JACC Vol. 6, No. 1
July 1985:124–32

Reentrant Ventricular Arrhythmias in the Late Myocardial Infarction Period. 12. Spontaneous Versus Induced Reentry and Intramural Versus Epicardial Circuits

NABIL EL-SHERIF, MD, FACC, WILLIAM B. GOUGH, PHD, ROBERT H. ZEILER, PHD, ROBERT HARIMAN, MD, FACC

Brooklyn, New York

One to 5 days after one-stage ligation of the left anterior descending coronary artery in dogs, reentrant excitation can be induced by programmed premature stimulation in the surviving electrophysiologically abnormal, thin epicardial layer overlying the infarct. In experiments in four dogs, reentrant excitation occurred "spontaneously" during a regular sinus or atrial rhythm. A tachycardia-dependent Wenckebach conduction sequence in a potentially reentrant pathway was the initiating mechanism for spontaneous reentrant tachycardias and was the basis for both manifest and concealed reentrant extrasystolic rhythms. In all dogs showing spontaneous reentry, reentrant excitation could also be induced by premature stimulation at cycle lengths much shorter than those associated with spontaneous reentry, and induced reentrant circuits were always different from those during spontaneous reentry. In two dogs, the reentrant circuit was located intramurally in close proximity to a patchy septal infarction.

The study illustrates that irrespective of the anatomic localization of reentrant circuits (epicardial or intramural), their dimension (large or small) or their mechanism of initiation (programmed premature stimulation or "spontaneous"), reentrant excitation always occurred in a figure 8 configuration (or a modification thereof). The figure 8 model, rather than the ring model or the leading circle model, may be the common model of reentry in the mammalian heart.

(J Am Coll Cardiol 1985;6:124–32)

We have shown (1,2) that in dogs 3 to 5 days after one-stage ligation of the left anterior descending coronary artery, reentrant excitation can be induced by programmed premature stimulation. The reentrant circuits are located in the surviving, although electrophysiologically abnormal, thin epicardial layer overlying the infarction. Reentrant activation has a figure 8 pattern in the form of two circulating wave fronts around arcs of functional conduction block that coalesce into a common wave front before reexciting myocardium on the other side of the arcs of block (1,2). This report describes experiments in which reentrant excitation developed "spontaneously" during a regular sinus or atrial rhythm with a critically short cycle length. A reentrant tachycardia or single reentrant beats in an extrasystolic arrangement could occur. Other experiments are also described in which the reentrant circuit had an intramural rather than an epicardial location. These experiments lend support to the argument that the figure 8 model of reentrant excitation may be central to a majority of atrial and ventricular arrhythmias based on reentry in the mammalian heart rather than being specific to a particular animal model.

Methods

Isochronal activation maps. Epicardial and intramural isochronal activation maps were studied in six dogs 3 to 5 days after one-stage ligation of the left anterior descending artery just distal to the anterior septal branch. Details of the experimental model, surgical techniques as well as the methods for recording simultaneous epicardial and intramural electrograms and for construction of isochronal activation maps have been previously described (1–3). Reentrant rhythms occurred spontaneously during a regular sinus or atrial paced rhythm or were induced by programmed premature ventricular stimulation. The stimulation protocol has been described (1,4) and will be detailed in the Results section.

From the Veterans Administration and State University of New York, Downstate Medical Center, Brooklyn, New York. This study was supported by Veterans Administration Medical Research Funds, Washington, D.C. Manuscript received October 9, 1984; revised manuscript received December 26, 1984, accepted January 9, 1985.

Address for reprints: Nabil El-Sherif, MD, Cardiology Division, State University of New York, Downstate Medical Center, Box 1199, 450 Clarkson Avenue, Brooklyn, New York 11203.

0735-1097/85/$3.30

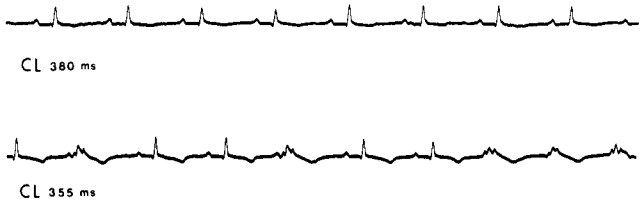
Anatomic correlations. After termination of the electrophysiologic study, the anatomic locations of intramural recording sites were determined and correlated with epicardial recording sites. The anatomic features of the infarction were also determined as previously described (1,2) and correlated with the recorded electrograms.

Results

Reentry. In four experiments, a reentrant rhythm developed spontaneously during sinus tachycardia (three dogs) or an atrial paced rhythm (one dog). Reentry was in the form of a single reentrant beat after two consecutive sinus or atrial beats (a trigeminal extrasystolic rhythm) in all four experiments. In three experiments, a repetitive reentrant rhythm (2 to 20 beats) also occurred. In all experiments, reentry developed after critical shortening of the sinus or atrial cycle length. In the experiment shown in Figure 1, spontaneous shortening of the sinus cycle length from 380 to 355 ms resulted in a regular trigeminal rhythm and occasionally in a repetitive reentrant rhythm. In all experiments, the reentrant circuits were located epicardially.

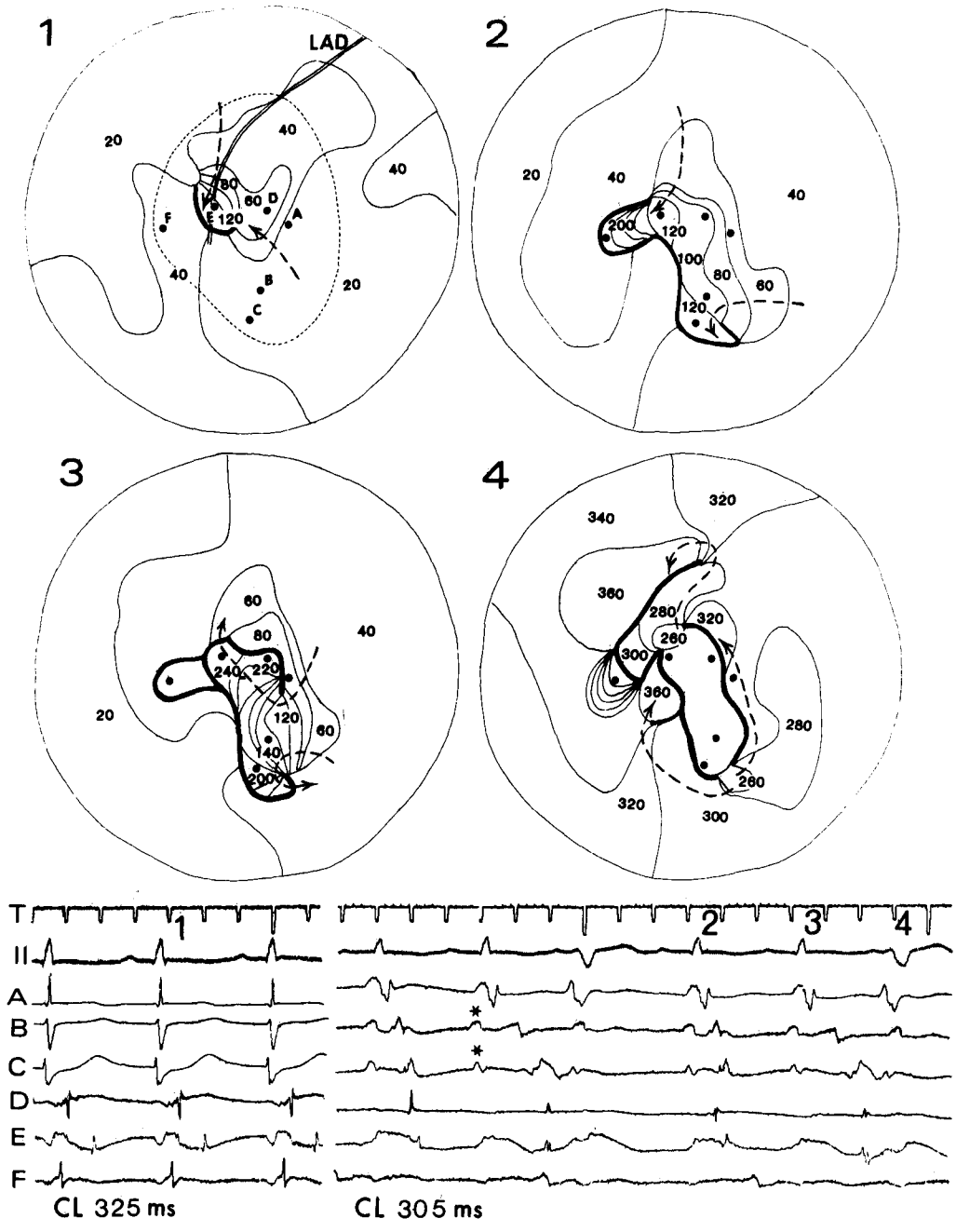
Reentrant extrasystolic rhythms. Figure 2 was obtained from another experiment and illustrates epicardial activation maps as well as selected electrographic recordings during a reentrant trigeminal rhythm. During sinus rhythm at a cycle length of 325 ms, there was a consistent small arc of functional conduction block near the apical region of the infarct and relatively slow activation of nearby myocardial zones. The activation pattern, however, was constant in successive beats, reflecting a 1:1 conduction pattern. Spontaneous shortening of the sinus cycle length of 305 ms resulted in the development of a single reentrant beat after every second sinus beat. During the reentrant trigeminal rhythm, the epicardial activation map of the first sinus beat showed the development of a longer arc of functional conduction block compared with that during sinus rhythm at a cycle length of 325 ms. The activation front circulated around both ends of the arc of block, but was not sufficiently delayed on the distal side of the arc of block. In contrast, the activation map of the second sinus beat showed some lengthening of the arc of block at one end, but more characteristically a much slower conduction of the two activation fronts circulating around both ends of the arc of block. The degree of conduction delay was sufficient for refractoriness to expire at two separate sites on the proximal side of the arc, resulting in two simultaneous breakthroughs close to the ends of the arc and thereby initiating reentrant excitation. The leading edge of the two reentrant wave fronts coalesced, but failed to conduct to the central part of the epicardial surface of the infarct (that is, to areas that were showing slow conduction during the preceding cycle). This limited the reentrant process to a single cycle. It also resulted in recovery of those myocardial zones in the central part of the infarct, allowing the next sinus beat to conduct with a lesser degree of conduction delay, thus perpetuating the reentrant trigeminal rhythm. Analysis of the two electrograms recorded from each of the two reentrant pathways (B and C, and D and E, respectively) shows a characteristic 3:2 Wenckebach-like conduction pattern. Figure 2 also illustrates the complexity of the conduction pattern in ischemic myocardium and the presence of a zone of dissociated conduction. This is represented by site F, which showed a 2:1 conduction pattern during the 3:2 Wenckebach cycle and reentrant trigeminal rhythm described previously.

Figure 1. Electrocardiographic recordings from a dog 4 days after infarction. A trigeminal ventricular rhythm and ventricular tachycardia occur during spontaneous shortening of the sinus cycle length (CL) from 380 to 355 ms. See text for details.



Manifest and concealed extrasystolic reentrant rhythms. In all experiments, a concealed reentrant trigeminal rhythm also occurred at cycle lengths equal to or slightly longer than those associated with manifest reentry. During concealed trigeminy, the 3:2 Wenckebach conduction sequence was usually maintained. However, the degree of conduction delay of the circulating wave front during the second sinus beat preceding a concealed trigeminal sequence was less than that during manifest reentry, and the wave front failed to reexcite myocardial zones proximal to the arc of block and failed to initiate a manifest reentrant cycle. More complicated conduction sequences could also occur during a concealed trigeminal rhythm. This is illustrated in Figure 3, which was obtained from the same experiment shown in Figure 2. Figure 3A shows a manifest trigeminal rhythm at a cycle length of 305 ms. Repetitive reentrant excitation is seen at the end of the recording. The epicardial activation map of the sinus beat that initiated reentry (labeled A) is shown on the top, while selected electrograms are shown on the bottom. The ladder diagram illustrates the condution pattern between site A located outside the arc of functional conduction block and sites C and E within the slow conduction zone. As shown previously in Figure 2, electrograms C, D and E show a 3:2 Wenckebach conduction sequence.

Figure 3B illustrates a concealed trigeminal rhythm at the same sinus cycle length during manifest reentry. The activation map of the second sinus beat that was supposed to initiate reentry (labeled B) is shown on the top. Compared with the map in Figure 3A, the arc of functional conduction block is shorter. More importantly, the circulating wave fronts around the two ends of the arc show less conduction



delay. The ladder diagram shows that a 3:2 Wenckebach conduction pattern continued during the concealed trigeminal sequence in one of the two reentrant pathways represented by electrogram C. However, the circulating wave front arrived at site C 20 ms earlier during concealed trigeminy compared with manifest reentry. In contrast, there was a concomitant 6:5 Wenckebach conduction sequence in the other reentrant pathway represented by electrograms D and E. This, in addition to other sites that were, at the same time, showing a 2:1 conduction pattern (site F in Fig. 2), illustrates the presence of a high degree of dissociated conduction in the ischemic zone even though the surface electrocardiogram may show a rather regular extrasystolic pattern.

Figure 2. Isochronal maps of a reentrant trigeminal rhythm. The figure illustrates epicardial activation maps (**top**) and selected electrographic recordings (**bottom**) from a dog 4 days after infarction in which a reentrant trigeminal rhythm developed during sinus tachycardia. Epicardial activation is displayed as if the heart is viewed from the cardiac apex located at the center of the circular map. The perimeter of the circle represents the atrioventricular junction. This display is helpful in illustrating reentrant circuits located around the cardiac apex. The epicardial map of a sinus beat at a cycle length (CL) of 325 ms is labeled 1. Spontaneous shortening of the sinus cycle length to 305 ms resulted in the development of a single reentrant beat after every second sinus beat. The maps labeled 2, 3 and 4 represent the first and second sinus beats and the reentrant beat, respectively. The **dotted line** in map 1 represents the epicardial outline of the ischemic zone. The left anterior descending artery (LAD) is represented by a **double line**. The **asterisks** refer to electrotonic deflections. See text for details.

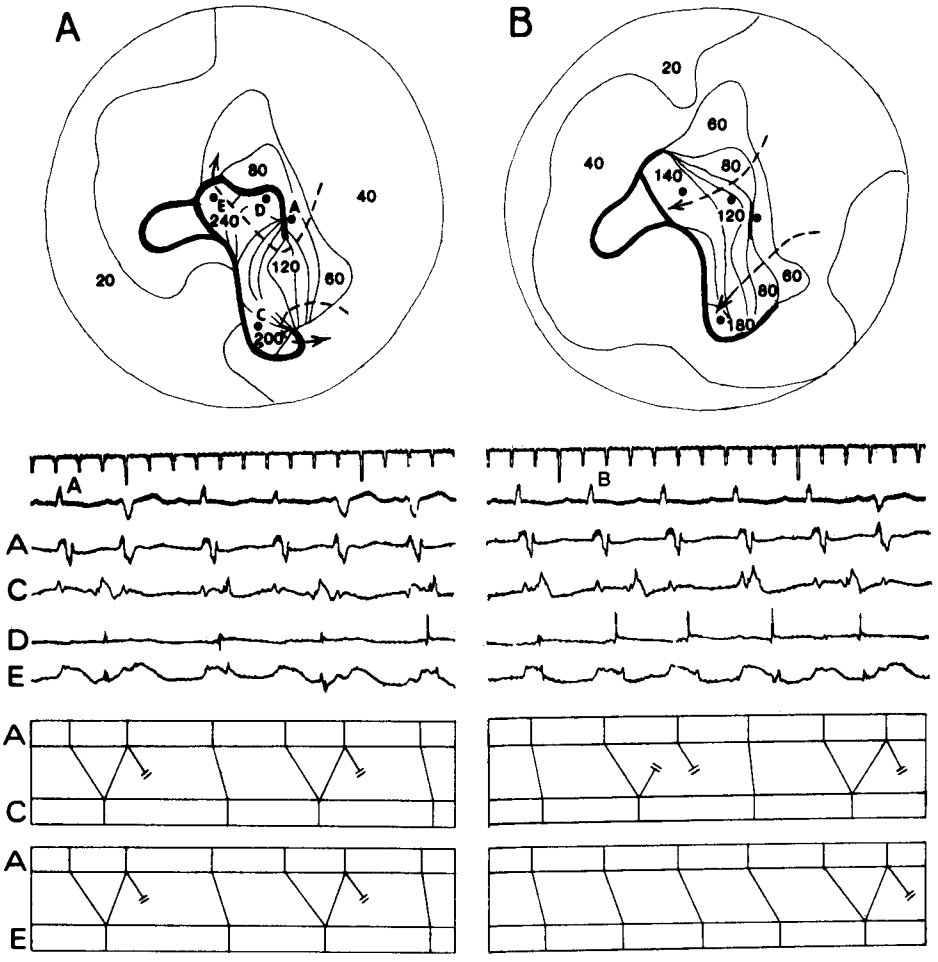


Figure 3. Recordings from the same experiment shown in Figure 2. **Panel A** shows a manifest trigeminal rhythm. The epicardial activation map of the sinus beat that initiated reentry (labeled A) is shown on the **top**, while selected electrograms are shown on the **bottom**. **Panel B** illustrates a concealed trigeminal rhythm at the same sinus cycle length as in **panel A**. The activation map of the second sinus beat that was supposed to initiate reentry (labeled B) is shown on the **top**, and selected electrograms are shown on the **bottom**. The ladder diagram illustrates the conduction pattern between site A, located outside the arc of conduction block, and sites C and E, located within the slow conduction zone during both manifest and concealed trigeminal rhythms. See text for details.

In three of the four experiments, a Wenckebach conduction sequence was also the initiating mechanism for a repetitive reentrant rhythm (Fig. 1 and 3A). The reentrant rhythm occurred at a cycle length of 270 to 320 ms. This was slightly shorter than the sinus cycle length associated with the Wenckebach sequence (290 to 355 ms). In two experiments, the activation pattern during repetitive reentrant excitation was slightly different from that during the reentrant trigeminal rhythm, but the reentrant circuits were also epicardially located. In the third experiment, subsequent "reentrant" cycles could not be localized on the epicardial surface and may have been located intramurally.

Spontaneous versus induced reentrant rhythms. In all experiments in which a spontaneous reentrant rhythm developed, reentry could also be induced by programmed ventricular premature stimulation. Stimulation was applied to the right ventricular outflow tract at an S_1S_1 cycle length of 360 to 400 ms, and a single premature stimulus (S_2) was introduced at a cycle length of 200 to 260 ms. In two experiments, a short pleomorphic rhythm was induced. In the other two experiments, a monomorphic reentrant tachycardia developed. The cycle length of the induced reentrant rhythms was much shorter compared with spontaneous reentrant rhythms (160 to 240 and 270 to 320 ms, respectively). The activation pattern of the induced reentrant rhythms was consistently different and usually much more complicated than that of the spontaneous rhythm.

Figure 4 was obtained from the same experiment shown in Figures 2 and 3 and illustrates the initiation of a fast monomorphic reentrant tachycardia by programmed premature stimulation. The activation maps of the S_1 and S_2 beats and first two reentrant beats (V_1 and V_2) are shown. During S_1 at a cycle length of 360 ms, there was a small arc of conduction block and the entire epicardial surface was activated within 120 ms. S_2 resulted in an extensive island of functional conduction block within the border of the epicardial ischemic zone. The area of block was much more extensive compared with that during spontaneous reentry in Figure 2. A slow wave front circulated around an extension of the zone of block along the septal border of the ischemic zone before reactivating myocardial sites proximal to the arc of block giving rise to a reentrant beat (V_1) with a positive QRS configuration in lead II. The reentrant process continued in a complex activation pattern with a circulating wave front near the apical and lower anterolateral part of the ischemic zone, giving rise to a second reentrant

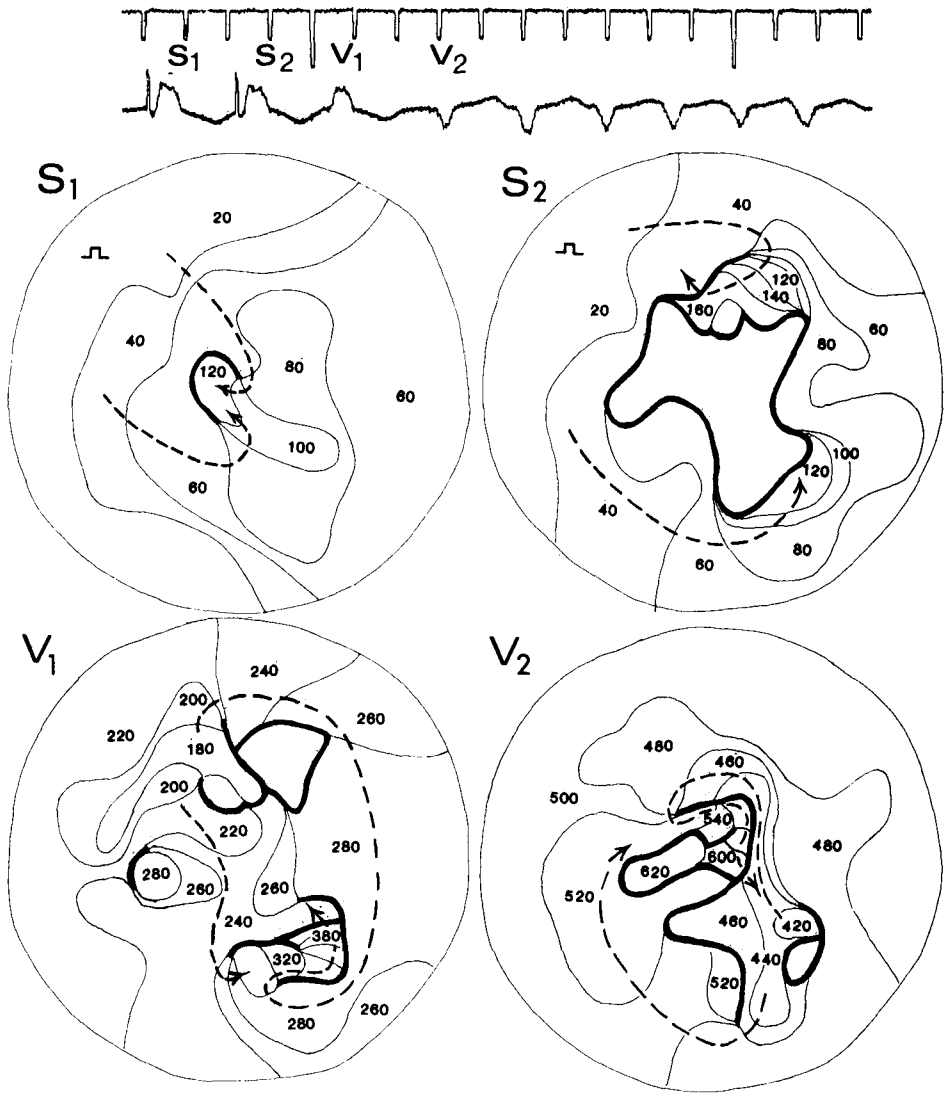


Figure 4. Recordings from the same experiment shown in Figures 2 and 3 illustrating the initiation of a fast monomorphic reentrant tachycardia by a single premature stimulus (S_2) during ventricular pacing (S_1). The activation maps of S_1 and S_2 and the first two reentrant beats (V_1 and V_2) are shown. See text for details.

beat (V_2) with a negative QRS configuration in lead II. A beat to beat variation of the configuration and site of the arcs of functional conduction block and circulating wave fronts continued to occur during the monomorphic reentrant tachycardia. However, the site of reactivation remained relatively stable near the apical and lower anterolateral part of the ischemic zone. This is seen on the activation map of V_2 where the site, configuration and direction of the circulating wave front were different from those in V_1. Yet the reactivation site was contiguous in the two maps. This resulted in an approximately similar activation pattern of the epicardial surface of the normal zone, that is, outside the slowly conducting ischemic zone (not shown in Fig. 4), and explains the similar QRS configuration of consecutive reentrant beats.

Intramural versus epicardial reentrant circuits. In two experiments, reentrant circuits could be localized in the septal intramural region. Postmortem examination of both dogs revealed a large anastomatic vessel arising from the posterior descending artery and curving around the cardiac apex to fill the left anterior descending artery distal to ligation. This resulted in a small and rather patchy anteroseptal infarction with a thick epicardial layer of surviving myocardium. The findings from one of these two experiments are illustrated in Figure 5. In this experiment, two premature stimuli (S_2S_3) could induce a run of nonstimulated beats. However, the epicardial activation map of S_3 failed to show evidence of reentrant excitation. Several intramural needle recordings were obtained from the upper septal and paraseptal regions (marked by solid circles on the epicardial map). The intramural recordings revealed the presence of a small reentrant circuit (approximately 10 mm in diameter) located in the septum, 6 to 10 mm deep to the anterior epicardial surface. A composite intramural map of S_3 at the level of intramural sites A, B and C is also shown in Figure 5. The map illustrates that S_3 resulted in a continuous arc of functional conduction block, with two circulating wave fronts around both ends of the arc of block. The two wave fronts coalesced, then advanced slowly within the arc before breaking through the arc to initiate the first reentrant beat.

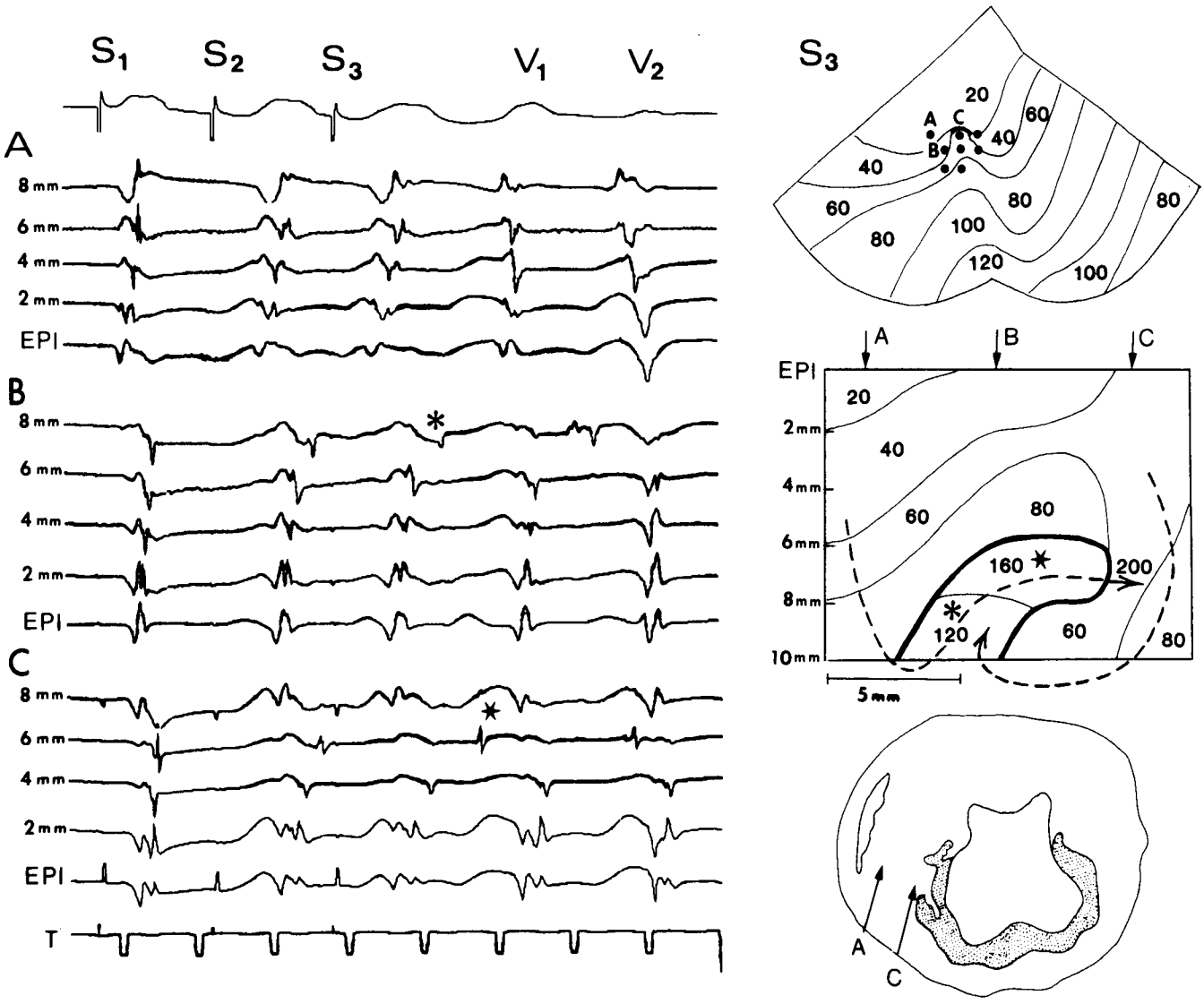


Figure 5. Epicardial and intramural activation maps from a dog 4 days after infarction showing an intramural reentrant circuit. The introduction of two premature stimuli (S_2 and S_3) resulted in the induction of two nonstimulated beats (V_1 and V_2). The epicardial isochronal map of S_3 is shown on the **top right**. The position of intramural needle recordings from the upper septal and paraseptal region are marked by **solid circles** on the epicardial map. Epicardial and intramural recordings at sites A, B and C are shown on the **left** of the figure. A composite intramural map of S_3 at the level of intramural sites A, B and C is shown at the right center. A cross section of the heart at the level of intramural recordings A and C is shown in the **lower right**. The **asterisks** illustrate the position of two delayed electrograms at intramural sites B and C on the intramural map. The epicardial (EPI) map is depicted as if the ventricles were folded out after a cut was made from the crux to the apex. See text for details.

Evidence that the reentrant circuit had a tridimensional configuration is shown by analysis of intramural recordings at site C. The recording shows that functional conduction block developed between electrograms recorded 6 mm deep to the epicardial surface and both myocardial layers anterior and posterior to this zone.

Discussion

Mechanism of spontaneous reentrant rhythms. The present study has shown that a tachycardia-dependent Wenckebach conduction sequence in a potentially reentrant pathway is the initiating mechanism for spontaneous reentry, that is, reentry occurring during a regular cardiac rhythm as contrasted with reentry induced by the introduction of one or more premature stimuli. This process can be repetitive giving rise to a reentrant tachycardia or it may result in a single reentrant cycle in a repetitive pattern giving rise to a reentrant extrasystolic rhythm. All the reentrant extrasystolic rhythms seen in the present study were in the form of manifest or concealed trigeminal rhythm. This was attributed to a tachycardia-dependent 3:2 Wenckebach conduction sequence in a reentrant pathway. Electrograms re-

corded from the reentrant pathway clearly demonstrated the 3:2 Wenckebach conduction sequence. At longer cardiac cycle lengths, those same sites showed a 1:1 conduction pattern. The in vitro correlate of these extracellular electrograms has been previously demonstrated in transmembrane recordings from cells in the ischemic epicardial layer (5). These cells showed variable degrees of partial depolarization, reduced action potential amplitude and decreased upstroke velocity. They also showed the phenomenon of postrepolarization refractoriness where full recovery of responsiveness outlasted the action potential duration.

A concealed trigeminal rhythm can occur during a 3:2 Wenckebach sequence if the slow wave front shows less conduction delay than is necessary for sites proximal to the arc of block to recover excitability. However, a concealed trigeminal rhythm may occasionally reflect a longer 6:5 Wenckebach conduction sequence. In a previous study (6) that utilized composite electrode recordings in the same animal model, examples of reentrant bigeminal rhythms were explained on the basis of a 2:1 conduction sequence in a potentially reentrant pathway. However, we did not have the chance to analyze activation maps of a reentrant bigeminal rhythm in the present study.

Reentrant rhythm induced by premature stimulation. A recent study (7) correlated activation and refractory maps during reentrant excitation induced by premature stimulation in the same canine model. Ischemia was found to result in a lengthening of refractoriness with a nonhomogeneous distribution usually in the form of concentric isochrones of refractoriness with a graded increase in refractoriness going from the border zone (effective refractory period of 150 to 190 ms) toward the center of the ischemic zone (effective refractory period up to 360 ms). A critically timed premature stimulus that succeeded in inducing reentry resulted in an arc of unidirectional block around which the reentrant wave front circulated. The arc of conduction block occurred between adjacent sites of short and long refractoriness, with the sites of longer refractory periods distal to the arc of block. Although we have not analyzed refractory maps in experiments in which a reentrant rhythm occurred spontaneously (as shown in the present study), it is possible to speculate on the changes of refractoriness in these experiments. Arcs of conduction block associated with spontaneous reentry occurred at cycle lengths of 290 to 355 ms. In contrast, the critical coupling interval of premature beats necessary to initiate reentry is usually much shorter (160 to 250 ms) (1,2). This suggests that the degree and possibly the distribution of ischemia-induced lengthening of refractoriness are greater in those dogs showing spontaneous reentry. In these dogs, reentrant excitation could also be induced by premature stimulation at cycle lengths much shorter than those associated with spontaneous reentry. It is expected in this case that the premature beat will result in a more extensive arc of conduction block compared with that associated with spontaneous reentry. A comparison of the activation maps in Figures 2 and 4 shows that this was indeed the case in the present study. The figures also show that the reentrant pathways during reentry induced by premature stimulation were always different from reentrant pathways during spontaneous reentry and were usually associated with a much more complex activation pattern.

Intramural versus epicardial reentrant circuits. Electrophysiologic-anatomic correlation of reentrant excitation in the present canine model has shown that both the arcs of functional conduction block and the slow activation fronts of reentrant circuits usually developed in the surviving electrophysiologically abnormal epicardial layer overlying the infarction. Conduction across the perpendicular axis of the thin epicardial layer usually was synchronous or showed only slight dispersion compared with conduction in the horizontal axis. Thus, a majority of reentrant circuits in this canine model could be viewed as having essentially a two-dimensional configuration. However, we have described experiments in which the reentrant circuit was located in a surviving subendocardial myocardial rim surrounding an apical infarction (8). These observations are of special interest because they simulate closely reentrant circuits described in the human heart around the scar of ventricular aneurysm (9). The latter may be similarly located in viable although electrophysiologically abnormal myocardial bundles in the subendocardial region and around the aneurysmal scar (10). The present study describes other experiments in which the reentrant circuit was located intramurally in close proximity to a patchy septal infarct. In these experiments, the infarction was rather small and had a thick layer of surviving epicardium, probably attributed to the presence of a brisk collateral circulation. In a limited way, these dogs may resemble the experimental infarction induced by coronary occlusion and reperfusion, which usually results in patchy necrosis. In the occlusion-reperfusion model, induced sustained tachycardias could not be attributed to an epicardial reentrant circuit (11). However, the possibility of an intramural circuit was not excluded. Obviously, a small intramural circuit may be difficult to demonstrate in the absence of extensive intramural mapping.

The individual examples of subendocardial and intramural reentry in the present canine model clearly establish that, depending on the particular anatomic features of the infarction and the geometric configuration of surviving ischemic myocardium, reentrant circuits could be located in epicardial, subendocardial or intramyocardial zones. However, irrespective of the anatomic localization of the circuit or its dimension (large or small), its configuration still conforms to the figure 8 activation pattern (or a modification thereof) (Fig. 2).

Models of reentrant excitation: ring model, leading circle model and figure 8 model. Early experimental observations (12–15) showed the existence of an entrapped

circuit wave (circus contractions or movement) in rings of living cardiac and other tissue cut from a variety of animals including mammals. The presence of a fixed anatomic obstacle was considered an important requirement for the occurrence of circus movement. Whether these experiments are related to human cardiac arrhythmias is the hypothesis itself, first proposed by Mines (14,15). Guided by his observations in rings of muscle, Lewis et al. (16) tried to prove that the atrial flutter wave circulated around a natural opening in the muscles of the auricle (the venae cavae). Schmitt and Erlanger (17) suggested that a loop composed of a branching peripheral Purkinje fiber bundle and ventricular muscle may sustain a circus movement similar to rings of muscle.

Although a circus movement in a Purkinje muscle loop is possible, it is difficult to demonstrate in the in situ heart. To date, the only two proven examples of ring model reentry in the intact mammalian heart are: 1) the pre-excitation syndrome first suggested by Mines (14) shortly after Kent (18) first demonstrated the multiple muscular connections between auricles and ventricles in human hearts, and 2) circus movement involving both bundle branches (bundle branch reentry) first suggested by the experimental observations of Moe et al. (19). What is common to the pre-excitation syndrome and bundle branch reentry is that the anatomic substrate is composed, in a large part, of pathways of excitable bundles that are not connected to adjacent atrial and ventricular myocardium. Thus, a single simple circulatory wave could be established. The circuit could be interrupted with ease by cutting at any point along the insulated excitable bundles, but most probably not at the less well defined atrial or ventricular connections of these pathways. However, there are no such insulated excitable bundles in the ventricles or atria but rather an interconnected syncytial structure. In this regard, the observations of Allessie and coworkers (20–22) that a fixed anatomic obstacle is not required for the development of a circus movement are of considerable significance. These investigators showed that in small pieces of atrial myocardium of the rabbit, a properly timed premature stimulus can initiate a circus movement tachycardia. The center of the circuit or the vortex is made of excitable tissue that has been rendered functionally inexcitable by invasion of the center by multiple centripetal wavelets from the leading circuit outside the vortex (Fig. 6C). The circumference of the circuit could be as small as 6 to 8 mm.

Leading circle model: figure 8 model. A critical analysis of the leading circle model of Allessie et al. (21) shows that it is indeed a special modification of the figure 8 model of reentry that can probably exist only in an isolated preparation but not in the intact heart. Figure 6 shows a slightly modified version of the isochronal maps of the premature stimulus that initiated circus movement reentry (labeled A) and the first reentrant beat (labeled B) from an atrial muscle preparation of the rabbit (21). The arcs of functional block are represented by heavy solid lines instead of double bars as in the original drawing. The S_2 map shows that a properly timed premature stimulus resulted in a continuous arc of functional conduction block. The activation wave front circulated around both ends of the arc, coalesced and then broke through the arc to reexcite myocardial zones on the proximal side of the arc. This resulted in the splitting of the original single arc into two separate arcs. Figure 6B shows that a circulating wave front continued around one of the two arcs. However, the second arc of block shifted its site significantly and developed in an area that was showing crowded isochrones in the S_2 map. More significantly, this arc joined the edge of the preparation so that a second circulating wave front around this arc was aborted. If the preparation shown in Figure 6B is reinserted in the in situ heart, the second aborted circulating wave front would be activated, thus resulting in a figure 8 activation pattern. The only situation in the in situ heart that would simulate the in vitro activation map shown in Figure 6B is that in which one of the two arcs of block joined the atrioventricular

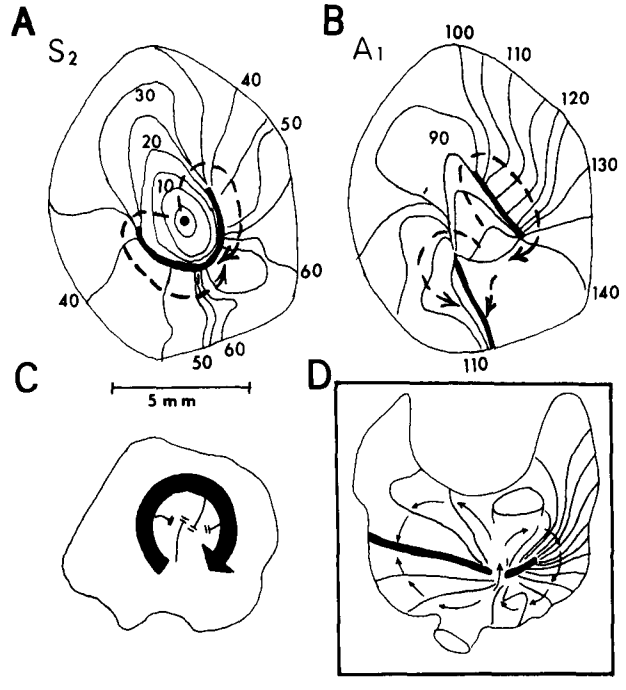


Figure 6. The leading circle model of reentry. **A** and **B** are isochronal maps of activation of a premature stimulus (S_2) and the first reentrant beat (A_1) from an in vitro preparation of atrial myocardium of the rabbit. See text for details. **C**, Diagrammatic illustration of the leading circle model. **D**, In vivo isochronal map of atrial activation during atrial flutter in a dog heart showing an activation pattern similar to that in **B**. See text for details. (**A** and **B** are modified from Allessie MA, et al. [21] and **C** is modified from Allessie et al. [22] with permission from the American Heart Association, Inc. **D** is modified from Boineau JP, et al. [23] with permission.)

junction. Such an example could, indeed, be found in some in vivo maps of atrial flutter in the canine heart as demonstrated by Boineau et al. (23) (Fig. 6D). In this example, a single clockwise circulating wave is seen around a zone (arc) of functional conduction block. The second potential circulating wave front in a figure 8 reentry model was prevented when the second arc of block connected to the atrioventricular junction. Thus, a figure 8 pattern seems central to the occurrence of "repetitive" reentrant excitation (short of fibrillation) in the interconnected syncytial structure of the atria and ventricles.

It should be emphasized that the dimension of the reentrant circuit in the ventricle could be as small as 10 mm (Fig. 5), and depending on the distribution of the pathologic features of the myocardium, these circuits can be located in the epicardial, intramural or subendocardial regions. Thus, the long arcs of functional conduction block that sustain large reentrant circuits in the canine postinfarction ventricle and the small vortices of functional block described by Allessie et al. (20–22) that sustain small reentrant circuits in rabbit atrial myocardium may represent two ends of a spectrum of the same electrophysiologic phenomenon.

References

1. Mehra R, Zeiler RH, Gough WB, El-Sherif N. Reentrant ventricular arrhythmias in the late myocardial infarction period. 9. Electrophysiologic-anatomic correlation of reentrant circuits. Circulation 1983;67:11–24.
2. El-Sherif N, Mehra R, Gough WB, Zeiler RH. Reentrant ventricular arrhythmias in the late myocardial infarction period. Interruption of reentrant circuits by cryothermal techniques. Circulation 1983; 68:644–56.
3. El-Sherif N, Scherlag BJ, Lazzara R. Reentrant ventricular arrhythmias in the late myocardial infarction period. 1. Conduction characteristics in the infarction zone. Circulation 1977;55:686–701.
4. El-Sherif N, Mehra R, Gough WB, Zeiler RH. Reentrant ventricular arrhythmias in the late myocardial infarction period. 11. Burst pacing versus multiple premature stimulation in the induction of reentry. J Am Coll Cardiol 1984;4:295–304.
5. El-Sherif N, Lazzara R. Reentrant ventricular arrhythmias in the late myocardial infarction period. 7. Effects of verapamil and D-600 and role of the "slow channel." Circulation 1979;60:605–15.
6. El-Sherif N, Lazzara R, Hope RR, Scherlag BJ. Reentrant ventricular arrhythmias in the late myocardial infarction period. 3. Manifest and concealed extrasystolic grouping. Circulation 1977;56:225–34.
7. El-Sherif N, Gough WB, Mehra R, Zeiler RH, Restivo M. The effect of differential shortening of refractoriness in the successive short cardiac cycles on the initiation and termination of reentry in the ischemic canine heart (abstr). J Am Coll Cardiol 1984;3:477.
8. El-Sherif N, Mehra R, Gough WB, Zeiler RH. Ventricular activation patterns of spontaneous and induced ventricular rhythms in canine one-day-old myocardial infarction. Evidence for focal and reentrant mechanisms. Circ Res 1982;51:152–66.
9. Horowitz LN, Josephson ME, Harken AH. Ventricular resection guided by epicardial and endocardial mapping for treatment of recurrent ventricular tachycardia. N Engl J Med 1980;302:589–97.
10. Fenoglio JJ, Pham TD, Harken AH, Horowitz LN, Josephson ME, Wit AL. Recurrent sustained ventricular tachycardia: structure and ultrastructure of subendocardial regions in which tachycardia originates. Circulation 1983;68:518–33.
11. Wit AL, Allessie MA, Bonke FIM, Lammers W, Sweets J, Fenoglio JJ. Electrophysiologic mapping to determine the mechanism of experimental ventricular tachycardia initiated by premature stimulation. Experimental approach and initial results demonstrating reentrant excitation. Am J Cardiol 1982;49:166–84.
12. Mayer AG. Rhythmical Pulsation in Scyphomedusae: II. Carnegie Institute. Papers. Washington Tortugas Lab 1:113–31. Carnegie Institute Publication 1908, no. 102, part VII.
13. Garrey WE. The nature of fibrillary contraction of the heart. Its relation to tissue mass and form. Am J Physiol 1914;33:497–8.
14. Mines GR. On circulating excitations in heart muscles and their possible relations to tachycardia and fibrillation. Trans R Soc Can 1914;8:43–52.
15. Mines GR. On dynamic equilibrium in the heart. J Physiol (Lond) 1913;46:350–83.
16. Lewis T, Feil HS, Stroud WD. Observations upon flutter and fibrillation, Part II. The nature of auricular flutter. Heart 1920;7:191–233.
17. Schmitt FO, Erlanger J. Directional differences in the conduction of the impulse through heart muscle and their possible relation to extrasystolic and fibrillary contractions. Am J Physiol 1928/1929;87:326–47.
18. Kent AFSA. Conducting path between the right auricle and the external wall of the right ventricle in the heart of the mammal. J Physiol (Lond) 1914;48:22–38.
19. Moe GK, Mendez C, Han J. Aberrant AV impulse propagation in the dog heart: a study of functional bundle branch block. Circ Res 1965;16:261–86.
20. Allessie MA, Bonke FIM, Schopman FJG. Circus movement in rabbit atrial muscle as a mechanism of tachycardia. Circ Res 1973;33:54–62.
21. Allessie MA, Bonke FIM, Schopman FJG. Circus movement in rabbit atrial muscle as a mechanism of tachycardia. II. The role of nonuniform recovery of excitability in the occurrence of unidirectional blocks, as studied with multiple microelectrodes. Circ Res 1976;39:168–77.
22. Allessie MA, Bonke FIM, Schopman FJC. 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. Circ Res 1977;41:9–18.
23. Boineau JP, Schussler RB, Mooney CR, et al. Natural and evoked atrial flutter due to circus movement in dogs. Role of abnormal atrial pathways, slow conduction, nonuniform refractory period distribution and premature beats. Am J Cardiol 1980;45:1167–81.