Editorial Comment

Stretching the Limits of the Electrocardiogram’s Diagnostic Utility*

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It cannot be disputed that the wide-spread use of noninvasive imaging techniques, in particular echocardiography, has greatly diminished the role of the electrocardiogram (ECG) in the assessment of the anatomic characteristics of cardiac chambers. From the vantage point of scientific evolution this was an unavoidable development.

The hold of the ECG on the diagnosis of cardiac chamber enlargement and hypertrophy has always been tenuous, dependent largely on the empirical correlations with autopsy and angiographic findings, but lacking a fundamental theoretical foundation. As long as the noninvasive methodology of sizing cardiac dimensions was limited to the physical examination and the flat chest X-ray, the ECG was capable of supplementing valuable input into the qualitative assessment of cardiac anatomy. This contribution was probably most evident in the diagnosis of left ventricular hypertrophy (LVH), a subject of countless studies striving to devise the most accurate, empirically derived diagnostic criteria translated into amplitudes in single leads or amplitude sums in several leads supplemented by the manifestations of the secondary repolarization changes. In one of the more recent studies (1), the ECG criteria for LVH were reanalyzed on the basis of correlations with echocardiography. A multiple logistic regression equation developed in the learning series in this study was tested prospectively and achieved a formula yielding a sensitivity of 51%, a specificity of 90%, and an overall accuracy of 76% (1). The dominant result of this and other similar studies is the tradeoff between the sensitivity and the specificity, a dilemma ubiquitous to other problems in the medical practice.

Echocardiographic studies have exposed the shortcomings of the ECG in its ability to differentiate between concentric hypertrophy, eccentric hypertrophy and dilation without hypertrophy (2). Echocardiography also has shown that in the diagnosis of asymmetric LVH, the presence of prominent abnormal Q waves attributed to septal hypertrophy is a poor predictor of increased septal thickness (2). As the ECG has failed in all contests against the imaging techniques measuring the chamber size and the wall thickness, the only lame excuse for defending the use of the ECG in the assessment of cardiac dimensions is the ECG’s lower cost relative to the echocardiogram. Yet would it not be more economically prudent to lower the cost of the superior product than to rely on the application of a cheaper but inferior one?

From a practical point of view no investigator who hopes for grant support will resort to electrocardiography in a proposal to study the effect of hypertension therapy on left ventricular and septal wall thickness or left ventricular mass, that is, parameters that are accurately measured by the echocardiogram.

Electrocardiographers should feel relieved for being absolved of the ungrateful tasks of making qualitative correlations between the electrical activity on the body surface and the anatomical parameters of the heart. This is not the task of electrocardiography even though one does not need to dismiss the empirical value of anatomic, pathologic and epidemiologic associations gathered in careful correlative studies made over nearly a century. An electrocardiographer can still contribute to the assessment of cardiac problems related to cardiac anatomy, pathology and differential clinical diagnosis while yielding to other techniques that can measure more accurately the anatomical parameters of the heart.

In acknowledging the imperfections of the ECG in its anatomical correlation, one should ask whether the ECG can perform more successfully in the assessment of the ventricular pumping function. In this issue of the Journal, Murkoñsky et al. (3) propose that “[I]t would be clinically useful and cost effective if the standard 12-lead ECG could be used to predict left ventricular dysfunction,” and proceed to correlate the QRS duration with the left ventricular ejection fraction (LVEF) at rest. Before discussing the results of this study, it may be in order to review briefly the methods and the results of measuring QRS duration in the normal adult population.

In 1952, Lepeschkin and I (4) examined the methodology of the QRS measurement and drew the following conclusions. First, differences in the apparent and real duration in different leads exist. If the lead axis is perpendicular to the initial or terminal vectors of the QRS complex, the respective parts of the complex will be isoelectric, and a complex may begin later and/or end earlier by as much as 20 ms. In the precordial leads the QRS duration was found to be longer than in the limb leads; in our material the beginning occurred up to 20 ms earlier in the lead V2 than in the limb leads. Second, the variability in successive complexes in the same lead was about 3 ms and was attributed to respiratory changes of the heart position and therefore to the differences in the projection of the QRS vector loop on a certain lead. Third, the end of the QRS complex is difficult to determine as there is no sharp demarcation of potential variations caused by ventricular repolarization near the end of the QRS complex, that is, there is a gradual transition between the QRS and the ST-segment. This is a source of intraobserver and interobserver differences.

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QRS duration was 65 (average 35) years, and found that in 21% of these subjects available recording cart in 1,254 white healthy men ages 19 to 82 years which tended to be lower in the larger individuals. We found that the increase of QRS duration from the lowest to the highest body size was 44%. We also took into consideration the effect of heart rate, which tended to be lower in the larger individuals. We found that the increase of QRS duration from the highest to the lowest heart rate was 23%, whereas the increase from the lowest to the highest body size was 44%. More recently, Selvester et al. (6) measured the QRS duration on a digital 12-simultaneous lead ECG by a commercially available recording cart in 1,254 white healthy men ages 19 to 65 (average 35) years, and found that in 21% of these subjects QRS duration was >100 ms and <120 ms, and that in 14 subjects QRS duration of normal morphology was 120 ms or longer. In this study, the 98% upper and lower bounds of QRS duration was 80 to 116 ms, with a peak of 96 ms. In a smaller group of women, the 98% bounds were 72 to 104 ms. The results of the above studies suggest that: 1) the QRS duration may differ in different leads (a finding corroborated in subsequent studies); 2) accurate measurement of the end of QRS complex may be difficult; 3) the QRS duration is longer in men than in women; 4) QRS duration appears to correlate with cardiac size. Systematic studies of QRS duration in patients with different types of cardiac pathology are not available to my knowledge, and are not listed in the references of Murkofsky et al. (3).

These investigators correlated QRS duration measured by the computer and manually confirmed on the 12-lead ECG at a paper speed of 25 mm/s with the LVEF obtained by radionuclide ventriculography. Of the 270 patients referred for the test during the study period, 44 were excluded because of bundle branch block (BBB), atrial flutter or fibrillation, pacemaker rhythm, recent myocardial infarction or treatment with antiarrhythmic drugs. The LVEF was <45% (abnormal) in 80 patients and >45% (normal) in 146 patients. Patients with abnormal LVEF were older, included a greater percentage of men (i.e., 68.8% vs. 51.4%) and had a greater incidence of ischemic heart disease. The QRS duration in the group with abnormal LVEF was significantly longer than in those with the normal LVEF (i.e., 102 ms vs. 91 ms) (p < 0.0001). Prolonged QRS duration defined as >0.10 s was found in 43.8% of patients with abnormal and in 16.4% of those with normal LVEF. With increasing QRS duration the specificity for the prediction of abnormal LVEF increased, and the sensitivity declined. At QRS duration >0.12 s, specificity was 99.3% and sensitivity was 13.8%, and at QRS duration >0.10 s, the specificity was 83.6% and the sensitivity was 43.8%. Murkofsky et al. (3) also measured the R score (defined as the sum of R waves [in milliVolts]) in leads aVL, aVF and V1-6. Patients with an abnormal LVEF had a significantly lower R-wave score. The combined criteria of R score of <4 mV and QRS duration of >0.10 s had a 15.3% sensitivity and 99% specificity for the prediction of decreased LVEF, but the two criteria did not correlate.

Are these results surprising? No and yes. Patients with left ventricular dysfunction, aside from being predominantly men in this study, may be expected to have an increased QRS duration even in the absence of a complete BBB. The increase in QRS duration may be caused by LVH, incomplete LBBB or other intraventricular conduction disturbances, including the peri-infarction block.

The surprising finding was the lower R score in patients with abnormal LVEF. Intuitively, one would expect greater incidence of LVH and thus a higher R score in patients with abnormal LVEF. The results opposite from those expected are open to a variety of speculations, one of which is that the results are fortuitous and probably will not be reproducible in another study because of the absent correlation between the QRS duration and the R score in this study. In any event, a pursuit of this problem is likely to end in a blind alley.

A more relevant question is whether one should heed the conclusion of Murkofsky et al. (3) that “larger studies may be warranted to assess the association between the severity of left ventricular dysfunction and the QRS duration.” It would be improper for me to advocate curtailling the scientific curiosity generated by this study. Another study as well designed as that of Murkofsky et al. (3) with statistically significant results has a good chance of being accepted for publication and could provide information which deserves to be stored in the crowded memory bins of the assorted trivia. But what is the practical value of such observations? How likely is it that the electrocardiographer will attempt to extrapolate the results of LVEF from the measured QRS duration, or that the nuclear cardiologist will attempt to derive the QRS duration from the measurement of LVEF? Unlikely, I venture to say.

An electrocardiogram is a record of electrical, not mechanical, activity and must be used to provide information that is unique to the electrical activity of the heart. Fortunately, this is a very large domain which includes the patterns of depolariza-
tion and repolarization, and the expressions of automaticity, refractoriness, excitability, vulnerability and injury currents, to mention some of the facets of normal and abnormal cardiac electrophysiology. Stretching the application of the ECG beyond the limits of its capability does not serve well either electrocardiography or clinical cardiology.

References