Inter-Relationship Between Electrocardiographic Left Ventricular Hypertrophy and QT Prolongation as Predictors of Increased Risk of Mortality in the General Population

Elsayed Z. Soliman, MD, MSc, MS; Amit J. Shah, MD; Andrew Boerkircher, DO; Yabing Li, MD; Pentti M. Rautaharju, MD, PhD

Background—Prolonged-QT commonly coexists in the ECG with left ventricular hypertrophy (ECG-LVH). However, it is unclear whether to what extent QT prolongation coexisting with ECG-LVH can explain the prognostic significance of ECG-LVH, and whether prolonged-QT coexisting with ECG-LVH should be considered as an innocent consequence of ECG-LVH.

Methods and Results—The study population consisted of 7506 participants (mean age, 59.4±13.3 years; 49% whites; and 47% men) from the US Third National Health and Nutrition Examination Survey. ECG-LVH was defined by Cornell voltage criteria. Prolonged heart-rate–adjusted QT (prolonged-QTa) was defined as QTa≥460 ms in women or 450 ms in men. Cox proportional hazards analysis was used to calculate the hazard ratios with 95% confidence intervals for the risk of all-cause mortality for various combinations of ECG-LVH and prolonged-QTa. ECG-LVH was present in 4.2% (N=312) of the participants, of whom 16.4% had prolonged-QTa. In a multivariable-adjusted model and compared with the group without ECG-LVH or prolonged-QTa, mortality risk was highest in the group with concomitant ECG-LVH and prolonged-QTa (hazard ratio, 1.63; 95% confidence interval, 1.12–2.36), followed by isolated ECG-LVH (1.48; 1.24–1.77), and then isolated prolonged-QTa (1.27; 1.12–1.46). In models with similar adjustment where ECG-LVH and prolonged-QTa were entered as 2 separate variables and subsequently additionally adjusted for each other, the mortality risk was essentially unchanged for both variables.

Conclusions—Although prolonged-QT commonly coexists with LVH, both are independent markers of poor prognosis. Concomitant presence of prolonged-QT and ECG-LVH carries a higher risk than either predictor alone. (Circ Arrhythm Electrophysiol. 2014;7:400-406.)

Key Words: electrocardiography ■ left ventricular hypertrophy ■ prolonged QT

Prolonged-QT interval and electrocardiographic left ventricular hypertrophy (ECG-LVH) have been associated with adverse outcomes, including all-cause mortality, coronary heart disease, stroke, and sudden cardiac death.1–4 Experimental studies have demonstrated that ECG-LVH could alter ventricular conduction and repolarization,6–9 which could be reflected as prolonged-QT interval in the resting ECG. Several studies on clinical populations also have shown the common concomitant presence of prolonged-QT and ECG-LVH in patients with hypertension and hypertrophic cardiomyopathy.10–14

Clinical Perspective on p 406

Because of their common coexistence, some studies have suggested that consideration of QT prolongation may improve the otherwise relatively low sensitivity of LVH detection by ECG criteria alone,12,13 and concomitant QT prolongation with LVH has been proposed as a potential explanation for the increased risk of cardiac death in patients with LVH.14–16

This inter-relationship between prolonged-QT and ECG-LVH, however, has not been thoroughly examined in the general population. It is also unclear whether QT prolongation in the presence of ECG-LVH should be considered as an innocent consequence of ECG-LVH, and whether the prognostic significance of ECG-LVH could be partially explained by the concomitant presence of QT prolongation.

We examined the prevalence of concomitant presence of prolonged-QT among those with ECG-LVH in the Third National Health and Nutrition Examination Survey (NHANES-III). We also evaluated the inter-relationship between prolonged-QT and ECG-LVH in terms of their association with all-cause mortality.
Methods

NHANES is a periodic survey of a representative sample of the civil-
nian noninstitutionalized US population aimed to determine estimates
of disease prevalence and health status of the US population. The
National Center for Health Statistics of the Center for Disease Control
and Prevention Institutional Review Board approved the protocol
for NHANES-III. All participants gave written informed consent.
Participant characteristics, ECG methodology, and ascertainment
of mortality in the NHANES-III Have been published elsewhere.17
Briefly, NHANES-III baseline data were collected during an in-home
interview and a subsequent visit to a mobile examination center from
1988 to 1994. The data collected during the in-home interview in-
cluded demographics and medical history, including smoking status
and the use of medications. Blood pressure data were the averaged
reading from 3 in-home measurements and 3 mobile center measure-
ments. Hypertension was defined as systolic and or diastolic blood
pressure >140/90 mm Hg or taking blood pressure–lowering drugs.
Using the height and weight measured during the visit to mobile ex-
amination center, the body mass index was calculated as the weight
in kilograms divided by the height in meters squared and obesity was
declared as body mass index >30 kg/m2. Diabetes mellitus was de-
defined as fasting plasma glucose ≥126 mg/dL, a nonfasting plasma
-glucose ≥200 mg/dL, or concurrent use of antidiabetic medications.
Dyslipidemia was defined as self-reported history of high cholesterol
or concurrent use of lipid-lowering drugs.

Study Population

The present analysis includes 7506 participants, who had good-
quality ECGs showing sinus rhythm and no major intraventricular
conduction delay (including complete bundle branch blocks, Wolf
Parkinson–White Syndrome, and QRS duration ≥120 ms) and avail-
able mortality data, medical history, medication use, and anthropo-
metric measurements.

Electrocardiography

Standard 12-lead ECG was recorded using a Marquette MAC 12 sys-
tem (Marquette Medical Systems, Milwaukee, WI) by trained techni-
cians during the visit of participants to a mobile examination center.
Computerized automated analysis of the electrocardiographic data
was performed with visual inspection of outlier values by a trained
ECG technician in a central ECG core laboratory.

QT interval was measured from global QRS–STT complex derived
from the standard 12-lead ECG as the interval from the QRS onset to
the T-wave offset (end). We used a linear formula for rate-corrected
QT as recommended by a task force sponsored by professional or-
manizations.16 Specifically, we set up a linear regression model with
QT interval as the dependent variable and heart rate interval as the
independent variable. On the basis of β-coefficient associated with
heart rate, the following formula was derived for heart-rate–adjusted
QT (QTa): QTa=QT+2.05×(heart rate–60). The application of this
formula in the study population effectively made the QT interval
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those without ECG-LVH was 2.30 (95% confidence interval, 1.66–3.19). Prolonged-QTa was present in 7.0% (N=528) of the participants, of whom 9.7% had ECG-LVH. There was a weak correlation between Cornell voltage and QT interval (r=0.10; P<0.01).

Table 1 shows characteristics of participants stratified by QT prolongation and ECG-LVH status. As shown, participants with both prolonged-QTa and ECG-LVH were more likely to be older, women, nonwhites, and have hypertension, diabetes mellitus, history of CVD, obesity, and prolonged QRS duration when compared with other groups, especially those without ECG-LVH or prolonged-QTa.

During a median follow-up of 13.8 years, 2569 deaths occurred at a rate of 26.9 deaths per 1000 person-years. The mortality rate was highest among participants with concomitant ECG-LVH and prolonged-QTa (58.2 deaths per 1000 person-years) and was the least among those without ECG-LVH or prolonged-QTa (24.8 deaths per 1000 person-years). However, the mortality rate was relatively similar in those with isolated ECG-LVH (47.7 deaths per 1000 person-years) and isolated prolonged-QTa (49.0 deaths per 1000 person-years; Figure 1). Figure 2 shows Kaplan–Meier survival curves for concomitant ECG-LVH and prolonged-QTa, isolated QT, and isolated ECG-LVH when compared with those for no ECG-LVH or prolonged-QTa.

Table 2 shows the results of Cox proportional hazard analysis, where combinations of ECG-LVH and prolonged-QTa were used as a 4-level variable (concomitant ECG-LVH and prolonged-QTa, isolated ECG-LVH, isolated prolonged-QTa, and no ECG-LVH or prolonged-QTa [reference group]). When compared with no ECG-LVH or prolonged-QTa, concomitant ECG-LVH and prolonged-QTa was associated with 2.5× the risk of mortality, whereas isolated ECG-LVH and isolated prolonged-QTa were associated with approximately twice the risk (P<0.01 for all). After adjustment for demographics, CVD risk factors, and potential confounders, the risk of mortality remained the highest in the concomitant ECG-LVH and prolonged-QTa group (63% increased risk; P<0.01), followed by isolated ECG-LVH (48% increased risk; P<0.01), and then isolated prolonged-QTa hazard ratio (27% increased risk; P<0.01; Table 2). Similar direction of the results was observed when we examined the association between different combinations of ECG-LVH and prolonged-QTa with mortality in subgroups of NHANES-III participants stratified by age, sex, race, hypertension, history of CVD, and obesity with no significant interactions between the components of each subgroup (Table 3). Also similar results were obtained when we used other ECG-LVH criteria, such as Sokolow–Lyon (results not shown).

Table 4 shows the risk of mortality associated with each of ECG-LVH and prolonged-QTa using separate unadjusted and adjusted models. As shown, prolonged-QTa and ECG-LVH, entered separately in different sets of models, were associated with almost double the risk of mortality, which remained significantly high in the multivariable-adjusted models. More importantly, the strength of association between ECG-LVH and prolonged-QTa with mortality was not significantly attenuated when both were entered in the same model as 2 separate variables. As shown in Table 4, the hazard ratio for ECG-LVH and prolonged-QTa group (63% increased risk; P<0.01), followed by isolated ECG-LVH (48% increased risk; P<0.01), and then isolated prolonged-QTa hazard ratio (27% increased risk; P<0.01; Table 2). Similar direction of the results was observed when we examined the association between different combinations of ECG-LVH and prolonged-QTa with mortality in subgroups of NHANES-III participants stratified by age, sex, race, hypertension, history of CVD, and obesity with no significant interactions between the components of each subgroup (Table 3). Also similar results were obtained when we used other ECG-LVH criteria, such as Sokolow–Lyon (results not shown).

![Figure 1](http://circep.ahajournals.org/)

**Figure 1.** Mortality rate for various combinations of ECG-left ventricular hypertrophy by Cornell Voltage criteria (ECG-LVH) and prolonged-heart-rate–adjusted QT interval (QTa).
only attenuated from 1.46 to 1.44 after further adjustment for prolonged-QTc in the multivariable adjusted. In a similar model, the hazard ratio for prolonged-QTc only attenuated from 1.27 to 1.26 (Table 4).

There was no significant interaction between prolonged-QTc and ECG-LVH as predictors for mortality (interaction \( P=0.489 \)).

### Discussion

We examined the inter-relationship between prolonged-QTc and ECG-LVH using data from the NHANES-III, which enrolled a representative sample from the US general population.

Our study revealed 3 main findings. First, prolonged-QTc commonly coexists with ECG-LVH; \( \approx 1 \) in 6 (16.4%) of our study participants with ECG-LVH had concomitant prolonged-QTc. Second, the magnitude of association between prolonged-QTc and mortality was not significantly attenuated after adjustment for ECG-LVH. Similarly, the magnitude of association between prolonged ECG-LVH and mortality was not significantly attenuated after adjustment for prolonged-QTc. These findings suggest that prolonged-QTc and ECG-LVH are 2 independent predictors of mortality, and the prognostic significance of one does not depend or explained by the other. Third, concomitant presence of prolonged-QTc and ECG-LVH carries a higher risk for mortality than the presence of each in isolation. This means that QT prolongation in the presence of ECG-LVH should not be considered as an innocent consequence of ECG-LVH, which is another finding supporting the independent prognostic information that could be obtained from each marker.

The relationship between LVH and prolonged-QT has a rational biological basis. In the hypertrophic myocardium, multiple pathological changes occur, such as myocardial fibrosis, myocyte hypertrophy, cell death, and disturbance in neurohormonal regulation. All of these pathological changes have an important effect on QT prolongation. Studies in human patients and animal models have demonstrated that cardiac hypertrophy significantly affects myocardial cell-to-cell coupling, leading to disturbances in action potential duration which subsequently can lead to potential malignant arrhythmia and sudden cardiac death. This explains our results and the results from previous studies, showing that ECG-LVH is commonly associated with QT prolongation. Other studies have also found that prolonged-QT in the setting of LVH denotes poor prognosis. In the Oregon Sudden Unexpected Death study, prolonged-QTc was significantly associated with sudden cardiac death in a case–control analysis using data from 158 subjects with ECG-LVH. Similarly, QT prolongation was a predictor of mortality in patients with hypertension and ECG-LVH in the Losartan Intervention for End point Reduction in Hypertension (LIFE) Study.

<table>
<thead>
<tr>
<th>Table 2. Hazard Ratios and 95% Confidence Intervals for All-Cause Mortality by ECG-LVH and Prolonged-QTc Status</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ECG-LVH</strong></td>
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<td><strong>Model 1</strong></td>
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<td>Absent</td>
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</table>

CI indicates confidence interval; ECG-LVH, ECG-left ventricular hypertrophy by Cornell Voltage criteria; HR, hazard ratio; and QTc, heart-rate–adjusted QT interval.

*Model 1, unadjusted.
†Model 2, adjusted for age, sex, and race.
‡Model 3, additional adjustment for diabetes mellitus, hypertension, dyslipidemia, obesity, smoking, history of cardiovascular disease, QT-modifying drugs, and QRS duration.
### Table 3. Hazard Ratios and 95% Confidence Intervals for All-Cause Mortality by ECG-LVH and Prolonged-QTa Status in Subgroup Analysis

<table>
<thead>
<tr>
<th>ECG-LVH</th>
<th>Prolonged-QTa Interval</th>
<th>Hazard Ratio</th>
<th>Hazard Ratio 95% Confidence Limits</th>
<th>( P ) Value for Interaction</th>
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<tbody>
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<td>0.83–3.40</td>
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<td>Absent</td>
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<td>1.16–1.63</td>
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<td>1.51</td>
<td>0.67–3.39</td>
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<td>1.03–2.38</td>
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<td>Present</td>
<td>1.73</td>
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</table>

Model adjusted for age, sex, race, diabetes mellitus, hypertension, dyslipidemia, obesity, smoking, history of cardiovascular disease, QT-modifying drugs, and QRS duration. ECG-LVH indicates ECG-left ventricular hypertrophy; QTa, heart-rate–adjusted QT interval; and reference group, no LVH or prolonged-QTa.
this is in agreement with our finding that LVH does not confound or interact with prolonged-QT as a predictor of death. Notably, the magnitude of risk in individuals with concomitant prolonged-QT and ECG-LVH in our study (63% increased risk in the fully adjusted model) was not much different than the sum of the risk associated with isolated prolonged-QT (27% increased risk) and isolated LVH (48% increased risk). This higher risk associated with concomitant prolonged-QT and ECG-LVH when compared with each one in isolation also suggests the independence and the additive prognostic information that could be obtained from ECG-LVH and prolonged-QT.

Our results should be read in the context of certain limitations. Sudden cardiac death is an adverse outcome that has been extensively examined in relationship to ECG-LVH and prolonged-QT. Because sudden cardiac death is not ascertained in NHANES, we could not use it as an outcome in our study. Nevertheless, this study is intended as a proof of concept; hence, a hard outcome, such as mortality, leaves little or no room for ascertainment bias. In contrast, a matter of debate has always been when an unexpected death should be called sudden and how the cardiac origin of the death should be ascertained.27 This could cause significant misclassification that could risk the credibility of the results and its replication in other studies.

Another limitation of our study which stems from the design of NHANES-III is the lack of echocardiographic data, which could be used to obtain LV mass. However, our focus on the inter-relationship between QT prolongation and ECG-LVH when compared with each one in isolation also suggests the independence and the additive prognostic information that could be obtained from ECG-LVH and prolonged-QT.

Conclusions

Prolonged-QT commonly coexists with ECG-LVH. However, both are totally independent and additive markers of poor prognosis. Hence, prolonged-QT in the setting of LVH should not be considered as an innocent consequence of LVH; instead both LVH and prolonged-QT should be evaluated and considered separately, despite their common concomitant coexistence. These findings could have potential applications in risk stratification and identifying individuals at high risk for poor outcomes.

Sources of Funding

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Disclosures

None.

References

Prolonged-QT intervals commonly coexist in the ECG with left ventricular hypertrophy (ECG-LVH). However, it is unclear to what extent QT prolongation coexisting with ECG-LVH can explain the prognostic significance of ECG-LVH, and whether prolonged QT coexisting with ECG-LVH should be considered as an innocent consequence of ECG-LVH. This analysis from the US Third National Health and Nutrition Examination Survey shows that prolonged QT and ECG-LVH are totally independent and additive markers of poor prognosis. Hence, prolonged QT in the setting of LVH should not be considered as an innocent consequence of LVH; instead, both LVH and prolonged QT should be evaluated and considered separately, despite their common coexistence.
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