Spontaneous Termination of Circus Movement Tachycardia Using an Atrioventricular Accessory Pathway: Incidence, Site of Block and Mechanisms

DAVID L. ROSS, M.B., B.S., JERONIMO FARRE, M.D., FRITS W.H.M. BAR, M.D., EDDY J. VANAGT, M.D., PEDRO BRUGADA, M.D., ISAAC WIENER, M.D., and HEIN J.J. WELLENS, M.D.

SUMMARY The incidence, mechanisms and sites of block of spontaneous termination of circus movement tachycardia (CMT) using an atrioventricular accessory pathway (AP) were analyzed in 24 consecutive patients (17 with Wolff-Parkinson-White syndrome and seven with a concealed AP) who were not receiving anti-arrhythmic drugs. Spontaneous termination of tachycardia occurred in 10 patients (105 episodes). A reduced "safety margin" of tachycardia was the only factor that was significantly more common in the patients who manifested spontaneous termination (p < 0.01). The site of spontaneous block was located in the AP in six patients (50 episodes), atrioventricular node (AVN) in six patients (37 episodes) and His-Parkinje system (HPS) in three patients (18 episodes).

At least 14 mechanisms leading to block in the tachycardia circuit were identified. Labile conduction during tachycardia occurred at multiple sites (AVN, His bundle, bundle branches, and AP). Analysis of the duration of tachycardia before spontaneous termination showed a characteristic time pattern for block at each site, consistent with the autonomic and electrophysiologic changes that occur after induction of tachycardia.

Spontaneous termination of CMT using an AP is a common phenomenon. Many mechanisms are involved, which are often complex and dependent on interplay of the electrophysiologic characteristics of the components of the tachycardia circuit.

PAROXYSMAL supraventricular tachycardia often involves the atrioventricular (AV) node or accessory pathway (AP) as part of the reentry circuit. Patients who have this form of arrhythmias frequently report a history of transient palpitations, suggesting that spontaneous termination of tachycardia is common under physiologic conditions. Programmed electrical stimulation of the heart combined with intracardiac recordings makes it possible to determine the sites and mechanisms of spontaneous termination of tachycardia. These observations on the site and mechanism of spontaneous termination of tachycardia might be relevant in choosing an effective antiarrhythmic drug regimen. We report on the incidence, sites of block, and detailed mechanisms of spontaneous termination of circus movement tachycardia using an AP in 24 patients who were not taking antiarrhythmic drugs.

Patients and Methods

We retrospectively analyzed 24 consecutive patients (beginning in January 1978), ages 11–60 years, in whom circus movement tachycardia using an AP was induced during clinical electrophysiologic study. No patients were excluded from analysis. Fifteen patients were male and nine were female. Seventeen patients had Wolff-Parkinson-White syndrome (WPW) and seven had a concealed AP (capable of retrograde conduction only). The AP was located on the left side in 20 patients, the right side in three patients and in the septum in one. Informed consent was obtained before electrophysiologic study. Antiarrhythmic drugs were discontinued at least five half-lives before electrophysiologic study. Lido-caine (2% solution), 5–10 ml, was used for local anesthesia. Our technique for electrophysiologic study has been reported.

Continuous recordings of at least eight simultaneous surface and intracardiac leads were obtained during all episodes of tachycardia at a paper speed of 100 mm/sec. All episodes of tachycardia in these patients were recorded and those that terminated spontaneously were analyzed. Termination of tachycardia due to extrastimuli or "spontaneous" premature complexes were excluded from analysis. Although termination of tachycardia by spontaneous premature complexes may be important in tachycardia termination, catheter-induced premature complexes during the study were considered to be likely enough to exclude this mechanism from analysis.

The following definitions were used:

Tachycardia: arbitrarily chosen to be at least three complete cycles.
**AP block**: failure of the reentrant impulse to conduct retrogradely over the AP to activate the atria.

**Atrioventricular nodal (AVN) block**: failure of the antegrade reentrant impulse to conduct over the AVN and activate the His bundle.

**His-Purkinje system (HPS) block**: failure of the antegrade reentrant impulse to conduct over the HPS distal to the His bundle and activate the ventricles.

**Safety margin of tachycardia**: the shortest recorded tachycardia cycle length minus the longest refractory period of the different components of the reentrant circuit measured at a basic cycle length of 600 msec (chosen from atrial (A) effective refractory period (ERP), AVN<sub>ERP</sub>, HPS<sub>ERP</sub>, ventricular (V)<sub>ERP</sub> and retrograde AP<sub>ERP</sub>).

Conventional definitions for cardiac refractory periods, conduction intervals and enhanced AVN function were otherwise used.4,5

Patients who manifested spontaneous termination of tachycardia were compared with those who did not by analysis of tachycardia cycle length, AH, HV and VA intervals during the tachycardia, safety margin of tachycardia, the presence or absence of enhanced AVN function, the diagnosis of WPW vs concealed AP, location of AP and the following refractory periods measured during driven rhythm: A<sub>ERP</sub>, AVN<sub>ERP</sub>, AVN<sub>PRP</sub>, V<sub>ERP</sub>, retrograde AP<sub>ERP</sub> (where FRP = functional refractory period).

The various sites of block in the reentry circuit causing spontaneous termination of tachycardia were analyzed. Patients who manifested block at any site were compared with the 14 patients who did not. In the patients with block in the AP, the following were analyzed: tachycardia cycle length; retrograde AP<sub>ERP</sub> measured at a basic cycle length of 600 msec; ventriculoatrial (VA) interval during tachycardia; type of AP (WPW vs concealed); presence of enhanced AVN function; incidence of bundle branch block abberation in tachycardia. In the patients with block in the AVN, the following were analyzed: tachycardia cycle length; AVN<sub>ERP</sub> measured at a basic cycle length of 600 msec; presence of enhanced AVN function; presence of bundle branch block abberation during tachycardia; AH interval during sinus rhythm (AH<sub>min</sub>); maximal AH recorded during electrophysiologic study (AH<sub>max</sub>). In patients who manifested block in the HPS, the following were analyzed: tachycardia cycle length; AVN<sub>ERP</sub> measured at a basic cycle length of 600 msec; presence of enhanced AVN function; HV interval; presence of bundle branch block abberation during tachycardia; and AH<sub>max</sub> recorded at electrophysiologic study.

Statistical analyses were performed using a two-tailed t test for populations with equal variances. A p value ≤0.05 was considered significant.

**Results**

**Incidence and Predictive Factors for Spontaneous Termination of Tachycardia**

Ten patients manifested spontaneous termination of tachycardia during electrophysiologic study, six with WPW syndrome and four with a concealed AP.

Patients with a concealed AP had a higher incidence of spontaneous termination of tachycardia than those with WPW (NS by Fisher's exact test). The three patients with right-sided APs showed spontaneous termination of tachycardia. The AP was a site of block in these three patients. No statistically significant differences were found in patients with spontaneous termination of tachycardia compared to those without in terms of location of AP; presence or absence of enhanced AVN function; AH, HV and VA intervals during tachycardia; A<sub>ERP</sub>; AVN<sub>ERP</sub>/FRP; V<sub>ERP</sub>; retrograde AP<sub>ERP</sub>; and tachycardia cycle length.

Patients with spontaneous termination of tachycardia had a safety margin of 20 ± 43 msec (mean ± SD) compared to 48 ± 36 msec in those without spontaneous termination (p < 0.01). In general, the site in the tachycardia circuit with the longest refractory period at a basic cycle length of 600 msec was the AP (13 of 22 cases in which these data were available.) In seven of 10 cases the longest refractory period in the tachycardia circuit at a basic cycle length of 600 msec coincided with one of the observed sites of block that caused termination tachycardia. Of interest was the negative safety margin of up to 40 msec noted in four patients, always the result of a long retrograde refractory period in the AP. Tachycardia in these cases clearly depended on rate-related shortening of refractoriness in the AP. Spontaneous termination of tachycardia occurred in three of these four patients at the level of the AP in one, the AVN in another, and either the AVN, HPS or AP in the remaining patient.

**Sites of Block Causing Spontaneous Termination of Tachycardia**

The number of episodes of spontaneous termination and sites of block are shown in table 1. The most frequent sites of spontaneous termination were located

<table>
<thead>
<tr>
<th>Pt</th>
<th>AVN</th>
<th>HPS</th>
<th>AP</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td></td>
<td></td>
<td>33</td>
</tr>
<tr>
<td>4</td>
<td>3</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>5</td>
<td></td>
<td></td>
<td>2</td>
</tr>
<tr>
<td>6</td>
<td></td>
<td></td>
<td>7</td>
</tr>
<tr>
<td>7</td>
<td>22</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>7</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>9</td>
<td>2</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>10</td>
<td></td>
<td>2</td>
<td></td>
</tr>
</tbody>
</table>

**Total episodes** 37 18 50

**No. of patients** 6 3 6

**Abbreviations**: AVN = atrioventricular node; HPS = His-Purkinje system; AP = accessory pathway.
in the AP or the AVN. Three patients manifested block at multiple sites in the tachycardia circuit.

Relation of Duration of Tachycardia to Site of Spontaneous Block in the Tachycardia Circuit

The duration of tachycardia before spontaneous termination is shown in figure 1. Three patterns are present. HPS block always occurred in the first 10 beats (<5 seconds) of tachycardia. AVN block occurred either in the first 5-10 seconds of tachycardia or in the late stages (>30 seconds) of tachycardia. No episodes of AVN block occurred in the 10-30-second time range. Block in the AP occurred at all stages in the first 25 seconds of tachycardia, and became less frequent, although it still occurred, in the late (>30 seconds) stages of tachycardia.

Possible Predictive Factors for Sites of Spontaneous Block in the Tachycardia Circuit

In searching for factors associated with block at a specific site, those with block at that site were compared with the other 14 patients who did not manifest block at that site. None of the measurements outlined in the methods section showed statistically significant correlation with a specific site of block in the tachycardia circuit. However, all right-sided APs showed spontaneous termination of tachycardia in the AP, although the numbers were too small to determine statistical significance. All tachycardias that terminated spontaneously at the level of the HPS showed either right or left bundle branch block aberrancy before block. Bundle branch block aberrancy per se, however, was not predictive of block in the HPS.

Mechanisms of Spontaneous Termination of Tachycardia

Block in the Accessory Pathway

Five mechanisms led to block in the AP:

1. Sudden spontaneous block in the accessory pathway without any preceding changes in the tachycardia (patients 3-6, 8 and 9). Sometimes, minimal changes in tachycardia cycle length of 5-10 msec were observed that were within the range of the natural variability of the tachycardia.

2. Block in the AP after a short cycle during cycle length alternation of tachycardia (generally in the AH interval) (fig. 2) (patients 3, 4 and 6). This was not related to a critical length of the short cycle. However, no episodes of block in the AP were observed after the long cycle during cycle length alternations.

3. Block in the AP after gradual shortening of the tachycardia cycle length due to a gradual decrease in the AH interval (patients 5 and 6). Occasionally, a VA increase was noted with decrease in cycle length, suggesting decremental conduction in the VA part of the tachycardia circuit (but not necessarily in the AP itself) (fig. 3).

4. Sudden shortening of AH interval during stable tachycardia (without any antecedent changes in AH interval) causing block in the AP by advancing ventricular activity during tachycardia (patients 3 and 8).

5. Loss of bundle branch block ipsilateral to the AP causing block in the AP by advancing ventricular activation in the region of the bypass tract without altering the onset of ventricular activity (fig. 4) (patient 6).

Block in the AV Node

Six mechanisms led to block in the AVN:

1. Sudden spontaneous AVN block during stable tachycardia without significant antecedent changes in AH interval (patients 2 and 7). In one patient the AH interval increased from 140 to 145 msec before spontaneous block in the AVN. This could be construed as an atypical Wenckebach sequence. This patient had enhanced AVN function and the maximum AH interval recorded before AVN block during atrial pacing was 150 msec, which would be in accord with the above explanation. Nevertheless, this tachycardia was sustained and stable with AH intervals of 140 msec. Five-millisecond variations in AH interval were within the natural variability of this tachycardia and did not previously lead to AVN block. In the other patient with the same phenomenon, no increase in AH interval was seen before block.

2. AVN Wenckebach sequences after induction of tachycardia leading to block in the AVN (patient 8). AVN Wenckebach sequences after advancement of atrial activation by improved HPS conduction were also observed, but are included in mechanism 4.

![Figure 1. Duration of circus movement tachycardia (CMT) before spontaneous termination in the His Purkinje system (HPS), atrioventricular node (AVN), or accessory pathway (AP).](image-url)
Figure 2. Case 6 — cycle length alternation due to AH alternation is present during tachycardia. Retrograde block in the accessory pathway causes spontaneous termination of tachycardia following a short cycle. The previous short cycle of identical cycle length fails to cause block in the accessory pathway. The ventriculoatrial interval is constant before block.

Tracings in this and other figures (except when labeled otherwise) are surface ECG leads 2 and V6, high right atrial (HRA); proximal and distal coronary sinus (CSp and CSd); and His bundle electrograms. The paper speed is 100 mm/sec. Electrograms are labeled A for atrial, H for His bundle and V for ventricular.

(3) Block in the AVN after a short cycle during cycle length alternation (fig. 5) (patients 4, 7, 8 and 10). Sometimes, a progressive increase in alternate AH intervals appeared before block after the short cycle (fig. 5), suggesting a Wenckebach sequence for alternate cycles. However, the presence of alternating long and short AH intervals clearly differentiates this mechanism from mechanism 2.

(4) Improved HPS conduction causing advancement of atrial activation both by loss of bundle branch block ipsilateral to the AP and also by shortening of the HV interval (patients 7–9). This caused AVN block either immediately (fig. 6) or after a few beats of unstable tachycardia.

(5) Impairment of HPS conduction (HV prolongation, split His, or bundle branch block) causing delay in atrial activation and subsequent unstable tachycardia (fig. 7) (patient 8). Unstable tachycardia was due to variation in at least one of the following: AH, HV, VA intervals or intraventricular conduction. Subsequent shortening of the HV interval during unstable tachycardia was often the final factor in termination in the AVN.

(6) Block in the AVN during left bundle branch block aberrancy dependent on attaining a critical HV interval (patient 4). Thus, delay of atrial activation due to HV prolongation paradoxically led to block in the AVN. We believe that the mechanism of termination of tachycardia in this case was retrograde concealed penetration of the AVN during tachycardia via the left bundle branch. This case will be reported separately.

Block in the His-Purkinje System

Three mechanisms led to spontaneous block in the HPS during tachycardia:

(1) Wenckebach block in the HPS after initiation of tachycardia (fig. 8) (patients 1 and 8). Because of the arbitrary definition of tachycardia as at least three complete revolutions through the reentry circuit, shorter HPS Wenckebach sequences were excluded from analysis, although they were rather common. Bundle branch block aberration was present before spontaneous termination.

(2) After cycle length alternation in AH interval (patient 1). No changes in HV interval preceded block. Bundle branch block aberration was present before termination. Block occurred after a short cycle.

(3) After cycle length alternation in HV (patient 4). Bundle branch block aberration was present. Variability in HV interval caused cycle length alternation (which at times was sustained for several seconds without spontaneous termination of tachycardia). Block in the HPS followed a short HH interval.

Discussion

This study confirms the clinical impression that spontaneous termination of tachycardia using an AP
SPONTANEOUS TERMINATION OF TACHYCARDIA/Ross et al.

Figure 3. Case 5 — after initiation of circus movement tachycardia there is a gradual decrease in tachycardia cycle length, beginning 6 seconds after the onset of tachycardia. A satisfactory His spike is not seen during tachycardia in these recordings, so atrioventricular (AV) conduction intervals are used to show that the decrease in tachycardia cycle length is due to decrease in AV conduction times. Concomitantly, the ventriculoatrial (VA) interval before block suggests Wenckebach block in the accessory pathway. The QRS configuration does not change during tachycardia. The VA interval is measured from the onset of ventricular activity to the onset of atrial activity in the coronary sinus (CS) lead. Times in seconds denote duration of tachycardia.

Figure 4. Case 6 — induction of circus movement tachycardia by an extrastimulus during right ventricular pacing at a basic cycle length of 600 msec. The accessory pathway is right-sided and earliest atrial activation during tachycardia occurs in the right atrial leads. Complete right bundle branch block (RBBB) aberration is present during tachycardia (QRS width 170 msec). The HV interval decreases as the tachycardia is sustained and RBBB aberrancy is lost in the last QRS complex. Both phenomena are due to adaptation of His-Purkinje system, refractoriness and conduction to the increased rate. The ventriculoatrial interval during RBBB is 240 msec. Loss of RBBB in the last beat advances right ventricular activation during tachycardia, although the time of onset of QRS is not advanced. Early arrival of the reentrant impulse causes block in the accessory pathway and spontaneous termination of tachycardia. S indicates a pacing artifact during tachycardia which fails to capture the ventricle. Abbreviations: see legend for figure 2.
FIGURE 5. Case 10 — five surface ECGs (I, II, III, V₁ and V₅). Cycle length alternation is present during circus movement tachycardia due to AH alternation. The ventriculoatrial interval is constant. Note that alternate AH intervals progressively increase before block. QRS complexes also show alternation of morphology. Block occurs in the atrioventricular node after a short cycle. The previous short cycle of identical cycle length fails to precipitate block in the atrioventricular node. Abbreviations: see legend for figure 2.

FIGURE 6. Case 7 — surface leads I and V₁ are included in this figure. A left lateral accessory pathway was present. Complete left bundle branch block (LBBB) was present during tachycardia with a ventriculoatrial (VA) interval of 230 msec. After spontaneous loss of LBBB, the HV interval shortens by 30 msec and the VA interval by 40 msec. This advances atrial activity by 70 msec, causing block in the atrioventricular node and spontaneous termination of tachycardia. After termination of tachycardia, there is fusion between a sinus beat and an escape ventricular beat. Sinus rhythm with normal conduction occurs with the next beat. HIS = His bundle electrogram; HRA = high right atrial electrogram.
is relatively frequent (10 of 24 patients). We observed at least three sites in the tachycardia circuit at which spontaneous block occurred in the absence of antiarrhythmic drugs. Block at the level of the AVN or AP is well known. Block at the level of the HPS is less well known in this type of tachycardia, although it has been observed after antiarrhythmic drug treatment.9, 10 Spontaneous block in the HPS may occur during tachycardia in the absence of drugs in patients with an electrophysiologically normal HPS. This may also occur in reentry AVN tachycardias but does not, of course, lead to spontaneous termination.11

Determinants of Spontaneous Termination of Tachycardia

Tachycardia cycle length, refractory periods of individual components of the tachycardia circuit, and other patient variables failed to correlate with spontaneous termination. As reported by Mines,12 continuation of tachycardia depends on a favorable relationship between the longest refractory period in the tachycardia circuit and the duration of the tachycardia cycle. We attempted to quantify this relationship by using the shortest recorded tachycardia cycle length and subtracting the longest refractory period in the circuit (measured at a basic cycle length of 600 msec). This safety margin of tachycardia was significantly reduced in the group with spontaneous termination (p < 0.01). This method of measuring safety margin has major limitations: After onset of tachycardia, refractory periods would clearly change to different degrees and in different directions. However, measurement of these refractory periods during tachycardia was not done in our retrospectively analyzed series of cases. The longest refractory period in the tachycardia circuit was most frequently retrograde APERP (13 of 22 cases). In four cases, this refractory period was up to 40 msec longer than the shortest recorded tachycardia cycle length. Rate-related shortening of AP refractoriness must therefore have occurred to sustain tachycardia. This is compatible with a previous study in which decreases of 10–60 msec occurred in retrograde APERP after an increase in heart rate.8 No patient variable analyzed in our study reliably predicted the site of block in spontaneous termination of tachycardia. The safety margin for tachycardia is generally small, ranging from approximately 6% of the tachycardia cycle length in patients with spontaneous termination to

Figure 7. Case 8 — circus movement tachycardia is induced by an extrastimulus during coronary sinus (CS) pacing at a basic cycle length (BCL) of 600 msec. The last four beats of tachycardia are labeled. H' is the second component of a split His bundle electrogram (HIS). Beat 1 is followed by delayed conduction in the His bundle producing a split His bundle electrogram. After this, there is unstable tachycardia with variable conduction intervals at several sites in the tachycardia circuit (AH, HV and VA) and variable aberrant conduction in the bundle branches (variable QRS morphology). Shortening of HV and ventriculoatrial (VA) intervals in beat 4 after a longer preceding HH interval advances atrial activation and is the final factor precipitating block in the atroventricular node, which terminates tachycardia. The ventriculoatrial (VA) intervals are measured from onset of QRS to the CS electrogram (which occurs 10 msec later than the atrial electrogram in the His lead).
Basic Mechanisms of Block in the Tachycardia Circuit

Block in the tachycardia circuit had three basic mechanisms.

Sudden Block

In a manner similar to Mobitz type II block, sudden failure of conduction occurred without preceding decremental conduction or change in rate. This phenomenon was not rate-related and occurred in both the AP and AVN. Its occurrence in the AVN is surprising. Both patients in whom it occurred had evidence of enhanced AVN function, but they nevertheless demonstrated increase in AVN transmission time with premature atrial stimuli. The underlying mechanism in these cases is probably a Wenckebach-type block, with little or no increase in AH interval before block.

Decremental Conduction Leading to Block

This was observed in the AP, AVN, and HPS. Decremental conduction in the AP and HPS is uncommon. In the case of the HPS it generally occurs in the presence of HPS disease. In our study, decremental conduction in the HPS occurred during tachycardia in patients who had no other evidence of significant impairment of HPS conduction. Decremental conduction in the AP has been noted previously in a few cases in both the antegrade and retrograde direction.

Tachycardia-dependent Block

This occurred in all three major components of the tachycardia circuit (AVN, HPS, AP) as a result of in-
crease in tachycardia rate or advancement of tachycardia by improved conduction in one of the components of the circuit (AP mechanisms 3, 4, and 5, AVN mechanism 4 and HPS mechanisms 2 and 3).

Labile Conduction in the Tachycardia Circuit

Although the labile component of the tachycardia circuit is considered to be the AVN, we also observed variable conduction during tachycardia in the His bundle, bundle branches and AP. Conduction either improved, was impaired, or alternated. Improved conduction may lead to block elsewhere in the circuit. Onset of impaired conduction at one site in the tachycardia circuit may herald block at that site or cause block at a distant site. This latter finding depends on the slope of the AVN function curve during tachycardia. If it is steep, small changes in the tachycardia cycle may be amplified. Thus, a delay in conduction may produce unstable tachycardia with oscillating long and short cycles (often with variable AH, HV and sometimes VA intervals) before the occurrence of spontaneous block at one site. Cycle length alternation during reentry supraventricular tachycardia due to alternating long and short AH intervals has been described. Currie and Krikler described its relationship to the relative refractory period of the AVN during simulated circus movement tachycardia. We also observed cycle length alternation due to HV alternation. Cycle length alternation during tachycardia (usually due to AH alternation) was associated with block at the level of the AVN, HPS and AP. Block always occurred after a short cycle. Often, preceding short cycles of similar duration failed to cause block (figs. 2 and 5). Thus, block during cycle length alternation may not be strictly rate-related phase 3 block. However, constant association of block with a preceding short cycle suggests a causal relationship rather than a fortuitous nonrelated phenomenon.

Multiplicity of Mechanisms of Spontaneous Termination

We identified mechanisms that lead to spontaneous termination of tachycardia. However, sudden spontaneous block without precipitating cause often occurred at the level of the AP, so the assumption that block at this level was due to other mechanisms must be examined. Occurrence of AP block in the same cycle as sudden shortening of AH interval or sudden loss of ipsilateral bundle branch block suggests that the two events were related. Similarly, the occurrence of VA Wenckebach block during a gradual decrease in tachycardia cycle length due to autonomic induced AH shortening suggests cause and effect. In the case of the AVN, the basic mechanism of block was most likely always a variation of Wenckebach block. However, the antecedent events clearly identified different sequences leading to AVN block.

Time Patterns of Spontaneous Block at Different Sites in the Tachycardia Circuit

Several phases occur before the establishment of sustained stable reentry tachycardia.

Induction of the First Echo Beat

Three conditions for reentry must be satisfied before the first echo beat can be induced: presence of a potential reentry circuit, one-way block in one of the limbs of the circuit, and slow conduction through the circuit to allow previously depolarized tissues ahead to recover excitability.

Rate-dependent Phase

This lasts for approximately the first 5 seconds of tachycardia and is largely independent of changes in autonomic tone, which take longer to have significant effects. Once the first echo has been achieved, continuation of the reentry cycle depends on the reentrant impulse not encountering refractoriness ahead in the circuit. The sudden increase in rate when tachycardia continues tends to cause decreased refractoriness in atrial and ventricular myocardium, AP and HPS. In contrast, an increase in rate tends to increase AVN refractoriness, with consequent increase in AH interval. Although this tends to slow the tachycardia cycle, protecting the tachycardia somewhat from spontaneous termination in the AVN, in this period the AVN would be expected to be the weak link in the tachycardia circuit.

Autonomic Phase

In phase 3a, increased sympathetic tone and decreased vagal tone are consequent upon drop in blood pressure after the initiation of tachycardia. This decreases AV nodal refractoriness markedly and makes it less likely to be the site of spontaneous termination. These autonomic changes probably have little effect on refractoriness at other sites in the circuit.

Phase 3b is a later phase of decreased sympathetic tone, and increased vagal tone may occur in some instances if blood pressure rises inappropriately as a result of phase 3a. This tends to increase AVN refractoriness and make it again a potential site for spontaneous termination of tachycardia. This is also the probable basis of the successful use of blood pressure elevating drugs like metaraminol in the treatment of AV junctional tachycardia.

Sustained Stable Tachycardia

All rate-related changes in refractoriness have occurred and a stable autonomic tone has been achieved. Tachycardia then continues until change in autonomic tone, drug administration, or premature complexes occur to terminate tachycardia. Curry et al. described the changes in autonomic tone involved in these phases.
The time patterns we observed for spontaneous termination of tachycardia by block in the AVN, HPS or AP (fig. 1) are in accord with the above phases of tachycardia. Thus, during the rate-dependent phase, all components of the circuit may be the site of block, with a predisposition to AVN block because of the increase in AVN refractoriness with increase in rate. His-Purkinje system block occurs only in this phase and probably indicates rate-dependent decrease in HPS refractoriness to a greater degree than occurs in other components of the circuit, such as the AP.9-7

During the phase of increased sympathetic tone (phase 3a), rate-dependent block would be expected to continue to occur because shortening of the AH interval increases tachycardia rate. The site of block during phase 3a would depend on the original refractory periods of the components of the tachycardia circuit and their subsequent changes due to increased rate and sympathetic tone. In our study, spontaneous termination of tachycardia in this phase was due to block in the AP only (10–30-second duration of tachycardia). Retrograde AP 

\[ R_\text{ERP} \]

tended to be the longest refractory period in the circuit, and AVN refractoriness was decreased by the increased sympathetic tone, so block in the AP might be expected at this stage. We postulate that a rate-related decrease in HPS refractoriness explains the lack of spontaneous block in the HPS in phase 3a of tachycardia.

The occurrence of some instances of spontaneous termination in the AVN after more than 30 seconds of tachycardia correlates with the increased vagal and decreased sympathetic tone phase (phase 3b) of tachycardia. Our instances of AVN block at this stage were associated with cycle length alternation in the AH interval, as might be expected with increased AVN refractoriness. Occasional episodes of spontaneous block in the AP occurred during sustained stable tachycardia, without any preceding change in tachycardia. This might represent fatigue in AP conduction.

Limitations of the Study

Electrophysiologic studies have inherent drawbacks in interpreting physiologic events. Thus, lidocaine local anesthesia,24 catheter-induced trauma to the AP or HPS, and posture25 may have significant effects. Block of conduction in the AP or HPS was not observed during sinus rhythm in our study, so any catheter-induced trauma was apparently minor. Autonomic tone is also difficult to assess, but it appears that electrophysiologic study does not provoke a hyperadrenergic state.26 Given these limitations, our results must be interpreted with caution when extrapolating to spontaneous termination of tachycardia under physiologic circumstances.

Therapeutic Implications of Spontaneous Termination of Tachycardia

Wu et al. reported that efficacy of antiarrhythmic drug therapy in patients with circus movement tachycardia using an AP often depends on promoting spontaneous termination of tachycardia after a few beats.10 Raper et al. suggested that observation of the site of spontaneous termination of tachycardia and the phase in which it occurs may aid in choosing antiarrhythmic drugs that will enhance spontaneous termination.9 Our data show that multiple mechanisms cause spontaneous termination and that these are often dependent on changes in the circuit remote from the site of the block. Understanding these mechanisms and the interplay involved may be useful in choosing an effective antiarrhythmic drug. This hypothesis must be tested in a prospective study.

Acknowledgment

We acknowledge the technical assistance of Jan Kersemakers and Adri van den Doel, and the cardiology fellows for their help in performing these studies.

References

17. Wellens HJJ, Durrer D: Combined conduction disturbances in
two A-V pathways in patients with Wolff-Parkinson-White syn-
drome. Eur J Cardiol 1: 23, 1973
18. Klein GJ, Prystowsky EN, Pritchett ELC, Davis D, Gallagher
JJ: Atypical patterns of retrograde conduction over accessory
atrophicventricular pathways in the Wolff-Parkinson-White syn-
drome. Circulation 60: 1477, 1979
19. Spurrell RAJ, Krikler D, Sowton E: Two or more intra AV
nodal pathways in association with either a James or Kent ex-
tranodal bypass in 3 patients with paroxysmal supraventricular
20. Vohra J, Hunt D, Stucray J, Sloman G: Cycle length alterna-
tion in supraventricular tachycardia after administration of
verapamil. Br Heart J 36: 570, 1974
21. Wellens HJJ, Tan SL, Bar FWH, Duren DR, Lie KI, Dohmen
HM: Effect of verapamil studied by programmed stimulation of
the heart in patients with paroxysmal re-entrant supraventric-
22. Curry PVL, Krikler DM: Significance of cycle length alterna-
tion during drug treatment of supraventricular tachycardia. Br
Heart J 38: 882, 1976
23. Curry PVL, Rowland E, Fox KM, Krikler DM: The
relationship between posture, blood pressure, and electrophysiological properties in patients with paroxysmal supraventric-
anesthesia for cardiac electrophysiologic studies. N Engl J Med
301: 418, 1979
25. Novick TL, Pritchett ELC, Cambell RWF, Rogers GC,
Wallace AG, Gallagher JJ: Temporary, catheter-induced block
tone of patients during electrophysiological catheterization. Am
Heart J 99: 51, 1980
Spontaneous termination of circus movement tachycardia using an atrioventricular accessory pathway: incidence, site of block and mechanisms.
D L Ross, J Farre, F W Bar, E J Vanagt, P Brugada, I Wiener and H J Wellens

doi: 10.1161/01.CIR.63.5.1129

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/63/5/1129