PATHOPHYSIOLOGY AND NATURAL HISTORY
VENTRICULAR TACHYCARDIA

Intermittent failure of local conduction during ventricular tachycardia

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ABSTRACT Forty-three patients with sustained ventricular tachycardia (VT) caused by prior myocardial infarction underwent intraoperative endocardial activation mapping during a total of 122 episodes of VT. Electrograms obtained during mapping were analyzed to determine the prevalence of local conduction failure during VT (defined as a portion of the local electrogram that did not repeat with every tachycardia cycle). Local conduction failure during VT was observed in 37 (86%) patients and 73 (65%) tachycardias. VT in which local conduction failure was observed were faster than VTs without local conduction failure (cycle length 315 vs 345 msec; p < .05). Local conduction failure occurred most frequently at or near sites having the earliest recorded electrical activity during VT ("site of origin"). Twenty-three patients also had sinus rhythm endocardial mapping at the time of surgery. Areas with abnormal or fractionated electrograms in sinus rhythm were more likely to demonstrate local conduction failure in VT than areas with normal electrograms in sinus rhythm (16% vs 8%; p < .01). Although the mechanism responsible for local conduction failure in VT is unclear, it is a common occurrence and is significant in that it can occasionally mimic "early" sites of endocardial activation, unless enough VT cycles are observed at a given site.

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INTRAOPERATIVE activation mapping during ventricular tachycardia (VT) has been used for several years to guide surgical therapy of malignant ventricular arrhythmias. With this technique, the local electrical activity from numerous endocardial or epicardial sites is sampled during induced VT with the aim of finding the earliest recorded site of electrical activity, which has been termed the "site of origin" of VT. Occasionally, part of the local electrogram obtained during mapping does not repeat with every tachycardia beat. We have termed this phenomenon intermittent local conduction failure (LCF). This study was undertaken to determine (1) the incidence of intermittent LCF during intraoperative activation mapping in VT, (2) factors associated with the development of LCF during VT, and (3) possible underlying mechanisms for this phenomenon.

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Methods

The study population consisted of 43 patients who underwent intraoperative mapping-guided subendocardial resection for drug refractory sustained VT. There were 35 men and eight women, with ages ranging from 39 to 72 years (mean 57). All patients had suffered prior transmural myocardial infarctions (37 anterior, six inferior), from 1 month to 10 years before surgery. All patients had failed a mean 4.7 drug trials before the consideration of surgical therapy.

The operative procedure consisted of a median sternotomy and initiation of normothermic cardiopulmonary bypass. The left ventricle was opened at the site of an aneurysm or previous infarction, and sustained VT was induced by programmed electrical stimulation. Endocardial activation mapping during VT was carried out with a single roving bipolar probe electrode (interelectrode distance 1 to 2 mm) according to a predetermined sequence of data acquisition (clockface format). A mean of 36 sites were sampled during each distinct VT morphology. Different VT morphologies in each patient were distinguished by comparison of three to four surface electrocardiographic leads as well as bipolar reference electrograms (using Teflon-coated stainless wires embedded in the endocardium) from each ventricle. Signals from the probe electrogram were amplified, filtered at 50 to 400 Hz, and recorded along with the surface and reference electrograms on paper (Siemens-Elema 16 Channel Ink Jet Recorder, 200 mm/sec paper speed) and on analog tape (Honeywell Model 5600). At least eight VT cycles from each endocardial site were recorded with the probe electrogram and analyzed postoperatively. Procainamide (500 to 1500 mg) was administered to 19 patients during intraoperative mapping to facilitate induction or mapping of VT (by slowing the cycle length). Twenty-three of the patients also underwent extensive sinus rhythm endocardial activation mapping of at least 20 en-
endocardial sites according to a clockface grid as described previously. Endocardial mapping time during normothermic cardiopulmonary bypass was 1 hr or less in all cases.

**Definitions.** LCF was said to occur when a repetitive portion of an endocardial electrogram did not repeat with every tachycardia beat.

Site of origin of VT was defined as the endocardial site from which the earliest electrogram in the latter half of diastole was recorded in that tachycardia.

Intraoperative sinus rhythm endocardial electrograms were arbitrarily classified as follows:

A normal electrogram was an electrogram with a discrete spike having an amplitude of greater than 1 mV and a duration less than 70 msec. All other electrograms were classified as abnormal.

A fractionated electrogram was an abnormal electrogram with multiple high-frequency components having an amplitude less than 0.3 mV and a duration greater than 90 msec.

Examples of these definitions are seen in figure 1.

Additional electrogram types were as follows:

- A split electrogram was an abnormal electrogram with at least two discrete spikes, separated by at least 30 msec.
- A late electrogram was one in which electrical activity extended beyond the end of the surface QRS complex.
- An individual electrogram could be classified in more than one way, i.e., both fractionated and late.

**Statistical analyses.** Continuous variables were compared by Students’ unpaired t test and categorical variables were compared by the chi-square test with Yates’ correction for continuity. All tests were subject to a p < .05 level of significance.

**Results**

**Patients.** Thirty-seven of the 43 patients (86%) in this series demonstrated intermittent LCF at least one endocardial site during VT. LCF was observed in all patients with anterior myocardial infarction and 31 out of 37 (84%) patients with previous anterior infarctions (p = NS). The administration of procainamide during mapping did not significantly influence the prevalence of LCF; 18 out of 19 patients who received procainamide demonstrated LCF, and 19 out of 24 who did not receive procainamide demonstrated LCF (p = NS).

VT: Seventy-three out of 112 (65%) tachycardias in this series demonstrated intermittent LCF. The overall incidence of LCF was approximately one out of eight sites recorded. The predominant pattern of conduction failure was 2:1; less common was Wenckebach periodicity (5% to 10% of instances) (figure 2) or variable conduction patterns. The mean cycle length of tachycardias during which LCF was observed was shorter than the cycle length of tachycardias not demonstrating intermittent LCF (315 ± 74 vs 345 ± 77 msec [mean ± SD]; p < .05).

Intermittent LCF occurred more frequently at or near sites of origin of the same or another VT. We have shown in a prior study of postinfarction patients with multiple morphologically distinct VT that different VTs are mapped to the same or adjacent areas (<3 cm distant) in 85% to 90% of patients. Twenty-two percent of instances of intermittent LCF were recorded at a site of origin of the same or another VT; 51% were observed within a 1 cm radius of the site from which the earliest electrogram during VT was recorded, and 75% of examples of LCF were recorded from within 2 cm of the site of origin of VT. Twenty-five percent of instances of LCF were recorded from sites more than 2 cm distant from a site of origin of VT. Although the distribution of endocardial sampling was more dense near sites of origin, the prevalences noted above are significantly different from those of all sites recorded during VT (p < .001).

The administration of procainamide during mapping again had no statistically significant effect on the prev-

![FIGURE 1](https://circ.ahajournals.org/). LCF recorded during VT. Surface ECG leads I, II, III, and V5,R are displayed with endocardial reference electrograms from the right (RV) and left (LV) ventricles, as well as a roving electrode (PROBE). Time and amplitude scales are also displayed. The vertical dashed line denotes the onset of the surface QRS complex. Part of the PROBE electrogram repeats with every tachycardia beat, but part (arrows) repeats every other beat. This is an example of LCF during VT. The PROBE electrogram is fractionated (see definitions) and was recorded from the “site of origin” of this tachycardia. The LV electrogram is an example of a normal electrogram (see definitions). Analog signals in this and subsequent figures traced from original.
alence of LCF in individual VTs. Twenty-six out of 39 (67%) of VTs mapped after administration of procainamide demonstrated LCF, and 47 out of 73 (64%) of VTs without drug administration demonstrated LCF (p = NS).

Procainamide was administered after mapping of one VT morphology but before mapping other VT morphologies in 5 patients. LCF was observed in all VTs in two of these patients; LCF was not observed in VTs mapped after procainamide in one patient; and LCF was observed only in VTs mapped after procainamide was given in the other two patients. Of note, in these latter two patients, procainamide facilitated the induction of a more rapid VT (cycle lengths 85 and 90 msec less than previous VT), which may have accounted for the occurrence of LCF in these VTs.

Intermittent LCF was recorded with approximately equal frequency from all regions of the endocardium; among patients in whom LCF was recorded, the percent of septal, anterior wall, and inferior wall sites demonstrating LCF was 15%, 13%, and 10%, respectively (p = NS). The surface electrocardiographic QRS morphology of VT had no influence on the prevalence of LCF during mapping (right vs left bundle branch block pattern, inferior vs superior axis, right or left axis).

Sinus rhythm mapping (table 1). Twenty-three patients had extensive sinus rhythm endocardial activation mapping. A mean 53 ± 15 sites were sampled during sinus rhythm. Eight percent of sites with normal electrograms in sinus rhythm demonstrated intermittent LCF during VT, whereas 16% of sites with abnormal electrograms in sinus rhythm demonstrated LCF in VT (p < .001). Among the subcategories of abnormal electrograms, intermittent LCF was also more likely to occur at sites with fractionated (16%), split (24%), and late (19%) electrograms in sinus rhythm than at normal sites (all p ≤ .01).

Discussion

Comparison with prior studies. Intermittent LCF has been observed in a canine preparation of infarction-associated VT as reported by Boineau and Cox and El-Sherif et al. These investigators demonstrated 2:1 conduction of late potentials recorded from the infarct zone using intramural and epicardial mapping. Bailey et al. reported a case of intermittent LCF recorded in man with an endocardial catheter. These investigators administered procainamide to the patient and observed a late potential change from 1:1 conduction to 2:1 and finally 2:1 conduction, and believed that procainamide was responsible for this effect. In the current study, administration of procainamide did not influence the incidence of intermittent LCF during the recording of large numbers of endocardial electrograms. LCF was first observed after administration of procainamide in two of our patients but not during the same VT. Procainamide might be expected to facilitate LCF by causing prolonged refractoriness, but this effect may be counterbalanced by the drug’s effect on conduction velocity and VT rate (making LCF less frequent).

Gilmour et al. studied cellular electrophysiologic

<table>
<thead>
<tr>
<th>Sinus rhythm electrogram</th>
<th>LCF Present</th>
<th>LCF Absent</th>
<th>p value (vs normal sites)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>45 (8%)</td>
<td>517 (92%)</td>
<td>—</td>
</tr>
<tr>
<td>Abnormal</td>
<td>109 (16%)</td>
<td>553 (84%)</td>
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<td>28 (16%)</td>
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<tr>
<td>Split</td>
<td>7 (24%)</td>
<td>22 (76%)</td>
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</tr>
<tr>
<td>Late</td>
<td>18 (19%)</td>
<td>83 (81%)</td>
<td>.001</td>
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can be read into a natural plain text representation as:

characteristics of endocardial resection specimens and observed intermittent LCF during pacing in several instances. Regions from which LCF was recorded had a decrease in resting membrane potential as well as a decrease in upstroke velocity of the action potential (dv/dt). Cellular electrophysiologic studies were not performed on endocardial resection specimens in the present series for comparison with the above studies. Additionally, these studies in vitro must be interpreted with caution because the abnormalities recorded may have resulted from tissue damage during removal or handling and have little relationship to events in vivo.

Cause of LCF. The exact electrophysiologic abnormalities responsible for the phenomenon of intermittent LCF are not clear, but several possibilities exist. Microanatomic factors may be responsible for failure of impulse transmission from one group of cells to another. Cardiac histopathologic findings in a population of patients with prior infarction and VT have shown isolated bundles of ventricular myocardium, five to 10 cells thick, separated by dense fibrous bands. Presumably, these thin bands of muscle are connected to more normal myocardium at the borders of the infarction. These thin bundles of ventricular muscle may be analogous to thin strips of atrial muscle forming a bridge between large muscle segments, as studied by de La Fuentes et al. These investigations observed 2:1 conduction of impulses across these thin bridges of atrial muscle and ascribed this to “impedance mismatch.” Likewise, the direction of impulse propagation relative to microanatomic fiber orientation, and resultant changes in intercellular resistance (anisotropy), may play a critical role in producing abnormal conduction. Spach et al. observed that, even in normal canine atrial muscle, anisotropic influences on impulse propagation could produce conduction delay, unidirectional block, Wenckebach periodicity, or 2:1 conduction failure. The abnormal microanatomy in patients with extensive endocardial scar (nonuniform anisotropy) could also yield abnormal patterns of conduction.

Abnormalities of cellular and tissue electrophysiology, independent of anatomic factors, may also be responsible for intermittent LCF. These would include increased refractoriness (local refractory period greater than VT cycle length), as has been observed in experimental infarctions. Calcium channel-dependent action potential prolongation has been invoked by El Sherif et al. as a cause of LCF in a canine preparation of VT. These investigators abolished LCF in these preparations by administering D-600, a methoxy derivative of verapamil. Altered time dependence of recovery of excitability has been observed to be responsible for the creation of block in certain circumstances. Saltatory conduction via passive electrotropic influences can result in asynchronous and markedly abnormal conduction. Finally, the decreasing efficacy of a wavefront to elicit a downstream action potential, such as occurs normally in the atrioventricular node, may also be responsible for LCF. Regardless of what abnormalities account for LCF on the cellular or tissue level, both the cycle length dependence of intermittent LCF and its occurrence in areas with abnormal sinus rhythm electrograms (as shown in this study) suggest that significantly diseased tissue is involved. The anatomic correlate of abnormal and fractionated sinus rhythm electrograms is not clear, although preliminary studies suggest that fractionated electrograms can be caused by myocardial cells with normal action potentials and upstroke velocity of the action potential, but poor cell-to-cell coupling. Thus, further studies are needed to evaluate the cellular and anatomic features associated with, and perhaps responsible for, intermittent LCF.

It is conceivable that LCF is actually recording atrial electrical activity during VT, which often is observed in a 2:1 Wenckebach conduction ratio. Atrial electrograms were not routinely recorded during intraoperative studies in this series, but we believe atrial activity is unlikely to be the cause of LCF because (1) LCF was observed frequently in sites near the left ventricular apex, several centimeters removed from atrial tissue; (2) five of the patients who demonstrated LCF were in chronic atrial fibrillation during intraoperative mapping; and (3) LCF was occasionally recorded from extremely localized areas (figure 3). It is also possible that intermittent recording of subendocardial Purkinje fibers could account for examples of LCF. Histopathologic study of endocardial resection specimens has revealed Purkinje fibers in septal and papillary muscle endocardial specimens but not in free-wall endocardium. LCF was observed in equal frequency throughout the left ventricular endocardium in the present study, however, which is at variance with the distribution of endocardial Purkinje fibers in this patient population. Although we cannot exclude Purkinje tissue as the site and source of intermittent LCF, ventricular myocardial cells may be a more plausible source of intermittent LCF. Cellular electrophysiologic abnormalities recorded from surviving ventricular myocardial cells in regions of previous infarction are similar to findings in animal preparations in which intermittent LCF was also observed. Regardless of the cells responsible for intermittent LCF, it is clear that they
are not necessary for the perpetuation of VT, since they are activated only every other beat or less frequently.

Finally, it is also possible that the phenomenon we observed is not really "failure" of local conduction but rather a beat-by-beat change in the direction of part of the excitation wavefront as it passed beneath the recording bipolar (perpendicularly) so that no signal was recorded on every other beat, although the tissue was still being activated. This would most likely result in an alteration in configuration or amplitude of part of the electrogram rather than complete absence. Additionally, figure 3 suggests that at least in some instances, LCF is not caused by such changes in wavefront direction relative to the recording bipolar, since several closely spaced bipoles still failed to record some of the intermittent potentials. Thus, although changes in wavefront direction could conceivably account for the appearance of LCF in some cases, we believe it is not a frequent cause of the phenomenon.

**Significance.** Since 75% of sites demonstrating intermittent LCF are found within 2 cm of a VT site of origin, it may be argued that subendocardial resection or other surgical procedures could be directed at areas demonstrating LCF. This would not seem to hold much promise, in that it is still necessary to extensively map VT to find sites with intermittent LCF, and 25% of instances of LCF occur at sites greater than 2 cm distant from any VT site of origin. Thus more extensive resections would be undertaken than would be necessary to eradicate VT, and little would be gained by directing surgery in this way.

Occasionally, intermittent LCF can mimic a site of origin, as shown in figure 4. In this example, the timing of the portion of the local electrogram that undergoes LCF is such that it appears to be significantly presystolic, but if observed for enough cycles, it eventually demonstrates 2:1 or Wenckebach block. The portion of the local electrogram that is repetitive from cycle to cycle (1:1) is clearly not "early." This "pseudo-early site" is unfortunately an uncommon phenomenon, occurring in only five of the tachycardias in the present series. Nonetheless, caution must be exercised in interpreting "early" sites that occur in isolation, with sites immediately surrounding it having a substantially later electrogram timing. In each of the five cases in which LCF mimicked an early site in this series, it was recognized as being spurious because of the large dis-
parity in timing of electrograms from adjacent sites. The use of sophisticated and expensive computer systems that simultaneously analyze multiple endocardial sites during only a few VT beats could obviously yield misleading information as a result of LCF. Analysis of a large number of VT complexes may thus be required to exclude LCF as a cause of "early" electrograms.

Limitations. The administration of antiarrhythmic drug during intraoperative mapping, which could conceivably produce or eliminate intermittent LCF by altering conduction and/or refractoriness, was not controlled in this series. However, there was no statistical difference in the prevalence of intermittent LCF in patients who received drug vs those not receiving drug during mapping, nor among individual tachycardias that were mapped with or without the influence of antiarrhythmic drug; however, the numbers of patients in each subgroup were small. Additionally, the possibility that intermittent LCF may be caused by the recording of a distant atrial electrogram has not been definitely excluded in this study by systematic recording of atrial activity; this limitation has been discussed above.

A major limitation is the reproducibility of location of endocardial sites in comparing sinus rhythm electrograms and those during VT. This may account for at least some of the instances of intermittent LCF recorded at sites that had normal electrograms in sinus rhythm, although anisotropy or other anatomic or physiologic factors may have been responsible as well. Electrograms of a given variety (normal, abnormal, fractionated) tend to occur in clusters on the endocardium, and thus a small (1 cm) error in location is unlikely to have a profound influence on the type of electrogram obtained. Nonetheless, this is a significant limitation in the current study.

Conclusions. This study demonstrates that intermittent failure of local endocardial conduction during intraoperative endocardial mapping of VT is a frequent finding. LCF is more commonly observed in tachycardias with a shorter cycle length and more frequently occurs in sites having abnormal or fractionated electrical activity than at sites with normal electrograms in sinus rhythm. Intermittent LCF is frequently observed at or near sites of origin of VT and can on occasion mimic a site of origin because of fortuitous timing of the intermittent electrogram relative to the onset of the surface QRS. The exact cause of this phenomenon, as well as the tissues responsible, are unknown at present. Further work is needed to resolve these issues.

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