The Anatomic Basis for High-Frequency Components in the Electrocardiogram

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SUMMARY
In a correlative study between selected body-surface potential recordings and anatomic findings, multilead sets of high-fidelity, high-speed records from 128 persons were studied in connection with the results of our detailed postmortem dissections of their hearts. Attention was focused on high-frequency components (notching and slurring of the expanded QRS) as described by Langner.

We found that (1) groups with ventricular enlargement without scarring were indistinguishable from groups with infarction on the basis of the number of high-frequency components; (2) both of these groups were clearly distinguishable from normal subjects on this basis alone; and (3) the high-frequency component count in the group with ventricular enlargement showed high negative correlation with age and high positive correlation with right and left ventricular weights.

These findings suggest that the appearance of high-frequency components in the vectorcardiogram or electrocardiogram may relate to the struggle between competing generator sites of ventricular enlargement as well as to the classic concept of shattering of the wave of activation on the shoals of infarction.

Additional Indexing Words:
Anisotropy Biventricular enlargement First derivative of ECG
High-frequency notching Myocardial infarction QRS notching and slurring
Radial spread of activation

The use of an expanded time scale or increased frequency response to increase electrocardiographic detail has occasionally been reported since the early 1930's. However, it was Langner in the early 1950's who first appreciated that clinically pertinent, otherwise obscure electrocardiographic information might be detected in this fashion. In 1949, Gilford, and in 1960, both Scher and Young and Langner with Geselowitz looked into the frequency spectrum of the electrocardiogram. The latter investigators recognized in 1962 the value of the first derivative of the electrocardiogram. By the derivative's representation of the rate of change of the original electrocardiographic potential with respect to time, notches in the original QRS complex of even very small amplitudes and durations may cause relatively wide swings across the base line of the derivative, thus aiding detection. A number of investigators have in one fashion or another attempted to assign meaning to bites and notches out of...
occurrence of notches and slurs in the high-fidelity electrocardiogram? (2) If notches and slurs do prove to be as frequent in the records of persons without infarction, can we find a direct dependence of the number of notches on a variable such as ventricular mass or age?

Methods

For the purpose of this study notches will be defined as any departure in both slope and sign from the primary electrocardiographic or vectorcardiographic curve after the artifacts of noise have been excluded. Furthermore, these departures will be exclusive of the fundamental directional changes of the QRS complex. For example, the nadir of a Q, the peak of an R, or nadir of an S would not be included. Slurs will be defined as changes of slope without changes of sign (fig. 1). Except where indicated notches and slurs will be summed.

On 1,300 male patients admitted to Kennedy Veterans Administration Hospital, McFee-axial system\textsuperscript{18} vectorcardiograms in both scalar (XYZ leads) and loop form as well as electrocardiographic leads I, V_{6}, and V_{1} to V_{5} were recorded at the time of each admission by means of Polaroid photography from the face of a cathode ray oscilloscope. Sweep speeds were at 500 and 200 mm/sec. V_{4} was constantly recorded as a control lead.

Figure 1

High-speed, high-fidelity scalar leads (XYZ) of the axial vectorcardiogram (VCG) and leads I, V_{f} and V_{1} to V_{5} of the electrocardiogram (ECG) from a patient with both biventricular enlargement and a large apical myocardial infarction. Note the prominent notching (n) and slurring(s) in both XYZ and V_{4} to V_{5}. This is particularly prominent in V_{4} and V_{5}. Notching represents both change in slope and change in sign (direction) while slurring represents change in slope alone. Primary directional changes are not counted, for example, peak of R.

the vectorcardiographic loop\textsuperscript{11–13} and Pipberger and Carter\textsuperscript{12} in a well-controlled study, set the limits of normal.

Except for Reynolds and co-workers\textsuperscript{14} in a recent study of patients with primary myocardial disease, most of the emphasis previously has been placed on a relationship between coronary artery disease and the appearance of notches and slurs of frequencies higher than that of the basic QRS complex\textsuperscript{5, 15–17}.

Because most of the previous studies through no fault of the investigators were without benefit of anatomic correlation, we set about to ask the following questions: (1) Can subjects with autopsy-proven myocardial infarction be distinguished as a population group from normals or from people with other forms of heart disease (without anatomic evidence of myocardial scarring) on the basis of the

Figure 2

Scalar leads of a 69-year-old man with posterior-inferior myocardial scarring. Note the relatively smoother QRS complexes when compared with figures 1 and 4. In lower left corner direct writer recordings are shown with residual q waves of old infarction.

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Surface potential from each site was recorded at both sweep speeds and a minimum of two such sets for each lead was obtained. At the time of each repeated admission, follow-up sets were obtained. Identical leads were recorded on 36 healthy male medical students. The frequency response of the system was 0.2 to 10,000 cycles/sec. Notches and slurs were counted by two independent observers using the technics of Langner, Geselowitz, and Mansure. The totals represent the mean of the sums of notches and slurs in XYZ and V1 to V5.

At the conclusion of 4 years, 128 of the original 1,300 patients had expired in the hospital and autopsy permission had been obtained. Two or more of the authors performed fresh dissections of the hearts. The coronary tree was serially sectioned and again opened longitudinally from the ostium distally. The right ventricle was dissected free from the interventricular septum, and after removal of the fat was measured planimetrically and weighed. Fat was removed from the left ventricular free wall which was then weighed with the septum. Both the left ventricular free wall and septum were sliced longitudinally at 0.5-cm levels from apex to base. The results were recorded on special protocol sheets with notation of right and left ventricular masses, right ventricular area, lesions in the coronary arterial tree, and the exact site and size of the lesions in the myocardium. These lesions were also photographed in color. Thirteen subjects were eliminated from the study because of advanced conduction defects.

Results

In the course of study we developed a strong impression that notching and slurring

![Figure 3](image-url)
Scalar leads of a 47-year-old man with rheumatic aortic insufficiency and left ventricle weighing 578 g. Right ventricle weighed 100 g. No myocardial scarring in spite of prominent notching (see text).

occurred at least as frequently in recordings from subjects with large, uninfarcted hearts as in recordings from those with myocardial scars or fresh infarctions. Two extreme examples follow: In figure 2 note the relatively smooth QRS complex in the scalar leads of this subject who had postero-inferior myocardial scarring seen in figure 3. In the electrocardiogram of a 47-year-old man with longstanding aortic insufficiency of rheumatic origin, however, notching is evident in many leads (fig. 4). Ventricular weights were excessive, the right ventricle weighing 100 g and the left weighing 578 g. Coronary arteries were large and wide open without evidence of even minimal atherosclerosis.

We set about to see if these impressions held up under statistical analysis. The criteria for ventricular enlargement were based upon our own work involving over 2,000 dissections and several autopsy studies reported by Saphir. The right ventricular upper limit of normal was assumed to be 50 g, while the left ventricle and septum were considered enlarged if they exceeded 180 g. The all-male groups represented in the bar graph (fig. 5) include 36 healthy medical students of mean age 25 years ("young normals"), and six persons with a mean age of 71 who had died from other than cardiac causes in whom no evidence of scarring or enlargement was found ("old normals"). These have been graphed separately for reasons which will become obvious.

In the ventricular enlargement group there were 46 subjects, with a mean age of 59. Hearts were included in this group if they (1) fulfilled the weight requirements and (2) had no gross evidence of myocardial scarring of any size. Though not a pre-set criterion for being included, no occlusion of any of the coronary arteries through the third

| Principal Etiologies of Heart Disease in Subjects with Ventricular Enlargement |
|---------------------------------|--------|
| Systemic hypertension           | 18     |
| Pulmonary                       | 5      |
| Rheumatic                       | 6      |
| Infections                      | 1      |
| Primary or secondary myocardial disease | 13 |
| Unclear                         | 3      |

The mean of the sums of notches in X, Y, Z, and V1 to V4 (along ordinate) of the three autopsied groups on right and the 36 young normals, left. Standard error is graphed. The number of subjects in each group is noted along abscissa. The ventricular enlargement and infarction groups were indistinguishable, but both were distinct from young normals: P < 0.01 and old normals: P < 0.05.

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Figure 6

Total number of notches from each subject (XYZ plus V₁ to V₅) plotted against subject's own combined ventricular weights. Note positive correlation in enlargement groups but less in infarction group illustrated by regression lines. The very high correlation (0.97) in the old normals was not plotted due to small sample.

Table 2

Comparative Correlations When Notching in XYZ and V₁ to V₅ were Analyzed Separately in the Noninfarction and the Infarction Groups

<table>
<thead>
<tr>
<th></th>
<th>Bivariate (r)</th>
<th>Multivariate (t)</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Age</td>
<td>RVM</td>
</tr>
<tr>
<td>Notches</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No infarct</td>
<td>-0.42</td>
<td>0.42</td>
</tr>
<tr>
<td>Infarct</td>
<td>-0.11</td>
<td>0.14</td>
</tr>
<tr>
<td>V₁-V₅</td>
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<td></td>
</tr>
<tr>
<td>No infarct</td>
<td>-0.48</td>
<td>0.26</td>
</tr>
<tr>
<td>Infarct</td>
<td>-0.04</td>
<td>-0.00</td>
</tr>
</tbody>
</table>

Note: The numbers in the three bivariate analysis columns on the left are correlation coefficients as is the final column under multivariate analysis. Note that composite correlation exceeds any partial correlation for a group. The first three columns in the multivariate section on the right are all t values; those in italics are significant with P < 0.05 and the value in bold face is significant with P < 0.01.

Note greater dependence on right ventricular mass for V₁-V₅ notching in the noninfarction group. Left ventricular mass shows the highest correlation with notching in XYZ in the noninfarction group. Inverse age correlates well with notching in both XYZ and V₁-V₅ for the noninfarction group.

Note lack of correlations in the infarction group.

Branching was noted in any of the enlargement group. The cardiac diagnoses of this group are noted in table 1 by etiology.

In the infarction group there were 63 subjects with a mean age of 64. Hearts with diffuse or spotty, or tiny and single scars
were included as well as those with coalescent large scars. All had either complete occlusion or marked atherosclerotic narrowing of one or more branches.

By Cochran’s approximations of Student’s t-test, the myocardial infarction group and the ventricular enlargement group were each clearly distinguishable from both groups of normal subjects with $P < 0.01$ in the case of the “young normals,” and $P < 0.05$ in the case of the “old normals.” The infarction and enlargement groups, however, were indistinguishable from each other. It is obvious, then, from the graph that the “old normals” had more notches than the “young normals,” but the most notches were seen in the enlargement group, with the infarction group following very closely.

Does the number of notches depend on age or ventricular mass? The answer is yes under certain circumstances, as illustrated in figure 6. Note that in the noninfarction group there is dependency of notch frequency on total ventricular mass as indicated by the regression line ($r = 0.35$), but in the infarction group, this dependency appears to be lacking, with an $r$ value of 0.09. In the enlargement (“old normal”) group the dependency of notching on weight was striking ($r = 0.97$), but the sample was small. When the variables age, left ventricular mass, and right ventricular mass were each allowed to act singly as the
Table 3

<table>
<thead>
<tr>
<th>Notches</th>
<th>Bivariate (r)</th>
<th>Multivariate (t)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Age</td>
<td>RVM</td>
</tr>
<tr>
<td>No infarct</td>
<td>-0.47</td>
<td>0.39</td>
</tr>
<tr>
<td>Infarct</td>
<td>-0.10</td>
<td>0.04</td>
</tr>
</tbody>
</table>

Note: High negative correlation with age and positive correlation with right and left ventricular masses in the noninfarction groups. Correlations are much lower in the infarction group.

The greater dependency on right ventricular mass for notching in the precordial leads is thought to relate to the proximity effect of $V_1$ and $V_2$ especially to the enlarging right ventricle. The vectorcardiogram, by its very nature a more perfect vectorial averager, favors the expression of the naturally dominant left chamber. We suspect the explanations for left ventricular dependency for notching in the vectorcardiogram might be so related.

What then are the casual possibilities for notching in general (table 4)? The Purkinje block, suggested by Oppenheimer and Rothschild in 1917 to explain interruption of the smooth progress of the wave of activation, still remains a tenable explanation in certain cases. The alteration of the power spectrum in acute infarction with ultimate resolution with healing, as suggested by Franke and associates, may well be due to functional block in reversibly injured tissue. The work of Langner and Geselowitz relating the number of notches to clinical coronary disease and Reynolds' relationship of notching to primary myocardial disease point to probable structural interruption of the activation wave of poorly conductive tissue. But the

Table 4

Hypotheses as to Notch Formation in the ECG

1. Classical shattering of the wave of activation on the zone of infarction
2. Departure from even radial spread of activation through the ventricular wall due to
   a. Microinfarction
   b. Functional microblock
3. Accentuation of 2 by ventricular hypertrophy (competing generator sites)
4. Relationship of anisotropy to above
striking occurrence of the most notches in patients with ventricular enlargement from many etiologies other than atherosclerotic suggests, at the least, multiple causation.

From whatever cause, when the normally smooth, radial spread of activation from the endocardium outward is interrupted, and more tangential, less efficient detours are taken, distortion of the QRS curve results. Anisotropy of propagation in the case of the myocardial syncytium implies differences in conduction along the long axis as compared to conduction across the muscle fiber. We would like to suggest tentatively that the anisotropic properties of the myocardium may be accentuated and compounded by many structural or functional changes. More practically, we may presuppose a number of necessary deviations in the activation pathway, but in the presence of ventricular hypertrophy the powerful pull of the generator in either ventricle tends to accentuate any unevenness of the spread of activation in the alternate ventricle.

The detection of high-frequency notching may prove to be a screening device for heart disease of any etiology leading to biventricular enlargement, as well as for classical atherosclerotic heart disease.

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References


Types of Critics

The capacity of public somnolence to retard change illuminates the role of the critic. In the early years of this century Abraham Flexner touched off a revolution in medical education by placing before the public a brilliant exposé of existing medical schools. Critics who call attention to an area that requires renewal are very much a part of the innovative process. (Of course, all critics are not heralds of the new. Some are elegant connoisseurs of that which has arrived, and when they approve of something it is likely to be long past its creative period. Like Hermes conducting the souls of the dead to Hades, they usher ideas and art forms into the mausoleums of “the accepted.”) — John W. Gardner: Self-Renewal: The Individual and the Innovative Society. New York, Harper & Row, Publishers, 1963 and 1964, p. 30.
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