Extrema of QRST Isointegral Maps in Left Ventricular Hypertrophy

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Abstract. *Left ventricular hypertrophy may both increase or decrease values of time integrals in QRST isointegral maps (IIM). We wanted to find out the change of IIM QRST in hypertensive patients with and without LVH.*

We analysed IIM QRST of 38 hypertensive patients – 15 without and 23 with left ventricular hypertrophy (left ventricular mass 107 ± 11 g/m^2 versus 158 ± 29 g/m^2, $p < 0.0001$) and compared their data with 12 controls of comparable age.

We found significantly decreased mean maxima and peak-to-peak values in patients with hypertrophy in comparison to controls (maxima: 72.4 ± 37.3 mV.ms versus 100.6 ± 18.9 mV.ms, $p < 0.05$; peak-to-peak: 100.5 ± 45.6 mV.ms versus 136.2 ± 24.4 mV.ms, $p < 0.05$). Minima increased not significantly. Any significant changes in map extrema values were found neither between patients with and without hypertrophy, nor between "pure" hypertensives and controls.

As both possibilities, i.e. increased and decreased IIM QRST values were published, this topic needs more detailed studies.

Keywords: ECG Body Surface Mapping, Isointegral Map, QT Interval, Hypertension, Hypertrophy

1. Introduction

The voltage-duration products of QRS complex or more precise time integrals in chest and limb leads may be used as criteria for left ventricular hypertrophy (LVH) [1, 2]. Increased voltage and prolonged duration of QRS complex is assumed. But repolarisation changes are displayed as opposite direction of ST-segment and T-wave to QRS complex (left ventricular strain) [3]. This may result either in increase or in decrease of QRTS time integrals based on the fact which of these phenomena dominates.

Spatial analogy of single leads time integral is the isointegral map (IIM) when using electrocardiographic body surface mapping [4]. The aim of this study was to find out the change of IIM QRST in hypertensive patients without and with LVH.

2. Subject and Methods

Electrocardiograms were recorded and body surface maps constructed in 38 hypertensive patients (24 men; 50 ± 10 years (y) old) divided into 2 groups: 15 patients without LVH (HT group: 9 men; 48 ± 11 y) and 23 patients with LVH (LVH group: 15 men; 51 ± 10 y).

LVH was based on echocardiographic examination [5]. Left ventricular mass (LVM in gram) was computed according to the formula:

$$LVM = 1.04 \cdot [(IVSd + LVIDd + LVPWd)^3 - LVIDd^3] - 13.6 \quad (1)$$

where IVSDd is the thickness of interventricular septum, LVIDd is the internal diameter of the LV, LVPWd is the thickness of LV posterior wall, all measured in cm during diastole.

LVH was taken as present if the LVM index (LVMI in g/m^2) based on body surface area (BSA):

$$LVMI = \frac{LVM}{BSA} \quad (2)$$

fulfilled the condition:

$$LVMI_{man} \geq 125 \ g/m^2 \quad \text{or} \quad LVMI_{woman} \geq 110 \ g/m^2 \ . \quad (3)$$

Mean IIM of QT interval was constructed using the lead system after Barr as described earlier for each examination [6, 7]. Patients' data were compared with 12 controls (43 ± 10 y, 5 men) with no history of cardiovascular diseases and normal electrocardiographic and echocardiographic findings.

Values of mean map extrema (maximum, minimum, peak-to-peak value = maximum – minimum) were analysed [7]. Statistical evaluation was done using t-test and analysis of variance with least square differences methods for multiple comparisons [8]. Statistically significant differences were assumed for $p < 0.05$ or less.

3. Results

Echocardiographic characteristics of patients are given in Table 1. Significantly higher values were obtained for the LVH group in all evaluated parameters.

Table 1: Echocardiographic characteristics of patients (mean ± standard deviation).

Group	IVSd [cm]	LVIDd [cm]	LVPWd [cm]	LVM [g]	LVMI [g/m^2]
HT	1.1 ± 0.1	4.9 ± 0.4	1.0 ± 0.1	214 ± 35	107 ± 11
LVH	1.3 ± 0.2$^{\#}$	5.2 ± 0.5*	1.2 ± 0.1$^{\#}$	320 ± 68$^{\#}$	158 ± 29$^{\#}$

Statistically significant difference between both groups: * $p < 0.05$; $^{\#}p < 0.0001$

Mean group IIM QRST revealed smooth dipolar distribution in all groups (Fig. 1). In controls, positive time integrals covered the majority of the left anterior chest and almost the whole inferior part of torso with maximum located in the left precordial area. Negative time integrals occurred in the upper half of the right chest and on the back with minimum located in the right clavicular region. In hypertensives without hypertrophy (group HT), the distribution was similar, but the positive time integrals covered smaller part of the chest. Zero line between positive and negative time integrals became more vertical. This verticality was

stressed in patients with LVH, where the whole right chest (anterior and posterior) was negative. IIM QRST became "flatter", the maxima decreased and the minima increased.

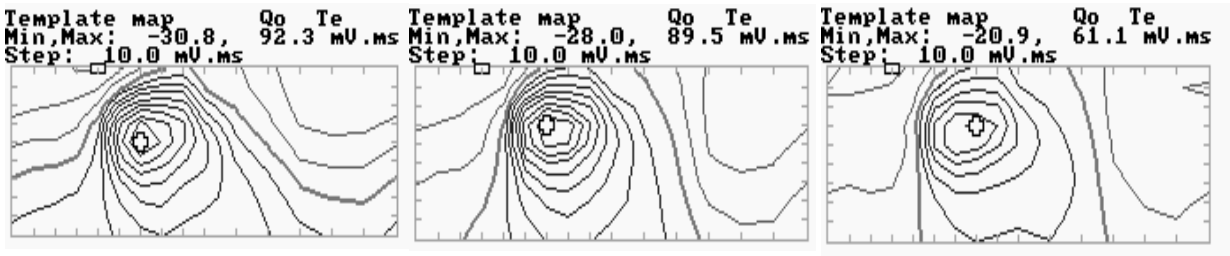

Figure 1: Mean IIM QRST (from left to right: controls, group HT, group LVH).

Values of absolute extrema of IIM QRST are given in Table 2. We confirmed the decreased values of all evaluated extrema in LVH group compared to both controls and hypertensives (minima taken in their absolute value). Statistically significant differences were found only between patients with hypertrophy and controls for maxima and peak-to-peak values. Differences between HT and LVH group were not significant, probably because of large variability of data in both groups. Differences between controls and hypertensives were negligible.

Table 2: Extreme values of QRST isointegral maps (mean ± standard deviation).

Group	Maximum [mV.ms]	Minimum [mV.ms]	Peak-to-peak [mV.ms]
Control	100.6 ± 18.9	- 35.6 ± 10.0	136.2 ± 24.4
HT	98.2 ± 40.8	- 32.1 ± 9.5	130.3 ± 45.6
LVH	72.4 ± 37.3*	- 28.1 ± 9.8	100.5 ± 44.2*

Statistically significant difference between LVH group and controls: * $p < 0.05$

4. Discussion and Conclusions

Increased extreme values in IIM QRST were published in patients with increased LV mass [9], while others published decreased values in IIM QRST [10, 11] in comparison to healthy subjects. Types of included hypertrophies were not discussed there, so both concentric and eccentric hypertrophy could be involved as well as left ventricular remodelling [12].

In our study, we found significantly decreased mean values of maximum and peak-to-peak in patients with LVH. Probably, repolarisation changes dominated against the expected increased voltage during QRS complex. But a few single patients (from both HT and LVH group) had higher extrema than the controls. The reason for different values is not known yet.

According to obtained results we assume that the mass increase of the left ventricle is not enough to increase the electric potential on the body surface. Other factors, which are not clear yet, may play an important role. More detailed studies have to be performed.

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