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CASE REPORTS

Complete Sinoatrial Block in Two Patients With Bradycardia-Tachycardia Syndrome

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Electrophysiologic studies with recordings of sinus node electrograms were performed in two patients with bradycardia-tachycardia syndrome. In both patients, the rest electrocardiogram showed apparent sinus bradycardia. Patient 1 had frequent paroxysms of atrial tachycardia with long pauses of up to 10 seconds; Patient 2 had paroxysmal atrial flutter and atrial pauses of up to 8 seconds. Multiple, repetitive, low frequency deflections, with a cycle length ranging from 730 to 960 ms in Case 1 and 570 to 750 ms in Case 2, suggestive of sinus node electrograms, were recorded at a critical area at the junction between the superior vena cava and the right atrium. These low frequency deflections had no relation to spontaneous junctional beats or the spontaneous atrial beats that showed high frequency deflections on the atrial electrogram. However, they could be suppressed by spontaneous or paced atrial beats. Pharmacologic interventions in Case 2 showed that the cycle length of the low frequency deflections shortened after administration of isoproterenol and did not change after propranolol or atropine.

Thus, complete sinoatrial exit block with intact entrance conduction can occur in patients with bradycardia-tachycardia syndrome. Under such circumstances, the surface electrocardiographic manifestation of sinus bradycardia may not be of sinus origin.

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Both sinoatrial exit block and suppression of sinus node automaticity have been proposed as mechanisms of prolonged atrial pauses in patients with sick sinus syndrome (1). However, recent recordings of sinus node electrograms have suggested that sinoatrial exit block is the major mechanism responsible for spontaneous or pacing-induced prolonged atrial pauses (2–5). Herein, we describe two patients with bradycardia-tachycardia syndrome whose sinus node electrograms suggest that complete sinoatrial exit block is the mechanism of such pauses.

Methods

Electrophysiologic study. Electrophysiologic study was performed with patients in the postabsorptive, nonsedated state after informed written consent had been given. Two size 7 quadripolar electrocatheters with an interelectrode distance of 10 mm were introduced percutaneously through right and left femoral veins. The first electrocatheter was placed across the tricuspid valve for recordings of the His bundle electrogram at filter frequencies of 30 and 500 Hz; the second was positioned in the high right atrium in the region of the sinus node. The two distal electrodes of the latter electrocatheter were used to record the sinus node electrogram at filter frequencies of 0.1 to 50 Hz as previously described (6); the proximal two electrodes were used to record the high right atrial electrogram at filter frequencies of 30 to 500 Hz. A third bipolar electrocatheter, size 5, was inserted through the right antecubital vein and advanced to the right atrium for atrial stimulation. The surface and intracardiac electrograms were simultaneously displayed on a multichannel oscilloscope (VR-16, Electronics for Medicine) and recorded at paper speeds of 100, 150 and 250 mm/s. The stimuli were provided by a digital programmable stimulator (Bloom and Associates), and were approximately twice the diastolic threshold and 2 ms in duration.

Validation of sinus node electrogram. The sinus node electrogram was validated by the recording of multiple, repetitive, identical, low frequency deflections free of baseline drifting during atrial quiescence that was noted on both high right atrial electrogram and surface electrocardiograms. These low frequency deflections were recorded only at a critical area over the anterior aspect of the junction between the superior vena cava and the right atrium. Moving the electrocatheter a few millimeters away from this area resulted in disappearance of these deflections.

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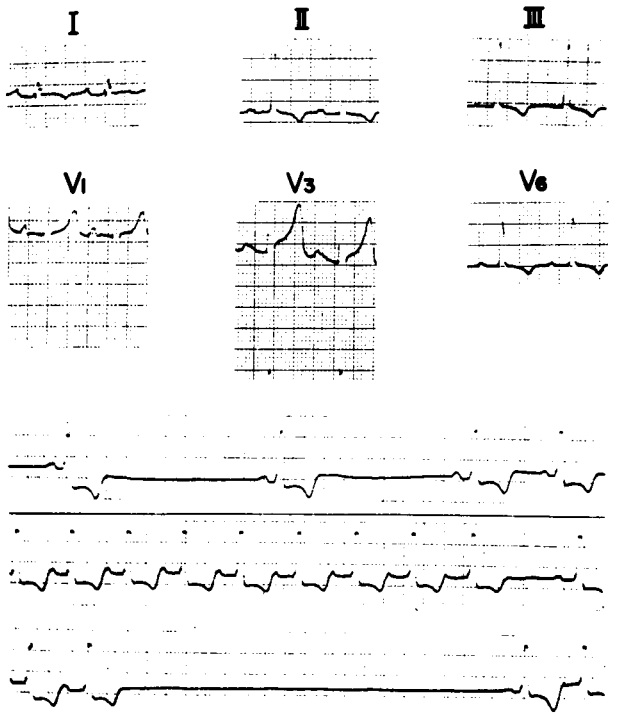


Figure 1. Case 1. Electrocardiogram with a lead II rhythm strip (**below**) showing a rate of 79/min, a PR interval of 0.24 s, left ventricular hypertrophy and bradycardia-tachycardia syndrome with prolonged atrial pauses.

Results

Case 1

A 48 year old man with chronic renal failure was referred because of recurrent episodes of near syncope for 3 days. Physical examination on admission revealed a blood pressure of 160/100 mm Hg. The heart rate was 100 beats/min, and an intermittent slow rate with long pauses was noted. The lungs were clear. A grade 2/6 systolic murmur was audible over the apex. The rest of the physical examination was normal. Blood urea nitrogen was 64 mg/100 ml and creatinine 5.8mg/100 ml. The chest X-ray film showed mild cardiomegaly. The echocardiogram disclosed dilation of the left atrium and left ventricle; the left ventricular ejection fraction was 50%.

The electrocardiogram revealed left ventricular hypertrophy with ST-T wave changes (Fig. 1). The rhythm strip showed a bradycardia-tachycardia syndrome. The cycle length during bradycardia varied from 1,920 to 2,200 ms, whereas that during tachycardia was 580 ms and regular. Prolonged atrial pauses of up to 10 seconds were noted on termination of atrial tachycardia.

Electrophysiologic study. The PP interval ranged from 900 to 1,580 ms; the PA interval was 40, the AH interval 75 and the HV interval 55 ms. The sequence of atrial activation was from high to low. Paroxysms of nonsustained atrial tachycardia with prolonged atrial pauses of 3,100 to 6,350 ms occurred almost continuously. Low frequency deflections were not observed before each P wave during episodes of bradycardia or tachycardia, but were evident during the episodes of long pauses after the termination of atrial tachycardia (Fig. 2 and 3). These low frequency deflections had a cycle length ranging from 730 to 960 ms.

Figure 2 is a continuous recording during an episode of a spontaneous long pause. At the beginning of Panel A, a low frequency deflection is noted on the sinus node elec-

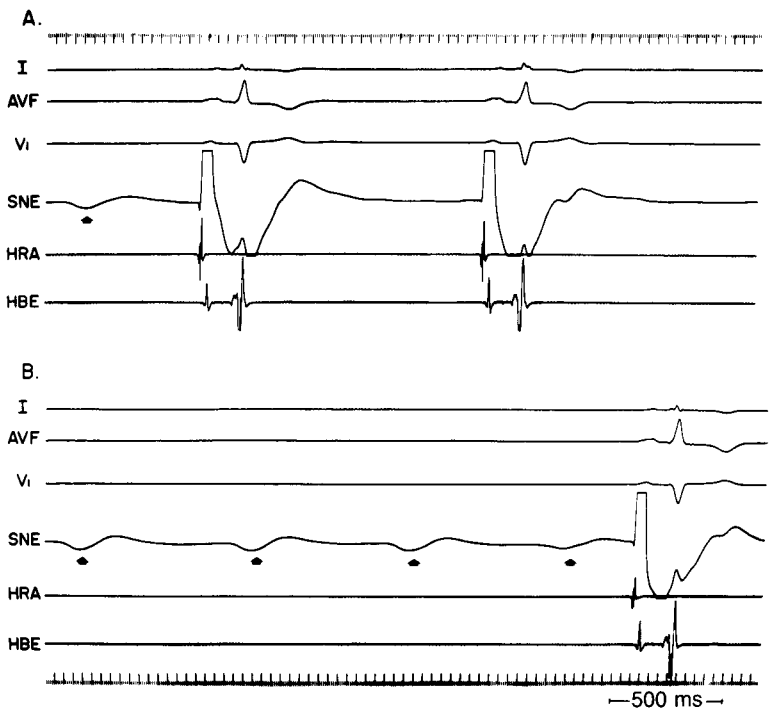


Figure 2. Case 1. Continuous recordings showing spontaneous complete sinoatrial exit block resulting in an abnormally prolonged atrial pause. Shown are electrocardiographic leads I, aVF and V_1, sinus node electrogram (SNE), high right atrial electrogram (HRA) and His bundle electrogram (HBE). **Arrowheads** indicate the sinus node electrograms (see text for discussion).

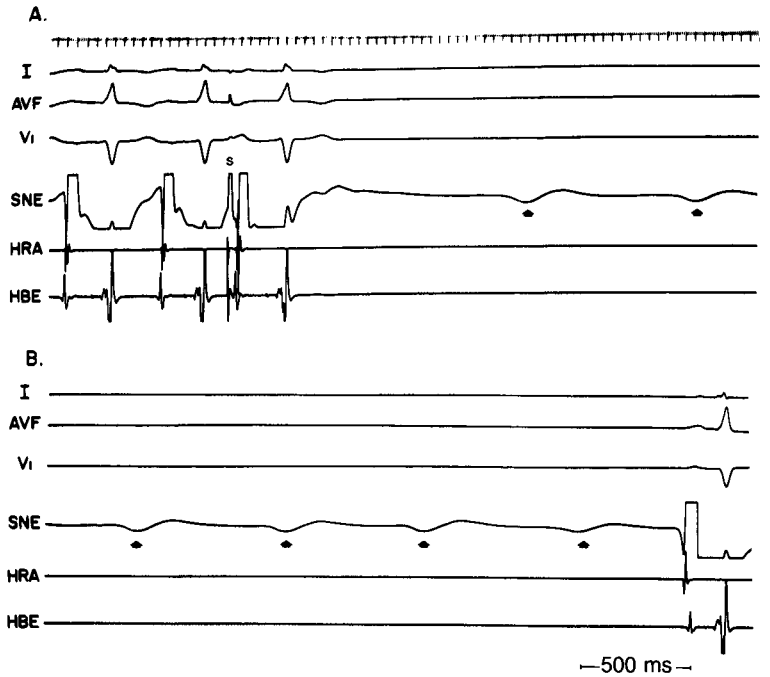


Figure 3. Case 1. Continuous recordings showing complete sinoatrial exit block (indicated by **arrowheads**) resulting in prolonged atrial pauses after termination of an episode of atrial tachycardia by a timed premature atrial beat (S) (see text for discussion). Other abbreviations as in Figure 1.

trogram and is followed by two atrial beats. These two beats bear no relation to the low frequency deflection but do suppress the low frequency deflections. In Panel B, four repetitive low frequency deflections are recorded on the sinus node electrogram during the atrial pause after the two atrial beats. The interval between the second atrial beat and the first low frequency deflection is 1,470 ms. Minor acceleration of the low frequency deflection is noted. This episode of atrial pause is terminated by another atrial beat.

Figure 3 is a continuous recording of an episode of a long pause after an episode of atrial tachycardia that was terminated by an atrial extrastimulus. Six repetitive low frequency deflections are observed. Again, suppression of the first posttachycardia low frequency deflection with subsequent minor acceleration is noted.

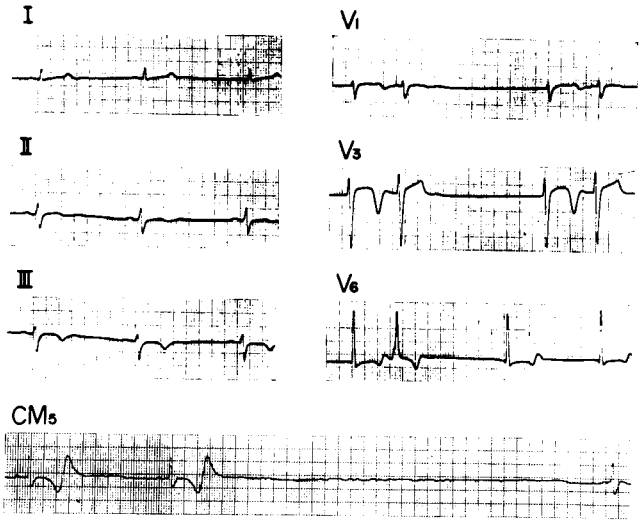
Case 2

A 32 year old man was admitted to the hospital because of recurrent episodes of near syncope during heavy exercise. Although he has had an irregular slow heart rate since childhood, he had no limitation of daily activities. His blood pressure was 120/80 mm Hg and pulse rate 40/min. The point of maximal cardiac impulse was located at the fifth intercostal space 3 cm lateral to the left midclavicular line. A grade 2/6 systolic murmur was audible over the apical area. The electrocardiogram showed apparent sinus bradycardia at a rate of 30 beats/min and junctional escape beats (Fig. 4). The M-mode echocardiogram showed a left ventricular dimension of 41 and 58 mm at end-systole and end-diastole, respectively, and a left ventricular ejection fraction of 56%.

A 24 hour Holter monitoring study showed persistent sinus bradycardia with sinus pauses lasting 7 to 8 seconds, junctional escape beats, atrial ectopic beats and paroxysms of atrial flutter and fibrillation (Fig. 4). Two exercise tests on separate days showed reproducible provocation of atrial flutter with 1:1 ventricular response at a rate of 260 beats/min, which was associated with severe dizziness.

Electrophysiologic study. Junctional rhythm with an HH interval ranging from 1,060 to 1,150 ms and an HV interval of 55 ms was noted (Fig. 5 and 6). No high frequency atrial deflections were recorded in the right atrium or coronary sinus. However, low frequency deflections with

Figure 4. Case 2. Electrocardiogram with a rhythm strip (CM5) recorded from the Holter monitor showing sinus bradycardia, sinus arrhythmia, junctional escape beats, left axis deviation, nonspecific ST-T changes and a long pause of 7 s.



a cycle length of 570 to 750 ms were observed when the electrocatheter was positioned at the anterior aspect of the junction between the superior vena cava and right atrium (Fig. 5, top panel). These deflections had no relation to the junctional beats. Minor suppression of the low frequency deflection was noted with incremental atrial pacing at paced cycle lengths between 600 and 400 ms (Fig. 6). The interval between the last paced atrial response to the first postpacing low frequency deflection ranged from 820 to 1,200 ms.

Effect of isoproterenol, propranolol and atropine. After the control study, an isoproterenol infusion was started at a rate of 0.5 μg/min and then increased to 1 μg/min. The junctional cycle length shortened to a range of 600 to 890 ms; frequent ectopic atrial beats were also noted (Fig. 5, second panel from top). The cycle length of the low frequency deflections decreased to a range of 500 to 550 ms; however, there was still no relation between the low frequency deflections and the junctional or ectopic atrial beats.

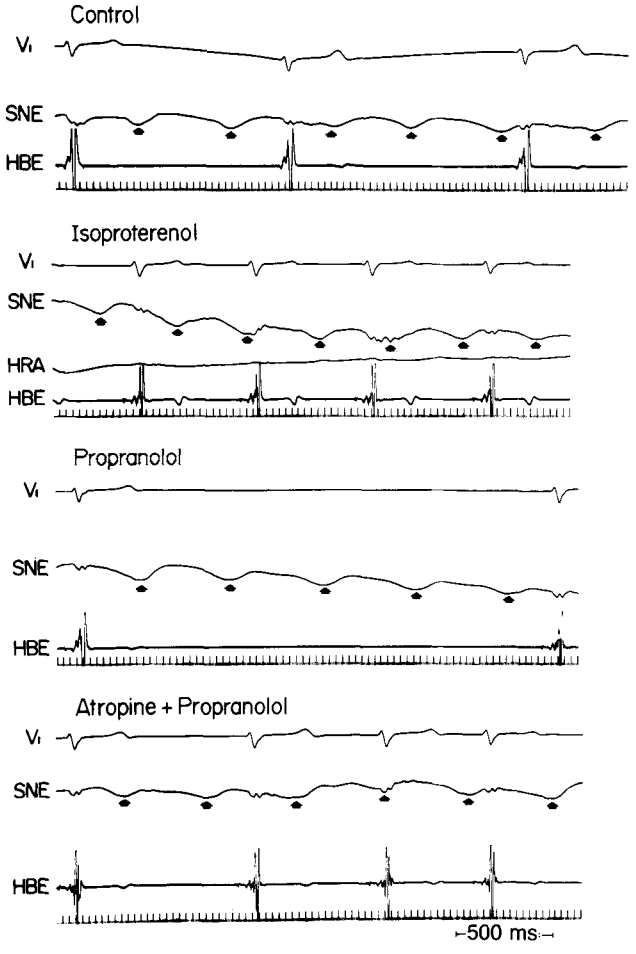


Figure 5. Case 2. Recordings showing complete sinoatrial exit block, junctional escape rhythm and the effects of isoproterenol, propranolol and atropine plus propranolol on sinus node activity. **Arrowheads** indicate the sinus node electrograms (see text for discussion). Abbreviations as in Figure 2.

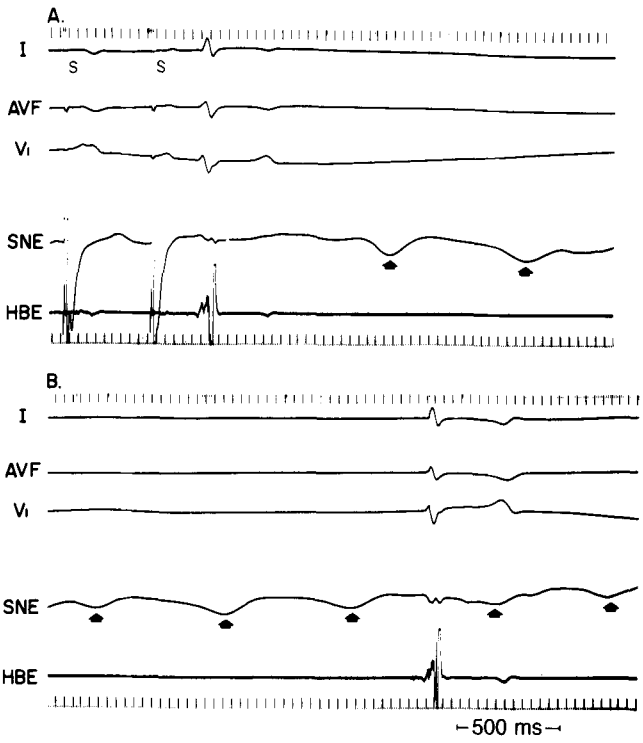


Figure 6. Case 2. Continuous recordings showing the effect of rapid atrial pacing on the sinus node electrogram (see text for discussion). Abbreviations as in Figure 2.

Minor suppression of the low frequency deflection was still noted after termination of atrial pacing. Fifteen minutes after discontinuation of isoproterenol infusion, propranolol, 10 mg, was administered intravenously over 10 minutes. The junctional cycle length increased to a range of 940 to 3,650 ms, and the cycle length of the low frequency deflection ranged from 650 to 700 ms (Fig. 5, third panel from top). Postpacing suppression of the low frequency deflection was again noted on termination of rapid atrial pacing. Atropine, 2.5 mg (0.04 mg/kg body weight), was then administered intravenously. The junctional cycle length shortened to a range of 790 to 1,450 ms, and the cycle length of the low frequency deflection ranged from 600 to 650 ms (Fig. 5, bottom panel). Postpacing suppression was still noted.

Discussion

Overdrive suppression and sinus pauses. Overdrive suppression of sinus node automaticity and sinoatrial exit block have been proposed as mechanisms of prolonged sinus pauses in patients with sick sinus syndrome. Physiologic suppression of sinus node activity after termination of overdrive pacing is a well known phenomenon, having been demonstrated in many in vitro and in vivo studies (7–9). The release of endogenous acetylcholine from cardiac tissue elicited by electrical stimulation has been considered to be the major factor responsible for the suppression. Previous

study suggested that rapid electrical stimulation could induce hyperpolarization of the sinus node cells, leading to suppression of automaticity. However, recent studies (9) showed that such stimulation caused hypopolarization rather than hyperpolarization and that ionic shifts resulting from overdrive pacing may be related to postpacing hypopolarization of sinus node cells. Whether overdrive suppression is a cause of abnormally long sinus pauses in patients with sick sinus syndrome has remained unclarified.

Sinoatrial exit block and sinus pauses. Recordings of sinus node electrograms have contributed significantly to the understanding of both normal and abnormal sinus node physiology. Several investigators (2–4) demonstrated that sinoatrial exit block rather than sinus node suppression was the major mechanism responsible for abnormally prolonged spontaneous or postpacing atrial pauses in patients with sick sinus syndrome. In their cases, however, Mobitz type II or type I exit block with repetitive sinoatrial exit block was the mechanism of prolonged atrial pauses. To the best of our knowledge, complete sinoatrial exit block has not been described previously in patients with sick sinus syndrome.

In our two patients with the bradycardia-tachycardia syndrome, validation of sinus node electrograms was supported by the finding that the low frequency, repetitive deflections were recorded only in a critical area at the anterior aspect of the junction between the superior vena cava and the right atrium. Moving the electrocatheter only a few millimeters away from this area resulted in disappearance of the low frequency deflections. In both patients, the low frequency deflections had no relation to junctional beats or the atrial beats that showed high frequency deflections on the atrial electrogram, and were suppressed by spontaneous or paced atrial beats. In Case 2, the low frequency deflections were facilitated by isoproterenol infusion, although they were not significantly affected by administration of propranolol or atropine. These findings further support the suggestion that these deflections reflected sinus node activities. Suppression of the low frequency deflection by spontaneous or paced atrial beats also suggests that complete sinoatrial exit block with intact entrance conduction occurred in these two patients. In Case 1, the bradycardia reflected an atrial escape rhythm. Suppression of the slow atrial escape rhythm by the tachycardia resulted in prolonged atrial pauses. In Case 2, the origin of the spontaneous slow atrial rhythm cannot be ascertained, because it was not observed during electrophysiologic studies. It could reflect either sinus rhythm with high grade sinoatrial exit block or a slow atrial escape rhythm. The episodes of near syncope that occurred during exercise in this patient reflected an exercise-triggered atrial flutter with 1:1 ventricular response.

Pathophysiologic consideration. The mechanism and sites of sinoatrial exit block in these two patients are not known. The observations that the sinus node electrograms are well preserved and the sinus node functions are rather intact suggest a site of block to be located in the perinodal tissue. Previous pathologic studies in patients with sick sinus syndrome (10,11) have consistently demonstrated degeneration and fibrosis of approaches to the sinus node and atria, suggesting that this syndrome is a disease process primarily involving the atria with invasion of the sinus node. The presence of paroxysmal atrial tachycardia and atrial flutter in our two patients is in accord with this hypothesis.

Conclusions and implications. This study demonstrates that unidirectional block with complete sinoatrial exit block and intact entrance conduction can be clinically manifested as a bradycardia-tachycardia syndrome. Under such circumstances, the surface electrocardiographic manifestation of sinus bradycardia may not be of sinus origin. Recordings of the sinus node electrogram are useful in unraveling the mechanism of the bradycardia-tachycardia syndrome.

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