Contents lists available at ScienceDirect



Computers in Biology and Medicine



journal homepage: www.elsevier.com/locate/cbm

Effect of aerobic conditioning on ventricular activation: A principal components analysis approach to high-resolution electrocardiogram



O. Nasario-Junior ^{a,1}, P.R. Benchimol-Barbosa ^{b,*}, G.A. Trevizani ^{a,1}, M. Marocolo ^{c,2}, J. Nadal ^{a,1}

^a Laboratório de Processamento de Sinais, Programa de Engenharia Biomédica, COPPE, Universidade Federal do Rio de Janeiro, PO box: 68510, Rio de Janeiro, RJ 21941-972, Brazil

^b Hospital Universitário Pedro Ernesto, Universidade do Estado do Rio de Janeiro, Boulevard Vinte e Oito de Setembro, 77, Board of Directors Suite, 20551-030 Rio de Janeiro, RJ, Brazil

^c Postgraduating Program in Physical Education and Sports, Universidade Federal do Triângulo Mineiro, Av. Tutunas, 490, Uberaba, MG 38061-500, Brazil

ARTICLE INFO

Article history: Received 24 May 2013 Accepted 7 September 2013

Keywords: Maximal aerobic power High-resolution electrocardiogram Ventricular activation Cardiac remodeling Intra-QRS patterns Principal component analysis Physical conditioning Athlete's heart

ABSTRACT

Background: The athlete's heart represents a reversible structural and functional adaptations of myocardial tissue developed through physical conditioning. Surface electrocardiogram (ECG) has the capability to detect myocardial hypertrophy but has limited performance in monitoring physical conditioning-induced myocardial remodeling. The aim of this study was to develop an ECG-derived test for detecting incipient myocardial hypertrophy in well-conditioned athletes based on a principal components (PC) analysis.

Methods: Two groups of study composed of 14 sedentary healthy volunteers (Control group) and 14 professional long distance runners (Athlete group) had their maximal metabolic equivalents (MET) estimated (mean \pm SD: Control group: 9 \pm 2 METs vs. Athlete group: 20 \pm 1 METs, *p* < 0.05). All participants had their high-resolution ECG (HRECG) recorded, and a 120 ms segment starting at the QRS complex onset and ending in the ST segment was extracted to build a data matrix for PC analysis. The Mahalanobis distance was evaluated by a logistic regression model to determine the optimal separation threshold between groups. HRECG was also analyzed using the classical time domain approach. The comparison of areas under the receiver operating characteristic curve (*c*-statistic) in 10,000 bootstrap re-samplings measured how well each method detected physical conditioning (α < 0.05).

Results: Average bootstrap c-statistic for PC analysis and time domain approaches were 0.98 and 0.79 (p < 0.05), respectively. PC analysis and maximal oxygen consumption exhibited comparable performances to distinguish between groups.

Discussion: The PC analysis method applied to HRECG signals appropriately discriminates well-conditioned athletes from healthy, sedentary subjects.

© 2013 Elsevier Ltd. All rights reserved.

1. Introduction

Regular aerobic exercise provides beneficial changes to the cardiovascular system [1,2], being characterized by mechanical, autonomic, and electrophysiological remodeling. Mechanical remodeling, usually related to aerobic exercise training, is characterized by mild to moderate increases in left ventricular mass and volume [3–6]. Autonomic remodeling is observed in reduced resting heart rates and increases in cardiac autonomic modulation [7–12]. Additionally, electrical changes are characterized by the

isamjf@gmail.com (M. Marocolo), jn@peb.ufrj.br (J. Nadal).

redistribution of electrical charges on myocardial surfaces, as indicated by increases in both ventricular activation amplitude and repolarization times, which are observed in the resting electrocardiogram waveforms of athletes [13–17].

While mechanical remodeling is more directly related to cardiovascular fitness, electrical remodeling during periods of increased fitness activity has been less commonly investigated, most likely because of methodological limitations [18]. Thus, an increase in the maximum aerobic power in healthy subjects has been shown to be related to an increase in left ventricular mass [19].

Marocolo et al. (2007) demonstrated a positive correlation between the estimated VO_{2MAX} and the high frequency amplitude of ventricular activation, which was derived from the high-resolution electrocardiogram (HRECG). This finding indicates a potential application of this method for studying cardiovascular fitness [11].

In a recent study, to detect intra-QRS high-frequency potentials, Nasario-Junio et al. (2010) proposed a method for unfiltered

^{*} Corresponding author. Tel.: +55 21 28688100; fax: +55 21 28688101. *E-mail addresses:* olivasse@hotmail.com (O. Nasario-Junior), ecgar@yahoo.com (P.R. Benchimol-Barbosa), gabytrevizani@yahoo.com.br (G.A. Trevizani),

¹ Tel.: +55 21 2562 8577.

² Tel.: +55 34 3318 5504.

^{0010-4825/\$ -} see front matter © 2013 Elsevier Ltd. All rights reserved. http://dx.doi.org/10.1016/j.compbiomed.2013.09.006

HRECG signal assessment based on a principal components (PC) analysis [20]. PC analysis is a statistical technique employed to study data variability and provides a privileged data set view [21]. PC analysis is performed on a database composed of data segments with equivalent time durations extracted from several synchronized signals [22]. The morphological classification of cardiac waveforms based on PC analysis, in which a subset of components serves as indices employed to detect intra-QRS high-frequency transients, can be used to identify greater energy potentials alongside ventricular activation.

The purpose of this study was to investigate the PC analysis of unfiltered HRECG in athletes and healthy sedentary subjects such that intra-QRS transients can be assessed as markers that correlate electrical remodeling to physical fitness.

2. Methods

2.1. Study population

The analyzed signals were extracted from an existing HRECG database as described previously [11]. The study protocol was approved by the National Institute of Cardiology Ethics Committee, and informed consent was obtained from each volunteer. Fourteen elite runners ([mean \pm SD] 8.9 \pm 3.2 years of training; 6–8 training sessions/week; 90–120 min/session; 90–110 km/week) were enrolled (Athlete group).

A group of 14 healthy volunteers were included as controls (Control group). Inclusion criteria required an age between 18 and 40 years old, good mental and physical health, and no previous history of cardiovascular disease. Both groups had identical gender distribution. None of the study subjects had a history of lower limb musculoskeletal injury diseases that could affect results. Additionally, all participants met the following criteria: (i) no intake of nutritional supplements or potential ergogenic aids of any type (e.g., exogenous anabolic androgenic steroids); (ii) non-smokers; (iii) normal blood pressure; (iv) non-diabetic; (v) no history of alcohol addiction; (vi) no history of thyroid dysfunction; (vii) not taking medications that affect cardiac electrical properties and (viii) familiarization with the exercises used in the evaluation. The subjects participating in the present study were slightly younger, but had similar anthropometric characteristics and gender distribution as those in the studies used as references in the sampling procedure (Table 1) [13,23].

2.2. Group separation

Subjects had their maximal oxygen consumption (VO_{2MAX}) estimated by the Cooper 12 min field test (Table 1) and calculated

Table 1

Anthropometric and demographic characteristics (mean \pm SD) of the subjects who participated in the study.

	Controls	Athletes
Age (years)	28.6 ± 4.7	24.5 ± 6.4
BSA (m ²)	1.8 ± 0.2	1.8 ± 0.2
APTD (cm)	20.9 ± 2	21.2 ± 1.1
LLTD (cm)	28 ± 3.3	28.1 ± 1.4
EAHFP (degree)	51.9 ± 12.3	44.7 ± 9
VO _{2MAX} (METs)	8.6 ± 2.1	$19.6\pm1.4^{^\circ}$

Data are reported as means \pm SD for 14 subjects in each group. BSA=body surface area; APTD=anteroposterior thoracic diameter; LLTD=laterolateral thoracic diameter; EAHFP=electrical axis of heart in frontal plane; VO₂MAX (METs)=estimated maximum aerobic power in mL kg min⁻¹.

* p < 0.05 compared to control subjects (unpaired Student's *t*-test). See text for details.

by the following equation [24]

$$VO_2 \max = \frac{D - 466.3}{45.7},\tag{1}$$

where *D* is the distance achieved in the field test, expressed in meters, and VO_{2MAX} is estimated as mL kg⁻¹ min⁻¹. Next, the resulting VO_{2MAX} was divided by the constant 3.5 mL kg⁻¹ min⁻¹ to be converted into metabolic equivalents (METs). The Control and Athlete groups were separated according to VO_{2MAX} as determined by the Cooper test, arbitrarily defined as less than 11.5 METs for controls and more than 16.0 METs for athletes (to separate both groups and to characterize sedentary and top athletes). The gap between groups was defined *a priori* with the purpose of enhancing eventual differences in the HRECG parameters caused by the conditioning level [3].

2.3. Physical assessment procedures

The athletes discontinued training 24 h before testing and fasted for 4 h before signal recording (all ECG signals were acquired in a quiet and air-conditioned environment, with a temperature of 25 °C \pm 2 °C, between 8:00 and 11:00 am). All subjects replied to a questionnaire about age, health condition, use of medication, and physical activities; their height, weight, anteroposterior and laterolateral thoracic diameters were measured under the supervision of a primary care physician. Both groups comprised 14 subjects matched by age, gender and body surface area (Table 1) calculated by a nomogram to minimize inter-group physiological and anthropometric variability (e.g., chest diameter) and to reduce the potential effect of thoracic geometry on the surface ECG signals.

2.4. HRECG acquisition and processing

HRECG was acquired shortly after application of the questionnaire and physical examination. Before a 15 min continuous signal acquisition, the subjects remained in the supine position for 5 min for the stabilization of autonomic modulation after changing from the orthostatic position, thereby preventing an "autonomic memory" in the acquired signals [25,26].

The ECG signals were acquired using modified bipolar Frank XYZ orthogonal leads. Self-adhesive electrodes (Ag/AgCl) were attached to the skin that were carefully prepared with a mildly abrasive pad and washed with alcohol. The paired electrode-to-electrode impedance was less than 7 k Ω for all electrode pairs. An electrocardiograph amplifier model AEG03 (Lynx Tecnologia Eletrônica, São Paulo, SP, Brazil), with 10-G Ω input impedance, 120-dB/channel common mode rejection, and a 12-V DC power supply, was used to measure ECGs. Each ECG lead was amplified (gain=1000), analogically filtered from 0.05 to 300 Hz by a third-order Butterworth filter, and converted into digital format by a data acquisition system with a 14-bit AD converter at a \pm 10 V dynamic range, and a 1000 Hz/channel sampling frequency (Lynx Tecnologia). The ECG was digitized and immediately stored in a PC for off-line processing.

Digital data were processed with pattern recognition software to reject ectopic or excessively noisy beats. The coherent weighted averaging was carried out independently on each lead using a previously described and validated technique for R-wave alignment and was stopped at a residual noise level below 0.35 μ V [27,28]. The HRECG was analyzed in the time domain on the vector magnitude ($Vm = \sqrt{X^2 + Y^2 + Z^2}$), where *X*, *Y* and *Z* are the averaged orthogonal leads after a 4-pole bi-directional Butterworth filtering, with a band-pass ranging from 40 to 250 Hz [29,30].

Download English Version:

https://daneshyari.com/en/article/10351697

Download Persian Version:

https://daneshyari.com/article/10351697

Daneshyari.com