The Human Sinus Node Electrogram:  
A Transvenous Catheter Technique and a Comparison of Directly Measured and Indirectly Estimated Sinoatrial Conduction Time in Adults  

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SUMMARY To improve methods for evaluating human sinus node function (SNF), we developed a transvenous electrode catheter technique for direct recording of sinus node electrograms in adults. Sinus node electrograms (SNE) characterized by low-frequency, anatomically localized pre-P-wave potentials were obtained in 19 of 23 patients. The SNE configuration was similar to that previously found for endocardial SNE recordings in vitro atrial preparations, in open-chest dogs and during human open heart surgery. In 16 patients with normal SNF, directly recorded sinoatrial conduction times (SACTs) were 46–116 msec. In three patients with sick sinus syndrome, SACT was 110–126 msec. In 15 of the 19 patients, SACT was estimated by the atrial premature stimulus technique and was compared with the directly measured SACT. When atrial premature depolarizations produced no sinus node depression, the mean difference between the direct and estimated SACT was 1.8 ± 5.6 msec.  

ALTHOUGH THE SINUS NODE (SN) is the dominant cardiac pacemaker, neither SN impulse initiation nor conduction are visible on the body surface ECG or on standard intracardiac electrograms because depolarization within the node is of very low amplitude. SN function, therefore, must be assessed indirectly. Normal SN function is assumed when the atrial musculature is depolarized at a normal rate and in a normal temporal sequence — so-called normal sinus rhythm (NSR). In NSR the atrial rate is assumed to correspond to the rate of impulse formation within the SN; however, the rate of impulse conduction from the SN to the atrium cannot be ascertained.  

Because conduction from the SN to the atrium cannot be assessed directly, an indirect method for estimating the sinoatrial conduction time (SACT) was developed: the premature atrial stimulation technique. This test has been used extensively to evaluate SACT in normal subjects and in those with SN dysfunction. In normal persons, the calculated SACT averages about 80 ± 20 msec and does not exceed 130 msec.2-4 However, this estimation is indirect and has not been directly verified. Also, some conditions prevent accurate estimation of SACT by premature atrial stimulation: SN suppression by the atrial premature depolarization (APD), sinoatrial (SA) entrance block, and marked sinus arrhythmia.2,3 A direct method of assessing SACT is needed for such instances.  

The initial steps towards achieving direct recordings from the SN in man were taken in 1977 when Cramer and associates identified the extracellular potential changes associated with electrical activity of the SA pacemaker in the isolated rabbit right atrium.6 They recorded an extracellular SN electrogram (SNE) simultaneously with the transmembrane action potential of the SN pacemaker (fig. 1). The microelectrode within the node was immediately subjacent to the extracellular electrode. In 1978, Cramer et al. reported similar findings in canine right atrial preparations.6,7 The SNE could be recorded over the SN from either the epicardial or the endocardial surfaces. In addition, the SNE was recorded from the epicardial surface of the beating dog heart using hand-held probes and by conventional bipolar electrode catheters held over the endocardial surface of the SN.6,7 However, in the beating heart the diastolic phase 4 slope, which was visible in isolated atrial preparations, was difficult or impossible to define because ventricular QRS- and T-wave events were superimposed upon it. Later in 1978, Harriman et al.8 used the same endocardial recording technique to record the SNE in patients during open heart surgery. These developments suggested the feasibility of recording the SNE in intact, awake man using transvenous catheter electrodes.  

Krongrad et al.9 reported preliminary work with a transvenous method for recording the SNE during human cardiac catheterization, predominantly in children. We have extended this work. This report details our development and use of this new intracardiac electrocardiographic technique in adults. The direct SNEs we obtained permitted us to measure normal SA conduction intervals directly, to evaluate the accuracy of the SACT estimated by the premature atrial stimulation technique, and to verify the existence of SA exit block in man.

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1324
Materials and Methods

Twenty-three patients were enrolled for study. The SNE was successfully recorded in 19 (83%). Of these 19 patients, eight were male and 11 were female; their ages ranged from 40–84 years (mean 68 years). Patients 2, 4 and 7 had sick sinus syndrome, defined by undue sinus bradycardia (patients 2 and 4), SA arrest (patient 2), SA exit block (patient 4), or the bradycardia-tachycardia syndrome (patient 7).4 Patients 2 and 4 had episodic dizziness and syncope; patient 7 had periodic palpitations and dizziness. The remaining 16 patients had no known sinus node dysfunction. All were studied in the nonsedated, postabsorptive state. All patients gave informed, written consent.

Our technique for recording the SNE was based on the procedures developed for recording SNEs in atrial preparations,5,6 in intact dogs,6,7 in humans during open heart surgery,8 and in the pediatric cardiac catheterization laboratory.9 In our study, bipolar SNEs were obtained in the following manner. We used #6 or #7F tripolar or quadrupolar catheters with the same convex distal curve used for His bundle electrograms. Inter electrode distances were 1 cm. Prior animal investigation showed that interelectrode distances of 0.7–1.0 cm would be suitable for recording the SN potential. For recording, we used high-amplification (up to 100 µV/cm) and low-pass filters of 0.1–50 Hz, as these were found successful in prior investigations.** In the last five patients studied we also attempted to record the SNE after the low-end filter was changed to 1 Hz and then 3 Hz. In one patient we recorded the SNE during midlateral right atrial pacing.

After percutaneous insertion into the femoral vein, the recording poles were advanced under fluoroscopic monitoring to the superior vena cava and then pulled back slowly to the right atrial–superior vena cava junction, with the convex curve of the catheter against the convex curve of the atrial border (fig. 2). The catheter was then rotated or pulled back slightly further in small increments until a pre-P-wave depolarization was detected. Fine adjustments in catheter position were then made until the maximum amplitude of the potential was established. Catheter positioning and localization of the SN potential usually took 10–20 minutes after femoral insertion. Occasionally, periods longer than 30 minutes were required. The configuration of the depolarization so obtained was that of a smooth, low-frequency upstroke slope that began before and merged into the P wave.
(figs. 3–6). As such, it always resembled the SNE obtained in the atrial, canine, and prior human investigations. Unless the cycle length was short or the PR interval prolonged, the SN potential was separated from the preceding T wave by an isoelectric plateau. By convention,5,9 the bipolar recording was displayed so that the SN upstroke slope was upright on the SNE, i.e., negative potentials gave positive deflections.

Localization of the SNE was verified in the first four patients by recording from multiple atrial sites with the same catheter at the same filter settings (0.1–50 Hz) and demonstrating absence of pre-P-wave activity elsewhere. In the last 15 patients, verification was obtained by simultaneously recording the SNE and one or more atrial electrograms from multiple other sites in the atria with a second catheter using the same filter settings (0.1–50 Hz) as well as settings of 10–500 Hz. These also showed absence of pre-P-wave activity at all other locations. Additional verification of the source of the SNE was obtained by recording during spontaneous SA exit block in one patient or during SA block induced by carotid sinus massage in three patients, and during atrial pacing in one patient. In four patients the SNE was not successfully recorded because of catheter instability, severe baseline drift, short cycle length or long PR interval (see below), or unknown reasons.

In some patients, low-frequency activity linked to the termination of the T wave was also recorded on the intracardiac lead (fig. 7). It occurred after the T wave had apparently ended on the body surface leads and probably represents the U wave. When present, the post-T-wave potential could be increased by moving the catheter toward the ventricle. Although this potential was often quite large on the intracardiac leads, it was not usually apparent on the body surface ECG leads.

At long cycle lengths, the presence of a U wave did not interfere with the recording and recognition of the SN potential because the SN potential was still separated from ventricular repolarization (TU complex) by a clear isoelectric plateau. This isoelectric plateau allowed the onset of the SN potential to be identified and made it possible to recognize the SNE.

When the cycle length is short, however, a U wave can make the recording procedure more difficult. At very short cycle lengths, the U wave could merge with the SN potential, making its onset impossible to define, or the U wave could merge with the P wave itself. A UP merger poses two problems. First, it obliterates the SN potential. Second, if the U wave is negative the merger of the upwards sloped terminal portion with the ensuing P wave could mimic a SN potential. This did not occur in the patients we chose to study. At short cycle lengths such a UP merger could be detected only by recognizing the U wave. As

**Figure 2.** Catheter location for recording the sinus node electrogram. **SVC** = superior vena cava; **RA** = right atrium; **IVC** = inferior vena cava.

**Figure 3.** Schematic illustration showing the direct measurement of sinoatrial conduction time (SACT). A schematic copy of a sinus node electrogram (SNE) is shown. On the SNE, high right atrial depolarization (A), ventricular depolarization (V), the T wave (T) and the sinus node potential (SN) are identified. In the second beat, reference lines are drawn through the point at which the SN potential first becomes evident and the point at which atrial activation begins. The SACT is the interval between these two reference lines.
suggested above, the U wave would be identified when the pre-P-wave upstroke was in fixed relationship to the prior QRST as cycle length varied and when the complex increased in size, rather than disappeared, as the catheter was moved to the ventricle. At slightly longer cycle lengths, a U-SN potential merger rather than a UP merger may occur. This can be identified by the use of multiple recording catheters or by recording at multiple sites. These recordings must be made at the same filter settings (0.1–50 Hz). In this circumstance, the catheter over the SN should record a continued upslope from the U wave to the P wave, while a recording elsewhere in the atria should reveal a U wave followed by an isoelectric baseline before the P wave. Figure 7 shows this situation. The recording was made from one of the patients we considered to be a recording failure. Although the recordings in this figure suggest that a SN potential may be present over the SN (fig. 7B), its usefulness is limited because the termination of the U wave and onset of the SN potential cannot be clearly defined, so the SACT cannot be measured. If only panel B were available, it would not be reasonable even to suggest the presence of an SN potential. A prolonged PR interval could impose the same problems as a very short cycle length, as this, too, could cause the SN potential and the preceding U or T wave to superimpose.

After SNEs were recorded in four of the first five patients attempted, additional studies were added to the protocol. In the last 15 patients, after the SNE was identified, a quadripolar catheter was also inserted. This catheter was used to record atrial electrograms simultaneously with the SNE and to stimulate the atrium. The distal pair of electrodes was positioned against the lateral wall of the midright atrium and the proximal pair of electrodes was positioned in the high right atrium. After the SNE and two simultaneous bipolar atrial electrograms were recorded, the catheter used for the SNE was repositioned and the filter settings were changed to provide a His bundle electrogram. No intervals were measured at this point. Then, using the distal electrode pair on the quadripolar catheter for stimulation and the proximal pair for recording, atrial premature stimuli, twice diastolic threshold and 2 msec long, were introduced via a programmable Ortec stimulator and an isolation transformer after every eighth spontaneous sinus cycle. They were moved in 20-msec increments until the entire atrial diastolic period had been scanned. For processing, all electrograms were displayed on an Electronics for Medicine multichannel oscillograph simultaneously with standard ECG leads I, II and III. Using the Electronics for Medicine recorder, the data were transferred to photographic paper at a paper speed of 100 mm/sec and were then analyzed. When the spontaneous sinus cycle was interrupted by an induced APD, the following intervals were measured: (1) A1A2, the interval between the last two spontaneous sinus P waves before the APD (the sinus cycle length); (2) A1A3, the interval between the last sinus beat and the APD; (3) A2A4, the interval between the APD and the returning sinus beat; and (4) A3A4, the interval between the returning sinus beat and the subsequent sinus beat (the post-return cycle).1,5,9

The direct SACT was measured from the SNE as the interval from the onset of the upstroke slope to the onset of atrial activation (figs. 3 and 4), as suggested by Cramer et al. and Harriman et al.6,9 The average of 10 consecutive beats was used to determine the direct SACT. In five patients in whom we had doubt about the clarity of the onset of SN potential, a second investigator independently repeated the measurements and the average of both sets was used. The indirectly estimated SACT was calculated in our standard
manner\textsuperscript{1-3, 10} from $A_3A_3$ responses after APDs that reset the SN using the formula $SACT = (A_2A_3 - A_1A_1)/2$. Direct and indirect SACTs were estimated by different investigators; neither had any knowledge of the other's results. In patients in whom the post-return cycles $A_3A_4$ equalled $A_1A_1$, most investigators assume that SN suppression is not produced by the APD and that the calculation is accurate\textsuperscript{2, 3} although SN suppression could be present for only the return cycles. In patients in whom the post-return cycles...
A\textsubscript{3}A\textsubscript{4} exceed A\textsubscript{1}A\textsubscript{1}, most investigators assume that SN suppression is produced by the APD.\textsuperscript{2,3} In these latter cases the estimated SACT is presumed to be longer than the true value because the return cycle will include an increment of sinus cycle length prolongation because of suppressed SN automaticity.\textsuperscript{2,3} Some investigators have suggested that prolongation of A\textsubscript{3}A\textsubscript{4} may occasionally result without SN suppression. In these instances, A\textsubscript{3}A\textsubscript{4} is prolonged because A\textsubscript{3} occurs prematurely due to shortening of the return cycle after the APD by one or more mechanisms.\textsuperscript{2} If this effect occurred with all reset cycles, it would cause the indirect technique to underestimate the SACT. However, it occurs infrequently, is predominantly

**Figure 7.** Nonsinus, post-T-wave activity. Each panel shows simultaneous recordings of standard leads I, II and III, a reference high mid-right atrial electrogram (AEG) recorded at a filter setting of 10–500 Hz, and a second atrial electrogram recorded at a filter setting of 0.1–50 Hz. This latter electrogram is unlabeled as to location because the catheter is moved between panels A and B. (A) The unlabeled atrial electrogram is recorded I–2 cm inferior to the catheter position usually used to obtain the sinus node electrogram (SNE). After the QRS there is T-wave (outlined on the surface and intracardiac leads by bracketing reference lines) and post-T-wave (labeled U) activity. After the U wave, the baseline becomes isoelectric until atrial activation restarts the cycle. (B) The unlabeled atrial electrogram is recorded over the sinus node (SN) region. Note that there is now an upslope from the U wave to the onset of atrial activation rather than an isoelectric plateau. If panel B were the only recording made, it would be impossible to tell whether there was a SN potential or whether there was only a wide U wave with a UP merger (see text). The addition of the recording in panel A suggests that a SN potential is probably present. The cycle length in panels A and B are identical. The short line on the unlabeled electrogram in panel B indicates the time at which the U wave ended and the isoelectric plateau began in panel A. The long line indicates the onset of atrial activation. Panel B, examined alone, indicates the limitations of the technique that may be encountered when the SN potential merges with ventricular repolarization potentials from the preceding beat. However, even when the recordings in panels A and B are examined together and the presence of a SN upstroke is suggested, the onset of the SN potential cannot be identified and the sinoatrial conduction time cannot be measured. These limitations, which result from TU and SN merger or superimposition, can result when the cycle length is short and/or the PR interval is prolonged. The recordings in this figure were taken from one of our four unsuccessful studies.

**Figure 6.** Simultaneous intracardiac electrograms demonstrating the pre-P-wave localization of the sinus node (SN) potential. Lead II is recorded simultaneously with three right atrial bipolar electrograms: a sinus node electrogram (SNE), a high right atrial electrogram (HRA), and a mid-lateral right atrial electrogram (MRA). As in figure 5, the SNE is recorded with bandpass filters of 0.1–50 Hz and has an arrow over the SN potential. For emphasis, the lower end of this filtering range is indicated on the tracing. The HRA is recorded from an electrode pair 1–2 cm medial to the SNE, near the atrial appendage. Both the HRA and MRA are recorded with bandpass filters of 10–500 Hz and the lower end of this filtering range is also indicated. When the low-end filter on the SNE was set to 10 rather than 0.1 Hz (not shown), the SN potential disappeared. When the lower end filter was set to 0.1 rather than 10 Hz on either the HRA or MRA (not shown), no SN potential was seen. Simultaneous SNE and HRA using the same low-pass filters (0.1–50 Hz) show this latter finding in figure 8 (different patient).
noted after APDs only early in diastole, and thus, should only rarely affect the SACT calculations, which are generally made from cycles in the middle or latter portion of the reset plateau. Cycles beyond A2A4 were not measured in this study. The t test for paired samples was used to test the hypothesis that there is no difference between the direct and indirect estimates of SACT.

Results

Sinus Node Electrograms

We recorded the SNE in 19 of our 23 patients (83%). On the SNE, the SN deflection was recognized as a smooth, low-frequency upstroke slope before depolarization of atrial muscle began and its onset was defined by the change in slope from the post-T-wave baseline plateau (figs. 3-6). Often, the post-T-wave plateau had a mild upsloping character; Cramer et al. suggested that in the dog this may represent the SN slow diastolic (phase 4) slope. This effect occasionally made difficult the precise demarcation of the onset of the SN potential. This difficulty is analogous to the occasional difficulty in recognizing the onset of atrial activation on a His bundle electrogram with low-amplitude atrial activity. In such cases, measurements were made by two investigators (see Methods section). The pair of measurements were within 1 mm (10 msec) in all cases except two, in which interobserver variation was 2 mm (20 msec). Furthermore, the interobserver differences were not systematic, so the average difference of all such measurement pairs was not significant. Occasionally there was beat-to-beat variation in the configuration of the SN deflection; this was most marked in patients with significant sinus arrhythmia. Moving the recording electrodes at least 1 cm usually caused loss of the potential.
Although the recordings were usually reasonably stable once the SN potential had been localized, in some patients, as expected, baseline drift occurred at the low filter frequencies. Such drift seemed to be related to respiration or catheter movement/pressure, although other factors may also be responsible. When spontaneous APDs occurred, they were also frequently followed by baseline drift. Presumably, this was caused by mechanical factors or alterations in atrial volume that affected the position of the catheter tip in relation to the atrial wall or the pressure of the electrode against the atrium. When baseline drift was present in a single lead, the SN deflection in that lead could be recognized with certainty only if there was constancy of the potential despite baseline fluctuations and especially by a positively sloped pre-P-wave potential despite negatively sloping baseline (see second complex, figure 5B and figure 8). Baseline drift did not simulate the SNE but did make it impossible to record the SNE in two cases. Baseline drift may be the major limitation of the current technique.

In the five patients in whom the effect of changing the low-end filter was examined, changing to 1 Hz caused substantial diminution of the potential in four patients and loss in one; changing to 3 Hz caused loss of the potential in all five patients.

In the 15 patients in whom the SNE was recorded simultaneously with the other atrial electrograms, no deflection occurred as early as the SN potential, either with bandpass filters of 0.1–50 Hz or 10–500 Hz (figures 6–8).

In 10 patients with sinus arrhythmia, the SN deflection, as expected, was coupled to the succeeding P wave rather than to the preceding T wave, as a U wave might be. In each case the coupling of the SN deflection to the ensuing P wave remained stable, while the coupling to the preceding T wave varied appropriately with the cycle length. That is, with longer sinus cycle lengths there was a longer plateau between the T-wave terminus and the SN deflection, and vice versa. In one patient with sinus arrhythmia, the SNE was recorded during atrial pacing (fig. 8). Pacing was performed at several cycle lengths equal to or just shorter than the shortest spontaneous cycle length. When the paced cycle length was slightly longer than the shortest of the spontaneous cycles (fig. 8B), there was stable isorhythmic dissociation between the SN deflection and the atria. When the paced cycle length was further shortened (fig. 8C), stable SN capture occurred and the SN deflection no longer preceded the atrial depolarizations, although it resumed again after pacing.

In one patient, spontaneous 2:1 SA exit block occurred during the recording period (fig. 9). The non-conducted beat had a SN deflection without associated atrial activation. The configuration of the

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**Figure 8. Atrial pacing and the sinus node electrogram.** Atrial pacing was performed in one patient with sinus arrhythmia. Spontaneous cycle lengths were 700–820 msec. They tended to be at the longer end of the range when she was resting unperturbed and at the shorter end when she was aware that stimulation was being or had just been performed. Each panel shows standard lead II, a high atrial electrogram (ATR) at 10–500 Hz, and a sinus node electrogram (SNE) at 0.1–50 Hz, the same filtering frequencies as in the previous figures. To avoid further crowding of this already complex figure, however, we did not label the low-end filter frequency directly on the tracing as in figure 6. During atrial pacing stimulation artifacts are labeled S. Time lines are 1000 msec apart. Consecutive atrial cycle lengths are labeled horizontally in msec. A bar in panel A (first cycle) shows this measurement. Additional vertical reference timing bars have been drawn in panels A and C. They mark the interval from the onset of prior atrial activation to the indicated point in time. They are 50 msec apart and the first in each set is at 600 msec into the cycle. Panel A was taken at rest. Note that despite some baseline drift, the configuration of the sinus node (SN) deflection is stable. Panel B was taken during atrial pacing at a cycle length of 720 msec. Note that there is isorhythmic dissociation between the SN deflection (marked with arrows) and the paced depolarizations. A glimpse of the SN deflection is seen before the first, second, third, and perhaps the fourth complexes. It is absent before the fifth. During the last two cycles, the sinus cycle length shortens and the SN deflection reappears and captures the atria. Panel C was taken at the termination of atrial pacing at a cycle length of 700 msec. During pacing, SN deflections no longer precede any of the atrial depolarizations (although there may be a suggestion of its presence before the third complex). After pacing the SN deflection reappears. Above the last cycle to the right, the interval from the onset of atrial depolarization to the onset of the next SN deflection is specifically identified (A-SN). This A-SN interval is shorter than was the 700-msec paced cycle length. Thus, the absence of the SN deflection during pacing at a 700-msec cycle length suggests that either the sinus cycle length was prolonged so that the A-SN interval was greater than 700 msec or that the SN was captured during pacing. The latter is more likely since the SN deflection was absent for 30 seconds of pacing. In panel C, note also that the first complex after pacing, which terminates the sinus recovery time (SRT), is associated with a different atrial electrogram configuration than is present during subsequent cycles and with absence of the typical SN deflection despite a "sinus" P wave. This may represent a transient change in the pacemaker focus within the node and/or a transient change in exit pathway within or from the node; or it may represent loss of the SN deflection due to displacement of the catheter secondarily to increased atrial filling during this prolonged cycle. However, that at the end of the TP interval during this SRT cycle, after the 800-msec time bar, there is a slight negative deflection preceding atrial activation. If this is not artifact, it may favor one of the former two explanations rather than the latter.
SN deflection resembled the configuration of the SN deflections on the extracellular SNE noted by Cramer et al.5 during SA block. In three other patients, carotid sinus massage produced SA block; however, this maneuver also was associated with baseline drift. The drift makes it difficult to comment on the exact configuration of the SN potential, but the mechanism of the pause in rhythm could be identified as SA block rather than SN arrest. In several other patients carotid sinus massage merely lengthened the SACT and slowed the sinus rate (fig. 10) without inducing SA arrest or exit block.

Sinoatrial Conduction Times

In 16 patients with normal SN function, the directly recorded SACT ranged from 46–116 msec (average 90 ± 18 msec). In three patients with sick sinus syndrome, directly recorded SACT ranged from 110–126 msec (average 120 ± 9 msec). In 15 of these 19 patients SACT was estimated by the atrial premature stimulation technique (fig. 11). Each had an A2A3 plateau during SN reset, i.e., stable return cycle lengths after midcycle APDs. In 11 of the 15 patients, the post-return cycle A3A4 equalled the mean sinus cycle length A1A1. Conversely, in four of the 15 patients, A2A3 exceeded A1A1, suggesting SN depression by the APD. In 10 of the 11 patients in whom the A3A4 was the same as the A1A1, the mean difference between the directly recorded SACT and the SACT estimated by the atrial premature stimulation technique was 1.8 ± 5.6 msec (NS). In patient 14, A1A1 was short and the SN deflection merged with the preceding T wave. In this patient, whose data are not included in figure 10, the direct SACT could only be determined as greater than 75

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**Figure 9.** Two-to-one sinoatrial exit block. ECG leads I and II, a sinus node electrogram (SNE) and a right atrial electrogram (ATR) are recorded simultaneously. The atrial electrogram is recorded in the high right atrium near the atrial appendage. Bandpass filters for both the SNE and ATR are 0.1–50 Hz. The SNE recording has been inverted by convention (see text) so as to display the SN potential as upright. Note the sinus node (SN) deflection (arrow) on the SNE during the blocked cycle while only a pause is seen on I, II and ATR. If the identified deflection on the SNE were anything other than a localized SN event, such as a T or U wave, it should probably be visible on the immediately adjacent ATR and on the SNE. After block, the SN deflection of the first depolarization is slightly altered. Slight change in the pacemaker focus or exit pathway within the SN may be the cause.5,4 After the blocked complex, baseline drift is recorded on both the SNE and ATR. This may be caused by catheter movement associated with the change in atrial filling pattern due to the prolonged cycle length.

**Figure 10.** The effect of carotid sinus massage on sinoatrial (SA) conduction. The sinus node electrogram (SNE) is recorded simultaneously with ECG leads I and II before and during carotid sinus massage (CSM). (A) The last beat before and the first beat during CSM. (B) Two beats during the fourth second of CSM. Dark bars indicate the onset of each sinus node deflection (upstroke slope). Between panel A and panel B the cycle length increases from 1070 to 1165 msec and the duration of SA conduction increases from 126 to 207 msec. Compare these recordings to panels A and B of figure 1.
msec; the indirect estimate of SACT was 96 msec. As expected, the estimated SACT exceeded the value obtained directly from the SNE electrogram in the four patients in whom $A_3A_4$ was greater than $A_1A_1$. However, the mean difference was less than 15 msec. We gave two patients 0.75 mg of i.v. digoxin during the study. In both, the direct SACT and indirectly estimated SACT increased in parallel during the 45 minutes after the digoxin dose.

**Discussion**

Our transvenous catheter method for recording the SNE stems from the work of Cramer et al. They developed a method for obtaining extracellular SN potentials, initially in vitro and then in vivo. In the in vitro studies the SN deflection was identified extracellularly only when the electrogram was recorded in the region of the SN and only when the recording was highly amplified and was recorded with low-pass filters. Its origin was verified by simultaneous transmembrane action potentials and by its behavior during induced SA block. In the in vivo studies the pre-P-wave SN potentials could be recorded with conventional electrode catheters positioned over the SN in the beating heart. The SN upstroke slope so recorded always began before the onset of the P wave and strongly resembled, in timing and configuration, the SN potential obtained in the in vitro studies. This potential was only obtainable in the region of the SN. Progression from the beating dog heart to the human adult catheterization laboratory then went quickly.

We initiated the present study in the adult catheterization laboratory to develop a method for recording SNEs in intact, awake, adult patients.

We have shown that SNEs may be obtained in most adults using our technique. Thirty minutes or less is usually required for this procedure. The electrograms recorded with catheters in man have a configuration and behavior similar to those in prior in vitro studies. While Cramer et al. believed that in vivo extracellular SN potentials were a true recording of the SN pacemaker tissue activation, the extracellular, catheter-recorded potential might include intra- and perinodal conduction elements as well. The ability to record during SA block, however, both in vitro and in vivo, makes it likely that activity within the SN proper forms a substantial part of the SN potential and that activity outside the node does not.

Our technique does have limitations. Using the low-pass filters required, baseline drift may pose a significant problem, particularly in patients who are tachyarrhythmic, have large atria, and/or have frequent atrial ectopic activity, making the recording impossible to obtain. Respiratory activity and atrial ectopy can cause baseline fluctuations. Simultaneous display from multiple leads over the SN and simultaneous referencing to other atrial electrograms helps identify the SN potential in these circumstances. Atrial enlargement or atrial ectopy may make catheter placement and/or stability against the atrial wall difficult. Under these circumstances, the SNE may be impossible to record. When the SN depolarization is contiguous with the T or U wave, its onset may be difficult to define and, unless additional atrial electrograms clearly reveal the end of ventricular repolarization potentials, it may not be possible to be certain that the SN potential is even present. Fortunately, most patients who are being evaluated for sinus node function have slow heart rates.

Beat-to-beat variation in the configuration of the SN deflection does occur. It seems most marked in patients with significant sinus arrhythmia, although we have little experience with this problem. When similar morphologic variation was seen in isolated atrial preparations, it represented changes in pacemaker focus or exit pathway within the node. Occasionally it is difficult to recognize precisely the onset of the SN upstroke slope. However, interobserver variation in defining the point of takeoff is small. Interelectrode distances shorter than the 1 cm used for this study might more clearly define the SN potential in such cases. Unipolar recordings could likewise offer some advantages, and are being studied. However, in the animal laboratory, the method used in our study appeared reasonably effective for obtaining the SN electrogram.

The SNE obtained as above can usually provide direct information on SA conduction. Unless the cycle length is short, the antegrade SA conduction time can be directly measured on the SNE. When the recording is stable, and no U wave is present, and significant sinus arrhythmia is absent, only a single catheter is necessary to identify the SN potential and measure the SACT. Although the time required to obtain the SNE...
may be only slightly shorter than that necessary to perform the atrial extrastimulation procedure, the time for data processing directly from the SNE and, therefore, determination of the SACT value, are considerably shorter in most patients. However, when conditions such as these are present, additional atrial electrograms are needed before the SNE can be interpreted with confidence. In the latter circumstance, the direct measurement of SACT may be considerably less practical than the indirect technique.

If significant sinus arrhythmia is present, both the direct and indirect techniques for determining the SACT could be inaccurate. Marked sinus arrhythmia may bias the direct recordings if the SN potential is only definable at longer cycles. However, marked sinus arrhythmia may also make the $A_3A_5$ vs $A_1A_3$ plot very difficult to interpret.

Both the direct and indirect methods for determining SACT may be affected by measurement error, as precise definition for the onset of the SN potential on the SNE and the atrial depolarization on the standard atrial electrogram may occasionally be difficult. However, our experience, though limited, suggests that interobserver variation is relatively small in both instances and that the mean error over a series of measurements is probably not significant. The direct SACT determination from the SNE is probably accurate to about 10-20 msec; however, the same may be true for the measurements used in the atrial extrastimulus method. Even a 10-msec error is only about a 10% error, because the normal SACT is usually about 80 msec.

Despite these limitations, the direct and indirect estimates of SACT correlate quite well. We found, for example, that the atrial premature stimulation method for estimating SACT is quite accurate when the return cycles are stable during the zone of SN reset and there is no evidence for SN suppression by the APD. That is, when $A_2A_3$ was relatively constant during the SN reset zone and $A_3A_5 = A_1A_3$, the estimated SACT and directly recorded SACT were virtually identical. When $A_2A_3$ was steady but $A_3A_5$ exceeded $A_1A_3$, suggesting SN suppression by APD, then, as predicted the indirect method overestimated the directly measured SACT.

It therefore appears to be possible to record the SNE in most adults using readily available equipment. The clinical application of the technique may have some limitations. Perhaps the most important use for the human SNE is in accessing the validity of indirect stimulation techniques for determining SN function. This is of major importance, considering the number of pharmacologic and physiologic studies in the past decade that have been based on the indirect techniques. Our data indicate, for example, that those assumptions that underlie the indirect method for assessing SACT are reasonable. If considerable SN depression, pacemaker shift or refractoriness were to result from single APDs, a much poorer correlation would be expected between the indirect and direct SACT estimates than we obtained. This conclusion would probably not be affected even if the direct measurement were in error by 10%. We believe that the SNE will be equally useful in assessing situations in which the atrial premature stimulation technique may be imprecise for estimating the SACT and in testing the assumptions that underlie the interpretation of SN responses after overdrive stimulation. The technique might also be useful in analyzing the relative contributions of changes in SN automaticity and SA conduction during a variety of physiologic states and pharmacologic interventions. The SNE is therefore a promising technique for evaluating SN function and should permit a direct test of many of the conclusions about diagnostic tests or pharmacologic actions that have been based on indirect measurements.

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