Electrophysiological and Clinical Study of Sick Sinus Syndrome Using the Overdrive Suppression Test

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1) Right atrial pacing was performed at rates of 80 to 200 beats/min for 30 seconds. The pacing rate was increased by an increment of 10 beats/min, with a 30 second rest period after each atrial pacing. One hundred three patients with sick sinus syndrome (SSS) and 25 normal subjects were studied. Recovery time (RT) was defined as the longest R-R interval of the five subsequent beats after cessation of overdrive stimulation at each rate, and was expressed as the ratio of RT to resting R-R interval (%RT). According to the origin of the beat following the longest pause, RT was divided into sinus node recovery time (SNRT) and A-V junctional recovery time (JRT). The longest RT in all pacing rates was defined as the maximum recovery time (max RT).

2) In normal subjects, the ratio of SNRT to the resting R-R interval (%SNRT) was only slightly affected by the pacing rate, though showed a two-phase phenomenon. Among cases with SSS, in patients with a maximum %SNRT of more than 200%, %SNRT progressively increased as the pacing rate increased. In patients with a maximum %SNRT less than 200%, however, %SNRT remained essentially constant over a wide range of pacing rates. JRT in cases with SSS showed a two-phase pattern with a maximum pause at a pacing rate of 260–280% with reference to the resting heart rate.

3) Patients with SSS were classified into three different response groups according to the above values and patterns of %RT against the pacing rate, with each group showing different clinical and electrocardiographic features.

In the 43-case low-rate response group (%RT already more than 200% below a pacing rate of 200%), sinus arrest was detected in 65% of the cases and paroxysmal tachycardia and syncopal attack in about half. In the 31-case high-rate response group (%RT smaller than 200% at a pacing rate lower than 200% but %RT over 200% at a pacing rate more than 200%), SA block was found in 74% of the cases, paroxysmal tachycardia in 81% and syncopal attack in 81%. Maximum suppression of sinus node automaticity occurred at

Key Words:

Sick sinus syndrome
Overdrive suppression
Percentage recovery time
Pacing rate (%)
Low-rate response group
High-rate response group
Normal response group

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a higher pacing rate of 300–400% in some cases. In the 29-case normal response group (%RT smaller than 200% at all pacing rates and little affected by the pacing rate), sinus bradycardia was found in 72% of the cases, but syncopal attack in 24% of the cases despite occurrence of paroxysmal tachycardia in about half of the cases.

The importance of pacing rate effect on RT should be stressed in the overdrive suppression test. Hence, our classification was thought to be useful in unmasking the presence of sinus node dysfunction and in confirming its severity in patients with SSS.

SICK sinus syndrome^{1,2} (SSS), including sinus bradycardia, sinus arrest or sinoatrial (SA) block and bradycardia-tachycardia syndrome is one of the causes of Adams-Stokes attack and sudden death. SSS recently has elicited a great interest in view of the availability of effective pacemaker therapy.³ SSS is diagnosed by the electrocardiogram (ECG), but it is rather difficult to precisely evaluate the sinus node dysfunction only by ECG findings. The atrial overdrive suppression test, first applied to the evaluation of sinus node dysfunction by Mandel,⁴ has been widely used as an important examination to diagnose SSS and to decide the treatment for patients with SSS. In SSS, recovery time (RT) is prolonged by overdrive stimulation more than one in normal subjects. However occurrence rates of abnormally prolonged RT in SSS widely differ from author to author ranging from 35 to 100%^{4–8}. Hence its usefulness is still being discussed. The reason why its occurrence rates differ so widely was mainly explained by assuming that SSS includes many different entities.

Therefore we decided to classify patients with SSS in three groups – low-rate, high-rate and normal response group – using RT response against pacing rate of overdrive stimulation. Clinical and electrocardiographic findings were studied in each of these groups.

PATIENTS AND METHODS

One hundred three patients – 46 males and 57 females – diagnosed as having SSS by ECG criteria were studied. They ranged in age from 10 to 70 years old, and their average age was 51. Sinus bradycardia cases, who showed a sinus rate of 50 beats/min or less, numbered 14, sinus arrest 11, SA block 14 and bradycardia-tachycardia syndrome 64.

Twenty five patients without SSS were also studied as control subjects; this group included 15 males and 10 females, ranging in age from 16 to 73, whose average age was 46.

Two tripolar catheters (No. 4, 5 or 6 Fr) were passed through the right femoral vein, with one placed at the level of bundle of His in the right ventricle and the other high in the right atrium. The His bundle electrocardiogram (HBE) and surface ECG (lead I, II and V₁), were recorded at the speed of 50 or 100 mm/sec. Fig. 1 shows overdrive suppression test method. Atrial pacing was delivered for 30 seconds then abruptly stopped. The initial pacing rate was at least 10 beats/min above the control heart rate. The rate was then increased by an increment of 10 beat/min up to 200 beats/min. After each atrial pacing a pause, i.e., a 30-second resting period, was given.

Recovery time was defined as the longest R-R interval among the five subsequent intervals after cessation of atrial stimulation at each pacing rate. The longest RT in all pacing rates was called the maximum recovery time (max RT). P and QRS wave configurations found on the surface ECG were used to ascertain origin of the escape beat following the longest pause. According to origin, RT was divided into sinus node recovery time (SNRT) and A-V junctional recovery time (JRT). The ratio of RT to the resting R-R interval was defined as percentage recovery time (%RT):

$$\%RT = \frac{RT \text{ (msec)}}{\text{resting R-R interval (msec)}} \times 100 (\%).$$

Corrected recovery time (CRT) is defined as RT in excess of average cycle length:

$$CRT = RT - \text{resting R-R interval (msec)}.$$

In each case, the pacing rate was decided with reference to the patient's resting heart rate:

$$\text{pacing rate } (\%) = \frac{\text{resting heart rate (beats/min)}}{\text{atrial pacing rate (beats/min)}} \times 100 (\%).$$

Before testing, cardiac drugs were discontinued for at least 48 hours or more than twice their half-decay period.

Fig.1. The method of overdrive suppression test.

The control R-R interval is indicated by 'a', and maximum recovery time by 'b'. Atrial pacing for 30 sec was performed at rate of 100 to 200 beats/min with a 30-sec rest period before the next pacing. The pacing rate was increased 10 beats/min.

Fig.2. The effect of the atrial pacing rate on the SNRT in control subjects.

Ordinate; sinus node recovery time in percentage change (%SNRT).

Abscissa; the pacing rate in percentage change $\left[\frac{\text{pacing rate}}{\text{resting heart rate}} \times 100\% \right]$.

Values are expressed as mean $\pm$ SEM. %SNRTs at pacing rates of 100–120% and 200% or greater were significantly (*) lower than that at a pacing rate of 140–160%.

Fig.3. (A) The effect of the atrial pacing rate on SNRT in patients with SSS. Values are expressed as mean $\pm$ SEM. The average %SNRT at pacing rates over 140% were significantly higher (*) than that at a pacing rate of 100–120%. Note that %SNRT are widely distributed at each pacing rate.

(b) The effect of the pacing rate on SNRT in two SSS groups.

In the first group, whose max %SNRT was less than 200%, %SNRT was not significantly influenced by different pacing rate ($\bullet$ - $\bullet$). In the second whose max %SNRT was more than 200%, mean values of %SNRT increased linearly as pacing rate increased ($\circ$ - $\circ$).

RESULTS

A. Pacing rate effect on RT.

a) Pacing rate effect on SNRT in control subjects

In 25 cases, maximum sinus recovery time (max SNRT) was 1181 ± 165 msec, maximum corrected sinus node recovery time (max CSNRT) 360 ± 106 msec and maximum percentage sinus node recovery time (max %SNRT) $144 \pm 12\%$. In Fig. 2 illustrating %SNRT at each pacing rate, %SNRT reached a maximum at a pacing rate of 140–160% increase in the resting heart rate and decreased slightly at higher pacing rates.

b) Pacing rate effect on SNRT in patients with SSS

Fifty cases were studied whose basic rhythm was sinus rhythm or sinus bradycardia where SNRT could be accurately measured at all

pacing rates. Thirty nine cases were excluded, since their basic rhythm was not consistent with the origin of beats following the maximum pause. In these cases, max SNRT was 3506 ± 2018 msec, max CSNRT 2380 ± 2002 msec and max %SNRT $318 \pm 187\%$. As shown in Fig. 3(A), the average %SNRT at pacing rates over 160–180% were significantly ($p < 0.05$) higher than that at a pacing rate of 100–120%. However, the %SNRT values were widely distributed at pacing rates of more than 200%. These cases were divided into two groups; in the first, max %SNRT was less than 200% and in the second more than 200% [Fig. 3(B)]. In the first group, %SNRT was not significantly influenced by the pacing rate, and on the other hand, in the second group, the average %SNRT was normal at a pacing rate of lower than 200% but progressively increased as the pacing rate was increased.

Fig. 4. The effect of the pacing rate on JRT in patients with SSS.

Ordinate; A-V junctional recovery time in percentage change (%JRT). Abscissa; the pacing rate in percentage. Values are expressed as mean $\pm$ SEM. %JRTs at pacing rates of 200% or less and 380% or greater were significantly (*) lower than that at a pacing rate of 260–280%.

c) Pacing rate effect on JRT in patients with SSS

In this study 14 patients were examined. Their basic rhythm was junctional escape rhythm where escape beats after the maximum pause at all pacing rates came from A-V junctional pacemaker. In these cases, maximum A-V junctional recovery time (max JRT) was 6173 ± 2679 msec, maximum corrected A-V junctional recovery time (max CJRT) 4672 ± 2513 msec and maximum percentage A-V junctional recovery time (max %RT) $398 \pm 109\%$. In Fig. 4, %JRT increased as the drive rate was increased, it reached a maximum at a pacing rate between 260–280% and then slightly decreased.

B. SSS classification by overdrive suppression test

The SSS cases were classified, according to

Fig. 5. Classification of SSS by overdrive suppression test.

Cases with SSS are classified into three groups according to the %RT response against the pacing rate: low-rate (upper left), high-rate (upper right) and normal response groups (lower left).

%RT response against the pacing rate, into low-rate, high-rate and normal response group. Fig. 5 shows schematic illustrations of %RT response in these groups. In control subjects, the pacing rate only slightly affected %RT. In the 43-case low-rate response group, most beats after the maximum pause originated in the A-V junctional tissue and some from the sinus node or other ectopic origins. %RT was already more than 200% below a pacing rate of 200%. Max RT was 6081 ± 1806 msec, max CRT 4681 ± 1757 msec and max %RT $474 \pm 189\%$. In the 31-case high-rate response group, most of the beats after maximum pauses came from the sinus node. %RT was smaller than 200% at a pacing rate of less than 200%, but %RT was over 200% at a pacing rate of more than 200%. Max RT was 4227 ± 1867 msec, max CRT 3408 ± 1793 msec and max %RT $418 \pm 165\%$. In the 29-case normal response group, most of the beats after maximum pauses came from the sinus node. Max RT was 1776 ± 505 msec, max CRT 653 ± 308 msec and max %RT $156 \pm 26\%$. %RT was little affected by the pacing rate. The response pat-

Fig. 6. A typical example of %RT as a function of atrial pacing rates in the low-rate response group.

This case is a 19 years old male, with primary myocardial disease. The ECG showed sinus arrest with escape junctional rhythm, %RT showed a two-phase pattern with maximum pause at a pacing rate of 260%, its origin is thought to be the A-V junctional pacemaker. Max RT was 5400 msec, max CRT 3800 msec and %RT 340%. He had episodes of syncope, paroxysmal atrial fibrillation and paroxysmal ventricular tachycardia. A pacemaker was implanted.

tern was similar to that of the normal subjects group. Figs. 6, 7 and 8 are typical examples of low-rate, high-rate and normal response group. As shown in Fig. 9, a fairly good reproducibility of %RT response against pacing rate was found at different times in one of the cases in the high-rate response group.

a) ECG findings

Fig. 10 shows the ECG findings in each group. In the low-rate response group, sinus arrest was found in 28 cases (65%) and SA block in 13 cases (30%), of which 11 cases had sinus bradycardia in their basic rhythm. In the high-rate response group, SA block was found in 23 cases (74%), 18 cases of them showed usually normal sinus rhythm. In the normal response group, 21 cases (72%) showed sinus bradycardia and 6 cases (21%) SA block.

Sixty four cases (62%) of SSS had episodes of paroxysmal tachycardia. Tachycardia arrhythmias in the bradycardia-tachycardia syndrome were atrial fibrillation or flutter (76%) and paroxysmal supraventricular tachycardia (23%).

Only one case had ventricular tachycardia.

b) HBE findings

In Fig. 11, intra-atrial conduction time (S-A interval) and H-V interval had no significant difference in each group. A-H intervals of the low-rate response group was significantly prolonged compared with those of other groups. A pacing rate producing Wenckebach type second degree A-H block was significantly low in the low-rate response group.

c) Syncopal attacks

Syncopal attacks were observed in 53 cases (51%) of 103 cases. As shown Fig. 12, syncopal attack occurrence was 49% in the low-rate response group, 81% in the high-rate response group and 24% in the normal response group. Syncopal attack was found in 41 cases (64%) of 64 patients with paroxysmal tachycardia and in 12 cases (31%) of 39 patients without paroxysmal tachycardia. Syncopal episodes incidence in cases with paroxysmal tachycardia of the high-rate response group was 92%, 52% in the low-rate response group 52% and 36% in the normal

Fig.7. A typical example in the high-rate response group.

This case is a 55 years old female, with hypertension. The ECG showed usually normal sinus rhythm, but transient SA block and sinus arrest (5040 msec) after cessation of paroxysmal atrial fibrillation. As shown in the right fourth and fifth tracing from the top, SNRT was 1280 msec at a pacing rate of 120 beats/min but longer than 5450 msec at a pacing rate of 200 beats/min. Note that %RT was of normal value at pacing rates lower than 200%, but %RT was markedly prolonged at pacing rates more than 200%. She had episodes of syncopal attack and a pacemaker was implanted.

Fig.8. A typical example in the normal response group. This case is a 63 years old male, with hypertension. The ECG showed sinus bradycardia (41 beats/min) and %RT remained essentially constant over a wide range of pacing rates. Max RT was 1480 msec, max CRT 480 msec and max %RT 148%. He suffered from paroxysmal atrial fibrillation but had no syncopal attack.

Fig.9. Reproducibility of the effect of the pacing rate on RT in the high-rate response group. This case is a 67 years old female, with hypertension. The ECG showed sinus rhythm of 63 beats/min and sinus arrest (4400 msec) after cessation of atrial flutter-fibrillation. At 3 different times, %RT was found increased over 200% at a pacing rate of more than 300%. She had episodes of syncopal attack and a pacemaker was implanted.

response group.

d) *Relationship between max RT and syncopal attack*

As shown Fig. 13, the average of max RT was significantly prolonged in the low-rate response group, but did not differ between cases with and without syncopal attack. In the high-rate response group, the average max RT prolongation was significantly lower than that in the low-rate response group and syncopal episodes were frequently found despite lower max RT. In 83% of the normal response group, max RT was smaller than 2000 msec, but 7 cases had syncope episodes. In 3 of them syncopal attack was not considered to be caused by sinus arrest.

e) *Clinical features of patients in each group*

Fig.14 shows the average age of patients which was higher in the high-rate response group. In regard to underlying disease, although, in many

ECG findings Groups	Sinus arrest	SA block		Sinus bradycardia	Sinus * rhythm
		Sinus bradycardia	Sinus rhythm		
Low-rate response group	●●●●●●●● ●●●●●●●● ○●○●○●○● ○●○●○●○●	●●●●●●●● ○●○●○●○●	●○	●○	
High-rate response group		●●●●●○	●●●●●●●● ●●●●●●●● ●○●○●○●○	●●●●●●●●	●●
Normal response group	● **	●●○●	●●	●●●●●●●● ●●●●●●●● ○●○●○●○● ○●○●○●○●	●

Fig.10. ECG findings in three groups.

The open circles show cases without paroxysmal tachycardia, and filled circles show cases with paroxysmal tachycardia. In the sinus rhythm column (*), cases which had only sinus arrest after cessation of paroxysmal tachycardia. Note that sinus arrest was mostly found in the low-rate response group, whereas SA block was found in the high-rate response group and sinus bradycardia in the normal response group. In one case with sinus arrest (**), max %JRT was lower than 200%, and was not significantly influenced by the pacing rate.

Fig.11. HBE findings in each group.

Ordinate; S-A interval, A-H interval and H-V interval in msec, and pacing rate producing Wenckebach type second degree AH block in beats/min. In the low-rate response group, A-H interval was significantly ($P < 0.05$) longer than and the pacing rate producing Wenckebach periodicity lower than those in other groups.

cases (43%), the etiology was unknown, diphtheria and primary myocardial disease seemed to be more frequently found in patients belong to the low-rate response group, and ischemic heart disease in the high-rate response group and diphtheria in the normal response group. The incidence of heart failure was higher in the low-rate response group than in other groups.

DISCUSSION

1. Parameters for evaluation of overdrive suppression

In control subjects^{4,5,9,10} and animals¹¹⁻¹⁴ atrial overdrive stimulation induced a suppression of sinus node automaticity. The magnitude of overdrive suppression is expressed as SNRT, which is usually measured as the interval from the last pacing artifact to the first sinus beat. However, in patients with SSS, cycle length is not always longest in the first cycle after overdrive stimulation, but occasionally longest in the second or later cycles^{4,9,15,16}. Benditt reported that there are two types of secondary pauses which are useful in evaluation of the magnitude of overdrive suppression¹⁶. The duration of longest cycle length after overdrive was considered to be clinically important.

Fig.12. An incidence of syncopal attack in each group.

The striped bar shows the case with paroxysmal tachycardia. Note that the occurrence rate of syncopal attack and paroxysmal tachycardia was markedly high in the high-rate response group, middle in the low-rate group and low in the normal response group.

Therefore, in this study, the longest duration after overdrive stimulation was measured, by defining it as RT.

In normal subjects, postdrive pause was generally terminated by a sinus beat, but rarely

Fig.13. The relationship between max RT and the occurrence of syncopal attack in each group.

Open circles show cases without syncopal attack episodes, and filled circles show those with syncopal attack. In the low-rate response group, syncopal episodes were frequently despite a lower max RT. On the other hand, in the high-rate response group, syncopal episodes were frequently despite a lower max RT.

by an A-V junctional, a ventricular⁹ or an atrial escape beat!¹⁰ In patients with SSS, postdrive

pause was frequently ended by an escape beat of ectopic origins^{5,8,17}. We considered it clinically useful to measure RT without consideration of its origin, what Itō called the maximum automaticity recovery time.¹⁸

The duration of SNRT has been used in three different ways: absolute value^{8,9}, CSNRT⁵ and %SNRT^{15,19}. Since resting heart rate before test stimulation is related to SNRT^{4,9,20}, electrophysiologically SNRT should be expressed in terms of the resting sinus interval, e.g., as the ratio of SNRT to the resting P-P interval.

2. Pacing rate effect on recovery time

a) Pacing rate effect on SNRT in control subjects

In our study %SNRT showed a maximum value at a pacing rate between 140–160% and slightly decreased at higher rates. This two-phase phenomenon conforms to results reported by Mandel⁴, Kulbertus⁹ and Yabek,¹⁰ with

Fig.14. Comparison of clinical features in each group.

The upper graphs show from left to right, age, cardio-thoracic ratio and the incidence of heart failure, lower graphs show the incidence of diphtheria, ischemic heart disease and primary myocardial disease in percentage in each group.

maximum values found at 120 beats/min and 130 beats/min by these authors.

b) *Pacing rate effect on SNRT in patients with SSS*

Some papers have studied pacing rate effect on SNRT in patients with SSS. According to these reports, gradual SNRT prolongation occurred by increasing the rate of atrial stimulation. The maximum value was reached at 90–140 beats/min,^{16,20,21} and in higher drive rates, SNRT was shortened.⁴ In our study, when the drive rate was increased up to 200 beats/min, prolongation of the %SNRT average was proportional with a wide range of values. Cases with SSS could be divided into two groups: In the first, max %SNRT was less than 200% and it was not significantly influenced by the pacing rate, in the second group, however, max %SNRT was more than 200%, and it increased progressively with the pacing rate. These findings in this second group were similar to the experimental results of Lange¹³ and Chadda¹⁴ in that SNRT increased linearly with the pacing rate up to 200 beats/min, although they found no clinical observations concerning this phenomenon. It seems difficult to compare our results with others, since our atrial pacing method and parameters were different.

Why SNRT was markedly reduced at more rapid pacing rates, are explained by SA entrance block,²² catecholamine release,^{4,13} sinus node reentrant tachycardia²⁴ or/and hemodynamic alteration.²³ Our results from patients with SSS showed that SNRT was markedly prolonged rather than shortened by the excessive high-rate stimulation, although the mechanism involved is not clearly understood; possibly, hindrance factors of suppression such as entrance block and catecholamine release were decreased, or the suppression effect was significantly amplified.

c) *JRT in patients with SSS*

Overdrive suppression was also observed in the A-V junctional pacemaker.^{13,25} In our study max JRT of patients with SSS was 6173 ± 2679 msec, and max %JRT $398 \pm 109\%$. JRT was much longer than SNRT in control subjects, but it was difficult to judge whether automaticity of the A-V junctional pacemaker in patients with SSS was reduced or not. Narula believes that the automatic capacity of the A-V junctional pacemaker is also compromised in patients with SSS, because of the long periods of asystole without escape of the lower pacemaker.⁵ In normal subjects, JRT cannot be measured be-

cause the A-V junctional pacemaker is more readily depressed by overdrive stimulation than the sinus nodal pacemaker.^{13,25} Therefore, in human being no control data is available to indicate normal value of the escape interval of the A-V junctional pacemaker following suppression by atrial pacing. Jordan, studying patients of advanced A-V block and by animal examination, reported that the magnitude of A-V junctional tissue suppression by ventricular pacing was larger than that of the sinus node.²⁵ We considered that JRT, in our study, did not always mean reduction of A-V junctional activity. At any rate, the JRT magnitude was considered to play an important role in the duration of cardiac arrest, and in the occurrence of Adams-Stokes attack.

d) *Pacing rate effect on JRT in patients with SSS*

Few investigators have studied the effect of the pacing rate on JRT. In our results, we found a two-phase pattern exhibiting maximum pause at a pacing rate of 260–280%. Jordan obtained similar results in patients with advanced A-V block, although it may be difficult to compare our results with his because of differences in pacing site and the underlying disease.

3. *SSS classification using the overdrive suppression test*

In the present study, 94 patients (91%) had an abnormal max RT, 86 patients (83%) an abnormal max CRT and 85 patients (83%) an abnormal max %RT. These occurrence rates were as high as those of Mandel⁴ (93%) and Simonin⁶ (100%) but higher than those of Gupta⁷ (35%), Rosen⁸ (40%) and Narula⁵ (57%). These differences may be due to different criteria of the normal limit of RT and different methods of pacing rate, duration and rest period and measurements of parameters of overdrive suppression. In addition, SSS includes many types of arrhythmias, such as sinus bradycardia, sinus arrest and SA block. In this study, the occurrence rates of RT prolongation differed in each arrhythmia: sinus bradycardia 41%, sinus arrest 97% and SA block 95%. Therefore, the reason why occurrence rates differed between author was mainly thought to be that they had studied cases including a different percentage for each arrhythmia.

Hence, we tried to classify patients with SSS into three groups depending on %RT response against the pacing rate: low-rate, high-

rate and normal response groups. ECG and HBE findings and clinical features were compared among these groups.

Low-rate response group: Max RT was markedly prolonged and most beats after maximum pauses originated in the A-V junctional tissue. Sinus arrest was detected in most cases. Paroxysmal tachycardia and syncopal attack were found in about half the cases. HBE findings showed more A-V nodal conduction disturbance in this group than in others, and more cases of diphtheria, primary myocardial disease and heart failure were found in this group, as well.

High-rate response group: Max RT was significantly prolonged and the beat after maximum pause originated mostly in the sinus node. ECG findings showed SA block and normal sinus rhythm in most cases. Paroxysmal tachycardia and syncopal attack were very frequent. The average patient age in this group was higher, and ischemic heart disease was the commonest underlying disease.

Normal response group: RT showed normal value in most cases, and most beats after maximum pauses came from sinus node. Sinus bradycardia was found in most cases, while about a half had episodes of paroxysmal tachycardia; syncopal attack was rather rare. Some cases had diphtheria as a underlying disease. Each classification showed different clinical and electrophysiological findings.

4. Evaluation of past episodes and future occurrence of syncopal attack using overdrive suppression test

We have tried to find a method to evaluate whether past syncopal attacks were related to sinus node dysfunction, and whether a case with SSS may suffer a syncopal attack in future. Such occurrence is difficult to evaluate only from ECG findings. Most patients in the high-rate response group, for example, showed normal sinus rhythm, and if no transient SA block or sinus arrest after terminating tachycardia was recorded, it is very difficult to determine whether that these cases were suffering from SSS rather than Adams-Stokes attack. On the other hand, patients with sinus bradycardia, sinus arrest, SA block and bradycardia-tachycardia syndrome did not always have syncopal attack episodes. Therefore, the overdrive suppression test becomes a useful method for unmasking the presence of sinus node dysfunction and estimating its severity.

The effect of the pacing rate on RT has not been previously stressed, on the assumption that SNRT is decreased at a higher pacing rate.⁴ Therefore, pacing rates of more than 150 beats/min were not used in earlier studies. In our study, however, the maximum suppression of sinus node automaticity occurred at a higher pacing rate of 300–400% in some cases. When the resting heart rate was 70 beats/min, a pacing rate higher than 210 beats/min were required to obtain a pacing rate of 300%. We also obtained an interesting result of SNRT prolongation by using high rate stimulation in most cases with SA block. This is not consistent with the sinoatrial entrance block assumed by Strauss.²²

In the present study, about 50% of the patients with SSS complained of syncopal attack. This value was nearly similar to that of previous investigators. The incidence of syncopal attack episodes was very high (81%) in the high-rate response group, but lower in cases of normal response (24%) and low-rate response (48%) groups. Generally, the magnitude of overdrive suppression, shown by SNRT, is used as a criterion showing the severity of sinus node dysfunction. Itō proposed that if maximum automaticity recovery time was longer than 5000 msec, a pacemaker should be implanted,¹⁸ but in our study, syncopal attacks occurred also in cases with RT shorter than 4000 msec, especially in the high-rate response group. Therefore, we would like to recommend that for cases in the high-rate response group, pacemaker implantation should be taken into account even when RT is less than 4000 msec. Most these cases had episodes of paroxysmal tachycardia, usually treated with digitalis and antiarrhythmic drugs. According to our data, RT was strongly prolonged by digoxin injection;²⁶ we felt it was dangerous to use digoxin and other antiarrhythmic drugs in these cases, and therefore this group was thought to provide good candidates for pacemaker implantation. In the low-response group, heart failure was found in comparatively many cases and RT was prolonged by digoxin injection in half.²⁶ We felt it is advisable to implant a pacemaker in cases needing digitalis treatment. Although it has been widely reported that many patients with bradycardia-tachycardia syndrome complained of syncopal attack, few syncopal attacks occurred in cases with this syndrome belong to the normal response group. Therefore, our

classification appears to be useful for prediction of syncopal attack and for indication for pacemaker implantation in patients with bradycardia-tachycardia syndrome.

Acknowledgement

I thank Professor Hirosawa, K., Associate Professor Kimata, S. and Dr. Tanaka, T., Department of Internal Medicine, and Professor Kikuchi, R., Department of Physiology, Tokyo Women's Medical College, for advice and for correcting the manuscript.

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