Cycle Length Alternation in Circus Movement Tachycardia Using an Atrioventricular Accessory Pathway

A Study of the Role of the Atrioventricular Node Using a Computer Model of Tachycardia

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SUMMARY The possible role of the atrioventricular nodal (AVN) function curve during tachycardia as a cause of cycle length alternation was investigated using a computer model of circus movement tachycardia utilizing an atrioventricular accessory pathway. Two types of AVN function curves during tachycardia were entered into the computer: straight lines of various gradients and representative examples of patient-based AVN function curves obtained during clinical electrophysiologic studies. Perturbations of the tachycardia model were induced by introducing a short cycle, by suddenly prolonging a conduction interval, or by moving the AVN function curve relative to that in stable tachycardia. Using the straight line AVN function curves, stable, sustained cycle length alternation could be induced by perturbation of the tachycardia cycle if the gradient of the line was \(-1\) (slope = \(45^\circ\)). If the gradient was more than \(-1\) (slope < \(45^\circ\)), the perturbation was damped. If the gradient was less than \(-1\) (slope > \(45^\circ\)), the perturbation was amplified, leading to termination of tachycardia by block in the AVN. Similar but more complex responses to perturbation of tachycardia were found using patient-based AVN function curves. Thus, sustained cycle length alternation and amplification or damping of perturbation could be produced. Using physiologic AVN function curves, the response to perturbation of tachycardia depended on the interrelationship of the shape of the AVN function curve, the location of the cycle length of tachycardia on the curve, the magnitude and direction of the perturbation, and the AVN effective refractory period. We conclude that cycle length alternation during tachycardia may be explained by the characteristics of a single antegrade AVN function curve without postulating the presence of additional AVN pathways. The stability of circus movement tachycardias depends on the interaction of several variables.

CYCLE LENGTH ALTERNATION during supraventricular tachycardia has been noted in several reports.\(^1\)\(^-\)\(^9\) It is usually seen at the beginning or end of a paroxysm of tachycardia or after administration of drugs that increase atrioventricular nodal (AVN) refractoriness.\(^4\)\(^-\)\(^6\) The mechanisms have been a puzzle and a variety of explanations have been suggested, including supernormal conduction, alteration in the length or conduction velocity of the reentrant pathway, and alternate use of a previously concealed slow antegrade pathway.\(^1\)\(^-\)\(^4\)\(^,\)\(^7\)\(^,\)\(^8\) More recently, using electrophysiologic studies to define the location and mechanism of tachycardia, some investigators have concluded that cycle length alternation in circus movement tachycardias utilizing an atrioventricular accessory pathway requires the presence of dual AVN pathways.\(^7\)\(^,\)\(^8\) These investigators postulated 2:1 block in the antegrade fast AVN pathway as the cause of cycle length alternation. Other workers have implied that normal properties of the AVN may be sufficient to permit cycle length alternation to occur.\(^5\)\(^,\)\(^8\)\(^,\)\(^10\) In this study we investigated the role of the AVN function curve during tachycardia by means of a computer model of circus movement tachycardia utilizing an accessory pathway. Cycle length alternation in this type of tachycardia refers to simultaneous atrial and ventricular cycle length alternation. Ventricular cycle length alternation due to atrial tachycardia with 3:2 Wenckebach atrioventricular block will not be considered in this paper.

Methods

A mathematical model of circus movement tachycardia utilizing an accessory atrioventricular pathway was constructed and entered into an Apple II microcomputer:

Let AA = the cycle length of tachycardia; let AH = the conduction time in the antegrade limb of the tachycardia circuit for conduction from atrium through the AVN to the His bundle; let K = the sum of conduction times in the retrograde limb of the tachycardia circuit (the sum of conduction times from the His bundle to ventricle to accessory pathway and back to atrium adjacent to the AVN). Then AA = AH = K.

Assuming that K is a constant, AA is directly proportional to AH. For any given tachycardia operating at a stable basic cycle length with a stable AH interval, there exists an AVN function curve relating the coupling interval of premature or late beats during tachycardia to their resultant increase or decrease in AH interval. Thus, any perturbation of the tachycardia cycle causing a short or long cycle will cause a change in AH interval in the next cycle, and consequently a change in the next AA interval, which then changes the following AH, and so on. To investigate

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862
the role of the AVN function curve during tachycardia in determining the effects of perturbation of the tachycardia cycle, the following curves were used: (1) simple straight-line relationships between AA and AH of various gradients from 0 to $-\infty$ (slope 0–90°); and (2) patient-based AVN function curves obtained during clinical electrophysiologic studies by single extrastimulus testing during a regular atrial paced rhythm. Patient-based curves that were tested in the computer model were selected as representative examples of clinical continuous AVN function curves by virtue of their shapes and slopes. Steep, medium and flat curves, plus similar curves with flat “tails,” were used.

These AVN function curves were entered into an Apple II microcomputer and the effects of perturbing the tachycardia cycle were tested. The computer displayed the cycle length of stable tachycardia, the perturbing cycle and subsequent tachycardia cycle lengths. In the case of patient-based AVN function curves, several stable tachycardia cycle lengths located on different parts of the curve were tested. Perturbations were achieved by introducing a short cycle, or suddenly lengthening an AH interval causing a long cycle, or by movement of the AVN function curve relative to that in stable tachycardia. For the purposes of this study we assumed that the AVN function curve during tachycardia remained constant after perturbation.

Where appropriate, clinical examples similar to the findings predicted by the computer model were selected from the records of patients undergoing electrophysiologic study in our department.

**Results**

**Using Straight-line AVN Function Curves**

Three types of responses were found after perturbation of the tachycardia cycle, depending on the gradient of the AVN function curve.

**Straight-line AVN Function Curve with Gradient $= -1$ (Slope = 45°)**

Figure 1 shows the effects of perturbation of a tachycardia in which the AVN function curve is a straight line with a gradient of $-1$ (slope of 45°). The serial changes after a 40-msec increase in AH are shown in figure 1. The net effect on tachycardia is stable cycle length alternation with an amplitude of alternation twice that of the initial perturbation. The mean cycle length of tachycardia is unchanged. Alternation lasts indefinitely unless the AVN function curve shifts or changes slope. If a straight-line AVN function curve X of slope 45°, as in figure 2, is moved to the right during stable tachycardia, as in line Y figure 2, similar phenomena are seen except that alteration occurs about a higher mean tachycardia cycle length, the short cycles are identical to the previous stable tachycardia cycle length, and the time difference between the long and short cycles is equal to the initiating perturbation in AH or AA interval that occurred as a result of the shift in the AVN function “curve” (fig. 2B). If, during sustained cycle length alternation, the AVN function curve then shifts further to the right (as in line Z in figure 2), a variety of changes may occur, depending on the timing and degree of the shift. If the curve shifts to the right during a short cycle, the subsequent long cycle is lengthened, and amplification of alternation occurs (fig. 2C). If the curve shifts to the right during a long cycle, three changes can occur, depending on the degree of lengthening of the following “short” cycle that is produced. If this cycle is lengthened such that it becomes equal to a long cycle, the changes shown in figure 2D occur. If the “short” cycle is lengthened but still remains less than the long cycle, the changes

![Figure 1](http://circ.ahajournals.org/)

*Figure 1. A stable, sustained tachycardia of cycle length $AA_1$, with an AH interval of $AH_1$ (A), is suddenly perturbed by an increase of 40 msec in the AH interval (B). The atrioventricular nodal function curve during tachycardia is represented by a straight line of gradient $-1$ (slope = 45°). The change shown in panel B causes a long cycle $AA_2$, which then causes a shortening in the AH interval ($AH_2$) compared to $AH_1$ (C). This causes a short cycle ($AA_3$), which then causes an increase in the AH interval ($AH_4$) compared with $AH_1$ (D). The net effect is sustained cycle length alternation.*
shown in figure 2E occur. If the short cycle is lengthened to a greater degree than the long cycle, the changes shown in figure 2F occur. In all responses to shifting of the AVN function curve to the right, the net effect is an increase in mean cycle length of tachycardia (fig. 2).

Figure 3 shows the effects of i.v. verapamil in a patient with sustained, stable circus movement tachycardia utilizing an accessory pathway. The onset of alternation was gradual with only a minor degree of alternation initially. A variety of changes occur, similar to those predicted in figure 2. There is a variation in degree of alternation, sometimes with and sometimes without increase in the length of the short cycle. The net effect is an increase in mean cycle length of tachycardia. Tachycardia terminates after a short cycle, probably due to associated increase in AVN effective refractory period (ERP).

The schemata in figure 2 assume that the slope of the curve stays constant. If the slope also changes when the curve moves to the right, the effects described in subsequent sections occur.

*Straight-line AVN Function Curve with a Gradient of More Than \(-1\) (Slope < 45\(^\circ\))*

Figure 4 shows the effects of perturbation by a 40-msec increase in AH interval during a tachycardia in which the AVN function “curve” is a straight line with a slope of 30\(^\circ\). The effect on subsequent tachycardia cycles is shown in figure 4E. Thus, the initial perturbation is rapidly damped in further cycles, and stable tachycardia continues at the previous stable cycle length. In the extreme example of gradient = 0 (slope = 0\(^\circ\)), the tachycardia returns to the baseline state immediately.

Figure 5 shows the effect of perturbation of a sustained stable tachycardia by induced atrial premature beats in a patient with circus movement tachycardia utilizing an accessory pathway. The resulting changes

![Figure 2](image-url)

*Figure 2.* Stable tachycardia of cycle length AA\(_1\) is present with an atrioventricular nodal (AVN) function curve X of gradient \(= -1\) (slope = 45\(^\circ\)) (A). Parallel movement of the AVN function curve to the right (line Y) during stable tachycardia induces sustained cycle length alternation (B). If the AVN function curve is shifted further to the right during sustained cycle length alternation, as in line Z, a variety of changes may occur, depending on the timing and degree of the shift.

![Figure 3](image-url)

*Figure 3.* The effects of i.v. verapamil on tachycardia cycle length in a patient with circus movement tachycardia utilizing an accessory pathway. Tachycardia cycle length was 360 msec before the onset of alternation. Note the gradual onset and variation in amplitude of alternation, increase in length of the short cycle, and increase in mean tachycardia cycle length. The tachycardia terminates after a short cycle.
in subsequent tachycardia AH intervals are similar to those predicted by the mathematical model.

Straight-line AVN Function Curve with Gradient of Less Than \(-1\) (Slope > 45°)

Perturbations were amplified in subsequent cycles in tachycardias with a straight-line AVN function curve of slope > 45° as would be predicted using similar reasoning to that used to construct figures 1 and 4. Thus, increasing longer and shorter cycles alternate until a short cycle falls within the ERP of the AVN causing termination of tachycardia. The steepness of the slope above 45° and the AVN ERP determine the degree of amplification of a perturbation and the number of subsequent tachycardia cycles before termination by block in the AVN, which always occurs after a short cycle. In the extreme example of gradient \(= -\infty\) (slope = 90°), tachycardia in infinitely unstable.

Patient-based AVN Function Curves

Findings similar to those seen with straight-line AVN function curves were obtained using patient-based AVN function curves. Thus, sustained cycle length alternation, damping and amplification of perturbation leading to AVN block could be produced (figs. 6A–C). However, the types of responses were more complicated than with the straight-line model and depended on the shape of the AVN function curve, the location of the point of stable tachycardia on that curve, the magnitude and direction of the perturbation, and the AVN ERP. A perturbation could be damped or amplified in subsequent cycles before the delayed onset of sustained stable cycle length alternation (figs. 6D and E). Thus, cycle length alternation could be induced using a variety of curves, in contrast to the straight-line model, which required a single, specific gradient. A flat tail on the AVN function curve (i.e., the right part of the curve) tended to increase the incidence of sustained cycle length alternation, especially if it had a gradient of 0 (provided the point of intersection of stable tachycardia fell on the steeper part of the curve). In contrast, if the point of intersection of stable tachycardia fell on the flat part of the curve, perturbations tended to be damped rapidly. If the gradient is 0 in this latter situation, all premature beats that did not immediately cause block in the AVN had no effect on subsequent tachycardia cycles (fig. 6F).

Discussion

This study demonstrates that cycle length alternation can be produced in a computer model of circus movement tachycardia utilizing an accessory pathway by assuming normal properties of AVN function. Several observations support the idea that the findings from this tachycardia model can be extrapolated to the clinical situation:

Cycle length alternation in the tachycardia model requires some part of the AVN function curve to have a gradient of \(-1\) or greater. Thus, some part of the AVN function curve must be at least moderately steep. This suggests that the AVN must be in a state of at least moderate refractoriness. This is compatible with the clinical situations in which cycle length alternation occurs, which are generally times of increased AVN refractoriness, such as onset of tachycardia, or just before spontaneous termination, or after using drugs that increase AVN refractoriness.
Cycle length alternation in the tachycardia model may be induced by movement of the AVN function curve to the right. Movement of the AVN function curve in this direction occurs during administration of drugs that increase AVN refractoriness. This could explain why clinical cycle length alternation is often induced by these drugs. Furthermore, the same drugs increase the AVN ERP, which, according to the tachycardia model, would predispose to termination of tachycardia at the level of the AVN after a short cycle during cycle length alternation. This is consistent with previous clinical observations.4,6

Induction of cycle length alternation in the tachycardia model by gradual movement of the AVN refractory curve to the right tends to cause a gradual onset of alternation, with a gradual increase in amplitude. Gradual onset is frequently seen in clinical cycle length alternation (fig. 3). In contrast, the onset of alternation according to the theory of 2:1 block in an antegrade fast AVN pathway should be sudden and marked, since block of fast AVN pathway conduction is usually manifested clinically by a sudden, marked increase in AVN conduction time.12 In our clinical experience, this type of onset of alternation is uncommon.

Perturbations of sustained clinical tachycardias by introduced conducted premature beats are usually rapidly damped (fig. 5). Thus, these tachycardias are generally resistant to perturbation. This suggests that AVN function curves during sustained stable tachycardias are usually flat, probably secondary to reflex increase in sympathetic tone. Thus, cycle length alternation due to the mechanism postulated in the tachycardia model should only be seen in situations of increased AVN refractoriness. Again, this is compatible with the clinical context in which cycle length alternation usually occurs.

Limitations of the Tachycardia Model

The major limitation of the tachycardia model used in this study is the assumption that perturbations of

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**Figure 5.** The effects on AH interval during tachycardia of induced premature beats in a patient with stable circus movement tachycardia utilizing an accessory pathway. The changes shown in AH interval caused identical changes in tachycardia cycle length. Thus, the perturbations were rapidly damped. Measurements were made at a paper speed of 1000 mm/sec. mAA = the cycle length of stable sustained tachycardia; AA = the coupling interval of the induced premature beats.

**Figure 6.** Effects of perturbing sustained stable tachycardias in the computer model using patient-based atrioventricular nodal function curves of various shapes. The locations on the curves of the tachycardia cycle lengths before perturbation are represented by dots. The initiating perturbations are shown in the first deflections from baseline in the right-hand panels.
the tachycardia cycle do not affect the AVN function curve in subsequent cycles. This is almost certainly not so. A short cycle would be expected to move the AVN function curve to the right in the next cycle, and a long cycle to move it to the left. These changes in the AVN function curve would make it more difficult to induce sustained cycle alternation according to our proposed mechanism. However, cycle length alternation can be produced by verapamil in the intact human heart using the technique of simulated circus movement tachycardia (fig. 7). Thus, even when the AVN function curve is physiologically responsive to beat-to-beat changes, cycle length alternation can be induced. This example is an exact physiologic parallel to the computer model, because the alternative theory of multiple AVN pathways could be excluded by comprehensive electrophysiologic testing before and after verapamil. It therefore seems likely that the findings of the computer model can be extended to the clinical situation despite the assumptions involved.

The other basic assumption of the tachycardia model is that the AVN is the only site in the tachycardia circuit at which conduction may vary. Although this is usually so, exceptions are not uncommon. Any increases in conduction time in the retrograde limb of the tachycardia circuit after a short cycle would tend to decrease the tendency toward cycle length alternation. However, this is rare in clinical cycle length alternation, when the VA interval usually remains constant, although a minor degree of QRS aberrancy may occur during the short cycles.

**Cycle Length Alternation in AVN Tachycardia**

Cycle length alternation also occurs in intranodal reentry tachycardia. The conduction time in the retrograde pathway is fixed in the majority of these patients; thus, the mathematical model of tachycardia and its conclusions should be applicable. The stability of tachycardia and the tendency toward cycle length alternation during tachycardia will be determined by the characteristics of the antegrade slow AVN pathway function curve. If the retrograde pathway can also manifest delayed conduction in response to prematurity, as in the remaining patients with AVN reentry, then the tendency to cycle length alternation and the effects of perturbation of tachycardia will be reduced.

**Stability of Tachycardia**

Simson et al. studied the stability of circus movement tachycardias utilizing an accessory pathway in computer and canine models, and concluded that tachycardias operating on a steep part of the AVN function curve (gradient \(-1\) or less) were potentially
unstable. Our data are in general agreement with their conclusions. However, the gradient of the curve at the point at which the tachycardia is operating is not the only important factor if physiologic human AVN function curves are used. The effects of perturbation are determined by the complex interrelationship of the shape of the AVN function curve, the location on the curve of the cycle length of stable tachycardia, the AVN effective refractory period, and the magnitude and direction of perturbation.

Conclusions and Clinical Implications

Cycle length alternation during circus movement tachycardia may be explained by assuming normal characteristics of a single antegrade AVN pathway. Consequently, cycle length alternation during tachycardia should not be used as sole evidence for the presence of dual AVN pathways in patients manifesting this phenomenon. This study also provides an explanation for the clinical observation that drugs like verapamil may induce cycle length alternation before termination of tachycardia by block in the AVN. Finally, the characteristics of the AVN function curve, the cycle length of tachycardia, and the magnitude and direction of any perturbations are important in determining the stability of the tachycardia circuit.

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