Correlation of Precordial Voltages to Left Ventricular Mass on Echocardiogram in Adolescent Patients With Hypertrophic Cardiomyopathy Compared With that in Adolescent Athletes

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Electrocardiograms continue to be part of screening programs for athletes and familial hypertrophic cardiomyopathy (HC). Whether electrocardiographic (ECG) findings of left ventricular (LV) hypertrophy can distinguish between healthy populations and those with HC remains unclear. We sought to (1) analyze the relation between ECG voltage and LV mass in patients with HC and (2) evaluate ECG characteristics of patients with phenotypical HC. Retrospective cohort of patients with HC aged 13 to 18 years. Relation between ECG voltages (RV_6, SV_1, and $RV_6 + SV_1$) and echocardiogram measurements of LV mass was investigated using smoothing splines to display relations and compared with those in a prospectively obtained population of adolescents. Frequency of abnormal LV voltages and nonvoltage ECG changes (Q waves, T-wave changes, and ST changes) were analyzed for association with HC. Fifty-three patients with HC (72% men) were age and gender matched to 104 control patients. Smoothing splines demonstrated that parabolic rather than linear relations existed between LV mass and SV_1, RV_6, and $RV_6 + SV_1$ in patients with HC and not the control cohort. LV hypertrophy by ECG voltage criteria was present in 34% of patients with HC and associated with poor sensitivity (29%). In patients with HC, 56% demonstrated nonvoltage ECG abnormalities and were associated with improved sensitivity (68%) and high specificity (94%). In conclusion, there is a parabolic relation between LV voltages and LV mass in adolescents with HC that may lead to "pseudonormalization." Voltage abnormalities were associated with poor sensitivity, whereas nonvoltage criteria were associated with improved sensitivity with high specificity. **(Am J Cardiol 2015;115:956–961)**

Hypertrophic cardiomyopathy (HC) is the most common genetic heart disease in the United States, with a predilection for sudden cardiac death, particularly in young athletes.[1] In efforts to identify at-risk patients, a number of screening processes have been proposed.[2–5] Echocardiography offers noninvasive assessment of left ventricular (LV) size, wall thickness, and outflow obstruction. Electrocardiograms are assessed for excessive LV voltage, ST-segment changes, and abnormal Q waves. Although echocardiograms and electrocardiograms are part of the diagnostic workup for HC, there is conflicting evidence regarding the correlation of electrocardiographic (ECG) voltages to echocardiogram measurement of LV mass in pediatric patients with HC. Previous studies suggest that electrocardiogram has low sensitivity and high specificity in relation to LV mass on echocardiogram.[6–8] Other studies suggest that extended ECG LV voltage criteria, including measures of Q waves and combination of voltage criteria, are better correlated to LV mass on echocardiogram than standard measurements of R waves in precordial leads.[9,10] Whether ECG findings of LV hypertrophy can be used to distinguish between healthy population and those with HC remains unclear. The purpose of this study was to (1) analyze the relation between ECG voltage and LV mass in adolescent patients with HC, (2) evaluate ECG testing characteristics of patients with phenotypical HC, and (3) determine whether ECG characteristics can be used to differentiate between patients with phenotypical HC and adolescent athlete controls.

Methods

This was a retrospective cohort study of pediatric patients evaluated for HC. This study was approved by the Cincinnati Children's Hospital Internal Review Board (study# 2013-4304). Consecutive adolescent patients aged 13 to 18 years with a history of HC from January 1, 2003, to June 30, 2013, were included. An electrocardiogram, echocardiogram, and genetic testing were part of the evaluation. Genetic testing was completed between 2008 and 2012. Patients with a gene positive family history of HC but without genetic screening specific to them were included in analysis if LV hypertrophy was present on echocardiogram.

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See page 961 for disclosure information.

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Table 1
Demographics of study patients and controls at time of echocardiogram

	Phenotype negative (n=12)	Phenotype positive (n=40)	Controls (n=104)	*p* value
Age (years)	16 ± 2	16 ± 2	16 ± 2	0.12
Male	5 (42%)	33 (83%)	76 (73%)	0.24
White	11 (92%)	31 (78%)	96 (92%)	0.02
Weight (kg)	69 ± 19	88 ± 30	68 ± 16	0.0003
Body Surface Area (m2)	1.8 ± 0.2	2.0 ± 0.3	1.8 ± 0.2	0.003
Left Ventricular Mass (gm)	122 ± 41	238 ± 119	146± 37	<0.0001
Gene positive	12	22 (54%)	*N/A*	*N/A*

Data expressed as mean +/- standard deviation. The phenotype negative group is comprised of genotype positive but phenotype negative hypertrophic cardiomyopathy patients. The phenotype positive group includes the following subgroups: genotype positive/phenotype positive, genotype positive but phenotype negative, and genotype unknown but phenotype positive. p-values represent the comparison between the phenotype positive group and controls.

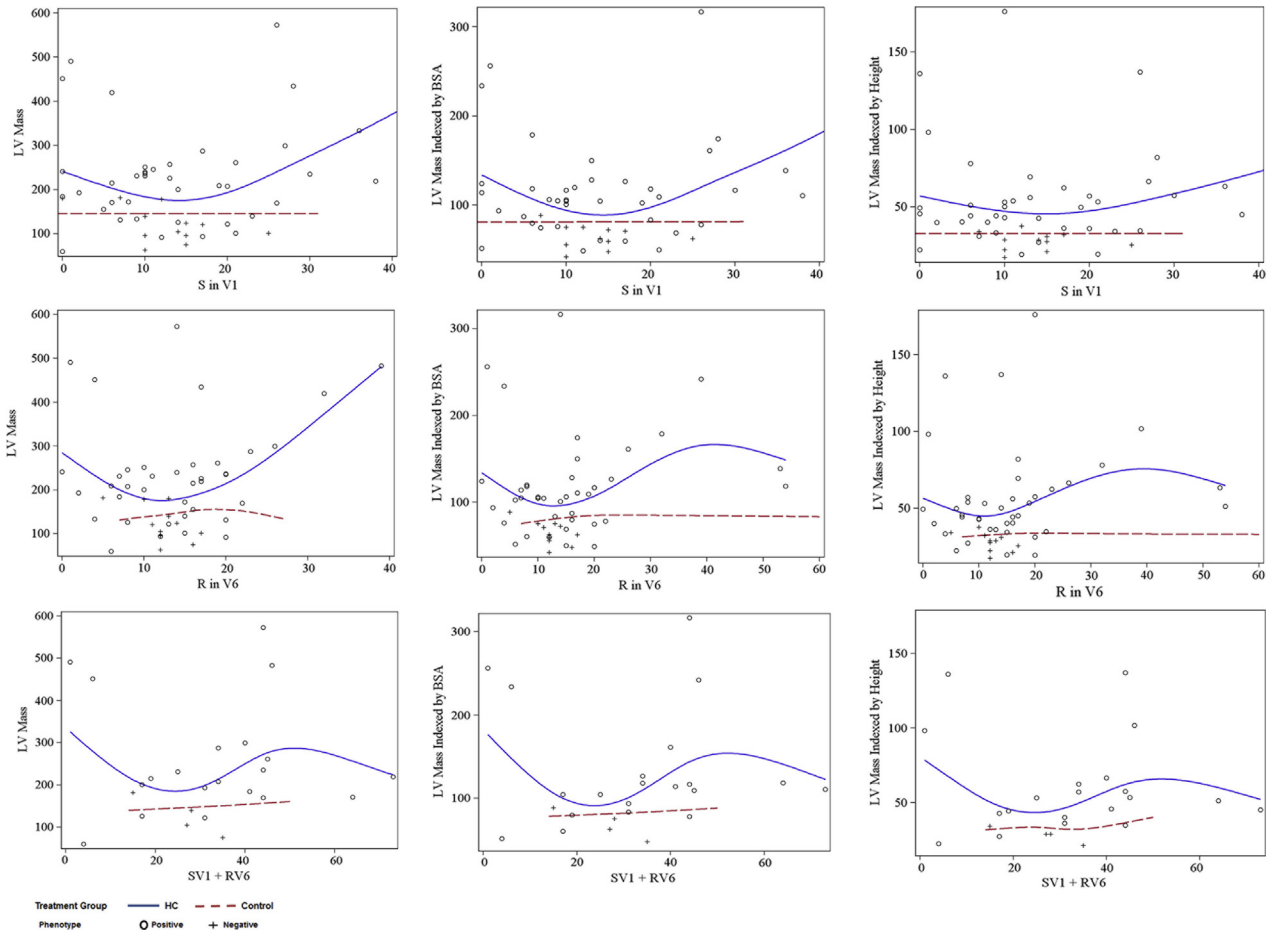

Figure 1. Scatterplot of electrocardiographic LV precordial voltage in relation to LV mass on echocardiogram. *Circles* depict phenotype-positive patients with HC. *Plus signs* depict phenotype-negative patients with HC.

Each patient's most recent electrocardiogram and echocardiogram completed within 14 days of each other up to age 18 years were included in analysis.

Patients with a known clinical history of hypertension, aortic stenosis, subaortic membrane, Friedreich ataxia, muscular dystrophy, LV noncompaction, and glycogen storage disease were excluded as these conditions may lead to secondary increased wall thickness and LV hypertrophy. In addition, patients without genetic testing and no LV hypertrophy on echocardiogram were excluded from the study.

Relation between ECG voltages (R-wave V_6, S-wave V_1, and R-wave V_6 + S-wave V_1) and echocardiogram measurements of LV mass, LV mass per square-meters, and LV mass/height$^{2.7}$ was investigated. These were compared with measurements in a prospectively obtained population of adolescent athlete controls who underwent sports screening.[11] Patients in the screening study were aged 13 to 18 years and underwent screening with echocardiogram and electrocardiogram. The frequency of abnormal LV voltages, as well as nonvoltage ECG changes that serve as components of the Seattle Criteria