The Limitations of Epicardial Mapping as a Guide to the Surgical Therapy of Ventricular Tachycardia

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SUMMARY The adequacy of intraoperative epicardial mapping as a guide to surgical procedures performed to terminate ventricular arrhythmias was investigated. Ligation of the anterior septal or left anterior descending coronary artery in 28 dogs produced ventricular arrhythmias that were studied 24-36 hours following occlusion. The sites of origin of 26 tachycardias were determined to be in the subendocardium by using extensive epicardial, endocardial and intramural mapping techniques and were verified by demonstrating unaltered activation sequences during pacing from these earliest sites. Epicardial breakthrough followed earliest directly recordable ventricular activity by as little as 7 msec. Without endocardial mapping many of these tachycardias would have been incorrectly identified as originating in the fascicles or epicardium. The sites of epicardial breakthrough were anatomically distant from the sites of origin by a markedly varying extent (5 mm to 6 cm). Two rhythms might be close in their sites of earliest epicardial appearance yet distant on the endocardium or vice versa.

We conclude that epicardial mapping may not be sufficient to identify or predict the origins of many ventricular tachycardias and that the success of surgery to abolish these arrhythmias may be enhanced by preoperative and intraoperative endocardial mapping.

ALTHOUGH SEVERAL INVESTIGATORS HAVE EXAMINED THE MECHANISMS OF ACUTE AND CHRONIC VENTRICULAR TACHYCARDIA in animals and man, the pathways of conduction and/or sites of origin of these rhythms have not been adequately defined. In order for surgical approaches to the therapy of medically intractable ventricular tachycardias to be more consistently successful, the conduction pathways and sites of origin for each rhythm must be known. The poor results reported by some previous investigators in reliably abolishing ventricular tachycardias following simple aneurysmectomy, with or without revascularization procedures, may be related to a lack of consideration of these factors: mechanisms, origins and conduction pathways. Other operative interventions have been based on the identification or assumption of macro-re-entry as the mechanism and the use of intra-operative epicardial maps to identify or predict the origins and/or pathways of the tachycardias. These innovative attempts have met with occasionally noteworthy but not consistent success.

The present study was therefore undertaken to 1) determine the sites of origin and ventricular activation patterns of the late ventricular tachycardias that follow experimental coronary occlusions and 2) establish the limitations of epicardial mapping and the importance of endocardial mapping in accurately identifying or predicting the sites of origin of such tachycardias.

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Methods

Studies were performed on 28 healthy mongrel dogs weighing between 10 and 15 kg. The animals were anesthetized with intravenous sodium pentobarbital (30 mg/kg) and then ventilated with room air through a pharyngeotracheal tube using a volume-cycled positive pressure respirator. Body temperature was maintained with a thermal mattress. The heart was exposed through a left thoracotomy at the fifth intercostal space, the pericardium opened, and the left atrial appendage retracted. In 12 animals the left anterior descending coronary artery was occluded by the Harris two-stage procedure 3-7 mm from the border of the left atrial appendage. In 16 animals blunt dissection was used to expose the anterior septal coronary artery. This vessel originated, in the majority of animals, as the first branch of the left anterior descending or at the bifurcation of the left anterior descending and circumflex arteries. The anterior septal artery was then acutely ligated. Spontaneous ventricular fibrillation was not observed in any of our animals.

Twenty-four hours following the initial procedure, the animals were anesthetized with sodium pentobarbital (10 mg/kg) plus either diazepam (1 mg/kg) or morphine (2.5 mg/kg), each intravenously. Ventilation and body temperature were maintained as indicated above. For the electrophysiologic studies the chests were opened in the fifth left intercostal space for the left anterior descending experiments and in the right fifth intercostal space or through a median sternotomy for the anterior septal artery experiments.

The constraints of the mapping procedure made it necessary to establish a monofocal ventricular tachycardia or a stable pattern of unifocal ectopic ventricular depolarizations in the animals in this study. To allow the ventricular ectopic rhythms to establish themselves without competition from supraventricular beats, the sinus node was mechanically crushed and, in some animals, vagal stimulation was also applied. In three animals with left anterior descending occlusions, these techniques were unsuccessful and a sequence of 3-10 programmed supraventricular beats followed by a pause allowed stable ventricular complexes to be used for mapping.
Complete epicardial maps were obtained first as previously described. This technique involves the insertion of bipolar plunge electrodes into a noninfarcted portion of the free wall of either ventricle via a 23 gauge needle to serve as a reference electrode. The epicardial surface of the ventricles was mapped using a hand-held bipolar moving electrode with terminals located 2 mm apart. Individual electrograms were recorded from as many as 80 predetermined locations on the left and right ventricles and displayed on a Tektronics 565 storage oscilloscope. The intervals between the reference and moving electrograms were measured at the intrinsics to deflections by a custom designed interval counter. Isochronic maps were constructed from these data to illustrate the sequence and timing of epicardial activation. The reproducibility of the measurements at each location was within two msec.

Next endocardial mapping was accomplished by using bipolar plunge electrodes and/or catheter electrodes to define activation sequences within the His bundle and bundle branches and the peripheral Purkinje system, as well as within, at the margins of, and outside the area of myocardial infarction. Recordings from 5-15 endocardial sites in the early areas were obtained to locate precisely the earliest Purkinje fiber activation. Surface landmarks and palpation through the atrial and ventricular free walls were used to position the electrodes properly — their exact position was verified by postmortem examination. In addition, intramural electrograms were obtained using Scher-type electrodes from multiple sites in 15 dogs. The interval between the earliest recorded Purkinje fiber activation and the earliest site of epicardial activation was determined by subtracting the interval between these locations and the reference electrogram. Pacing through the electrodes located at the sites of earliest activation was used in all experiments to verify that the earliest area was the site of origin of the tachycardia.

Following the electrophysiologic studies the animals were sacrificed and their hearts removed with the plunge electrodes left in place. The relationship of the electrodes to the bundle branch-Purkinje system was delineated by staining with a 2% tincture of iodine solution. The relationships of the electrodes to infarcted muscle were verified visually and histologically and by staining 2-4 mm thick slices of the septum with nitro-blue tetrazoleum.

**Results**

All animals developed myocardial infarctions documented by electrocardiographic and histochemical criteria. Broad low amplitude potentials were accepted as defining electrically dead tissue and corresponded to the areas of proven necrosis.

Complete electrophysiologic studies were performed on 13 tachycardias in 12 animals produced by left anterior descending occlusion and 13 tachycardias in 10 animals produced by anteroseptal artery occlusion. Two separate tachycardias were seen and mapped at different times in the same animal for one of the left anterior descending experiments and for three of the anteroseptal artery experiments. Two of the remaining six animals were utilized as sham-operated controls. In another animal the left anterior descending artery was inadvertently occluded along with the anteroseptal artery and a fourth animal was excluded because of heart worms. Technical problems precluded complete electrophysiologic studies in the two remaining dogs.

The earliest electrical activity for each of the 26 rhythms was recorded from surviving subendocardial Purkinje fibers either within the infarct for the left anterior descending occlusions or at its margins for the anteroseptal artery occlusions. The sites of origin were confirmed by pacing through electrodes located at the sites of earliest recordable activity and demonstrating unchanged endocardial and epicardial activation sequences. Using both multiple recording and pacing techniques, we were able to locate the earliest activation sites. The limit of resolution of these methods was 5-7 mm. Intramural multiple electrode recordings obtained from as many as 50 sites within the right and left ventricular free walls and interventricular septal myocardium using 5-15 separate electrodes excluded the possibility that any muscle activation preceded the earliest subendocardial sites. In addition, none of the arrhythmias arose from the His bundle or bundle branches despite the fact that in anteroseptal artery occlusions these tissues course directly through the necrotic septum.

Figure 1 examines the relationships between epicardial and endocardial activation during spontaneous ventricular tachycardia for a typical anteroseptal artery experiment. The epicardial activation map identifies the point of earliest epicardial breakthrough near the left ventricular apex. Activation spreads to the posterior surface of the left ventricle with right ventricular activation slightly delayed in comparison. As the schematic in the top right panel illustrates, this tachycardia originated in the mid-inferior portion of the left side of the septum with earliest left ventricular epicardial breakthrough occurring just 7 msec later. Without complete endocardial mapping and pacing, this early epicardial breakthrough site may have been erroneously considered to account for the initial forces of the QRS, to be the earliest site, and to be the source of the tachycardia.

The epicardial activation map in the left oblique view shows a second area of very early left ventricular activation occurring on the anterior surface along the interventricular septum. The appearance of multiple early epicardial breakthrough sites was at times the only indication that even earlier areas were yet to be located in the subendocardium. Such secondary sites may indicate that local conduction within the Purkinje system, emanating from the subendocardial sites of origin, may contribute to the activation patterns within that ventricle.

Figure 2 examines the relationship between epicardial and endocardial activation during spontaneous ventricular tachycardia for a typical left anterior descending experiment. Epicardial activation begins inferiorly along the left border of the infarct and spreads initially in a posterolateral direction. The left bundle is activated only 13 msec following the Purkinje fiber that accounts for the site of origin of the tachycardia in the subendocardium of the left ventricular free wall. When added to standard epicardial mapping, His bundle and bundle branch recordings may well have been considered sufficient to identify this rhythm as a fascicular tachycardia.

Figure 3 presents the relationships between the septal origin and the sites of epicardial breakthrough for all 13 tachycardias associated with anteroseptal artery occlusion.
The eight tachycardias that originated on the right side of the septum broke through to the epicardium on the right ventricle along the interventricular septum. The five tachycardias that originated on the left side of the septum broke through to the surface on the left ventricle and tended to emerge along the interventricular septum as well.

When viewed in this summary fashion some of the limitations of epicardial mapping in identifying or predicting the
sites of origin of the tachycardias become apparent. Epicardial activation for rhythms 1, 2, and 3 begins anteriorly on the right ventricle along the interventricular septum and directly overlies the sites of origin for these tachycardias; yet, for rhythms 10, 11, and 12, despite anterior epicardial breakthrough sites on the left ventricle, the sites of origin are 5.5 to 6 cm away on the far posterior aspect of the left side of the septum. Although the left septal sites of origin for tachycardias 11 and 13 are within a few millimeters of each other, they are more than 6 cm apart where they break through onto the epicardial surface. Furthermore, the epicardial appearance of one is anterior and of the other posterior.

Figure 4 examines similar relationships for the 13 tachycardias associated with left anterior descending occlusions. Tachycardias 5 and 6 which break onto the epicardium at virtually identical points are 6 cm distant from each other in their subendocardial origin. While epicardial breakthrough for rhythm 5 directly overlies the site of origin of the tachycardia, epicardial breakthrough for rhythm 6 clearly does not. Tachycardias 2 and 7, on the other hand, which are within one centimeter of each other in their origin, are 5 cm apart at their site of epicardial breakthrough.

**Discussion**

This study reports the results of extensive endocardial, epicardial, and intramural mapping studies in 28 animals with ventricular tachycardias 24–36 hours following experimental occlusion of the left anterior descending and anterior septal coronary arteries. These animals had physiologic studies to determine the sites of origin of the tachycardias and to assess the effectiveness of epicardial mapping in identifying or predicting these sites. The earliest electrical activity for each of 26 tachycardias was recorded from surviving subendocardial Purkinje fibers either within the infarct for the left anterior descending occlusions or at its margins for the anteroseptal artery occlusions. The sites of origin were confirmed by pacing through the electrodes located at the sites of earliest activation and those demonstrating unchanged activation sequences in both endocardial and epicardial recordings.

In the present experiments, the earliest epicardial breakthrough sites may appear so early in the overall activation sequence that they might easily be assumed to account for the initial forces of the QRS and to identify the origins of the tachycardias. This is particularly true for rhythms that arise in the septum near the left ventricular apex where anatomic proximity may allow rapid conduction to the left ventricular epicardial surface — as little as 7 msec following the true origin of the tachycardia in the subendocardium. The addition of standard His bundle-bundle branch recordings to epicardial mapping may be misleading as well. His or bundle branch electrograms may precede all epicardial activation and may appear to identify a rhythm as a fascicular tachycardia — without extensive endocardial mapping, subendocardial Purkinje fiber activity inside the infarct, occurring just 10–15 msec earlier, goes unrecognized.

The correlation between the earliest epicardial breakthrough sites and the sites of subendocardial origin for these tachycardias is extremely poor; that is, two rhythms may be quite close in their appearance on the epicardium yet
distant on the endocardium or vice versa. Epicardial breakthrough may directly overlie the endocardial site of origin for one rhythm and be 4–6 cm away for another rhythm, the epicardial appearance of which is virtually identical. A rhythm may arise in the posterior portion of the septum and break through onto the epicardium along the anterior aspect of the interventricular septum.

In this study we have used the ventricular tachycardias that follow experimental infarction as a model for these activation studies. This model was chosen because it was stable and reproducible. Although several previous studies indicate that the mechanism of these arrhythmias is probably enhanced automaticity,6,14 an automatic focus and a micro re-entrant pathway are, for the purposes of this study, identical in that each is a local, well-circumscribed process. Recent studies have suggested that enhanced automaticity and localized micro re-entry are the most common mechanisms of clinically significant sustained ventricular tachycardia.18 Therefore, this model appears to be a relevant one in which to study activation sequences of ventricular tachycardias.

Previous studies by El-Sherif, Wells and others1–6 have directed themselves to the analysis of the mechanisms of acute and chronic ventricular tachycardia. The sites of origin and pathways of conduction of these rhythms have not, however, been adequately defined. Although surgical approaches to chronic, medically intractable ventricular tachycardias are technically feasible, current surgical therapy has not met with consistent success. Some operative interventions have involved aneurysmectomy and/or revascularization7,8 without operative mapping, while others have relied on epicardial maps to identify or predict the sites of origin of the rhythms. Fontaine11 and Spurrell9,10 have based operative interventions on ventricular activation sequences obtained by epicardial mapping. In no patient, however, were endocardial or intramural electrograms recorded to define further the sites of origin or to determine overall activation sequence. In addition, detailed mapping of the conduction pathways was not performed. In contradistinction to the above reports, in animals and in three patients studied by Wittig and Boineau,17 the sites of origin of both re-entrant and automatic ventricular tachycardias were determined successfully using endocardial and transmural recordings in addition to standard epicardial maps. The authors emphasized the necessity for complete mapping by showing that epicardial breakthrough may not overlie the earliest premature activity on the endocardium, which may be a focus some centimeters away in subendocardial scar. They also demonstrated in one case that aneurysmectomy without mapping was inappropriate because the site of ventricular irritability located by endocardial mapping was some distance from the aneurysm to be excised.

Our findings suggest that extensive endocardial mapping should be performed in addition to epicardial and transmural recordings in order to identify the sites of origin and activation patterns of ventricular tachycardias. This model has particular applicability for rhythms of localized origin — whether automatic or micro-re-entrant — in which endocardial mapping may provide a more rational basis for operative interventions designed to terminate these arrhythmias.

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S R Spielman, E L Michelson, L N Horowitz, J F Spear and E N Moore

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