RECIROCATING SUPRAVENTRICULAR TACHYCARDIA INITIATED BY PAIRED PREMATURE BEATS

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THE clinical association between atrial premature beats and paroxysmal supraventricular tachycardia is well recognized.\textsuperscript{1,2,3,6,8,12,16,25,33,50} Under cardiac catheterization Goldreyer and Damato\textsuperscript{12} not only initiated but also terminated multiple episode of supraventricular tachycardia by single atrial premature stimuli in 4 patients with a history of paroxysmal supraventricular tachycardia. It was concluded by them that supraventricular tachycardia results from atrial reentry via the A-V node. Han\textsuperscript{15} expressed the same conclusion. Whereas it was shown by Janse et al\textsuperscript{17} in isolated rabbit heart preparation that a cirsus movement in the A-V node, based on functional longitudinal dissociation of the upper A-V node, was the underlying mechanism of the arrhythmia. Luria\textsuperscript{24} found one atrial reciprocating tachycardia and two repetitive tachycardia in 120 cases of paroxysmal tachycardia. Levine and Smith\textsuperscript{20} found 38 repetitive tachycardia and in 2 of them in whom the tachycardia was supraventricular in origin, a reciprocation was demonstrated.

In this paper, 2 cases of paroxysmal reciprocating supraventricular tachycardia were presented. In both cases supraventricular tachycardia was initiated by paired supraventricular premature beats, the first of the 2 occurring within a specific portion of the relative refractory period of the A-V conduction system. Both appeared in the form of repetitive tachycardia\textsuperscript{19,20} one with and the other without block.

REPORT OF CASES

In case 1, the coupling interval of supraventricular premature beats was measured before and during drip infusion of atropine (Fig. 1). Before drip infusion, premature beats with a coupling interval of 0.49 to 0.52 second (35 beats) initiated a paroxysm of supraventricular tachycardia, and those with a coupling interval of 0.53 to 0.67 second (56 beats) failed to initiate it. Premature beats with a coupling interval of 0.48 second and less (3 beats) did not initiate supraventricular tachycardia either. The R-R interval during tachycardia was 0.46 to 0.50 second although the first few cycles were slightly longer than those after the tachycardia was stabilized. During drip infusion of atropine, the number of paroxysmal tachycardia and pancardiac asystole decreased. Among 38 supraventricular premature beats with a coupling interval of 0.46 to 0.54 second, 32 induced supraventricular tachycardia, but the remaining 6 failed to initiate it. Whereas among 14 supraventricular premature beats with a coupling interval of 0.55 to 0.78 second, 13 failed to induce supraventricular tachycardia, but the remaining one premature beat with a coupling interval of 0.55 second initiated it. This case showed no P waves in the standard 12 lead ECGs. It was demonstrated only in esophageal leads as shown in Fig. 2. During sinus rhythm, the P wave in E37 was positive-negative biphasic. When the ventricular rhythm was irregular with neither pancardiac asystole nor supraventricular tachycardia, it showed atrial flutter or atrial tachycardia with block. During paroxysmal

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Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

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supraventricular tachycardia, the intervening P wave in E35 was inverted as shown in Fig. 3. The P wave of the esophageal leads, however, gave no convincing evidence as to whether the impulse is of atrial or A-V nodal origin because it reflects mainly the anteroposterior spread of the impulse. In Fig. 4, panchardiac asystole of ca. 7.7 second duration was first followed by 6 supraventricular escape beats and then by supraventricular tachycardia initiated by a supraventricular premature beat (R’1) with a coupling interval of 0.50 second. This premature beat must have occurred during the relative refractory period of one of the A-V conduction pathways which had been discharged by a preceding supraventricular impulse because it showed an aberrant ventricular conduction of an RBBB configuration. The following QRS complex (R’2) with a coupling interval of 0.38 second also showed an rSr’ pattern but of normal duration, which indicates that this impulse must have spread the other pathway which had not been discharged by the preceding premature impulse. These two supraventricular premature impulses were followed by the QRS complexes of the same configuration of normal duration with an R’-R’ interval of 0.42 to 0.44 second. This seems to indicate that paroxysmal supraventricular tachycardia is initiated not by a single premature impulse but in cooperation with another premature impulse which spreads forward through the other pathway and turns back to the atria through the pathway which had been discharged by the first premature impulse, thus producing a sustained reciprocation between the A-V node and the atria. Such supraventricular tachycardia alternated repeatedly with panchardiac asystole of various duration. This makes it unlikely that we are dealing with a rapidly firing ectopic pacemaker in the atrium or the A-V node. Because it would be unexplainable without an assumption of reciprocation of an impulse initiated by an escape or a premature impulse that a rapidly firing supraventricular ectopic focus could alternate with its complete
arrest of considerable duration.

In case 2, tachycardia occurred also in a repetitive form without S-A block. In Fig. 5, the first atrial premature beat (1) occurring with an R-R' interval of 0.44 second and a P-P' interval of 0.40 second did not initiate supraventricular tachycardia. The second atrial premature beat (2) occurring with an R-R' interval of 0.40 second and a P-P' interval of 0.32 second induced paroxysmal supraventricular tachycardia. However, in lead II, an inverted, retrograde P wave appeared not after the first atrial premature beat but after the second atrial premature beat. This seemingly interpolated atrial premature beat (2) showed an aberrant ventricular conduction of an RBBB configuration. The R'-R' interval during paroxysmal tachycardia was 0.40 to 0.42 second, and the QRS complexes were of normal duration. The paroxysmal supraventricular tachycardia was discontinued spontaneously after 6 seconds when the R'-R' interval (R'13-R'14) became 0.44 second. The third atrial premature beat (3) occurring with an R-R' interval of 0.40 second and a P-P' interval of 0.32 second was also followed by another atrial premature beat which was preceded by an upward P wave in lead II. This second atrial premature impulse initiated paroxysmal supraventricular tachycardia consisting of alternate appearance of a retrograde P wave and a normal QRS complex. The polarity of the sinus and the retrograde P waves was clear in leads aVR and aVF as shown in Fig. 6. In the precordial leads of Fig. 7, paired supraventricular premature beats were clearly observed especially in leads V1 to V3. The first atrial premature beat (R'1) showed its own P wave on the top of the preceding T wave with a P-P' interval of 0.32 second together with P-R prolongation and ventricular aberration (V1). The next QRS complex following this premature beat (R'2) showed its upward P wave (V2 to V3) although it was slightly sharper with a shorter P-R interval than other sinus P waves. The succeeding QRS complexes (R') were all preceded by a negative P wave, indicating the opposite direction of the P wave vector during paroxysmal tachycardia and also supporting the underlying mechanism of reciprocation of an impulse. In Fig. 8, the sinus interval was 1.04 second to 1.10 seconds. Early atrial premature impulses (P'1 and P'2) with a
P-P' interval of 0.34 to 0.36 second were blocked, the retrograde conduction of which, however, reset the sinus cycle. The antegrade block and the retrograde conduction of early atrial premature impulses strongly indicate the possibility that the antegrade A-V conduction pathway is more refractory than the retrograde A-S conduction pathway. Whereas the resetting of the sinus cycle by retrograde conduction of an early atrial premature impulse indicates the probability that an atrial premature impulse, having reached the sinus node, returns back to the atria as an echo. The third atrial premature impulse (3) was conducted with a P-P' interval of 0.40 second and followed by another atrial premature beat with an R'1-R'2 interval of 0.40 second. After the second atrial premature beat (R'2), a retrograde P wave and a normal QRS complex appeared alternately. Also in Fig. 9, the first atrial premature impulse (P'1) with a P-P' interval of 0.36 second was blocked antegradely but its retrograde conduction reset the sinus cycle. The second atrial premature impulse (P'2) with a P-P' interval of 0.44 second was conducted with an R-R' interval of 0.48 second and ventricular aberration but failed to initiate paroxysmal supraventricular tachycardia. Its retrograde conduction also reset the sinus cycle. The atrial premature beat (P'3) coupled with another atrial premature beat was followed by a retrograde P wave and a QRS complex appearing alternately for several seconds. These observations strongly indicates the possibility that as a result of retrograde conduction an echo impulse via the S-A node can occur as the second component of the paired premature beats. On the basis of the illustrations and documentations above, it can safely be stated that paroxysmal supraventricular tachycardia, at least in these cases, is initiated by paired supraventricular premature impulses. The pair may consist of a) a supraventricular premature beat and a following sinus beat b) paired supraventricular premature beats or c) a supraventricular premature beat and an echo beat via the S-A node. In Fig. 10, the effect of the oculovagal reflex was demonstrated. In strips A and B, two paroxysms of supraventricular tachycardia occurred. After oppression of the eye balls, two pairs of supraventricular premature beats (R'1-R'2) appeared in strips D.
and E without initiating paroxysmal tachycardia. This indicates that the pathway which has been discharged by the first premature beat is still refractory to the retrograde conduction of the second premature beat coming through the other pathway. This retrograde block of the second premature beat must be due to the oculovagal reflex. And the fact that two supraventricular premature beats appeared as a pair in strips D and E (R'1-R'2) strongly indicates that the R'2 is causally linked to the R'1, i.e., an echo beat via the S-A node.

**DISCUSSION**

There are numerous experimental and clinical reports on the role of a premature beat on reciprocation and reciprocating supraventricular tachycardia\(^1\)–\(^13\),\(^15\)–\(^19\),\(^21\)–\(^29\),\(^31\)–\(^33\),\(^36\)–\(^40\),\(^42\)–\(^50\). Reciprocation through the A-V node has long been considered as a likely explanation for paroxysmal A-V nodal tachycardia in the human heart\(^1\)–\(^3\),\(^5\),\(^12\),\(^13\),\(^15\)–\(^18\),\(^22\),\(^23\),\(^28\)–\(^32\),\(^36\),\(^40\),\(^47\)–\(^50\). According to Mendez et al.\(^28\) the necessary condition for such a self-sustained reciprocal rhythm would be: a) the round-trip conduction time for a premature beat must exceed the refractory period of the retrograde conduction pathway, b) lateral propagation must be impossible for the return impulse above the level of the dissociation, so that the retrograde pathway is not occluded by a short circuit, and c) an equilibrium must be reached at which the round-trip circuit time and the refractory period of the limiting components of the pathway remain in balance. It is obvious that the necessary conditions could only be achieved under the influence of an agency which facilitates intra-nodal propagation, particularly in the retrograde direction and simultaneously abbreviates the effective refractory period of the limiting element in the pathway. Goal and Han\(^1\) stated that ectopic activity is increased in the atrium when the basic heart rate is slowed just as it is in the ventricle, indicating the possible role of the vagus nerve in facilitating the induction of atrial tachyarrhythmias. In the human heart without medication, such an agency is the autonomic nervous system affected by mental situations.

It is well known that “R on T” phenomenon\(^43\) is often followed by various ventricular arrhythmias because it corresponds to the ventricular vulnerable period. Likewise, there must be an atrial vulnerable period which is referred to as “R on P” phenomenon by Csapó. However, it was shown in the present cases that the atrial vulnerability to a premature beat is not applicable to paroxysmal reciprocating supraventricular tachycardia. As indicated in the previous reports\(^1\),\(^3\)–\(^5\) an atrial echo can occur not only through the A-V node but also through the S-A node. An impulse that originates in the atria is conducted retrogradely to the S-A node and turns back to reactivate the atria and the ventricles. In case 2, as shown in Fig. 9, a premature beat obviously spreads backward to the S-A node, resetting the sinus cycle. If, like an intranodal dissociation, an intra-sinus dissociation occurs, a supraventricular premature impulse,
having reached a portion of the S-A node, turns
back to the atria through the non-refractory
pathway which has not been discharged by the
retrograde conduction. The P-P' interval which
reset the sinus cycle by retrograde conduction
was between 0.36 and 0.44 second, and that
which initiated a sustained supraventricular
tachycardia was 0.40 second. The probable
mechanism thereby is shown in
Fig. 11. Effects of the paired stimuli to the heart
have been reported\cite{41,45} which, however, had no
connection to the present report.

**Summary**

Two cases of paroxysmal supraventricular
tachycardia were documented, and its underlying
mechanism was discussed.

1) An antegrade A-V conduction pathway was
found to be more refractory than a retrograde A-S conduction pathway because an antegrade A-V block was usually accompanied by a retro-
grade A-S conduction, resetting the sinus cycle.

2) A single supraventricular premature beat did
not initiate paroxysmal tachycardia.

3) Paroxysmal tachycardia consisting of an
alternate appearance of a retrograde P wave and a
QRS complex of normal configuration (reciprocation) always followed a pair of two premature
beats.

4) The first premature beat showed an aberrant
ventricular conduction, indicating that the im-
pulse spread the same way as that of the
preceding sinus impulse.

5) The second premature impulse with an ante-
grade P wave showed a QRS complex of normal
configuration similar to those appearing during
paroxysmal tachycardia with a shorter coupling
interval, indicating that the second premature
impulse spread the pathway which had not been
discharged by the preceding premature impulse.

6) Even when the reciprocating supraventricular
tachycardia was suppressed by oculovagal reflex,
these two premature beats appeared as a pair,
indicating that the second premature impulse is
causally linked to the first, i.e., an atrial echo via
the S-A node.

7) It was inferred that this echo impulse via the
S-A node spreads the pathway which has not been
discharged by the first premature impulse and turns back to the atria through the pathway
which has been discharged by the first premature
impulse but has then recovered, thus initiating a
sustained reciprocation of an impulse.

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LEGENDS FOR FIGURES

Fig. 1. R-R' Interval and Reciprocation.
Fig. 2. ECGs during Sinus Rhythm.
During sinus rhythm the P wave in E37 was positive-negative biphasic. The P-Q interval was 0.20 second.
Fig. 3. ECGs during Paroxysmal Tachycardia.
During paroxysmal supraventricular tachycardia, the P wave was negative in E35 and appeared on the later half of the preceding T wave. The P-Q interval was 0.22 second.
Fig. 4. Paroxysmal Atrial and Paroxysmal Tachycardia.
A paroxysm of paracardiac asystole of ca. 7.7 second duration was first followed by 6 supraventricular escape beats. Then, these were followed by a supraventricular premature beat with aberrant conduction (R'1). The second premature beat (R'2) had also an rSR' pattern but of normal duration. These two premature beats were followed by a paroxysmal supraventricular tachycardia consisting of the same QRS complexes.
Fig. 5. Sinus Rhythm and Paroxysmal Tachycardia.
The first atrial premature impulse (marked by an arrow) was blocked antegrade but conducted retrogradely, resetting the sinus cycle. The first conducted atrial premature beat (1) was coupled to the preceding sinus beat with an R-R' interval of 0.44 second and a P-P' interval of 0.40 second without initiating a paroxysmal supraventricular tachycardia. The second atrial premature beat (2) was coupled to the sinus beat with an R-R' interval of 0.40 second and a P-P' interval of 0.32 second. This was followed by another supraventricular premature beat with a positive P wave as observed in lead II (paired atrial premature beats). These two successive atrial premature beats initiated a paroxysmal supraventricular tachycardia consisting of alternate appearance of a retrograde P wave and a QRS complex of normal duration. The paroxysmal tachycardia ceased spontaneously when the R-R' interval (R13-R14) became 0.44 second. The R-R' interval during the tachycardia was 0.40 to 0.42 second. The third atrial premature beat (3) which occurred with the same coupling interval as that of the second one also initiated a paroxysmal supraventricular tachycardia.
Fig. 6. Polarity of the P Waves in the Limb Leads.
In lead aVR, the first atrial premature beat (R'1) was preceded by a negative P wave superimposed on the preceding T wave. The ventricular complex of the R'1 was aberrant but the second one (R'2) was almost normal in duration and configuration, which indicates that the latter impulse must have passed the different pathway. These two atrial premature beats were followed by alternate appearance of a retrograde P wave and a normal QRS complex of the same configuration. In lead aVF, the P wave of the R'1 was positive superimposed on the preceding T wave, and that of the R'2 was positive-negative biphasic which was different from both the sinus and the retrograde P waves.
Fig. 7. Polarity of the P Waves in the Chest Leads.
The fact that the P waves of the first two premature beats (paired premature beats) are different from the following P waves of the paroxysmal supraventricular tachycardia was clearly seen in lead V1 to V3 in which the former were positive and the latter negative. The fact that the first premature beat (R'1) with a coupling interval of 0.42 second spreads the same pathway as that of the sinus beat was shown by its aberrant form and that the second premature beat (R'2) spreads the different pathway was shown by its normal configuration in spite of a shorter R'-R' interval of 0.38 second. The QRS complexes of paroxysmal tachycardia were similar to that of the second premature beat (R'2) in configuration, which indicates that both impulses spread the same pathway.
Fig. 8. Resetting of the Sinus Cycle by Retrograde Conduction.
The preceding paroxysmal tachycardia was discontinued spontaneously without prolongation of the R-R' interval. The first (P'1) and the second (P'2) atrial premature impulses were blocked antegrade but conducted retrogradely, resetting the sinus cycle. The third atrial premature impulse (P'3) was conducted to the ventricles with ventricular aberration which was followed by an antegrade P wave (P'4) and a normally configured QRS complex. These two atrial premature beats were followed alternately by a retrograde P wave and a QRS complex of similar configuration to that of the R'2. This indicates that paired atrial premature beats with an appropriate timing initiate a reciprocating supraventricular tachycardia.

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Fig.9. Possibility of the R'2 to be an Echo Beat via the S-A Node.

As in Fig.8, the first atrial premature impulse (P'1) was blocked antegradely but conducted retrogradely to the sinus node, resetting the sinus cycle. The second atrial premature impulse (P'2) was conducted antegradely to the ventricles with aberrant conduction and also retrogradely to the sinus node, resetting the sinus cycle. The third atrial premature impulse (P'3) was conducted antegradely to the ventricles with aberrant conduction which was followed by an antegrade P wave (P') without ventricular aberration. This indicates the possibility that retrograde conduction of the first atrial premature impulse, having reached the sinus node, returns back to the atria, producing an atrial echo which is further conducted to the ventricles through one pathway which has not been previously discharged. A part of impulse is again returns back to the atria through the other pathway which has been discharged by the first premature beat and has recovered when the atrial echo impulse reaches the common pathway, thus initiating a sustained reciprocation.

Fig.10. Suppression of Reciprocation by Oculovagal Reflex.

In strip A, the P-R interval of the sinus beat was 0.12 second and that of the first atrial premature beat (R'1) 0.20 second. The P-R interval of the second atrial premature beat (R'2) was also 0.20 second. However, the P-R interval during supraventricular tachycardia was 0.16 second. These two premature beats were followed alternately by a retrograde P wave and a QRS complex similar to that of the R'2. The similarity was shown by the first deflection of the QRS complex, i.e., the sinus beat showed an rS pattern and both the R'2 and the succeeding QRS complexes a Qr or a QS pattern. In strip B, the atrial premature impulse (P'1) was blocked antegradely with retrograde resetting of the sinus cycle. The second atrial premature impulse (P'2) initiated a paroxysmal supraventricular tachycardia as in strip A. In both strips, the paroxysm ceased spontaneously with a slight increase in the R-R interval. After oppression of the eye balls, the paroxysmal tachycardia was completely suppressed. In strip C, the first atrial premature impulse (P'1) was blocked antegradely with retrograde resetting of the sinus cycle. In strips D and E, the first atrial premature impulse was conducted to the ventricles with aberrant conduction which was followed by a QRS complex of a Qr pattern similar to those occurring during paroxysmal tachycardia. It is interesting to note that these two premature beats appeared as a pair, indicating that the second premature beat (R'2) is causally linked to the first (R'1). This fact strongly indicates that the retrograde conduction pathway which had been discharged antegradely by the first premature beat was still refractory as a result of the vagal effect when the reciprocal impulse via the the sinus node reached the A-V node through the other pathway which had not been discharged. In other words, the retrograde portion of the round-trip circuit was refractory as a result of oculovagal reflex.

Fig.11. Probable Mechanism of Initiation of Reciprocation.

The sinus impulse spreads to the ventricles through the alpha pathway. The first atrial premature impulse spreads also through the alpha pathway to the ventricles with aberrant ventricular conduction. The second atrial premature impulse, however, spreads to the ventricles through the beta pathway without aberrant ventricular conduction. Since it was repeatedly demonstrated that a retrograde pathway is less refractory than an antegrade pathway and that the two premature beats appear as a pair, this second atrial premature impulse can be an atrial echo via the S-A node. A part of this reciprocal impulse via the S-A node, having reached the A-V node, again turns back to the atria through the alpha pathway which has been recovered. A repetition of the same sequence establishes a sustained reciprocating supraventricular tachycardia.