulation method is either imprecise or in those in whom the absence of a zone of reset makes the assessment of sinoatrial conduction impossible; (3) patients with frequent premature atrial contraction which cannot be overdrive suppressed at rates ≤ 10 beats/min above the sinus rate; and (4) analyzing the relative contributions of changes in sinus node automaticity and sinoatrial conduction during a variety of physiologic maneuvers, such as carotid sinus massage and overdrive pacing for estimation of sinus node recovery time.

**Acknowledgment**

The authors acknowledge Juliann Lyons and Robert Zeiler for technical assistance, Mike Yu for artwork, and Phyllis Blevins for preparation of the manuscript.

**References**

8. Kang PS, Gomes JAC, Kelen G, El-Sherif N: Role of autonomic regulatory mechanism on sino-atrial conduction and sinus node
29. Meek WJ, Eyster JAE: Experiments on the origin and propagation of the impulse in the heart. IV. The effect of vagal stimulation and cooling on the location of the pacemaker within the sinoauricular node. Am J Physiol 34: 368, 1914
The sinus node electrogram in patients with and without sick sinus syndrome: techniques and correlation between directly measured and indirectly estimated sinoatrial conduction time.

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Circulation. 1982;66:864-873
doi: 10.1161/01.CIR.66.4.864

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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