Localizaton of the Site of Myocardial Scarring in Man by High-Frequency Components

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SUMMARY
The increased incidence of high-frequency components (notching) in electrocardiographic (ECG) recordings previously has been related to coronary artery disease, primary myocardial disease, and biventricular enlargement.

This study included 130 subjects on whom autopsy permission was obtained, who had one or more sets of high-frequency, orthogonal ECG (XYZ) leads and direct-writing standard ECG leads recorded ante mortem. In each instance careful dissection of the heart was performed by two or more of the authors according to fixed protocol.

Correlations were made between the site of infarction and the occurrence of notching in specific high-frequency ECG leads. Postero-inferior infarctions tended to express themselves with a predominance of increased notching in the Y lead, while anterior infarction manifested dominantly in the X lead. This was true in intramural as well as transmural lesions.

The greatest value of notch recognition in diagnosing and localizing infarction appeared to be in subjects with normal sized hearts, with none of the classic ECG criteria for infarction, but who ultimately proved to have intramural scarring. In these subjects the increased incidence of notching clearly could not be attributed to biventricular enlargement, nor could the ECG diagnosis of infarction be arrived at by conventional criteria.

Additional Indexing Words:
Intramural infarction QRS notching and slurring

The clinical importance of high-frequency notching of the QRS complex in the absence of conduction delay has been the subject of intermittent reports since Langner began his studies in 1952. This work ultimately led to a correlation between high-frequency notching and coronary artery disease.1-8 Burch's group9-11 and Selvester and co-workers12 appreciated the significance of bites and notches in the vectorcardiographic loop (VCG) and related these to overt myocardial infarction as well as to patchy scarring. In a well-controlled computer study, Pipberger and Carter13 attempted to establish the upper limit of normal for the numbers of high-frequency departures in the vectorcardiographic loop.

More recently Reynolds and colleagues14 related high-frequency components to primary myocardial disease. Still more recently, we pointed out with detailed postmortem correlation, the fact that patients with biventricular enlargement also have an extremely high

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incidence of high-frequency components. In fact, patients with biventricular enlargement cannot be separated as a population group from patients with myocardial scarring on the basis of notch count. Furthermore, the groups of patients with either scarring or biventricular enlargement are clearly distinguishable from normal subjects \((P < 0.01)\).15 With epicardial and plunge electrode recordings, Durrer and associates16 demonstrated alteration of conduction pathway around induced scars in the dog’s ventricle. In 1966 Langner17 showed that intramural lesions created in nine experimental animals were expressed in six of them by high-frequency notching of the QRS complex.

Our present study was undertaken to determine whether the particular orthogonal lead (XYZ) with maximal notching reliably pointed to the site of myocardial infarction. Especially were we concerned with whether the incidence of high-frequency components in specific leads indicated the presence and location of intramural myocardial scars otherwise not apparent. Finally, we studied the value of notching or slurning on the downstroke of the R wave in leads II, III, and aVF of the direct-writing ECG as a possible predictor of inferior myocardial infarction.

**Methods**

From 1963 through 1967, on 1,300 male patients admitted to the Kennedy Veterans Administration Hospital, vectorcardiographic loops and scalar leads XYZ of the McFee-axial lead system were obtained.18 At the same sitting, high-frequency ECG leads I, V_{F}, and V_{i} to V_{6} were recorded with a constant V_{4} control for all frames. Direct-writing standard ECGs were also obtained. The high-frequency recordings were obtained by photography from the face of a cathode-ray oscilloscope at sweep speeds of 500 and 200 mm/sec. A minimum of two such sets for each lead was obtained. Follow-up sets were obtained at the time of each subsequent admission. The same XYZ leads were recorded on 36 apparently healthy, male medical students and faculty. The frequency response of the high-frequency system was 0.2 to 10,000 cycles/sec \((\text{cps})\). To be included, a notch or slur would have to appear consistently in two or more QRS complexes at each sweep speed. By the technics described by Langner and associates,5, 6, 19 notches and slurs in the QRS complex were counted in the high-frequency recordings. Notches will be defined as departures in both slope and sign from the primary QRS curve after the artifacts of noise have been excluded. These departures will be exclusive of the fundamental directional changes of the complex; in other words, the nadir of a Q or an S, or the peak of an R. Slurs will be defined as changes of slope without changes of sign. Unless indicated, slurs and notches will be counted together and referred to as “notches.”

The presence of slurking or notching in the latter half of the QRS complex of II, III, aVF or V_{i}, and V_{6} were tabulated from the standard direct-writing ECG (upper frequency response about 70 cps).

At the end of 4 years, 130 subjects had come to autopsy. In each case, two or more of the authors personally performed fresh dissections of each heart according to prospective protocol. The vascular tree was serially sectioned and again opened longitudinally from the ostia distally. The left ventricle and the interventricular septum were separated from the free wall of the right ventricle after complete removal of the fat. The perimeter of the right ventricle was traced and measured planimetrically to establish area, and the right ventricle was weighed separately. The left ventricular free wall and septum were weighed together. Both the free wall and septum were sliced longitudinally at 0.5-cm levels from apex to base to expose the intramural surfaces. The results of the dissections were recorded on protocol sheets with notations of the right ventricular area, right and left ventricular weights, lesions in the coronary arterial tree, and the exact size and site of myocardial lesions. These lesions were photographed in color.

Because of advanced conduction defects, 13 subjects were eliminated from the study. The groups were divided after autopsy into subjects with predominantly anterior infarction, predominantly postero-inferior infarction, biventricular enlargement, left ventricular enlargement, right ventricular enlargement, spotty myocardial infarction, and a group of autopsied subjects with normal hearts, “old normals.” The 36 medical students comprised a nonautopsied group of “young normals.” Comparisons of the relative frequency of notching in each lead within a given group of subjects were tested by Student’s \(t\)-test while comparisons between groups were tested by Cochran’s modification of the \(t\)-test.20

**Results**

In table 1 the lead showing predominant notching in each group is indicated. In the
Table 1

Predominance of Notching in Groups

<table>
<thead>
<tr>
<th>No. of subjects</th>
<th>Lead of predominant notching</th>
<th>X</th>
<th>Y</th>
<th>Z</th>
<th>XY</th>
<th>YZ</th>
<th>ZX</th>
<th>XYZ</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior</td>
<td>Groups with infarction</td>
<td>24</td>
<td>9</td>
<td>5</td>
<td>5</td>
<td>4</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>37.5%</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Posterio-inferior</td>
<td>Groups with no infarction</td>
<td>24</td>
<td>1</td>
<td>13</td>
<td>4</td>
<td>4</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>54.2%</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spotty</td>
<td>17</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>4</td>
<td>3</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>BVE</td>
<td>35</td>
<td>10</td>
<td>12</td>
<td>6</td>
<td>2</td>
<td>3</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>LVE</td>
<td>6</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RVE</td>
<td>5</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normals</td>
<td>42</td>
<td>6</td>
<td>9</td>
<td>15</td>
<td>4</td>
<td>3</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Mean age of normals</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>71 yr.</td>
<td>6</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>25 yr.</td>
<td>36</td>
<td>3</td>
<td>8</td>
<td>15</td>
<td>4</td>
<td>2</td>
<td>4</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: BVE = biventricular enlargement; LVE = left ventricular enlargement; RVE = right ventricular enlargement.

antior myocardial infarction group notching predominates in the X lead while in the postero-inferior group, predominance is in the Y lead. When more than one lead tied for the greatest number of notches, this is indicated. X and Y leads are heavily represented in biventricular enlargement, and in the group with spotty myocardial infarction there is almost an equal distribution through each of the leads. Notching, while less common, predominated in the Z lead in the combined group of normal subjects, though in the smaller subgroup of old normals it predominated in the X lead. It should be emphasized, however, that the autopsied normal subjects can be separated by notch count alone from those with biventricular enlargement, and from infarction subjects with a P value of less than 0.05, and the 36 “young normals” may be separated from the enlargement and infarction groups with a P value of less than 0.01. Thus, notch count remains very low even in the lead of predominance in both categories of normal subjects.15

Figure 1 illustrates the data distribution. It shows the number of notches per lead of each subject for the normal and the infarction groups. The normals tend to show peak distribution at the zero notch count with the count of 3 notches as the maximum for any lead. While there is overlap, the anterior group of infarctions are distributed about a relatively high mean notch count in lead X, while the subjects with postero-inferior infarctions show a high notch count in lead Y and peak at a relatively lower count in lead X. The spotty group is more equally distributed between leads X, Y, and Z.

Table 2 shows the mean notch count for the leads X, Y, and Z (both individually and in combination) for each group. Note the low notch counts in the normal subjects and the groups with single ventricular enlargement. The infarction and biventricular enlargement groups all have high total counts. The predominance of notching in X in the anterior infarction group over the postero-inferior infarction group is significant with a P value of less than 0.05.

Figure 2 shows examples of the standard ECG and XYZ leads of an 89-year-old man obtained 2 months prior to death. Less than 24 hours before death, the ECG was unchanged. None of the classic ECG features of myocar-
The data distribution is illustrated for the normal and infarction subjects. Note that the normals cluster toward the zero end of the scale with 3 notches as the maximal number reached in any lead. The anterior group is distributed about a relatively high mean notch count in X while the postero-inferior group shows a higher count in Y. The spotty group is more equally distributed in X, Y, and Z, and has relatively high counts.

**Table 2**

Lesions by Group with Average Number of Notches per Lead and All Three Leads Combined

<table>
<thead>
<tr>
<th>No. of subjects</th>
<th>Mean no. of notches by lead</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>X</td>
</tr>
<tr>
<td>Anterior</td>
<td>24</td>
</tr>
<tr>
<td>Postero-inferior</td>
<td>24</td>
</tr>
<tr>
<td>Spotty</td>
<td>17</td>
</tr>
<tr>
<td>BVE</td>
<td>36</td>
</tr>
<tr>
<td>LVE</td>
<td>6</td>
</tr>
<tr>
<td>RVE</td>
<td>5</td>
</tr>
<tr>
<td>Normal (old)</td>
<td>6</td>
</tr>
<tr>
<td>Normal (young)</td>
<td>36</td>
</tr>
</tbody>
</table>

Abbreviations same as in table 1.

Dial infarction were present in any set. The strict localization of high-frequency notching in the Y lead, however, pointed to a coalescent, old, intramural postero-inferior infarct, 2.5 by 1 by 0.5 cm, found at postmortem examination.

In figure 3 is an example of an extensive intramural anteroseptal and postero-inferior infarction in a second patient, 66 years old, again with extensive notching in orthogonal leads X and Y, but only a questionably wide Q in X.

In a third patient (fig. 4), the evidence for
LOCALIZATION OF MYOCARDIAL SCARRING

Figure 2

The standard ECG (A) and orthogonal XYZ leads (B) of an 89-year-old man obtained 2 months prior to death. An ECG less than 24 hours before death was identical to its predecessor. Note the absence of classic diagnostic signs of infarction but the localized occurrence of high-frequency notching in the Y lead. At autopsy the patient had a coalescent, old inferior infarct confined to the intramural aspect.

Figure 3

Extensive notching in X and Y without diagnostic Q waves or other signs of infarction. Patient had extensive intramural anteroseptal and intramural postero-inferior scarring.

an anteroseptal myocardial infarct is present as classic QS complexes in V₁ to V₃ in the standard ECG taken the last week of life. However, the only allusion to the inferior scarring also found at autopsy is suggested by the notching noted in the Y lead of the high-frequency recording taken 72 hours before death and possibly by the slurring on the downstroke in direct-writing leads II, III, and aVF. The classic lack of the normal anteriorly directed activation (no Q in Z) reflects the anteroseptal infarction.

Table 3 indicates notch incidence in XYZ leads in intramural infarctions only. This table may be compared with table 2 which includes all groups. Note that in the intramural infarction group notches are frequent and appear in greatest number in the X lead with anterior intramural scars, in the Y lead with postero-inferior intramural scars, and are more equally distributed in X, Y, and Z in the spotty intramural infarctions.

From table 4 it may be noted that slurring and noting of II, III, and aVF in the direct-writing ECG are frequent in inferior myocardial infarction and may predict the information before the more classic signs are manifest.

Table 4

<table>
<thead>
<tr>
<th>Type of Infarction</th>
<th>No. of Subjects</th>
<th>Pre-Q Notching</th>
<th>Post-Q Notching</th>
<th>No Notching</th>
</tr>
</thead>
<tbody>
<tr>
<td>Postero-inferior</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Transmural</td>
<td>13</td>
<td>7</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>Intramural</td>
<td>11</td>
<td>8</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Anterior</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Transmural</td>
<td>17</td>
<td>7</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>Intramural</td>
<td>7</td>
<td>5</td>
<td>0</td>
<td>2</td>
</tr>
</tbody>
</table>

* Pre-Q notching implies the presence of notching or slurring in leads II, III, and aVF prior to other ECG or VCG diagnostic evidence of infarction.
† Post-Q notching includes patients who developed notching only after the development of other classic ECG or VCG criteria of infarction. Patients in whom chronologic relationship between the occurrence of notching and the development of significant Q waves was not known, but in whose recordings both are present, were included in this group.
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frequent however, suggestion, when limited points (A). The absence of a Q wave in Z also points to the anteroseptal lesion (B). The only suggestion, however, of the intramural inferior infarction also found at autopsy is the notch and slurring in II, III, and aVp (A) and the prominent notch in the high-fidelity Y lead (B).

However, the value of this observation is limited when it is realized that notchings are also frequent in one or more of these leads in anterior infarction as well. We do not have a large enough group of autopsied "old normals" to make a valid statement regarding incidence of pre-Q notchings in that group. Notching occurred in eight of the 36 "young normals" who had high-frequency Vp leads run.

Though many correlations were attempted, table 5 summarizes those of greatest significance. In the body of the text certain highly significant correlations previously made are noted.15, 20

Discussion

It seems, then, that there is some localization value in noting the lead of greatest occurrence of high-frequency components. Especially useful is this relationship when a diagnostic Q wave is absent. In the case of nontransmural infarction the only hint may be an increased incidence of notchings in specific leads.

It is logical that the lesions that are more nearly along the Y axis, as in the case of postero-inferior lesions, would manifest increased notchings in Y. Anterior lesions, however, express themselves predominantly in lead X. It is difficult to understand why notchings is not also strongly expressed in the anteriorly oriented Z lead in anterior infarction. Perhaps the reason relates to the fact that while 11 of 24 postero-inferior infarctions were intramural, only seven of 24 anterior lesions were confined to the inner wall. In general, the anterior infarctions were larger than the postero-inferior ones. These two

Table 5

<table>
<thead>
<tr>
<th>Significant Correlations*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Postero-inferior infarction</td>
</tr>
<tr>
<td>Notching in Y &gt; notching in X</td>
</tr>
<tr>
<td>Notching in Y &gt; notching in Z</td>
</tr>
<tr>
<td>X lead notching</td>
</tr>
<tr>
<td>X notching in anterior infarction &gt; than in postero-inferior</td>
</tr>
<tr>
<td>Young normals</td>
</tr>
<tr>
<td>Notching in Y &gt; notching in X</td>
</tr>
<tr>
<td>Notching in Z &gt; notching in X</td>
</tr>
</tbody>
</table>

* Note predominance of Y notchings over X and Z notchings in postero-inferior infarction. X notchings are significantly greater in anterior infarction as compared with postero-inferior infarction. Young normals show notchings predominating in Z and Y over X, but in each case a very small total notch count (average 3.1 notchies for X + Y + Z).

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observations suggest that high-frequency notching in infarction probably originates from areas peripheral to the main coalescent scar in transmural infarction where spottily interrupted conduction may occur. In the larger, and more frequently transmural, anterior lesions these peripheral areas may spill over laterally or along the X axis while the Z axis senses an electrically silent area. In the relatively smaller, more frequently intramural postero-inferior lesions the peripheral or fringe zone of impaired conduction still lies predominantly along the Y axis.

We want to point out again that high-frequency notching in infarction may be simply an earlier stage of Purkinje block as originally described by Oppenheimer and Rothschild, a stage prior to QRS prolongation and detectable only with high-frequency recording technics.15, 21

The lower frequency notching (greater in frequency than the basic QRS but below 100 cps) seen on the descending limbs of direct-writing leads II, III, and aVF are intriguing and seem frequently to accompany infarction, both transmural and intramural. This type of slur or notch of relatively lower frequency than that detectable in a high-frequency system offers some diagnostic help but is of limited aid to localization. This is mainly true because of the high incidence in anterior as well as posterior infarction.

We would like to emphasize the need of a convenient, frictionless recording system above a frequency response of 500 cps and a sweep speed of 250 mm/sec. We believe a source of early diagnostic, prognostic, and screening information may be otherwise untapped.

It appears that Durrer's and later Langner's experimental studies of induced local myocardial injury in dogs may be borne out in the clinical situation; but because high-frequency notching may point to other myocardial abnormalities, rather than to intramural scarring, it is most helpful to have eliminated biventricular enlargement as a cause by roentgenograms, careful history and physical examination.15-17 The greatest clinical fruitfulness in infarction appears to be in diagnosing and localizing scars in the small heart, with intramural lesions and an absence of the usual ECG evidence of infarction. These may have high notch counts not clouded by the increased notch counts expected in ventricular enlargement. In these cases, the leads may offer some pertinent information as to localization of scars as well as to simply announce their presence.

References


Savoring the Thoughts of Writers

... The purpose of reading is not to gobble up the words—even though the reader can afterward itemize his intake from memory—but to experience a mood, to meander through thoughts touched off by an idea, to provide food for the imagination. What makes the present emphasis on rapid reading all the more baleful is that it occurs in the context of an age that worships speed. The consciousness needs the kind of breather the book can provide. It is absurd to say that the speed-readers, if tested, will be able to give everything back. This is not reading; it is regurgitation. What speed-readers too often can't give back is the beauty of a line or the melody of a thought.—From Norman Cousins: Editorial: Books and Transplants. Saturday Review (July 5) 18, 1969.
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