Circus Movement in Rabbit Atrial Muscle as a Mechanism of Tachycardia

III. The "Leading Circle" Concept: A New Model of Circus Movement in Cardiac Tissue without the Involvement of an Anatomical Obstacle

MAURITS A. ALLESSIE, FELIX I.M. BONKE, AND FRANCIEN J.G. SCHOPMAN

SUMMARY In small pieces of rabbit atrial myocardium, sustained periods of circus movement tachycardia were produced by the induction of a single properly timed premature impulse. By use of multiple intracellular and extracellular electrodes the spread of activation during the tachycardia could be analyzed accurately. Because in the present experiments there was no gross anatomical obstacle for the impulse to circulate around, we paid special attention to phenomena occurring in the center of the circus movement. We found that in the absence of an inexcitable central obstacle the center of a circus movement was invaded by multiple centripetal wavelets which converged in the very center of the circuit. On the basis of these observations we developed a new model of circulating excitation in cardiac tissue. The properties of this model (referred to as the "leading circle concept") were compared with the behavior of circus movement around the anatomical obstacle. It turned out that both types of circus movement tachycardia responded differently to changes in basic electrophysiological properties such as conduction velocity and refractory period. For example, addition of carbamylcholine to the tissue bath caused a marked acceleration of the leading circle tachycardia, whereas circus movement in a ring of atrial tissue was hardly affected. On the other hand, depression of conduction velocity by exposure to moderate concentrations of tetrodotoxin had a more pronounced effect on circus movement in the ring preparations than on tachycardias based on a leading circle mechanism. Finally we suggest the use of the strength-interval curve—after some modification—to describe and predict the behavior of a leading circle tachycardia.

IN OUR SERIES of papers on circus movement in rabbit atrial muscle we were able to show that in a small piece of atrial myocardium the induction of a properly timed premature beat can force the impulse to conduct in a circuitous route and thus set the stage for a period of tachycardia. We gathered evidence that the naturally existing nonuniform recovery of excitability in the atrium was of major importance for the occurrence of unidirectional block of the premature impulse, which, of course, is a prerequisite for the onset of circus movement. Furthermore, by use of a technique for synchronous multiple microelectrode recordings we obtained detailed information about the cellular responses during the initiation of the circus movement. However, thus far no conclusive model of circus movement in the absence of an anatomical obstacle could be derived. This was due mainly to a lack of information about what happens in the center of a circulating impulse. In most studies on circus movement and reentry, the model introduced by Mines in 1913 has been used. This model is based on observations in ring-shaped strips of cardiac tissue and implicitly supposes the presence of some kind of gross anatomical obstacle. However, in many cases of tachycardia, as in our experimental studies, circus movement without the involvement of a central obstacle seems likely. Therefore, we do not consider the model of Mines suitable to describe such a type of circus movement tachycardia.

In this paper we report the results of multiple microelectrode recordings from the center of sustained circus movement in small segments of isolated rabbit atrium. From these complementary studies a new model of circus movement can be derived that is applicable to circulating excitation in the absence of a nonexcitable central obstacle. This model, which we called the "leading circle" concept, differs in various respects from the conventional model of circus movement. Both models respond differently to changes in basic electrophysiological conditions such as the steepness of phase zero of the action potential, the conduction velocity of the impulse, and the duration of effective and relative refractory periods. Also the mechanism for interruption of the circulating excitation may differ in both types of circus movement.

Methods

The preparation, perfusion system, stimulating and recording techniques, and the method for data processing were the same as described previously. The isolated left atrium of the rabbit was regularly paced at an interval of 500 msec. Tachycardias were produced experimentally by the induction of a single properly timed premature stimulus (duration, 1 msec; intensity 4 times diastolic thresh-
The spread of activation through the preparation was measured either with 10 unipolar surface electrodes or with a multiple microelectrode technique. To study the course of the excitation during tachycardia with a limited number of recording leads, several precautions have to be taken. One possible approach, which we have used, is to induce tachycardias in a reproducible way. If the subsequent periods of tachycardia are identical, the course of the excitation wave can be mapped by combining the results obtained during each separate period of tachycardia. However, since slight differences in the time course of the tachycardias are cumulative, in most cases the requirement for identity was fulfilled only during the first beats. In a later stage of the arrhythmia it was inappropriate to relate time measurements obtained from different episodes of tachycardia. To map the excitation process, then, another procedure need be followed. The complete mapping must be accomplished during the progress of one single sustained tachycardia. Furthermore, during the mapping procedure the rate and rhythm of the tachycardia must stay constant and the mechanism underlying the arrhythmia must not change. Under normal conditions, generally the induction of a premature beat resulted only in short runs of rapid repetitive activity. However, the addition of carbamylcholine to the tissue bath (10^{-6} to 10^{-7} g/ml), which shortened the refractory periods to about 40 to 50 msec, was an appropriate method not only to facilitate the induction, but also to favor continuation, of the tachycardia. Under these conditions we frequently could produce tachycardias which were sustained for more than 30 minutes; during that time rate and rhythm of the tachycardia varied only by a trifling amount. This period was sufficient to map the spread of activation either with 10 extracellular or with 10 intracellular electrodes. Successive registrations were time-aligned relating the measurements to records obtained through one or more fixed extracellular reference leads. This procedure not only resulted in an accurate measurement of the excitation pattern during sustained tachycardia, but also allowed us to make intracellular recordings from the center of the circus movement.

Results

INTRACELLULAR RECORDINGS FROM THE CENTER OF CIRCUS MOVEMENT

Figures 1 and 2 show the results of an experiment in which the excitation process during sustained tachycardia was mapped with the multiple microelectrode technique. Similar results were obtained in two other experiments. In this case it was very difficult to evoke tachyarrhythmias under normal conditions. Only one area could be found where a premature stimulus (coupling interval = 70 msec) resulted in just one extra nonstimulated beat. After addition of carbamylcholine (3 x 10^{-7} g/ml) the refractory period decreased to about 40 msec. Single early premature stimuli now easily induced tachycardias which had a strong tendency to continue. Not only the timing of the test stimulus but also its point of application now were much less critical. This marked effect of carbamylcholine was observed in most instances. The map at the right side of Figure 1 clearly shows that the tachycardia was based on circus movement of the impulse through the myocardium. This activation pattern resulted from measurements of the time of action potentials of 94 different fibers. All these intracellular recordings were made within a period of 42 minutes, during which the tachycardia showed only minimal variations in cycle length (105 ± 5 msec). At the left of the map the transmembrane potentials of five fibers (A-E).
CIRCUS MOVEMENT IN RABBIT ATRIA/Allessie et al.

FIGURE 2  The same experiment as in Figure 1. The membrane potentials of seven fibers (marked A, D, and 1–5) located on a straight line through the center of the circus movement are shown. Fibers A and D are the same as in Figure 1. These records demonstrate that the central area is activated by centripetal wavelets. Note that the fibers in the central point of the circuit (fibers 3 and 4) show double responses of subnormal amplitude. Both responses are unable to propagate beyond the center, thus preventing the impulse from shortcutting the circuit. Below the map the activation pattern is given schematically. It shows the leading circuit with the converging wavelets in the center. Block is indicated by double bars.

...lying along the circular route, are shown. The action potentials are of markedly short duration; this is caused by the influence of carbamylcholine. It should be emphasized, however, that although the effective refractory period also was shortened by carbamylcholine, the effect on the action potential duration was greater than the effect on the refractory period. Therefore, under the influence of carbamylcholine the effective refractory period outlasted the duration of the action potential, i.e., for some time after full repolarization the fibers still were inexcitable.

Figure 2 shows intracellular recordings from seven fibers located on a straight line through the center of the circus movement. The most peripheral fibers (A and D) are the same as in Figure 1. The fibers in the center are marked by digits (1–5). As can be seen from the measurements of activation times, of these fibers, the central area of the circuit was activated in a centripetal direction. From fiber A the impulse excited fibers 1, 2, 3, and 4 in that order. When penetrating deeper into the center of the vortex, the centripetal wavelet lost more and more of its "stimulating efficacy" until it was unable to excite the tissue ahead. Going from fiber 1 to 4, the amplitude, rate of rise, and duration of the responses all gradually decrease and finally result in complete extinction of the impulse somewhere between fibers 4 and 5. At the opposite side of the circuit essentially the same sequence of events takes place. There, half a revolution time later, the circulating impulse penetrates the center again, traveling from fiber D to fibers 5, 4, and 3. Again the centripetal wavelet is conducted with decrement that results in extinction of the impulse between fibers 3 and 2. As a result of this course of events, fibers 3 and 4, which were located in the very center of the circus movement, showed only local responses. Because this area was invaded twice during each revolution of the impulse, the frequency of these local responses was double the rate of the tachycardia.

In a previous paper we speculated about the mechanism which might explain why, during tachycardia, the fibers in the center of the circus movement are not activated although during regular driving of the preparation the same fibers show a normal response: "A possible explanation is that the membrane potential of these fibers is held above threshold by the electronic influence of the depolarization front which continuously turns around this area. In this way the center is functionally inexcitable, and the activation wave will travel around this functional ob-
The intracellular recordings from the center of sustained circus movement, presented here, show that this speculation is incorrect. The membrane resting potential of fibers 3 and 4 does not show such a degree of depolarization that they can be expected to be inexcitable. On the contrary, the center of the vortex is continuously invaded by multiple centripetal wavelets which are blocked in the very central circuit. Here the impulse encounters fibers that are still in their refractory phase because they were already activated by another centripetal wavelet just half a revolution time earlier. In this way the circulating impulse is prevented from shortcircuiting the circuit, whereas the area of converging wavelets serves as an "obstacle" for the impulse to turn around. In the diagram beneath the map in Figure 2 the sequence of excitation is summarized schematically. It can be described as a "leading" circulating wavefront which not only activates the periphery but from which also centripetal wavelets are emerging to collide in the center of the circuit. In this experiment the diameter of the leading circle was about 6 mm. Hence the total length of the circular pathway can be estimated to be about 20 mm.

**EXTRACELLULAR RECORDINGS FROM THE CENTER OF CIRCUS MOVEMENT**

Although the technique of multiple microelectrode recording allows detailed analysis of the electrical behavior of different individual fibers in a wide range of experimental circumstances, its use is limited because of the delicacy of the technique. Since measurement of the course of excitation through cardiac muscle with extracellular leads is much more simple, and because surface electrodes have a much wider field of application, we also examined whether the mechanism in the center of circus movement could be studied with this method. Figure 3 shows the results of an experiment in which the spread of excitation during a sustained tachycardia was mapped with multiple extracellular leads. In this experiment again carbamylcholine (2 × 10⁻⁶ g/ml) was added to the tissue bath to facilitate the induction and to favor the perpetuation of tachycardias. The map reveals that during tachycardia the impulse was rotating in a clockwise direction with a revolution time of 80 msec. At the bottom of the figure the central part of the map is enlarged and is given together with a number of electrograms recorded from this area. Accurate analysis of these electrograms again demonstrates the existence of multiple centripetal wavelets converging into the center of the circuit. In this case, however, rather than collision of the wavelets at one single point, there was an extended area in which the centripetal impulses were blocked. In the diagram in the right upper corner of the figure the spread of excitation in the central area is given schematically as it can be deduced from the multiple electrograms. The area of block is indicated by hatching. In the tracings the corresponding blocks are indicated by double bars. In all panels the tracing indicated by an asterisk is recorded at the most peripheral site. (Note that the lower trace of panel D is the same as the upper trace of panel E). As in the case of intracellular recording (Fig. 2), in the area of collision the electrograms show two clearly separate responses. For instance, in panel C, the upper trace (recorded from the left site) shows an activation 75 msec after the zero reference. The lower trace (right site) shows a single response at 22 msec. Since the cycle length of the circus movement was 80 msec, this means a time lapse of 27 msec between the activation of the two closely approximated sites. The middle trace of panel C shows that this delay is not caused by slow conduction, but by a conduction block between the two sites. During each cycle of the circus movement the middle trace shows two distinct complexes. The first, of low amplitude (t = 77 msec), is almost simultaneous with the deflection in the upper trace. The second complex (t = 25 msec) is temporally associated with the lower recording. Thus, each of the two complexes of the middle trace belongs to a separate centripetal wavelet coming from an opposite direction and colliding at the middle one of the three recording sites. The same can be seen in panel D. In the other panels block of the centripetal wavelets also can be recognized. There, however, the double complexes in the middle trace are less clearly separated because the detour of the impulse around the site of block is relatively small. Thus also with extracellular recordings one can get the necessary information to identify this type of circus movement.

**THE EFFECT OF CARBAMYLCHOLINE, TETRODOTOXIN, AND TEMPERATURE ON CIRCUS MOVEMENT TACHYCARDIA**

To elucidate whether the type of circus movement described above responds differently to changes in electrophysiological properties from circus movement around an anatomical obstacle, we studied the effect of carbamylcholine, tetrodotoxin (TTX), and temperature on the rate of both types of tachycardia. In these experiments the left atria of two rabbits were isolated and put together in the same tissue bath. One preparation was kept intact while the other was transformed into a ringlike structure by making multiple incisions as indicated in Figure 4. Cutting the preparation in this way, the length of the ring was about 40 mm. With the single-stimulus method sustained circus movement was then induced in either preparation. Generally in ring preparations tachycardia could be induced more easily than in the intact segments of the atrium; the tachycardias in the ring also tended to last longer. Multiple extracellular recordings confirmed that the tachycardia was based on a circuitous sequence of excitation through the ring. As in the intact preparation, in the ring preparations also the site of application of the test stimulus was important, but in most preparations it was not too difficult to find an area where the test stimulus started tachycardia. The rate of tachycardia in the ring always was lower than the one in the intact segment of atrium. After revolution time of both independent circulating excitations had become constant, carbamylcholine or TTX was added to the tissue bath and the effect on cycle length of the two tachycardias was recorded. Figure 5 shows the effect of carbamylcholine (2 × 10⁻⁴ g/ml). The example is representative of results of four experiments. The concentration of carbamylcholine used caused a shortening of the effective refractory period up to about 40-50 msec; conduction velocity increased only slightly.
FIGURE 3  Analysis of excitation during sustained tachycardia with multiple extracellular electrodes. At the top the map of the spread of activation is shown together with a schematic representation. In the latter the dimensions of the leading circuit and the centripetal wavelets are shown. The hatched area in the scheme indicates the area where the centripetal wavelets are blocked. In the lower part of the figure, the central part of the map is enlarged and given together with groups of three unipolar electrograms (A–F) recorded from that area. The sites of recording are indicated on the map, the most peripheral registrations being marked by asterisks. In the groups of electrograms the corresponding sites of conduction block are indicated by double bars. See text for further explanation.
As a control, the cycle length of a spontaneously beating isolated right atrium was also recorded. Prior to the administration of carbamylcholine the cycle length of sinus rhythm was 365 msec. The revolution time of the impulse through the ring of atrial muscle amounted to 168 msec, whereas the cycle length of the tachycardia in the intact preparation was 143 msec. All three rhythms responded differently to the action of carbamylcholine. As expected, sinus rhythm slowed, and the beat-to-beat interval of the isolated right atrium increased from 365 to 800 msec. In contrast the circus movement in the ring of atrial muscle was hardly affected. Carbamylcholine caused only a slight shortening of the cycle length from 168 to 162 msec and this change can adequately be explained by the minute increase in conduction velocity brought about by the drug. The tachycardia in the intact piece of atrial muscle, however, showed a marked acceleration, the circulation time being substantially reduced from 143 to 116 msec. Frequently the application of carbamylcholine caused a sudden termination of the tachycardia in the intact preparation; in the ringlike preparations this was never observed. This marked difference in the effects of carbamylcholine on the two different atrial preparations strongly points to the existence of two different types of circus movement tachycardia (see Discussion).

Three experiments were made to study the effect of TTX on circus movement with and without an anatomical obstacle. Figure 6 shows an example. TTX was used in such low concentration (3 × 10⁻⁷ g/ml) that impulse conduction was slowed but not completely blocked. Measurement of strength-interval curves before and during the administration of TTX made clear that in these concentrations TTX did not affect the time course of the restoration of excitability. In both models of circus movement the

---

**Figure 4** Panel A: endocardial view of intact segment of left atrium consisting of part of the body of the atrium with the adjacent appendage. To transform the preparation into a ringlike structure, multiple cuts were made as indicated by the dotted lines. Panel B: ring preparation of atrial muscle as it results from the multiple incisions.

**Figure 5** The effect of carbamylcholine (2 × 10⁻⁶ g/ml) on sinus rhythm, tachycardia in a ring preparation, and tachycardia in an intact segment of the atrium. All three rhythms respond differently to the administration of carbamylcholine. Whereas sinus rhythm is slowed and the tachycardia in the intact atrium is accelerated, the circus movement in the ring of atrial muscle is hardly influenced by this intervention.
decrease in stimulating efficacy and conduction velocity of the impulse induced by TTX resulted in a deceleration of the tachycardia. However, the effect was more pronounced in the ring preparation than it was in the intact segment of the atrium. In this example the revolution time of the circulating excitation in the ring preparation increased from 138 to 172 msec (25%), whereas in the intact piece of atrial muscle revolution time increased from 117 to 129 msec (11%). This difference in behavior again suggests a different underlying mechanism.

The effect of temperature on circus movement tachycardia was studied in three experiments. It is known that cooling of the heart causes a decrease in the rate of depolarization, whereas the duration of the action potential is markedly prolonged. Consequently conduction velocity is depressed and the refractory period is lengthened. These changes in electrophysiological properties resulted in a marked diminution of the rate of the tachycardia. Figure 7 shows the temperature-dependency of circus movement in a ringlike structure and in an intact piece of the myocardium. At 37°C revolution time was 172 and 134 msec, respectively. Lowering the temperature resulted in a gradual lengthening of the beat-to-beat interval. At 29°C the cycle length of the tachycardia in the ring of atrial tissue had increased from 172 to 288 msec (67%), whereas the interval in the intact preparation had become 215 msec (60% increase). Thus, although in both models the effect on the circulation time was considerable, no clear difference in their response to temperature change could be observed.

Discussion

THE LEADING CIRCLE CONCEPT

From the results described above, a new model of circus movement in cardiac tissue emerges. When there is no anatomical obstacle which defines the length of a circular pathway, the circuit in which the impulse circulates is completely defined by the electrophysiological properties of the fibers composing the circuit. Under these circumstances the smallest possible pathway in which the impulse can continue to circulate is the circuit in which the stimulating efficacy of the circulating wavefront is just enough to excite the tissue ahead which is still in its relative refractory phase. In other words, in this smallest circuit possible, which we designated as the “leading circle,” the head of the circulating wavefront is continuously biting in its own tail of refractoriness. Because of this tight fit, the length of the circular pathway equals the “wavelength” of the circulating impulse (i.e., product of conduction velocity and
refractory period). In the center of the leading circle, dimensions are too small for a sustained circus movement. Within this area a circulating impulse would encounter tissue in which excitability has not yet recovered sufficiently. As a consequence, the conduction velocity of the impulse would be secondarily depressed below some minimal value at which successful impulse propagation is no longer possible. In fact, the area within the leading circle is activated by centrifugal wavelets that arise from the leading circle and collide in the very center of the circus movement. Going from the leading circuit to the periphery, the length of a circular pathway becomes longer and longer. Since a longer pathway implies a longer revolution time, it also is evident that the fibers in the periphery will be excited by wavefronts emerging from the leading circle, rather than forming part of a longer circuit. In Figure 8 the leading circle concept is compared with the model of circus movement around an obstacle as proposed by Mines. The main features of the leading circle model are: (1) The length of the circuit is determined not by anatomical but by electrophysiological properties. (2) The dimensions of the circuit are not fixed but may change with alterations in electrophysiological properties, such as conduction velocity, upstroke of the action potential, and time course of recovery of excitability. (3) Because there is a tight fit between the crest and the tail of the impulse there is no excitatory gap in the circuit. This implies that a wavefront (or stimulus) of greater efficacy than the circulating impulse is required to interfere with the leading circle. This may have some important implications for the degree of protection of such a focus of reentrant activity. (4) Because the center of the circuit consists of excitable tissue, under some circumstances the impulse may succeed in crossing the center. In comparison with the Mines model such a short circuit offers an additional mechanism for the interruption of tachycardia. (5) Whereas the revolution time of a circus movement in a large anatomically defined circuit is inversely related to the conduction velocity of the impulse, in the leading circle model revolution time is primarily proportional to the time course of recovery of excitability of the fibers composing the circuit (see below).

**THEORETICAL CONSIDERATIONS ON THE LEADING CIRCLE MODEL**

As indicated by Lewis, three factors must be considered in any theory of circus movement, namely, the length of the conduction pathway (P), the conduction velocity (V), and the duration of the refractory period (R). According to this theory, circulating excitation can take place only when the path length equals or exceeds the product of conduction velocity and refractory period (P ≤ VR). When, in case of circus movement within an anatomically defined loop of excitable tissue, the wavelength of the impulse (VR) is shorter than the length of the circuit (P), there is a gap of full excitability between the crest and the tail of the circulating impulse. In this situation conduction velocity is not influenced by changes in refractory period. Since revolution time = P/V, and the length of the pathway is fixed, in this type of circus movement revolution time varies inversely with conduction velocity (revolution time ~ 1/V). However, in many cases the situation cannot be described adequately in this simple manner. When the revolution time of circulating excitation is too short to allow full recovery of excitability of the fibers in the circuit the depolarization wave will travel through relatively refractory tissue and consequently the conduction velocity will be reduced. In this situation conduction velocity (V) and refractory period (R) are no longer independent variables but on the contrary they are inextricably linked.

In the leading circle concept the length of the circuit is variable, being defined by and equal to the wavelength of the circulating impulse (P = VR). This means that the revolution time of the impulse is not determined by the length of a given circuit but by the length of the circulating impulse itself. Since revolution time = P/V, and P = VR, in the leading circle model the revolution time is proportional to the refractory period (revolution time ~ R). In fact, however, the process of recovery of excitability is too complex to be described by just one single value of the duration of refractoriness. A more accurate way to express the time course of restoration of excitability is to measure the strength-interval curve. Such a curve depicts the minimal stimulus strength which is required to induce propagated excitation at different moments of prematurity, or, vice versa, it gives the refractory period for stimuli of different intensities. With some modification, this well known and easily measurable electrophysiological relationship gives a much better description of the properties of the leading circle than the oversimplified relation, revolution time ~ R. In the pathway of the leading circle the situation is very similar to the situation described by the strength-interval curve. In either case, the strength of the stimulus or the stimulating efficacy of the circulating wavefront, if just enough to initiate, or maintain propagation. Therefore on the ordinate of the strength-interval curve we can replace stimulus strength by stimulating efficacy of the circulating impulse. On the abscissa the interval then represents the interval of the tachycardia, i.e., the revolution time of the circus movement (see Fig. 9). This modi-

---

**Figure 8** Comparison of the properties of circus movement with and without the involvement of a central anatomical obstacle.

1. Length of circuit determined by conduction velocity, stimulating efficacy and refractory period
2. Length of circular pathway fixed
3. Excitable gap between crest and tail of the impulse (entire part of circuit)
4. Impulse can not penetrate the circuit
5. Revolution time inversely related to conduction velocity
6. Length of circuit determined by conduction velocity, stimulating efficacy and refractory period
7. Length of circuit can change with alterations in electrophysiological properties
8. No gap of full excitability
9. Stimulus of the circuit possibly
10. Revolution time proportional to refractory period
**Changes in Rate of Leading Circle Tachycardia by Alterations in Electrophysiological Properties**

Figure 9 shows how the strength-interval curve can be used to predict changes in revolution time of a leading circle tachycardia in response to changes in some basic electrophysiological properties. Panel A illustrates the effect of a decrease in amplitude and rate of rise of the action potential (taken together as stimulating efficacy). When during a certain tachycardia with a revolution time \( a \), the stimulating efficacy of the circulating wavefront is diminished, according to the modified strength-interval curve, the revolution time will increase from \( a \) to \( b \). The experimental imitation of this situation by the application of TTX indeed revealed such a decrease of the rate of the tachycardia (see Fig. 6). In case of a shortening of the refractory period the strength-interval curve shifts to the left (broken line, panel B). If one assumes that the stimulating efficacy of the circulating excitation is constant, the given shift in the strength-interval curve results in a shortening of the revolution time from \( a \) to \( b \). The observed acceleration of the leading circle tachycardia caused by the application of carbamylcholine (Fig. 5) is in good agreement with this explanation.

However, a selective change either in stimulating efficacy or in refractory period is rarely met in nature. Most changes in electrophysiological properties, occurring spontaneously or induced by certain cardiac drugs, affect both the upstroke of the action potential and the time course of restoration of excitability. In panels C and D of Figure 9 are two examples of such an influence on the leading circle model. In panel C the combination of a decrease in stimulating efficacy and a lengthening of the refractory period is shown. Both the shift in the strength-interval curve to the right (broken line curve in panel C) and the decrease in the stimulating efficacy of the action potential cooperate to prolong the revolution time of the leading circle from \( a \) to \( b \). As a consequence the tachycardia will decelerate considerably. Since cooling of the heart exerts the abovementioned combination of effects, we can take the temperature experiments for comparison (Fig. 7). The relatively strong slowing of the tachycardia which we found to be associated with a decrease in temperature again seems to confirm the validity of the model. The same effect can be expected from cardiac drugs such as quinidine, procainamide, and \( \beta \)-adrenergic blocking agents.

In other situations the changes in stimulating efficacy and refractory period will have an opposite effect on the interval of the tachycardia. An example of a situation in which a decrease in stimulating efficacy is coupled with a shortening of the refractory period is a sudden increase in heart rate. After a sudden increase in heart rate, amplitude and \( dV/dt_{\text{max}} \) of the action potential gradually decrease until only after a large number of beats (100 or more) a new steady state is reached. At the same time the refractory period is gradually shortened. The sudden onset of a paroxysm of tachycardia thus is followed by a period in which the stimulating efficacy of the impulse gradually decreases while the refractory period is shortened. Whether, as a result of these changes, the cycle length of the tachycardia is gradually shortening ("warm-up" phenomenon) or lengthening will depend on the degree to which both parameters are influenced. The paroxysms of tachycardia produced experimentally in the isolated left atrium of the rabbit always showed a gradual increase in cycle length during their initial phase. As tachycardia progressed, cycle length became constant after about 100 beats. Panel D of Figure 9 shows how this phenomenon can be explained on the basis of the leading circle model. The solid curve represents the strength-interval curve during basic rhythm (interval = 500 msec). The dotted line represents the strength-interval curve after...
the tachycardia has reached a steady state (interval = about 130 msec). During the first 100 beats of the tachycardia the strength-interval curve is gradually shifting to this steady state value. At the same time the stimulating efficacy of the circulating wavefront is gradually decreasing. When, as in this example, the effect of the decrease of the simulating efficacy on the revolution time exceeds the effect of the shortening of the refractory period, the net result will be a gradual lengthening of the interval of the tachycardia from a to b. When, on the other hand, the shortening of the refractory period is more marked and the change in the upstroke of the action potential is more moderate, the net result can be the opposite. After initiation of circus movement the revolution time will then get shorter and the tachycardia will show the "warming-up" phenomenon.

CHANGES IN DIMENSIONS OF THE LEADING CIRCUIT

The diameter of the leading circuit in the present experiments was about 6–8 mm. This means that a minimal area of approximately 30–50 mm² of atrial muscle is required to accommodate the circus movement. This is in remarkably good agreement with previous studies of West and Landa,12 who demonstrated that it is not possible to induce a sustained arrhythmia in segments of rabbit atrial muscle smaller than a critical mass of 30 mg. An important feature of the leading circle model is that the length of the reentrant pathway is determined by the electrophysiologic properties of the tissue. Changes in refractory period, conduction velocity, and stimulating efficacy will all influence the dimensions of the circuit. An acceleration of the process of recovery of excitability shortens the wavelength of the circulating excitation. Since in the leading circle the length of the circular pathway equals the wavelength of the impulse, a shortening of the refractory period will lead to a shortening of the circuit. The decrease in cycle length of tachycardias in intact pieces of atrial myocardium as observed after application of carbachol (Fig. 5) is therefore probably associated with a shortening of the circular pathway. Such a reduction in the dimensions for circus movement also can explain why acetylcholine and its derivatives highly favor the induction and perpetuation of tachycardia13 and why these drugs frequently convert tachycardia into fibrillation.14 If one considers fibrillation as a state in which more than one wavelet is circulating through the myocardium,15 the chance for the development of this situation is high either in large hearts or in tissue in which small circuits are possible.

Finally, we want to emphasize that the two different types of circulating excitation, as contrasted in this paper, should be considered as extremes. In such a complex organ as the heart there is a good chance for the intermingling of these forms of circus movement. If, instead of the presence of a gross anatomical obstacle, there are pathways of preferential conduction, circus movement tachycardia may arise, with properties somewhere in between those of the leading circle model and circus movement in a large loop of cardiac tissue.

Acknowledgments

We are grateful to Prof. Dr. L.N. Bouman and Dr. T. Blangé for the helpful discussion during this study, and to Bebbvy van der Mars for secretarial help.

References

14. Lewis T, Drury AN, Bulger HA: Observations upon flutter and fibrillation. VII. The effects of vagal stimulation. Heart 8: 141-170, 1921
Circus movement in rabbit atrial muscle as a mechanism of tachycardia. III. The "leading circle" concept: a new model of circus movement in cardiac tissue without the involvement of an anatomical obstacle.

M A Allessie, F I Bonke and F J Schopman

_Circ Res._ 1977;41:9-18
doi: 10.1161/01.RES.41.1.9

_Circulation Research_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1977 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circres.ahajournals.org/content/41/1/9.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation Research_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation Research_ is online at:
http://circres.ahajournals.org/subscriptions/