Critical Value of the Electrocardiogram in LVH: From Predictive Index to Therapeutic Reassessment

Letter to the Editor:

Although QRS voltages for left ventricular hypertrophy (LVH) are widely recognized as an independent factor for coronary artery disease (CAD), little information is available on reversion of ST-T alterations in hypertensive subjects with electrocardiographic (ECG) criteria for LVH.

Two recent articles in Hypertension by Fagard et al.1 and Schneider et al.2 presented the regression of QRS voltages in treated hypertensive subjects without evidence of improvement on ventricular repolarization abnormalities. The first study, the Systolic Hypertension in Europe (Syst-Eur) Trial based on nitrendipine therapy to which enalapril or thiazide could be added, showed that serial 1 mV increase in QRS voltage predicts cardiovascular morbidity and mortality, whereas a −1 mV regression was associated with lower cardiovascular events, without mention of the ST-T alterations. In the second study, the Cardiovascular Irbesartan Project, the reduction of QRS voltage at 6- and 18-month periods was more significant with irbesartan as compared with atenolol, but no precise information was given on the ST-T changes (see Tables 2 and 4 in the article). It is, therefore, uncertain whether all of these therapeutic measures failed to improve the ventricular repolarization abnormalities or if the investigators failed to report them. This is a critical point because QRS voltage may decrease with the lowering of blood pressure irrespective of derangement of coronary blood supply relative to LV mass. In fact, ST-T changes may precede, occur simultaneously with, or appear after the increase in QRS voltages,3 whereas the T wave inversion may carry an elevated risk for ischemic heart disease, whether in conjunction with ST depression or increased QRS voltages.4

Such “minor” alterations, often discarded as clinically meaningless and receiving little attention, provide some explanation for the increasing number of CAD reported in hypertensive subjects despite improved BP control.5 In this context, we have recently documented how these ST-T alterations in hypertensive subjects with LVH or CAD can be improved or reversed in a significant number of subjects within the first 6 months of treatment, when closer attention is given to each ECG abnormality.6

In general, these uncompleted reports on ST-T changes in hypertensive subjects with ECG-LVH1,2 probably result from the use of single QRS voltages criteria, as in the Syst-Eur trial or by the misconception that only “classical” LV strain pattern in V5, V6 is important. It should be remembered that the earliest ECG criteria by Sokolow-Lyon (1949)7 or DW Romhilt and EH Estes Jr (1968)8 included both QRS voltage with any ST segment shifts opposed to mean QRS and diphasic or inverted T wave, and that the classical LV strain pattern was included as ECG score for patients on digitalis. It is thus surprising that after several reports from the Framingham Heart Study3–9 on the predictive value of ECG-LVH (QRS voltage, ST segment shift, flat or inverted T waves), the prognostic significance of ECG-LVH is universally related to the increased QRS voltages. Other reports, such as LIFE study patients,10 included typical LV strain but on ECG recorded at a 1-year interval. In this context, a policy of obtaining ECG recordings on a more regular basis (ie, yearly in the absence of ST-T changes, but every 3 months if ST-T minor changes appear) can certainly contribute to an improved ECG diagnosis of subclinical heart disease in hypertensive patients and, more importantly, to a critical reassessment of the therapeutic measures in these patients with LVH.

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