Sinus Node Function in Children
Factors Influencing its Evaluation

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SUMMARY Overdrive atrial stimulation was performed in 20 electrophysiologically normal children at the time of routine diagnostic cardiac catheterization, for the determination of the sinus node recovery time (SNRT). Measurements were made at pacing rates of 120, 150, 180, and 200 beats/min. The absolute recovery times demonstrated a wide range of values (420–1280 msec). Expressed as a percent of the resting P-P interval, values at pacing rates of 120 and 150 beats/min were 109–166%, with significantly less overdrive suppression ($P < 0.001$) occurring at the two faster pacing rates. Atropine diminished the maximum SNRT in all patients tested. This study reports normal values for the SNRT in children. Factors which influence the SNRT are the resting heart rate, the rate of overdrive, and the autonomic tone. In children, the sinus node recovery time should be expressed in terms of the resting sinus interval.

☐ SINUS NODE DYSFUNCTION is not a disease solely of adults, but is now also recognized to occur in children with congenital heart disease, both before and after intracardiac surgery. Those children reported have presented with sinus arrest, marked sinus bradycardia, and/or brady-tachyarrhythmias.

Assessment of sinus node function in adult patients includes overdrive atrial stimulation to determine the sinus node recovery time (SNRT). Although this electrophysiological technique has been previously used in children, the effects of pacing rate and resting heart rate on the interpretation of normal values have not been emphasized. This study was designed, therefore, to define normal values for SNRT in children, taking into account the important influences of resting heart rate and rate of overdrive stimulation. In addition, the effects of parasympathetic blockade on SNRT are evaluated.

Materials and Method

Atrial pacing was conducted in 20 children undergoing right heart catheterization for the evaluation of congenital heart disease. The patients, aged 2 to 18 years, were arbitrarily divided into three age groups: 2 to 5 years (six patients); 6 to 9 years (seven patients); and 10 years and over (seven patients). No patient demonstrated clinical or electrophysiological evidence of arrhythmia or abnormal conduction. The diagnoses of the patients, as determined by catheterization, included five patients each with tetralogy of Fallot, ventricular septal defect, and pulmonic stenosis; and one patient each with idiopathic hypertrophic subaortic stenosis, total anomalous pulmonary venous return, and truncus arteriosus, Type I. Two patients had normal hemodynamics. No patient having undergone open heart surgery was included. Two patients with tetralogy of Fallot, however, had previously undergone aortic to pulmonary anastomoses and one patient with ventricular septal defect had previous pulmonary artery banding. No patient was receiving digitalis at the time of catheterization. After obtaining informed consent, all patients were routinely premedicated with meperidine, 1 mg per kg, chlorpromazine, 0.5 mg per kg, and promethazine, 0.5 mg per kg intramuscularly. Following His bundle electrocardiography, which was performed to confirm normal conduction, a #6 or #7 French quadripolar electrode catheter was positioned along the high right atrial lateral wall so that normal or near-normal P wave morphology could be obtained by continuous pacing. High right atrial electrograms recorded from the same catheter, and standard leads, 1, 2, and 3 were all simultaneously displayed on a multi-channel oscilloscopic recorder (Electronics for Medicine DR-12), and photographed on paper running at 50 or 100 mm/sec (fig. 1). Pacing was provided by either a battery operated pulse generator (Medtronic 5837) or a Grass SD9 stimulator at rates of 120, 150, 180, and 200 beats per min. Rectangular pulses of at least 2 msec duration were provided with the current set at approximately twice the diastolic threshold (usually 2–4 milliamperes). All patients were paced for 30 seconds at the different rates. At least 30 seconds of equilibration were allowed between periods of pacing. Intravascular or cuff blood pressures were monitored during pacing in all patients.

The absolute SNRT (in msec) was measured from the last pacing artifact to the onset of the first spontaneously occurring P wave of sinus origin as seen in any of the three standard ECG leads. This value was also expressed as a percentage of the resting P-P interval. The resting P-P interval was determined by averaging the P-P of all beats during the last 10 seconds prior to each pacing period. After cessation of pacing, successive P-P intervals were also measured until the resting interval was reestablished. In ten patients, the pacing studies were repeated 5 minutes after the intravenous administration of atropine in the dosage of 0.01 mg/kg.

Results

Atrial Pacing

Stable atrial pacing was accomplished in all patients. Two patients developed Wenckebach block at pacing rates less than 200 beats/min and were not studied at that rate. One

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A patient had a resting heart rate greater than our lowest pacing rate of 120 beats/min. All patients, except one, maintained normal systemic blood pressure with pacing to 200 beats/min. One patient, with moderately severe pulmonary valvular stenosis, became hypotensive at pacing rates above 150 beats/min and was therefore studied only at the two lower pacing rates. Occasionally, the first post-pacing P wave was accompanied by an abbreviated P-R interval or had an axis different from the pre-pacing P wave axis (fig. 2). In those cases, the SNRT was not included, and after an

**FIGURE 1.** Continuous recording which shows the results of atrial pacing at 120 beats/min. Shown in panel A are the ECG (leads 1, 2 and 3), high right atrial electrogram (AE), the arterial blood pressure (BP) and a resting P-P interval of 640-660 msec. In panel B, the SNRT is measured at 860 msec (132% resting P-P) with the heart rate returning to the resting rate within three post-pacing beats. Time lines equal 1 second.

**FIGURE 2.** Tracing from a patient paced at 180 beats/min which shows, in panel A, normal P wave morphology and axis (+60°). Following pacing, panel B shows a P wave axis of 0°. This patient had normal SNRTs and postpacing P waves when paced at 120 and 150 beats/min. Symbols as in figure 1. Time lines equal 1 second.
adequate period for rate stabilization, atrial pacing was repeated. No complications were encountered as a result of these studies.

### Sinus Node Recovery Time

The absolute recovery times demonstrated a wide range of values (420–1280 msec), with the two lower pacing rates generally showing greater degrees of sinus node suppression (table 1). Linear correlations were found between the absolute recovery times and the resting P-P intervals at each pacing rate (fig. 3). Insofar as the older children generally had lower resting heart rates, and therefore longer P-P intervals, they also had longer absolute pauses.

The range of SNRTs, expressed as a % of the resting interval, was 81–166% (fig. 4, table 1). When expressed in this way, there were no significant differences in SNRT between children in the various age groups. Although suppression occurred at all pacing rates, SNRTs were significantly longer \((P < 0.001)\) at 120 and 150 beats/min with values of 136 ± 13% and 131 ± 15% (mean ± SD) respectively and a combined range of 109–166%. Markedly reduced suppression occurred at pacing rates of 180 and 200 beats/min with values of 108 ± 10% and 102 ± 9% (mean ± SD). Recovery times less than 100% of the resting P-P interval were seen in four patients and occurred only at the two faster pacing rates. The SNRTs in these four patients were 81%, 82%, 88% and 96% of the resting P-P interval. Each patient had a gradual increase in the duration of successive intervals and returned to pre-pacing levels within five post-pacing beats.

At the termination of pacing, all patients, regardless of

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### Table 1. Results of Overdrive Atrial Stimulation at Varying Rates in 20 Electrophysiologically Normal Children

<table>
<thead>
<tr>
<th>Resting P-P range (msec)</th>
<th>Pacing rate (beats/min)</th>
<th>SNRT range (msec)</th>
<th>SNRT (%) range</th>
<th>Mean</th>
<th>SD</th>
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<td>120</td>
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## Figure 3

Correlations of the absolute recovery times with the resting P-P intervals at each of the pacing rates studied. All times in msec.
pacing rate, returned to their pre-pacing P-P intervals within six beats (fig. 1). The two lower pacing rates produced gradually decreasing successive intervals while pacing at 180 and 200 beats/min generally caused acceleration for the first few post-pacing beats. After returning to the resting interval, no patient demonstrated acceleration in sinus rate for the next 30 seconds.

Atropine

Administration of atropine to ten patients produced a rise in the resting heart rate of at least 20%. In two patients, this resulted in a heart rate greater than 120 beats/min, making it impossible to pace at that rate. Atropine diminished the maximum SNRT in all patients tested by an average of 17%, but in no case did it completely abolish the post-pacing suppression (fig. 5). In addition, the pacing rate related differences in SNRT noted before the administration of atropine were no longer apparent, and patients required fewer post-pacing beats to resume their baseline P-P interval. Likewise, shortened P-R intervals and aberrated P wave axes did not accompany any initial post-pacing P wave following atropine.

Discussion

In 1884, Gaskell first demonstrated the ability of overdrive stimulation to suppress the pacemaker activity of the heart.6 In 1961, West reported that repetitive stimulation of the sinus node was followed by a brief period of asystole and then a period of accelerated firing rate. Both in situ and tissue preparation studies7,8 demonstrated a linear response between the pacing rate and the degree of sinus node suppression.

Overdrive atrial stimulation has recently been shown to be useful in evaluating sinus node function in patients with the sick sinus syndrome presenting with sinus bradycardia or bradytachyarrhythmias.9-12 In these studies, 35 to 93% of the patients with clinical sinus node dysfunction demonstrated markedly prolonged SNRTs. Although the recent reports of sinus node evaluation have concentrated on adults, sinus node dysfunction is well known to occur in infants and children. Studies on children and young athletes dying suddenly have revealed isolated pathologic disturbances in the sinus node.13 Even more common is the sinus node dysfunction which complicates congenital heart disease and intracardiac surgery.14

Stable atrial pacing was achieved in our group of 20 electrophysiologically normal patients without difficulty and without added risk or morbidity. Mandel et al.9 showed no difference in the degree of overdrive suppression when a range of pacing duration of 15 sec to 3 min was used. The 30 seconds of atrial pacing used in our studies, therefore, appear adequate. We found, as have others,9,10 that upon cessation of pacing, all patients returned to their pre-pacing P-P intervals within six post-pacing beats with no evidence of later suppression or acceleration. Because of this finding, 30 seconds of stabilization between pacing periods seemed adequate.

The finding of a good linear correlation between the absolute recovery time and the resting heart rate is in agreement with studies in adults.9 The wide range of age-related resting heart rates found in children necessitates correcting the SNRT for the resting P-P interval. Therefore, SNRT is to be expressed as a percent of the resting cycle for the rest of the discussion.

Pacing at 120 and 150 beats/min yielded SNRTs of 109-166%, which are similar to those obtained by Narula et al.10 in normal adults (115-159%). Mandel et al.9 obtained maximum pauses when pacing at 130 beats/min compared to our maximum pauses obtained at 120 beats/min. Our data showed little or no overdrive suppression at high pacing rates (180 and 200 beats/min), a result alluded to by Mandel et al.,9 but not previously stressed perhaps because pacing rates in earlier studies have generally not exceeded 150 beats/min. Pacing at rates of 180 and 200 beats/min
produced, in four instances, SNRTs less than the resting P-P interval (<100%), suggesting transient acceleration in automaticity. In these and the other patients who had abbreviated suppression following rapid pacing, accelerated sinus node function frequently occurred for up to five successive post-pacing beats. Our results in children, therefore, are comparable to normal values for SNRT obtained from studies in adults, particularly when the pacing rate does not exceed 150 beats/min and the SNRT is expressed in terms of the resting sinus cycle.

The first post-pacing P wave in most cases was of sinus origin. Pacing at 180 and 200 beats/min in some patients, however, resulted in what appeared to be an ectopic atrial pacemaker escaping before the first sinus P wave (fig. 2). Animal experiments have shown that the sinus node is less readily depressed than other cardiac pacemakers and recovers more quickly following rapid stimulation. The possibility exists that occasionally subsidiary pacemakers escaped from being depolarized during atrial pacing and were, therefore, not subject to overdrive suppression. An alternative explanation might be a transient pacing induced shift from true to latent pacemaker cells within the sinus node, resulting in alteration of the usual pattern of exit from the node. Temporary block in one or more of the atrial preferential pathways following pacing may also occur. Both of these situations could conceivably result in aberrated atrial depolarization and an abnormal P wave axis. We could not conclude, however, that this was a sign of sinus node dysfunction because this phenomenon occurred only at the two higher pacing rates.

The markedly reduced post-drive suppression found at high pacing rates can be explained on the basis of sinus node entrance block as recently described by Strauss et al. They showed that rapid atrial stimulation results in rate related incomplete sinus node entrance block, allowing only a portion of the paced beats to enter and depolarize the sinus node. This invariably leads to minimal overdrive suppression. If, at very high pacing rates, entrance block were complete, the sinus node would be totally protected and no overdrive suppression would occur.

Although sinus node entrance block is almost certainly the explanation for the lack of significant overdrive suppression at the more rapid pacing rates, it is hard to explain the occasional occurrence of transient pacemaker acceleration which we found to occur in four instances. Since the release of catecholamines has been shown to occur during overdrive, sinus node acceleration could be the result of local catecholamine release in excess of acetylcholine during rapid rates of overdrive. Alternatively a marked sinus arrhythmia could be responsible for the observed pacemaker acceleration.

Increase in vagal tone has been previously shown to enhance post-drive suppression by its effect on pacemaker cells. In the present study, atropine was shown to decrease the maximum duration of suppression in all patients, indicating that much of the overdrive suppression was mediated by vagal tone and acetylcholine release. The increased heart rate of at least 20% over resting levels was evidence of the reduction in vagal tone following atropine administration. Other factors, however, must be involved since in no patient did atropine completely abolish the suppressant effect of pacing. Studies have suggested a pacing-induced increase in extracellular \( K^+ \) as an additional factor and Lu et al. have demonstrated in animals that increasing extracellular \( K^+ \) by 50–100% results in increased post-drive suppression.

Regarding the use of premedication, it is known that meperidine may have some anticholinergic properties and that the phenothiazines in general may weakly block the actions of atropine. We compared the resting electrocardiograms, obtained one day prior to catheterization, to the continuously recorded rhythm strips obtained following premedication. We found no significant differences in P-R interval or resting heart rate. In addition, the A-H interval was normal in every patient studied, indicating normal A-V nodal conduction. We concluded, therefore, that the degree of parasympathetic tone present at the time of study (prior to atropine administration) was not significantly different from that on the previous day and that the premedication did not adversely affect the results of our studies.

The clinical entity of sick sinus syndrome occurs in children in association with congenital heart disease and intracardiac surgery. A normal sinus rate and rhythm is no assurance of normal sinus node function and all patients with sinus bradycardia are not equally subject to syncopal episodes. Evaluation of the SNRT as part of an electrophysiological study may effectively and safely determine which patients are prone to the development of prolonged periods of asystole following spontaneous tachyarrhythmias. Factors which we have shown to influence the SNRT in children, and which must be considered in its interpretation are: the resting heart rate; the rate of overdrive stimulation used; and the degree of vagal tone at the time of study.

Acknowledgment

The technical expertise of Mr. George Oku and Ms. Jean Gordon was invaluable and is gratefully acknowledged.

References

Effects of the Pacing Site on A-H Conduction and Refractoriness in Patients with Short P-R Intervals

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SUMMARY His bundle recordings were studied in four patients with short P-R and A-H intervals, and narrow QRS complexes, who had experienced several episodes of supraventricular tachyarrhythmias. The heart was paced from the high right atrium (HRA) and the coronary sinus (CS). In three patients the A-H Wenckebach phenomenon occurred at higher rates (greater than 200 pacing beats/min) when the CS was paced than when pacing was performed from the HRA. Moreover, CS pacing produced smaller increments in the A-H interval than did pacing from HRA. The extrastimulus method of testing was done. In cases 1 and 2 the functional refractory period of the A-H tissues was 15 to 25 msec shorter during CS pacing than when pacing from the HRA. In case 3, the low right atrium (LRA) as well as the other two sites were paced. A type 1 gap was seen from HRA, a type 2 gap from CS, and both types appeared when the LRA was paced. Case 4, in which the mid-right atrium (MRA) was also stimulated, had a double pathway from HRA and CS with conduction through the accessory pathway late in the cycle and through the A-V node earlier in the cycle. However, the A-V node could not be penetrated during MRA stimulation. It appeared that the pacing site influenced the A-H conduction pattern and refractoriness, possibly by changing the site and/or mode of entry of the stimulus into the pathways that are responsible for this syndrome.

\[ \text{DCL} = \text{driving cycle length.} \]

Material and Methods

Conventional cardiac catheterization and specialized conducting system studies were performed in four patients with short (less than 120 msec) P-R intervals and narrow QRS complexes. Informed consent was obtained after explaining the procedures. Pertinent clinical and surface electrocardiographical information is presented in table 1. Anti-arrhythmic drugs and digoxin were withheld for five days prior to the study.

\[ \text{Definition of Terms and Graphs} \]

The A-H interval was the time interval between atrial and His bundle deflections recorded from the bipolar lead located over the His bundle area. In our four patients, unlike those with normal P-R intervals, the A-H interval did not necessarily reflect A-V nodal conduction time.

The terms A-H Wenckebach phenomenon and A-H tissues will be used because we could not be certain which structure(s) (A-V node, accessory pathway, or both) caused the conduction disturbance. The H-V interval gave a measure of His-Purkinje conduction time.

The St-A interval reflected conduction time from the paced site to the right atrium in the vicinity of the A-V node as recorded in the His bundle lead.
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