Activation Mapping of Reentry Around an Anatomic Barrier in the Canine Atrium: Observations During Entrainment and Termination

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We determined total right atrial activation sequences during entrainment and termination of flutter induced in dogs with a surgically induced atrial lesion. This type of atrial flutter is due to a complex interaction between atrial and ventricular tissue. By constructing isochronal maps, we demonstrated the pattern of atrial activation during pacing protocols that either entrained or entrained and then terminated the reentrant rhythm. We show that during pacing, the orthodromic wavefront from the paced impulse (A_n) collides with the anterograde wavefront from the previous paced impulse (O_{n-1}). During entrainment, the site of collision of the orthodromic and anterograde wavefronts is constant during pacing at a fixed rate but shifted in the anterograde direction as the pacing rate increased. Furthermore, the last paced beat was entrained only up to the site of collision of the previous paced beat. During one period of entrainment, termination of the reentrant arrhythmia occurred because O_{n-1} blocked in the reentrant pathway due to refractory tissue left by O_{n-2}. However, subsequent A_n did not collide directly with O_n, as was expected, but rather O_n blocked by an interaction with tissue left refractory by A_n. Because O_n was blocked, no reentry occurred when pacing ended. (Circulation 1989;79:406-416)

Entrainment of a tachycardia is “an increase in the rate of the tachycardia to a faster pacing rate, with resumption of the intrinsic rate of tachycardia upon either abrupt cessation of pacing or slowing of the pacing rate below the intrinsic rate of tachycardia.”1 Atrial and ventricular arrhythmias occurring in animals and humans have been entrained.1-7 For human atrial flutter, Waldo and his colleagues1-5 have described several criteria, any one of which, if present, establishes entrainment of a tachyarrhythmia. They judged that a rhythm that could be entrained and met these criteria was most likely caused by reentrant excitation due to circus movement. Satisfaction of criteria during entrainment protocols seemed necessary to rule out other possible types of activation that could occur during rapid pacing during a tachycardia. For instance, rapid pacing could cause overdrive acceleration of an arrhythmogenic nonreentrant focus8-12 or could cause termination of one and reinitiation of another reentrant tachycardia by the paced impulses. Finally, rapid atrial pacing could cause one arrhythmia to change to another with the same or a different site of origin.

Waldo et al established criteria through studies on patients with atrial flutter or preexcitation using multiple (up to five) recordings obtained simultaneously during the rhythm, during pacing, and during its termination. However, in these studies, total sequence of activation in and around the reentrant circuit during reentry and entrainment was assumed but not documented.

We have described a model of canine atrial flutter that satisfies Waldo et al’s criteria for entrainment. By recording simultaneously from 96 endocardial sites, we have shown that the atrial flutter is due to movement of the impulse in the supraventricular annulus of the tricuspid ring.6,13 Fast response action potentials are recorded from the fibers throughout the reentrant pathway and analysis of premature stimuli that reset the tachycardia shows that during the tachycardia there is a partially excitable gap.13

In this report, we describe the activation sequences of the atrial tissue in and immediately surrounding the reentrant circuit during entrainment of this type of reentrant rhythm. We also describe a mechanism...
by which overdrive pacing entrains and then terminates this type of reentry and that has not yet been described for reentrant tachycardias.

Methods

For these studies, we mapped right atrial endocardial activation sequences in the isolated, coronary artery perfused canine heart during sustained flutter and during pacing. To concentrate on the details of the activation pattern in and around the known reentrant circuit, all electrodes were placed on right atrial sites. We did not determine the sequence of activation of the adjoining left atrium. In a previous study, we have shown that sites in the left atrium were not part of the reentrant pathway and that during flutter the left side of the intraatrial septum was activated by a broad wavefront that spread from the right side. For this reason, data from reentry protocols were analyzed from experiments on three hearts with stable flutter.

To induce stable flutter, a Y-shaped incision was made in the right atrium extending from the superior to inferior vena cava and across the right atrium to the base of the appendage. In two dogs, this lesion was made just after the heart had been isolated and the electrode arrays put in place. In the third dog, the lesion had been made 2.5 months before isolation of the heart. During this interval, stable flutter could be induced at will and pacing protocols gave results similar to those previously described.

Isolated Heart Technique and Electrode Arrays

The procedure we used for the isolated blood-perfused canine heart has been described previously. The fixed array of bipolar electrodes used to record endocardial electrograms from 96 sites on the right atrium has also been described. This flexible right atrial (RA) electrode array was inserted into the atrium through a ventriculotomy and then through the tricuspid ring. It contained 96 bipolar pairs of electrodes and signals from all pairs could be recorded simultaneously.

Study Protocol

During pacing from an epicardial site that was adjacent to the circus path, either a critically timed premature stimulus or a burst of rapid stimuli induced atrial flutter in all three hearts. After recording the bipolar electrograms from 96 electrode sites during the flutter, pacing protocols were started with stimuli applied at the same epicardial site.

Figure 1. Panel A: Maps of entire two-dimensional representation of the electrode array used. For this projection, the right atrium has been opened, the free wall folded downward and the tissue to the left of the intercaval lesion flattened to the left. Numbers correspond to the electrode number and its location within the array. Superior and inferior vena cavae (SVC, IVC), right atrial appendage (App), and tricuspid valve orifice (TO) are indicated. Interrupted lines indicate the portion of the grid array that is represented in subsequent maps. Panel B: Tracings of representative electrograms recorded simultaneously from multiple endocardial sites during an episode of atrial flutter. The number below each trace indicates the electrode number and corresponds to an atrial site indicated in Panel A. The response to the last two stimuli (S_n-1, S_n) of a train (BCL, 150 msec) and the next two beats of the tachycardia (T1, T2) are shown. Numbers just above electrograms indicate cycle length. The time bar is 150 msec. V, time of ventricular activation; dots, site of earliest activation by the pacing stimuli; arrows, direction of activation.
Rapid atrial pacing was started at a cycle length 5–10 msec less than the average cycle length of the stable flutter. Epicardial pacing electrodes were located on the right atrial freewall below the surgical lesion and adjacent to the tissue of the supravalvular annulus of the tricuspid ring. In one heart, an epicardial pacing electrode was also sutured on the aortic wall adjacent to Bachmanns bundle. During a protocol, pacing was continued for 5–10 seconds to determine if entrainment had occurred. Then pacing was terminated abruptly. If the flutter persisted, rapid atrial pacing from the same site was started at a cycle length 5–10 msec less than the first overdrive cycle length tested. The pacing cycle length was shortened in steps of 10 msec until the arrhythmia was terminated or until atrial refractoriness prevented capture by every stimulus.

**Data Acquisition and Analysis**

For data acquisition, electrograms were amplified by a bank of programmable amplifiers (band width, 10–1,000 Hz) and then fed into an eight-bit analog-to-digital converter and multiplexing system (Preston, Anaheim, California). An automatic gain control was used periodically during the experiment to adjust the gain of each amplifier so that the amplified electrogram used the 10-V range of the A/D converter. When recording the 96 electrograms, the sampling frequency was 4,000 samples/sec and the sampling interval was 0.25 msec/channel. The digitized data were recorded on tape with an Ampex (Redwood City, California) high-bit tape recorder. Files constructed from tape were stored on disk for analysis. Activation sequences during selected time periods were constructed after the experiment with a PDP 11/34 or Microwax system (Digital Equipment Corp, Maynard, Massachusetts). A Tektronix (Portland, Oregon) 4012 high resolution graphics terminal and hard copy unit were used to display electrograms for the marking of activation times by the user. These times were then displayed on a two-dimensional representation of the electrode array used in the experiment. Figure 1A shows a two-dimensional representation of the right atrial endocardial electrode used to construct maps in this study. In general, the activation time was marked at a point where a predominantly biphasic electrogram crossed the baseline after the first major deflection. In some recordings, multiple component electrograms were seen. Care was taken to first identify all components that were due to ventricular activation. This was done by comparing the timing of the electrogram component with the time of a ventricular electrogram. For multiphasic atrial electrograms, the time of the major deflection was marked, and in electrograms of apparent long duration, a midpoint was defined and marked. Each of the deflections in a multiphasic electrogram may indicate activity at the recording site particularly when the atrial tissue is trabeculated. Isochrones were drawn by computer or by hand.

**Results**

*Demonstration that there is a constant site of collision during pacing at a constant rate faster than the rate of the tachycardia except for the last paced beat, which is entrained but does not collide.*

We have previously described the pattern of total right atrial activation during sustained atrial flutter in these hearts. During overdrive, after several pacing stimuli, the fibers in the reentrant pathway are captured and the rhythm is entrained. Figure 1B illustrates the electrograms recorded, and Figure 2 shows the sequence of right atrial activation during one episode of documented entrainment of an episode of flutter [atrial cycle length (ACL), 170 msec] at a cycle length (CL) of 150 msec. Pacing in this example and all figures was from the freewall electrode site. In Figure 2, the sequences of activation caused by the last two entraining stimuli (S<sub>n−1</sub> and S<sub>n</sub>) are shown as are the first two beats of the subsequent tachycardia (T1 and T2). In this figure, each panel represents a small time window during the episode, but the time windows are continuous. That is, the front of the wave depicted by the white arrow (depicted by O<sub>n−1</sub>) in Figure 2A continues and becomes the stippled arrow in Panel B, and the white arrow (O<sub>n</sub>) in Panel B continues in Panel C as the stippled arrow.

During circus movement of the impulse around the tricuspid valve orifice, entrainment resulted from entry of the paced wavefront during the partially excitable gap. Subsequently, during each paced beat there is a constant site of collision of the antidromic wavefront from the paced impulse (e.g., A<sub>n</sub>) with the orthodromic wavefront of the previous circulating paced impulse in the reentrant path (O<sub>n−1</sub>) (Figure 2B). In addition, when the rhythm is entrained, the orthodromic impulse follows a pathway identical to that of the nonentrained reentrant impulses of the flutter (Figure 2D).

The activation patterns observed during an episode of entrainment are different than the activation patterns observed during atrial pacing from the same site during normal rhythm. Maps during pacing in the absence of flutter show that there was a significant clockwise and counterclockwise penetration of the impulse into the atrial tissue surrounding the valve (data not shown). The paced wavefronts eventually collided but at a site nearly opposite to the site on the ring where the paced wavefronts entered the circuit. Furthermore, during pacing in the absence of flutter, it was always the counterclockwise and clockwise wavefronts resulting from the *same* paced beat that collided.

Throughout the period of entrainment, the site of collision was constant except for the last paced beat. The last paced impulse was still entrained but only up to the site of the previous collision. In electrograms proximal to the site of collision (e.g., electrogram 92), the coupling intervals between S<sub>n−1</sub> and S<sub>n−2</sub> and S<sub>n</sub> and S<sub>n−2</sub> and the next tachycardia beat...
Figure 2. Panels A and B: Maps of activation patterns during entrainment of the rhythm during two consecutively paced beats at an overdrive cycle length 20 msec less than the flutter cycle length (170 msec) (see also Figure 1B). In each, the map is an enlargement of the area within the dashed lines in Figure 1A. The tricuspid valve orifice (TO) is in the center. Small numbers represent the time of activation at individual electrode sites. Large numbers indicate the edge of selected isochrones (drawn at 10-msec intervals). For each map, activation times are relative to an arbitrary reference time. The four maps represented in this figure are continuous in time. In Panels A and B, stimuli (black) were delivered at +60 msec relative to the zero time of this window. Therefore, in each map, a wavefront arising from the previous stimulus (stippled arrows) and wavefronts arising from the present stimulus (white arrows) are seen. Note that with consecutive stimuli \( S_{n-1}, S_n \), there is a constant site of collision between \( A_{n-1} \) and \( O_{n-2} \) and \( A_n \) and \( O_{n-1} \), and the patterns of activation are identical. Panels C and D: Activation patterns of the first two beats of the tachycardia (T1, T2) immediately after the stimulator was turned off. Panel C shows no collision, yet \( O_n \) is still entrained up to the site where collision occurred on previous beats (*). In Panel D, the pathway of the reentering impulse is illustrated by the stippled arrow and is the same as the pathway of the orthodromic impulses during the entrainment period.
(T1) are nearly equivalent (Figure 1B). In contrast, for electrograms distal to the site of collision (e.g., electrogram 71), the coupling interval of the next tachycardia beat is greater than the pacing interval. In Figure 2C, the map clearly shows that there is no site of collision and that the impulse is entrained only up to the site of the previous beat’s collision (*). At this point, because there is no opposing wavefront, the last paced impulse continues to circulate around the path to perpetuate the circus movement (Figure 2D).

Careful inspection of maps T1 and T2 illustrates an important characteristic of entrainment in this reentrant circuit. Although the activation pattern for map T1 is similar to that of T2, there are several bunched isochrones in T1 that are not seen in T2, suggesting that conduction velocity through this area changes from T1 to T2. Beat T1 is the first beat at the end of a train of paced stimuli at BCL 150 msec. That is, pacing occurred at a coupling interval 20 msec shorter than the unperturbed flutter CL. As described above, in map T1, from the time of the 0–10 isochrone to the time of the asterisk, capture of several sites within the reentrant path by the last paced impulse is evident. We know from our previous work that the normal reentrant impulse propagates through tissue that has not completely recovered its excitability. Therefore, the slowed conduction observed in the portion of the reentrant circuit that was captured and still entrained in map T1 is expected. Slowed conduction in this part of the reentrant path increased the total time for the impulse to travel around the entire path (revolution time, 220 msec). For map T2, which follows map T1, the local cycle length preceding the activation at several sites within the reentrant path is longer, therefore there is faster conduction of the impulse through these areas of the circus path resulting in a decrease in the amount of time needed to complete one circuit (revolution time, 170 msec). This particular behavior of an impulse in this type of reentrant circuit has also been observed in the in vivo entrainment studies in these dogs, as well as during resetting experiments using the isolated ring model (see Figure 7 of ) and the isolated heart.

Results presented above are representative of the six entrainment protocols completed in the three canine hearts. In all individual episodes (n = 22) of documented entrainment of the induced flutter (CLs, 165–175 msec), entrainment was possible with stimuli delivered at CLs of 165–140 msec. In one heart in which pacing was performed from more than one site, the site of collision of the orthodromic and antidromic wavefronts was similar during episodes of pacing at the same CLs but depended on the site of stimulation and whether the flutter was clockwise or counterclockwise in its direction around the tricuspid ring.

In this heart, the epicardial pacing electrode was placed on the aortic wall of the right atrium adjacent to the area of Bachmanns bundle. Paced beats from this site entered the reentrant circuit at a site 180° from the freewall epicardial site. During a counterclockwise flutter (CL, 163 msec), entrainment with paced beats (CLs, 150 and 140 msec) was possible. The site of collision of A with O was fixed during pacing at any one cycle length. However, collision of the two wavefronts occurred in an area of the reentrant circuit different from that shown in Figure 2 (data not shown).

Figure 7 1D

Demonstration of the effect of pacing cycle length on the site of collision during entrainment. When a reentrant rhythm is entrained by progressively shorter cycle lengths, the paced impulses from the short CLs should result in early entry of the paced wavefronts into the reentrant circuit. In theory, the antidromic wavefront from the shortest paced CL should penetrate the farthest into the reentrant path and the site of collision between A and O should shift with different pacing CLs. Figures 3 and 4 illustrate the electrograms and the patterns of activation recorded as the sustained flutter was entrained with stimuli at BCL at 160 (Figure 3) and 140 (Figure 4) msec. Each map shows how the wavefront resulting from the last paced stimulus (S) invades the reentrant pathway. In the map of BCL at 160 msec (5 msec less than cycle length of flutter), the extent to which the antidromic impulses invade the loop is small. The site of collision of the orthodromic (O) and antidromic (A) wavefronts is within 5 mm of the breakthrough from the epicardial pacing site. Electrograms recorded from various sites on the endocardial surface (electrograms 66, 19, and 67) involved in the area of collision are noted by the larger bold numbers in Figure 3A. O entrains this reentrant rhythm because careful comparison of the maps in Figure 3B and 3C show that activation of sites on the O path by a paced stimulus occurs 5–6 msec earlier than it does during the unperturbed flutter.

With the decrease in the pacing CL to 140 msec (Figure 4), the antidromic wave occurs sooner than that in Figure 3. Thus, this wavefront is able to invade and travel along the reentrant path for a greater distance before colliding with the preceding orthodromic impulse. Therefore, the site of collision is different from that in Figure 3. The site of collision now occurs between electrodes 19 and 66.

Figure 7 1D

Demonstration that termination of flutter after a period of entrainment is associated with conduction block localized to a site in the reentrant pathway for one beat, followed by activation of that site from a different direction and with a shorter conduction time by subsequent paced impulses. A critical overdrive cycle length (135–145 msec), approximately 17–23% of the flutter CL, resulted in termination of each of six episodes during which pacing with a range of CLs was tested. In one case, a clearcut termination of flutter with restoration of normal rhythm was observed after entrainment. In the other five cases, entrainment with rapid drive produced irregular capture of the tissue stopping the
original rhythm and initiating another more complicated irregular rhythm. On cessation of stimulation, this irregular rhythm was nonsustained and normal rhythm resumed. Our results from analysis of maps of the first example are presented below.

 Interruption of a reentrant tachycardia by rapid atrial pacing should be associated with conduction block of the orthodromic impulse before it collides with the antidromic impulse. Any further pacing of tissues in the circuit should result in a direct collision of $A_n$ with its counterpart $O_n$. Direct analysis of events that occurred in the reentrant circuit during this episode of entrainment illustrates that although reentry was terminated, further pacing resulted in unexpected activation patterns. Furthermore, termination was associated with a rate-related conduction block of the orthodromic impulse within the reentrant path. In this case, antidromic penetration was not necessary for the localized conduction block.

Figures 5-7 illustrate this mechanism of termina-
tion of atrial flutter after a period of transient entrainment. The activation pattern during flutter shows the reentrant impulse traveling in a clockwise direction (Figures 5A and 5C). Rapid atrial pacing at a CL 30 msec less than the flutter cycle length entrained the flutter (Figures 5B and 5D). We can locate the site of collision from changes in the morphology of the electrograms recorded near the site (note the morphology of electrograms 24 and 72 in Figure 5A and compare it with the recordings from the same electrode sites during S5 in Figure 5B). The site of the collision of O₇ and A₇ is between electrode sites 24 and 72. This pattern was similar for S6 and S7, except in S6 there is some indication that at site 96 there is electrical activity that is secondary to activation from both the antidromic and orthodromic wavefronts (note the multiple electrograms). Just before the time of the eighth stimulus (S8), the orthodromic impulse from S7 blocks at a site within the reentrant loop (noted by the thickened black line at 40-msec isochrone) (Figures 6A and 7A). The reason for this block is not known but from maps of S5, S6, and S7 it is clear that conduction velocity through this region of the anatomic path was frequency dependent. For instance, in the map of S5 there is a long delay (45 msec, see the dashed lines in Figure 5B) between the activation of electrode site 45 and 92 (circled activation times in Figure 7A). In S6, this delay becomes 52 msec. By S7, the delay is 57 msec (S6 and S7 maps not shown). This delay allowed site 72 to be activated by the antidromic wavefront A₇ (note the relation between electrograms 24 and 72 in Figure 5B). Block of A₇ by O₈ then occurs between 96 and 72.

The orthodromic wavefront O₇ proceeds around the path until it blocks between 45 and 92 (see Figures 6A and 7A). This terminates the tachycardia. The major activation of the tissue at sites 96 and 92 is due to A₈, which now activates site 72.

Just after the time of the localized conduction block that occurs between the two sites 45 and 92 in map S8, there is a dramatic change in the relation and configuration of electrograms 96 and 92, electrode sites distal to the site of conduction block (cf, Figure 6A to 5A). This further supports the conclusion that the orthodromic impulse (O₇) blocked proximal to site 92. As a consequence of conduction block at this site, the antidromic wavefront (A₈) of S8 can propagate further around the reentrant path before it completely blocks between sites 92 and 45 (Figure 7A). Of importance, this map does not demonstrate a DIRECT collision or interaction between A₈ and O₇. In a direct collision, we would have expected A₈ and O₇ to have arrived at the same site at the same time. Rather, A₈ is blocked because of the barrier of persistent refractoriness caused by O₇. If the stimulus had been turned off here, no reentry would have been possible because the A₈ wavefront in turn would produce refractory tissue that would result in block of the O₈ wavefront.

This pattern of activation continues for several more stimuli. By the 17th stimulus (S17) in this train (Figure 7B), conduction has changed so that the A₁₇ wavefront now arrives at the barrier of refractory tissue resulting from O₁₆ at a later time (at +126
FIGURE 5. Panel A: Electrograms during another episode of flutter just before the stimulation protocol began. The time bar in this panel is 40 msec. Panel B: Electrograms resulting from the fifth (S5) and sixth (S6) stimuli in a train of stimuli (BCL, 140 msec). Time bar represents 100 msec. Maps of activation during flutter (F) and resulting from the S5 stimulus are shown in Panels C and D, respectively. Note that S5 entrains this reentrant rhythm.

msec instead of +110 msec as in Figure 7A). Again, there is not a direct collision of A17 and O17 but rather the O16 wavefront produces refractory tissue that blocks A17, and tissue refractoriness that is due to A17 causes block of the O17 wavefront. Restoration to normal rhythm finally occurs when just after the last paced impulse, activation due to O17 proceeds until it blocks at the 50 msec isochrone (between 45 and 92) (Figure 7C). No reentry is possible. The small deflections seen in 92 and 96 at this time are not atrial but ventricular in origin (see Figure 6A legend).

Discussion

We have previously described a model of atrial flutter that can be entrained in the conscious dog.6 The flutter is due to circus movement around a fixed barrier with a partially excitable gap.13 We now provide endocardial right atrial activation sequences during entrainment of this rhythm. From these
results we can make some general statements concerning the relation between sequences of activation in a reentrant circuit during entrainment and the surface electrocardiographic criteria that are often used in entrainment studies.\(^1\)\(^-\)\(^5\)

For the first electrocardiographic criterion,\(^1\) constant fusion means that the timing of the paced impulse and the reentrant impulse have the same relation on each beat. In our maps, this is represented by a fixed site of collision for each paced beat as well as a similar activation sequence in the reentrant pathway and surrounding right atrium during the pacing stimuli that entrain. This would have resulted in a constant fusion during pacing at a fixed rate in a surface electrocardiogram if left atrial activation were the same with each paced beat. Furthermore, the rate of impulses emerging from the reentrant path accelerated to the rate of the pacing, and, therefore, these impulses were still central to the total atrial activation pattern. Finally, our maps clearly show that at the end of a period of entrainment, the last paced beat was entrained only up to the site where collision of the orthodromic and antidromic wavefronts had occurred on previous paced beats (see Figure 2). Thus, the overall activation sequence of the atrium would have resulted in an entrained but not fused atrial complex in the surface electrocardiogram. Our results describing entrainment of this flutter in the conscious animal certainly support this idea (see Figure 10 in Frame et al\(^6\)).

By electrocardiographic criteria, progressive fusion in the electrocardiogram waveform means that the temporal relation between two impulses varies as the pacing rate is increased during entrainment. In our activation maps, this would be represented by a shift in the site of collision between the paced impulse and the impulse already propagating in the reentrant path. In our maps, the site of collision is shifted in the antidromic direction as predicted by Waldo et al\(^1\) and our own studies.\(^6\) It is unlikely that the shift seen is due to movement of endocardial egg electrode inside the atrial cavity in the orthodromic direction because by careful inspection of Figures 3 and 4 one can see that not only are isochrones near the endocardial breakthrough site of the paced beat different, but the whole area encompassed by the first several isochrones is different in the two figures. That is, if an egg movement had occurred and produced the "apparent shift" in the collision site, we would have expected the left and right extent of these isochrones at the time of the collision to be the same in the two figures and they are not.

By decreasing the pacing CL by 20 msec we might have expected to see a comparable change in the penetration of the antidromic impulse and thus a more dramatic shift in the site of collision during entrainment at this more rapid rate. However, we did not see this (see Figures 3B and 4B). Presumably this is due to the slowing of conduction that occurs in the incompletely repolarized tissue within the path\(^1\)\(^3\) with the decrease in pacing CL. Furthermore, it seems reasonable to suggest that the degree to which the site of collision is shifted along the reentrant path depends on several factors includ-
Figure 7. Panels A, B, and C: Maps of activation patterns during the periods indicated in Figure 6. Panel A: Activation pattern at the point when entrainment of the rhythm ceases. Panel B: Activation pattern of the last paced beat in this train (S17). Panel C: Activation pattern immediately after the period shown in Panel B. The subsequent beat was a normal sinus beat (map not shown).
An example of refractoriness to the atrial activation is the block of the orthodromic impulse before it collided with the antidromic impulse. What is presumed to be the usual mechanism of termination after a period of entrainment is that $O_{n-1}$ blocks the pathway due to the refractory tissue left by $O_n$. Therefore, $O_{n-1}$ can no longer conduct around the path to collide with $A_n$. As a result, $A_n$ conducts until it collides with $O_n$. If there is no subsequent paced impulse, then reentry will not resume because $O_n$ is blocked.

Our results detail a variation on the termination of reentry after a period of entrainment. The critical event that causes termination is the block of $O_n$ due to the refractoriness left by $O_n$ before $O_n$ collides with $A_n$ and is similar to what is expected to occur. In other cases of documented entrainment the critical event did not necessarily occur with the seventh paced beat but varied. However, our maps illustrate that $A_n$ (and subsequent paced beats $A_n$) does not subsequently directly collide with $O_n$ (and subsequent $O_n$). Rather, $O_n$ blocks between sites 45 and 92 due to the refractoriness left by $A_n$. Furthermore, the condition that prevents reinitiation of reentry by the last $O_n$ impulse when pacing is stopped is that it is blocked by an interaction with $A_n$. The basic difference between our example and the predicted or assumed usual mechanism is that the interaction is the block of $O_n$ by the refractoriness left by $A_n$, rather than the direct collision with $A_n$. Furthermore, a continued block of $A_n$ occurs due to the refractoriness left by $O_{n-1}$, but this block is not critical for preventing reentry but is necessary to preserve a condition by which $O_n$ will be blocked. If $A_n$ failed to block as is illustrated, it would have eventually collided with $O_n$ directly, thus resulting in a pattern of activation that is assumed to occur during overdrive pacing after termination.

References


**KEY WORDS** • atrial flutter • entrainment • mapping • reentry
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