A New Method for Measurement of Sinoatrial Conduction Time

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SUMMARY This study describes a new method (NM) for estimation of sinoatrial conduction time (SACT), which utilizes constant atrial pacing (AP) instead of the premature atrial beats (PABs) used in the method reported in 1973 by Strauss et al. The SACTs were obtained by both methods in 20 patients. The SACT by the Strauss method (SM) was calculated as A3A2 minus A1A2. The NM consists of high right AP for a train of eight consecutive beats at rates ≤10 beats/min faster than the sinus rhythm. The interval between the last paced atrial electrogram (AP) and the first escape atrial electrogram (A) of sinus origin (Ap-A) was measured along with several post pacing sinus cycles. The SACT by the NM was calculated as follows: SACT = Ap-A minus A1A2. The effect of AP at higher rates was also analyzed. In two patients, the SACT with the SM could not be defined, as all the A1A2 intervals were fully compensatory; with the NM the SACT was 217 and 320 msec. In the remaining 18 patients the SACT was obtainable by both methods. With SM, the SACT ranged 105-452 msec (mean 219 ± 102 sd) and with the NM it was 85-492 msec (mean 201 ± 112 sd), and the difference was statistically significant (P = 0.0162). The coefficient of correlation between the two methods was r = 0.97. During AP at faster rates, a rate related increment in Ap-A intervals and also post pacing sinus cycles was noted. This study describes a new and simple method for measurement of SACT in man.

THE POSSIBLE EXISTENCE of sinoatrial (SA) block in man was first suspected at the beginning of this century.2 Second degree SA block was interpreted on the basis of long atrial pauses.3 However, at that time, there were no clinical means available to diagnose first degree SA block. In recent years, intracardiac catheter recordings in conjunction with electrically induced premature atrial beats have been used to elucidate first degree SA block in man. The effect of electrically induced premature atrial beats (PABs) in mammalian hearts was studied by Cushnry in 1897.4 A few years later Wenckebach evaluated the effect of PABs on the sinus rhythm.5, 6

He postulated that an atrial response to a PAB would be determined by the retrograde and antegrade conduction times between the sinus node (SN) and the atrium and the sinus return cycle.4 Almost six decades later, others utilized the effect of PABs in patients with atrial parasystole to estimate sinoatrial conduction time (SACT).7, 8 Based on this previous work, Strauss et al. proposed the use of programmed PABs to estimate the SACT.9

In this study we report a new, simpler method for the estimation of SACT which utilizes constant atrial pacing (AP) instead of the PABs.

Materials and Methods

Twenty patients with sinus rhythm were studied during diagnostic electrophysiologic studies. The SACT was measured by the two methods: the method of Strauss et al. (SM) and the new method (NM), which is described below. The patients ranged in age from 30-75 years, with a mean age of 57.8 years (table 1). The sinus cycle length was >1000 msec in three cases (11, 16 and 19), and was <1000 msec in 17
patients. Four patients (cases 9, 13, 16 and 18) exhibited sinus arrhythmia with a variation in spontaneous cycle length >120 msec. The variations were 200, 130, 150 and 165 msec, respectively. One patient (case 18) exhibited ECG findings consistent with spontaneous periods of 2:1 SA block both before and during the electrophysiological study. The corrected SN recovery time was normal (<525 msec) in all except three patients (cases 11, 18 and 19), in whom it was 614, 3000 and 2375 msec, respectively. The QRS duration was normal (≤0.10 sec) in 18 and prolonged in two patients. The P-R interval was prolonged (>0.20 sec) in seven of the 20 patients.

The patients were receiving no cardioactive drugs at the time of the study; the drugs had been discontinued for an interval exceeding three drug half-lives. No patient had had a recent myocardial infarction, i.e., within eight to 10 weeks before the study, nor were any in the immediate postoperative period. All patients were studied in the postabsorptive state and were premedicated with 100 mg of pentobarbital (Nembutal), administered intramuscularly 30 minutes before the study. Our previous observations indicate that this dosage has no direct effect on the heart rate or rhythm. Informed consent was obtained from each patient.

A quadrupolar electrode catheter (with ring electrodes 10 mm apart) was placed in the right atrium (RA) so that the proximal electrode pair was located at the junction of the superior vena cava and the high RA for recording high right atrial bipolar electrograms, and the distal pair was used for atrial stimulation. Another bipolar electrode catheter was introduced percutaneously via a femoral vein and placed in the His bundle (BH) region for recording BH electrograms. Multiple ECG leads in combinations of L-1, L-2, L-3 and Vr, or L-1, aVF and Vr, were recorded simultaneously with high right atrial and BH electrograms. The standard ECG leads and the bipolar electrograms were displayed at frequency settings of 0-25 Hz and 30-250 Hz, respectively. All recordings were made at paper speeds of 50 and 100 mm/sec on the Electronics for Medicine (VR-12) recorder. Atrial stimulation studies were performed at double the diastolic threshold with stimuli 2 msec in duration. During measurements of SACT by the two methods, the position of the quadripolar catheter was fluoroscopically and electrocardiographically monitored and kept constant throughout each study. This enabled an accurate measurement and comparison of the SACTs by the two methods from the same and constant right atrial sites.

Programmed PABs were introduced during sinus rhythm for estimation of SACT as previously described by Strauss et al. The following four consecutive atrial intervals were measured: 1) spontaneous cycle length (A2A3), i.e., the last two spontaneous sinus P waves immediately preceding the PAB; 2) the test cycle (A1A2); 3) the return cycle (A2A3); and 4) the post return cycle (A3A4). In addition, the subsequent spontaneous sinus cycles (third to seventh) were also analyzed. The number of various intervals analyzed corresponded to the number of programmed PABs induced in any given patient. However, only the responses to PABs in the latter third of zone II (zone of SN reset) were utilized for calculating mean values of A1A2, A2A3 and A3A4 and the number of these cycles ranged from 12-32 (table 1). The A1A2 and A2A3 intervals were normalized by dividing them with the A1A3 cycle. The SACT was calculated according to the most recently described criteria of Strauss et al. (SACT = A2A3-A1A2). The interval thus obtained represents the sum total of the conduction time into and out of the sinus node.

New Method For SA Conduction Time

Following the sequence of PABs, sinus rhythm was observed for 2 minutes without any interventions. Immediately preceding AP by the NM, 10 sinus cycles were recorded to obtain the mean cycle length. In each patient, constant AP was performed at a rate slightly faster (≤10 beats/min) than the control sinus rhythm for a train of eight consecutive beats. On cessation of AP the recordings were continued for the subsequent eight or more spontaneous SN cycles for purposes of analysis (fig. 1). This procedure was repeated four to five times at the same pacing cycle length.

The SACT by the NM was calculated by deducting the mean sinus cycle length from the interval between the last paced atrial electrogram (Ap) and the atrial electrogram (A) of the first escape SN cycle. This interval (Ap-A minus A1A3) represents the sum total of the conduction time into and out of the SN if there is no depression of its automaticity. The measurement of SACT during several repeat observations at the same AP cycle length was used to calculate the mean SACT and provided the range of variation in reproducibility.

AP was similarly performed at two or more additional pacing rates to assess the effect of pacing rate on the data obtained. Immediately preceding each change in AP cycle length, 10 spontaneous SN cycles were recorded to obtain a new mean SN cycle length.

The study of SACT by the two methods (SM and NM) was generally completed in less than 15 minutes. No arrhythmias or other complications occurred in any patient either during or after the stimulation studies. Statistical evaluation was performed by use of the t test for paired data, whereas regression analysis was done by the use of a computer program.

Results

The SACTs could be measured with the NM in all 20 patients and with the method of Strauss et al. in only 18 patients. In two patients the SACT could not be defined with SM because all of the A3A4 responses were fully compensatory.

In the 18 patients, the SACTs by the SM ranged 105-452 msec (mean = 219 ± 102 s) and with the NM ranged 85-492 msec (mean = 201 ± 112 s). The differences in SACTs calculated by the two methods were statistically significant (P = 0.0162).
Table 1. Comparison of Sinoatrial Conduction Time by the Strauss and New Methods

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Abbreviations: A1A1 = spontaneous sinus cycle; A2A4 = return cycle; A3A4 = post return cycle; AP = atrial pacing; NM = new method; Ap-A = interval between the last atrial paced (Ap) A wave and the first spontaneous sinus A wave; CL = cycle length; ND = nondefinable; N = number of cycles used to calculate mean; SACT = sinoatrial conduction time; SC = sinus cycle; SM = method of Strauss et al. All measurements are provided in msec.
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The SACT with the NM was identical to that of SM in one patient (case 8) and was longer by 21, 6 and 40 msec in three other patients (cases 13, 14 and 16). In the remaining 14 of the 18 patients, the SACTs with the NM were shorter than by the SM by a range of 2–77 msec (figs. 1 and 2) (table 1). In general, even in patients with a long SACT or SN dysfunction, the SACTs with the NM were shorter than with the SM. The coefficient of correlation between SACTs by the two methods was $r = 0.97$ (fig. 3).

In the remaining two (cases 12 and 18) of the 20 patients, the SACT could not be calculated by SM as

**FIGURE 1.** Estimation of the sinoatrial conduction time (SACT) by the new method (NM), case 5. A) Shows control recordings of sinus rhythm with a mean sinus cycle length (CL) of 730 msec. B) Shows termination of atrial pacing (AP) after eight beats at a CL of 650 msec. The interval between the last paced atrial electrogram (Ap) and the first escape A wave of sinus origin is 850 msec. The SACT is calculated by deducting the mean sinus cycle length from the Ap-A interval (850–730 = 120 msec). C) Shows a repeat measurement of SACT with the NM at a pacing CL of 650 msec. D) and E) Despite a faster AP rate (CL = 550 msec), the SACT (110–120 msec) is similar to that in panels B and C. Note that the subsequent post pacing sinus cycles are similar to those of control. This excludes depression of sinus node automaticity. PI = pacing impulse; A = atrial electrogram; RA = high right atrial bipolar electrogram; BH = bipolar recordings from the His bundle (BH) region. Time lines on this and subsequent figures are at 1-second intervals unless otherwise indicated.

**FIGURE 2.** Shows that the estimation of SACT by the Strauss method (SM) is longer than with the new method (NM) in the same patient as in figure 1. A–F) Show the return cycles ($A_1A_2A_3$) following premature atrial beats ($A_1A_2$) at various coupling intervals. On top of each panel the sum total of $A_1A_2 + A_2A_3$ intervals is given. Sinus node reset can be clearly defined only in panel F, as the rest of the premature atrial beats were fully compensatory.
the onset of zone of SN reset (zone II) was not clearly definable. The A2A3 responses after PABs were fully compensatory and suggested SN entrance block. In one patient (case 12) there was no clinical, ECG or electrophysiological evidence of SN dysfunction. In this patient, the SACT by the NM was determined to be 217 msec and the corrected SN recovery time was 430 msec. The other patient (case 18) occasionally exhibited spontaneous periods of 2:1 SA block during sinus rhythm. Following PABs all of the A2A3 responses were fully compensatory (fig. 4). The SACT by the NM was 320 msec and the longest corrected SN recovery time was 3000 msec (fig. 5).

In this study the fluctuations in spontaneous sinus cycle length ranged from 10–200 msec (mean = 73). In 18 patients, the A2A3 intervals clearly definable to be in the last third of zone II responses and utilized for estimation of SACT with SM showed fluctuations ranging from 10–140 msec (mean = 46). On the other hand, during repeat measurements of SACT with NM at a constant AP cycle length, the Ap-A intervals fluctuated by a range of 0–110 msec (mean = 38). The fluctuations in mean Ap-A intervals were shorter by 48% compared to the spontaneous fluctuations in sinus cycles, and the difference was statistically significant (P = <0.005). Although the fluctuations in mean Ap-A intervals were shorter by 17% than those noted in the A2A3 intervals, the difference was not statistically significant.

A comparison of the mean sinus cycle length used for calculating SACTs by the SM and NM showed a minimal but definite difference in 16 of the 20 patients, and in four it was essentially unchanged (≤5 msec). During SACT measurements with the NM, the mean sinus cycle length was longer by 11–72 msec (mean = 31) in 10 and shorter by 10–40 msec (mean = 19) in six patients, compared to that during SM (table 1).

**Post Pacing Sinus Cycles**

Following atrial stimulation the subsequent spontaneous sinus cycles (numbering 2–7), were analyzed both during the SM and the NM. With the SM the post return sinus cycles (A2A4) were longer than the mean sinus cycle length by a range of 0–11% (mean = 2.1%). During the NM the second post pacing sinus cycles were longer by 0–8.6% (mean = 2.21%) than the mean sinus cycle length. The remaining post pacing sinus cycles (three to seven) were essentially similar to the control sinus cycles.

**Effect of Pacing Rate on SACT**

Most of the patients exhibited a slight lengthening of Ap-A intervals when the AP rates were faster than the sinus rhythm by >10 beats/min. Occasionally, no appreciable differences were noted (cases 6 and 7) (table 1). The pacing rate-related increments in Ap-A intervals ranged from 10–142 msec. At times, this increment could not be appreciated if data after a single train of atrial paced beats was inspected. These increments usually became apparent only when mean values of repeated reproductions of Ap-A intervals after AP at a faster but constant rate were compared.
FIGURE 4. A patient (case 18) with spontaneous but very occasional periods of 2:1 sinoatrial block in whom sinoatrial conduction time (SACT) could not be defined by the Strauss method (SM). A–H) The induced premature atrial beats (A1A2) are followed by return cycles (A2A3) which are more than fully compensatory and suggest sinus node entrance block. The sum total of A1A2 and A2A3 intervals is listed on top of each panel.

FIGURE 5. Measurement of sinoatrial conduction time (SACT) with the new method (NM) in the same patient as in figure 4. A) Shows spontaneous sinus rhythm with an occasional period of 2:1 sinoatrial block. B) Shows SACT (310 and 330 msec) during repeat measurements with the NM after atrial pacing at a cycle length of 700 msec.
with those at a comparatively slower rate. At faster pacing rates, the increase in Ap-A intervals was associated with a slight but obvious prolongation of the post pacing sinus cycles which gradually returned to control levels by the sixth or seventh beats. At faster pacing rates, the duration of Ap-A and of the subsequent post pacing sinus cycles appeared to be directly related to the pacing rate, and the increments were more pronounced in patients with SN dysfunction.

Discussion

The method for measurement of SACT as previously described by Strauss et al. is based on depolarization and reset of the SN by induced PABs.12 The NM is similarly based on artificial depolarization of the SN; however, it utilizes constant AP at rates slightly faster than the spontaneous sinus rhythm (≤10 beats/min) which are sufficient to capture the SN but do not depress it appreciably.

This is supported by our results, as in the majority of patients the SACT with the NM was slightly shorter than with the SM. With the NM, the second post pacing sinus cycle showed only a slight prolongation (a mean increment of 2.2%) compared with the mean sinus cycle length. This increment is similar in magnitude to that noted following PABs (2.1%) in the post return cycle (A2A3) with the SM. Others have also noted that the A2A3 intervals are generally longer during the SM by 3–4% than the mean sinus cycles.12, 14–19 Others have indicated that an analysis of A2A3 intervals during the SM does not assist in evaluating or excluding SN depression by the PABs.15

In the NM, AP was performed for a train of eight consecutive beats. The number of eight beats was arbitrarily chosen for the following reasons: 1) several paced beats may be needed to capture the SN, as the pacing rate was only slightly faster than the sinus rhythm; 2) if the SN is initially captured prematurely during early diastole, by the first pacing impulse, additional paced beats would be required to permit return of sinus cycles to near control levels.

The results of this study clearly demonstrate the feasibility of measuring reproducible SACTs by the NM. These values are similar to that obtained with the SM. However, further experimental studies are required to compare these indirectly measured values to those obtained directly. In this study only a limited number of patients (four out of 20) showed sinus arrhythmia. Therefore, the efficacy of the NM for measurement of SACT in patients with sinus arrhythmia cannot be fully assessed. The SACTs by the NM were obtained in all cases, but failed to accomplish this goal in two patients (cases 12 and 18) with the SM. In the latter patients, all of the A2A3 intervals were fully compensatory and indicated SN entrance block (fig. 4). In these patients, the possibility of entrance block is excluded by the demonstration of longer corrected SN recovery time which could not have been obtained without SN suppression or penetration by the AP impulses. These observations suggest that SN entrance block may be misinterpreted when PABs are used for estimation of SACT.

The reason for failure to define zone II or reset of the SN with the PABs is probably an inherent problem in the use of PABs, since: 1) In some patients with an increase in prematurity, the atrio-sinus conduction time may be proportionately lengthened, and thus the premature impulse may never reach the dominant pacemaker in the SN,20 and 2) it is possible that the premature impulse in reality may reach and reset the SN; however, it may not be obvious from the A2A3 intervals if the conduction times are disproportionately longer on either limb (conduction into or out of the SN). Despite SN reset, a marked degree of conduction delay may result in A2A3 intervals which are fully compensatory. Similar mechanisms may explain other cases in which the SACT cannot be defined. Difficulties in defining SACTs with SM are quite frequent, as reported in a recent study where in five out of 17 cases zone II responses could not be defined.18

Additional problems exist in estimating SACT by using PABs: 1) The transition from compensatory to the constant and less than compensatory A2A3 cycle is gradual. This adds a significant margin of error in estimation of SACT. The defining of onset of zone II responses is further complicated by the spontaneous fluctuations in sinus cycle length. In the same patient the onset of zone II responses may easily vary by as much as 40–50 msec (mean 42). 2) The shortening of SN-SN intervals resulting from PABs cannot be detected from A2A3 intervals.15, 20–22 3) However remote, there is a theoretical and to some degree a real concern for inducing atrial arrhythmias during estimation of SACT by the PABs. 4) Estimation of SACT with PABs is cumbersome and requires a large number of measurements. This has led some investigators to devise a computer program for the analysis of SACT. In addition, the SM requires the use of a special programmable stimulator. These problems in measuring SACT with SM listed above are remedied by the NM, as they are a consequence of using PABs. The NM does not utilize PABs, and instead, constant AP at rates only slightly faster than the sinus rhythm is used. The cumbersome problem of estimating SACT with PABs is also solved, as only a few beats need be measured and any ordinary pulse generator should suffice with the NM.

The magnitude of fluctuations in A2A3 intervals used for estimation of SACT with SM cannot be fully detected, as the point of transition between zone I and II responses cannot always be precisely demarcated. In this study, only those A2A3 intervals which could not be confused and were clearly definable to be less than fully compensatory, within the last third of zone II, were used to calculate SACT by the SM. Therefore, the range of fluctuations in A2A3 intervals with SM (as listed under results) may have been artificially minimized by the arbitrary elimination of transitional A2A3 responses.

With the NM there is no difficulty in defining the range of fluctuations in Ap-A intervals, as none of the
measured Ap-A intervals were eliminated. Following AP at a constant cycle length, several repeat Ap-A responses were used to determine the range of fluctuations and to obtain a mean of Ap-A intervals which provides a valid comparison with a mean of spontaneous sinus cycles (A1,A2). Thus, the influence of fluctuations in both latter intervals should be negated in a realistic fashion. The degree of fluctuations in mean Ap-A intervals were significantly (P = 0.0005) shorter than that noted in the mean spontaneous sinus cycle length. This suggests a possible reduction in spontaneous sinus fluctuations by a regular depolarization of the SN with constant AP.

Our studies demonstrate a pacing rate related prolongation in Ap-A intervals with AP rates faster by >10 beats/min than the sinus rhythm. This lengthening in the Ap-A interval may be explained by either or both of the following explanations: 1) that analogous to the atroventricular node the conduction time into the SN is rate-related; and 2) SN automaticity is depressed by AP. At faster AP rates in some patients the subsequent post pacing sinus cycles were also slightly prolonged in association with a prolongation of Ap-A intervals. This suggests that at fast pacing rates SN automaticity is depressed and contributes not only to the duration of Ap-A interval, but also to the duration of the post pacing SN cycles. In view of these observations it is recommended that for estimation of SACT with the NM, the AP rates used should not exceed the spontaneous sinus rate by ≥10 beats/min. This should be given special consideration when SACT is being analyzed in patients with SN dysfunction.

In summary, this study describes a new method for estimation of SACT by an alternative approach which is simpler and quicker. We hope this report will stimulate microelectrode studies by others for an independent validation of this technique.

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