The Role of Premature Beats in the Initiation and the Termination of Supraventricular Tachycardia in the Wolff-Parkinson-White Syndrome

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SUMMARY
In four patients with WPW syndrome atrial and ventricular premature beats were induced and the changes in form of the ventricular and atrial complexes were studied. Results indicate that, depending upon the timing of the premature atrial beat and the state of refractoriness of the His and Kent bundles, excitation of the ventricles occurs predominantly through the atroventricular nodal system, predominantly through the Kent bundle or exclusively through one or both conduction systems. With short delays conduction through the Kent bundle may be blocked and only normal excitation of the ventricles occurs. In one patient with a history of attacks of tachycardia these normal QRS complexes were followed by retrograde activation of the atria by the Kent bundle, and attacks of supraventricular tachycardia of shorter or larger duration occurred. They stopped spontaneously, sometimes by delay or block, either of retrograde Kent conduction or of antegrade A-V nodal conduction, making it possible for the sinus node to capture the ventricles. They also could be terminated by induced atrial premature beats.

In two patients tachycardias could be induced by appropriately timed ventricular premature beats during regular driving of the right ventricle. In one of these patients a circus movement, involving the Kent bundle, is probably present. By appropriate stimulation of the atria or ventricles during an attack of supraventricular tachycardia in this patient, one cycle length could be shortened without changing those of the following beats. These results suggest that a circus movement involving the atria, the normal atrioventricular conduction system and the Kent bundle is present. In the other patient, not fulfilling the WPW criteria, ventricular or atrial premature beats did not interfere with the basic rhythm of the tachycardia. Two hypotheses for this tachycardia are given: nodal tachycardia caused by rapid firing of the A-V node or a nodal tachycardia caused by a reciprocal mechanism in the A-V junction. The attacks could be blocked too by appropriately timed atrial and ventricular premature beats. No ventricular type of tachycardia could be demonstrated.

Additional Indexing Words:
- Atrioventricular nodal tachycardia
- Circus movement
- Clinical stimulator
- Kent conduction
- Reciprocal rhythm
- Ventricular tachycardia
- Atrioventricular conduction

The Wolff-Parkinson-White (WPW) syndrome is associated in a fairly large percentage of cases with attacks of supraventricular tachycardia. These attacks can last...

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SUPRAVENTRICULAR TACHYCARDIA IN WPW SYNDROME

A. normal human heart

B. W.P.W. (type B) heart

C. supraventricular tachycardia

D. ventricular tachycardia

Figure 1

Schematic representation of excitation pattern in the normal heart and in the WPW syndrome, type B. The beginning of the P wave is taken as the zero point; isochrones are depicted. The figures indicate milliseconds. (A) Normal pattern.

(B) Antegrade Kent conduction time is assumed to be 40 msec.

(C) During SVT the zero point is the beginning of excitation of the ventricles. The atria are activated retrogradely by way of the Kent bundle, the retrograde Kent conduction time is taken to be 40 msec. The A-V node is activated at about 150 msec, and normal conduction occurs.

(D) During ventricular tachycardia the beginning of ventricular depolarization by way of the anomalous bundle is taken as the zero point. The A-V node is activated about 100 msec later. A delay of 100 msec in retrograde A-V conduction results in the beginning of atrial depolarization at about 200 msec.

The figures indicated in all schematic representations are hypothetical.

very long, can damage the heart, and may greatly influence the social status of the pa-
tient. The demonstration of an anomalous excitation of the right ventricle in a patient
with WPW syndrome, type B (fig. 1B) pro-
ved the thought that a mechanism respon-
sible for the attacks of tachycardia involving
the Kent bundle might have surgical con-
sequences. This study was performed after
developing a safe and very accurate stimu-
lar, acceptable for clinical use.

De Boer in 19267 postulated that a circus
movement of the excitatory wave, made pos-
sible by the presence of an anomalous path-
way connecting adjacent parts of the atria
and the ventricles, is responsible for attacks
of supraventricular tachycardia (SVT). If
antegrade conduction is only possible in
the normal atrioventricular conduction system,
because of a temporary state of refractoriness
in the Kent bundle, normal excitation of
the ventricles will occur with activation of the
basal part of the ventricles at a time when
the Kent bundle is not refractory any longer,
and this will be followed by activation of the
atria from the atrial end of the Kent bundle
(fig. 1C). When the atrioventricular node is
activated again, this sequence of excitation is
repeated, with resulting tachycardia.

Experimental evidence for this hypothesis
was already given by Butterworth and Poin-
dexter9 in the cat heart, where they produced
a tachycardia by retrograde stimulation of
the auricle by the amplified QRS current.

Clinical arguments in favor of retrograde
conduction via accessory pathway during
SVT were reported by Wolff8 and Harnisch-
feger.9 Penetration into the anomalous bundle
was described by Langendorff and asso-
ciates10 in a patient with atrial fibrillation
and the WPW syndrome.

Certain deductions from these concepts can
be tested with induced atrial and ventricular
beats during sinus rhythm and paroxysmal
tachycardia. If one considers the QRS com-
plexes in WPW syndrome to be fusion beats,
the changes in configuration and duration of
the QRS complex and of the PQ interval,
following induced atrial premature beats with
varying delay, indicate roughly the degree of
fusion of normal and anomalous excitation.
A QRS complex with a normal configuration and a duration of 0.10 sec, following an atrial premature beat at a PQ interval of 0.20 sec or longer, indicates exclusive conduction through the normal atroventricular conduction system (fig. 1A). A ventricular complex, following an atrial premature beat at 0.12 sec or shorter, with a QRS duration of 0.12 sec or slightly more and pre-excitation configuration, is caused by partial excitation of the ventricles through the Kent bundle. If the PJ time of this fusion complex is about the same as that present during sinus rhythm, excitation of a part of the ventricles through the normal atroventricular conduction system is present. With longer duration of the PJ and QRS intervals and concomitant increased deformation of the QRS complex, predominant or even exclusive excitation of the ventricles by way of the Kent bundle must be assumed.

Attacks of SVT may be initiated by an atrial or ventricular premature beat, if differences in the state of refractoriness of both bundles are present. The atrial premature beat will have to be blocked in the Kent bundle but conducted to the ventricles through the normal atroventricular conduction system (fig. 1C). If a ventricular premature beat could initiate an attack of SVT, conduction in the Kent bundle from ventricles to atria has to be present, and the atroventricular conduction system must be refractory temporarily, with the refractoriness disappearing when the retrograde atrial impulse reaches the atrioventricular node. No such attack will occur, if retrograde Kent conduction is blocked.

For a ventricular type of tachycardia—if possible at all in the WPW syndrome (fig. 1D)—the postulated pathway of excitation during the initiating atrial or ventricular beat, as outlined above, has to be reversed. In this instance an atrial beat has to be blocked in or near the atrioventricular node, but conducted to the ventricles through the Kent bundle, with retrograde activation of the atroventricular conduction system, and atrial activation by way of the atrioventricular node. For a ventricular premature complex to initiate a circus movement of this type, an essential condition is the presence of retrograde block in the Kent bundle during this beat.

Some consequences of the circus-movement theory have to be described. During tachycardia, caused by a circus movement as described, stimulation of nonrefractory parts of ventricles or atria, awaiting the arrival of the excitatory wave, can shorten the duration of one cycle, whereas the cycle length of the following beats is unchanged.

According to Wenckebach and Winterberg no circus movement can be present when an induced premature beat is followed by a compensatory pause. There is an analogy between the circus movement we postulate along two anatomically separate pathways and the reciprocal rhythm during functional dissociation of two pathways within the A-V junction. Moe and Mendez demonstrated that in a nodal tachycardia, caused by the latter mechanism within the A-V junction, the pause following a premature response may be fully compensatory and, therefore, this finding does not constitute evidence against a reciprocal mechanism of this type.

**Methods**

In essence the same methods were used as previously published for the determination of the excitability of the heart in dogs.

A current stimulator, able to deliver driving and testing stimuli, which could be varied in strength and duration, was designed. By use of a transformer the current pulses are separated from ground, preventing unwanted dangerous interference from the mains. In the design special caution was taken to prevent spurious pulses due to disturbances in the mains or the ground, including switching on and off the apparatus.

Two independent stimuli could be used separately or combined. The strength of the square wave pulses is adjustable from 25 µamp to 30 mA, with a duration of 1, 2, 3, 4, 8, or 16 msec.

Because the internal resistance of the stimulator is very high, the resistance of the tissue and the electrode has only a negligible influence upon the strength of the testing stimulus. The test-pulse can be presented after a chosen delay, accurate to 1 msec. It can be applied after each beat, each second beat, and so on, up to once for each 16 beats. The delay is started by the driving stimulus or by the electric activity of the heart; in
the present study it was the intra-atrial or intra-ventricular depolarization complex. There is a blocking circuit in the synchronizer, limiting the frequency of the test pulses. For the study of antegrade and retrograde conduction in the Kent or the His bundle, stimuli were often delivered through a catheter with two electrodes, situated at a distance of 1 cm, introduced into the right atrial or ventricular cavities, and placed against the endocardial surface. In all instances test stimuli with the longest delay were applied first, and then the delay was gradually decreased.

An attempt was made to drive the atria regularly by delivering driving stimuli of 2-msec duration and a strength of 1 ma to the right atrium or right ventricle. Sometimes this was not possible because of rapid spontaneous sinus rhythm.

A direct writing Elema 8-channel electrocardiograph with a high frequency range was used for recording. In all cases the three extremity leads joins together with a unipolar right intra-atrial or intraventricular lead were recorded.

For reasons of presentation the duration of the QRS complexes are often expressed in milliseconds, without suggesting an accuracy of this degree.

Case Histories

Patient A

This patient, a truck driver, 43 years old, had attacks of stabbing, nonradiating precordial pain, lasting from 1 hour to 1 day. The pain was not related to exercise. These attacks started 1 year prior to admission. The patient denied ever having had palpitations. The ECG showed a sinus rhythm with a rate of 70/min and a WPW syndrome, type A (QRS duration, 0.14 sec).

Patient B

A 20-year-old soldier was referred to our department because of attacks of palpitation and dyspnea on exertion. The ECG showed a sinus rhythm with WPW syndrome, type B18 (QRS duration, 0.14 sec).

Patient C

This woman, a 47-year-old school teacher, had attacks of palpitations for 4 years, proven to be attacks of SVT. Since the frequency and duration of the attacks had increased considerably in the last half year, despite medical treatment, she was referred to our department. The electrocardiograms, made during this admission, showed attacks of SVT or a sinus rhythm with either a WPW syndrome, type B, or a complete right bundle-branch block. In the absence of pre-excitation the P-R interval was 0.15 sec. Sometimes pre-excitation was present in alternating beats. The SVT had a rate of 165/min.

Patient D

This 15-year-old girl had had attacks of palpitation during the last 8 years, the frequency of which had gradually increased in the last years. Prior to admission she had one or two attacks a month, lasting from 2 hours to 2 days, during which she was short of breath and felt nauseated. She was admitted for the first time during a tachycardia, that had started 2 hours earlier.

On physical examination the blood pressure was 100/80 mm Hg. She had a regular pulse of about 200/min. The venous pressure was not elevated. Apart from the rapid, regular heart action of 200/min, the examination disclosed normal conditions. The ECG on admission showed a regular SVT (rate, 210/min) with a QRS duration of 0.08 sec; no P waves could be identified.

Figure 2

Patient A. Lead I. Sinus rhythm with rate of 60 to 65/min. Induced right atrial premature beats with decreasing delay after each fourth sinus P wave. The QRS complex of the premature beat shows a gradually more pronounced pre-excitation pattern with delays decreasing to 366 msec. At a delay of 365 msec the atrial premature beat is followed by a QRS complex with a normal configuration, a duration of 100 msec, and a PQ interval of 260 msec increasing to 290 msec at a delay of 300 msec. At delays of 295 msec or less, atrial depolarization could only be provoked by a tenfold increase in current strength.
Immediately after admission venous catheterization was performed, the results of which and those of the subsequent one will be described later. The ECG during sinus rhythm had a frequency of 60/min with a PQ interval of 0.11 sec, a QRS duration of 0.10 sec with delta waves in several leads, and a short PJ time of 0.21 sec. The complexes resembled those found in the WPW syndrome, type A.\textsuperscript{16}

Results

Induced Atrial Premature Beats During Sinus Rhythm or Regular Driving of the Atria

Patient A

During spontaneous sinus rhythm, varying between 60 and 65/min, after every 4 beats one testing stimulus was delivered to the right atrial muscle (fig. 2). With a delay of 700 msec slight changes in the form of the terminal part of the QRS complex occurred, the initial part remaining the same. The QRS duration increased from 140 msec during regular sinus rhythm, to 150 msec in the complex following the test-impulse of 700-msec delay, and to 180 msec when the delay was 366 msec. The values for the duration of the PJ time of the ventricular premature complex after different delays varied. It had a constant value of 240 msec up to a delay of 450 msec, increased to 260 msec at a delay of 400 msec, and reached 300 msec at a delay of 370 msec. At a delay of 365 msec the atrial premature beat was followed by a normal QRS complex (duration, 100 msec), and a PQ time of 260 msec, increasing to 290 msec, with a delay of 300 msec. Decrease of the delay to 295 msec, with constant strength and duration of the testing impulse, did not give rise to atrial depolarization any more, which, however, occurred if the current strength was increased to 30 ma. At 255 msec with a current strength of 30 ma the P wave had a duration of 160 msec; the PQ time was 230 msec. These figures are not very accurate, because the beginning of atrial depolarization after the testing stimuli cannot be ascertained accurately.

Patient B

The atria were stimulated with a frequency of 113/min (the basic cycle length was 530 msec) with a bipolar stimulating electrode lo-

**Figure 3**

*Patient B. Regular driving of the right atrium with a basic cycle length (B.C.L.) of 530 msec. Induced right atrial premature beat with a delay of 300 msec. The QRS complex of the premature beat shows a more pronounced pre-excitation pattern than the regularly driven beats.*

**Figure 4**

*Patient B. Regular driving of the right atrium with a basic cycle length of 530 msec. Induced atrial premature beat with a delay of 280 msec. The QRS complex of the premature beat shows no pre-excitation and has a width of 80 msec, and a PQ interval of 250 msec. Therefore, only His conduction is present in this complex.*
cated high in the right atrial cavity. The delay of the testing stimuli was varied from 400 to 280 msec. They were followed by WPW complexes (fig. 3) with increase in duration of the QRS complex up to 160 msec, in the range of 400 to 290 msec. With a delay of 280 msec (fig. 4) normal QRS complexes, with a duration of 80 msec, followed the premature atrial depolarization (PQ time, 250 msec; artefact-J time, 330 msec). A very interesting phenomenon often happened: spontaneous P waves, with a duration of 50 msec, followed the normal QRS complex (PQ time, 250 msec; artefact-J time, 330 msec).

**Figure 5**

Patient B. Regular driving of the right atrium with a basic cycle length of 530 msec. Induced atrial premature beat with a delay of 280 msec. The QRS complex of the premature beat has the same configuration and timing as the premature beat shown in figure 4, but now it is followed 20 msec after the end of the QRS complex by a P wave (indicated as d), which is negative in leads II and III and is caused by retrograde Kent (R.K.) conduction.

**Figure 6**

Patient B. Regular driving of the right atrium with a basic cycle length of 530 msec. Induced atrial premature beat with a delay of 278 msec. The QRS complex of the premature beat has the same configuration and timing as the premature beats shown in figures 4 and 5. Like the sequence shown in figure 5, it is followed 20 msec after the end of the QRS complex by a P wave, which in its turn is followed now at 180 msec by a QRS complex without pre-excitation pattern, but possibly with right ventricular conduction delay (QRS width, 110 msec). This beat is the first complex of a regular supraventricular tachycardia (rate, 220/min), which after a short while ends spontaneously, the last QRS complex not being followed by a P wave.
caused by a right ventricular conduction delay. No appreciable changes in length of the time interval between the end of QRS of these complexes and the beginning of the retrograde P wave, which was often highly notched in the intra-atrial electrocardiogram were found. These measurements are inaccurate, however, because both the end of QRS and the beginning of the P wave could not be identified exactly.

**Patient C**

A rapid sinus rhythm was present. The driving stimuli, which had to be of a higher frequency than the sinus rhythm, did result in multiple premature beats, and attacks of tachycardia occurred frequently.

**Patient D**

In this patient also it was difficult to obtain the relevant data, because of the frequent occurrence of attacks of SVT during regular driving of the atria. The intrinsic deflection of the intracavitary atrial complex, present during sinus rhythm varying in rate between 73 and 81/min, was used for the triggering of the delay circuit for the testing stimulus, given after each 8 sinus beats. The delay varied between 450 and 270 msec. In figure 7 the changes in form of the WPW complexes, caused by the testing stimuli, are shown; a gradual increase in aberrant excitation of the ventricles may be noted. The PJ time was 230 msec until a delay of 390 msec, 250 msec in the interval from 390 to 330 msec, and reached 260 msec at a delay of 290 msec. At a delay of 270 msec SVT repeatedly occurred (figs. 7 and 8). The induced premature atrial complex was followed after 280 msec by a QRS complex without any pre-excitation, probably showing right ventricular conduction delay (QRS duration, 110 msec); this is the first complex of the always occurring SVT.

**Induced Right Ventricular Premature Beats During Regular Driving of the Right Ventricle**

**Patient A**

Stimuli of 90/min, with the same duration and strength as used during atrial stimulation, were delivered to the ventricles. No retrograde conduction of the atria occurred, and the form and polarity of the P waves remained the same as those present during normal sinus rhythm.

**Patient B**

Driving stimuli were delivered to the right ventricle with a frequency of 113/min (strength, 1.5 ma; duration, 2 msec).

The ventricular complexes (duration, 140 msec) were followed by P waves, which were negative in leads II and III. The Q-P time,
measured as the interval between the beginning of the QRS complex and the beginning of the P wave, was 140 msec. Testing stimuli were delivered to the right ventricle with gradual decrease in delay from 350 to 264 msec. This resulted in a gradual increase in Q-P time without initiating a tachycardia. Testing stimuli with a delay of 266 msec or less were not followed by retrograde P waves.

An unusual P wave was recorded during massage of the right carotid sinus, when the delay was 264 msec (fig. 9). A notched negative P wave of nearly the same form as that present during the SVT, but broader (90 msec), was recorded in the intra-atrial complex, occurring 60 msec after the end of the ventricular complex. This P wave was not clearly visible in the extremity leads. It was followed after 160 msec by a QRS complex of normal configuration, after which regular ventricular stimulation was resumed.

Patient C

The right ventricle was driven with a regular rate of 102/min. Retrograde P waves were visible 200 msec after the beginning of the QRS complexes. Right ventricular premature beats with a delay down to 320 msec were followed by P waves with the same Q-P interval, without appreciable changes in configuration, compared to that with the complexes of the basal rhythm. At a delay of 320 msec these P waves were followed repeatedly by SVT and QRS complexes, suggesting a right ventricular conduction delay (fig. 10).

Patient D

Driving stimuli (3 ma, 2 msec) were applied to the endocardial surface of the right ventricle at a rate of 80/min, and testing stimuli with a delay varying from 600 to 360 msec were given after each 8th beat (fig. 11). The regularly driven ventricular beats were followed about 140 msec after the beginning of the QRS complex by P waves, which were negative in leads II and III. After an induced ventricular premature beat, caused by a stimulus with a delay of 365 msec, an attack of SVT (rate, 230/min) occurred in which the P waves did not clearly differ from those recorded during right ventricular stimulation, and
the QRS complexes had a normal configuration with a duration of 0.08 sec.

**Induced Atrial Premature Beats During Supraventricular Tachycardia**

Patient A did not have attacks of tachycardia. In patient B stimuli were delivered to the atria. This could not be done in a systematic way, because of the short duration of the tachycardia. Therefore, regularly spaced stimuli were applied to the right atrium at a slower rate than the rate of the SVT. Termination of the SVT in this way is demonstrated in figure 12.

**Patient C**

Testing stimuli, applied to the right atrium during tachycardia, with delays after the foregoing QRS complex short enough to activate the right atrium earlier than the atrial activation expected in the course of the tachycardia, gave rise to shortening of the RR interval of this cycle. The following cycle had again the basic cycle length of the tachycardia. Figure 13 gives an example of this phenomenon, occurring with a delay of 90 msec. The premature atrial activation occurs 100 msec earlier than the expected atrial activation. In another instance (fig. 14) atrial premature beats with the same delay resulted in termination of the tachycardia. Probably the premature atrial activation found the atrioventricular node in its refractory phase.

*Figure 9*

Patient B. Regular driving of the right ventricle with a basic cycle length of 530 msec. Induced right ventricular premature beat after each eighth driving beat, with a delay of 264 msec. Right carotid sinus massage. The regularly driven beats are followed by P waves at about 140 msec after the beginning of the QRS complex. A broad notched P wave (indicated as \( P(K) \)) began 60 msec after the end of the ventricular premature complex (Q-P interval, 220 msec). This P wave is followed after 160 msec by a QRS complex of normal configuration. Therefore the atrial beat, indicated as \( P_{(K)} \), is probably caused by retrograde Kent conduction, a spontaneous atrial beat being unlikely.
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Figure 10

Patient C. Regular driving of the right ventricle with a basic cycle length of 585 msec. Induced ventricular premature beat with a delay of 320 msec. The premature ventricular beat is followed by a P wave with about the same configuration and timing as the P waves following the regularly driven ventricular beats. This P wave is followed by a QRS complex without pre-excitation pattern, but with a (nearly) complete right bundle-branch block (PQ interval, 150 msec). This complex is the first of a regular supraventricular tachycardia.

Patient D

Testing stimuli were applied during tachycardia to the right atrium with gradually decreasing delay. Figure 15 shows what happens with a delay of 340 msec after the beginning of the foregoing QRS complex, 180 msec earlier than the expected arrival of the atrial excitation wave. The first premature complex in this figure does not change the time of occurrence of the following ventricular and atrial complexes. The next premature complex with the same delay results in termination of the SVT.

Induced Ventricular Premature Beats During Supraventricular Tachycardia

Patient A did not have tachycardia, and in patient B ventricular premature beats were not made during SVT.

Patient C

Testing stimuli, applied to the right ventricle with an appropriate delay after the foregoing QRS complex, gave rise to ventricular premature beats, followed immediately by a P wave, which occurred about 130 msec earlier than the P wave expected in the course of the tachycardia and resulted in advancing all subsequent atrial and ventricular complexes to an earlier moment (fig. 16).

Testing stimuli, applied slightly earlier (about 10 msec), terminated the tachycardia, the retrograde activation of the atria being blocked (fig. 17).

Patient D

Premature stimulation of the right ventricle (fig. 18) with a delay of 200 msec did result in
production of tachycardia by a right ventricular premature beat (delay 365 msec)

right atrial cavitary complex

initiation and termination of supraventricular tachycardia during stimulation of right atrium

Figures 11 (Upper) and 12 (Lower)

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Figure 13

Patient C. Supraventricular tachycardia with rate of 150/min. Induced right atrial premature beat with a delay of 90 msec after the beginning of the foregoing QRS complex. The atrial premature beat gives rise to a shortening of the RR cycle length comprising this premature beat. The following RR interval has again the basic length of this tachycardia.

Figure 11

Patient D. Regular driving of the right ventricle with a basic cycle length of 750 msec. Induced right ventricular premature beat with a delay of 365 msec. The regularly driven ventricular complexes are followed by P waves with a Q-P interval of about 120 msec. The premature ventricular beat is followed by a P wave with about the same configuration, but a slightly longer Q-P interval (about 140 msec). After this P wave SVT with QRS complexes lacking pre-excitation is established.

Figure 12

Patient B. Regular driving of the right atrium (basic cycle length, 530 msec). Initiation of SVT by an induced atrial premature beat (delay, 280 msec). Termination of the SVT by induced right atrial depolarization.
supraventricular tachycardia, termination by induced right atrial premature beat

intra-atrial complex coronary sinus

induced right atrial premature beat during tachycardia

Figures 14 (Upper) and 15 (Lower)
Figure 16
Patient C. SVT with rate of 162/min. An induced right ventricular premature beat with a delay of 300 msec after the foregoing QRS complex resulted in shortening of the atrial cycle comprising the premature beat. The following QRS complex occurred correspondingly 50 to 60 msec earlier than expected.

Figure 14
Patient C. SVT with rate of 162/min. An induced right atrial premature beat applied 90 msec after the beginning of the foregoing QRS complex results in termination of the SVT. The premature atrial complex has a configuration, which slightly differs from that of the premature atrial complex shown in figure 13.

Figure 15
Patient D. SVT with rate of 170/min. Induced right atrial premature beats with a delay of 340 msec after the beginning of the foregoing QRS complexes. The first premature atrial beat occurs 180 msec earlier than the expected arrival of the atrial excitation wave but does not change the time of occurrence of the following ventricular and atrial complexes. The second premature atrial beat with the same delay results in termination of the SVT.
Termination of Attacks of Supraventricular Tachycardia

As described, it was possible in most instances of SVT to terminate the attacks by premature atrial and ventricular beats applied at an appropriate time. Spontaneous termination of SVT was often seen, sometimes by blocking or delay of retrograde Kent conduction (fig. 6), sometimes by blocking or delay of antegradde A-V nodal conduction.

Discussion

Patients A, B, and C fulfilled the diagnostic criteria for the WPW syndrome. Patient D had a very short PQ time during sinus rhythm; the duration of the QRS complex however was 0.10 sec despite the presence of a delta wave, and the PJ time was very short. The changes in QRS configuration during induced premature atrial beats (fig. 7) were similar to those found in patient A with typical WPW syndrome, type A (fig. 2), and therefore patient D was included in this study.

Influence of Atrial Premature Beats During Sinus Rhythm

Because heart rate has a very important...
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influence on the duration of the refractory period, it would have been necessary to avoid any possible influence of natural irregularities in cardiac rhythm, to drive the atria at a constant rate. This was often not possible, but the slight variations in sinus rate very probably do not influence the gross changes in form of the QRS complex, following atrial premature beats.

The changes in the QRS complex occurring after induced atrial premature beats indicate that with shortening of the delay an increasing degree of pre-excitation of the ventricle is present. With short delays excitation occurs predominantly by way of the anomalous bundle responsible for the delta wave found during sinus rhythm, because the polarity and configuration of the initial part of these complexes are the same as those of the delta waves in all extremity and intracavitary leads, and the PQ interval is very short (0.12 sec).

In the atrial premature beat with very short delay, slightly longer than the delay that gives rise to exclusive but slowed normal atrioventricular conduction, broad pre-excitation complexes with a short PR interval, indicating exclusive Kent excitation, occur.

Genesis of Atrial Tachycardia after Atrial Premature Beats

The changes in excitation pattern of the heart heralding the onset of SVT in patient B with a history of palpitations are illustrated in figures 3 to 5. The atrial testing stimulus, applied after each 8 beats, beginning with

Figure 18

Patient D. SVT with rate of about 162/min. A stimulus applied to the right ventricle with a delay of 195 msec after the beginning of the foregoing QRS complex did not result in ventricular activation. A stimulus applied with a delay of 200 msec produced a ventricular premature beat which did not give rise to a shift in the basic rhythm of the tachycardia, the pause between the premature and the next ventricular complex being fully compensatory.

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termination of tachycardia by right ventricular premature beat

Figure 19

Patient D. SVT with rate of 162/min. An induced right ventricular premature beat with a delay of 260 msec resulted in termination of the SVT.

a long delay, gradually shortened down to a delay of 280 msec and resulted in increasing anomalous excitation of the ventricles. In most instances at a short delay of 280 msec these atrial beats were followed 240 msec after the beginning of the induced P wave by normal QRS complexes of 0.10-sec duration. Exclusive excitation of the ventricles through the normal atrioventricular conduction system is present. The Kent bundle is blocked. Nearly always P waves, negative in the right intracavitary lead and in leads II and III, became visible 20 to 40 msec after the end of this QRS complex with a form and polarity compatible with atrial excitation due to retrograde Kent conduction. This conclusion is strengthened by the frequent occurrence of normal QRS complexes after these P waves and shorter or longer runs of tachycardia, with about the same time relations between QRS and P as outlined above for the first initiating beat of the SVT. No attacks of tachycardia could be induced in patient A, who did not have a history of paroxysmal VT. In patient D attacks of VT always occurred after the first premature atrial beat, which was followed by a normal QRS complex.

Genesis of Atrial Tachycardia after Ventricular Premature Beats

During regular driving of the right ventricle, retrograde activation of the atria occurred in patients B, C, and D. In patient C premature right ventricular complexes during regular driving of the right ventricle gave rise to
retrograde P waves, which had the same timing and configuration as those of the complexes of the basic rhythm. If the premature stimulus was given in a sharply defined delay interval (about 320 msec), these retrograde P waves were always followed by a SVT with normal excitation of the ventricles (fig. 10). This sequence of events therefore suggests that the atria are activated in a retrograde way through the Kent bundle. There are other arguments also for the presence of circus movement in this patient.

In patient B in only one instance was a broad P wave found after one induced ventricular premature beat with short delay (fig. 9). This P wave was followed by a normal QRS complex, with a PQ interval of 160 msec. This sequence, the time relations, and also the polarity of the intra-atrial and extremity P wave suggest that retrograde Kent conduction is present in the beat indicated as $P_{(K)}$.

Patient D also had attacks of SVT, occurring after a right ventricular premature beat, elicited during regular driving of the right ventricle. Here too the time interval, during which this phenomenon occurred, was narrow. A tentative explanation for the genesis of the tachycardia in this patient will be discussed later.

**Effects of Atrial and Ventricular Premature Beats on Supraventricular Tachycardia**

**Effect on Cycle Length Comprising the Premature Beat**

The circus movement theory prompted us to study the influence of induced atrial and ventricular premature beats during tachycardia. Either shortening of one cycle or interruption of the tachycardia was expected.

In patient C a properly timed ventricular premature beat indeed shortened one atrial cycle length (fig. 16), and a properly timed atrial premature beat shortened one ventricular cycle length (fig. 13). All following cycles are shifted to an earlier time than would be expected without shortening of one cycle by the premature beat. In patient D the premature atrial (fig. 15) and ventricular (fig. 18) complexes did change the time of occurrence of the P wave, but the following pause was fully compensatory; no shift did occur as described above.

According to Wenckebach and Winterberg this is not compatible with a circus movement, comprising an accessory pathway outside the A-V node. It is in agreement with a circus movement, comprising two functional pathways within the A-V node and the atrium, as demonstrated by Moe and Mendez.

**Termination of the Attacks of Supraventricular Tachycardia**

In patient B (fig. 12) a right atrial premature beat, occurring at the right delay, terminated the attack, because retrograde Kent conduction of the ventricular complex was blocked, the atrium being refractory.

In patients C and D both atrial and ventricular premature beats with appropriate timing did the same (figs. 14 and 17 and figs. 15 and 19).

**Genesis of Tachycardia in Patient D**

The premature atrial beat, initiating the SVT, was always followed by a complex without pre-excitation, so that the Kent bundle was blocked. It is possible that, after completion of this nonanomalous ventricular depolarization wave, retrograde conduction in the Kent bundle occurs and that this early atrial excitation initiates a nodal tachycardia. Scherf and associates postulated that an atrial excitatory wave arriving at the atrioventricular nodal centers early in diastole may precipitate rapid firing at this area.

An alternative hypothesis, in view of the long P-R interval (0.25 sec) of the premature atrial beat conducted via the His bundle (and with right bundle-branch block), is the occurrence of an atrial echo rather than retrograde conduction via the Kent bundle, initiating a self-sustained reentry mechanism in the A-V junction, entirely independent of the accessory bundle. In a nodal tachycardia of this type atrial or ventricular premature beats may be followed by a compensatory pause as demonstrated by Moe and Mendez.

**Ventricular Tachycardia (Fig. 1D)**

In no instance was it possible to induce a ventricular type of tachycardia. It must be
pointed out that the same processes, which cause tachycardia, either atrial or ventricular, in the normal heart, can be expected to do so in the WPW heart. The ordinary diagnostic criteria can be applied. The only specific mechanism, inducing ventricular tachycardia unique for the WPW syndrome that we can conjecture at this moment, is a circus movement in the reverse direction compared with the one present in SVT. Basically this is a supraventricular type of tachycardia with exclusive antegrade anomalous A-V conduction and retrograde His conduction. The diagnostic criteria then would be regular broad ventricular complexes of about the same rate as the rate during SVT, with pre-excitation configuration and 1:1 atrioventricular nodal P waves. A survey of the literature gives only one case probably fulfilling these criteria.18

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