Electrocardiographic effects of experimental nontransmural myocardial infarction

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ABSTRACT Clinical and experimental data have documented the ability of nontransmural myocardial infarction to produce abnormal Q waves on both the epicardial and body surfaces. We undertook this study to define the anatomic determinants of such Q wave development. Thirty dogs were studied before and after occlusion-reperfusion (26 dogs) or latex embolization (four dogs) of the left circumflex coronary artery. Occlusion was maintained for 60 to 240 min before reperfusion to produce nontransmural lesions of various sizes. Electrocardiographic data were registered from 84 torso electrodes by body surface mapping techniques before and 1 week after infarction. Infarct size was quantitated by computer analysis of heart slices stained with triphenyl tetrazolium chloride. Six dogs did not develop infarction. Of the remaining 24, 10 did and 14 did not develop significant changes in body surface Q wave duration and width. The incidence of Q wave changes was not different in dogs with nontransmural and those with transmural lesions. Infarct size (expressed as a percentage of the left ventricle infarcted), the percentage of endocardium subjacent to infarction, the average depth of necrosis, the percent of the four outer fifths of the ventricular wall infarcted, and the duration of occlusion were significantly (p < .05) greater in dogs with than in those without Q wave changes. Logistic regression modeling demonstrated that only two anatomic parameters — percentage of left ventricle infarcted and average lesion depth — significantly and independently predicted Q wave development. A model including only these two variables accurately classified all 24 cases. Thus, nontransmural infarction can produce abnormal body surface Q waves, and the development of new Q waves after infarction is statistically dependent on overall infarct size as well as average transmural extent of necrosis.


The recent emphasis on the clinical similarities and differences between these two electrocardiographic classes of infarction14, 15 suggested to us that a reevaluation of the pathophysiologic variables determining Q wave evolution was warranted. To do so, we produced myocardial infarction of various sizes by transmural occlusion16–18 or embolization18, 19 of the left circumflex coronary artery in dogs. We then determined the anatomic features of the resulting lesions, correlated them with body surface electrocardiographic features, and developed a statistical model of the factors interacting to produce abnormal body surface Q wave patterns.

**Methods**

Experimental preparations. We successfully studied 30 adult mongrel dogs weighing 16.1 to 20.4 kg. Each underwent a left thoracotomy under halothane–nitrous oxide–oxygen anesthesia. Two experimental protocols were used. In 26 animals, a hydraulic balloon vascular occluder with an internal diameter of 4 mm was placed about the proximal left circumflex artery. The tubing was tracked subcutaneously and exteriorized at the nape of the neck.

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One week later, when all animals were ambulatory, we inflated the occluder device by injecting mineral oil into the exteriorized tubing until we met significant resistance to further infusion. Occlusion was maintained by clamping the tubing for 60 min in five dogs, for 90 min in eight dogs, for 2 hr in eight animals, and for 4 hr in five dogs. At the end of the designated time period, we aspirated the mineral oil from the occluder and left the tubing unclamped.

In the remaining four dogs, we first ligated the circumflex artery. We then performed an arteriotomy and injected 1 ml of liquid latex to "embolize" the distal vasculature. The chest was then closed and air was evacuated. The positions of vascular obstruction were standardized in all cases.

**Electrocardiographic recordings.** We recorded electrocardiographic signals twice. The first set was acquired before coronary occlusion or embolization. For occlusion-reperfusion subgroups, this was done immediately before balloon inflation, 1 week after thoracotomy. For those undergoing coronary embolization, the initial data were registered just before surgery. The second set was recorded, for both groups, 1 week after infarction was produced.

Animals were sedated at each recording session with intramuscular Innovar-Vet (20 mg droperidol and 0.4 mg fentanyl/ml). Eighty-four chloridized silver electrode disks were then fixed to each dog’s shaved chest in vertical strips extending from the clavicles to below the inferior rib margins, on both the anterior and posterior hemithoraces. Additional electrodes were placed on the extremities to record limb leads and to derive a Wilson central terminal potential. Electrode positions were carefully standardized for both recording sessions in each dog. After electrode placement, dogs were placed in an upright position with use of a support sling to further increase recording reproducibility.

Signals from the 88 electrodes were amplified by a bank of low-noise differential (electrode vs Wilson central terminal potential) amplifiers. Gains of each amplifier were individually set at 1000 to 16,000 under computer control so that the output filled the input range of the analog-to-digital converter. Analog signals were then converted to digital form at a rate of 500 samples per channel per second, with simultaneous sampling of all electrode voltages. Baseline stability and low noise levels were visually verified at each session.

**Determination of infarct size and location.** We killed the dogs immediately after the second electocardiographic recording session. Excised hearts were sliced into 8 mm sections, parallel to the atrioventricular groove, with a brain macrotome. These sections were incubated for 30 min in triphenyl tetrazolium chloride (1% solution in phosphate buffer) at 37°C to identify necrotic myocardium. Infarcted tissue remains pale whereas viable myocardium is stained deep red.

Stained slices were photographed and images were traced onto a microprocessor-based graphics digitizing tablet. Outlines digitized included the left ventricular epicardial and endocardial borders and the rim of the infarcted zone (figure 1). From these data, we quantitated the following variables: (1) the percentage of left ventricle that was infarcted, (2) the maximum and average depth of the infarction, expressed as a percentage of transmural wall thickness, and (3) the percentage of left ventricular endocardial area subjacent to the infarction.

In addition, the ventricular wall was subdivided into five segments of equal thickness, with segment 1 being on the endocardial and segment 5 being on the epicardial surface. Each segment was bordered laterally by radii drawn from the geometric centroid of the left ventricle to the endocardial margins of the infarction and extended to the epicardial surface. The percent of volume in each that was necrotic was then computed. A transmural lesion was defined by the presence of greater than 10% infarction in the most epicardial segment (segment 5).

Simpson’s rule was used to compute areas. Volumes were computed assuming a paraboloid model of the left ventricle.

**Analysis of electrocardiographic data.** We acquired 14 sec of data at each recording session. Serial PQRS waveforms in each lead with similar morphologies were averaged to reduce random noise. Onsets and offsets of the PR interval, the QRS complex, and the ST-T interval were then manually determined from plots of orthogonal leads. We selected and averaged a 10 msec period of the terminal TP segment as a zero potential baseline.

Beginning and end of Q waves were determined in each lead by visual observation of each of the 84 averaged waveforms. An
initial negative deflection lasting longer than 4 msec was considered to be a Q wave. The end of the Q wave was defined as the instant at which the initial negative wave reached the predetermined TP segment baseline (figure 2).

From these data, we quantitated the duration as well as the depth of the Q wave in each torso lead (insert in figure 2, A). A duration and a depth of zero were assigned to records with initial R waves (figure 2, A).

These measurements were then processed to construct “isointerval” and “isodepth” maps displaying the spatial distribution of Q wave durations and depths, respectively, on the dog’s torso. Examples of each are shown in figure 3. Numbers in figure 3, A, mark electrode locations, with the value equaling the Q wave duration, in milliseconds, recorded at that site. The center of each map is along the sternum, and the left and right margins correspond to the right and left paravertebral zones, respectively. In the isodepth map in figure 3, B, values equal the Q wave depth, in microvolts, at each locus. Contour lines connecting points with equal values were then drawn. Findings in these examples will be discussed below.

The duration and depth of the Q wave in each lead before infarction was subtracted from the corresponding value after infarction to define the effects of the lesion. These differences were then used to construct isointerval and isodepth “difference” maps. A change in Q wave duration of more than 4 msec or in Q wave depth of more than 100 μV was considered to be significant.

Statistical methods. All data are presented as mean ± SD. Comparisons of infarct size parameters in subsets of animals with and without Q wave changes were performed by analysis of variance methods or by Fisher’s exact test. 5% was used as the level of significance.

A stepwise logistic regression model was then used to determine which experimental factors predicted the development of body surface Q waves. The dependent variable was the coded presence or absence of new Q waves. Independent variables included the duration of occlusion, percentage of left ventricle infarcted, average transmural depth of necrosis, maximum transmural lesion depth, and the percentage of each of the five intramural sectors that were necrotic. Duration of occlusion was coded 1 to 5 (1 = 60 min; 2 = 90 min; 3 = 120 min; 4 = 240 min; 5 = latex embolization). Variables were added to the model until the step fit a good chi square was computed to test the hypothesis that the statistical model at that step fit the data adequately. A high p value indicated a good fit. Also computed was an “improvement” chi square, testing the hypothesis that the term entered or removed at that step significantly improved the model. A small p value indicated a significant improvement.

Results

Control Q wave patterns. We first evaluated the distribution of Q waves before coronary occlusion to define the normal patterns. Examples of electrocardiographic records without and with normal Q waves are shown in figure 2. The maps in figure 3 display the spatial distributions of these normal Q waves. Q wave durations (panel A) varied from 0 (not present) to 30 msec. Q wave depth (panel B) varied from 0 to over 900 μV. Q waves were spatially located over the posterior and left lateral anterior torso, with widest and deepest deflections recorded on the superior posterior chest.
Data from control recordings in all 30 animals were averaged. The number of dogs with Q waves at each site is displayed in figure 4, A. As in the example shown in figure 3, Q waves were most common on the lateral and posterior torso surfaces. All 30 dogs had Q waves along the vertebral column. They were uncommon on the anterior chest, and no normal dog had Q waves in leads from the central and upper sternal regions.

Mean durations of normal Q waves (figure 4, B) were also greatest over the posterior superior chest and shortest over the anterior inferior zones. Similarly, Q waves were deepest (figure 4, C) on the posterior chest.

SDs about the mean values for these parameters were also computed. For Q wave duration, the SD ranged from 1.59 to 16.54 msec. Values for Q wave depth ranged from 14.57 to 456.0 μV.

**Measurements of infarct size.** In six dogs no necrosis was demonstrable; the circumflex artery was occluded for 60 min in three, for 90 min in two, and for 2 hr in one. Descriptive statistics for the quantitated infarct measurements in the remaining 24 dogs are listed in table 1. A wide range of lesion sizes was produced in both the occlusion-reperfusion and the embolization preparations. Eight lesions were transmural; 16 were nontransmural. Although larger infarctions generally resulted from longer durations of occlusion and from embolization than from short occlusions, considerable overlap was observed. For example, from 3.79% to 14.2% of the left ventricle was damaged after 90 min of occlusion and values after 120 min ranged from 1.51% to 23%.

**Postinfarction Q waves.** QRS duration after infarction was identical to that before occlusion; differences equaled 1.13 ± 1.86 msec. Examples of QRS changes produced by infarction are shown in figure 2. As shown in panel A, the record before occlusion had an initial R wave; Q wave duration and depth were therefore 0. After occlusion, a 10 msec Q wave developed with a depth of 134 μV. At other sites, such as that from which the waveform in panel B was registered, a normal Q wave was recorded before occlusion. After infarction, however, this normal Q wave increased in duration to 40 msec and in depth to over 600 μV.

Six dogs did not develop myocardial necrosis after transient coronary occlusion. Comparison of preocclusion and postocclusion Q wave maps in these cases would therefore serve to define the variation due only to time. The maximum difference in Q wave duration was 4 msec, and the maximum difference in Q wave depth was 98 μV. These small changes confirm the stability of measurement techniques over the 1 week experimental period.

Twenty-four animals developed areas of necrosis. Of these, 10 did and 14 did not develop significant Q wave changes. One example from each group demon-

**TABLE 1**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean ± SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>% of LV infarcted</td>
<td>13.09 ± 11.98</td>
<td>0.33-36.51</td>
</tr>
<tr>
<td>Endocardial surface infarcted (%)</td>
<td>29.33 ± 12.37</td>
<td>3.0-53.0</td>
</tr>
<tr>
<td>Average transmural depth</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(% of wall thickness)</td>
<td>43.87 ± 22.22</td>
<td>11.12-86.82</td>
</tr>
<tr>
<td>Maximum transmural depth</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(% of wall thickness)</td>
<td>88.49 ± 20.66</td>
<td>24.45-100.0</td>
</tr>
<tr>
<td>% of intramural segment infarcted</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Segment 1</td>
<td>61.35 ± 20.36</td>
<td>19.52-88.51</td>
</tr>
<tr>
<td>Segment 2</td>
<td>46.69 ± 25.21</td>
<td>2.35-81.12</td>
</tr>
<tr>
<td>Segment 3</td>
<td>33.92 ± 25.31</td>
<td>0.00-75.06</td>
</tr>
<tr>
<td>Segment 4</td>
<td>23.47 ± 26.39</td>
<td>0.00-76.9</td>
</tr>
<tr>
<td>Segment 5</td>
<td>17.18 ± 26.49</td>
<td>0.00-76.58</td>
</tr>
</tbody>
</table>

LV = left ventricle.

*Includes only data from 24 dogs with infarction.

**FIGURE 4.** A. Spatial distribution of the prevalence of Q waves at each of 84 torso loci in 30 preocclusion data sets. Values indicate the number of dogs with Q waves at that site, and contour lines connect sites with equal frequencies (0, 12, and 24 dogs). B. The spatial distribution of the mean value of Q wave duration at torso sites. C. Mean values for Q wave depth.
FIGURE 5. Tracings in A anterior electrode sites to determine normal Q waves by postinfarction and 13 others dog are shown A, magnitudes decreased in figure chest and nine other in duration than 4 sites (by from the animal 11.63% average 23.06% of former, 11.63% of the ventricle was necrotic, with an average transthal depth of 67.45%. In the latter, only 11.63% of the ventricle was infarcted and the average transthal depth measured 44.43%.

Q wave isointerval and isodepth difference maps from the animal whose infarct is depicted in figure 5, A, are shown in figure 6. New and significant (greater than 4 msec) Q waves appeared after infarction at 12 sites while preexistent Q waves increased in duration (by over 4 msec) at 35 other positions. These changes in duration were accompanied by increases in Q wave depth. Both were located in posterior and inferior-anterior chest regions. Based on these changes, this and nine other similar dogs were considered to have postinfarction Q wave alterations.

Difference maps in figure 7 are from the dog in figure 5, B. Q wave durations (figure 7, A) increased or decreased in a seemingly random manner by no more than 4 msec. Durations (panel B) likewise changed, without pattern, by no more than 88 μV. Based on the small magnitudes of the changes, which were similar to those seen in the six dogs without infarction, this dog and 13 others were grouped together as not having postinfarction Q wave differences.

We also determined the incidence of Q wave abnormalities by comparing postinfarction Q wave patterns with the normal Q wave distribution computed from all 30 control data sets. To do so, the upper limits of normal Q wave duration and depth at each site were determined to be the mean value plus 1.5 SDs. All electrode sites with values exceeding these limits were identified, and the dogs were reclassified as normal or abnormal. Only six dogs had abnormal Q waves when determined by this second method; four of 10 animals determined to have new Q waves based on the com-
with Q waves had transmural lesions, whereas only two of the 14 without Q waves had such. These differences were not statistically significant (p > .05, Fisher's exact test). Thus, Q waves were not more common in those with full-thickness than in those with nontransmural lesions; 40% of lesions accompanied by new Q waves were nontransmural.

Infarct parameters for the subsets of dogs with and without new Q waves are compared in table 2. Statistically significant differences between these two groups were found for (1) the percentage of the left ventricle infarcted, (2) the percentage of the endocardial surface subjacent to the infarction, (3) the average depth of infarction, (4) the percentage of infarction in all but the innermost fifth of the ventricular wall, and (5) the duration of occlusion. In each case, the group with Q waves had larger values than did the group without Q waves. Values for the other variables, including maximum depth of infarction, were not significantly different.

A logistic regression model was then developed to determine which anatomic parameters contributed independently to Q wave development. Results are tabulated in table 3. Only two anatomic variables had independent predictive value. Infarct size, measured as the percentage of the left ventricle infarcted, was the most important term. The goodness of fit chi square after addition of this single variable resulted in p value of 1.00. The second parameter entered was the average transmural depth of the lesion. These two variables were not significantly correlated with each other (r = .22). Other variables did not add to the fit of the data.

Thus, the statistical model best predicting development of new Q waves included only these two anatomic measures, plus a constant term. The resultant equation fit the data with a perfect p value of 1.00. In addition, each of the 24 cases was classified by the model as being predicted to have or not have Q wave changes. All cases were correctly classified, corresponding to false-positive and false-negative rates of zero.

**Discussion**

This pathologic-electrocardiographic correlative study demonstrated that new abnormal Q wave patterns can develop after nontransmural infarction. Statistical determinants of such changes include the size and the average transmural depth of the infarction.

**Prior studies.** Numerous other studies have documented Q wave changes after nontransmural infarction. Experimental reports have correlated epicardial and/or intramural recordings with infarction size and location. In 1935 Wilson et al. reported epicardial Q waves from sites in which "the outer layers of muscle were alive and were responding to the excitatory process." Durrer et al. recorded epicardial Q waves "even with the smallest infarction . . . with the largest diameter of less than 1 cm and an intramural extension of less than one-fourth of the thickness of the ventricular wall . . . ." Q wave changes were similarly observed over infarctions with diameters greater than only 5 mm by Daniel et al., and over subendocardial lesions produced by occlusion-reperfusion by, for example, Mickleborough et al.

In contrast to these studies showing Q waves with and due to subendocardial damage, Prinzmetal et al. attempted to prove that QR waves were due to incomplete damage to outer rather than inner muscle layers. As noted by Pipberger and Lopez and by Phibbs, this theory was contradicted by later studies from the same
laboratory confirming a role of the subendocardium in Q wave generation. Thus, the bulk of experimental data confirm the evolution of epicardial Q waves after nontransmural infarction.

Clinical studies have reported similar conclusions. Cook et al. concluded that large lesions extending through approximately one-half of the ventricular wall would cause Q wave abnormalities on the standard electrocardiogram. Savage et al. reported significant Q waves in six of 11 patients with autopsy-proven subendocardial infarction. Sullivan et al. likewise documented a high incidence of Q waves in such lesions. Bodenheimer et al. correlated myocardial biopsy pathology to body surface and epicardial electrograms in patients undergoing coronary artery surgery; epicardial Q waves occurred with less than 50% fibrosis and body surface Q waves were found only in those with greater than 50% but less than 100% fibrosis. Finally, Raunio et al. documented a 40% incidence of abnormal Q waves in patients with recent subendocardial infarction. Thus, the clinical evidence uniformly supports a role of subendocardial infarction in producing Q wave patterns.

**Consideration of methods.** This study differs in several key ways from these prior efforts. First, body surface rather than epicardial responses to experimental lesions were studied. It is the former that is analogous to the clinical problem. Although records from the two sites must be related, this relationship is complex because of intervening factors such as distance and electrical inhomogeneities. This has been documented by Bodenheimer et al., who reported a 30% incidence of epicardial Q waves that were not manifest on the body surface of patients with coronary artery disease. These investigators as well as others concluded that larger lesions would be needed to produce body surface than epicardial Q waves.

Second, the use of experimentally produced lesions permits comparison of preinfarction and postinfarction electrocardiograms, a comparison recognized to be important but not often possible in clinical studies. In addition, the frequent finding of multiple lesions confounds interpretation of clinical records. For example, in the study of Raunio et al., 11 of 15 subjects with recent subendocardial infarction also had other older scars.

The specific methods that we used have advantages as well as limitations. The occlusion-reperfusion preparation of infarction was selected because it permitted creation of a wide range of infarction sizes and geometries. It has the additional advantage of mimicking the histopathology of human subendocardial infarction. As reported by Freifeld et al., 70% of subendocardial and all of "mixed" lesions demonstrated contraction band necrosis suggestive of an occlusion-reperfusion mechanism. Thus, although Q waves may appear sooner and more frequently after reperfusion than with sustained occlusion, the former preparation is more clinically relevant. Embolization of distal vasculature with latex produces larger, usually transmural infarction by occlusion of collateral vessels responsible for epicardial sparing in preparations of subendocardial infarction.

We used body surface isopotential mapping methods to take full advantage of "proximity potentials" in detecting postinfarction Q wave changes.

Limitations are also important. First, we studied only QRS changes produced by occlusion of one vessel. Results may be different after obstruction of other beds. Second, because we did not record epicardial and intramural electrograms, we can only speculate as to the mechanisms for new Q wave development and we cannot be sure that abnormal epicardial Q waves emerged that were not projected to the body surface.

Third, we recorded data only once after coronary occlusion; we cannot therefore exclude the possibility of the early disappearance of Q waves previously present. Fourth, although mid and late QRS abnormalities do result from infarction, we limited our attention in this report to early QRS effects. Finally, differences in the torso and cardiac anatomy and between the physiology of dog and man may affect the application of the conclusions of an experimental study to a clinical problem.

**Normal Q wave patterns.** The first findings in this study were derived from examination of the Q wave pattern in normal dogs. As shown in figures 3 and 4, Q waves are most prevalent over the posterior and lateral body surfaces, with greater duration and depth superiorly within these zones. This pattern corresponds to a Q wave vector pointing anteriorly and inferiorly, i.e., the same direction found by Grant and Murray in man.

These normal waveforms reflect earliest ventricular activation on the left septal and adjacent ventricular zones. This results in an epicardial maximum over the anterior right ventricle and a minimum on the posterobasal left ventricle, corresponding to a dipole oriented anteriorly and inferiorly projecting negative potentials over the posterolateral superior torso.

This normal Q wave distribution conforms closely to the perfusion territory of the left circumflex artery, i.e., the posterobasal left ventricle. This was demonstrated by the similarities in the normal Q wave maps
(figures 3, A and 4) and the isoduration and isodepth difference maps after infarction (figure 6). It is also demonstrated by the finding that the predominant effect of infarction was to increase duration and depth of normal Q waves rather than to induce evolution of new negative waves.

The interanimal variability in this normal pattern had direct consequences on the detection of myocardial infarction. Forty percent of animals with significant new Q waves after coronary occlusion had Q wave patterns not beyond normal limits, as defined in the total population of dogs studied. Thus, "new" Q waves are not equivalent to "abnormal" Q waves. This confirms the importance of comparison of serial electrocardiograms; changes in a pattern may well be significant even when the absolute magnitude of a deflection may not exceed fixed, standard norms.

Determinants of postinfarction Q waves. A second and major set of findings identified the anatomic variables determining Q wave development after infarction. In the preparation used here, Q waves were no more common after transmural than after nontransmural infarction. Lesions causing new Q waves involved a greater percentage of the left ventricle and of the endocardial surface, had greater average transmural depths, and resulted from more prolonged periods of occlusion than did infarctions that did not result in early QRS aberrations (table 2). When all anatomic variables were entered into the logistic regression model to determine which, if any, independently predicted Q wave changes, two were selected — infarct size and average transmural depth. A statistical model using only these functions accurately predicted the Q wave pattern in 100% of test cases. Since the two selected measures were independent of each other (correlation coefficient of .22, p > 0.1), lesions that are large or of great average depth or both may be expected to produce new body surface Q waves.

Specifically not included in the final model was the maximal transmural depth and the percent of the outermost sectors of the ventricular wall. These are the indexes typically used to classify lesions as transmural or nontransmural. Thus, a narrow region of transmural necrosis may not be associated with Q wave abnormalities, whereas extensive lesions limited to the inner wall regions may produce Q waves.

These findings are in accord with limited clinical data. Cook et al. described abnormal Q waves in patients with large but not in those with small nontransmural lesions. Ideker et al. compared lesion anatomy in patients with and without abnormal Q waves; those with Q waves had larger (but not necessarily transmural) lesions than did those without Q waves. Thus, clinical correlations support our experimental conclusions.

Mechanisms of Q wave generation. Our findings are also consistent with electrophysiologic mechanisms responsible for epicardial Q waves. Wilson et al. suggested that epicardial Q waves resulted from passive transmission of intracavitary potentials reflecting distant activation to the heart surface. This passive effect is permitted by the electrical inactivity of the infarcted tissue plus, in cases of nontransmural infarction, delay in excitation of surviving subepicardial tissue.8 This latter effect is due to slowed intramural conduction and late activation of surface tissue by wavefronts traveling abnormally long distances around inactive tissue and, perhaps, in a tangential direction from neighboring intact myocardium.9 In studies by Daniel et al.,25 Q waves overlying subendocardial lesions were only seen in cases in which such epicardial delay was present.

If, as suggested by these prior studies, epicardial delay contributes to Q wave appearance and is quantitatively related to the distance the excitation wave must travel to circumvent a necrotic subendocardium, then larger and deeper nontransmural lesions should create longer paths, more epicardial delay and, hence, more and larger epicardial Q waves. This has been shown in the experiments of Durrer et al.,9 in which epicardial delay was seen only with larger nontransmural lesions. For these Q waves to appear in body surface leads, these initial forces must be sufficiently intense and widely distributed spatially within the solid angle subtended by those leads. Larger lesions would likewise facilitate this transfer.

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