Sinus Node Recovery Time and Calculated Sinoatrial Conduction Time in Normal Subjects and Patients with Sinus Node Dysfunction

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SUMMARY In 61 patients sinus node function was tested by programmed (sinoatrial conduction time, SACT) and overdrive atrial pacing (sinus node recovery time, SRT). In the control group (N = 20), mean sinus cycle length was 773 ± 140.2 msec, mean absolute SRT 1044 ± 215.8 msec [corrected SRT (CSRT)] 270 ± 112.5 msec; mean ± SD) and calculated SACT was 82 ± 19.2 msec. The upper limit of normal SACT was defined as 120 msec. In 41 patients with sinus node dysfunction, mean age (55 ± 14.7 years), mean spontaneous cycle length during the study (1094 ± 248.0 msec), the lowest heart rate observed (42 ± 7.5 beats/min), maximal SRT (2110 ± 1269.1 msec), maximal CSRT (1016 ± 1182.8 msec), and calculated SACT (126 ± 47.3 msec) were significantly longer than in the control group. Abnormalities of sinus node function, as evidenced by the degree of spontaneous bradycardia, SRT and calculated SACT were more frequent in patients with bradycardia-tachycardia syndrome or spontaneous sinoatrial block than in those exhibiting isolated sinus bradycardia.

We conclude 1) that in patients with sinus node dysfunction both sinus node automaticity and sinoatrial conduction may be abnormal, and 2) that overdrive and programmed premature atrial stimulation can separate patients with sinus node dysfunction according to their clinical presentation.

DISTURBANCES OF SINUS NODE AUTOMATICITY or of sinoatrial conduction or both have been assumed to occur in the sick sinus syndrome.1-6 A direct estimation of these parameters is not yet possible in man. Indirect methods have therefore been used for evaluation of sinus node function. Estimation of sinus node recovery time after overdrive suppression is a clinically useful method for evaluation of sinus node automaticity.1, 4, 5, 7-11 Sinoatrial conduction time may be evaluated by premature atrial stimulation as proposed by Strauss et al.12 Studies evaluating both parameters of sinus node function in the same group of patients have been rare.7, 10, 11 Therefore we compared sinus node recovery time (SRT) and calculated sinoatrial conduction time (SACT) in 41 patients with sinus node dysfunction to these measures in 20 normal subjects.

Methods

All patients gave written informed consent. They were studied in the cardiac catheterization laboratory in the resting, postabsorptive, nonsedated state. All cardioactive drugs had been withdrawn at least 48 hours before the study. In case of digitalis or long-acting antiarrhythmic drugs medication was stopped at least one week before.

Electrode catheters were passed percutaneously and positioned under fluoroscopic control. A quadripolar catheter was passed via the right femoral vein into the high right atrium. The distal electrodes were used for stimulation and positioned near the junction of the superior vena cava and the right atrium; the proximal pair of electrodes was used for recording a high right atrial electrogram. A second tri- or hexapolar electrode catheter was inserted just below the first catheter and advanced under fluoroscopic inspection to the tricuspid orifice to record the His bundle electrogram.17

All electrodes were connected to the recording device via a keyboard switchbox (Siemens-Elema) that contained 50 Hz high-pass filters and current-limiting circuits. The right atrial electrogram, the His bundle electrogram, and the surface ECG (lead I, II and III) were simultaneously recorded on a six-channel direct-writing recorder (Siemens Cardiowriter) at a paper speed of 50 or 100 mm/sec. The right atrial electrogram was used to trigger a programmable stimulator (Medtronic 5837, modified). The pulse width was 2 msec, and the amplitude was adjusted to twice diastolic threshold. After a 10 min control period, single premature atrial stimuli were introduced via the stimulating electrodes during spontaneous sinus rhythm beginning late in diastole after each sixth to eighth beat.

The following intervals were measured from the atrial electrogram: A1-A2 interval: the spontaneous cycle length immediately preceding the stimulated atrial complex; A1-A3 interval: the test cycle or the coupling interval of the premature stimulated atrial beat; A2-A3 interval: the return cycle or the postextrasystolic pause.

The results were plotted in a coordinate system using normalized data with the relative prematurity of the test stimulus [A1-A2 / A1] on the abscissa and the relative length of the return cycle [A2-A3 / A1] on the ordinate. Sinoatrial conduction time (SACT) was calculated according to the following equation during reset responses of the sinus node:12, 18, 19

\[
\text{SACT} = \frac{A2-A3}{A1-A1} \times A1-A1
\]

The value thus obtained represents single sinoatrial conduction time as originally proposed by Strauss et al.12 and adopted by several investigators.10, 14, 17, 18, 19, 20, 21 It is the average of both retrograde and antegrade conduction time which, however, need not be identical. That is why some authors prefer to use total SACT.19, 21

Sinus node recovery time (SRT) was estimated by atrial overdrive pacing. The duration of stimulation was 30 sec. Stimulation was performed selecting an initial rate just above the spontaneous sinus rate. The rate of stimulation was increased by 10-20 beats/min until a rate of 160 beats/min was attained. Sinus node recovery time was mea-
sured from the last paced atrial beat and the first beat of sinus origin (high to low right atrial sequence) or, in case of escape beats from a lower focus, to the first atrial depolarization giving a minimal SRT. For the present study only the maximal pause after cessation of high rate atrial stimulation was further considered. Sinus node recovery time was corrected (CSRT) for the spontaneous cycle length by calculating the difference between both.7

Twenty patients served as the control group. Electrophysiologic study was performed because of a history of palpitations or for exclusion of a WPW syndrome (5 patients). Holter monitoring of the ECG, electrophysiological studies, and cardiac catheterization data were normal (table 1).

Forty-one patients demonstrated sinus node dysfunction. History of dizziness or syncope prior to the study was determined and one 12-hour ambulatory ECG recording, or more was made. On the basis of these clinical data, the patients were divided into four subgroups (tables 2 and 3): subgroup A: patients with sinus bradycardia without complaints (1–8); subgroup B: patients with sinus bradycardia with complaints (9–21); subgroup C: patients with bradycardia-tachycardia (22–24); subgroup D: patients exibiting sinoatrial block (25–41). Four patients of subgroup D additionally showed clinical features of subgroup C. Isolated sinus bradycardia (subgroups A and B) was diagnosed if the routine ECG and the long-term ambulatory recording of the ECG did not demonstrate any disturbances of sinus nodal or atrial function other than sinus bradycardia. Patients of subgroups C and D were regarded as representing the sick sinus syndrome proper. In all symptomatic patients a neurological study was done before the electrophysiological examination. Only those patients were included whose neurological evaluation was negative. Furthermore, no patient had any evidence of valvular heart disease. Digitalis excess as a cause of sinus bradycardia had been excluded in all cases.

Statistical evaluation used the Wilcoxon, Mann, and Whitney test.22

Results

Control Group

The results in the control group are listed in table 1. The upper limit of calculated SACT in normal subjects was considered to be 120 msec (mean ± 2 SD).

 Patients with Sinus Node Dysfunction (tables 2 and 3)

Nine of the 41 patients with sinus node dysfunction also had atrioventricular or intraventricular conduction disturbances. Eight of 21 patients with isolated sinus bradycardia were asymptomatic (subgroup A), and 13 were symptomatic (subgroup B). Six of the latter patients complained of dizziness and seven of syncope. In patients with bradycardia-tachycardia syndrome (subgroup C), two of three patients were symptomatic (dizziness in both patients). Thirteen of 17 patients who exhibited spontaneous sinoatrial block (subgroup D) were also symptomatic (8 with dizziness and 5 patients with syncope). Throughout the electrophysiologic study mean spontaneous cycle length was greater than 1000 msec in 27 of 41 patients (range 1030 msec to 1865 msec); in the rest, mean spontaneous cycle length ranged between 637 msec to 946 msec.

Mean age (55 ± 14.7 years), mean spontaneous cycle length during the study (1094 ± 248.0 msec), the lowest heart rate ever observed (42 ± 7.5 beats/min), maximal sinus node recovery time (SRT) (2110 ± 1269.1 msec) and maximal corrected SRT (CSRT) (1016 ± 1182.8 msec) were significantly different from the control group. Calculated sinoatrial conduction time (126 ± 47.3 msec) was significantly longer than in the control group in those patients in whom it could be calculated.

Figure 1 gives some representative examples of the return cycle pattern in four patients with sinus node dysfunction. Calculated SACT in the control group and in the various subgroups with sinus node dysfunction is plotted in figure 2. In table 3 mean data are presented for the four subgroups.

Subgroups A and B: Patients with Isolated Sinus Bradycardia with and without Complaints

Mean age (49 ± 16.6 years), mean spontaneous cycle length during the study (1071 ± 202.8 msec), and the lowest heart rate ever observed (45 ± 6.1 beats/min) were significantly different from the control group. Maximal CSRT (232 ± 187.2 msec) and calculated SACT (95 ± 30.2 msec) in patients without complaints (1 to 8) were not different from the control group, whereas in patients with complaints (9 to 21), all three parameters (1675 ± 646.7 msec, 627 ± 608.7 msec, and 109 ± 29.6 msec respectively) were significantly longer. In one patient with sinus bradycardia and syncope, a chaotic pattern of the return cycles was observed. Three patients exhibited an ectopic atrial rhythm during the study. Therefore, the calculated value did not yield a conduction time between the atrium and the sinus node, but instead between the site of stimulation and the ectopic focus. These data were not considered when calculating SACT.

Subgroup C: Patients with Bradycardia-Tachycardia Syndrome

In this small subgroup mean age, the lowest heart rates ever observed, mean spontaneous cycle length during the study, maximal SRT, and CSRT were different from those values in the control group (tables 2, 3). The prolongation of calculated SACT was not significantly different.

| TABLE 1. Clinical and Electrophysiological Data in the Control Group |
|---------------------|---------------------|---------------------|---------------------|---------------------|
|                    | Age (yr)            | AAI1               | SRTmax             | CSRTmax             | SACT |
| Mean ± SD          | 32 ± 10.7           | 773 ± 142.0        | 1044 ± 215.8       | 270 ± 112.5         | 82 ± 19.2 |
| Range              | 15—55               | 574—1004           | 640—1480           | 66—508              | 48—112 |

All values except age given in msec.

Abbreviations: SRT = sinus node recovery time; CSRT = corrected sinus node recovery time; SACT = sinoatrial conduction time.
<table>
<thead>
<tr>
<th>No./Age/Sex</th>
<th>Subgroup A</th>
<th>Subgroup B</th>
<th>Subgroup C</th>
<th>Subgroup D</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/6/m</td>
<td>SB, 30bp</td>
<td>SB, 45bp</td>
<td>SB, 50bp</td>
<td>SB, 29bp</td>
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<tr>
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<td>SB, 35bp</td>
<td>SB, 54bp</td>
<td>SB, 35bp</td>
<td>SB, 45bp</td>
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<tr>
<td>3/20/m</td>
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<tr>
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<td>SB, 52bp</td>
<td>SB, 45bp</td>
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<tr>
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<tr>
<td>7/56/m</td>
<td>SB, 52bp</td>
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<td>SB, 48bp</td>
<td>SB, 29bp</td>
</tr>
<tr>
<td>8/52/f</td>
<td>SB, 43bp</td>
<td>SB, 46bp</td>
<td>SB, 50bp</td>
<td>SB, 29bp</td>
</tr>
</tbody>
</table>

**Table 2. Clinical and Electrophysiological Data in 41 Patients with Sinus Node Dysfunction**

<table>
<thead>
<tr>
<th>No./Age/Sex</th>
<th>Subgroup A</th>
<th>Subgroup B</th>
<th>Subgroup C</th>
<th>Subgroup D</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/6/m</td>
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<td>SB, 50bp</td>
<td>SB, 29bp</td>
</tr>
</tbody>
</table>

**Abbreviations:**
- m = male; f = female; SB = sinus bradycardia; bpm = beats per minute (lowest spontaneous heart rate); SA-BI = I° sinoatrial block; AF = atrial fibrillation; AEB = atrial ectopic beats; AT = atrial tachycardia; LAH = left anterior hemiblock; LBBB = left bundle branch block; RBBB = right bundle branch block; AV I° = first degree a-v block; VEB = ventricular ectopic beat; SRT = sinus node recovery time; CSRT = corrected SRT; max SN depression (rate) = rate at which the longest SRT was observed; D = dizziness; S = syncope; CP = chaotic pattern of the return cycles; AER = atrial ectopic rate as the dominant rhythm during the study.
Subgroup C: Patients with Sinus Node Dysfunction (N = 41) Grouped according to their Clinical Presentation

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>Age (yrs)</th>
<th>Lowest heart rate observed (beats/min)</th>
<th>$\bar{A}_t$ (msec)</th>
<th>SRT$_{max}$ (msec)</th>
<th>CSRT$_{max}$ (msec)</th>
<th>SACT (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All pts with sinus node dysfunction (N = 41)</td>
<td>55 ± 14.7</td>
<td>42 ± 7.5</td>
<td>1094 ± 248.0</td>
<td>2110 ± 1269.1</td>
<td>1016 ± 1182.8</td>
<td>126 ± 47.3</td>
</tr>
<tr>
<td>Subgroup A</td>
<td>47 ± 14.9</td>
<td>44 ± 7.7</td>
<td>1109 ± 195.5</td>
<td>1341 ± 279.5</td>
<td>232 ± 187.2</td>
<td>95 ± 30.2</td>
</tr>
<tr>
<td>(pts 1 to 8, N = 8)</td>
<td>P &lt; 0.01</td>
<td>P &lt; 0.01</td>
<td>P &lt; 0.01</td>
<td>P &lt; 0.01</td>
<td>P &lt; 0.01</td>
<td>P &lt; 0.01</td>
</tr>
<tr>
<td>Subgroup B</td>
<td>50 ± 18.1</td>
<td>45 ± 5.2</td>
<td>1048 ± 211.4</td>
<td>1675 ± 646.7</td>
<td>627 ± 608.7</td>
<td>109 ± 29.6</td>
</tr>
<tr>
<td>(pts 9 to 21, N = 13)</td>
<td>P &lt; 0.01</td>
<td>P &lt; 0.01</td>
<td>P &lt; 0.01</td>
<td>P &lt; 0.01</td>
<td>P &lt; 0.01</td>
<td>P &lt; 0.01</td>
</tr>
<tr>
<td>Subgroup C</td>
<td>56 ± 10.6</td>
<td>39 ± 9.3</td>
<td>1358 ± 158.2</td>
<td>3467 ± 2821.9</td>
<td>2109 ± 2685.8</td>
<td>150 ± 80.4</td>
</tr>
<tr>
<td>(pts 22 to 24, N = 3)</td>
<td>P &lt; 0.01</td>
<td>P &lt; 0.01</td>
<td>P &lt; 0.002</td>
<td>P &lt; 0.05</td>
<td>NS</td>
<td>1048</td>
</tr>
<tr>
<td>Subgroup D</td>
<td>63 ± 8.1</td>
<td>40 ± 8.1</td>
<td>1076 ± 291.7</td>
<td>2565 ± 1291.8</td>
<td>1489 ± 1187.6</td>
<td>152 ± 43.7</td>
</tr>
<tr>
<td>(pts 25 to 41, N = 17)</td>
<td>P &lt; 0.01</td>
<td>P &lt; 0.01</td>
<td>P &lt; 0.01</td>
<td>P &lt; 0.01</td>
<td>NS</td>
<td>1048</td>
</tr>
</tbody>
</table>

Values are mean ± standard deviation.

Patients 25 to 28 were considered only in subgroup D though they also exhibited features of subgroup C such as tachyarhythmias.

All data were compared to the control group using the Wilcoxon, Mann, and Whitney test.83

**Subgroup D: Patients with Spontaneous Sinoatrial Block**

Four patients of this subgroup showed clinical features of subgroup C. The mean age (63 ± 8.1 years), the lowest heart rate observed (40 ± 8.1 beats/min), mean spontaneous cycle length during the study (1076 ± 291.7 msec), maximal SRT (2565 ± 1291.8 msec), CSRT (1489 ± 1187.6 msec), and calculated SACT (152 ± 43.7 msec) did not show any significant difference when compared to the bradycardia-tachycardia group. However, these parameters differed significantly from the control group. Mean age, maximal SRT and CSRT, and calculated SACT were also signifi-
SINUS NODE FUNCTION/Breithardt, Seipel, Loogen

FIGURE 2. Calculated SACT in control subjects and in patients with sinus node dysfunction. The latter group was subdivided into four groups according to their clinical presentation. (+) = ectopic atrial rhythm during the study; CP = chaotic pattern of the return cycles.

FIGURE 3. A plot of the calculated SACT and CSRT in the control group (right panel) and in patients with sinus node dysfunction (left panel). Patients with sinus node dysfunction are represented by different symbols according to their clinical presentation (see upper insert). In the control group all values of calculated SACT and CSRT were within the limits of normal (broken lines). Similar results were obtained in many patients with isolated sinus bradycardia. However, in patients with more complex sinus node dysfunction, most values were abnormal. The longest values of CSRT in patients with bradycardia-tachycardia syndrome or spontaneous sinoatrial block were usually found in those who had relatively short calculated SACT. This indicates the importance of adequate discharge of the sinus node for achieving maximal overdrive suppression.
cantly different from patients with isolated sinus brady-
cardia (subgroups A and B).

In five cases, a chaotic pattern of the return cycles was
observed after premature atrial depolarization (fig. 1, panel
D). Obviously, SACT cannot be calculated in these patients.

In figure 3 calculated SACT is plotted versus CSRT in the
control group and in patients with sinus node dysfunction.
Patients with a chaotic pattern are not represented.

In each case of the control group, CSRT was within the
limits of normal, which according to Narula et al.,7 is less
than 525 msec. In patients with isolated sinus bradycardia,
CSRT was normal in 14 patients, reaching the upper limit of
normal in two, and slightly above normal in three patients.
CSRT was prolonged (2590 msec) only in patient 20. An ab-
normal value of calculated SACT was found in six patients
with sinus bradycardia. In the remaining patients, calculated
SACT was in the range of normal except for one case who
demonstrated a chaotic pattern of the return cycles. In pa-
tients with bradycardia-tachycardia and/or sinoatrial block,
both CSRT and calculated SACT were abnormal in 13 out
of 15 patients. Patients with a chaotic pattern are not re-
presented in figure 3. In three out of five patients with a
chaotic pattern of the return cycles, CSRT was abnormal.

In eight out of 11 patients with spontaneous sinoatrial
block, calculated SACT was greater than 140 msec. How-
ever, there was a great overlap of calculated SACT in
patients with and without sinoatrial block. Excluding pa-
tients in subgroup A, mean calculated SACT in cases with
spontaneous sinoatrial block was 119 ± 46.5 msec, and
155 ± 44.4 msec in those with sinoatrial block (P < 0.05). Cases demonstrating an ectopic atrial rhythm
were not included in this evaluation.

The presence or absence of complaints did not sig-
nificantly affect the results of electrophysiological test-
ing. Mean SRT in asymptomatic patients was 2428 ± 562
msec (CSRT: 1160 ± 471 msec), and 2791 ± 1751 msec
(CSRT: 1723 ± 1602 msec) in symptomatic cases (NS).
Mean calculated SACT was practically identical among
symptomatic and asymptomatic patients (150 ± 35 msec
and 156 ± 59 msec, respectively). All patients with a chaotic
pattern of the return cycles were symptomatic.

Discussion

The focus of the present report was directed toward the
role of electrophysiological procedures in separating pa-
tients with sinus node dysfunction.1-4, 23-28 Sinus node
automaticity and sinoatrial conduction time were evaluated
by use of atrial overdrive pacing, 6, 10 and programmed pre-
mature atrial pacing. A detailed discussion of the problems
inherent in the latter approach is beyond the scope of this
paper and has recently been presented. 15, 16, 19a, 20-25

Patients were grouped with regard to their clinical presen-
tation and then compared to a control group. Some reser-
vations should be made about the use of this control group
because these subjects were not age-matched to the patients
with sinus node dysfunction. The following arguments might
render the use of our control group feasible. Calculated
SACT, SRT, and CSRT in the control group and in pa-
tients with isolated sinus bradycardia (without complaints)
were not different although these patients differed as to their
age, mean cycle length, and lowest heart rate observed. This
suggests that at least in the age groups studied, calculated
SACT and SRT are independent of age. This has recently
been suggested with regard to SRT. 26

The mean value of calculated SACT (82 ± 19.2 msec)
and the upper limit of normal (120 msec) in our study
are consonant with those recently published by several au-
thors.14, 16, 18b, 21, 37, 38 In two studies1, 20 which included
patients with conduction defects and organic heart disease in
the control group, different values were found. According to
our own experience in 81 patients with atrioventricular or in-
traventricular conduction disturbances, about one fifth of
patients exhibited abnormal values of calculated SACT
when compared to the control group (unpublished data).
Abnormalities of sinoatrial automaticity and of atrio-
ventricular conduction frequently coexist. 24, 39-41 The respon-
siveness of the sinus node to isoprenaline has been found to
be diminished in many patients with total A-V block. 42
These findings suggest that in a great number of patients
pervasive conduction system disease25 exists. Therefore, it
does not seem valid to include patients with conduction
defects in a control group considered to represent normal
sinus node function.

Among patients with sinus bradycardia or the sick sinus
syndrome, many have abnormal sinus node function as
evinced by premature atrial stimulation10, 12-15, 19b, 38 or over-
drive atrial pacing. 1, 4, 7, 8, 11, 24, 26, 28 However, only a few
studies exist in which the effect of both procedures on sinus
node function was tested in a large number of patients2 38 ex-
cept for the most recent report by Strauss et al. 13

Our study demonstrates that marked differences exist
between the various subgroups of patients with sinus node
dysfunction with regard to the electrophysiologic para-
eters tested. Sinus node recovery time and calculated SACT
were commonly within normal limits in patients with
isolated sinus bradycardia (subgroup A and B). This might
represent less disturbance of sinus node function but could
also be explained by other factors. First, SA entrance block
may produce an apparently normal SRT: 5, 6 second, a shift
of the pacemaker to a more peripheral site in the SA node
may produce apparent normalization of calculated
SACT. 20, 31 As both parameters were within normal limits in
most patients with isolated sinus bradycardia, both
mechanisms would have to be active in the same patient.
This combination does not seem likely. Nevertheless, to date
no corroborating clinical information in terms of follow-up
study is available to support the hypothesis that isolated
sinus bradycardia is a milder form of sinus node disease with
a more benign prognosis.

The overall function of the sinus node and the perinodal
region seems to be more severely disturbed in patients with
bradycardia-tachycardia syndrome or with spontaneous
sinoatrial block. Most of them were symptomatic. These pa-
tients had the lowest heart rates of all groups and far more
abnormal values of calculated SACT and SRT than pa-
tients with isolated sinus bradycardia. It is noteworthy that
some previous studies were not able to separate patients with
normal and abnormal sinus node function by the results ob-
tained by premature atrial stimulation. 7, 15 This is probably
due to the selection of control subjects whose heart rates did
not closely compare with that of the abnormal group, as well
as the number of patients with isolated sinus bradycardia. 2
This has been definitely demonstrated by the different results in our subgroups with sinus node dysfunction.

Spontaneous heart rates were slightly lower in patients with bradycardia-tachycardia syndrome or spontaneous sinoatrial block than in patients with isolated sinus bradycardia. However, in the individual case it may be difficult to decide, using the lowest heart rate as the only parameter, in which group of patients with sinus node dysfunction he belongs. Therefore, both electrophysiological procedures used in this study give important additional information to characterize the state of sinus node function more precisely.

Patients with abnormally long calculated SACT exhibited spontaneous sinoatrial block more frequently. This suggests that a pre-existing delay of sinoatrial conduction is a prerequisite for second degree sinoatrial block to occur. However, other mechanisms cannot be ruled out on the basis of clinical studies.

A chaotic pattern of the return cycles deserves some comments. We did not observe it in any normal person, but usually in patients with severe disturbances of sinus node function, all of whom were asymptomatic. Only one patient with this pattern belonged to the patients with isolated sinus bradycardia. It is possible that in this case the clinical evaluation had underestimated the severity of sinus node dysfunction. The pathophysiological mechanism leading to this abnormal pattern of the return cycles is unknown and has not yet been described in animal preparations. In these patients the variation of the spontaneous cycle length was not greater than in others. Thus the variation of the spontaneous rhythm could not entirely explain the great variation of the return cycles. Atropine has been shown to make this abnormal pattern return to the normal biphasic pattern. We assume that probably abnormalities both of sinus node automaticity and of sinoatrial conduction contribute to this abnormal pattern.

What weight electrophysiological parameters should be given when deciding whether to implant a pacemaker is not known. The lack of correlation between these parameters and the complaints of our patients may indicate that in vivo testing of sinus node function does not uncover the whole spectrum of sinus node disease. In the same individual periods of normal sinus node function may alternate with those of abnormal function, as is suggested by some case reports. On the other hand, the symptoms of the patients may not be related to sinus node dysfunction at all, but to some other cause such as cerebrovascular disease. Furthermore, it should be taken into account that estimation of SRT is only a measurement of sinus node automaticity after overdrive pacing. It is conceivable that in a given symptomatic patient with a normal SRT the symptoms are due to the severity of spontaneous bradycardia, or symptoms may arise because of hemodynamic impairment during atrial tachyarrhythmias. To exclude these mechanisms, ambulatory tape recording of the ECG is of utmost importance. The lack of symptoms in a patient with an extremely prolonged SRT may indicate that this patient has not yet experienced episodes of spontaneous tachycardias uncovering the underlying disturbance of sinus node automaticity. As far as the indication for pacemaker implantation is concerned, estimation of calculated SACT seems to be of greater value than estimation of calculated SACT. However, for diagnostic purposes programmed premature atrial stimulation is of additional value in separating patients with normal and abnormal sinus node function.

The differences of the electrophysiological data between the various subgroups with sinus node dysfunction suggest either that isolated sinus bradycardia and the sick sinus syndrome are different entities or that both represent different stages of the same disease spectrum. However, follow-up studies are needed.

References

An Unusual Variety of Atrioventricular Nodal Re-entry due to Retrograde Dual Atrioventricular Nodal Pathways

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SUMMARY Three patients with paroxysmal supraventricular tachycardia (PSVT) had discontinuous ventriculo-atrial conduction curves (V1-V2, A1-A2), suggesting dual A-V nodal pathways. Ventricular echoes occurred simultaneously with sudden increase of V-A interval. These echoes were characterized by retrograde P waves occurring in front of QRS, suggesting utilization of a slow pathway for retrograde conduction and a fast pathway for antegrade conduction. In case one, atropine improved retrograde slow pathway and antegrade fast pathway conduction and made A-V nodal re-entry sustained, resulting in PSVT (with retrograde P in front of the QRS). In cases 2 and 3, atropine markedly shortened retrograde fast pathway refractory period and slightly improved antegrade slow pathway conduction. The discontinuous V1-V2, A1-A2 curves and echoes were no longer demonstrable. However, with improvement of retrograde fast pathway and antegrade slow pathway conduction, A-V nodal re-entrant echoes and PSVT were observed, utilizing the slow pathway for antegrade conduction and the fast pathway for retrograde conduction (P simultaneous with QRS).

In this study of three patients with PSVT, we demonstrate an unusual form of A-V nodal re-entry utilizing an A-V nodal fast pathway for antegrade conduction and an A-V nodal slow pathway for retrograde conduction. All three patients demonstrated retrograde discontinuous conduction curves (V1-V2, A1-A2). The electrophysiological mechanisms of this unusual variety of A-V nodal re-entry are discussed.

Methods
Electrophysiological Studies
Three patients with documented recurrent PSVT were studied. Electrophysiological studies were performed in the nonsedated, supine state. Cardiac medications were discontinued at least 72 hours prior to the study. Informed written consent was obtained. A percutaneously passed tripolar electrode catheter was placed at the tricuspid valve for His bundle recording. A hexapolar electrode catheter was placed at the right ventricular apex via an antecubital vein. The distal two electrodes were utilized for ventricular
Sinus node recovery time and calculated sinoatrial conduction time in normal subjects and patients with sinus node dysfunction.

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