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Sinus Automaticity and Sinoatrial Conduction in Severe Symptomatic Sick Sinus Syndrome

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Electrophysiologic studies with recordings of sinus node electrograms were performed in 38 patients with severe symptomatic sick sinus syndrome. Thirty-two of the 38 patients had episodic tachyarrhythmias and 17 presented with syncope. The clinically documented sinus or atrial pause was 5.6 ± 2.8 s (mean ± SD).

Patients were divided into three groups according to electrophysiologic findings. Group I consisted of nine patients with complete sinoatrial block. Sinus node electrograms were recorded during the episodes of long pauses. Seven patients had unidirectional exit block, with the atrial impulse being capable of retrograde penetration to the sinus node causing suppression of sinus automaticity; two had bidirectional sinoatrial block.

Group II consisted of 22 patients with either 1:1 sinoatrial conduction (group IIa = 13 patients) or second degree sinoatrial exit block (group IIb = 9 patients) during spontaneous sinus rhythm. Sinoatrial exit block, ranging from 1 to >14 sinus beats, was observed during postpacing pauses that ranged from 1,650 to 37,000 ms (mean 7,286 ± 6,989). The maximal sinus node recovery time ranged from 770 to 5,580 ms (mean 3,004 ± 1,686) and was normal in 5 patients and prolonged in 17.

Group III consisted of seven patients with no recordable sinus node electrogram, reflecting either a technical failure or a quiescence of sinus activity. The sinus node recovery time in these seven patients ranged from 1,190 to 4,260 ms (mean 2,949 ± 1,121).

Thus, abnormalities in both sinus node automaticity and sinoatrial conduction are responsible for the long sinus or atrial pauses in the sick sinus syndrome. However, complete sinoatrial exit block can occur and cause severe bradycardia with escape rhythm; repetitive sinoatrial exit block plays a major role in producing posttachycardia pauses.

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Sick sinus syndrome is characterized by the presence of persistent sinus bradycardia with occurrence of episodic sinus or atrial pauses due to sinus arrest or sinoatrial exit block (1,2). This syndrome is frequently accompanied by episodic attacks of atrial tachyarrhythmias, including atrial fibrillation, atrial flutter and atrial tachycardia. Previous studies (1,2) suggest that abnormalities in sinus node automaticity or sinoatrial conduction, or both, produce clinical manifestations of the sick sinus syndrome. However, information is limited (3–6) regarding sinus node automaticity, sinoatrial conduction and the mechanisms responsible for long sinus or atrial pauses. The purpose of this study, therefore, was to evaluate systematically the electrophysiologic function of the sinus node in a large group of consecutive patients with severe symptomatic sick sinus syndrome to delineate the functional status of sinus node automaticity and sinoatrial conduction and their contributions to the cause of long sinus or atrial pauses.

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Methods

Study patients. The study group consisted of 38 patients with symptomatic sick sinus syndrome. All 38 patients had one or more of the following criteria for inclusion in the study: 1) syncope; 2) electrocardiographically documented sinus or atrial pause ≥3 s; and 3) severe sinus bradycardia with or without escape rhythm and with a rate <40 beats/min. There were 18 men and 20 women aged 23 to 83 years (mean 61 ± 15). Twenty-seven patients had no obvious organic heart disease, six had hypertension, two had aortic valve disease, two had uremic pericarditis and one had hypertrophic cardiomyopathy. Seventeen patients presented with syncope, 8 with near syncope and the others with dizziness (10 patients), fatigue (4 patients) or dyspnea (1 patient). Sinus bradycardia was present in 29 patients, junctional escape rhythm in 8 and ventricular escape rhythm in 1. Heart rate ranged between 19 and 57 beats/min (mean 41 ± 10). The longest documented sinus or atrial pauses ranged from 1.8 to 15 s (mean 5.6 ± 2.8). The clinical pause was recorded on the 24-h ambulatory electrocardiogram (ECG) when administration of digitalis, a beta-adrenergic blocking agent, a calcium channel blocking agent or an antiarrhythmic agent was discontinued.

Thirty-two patients had paroxysmal tachyarrhythmias, including atrial fibrillation in 15, atrial flutter in 4, atrial

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tachycardia in 17, ventricular tachycardia in 3 and paroxysmal supraventricular tachycardia in 1. Of these latter 32 patients with episodic tachyarrhythmias, the clinical rhythm was frequently dominated by the tachyarrhythmia.

Electrophysiologic studies. The study protocol was reviewed and approved by the Institutional Research Committee and informed written consent was obtained from all patients. Electrophysiologic studies were performed during the postabsorptive, nonsedated state. All cardiac medications were discontinued ≥72 h (or 5 plasma half-lives) before the study.

Two 6F quadripolar catheters and one 5F bipolar electrode catheter with an interelectrode distance of 1 cm were introduced percutaneously through the femoral veins. The first quadripolar catheter was placed across the tricuspid valve for recording the His bundle electrogram (proximal two electrodes) and for right ventricular pacing (distal two electrodes). The second electrode catheter was positioned in the high right atrium. The distal two electrodes of this catheter were positioned at the junction of the superior vena cava and the right atrium for recording the sinus node electrogram with use of filter frequencies of 0.2 to 50 Hz. The proximal two electrodes of this catheter were used to record the right atrial electrogram. The third bipolar electrode catheter was positioned at the right atrium for pacing. Surface ECG leads I, aVF and V_1 as well as intracardiac electrograms were simultaneously displayed and recorded on a multichannel oscilloscopic recorder (VR-16; Electronics for Medicine) at a paper speed of 50, 100 and 150 mm/s. A stimulus of 2 ms in duration and approximately twice the diastolic threshold was provided by a programmable digital stimulator (DTU PG 100; Bloom and Associates).

Anterograde and retrograde conduction properties were evaluated by atrial and ventricular incremental and extrastimulus testing techniques (7). The sinus node electrogram was recorded according to previously described techniques (8–10). The sinus cycle length referred to the cycle length of the sinus node electrogram when this was recordable. When the sinus node electrogram was not recordable, sinus cycle length was measured from P waves that were considered to be of sinus origin. The sinus node recovery time (3,4) was evaluated after sudden termination of overdrive atrial pacing for a minimum of 30 s and was measured as the interval from the last stimulus artifact to the first returning sinus node electrogram when it was recordable. When the sinus node electrogram was not recordable, sinus node recovery time was measured as the interval from the last stimulus artifact to the first returning P wave considered to be of sinus origin. The upper limit of normal maximal sinus node recovery time is 1,600 ms in our laboratory.

Sinoatrial conduction time was measured directly from the sinus node electrogram as the interval between the onset of the upstroke slope to the onset of atrial depolarization (9,10). The upper limit of normal sinoatrial conduction time is 130 ms in our laboratory. First degree sinoatrial block is defined as a delay in sinoatrial conduction time >130 ms, with atrial activation in response to all the sinus activity. Second degree sinoatrial block refers to the condition when one or more sinus node activities are not followed by atrial activation.

Type I sinoatrial block refers to the type of second degree block with a progressive prolongation of sinoatrial conduction time until a sinus activity is not followed by atrial activation. Type II sinoatrial block refers to the type of second degree sinoatrial block with sudden failure of sinus node activity to be followed by an atrial response without an antecedent prolongation of sinoatrial conduction time. Repetitive sinoatrial block refers to the type of second degree sinoatrial block with two or more consecutive sinus activities that are not followed by atrial responses.

Validation of sinus node electrogram. The recordings of the sinus node electrogram were validated by noting repetitive identical low frequency deflections free of baseline drifting during periods of long atrial pauses (8–10). These low frequency deflections are recorded only at a critical area over the anterior aspect of the junction between the superior vena cava and right atrium. Moving the electrode catheter a few millimeters away from this area results in disappearance of these deflections. The recordings of the sinus node electrogram were further validated by administration of isoproterenol, propranolol and atropine (11). After the control study, an isoproterenal infusion was started at a rate of 0.5 μg/min and then increased to 1 μg/min. Fifteen minutes after discontinuation of isoproterenol infusion, propranolol (0.1 mg/kg) and then atropine (0.04 mg/kg) were administered intravenously.

Statistical analysis. Values are presented as mean values ± SD. A p value ≤ 0.05 was considered statistically significant.

Results

The sinus node electrogram was recordable in 31 of the 38 patients during electrophysiologic study. The patients were classified into three groups according to the status of sinoatrial conduction and whether the sinus node electrogram was recordable. The clinical characteristics and electrophysiologic findings of these 38 patients are summarized in Table 1. Cases 1 and 2 have been reported previously (11).

Group I: complete sinoatrial block (Table 1). In nine patients (Cases 1 to 9), sinus activities were recorded on the sinus node electrogram especially during the periods of posttachycardia or postpacing long atrial pauses (Fig. 1). The average sinus cycle length in these nine patients ranged from 660 to 6,075 ms (mean 2,269 ± 1,704). Pharmacologic interventions were performed in eight of the nine patients; the cycle length of the sinus activities shortened during isoproterenol infusion, lengthened after intravenous administration of propranolol and again shortened slightly after atropine administration (11) (Fig. 1). These sinus node deflections were not related to the spontaneous atrial beats during either bradycardia or tachycardia, suggesting the

Table 1. Clinical Characteristics and Electrophysiologic Findings in 38 Patients

Case No.	Age (yr)/ Gender	Heart Disease	Symptoms	Clinical Bradycardia (beats/min)	Longest Clinical Pause (s)	Tachyarrhythmias	Sinus CL During EPS (ms)	Direct SACT (ms)	Maximal SNRT (ms)	Longest Sinus or Atrial Pause (ms)	Maximal No. of SAB
					Group I						
1	32/M	None	Dizziness	SB (37)	8	AF1	660 (570–1,200)		1,200	5,040	
2	48/M	Uremic pericarditis	Dizziness	SB (30)	10	AT	845 (720–1,650)		1,650	6,350	
3	23/M	None	Syncope	SB (47)	3	AT	6,075 (1,800–14,500)		14,500	3,050	
4	55/F	None	Fatigue	JER (37)	3.4	AT	3,816 (870–8,660)		8,660	4,350	
5	71/M	None	Near syncope	SB (50)	4.6	AT	1,580 (900–3,000)		3,000	3,650	
6	57/M	None	Syncope	JER (30)	4.4	AF,AT	2,070 (740–6,000)		6,000	13,000	
7	54/F	None	Near syncope	SB (38)	7.9	AT	2,380 (420–6,800)		6,800	7,800	
8	54/F	None	Syncope	JER (34)	8.2	AF,AT	1,625 (850–2,400)			6,580	
9	71/F	Hypertrophic cardiomyopathy	Syncope	SB (57)	10	AF	1,373 (600–2,350)			>17,000	
					Group IIa						
10	83/M	None	Near syncope	VER (37)	15	None	1,160	95	1,750	6,600	5
11	59/F	HCVD	Syncope	SB (50)	2.5	AT,VT	830	110	1,840	9,160	5
12	63/F	None	Syncope	SB (50)	3.4	AT,AF	670	70	2,240	8,800	4
13	78/F	None	Near syncope	SB (40)	3.8	AT	1,500	120	5,550	6,750	3
14	60/M	HCVD	Syncope	SB (48)	1.8	AF	640	60	5,550	5,550	1
15	71/F	None	Near syncope	SB (42)	6	AF	810	120	5,580	5,900	1
16	61/M	None	Dyspnea, dizziness	SB (33)	4.4	None	1,200	150	825	1,650	1
17	87/F	None	Syncope	SB (43)	5.8	AF	1,570	175	770	3,500	3
18	48/F	None	Syncope	SB (50)	6.1	PSVT	1,170	280	2,330	5,440	5
19	81/F	None	Syncope	SB (50)	8	AF,AF1,VT	690	160	2,600	8,000	4
20	24/M	None	Dizziness	SB (25)	7	AT	920	150	3,450	6,200	3
21	75/F	None	Syncope	SB (30)	8.5	AF	1,940	245	5,420	5,820	3
22	69/M	None	Fatigue	JER (30)	3	AF,AF1	1,000	140	3,200	37,000	>14
					Group IIb						
23	35/M	None	Syncope	SB (52)	2.6	AF1	1,180	Type I SAB and 2:1 SAB	1,600	9,750	8
24	70/M	None	Syncope	SB (56)	2.6	None	1,080	2:1 SAB	1,200	3,220	2
25	62/F	HCVD	Dizziness	SB (43)	4.1	AT	1,280	Type II SAB	1,540	4,080	2
26	64/F	None	Dizziness	SB (43)	3.3	None	840	Type II SAB	1,800	3,780	3
27	45/M	None	Syncope	JER (27)	2.2	None	900	2:1 SAB	2,140	4,440	2
28	59/M	HCVD	Syncope	SB (50)	3.8	AT	1,080	2:1 SAB	3,000	3,300	1
29	62/M	None	Fatigue, dizziness	SB (42)	4.2	None	730	Type I SAB	5,200	5,750	1
30	53/M	None	Near syncope	JER (43)	8.2	AF	1,130	Type II SAB and repetitive SAB	3,100	9,510	6
31	77/F	AR	Near syncope	JER (57)	7	AT,VT	920	Type II SAB	5,400	6,100	1
					Group III						
32	83/M	HCVD	Syncope	SB (52)	7.1	AT	730		1,190		
33	65/F	HCVD	Near syncope	SB (19)	7	AT	930		2,000		
34	61/F	Uremic pericarditis	Fatigue	JER (25)	5.6	AF	920		2,500		
35	53/M	None	Syncope	SB (40)	8.2	AF	1,050		3,120		
36	75/F	None	Dizziness	SB (48)	3.4	AT	960		4,260		
37	66/F	AS,AR	Dizziness	SB (40)	4.4	AF	1,000		4,100		
38	56/F	None	Dizziness	SB (38)	3.4	AF	610		3,470		

AF = atrial fibrillation; AFl = atrial flutter; AR = aortic regurgitation; AS = aortic stenosis; AT = atrial tachycardia; CL = cycle length; EPS = electrophysiologic study; F = female; HCVD = hypertensive cardiovascular disease; JER = junctional escape rhythm; M = male; PSVT = paroxysmal supraventricular tachycardia; SAB = sinoatrial block; SACT = sinoatrial conduction time; SB = sinus bradycardia; SNRT = sinus node recovery time; VER = ventricular escape rhythm; VT = ventricular tachycardia.

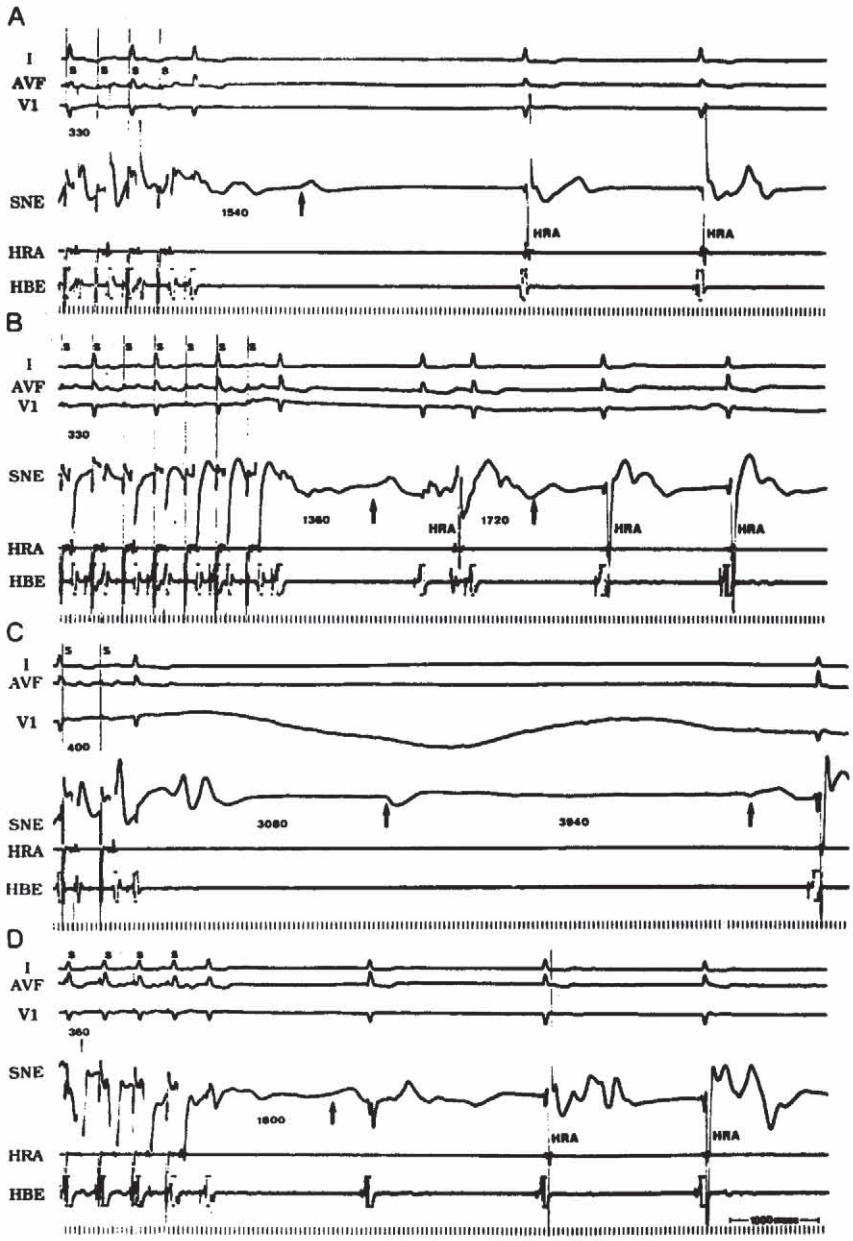

Figure 1. Case 4. Recordings showing emergence of sinus node deflections (**arrows**) during the postpacing long atrial pause (**A**) and the effects of isoproterenol (**B**), propranolol (**C**) and atropine (**D**) on sinus node recovery time and cycle length of sinus activities. Shown are electrocardiographic leads I, aVF and V_1, sinus node electrogram (SNE), high right atrial electrogram (HRA) and His bundle electrogram (HBE). The paper speed was 50 mm/s. **Panel A** shows a sinus deflection occurring 1,540 ms after cessation of atrial pacing at a paced cycle length of 330 ms before drug intervention. **Panel B** shows two sinus deflections after atrial pacing during isoproterenol infusion. The first deflection occurred 1,360 ms after pacing and the second 1,720 ms after the first deflection. Thus, isoproterenol shortened the sinus node recovery time as well as the cycle length of sinus activities. **Panel C** shows two sinus deflections after atrial pacing performed after administration of propranolol. The first deflection occurred 3,080 ms after pacing and the second 3,940 ms after the first deflection. Thus, propranolol lengthened both the sinus node recovery time and the cycle length of sinus activities. **Panel D** shows a sinus deflection occurring 1,800 ms after pacing performed after administration of propranolol and atropine. Thus, atropine shortened sinus node recovery time. S = stimulus artifact.

presence of complete sinoatrial exit block (Fig. 2 to 5). The apparent "sinus" rhythm on the surface ECG in these nine patients, therefore, reflects an atrial escape rhythm. In seven patients, slowing of sinus activities with subsequent acceleration was observed after sudden termination of overdrive atrial pacing, a finding suggesting that the atrial impulse was capable of retrograde penetration to the sinus node, causing suppression of sinus node automaticity (Fig. 3). The sinus node recovery time was normal in one patient (Case 1), slightly prolonged in another (Case 2) and markedly lengthened in the other five patients (Cases 3 to 7). In the remaining two patients (Cases 8 and 9), the sinus activities did not appear to be disturbed by overdrive atrial pacing, suggesting that both exit and entrance sinoatrial block were present (Fig. 4 and 5).

Group II: second degree sinoatrial exit block (Table 1). In 22 patients (Cases 10 to 31), sinus node activity was recorded before each atrial depolarization during spontaneous sinus rhythm. The patients were further classified into two groups according to whether spontaneous second degree sinoatrial block occurred during sinus rhythm. Group IIa consisted of 13 patients (Cases 10 to 22) with 1:1 sinoatrial conduction during sinus rhythm. The sinus cycle length ranged from 640 to 1,940 ms (mean of 1,085 ± 394) and the sinoatrial conduction time ranged from 60 to 280 ms (mean 144 ± 63). Six patients (Cases 10 to 15) had a normal and seven (Cases 16 to 22) had a prolonged sinoatrial conduction time (Fig. 6). Group IIb consisted of nine patients (Cases 23 to 31) with spontaneous second degree sinoatrial exit block during sinus rhythm. This involved type I block in two patients (Cases 23

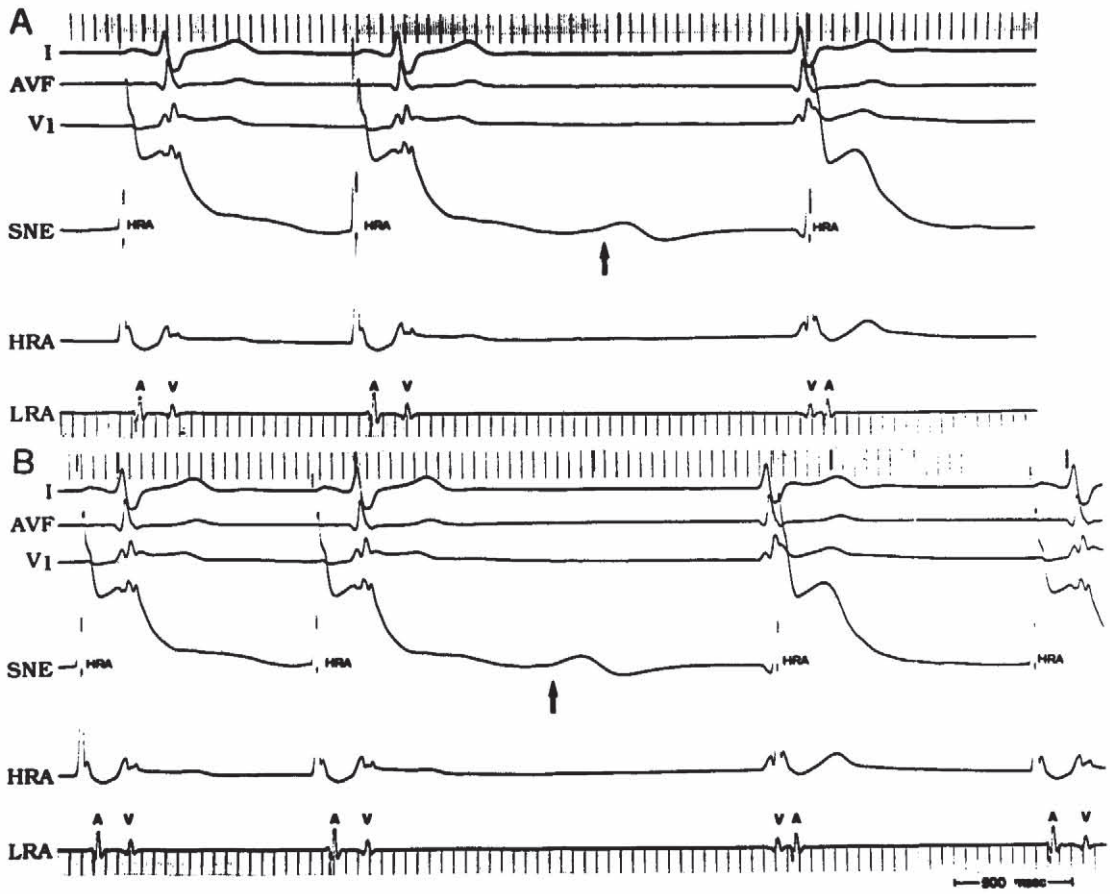

Figure 2. Case 3. Continuous recordings during spontaneous rhythm showing complete sinoatrial exit block with intermittent sinus node activity. The first two beats in **panels A** and **B** are atrial escape beats that were not preceded by a sinus deflection; the third beat on both panels is a junctional escape beat that was not preceded by a P wave. Low frequency deflections suggesting sinus activities (**arrows**) are noted intermittently on sinus node electrogram recordings. The paper speed was 100 mm/s. A = atrial depolarization; LRA = low right atrial electrogram; V = ventricular depolarization; other abbreviations as in Figure 1.

and 29) (Fig. 7), type II block in three (Cases 25, 26 and 31), 2:1 block in three (Cases 24, 27 and 28) and type II block with spontaneous high grade repetitive block accompanied by spontaneous sudden slowing of sinus activity in one (Case 30) (Fig. 8).

A long sinus or atrial pause after termination of spontaneous tachycardia or sudden cessation of overdrive atrial pacing occurred in all 22 patients. Sinoatrial exit block was observed during the period of long pauses in all 22 patients (Fig. 6B, 7B, 7C and 9). The maximal number of sinoatrial exit block beats ranged from 1 to >14 sinus beats. Repetitive sinoatrial exit block with block of more than one sinus impulse was observed in 16 patients (Cases 10 to 13, 17 to 27 and 30). The maximal sinus node recovery time was normal in 5 patients (Cases 16, 17 and 23 to 25) and prolonged in the remaining 17 (Fig. 6, 7 and 9).

Group III: no recordable sinus activity (Table 1). In seven patients (Cases 32 to 38), a sinus node electrogram could not be recorded before each spontaneous atrial depolarization or during the episodes of prolonged sinus or atrial pauses despite a meticulous search around the area between the junction of the superior vena cava and right atrium, even during isoproterenol infusion (four patients). The sinus cycle length in these seven patients ranged from 610 to 1,050 ms (mean 886 ± 158). The maximal sinus node recovery time was normal in one patient (Case 32) and prolonged in six (Cases 33 to 38).

Discussion

Mechanism of long pauses in sick sinus syndrome. The sick sinus syndrome encompasses a number of features, including persistent sinus bradycardia, sinus arrest or sinoatrial exit block and frequent episodic atrial tachyarrhythmias (1,2). A prolonged sinus or atrial pause with ventricular asystole usually occurs on termination of atrial tachyarrhythmia and results in clinical syncope or severe dizziness. Abnormalities in sinus node automaticity and in sinoatrial conduction have been proposed as mechanisms responsible for the long pause. Indeed, persistent sinus bradycardia is

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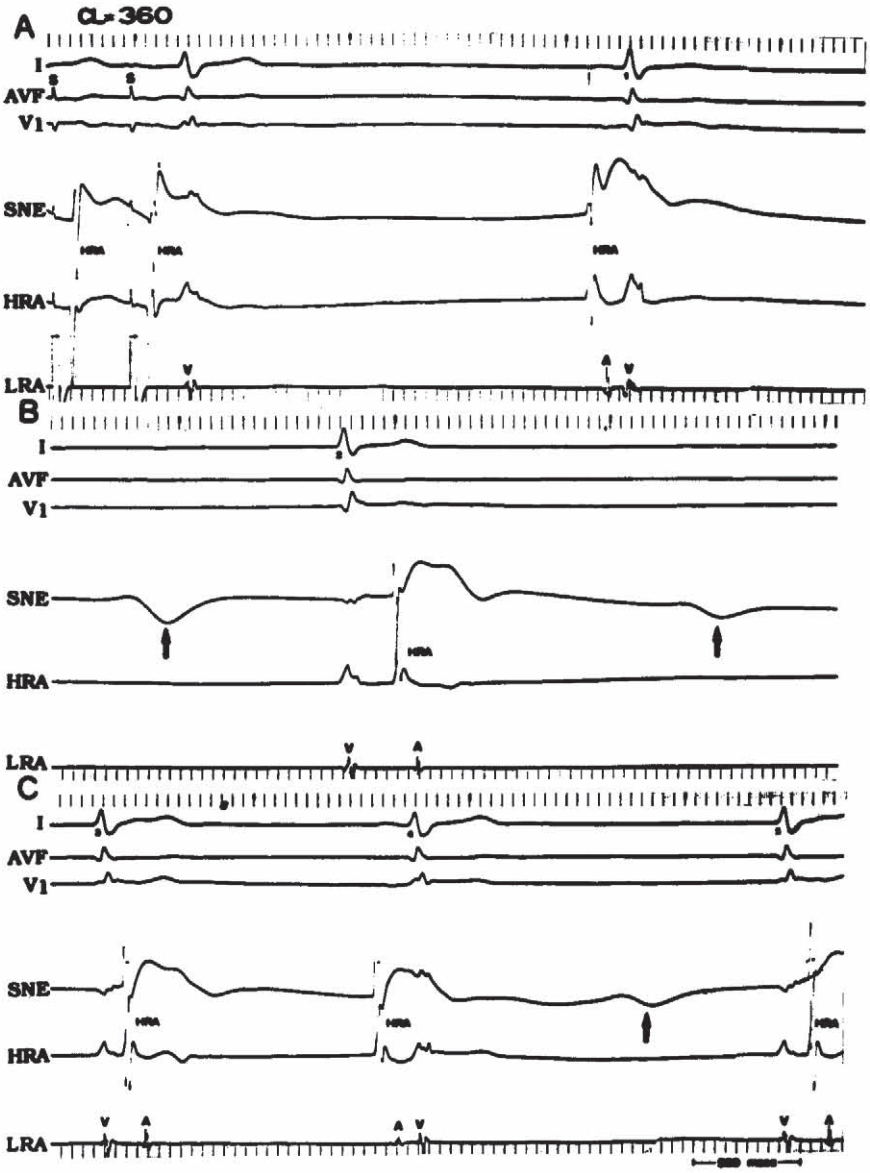

Figure 3. Case 3. Continuous recordings showing complete unidirectional sinoatrial exit block with suppression of sinus automaticity after termination of overdrive atrial pacing at a paced cycle length (CL) of 360 ms. The Arabic numerals under the QRS complex indicate the postpacing ventricular beats. The first and the fourth beats after overdrive pacing are atrial escape beats that were not preceded by a sinus deflection; the second, third and fifth beats are junctional escape beats that were not preceded by a P wave. Three low frequency deflections suggesting sinus activities (**arrows**) are observed. The first deflection occurred 3,660 ms after termination of overdrive pacing (sinus node recovery time). Abbreviations as in Figures 1 and 2.

strong evidence of suppressed sinus automaticity. Because overdrive suppression of sinus node automaticity is a physiologic phenomenon, it is logical to consider an exaggerated suppression of sinus node automaticity on termination of atrial tachyarrhythmia as the mechanism of sinus or atrial pause. The measurement of sinus node recovery time with overdrive atrial pacing is designated for this purpose (3,4).

Nonetheless, with the innovation of sinus node electrogram recording, sinoatrial exit block has emerged as the major mechanism responsible for the long pause (5,6,9,11,12). Using this technique, Reiffel et al. (9) first demonstrated a sinus depolarization without atrial activation in a patient with spontaneous sinoatrial exit block. Other workers (5,6) demonstrated repetitive sinoatrial exit block on termination of overdrive atrial pacing in several patients with the sick sinus syndrome. In a preliminary report from our laboratory, Yeh et al. (11) demonstrated complete sinoatrial exit block with a slow atrial escape rhythm in two patients with the tachycardia-bradycardia syndrome. The present study further demonstrates a wide spectrum of abnormalities in sinus node automaticity and sinoatrial conduction in patients with the sick sinus syndrome.

Complete sinoatrial exit block. Complete sinoatrial exit block was noted in 24% of our patients. In these patients, the surface ECG "sinus" bradycardia was, in fact, a slow atrial escape rhythm, and the long pause was the result of overdrive suppression of the atrial escape pacemaker. It should be noted that most patients had preserved atriosinus conduction; the atrial impulse was capable of retrograde penetration to the sinus node, causing overdrive suppression. Also, most patients had marked slowing of sinus activity, with striking overdrive suppression of sinus node automaticity. Thus, these patients had very abnormal sinus node automaticity and sinoatrial conduction. Marked suppression

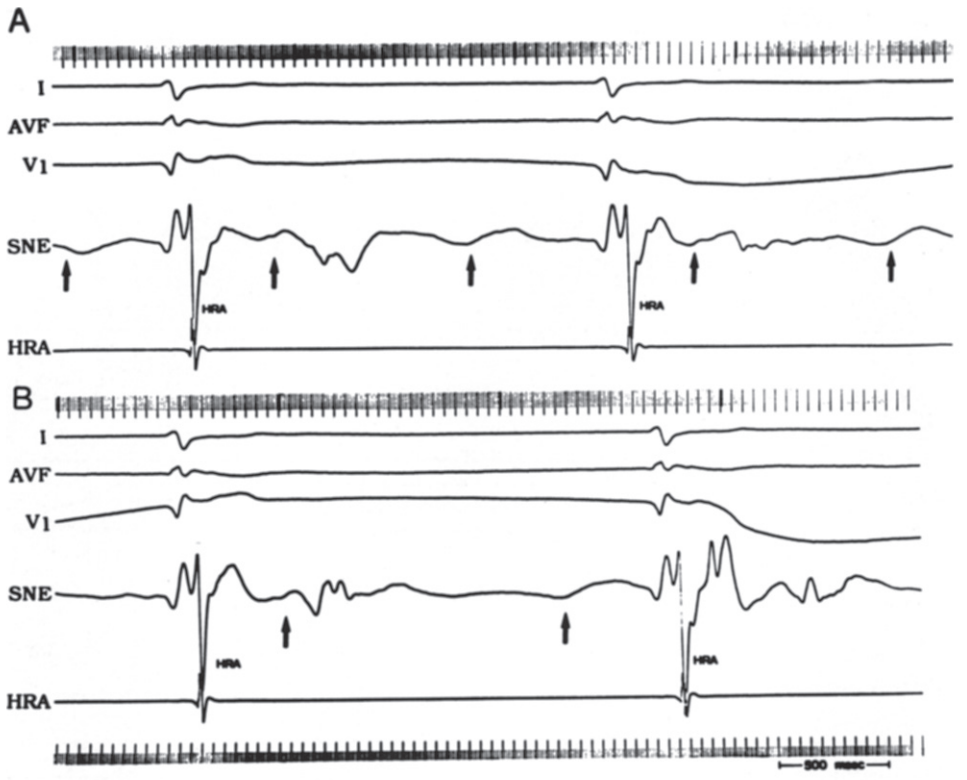

Figure 4. Case 8. Continuous recording during spontaneous junctional rhythm showing complete bidirectional sinoatrial block. The junctional rhythm has 1:1 retrograde junctionoatrial conduction. The sinus node electrogram (SNE) recorded low frequency deflections (**arrows**) suggesting sinus activities with a cycle length ranging from 850 to 1,250 ms. These low frequency deflections were not interfered with by the atrial activations. Abbreviations as in Figure 1.

of sinus node automaticity in these patients with preserved atriosinus conduction raises the possibility that sinoatrial exit block could be incomplete because the sinus activities might also be suppressed repetitively by retrograde atrial impulses. This possibility could also explain why the sinus electrograms in these patients were not readily discernible during spontaneous atrial escape rhythm and were only apparent during the periods of long pauses.

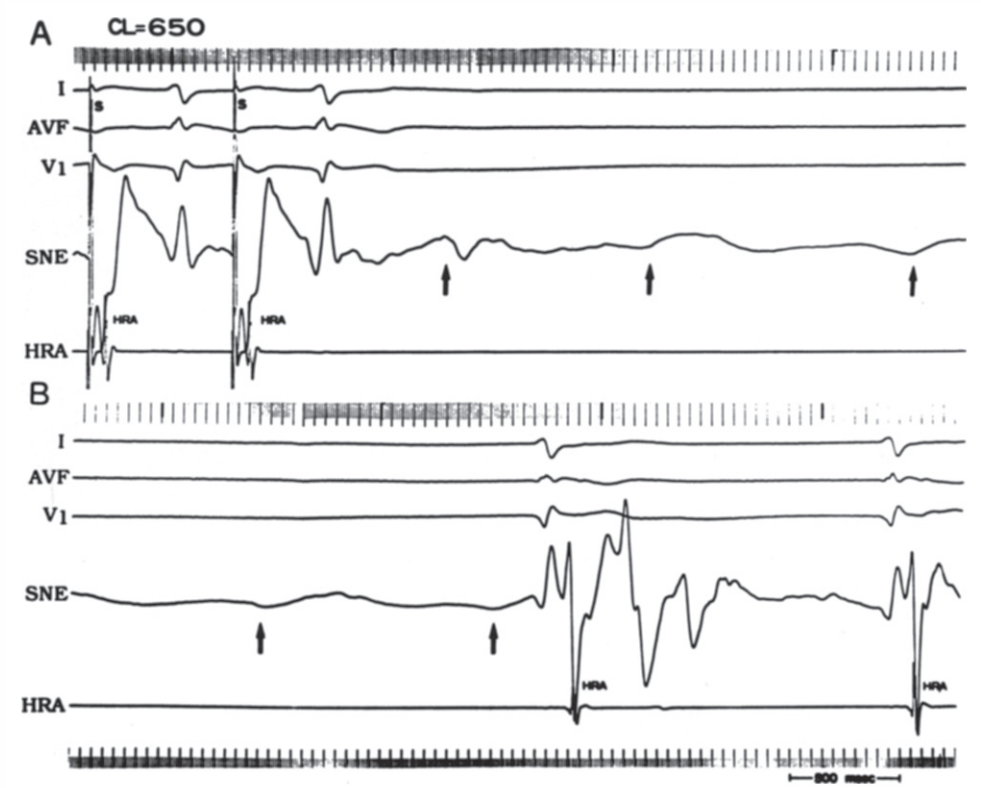

Figure 5. Case 8. Continuous recording showing complete bidirectional sinoatrial block after termination of overdrive atrial pacing at a paced cycle length (CL) of 650 ms. Five repetitive low frequency deflections (**arrows**) suggesting sinus activities are observed. The cycle length varies from 950 to 1,200 ms and was not disturbed by overdrive atrial pacing. Abbreviations as in Figure 1.

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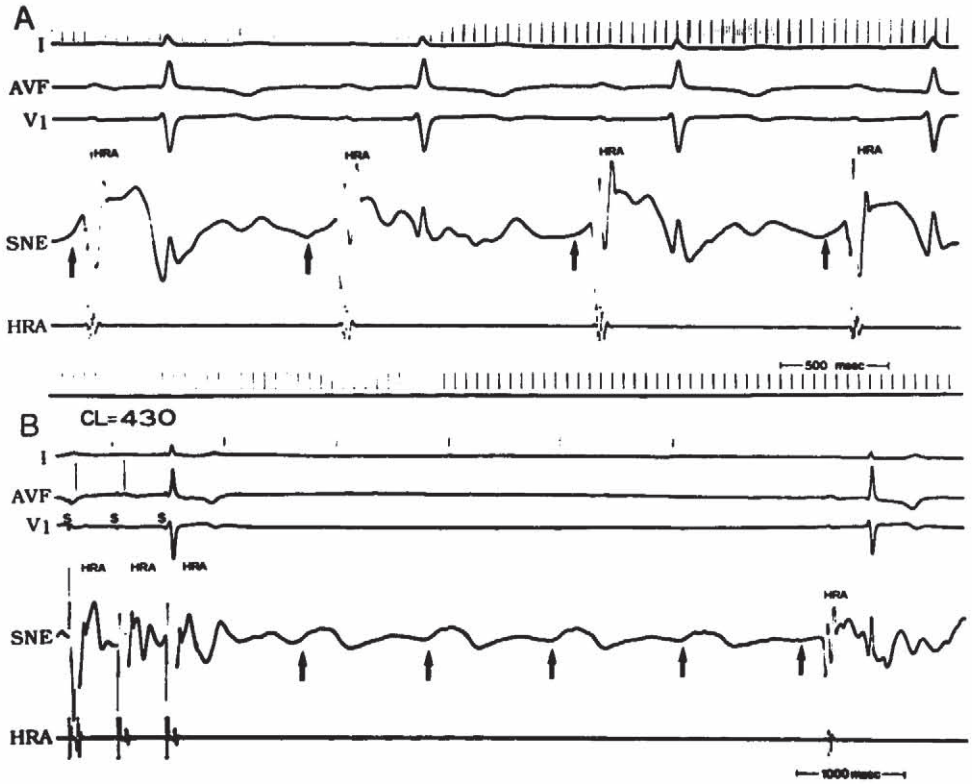

Figure 6. Case 10. Recordings showing 1:1 sinoatrial conduction during spontaneous sinus rhythm (**A**) and repetitive sinoatrial exit block after termination of overdrive atrial pacing (**B**). The paper speed was 100 mm/s in **panel A** and 50 mm/s in **panel B**. **Panel A** shows 1:1 sinoatrial conduction during spontaneous sinus rhythm. The sinoatrial conduction time varies from 70 to 130 ms and averages 95 ms. **Panel B** shows a prolonged sinus pause of 5,850 ms after termination of overdrive atrial pacing at a paced cycle length (CL) of 430 ms. Four low frequency deflections (**arrows**) suggesting sinus activities are observed during the pause; the fifth deflection is followed by an atrial response with resumption of sinus rhythm. The cycle length of the sinus activities is 1,160 ms and was reset by overdrive atrial pacing with minimal suppression (sinus node recovery time of 1,350 ms). Abbreviations as in Figure 1.

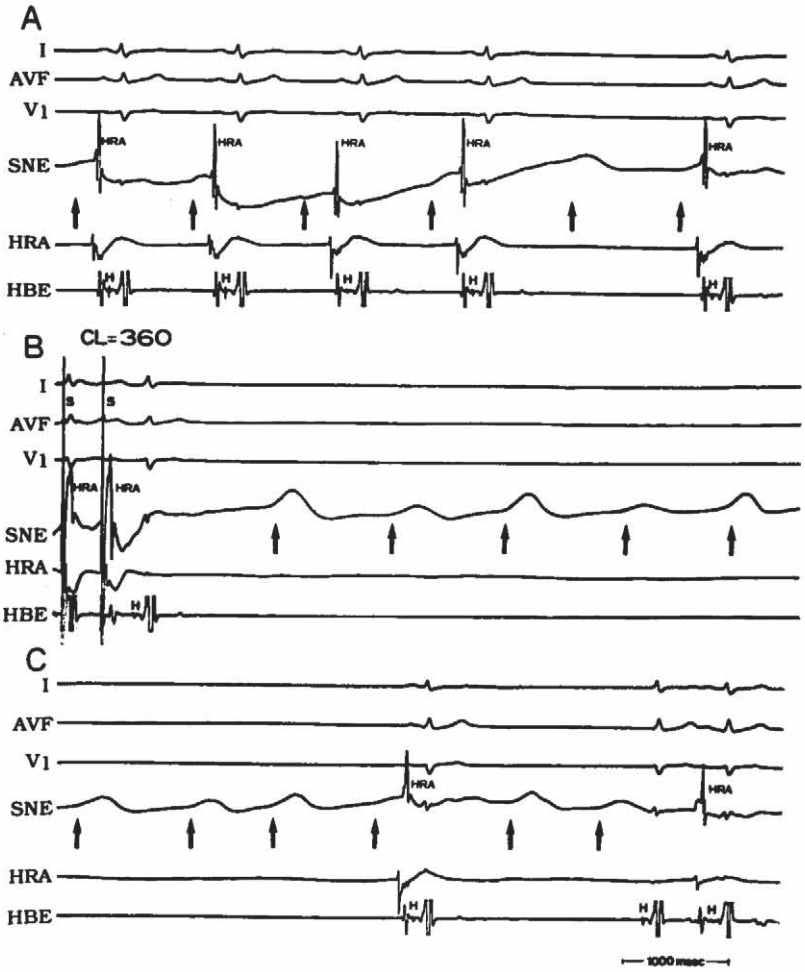

Figure 7. Case 23. Recordings showing type I sinoatrial block during spontaneous sinus rhythm (**A**) and repetitive sinoatrial exit block after termination of overdrive atrial pacing (**B** and **C**). **Arrows** indicate onset of the sinus node electrogram. **Panel A** shows type I sinoatrial exit block during spontaneous sinus rhythm; the sinoatrial conduction time lengthened progressively from 155 to 275 ms before a blocked sinus impulse occurred. **Panels B** and **C** are continuous recordings and show a prolonged sinus pause of 9,750 ms after termination of overdrive atrial pacing at a paced cycle length (CL) of 360 ms. Eight low frequency deflections (**arrows**) suggesting sinus activities are observed during the pause. The ninth deflection is followed by atrial activation. The subsequent two low frequency sinus deflections are again blocked before resumption of spontaneous sinus rhythm. The cycle length of sinus activities varies from 780 to 1,300 ms and averages 1,180 ms. The low frequency sinus deflection was minimally suppressed by overdrive atrial pacing (sinus node recovery time of 1,600 ms). Abbreviations as in Figure 1.

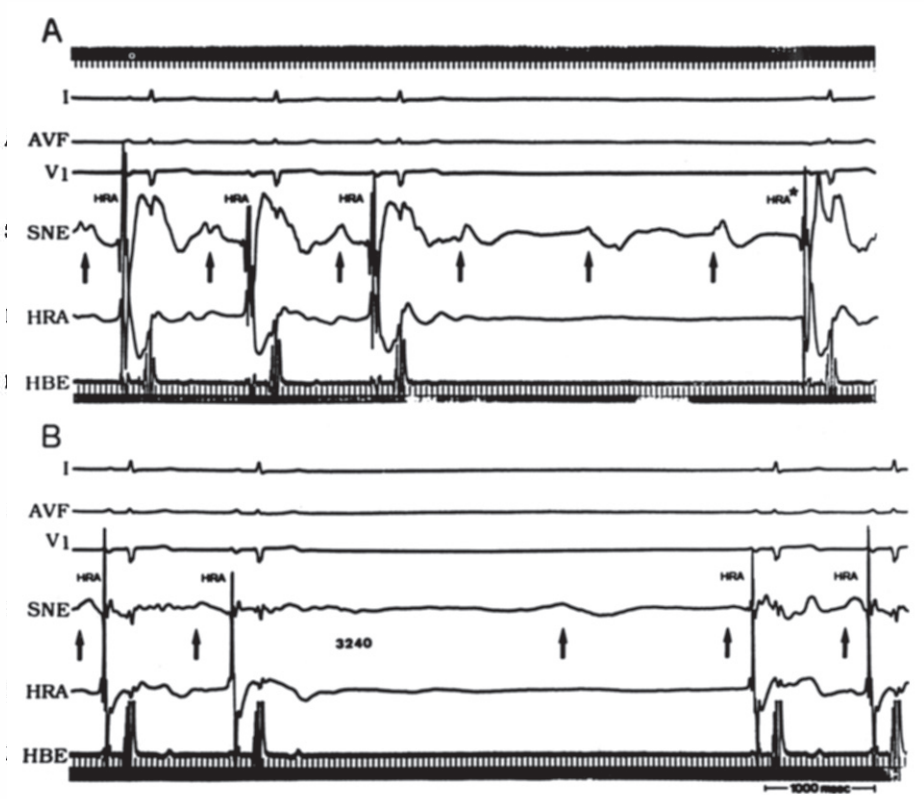

Figure 8. Case 30. Recordings during sinus rhythm showing spontaneous repetitive sinoatrial block (**A**) and spontaneous slowing of sinus automaticity (**B**). The paper speed was 50 mm/s. **Panel A** shows a sudden unexpected sinus arrest with an atrial escape beat (indicated by a **star**). Three low frequency deflections suggesting sinus activities (**arrows**) are observed during the arrest. The cycle length of the low frequency deflections is similar to that of sinus rhythm and is 1,130 ms. The sinoatrial conduction time is 310 ms and did not show a prolongation before sinoatrial block occurred. **Panel B** shows a sudden unexpected pause of 4,600 ms. One low frequency sinus deflection (**arrow**) without an atrial response is noted 3,240 ms after the last conducted sinus impulse. The low frequency sinus deflections then accelerate to the preceding cycle length. Abbreviations as in Figure 1.

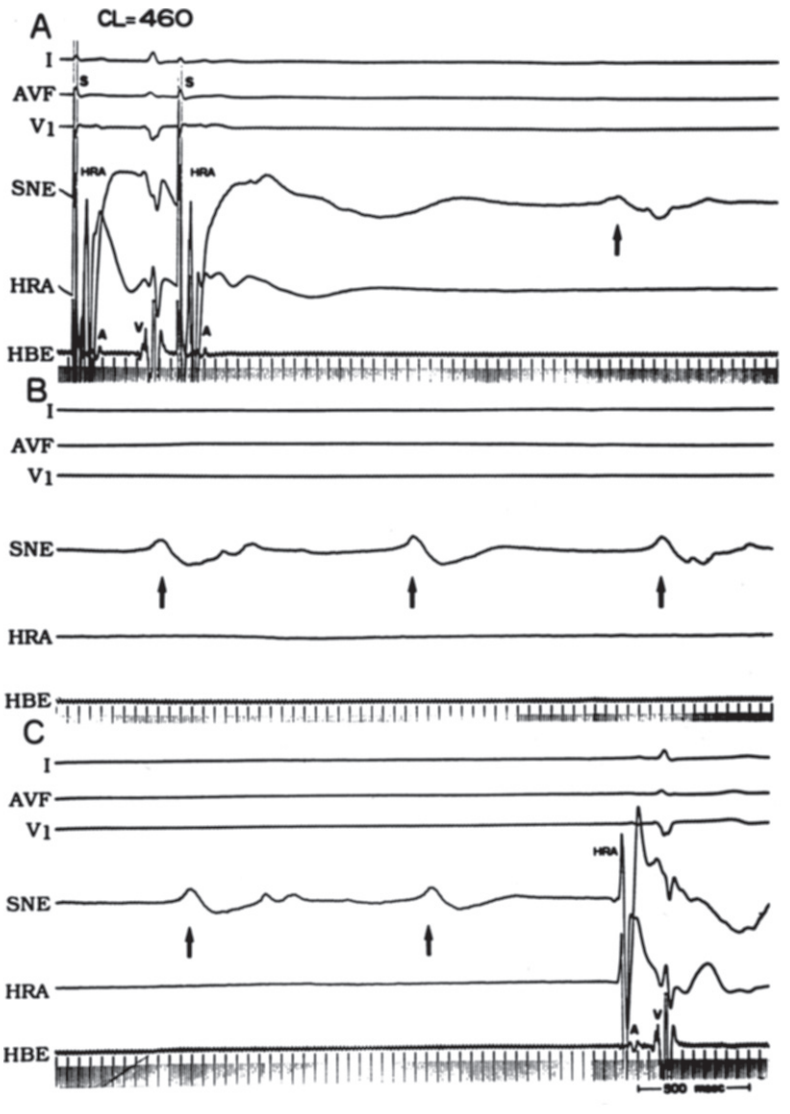

Figure 9. Case 30. Continuous recording showing repetitive sinoatrial exit block after termination of overdrive atrial pacing. A prolonged sinus pause of 8,250 ms occurs after termination of overdrive atrial pacing at a paced cycle length (CL) of 460 ms. Six low frequency deflections are observed during the pause. The first atrial beat is either a sinus beat conducted from the last low frequency sinus deflection or an atrial escape beat. The sinus node recovery time is 1,800 ms. Abbreviations as in Figure 1.

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Second degree sinoatrial exit block. Fifty-eight percent of our patients manifested a single or repetitive sinoatrial exit block during long sinus or atrial pauses. These patients had either normal sinoatrial conduction or various degrees of incomplete sinoatrial exit block during the spontaneous sinus rhythm. Repetitive sinoatrial exit block alone without suppression of sinus node automaticity was responsible for the long pauses on termination of overdrive pacing in some patients. In most patients, however, a combination of repetitive sinoatrial exit block and overdrive sinus suppression was responsible for the long pauses.

No recordable sinus node electrograms. Eighteen percent of our patients had no recordable sinus node electrogram. This lack may reflect a technical failure as sinus node electrograms frequently cannot be recorded because of baseline drift. However, it is possible that the lack was a result of complete sinus node quiescence. In this case the ECG "sinus" rhythm in these patients would reflect an atrial escape rhythm, and the long pause would reflect overdrive suppression of the atrial escape pacemaker.

Pathophysiologic considerations. The anatomic substrate of the sick sinus syndrome has not been adequately studied. There has been no good study correlating the electrophysiologic findings and the morphologic changes of the sinus node. Thery et al. (13) examined the sinus node in 111 patients. Twelve had sinoatrial block, six had the bradycardia-tachycardia syndrome and one had atrial standstill. Degeneration, loss of node cells and fibrosis of the sinus node were noted in these patients. Evans and Shaw (14) examined the sinus node in eight patients with the sick sinus syndrome and found abnormalities in seven. The sinus node was atrophic, completely surrounded by adipose tissue and appeared to be cut off from the atrial myocardium on the sinoatrial node approaches. Bharati et al. (15) studied two adolescent patients with the sick sinus syndrome and found degeneration, fatty infiltration and fibrosis of the approaches to the sinus node, with only minimal changes in the sinus node itself. These findings provide a morphologic basis for abnormalities in both sinus node automaticity and sinoatrial conduction in patients with sick sinus syndrome.

Conclusions. Several conclusions can be drawn from the present study. First, a combined abnormality in sinus node automaticity and sinoatrial conduction is usually present in patients with the sick sinus syndrome. Second, complete sinoatrial block can occur. Under such circumstances, the apparent "sinus" rhythm on the surface ECG actually reflects an atrial escape rhythm. Third, unidirectional block with complete sinoatrial exit block and a relatively well preserved retrograde atriosinus conduction can occur. Fourth, repetitive sinoatrial exit block plays a major role in causing the long sinus or atrial pause after termination of atrial tachyarrhythmia in those patients with preserved anterograde sinoatrial conduction during spontaneous sinus rhythm. Overdrive suppression of sinus node automaticity usually also contributes to the long pause.

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