Mechanism of Supraventricular Tachycardia and Pharmacological Effects on It

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AN ELECTROPHYSIOLOGICAL study was performed in human patients in order 1) to classify the mechanisms of supraventricular tachycardia (SVT) in the cases with or without WPW syndrome, 2) to assess the underlying electrophysiological properties which allow the induction of SVT and 3) to elucidate the effects of anti-SVT drugs on such a mechanism associated with the development of SVT.

SUBJECTS AND METHODS

The study group consisted of 30 patients with previously documented SVT attacks. Sixteen had preexcitation (WPW group) and the others did not (SVT group). Twenty four subjects who did not have a history of SVT were served as control (control group). They included valvular heart disease, congenital heart disease, sclerotic heart disease and cardiac neurosis.

For electrophysiologic study, two multipolar electrodes catheters were introduced into the right heart through the femoral vein. A programmed electrical stimulus was applied to the right atrium after every 5 or 6 beats. Electrocardiographic lead as well as right atrial potential and His bundle electrogram were recorded simultaneously on a multichannel recorder and on a magnetic tape system.

Sinoatrial conduction time (SACT) was determined according to *Strauss et al.*¹ Functional refractory period of the AV node (AVFRP) was defined as the shortest HH' interval as the coupling interval of premature atrial contraction (AA') was shortened. The echo zone was defined in terms of the longest and the shortest AA' interval when atrial echo or SVT was induced. The study was repeated following an intravenous

injection of propranolol, 0.2 mg/kg of body weight, or procaine amide, 500 mg.

CRITERIA FOR CLASSIFICATION OF SVT MECHANISM

The mechanisms of SVT were classified into four types; AV nodal reentry (type Ia), SA nodal or atrial reentry (type Ib), ectopic atrial automaticity (type II) and reentry utilizing accessory pathway (type III).

The criteria for the diagnosis of type Ia included the following; 1) initiation and termination of SVT with properly applied atrial extra-stimulus, 2) a critical AH prolongation necessary for induction of SVT, 3) changes in VA conduction time related to AV conduction. The criteria for the diagnosis of type III included; 1) delineation of an echo zone which coincided with the effective refractory periods of the accessory pathway and the AV node, 2) in cases of reentry involving accessory pathway in VA direction, appropriately short and constant VA conduction time regardless of the variation of AV conduction time, 3) slowing in the rate of SVT following intraventricular conduction delay as shown in Figure 1. The diagnosis of type Ib in the present series of cases depended on the exclusion of type Ia and type III in those with the evidence of reentrant SVT. Criteria for the diagnosis of type II included; 1) resetting of the rhythm of SVT with premature atrial contraction, 2) temporary pause of SVT only after the cessation of atrial pacing at very high rate. Although the activation sequence of atrial excitation is very important for the diagnosis of the mechanism of SVT, the present study did not deal with this because the detailed data were available in only limited

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TABLE I CLASSIFICATION OF MECHANISMS

	SVT group	WPW group	
		preexcitation during the study	normal conduction during the study
Type Ia (AV nodal reentry)	6	1	1
Type Ib (SA nodal or atrial reentry)	1	—	—
Type II (ectopic automaticity)	2	—	1
Type III (reentry through accessory pathway)	—	5	3
undetermined	3	1	—
SVT not induced	2	1	—
Total	14	9	7

number of cases.

RESULTS

1) Classification of SVT mechanisms

SVT was observed during the study in 27 of 30 patients. The mechanisms were classified by accepting only the positive findings described above. The results are summarized in Table I. Of the SVT group patients, 6 were considered to have their reentry circuits in the AV node (type Ia), 2 were regarded as of ectopic automaticity (type II) and in 5 the mechanism of SVT could not be identified. The 2 patients diagnosed as of type II had chronic SVT which persisted for more than 2 months. Eight patients of the WPW group used their accessory pathways during SVT (type III). In two, reentry seemed to be confined to the AV node (type Ia) and, in one, ectopic automaticity was most likely as the mechanism (type II). In the remaining 5 patients the mechanisms were not clear.

Figure 2 shows a case with preexcitation and the AV conduction curve suggestive of dual AV nodal pathway. In this case, SVT was considered to be due to circus movement using both fast and slow pathways in AV direction and an accessory pathway in VA direction. Also depicted in Figure 3 is a case with WPW syndrome in which the reentrance seemed not to utilize the accessory pathway but to be confined to the AV node.

2) Underlying electrophysiological properties in cases with SVT

The SACT, AVFRP and AH interval were compared between the SVT, the WPW and the control groups. However, no significant differences were observed in those values between the three groups (Figure 4). Conduction patterns consistent with dual AV nodal pathway was demonstrated in 2 cases, one in the SVT and the other in the WPW group, and not found in the control group. There were also no significant difference between the three group subjects in the magnitude of the changes of electrophysiological activities following the administration of propranolol or procaine amide (Figure 5). In no cases, a dual AV nodal pathway appeared after the administration of the drugs.

3) Effects of drugs on AV conduction system or accessory pathway and echo zone

In order to exclude the effects of heart rate change on the drug-induced changes in AVFRP, AVFRP was corrected with heart rate by calculating the predicted normal value for the observed heart rate from the regression equation previously obtained by us, $Y = 0.186X + 273$, and subtracting the measured from this value. As shown in the Figure 6 the average corrected AVFRP increased from -15 to $+15$ msec and from -7 to $+47$ msec following propranolol and procaine amide, respectively. The refractory period of accessory pathway remained unchanged after propranolol, while increased after procaine amide in all 5 patients, in 2 of which the accessory pathways were completely blocked

Fig.1. Relationship between intraventricular conduction delay and the rate of SVT. In A, SVT is initiated by atrial stimulation when prolongation of A'H' interval reaches to a critical value. During SVT, VA conduction time varies as the conduction time in AV direction changes. Therefore the mechanism of SVT is diagnosed as of type Ia. It is noted here that the acceleration of the rate of SVT is accompanied with aberrant ventricular conduction. Shown in B is a case with WPW syndrome (type B) who demonstrates normal AV conduction during the study. SVT is initiated by premature atrial contraction. During SVT, development of intraventricular conduction delay results in prolongation of VA conduction time, which is followed by slowing of the rate of SVT. This suggests that the SVT circuit involves a part of ventricle. Therefore, type III is most likely as the mechanism of SVT. The width of dotted bar shown in the column of H depicts the interval from H to the end of QRS.

(Figure 7).

The effects on echo zone of these drugs are shown in Figure 8. Propranolol abolished the echo zone in one case of type Ib, increased in 2 and caused no appreciable changes in the other cases. After procaine amide, the echo zone was abolished or decreased in 5, unchanged in 2 and increased or created in 2.

DISCUSSION

Several kinds of mechanisms have been known to be involved in SVT. Since *Bigger* and *Goldreyer*^{2,3} demonstrated the initiation and the termination of SVT with atrial extrastimulus technique, the AV nodal reentry has been widely accepted as the most common mechanism of SVT. Later, SA nodal reentry and atrial reentry

Fig.2. SVT in a case with WPW syndrome (type B) as well as dual AV nodal pathway. During the study, the ventricular complex shows normal conduction pattern. Shortening of AA' interval results in lengthening of A'H'. At a critical AA' interval, a sudden increase of A'H' is seen. This supports the existence of dual AV nodal pathway. Echo beats (filled circles in the figure) are present on both fast and slow pathway conduction curves. This suggests that the failure of the fast pathway conduction in AV direction is not necessary for the echo phenomenon. Considering the fact that the VA conduction time is constant regardless of the changes in AV conduction time, the SVT circuit can be regarded to involve either slow or fast AV nodal pathway in AV direction and an accessory pathway in VA direction. EZ=echo zone.

Fig.3. AV nodal reentrant SVT in a case with WPW syndrome (type B). Echo beats are induced with A₁. It should be noted that A₁ is followed by both normal AV conduction (H₁) and anomalous excitation and that SVT persists in spite of A₃ blocked in both normal and anomalous pathways. This suggests that an accessory pathway is not involved in SVT mechanism. The induction of SVT is associated with a critical prolongation of AH interval and nonconducted A₃ is accompanied with a prolonged A₃H₃ interval. The latter suggests the deep penetration of previous excitation in the AV node. Therefore, the SVT in this case can be regarded as of reentry not through accessory pathway but confined to the AV node.

Fig.4. The electrophysiological properties in SVT, WPW and control subjects.

have been proposed as other additional mechanisms^{3,5-7} and an ectopic atrial focus as the mechanism for most of the persistent or chronic SVT^{4,8}. Although in cases with WPW syndrome, the accessory pathway has been regarded to serve as a pathway participating in the circus movement of excitation, the mechanisms other than

reentry through accessory pathway has been also manifested in some patients^{9,12,13}. The findings obtained in the present study were: 1) the AV nodal reentry was the mechanism of SVT in 6 cases of SVT group; 2) the mechanism of persistent SVT observed in 2 cases was attributed to ectopic automaticity; 3) three of WPW group

Fig.5. Pharmacological responses in electrophysiological activity in SVT, WPW and control subjects. BCL=basic cycle length.

patients did not use their accessory pathways during SVT; 4) three of the patients not showing preexcitation in AV direction during the study seemed to use accessory pathways in VA direction during the SVT. The classification of SVT

patients according to the mechanisms involved must be important because the various mechanisms suggest the differences in the clinical behavior and pharmacological responses of SVT. The study also suggests that an accessory path-

Fig. 6. The effects of propranolol and procaine amide on AVFRP corrected with heart rate change. B=before the drugs; A=after the drugs.

way is not always involved in the mechanism of SVT in the cases with WPW syndrome, while there might be such an accessory pathway which is concealed in AV direction but activated in VA direction^{10,11,13} in the cases without documented WPW syndrome. These are of important therapeutic implication in regard to current surgical interventions, since the surgical destruction of accessory pathways would not benefit in the former patients while it would in the latter patients if the accessory pathway could be detected.

Concerning the underlying electrophysiological properties in cases with SVT, dual AV nodal pathways have been described in some patients with AV nodal reentrant SVT.¹⁴ Coumel¹⁵ observed a prolongation of SACT in 2 of 11 subjects with intraatrial reentrant SVT. Bissett *et al.*¹⁶ reported that there were patients who developed dual AV nodal pathways following propranolol. However, in the present study, no characteristic electrophysiological properties of SA or AV conduction systems were observed in both SVT and

Fig. 7. The effects of propranolol and procaine amide on refractory period of accessory pathway. B=before the drugs; A=after the drugs.

WPW group patients. No cases demonstrated abnormal pharmacological responses in these properties and no appearance of dual AV nodal pathways was observed after the drugs. Rather, it was noted that, in a case who had both preexcitation and a dual AV nodal pathway, VA conduction through an accessory pathway was diagnosed as the mechanism of SVT.

Propranolol and procaine amide are the drugs which have been widely used in the management of SVT. Propranolol has been known to slow the sinus rate, prolong the AV conduction time and lengthen the AVFRP.¹⁷ As shown in the present study, the actions of propranolol on echo zone are variable. Therefore, propranolol should be regarded to be beneficial because of slowing of the rate of SVT, loss of ability to sustain SVT or protection against spontaneous premature beats which might initiate SVT. In contrast to the previous observations¹⁸⁻²⁰ demonstrating the minimal effects of procaine amide on AV conduction system, we noticed it prolonged AVFRP to the same degree as propranolol. However, procaine amide was found to have a beneficial effect on the echo zone not only in the cases with reentry utilizing accessory pathways but also in those with AV nodal reentrant SVT. Since procaine amide makes the accessory pathway refractory, it will interrupt SVT using an accessory pathway but might influence the SVT circuit confined to the AV node in the same manner as propranolol. These are two possible

Echo zone after propranolol

Echo zone after procaine amide

Fig.8. The effects of propranolol and procaine amide on echo zone.
White bar = before the drugs; dotted bar = after the drugs.

explanations of the difference on the effect on echo zone of these two drugs. 1) The reentry circuit during SVT, which fails to be initiated following procaine amide, may include the accessory pathway and not be confined to the AV

node. 2) The effect on AV and VA conduction over the AV node following administration of procaine amide may not be the same.

SUMMARY

Electrophysiologic study in 30 patients with documented SVT, 16 of whom had preexcitation, and in 24 control subjects were reported.

The mechanisms of SVT were found to be various and to include reentry confined to the AV node, SA node or the atrium, reentry through accessory pathway and ectopic atrial automaticity. It was noted that some of SVT might use concealed accessory pathway in VA direction while the some of SVT in WPW syndrome might not utilize accessory pathways.

Electrophysiological properties in cases with SVT did not differ from those observed in controls. There were no differences in the mode of electrophysiological action of drugs between SVT and control groups.

Both propranolol and procaine amide slightly depressed AV conduction. However, the action of the drugs on echo zone were not the same. The basic mechanisms of the action of the drugs on SVT were discussed.

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