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Computerized Q wave dimensions in athletes and hypertrophic cardiomyopathy patients☆

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Abstract

Background: There is controversy regarding Q wave criteria for assessing risk for hypertrophic cardiomyopathy (HCM) in young athletes.

Methods: The 12-lead ECGs from Preparticipation screening in healthy athletes and patients with HCM were studied retrospectively. All 12 leads were measured using the same automated ECG analysis program.

Results: There were a total of 225 HCM patients and 1124 athletes with 12-lead electrocardiograms available for analysis. Athletes were on average 20 years of age, 65% were male and 24% were African–American. Patients with HCM were on average 51 years of age, 56% were male and 5.8% were African–American. Q waves by either amplitude, duration or area criteria were more prevalent in males than females, in lateral leads than inferior and in HCM patients than athletes. The most striking difference in Q waves between the groups was in Limb lead I and in the females. Tall, skinny Q waves were rare in athletes and had the highest prevalence of only 3.7% in male HCM patients.

Conclusion: Q waves are more common in males compared to females and in patients with HCM compared to athletes. Q waves of 30 ms or more in limb lead I appear to offer the greatest discriminatory value for separating patients with HCM from athletes.

Keywords: Athlete; Screening; Electrocardiography; Hypertrophic cardiomyopathy

Introduction

One of the leading causes of early sudden cardiac death (SCD) in athletes is hypertrophic cardiomyopathy (HCM) [1], an inherited heart condition characterized by pathological thickening of the heart wall [2]. Since current pre-participation athletic screening recommendations in the US include only family history and physical examination [3], which have limited sensitivity and specificity [4], there is a clear need for improvement. Routine electrocardiogram (ECG) pre-participation screening of competitive athletes has been proposed to identify athletes at risk of SCD due to HCM but controversy exists in the guidelines as to appropriate Q wave criteria [5–7]. Even Q wave criteria for myocardial infarction, a different pathological process, have evolved from the classic 40 ms and more than 25% of the following R wave [8].

No previous study has compared computer Q wave measurement from athletes to those from patients with HCM. We hypothesized that direct comparison of digital ECGs using the same computerized algorithm applying various cut points in patients with HCM and young athletes, will demonstrate the optimal criteria for identifying Q waves associated with HCM in young athletes.

Methods

Participants

Consecutive athletes referred to the Stanford Sports Medicine program from June 2010 to July 2013 for pre-participation screening signed informed consent approved by the Stanford Investigational Review Board and underwent 12-lead computerized ECG recording (Cardea Associates, Seattle, WA). Data were collected and saved at a sample rate of 1000 samples/s and a resolution of ±0.5 μV. The data were sub-sampled to 500 samples per second for analysis. The athletes referred for screening included Stanford Collegiate participants, National Football and Basketball League players

☆ Disclosures: Victor Froelicher and David Hadley, Co-owners, Cardea Associates (Seattle, Washington).

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Table 1
Demographic data (mean ± SD; n (%)).

	HCM (n = 240)	Athletes (n = 1123)	p
Age	50.9 ± 16.4	20.4 ± 3.8	<0.001
Males	134 (56)	728 (65)	0.008
Ethnicity			<0.001
Caucasian	197 (82)	716 (64)	
African–American	14 (5.8)	269 (23.9)	
Asian	23 (10)	73 (7)	
Hispanic	3 (1)	35 (3)	
Other	3 (1)	31 (2.8)	
Heart rate (HR)	70.7 ± 16	62 ± 11	<0.001

and local high school athletes. Basic demographic information was collected at the time of ECG recording and race was self-reported. Need for follow-up testing was determined by the clinical team performing the pre-participation screen based on physical exam, electrocardiogram and clinical history. None of our cohort had been diagnosed with HCM. All athletes at Stanford (Football, basketball, volley ball and swimming) had screening Echocardiograms and of the professional athletes have had rest and exercise Echocardiograms within a 2 year window of the ECG.

Between 2006 and 2013, all patients with a clinical diagnosis of HCM followed at the Stanford Center for Inherited Cardiovascular Disease who had undergone digital 12-lead ECG recording (Phillips Healthcare, Andover, MA) at Stanford were enrolled. Patients with HCM received a clinical diagnosis based on the presence of a hypertrophied, non-dilated left ventricle in the absence of other primary causes of left ventricular hypertrophy [9]. Demographic information and medical histories were obtained from medical records review. Electrocardiograms performed while pacing, after myectomy, left bundle branch block or that contained atrial arrhythmias were excluded.

ECG processing

Digital electrocardiograms from the clinical and athlete populations were processed using the same software (Cardea Associates, Seattle, WA). Computer measurements were

Table 2

Q wave Measurement Cut Points	Athletes, male (N = 728)					
	II	aVF	I	V4	V5	V6
N (%)						
<−300microV (3 mm)	1 (0.1)	1 (0.1)	0 (0)	6 (0.8)	11 (1.5)	7 (1.0)
<−350 (3.5 mm)	0 (0)	1 (0.1)	0 (0)	3 (0.4)	5 (0.7)	3 (0.4)
<−400 (4 mm)	0 (0)	0 (0)	0 (0)	1 (0.1)	2 (0.3)	1 (0.1)
>30 msec	12 (1.7)	12 (2.9)	12 (1.7)	3 (0.4)	17 (2.3)	36 (5.0)
>40 msec	0 (0)	1 (0.1)	0 (0)	0 (0)	0 (0)	1 (0.1)
Area >10000	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
	HCM, male (N = 134)					
	II	aVF	I	V4	V5	V6
<−300microV (3 mm)	5 (3.7)	4 (3.0)	7 (5.2)	3 (2.2)	5 (3.7)	5 (3.7)
<−350 (3.5 mm)	4 (3.0)	4 (3.0)	5 (3.7)	3 (2.2)	5 (3.7)	4 (3.0)
<−400 (4 mm)	2 (1.5)	3 (2.2)	3 (2.2)	3 (2.2)	5 (3.7)	4 (3.0)
>30 msec	8 (6.0)	13 (9.7)	17 (12.7)	6 (4.5)	8 (6.0)	13 (9.7)
>40 msec	4 (3.0)	5 (3.7)	4 (3.0)	3 (2.2)	4 (3.0)	6 (4.5)
Area >10000	2 (1.5)	3 (2.2)	1 (0.8)	2 (1.5)	2 (1.5)	2 (1.5)
	Athletes, female (N = 395)					
	II	aVF	I	V4	V5	V6
<−300microV (3 mm)	1 (0.3)	2 (0.5)	0 (0)	0 (0)	2 (0.5)	0 (0)
<−350 (3.5 mm)	0 (0)	2 (0.5)	0 (0)	0 (0)	0 (0)	0 (0)
<−400 (4 mm)	0 (0)	1 (0.3)	0 (0)	0 (0)	0 (0)	0 (0)
>30 msec	4 (1.0)	6 (1.5)	1 (0.3)	2 (0.5)	12 (3.0)	10 (2.5)
>40 msec	0 (0)	0 (0)	1 (0.3)	0 (0)	1 (0.3)	1 (0.3)
Area >10000	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
	HCM, female (N = 106)					
	II	aVF	I	V4	V5	V6
<−300microV (3 mm)	4 (3.8)	4 (3.8)	3 (2.8)	3 (2.8)	5 (4.7)	4 (3.8)
<−350 (3.5 mm)	2 (1.9)	4 (3.8)	2 (1.9)	3 (2.8)	2 (1.9)	4 (3.8)
<−400 (4 mm)	1 (0.9)	4 (3.8)	0 (0)	2 (1.9)	2 (1.9)	4 (3.8)
>30 msec	5 (4.7)	12 (11.3)	6 (5.7)	2 (1.9)	3 (2.8)	5 (4.7)
>40 msec	3 (2.8)	7 (6.6)	3 (2.8)	2 (1.9)	2 (1.9)	1 (0.9)
Area >10000	1 (0.9)	4 (3.8)	0 (0)	1 (0.9)	1 (0.9)	1 (0.9)

The shaded areas indicate that lead I with a duration greater than 30 msec and any amplitudehas a highly significant ($P < 0.001$) percentage difference between the HCM patients and the athletes. difference between the HCM patients (10-19%) and the athletes (2-5%)