
SUMMARY Endocardial ventricular mapping of 21 ventricular tachycardias (VT) in 17 patients was performed using electrode catheters. Activation at multiple left and right ventricular sites was utilized to determine the site of origin of the VT. Eleven VT had a left bundle branch block pattern (VT-LBBB) and 10 VT had right bundle branch block pattern (VT-RBBB). In all VT-RBBB the earliest site of activation was in the LV or septum. In VT-LBBB the earliest site was RV (4/11), LV (5/11) and septum (2/11). All ventricular tachycardias with QRS < 140 msec arose in the septum. In patients with an aneurysm, the site of origin of ventricular tachycardia was always in the aneurysm. All VT-LBBB arising from the left ventricle originated in an aneurysm involving the septum. QRS changes during ventricular tachycardia were associated with alterations in the pattern of ventricular activation without alteration of the site of origin. In three patients the site of origin predicted by endocardial ventricular mapping was confirmed intraoperatively by epicardial or endocardial mapping.

We conclude that endocardial ventricular mapping demonstrates the limitations of the surface electrocardiogram in localizing the site of origin of ventricular tachycardia. The method may provide important data upon which the surgical therapy of ventricular tachycardia is based.

THE SURGICAL APPROACH TO THE THERAPY of medically resistant ventricular tachycardia has continued to evolve over the past ten years. However, the success of ventricular aneurysmectomy and/or coronary artery bypass grafting in terminating this arrhythmia varies, and in many instances may be accompanied by a high surgical mortality.1-4 Recently, intraoperative epicardial mapping has been used as a surgical guide to localize more accurately the site of origin of the arrhythmia.5-7 In spite of this technique these surgical interventions for ventricular tachycardia are still not universally successful. The present report concerns the development of a new technique, ventricular endocardial mapping, which provides useful data that may improve the efficiency of these surgical interventions.

Methods and Materials

Seventeen patients with sustained recurrent ventricular tachycardia underwent ventricular endocardial mapping as a part of the electrophysiologic evaluation of their arrhythmia. The clinical data for these patients are listed in table 1. Eight patients had ischemic heart disease and seven of these patients had a clinical angiographically documented ventricular aneurysm. Twenty-one morphologically distinct ventricular tachycardias were studied in the 17 patients. Four patients demonstrated two ventricular tachycardia morphologies. Ten ventricular tachycardias showed a right bundle branch block morphology (R, qR, rsR’, Rs in V1) and 11 demonstrated a left bundle branch block pattern (QS or Rs in V1). In each patient three to six electrode catheters were percutaneously introduced and positioned at the following endocardial sites: 1) high right atrium; 2) atrioventricular (A-V) junction at the point where the His bundle was recorded; 3) right ventricular apex; 4) coronary sinus; 5) right ventricular outflow tract; and 6) left ventricle. The left ventricular catheter was positioned using the retrograde arterial approach. The A-V junction and coronary sinus catheters, when used, remained fixed so that right ventricular activation at the atrioventricular junction and left ventricular activation along the atrioventricular groove were continuously recorded. In addition, the catheter in the right ventricular apex was left in a stable position whenever possible. One or more ventricular catheters were used as exploring electrodes. The mapping sites included the fixed sites and the midseptum, anterior wall, outflow tract, lateral inflow tract...
Table 1. Endocardial Mapping

<table>
<thead>
<tr>
<th>Pt/Age/Sex</th>
<th>Cardiac Dx</th>
<th>ECG</th>
<th>Morphology of VT mapped</th>
<th>VT-QRS width</th>
<th>Site of origin of VT</th>
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<td>RBBB</td>
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<td>Septum</td>
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<td>ASMI, LAH</td>
<td>LBBB</td>
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<td>LV</td>
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<td>ASMI, IMI</td>
<td>RBBB</td>
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<tr>
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<td>IMI, ASMI</td>
<td>LBBB</td>
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<td>LV</td>
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<td>LV</td>
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<td>RBBB</td>
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Abbreviations: ASMI = antero-septal myocardial infarction; ASHD = atherosclerotic heart disease; AVB = atrioventricular block; CAA = coronary artery anomaly; F = female; ICRBBB = incomplete right bundle branch block; IMI = inferior myocardial infarction; LAH = left anterior hemiblock; LBBB = left bundle branch block; LV-An = left ventricular aneurysm; LV = left ventricular myocardial infarction; NTMI = nontransmural myocardial infarction; RBBB = right bundle branch block; RHD = rheumatic heart disease; RV = right ventricle; WNL = within normal limits.

of the right ventricle; and three sites (high, middle and low) along the septum, apex, anterior and lateral walls, and inferobasal area of the left ventricle. These are schematically represented in figure 1. Whenever possible recordings from all sites were obtained. When an aneurysm was present, mapping the endocardial surface of the aneurysm was attempted. Positions were verified by fluoroscopy in multiple planes. A typical analog record is shown in figure 2. The site of origin was determined by location of the earliest recorded ventricular electrogram. A septal site of origin was suggested by recording right and left ventricular electrograms almost simultaneously, implying the site of origin was equidistant from the catheters, i.e., in the septum. Standard #6 French electrode catheters with a 1 cm interelectrode distance were used. Intracardiac electrograms were displayed simultaneously with 2 or 3 surface ECG leads on a switch-beam oscilloscope at filter frequencies of 40 to 500 Hz. Care was taken to assure electrical isolation of the patient and recording equipment.

All data were recorded on magnetic tape and subsequently retrieved on photographic paper at speeds of 150 to 400 mm/sec. The time measurement was taken at the point at which the largest rapid deflection crossed the baseline, using the mean value of six consecutive beats. We used this method because the intrinsic deflection of the electrogram (rapid plus to minus deflection) signals the time of depolarization of the tissues in the immediate vicinity of the electrode. Two references were always utilized; one being the earliest onset of the QRS in any of the recorded ECG leads, and the other being a ventricular electrogram recorded from a fixed right ventricular site (usually the A-V junction or the right ventricular apex). An intracardiac reference was always used because 1) it may be impossible to localize the beginning of the QRS in the recorded leads precisely, and 2) the earliest time of intracardiac activation may either precede QRS or occur during an isoelectric period of the QRS. The variation of activation times during any ventricular tachycardia with constant rate and QRS morphology varied less than ± 2 msec over the time period of measurement (fig. 2). In three patients intraoperative epicardial and in one endocardial activation sequences were obtained using standard techniques.3, 6, 9

Results

All patients underwent mapping procedures without embolic, hemorrhagic, or thrombotic complications. The entire endocardial map of the ventricular tachycardia required 5 to 15 min during the electrophysiologic study. At least four right ventricular and three left ventricular sites were obtained. Detailed exploration of the ventricular aneurysm was accomplished in five patients (cases 3, 4, 7, 12, 13).

In all patients with a ventricular tachycardia displaying a right bundle branch block morphology, the earliest site of activation was in the left ventricle (fig. 3), or septum (fig. 4). Of the tachycardias with left bundle branch block morphology, four had an early right ventricular site (fig. 2), and five cases had an early left ventricular site (fig. 5), and two originated in the septum. Of five patients with the left bundle branch block type of ventricular tachycardia originating in the left ventricle, analysis of the QRS during sinus rhythm and during the tachycardia did not provide any clues to the site of origin. In the three patients with ventricular tachycardias manifesting either right or left bundle branch block morphology in whom the QRS was less than 140 msec, activation data suggested that these tachycardias originated within the interventricular septum because the electrograms recorded from both the right and left sides of the septum and the atrio-ventricular junction occurred early and almost simultaneously (figs. 6 and 7).

In the patients with left ventricular aneurysms the earliest site of endocardial activation of the ventricular tachycardia was always within the aneurysm. In one patient electrical activation within segments of the aneurysm occurred prior to
Figure 1. Schematic view of the heart in serial sections indicating the endocardial mapping sites. The inset shows the level of transection for each section. The sites are 1) right ventricular (RV) apex; 2) RV midseptum; 3) RV free anterior wall; 4) A-V junction; 5) RV inflow tract; 6) RV outflow tract; 7) left ventricular (LV) apex; 8) LV low septum; 9) LV midseptum; 10) LV anterior free wall; 11) LV inferoposterior wall; 12) LV high septum (under aortic valve); 13) LV lateral wall (under mitral valve), and 14) posterobasilar LV (recorded from coronary sinus). Ao = aorta; MV = mitral valve; RVOT = right ventricular outflow tract; RA = right atrium; LA = left atrium.

Figure 2. Typical endocardial map during ventricular tachycardia with left bundle branch block morphology (case 15). The records are organized from top to bottom: ECG leads 2, V1, and electrograms from the right ventricular outflow tract (RVOT), atrioventricular junction (AVJ), right ventricular septum at the apex (RVSA), right ventricular anterior wall (RVAW) and time lines (T). Note the stability of the electrograms. The earliest site was the RVOT which occurred 8 msec after the onset of the QRS. The earliest left ventricular site was recorded at the high septum 32 msec after the onset of the QRS.

Figure 3. Earliest endocardial site of activation at left ventricular apex in a ventricular tachycardia with right bundle branch block morphology and wide QRS (case 10). ECG leads 1, aVF, and V1 and electrograms at the right ventricular apex (RVA) and left ventricular apex (LVA). Of all sites mapped the LVA was the earliest site of activation, occurring 30 msec prior to the inscription of the QRS.
the inscription of the QRS while other segments showed activity occurring at various times throughout the cardiac cycle (fig. 8).

In three patients intraoperative epicardial (3 patients) and endocardial (1 patient) activation mapping confirmed the site of origin of the ventricular tachycardia within the aneurysm, in the area predicted by catheter mapping techniques.

Endocardial Activation with Changing QRS Morphology

Variations in morphology of the ventricular tachycardia were frequently observed. Abrupt morphologic changes were accompanied by obvious changes in the ventricular endocardial activation sequence (fig. 9). There was usually a transient (1 beat) change in cycle length at the time of the QRS morphology change, but the subsequent tachycardia had the same rate as the initial tachycardia despite its different ventricular endocardial activation sequence (fig. 9).

These observations were most apparent in those ventricular tachycardias originating within ventricular aneurysms. In these cases, despite varying degrees of change in QRS morphology, in each instance the earliest site of activation remained within the aneurysm (fig. 10). Moreover, subtle changes in morphology were frequently accompanied by marked changes in endocardial activation sequence (fig. 10).
Figure 7. Septal origin of ventricular tachycardia with narrow QRS and left bundle branch block morphology (case 16). The panel is organized as in figure 5 but a high right atrial electrogram (HRA) is also recorded. Note that ventricular electrograms (V) recorded from the right (HBE, RVA) and left ventricles (CS, LVA) occur almost simultaneously, suggesting a septal site of origin.

Figure 8. Mapping from a left ventricular aneurysm (case 12). The records are organized as in figure 4 but the LV recording is from an aneurysm. Note that electrical activity is present during systole and diastole with two sharp deflections during systole and diastole seen 100 and 20 msec prior to the onset of the QRS.

Figure 9. Abrupt change in QRS morphology and endocardial activation sequence of ventricular tachycardia (case 12). The panel is organized similarly to figure 8 but the LV catheter has been moved slightly. The tachycardia as recorded in the LV aneurysm (LV-An) is unaltered by the change in QRS noted by the arrow. However, the morphologic change in QRS is associated with a one cycle delay (370 to 405 msec) in the electrogram recorded at the right ventricular apex along the septum (RVSA), and subsequent change in ventricular activation recorded in the CS, AVJ, and RVSA (vertical lines). This suggests that a change in exit site has occurred without disturbance of the underlying tachycardia.
Furthermore, exit block from within the aneurysm was demonstrated in two cases. In one patient the appearance of left bundle branch block type of ventricular tachycardia was associated with exit block out of the left ventricular aneurysm (fig. 11).

Fusion complexes were analyzed in several patients and were of two types: those due to partial supraventricular capture (fig. 12), and those due to spontaneous premature ventricular depolarization with the site other than the site of origin of ventricular tachycardia (fig. 13).

![Diagram](https://i.imgur.com/350.jpg)  
**Figure 10.** Subtle change in QRS morphology during ventricular tachycardia associated with marked change in ventricular activation (case 12). The tracing is organized as in figure 8. The two QRS morphologies of the ventricular tachycardia shown in the panels are similar but the ventricular activation sequences (vertical lines) are grossly different. Note that the rate of the tachycardia remains identical.

![Diagram](https://i.imgur.com/350.jpg)  
**Figure 11.** Two-to-one exit block during ventricular tachycardia (case 12). The tracing is organized from top to bottom: ECG leads 2 and V1; intracardiac electrograms from the A-V junction (AVJ), right ventricular apex (RVA), and lateral aspect of an apical left ventricular aneurysm (LV-An); and time lines (T). In the left panel ventricular tachycardia with right bundle branch block morphology, previously shown to originate within the aneurysm near the apex (fig. 8), is present. On the right 2-to-1 exit block is present at the LV-An which is associated with a change in ventricular tachycardia rate (350 to 305 msec) and QRS morphology to a left bundle branch block pattern. These findings suggest that during 2-to-1 LV-An exit block the re-entrant pathway was shorter (hence the decreased cycle length) and the exit site shifted to produce the different QRS.
Discussion

The present report represents the first demonstration of the technique of catheter endocardial mapping in the analysis of ventricular tachycardia in man. Endocardial mapping has been extensively employed in the analysis of supraventricular tachycardias, intra- and interatrial conduction in normals and subjects with electrocardiographic left atrial enlargement, and patterns of ventriculoatrial conduction in various conditions. Our technique is an adaptation of those previously employed in the analysis of experimental ventricular tachycardia in the open chest canine model. The validity of our mapping technique depends upon the stability, reproducibility, and verification of catheter placement. In each case the beat-to-beat variation in activation times at each mapping site was ±2 msec, thus, demonstrating the stability of our recordings. Our data were reproducible in that catheters could be returned to previously recorded sites with no significant change in activation times. Verification of catheter sites was made using fluoroscopy in multiple planes.

The recording sites in both right and left ventricles were arbitrarily chosen to assess major subdivisions of the ventricular endocardial surface with special attention to the interventricular septum. The number of sites utilized varied from patient to patient for technical reasons, but at least one recording site was obtained in each of the major segments of the ventricular endocardial surface, e.g., septum, apex, and free wall. Utilizing from 7 to 14 recording sites, we were able to localize the origin of the ventricular tachycardia to an area approximately 10 to 12 cm² within the ventricles or specifically within an aneurysm when present. It is impossible to localize more precisely the site of origin due to the inherent limitations of catheter positioning capability. Increased precision may have been achieved by mapping additional sites within the ventricle; however, the previously discussed limitations make mapping more than 15 reproducible and identifiable sites an unrealistic objective. Therefore, only sites which could be reproducibly obtained were used. Despite the limitations imposed by the technique, in three patients the actual site of origin of the tachycardia determined by detailed intraoperative activation mapping was located within 2 to 3 cm of the site predicted by catheter endocardial mapping.

Relationship of the Site of Ventricular Tachycardia to the QRS Morphology

It has been stated that ventricular tachycardias originating in the left ventricle have a right bundle branch block configuration and those from the right ventricle have a left bundle branch block configuration. This contention has not been verified and our data suggest that this generalization is frequently incorrect.

While all tachycardias with right bundle branch block morphology originated within the left ventricle or septum, seven of eleven ventricular tachycardias with left bundle branch block morphology also had their endocardial site of origin in the left ventricle or septum. Of particular note was that in each patient with a left ventricular aneurysm and a ventricular tachycardia with a left bundle branch configuration, the site of origin was always in the aneurysm, which in
variably involved the septum. Possible explanations for a left ventricular site of origin of ventricular tachycardia with left bundle branch block configuration are 1) preferential left-to-right transseptal activation; 2) exit block from the re-entrant site within the aneurysm to the left ventricular free wall; and 3) underlying disease of the left ventricular muscle and/or conduction system which alter conduction patterns and make it impossible to predict the QRS morphology on the basis on the site of impulse origin. In most cases all three mechanisms were probably operative. The apparent paradoxical observations that the left ventricular aneurysmectomy have resulted in cure of ventricular tachycardias with left bundle branch block configuration are best explained by these mechanisms.4, 19-21 We suspect that the endocardial activation sequence been evaluated in these cases, the left ventricular aneurysm would have been demonstrated to be the source of the arrhythmia.

Recent data in experimental ventricular tachycardia produced by left anterior descending or septal artery occlusion in dogs have shown that the QRS morphology on the surface ECG reflects epicardial activation which may not be the reliable indication of the site of origin of the ventricular tachycardia.3, 22 In the case of septal origin of the ventricular tachycardia the endocardial site of origin has been found to be as much as 6 cm away from the earliest site of endocardial breakthrough.23 Thus, intraoperative epicardial mapping alone may not accurately localize the site of origin of ventricular tachycardias. This was previously suggested in a case report by Gallagher et al.4 which revealed the earliest site of epicardial activation occurred 25 msec after the QRS; thereby implying an endocardial site of origin.

Significance of Endocardial Mapping

Successful surgical ablation of an arrhythmia is dependent upon the correction of factors responsible for the arrhythmia. The technique of intracardiac stimulation and recording to define arrhythmia mechanism and site of origin can play an important role in the planning and execution of medical and surgical therapy. In patients refractory to pharmacologic and/or pacemaker therapy the consideration of surgical intervention may be influenced by the site of origin of the ventricular tachycardia as determined by endocardial mapping. If aneurysmectomy is contemplated solely for arrhythmia control, the ventricular aneurysm should be demonstrated to be site of origin of the arrhythmia prior to surgery. If coronary artery bypass grafting is considered as a therapeutic modality for arrhythmia treatment, the area supplied by the proposed graft should be demonstrated to be the site of origin of the tachycardia. While extremely precise localization with 1 cm² of the ventricular tachycardia is not possible with current in vivo mapping techniques, it is clearly possible to state whether an aneurysm or some other anatomic structure is responsible for the arrhythmia. The ability to localize ventricular tachycardias in this manner provides information upon which the feasibility of a surgical intervention can be assessed. Furthermore, endocardial mapping can identify particularly difficult surgical candidates preoperatively. The demonstration of septal origin of a ventricular tachycardia would make surgical excision extremely difficult, although not impossible.8

Refinement of preoperative and intraoperative endocardial mapping, coupled with intraoperative epicardial mapping, should provide the information to enable the most effective surgical approach to the therapy of intractable ventricular arrhythmias.

References

Recurrent sustained ventricular tachycardia. 2. Endocardial mapping.
M E Josephson, L N Horowitz, A Farshidi, J F Spear, J A Kastor and E N Moore

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