Automaticity (postpacing impulse recovery time) of the sinus node or subsidiary pacemakers (or both) was determined by intracardiac recordings and atrial overdrive pacing in three patients with disease of the sinus node. Their electrocardiographic manifestations included sinus bradycardia, sinoatrial block, sinus arrest, atrioventricular junctional rhythm, and atrioventricular dissociation. One patient, who initially had syncope, demonstrated prolonged time for recovery of the sinus node (maximum corrected value, 3,485 msec) and impaired atrioventricular junctional automaticity (maximum corrected junctional recovery time, 1,460 msec). Of the remaining two patients without syncopal attacks, one had an adequate low atrial or junctional escape mechanism following cessation of pacing. The maximum low atrial escape interval was 1,660 msec, and the maximum junctional recovery time was 1,770 msec (corrected value, 140 msec). The third patient disclosed a prolonged maximum corrected recovery time for the sinus node (870 msec), while the maximum junctional recovery time was only 1,810 msec (corrected value, 160 msec). We conclude that automaticity of the subsidiary pacemakers in disease of the sinus node can be determined in the presence of atrioventricular dissociation or total sinus arrest, and that determination of the recovery time of the subsidiary pacemakers may be useful in identifying symptomatic patients with sinus nodal dysfunction.

**Disorders of sinus nodal function and failure of subsidiary pacemakers to escape at a physiologic rate are the electrophysiologic abnormalities common to a diverse group of dysrhythmias included in the sick sinus syndrome.** Although there have been extensive electrophysiologic studies in this clinical setting, the mechanism of failure to escape in lower atrial or subsidiary pacemakers is still unsolved. Narula and Narula studied the recovery time of the junctional pacemaker following cardiac pacing in five patients with sinus nodal dysfunction but without atrioventricular block. These investigators found that the symptoms of syncope or severe dizziness was correlated with a prolonged junctional recovery time.

We have studied automaticity (postpacing impulse recovery time) of the sinus node or subsidiary pacemakers (or both) by intracardiac recordings and atrial overdrive pacing in three patients with sinus nodal disease. Their electrocardiographic manifestations included (1) atrioventricular dissociation and sinus bradycardia, with or without sinoatrial block, in two patients, and (2) total sinus arrest, atrioventricular junctional rhythm and episodic sinus bradycardia or sinus bradycardia with atrioventricular dissociation in one patient. The results suggest that automaticity of the subsidiary pacemaker may not be so much impaired so as to produce syncopal attack even in those with advanced sinus nodal dysfunction.

**MATERIALS AND METHODS**

Three patients with sinus nodal dysfunction necessitating consideration of therapy with a pacemaker were studied. All were women, with ages of 22, 54, and 47 years. Syncopal attacks and paroxysmal palpitation were present in one patient, easy fatigability was present in another, and the remaining patient was asymptomatic.

Electrophysiologic studies were performed with the patient in the resting nonsedated postabsorptive state. Informed written consent was obtained in all patients. The His bundle electrogram was recorded by use of a previously described technique with a bipolar catheter percutaneously passed from the right femoral vein. Two additional bipolar catheters were passed via the right arm vein or veins, together or separately; one was positioned along the lateral wall of the right atrium for the high right atrial electrogram. Recordings were obtained on an eight-channel oscilloscopic photographic recorder (Electronics for Medicine DR-8) at paper speeds of 100 and 200 mm/sec. Simultaneous two-surface electrocardiographic leads (leads 1 and 3) were recorded.

Overdrive atrial pacing was performed, usually at a rate slightly faster than sinus rate and then at increments of 10 to 20 beats until a maximal pacing rate of 150 beats per minute was achieved. Each pacing period lasted for three minutes. In each patient the pacing stimuli were less than 3 mamp. The maximum corrected recovery time for the sinus node was defined as the longest atrial asystolic period (the last paced
high right atrial deflection (HRA) to the first spontaneous high right atrial deflection after sudden cessation of pacing at the rate) in excess of the average value of ten sinus cycle lengths prior to atrial pacing. The atrioventricular junctional impulse recovery time was defined as the interval between the His deflection of the last pacing beat to the His deflection of the atrioventricular junctional escape beat. The value was measured from the average of ten determinations and was corrected by reduction of the average value for the preceding ten atrioventricular junctional cycle lengths. In one patient a lower atrial escape beat constantly appeared following cessation of overdrive atrial pacing. In this patient, low atrial recovery time was defined as the interval between the low atrial impulse deflection (A) of the last pacing beat and the low atrial impulse deflection of the low atrial escape beat.

Sinoatrial conduction time was calculated using the atrial extrastimulus technique as described by Strauss et al. and could be performed in only one patient. The conduction time was obtained by measuring the difference between the A1-A2 interval and A1-A3 interval during the zone of reset and dividing by two. A mean value was calculated using all reset responses. The atrial effective refractory period was measured with the atrial extrastimulus technique in two patients during sinus rhythm. Atropine was administered intravenously in one patient after control studies, and studies were repeated ten minutes later.

**CASE REPORTS**

**Case 1**

A 22-year-old woman was admitted to the hospital because of dizzy spells and syncope. She had had syncopal attacks ever since her childhood; the heart rate was around 35 to 50 beats per minute. Electrocardiograms showed sinus bradycardia, sinoatrial block, junctional escape rhythm, and atrioventricular dissociation. The duration of the QRS complex was normal, and the axis was +80°. Therapy with a pacemaker was offered, but the patient refused. She continued to have syncopal attacks despite administration of atropine.

During electrophysiologic studies the sinus cycle length varied between 1,370 and 3,285 msec (mean, 1,783 ± 381 msec), while the junctional cycle length varied from 880 to 1,570 msec (mean, 1,413 ± 148 msec) (Fig 1A). Sinus capture beats had atrio-His (A-H) intervals between 340 and 355 msec and a His-ventricle (H-V) interval of 53 msec. Atrial overdrive pacings were performed, and both sinus and junctional recovery times were measured. The effect of termination of atrial pacing (90 beats per minute) after three minutes is shown in Figure 1B. The results of the atrial overdrive pacing are summarized in Figure 2. The maximum corrected sinus nodal recovery time was 3,485 msec. The junctional recovery time varied from 1,865 to 2,970 msec, with a corrected maximum value of 1,460 msec. The calculated mean sinoatrial conduction time was 170 msec; the atrial effective refractory period was 435 msec. Electrophysiologic studies were repeated after administration of atropine (1 mg) intravenously. The sinus cycle length ranged between 675 and 705 msec (mean 684 ± 15 msec). The maximum corrected sinus nodal recovery time was 698 msec. Junctional escape rhythm was no longer observed.

**Case 2**

A 54-year-old woman was admitted to the hospital because of general malaise and loss of weight. Seventeen years prior to admission, she had had hyperthyroidism and was treated

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Figure 2. Results of atrial overdrive pacing. Maximum corrected sinus nodal recovery time was 3,485 msec before intravenous (IV) administration of atropine (1 mg). Junctional recovery time varied from 1,865 to 2,970 msec, with corrected maximum value of 1,460 msec. Maximum corrected sinus nodal recovery time was shortened (698 msec) and junctional escape rhythm was no longer observed after administration of atropine. SB, Sinus bradycardia; SA, sinus arrest; SAB, sinoatrial block; and AV, atrioventricular (case 1).

With antithyroid medicines. In the last six years, she has had a slow and irregular pulse. Electrocardiograms revealed sinus bradycardia with rates between 36 and 46 beats per minute, sinus arrest, junctional escape beats, premature atrial beats, and left ventricular hypertrophy; the P-R interval was 0.18 second. One episode of atrial fibrillation was also recorded. The results of the uptake of radioactive iodine and a thyroid scan were compatible with thyrotoxicosis, and she was placed on therapy with methimazole (Tapazole), with improvement. Syncope was not noted throughout the whole clinical course.

During electrophysiologic studies, sinus bradycardia with anterograde conduction, junctional escape rhythm with atrioventricular dissociation, or junctional escape rhythm with retrograde atrial conduction (Fig 3A) was noted. The sinus cycle length ranged from 1,325 to 1,630 msec (mean, 1,438 ± 95 msec), and the cycle length of junctional escape rhythm ranged from 1,425 to 1,690 msec (mean, 1,586 ± 72 msec). During intact atrioventricular conduction the HRA-A interval was 35 to 40 msec, the A-H interval was 60 msec and the H-V interval was 45 msec. The results of atrial overdrive pacing are summarized in Figure 4. In all but one instance a low atrial pacemaker was the escape mechanism (Fig 3B), with a low atrial impulse recovery time ranging from 1,175 to 1,660 msec. In one instance a junctional pacemaker was the escape mechanism. The junctional recovery time was 1,770 msec, and the corrected recovery time was 140 msec.

Case 3

A 47-year-old woman was admitted to the hospital because of discomfort in the chest, palpitation, and exertional dyspnea. Twelve years ago she had undergone thyroideectomy due to thyrotoxicosis. One year prior to admission, the patient had begun to note a slow pulse rate, but there were no dizzy spells or syncope. Electrocardiograms showed sinus bradycardia, premature atrial beats, sinoatrial block, junctional escape beats, and nonspecific changes in the S-T segment and T wave. The ventricular rate was 45 to 48 beats

Figure 3. A. Control tracing. Atrioventricular (AV) junctional escape rhythm with constant retrograde atrial conduction and absent sinus activity. Junctional cycle lengths were between 1,510 and 1,520 msec, and retrograde conduction time (V-A interval) was 190 msec. B. Example of atrial overdrive pacing (100 beats per minute) in which low atrial pacemaker was escape mechanism after sudden cessation of pacing. First escape beat had P wave which was different from that of atrial pacing, and low atrial deflection (A) preceded high right atrial activity (HRA) by 10 msec. Low atrial impulse recovery time was 1,530 msec. HRAE, High right atrial electrogram; and HBE, His bundle electrogram (case 2).
duced by the dysfunction of the sinus node are actually related to the markedly delayed or absent escape mechanism of the low atrial or subsidiary pacemaker. In patients without sinus nodal disease, pacemaker cells in the atrioventricular junction provide escape beats at rates of 40 to 50 beats per minute when the sinus node defaults. On the contrary, many symptomatic patients with sinus nodal disease fail to generate atrioventricular junctional escape beats at the usual physiologic rates, resulting in ventricular responses as low as 30 beats per minute. Because of this, a new term, "sick escape pacemaker syndrome," rather than the conventional term of “sick sinus syndrome,” has recently been suggested to better define this setting of dysfunction in a more practical sense.24 Although the impaired formation of impulses by the lower pacemakers has been well documented clinically in patients with sinus nodal disease, detailed functional studies (automaticity) of the lower pacemakers in these patients have been scanty.22

Atrial overdrive pacing has been proved to be a valuable method of evaluating patients with dysfunction of the sinus node,10,11,15,18,20,24 however, application of this technique for determining the automaticity of low atrial or atrioventricular junctional tissue may not be ideal. Gaskell25 first demonstrated the depression of the intrinsic pacemaker's activity by driving the heart at a rate faster than the dominant pacemaker. In a canine study with crushed sinus node, Lange26 found that an imposed fast drive depressed the junctional pacemaker much more readily than the sinus nodal pacemaker. He also observed augmentation of the depression by sectioning the sympathetic cardiac accelerator nerves with removal of the thoracic ganglia. The normal escape interval of the lower or subsidiary pacemakers following suppression of the sinus node by atrial overdrive pacing in man is not yet available. In patients with sinus nodal dysfunction, manifesting with either sinus bradycardia with atrioventricular dissociation or total sinus arrest with low atrial or atrioventricular junctional escape rhythm, automaticity of the subsidiary pacemaker may be determined. The escape interval of the subsidiary pacemakers following termination of cardiac pacing might be of clinical significance in identifying symptomatic patients.22

Narula and Narula22 have recently studied five patients with sinus nodal dysfunction and 16 patients with complete atrioventricular block with overdrive atrial or ventricular pacing. These investigators22 found that the presence or absence of symptoms of syncope and dizziness was correlated with a corrected junctional recovery time of greater...
or less than 200 msec. In this study of three patients, the one with a history of syncopal attacks had a marked prolongation of the junctional recovery time (1,460 msec), while the two patients without a history of syncope had a corrected recovery time of less than 200 msec. These findings are consistent with those of Narula and Narula.22

It is not clear why the escape mechanism of the subsidiary pacemaker is inadequate in some patients with sinus nodal disease but not in the other; however, atrial disease occurred rather commonly in patients with sinus nodal dysfunction, and the same process could involve the atrioventricular junctional area, causing an inadequate escape mechanism.27-30

On the other hand, Demoulin and Kulbertus have recently found that the nervous structures surrounding the sinus node (perinodal ganglia and fibers) are also often the site of pathologic changes in sinus nodal disease. This involvement may cause abnormal sympathetic input and result in inappropriate escape of the junctional pacemaker. A normal sympathetic input has been stated to be essential for proper automatic function of the junctional pacemaker.31

In conclusion, determination of the recovery time of the subsidiary pacemakers is feasible in patients with sinus nodal dysfunction by overdrive atrial pacing. This technique may be useful in identifying those patients requiring therapy with a pacemaker. The automaticity of the subsidiary pacemakers may not be so much impaired as to produce syncopal attack, even in patients with advanced sinus nodal dysfunction.

ADDENDUM

Since the submission of this manuscript, another two patients have been studied. One patient who showed sinus bradycardia, sinoatrial block or sinus arrest, junctional escape beats, and paroxysmal atrial fibrillation on ECGs and had syncopal attacks disclosed total sinus arrest with atrioventricular junctional rhythm (no ventriculoatrial conduction) during electrophysiological study. Atrial overdrive pacing could not be successful by any means. The maximum corrected junctional recovery time determined by ventricular overdrive pacings was 2,610 msec. Another patient, with normal sinus rate, sinoatrial block, or sinus arrest who had severe dizzy spells disclosed the first escape beat of the atrioventricular junctional mechanism after the cessation of atrial overdrive pacings all the time, following intravenous administration of atropine (1 mg). Atrioventricular dissociation ensued, and both the sinus nodal and junctional recovery times could be determined. No junctional escape beat after termination of atrial overdrive pacing was noted at any pacing level before the administration of the atropine. Junctional recovery times were invariably shorter than any sinus nodal recovery time noted before administration of atropine. The maximum corrected sinus nodal recovery time before administration of atropine was 1,622 msec. After administration of atropine, the maximum corrected sinus nodal recovery time became shorter (1,210 msec); the maximum corrected junctional recovery time was 970 msec. Calculated mean sinoatrial conduction times before and after administration of atropine were 155 msec and 86.5 msec, respectively.

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