Sinoatrial Conduction in Children: An Index of Sinoatrial Node Function

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SUMMARY The premature atrial stimulus technique was used to evaluate sinoatrial conduction (SAC) in 50 children (age range 6 months to 21 years; median 6.5 years) to obtain normal values of sinoatrial conduction time (SACT) and to determine the usefulness of the technique in evaluation of sinoatrial node (SAN) dysfunction in children. The results in the normal group (n = 20) were compared with those of patients with documented SAN dysfunction (group 1, n = 20) or at risk to develop SAN dysfunction because of a previous intracardiac operation (group 2, n = 10). Among the 30 patients in groups 1 and 2, 23 had undergone an intracardiac operation — 13 Mustard operations and five closures of an atrial septal defect.

In group 1, the mean value of total SACT was 172 msec ± 42 (SD) compared with 124 msec ± 38 in the normal group (p < 0.001). Each of the 20 patients in group 1 had abnormal SAN automaticity (prolonged corrected sinus node recovery time, CSNRT) and 13 of the 20 had abnormal SAC. While each of the 10 patients in group 2 had normal CSNRT, two of them had abnormal SAC.

We conclude that the evaluation of SAC should be included in the assessment of children with suspected SAN dysfunction.

CORRECTED SINUS NODE RECOVERY TIME (CSNRT) is an indicator of the automaticity of the sinoatrial node (SAN) and has been useful in confirming known or suspected SAN dysfunction.1-7 Some patients, however, have clinical manifestations of SAN dysfunction but have normal CSNRT.5-10 The presence of prolonged sinoatrial conduction time (SACT) invalidates the reliability of CSNRT, since the rapidly paced atrial impulses may not reach the SAN in sufficient number to suppress the SAN. Normal recovery of the unstressed SAN could then occur at the termination of the pacing despite SAN dysfunction. The recent introduction by Strauss et al.11 of an indirect method to calculate SACT in man has been valuable in identifying previously unrecognized SAN dysfunction in adult patients.8-10 Based on preliminary reports,14, 17 the technique also may be valuable in assessing SAN function in children.

This study was undertaken to 1) obtain normal values of SAC in children without SAN dysfunction; 2) determine SACT in children with abnormal SAN automaticity; 3) determine SACT in patients with normal CSNRT but who have had an intracardiac operation, with emphasis on children who have had operations involving the right atrium.

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Methods

Subjects

The response to premature atrial stimuli was recorded in 20 subjects without clinical or electrophysiologic evidence of SAN or atrial muscle disease and compared with the data in 30 patients assigned to two groups based on a prolonged CSNRT (group 1, n = 20) or a normal CSNRT (group 2, n = 10). The "normal" group, as indicated in table 1, included 13 subjects with unoperated congenital heart defects (eight had hemodynamic abnormalities and five had anomalous conduction pathways) and seven with normal hearts (five with completely normal hearts and two with atrioventricular nodal reentry tachycardia). Subjects with dysrhythmias due to intrartrial reentry were excluded from our normal group, even though they satisfied all other criteria. Clinical criteria for normal SAN function included no history of dizziness, syncope, or chest pain associated with SAN dysfunction. Using the 12-24-hour ambulatory monitor or the standard 12-lead ECG, criteria for normal SAN function were absence of any of the following: 1) sinus bradycardia for age,18 2) sinus arrest, 3) low-amplitude P waves (<1.0 mm in both standard lead I and II),19 4) Wenckebach and other types of SAN exit block20 or 5) manifestations of bradycardia-tachycardia syndrome. Further criteria were the following: none of the normal subjects had undergone a cardiac operation and all had normal CSNRT. Forty-three ambulatory recordings were performed in 50 subjects.

The criterion for inclusion in group 1 was a prolonged CSNRT (table 1). Thirteen of the 20 patients had undergone a cardiac operation and seven of 20 showed electrocardiographic evidence of SAN dysfunction. Two patients had symptoms related to SAN dysfunction. Group 1 was further classified into two subgroups. Group 1A included patients who had undergone the Mustard operation (n = 9). Group 1B
included patients with increased CSNRT who did not have the Mustard operation (n = 11). In four of them intracardiac surgery had been performed (tables 1 and 2).

Group 2 consisted of 10 patients with normal CSNRT, but all patients had undergone an intracardiac operation. Since there is a higher incidence of SAN dysfunction in children who have had cardiac operations, these patients were excluded from our normal group even though they had normal CSNRT. Those patients who had had the Mustard operation were subgrouped into group 2A (n = 4). One patient in group 2B had an episode of dizziness, but it was difficult to attribute the symptoms to SAN dysfunction since the patient also had supraventricular and ventricular tachycardia.

Techniques

All subjects were studied in the cardiac catheterization laboratory in the sedated state. Meperidine (2 mg/kg) and promethazine (0.5 mg/kg) were given intramuscularly 30 minutes before the catheterization. Twenty-eight of the 50 subjects also were given chlorpromazine (0.5 mg/kg) at the same time and by the same route. If additional sedation was needed during the course of the procedure either diazepam (0.2 mg/kg) or ketamine (1 mg/kg) was administered by the intravenous route. The only cardiovascular drugs taken by the subjects were digoxin, propranolol, quinidine sulfate, or furosemide and all of these drugs were withheld at least 36 hours before study.

Using the percutaneous technique, electrode catheters were positioned with fluoroscopic guidance. In each subject, a quadrupolar catheter (with inter-electrode distance of 3 mm) was advanced from a femoral vein and placed in the high lateral right atrium at the junction of the superior vena cava. In each patient who had had the Mustard operation this position was achieved by manipulating the catheter along the intra-atrial baffle. The distal electrodes were used for stimulation and the proximal electrodes were used for recording the right atrial electrogram. In 32 of the subjects, a tripolar electrode catheter was advanced from a femoral vein and positioned across the tricuspid valve to record the His bundle potential. Simultaneous surface ECG leads I, II, III or I, aVF, V1, were recorded with the electrograms on an Electronics for Medicine model DR8 or VR12 photographic recorder at a paper speed of 100 or 200 mm/sec.

Determination of Sinoatrial Conduction Time

Atrial premature depolarizations (APD) were produced with a Medtronics model 5837 stimulator and coupled to the preceding spontaneous beat. The stimuli were 2 msec long and the amplitude was twice diastolic threshold. At least eight spontaneous sinus cycles were allowed between each stimulus. The single premature atrial stimulus was given throughout diastole at successively more premature intervals of 10−40 msec until the atrial refractory period was reached. A prerequisite for performing the test was the presence of sinus rhythm, defined as normal P-wave axis on the surface ECG leads plus normal atrial activation on the atrial electrograms.

The following intervals were measured from the right atrial electromgram as illustrated on the diagram in figure 1: 1) A2-A1, the spontaneous cycle length immediately before the APD; 2) A1-A2, the interval from the last spontaneous depolarization (initiated from SAN) to the induced APD (A2); 3) A2-A3, the interval from the APD to the next spontaneous depolarization (A3); 4) A3-A4, the first spontaneous cycle after the return cycle (A4). The A3-A4 interval was compared with the A2-A1 interval for the same sequence to assess the sinus cycle variability. The A2-A4 interval was not measured in the early studies, since its importance has been emphasized recently. The sinus origin of the A2 and A4 responses was demonstrated indirectly by the unchanged P-wave axis and morphology and by unchanged atrial activation sequence.

Definitions and diagrammatic examples of nonreset and reset responses, as well as a sample calculation of SACT for a single reset response, are shown in figure 1.

Some basic assumptions are inherent in the indirect calculation of SACT. It is assumed that the APD enters and resets the SAN without changing the SAN automaticity. Also, the sinus cycle length is assumed to be identical to A1-A2 (fig. 1). By knowing the sinus cycle length and measuring the A2-A3 interval of a reset response, the SACT is calculated indirectly by subtracting A2-A3 from A2-A4 to derive the total time of conduction into and out of the SAN (reset step 2, fig. 1). Some investigators use one-half of this time as the calculated SACT (reset step 3, fig. 1). It should be emphasized that antegrade and retrograde conduction may not necessarily be the same. Therefore we and others prefer to express calculated SACT as the total time (total SACT) rather than an average of antegrade plus retrograde conduction time.

To clarify the transition from nonreset to reset zone, a plot of normalized intervals (A2-A3/A2-A4 or A2-A3/A2-A1) was made in each patient (fig. 2). The corresponding A2-A1 and A2-A3 intervals for the first five reset responses (which encompassed no more than one-half of the reset zone) in each patient were averaged to calculate an average value of total SACT: Ave A2-A3 - Ave A2-A1 = Ave total SACT of the first five responses in reset zone.

Definitions

Abnormal Sinoatrial Conduction

Abnormal sinoatrial conduction (SAC) was defined as either prolonged SACT (greater than 2 standard deviations from the mean value of our normal group) or evidence of SAN entrance block (SAN EB). Complete SAN EB was defined when only a nonreset zone was obtained regardless of how short the A2-A3 that was induced (fig. 3). In this study, we defined intermittent SAN EB (fig. 4) when nonreset responses
TABLE 1. Clinical Data

<table>
<thead>
<tr>
<th>Pt no.</th>
<th>Age (years)</th>
<th>Diagnosis</th>
<th>ECG evidence of SAN dysfunction</th>
<th>Symptoms related to SAN dysfunction</th>
<th>SCL (msec)</th>
<th>AERP (msec)</th>
<th>CSNRT (msec)</th>
<th>SACT (msec)</th>
<th>SAN entrance block</th>
<th>Abnormal SAC</th>
</tr>
</thead>
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Normal group

1  4  N  None  None  528  195  228  92  No  No
2  14  PS  None  None  590  192  235  164  No  No
3  3  PSVT (AVN-R)  None  None  500  140  130  97  No  No
4  3  PSVT (LCK)  None  None  443  137  122  123  No  No
5  4  VSD/PS  None  None  392  136  42  101  No  No
6  21  WPW  None  None  787  254  157  127  No  No
7  0.5  WPW  None  None  589  <220  177  136  No  No
8  1  VSD  None  None  415  142  63  85  No  No
9  4  PDA  None  None  556  190  186  80  No  No
10  3  N  None  None  490  148  102  92  No  No
11  14  N  None  None  760  207  200  193  No  No
12  5  PS  None  None  517  <195  205  120  No  No
13  0.5  PDA  None  None  440  <205  90  94  No  No
14  5  PSVT (AVN-R)  None  None  544  120  200  172  No  No
15  14  LGL  None  None  409  135  135  119  No  No
16  15  WPW  None  None  776  190  190  188  No  No
17  1  N  None  None  510  <230  80  96  No  No
18  10  N  None  None  750  <270  150  192  No  No
19  1  LTGA/VSD  None  None  471  140  90  90  No  No
20  0.5  VSD  None  None  544  155  260  106  No  No

Group 1A

21  5  PO M  None  None  640  117  295  181  No  No
22  6  PO M  None  None  481  170  295  80  No  No
23  1  PO M  B/T  None  None  463  150  1050(J)  138  Yes  Yes
24  12  PO M  SB  None  513  170  307  198  No  No
25  2  PO M  SB  None  708  218  389  —  Yes  Yes
26  12  PO M  SA  Diz  801  263  335  201  No  Yes
27  8  PO M  None  None  651  179  504(J)  —  Yes  Yes
28  10  PO M  None  None  525  <215  775(J)  —  Yes  Yes
29  2  PO M  None  None  550  150  390(J)  —  Yes  Yes

Group 1B

30  4  PO TF  None  None  647  180  290  117  No  No
31  8  WPW  SB  None  661  230  420  144  No  No
32  4  PS/ASD  None  None  643  170  294  128  No  No
33  17  CCM  None  None  658  189  367  232  No  Yes
34  10  PO ASD  None  None  767  176  348  197  Yes  Yes
35  17  PO ASD  B/T  Sync  727  247  450  206  Yes  Yes
36  4  PO ASD/VSD  None  None  572  180  329  213  No  Yes
37  8  N  SB  None  954  315  305  228  No  Yes
38  12  N  None  None  682  220  280  152  Yes  Yes
39  4  ASD/PS  None  None  636  <210  399  146  No  No
40  10  ASD  None  None  676  <215  290  192  Yes  Yes

Group 2A

41  13  PO M  SB  Sync  729  190  200  144  No  No
42  3  PO M  SB  None  531  170  70  151  No  No
43  3  PO M  None  None  522  205  225  151  No  No
44  2  PO M  None  None  621  211  239  181  Yes  Yes

Group 2B

45  8  PO ASD/VSD  None  None  618  170  180  119  No  No
46  1  PO VSD/PS  None  None  754  254  190  169  No  No
47  7  PO ECD  None  None  554  210  210  173  No  No
48  12  PO TF  None  None  712  205  180  112  No  No
49  10  PO AS  None  Diz  616  180  70  158  Yes  Yes
50  5  PO VSD  None  None  598  235  265  172  No  No

Abbreviations: N = normal heart; PS = pulmonary stenosis; PSVT = paroxysmal supraventricular tachycardia; AVN-R = atrioventricular nodal reentry; LCK = left-sided concealed bundle of Kent; VSD = ventricular septal defect; WPW = Wolff-Parkinson-White syndrome; PDA = patent ductus arteriosus; LGL = Lown-Ganong-Levine syndrome; L-TGA = l-transposition of the great arteries; PS = postoperative Mustard operation; PO M = postoperative Mustard operation; PO TF = postoperative tetralogy of Fallot; ASD = secundum atrial septal defect; CCM = congestive myxomatous; PO AS = postoperative atrioventricular septal defect; PO AS = postoperative endocardial cushion defect; PO AS = postoperative valvotomy for aortic stenosis; SAN = sinoatrial node; B/T = bradycardia/tachycardia syndrome; SB = sinus bradycardia; SA = sinus arrest; Diz = dizziness; Sync = syncope; SCL = sinus cycle length; AERP = atrial effective refractory period; CSNRT = corrected sinus node recovery time; J = junctional; SACT = sinoatrial conduction time; SAC = sinoatrial conduction.
occurred after an initial reset zone was demonstrated while ensuring that the automaticity was essentially unchanged (A2-A4 intervals being approximately the same).

Abnormal SAN Automaticity

Sinus node automaticity was evaluated by measuring sinus node recovery time after rapid atrial pacing for 30 seconds. Beginning with a pacing rate slightly faster than the spontaneous sinus cycle length and increasing by 10-20 beats/min after each recording, the CSNRT was measured after each paced cycle length. The maximum pacing rate was approximately 200 beats/minute. However, usually the longest CSNRT was obtained at pacing rates only slightly faster than the patient’s resting cycle length. The interval from the last paced, captured atrial beat to the first sinus atrial beat was defined as the sinus node recovery time. Since the sinus node recovery time should be expressed in terms of some relationship to the resting sinus cycle length, for each paced cycle length the baseline sinus cycle length was subtracted from the sinus node recovery time to obtain the CSNRT.

The longest calculated time was used to express the CSNRT for each subject and the value of 275 msec (2 standard deviations from the mean of the normal group) was used as the upper limit of normal. When a junctional pacemaker was the first to recover, the interval from the last paced, captured atrial beat to the junctional beat was measured. The pre-pacing sinus cycle length was subtracted from this interval to calculate the corrected pacemaker recovery time. Although this is obviously not a true CSNRT, it is an abnormal response. We excluded these corrected pacemaker recovery times in the calculation of group data.

Atrial Refractoriness

The atrial effective refractory period (AERP) was defined as the longest A1-A2 interval at which the APD (A2) failed to capture the atrium. In the early studies, a few instances occurred when the shortest A1-A2 interval given still captured the atrium. Even allowing that the values of the AERP would have been shorter in these patients (if shorter A1-A2 had been given), none of them had prolonged atrial refractory periods according to the values in normal children reported by Dubrow et al. However, for statistical analysis, the mean value of the normal group was computed from the individual values in only those patients in whom the AERP was reached (table 1). Values were considered prolonged when greater than 2 standard deviations from the mean of our normal group.

Sinus Cycle Length

The sinus cycle length (SCL) was obtained by averaging the A1-A2 intervals from the first five reset determinations in each patient. In those patients in whom a reset zone was not obtained, the SCL was obtained from the average of five nonreset responses.
Since the $A_1$-$A_2$ intervals used to calculate SACT were taken at approximately the same $A_1$-$A_2/A_1$-$A_1$ in each patient, this allowed for more appropriate comparison of data. The individual and group SCL intervals listed in the tables therefore were significantly less ($p < 0.001$) than those from the resting nonsedated ECGs.

### Statistics

The $t$ test for independent means was used to compare the mean data for the study groups with that from the normal group. In addition, multiple linear regression was used to test correlations between SCL and SACT and between CSNRT and SACT in the normal group.

### Results

#### SACT in the Normal Group

In the normal group of children, calculated total SACT was $124 \pm 38$ msec (mean ± sd) (table 2). Thus, 200 msec was selected as the upper limit of normal (2 standard deviations) and none of these subjects had values of total SACT greater than 200 msec (table 1). The total SACT was unrelated to SCL ($r = 0.17$). The mean SCL was $551$ msec ± $125$ (SD). The transition from nonreset to reset zone was expressed as the initial $A_1$-$A_2/A_1$-$A_1$ of the reset zone. The mean value of the normal group was $67.8\% \pm 5$ (SD). As illustrated by the example of a normal response in figure 2, all the normal subjects had a clear transition from nonreset to reset zone.

#### SAC in Group 1 — Patients with Abnormal SAN Automaticity

The mean value of total SACT in group 1 (172 msec ± 10 SEM) was prolonged significantly ($p < 0.001$) compared with the normal group and could be calculated in 16 of the 20 patients (tables 1 and 2). Because only nonreset responses were elicited in the other four patients in group 1, SACT could not be calculated. An example of such a response is shown in figure 3.

Including patients with prolonged SACT and complete or intermittent SAN EB, 13 of the 20 patients in group 1 had evidence of abnormal SAC (tables 1 and 2). Of the 13 patients, five patients had prolonged SACT, four other patients had complete SAN EB, and four other patients had normal calculated SACT, but at shorter $A_1$-$A_2$ intervals showed SAN EB (fig. 4). After normal nonreset and reset responses, there were more nonreset responses followed by more reset responses. With shorter $A_1$-$A_2$ intervals, nonreset responses followed again, similar to those found at short $A_1$-$A_2$ in the patient with complete SAN EB (fig. 3). Reset responses followed by nonreset responses indicated intermittent SAN EB. Normal P-wave morphology and atrial activation sequence occurred in each response. Moreover, the $A_3$-$A_4$ intervals after both the reset and nonreset responses were similar, probably indicating that neither variability in the sinus cycle length nor changes in SAN automaticity accounted for the findings.
Findings from a patient who had undergone a Mustard operation demonstrated that the A₂-A₄ interval may be misleading (fig. 5). With decreasing A₁-A₂, SAN EB occurred and was associated with a normal A₃ and A₄ P-wave axis and A₃-A₄ intervals (figs. 5A and B). Decreasing the A₁-A₂ interval further resulted in apparent further SAN EB as the A₂-A₃ intervals follow the nonreset line and the A₃-A₄ intervals remained approximately the same (fig. 5A). The A₃ P-wave morphology of this (fig. 5C) and the next five nonreset responses showed an abnormal P-wave axis accompanied by an altered right atrial activation sequence (HRA₃-LRA₃ = 10 msec vs HRA₁-LRA₁ = 40 msec). Thus we could not confirm the existence of SAN EB from these five responses. However, in this particular patient SAN EB was apparent earlier (longer A₁-A₂).

SAC in Group 2 — Postoperative Patients with Apparently Normal SAN Automaticity

The mean calculated total SACT was not significantly different from that in the normal group (table 2). Although no patients had prolonged calculated SACT, one patient from each subgroup showed evidence of SAN EB after having sufficient reset responses to calculate SACT (table 1). The reset zone first was demonstrated at an average A₁₋₂-A₁₋₄/A₁₋₁-A₁ of 65.8 ± 2.1 sem% (p > 0.1).

Relation Between Previous Cardiac Operation and SAC

Among the 13 patients in group 1 with abnormal SAC, nine had undergone an intracardiac operation (tables 1 and 2), including six who had had a Mustard operation (group 1A) and three in group 1B who had undergone closure of an atrial septal defect. The other three patients in group 1A and the one other postoperative patient in 1B had normal SAC after intracardiac operation.

By selection, all of the patients in group 2 had undergone an intracardiac operation. Although no patient had a prolonged calculated SACT, two patients, one in each subgroup, had evidence of abnormal SAC by intermittent SAN EB.

**Figure 2.** A plot of A₂₋₃_A₁₋₄ and A₂₋₄/A₁₋₁, vs corresponding A₁₋₂_A₁, showing an example of a normal response. The nonreset zone is distinctly separated from the plateau reset zone. The circle around the first five reset points indicates the responses from which the average sinoatrial conduction time was calculated. The closeness to which the A₂₋₄ intervals fall along the 1.00 A₂₋₄/A₁₋₁ line suggests that there has been little change in automaticity.

**Figure 3.** A graph of the sinoatrial node response to the extrastimulus method from a patient who underwent a Mustard operation. A plot is shown of the normalized A₂₋₃_A₁ and A₂₋₄/A₁₋₁ intervals vs the A₁₋₂_A₂ interval. Decreasing the A₁₋₂ results in a continuing nonreset zone with no reset zone. In this patient, some A₂₋₄ intervals were not recorded, but for those which were recorded, they are similar to the normal response from the patient in figure 2.
Sinus Cycle Length

With the predominant influence of group 1B, group 1 had a longer ($p < 0.01$) mean sinus cycle length than the normal group (table 2). Groups 1A, 2A and 2B each had a mean sinus cycle length which was not different ($p > 0.05$) from that in the normal group (table 2).

Atrial Effective Refractory Period

The mean AERP of the normal group was 169 ± 38 (SD) msec, making 245 msec ± 2 SD the upper limit of normal. One patient from group 1A had a prolonged value, as did two in group 1B and one in group 2B (table 1).

Discussion

The calculated total SACT in our normal group was lower than that reported in some adult series. Three investigators have reported total SACT values in normal adult subjects ranging from 240–320 msec as the upper limit of normal. This is in contrast to our finding of 200 msec (mean ± 2 SD) as the upper limit of normal. The study of Jordan et al. in adults with coronary artery disease helps explain the discrepancy. They discovered that patients with significant atherosclerotic lesions of the SAN artery or in vessels proximal to the origin of the SAN artery had longer mean total SACT (238 ± 30 msec) than patients without the lesions (144 ± 10 msec). Dhinghra et al. included some patients with intraventricular conduction delay who may have had clinically inapparent coronary artery disease proximal to the origin of the sinus node artery. As emphasized by Jordan et al., this could account for the higher value of the upper limit of normal (304 msec) in the study of Dhinghra et al.

Lower normal values of SACT in adults (similar to our normal values in children) have been reported by two investigators using similar methods of calculation. Steinbeck and Lüderitz used only the first reset response and Masini et al. used the first 20% of the reset responses. We used the first five responses in the reset zone in order to avoid the possibility of using reset responses near or within the functional atrial refractory period. However, this aspect of calculation may be less important than previously thought, since all of the reset responses were used in the adult study of Scheinman et al., who found values of total SACT (142 ± 32 msec (SD)) similar to ours. Except for our preliminary report, only Dubrow et al. have reported values of SACT in children, and their technique is not stated. Thus, most evidence suggests that children and adults have similar SACT.

Abnormal Sinoatrial Conduction

SAC was considered abnormal not only if SACT was prolonged but also if there was evidence of SAN EB. Patterns of complete SAN EB and "chaotic" responses have been described with the premature atrial stimulus technique and have been considered abnormal findings. Several of our patients without prolonged calculated SACT showed evidence of intermittent SAN EB. We considered this abnormal if the responses were constant, reproducible, and not accompanied by extreme sinus cycle variability (stable $A_2$-$A_4$). The CSNR in these patients with intermittent SAN EB probably was falsely normal because of the SAN EB. Thus, when CSNR was determined after rapid atrial pacing, the abnormal SAC protected the SAN from overdrive suppression. This phenomenon has been discussed previously. If one of the advantages of using the premature atrial stimulus technique is to identify this type of patient who has abnormal SAC and normal CSNR, our

Figure 4. A) A plot of normalized $A_2$-$A_3$ and $A_3$-$A_4$ vs $A_1$-$A_2$ is shown from a patient who had had a valvotomy for valvular aortic stenosis. At an $A_1$-$A_2$/$A_1$-$A_0$ of 0.84–0.86, increased nonreset $A_2$-$A_4$ responses accompanied by increased $A_1$-$A_4$ intervals occur which probably resulted from sinus arrhythmia as suspected by the associated increased $A_2$-$A_4$. The transition from nonreset to reset is not as distinct as found in plots from normal subjects (fig. 2). Two reset responses are initially apparent at $A_1$-$A_2$/$A_1$-$A_0$, 0.63 (fig. 4B) and 0.65. However, with earlier $A_1$-$A_2$/$A_1$-$A_0$ (0.62–0.55) nonreset is demonstrated as the points follow the nonreset line (the nonreset response at 0.60 is shown in fig. 4C). When $A_1$-$A_2$/$A_1$-$A_0$ is decreased further, five more reset responses occur; intermittent nonreset responses are also demonstrated. Therefore, this is an example of intermittent sinoatrial entrance block. During the intermittent reset and nonreset responses the $A_2$-$A_4$ intervals show no significant variation, suggesting no apparent change in sinoatrial node automaticity.

B) This tracing from the same patient depicted in figure 4A shows the recording of the first reset response. Labeled from top to bottom are: standard surface ECG leads (I, II and III), high right atrial (HRA) electrogram, proximal coronary sinus (PCS) electrogram, His bundle electrogram (HBE) and the femoral artery pressure. The distance between time lines is 1000 msec. At a $A_1$-$A_2$/$A_1$-$A_0$ of 0.63 the SAN is reset (fig. 1 and text), since $A_1$-$A_2$ (1170 msec) is less than $2 \times A_1$-$A_3$ (1240 msec). Because the P waves ($A_4$ and $A_1$), atrial activation, and $A_2$-$A_4$ intervals are all unchanged, it is unlikely that sinoatrial node (SAN) automaticity has been affected. C) In a tracing from the same patient, a nonreset response is demonstrated with a shorter $A_1$-$A_2$/$A_1$-$A_0$ (0.60). The $A_1$-$A_2$ interval is approximately twice the $A_1$-$A_3$ (1190 msec vs 1200 msec), indicating the SAN is not reset (fig. 1 and text). Again, there is no evidence of a change in SAN automaticity. The paper speed and electrograms are the same as in figure 4B.
findings are not surprising. The importance of plotting all of the responses in each patient also is illustrated by these patients: SAN EB may be identified (with shorter A1-A2) after SACT is calculated from the reset responses already elicited with longer A1-A2.

Limitations

The patient with abnormal SAN automaticity presents a particular problem because the effect of the induced APD on the automaticity of the diseased SAN is unknown in man. Although attention to the A3-A4 interval should detect suppression of automaticity,27 in these patients automaticity may be affected by the APD and remain undetected with present techniques. The solution to this problem ultimately is not in the present methods but in the direct calculation of SACT. An animal study88 using specialized catheter techniques to record the endocardial SAN potential, suggests that direct calculation of anterograde SACT may be feasible in man. With this method, the dependence on the assumed sinus cycle length would be eliminated (and therefore any question of delay due to depressed SAN automaticity would be eliminated) and the direct calculation of time from the SAN potential to the atrial depolarization would be measured.

Implications

As has been stated for adults with absence of a history of syncope10, 44 more data are needed before we can conclude which invasive or noninvasive test is the most accurate index of SAN function. Clearly, however, invasive testing should include both CSNRT and SACT. Frequently, SACT was abnormal in our patients with abnormal SAN automaticity. Since SAC was abnormal in two patients without apparent depressed automaticity, some patients with SAN dysfunction may be undetected if only CSNRT is measured.

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