The Estimation of Sinoatrial Conduction Time in Rabbit Heart by the Constant Atrial Pacing Technique

Augustus O. Grant, M.B., Ch.B., Ph.D., Gilbert Kirkorian, M.D., David G. Benditt, M.D., and Harold C. Strauss, M.D., C.M.

SUMMARY This study compared estimates of sinoatrial conduction time (SACT) obtained by constant atrial pacing (CAP) and premature atrial stimulation (PAS) with measured SACT in isolated rabbit right atrial preparations. Transmembrane potentials and surface electrograms were recorded from the sinus node and crista terminals, respectively. The crista terminals was paced 5, 10 and 15 beats/min faster than the spontaneous sinus rate with a train of eight pulses. Estimate of SACT by CAP was taken as the difference between the first atrial return cycle and the mean spontaneous cycle length. SACTs at 5, 10 and 15 beats/min faster were 76 ± 10, 86 ± 10 and 96 ± 10 msec (mean ± SEM; n = 12), respectively; correlation coefficients with the true SACT were 0.7, 0.54 and 0.4. Consecutive determinations of SACT by PAS and CAP in the same preparation (n = 6) at 10 beats/min faster gave SACTs of 86 ± 13 and 79 ± 14 msec, respectively, compared with true SACTs of 79 ± 10 msec. Shortening of sinus node action potential, depression of automaticity and shifts in the site of the primary pacemaker contributed to the errors in both techniques. Estimation of SACT by CAP may be further complicated by failure of sinus node capture. Principles to minimize some of these errors are also presented.

DIRECT RECORDING of the electrical activity of the sinus node by catheter techniques is in a preliminary stage of development. The currently used techniques of assessing sinus node automaticity and sinoatrial conduction are based on inferences drawn from the atrial cycle length. In 1973 Strauss et al. proposed programmed atrial stimulation for estimating sinoatrial conduction (SACT) in the in situ heart. However, when such estimates were compared with direct measurements of SACT in the rabbit heart in vitro, a number of confounding influences not immediately apparent from the atrial electrogram became evident. Klein et al. pointed out how misleading the atrial electrogram can be with regard to activity in the sinus node. Recently, Narula et al. suggested that SACT can be estimated from the atrial return cycle after a brief period of constant atrial pacing, and pointed out advantages over the premature atrial stimulation technique. We thought it important to validate the estimates of SACT obtained by constant pacing with direct measurements in the rabbit heart in vitro. We also compared SACTs obtained by constant pacing with those obtained by premature atrial stimulation in the same heart. Our results indicate that both estimates are subject to similar errors and can occasionally be misleading, but principles to minimize some of these errors are also presented.

Methods

Fourteen rabbits that weighed 2–4 kg were anesthetized with sodium pentobarbital 25–50 mg/kg i.v.; heparin 1000 U i.v. was then given. The hearts were rapidly excised and dissected in cool, modified Tyrode solution. The dissected preparation consisted of the area between the interatrial septum and the lateral margin of the crista terminalis, and the superior and inferior venae cavae, excluding the atrioventricular (AV) node. The preparation, endocardial surface uppermost, was pinned to the waxed base of a 5-ml Lucite tissue chamber and superfused with oxygenated Tyrode solution at 5 ml/min.

The modified Tyrode solution had the following composition (mM): NaCl 130.0, KCl 4.0, NaH2PO4 1.8, CaCl2 2.7, MgCl2 0.5, and NaHCO3 18.0. The solution was bubbled with a 95% O2-5% CO2 gas mixture. The temperature of the bath was monitored with a telethermometer (Yellow Springs Instrument Co) and maintained at 36 ± 0.2°C.

The transmembrane potential was recorded with glass microelectrodes filled with 3M KCl and that had tip resistances of 20–40 MΩ. The microelectrode was coupled by an Ag/AgCl wire to a high-input impedance amplifier having input capacitance neutralization. A bipolar electrogram was recorded with Teflon-coated silver wires from the upper end of the crista terminalis. The interelectrode distance of the bipolar electrodes was approximately 0.5 mm. The signal was amplified by a differential amplifier (Tektronix 26A2). The transmembrane potential, surface electrogram, and 100-msec time marks (provided by a Tektronix Model 2901 time mark generator) were displayed on a dual-beam oscilloscope (Tektronix 565). The signals were also stored on magnetic tape (7.5 inches/sec) and hard copy was provided by an Elema Mingograf 803 recorder with a frequency response of 0–750 Hz at a paper speed of 200 mm/sec.

Stimuli for constant or premature atrial pacing were isolated from ground (Grass SIU Model 478A)
and applied to the preparation via a pair of Teflon-coated silver wires (interelectrode distance ~ 0.5 mm). The stimulating electrodes were applied within 2 mm of the electrodes recording the surface electrogram. For both patterns of stimulation, the stimuli were constant 2-msec voltage pulses 1.5 times threshold. For constant atrial pacing, signals were provided by a Grass Model S4 stimulator triggered by a digital timing circuit. The digital timing circuit allowed the selection of pacing cycle length correct to the nearest millisecond. Stimuli for premature atrial stimulation were provided by a combination of waveform and pulse generators (Tektronix 160 series). By triggering the waveform generator with the surface electrogram, the premature stimuli could be delivered at any predetermined coupling interval during the spontaneous sinus node cycle. The preparation was allowed to equilibrate for 1 hour. The region of the sinus node was then systematically explored for a cell having the following characteristics: 1) an action potential amplitude of at least 50 mV; 2) a smooth transition between phases 4 and 0 of the action potential; and 3) an onset of phase 0 preceding the first rapid component of the atrial electrogram by at least 25 msec. Cells fulfilling all of these characteristics were usually found in a very small area (1 mm²), which is referred to as the primary pacemaker area. In some preparations, cells fulfilling the above criteria could not be found after several hours of search, and those experiments were abandoned. All the data reported in the present paper were obtained during the continuous impalement of single cells.

After the primary pacemaker area was located, the spontaneous sinus cycle length was measured from the chart record. A cycle length equivalent to a rate 5 beats/min faster was calculated and the preparation was driven for a train of eight cycles at the calculated cycle length. The train was introduced eight to 10 times, with an interval of 10–20 spontaneous cycles between each train. Five minutes after the series of eight to 10 trains was introduced, the spontaneous cycle length was remeasured. The protocol was repeated with pacing rates 10 and then 15 beats/min faster than the spontaneous rate. In six experiments, the SACT was also estimated by introducing premature stimuli in diastole during the continued impalement of the same cell. In three experiments, the protocol of constant atrial pacing was repeated after the preparation was exposed to 3.5 × 10⁻⁴ M atropine sulfate and 1.7 × 10⁻⁴ M propranolol for 15 minutes. In two experiments, the preparation was exposed to atropine and propranolol before constant pacing was performed.

Data Analysis

The onset of the sinus node action potential was taken as the point of intersection between tangents along phase 0 and the terminal portion of phase 4. Sinus node action potential duration was measured from the onset, as defined above, to repolarization to 90% of the maximum diastolic potential. During simultaneous bipolar extracellular and intracellular microelectrode recordings from the crista terminalis, phase 0 of the intracellular action potential coincided with the rapid component of the atrial electrogram. Therefore, the onset of atrial activity was measured from the first rapid component of the atrial electrogram.

Throughout the text, SACT refers to the sum of antegrade and retrograde SACTs. For estimates of SACT by constant atrial pacing, the following cycles were measured:

1) The dominant cycle (AA): the interval between two spontaneous beats. The three atrial cycles immediately preceding each pacing train were measured and averaged.

2) The return cycle: the interval between the last beat in the train of paced beats and the subsequent spontaneous cycle.

3) The first postreturn cycle: the interval between the first and second spontaneous cycles after the train of constant pacing. The fifth and tenth postreturn cycles are similarly defined.

The data in the tables are given as mean ± SEM. The percentage error of the estimated SACT is defined as |measured SACT – estimated SACT|/measured SACT × 100, where | | means the modulus or absolute value of the difference. Statistical comparisons were made by the paired t test or an analysis of variance.

Results

SACT was estimated at pacing rates 5, 10 and 15 beats/min faster than the spontaneous sinus rate. The results at a pacing rate 10 beats/min faster are presented in detail and then contrasted with those at pacing rates 5 and 15 beats/min faster.

Pacing at 10 Beats/min Faster

Table 1 summarizes the results of 12 experiments. The variability of spontaneous cycle length was similar to that previously reported from this laboratory. In most experiments, measured antegrade and retrograde conduction times were not equal. This is evidence in favor of quoting estimated SACT as total conduction time rather than assuming equal antegrade and retrograde times and dividing by two. The measured SACT was quite variable between preparations, which could reflect variation between the relative positions of the surface and intracellular microelectrodes. The surface electrogram was recorded from the rostral end of the crista terminalis, but the actual position on the crista terminalis should account for little of the variability. Sano and Yamagishi have shown that the crista terminalis is activated almost synchronously during normal sinus rhythm. The position of the primary pacemaker site in relation to the crista terminalis varied from one preparation to the next. Conduction within the sinus node is slow; therefore, small variations in the distance between the recording sites could account for
some of the variability in measured conduction times.

Figure 1 shows the measurements on which the estimate of SACT was based. The estimate was based entirely on the surface electrogram. Panel A shows two spontaneous action potentials preceding the train. The dominant cycle length was 400 msec, and the antegrade conduction time 34 msec. There was a direct correspondence between the atrial cycle and the sinus node cycle measured with the intracellular microelectrode. Panel B shows the return cycle at the end of a pacing train. The interval on the atrial electrogram, 444 msec, includes the retrograde conduction time, the sinus return cycle, and the antegrade conduction time. Subtraction of the mean dominant cycle length, 400 msec (the mean of three consecutive cycles preceding this train), from the atrial return cycle gave an estimated SACT of 44 msec. In each experiment this procedure was performed eight times, and the means of these eight determinations are listed in table 1. This technique may over- or underestimate SACT. The correlation coefficient between measured and estimated conduction time was 0.54.

We explored some sources of error in the estimate by combining the information contained in both the intracellular and extracellular records. The atrial return cycle includes the retrograde conduction time, the sinus node return cycle (which includes the duration of the last paced action potential) and the antegrade conduction time. The duration of the last paced action potential was shorter than the last spontaneous action potential preceding the train (table 2). As the sinus node return cycle includes the duration of the last paced action potential and phase 4 from the maximum diastolic potential to onset of the first escape beat, the shorter paced action potential leads to an earlier onset of phase 4 and a relative shortening of the sinus node return cycle.

The effect of the brief period of constant pacing on sinus node automaticity is also shown in table 2. The mean spontaneous sinus cycle preceding the train was 452 msec, and the mean sinus node return cycle was 469 msec \( (p > 0.05) \). However, if we consider the action potential shortening discussed above, the diastolic period in the sinus return cycle was significantly prolonged. This is consistent with studies showing that constant pacing or even a single premature response may depress sinus node function. Suppression of sinus node automaticity would lead to an overestimate of SACT. In a study on the estimate of SACT by premature atrial stimulation, Miller and Strauss also showed that pretreatment with atropine and propranolol did not prevent shortening of the sinus node action potential by atrial premature depolarizations. To determine whether pretreatment with atropine and propranolol would prevent action potential shortening during pacing, and prolongation of the sinus node return cycle, five similar experiments were performed in the present study. The pretreatment did not prevent the shortening of action potential duration or the prolongation of the sinus node return cycle.

A tacit assumption in estimating SACT is that the antegrade conduction time during spontaneous rhythm preceding the train is the same as that for the first sinus escape beat. This was clearly not the case. The results of a typical experiment are shown in figure 2. The antegrade conduction time preceding the train was 30 msec and 10 msec for the first sinus escape beat. Occasionally the atrial electrogram preceded the

<table>
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<tr>
<th>Table 1. Sinoatrial Conduction Time Estimated by Constant Atrial Pacing Compared with Measured Sinoatrial Conduction Time</th>
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<td><strong>Experiment</strong></td>
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<td>10</td>
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<tr>
<td>11</td>
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<tr>
<td>12</td>
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</table>

Mean \( 80 ± 20 \) \( (p > 0.05) \)

*Twenty-four determinations in each experiment.
†Eight determinations in each experiment.
Abbreviation: CT = conduction time.
FIGURE 1. Estimate of sinoatrial conduction time by constant atrial pacing. In each panel the upper trace is the transmembrane potential recorded from a sinus node cell; the middle trace is the surface electrogram recorded from the crista terminalis. In the lower part of each panel, a ladder diagram depicts the presumed activation sequence during spontaneous rhythm and atrial pacing. Voltage and time calibrations are shown in the lower right-hand corner of the figure. A) Two spontaneous action potentials before the onset of a pacing train; B) the last action potential of a train of eight beats and the sinus node return cycle. \(SAN = \) sinoatrial node; \(SAJ = \) sinoatrial junction; \(AT = \) atrium.

TABLE 2. Effect of Constant Atrial Pacing on Sinus Node Action Potential Duration, Sinus Node Return Cycle Length, and Antegrade Conduction Time

<table>
<thead>
<tr>
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<th>Spontaneous activity preceding train (msec)</th>
<th>First sinus node cycle after train (msec)</th>
</tr>
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<tbody>
<tr>
<td>Sinus node AP duration</td>
<td>161 ± 7</td>
<td>140 ± 6*  (\text{p} &lt; 0.05)</td>
</tr>
<tr>
<td>Sinus node cycle</td>
<td>452 ± 18</td>
<td>469 ± 22  (\text{p} &lt; 0.05)</td>
</tr>
<tr>
<td>Antegrade conduction time</td>
<td>42 ± 36</td>
<td>30 ± 5 (\text{p} &lt; 0.05)</td>
</tr>
</tbody>
</table>

*Last paced AP of the train of eight beats.
†n = 12.
Abbreviation: AP = action potential.

onset of the action potential in the “primary pacemaker area.” These changes in antegrade conduction time may reflect a shift in the pacemaker site toward the stimulating and recording sites on the crista terminalis.\(^7,\)\(^8\) The conduction time usually returned to its initial value in five to 10 cycles. An apparent shift of the primary pacemaker toward the crista terminalis would lead to an underestimate of the SACT.

Pacing at 5 and 15 Beats/Min Faster

Estimates of SACT with a pacing rate 5 beats/min faster than the spontaneous cycle were less than those at a pacing rate 10 beats/min faster. The measured SACT was also shorter at the slower rate of stimula-
tion. In addition, there were a number of problems unique to this pacing rate. First, in four experiments the sinus node was not captured by the last beat of one or more pacing trains. Factors leading to failure of capture included atrial capture, but failure of sinus node capture, and initial sinus node capture with subsequent sinus node acceleration.

That there was atrial but no sinus node capture (clearly seen on the intracellular recording) could usually be inferred from the atrial return cycle, as this was shorter than the atrial cycles in which sinus node capture occurred. The results of an experiment depicting atrial but no sinus node capture are shown in figure 3. The atrial return cycle in panel B was prolonged by 16 msec. The prolongation was equal to the relative prematurity of the atrial stimulus.

Acceleration of the sinus node may be observed during pacing at 5 beats/min faster (fig. 4). Panel A shows a spontaneous cycle length of 365 msec preceding the train. The first beat at a paced cycle length of 345 msec captured the sinus node (panel B). However, the sinus node cycle shortened with successive beats (panel C) until the last stimulus of the train failed to capture the sinus node (panel D). The atrial return cycle was 392 msec. The estimate of the SACT was therefore 27 msec. However, this result was clearly not meaningful, since sinus node capture by the last paced beat of the train, a requirement of the method, was not met. This observation was repeated several times in this experiment, and similar observations were made in another preparation. An approximation method for determining the number of cycles required for sinus node capture is presented in the Appendix.

Pacing at 15 beats/min faster caused a further increase in estimated and measured SACT. In fact, for the three rates used, mean estimated SACTs were 76, 86 and 96 msec for 5, 10 and 15 beats/min faster, respectively. The increase in SACT was largely the result of an increase in retrograde conduction time with increased rate of pacing. The mean errors of the estimate at each pacing rate are plotted in figure 5. The errors were large and similar. Narula et al. proposed a pacing rate ≤10 beats/min faster than the spontaneous rate. Although the overall errors at all three pacing rates are similar, the frequency of failure to capture at a rate of 5 beats/min faster suggests that this may not be an appropriate pacing rate.

**Comparison of Estimates of SACT by Constant and Premature Atrial Stimulation**

As the other commonly applied technique of measuring SACT is indirect, the more clinically relevant comparison is of the estimates of SACT obtained by both indirect techniques. In six experiments, SACT was measured by each technique in turn during the impalement of the same cell. The result of these experiments are summarized in table 3. The mean ± SEE were similar for both techniques. Either technique may over- or underestimate SACT. Further, the direction of the trend was similar. SACTs overestimated by constant pacing tended to be overestimated by premature atrial stimulation. The correlation coefficient between the estimates of SACT by constant and premature stimulation was 0.85. When the two estimates were compared with the measured SACTs, the determinations were not significantly different from each other (p ≥ 0.9).

**Discussion**

We draw the following conclusions from this study:
1) The estimates of SACT by constant atrial pacing are dependent on the pacing rate. 2) The estimates at pacing rates 5, 10 and 15 beats/min faster than the spontaneous rate are subject to a mean error of approximately 30%. 3) The determination of SACT by premature atrial stimulation is subject to a similar error. 4) The estimate is not improved by treatment with atropine and propranolol.

From direct measurements of transmembrane potential in the primary pacemaker area, probable sources of error included shortening of the action potential duration with pacing, depression of automaticity, shift in the site of the pacemaker site after pacing, and failure of sinus node capture by the last paced beat with pacing rates 5 beats/min faster. We do not know whether these sources of error are unique to the isolated rabbit heart or whether they obtain in other species, including man.
measurements in feline atrial preparations by Lu et al. also showed that automaticity was depressed by constant pacing, and that the extent of sinus node depression observed in vitro was similar to that which occurs in vivo. Indirect evidence of the extent of depression of automaticity by single atrial premature depolarizations or by constant pacing in the in situ heart has been obtained by comparing the postreturn cycle (A3A4) with the mean spontaneous cycle (A1A4). However, this does not provide a true picture of the extent of pacemaker depression. A decrease in antegrade conduction time of the first escape beat, the result of pacemaker shift, contributes to prolongation of the postreturn cycle (A3A4) (fig. 2). The extent of pacemaker depression is probably less than the (A3A4 - A1A4) difference would suggest. It has been reported that a single premature depolarization may prolong the postreturn cycle 0.3–19%, with a mean prolongation of 4% in human hearts. This degree of prolongation appears small; however, at a mean spontaneous cycle length of 1000 msec, this represents a mean error of 40 msec in a parameter that averaged 189 msec in their subjects. It seems likely that pacing for more than a single beat would depress sinus node automaticity to the same or an even greater extent.

The depression of sinus node automaticity with pacing was associated with a decrease in antegrade conduction time, which may have resulted from a shift in the pacemaker site toward the crista terminalis. The available evidence suggests that changes in sinus rate, e.g., due to changes in [K]o, acetylcholine or drive, are accompanied by a shift in the site of the primary pacemaker. It appears that extrinsic influences that decrease the sinus rate do so in part by shifting the site of the primary pacemaker to sites of lesser intrinsic rates, closer to the crista terminalis. If sinus node

![Figure 4](attachment:image.png)

**Figure 4.** Acceleration of sinus node rate during atrial pacing. The traces are as in figure 1. A) A spontaneous cycle preceding the pacing train; B) the onset of the pacing train. In this and the subsequent panels, the numbered arrows indicate the paced beats of the train. Sinus node capture with beat 1 is indicated by a shortening of the duration and an increase in the amplitude of the sinus node action potential. In panel C and other paced cycles (not shown), the sinus node cycle shortens. As a result, the last beat (no. 8) of the train shown in panel D failed to capture the sinus node.

![Figure 5](attachment:image.png)

**Figure 5.** Error of the estimate of sinoatrial conduction time (SACT) at pacing rates 5, 10, and 15 beats/min faster. In each experiment, the error of the estimate, defined as | measured SACT - estimated SACT | /measured SACT, was calculated at each pacing rate. The mean error of all 12 experiments is plotted against the corresponding rate. Bars indicate mean ± SD of the mean errors.

**Table 3.** Comparison of Sinoatrial Conduction Time Estimated by Constant Atrial Pacing and Premature Atrial Stimulation with Measured Sinoatrial Conduction Time

<table>
<thead>
<tr>
<th>Experiment</th>
<th>Constant pacing* (msec)</th>
<th>Premature stimulation (msec)</th>
<th>Measured SACT (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>65 ± 6.5</td>
<td>94 ± 3.2</td>
<td>94 ± 1.8</td>
</tr>
<tr>
<td>3</td>
<td>59 ± 4.9</td>
<td>55 ± 1.6</td>
<td>67 ± 2.0</td>
</tr>
<tr>
<td>4</td>
<td>60 ± 1.4</td>
<td>56 ± 3.1</td>
<td>72 ± 1.9</td>
</tr>
<tr>
<td>5</td>
<td>71 ± 0.5</td>
<td>84 ± 5.2</td>
<td>53 ± 0.7</td>
</tr>
<tr>
<td>7</td>
<td>111 ± 1.5</td>
<td>88 ± 3.4</td>
<td>119 ± 0.8</td>
</tr>
<tr>
<td>12</td>
<td>127 ± 6.7</td>
<td>141 ± 2.0</td>
<td>70 ± 0.8</td>
</tr>
<tr>
<td>Mean ± SEM</td>
<td>79 ± 14</td>
<td>86 ± 13</td>
<td>79 ± 10</td>
</tr>
</tbody>
</table>

*10 beats/min faster.
Abbreviation: SACT = sinoatrial conduction time.
slowing occurs after drive in the human heart, one would anticipate a shift in pacemaker site, introducing an error into the estimate of SACT.

As a population, patients with sinus node disease have slow heart rates. In a proportion of these patients, the site of the primary pacemaker may be shifted permanently toward the crista terminalis. Such permanent shifts may lead to the apparent paradox of a short or normal SACT in the presence of sinus node disease. However, he stressed that with pacing rates ≤ 5 beats/min faster than the spontaneous rate, as many as 16 paced cycles may be required for sinus node capture. In their comparison of estimates of SACT by premature stimulation and constant atrial pacing, Breithardt and Seipel used rates that were approximately 3, 6 and 9 beats/min faster than the spontaneous rate for only nine paced cycles. They suggested that it was crucial to use the longest possible cycle length and reported mean SACTs of 96 ± 22 msec with constant pacing at a mean rate of 3 beats/min faster than the spontaneous rate vs 113 ± 27 msec with the premature atrial stimulation technique. Greater depression of sinus node automaticity by premature atrial stimulation than with constant atrial pacing was the favored explanation for the difference. However, a further increase of 3 beats/min in the constant pacing rate increased the SACT by as much as 62 msec, with a mean increase of 23 msec. The marked increases in SACT associated with small increases in the pacing rate seen in the same patients are probably more readily explained by capture of the sinus node at the faster pacing rate only.

There are purported advantages to the constant pacing technique: 1) It is simpler to perform than the premature atrial stimulation technique. 2) It eliminates the spontaneous fluctuation in sinus cycle length. 3) It eliminates the need for and programs in demarcating zone II responses. 4) It permits comparison of drug effects on SACT during a constant cycle length.

With regard to the first advantage, the estimate by constant pacing can be performed with a simple, fixed-rate pacemaker compared with the programmable delay requirement for the premature stimulation technique. This could make the assessment of SACT feasible in a larger number of clinical centers. An estimate of SACT is provided by a single pacing train; however, in our in vitro study, the variance of the estimate was large in some experiments. We would therefore recommend repeating the train several times in a given study. The validity of the second advantage is open to question. The estimate is based on the difference between the mean spontaneous sinus cycle length and the first return cycle after pacing. The return sinus cycle is subject to the same influences that cause beat-to-beat variation of the normal sinus cycle. Therefore, the period of constant pacing may not eliminate the effects of variation of sinus cycle on the estimate of SACT. The third advantage is a definite one. In some cases, zone II responses may be absent, or delineation of their onset may be subjective. The comments on the second advantage apply also to the fourth.

In spite of its advantages, the constant pacing technique is subject to sources of error similar to those of the premature atrial stimulation technique: shortening of the paced action potential duration, depression of automaticity, and shifts in the site of the primary pacemaker. Both techniques are subject to large and unpredictable errors. A clear distinction between normal and abnormal sinoatrial conduction may have to await the application of techniques for directly recording sinus node activity in the in situ heart.

References

Appendix

Number of Pacing Cycles Required for Sinus Node Capture

Given the spontaneous cycle length, the pacing cycle length, total conduction time (SACT or its estimate), and the prematurity of the first pacing stimulus, it was possible to predict the number of cycles required for sinus node capture (fig. 6). For sinus node capture during the first cycle, the prematurity of the atrial stimulus, P, must be greater than the total conduction time (antegrade and retrograde).

In the ladder diagram, the first pacing stimulus fails to capture the sinus node because of interference with the emerging sinus node impulse in the sinoatrial junction. However, with each paced beat the prematurity increases by an interval equal to the difference between the spontaneous cycle length (SCL) and the paced cycle length (PCL) (i.e., (SCL - PCL)/beat). This increase in prematurity enables capture of the sinus node by P2. In general, the number of beats required to capture the sinus node, n, is given by the following equation: n = (SACT - prematurity)/(SCL - PCL). Prematurity of pacing stimuli just preceding atrial activity is computed as positive, while prematurity immediately after atrial activity is computed as negative. Panel B gives an example with the first pacing stimulus just preceding the anticipated atrial capture. The number of beats predicted for capture by the above equation was 3.6; the observed number was 4. Similarly, panel C shows the first stimulus of the train just after "spontaneous" atrial activity. The predicted number of beats for capture was 7.9; the observed number was 8.

The utility of an equation to predict the number of pacing stimuli required capture is in choosing an appropriate pacing rate to assure sinus node capture before the end of the train. With short, spontaneous cycle lengths, the equations enable us to select a rough estimate of an appropriate pacing length for capture.

**Figure 6.** Prediction of the number of atrial cycles required for sinus node capture. SAN = sinoatrial node; SAJ = sinoatrial junction; AT = atrium; SCL = spontaneous cycle length; PCL = paced cycle length; P1, P2 = the prematurity of the first and second paced beat of a train. A) A ladder diagram during spontaneous and paced activity. The first paced beat (broken line) of prematurity P1 fails to capture the sinus node because of interference at the SAJ. The prematurity of the second paced beat, P2, increases by (SCL - PCL)/beat. P2 exceeds SACT; therefore, sinus node capture occurs. B) An actual experimental record. The dotted line indicates the timing of atrial activity if the atrial cycle were not curtailed by the paced beat. The difference between the onset of activity between the solid and dotted electrogram is the prematurity of the paced beat (positive). The number of cycles required to capture = (SACT - P)/SCL - PCL, in this case 3.6 beats. The observed number of cycles required for capture is 4. C) Similarly, the predicted number of cycles was 7.9 and the observed number was 8. In this case the prematurity is negative.
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