JACC Vol. 20, No. 4
October 1992:869–78

ELECTROPHYSIOLOGIC STUDIES

Endocardial Mapping of Ventricular Tachycardia in the Intact Human Heart. II. Evidence for Multiuse Reentry in a Functional Sheet of Surviving Myocardium

EUGENE DOWNAR, MD, FACC, SHANE KIMBER, MD, LOUISE HARRIS, MB, FACC, LYNDA MICKLEBOROUGH, MD, FACC, ELIAS SEVAPTSIDIS, DCS, STEPHANE MASSE, MS THOMAS C. K. CHEN, MD, ADRIANA GENGA, RN

Toronto, Ontario, Canada

Objective. **The purpose of this study was to obtain improved detection and characterization of reentrant circuits in the infarcted human ventricle.**

Background. **The return path of reentrant ventricular arrhythmias usually is not manifested in clinical mapping studies but is thought to be formed by isolated bundles of surviving myocytes whose presence is difficult to detect by standard recording techniques.**

Methods. **We obtained simultaneous unipolar and high gain bipolar recordings using a left ventricular endocardial balloon array in 10 patients with chronic ischemic heart disease undergoing intraoperative mapping of ventricular tachycardia.**

Results. **Three patients demonstrated seven separate ventricular tachycardias that utilized a return tract that was manifested on up to 20% of all left ventricular electrode sites. The recordings suggested an extensive sheet of surviving myocardial fibers with multiple entry and exit points allowing for different reentrant paths at different times all in the same heart. In one patient, five different ventricular tachycardias could be induced, four of which utilized such a sheet. Two of these tachycardias had the same exit point (site of origin) but two different entry points with a long and short return path resulting in long and short tachycardia cycle lengths. The same sheet sustained another tachycardia with one entry and two exit points resulting in two separate "sites of origin" on the endocardium. Such sheets also were seen to insert into the left bundle system. In one patient portions of the sheet could be detected epicardially.**

Conclusion. **The existence of such a structure of surviving myocardium with functional pleomorphism may account for unexplained changes in tachycardia cycle length, epicardial entrainment and spontaneous morphologic changes during ventricular tachycardia.**

(J Am Coll Cardiol 1992;20:869–78)

In chronic ischemic heart disease, ventricular tachycardia is believed to be due to reentry. Evidence for this cause is mostly indirect (1,2) because intraoperative mapping studies reveal the full reentrant circuit in only a minority of cases (3–5). In >70% of mapping studies, a site of origin is identified that is followed by radial spread of activation or an incomplete circle with no manifestation of the return path. Such paths are believed to exist but are thought to be formed by isolated bundles of surviving myocytes whose presence is difficult to detect by standard recording techniques (6). We used simultaneous unipolar and high gain bipolar recordings to facilitate this detection and were surprised to find in some patients evidence of an extensive sheet of surviving fibers rather than isolated tracts. Multiple connections between the sheet and the surrounding myocardium allowed for multiple circuits of reentry to occur. We present electrophysiologic data supporting such a structural arrangement of surviving myocardium, and discuss its implications.

Methods

Patient selection. From March 1990 to April 1991, 10 patients with spontaneous recurrent ventricular tachycardia and coronary artery disease underwent intraoperative balloon mapping with simultaneous unipolar and bipolar recordings of left ventricular endocardial activation. All patients underwent preoperative electrophysiologic studies during which their clinical arrhythmias were reproduced by programmed electrical stimulation. The indication for surgery was either refractory ventricular arrhythmias or unstable angina in patients with medically controlled arrhythmias. Informed consent was obtained for all preoperative and intraoperative procedures.

From the Divisions of Cardiology and Cardiovascular Surgery, Toronto General Hospital and Women's College Hospital, University of Toronto, Toronto, Ontario, Canada. Dr. Downar is a recipient of the Career Investigator Award of the Heart and Stroke Foundation of Ontario, Toronto. Dr. Harris is a recipient of a Scholarship from the Heart and Stroke Foundation of Canada, Ottawa, Ontario. This work was supported by grants from the Heart and Stroke Foundation of Ontario, Toronto.

Manuscript received October 31, 1991; revised manuscript received April 14, 1992, accepted April 20, 1992.

Address for correspondence: Eugene Downar, MD, Toronto General Hospital, 12 Eaton North-224A, 200 Elizabeth Street, Toronto, Ontario, M5G 2C4, Canada.

0735-1097/92/$5.00

Table 1. Findings in the Three Patients With a Broad Return Tract

Pt No.	Age (yr)/ Gender	Infarct Site	Aneurysm	Cardiac Arrest	No. of VTs	% of Endocardium With Diastolic Potentials	Postoperative Electrophysiologic Study
1	55/F	Anteroseptal	Yes	Yes	2	19	Positive
2	61/F	Anteroseptal	Yes	Yes	4	17	No clinical VT
3	55/M	Anteroseptal	Akinesia	Yes	1	12	Negative

All three patients underwent aneurysmectomy and map-directed scar resection and cryoablation. F = female; M = male; Pt = patient; VT = ventricular tachycardia.

Recording electrodes. An array of silver bead electrodes, mounted on a nylon mesh stretched over a double latex balloon, was used for endocardial mapping (7). Previously we used single beads to record unipolar electrograms and used adjacent beads in each row to provide bipolar electrograms. In this study, each electrode consisted of a couplet of two silver beads mounted 2 mm apart and sutured to the nylon mesh. Teflon-coated stainless steel was soldered to the electrodes, which were arranged in a matrix of 112 electrodes in 14 rows, each row extending from apex to base. The interelectrode distances varied from 1 to 3 cm. A matching sock array of button electrodes was used to obtain simultaneous epicardial maps (8).

Mapping procedure. At operation, the patient was placed on normothermic cardiopulmonary bypass. An electrode for ventricular stimulation was sutured over the right or left ventricle. The compact deflated endocardial balloon (1.5 to 2 cm in diameter) was introduced through a left atriotomy (9). The balloon was advanced across the mitral valve into the left ventricle so that the first row of electrodes approximated the course of the left anterior descending coronary artery. Stable electrograms were obtained by inflating the balloon with dextrose solution. Approximately 60 ml of solution was required and the pressure was monitored throughout to prevent subendocardial ischemia. Intrinsic balloon pressure was determined for incremental volumes of solution before balloon insertion. When the balloon was positioned and inflated in the left ventricle, pressure was again recorded. Left ventricular end-diastolic pressure equaled recorded pressure in the ventricle minus the intrinsic balloon pressure at the same volume. This pressure was maintained at 15 to 20 mm Hg to reduce the likelihood of compromising subendocardial blood flow. The sock array of electrodes was then pulled over the heart and positioned so that the first row of electrodes was aligned along the left anterior descending artery. Contact between the electrodes in the epicardium was facilitated by moistening the surfaces with saline solution and adjusting the tension of the suture threaded through the mouth of sock that was positioned over the atrioventricular groove. With the balloon and sock in position, programmed stimulation was then performed with use of the protocol that successfully induced ventricular tachycardia at the preoperative electrophysiologic study.

Recording system. Local unipolar electrograms from one of each of the silver couplets on the balloon array and from each of the button electrodes of the sock array were multiplexed and recorded on the video recorder as previously described (7). A second recorder linked to a common time code was used to record simultaneous high gain, close bipolar signals from each of the endocardial bead couplets. The input amplifiers were high impedance (field effect transistor input stage) and configured in a bipolar arrangement with a gain of up to 3,000 and a 3 dB bandwidth of 50 to 200 Hz. The off-line records were used to provide a manually generated activation map with an accuracy of +4 ms. Local activations were defined by the dominant negative deflection on the isoelectric line and activation times were measured from a common time reference provided by the time code. When an individual electrogram was not initially interpretable because of broad multiphasic deflections, review of adjacent electrograms with less complex waveforms often permitted the determination of an activation time.

Results

Patients with a return tract. A broad return tract was evident in 3 of the 10 patients who underwent simultaneous unipolar and high gain bipolar left ventricular endocardial mapping (Table 1). These three nonconsecutive patients had a total of seven separate ventricular tachycardias that utilized a return tract that was manifested on ≤20% of all left ventricular endocardial electrode sites. In the other seven patients, only a site of earliest activation, with no manifestation of a return tract, was found.

Figure 1. This figure shows left ventricular endocardial activation maps in a polar projection from one of these three patients (Patient 1, Table 1). Panels A and B depict in 12-ms isochrones the excitation sequence of two consecutive beats of a ventricular tachycardia whose initiation is shown in Figure 2. Each beat started with an elliptic band of excitation high on the left ventricular septum and ended at an inferoapical region 228 ms later. The next beat of the tachycardia also started on the septum, at 408 ms. There appeared to be a large spatial (7-cm) and a large temporal (180-ms) separation between the end of one beat and the beginning of the next. Close scrutiny of the local electrograms revealed diastolic potentials on virtually a full quadrant of the total endocardium linking the latest activation of one beat and the beginning of the next. These diastolic potentials could be identified stretching from apex to base on some electrode

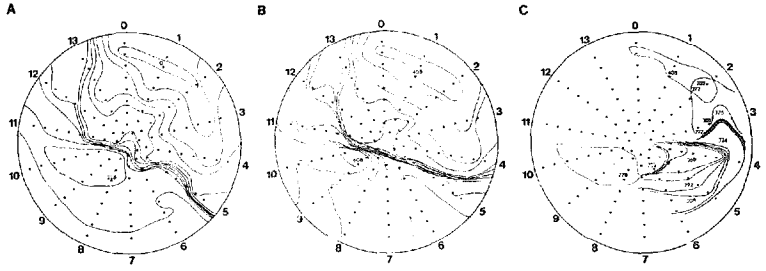
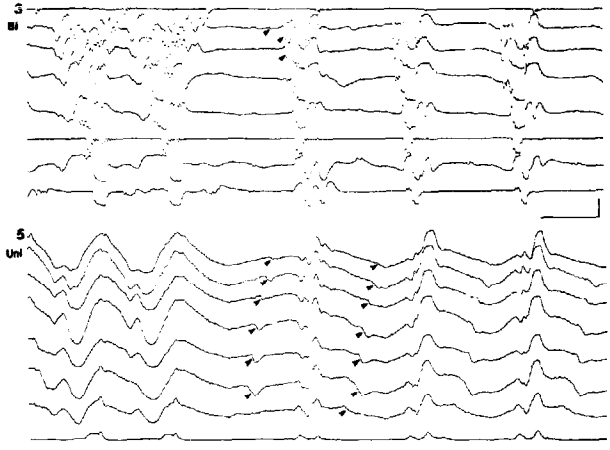


Figure 1 (above). Left ventricular endocardial activation maps shown in a polar projection. Fourteen electrode rows (0 to 13) extend from the base (periphery) to the apex (center). Row 0 is parallel to the course of the left anterior descending coronary artery. **A** and **B** depict 12-ms isochrones of two consecutive beats of the ventricular tachycardia shown in Figure 2. The first beat begins with an elliptic band of activation (0 ms) across electrode rows 0, 1 and 2 and ends at 228 ms in an inferoapical region. The next beat (**B**) starts at 408 ms and follows a similar spread of activation. **C** shows the activation sequence of diastolic potentials linking the end of the first beat and the start of the second (see text).

Figure 2 (below). Initiation of the ventricular tachycardia shown in Figure 1. The **upper panel** shows bipolar (**Bi**) electrograms from row 3 from the base (**upper tracings**) to the apex (**bottom tracings**), and the **lower panel** shows simultaneously recorded unipolar (Uni) electrodes from row 5. Two premature paced beats are followed by the first three beats of a sustained ventricular. **Arrowheads** in the **upper panel** show left bundle branch potentials and in the lower panel show diastolic potentials (see text). Calibration 100 ms, 10 mV unipolar: 1 mV bipolar.



JACC Vol. 20, No. 4
October 1992:869–78

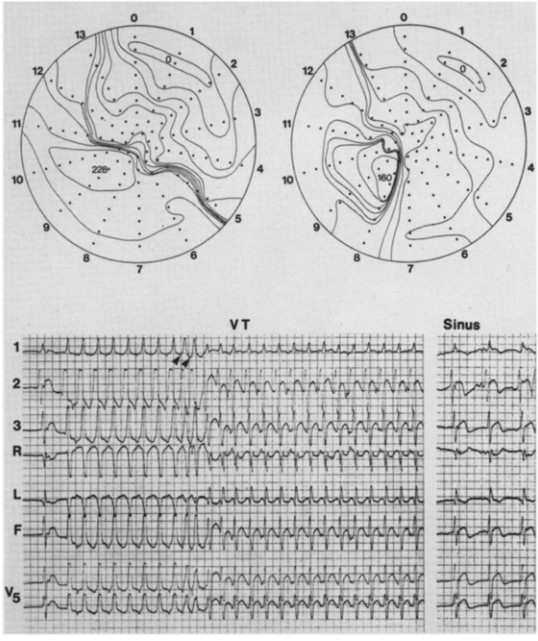


Figure 3. Left ventricular activation sequence (12-ms isochrones) during sinus rhythm and during the ventricular tachycardia shown in Figure 2. Surface electrocardiogram (**from top to bottom**, leads I, II, III, aVR, aVL, aVF and V_5) of the initiation of that tachycardia (VT) and of sinus rhythm (sinus) is shown in the **lower panels**. Note the similar site of earliest activation and initial activation sequence in both instances but slower conduction and different terminal isochrones. **Arrowheads** indicate premature stimuli.

rows such as row 5 in Figure 2, lower panel. Local unipolar electrograms of two premature stimuli are shown followed by the first 3 beats of the ventricular tachycardia. The arrowheads indicate diastolic potentials extending from the apex (bottom trace) to the base (top arrowhead). Conduction proceeded on a broad front (Fig. 1C) from the 232-ms isochrone to the 324-ms isochrone in row 3. There, after a 53-ms delay, the left bundle branch was engaged at 376 ms in row 3 and at 388 and 392 ms in row 2 to complete the return cycle to the start of the next beat at 408 ms.

Figure 2. The upper panel of Figure 2 shows the left bundle branch potentials (see arrowheads) on a bipolar recording of electrode row 3 obtained at the same time as the electrograms shown in the lower panel. Identical left bundle branch potentials were recorded from these electrodes during sinus rhythm, and the surface configuration of the ventricular tachycardia was virtually identical to that of sinus rhythm.

Figure 3. Figure 3 shows the surface electrocardiogram (ECG) during sinus rhythm (lower right panel) and during the same induction of the ventricular tachycardia shown in Figure 2. Cryoablation was applied during the tachycardia to the region between the retrograde potentials and the left bundle branch, and the tachycardia was terminated. Subsequently the tachycardia could not be reinduced at postoperative testing.

Figure 4. Figure 4, from the same patient, shows the epicardial activation sequence of the ventricular tachycardia shown in Figure 1. The earliest epicardial activation occurred simultaneously at two separate sites (asterisks) 48 ms after the earliest endocardial activation. The lower panel shows unipolar epicardial electrograms from electrode row 1 at the same moment of initiation of ventricular tachycardia shown in Figure 2. Electrode row 1 of the epicardial sock array was situated over row 5 of the endocardial electrode array. The diastolic potentials observed over so much of the endocardium also were manifested on the adjacent epicardium but on only two electrodes (arrowheads). They appeared 120 ms before the earliest epicardial breakthrough.

Figures 5 to 7. The electrogram in Figure 5 is from Patient 2, Table 1. Panels A and B show the left ventricular

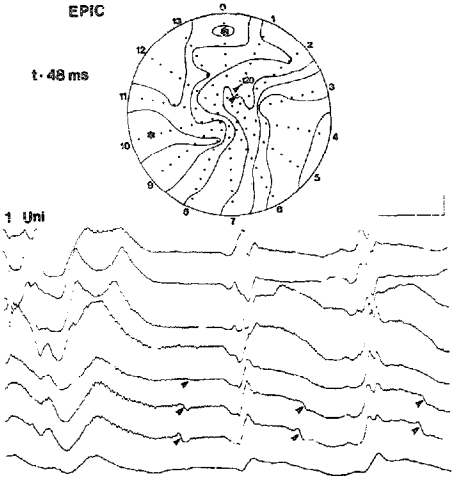


Figure 4. Global epicardial (EPIC) sequence of the same tachycardia. The earliest epicardial activation occurred simultaneously at two separate sites (asterisks) 48 ms after earliest endocardial activation. The **lower panel** shows local unipolar epicardial electrograms from electrode row 1 (1 Uni) at the same initiation of tachycardia shown in Figure 2. **Arrowheads** indicate that diastolic potentials were also manifested on the epicardium over a small region and preceded epicardial breakthrough by 120 ms (see text). Calibration 100 ms, 10 mV. t = time after earliest endocardial activation.

endocardial activation sequence during two consecutive beats of the ventricular tachycardia shown in Figure 6, upper panel. Each beat of the tachycardia started on the second electrode of row 3 (indicated as zero isochrone). Activation proceeded on two encircling fronts moving around the base with the latest activation occurring at the apex at 184 ms. The next beat started 268 ms later at the same electrode as before at 452 ms and followed a similar sequence. Superfi-

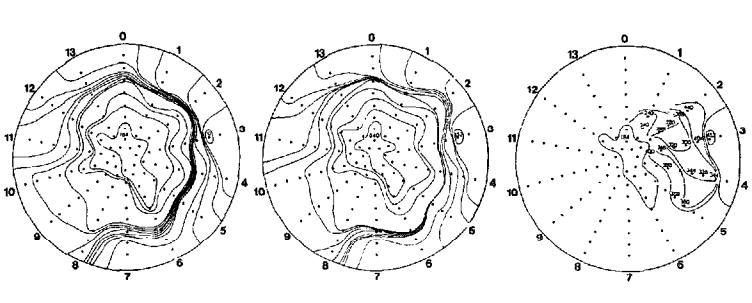


Figure 5 (below). **A** and **B**, Left ventricular endocardial activation sequence (12-ms isochrones) of two consecutive beats of tachycardia from a different patient. Each beat starts at a monofocal origin on the second electrode of row 3 and ends at the apex 184 ms later. **C**, The earliest and latest isochrones were linked over a distance of 7 cm by diastolic potentials that propagated slowly over a broad front (see text).

JACC Vol. 20, No. 4
October 1992:869–78

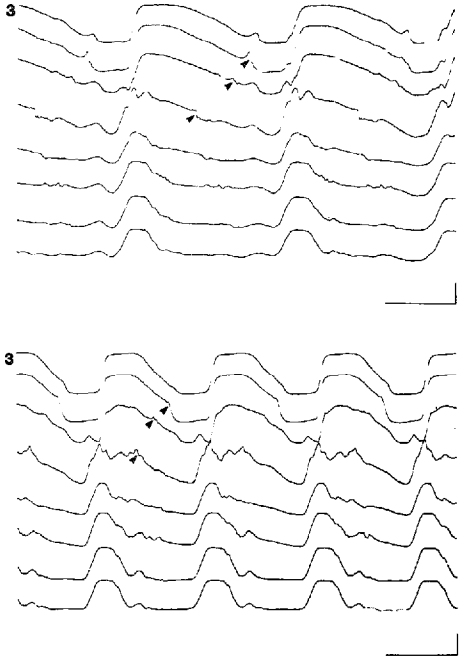


Figure 6. Local unipolar electrograms from two ventricular tachycardias with the same endocardial origin and the same surface configuration. Recordings are from electrode row 3. The **upper panel** relates to the activation maps shown in Figure 5. The **lower panel** is from the maps shown in Figure 7. **Arrowheads** indicate potentials forming part of the return path (see text). Calibration 100 ms, 10 mV.

cially the tachycardia appeared to be unifocal in origin with a spatial gap of at least 7 cm between the earliest and latest isochrones. A detailed review of the local electrograms revealed that a large sector of the endocardium exhibited diastolic potentials of the type shown in Figure 6, upper panel. The sequence of these potentials (Fig. 5, panel C) showed slow propagation of a broad activation front returning from the apex to the site of initiation at the base. As in Patient 1, there was evidence of an extensive sheet of subendocardial fibers; however, the left bundle branch system was not involved in the tachycardia circuit and there was no epicardial manifestation of the diastolic potentials.

In the same patient, a second tachycardia could be induced that had an identical surface configuration morphology to that of the first but was much faster (tachycardia cycle length 312 vs. 452 ms). The endocardial activation is shown in Figure 7, panel A. Each beat of this tachycardia originated at exactly the same site as before and proceeded in a similar manner to encircle the apex, which was last to be activated (at 168 ms). This time, however, diastolic potentials (Fig. 6, lower panel) were seen along electrode row 3 that traced a short path back to the site of origin. Figure 7, panel B shows this return and panel C shows detailed activation times along row 3. The fourth electrode from the base showed fractionated activity (Fig. 6, lower panel) after an initial activation at 112 ms with reactivation at 168 and 204 ms leading to reactivation of the third electrode at 252 ms and the start of the next cycle at 312 ms at the second electrode.

Figure 8. Panels A and B of Figure 8 are from the same patient and show the endocardial activation sequence of two beats of a third ventricular tachycardia, each beat of which originated on two separate sites. Panel A shows that the site of earliest activation occurred on the sixth electrode of row 7. Twelve milliseconds later a second site of activation appears on the fifth electrode of row 4, some 4 cm distant from the first. At 48 ms the two activation fronts had merged

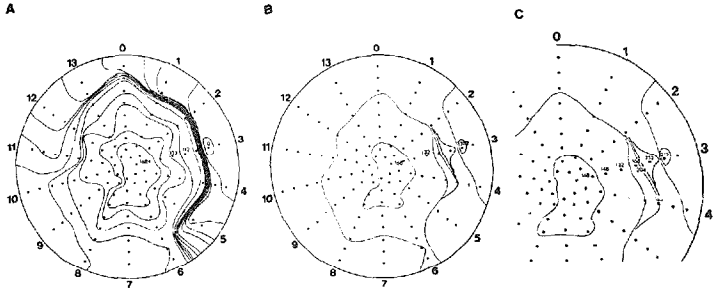


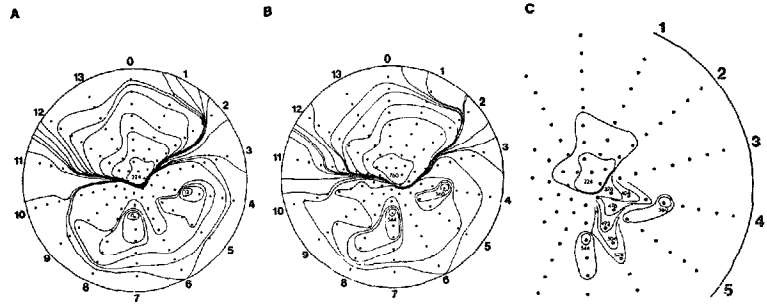
Figure 7. A, Left ventricular activation map (12-ms isochrones) of tachycardia are shown in the lower panel of Figure 6. B and C, Return path and activation times linking one beat to the next (see text).

but were subsequently forced to proceed around an arc of block across the apex. The latest activation occurred at the apex on the distal side of the block at 224 ms. The next beat of the tachycardia (panel B) started at the same two sites as before at 544 and 560 ms, both sites were about 3 cm distant from the region of latest activation. Close examination of the local electrograms revealed that this gap was bridged by a meandering reactivation front shown in panel C.

Figure 9. The diastolic potentials of the return front seen on electrode row 6 of Figure 8C are shown in Figure 9. High gain bipolar electrograms are shown in the lower panel and simultaneously recorded unipolar electrograms in the upper panel. The clearly discernible late diastolic potential indicated by the arrowheads in the lower panel are barely seen in the unipolar electrograms recorded from the same electrodes. Unipolar recordings alone would not have delineated the reentrant sequence.

Figure 10: summary of tachycardias. Figure 10 summarizes the ventricular tachycardias presented in a single conceptual synthesis. Rather than envisaging a single bundle forming a return path (panel A), an extensive sheet of surviving intramural fibers is proposed, linked to the subendocardium by six connections (panel B). Three of these act

Figure 8. Left ventricular endocardial activation of a tachycardia with two separate endocardial origins. A and B show activation of two consecutive beats. C shows details of diastolic activations linking the two beats.



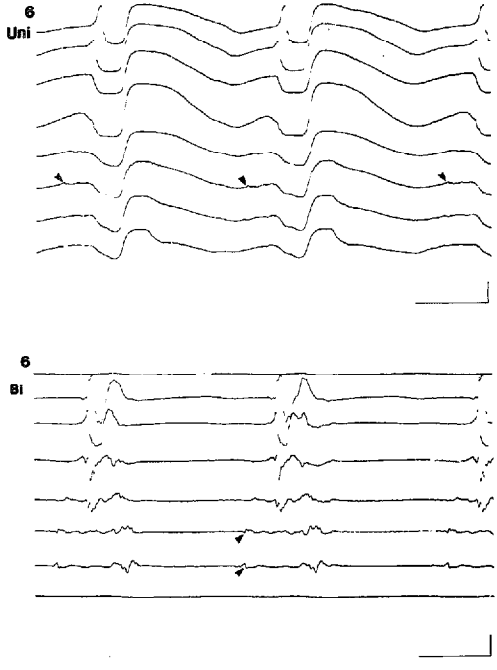


Figure 9. Unipolar (Uni) and bipolar (Bi) electrograms from electrode row 6 recorded during the tachycardia shown in Figure 8. Note that the diastolic potentials easily seen in the bipolar recordings (**arrowheads in lower panel**) are barely visible on the simultaneously recorded unipolar signals (**upper panel**) (see text). Calibration 100 ms, 10 mV **upper panel**, 1 mV **lower panel**.

as "sinkholes" (shaded ovals) providing access from the subendocardium, and three act as exit points (open ovals) providing egress from the sheet to the subendocardium. The functional characteristics of these connections all differ according to geometric and other considerations and their interplay in any given circumstance determines which reentrant route predominates. The connections may extend directly to involve portions of the left bundle branch system (panel C).

Discussion

Previous hypotheses of the mechanism of ventricular tachycardia. Most studies of ventricular tachycardia utilizing clinical mapping (3–5) identify a discrete region of earliest activation referred to as a "site of origin" but rarely establish the mechanism of the tachycardia. When the region of earliest activation is immediately adjacent to the latest activated region, diastolic potentials can sometimes be recorded spanning a small spatial temporal gap as in the case of figure eight reentry (4). Such findings suggest a short narrow path of reentry formed by a discrete bundle of surviving muscle fibers (6,10). Most often the region of latest activation is remote from the earliest activated region and no recordings are obtained of any diastolic activity. Such cases are still believed to be due to reentry despite the large spatial temporal gaps. The explanations offered (6,10) are that the tachycardia is caused by a microreentrant circuit smaller than the spatial resolution of the mapping process, or that there is a macroreentrant circuit whose return path remains undetected by the mapping. It has been proposed (6) that such a path may consist of a small isolated bundle of surviving cells whose extracellular potential would have an amplitude measured in the microvolt range. Such activity

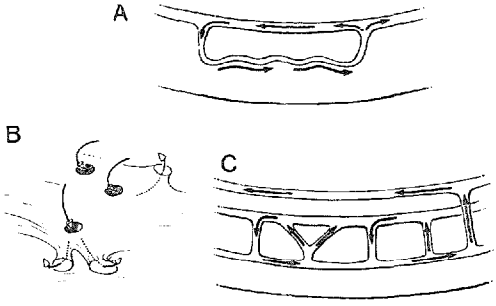


Figure 10. Functional schematic based on the cases discussed. Instead of envisaging a single bundle forming a return path (**A**), an extensive sheet is proposed, linked with the subendocardium through multiple connections (**B**). Some act as "sinkholes" (**shaded ovals**), whereas others provide exit points (**open ovals**) to the subendocardium. The connections may involve portions of the left bundle branch system (**C**) (see text).

would be undetectable to electrodes more than 1 mm away (11).

Present findings and the hypothesis of multiuse reentry in a functional sheet of surviving myocardium. In the hope of improving our ability to detect micropotentials, we used a balloon array and recording system that allowed register of normal electrograms and simultaneous high gain close-bipolar electrograms. There is always a concern that such bipolar recordings may have produced artifactual diastolic potentials. The fact that these recordings were not artifactual is attested to by recordings such as those in Figure 9, which show minimal unipolar manifestation of very clear bipolar electrograms. This approach was successful in clearly identifying activations that were not discernible on conventional unipolar recordings from the same electrodes. The two recording modes together were able to identify large regions of subendocardium that participated in the return portion of the macroreentrant circuit. These findings are not consistent with a small isolated bundle of surviving cell as has been proposed. Rather the finding suggests a functional sheet of surviving muscle, perhaps constituted by an extensive interweaving of many muscle bundles. Such a sheet could account for the meandering sequence of activation seen in some instances.

Two layers of surviving fibers? The subendocardial regions that demonstrated diastolic activation also exhibited fully developed local electrograms that swept rapidly over the same region in systole. The direction of this systolic activation was generally the opposite of that in diastole, and both activations often occurred within <150 ms of each other or within the presumed refractory period of any one site. These considerations strongly suggest two distinct layers of surviving fibers—one immediately below the endocardium forming a continuous mass of rapidly conducting myocardium. The second layer is deeper and probably discontinuous, formed by a smaller muscle mass of interwoven bundles. Because the existence of this functional sheet can best be detected from the endocardial electrode array, it must be located in close proximity to the subendocardium. However, it is possible that portions of the sheet are in a deeper intramural or even subepicardial location, as evidenced by the recording shown in Figure 4. Such externalization of portions of a macroreentrant circuit could allow for entrainment and successful laser photoablation of some ventricular tachycardias from the epicardial surface. Transmural penetration of reentrant circuits is not a novel concept, and successful treatment modalities directed from the epicardium have recently been reported (12).

There may be multiple connections between the functional sheet and the adjacent subendocardial muscle mass. These may function either as routes of access to the sheet or as exit points from the sheet to the subendocardium or even to the left bundle branch system. The limited histologic data available on connecting tracts describe abnormalities in geometric arrangement and narrowed passages that may engender anisotropic properties to the point of providing unidirectional block (10,13,14). In Figure 8 two exits are utilized almost simultaneously, giving rise to two separate sites of origin on the endocardium. It would not be difficult to envisage under slightly different circumstances the conducting properties of these exits changing to allow only one to operate. The futility of directing surgical ablation to a site of origin in this situation need not be emphasized. Equally, graded responses in one or both exits could provide an interplay of relative contributions to global cardiac excitation, which could manifest on the surface electrocardiogram as a polymorphic ventricular tachycardia beyond the scope of surgical ablation.

The routes of access to the sheet may have differing functional properties according to the geometric detail of each connecting tract. It might be reasonable to suppose that circumstances favoring one access route might at another time favor an alternative access without a change in the exit route. This occurrence would mean that the surface configuration of a tachycardia would remain unchanged even though the return route was changed, as in the example

shown in Figures 5 and 7. This possibility might explain some instances in which hemodynamically stable ventricular tachycardia accelerates in response to overpacing to a rapid malignant tachycardia with hemodynamic collapse. The concept of a surviving sheet of muscle fibers with multiple connections to a larger adjacent subendocardial muscle mass can be compared to an electronic matrix. A multiplicity of (reentrant) circuits is possible. Which circuit is actually realized in any given circumstance is determined by an interplay of the functional properties of the connections. These in turn may be determined by individual details of geometry, heart rate, autonomic tone, antiarrhythmic drugs and other as yet identified factors.

Limitations of this study and implications. Detailed histologic studies were not feasible in these intraoperative mapping studies. In the absence of histologic correlates, the concept that we propose has to be speculative and is further limited because it is based on a small number of observations.

A further limitation is the clinical applicability of our findings. We are not suggesting that all cases of ventricular tachycardia in the setting of chronic infarction are supported by such a "multiuse" reentrant sheet. Rather, we propose that chronic infarction can result in an arrangement of surviving muscle fibers that can range over a topologic spectrum. At one end of this spectrum is the classical isolated bundle with one entry and one exit point to the subendocardial muscle mass, and at the other is the sheet with multiple connections. The concept of a surviving sheet has the attraction of encompassing such disparate observations as pleomorphism, epicardial entrainment and accelerations in tachycardia rate.

References

1. Josephson ME, Horowitz LN, Farshidi A, Kastor JA. Recurrent sustained ventricular tachycardia. 1. Mechanisms. Circulation 1978;57:431–40.
2. Josephson ME, Horowitz LN, Farshidi A. Continuous local electrical activity. A mechanism of recurrent ventricular tachycardia. Circulation 1978;57:659–65.
3. Miller JM, Josephson ME. Intra-operative mapping of ventricular tachycardia: utility and pitfalls. Int J Cardiol 1985;8:173–5.
4. Downar E, Harris L, Mickleborough LL, Shaikh N, Parson ID. Endocardial mapping of ventricular tachycardia in the intact human heart: evidence for reentrant mechanisms. J Am Coll Cardiol 1988;11: 783–91.
5. Kaltenbrunner W, Cardinal R, Page P, et al. Ventricular tachycardia in remote myocardial infarction without evidence of superficial reentry (abstr). Circulation 1990;82(suppl III):III-579.
6. de Bakker JMT, van Capelle FJL, Janse MJ, et al. Reentry as a cause of ventricular tachycardia in patients with chronic ischemic heart disease: electrophysiologic and anatomic correlation. Circulation 1988;77:589–606.
7. Chen TCK, Parson ID, Downar E. The construction of endocardial balloon arrays for cardiac mapping. PACE 1991;14:470–9.
8. Downar E, Parson ID, Mickleborough LL, Cameron DA, Yao LC, Waxman MB. On-line epicardial mapping of intraoperative ventricular arrhythmias: initial clinical experience. J Am Coll Cardiol 1985;4:703–14.
9. Harris L, Downar E, Mickleborough LL, et al. Activation sequence of ventricular tachycardia: endocardial and epicardial mapping studies in the human ventricle. J Am Coll Cardiol 1987;10:1040–7.
10. de Bakker JMT, Coronel R, Tasseron S, et al. Ventricular tachycardia in the infarcted, Langendorff-perfused human heart: role of the arrangement of surviving cardiac fibers. J Am Coll Cardiol 1990;15:1594–607.
11. Spach MS, Dolber PC. Relating extracellular potentials and their derivatives to anisotropic propagation at a microscopic level in human cardiac muscle. Evidence for electrical uncoupling of side-to-side fibre connections with increasing age. Circ Res 1986;58:356–71.
12. Littman L, Svenson ..., Gallagher JJ, et al. Functional role of the epicardium in post-infarction ventricular tachycardia: observations derived from computerised epicardial activation mapping, entrainment and epicardial laser photoablation. Circulation 1991;83:1577–91.
13. Joynes RW. Mechanisms of unidirectional block in cardiac tissue. Biophys J 1981;35:113–25.
14. Spach MS, Miller WT 3rd, Dolber PC, Kootsey JM, Sommer JR, Mosher CE Jr. The functional role of structural complexities in the propagation of depolarization in the atrium of the dog: cardiac conduction disturbances due to discontinuities of effective axial resistivity. Circ Res 1982;50:175–91.