

AMPLITUDES OF ISOINTEGRAL MAPS DURING VENTRICULAR REPOLARISATION IN HYPERTENSION AND LEFT VENTRICULAR HYPERTROPHY

KOZLÍKOVÁ K.¹, MARTINKA J.¹, MURÍN J.², BULAS J.³

¹Institute of Medical Physics and Biophysics, Comenius University, Faculty of Medicine, Bratislava, Slovak Republic

²First Department of Internal Medicine, University Hospital, Bratislava, Slovak Republic

³First Department of Internal Medicine, Comenius University, Faculty of Medicine, Bratislava, Slovak Republic

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Abstract

Left ventricular hypertrophy may both increase or decrease values of time integrals in QRS or QRST isointegral maps (IIM). We wanted to find out what change appears during repolarisation alone in hypertensive patients with and without left ventricular hypertrophy.

We analysed the extrema in IIM QRS, STT, and QRST of 38 hypertensive patients – 15 without (group HT) and 23 with left ventricular hypertrophy (group LVH), and compared their data with age- and sex-matched 12 controls (group C).

There were no significant changes during the QRS complex, although peak-to-peak values increased with increasing ventricular mass. The highest maxima were found in group HT, the lowest in group C. The shallowest minima were in group LVH, the deepest in group HT.

During repolarisation (IIM STT, IIM QRST), the highest mean extrema were found in group C, the lowest (flat maps) in group LVH. Significant changes were found in group LVH compared to group C in IIM STT for minima ($p < 0.0005$) and peak-to-peak values ($p < 0.05$), in IIM QRST for maxima and peak-to-peak values (both $p < 0.05$); in group HT compared to group C in IIM STT for minima and peak-to-peak values (both $p < 0.05$). There were no significant differences between groups HT and LVH.

We found increasing values of time integrals with increasing left ventricular mass during depolarisation, but decreasing values during repolarisation. As both possibilities, i.e. increased and decreased values of IIM extrema were published, this topic needs more detailed studies taking into account not only the ventricular mass but also its geometry.

Key words

Electrocardiographic body surface mapping, Depolarisation, Repolarisation, Hypertension, Hypertrophy

INTRODUCTION

The increased voltage-duration products of the QRS complex or, more precise, the time integrals in chest and limb leads can be used as criteria for left ventricular hypertrophy (LVH) assuming increased voltage and prolonged duration of the QRS complex (1, 2). Repolarisation changes occur in opposite direction to QRS complex changes - left ventricular strain (3). This may result either in an increase or in a decrease of QRST time integrals, depending on 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 detect the change of IIM during repolarisation in hypertensive patients without and with LVH and whether these changes occur separately during the ST-T interval or only in the QT interval.

SUBJECTS AND METHODS

The electrocardiograms were recorded and body surface maps constructed in 38 patients with essential hypertension (24 men; 50 ± 10 years (y) old, range 25-68 y) 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). Hypertension was defined as prevailing systolic blood pressure (BP) exceeding 140 mmHg and/or diastolic BP exceeding 90 mmHg, or antihypertensive therapy lasting for more than 3 months. Patients with angina pectoris, myocardial infarction, or any conduction disturbances were excluded from this study.

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

$$\text{LVM} = 1.04 \sqrt{[\text{IVSd} + \text{LVIDd} + \text{LVPWd}]^3 - \text{LVIDd}^3} - 13.6$$

where IVSd is the thickness of the interventricular septum, LVIDd is the internal diameter of the left ventricle (LV), LVPWd is the thickness of the LV posterior wall, all measured in cm in diastole.

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

$$\text{LVMI} = \frac{\text{LVM}}{\text{BSA}}$$

fulfilled the condition $\text{LVMI}_{\text{man}} \geq 125 \text{ g}/\text{m}^2$ and/or $\text{LVMI}_{\text{woman}} \geq 110 \text{ g}/\text{m}^2$ (6, 7). According to this criterion and to the relative wall thickness

$$\text{RWT} = \frac{\text{IVSd} + \text{LVPWd}}{2}$$

the patients may be classified into 4 ventricular geometry groups (6) as follows: normal geometry (LVH not present, $\text{RWT} < 0.45$) - 13/15 patients in HT group; concentric remodelling (LVH not present, $\text{RWT} \geq 0.45$) - 2/15 patients in HT group; concentric hypertrophy (C_LVH; LVH present, $\text{RWT} \geq 0.45$) - 17/23 patients in LVH group; eccentric hypertrophy (E_LVH; LVH present,

RWT < 0.45) - 6/23 patients in LVH group. In the LVH group only 2 patients had increased LVIDd above normal limits (1 woman (E_LVH): 60 mm; 1 man (C_LVH): 62 mm).

Mean maps were recorded and constructed for each examination from single beat maps as described earlier (8, 9). The patients' data were compared with 12 controls (5 men; 43 ± 10 y, range 30–56 y) with no history of cardiovascular diseases, normal blood pressure, and normal electrocardiographic and echocardiographic findings. All studied groups were age- and sex-matched.

The values of mean map extrema (maximum, minimum, peak-to-peak value = maximum - minimum) were analysed, mean maps were calculated from each examination (9). Mean group maps were constructed from a single beat map of each subject. Statistical evaluation was done using the t-test; analysis of variance followed by the least square difference method for multiple comparisons, and testing of binomial proportions for sex (10). Statistically significant differences were assumed for $p < 0.05$ or less.

RESULTS

Significantly higher values of evaluated echocardiographic characteristics were obtained for the LVH group (Table 1). At the date of examination the systolic BP of all patients with hypertension was 151 ± 19 mmHg and the diastolic BP was 94 ± 12 mmHg.

Table 1
Echocardiographic characteristics of patients (mean \pm standard deviation)

Group	IVSd [cm]	LVIDd [cm]	LVPWd [cm]	LVM [g]	LVMI [g/m ²]	RWT
HT	1.1 ± 0.1	4.9 ± 0.4	1.0 ± 0.1	214 ± 35	107 ± 11	0.42 ± 0.03
LVH	$1.3 \pm 0.2^{\#}$	$5.2 \pm 0.5^*$	$1.2 \pm 0.1^{\#}$	$320 \pm 68^{\#}$	$158 \pm 29^{\#}$	$0.49 \pm 0.08^{**}$

Statistically significant difference between both groups:

* $p < 0.05$; ** $p < 0.005$; # $p < 0.0001$

The mean group IIM revealed a smooth dipolar distribution (Fig. 1). In controls, positive time integrals covered the majority of the left anterior chest and the inferior part of the torso with maximum located in the left precordial area. Negative time integrals were in the upper half of the right chest and on the back with minimum located in the left parasternal area (QRS), above the right scapula (STT), and in the right clavicular region (QRST), but always higher than the maximum.

In group HT, the distribution of mean maps was similar, but the positive time integrals covered smaller part of the chest, mainly the left half. The maxima were located in approximately the same positions as in controls, but the minimum in IIM QRS was located rightwards and upwards and in IIM STT downwards to the level of the maximum. The zero line between positive and negative time integrals became more vertical. This verticality was stressed in LVH patients, where the whole right chest (anterior and posterior) was negative. In IIM QRS and STT both extrema were at the same horizontal level (the 4th intercostal space). In IIM QRST

the maximum moved slightly leftwards. The mean repolarisation maps became “flatter”, the maxima decreased and the minima increased.

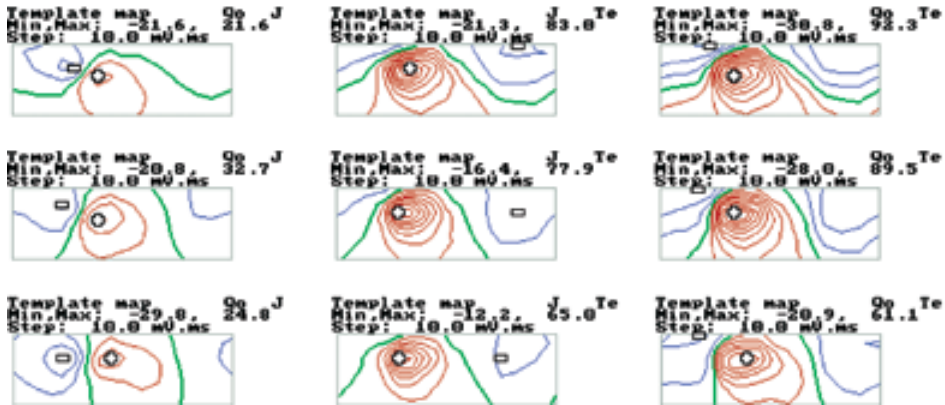


Fig. 1

Mean group isointegral maps

From top: controls, group HT, group LVH; from the left: IIM QRS (QoTe), IIM STT (JTe), IIM QRST (QoTe). Each rectangle represents the chest surface enrolled into plane along the right midaxillary line. The left half of the rectangle corresponds to the anterior chest, the right half to the back. Maximum and minimum are marked by plus and minus signs, respectively.

There were no significant differences between the mean absolute extrema of IIM QRS (Table 2), although peak-to-peak values increased with increasing LV mass. In IIM STT and QRST (Tables 3, 4), all evaluated extrema decreased with increasing LV mass (minima in absolute value). Statistically significant differences were found for STT minima between HT and LVH patients compared to controls and between LVH group and controls for peak-to-peak values (Table 3). The maxima and peak-to-peak values of IIM QRST differed significantly between HT and LVH patients compared to controls (Table 4). The differences between HT and LVH group were not significant.

Table 2
Extreme values of IIM QRS (mean \pm standard deviation)

Group	Maximum [mV.ms]	Minimum [mV.ms]	Peak-to-peak [mV.ms]
Control	27.9 \pm 11.8	- 32.0 \pm 19.4	59.9 \pm 18.6
HT	37.2 \pm 22.1	- 26.7 \pm 11.7	63.9 \pm 28.3
LVH	31.2 \pm 14.1	- 36.2 \pm 20.7	67.4 \pm 26.1

Table 3
Extreme values of IIM STT (mean \pm standard deviation)

Group	Maximum [mV.ms]	Minimum [mV.ms]	Peak-to-peak [mV.ms]
Control	95.7 \pm 27.3	- 24.7 \pm 8.7	120.4 \pm 30.2
HT	87.1 \pm 41.0	- 19.1 \pm 7.5*	106.2 \pm 44.5
LVH	71.1 \pm 36.1	- 15.2 \pm 5.8***	86.3 \pm 40.5*

Statistically significant difference between LVH (HT) group and controls:

*p < 0.05; ***p < 0.0005

Table 4
Extreme values of IIM QRST (mean \pm standard deviation)

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

Statistically significant difference between LVH (HT) group and controls:

*p < 0.05

DISCUSSION

Increased and decreased extreme values in isointegral maps were published in patients with increased LV mass (11, 12, 13) in comparison to healthy subjects. The types of included hypertrophies from the geometrical point of view were not discussed there, so both concentric and eccentric hypertrophy could be involved as well as LV remodelling (7).

Igarashi et al. (11) studied 42 men having essential hypertension (systolic BP over 160 mmHg, and/or diastolic BP over 95 mmHg), 32–72 years old, divided into 3 groups due to increasing LVM: group A (188 \pm 20 g), group B (247 \pm 19 g), and group C (373 \pm 59 g). They found significantly increased IIM QRS maxima in groups B and C compared to group A and to controls (40 men, 22–55 years old). The next studied extrema revealed no significant differences. Because of different LVM group values it is not possible to compare the data more exactly. Differences could also be caused by higher blood pressure than in our study and due to sex difference.

Corlan and de Ambroggi (12) studied 16 patients (LVH: 8 men, 68 ± 13 y), with LVH due to aortic stenosis having $\text{LVMI} = 182 \pm 51 \text{ g/m}^2$. Their maps were compared with 35 controls (C: 25 men, 33 ± 10 y). Significantly increased peak-to-peak values were found in IIM QRS (C: $69.3 \pm 26.2 \text{ mV.ms}$ versus LVH: $164.1 \pm 68.1 \text{ mV.ms}$, $p < 0.0001$) and IIM STT (C: $130.1 \pm 49.8 \text{ mV.ms}$ versus LVH: $186.5 \pm 96.7 \text{ mV.ms}$, $p < 0.05$). In IIM QRST they decreased significantly (C: $148.2 \pm 56.2 \text{ mV.ms}$ versus LVH: $104.8 \pm 38.9 \text{ mV.ms}$, $p < 0.005$). As no data concerning blood pressure were given it is not possible to compare the data more exactly.

Hirai et al. (13) studied 10 patients with aortic stenosis (representing concentric LVH, 7 men, 24–72 y; $\text{LVM} = 390 \pm 148 \text{ g}$), 12 patients with aortic regurgitation (eccentric LVH, 11 men, 36–65 y; $\text{LVM} = 403 \pm 116 \text{ g}$), and 22 patients with septal hypertrophy (hypertrophic cardiomyopathy, 19 men, 17–65 y; $\text{LVM} = 356 \pm 103 \text{ g}$). Their maps were compared with 50 controls (25 men, 18–52 y). In all patient groups, they found decreased values of IIM QRST compared to controls. No data concerning map extrema were given, so it is not possible to compare the data more exactly.

Oikarinen et al. (14) studied 42 LVH patients (25 men, 63 ± 12 y; $\text{LVM} = 344 \pm 93 \text{ g}$, $\text{LVMI} = 181 \pm 45 \text{ g/m}^2$), among them 27 patients with aortic valve stenosis and 15 with essential hypertension. Their maps were compared with 11 controls (8 men, 55 ± 7 y). The area maps used were constructed in a different way than used in our study and all the other mentioned studies, so direct comparison is again not possible, although significant differences between LVH group and control maps were found.

In our study, we found no significant changes of the QRS complex between controls and patients, although peak-to-peak values increased with increasing LV mass. This is in good agreement with some published data (11, 12). However, we found significantly decreased mean extrema in both HT and LVH groups. Repolarisation changes were found as early as in ST-T interval maps and not only in QT interval maps as in (12). They dominated against the expected increased voltage during the QRS complex. The differences between HT and LVH group were not significant.

Decreased values of IIM QRST from our study are in good agreement with some published data (12, 13). Discords in the results could be caused by differently defined groups of patients, a different aetiology of hypertrophy, racial differences, the possible influence of obesity, sex, and/or age. The different lead systems used may also influence the results.

According to the obtained results we assume that the mass increase of the LV is not enough to increase the electric potential on the body surface during repolarisation. More detailed studies have to be performed including not only the LV mass but also its geometry and trying to exclude the influence of earlier mentioned factors.

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Kozlíková K., Martinka J., Murín J., Bulas J.

AMPLITÚDY IZOINTEGRÁLOVÝCH MÁP POČAS KOMOROVEJ REPOLARIZÁCIE PRI HYPERTENZII A HYPERTROFII LAVEJ KOMORY

Súhrn

Pri hypertrofii ľavej komory sa hodnoty časových integrálov na izointegrálových mapách (IIM) QRS a QRST môžu zvýšiť aj znížiť. Zaujímalo nás, aké zmeny sa udejú počas samotnej repolarizácie u hypertenzných pacientov, ktorí môžu mať aj hypertrofiu ľavej komory (LK).

Analýzovali sme extrémny IIM QRS, STT a QRST u 38 pacientov s hypertenziou – 15 bez hypertrofiu LK (skupina HT) a 23 s hypertrofiou LK (skupina LVH), a porovnali sme ich údaje s kontrolnou skupinou (C, 12 osôb).

Nezistili sme žiadne významné rozdiely počas komplexu QRS hoci rozsah amplitúd narastal so zvyšujúcou sa hmotnosťou LK. Najvyššie maximá boli v skupine HT, najnižšie v skupine C. Najplytšie minimá boli v skupine LVH, najhlbšie v skupine HT.

Počas repolarizácie (IIM STT, IIM QRST) boli najvyššie extrémny v skupine C, najnižšie (ploché mapy) v skupine LVH. Významné rozdiely boli v skupine LVH voči skupine C na IIM STT pre minimá ($p < 0.0005$) a rozsah amplitúd ($p < 0.05$), na IIM QRST pre maximá a rozsah amplitúd ($p < 0.05$). Významné rozdiely boli v skupine HT voči skupine C na IIM STT pre minimá a rozsah amplitúd ($p < 0.05$). Nezistili sme žiadne významné rozdiely medzi skupinami HT a LVH.

Zistili sme zvýšenie časových integrálov pri zvyšujúcej sa hmotnosti LK počas depolarizácie, ale ich zníženie počas repolarizácie. Keďže dosiaľ boli publikované obe možnosti, táto problematika si vyžaduje detailnejšie štúdium so zohľadnením geometrie LK, nielen jej hmotnosti.

REFERENCES

1. Okin PM, Roman MJ, Devereux RB, Pickering TG, Borere JS, Kligfield P. Time-voltage QRS area of the 12-lead electrocardiogram. Detection of left ventricular hypertrophy. *Hypertension* 1998; 31(4): 937-942.
2. Jern S. Assessment of left ventricular hypertrophy in patients with essential hypertension. *Blood Press* 1997; 2 (Suppl): 16-23.
3. Milliken JA, Macfarlane PW, Veitch Lawrie TD. Enlargement and hypertrophy. In: Macfarlane PW, Veitch Lawrie TD, eds. *Comprehensive electrocardiology. Theory and practice*. New York: Pergamon Press, 1989: 631-670.
4. Abildskov JA, Burgess MJ, Urie P, Lux RL, Wyatt R. The unidentified information content of the electrocardiogram. *Circ Res* 1977; 40(1): 3-7.
5. Devereux RB. Detection of left ventricular hypertrophy by M-mode echocardiography. Anatomic validation, standardization and comparison to other methods. *Hypertension* 1987; 9 (Suppl II): II19-II26.
6. Bulas J, Murín J, Kozlíková K. Echokardiografická charakteristika hypertrofiu ľavej komory srdca [Echocardiographic characteristics of left ventricular hypertrophy]. *Kardiol/Cardiol* 1998; 7(2): 92-98.
7. Devereux RB, Alonso DR, Lutas EM et al. Echocardiographic assessment of left ventricular hypertrophy: comparison to necropsy findings. *Amer J Cardiol* 1986; 57 (6): 450-458.
8. Kozlíková K, Martinka J, Murín J. IIM QRS in concentric left ventricular hypertrophy. *Int J Bioelectromagnetism* 2003; 5(1): 199-200.
9. Kozlíková K. Povrchové integrálové mapy, ich charakteristiky a metódy kvantitatívnej analýzy [Body surface integral maps, their characteristics and methods of quantitative analysis]. *Bratisl Lek Listy* 1990; 91(11): 815-823.

10. Statgraphics® PLUS, version 3 for Windows. User manual. Rockville: Manugistics, Inc., 1997.
11. Igarashi H, Kubota I, Ikeda K, Yamaki M, Tsuiki K, Yasui S. Body surface mapping for the assessment of left ventricular hypertrophy in patients with essential hypertension. *Jpn Circ J* 1987; 51(3): 284-292.
12. Corlan AD, De Ambroggi L. New quantitative methods of ventricular repolarization analysis in patients with left ventricular hypertrophy. *Ital Heart J* 2000; 1(8): 542-548.
13. Hirai M, Hayashi H, Ichihara Y, et al. Body surface distribution of abnormally low QRST areas in patients with left ventricular hypertrophy. An index of repolarization abnormalities. *Circulation* 1991; 84(4): 1505-1515.
14. Oikarinen L, Karvonen M, Viitasalo M, et al. Electrocardiographic assessment of left ventricular hypertrophy with time-voltage QRS and QRST-wave areas. *J Human Hypertens* 2004; 18(1): 33-40.