Significance of the Diagnostic Q Wave of Myocardial Infarction

By Leo G. Horan, M.D., Nancy C. Flowers, M.D., and Jennifer C. Johnson, M.D.

SUMMARY
Correlation between the QRS complex and postmortem ventricular anatomy was made in 1184 instances of normal conduction:
(1) Mechanical reliance on the sheer presence or absence of a Q wave greater than 0.03 sec in duration led to "correct" diagnosis of infarction or not in 79% of the series.
(2) With normal conduction, abnormal Q waves isolated to either the anteroseptal (V1-V4) or inferior (II, III, aV_F) electrocardiographic zones were frequently false (46%). However, abnormal Q waves restricted to the lateral zone (V5-V6) or in a combination of more than one electrocardiographic zone, were rarely false predictors of the presence of infarction (4%).
(3) Classical localization of infarction with normal conduction was statistically relatively reliable as compared with bundle-branch block. The increased frequency of the anatomic pattern of lateral basal infarction in association with normal QRS complexes (but known infarction) suggests relative "electrical silence" of the latero-basal left ventricle in abnormal Q-wave genesis.
(4) Lesions confined to a given anatomic location in the left ventricle tended to place particular emphasis and limits on the spectrum of electrocardiographic expression but did not yield a uniform single pattern of Q-wave distribution.

Additional Indexing Words:
Abnormal Q wave  Clinicopathologic correlation  Myocardial scar
Electrocardiogram

The association between an abnormally wide Q wave and myocardial infarction has become so firmly entrenched in the mind of the electrocardiographer as to approach dogma. Monumental work of Fenichel and Kugell, Wilson and coworkers, and Myers and coworkers established the theoretical and empirical basis for association between myocardial necrosis and Q-wave abnormality. All of these workers recognized that the association was high but not absolute. In a classic experiment, Bayley and LaDue demonstrated appearance of an abnormal Q wave within minutes after transient coronary occlusion in the experimental animal—a finding later confirmed by many and shown to be reversible, thus providing evidence that the new Q wave is not synonymous with myocardial necrosis. Simonson, in examining the statistical basis of clinical and electrocardiographic associations, reminded us that the arbitrary division between populations of normal and abnormal (with respect to myocardial infarction) by the width of the Q wave in lead III is at best a crude one.

Our original interest in critical examination of the relationship between the presence of an
DIAGNOSTIC Q WAVE OF MYOCARDIAL INFARCTION

Table 1

| Number of Patients with Electrocardiograms in Which Q Wave ≤ 0.03 Seconds |
|-------------------------------|-------------------------------|-------------------------------|
|                               | With MI | Without MI |
| Normal QRS                   | 38      | 256         |
| Small septal Q (I, V_a, V_b) | 40      | 242         |
| No septal Q (I, V_a, V_b)    | 85      | 184         |
| Total                         | 163     | 682         |

Abbreviation: MI = myocardial infarction.

abnormal Q wave and a demonstrable anatomic deficit in the myocardium was related to the controversy surrounding the presence of Q waves or Q equivalents with left bundle-branch block (LBBB). If a rigid and orthodox pattern of depolarization in LBBB is assumed, the reliable diagnosis of infarction can be reasonably expected when this pattern is altered by distinctive QRS abnormalities. If this is not the case, diagnosis is uncertain. We found frequent examples of abnormal Q waves or Q-equivalent patterns in the presence of LBBB in electrocardiograms from subjects both with and without gross myocardial lesions.12

We felt that the findings specifying the degree of association between abnormal Q waves and myocardial infarction in the presence of conduction defects would have more meaning if viewed against a background of their relative association in the absence of conduction defects. We, therefore, undertook a comparable examination of the hearts associated with apparent normal conduction in the standard clinical electrocardiogram.

Methods

Between the years 1961 and 1967, we examined over 1500 hearts by prospective protocol for the distribution of coronary arterial lesions and ventricular myocardial lesions. The hearts of those patients who died on the Medical Service of the Kennedy Veterans Administration Hospital for whom electrocardiograms (ECGs) had been recorded during life were carefully dissected in the fresh state.

As previously described,12 we serially sectioned the coronary arteries noting, on prepared forms, sites and degrees of luminal encroachment, and (after freeing the atria from the ventricles and removing excess fat) we separated and weighed the right and left ventricles (including with the left ventricular mass that of the interventricular septum). We carefully laminated the left ventricle and septum and recorded the anatomic site and distribution of scars, infarction, or, occasionally, metastatic lesions. This correlated file has been examined carefully for instances of abnormal Q waves, defined as those greater than 0.03 sec in width in leads I, V_a, and V_b.

Diagrammatic representation of frequency of localization of lesions in the left ventricular myocardium relative to the occurrence of "normal septal Q waves." The upper left circle illustrates the longitudinal division of the left ventricle (viewed from the apex) along major arterial routes into septal, anterior, and posterolateral inferior thirds and latitudinal division into basal, central, and apical zone with further subdivision in the outer zones to give a total of 15 myocardial sectors. Only those hearts with known myocardial scar or infarct somewhere were included in these groups. In figures 1, 2, and 3 a sector entered by lesions in over one-half the subjects was shaded with horizontal lines; in over three-quarters, with bold dots.

Abbreviations: normal QRS = normally conducted QRS complexes with neither small nor absent septal Q waves; small septal Q = Q waves of 0.5 mm or less in depth and less than 0.03 sec in width in leads I, V_a, and V_b.

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that is 0.04 sec wide, many of them in actual practice "call" the Q wave that is slightly less than the 0.04-sec wide block, but not clearly as short as 0.03 sec.

We excluded the previously mentioned 192 instances of conduction defects and an additional 47 instances of classical electrocardiographic right ventricular enlargement (extreme right axis and peak of the early R' in V1 at 0.07 sec or sooner after the QRS onset). Miscellaneous exclusions were those where the manner of ventricular conduction in the available ECG was in doubt, as with hyperkalemia, ventricular tachycardia, idioventricular rhythm, etc. We included with the group of normal conduction those who had obvious left ventricular hypertrophy or enlargement, but without abnormally prolonged QRS complexes. Of the 1184 remaining examples, 845 had ECGs in which no Q wave was wider than 0.03 sec. As noted in Table I, the examples with ECGs in which no Q wave exceeded 0.03 sec in duration were divided into three groups: those with no septal Q wave—that is, no Q in I, V5, and V6; those with a small septal Q wave—that is, a Q 0.5 mm or less in depth in one or more of the same leads; and finally, those with normal QRS complexes—that is, with the common septal Q present in these "lateral" leads.

The question of localization of infarction was approached in the following fashion. The left ventricular myocardium was divided into 15 zones. As seen in Figure 1 where the left ventricle is represented diagrammatically as a bull's eye with the apex pointed toward and the base away from the viewer, the paths of the major arteries divided the ventricular cone into three major subdivisions: one lying between the left anterior descending and the right posterior descending coronary artery branches was the septal third, one between the left anterior descending and the circumflex artery was the anterior third, and one between the circumflex and right posterior descending artery was the posteroinferior and lateral free wall. These major subdivisions were further subdivided into apical, central, and basal portions, and, except for the apical sectors, a further subdivision was necessary to produce sectors of roughly equivalent area. Study of the pattern of localization was confined to the 416 specimens that had either infarction or scar. For each electrocardiographic group, we tallied the number of entries in a given myocardial sector. Thus, when more than one-half of the subjects with a given electrocardiographic pattern were found to have lesions in a given sector, that sector was shaded with horizontal lines. When more than three-quarters were involved, the sector was marked with bold dots. In none of our groups, was a sector involved in all instances.

Results

Detection of Myocardial Infarction or Scar by Abnormal Q Waves

It can be seen that there is a relatively higher incidence of infarction (slightly less than 1 out of 3) when the septal Q wave is absent than there is when it is present or small

Table 2

<table>
<thead>
<tr>
<th>Major Groupings of Q-wave Distribution</th>
<th>Q &gt; 0.03 sec</th>
<th>V1-2</th>
<th>V1-4</th>
<th>I, V5-6</th>
<th>II, III, aVF</th>
<th>-V1-2*</th>
<th>With MI</th>
<th>Without MI</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Uncomplicated anteroseptal or inferior patterns</td>
<td>Anterior</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td>4</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Septal</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td>18</td>
<td>21</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Anteroseptal</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td>47</td>
<td>26</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Inferior</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td>25</td>
<td>31</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Subtotal</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td>94</td>
<td>80</td>
<td></td>
</tr>
<tr>
<td>II. Lateral and combined patterns</td>
<td>Lateral</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td>8</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Anterolateral</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td>48</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Inferolateral</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td>24</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Inferoseptal†</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td>61</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Anteroinferior plus‡</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td>15</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Inferoposterior</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td>3</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Subtotal</td>
<td>159</td>
<td></td>
<td></td>
<td></td>
<td>6</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total with abnormal Q</td>
<td>253</td>
<td></td>
<td></td>
<td></td>
<td>86</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* - V1-2 indicates presence of widened R in V1V2.
†Include those with abnormal Q in II, III, aVF and either abnormal Q in V1V2 or absent septal Q in I, V5V6.
‡Those with abnormal Q waves in V5V6 and II, III, aVF plus abnormal Q waves in either V1V2 or V5V6—or both.

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(slightly less than 1 out of 7) (table 1). The overall incidence of infarction in the presence of a "normal QRS complex" was slightly under 20%.

It became evident to us that there were two major groupings of Q-wave distribution based on the occurrence of abnormal Q waves in specific electrocardiographic zones. As noted in table 2, we called these two major groupings (I) uncomplicated anterior or inferior Q-wave patterns and (II) lateral and combined Q-wave patterns. Uncomplicated Q-wave patterns were those in which an abnormally wide Q wave was found in only one of the electrocardiographic zones. The lateral and combined Q waves were those in which abnormally wide Q waves were found either in V3 and V6 or in more than one of the classical pair or trio of electrocardiographic leads making up a zone—e.g., (I, aVL), (II, III, aVF), (V1, V2), (V3, V4). One possible exception to this rule for lumping besides putting isolated Q waves in group II is apparent: Q waves in V1-V4 showed the characteristics of isolated Q waves; this is not unexpected, for in most of these, the Q-wave abnormality appeared in leads V2 and V3. Not infrequently, however, a small Q wave also preceded the R wave in V1 and V4.

Q waves which could be classified as uncomplicated anterior or inferior (group I) were not reliably associated with infarction or scar, but those classified as lateral or combined (group II) were very reliable predictors of myocardial deficit. The difference in percentage of false positives was 46% as compared to 4%. Another way of summarizing this difference in reliability would be to say that when abnormal Q waves were found either in V3-V6 or in II, III, and aVF in combination with some other leads, they were ten times more accurate in predicting infarction than when isolated to V1-V4 or II, III, and aVF.

Anatomic Localization of Myocardial Lesions Based on Abnormal Q-wave Patterns

Of the 38 subjects with myocardial lesions and with normally conducted QRS complexes and neither small nor absent septal Q wave, over half had scars in the basolateral sector (fig. 1). This was true also for those with small septal Q waves and those with no septal Q waves at all, except that in each of the latter groups, an inferior or lateral additional section was involved over one-half of the time. It would be unrealistic to conclude that laterobasal infarctions produced normal QRS complexes; it would be more reasonable to conclude that the laterobasal sector is relatively silent and that infarctions in this zone may not deform the QRS complex with characteristic Q-wave abnormalities. Figure 2 graphically illustrates the frequency of distinctive localization for the three patterns of abnormal Q in V1 and V2, V3 and V4, and V1-V4. Three of the small group of four patients with isolated Q waves in V8 and V4 shared extensive damage.

Figure 2

Frequency of localization of lesions in the left ventricular myocardium of hearts associated with electrocardiograms with abnormally wide Q waves in isolated anteroseptal or inferior leads. See figure 1 for convention.

Abbreviations: MIP = myocardial infarction pattern; anterior MIP = abnormal Q waves in V1V4; septal MIP = abnormal Q waves in V3V6; anteroseptal MIP = abnormal Q waves in V1V4; inferior MIP = abnormal Q waves in II, III, aVF.
in the septum and posteroinferior walls, but not in the anterior wall. In 18 patients with "septal infarct" Q waves in $V_1$ and $V_2$, the distribution of the pattern of scar was widely variable except that slightly over half did enter the inferior central septum, whereas, in 47 patients with Q waves in $V_1-V_4$ and myocardial infarction, over one-half of the infarctions entered the apical septal sector. By contrast (fig. 3), in the 48 patients with Q waves in $V_1-V_6$, over 75% had scar or infarction in the apical one-third of the left ventricle extending into the septum and inferior wall, and over one-half showed lesions in the surrounding septal, anterior, lateral, and inferior walls as noted in the diagram. Note the "lateralization" in pattern when just I, $V_5$, and $V_6$ held abnormally wide Q waves.

In figure 3 also are shown the patterns of myocardial localization in the groups with abnormally wide Q waves both in the "footward lead group" (II, III, $aV_R$) and in another lead pair or group. Notice that inferolateral myocardial infarction was well predicted by Q waves in $V_5$ and $V_6$, plus the footward leads. Similar good prediction of infarction sites with II, III, and $aV_R$ alone (fig. 2) was found, but the proviso must be

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recalled that in the particular group of patients under discussion the actual presence of infarction was not in question. Inferoseptal infarction was consistent in localization by its classical pattern or patterns as noted in the diagram (fig. 3).

**Frequency with Which Specific Anatomic Lesions Lead to Certain Electrocardiographic Patterns**

When we received the data from the "forward" point of view (i.e., heart current-to-skin voltage) instead of from the "inverse" approach (i.e., ECG finding-to-heart anatomy), we noted the following points. When the posterolateral third of the left ventricle was the sole site of a lesion, the most common electrocardiographic result was the absent septal-Q pattern and a wide variety of patterns was found. By contrast, when the anterior one-third was the sole site (as occurred less commonly), electrocardiographic patterns were normal, or inferoseptal infarction patterns were found. When both septum and posterolateral wall were damaged, the most frequent electrocardiographic pattern to result was that of wide Q in II, III, and aVF accompanied by loss of normal septal Q in I, V5, or V6. When all three sectors of myocardium were entered, the combined inferior and "anterolateral" pattern was the most frequent result. Figure 4 illustrates these relative distributions of the electrocardiographic sequellae of specific patterns of myocardial damage.

**Discussion**

**Diagnostic Efficiency of the Abnormal Q Wave Alone**

The data in tables 1 and 2 may be reduced to estimates of sensitivity and specificity. Thus, the abnormal Q wave was present 339 times out of 1184 in the total population. It was truly positive 253 times out of 416 in the abnormal population, and truly negative 682 times out of 768 in the noninfarct population. There was thus a sensitivity of 61%, a specificity of 89% averaging to a performance of 75%. Another estimate may be expressed in

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**Figure 4**

The relative frequency of electrocardiographic QRS patterns in the presence of myocardial lesions according to site.

**Abbreviations:** P = posterior = lesions in posterolateral inferior one-third of the left ventricular wall (see fig. 1); A = anterior = lesions in anterior one-third of left ventricular wall; S = septal = lesions in septal one-third of left ventricular wall.
this way: the number of "correct" positive and "correct" negative diagnoses divided by the total number of opportunities. When reduced to percentage, this reflects an absolute diagnostic reliability of the criterion—in this instance, 79%. Comparative estimates for abnormal Q waves in the presence of conduction defects suggested from previous work are 74% for RBBB, 67% for LBBB, and 57% for incomplete LBBB.\textsuperscript{12}

Study of the effect of separating ECGs with diminished septal Q waves from those with otherwise normal QRS complexes can be seen in table 1. No assurance is forthcoming as to the usefulness of predicting infarction from the diminution or absence of the normal septal Q in I, V\textsubscript{5}, and V\textsubscript{6} from these figures. However, it is interesting to note that the relative incidence of hidden infarction was 13% with normal septal Q waves, 14% with small septal Q waves, but 32% when septal Q waves were absent. Thus the clinician's uneasiness upon discovering the septal Q wave absent finds some justification here,\textsuperscript{14} but we were unable to extract individual diagnostic power from the observation.

These findings in the presence of normal conduction may be compared with the findings with conduction defects.\textsuperscript{12} In 72 subjects with LBBB, 28 were without infarction. It is of interest that there were eight instances of Q waves in II, III, and aV\textsubscript{F} with LBBB and proven myocardial infarction, and no instances of this Q-wave abnormality in the group without infarction. By way of contrast, 76 patients with RBBB were found in the series, and of the 40 of these without infarction at autopsy, there were five each with either abnormal Q waves in V\textsubscript{1}—V\textsubscript{4} or abnormal Q waves in II, III, and aV\textsubscript{F}. In each of these instances of false Q wave with RBBB, there was strong clinical evidence of either pulmonary embolism or chronic or acute obstructive lung disease, so that clinicians were provided with a good basis for suspecting the alternative explanation of Q waves of cor pulmonale in these anterior and footward electrocardiographic leads. Finally, there were 44 cases of incomplete LBBB in the study (QRS complexes of greater than 0.10 but not greater than 0.12 sec in the limb leads). Although the incidence of true positive abnormal Q waves associated with infarction was slightly higher than false negative Q waves, diagnostic performance of the Q wave was even poorer than with complete LBBB. The so-called septal Q waves in leads I, aV\textsubscript{L}, V\textsubscript{4}, and V\textsubscript{6} were either small or absent, as would be expected in any complete LBBB in the remainder without abnormally wide Q waves.

Because false abnormal Q waves in the presence of RBBB had been found consistently associated with hearts with large right ventricles,\textsuperscript{12} we searched for similar evidence in the absence of a conduction defect. As shown in table 3, the relative mass of right and left ventricular walls was compared for the three major groupings with and without myocardial infarction. The right ventricular weight was not found to be increased in the group with falsely abnormal Q waves in the isolated right precordial or foot leads, as might have been expected by analogy with the acute cor pulmonale patterns showing RBBB. (Obviously these data cannot exclude physiologic right loading insufficient in degree or duration to produce right ventricular hyper-

\begin{table}
\centering
\caption{Mass of Right and Left Ventricular Walls}
\label{tab:mass}
\begin{tabular}{|c|c|c|c|c|c|}
\hline
 & With MI & LV (g) & RV (g) & Without MI & LV (g) & RV (g) \\
\hline
"Normal" QRS patterns* & 163 & 220 \(\pm\) 83 & 60 \(\pm\) 17 & 682 & 194 \(\pm\) 73 & 56 \(\pm\) 19 \\
Uncomplicated anteroseptal or inferior Q-patterns & 94 & 229 \(\pm\) 73 & 62 \(\pm\) 19 & 80 & 221 \(\pm\) 80 & 62 \(\pm\) 24 \\
Lateral and combined Q-patterns & 159 & 246 \(\pm\) 80 & 67 \(\pm\) 23 & 6 & 220 \(\pm\) 83 & 67 \(\pm\) 17 \\
\hline
\end{tabular}
\begin{flushleft}
*Including ECGs with voltage and ST-T criteria for left ventricular hypertrophy, but not otherwise abnormal as to QRS morphology or duration.
\end{flushleft}
\end{table}

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trophy, as a cause of false but transient Q waves.) Indeed, the only item of interest noted in the comparison of ventricular weights is the finding of greater left ventricular mass in the groups with infarction than in the corresponding groups without infarction, but not to a statistically impressive degree. (The elevated mean right and left ventricular weights for the “normal” QRS patterns without anatomic infarction reflect the inclusion in this group of ECG voltage patterns of left ventricular enlargement but with no other QRS deformity, such as conduction delay. A better inclusive label for “normal” activation in hearts with and without left ventricular enlargement is obviously desirable.)

Accuracy of Localization of Lesion According to Orientation of Abnormal Q Wave

When considering localization as contrasted with absolute detection of infarction, the classical association between abnormal Q waves and regions of myocardium may be summarized as follows. The lateral free wall of the left ventricle is associated with leads I, V5, and V6. Infarction of the inferior or diaphragmatic aspect of the left ventricle is expected to be expressed by abnormal Q waves in leads II, III, and aVF. Lesions of the anterior or apical free wall of the left ventricle are reflected by Q waves in V3 and V4, and of the interventricular septum by Q waves in V1 and V2. Combinations of lesions in more than one myocardial region would be expected to produce Q waves in more than one corresponding electrocardiographic zone. Ordinarily, in the presence of normal conduction, infarction of the septum might also be expected to produce loss of Q wave in the “lateral leads” I, V5, and V6. The addition of true or strict posterior wall infarction to an inferior wall infarction might be expected to produce widening of the R wave in V1 and V2, or increase in the R/S ratio of V1 and V2.

As previously discussed, inspection of figures 1, 2, and 3 provides a portrait of the trends of actual anatomic localization with various combinations of abnormal Q waves in the standard electrocardiogram. Focusing on figure 3, we note that, in general, the words classifying the Q orientation describe the relative left ventricular concentration of lesions. However, we also notice that there are no 100% correlations and that frequent associations are omitted from the prediction: the inferior and anterior overlap is not predicted from the lateral myocardial infarction pattern (MIP) (wide Q in V5V6); the common inferior and septal involvement is not predicted from the anterolateral MIP (Q in V5V6); the septal extent is not predicted from the inferolateral MIP (Q in V3V6 plus Q in II, III, and aVF). It is not clear why the difference of adding Q in V3V4 to the “inferoseptal” combination (Q in II, III, and aVF plus Q in V1 and V2) should shift the basal aspect of localization from septum to lateral free wall but not increase the commonness of lesions in the anterior anterior free wall.

It is worthwhile to comment on the variation in localization seen in conduction defects as compared with these findings. Classical uncomplicated LBBB patterns with autopsy-proven myocardial infarctions showed consistent involvement by scar or infarction in the inferior basal sector near the point where the bundle of His sends over the left bundle and its ramifications. Similarly, in six out of 10 patients with classical patterns of RBBB and no abnormal Q waves, the same sector was involved consistently. Q waves in V1–V4, Q waves in I, V5, and V6, and Q waves in II, III, and aVF consistently predicted appropriate myocardial lesions in over one-half of the patients with known infarction and RBBB, but some paradoxes appeared in LBBB. For example, the only consistent lesion in those patients with Q waves in V1–V4 and LBBB was a scar or infarction in the posterolateral basal sectors. Q wave or Q-equivalent in I, V5, and V6 was associated with extensive lesions in the septum and apex in all seven patients with this pattern, with extension beyond this zone into the central anteroseptal region in five, and into all zones in four. Thus, this particular electrocardiographic pattern could be said to be predictive of septal, lateral, or wrap-around scar or infarction. All eight patients with abnormal Q waves in II, III, and
aVF and LBBB had scars or infarction in the inferior central zone with frequent extension into the surrounding zones to a lesser degree.

Clinical Significance of the Abnormal Q Wave

The clinical implications of these data should be approached with the customary caution to be exercised in translating information from a population sample selected by autopsy to the wider population faced by clinicians. Such provisos are to be considered as: (1) a physiologic event of importance (e.g., sudden right ventricular load, transient ischemic electrical shut-down of a segment of ventricular wall, or transient alteration of conduction in the distal fascicles of the conduction system) may leave no anatomic trace; (2) death may select a relatively higher percentage of these "physiologic abnormalities" than would be encountered among the unselected living. However, if our sampling of false positives and negatives is not drastically inappropriate, then table 2 tells the clinical electrocardiographer that the "cold reading" of abnormal Q waves in leads II, III, and aVF of an isolated uncomplexed electrocardiogram requires a variable response: if present in those leads alone, a causative myocardial infarction or scar is quite possible, but if another lead set also contained abnormal Q waves, it is highly probable. Any further refinement of interpretation requires individual clinical context for which electrocardiographic rules of thumb cannot substitute.

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