Clinical Spectrum of the Sick Sinus Syndrome

By Joel J. Rubenstein, M.D., Charles L. Schulman, M.D., Peter M. Yurchak, M.D., and Roman W. DeSanctis, M.D.

SUMMARY
The clinical spectrum of the sick sinus syndrome (SSS) is described in a series of 56 patients who demonstrated either persistent unexplained sinus bradycardia (group I: eight patients); sinus arrest (group II: 15 patients); or bradycardia with episodic supraventricular tachyarrhythmias (group III: 33 patients). Coronary disease was the most common form of heart disease where etiology could be determined, but in 25 patients no clear etiologic diagnoses could be established. Thirty-three patients showed associated electrocardiographic conduction disturbances. Symptoms were common and were produced by both bradycardia and tachycardia. Eight patients in the bradycardia-tachycardia group experienced cerebral embolization. Despite bothersome symptoms, only six of the 56 patients died over an average follow-up of 7 years, and only one of these deaths appeared related to an arrhythmia. Drug therapy of bradycardia was generally ineffective, but digitalis was frequently helpful in patients with tachycardia. Electrical pacing was uniformly successful in treating symptoms of bradycardia but was disappointing in preventing tachyarrhythmias.

Additional Indexing Words:
Bradytachycardia syndrome
Sinus arrest
Sinoatrial block
Conduction disturbances
Pacemakers
Systemic embolism
Sinus bradycardia

DEVELOPMENTS in electrical pacing of the heart during the past decade have stimulated interest in mechanisms of bradycardia in patients experiencing Adams-Stokes syncope. For the most part these attacks are due to atrioventricular (A-V) block. However, an increasing number of patients have been described in whom the basis of syncope has been profound and unexplained sinus bradycardia or sinus arrest. In addition to bradycardia, certain of these patients have recurrent episodes of supraventricular tachyarrhythmias.

The terms "sick sinus syndrome,"1-3 "inadequate sinus mechanisms,"4 "sluggish sinus node syndrome,"5 and "sinoatrial syncope"6 have all been used to describe this phenomenon. When complicating supraventricular arrhythmias have been present, the condition has been termed "the syndrome of alternating bradycardia and tachycardia,"7 or simply the "bradycardia-tachycardia syndrome" (BTS).8 We consider the sick sinus syndrome (SSS) to be present in patients exhibiting otherwise unexplained marked sinus bradycardia or sinus arrest with or without associated supraventricular tachyarrhythmias. The mechanism of bradycardia may be either disordered impulse generation within the sinus node or impaired conduction of impulses from the sinus node into the atrium.

Most previous reports have focused on the necessity for and results of pacing in patients with this type of bradycardia.2, 4, 6, 9-15 The purpose of this communication is to describe in broader terms the clinical spectrum of the sick sinus syndrome as observed in 56 patients.
with this disorder encountered at the Massachusetts General Hospital. In addition to therapy, attention is called to etiology, symptomatology, and associated electrocardiographic abnormalities.

**Patients and Classification**

Patients were collected from the clinical material of the Massachusetts General Hospital and from the private practices of the authors. The charts of 83 patients meeting the criteria of SSS were reviewed. Of these, 56 were selected for inclusion, while 27 were excluded because of the probability that the slow heart rates were the result of medications (19 patients) or for lack of sufficient clinical information (8 patients).

The 56 patients were divided into three groups on the basis of electrocardiographic manifestations of SSS.

Group I included eight patients with persistent and otherwise unexplained extreme sinus bradycardia at a heart rate of less than 50 beats/min. Generally the heart rate was between 40 and 50, though one patient had persistent bradycardia of 30 beats/min.

Group II included 15 patients with at least one documented episode of sinus arrest or sinoatrial block, with A-V junctional or ventricular escape beats. Of the 15 patients, five had sinus bradycardia, nine were in regular sinus rhythm, and one exhibited permanent atrial arrest with A-V junctional rhythm.

Group III included 33 patients with BTS. All these patients exhibited either of the bradycardic mechanisms of groups I and II. In addition, each had at least one documented episode of a supraventricular tachycardia, either paroxysmal atrial tachycardia (nine patients), atrial flutter (14 patients), or atrial fibrillation (27 patients). Thirteen patients exhibited more than one type of tachycardia, and in 28 the attacks were multiple. Mechanisms of bradycardia included sinus bradycardia in 14, intermittent sinus arrest in 16, and permanent sinus arrest in three.

The series included 31 women and 25 men ranging in age from 26 to 90 years with a mean age of 65 years. Table 1 shows the sex, age, of onset of either symptoms or electrocardiographic diagnosis of the syndrome, and mean duration of follow-up for each of the three groups. Note the female predominance in group III, the wide age ranges, and the long follow-up. Figure 1 demonstrates a breakdown of the group by decile at age of onset of SSS. Although the numbers are small, there is a suggestion of a bimodal distribution of the patients.

**Etiology**

The diagnosis of coronary artery disease was made in 20 patients on the basis of either historical or electrocardiographic evidence of angina pectoris or myocardial infarction. Three patients exhibited evidence of idiopathic cardiomyopathy. Hypertensive heart disease was present in four. Single cases of luetic aortic insufficiency, rheumatic heart disease, scleroderma, and congenital heart disease were noted. In 25 patients no clear etiology could be determined.

**Symptoms**

Table 2 lists symptoms noted in the three groups. Forty-one of the 56 patients experienced symptoms related to the arrhythmias. Bradycardia alone was associated with symptoms in 19 patients. In group III tachycardia alone caused symptoms in 12 patients, and 10 patients experienced symptoms from both bradycardia and tachycardia.

Syncope occurred in 25 patients and was recurrent in 16. An additional 15 patients reported a spectrum of symptoms varying from lightheadedness to near-syncope.

There were 10 cerebrovascular accidents in the entire group, eight of them in the bradycardia-tachycardia group, and two in group II. In eight cases, the strokes were thought to be due to emboli. Peripheral emboli were surgically removed from two patients in group III, one of whom had suffered a previous stroke.

**Associated Electrocardiographic Abnormalities**

Abnormalities in addition to the rhythm were encountered in the electrocardiograms of 37 of the 56 patients. These are documented in table 3. Some disturbance of conduction, either at the level of the A-V node or in the peripheral bundle branches, was seen in 33 patients. First-degree A-V block (19 patients) and left-axis deviation (± 30°) (20 patients) were most common. Complete bundle-branch block was noted in 10 patients. Combinations of disturbances frequently occurred. For example, left-axis deviation accompanied right bundle-branch block in four of

![Table 1](https://example.com/table1.png)

**Characteristics of the 56 Patients**

<table>
<thead>
<tr>
<th>Data</th>
<th>Group I</th>
<th>Group II</th>
<th>Group III</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male (no.)</td>
<td>5</td>
<td>7</td>
<td>13</td>
</tr>
<tr>
<td>Female (no.)</td>
<td>3</td>
<td>8</td>
<td>20</td>
</tr>
<tr>
<td>Mean age (yr)</td>
<td>61</td>
<td>63</td>
<td>68</td>
</tr>
<tr>
<td>Range</td>
<td>28-92</td>
<td>26-92</td>
<td>44-90</td>
</tr>
<tr>
<td>Age at onset (yr)</td>
<td>56</td>
<td>56</td>
<td>57</td>
</tr>
<tr>
<td>Range</td>
<td>27-76</td>
<td>25-74</td>
<td>12-87</td>
</tr>
<tr>
<td>Follow-up (yr)</td>
<td>4</td>
<td>6</td>
<td>9</td>
</tr>
</tbody>
</table>

*Note: Data represent the number of patients.*

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SICK SINUS SYNDROME

AGE AT ONSET

Figure 1

Age of onset of the sick sinus syndrome. The decades are broken down according to the three subgroups of the syndrome. Note the suggestion of a bimodal distribution.

Table 2

Symptoms in the 56 Patients with the Sick Sinus Syndrome

<table>
<thead>
<tr>
<th>Group</th>
<th>Symptoms</th>
<th>I</th>
<th>II</th>
<th>Brady</th>
<th>Tachy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Syncope</td>
<td>2</td>
<td>5</td>
<td>15</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Lightheadedness</td>
<td>1</td>
<td>4</td>
<td>5</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Palpitations</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>17</td>
<td></td>
</tr>
<tr>
<td>Dyspnea + edema</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>Angina</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Fatigue</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Cerebrovascular accidents</td>
<td>0</td>
<td>2</td>
<td>8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Symptomatic</td>
<td>3/8</td>
<td>8/15</td>
<td>30/33</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 3

Associated ECG Abnormalities

<table>
<thead>
<tr>
<th>Conduction disturbance</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>First-degree atroventricular block</td>
<td>33</td>
</tr>
<tr>
<td>With left-axis deviation $\leq -30^\circ$</td>
<td>9</td>
</tr>
<tr>
<td>With right-axis deviation $\geq 120^\circ$</td>
<td>2</td>
</tr>
<tr>
<td>Intraventricular conduction defect</td>
<td>1</td>
</tr>
<tr>
<td>Alone</td>
<td>7</td>
</tr>
<tr>
<td>Bundle-branch block</td>
<td>19</td>
</tr>
<tr>
<td>Right bundle-branch block</td>
<td>5</td>
</tr>
<tr>
<td>With left-axis deviation</td>
<td></td>
</tr>
<tr>
<td>Left bundle-branch block</td>
<td>4</td>
</tr>
<tr>
<td>Intraventricular conduction defect</td>
<td>3</td>
</tr>
<tr>
<td>Left-axis deviation $\leq -30^\circ$</td>
<td>20</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>7</td>
</tr>
<tr>
<td>Left ventricular hypertrophy</td>
<td>8</td>
</tr>
<tr>
<td>Nodal escape interval $&gt;2$ sec</td>
<td>7</td>
</tr>
</tbody>
</table>

Pharmacologic treatment of the SSS was usually unsuccessful. Belladonna alkaloids were ineffectual in speeding the sinus rate on 14 occasions, and in four cases patients were worse because of side effects in two and the induction of tachyarrhythmias in two. There were only two cases of bradycardia in whom the chronic administration of atropine appeared to help. Similarly, sympathomimetic...
amines were ineffective in 11 cases, and five patients were worse because of side effects or the induction of tachyarrhythmias. However, one patient from group III was improved from the chronic administration of ephedrine, and another in group III benefited from chronic isoproterenol administration.

Drug therapy directed at episodes of tachyarrhythmia in group III patients was similarly frustrating and unrewarding. Attempts to increase the basic heart rate and thus to suppress the arrhythmias with belladonna alkaloids on seven occasions, or sympathetic mimetic amines on seven occasions, were uniformly without benefit. Antiarrhythmic agents alone, including quinidine, procainamide, and propranolol, were of little help in 11 cases. Only four of 22 patients who received these agents were improved. Seven patients were worse, five because of increase in symptoms related to bradycardia, to sinus arrest, or to further depression of the A-V junctional escape focus. Digitalis glycosides were employed in 17 patients for symptomatic tachycardia and benefited 10 by either decreasing the frequency of tachyarrhythmia or controlling the ventricular response. However, despite improvement with digitalis therapy, additional antiarrhythmic drugs were deemed necessary in six of the 10 cases. In all, digitalis glycosides were used in 34 of the 56 patients for treatment of congestive heart failure or tachyarrhythmia. These drugs were frequently employed following development of SSS, but occasionally had been administered several years preceding the diagnosis. Digitalis was withdrawn from 14 patients in an effort to abolish the bradycardia, but without effect in any.

Cardioversion was used uneventfully on five occasions to convert arrhythmias. It was generally avoided because of the fear of an inadequate sinus rate following cardioversion.

Pacing was employed in 23 of 56 patients—permanently in 18 and temporarily in five. Long-term pacing was undertaken in 18 for symptomatic bradycardia. In 16 this was manifested as episodes of transient cerebral hypoxia secondary to long sinus pauses. Two were paced primarily to increase cardiac output, which was considered depressed by bradycardia. The original site of long-term pacing was the right ventricle in 14 cases and the right atrium in four. However, it was necessary to change the electrode site on two occasions. In one case erratic atrial pacing necessitated changing to ventricular pacing. In the other case serious hypotension related to retrograde V-A conduction during demand ventricular pacing was eliminated with demand atrial pacing. All 18 patients were improved.

An additional two patients with bradycardia were paced temporarily in an effort to improve cardiac output. In neither case was the clinical course altered, and the temporary electrode was withdrawn.

Among those 18 patients permanently paced primarily for symptoms related to bradycardia, 13 also had episodes of tachyarrhythmias. In only two patients did the tachyarrhythmias appear to be suppressed by the addition of the pacemaker alone. One of the two patients has recently reverted to permanent atrial fibrillation following 24 months of freedom from arrhythmia with ventricular pacing. In three others the pacemaker allowed more aggressive administration of antiarrhythmic drugs. This combination eliminated or reduced the frequency of tachyarrhythmias. Three additional patients were paced temporarily in an effort to suppress episodic tachyarrhythmia. When it was clear that the clinical course was altered, the temporary electrode was withdrawn.

There were six deaths in the series during an average follow-up of 7 years. However, in only one case was the arrhythmia closely related to the terminal episode.

Postmortem data were available in only four cases and revealed a spectrum of coronary artery disease. This ranged from sclerotic but widely patent vessels to marked atherosclerosis with severe focal obstruction of all major coronary vessels in a patient with scleroderma. The myocardial pathology reflected the severity of coronary artery disease, ranging from
mild cytoplasmic vacuolization of myocardial cells and small patches of myocardial sclerosis to severe, dense, and widespread myocardial fibrosis with particular involvement of atrial myocardium. Detailed examination of the specialized conduction system was not carried out.

**Discussion**

Altered function of the dominant pacemaker of the heart, the S-A node, has long been recognized. Levine first described the electrocardiographic features of S-A exit block in 1916. Most subsequent publications have dealt with electrocardiographic aspects of S-A block and atrial standstill. The first report of symptomatic sinus bradycardia with Adams-Stokes attacks was that of Laslett in 1909. Other isolated case reports followed. A review of the subject of sinus bradycardia appearing in 1952 emphasized exclusively the etiologic features. The relationship between symptomatic bradycardia and paroxysmal supraventricular arrhythmias was first described by Short in 1954.

The term sick sinus syndrome was coined by Lown to describe rhythm disturbances following DC cardioversion of chronic atrial fibrillation. These phenomena included “chaotic atrial activity, changing P wave contour, bradycardia, interspersed with multiple and recurrent ectopic beats, with runs of atrial and nodal tachycardia.” Ferrer broadened the definition to include (1) persistent, severe, and unexpected sinus bradycardia; (2) sinus arrest, brief or sustained, with escape atrial or A-V junctional rhythm; (3) prolonged sinus arrest with failure of subsidiary pacemaker resulting in total cardiac asystole; (4) chronic atrial fibrillation with slow ventricular response not due to drug therapy (she gave no explanation for inclusion of this “non-sinus” rhythm in her definition.); and (5) inability of the heart to resume sinus rhythm following electroconversion for atrial fibrillation. Other descriptive terms have been applied to this phenomenon, but sick sinus syndrome seems the simplest and most desirable.

The clinical spectrum of SSS seen in our material is wide, ranging from sinus bradycardia without symptoms to recurrent attacks of syncope or congestive failure. The question arises whether asymptomatic sinus bradycardia (group I) can really be considered abnormal, since sustained sinus bradycardia is found in association with many extracardiac factors. It is regarded as physiologic in well-trained athletes. Although only three of the six patients in group I had symptoms related to bradycardia, none was free of some evidence of heart disease. Thus, we believe them to comprise a portion of the spectrum of SSS, as did Ferrer. Long-term follow-up of patients in group I is not available, but it is very likely that some may ultimately develop the rhythm disturbances seen in groups II and III. In fact a number of patients in our groups II and III had sustained sinus bradycardia for years preceding the documentation of sinus arrest or tachyarrhythmia. There is convincing evidence for derangements of S-A nodal function in groups II and III. One patient in group II and three in group III had permanent atrial arrest. Half of group III had intermittent sinus arrest as the mechanism of bradycardia.

Most recent descriptions of SSS emphasize its frequent association with disturbances of consciousness and the need for long-term pacing. It is not generally recognized that SSS may exhibit a wide range of phenomena—from no symptoms at all, to fatigue and episodic lightheadedness, to frank syncope and/or congestive heart failure. One fourth of our patients experienced no symptoms, and those who were symptomatic had disturbances of consciousness of variable severity (40 of 56). A small number exhibited congestive heart failure as an important complication of their disease. Bouvain analyzed a series of 63 cases of marked bradycardia, mostly related to recurrent S-A block. Of this number, 37 had chronic S-A block, unrelated to drug effects. Most patients were elderly, and about one fourth had detectable underlying coronary artery disease. Most were asymptomatic, syncope occurring in 11 patients but posing a problem in only five. Rasmussen reported 21 cases of chronic S-A
block, 15 of whom had Adams-Stokes attacks. Fourteen patients also had paroxysmal atrial arrhythmias. Seventeen of the group eventually required permanent pacemaker implantation. Easley et al.\textsuperscript{6} reported 13 patients with what they termed sinoatrial syncope. Eight of the group had other symptoms as well, chiefly angina pectoris and congestive failure. They classified their series into two groups, those with only bradyarrhythmias, and those with alternating brady- and tachyarrhythmias. They emphasized, as have others, the difficult therapeutic problem presented by the latter group.

An unexpected and important feature of the BTS noted by us is the occurrence of systemic embolization in eight of 33 patients. Presumably stasis in the atria associated with the tachyarrhythmias promoted mural thrombus formation. On the basis of our experience, anticoagulant therapy should be considered in these patients if their general condition warrants it and if there is no contraindication to such therapy.

Remarkably absent from all patients reported in our series and in those of others are serious ventricular arrhythmias. Although slow heart rates are known to promote appearance of ventricular ectopic activity, this has not been a feature of SSS.

The exact etiology of SSS remains obscure. Information from pathologic examination is meager. The basic process is presumably degenerative change in the S-A node, either primary or secondary to some vascular disease. Myocardial biopsy in two family members with persistent atrial standstill showed amyloid deposits,\textsuperscript{31} but no parallel cases have appeared. Rasmussen\textsuperscript{15} believed the disease to be a sequel of diphtheria in his patients from Scandinavia, but could offer only one example to support this thesis. Two patients reported by Bouvrain et al.\textsuperscript{2} first developed SSS during an attack of diphtheria. Winternitz and Selye\textsuperscript{36} reported a patient with sinus bradycardia of 43–47 beats/min due to thrombosis of the artery to the S-A node. James\textsuperscript{37} has pointed out the vulnerability of the S-A node to vascular damage by virtue of receiving its entire blood supply from a single artery. The S-A node artery also supplies the atria, and disease of this vessel might be expected to affect stability of the atrial rhythm, a possible explanation for the BTS.

The most common etiology in our material was coronary disease, seen in 20 patients. Yet no specific diagnosis could be established in a still larger number (25 patients). It is probable that older subjects in this undiagnosed group will prove to have coronary disease, while some younger subjects will prove to have cardiomyopathy. It is worth pointing out that bradycardic rhythm disturbances have only rarely been mentioned in the large reported series of patients with cardiomyopathy.\textsuperscript{38–40} Some patients in either age group may have a primary degenerative process involving the S-A node artery, or the specialized conduction tissue. Detailed examination of the S-A node in one of our four patients coming to autopsy showed degenerative changes, but no evidence of arteriopathy involving its artery.

Several authors have pointed out that not infrequently more than the S-A node is diseased in SSS.\textsuperscript{2, 41, 42} Electrocardiographic evidence for widespread abnormalities of impulse formation and conduction among our patients with the SSS is impressive (table 3). Nineteen subjects had some degree of A-V block, and 10 had intraventricular conduction disturbances. Abnormal left-axis deviation in 20 subjects can be offered as evidence of impaired conduction in the anterior division of the main left bundle branch. Evidence of generalized dysfunction of the conduction system has been demonstrated with His bundle recordings and with atrial pacing.\textsuperscript{41, 42} These abnormalities include impaired impulse formation by lower automatic centers. Ordinarily pacemaker cells in the A-V junction provide escape beats at rates of 40–50 beats/min when the S-A node defaults. Seven of our patients failed to generate A-V junctional escape beats at the usual rate, permitting sinus rates as low as 30 beats/min.

\textit{Circulation, Volume XLVI, July 1972}
With regard to therapy, none is indicated for bradycardia without symptoms. Bradycardia leading to disturbances of consciousness or frank syncope is best treated with pacemaker implantation. Drug therapy alone appears to have little value, a conclusion voiced also by others.9, 14, 15 Nine of our series (16%) had syncope only once, and a solitary attack of syncope did not seem to merit pacemaker implantation. Syncope occurred repeatedly in 16 patients, all of whom required permanent pacemaker implantation. An average of 3½ years elapsed between onset of symptoms and the need for pacemaker implantation. Only one of the six deaths in our series appeared related in any way to the arrhythmia. The variability in symptomatology and prognosis emphasizes the need to individualize therapy.

Bradycardia associated with evidence of congestive heart failure can be managed by digitalis, with vigilance for any unexpected effects of this agent on the diseased S-A node. Should this occur, pacemaker implantation may be necessary as an adjunct to drug therapy of heart failure.

Treatment of BTS merits special comment. Previous authors have emphasized the difficult therapeutic problem posed by patients with this entity.2-6, 10-13, 43, 44 Therapy for tachyarrhythmia with digitalis and/or conventional antiarrhythmic agents may be associated with marked bradycardia and syncope following reversion to normal rhythm. This is presumably due to depression of endogenous pacemakers by the drugs. Atropine, given for bradycardia, may promote the appearance of tachyarrhythmia. It was not until the advent of pacemakers that a satisfactory solution of the bradycardic aspect of the BTS was at hand. Since the first report10 of a patient successfully treated with a permanent pacemaker a number of reports have reiterated its value.6, 3, 15

In general, pacing has failed to prevent bouts of tachyarrhythmia. However, implantation of a pacemaker may be of value by permitting the addition of digitalis and/or antiarrhythmic preparations to the therapeutic program without fear of aggravating the effects of bradycardia. Five of our patients with BTS treated by combined pacing and drugs have had a reduction in the frequency of tachyarrhythmia. In the truly refractory case of recurrent supraventricular tachycardia, consideration may be given to the very aggressive procedure of sectioning the His bundle in combination with ventricular pacing.2

With regard to the site of pacing, there is theoretic advantage to atrial pacing because the contribution of atrial systole to cardiac output is preserved. In one of our cases, atrial pacing was successful when ventricular pacing failed to maintain cardiac output. However, the lesser reliability of atrial pacing and the high incidence of A-V conduction disturbances in these patients has led us to select a ventricular site for pacing in the majority of our cases.

Finally, the recently developed "bifocal" demand pacemaker may prove useful in these patients.13 This pacemaker involves the use of two electrodes, one positioned in the right atrium and one in the right ventricle, with both atrial and ventricular pacing circuits. The ventricular electrode senses the ventricular electrocardiogram and sequentially paces the atrium and ventricle on demand. In the presence of sinus bradycardia and intact A-V conduction, the atrium is paced and the ventricular circuit is blocked by the conducted beat.

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Circulation, Volume XLVI, July 1972

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