Circus Movement Atrial Flutter in the Canine Sterile Pericarditis Model

Cryothermal Termination From the Epicardial Site of the Slow Zone of the Reentrant Circuit

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**Background.** We have shown that atrial flutter (AF) in dogs with sterile pericarditis is commonly due to a single-loop reentrant circuit in the lower right atrium comprised of a functional or functional/anatomic obstacle and a slow zone of conduction (SZ) between the central obstacle and the atrioventricular (AV) ring. The goals of the present study were 1) to establish that the epicardial SZ is the critical component of circus movement AF and 2) to identify the optimal site within the epicardial SZ at which interruption of circus movement can be accomplished by ablative techniques.

**Methods and Results.** We analyzed the atrial activation patterns during epicardial cooling of the SZ with an N₂O-cooled probe in eight dogs (five with clockwise [CW] reentrant circuit, one with counterclockwise [CCW] reentrant circuit, and two with both CW and CCW reentrant circuits around the same pathway). In all eight dogs, cooling (−5°C to +5°C for 5–20 seconds) narrowed the isthmus at the inferoposterior part of the SZ between the central obstacle and the AV ring reversibly terminated the reentrant circuit, whereas cooling outside this area failed to terminate the reentrant circuit. The circus movement was not observed to continue along alternate pathways when conduction in this critical zone was interrupted. Both CW and CCW reentrant circuits could be terminated from the same site within the SZ. Cooling resulted in slowing of conduction in the SZ (S5±15 msec) in both CW and CCW reentrant circuits. Cooling-induced termination of CW reentrant circuits was characteristically associated with oscillations of conduction in the cooled zone of the last three cycles before termination and conduction block occurred within the cooled zone. The last “manifest” reentrant cycle was associated with the longest conduction delay in the cooled zone. However, this delay was not necessarily reflected in the length of the last reentrant cycle because of compensatory acceleration of conduction in the rest of the pathway. On the other hand, in CCW reentrant circuits, conduction block occurred abruptly at the distal border of the SZ and without significant oscillations of conduction.

**Conclusions.** The present study provides convincing evidence that single-loop circus movement in this model is critically dependent on an obligatory conduction in a SZ in the inferoposterior portion of the free right atrial wall between a functional obstacle and the AV ring. Because the atrial myocardium behaves electrophysiologically as a two-dimensional surface, the results of this study may help to guide the endocardial electrode catheter ablative technique for treatment of clinical AF. (*Circulation* 1993;87:1649–1660)

**Key Words** • circus movement reentry • conduction • ablation • arrhythmias • atrial flutter

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We have recently described the epicardial activation patterns during circus movement atrial flutter in the canine sterile pericarditis model.1 Single-loop circus movement was shown to occur around functional or combined functional/anatomic obstacles primarily in the right atrium.1,2 An area of slow conduction, bounded by the central obstacle and the atrioventricular (AV) ring, was an integral part of the reentrant circuit. Spontaneous termination was consistently due to failure of propagation within the area of slow conduction.

The goals of the present study were 1) to establish that the epicardial zone of slow conduction is the critical component of circus movement atrial flutter (i.e., if conduction is blocked in this critical area, circus movement will not continue via parallel or alternate septal pathways) and 2) to identify the optimal site within the epicardial zone of slow conduction at which interruption of circus movement can be accomplished by ablative techniques. For this purpose, we used reversible cooling and/or cryoablation of specific areas of the epicardial surface of the reentrant circuit. Because atrial myocardium behaves electrophysiologically as a two-dimensional surface,3 the data obtained from cryoablative termination of atrial flutter from the epicardial surface may help to guide the endocardial electrode...
catheter ablative technique for treatment of atrial flutter in the clinical setting. Preliminary data have been published.4

Methods

Details of the model preparation, recording techniques, and methods for constructing isochronal activation maps have been described.1 Studies were performed in eight mongrel dogs (15–20 kg) with sterile pericarditis and inducible sustained atrial flutter. Sterile pericarditis was created by generously dusting the epicardial surface of both atria with talcum powder.5 After recovery from the initial surgical procedure, induction of atrial flutter by rapid atrial pacing was attempted daily with the dogs in the conscious, nonsedated state. Mapping studies were performed on the day after induction of sustained atrial flutter (3–5 days after the initial surgery). The chest was reopened, and a custom-designed epicardial jacket electrode array was placed on the epicardial atrial surface. Two electrode arrays were used in this study (Figure 1). The first array was, as described previously,1 designed to record from the epicardium of both atria. The atrial “jacket” contained 111 bipolar recording electrodes with an interpolar distance of 1–2 mm in a flexible nylon mesh. The interelectrode distance ranged from 3 to 5 mm but could reach 5–8 mm in certain areas (e.g., the atrial appendages) when the nylon mesh was stretched. Five small Teflon patches containing two to five electrode pairs provided 16 additional bipolar recordings from the area between the pulmonary veins, the area below the inferior vena cava, and the anterior aspect of the atrial appendages. Because we have previously shown that the majority of reentrant circuits in this model are located in the right atrial wall,1 the second electrode array was designed to record from the epicardial surface of the right atrium with a higher resolution (1.5–2.5-mm interelectrode distance). The high-resolution electrode array was used in five of the eight experiments. After placement of the electrode array, the chest was covered with gauze. Epicardial and core temperatures were continuously monitored. A heating blanket was used to keep the epicardial temperature at 37±1°C.

Induction of atrial flutter then was attempted by either rapid burst pacing or critically timed premature stimuli provided by a DTU-101 digital stimulator (Bloom, Reading, Pa.). Once sustained atrial flutter was induced, recordings from all 127 electrode sites were obtained. The cycle length and ECG morphology were observed over a time course of at least 30 minutes to confirm the stability of the arrhythmia. Data were stored and analyzed using a 128-channel computerized multiplexer recording system. Activation times were marked manually after review of each individual recording. In electrograms showing a sharp intrinsic deflection, the maximum first derivative was taken as the moment of activation. For multiphasic electrograms, the contribution of the ventricular activation to the recorded signal was excluded by comparing the timing of the electrogram components with the surface ECG and with other epicardial recordings. The peak of the major deflection then was chosen as the moment of activation. From these activation times, isochronal epicardial activation maps were constructed manually at 10-msec intervals. As in our previous studies, functional conduction block was considered to be present when the activation time difference between adjacent sites was ≥40 msec.6,7 Additional criteria for functional block have been described previously.8 During the mapping experiments, ECG lead II, a left or right atrial electrogram, and aortic blood pressure were continuously monitored on a VR12 recorder (Electronics for Medicine, Lenexa, Kan.).

Cryothermal Protocol

The cryothermal system used in the present study was a Spembly-Amois BMS 411 cryo unit.9 This apparatus regulates the flow of nitrous oxide through the tip of the cryoprobe. The cryoprobe used in the study (No. 7107) had a flat tip 6 mm in diameter. Local epicardial temperature could be measured by a ther-
FIGURE 2. Panel A: Epicardial right atrial isochronal map of a control atrial flutter single-loop clockwise reentrant circuit (left). Selected electrograms along the reentrant pathway are shown to the right, and their positions are indicated by the black dots on the isochronal map. Numbers represent the activation times in msec. The arrow indicates the direction of the reentrant wave front. The arc of functional conduction block is represented by the heavy solid line. (See text for details.) Panel B: The effects of reversible cooling along the broad superior border of the zone of slow conduction. The position of the cryoprobe is represented by the shaded circle. Note that cooling at this site did not interrupt the reentrant circuit or change the overall reentrant pathway; however, it resulted in conduction block between sites C and D (represented by asterisks on the electrograms on the right panel). This was represented in the isochronal map as an extension of the superior edge of the arc of block to between sites C and D as well as by crowding of the isochrones at this site reflecting slowed conduction.
FIGURE 3. Panel A: Isochronal activation maps from the same experiment shown in Figure 2 illustrating interruption of atrial flutter circus movement when the cryoprobe (shaded circle) was applied to an epicardial site along the narrow inferior border of the zone of slow conduction. The activation maps of the last four reentrant cycles before termination of reentry are labeled N-3, N-2, N-1, and N, respectively. Cooling resulted in marked slowing of conduction in this zone (depicted as crowding of activation isochrones). The N map shows that the reentrant circuit was interrupted when the inferior edge of the arc of functional conduction block extended to the atrioventricular ring. Panel B (facing pages): Selected electrograms along the reentrant pathway from the experiment shown in Figure 3A illustrate the long-short-long oscillations of conduction time in the cooled zone before termination of reentry. The horizontal arrows represent conduction times between sites G and I that span the cooled zone. Conduction block between sites H and I is represented by a double bar. (See text for details.) Numbers are given in msec.

mocouple at the tip of the probe. For reversible interruption of reentrant activation, the myocardial temperature at a localized epicardial site was reduced to between −5 and +5°C for 10–30 seconds. The cryoprobe was applied over the nylon mesh jacket electrode. Different epicardial sites were tested (three to six sites per experiment), and the effects of transient epicardial cooling on atrial activation patterns were analyzed. To achieve cryoablation, the temperature at the tip of the cryoprobe was reduced to between −55°C and −65°C for 2 minutes.9

Results

Eight dogs in which atrial flutter was due to a single-loop reentrant circuit around a functional arc of conduction block in the lower posterior wall of the right atrium were included in the study. In five dogs, the reentrant wave front circulated in a clockwise direction, and in one dog, it circulated in a counterclockwise direction only. In the remaining two dogs, clockwise and counterclockwise reentrant circuits could be induced around the same pathway. The atrial flutter mean cycle length was 163±31 msec (range, 130–210 msec).

Critical Site for Termination of Atrial Flutter Single-Loop Reentrant Circuit

Figures 2 and 3 illustrate the effects of reversible cooling at different epicardial sites along the reentrant
pathway during sustained circus movement atrial flutter. The left panel of Figure 2A shows the epicardial right atrial isochronal map of the control reentrant circuit, and the right panel shows selected electrograms along the reentrant pathway. The reentrant circuit had a characteristic single-loop pattern with the activation wave front circulating in a clockwise direction around a functional arc of conduction block. Conduction along the left side of the arc of block was relatively fast, whereas conduction was much slower in a zone located between the arc of block and the AV ring. The cryo-probe then was applied at different epicardial sites along the reentrant pathway.

Figure 2B illustrates the effects of reversible cooling of an epicardial site along the broad superior border of the slow zone. Cooling of this site resulted in distinct changes in local conduction. The conduction time between sites C and D (marked by an asterisk) increased from 15 msec during control to 42 msec after application of the cryo-probe. The change in the configuration of the electrogram at site C could be explained by the change in the activation pattern (i.e., the development of an arc of conduction block between sites C and D). However, the reentrant circuit remained uninterrupted with no significant change in the overall activation pattern and conduction time.

Figures 3A and 3B show that the reentrant circuit could be interrupted when the cryo-probe was applied to an epicardial site along the narrow inferior border of the slow zone between the inferior edge of the arc of block and the AV ring. The activation maps of the last four reentrant cycles before termination are shown in Figure 3A, and electrograms from selected sites along the reentrant pathway are shown in Figure 3B. Cooling of this site resulted in distinct changes in the local activation pattern. The inferior edge of the arc of conduction block moved closer to the AV ring, thus decreasing the width of the isthmus of slow conduction. Furthermore, cooling resulted in marked slowing of conduction in this zone (depicted as crowding of activation isochrones). The conduction time between sites G and I that spanned the site of the cooled zone was 30 msec during control (Figure 2A) and increased to 80–90 msec during the last few cycles preceding the termination of circus movement. Conduction accelerated in the remainder of the reentrant pathway. However, the accelerated conduction in the remainder of the reentrant pathway did not fully compensate for the marked slowing of conduction in the cooled zone. This resulted in lengthening of the overall reentrant circuit conduction time by approximately 20 msec (from 150 msec during control to 170 msec before termination). Cooling-induced termination of reentry occurred when the inferior edge of the arc of functional conduction block extended to the AV ring (Figure 3A, map N). In this experiment, termination repeatedly occurred 5–15 seconds after application of cooling.
Characteristic Oscillations of Conduction Before Cooling-Induced Termination of Clockwise Reentrant Circuit

Gradual lengthening of the reentrant circuit conduction time occurred during cooling, as discussed above. This sometimes was associated with slight (2–5 msec) oscillations of reentrant circuit cycle length that occasionally could revert to constant cycles as cooling continued. However, cooling-induced termination of clockwise reentrant circuits was consistently associated with characteristic oscillations of conduction of the last three manifest reentrant cycles before termination. This is illustrated in Figures 3A and 3B. For description, a “manifest” cycle is defined as one associated with complete revolution around the reentrant pathway. By contrast, the last revolution before termination is considered a “concealed” cycle because its conduction time could not be evaluated from surface ECG recordings. The oscillations were initiated by a 5–10-msec lengthening of the third manifest cycle before termination. This was followed by 10–20-msec shortening of the second-to-last cycle and 20–30-msec lengthening of the last manifest cycle. The last manifest cycle had the longest conduction time of all preceding cycles.

The characteristic long-short-long oscillations of conduction occurred primarily in the cooled zone with markedly slowed conduction. This could be best illustrated at the site of cooling-induced conduction block (between sites G and I in Figure 3B). The conduction times between sites G and I were 90, 95, 85, and 105 msec, respectively, during the last four cycles before termination.

The long-short-long oscillations of conduction at the critical site of cooling-induced termination of reentry usually were reflected in the overall reentrant cycle length, as shown in Figure 3. However, in some experiments, the marked slowing of conduction at the critical site of cooling-induced termination of reentry during the last manifest cycle was associated with compensatory acceleration of conduction in the rest of the reentrant pathway. In this case, the last manifest reentrant cycle was not necessarily the longest cycle of atrial flutter. This is illustrated in Figures 4 and 5, which were obtained from a different experiment. In this experiment, both a clockwise and a counterclockwise reentrant circuit could be induced along the same reentrant pathway. Figure 4 shows the epicardial isochronal map and selected electrograms along the reentrant pathway of the clockwise circuit. The activation wave front circulated around a functional arc of conduction block in the lower part of the right atrium. A narrow isthmus of slow conduction was bounded by the inferior end of the arc of block and the AV ring. Cooling of this zone resulted in interruption of circus movement. The activation maps of the last four cycles before termination are shown in Figure 5A, and selected electrograms along the reentrant pathway are shown in Figure 5B. Similar to the example shown in Figures 3A and 3B, cooling resulted in marked slowing of conduction in the inferior portion of the zone of slow conduction. The cooling-induced local slowing of conduction was only partially compensated for by faster conduction in the remaining parts of the reentrant circuit. This resulted in lengthening of the overall reentrant circuit conduction time from 205 msec during control to approximately 220 msec a few beats before termination. Reentry terminated when the inferior edge of the arc of block extended to the AV ring (Figure 5A, map N). The last three cycles before termination showed characteristic long-short-long oscillations of conduction at the critical site of the cooled zone where reentry terminated. Figure 5B shows that the conduction times between sites F and J (spanning the cooled zone) were 90, 95, 65, and 110 msec, respectively, during the last four cycles before termination. The marked local slowing of conduction during the last cycle lengthened the reentrant...
circuit cycle length to 245 msec at sites immediately distal to the site of conduction block (site J). However, compensatory acceleration of conduction in the remaining parts of the reentrant circuit resulted in a shorter cycle length of the last manifest reentrant beat (220 msec).

**Termination of Both Clockwise and Counterclockwise Reentrant Circuits From the Same Critical Site**

In two dogs, both clockwise and counterclockwise reentrant circuits were induced around the same reentrant pathway and could be terminated from the same site in the slow zone of the reentrant circuit. Figures 6 and 7 illustrate termination of the counterclockwise reentrant circuit from the same experiment shown in Figures 4 and 5. Figure 6 shows the control reentrant circuit that used approximately the same pathway as the clockwise circuit shown in Figure 4. However, the reentrant circuit conduction time of 190 msec was shorter than that of the clockwise circuit. Figure 7 shows that the reentrant circuit could be terminated when cooling was applied to the same site in the zone of slow conduction as during the clockwise circuit as shown in Figure 5. The isochronal activation maps of the three cycles preceding termination are shown as well as selected electrograms along the reentrant pathway. Cooling resulted in marked slowing of local conduction. A partial compensatory acceleration of conduction in the remainder of the reentrant pathway resulted in an overall increase of the reentrant circuit conduction time from 190 to 205 msec. Contrary to the characteristic oscillations of conduction in the cooled zone before termination of the clockwise reentrant circuit (Figure 5), termination of the counterclockwise circuit occurred abruptly without prior oscillations of conduction times. Conduction block occurred at the inferior border of the cooled zone. Thus, the last activation wave front failed abruptly to conduct through the cooled zone. This was in contrast to termination of the clockwise circuit (Figure 5) where the last activation wave front conducted through the slow zone and then blocked within the cooled zone. The pattern of cooling-induced termination of counterclockwise circuits shown in Figure 7 also was seen in the two other counterclockwise circuits that were analyzed in the present study.

Figure 8 diagrammatically illustrates the site of termination of single-loop reentrant circuit atrial flutter in eight dogs. Figure 8A illustrates the experiment in which only a counterclockwise circuit was induced; Figures 8B–8F show the five experiments in which clockwise reentrant circuits were induced. Figures 8G and 8H represent the clockwise reentrant circuit from the two dogs in which a counterclockwise circuit around a similar pathway also was induced. Reentrant activation could be consistently terminated when cooling was applied to the epicardial site within the narrow isthmus of the slow conduction zone between the inferior edge of the functional arc of block and the AV ring. Two to four attempts to reversibly terminate atrial flutter from the same critical site were successfully tested in each experiment. Termination of re-entry always occurred when the inferior edge of the arc of the block extended to the AV ring. In four experiments in which cryoblation was applied to the same critical site in the slow zone, atrial flutter could no longer be induced. Atrial fibrillation was not induced during cryoprobe application in any of the experiments.

**Discussion**

**Effects of Cooling on the Reentrant Circuit**

We have shown that the reentrant circuit in the sterile pericarditis model occurs in the surviving endocardial layer of grossly intact myocardium. The thickness of the canine atrial wall is 1.5–2 mm. It is a reasonable assumption that cryoprobe application to the epicardial surface resulted in sufficient cooling across the atrial wall where the reentrant circuit is located. Moderate cooling results in marked increase of the duration of the action potential without marked changes in the resting potential. The increase in action potential duration results from a decreased slope of phase 2. However, if the myocardium is cooled sufficiently, resting potential is decreased, resulting in diminished excitability and blocked conduction. In the in vivo canine heart, cooling of the normal ventricular epicardial layer results in a lengthening of the effective refractory period of the cooled region and cycle length-dependent conduction delays and conduction block. During sustained reentrant activation, the wave front will move at the maximum velocity permitted by the state of recovery of excitability of the myocardium. Any lengthening of refractoriness of localized, but critically situated myocardial zones along the reentrant pathway will be directly reflected in changes in conduction. In the present study, it is probable that cooling-induced lengthening of the effective refractory period of localized zones of the epicardial surface of the zone of slow conduction resulted in slowing of conduction, conduction block, or both.

**Critical Area for Interruption of Circus Movement Atrial Flutter**

The present study demonstrates that single-loop circus movement atrial flutter can be successfully interrupted when cooling or cryoablation is applied to a critical part of the zone of slow conduction between the inferior end of the central obstacle (i.e., the arc of functional block) and the AV ring. This site represented the narrow isthmus between the central obstacle and the AV ring. Conduction at this narrow isthmus usually was very slow. However, it should be emphasized that it is not the site(s) of slowest conduction but rather the anatomic site of the narrow isthmus that is critical for successful interruption of the reentrant circuit by a localized ablative lesion. In this model of atrial flutter, the apparent conduction along the septal side of the central obstacle is relatively fast. Because simultaneous endocardial recordings were not obtained, the possibility that the epicardial activation in this area might not have represented the actual conduction of the reentrant wave front has been previously entertained. A spread of activation around the portion of the AV ring under the right atrial appendage through the septum with consecutive activation of the paraseptal myocardium could have been possible. The present study provides convincing evidence that single-loop circus movement in this model is critically dependent on an obligatory conduction in the zone of slow conduction in the inferoposterior portion of the free right atrial wall bounded by a functional or functional/anatomic obsta-
Oscillations of Conduction Before Cooling-Induced Termination of Clockwise Circus Movement

The present study has shown that cooling-induced termination of clockwise circus movement atrial flutter in the right atrium was associated with characteristic oscillations of conduction in the cooled zone just before termination. After 5–15 seconds of cooling that induced significant slowing of conduction in the cooled zone, a slight additional slowing of conduction for one cycle initiated oscillations of conduction for the last two cycles before termination. The oscillations took the form of a relatively fast conduction in the cooled zone of the second-to-last manifest reentrant cycle followed by relatively slow conduction of the last manifest reentrant cycle. Because of compensatory acceleration of conduction in the remainder of the reentrant pathway, the duration of the last manifest reentrant cycle could be the same or even shorter than the third cycle before termination (Figure 5). Thus, analysis of cycle length changes of atrial flutter from surface ECG leads or local electrograms outside the zone of conduction block may fail to show the characteristic long-short-long oscillations of conduction before termination.

In an earlier report, we described oscillations of conduction in the slow common reentrant pathway of figure-eight circus movement before cooling-induced termination of reentrant ventricular tachycardia in the canine postinfarction model.14 Frame and Simson15 and Frame and Rhee16 described oscillations of conduction, action potential duration, and refractoriness before spontaneous termination of circus movement in vitro in rings of canine atrial tissue from around the tricuspid valve orifice. Reentry occurred around a fixed path with incomplete recovery of excitability. Local changes in conduction during oscillations resulted from the dependence of both conduction velocity and action potential duration on the preceding local diastolic interval. A more complex pattern of oscillations was seen when conduction velocity varied at more than one site in the circuit. The critical event in this model was an exceptionally long diastolic interval preceding the next-to-last cycle that accelerated local conduction, which tended to

**FIGURE 5.** Panel A: Isochronal maps from the same experiment shown in Figure 4 illustrating interruption of circus movement atrial flutter when the cryoprobe (shaded circle) was applied to an epicardial site along the narrow isthmus of slow conduction between the inferior edge of the arc of functional conduction block and the atrioventricular ring. The activation maps of the last four reentrant cycles before termination of reentry are labeled N-3, N-2, N-1, and N, respectively. The N map shows that the reentrant circuit was interrupted when the inferior edge of the arc of block extended to the atrioventricular ring. Panel B: Selected electrograms along the reentrant pathway from the experiment shown in Figure 5A illustrate the long-short-long oscillations of conduction time in the cooled zone before termination of reentry. The horizontal arrows represent conduction times between sites F and J that span the cooled zone. Conduction block between sites I and J is represented by a double bar. Note that the marked local slowing of conduction during the last “manifest” reentrant cycle is evident only at the site of conduction block. This delay could not be detected from analysis of flutter waves in the surface ECG lead or from electrograms outside the site of conduction block because of compensatory acceleration of conduction in the remainder of the pathway during the last “concealed” cycle. (See text for details.)

**FIGURE 6.** Epicardial isochronal map from the same experiment shown in Figures 4 and 5 illustrating a control counterclockwise reentrant circuit using approximately the same pathway as the clockwise circuit shown in Figure 4. Selected electrograms along the reentrant pathway are shown on the right, and their position is indicated by the black dots on the isochronal map. The last QRS deflection in the surface ECG is a ventricular paced beat. The paced spike is barely discernible in few of the local electrograms.
shorten the last cycle, and prolonged action potential duration and refractoriness at the site of block.

A similar mechanism may explain cooling-induced termination of circus movement atrial flutter in the present study. The marked local slowing of conduction during the last manifest cycle would result in 1) acceleration of conduction in the remainder of the reentrant pathway of the last concealed circus movement, 2) a rightward shift of the tail of refractoriness at the site of block, and 3) lengthening of refractoriness at the site of block. The latter could be explained by interval-dependent lengthening of refractoriness because of the significant prolongation of the preceding cycle length at this site.

The sudden shortening of conduction delay during the second-to-last manifest reentrant cycle following the slight increase in conduction delay in the preceding cycle is, however, more difficult to explain. It is possible that the lengthening of the preceding cycle length resulted in an improvement of conduction in a critical part of the cooled zone. In the study by Frame and Simson, changes in cycle length were poorly correlated with changes in diastolic interval and with local conduction velocity. Longitudinal dissociation of conduction in the cooled zone also could explain an "apparent" improvement of conduction delay. In the absence of very-high-resolution recordings from this zone, this latter mechanism could not be substantiated.

It also is important to note that oscillations of conduction during cooling-induced termination should not be fully equated with similar oscillations during spontaneous termination because of the continuously changing electrophysiological properties of the cooled zone. However, it is interesting to note that the termination of clockwise circus movement occurred only after a brief period of oscillations of conduction rather than a grad-

FIGURE 7. Epicardial isochronal maps of the counterclockwise circuit illustrating interruption of reentry when the cryoprobe (shaded circle) was applied to the same site in the zone of slow conduction as during the clockwise circuit shown in Figure 5. The activation maps of the last three reentrant cycles are labeled N-2, N-1, and N, respectively. Termination of the counterclockwise circuit occurred without prior oscillations of conduction time (see the selected electrograms in the lower right panel). Conduction block occurred at the inferior border of the cooled zone between sites E and F (represented by double bar). The QRS deflection in the surface ECG is a ventricular paced beat.
ual cycle-to-cycle lengthening of conduction delay. These observations tend to emphasize the importance of local oscillations of conduction in the termination of circus movement reentry in general. This is further emphasized when contrasting the termination of clockwise and counterclockwise circus movements in the present study. Cooling-induced termination of counterclockwise circus movement was abrupt and occurred at the border of the cooled zone without preceding cycle length oscillations. It is probable that conduction block of the counterclockwise circulating wave front occurred the moment cooling resulted in “sufficient” lengthening of refractoriness and/or decrease in excitability of the cooled zone.

**Ablative Therapy for Clinical Atrial Flutter**

In humans, limited mapping techniques have provided evidence to support the concept that classic atrial flutter is due to circus movement in the lower right atrium. Electrode catheter recordings of Puech et al., Olsansky et al., and Cosio et al. suggested the presence of circus movement in the posterior inferior wall of the right atrium. Several have described an isthmus of slow conduction during atrial flutter in the region of Koch’s triangle between the orifices of the inferior vena cava, coronary sinus, and tricuspid ring. Klein et al. described slow conduction in this area in two patients with atrial flutter who underwent epicardial mapping in the operating room and cryosurgical ablation of the arrhythmia. Electrode catheter ablation techniques applied to roughly the same area could permanently interrupt atrial flutter. These observations are remarkably similar to the finding in the present study that the critical site of interruption of circus movement atrial flutter was in the lower posterior right atrium. In a study by Stone et al., simultaneous right atrial free wall epicardial and endocardial recordings during sinus rhythm and atrial flutter in normal dogs revealed minor differences in the activation pattern, confirming that the atrial myocardium behaves electrophysiologically as a two-dimensional surface. This strongly suggests that ablative energy applied through an electrode catheter to the endocardial site of the critical zone of the atrial flutter reentrant circuit could be equally effective in interrupting the arrhythmia. Recently, Feld et al. used radiofrequency current applied through an endocardial electrode catheter to ablate type 1 atrial flutter in humans. Sites where ablation was successful, located just inferior or posterior to the coronary sinus ostium, were characterized by discrete electrograms with activation times of −20 to −50 msec before P wave onset and exact entrainment pace maps with a stimulus–P wave interval of 20–40 msec, consistent with the exit site from the area of slow conduction. However, there is a need for a more accurate interpretation of endocardial electrode catheter recordings in relation to the atrial flutter reentrant circuit. Precise localization of the endocardial critical zone for interruption of the arrhythmia can have direct clinical application.
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