Comparison of Endocardial Catheter Mapping with Intraoperative Mapping of Ventricular Tachycardia

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SUMMARY To validate the accuracy of catheter endocardial mapping to localize the origin of ventricular tachycardia (VT), we compared catheter endocardial mapping with intraoperative epicardial and endocardial mapping of 24 morphologically distinct VTs in 18 patients undergoing surgery. Twelve had VT with left bundle branch block morphology and 12 had VT with right bundle branch block morphology. Catheter endocardial mapping localized 23 VT morphologies to the border of a left ventricular aneurysm or myocardial infarction and one VT to a right ventriculotomy scar. Intraoperative epicardial mapping showed epicardial breakthrough on the right ventricle in 10 VTs with left bundle branch block morphology and on the left ventricle in two. In 12 VTs with right bundle branch block morphology, intraoperative epicardial mapping showed epicardial breakthrough at the border of a left ventricular aneurysm. Intraoperative endocardial mapping revealed the earliest site of VT with left bundle branch block morphology (11 patients) and VT with right bundle branch block morphology (12 patients) at the border of a left ventricular aneurysm, and one VT with left bundle branch block morphology in the right ventricle. Catheter endocardial mapping predicted the origin of VT within 4–8 cm² of that determined by intraoperative endocardial mapping, which always identified the earliest site. These data validate the accuracy of catheter endocardial mapping in localizing the origin of VT.

WE HAVE RECENTLY DEVELOPED the technique of mapping ventricular tachycardia (VT) using an endocardial catheter. This technique has allowed us to analyze the sites of origin of 51 morphologically distinct VTs in 32 patients, including 24 with ventricular aneurysms. We used mapping to localize the origin of the tachycardias to aneurysms or regions of the heart considered to be ischemic, providing a rational basis for either aneurysmectomy or coronary artery bypass grafting for the management of drug-resistant VT. The success of catheter mapping in this endeavor, however, depends on the accuracy of the catheter recordings. We compared the origin of VT predicted by catheter endocardial mapping with the results of intraoperative epicardial and endocardial mapping in 18 patients. This report details our experience, which validates the ability of catheter recordings to locate the origin of VT, and further stresses the limitations of the surface ECG and epicardial mapping for this purpose.

Materials and Methods

Eighteen patients in whom preoperative and intraoperative mapping of VT was performed form the basis of this report (table 1). There were 16 men and two women, ages 23–68 years. Seventeen had ischemic heart disease, all with prior infarction, and one (patient 17) had undergone repair of tetralogy of Fallot 15 years previously. In all patients, VT was refractory to medical therapy. In each instance, the tachycardia could be reproducibly initiated and in 15 patients could be reproducibly terminated by programmed stimulation, which included rapid ventricular pacing and the introduction of one or more ventricular extrastimuli, as previously described. In the other three patients, hemodynamic deterioration or acceleration of VT necessitated electric cardioversion. Six patients had two VT morphologies (e.g., right bundle branch block and left bundle branch block patterns) which were mapped by catheter and intraoperatively. Thus, 24 VT morphologies were mapped preoperatively and intraoperatively.

Each patient underwent preoperative catheter endocardial mapping using a modification of the procedure we recently described. In each patient, two or three standard electrocardiographic leads were simultaneously displayed with recordings from the right ventricular apex and the atroventricular junction (His bundle position) and in 15 patients from the coronary sinus to record posterobasal left ventricular activity. An additional right ventricular catheter was used to map the right ventricular outflow tract, anterior right ventricular wall and right ventricular inflow tract. The left ventricle was entered via the retrograde arterial approach. Mapping was accomplished using #6 French bi- or quadripolar USCI catheters with a 1-cm interelectrode distance or a specially designed hexapolar catheter (Webster Labs) with electrodes 1 mm apart that allowed recording from the second and fifth electrode pair (8 mm) and stimulation from the first and sixth electrode (11 mm),
bipolar electrograms were recorded at 54 preselected epicardial sites on both ventricles using either a ring or a hand-held probe electrode, which has interelectrode distances of 1–2 mm (fig. 1). Particular attention was directed toward the margins of the aneurysm or infarction, where more detailed mapping was performed. Epicardial mapping was performed during partial or complete bypass, depending upon mean central aortic pressure. If hypotension developed, full cardiopulmonary bypass was used to maintain a mean central aortic pressure of 80 mm Hg.

After the completion of epicardial mapping, during full cardiopulmonary bypass, the infarction, aneurysm or, in one case, ventriculotomy scar, was opened at its center and resection was performed. After resection, the tachycardia was reinduced if it had not persisted and endocardial mapping using a ring or hand-held probe electrode was accomplished under direct vision. Epicardial electrograms were recorded at 25–55 preselected endocardial sites in the left ventricle (in the right ventricle in patient 17) in a radial pattern around the margin of the excised area in successive rows 1 cm apart. In patients in whom the aneurysm involved the interventricular septum, electrograms were recorded from the left side of the septum.

The reference and mapping electrograms were filtered at 40–500 Hz and were simultaneously displayed with three electrocardiographic leads (1, 2 or aV_F and either V_6, V_5 or V_6). Data were recorded directly on an eight-channel, ink-jet recorder (Elema Mingograf) at paper speeds of 200 mm/sec and simultaneously recorded on eight-channel magnetic tape (Honeywell 5600C). Data were later retrieved on photographic paper at speeds of 100–200 mm/sec for illustrative purposes. Epicardial and endocardial activation times were taken as a point at which the rapid deflection crossed the baseline in three consecutive complexes of the same morphology. Measurements were made between the mapping electrogram and both right and left ventricular reference electrograms. Subsequently, the data were standardized to indicate the time from the onset of the QRS complex by adding the interval between that point and the reference electrogram obtained when the heart’s position was undisturbed in the pericardial cradle.

Epicardial isochronic maps and endocardial activation times from the border of the resection were constructed and compared with catheter recordings obtained preoperatively.

Results

Preoperative endocardial catheter activation mapping of 24 morphologically distinct VTs in the 18 patients was compared with intraoperative epicardial and endocardial mapping. There were 12 tachycardias with right bundle branch block and 12 with left bundle branch block morphologies.

Preoperative Endocardial Mapping

The earliest site of electrical activity in all but one VT morphology was in the area of the left ventricular

<table>
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<tr>
<th>Patient</th>
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<th>Sex</th>
<th>Aneurysm</th>
</tr>
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<td>M</td>
<td>Apical*</td>
</tr>
<tr>
<td>2</td>
<td>50</td>
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</tr>
<tr>
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<td>M</td>
<td>Inferior*†</td>
</tr>
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<td>4</td>
<td>59</td>
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<td>Apical</td>
</tr>
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<td>M</td>
<td>Apical*</td>
</tr>
<tr>
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<td>68</td>
<td>F</td>
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</tr>
<tr>
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<td>M</td>
<td>Apical*</td>
</tr>
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<td>9</td>
<td>61</td>
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<td>M</td>
<td>Inferoposterolateral</td>
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<td>—</td>
</tr>
<tr>
<td>18</td>
<td>58</td>
<td>M</td>
<td>Apical*</td>
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</tbody>
</table>

*Involved the interventricular septum.
†No discrete aneurysm; inferior akinetic scar.

thereby allowing recording and pacing capability from essentially the same site.

Since our previous publication, the mapping sites in the left ventricle have been modified as shown in figure 1. In addition, exploration of an aneurysm or akinetic area was performed when present so that medial, lateral and central recording sites from that area were obtained. Electrograms were recorded at an average of nine sites in the left ventricle, which allowed for localization to segmental areas of the left ventricle of 10–15 cm². This number of sites was the maximum number from which reproducible recordings could be obtained. Verification of catheter position was made using multiple-view cinefluoroscopy. Activation times were measured at the point at which the first rapid deflection crossed the baseline and the mean value of three consecutive complexes of uniform morphology was calculated. Particular note was made of presystolic or holodiastolic activity, which we considered to represent recording of potential reentrant circuits, thus suggesting the area of origin.3, 4

Intraoperative mapping was performed after cannulation for cardiopulmonary bypass. Bipolar plunge electrodes (0.005-inch diameter teflon-coated stainless steel wire) were inserted in the right and left ventricles by means of a 23-gauge needle to record reference electrograms or for pacing (18 patients). In 10 patients, right ventricular stimulation and recording were performed using a quadripolar electrode catheter (1 cm interelectrode distance) positioned at the right ventricular apex before surgery. After VT was initiated by programmed stimulation,
Figure 1. A) Catheter endocardial mapping sites in the left ventricle are displayed on top. Site 1 = apex. Sites, 2, 3, 4 = apical, mid, and basal septum. Site 5 = inferior. Site 6 = inferoposterior. Site 7 = apical low lateral. Site 8 = inferoposterolateral. Site 9 = anterolateral. Site 10 = lateral basal. Site 11 = midanterior. Site 12 = superior basal. In the bottom panel, corresponding epicardial sites are displayed. B) Standard epicardial mapping sites in anterior (left), lateral (middle) and posterior (right) views.
aneurysm (23 morphologies). In the remaining patient, the earliest activity was recorded at the site of right ventriculotomy scar (patient 17; table 2). Holo- or nonholodiastolic activity was detected in 11 patients and in 17 morphologies. Eleven of the morphologically distinct VTs arose on the septal side of an apical aneurysm, including three patients with pleomorphic (right and left bundle branch block) patterns. Seven morphologically distinct tachycardias arose from the free wall, either anterior or anterolateral, aspects of left ventricular aneurysm, including two morphologies in one patient. Five morphologically distinct VTs in three patients arose from the border of an inferoposterior aneurysm, three from the posteromedial border at or near the septum and two from the posterolateral border. These five tachycardias included right and left bundle branch forms in two patients. In the third patient, who had an inferior scar without a discrete aneurysm, the tachycardia arose from the posterior septum. In the other patient (patient 17), the tachycardia arose from the right ventricular outflow tract at the ventriculotomy scar for repair of tetralogy of Fallot. In the six patients with pleomorphic VT, both

Table 2. Comparison of Preoperative and Intraoperative Mapping

<table>
<thead>
<tr>
<th>Pt</th>
<th>VT morphology</th>
<th>Endocardial catheter origin*</th>
<th>Epicardial origin†</th>
<th>Endocardial origin*</th>
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<td>1</td>
<td>LBBB</td>
<td>LV-An, septum (3)</td>
<td>LV-AIVG (28)</td>
<td>Anterior LV septum (2)</td>
</tr>
<tr>
<td></td>
<td>RBBB</td>
<td>LV-An, septum (2)</td>
<td>LV-PIVG (54)</td>
<td>Anterior LV septum (2)</td>
</tr>
<tr>
<td>2</td>
<td>LBBB</td>
<td>LV-An, septum (2)</td>
<td>RV-AIVG (23)</td>
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</tr>
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<td>Posterior LV septum (between 4 and 6)</td>
<td>RV-PIVG (1)</td>
<td>Posterior LV septum (4)</td>
</tr>
<tr>
<td>4</td>
<td>RBBB</td>
<td>LV-An, anterolateral (7)</td>
<td>Anterolateral edge of LV-An (38)</td>
<td>Anterolateral edge of LV-An (9)</td>
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<td>5</td>
<td>RBBB</td>
<td>LV-An, apex (between 1 and 7)</td>
<td>Anterolateral edge of LV-An (28)</td>
<td>Anterolateral edge of LV-An (7)</td>
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<td>6</td>
<td>RBBB</td>
<td>LV-An, septum near apex (between 1 and 2)</td>
<td>Anterior edge of LV-An (33)</td>
<td>Anterior septum near apex (2)</td>
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<td>LBBB</td>
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<td>RV-AIVG (25)</td>
<td>Anterior septum 1-2 cm from apex (2)</td>
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<tr>
<td>7</td>
<td>RBBB</td>
<td>LV-An, anterolateral (between 7 and 9)</td>
<td>Anterolateral edge of LV-An (between 37 and 38)</td>
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<td>LBBB</td>
<td>LV-An, midseptum (3)</td>
<td>RV-AIVG (24)</td>
<td>Mid LV septum (3)</td>
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<td>RBBB</td>
<td>LV-An, anterolateral (9)</td>
<td>Anterolateral edge of LV-An (between 33 and 38)</td>
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</tr>
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<td>10</td>
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<td>LV-An, inferoposterior near septum (between 4 and 6)</td>
<td>RV-PIVG (between 1 and 2)</td>
<td>Posteroemidal edge of LV-An near septum (between 4 and 6)</td>
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<tr>
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<td>RV-PIVG (1)</td>
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<td>16</td>
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<td>LV-AIVG (28)</td>
<td>Anterior LV septum (2)</td>
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<tr>
<td>LBBB</td>
<td>LV-An, septum near apex (between 1 and 2)</td>
<td>RV-PIVG (3)</td>
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<tr>
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<td>LBBB</td>
<td>RVOT</td>
<td>RVOT (18)</td>
<td>RVOT</td>
</tr>
<tr>
<td>18</td>
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<td>LV-AIVG (29)</td>
<td>Anterior mid LV septum (between 2 and 3)</td>
<td></td>
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</tbody>
</table>

*See figure 1A to key numbers.
†See figure 1B to key numbers.

Abbreviations: LV = left ventricle; LV-An = left ventricular aneurysm; LV-AIVG = left ventricular side of anterior interventricular groove; RV-AIVG = right ventricular side of anterior interventricular groove; RV-PIVG = right ventricular side of posterior interventricular groove; LV-PIVG = left ventricular side of posterior interventricular groove.
morphologies arose from the same area. Recording
the earliest site of both morphologies required either
no movement of the catheter or slight repositioning of
1-2 cm. In four patients, pacing at the earliest activa-
tion site produced the same morphologic
characteristics as the induced and spontaneous
tachycardia (fig. 2).

Intraoperative Mapping

In all 12 right bundle branch block morphologies,
the site of earliest epicardial breakthrough was either
on the free wall of the left ventricle or along the left
ventricular side of the anterior or posterior interven-
tricular groove (table 2). In 10 of the 12 left bundle
branch block morphologies, the earliest epicardial
breakthrough was along the right ventricular side of
the anterior or posterior interventricular groove. In
patients 1 and 18, the earliest epicardial break-
through appeared on the left side of the interven-
tricular groove anteriorly, and in patient 17, the
earliest epicardial breakthrough was at the right ven-
tricular outflow tract at the site of a ventriculotomy
scar. Intraoperative endocardial mapping revealed
disparate results from the epicardial maps (table 2). In
all cases, the earliest site of activation was recorded
during endocardial mapping and always preceded the
onset of the QRS. In fact, only in patient 14 did the
earliest epicardial site occur at the onset of the QRS;
endocardial mapping revealed an earlier site just in-
fierior to the early epicardial site at the border of a
thin-walled aneurysm (fig. 3). All morphologic forms
arose from the left ventricle, except in the patient
without coronary artery disease, in whom the tachycardia arose from the region of the ven-
triculotomy scar in the right ventricular outflow tract.
In the other patients, all VTs, including those with left
bundle branch block morphologies (11 patients), arose
at the border of the aneurysm or akinetic area in the
left ventricle. Excluding the patient with the right ven-
tricular outflow tract origin, there were 23 tachycar-
dias arising from the left ventricle. Eleven arose from
the septum anteriorly and three arose from or adja-
cent to the septum posteriorly. Seven came from the
anterolateral or anterior region of anterior or apical
aneurysms and three came from the posterior medial
or lateral aspects of an inferoposterior aneurysm.

Comparison of Intraoperative and Preoperative Mapping

In all patients, the catheter mapping localized the
origin of the tachycardia to the region eventually
found to have the earliest activation site, which was
always that obtained during intraoperative endocar-
dial mapping (table 2). The site of origin predicted by
catheter recording was within one site (fig. 1) of that
shown to be earliest by intraoperative endocardial
mapping (table 2). This held true in patients with right
dbundle branch block morphology (figs. 2 and 3), left
bundle branch block morphologies (figs. 4 and 5) and
pleomorphic VT (figs. 6 and 7), regardless of whether
the tachycardia arose posteriorly (figs. 8 and 9),
anteriorly (figs. 2 and 3) or from the septum (figs. 4
and 5). The correspondence of the two methods of en-
cardial mapping held true despite discrepancies of
intraoperative, epi- and endocardial mapping (table 2).

Discussion

Endocardial catheter mapping was developed in our
laboratory as a means of preoperatively localizing
the origin of VT to a region of the heart considered re-
sectable. The technique was used initially to localize
VT to ventricular aneurysms, thus providing a ration-
ale for aneurysctomy. In developing the tech-
nique, we obtained data that showed that multiple
morphologic forms of VT could arise from a left ven-

![Figure 2](http://circ.ahajournals.org/)

**Figure 2.** Endocardial catheter mapping
of ventricular tachycardia (VT) in patient
14. The panels are arranged from top to bottom
as ECG leads 1, 2 and V1 with electrog-
grams from the right ventricular apex
(RVA) and at site 7, the apical low lateral
aspect of the aneurysm (An). LVM = left
ventricular mapping catheter. Site 7 was the
earliest endocardial site during VT and just
preceded the onset of the QRS. The cycle
length of VT is 425 msec. In the bottom pan-
el, pacing from site 7 at a cycle length of 400
msec produces the same morphology and ac-
tivation sequence as VT.
Figure 3. Intraoperative mapping of ventricular tachycardia in patient 14. The anterior, lateral and posterior views of epicardial mapping sites are shown clockwise from the upper left with ECG leads 1, 2 and V5. The stippled area is the aneurysm. In the lower left are the data from the endocardial map after aneurysmectomy. Epicardial activation is displayed in 20-msec isochrones. The earliest epicardial breakthrough occurs at the onset of the QRS at site 38. The earliest endocardial site occurs approximately 1 cm inferior to this site (filled circle at edge of aneurysm). These sites are at site 7, which was the earliest site recorded by catheter (see fig. 2).

Figure 4. Endocardial mapping of ventricular tachycardia in patient 3. Ventricular tachycardia with left bundle branch block pattern is present at a cycle length of 340 msec. Leads 2 and V5 are displayed with a left ventricular (LV) site between 4 and 6, the coronary sinus (CS), atrioventricular junction (AVJ), right ventricular apex (RVA) and LV sites 4, 8, 3, 2 and 7. T = time lines. The earliest sites are along the posterobasal septum between sites 4 and 6. RVOT = right ventricular outflow tract.

Ventricular aneurysm and that pleomorphic VT need not be considered a contraindication of surgery. Several inherent limitations of catheter mapping techniques exist, including difficulty in obtaining accurate and reproducible catheter position and the inability to precisely localize ventricular tachycardia to regions smaller than a few cm². Validation of the ability of endocardial catheter mapping to localize the origin of VT was therefore necessary. Validation required comparing the origin of VT determined by the catheter technique with that determined by intraoperative epicardial and endocardial mapping techniques. We made this comparison in 24 morphologically distinct VTs in 18 patients. Although 12 patients had both right and left bundle branch block morphologies during the clinical electrophysiologic study, in only six patients could endo-
cardial and epicardial mapping during both forms be performed at operation.

In all instances, the catheter map localized VT to the region shown to be the earliest during intraoperative mapping. Specifically, the earliest site of activation was always within 4–8 cm² of the site predicted by catheter mapping. Epicardial mapping, the most commonly used intraoperative technique for localizing the origin of VT, incorrectly localized nine VTs with left bundle branch block morphology to the right ventricle. In each instance, the endocardial intraoperative map corresponded to the preoperative catheter map that showed a left ventricular origin at the border of an aneurysm.

The limitations of epicardial mapping as a method of localizing the origin of VT in experimental animals and man have been reported. Epicardial breakthrough followed the onset of QRS in all but one patient, suggesting either septal origin or endocar-dial/myocardial origin of the VT and, therefore, supporting the need for endocardial and perhaps intramural mapping of the tachycardia.

Abolition of the VT by surgical resection was proof that the site predicted by the catheter and intraoperative endocardial mapping was indeed the origin of the tachycardia. These data and reported data suggest that epicardial breakthrough and the subsequent pattern of ventricular activation deter-

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**Figure 5.** Intraoperative mapping of ventricular tachycardia in patient 3. The epicardial map is displayed as in figure 3 with ECG leads 1, 2 and V₅. The stippled area represents an infarct without aneurysm. The earliest epicardial breakthrough appears on the right ventricle 40 msec after the onset of QRS near the posterior interventricular groove at epicardial site 1. The endocardial map displays data obtained through a ventriculotomy in the infarct. The earliest endocardial site is along the basal septum and appeared 2 msec before the onset of QRS. This corresponded to the earliest site obtained by catheter mapping (see fig. 4).

**Figure 6.** Catheter endocardial mapping during right and left bundle branch block morphologies in patient 1. Lead V₁ is displayed with electrograms from the right ventricular apex (RVA), atrioventricular junction (AVJ), coronary sinus (CS) and site 2 at the apical septal segment of a left ventricular (LV) aneurysm. During both right (left panel) and left (right panel) bundle branch block morphologies the earliest endocardial site was site 2. Presystolic activity is clearly seen during right bundle branch block morphology. T = time lines.
Figure 7. Intraoperative mapping of right and left bundle branch block morphologies in case 1. Epicardial mapping in both panels is displayed as in figure 3 with ECG leads 1, 2, V5. The stippled area represents an apical aneurysm and the endocardial mapping data are shown from the cut edges of the aneurysm at the lower left of each panel. The data during ventricular tachycardia (VT) with right bundle branch block (RBBB) morphology are shown on top and that during left bundle branch block (LBBB) morphology on the bottom. Early epicardial breakthrough during VT-RBBB (upper panel) appears 24 msec after the onset of the QRS along the posterior interventricular groove at the apex at epicardial site 54. The endocardial site of origin was on the septum near the apex at endocardial mapping site 2. Data for VT-LBBB are displayed in the lower panel. Early epicardial breakthrough appears along the anterior interventricular groove at the border of the aneurysm 35 msec after the onset of the QRS, approximately 10 cm from the epicardial origin of VT-RBBB. The earliest endocardial site was along the septum at endocardial site 2. Thus, despite marked differences in epicardial breakthrough, both VT morphologies arose from the anterior septum as predicted by catheter mapping (see fig. 6).

mine the QRS morphology of the VT but fail to predict the site of origin reliably. Extensive endocardial and intramural recordings are necessary to localize the origin of the tachycardia accurately and assure successful surgical extirpation of the arrhythmia.

Role of Catheter Mapping

The clinical significance of endocardial catheter mapping relates to its ability to localize VT to areas not usually resected during routine aneurysmectomy. The ability to perform intraoperative mapping in no way detracts from the value of endocardial catheter mapping. We believe that preoperative endocardial catheter mapping is of value for several reasons:

1) In some patients, the VT is not inducible in the operating room (including those with automatic VT in whom a spontaneous episode can be mapped in the catheterization laboratory).

2) In some patients, not all morphologic forms of VT can be induced or mapped intraoperatively.

3) Intraoperative endocardial mapping occasionally cannot be performed because of lack of technical skills, physical factors such as mural thrombosis, or the inability to induce VT after aneurysmectomy.

We conclude that it is not unreasonable to perform preoperative endocardial catheter mapping to provide data upon which surgical ablation can be planned. Although endocardial catheter mapping should not replace detailed intraoperative mapping, which can provide more precise data, our experience suggests that it is a reasonable alternative when intraoperative studies cannot be performed.
Figure 8. Catheter endocardial map during ventricular tachycardia in patient 12. Surface leads 1, 2 and V1 are shown with electrograms from the right ventricular apex (RVA) and left ventricular sites 2, 7, 9, 1 and 8. The electrogram from site 8 at the inferoposterolateral left ventricle is earliest, preceding the onset of the QRS. T = time lines.

Figure 9. Intraoperative mapping during ventricular tachycardia in patient 12. The epicardial map is displayed as in figure 3 with ECG leads 1, 2 and V5R. The stippled area represents an aneurysm. Early epicardial breakthrough occurs 32 msec after the onset of the QRS at site 42. Earliest endocardial activation is located 2–3 cm away under epicardial site 44, which corresponds to endocardial catheter mapping site 8, which was the origin predicted by catheter mapping (see fig. 8).
Quantification of Aortic Valvular Regurgitation in Dogs by Nuclear Imaging

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SUMMARY Radionuclide gated cardiac blood pool (GBP) imaging was used to quantitatively assess the severity of acute aortic valvular regurgitation produced experimentally in 10 anesthetized dogs. Right ventricular (RV) and left ventricular (LV) stroke counts (end-diastolic minus end-systolic counts in RV and LV regions of interest) were used as indices of the stroke volumes of the two ventricles. Regurgitant fraction (RFGBP) was derived by assuming that an excess of LV stroke counts compared to RV stroke counts was due to regurgitant flow: RFGBP = LV stroke counts - RV stroke counts/LV stroke counts × 100. Regurgitant fraction (RFEMF) was also estimated directly from an electromagnetic flowmeter (EMF) on the ascending aorta. Mean RFEMF was 55.8 ± 17.9% (±SD). Close agreement was found between regurgitant fractions measured by GBP and EMF (RFGBP = 1.09, RFEMF = 4.7%, r = 0.88, p < 0.001, SEE = 9.98%). The severity of regurgitation from blood pool images also correlated closely with aortic pulse pressure (r = 0.89) and the length of the tear in the aortic valve (r = 0.84). These results suggest that blood pool imaging may be useful for noninvasive quantification of regurgitant flow in patients with valvar insufficiency.

NONINVASIVE ASSESSMENT of the severity of aortic and mitral regurgitation in patients is usually based on clinical evaluation, supplemented by indirect techniques such as electrocardiography, chest radiography, and echocardiography, which assess the effects of regurgitation and other factors on ventricular myocardium and chamber size, but which do not measure regurgitation directly. Quantification of regurgitation in man is most often based on measurements of total left ventricular stroke volume and effective forward stroke volume. This requires invasive cardiac catheterization with biplane cineangiography and measurement of forward cardiac output by indicator dilution or Fick techniques. Unfortunately, this quantitative ventriculographic approach is technically difficult and most angiographers therefore use some form of subjective grading of the severity of regurgitation.

A new, noninvasive method for measuring the severity of mitral and aortic regurgitation based on nuclear imaging has been recently described. The difference in activity measured within end-systolic and end-diastolic areas of interest over the left and right ventricles are measured from a gated blood pool image. Since the blood pool activity is proportional to blood volume, a left ventricular/right ventricular...
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