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Clinical Significance of Wide QRS Complexes at the Termination of Paroxysmal Supraventricular Tachycardias

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Abstract

Background: A wide QRS complex is not a rare electrocardiographic phenomenon at the termination of paroxysmal supraventricular tachycardia (PSVT), but no plausible underlying mechanism has yet been proposed. The purpose of the present study was to elucidate the frequency and the underlying mechanism of the wide QRS complexes at the termination of PSVT.

Methods: We retrospectively reviewed 305 electrocardiograms (ECGs) from 100 patients, on which PSVT termination was recorded. The frequency of the wide QRS complexes was analyzed in 181 ECGs to avoid duplication, because there were 124 ECGs obtained from the same patients with same methods. The 181 ECGs were divided by morphology into three groups: Type A, termination with wide QRS complex without pause; Type B, wide QRS complex following initial pause after termination; Type C, wide QRS complex following the first narrow QRS after termination.

Results: The wide QRS complex was recorded in 81/181 (44.8%) ECGs (Type A; 3/81 (3.7%), Type B; 44/81 (54.3%), Type C; 62/81 (55.6%)) and its frequency was not dependent on the mechanism of PSVT. It was more frequently observed after a long pause, and was frequently induced by procedures that increase vagal tone, such as intravenous adenosine 5'-triphosphate administration (16/22: 72.7%) and vagal stimulation maneuvers (16/32: 50%). There were a total of 41 wide QRS complexes (44.6%) which had a preceding sinus P wave, out of a total of 92 wide QRS complexes in all three types. These 41 wide QRS complexes included 30/44 (68.2%) Type B wide QRS, and 11 (24.4%) Type C wide QRS complexes. Conclusion. The aberrant conduction or escaped ventricular contraction was suggested to be the underlying mechanism of the majority of wide QRS complexes and ventricular premature contraction is less frequent. (J Nippon Med Sch 2002; 69: 525–533)

Key words: paroxysmal supraventricular tachycardia, wide QRS complex, vagal tone, antiarrhythmic drug, ventricular premature contraction

Introduction

Unexpected wide QRS complexes at the termination of paroxysmal supraventricular tachycardia (PSVT) are curious electrocardiographic phenomena. Their occurrence and clinical significance are not fully understood. They have been observed at the termination of PSVT with intravenous verapamil, adenosine 5'-triphosphate (ATP)[1–14] and carotid

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sinus massage[15], and are described as development of ventricular premature complexes at the termination of PSVT. It is thought that they result from the activation of multiple factors, although the mechanisms are unknown. In clinical practice, we frequently note the unexpected occurrence of a wide QRS complex on the cessation of PSVT with verapamil, but not in cases in which class I antiarrhythmic drugs such as disopyramide are administrated. To elucidate these very interesting electrophysiologic phenomena, we retrospectively investigated the frequency and the clinical significance of wide QRS complexes at the reversion of PSVT, and proposed possible mechanisms for these complexes.

Materials and Methods

From 1990 to 1994, in the out-patient Clinic of Nippon Medical School Hospital, 305 consecutive electrocardiograms documenting the termination of PSVT from 100 patients (65 men, 35 women, mean age 50±15 years) were evaluated retrospectively. Seven patients had ischemic heart disease, 4 valvular disease, 4 hypertension, 2 cardiomyopathy and 2 sick sinus syndrome as underlying heart diseases, and the remaining 82 patients had no obvious underlying heart disease. Patients who had a history of frequent ventricular arrhythmia or other types of atrial tachyarrhythmias were excluded. The mechanism of the PSVT was defined on the basis of the electrophysiologic profiles obtained from electrophysiologic study. The frequency of the wide QRS complexes were analyzed in 181 electrocardiograms to avoid duplication, because there were 124 electrocardiograms obtained from same patients with same methods.

In this study, the width of a wide QRS complex was defined as grater than 120 msec, and its morphology is clearly different from that of the QRS complex in sinus rhythm or in supraventricular tachycardia on the standard electrocardiogram recorded at a paper speed of 25 mm/sec. Tracings of the wide QRS complexes were evaluated from the reversion to the sinus rhythm immediately following the termination of PSVT for a period of 10 seconds, and the complexes were classified into the following three categories on the basis of the timing of their appearance and their electrocardiographic characteristics. The type of wide QRS complex was evaluated only for the first one in each type in each strip.

Definitions

Type A: Wide QRS complex that appeared with shorter coupling intervals than the cycle length of PSVT. PSVT is terminated by a type A wide QRS which is usually diagnosed as ventricular premature complex.

Type B: Wide QRS complex that was the first QRS complex, appearing after the termination of the PSVT. This type of wide QRS complex is accompanied by longer preceding coupling intervals than the PSVT cycle length.

Type C: Wide QRS complex following the first narrow QRS after termination.

These three types of wide QRS complexes are represented in **Fig. 1**. If we observed two or more wide QRS complexes at a reversion of tachycardia, every wide QRS complex was evaluated and classified as above.

Electrocardiographic measurements

For each electrocardiogram, the following four measurements were made.

1) Maximum RR (Max RR) : The longest RR interval during the 10 seconds following the termination of tachycardia.

2) Preceding RR (Prec RR) : The RR interval preceding the wide QRS; this means the coupling interval of the wide QRS complex.

3) RP interval: The interval between the onset of the R wave of the final QRS complex of tachycardia and the onset of the first sinus P wave after the termination of tachycardia

Statistics

Descriptive data are expressed as mean±SD. The three groups were compared by Student's t test. We used chi-square tests to compare the frequencies

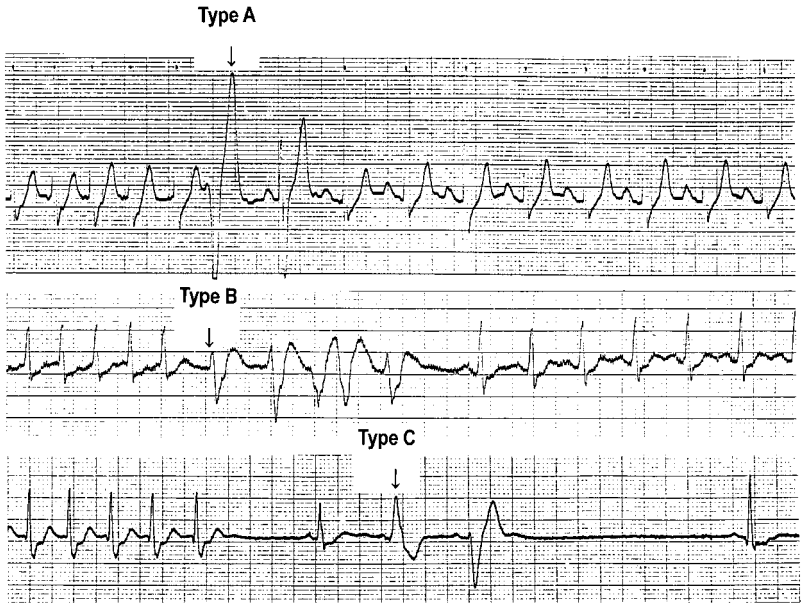

Fig. 1 Three types of wide QRS complexes at the termination of PSVT.

with which wide QRS complex was found in the patient population in relation to sex, underlying cardiovascular disease and mechanism of tachycardia. For all comparisons, statistical significance was assessed at the 0.05 level by using two-tailed p values.

Results

Characteristics of the patients

In all, 305 electrocardiograms in which the termination of PSVT was recorded were obtained from 100 patients, including 50 with atrioventricular reentrant tachycardia, 43 with atrioventricular node reentrant tachycardia, and 2 with intra-atrial reentrant tachycardia. The remaining 5 were not evaluated on electrophysiological study. In these 305 tracings, 193 episodes were terminated by intravenous ATP or antiarrhythmic drugs, and 60 were terminated by vagal maneuvers such as carotid sinus massage or the Valsalva maneuver. The remaining 52 were terminated by means of transesophageal pacing.

Frequency of wide QRS complexes

Wide QRS complexes occurring at the termination of PSVT were observed in 156 (51.1%) of the 305 ECGs, and in 81 (44.8%) of the selected 181 ECG strips without duplication. These 81 ECGs were recorded from 61 patients, and 100 ECGs that had no wide QRS complexes were from 63 patients. There was no statistical difference between the two patients groups in clinical background (**Table 1**).

A total of 202 wide QRS complexes were recorded in these 81 ECG strips, which contained 80 isolated wide QRS complexes, 27 episodes of 2 successive wide QRS complexes, 11 episodes of 3 successive complexes, 5 episodes of 4 successive complexes, one episode of 5, and one episode of 10 successive complexes. In none of the consecutive wide QRS complexes did the heart rate exceed 100 beats/min. Of 81 ECG strips recording terminations of tachycardia, 45 had two or more wide QRS complexes.

The frequency of a wide QRS complex in each procedure is shown in **Table 2**. Of 22 episodes terminated by intravenous administration of ATP, 16 episodes (72.7%) showed one or more wide QRS complex. As **Fig. 2** shows, the induction of wide QRS complexes by ATP was the most frequent, and was followed by that by esophageal pacing (56.5%), calcium antagonists (51.0%), and vagal maneuvers (50.0%). The development of wide QRS complexes was less frequent, however, after the use of intravenous Class Ia, and Ic antiarrhythmic agents (19.1%, 25.0%). In relation to the mechanisms of

Table 1 Characteristics of patients

	WQC (−) (N = 63)	WQC (+) (N = 61)	
Age, y	49 ± 14	51 ± 15	NS †
Sex, M/F	40/23	43/18	NS ‡
Underlyineg cardiovascular disease	9	14	NS ‡
cardiomyopathy	0	2	
ischemic heart disease	5	4	
hypertension	3	3	
valvular disease	1	4	
sick sinus syndrome	0	2	
others	1	1	
Mechanism of tachycardia			
AVNRT *	25	27	NS ‡
AVRT **	33	31	NS ‡
IART ***	2	1	
unknown	3	2	

WQC: wide QRS complexes.
AVNRT *: strioventricular nodal reentrant tachycardia
AVRT **: atrioventricular reentrant tachycardia
IART ***: intra-atrial reentrant tachycardia
†: t test, ‡: chi-square test
Plus-minus values are means ± SD

Table 2 Drugs and interventions for the termination of tachycardia

	ECG	WQC (−)	WQC (+)
Class Ia	47	38 (80.9%)	9 (19.1%)
aprindine	3	2 (66.7%)	1 (33.3%)
disopyramide	40	33 (82.5%)	7 (22.5%)
procainamide	4	3 (75.0%)	1 (25.0%)
Class Ic	8	6 (75.0%)	2 (25.0%)
flecainide	4	2 (50.0%)	2 (0.0%)
pilsicainide	3	3 (100%)	0
propafenone	1	1 (100%)	0
Ca antagonist	49	24 (49.0%)	25 (51.0%)
diltiazem	4	2 (50.0%)	2 (50.0%)
garapamil	2	0	2 (100%)
verapamil	43	22 (51.2%)	21 (48.8%)
ATP	22	6 (27.3%)	16 (72.7%)
Vagal maneuver	32	16 (50.0%)	16 (50.0%)
Ascher's reflex	17	6 (35.3%)	11 (64.7%)
carotid massage	3	3 (100%)	0
cold water	2	1 (50.0%)	1 (50.0%)
diving reflex	1	0	1 (100%)
Valsalva maneuver	9	6 (66.7%)	3 (33.3%)
Esophageal pacing	23	10 (43.5%)	13 (56.5%)

WQC: wide QRS complexes.

tachycardia, the ECG recorded wide QRS complexes were 44.7% (34/76) in 27 patients with atrioventricular node reentrant tachycardia, 45.7% (42/92) in 31 patients with atrioventricular reciprocating tachycardia, and 50.0% (3/6) in 2 patients with intra-atrial reentrant tachycardia.

Type of the wide QRS complex

Type A wide QRS was recorded in only 3 strips (3.7%), Type B was recorded in 44 (54.3%) and Type C was recorded in 45 (55.6%). The frequencies of each type of wide QRS complex in various termination methods are represented in **Fig. 3**. Type B and Type C was common wide QRS complexes caused by a calcium antagonist. There were 11 (24.4%) episodes of Type C accompanied by Type B wide QRS in this series of ECG strips. Among the 3 types of wide QRS complexes, there were no significant differences in the patients' backgrounds.

Electrocardiographic parameters

In the 81 strips in which wide QRS complexes were present, the Max RR interval was longer than in cases without wide QRS (1,727 ± 1,748 msec vs. 979 ± 331 msec: P = 0.00027). Among the 81 termination episodes accompanied by wide QRS complexes, Max RR was 787 ± 367 msec in the case of Type A wide QRS complexes, 1,535 ± 1,199 msec in Type B, and

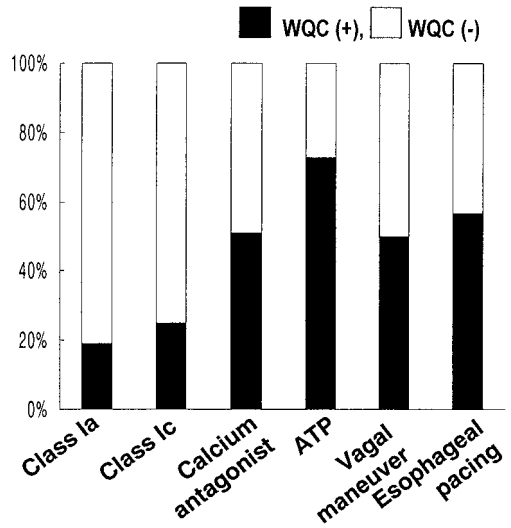

Fig. 2 Frequency of wide QRS complexes in each procedure.
Bar graphs indicate the percentage of trial induced of wide QRS.

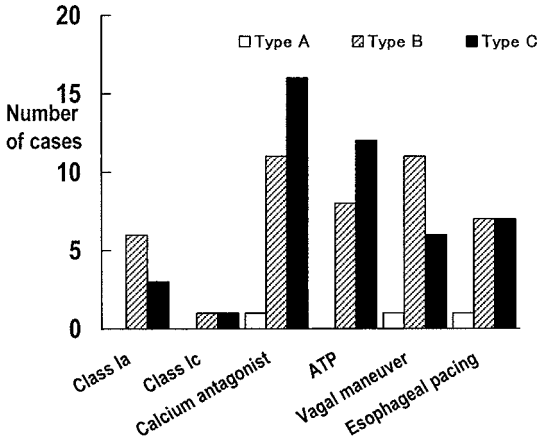

Fig. 3 Frequency of each type of wide QRS complex.
Bar graphs indicate the actual number of trial induced of wide QRS.

1921±2031 msec in Type C. The Max RR with Type B and Type C wide QRS complexes was significantly longer than with Type A ($p=0.037$, $p=0.0075$). Furthermore, Max RRs occurring after intravenous injection of ATP or vagal maneuvers were significantly greater than those after other procedures ($p<0.05$, $p<0.05$ respectively).

The mean Prec RR was 353±31 msec in Type A wide QRS complexes, 1070±1010 msec in Type B, and 616±564 msec in Type C. In Type B and Type C wide QRS complexes, it was significantly longer than that in Type A ($p<0.001$, $p<0.005$). Furthermore, in the case of intravenous ATP, Prec RR was significantly longer than with calcium antagonist ($p=0.01$) and transesophageal pacing ($p=0.002$).

The analysis of the RP interval excluded reversion due to transesophageal pacing, because of the difficulty of recognizing the P wave during pacing. The mean RP interval was 1,452±1,577 msec in cases of wide QRS complexes, and 874±451 msec without wide QRS complexes ($p=0.0024$).

The relationships between the Prec RR interval and the Max RR interval in Types B and C are shown in **Fig. 4**. About half (20/44; 45.5%) of the Type B wide QRS complexes are in a line at an angle of 45° to the axes, but only four (4/45; 8.9%) Type C wide QRS complexes are to be found there. The relationship between the Prec RR interval and the RP interval in Type B wide QRS complexes is shown in **Fig. 5**. Of Type B, 75.7% (28/37) were almost on the 45-degree line. But of Type C, only 18.4% (7/38) were to be found there.

There were a total of 41 wide QRS complexes (44.6%) which had a preceding sinus P wave, out of a total of 92 wide QRS complexes in all three types. These 41 wide QRS complexes included 30/44 (68.2%) Type B wide QRS, and 11 (24.4%) Type C wide QRS complexes. The mean PR interval was 152±34 msec in Type B, and 156±33 msec in Type C. The frequency of the wide QRS complex accompanied by a preceding sinus P wave was highest after termination by the Valsalva maneuver (3/4; 75.0%), which was followed in frequency by intravenous ATP administration (30/62; 48.4%), disopyramide (7/16; 43.8%) and verapamil (15/44; 34.1%). In each procedure, Type B wide QRS complexes tended to have a preceding sinus P wave.

Successive wide QRS complexes were demonstrated in a total of 36 strips. These were seen most frequently with intravenous ATP (54.5%), vagal maneuvers (21.9%), and transesophageal pacing (21.7%).

Reproducibility

Finally, duplication analysis was made in a total of

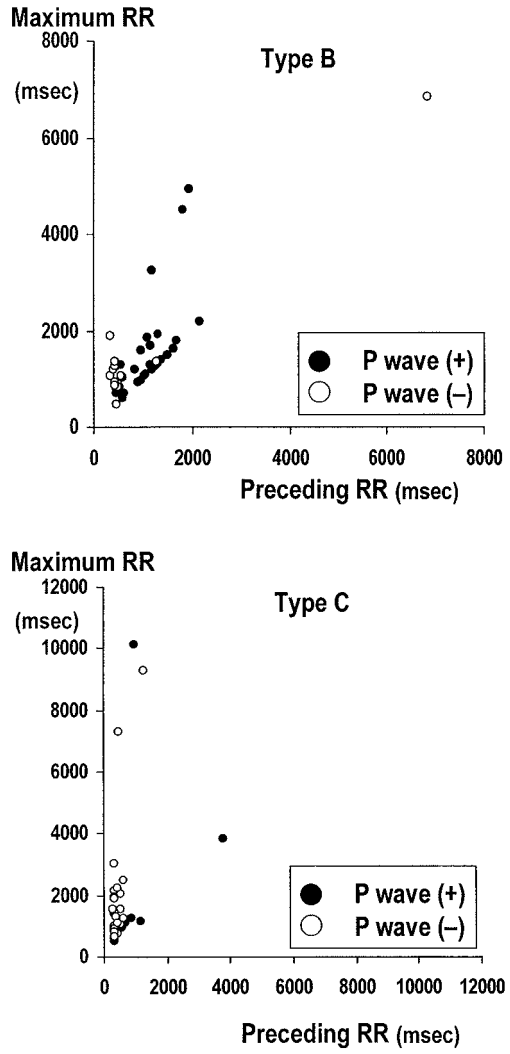

Fig. 4 Relationship between preceding RR interval and maximum RR interval in Type B and C.

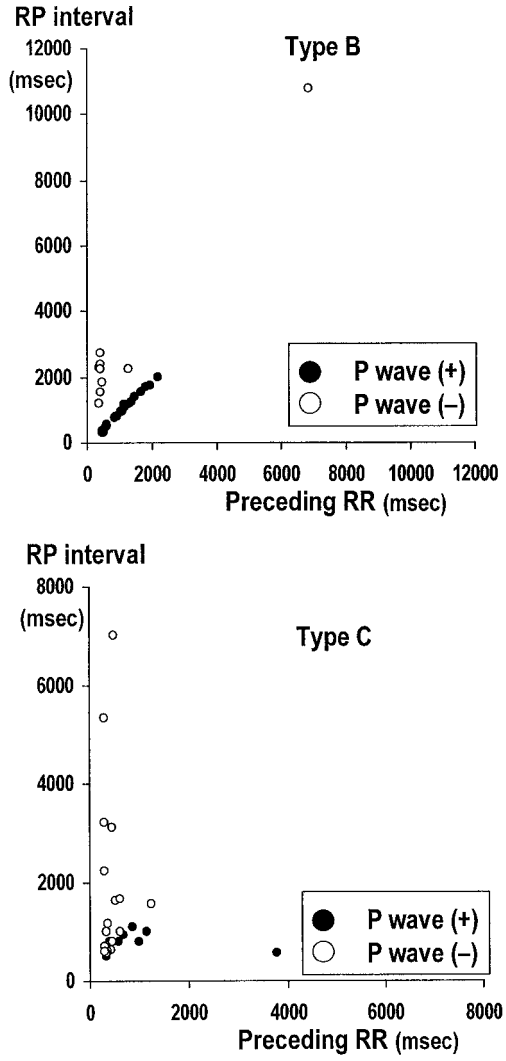

Fig. 5 Relationship between preceding RR interval and RP interval in Type B and C.

125 ECGs; 23 ECGs from 8 patients with ATP, 17 from 5 patients with disopyramide, 48 from 10 patients with verapamil, and 37 from 8 patients with esophageal pacing (**Fig. 6**). The average number of repeated trials was 4.3±4.0 times (range 2 to 17) for each procedure in each patient. The wide QRS complex developed most frequently with intravenous ATP, and had a high total frequency of 21/23 (91.3%), and the average reproducibility for each of seven patients was 93.3% (66.7% to 100%). In verapamil and esophageal pacing, the reproducibility in each patient varied from 0 to 93.8% (15/16). In the case of intravenous disopyramide, only two patients out of five showed the wide QRS complexes and reproducibility was the lowest in these four interventions.

Discussion

For the termination of various atrial tachyarrhythmias such as paroxysmal atrial tachycardia and paroxysmal atrial fibrillation, many kinds of antiarrhythmic agent are administered intravenously. But a wide QRS complex is rarely observed at the termination of tachycardia except in cases of the PSVT. In this study, we found a high frequency of wide QRS complexes at the reversion of PSVT: in 51.1% of all 305 strips, and in 44.8% of 181 strips from 61 patients. The wide QRS complexes were induced most frequently by intravenous ATP, followed, in decreasing frequency, by esophageal pacing, calcium antagonist administration, vagal

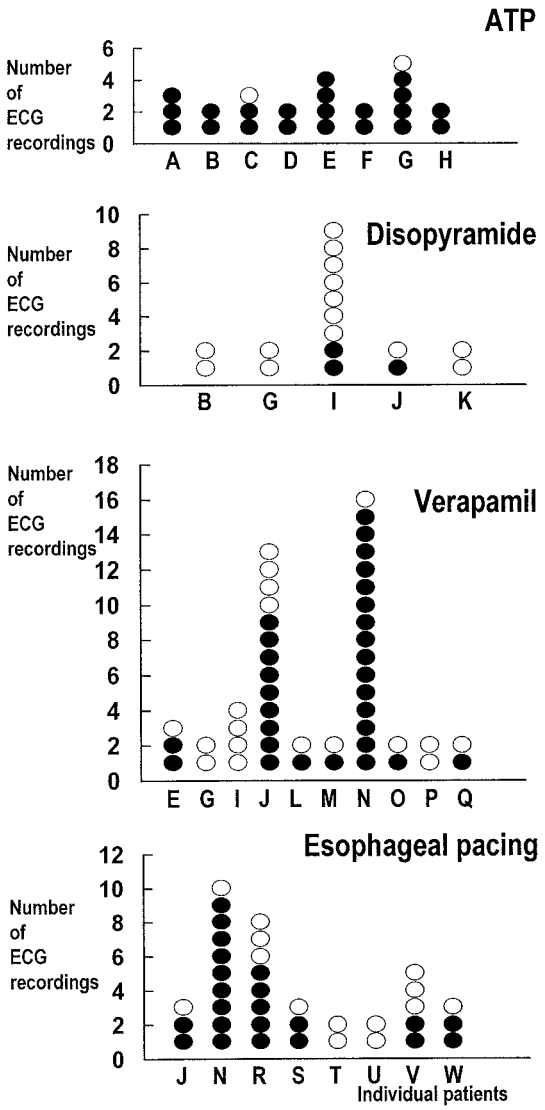

Fig. 6 Reproducibility of wide QRS complex. Open circles: ECG recording without wide QRS complexes. Closed circles: ECG recording with wide QRS complexes. Alphabetical letter indicates the individual patient.

maneuvers, and less frequently by administration of class Ia or Ic antiarrhythmic drugs.

Type of wide QRS complex and their mechanism

To elucidate the underlying mechanism, we divided the wide QRS complexes into three types. In our series of patients, wide QRS complexes of Types B and C were more frequent than those of Type A. Type B and C have a longer Max RR, and preceding RR than Type A. The wide QRS complex tends to develop with a longer Max RR, preceding RR and RP interval. Also, the frequency of wide QRS complexes did not depend on the type of PSVT.

The development of type A wide QRS complexes was the most unusual phenomenon. Type A wide QRS complexes were observed in only three trials. Vohra[1] was the first to report this type of wide QRS complex, and later Hamer[5] et al described the spontaneous development of ventricular complexes at the termination of PSVT in an electrophysiological study. In their cases, following intravenous verapamil administration, Type A wide QRS complexes developed and the PSVT was terminated following the retrograde atrial complex. They concluded that the underlying mechanism of a Type A wide QRS complex may be ventricular early depolarization, because of its electrocardiographic manifestations and its electrophysiological findings. In this study, Type A wide QRS complexes were induced at the termination by intravenous verapamil, and esophageal pacing. But the causal relationship with verapamil is still unknown.

More than half of Type B wide QRS complexes have a preceding sinus P wave. In 45.5% of Type B and 8.9% of Type C, the Prec RR intervals were almost equal to Max RR. In 75.5% of Type B and 18.4% of Type C, the Prec RR intervals were almost equal to the RP intervals. Since some wide QRS complexes had no preceding sinus P waves, but followed long pauses, it was speculated that their mechanism was escaped beats. When the wide QRS complexes had a preceding sinus P wave, intraventricular aberrant conduction was strongly suggested. The preceding PR intervals in these Type B and C wide QRS complexes were consistent with normal sinus PR intervals, but some of them may be fusions of complexes of ventricular origin and propagated sinus origin narrow QRS complexes. An RP interval that was equal to the preceding RR in Type B or Type C may also represent the sinus node recovery time. If the development of a Type B or C complex is related to a longer sinus node recovery time, increased vagal tone may contribute to the development of these types of wide QRS complex. But some Type B complexes were not accompanied by a P wave in a shorter Prec RR, and so did not indicate aberrant conduction. These may be ventricular complexes due to enhanced abnormal

automaticity. The wide QRS complexes with longer Prec RR in both Type B and C tend to have preceding P waves, but those with shorter Prec RR intervals do not. The wide QRS complex with a shorter Prec RR in Types B and C was not accompanied by a P wave, which may also indicate ventricular origin. These QRS complexes may be diagnosed as ventricular premature complexes.

Wide QRS complex and interventions

In the present study, a wide QRS complex developed frequently at terminations resulting from ATP treatment, esophageal pacing and a calcium antagonist administration. Recently, both intravenous adenosine administration and combined intravenous administration of isoproterenol and verapamil brought about clear manifestation of the conduction via a latent accessory pathway[18]. These drugs selectively suppress the atrioventricular conduction system. If these patients have an accessory pathway, it may manifest on the electrocardiogram. Also, it is known that esophageal pacing induces unmasking of the latent preexcitation[19]. On the other hand, intravenous administration of disopyramide mainly suppresses accessory pathway conduction[20] and may accelerate atrioventricular conduction by its anticholinergic action[21]. In this study, a wide QRS complex developed less frequently after termination with disopyramide and other class Ia or Ic antiarrhythmic drugs. If these patients had an accessory pathway, wide QRS complexes may have developed when atrioventricular conduction via an atrioventricular node was selectively suppressed without obvious effect on the accessory pathway. In this study, the type of PSVT did not seem to play a role in the development of wide QRS complexes. Therefore, observed wide QRS complexes in this study may not relate with the electrical potential of the accessory pathways.

Our results indicated that wide QRS complexes tend to develop after a long diastolic interval. This may be explained by the contribution of increased vagal tone to the development of a wide QRS complex (if indeed such contribution occurs), and also by the longer diastolic intervals that result from activation of a stretch-mediated calcium[22] or sodium channel[23]. Nearly half a century ago, it was reported that vagal stimulation such as carotid sinus massage occasionally leads to the appearance of extra systoles, which are thought to be of ventricular origin[14–16]. Furthermore, it was also reported that, experimentally, stretch increases cellular excitability, which makes the heart prone to ectopic activity[24]. However, we could not speculate on the more detailed underlying mechanisms concerned with the development of wide QRS complexes.

Finally, it seems important to evaluate the reproducibility of this arrhythmogenicity. In our 24 patients, multiple ECGs were recorded by the same method. The levels of reproducibility with intravenous ATP, verapamil and esophageal pacing were high, as was the reproducibility of negative results with disopyramide. From these observations, the development of a wide QRS is not accidental and indicates that some underlying mechanisms spontaneously provoke such a complex in those circumstances. The procedure that provoked wide QRS complexes with a high level of reproducibility enhanced vagal activity. This supported the concept that the wide QRS complex was induced by the enhancement of vagal tone or vagotonia.

Limitations of the study

In the present study, various mechanisms for wide QRS complexes after the termination of PSVT were proposed. But these phenomena were studied retrospectively and were observed using three-lead recording from body surface without intracardiac information, so no hemodynamic or electrophysiologic information on the development with wide QRS complexes was obtained, nor was the concentration of the antiarrhythmic agent at the termination of tachycardia evaluated. Further studies are required to elucidate the precise mechanism of development of wide QRS complexes, and, in particular, detailed electrophysiological study should provide more useful information. In electrophysiological study, observation of the spontaneous rhythm occurring after successful catheter ablation may provide further information.

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