Clinical Studies on the Sick Sinus Syndrome

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The diagnosis of the sick sinus syndrome is usually made by electrocardiographic findings. Therefore, many different etiologies and mechanisms of arrhythmias may be involved in patients with this syndrome.

The purpose of the present study is to assess the cardiac automaticity and the atrioventricular (A-V) conductivity of these patients, and to correlate the obtained results with their clinical symptoms and electrocardiographic findings.

MATERIALS AND METHODS

The clinical characteristics of materials are summarized in Fig. 1. They were composed of 22 females and 28 males, ranging in age from 6 to 74 years with mean age of 49.6 years, all who meet the diagnostic criteria proposed by Ferrer; 5 patients with sinus bradycardia (HR < 50 beats/min), 22 with sinoatrial block (S-A block) or sinus arrest, and 23 with bradycardia-tachycardia syndrome (BTS). Twenty four of these patients experienced Adams-Stokes syncope and 22 complained of recurrent dizziness, but the remaining 14 had no symptom associated with cerebral hypoperfusion. Parenthesized numbers in Fig. 1 indicate numbers of patients who had permanent pacemaker implantation.

The methods of overdrive suppression and recordings of His bundle electrograms were the same as previously described. In brief, atrial overdrive pacings were performed at various rates from 60 to 180 beats/min with increment of 20 beats/min for 15 and at a rate of 100 beats/min for various durations from 15 to 180 sec. Overdrive suppression was repeated over 10 times in each patient and the length of asystolic pause after cessation of the pacing was measured in each time. Then, the longest (the maximum pause) of the measured pauses in each patient was used for a parameter which evaluates depression of the cardiac automaticity. His bundle electrograms were recorded during atrial pacing at a rate of 80 beats/min and the following atrioventricular conduction times were measured; St-A time (stimulus to peak of the initial sharp deflection of A wave), A-H time and H-V time.

RESULTS

Fig. 1. Clinical characteristics of subjects. Parenthesized number indicates the number of cases having permanent pacemaker implantation. Abbreviations: SB, Sinus bradycardia; SAB or SA, sinoatrial block or sinus arrest; BTS, bradycardia-tachycardia syndrome.

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Fig.2. Relationship between cerebral symptoms and overdrive suppression. Shaded zone indicates a normal upper limit of maximum pause.

Fig.3. Relationship between cerebral symptoms and A-V conduction times.

70.1 ± 34.3 msec in syncope group, respectively. A significant difference (p < 0.005) was noted only between the first group and either of the remaining two. It is noticable that no patient in the first, 3 in the second and 7 in the third group exceeded the normal upper limit of the ST-A time (70 msec).

Relationship between electrocardiographic findings and the electrophysiologic examinations

The average maximum pause was 2.0 ± 0.9 sec in sinus bradycardia group, 5.0 ± 2.6 sec in S-A block or sinus arrest group, and 5.4 ± 2.3 sec in BTS group. A statistically significant differences (P < 0.005) were observed between litter of the former two and the last group (Fig. 4.).

Relationship between electrocardiographic findings and atioventricular conduction times are illustrated in Fig. 5. It is noteworthy that 9 of 10 patients who exhibited ST-A time prolonged beyond 70 msec belonged to BTS group. Consequently, the average ST-A time in this group was up to 76.5 ± 32.4 msec, showing a statistically significant difference (P < 0.005) from these in the remaining two groups. In A-H time, it was prolonged in nearly half the patients in both SAB or SA and BTS group but none is in the SB group. There were 6 patients in the

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Fig. 4. Relationship between electrocardiographic findings and overdrive suppression.

Fig. 5. Relationship between electrocardiographic findings and A-V conduction times.

former two groups, but not in the remaining group, who had prolonged H-V time over than 55 msec. However, any significant difference was not obtained among average A-H and H-V times in the three groups to each other.

**DISCUSSION**

**Evaluation of cardiac automaticity by overdrive suppression**

Several investigators\textsuperscript{12,13} reported in their animal experiments that asystolic pause after interruption of the overdrive pacing was prolonged, within limits, in proportion to increase in both pacing rate and duration. However, clinical result\textsuperscript{9,14,15} have not demonstrated such a clear data as the animal experiments, and reason of this discrepancy remains to be solved. Furthermore, the clinical application of overdrive suppression to the sick sinus syndrome offers a more complicating problem\textsuperscript{16} as follows; as the sick sinus syndrome may have more or less conduction disturbance in the sinoatrial junction in addition to depression of the automaticity, the rapid atrial impulses which are delivered by overdrive pacing can not always depolarize the pacemaker cells in the S-A node. Therefore, the most effective rate for overdrive suppression in these pacemaker cells can not be predicted, and hence, it may become reasonable to evaluate the depressed automaticity in the sick sinus syndrome by the longest (maximum pause) of all pauses that were obtained in each patient with changing overdrive pacing rates and durations. We reported\textsuperscript{11} previously that the normal value of the maximum pause thus obtained ranged from 0.7 to 1.5 sec (mean, 1.2 ± 0.14 sec, N = 10), and similar values for normal person were also reported by several authors\textsuperscript{7,14,15}. In present study, the maximum pause exceeded the normal upper limit in the all but two patients, and also it tended to be lengthened with development of the cerebral symptom.

These results imply that the maximum pause thus obtained is a useful parameter to evaluate depression of the cardiac automaticity.

**Evaluation of A-V conduction times by His bundle electrograms**

It is generally accepted\textsuperscript{17} that P-A time, the interval measured from the beginning of the P
wave of the surface electrocardiogram to the first rapid deflection of the A wave of the His bundle electrogram, is a measure of the conduction time from the S-A node to the approaches of the A-V node, and its normal value ranges from 25 to 40 msec. However, Waldo et al. indicated that a P wave in the body surface electrogram commonly began to inscribe 10 to 25 msec after initiation of the earliest atrial depolarization and, consequently, it is a poor indicator to recognize onset of the atrial excitation. Accordingly in the present paper, St-A time is used for a measure of the sinoatrial conduction time and its normal value was defined within 70 msec which is 25 msec longer than normal P-A time. Several authors have reported patients with intra-atrial conduction delay, but they have hardly referred to its clinical significance. Nevertheless a severe intra-atrial conduction defect may provide an indirect, but strong evidence for an extensive atrial damage, and this atrial damage may play a role to produce re-entrant tachycardia in BTS.

It is also accepted A-H and H-V times are measures of conduction time in the A-V node and His-Purkinje system, respectively.

Several electrophysiologic studies on the sick sinus syndrome revealed frequent association of prolongation in these conduction times with the depressed automaticity. Samely, nearly half the patient in both S-A block or sinus arrest and BTS groups had prolonged A-H time, but there were few who had prolonged H-V time. Therefore, pathologic changes in sick sinus syndrome may usually be confined to the supraventricular regions.

Conclusion

1) Overdrive suppression is clinically useful to evaluate depressed cardiac automaticity in the sick sinus syndrome.
2) His bundle electrograms revealed frequent association of atrioventricular conduction defects with the depressed automaticity in this syndrome.
3) Especially in BTS, marked intra-atrial conduction defect was observed, and this conduction defect may play a role for development of re-entrant tachycardia.
4) Pathologic changes in the sick sinus syndrome are supposed to involve the whole atrial tissues including not only the S-A nodal region but the A-V node and its approaches.

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REFERENCES