

## The Opposite Polarity Of The PQ Segment Compared To The P Wave Isointegral Maps

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Opposite Polarity of PQ Segment and P Wave

## Summary

The aim of our work was to study the opposite polarity of the PQ segment to the P wave body surface potential maps in different groups of patients. We constructed isointegral maps (IIM) in 26 healthy controls (C), 16 hypertensives (HT), 26 patients with arterial hypertension and left ventricular hypertrophy (LVH) and 15 patients with myocardial infarction (MI). We analyzed values and positions of map extrema and compared the polarity of maps using the correlation coefficient. The IIM P maxima appeared mainly over the precordium, the minima mainly in the right subclavicular area. The highest maxima were in the MI group, being significantly higher than in the HT and LVH groups. No differences concerning any values of next extrema were significant. The IIM PQ maxima were distributed over the upper half of the chest; the minima mainly over the middle sternum. A statistically significant opposite polarity between the IIM P and IIM PQ was found in 80%. The opposite polarity of the P wave and the PQ segment was proved in isointegral body surface maps. The extrema occurred in areas not examined by the standard chest leads. This has to be considered for diagnostic purposes.

## Keywords

body surface potential mapping, atrial repolarisation, arterial hypertension, left ventricular hypertrophy, myocardial infarction

## Introduction

In the standard 12-lead electrocardiogram, the atrial depolarisation is represented by the P wave and the atrial repolarisation occurs during the PQ segment and the QRS complex. The polarity of the atrial repolarisation wave  $T_a$  is normally opposite to that of the P wave. The potentials generated by the atrial repolarisation are difficult to study for two reasons. Firstly, their amplitude is very low, usually under 0.1 mV and, therefore, the  $T_a$  wave is very often not recorded on the standard electrocardiogram. Secondly, ventricular activation may begin before the atrial repolarisation ends; therefore, then the QRS complex overlaps it. When a positive P wave is recorded and a  $T_a$  wave exists, the electrocardiographic curve between the P wave and the beginning of the QRS complex can be oblique. During tachycardia, the PQ segment can be even more depressed. The atrial repolarisation can be recorded even during the ST segment. Changes of the PQ segment shape were found in myocardial infarction of the right atrium. More often, a change in the PQ duration is found in preexcitation or atrioventricular conduction blockade. Therefore, correct evaluation of the PQ segment has high diagnostic importance (Cagán and Hulín 1983; Mirvis 1993).

On the other side, the flat portion of the PQ segment is often taken as the zero baseline in body surface potential mapping, for example when diagnosing myocardial infarction or when the normally flat TP segment is not usable for some reason, mainly high heart rate (Takala et al. 2001; Vesterinen et al. 2004; Yamaki et al. 1988).

The research on body surface potential mapping deals predominantly with the ventricular excitation process. A limited number of data describing the atrial electric events is available. They are mainly devoted to isopotential maps as well as papers concerning the PQ segment mapping (Ihara et al. 2006; Kozlíková 2007; Mirvis 1980; Spach et al. 1979; Stilli et al. 1983). Only a few papers deal with body surface isointegral P wave maps as reviewed previously (Kozlíková 2007).

No data dealing with isointegral maps during the PQ segment except for our studies have been found yet.

The limited number of data concerning body surface mapping of atrial activation may be also due to small amplitudes of the P waves and even smaller amplitudes of the PQ segments. Therefore, both can easily be distorted by noise. This can be at least partially avoided by using isointegral maps as they can stress small changes lasting longer time.

The aim of this retrospective study was to find out whether the opposite polarity during the PQ segment to the P wave can be recorded when using body surface potential mapping – isointegral maps and whether these isointegral maps differ between evaluated groups of patients.

## Methods

### Patients and Controls

We studied isointegral maps of 83 subjects with different diagnosis. Group HT involved 16 patients (10 men, 6 women) with arterial hypertension, at age 25 – 75 years ( $50 \pm 13$  y). Hypertension with left ventricular hypertrophy (group LVH) had 26 patients (15 men, 11 women), at age 32 – 72 years ( $54 \pm 12$  y). Group MI involved 15 men, three months after acute myocardial infarction, at age 40 – 70 years ( $53 \pm 9$  y). Control group C involved 26 healthy controls (16 men, 10 women), at age 21 – 56 years ( $33 \pm 13$  y). None of the controls had any sign of cardiovascular disease; all had normal 12-lead standard electrocardiographic as well as echocardiographic findings.

All examined patients were hospitalised for cardiovascular reasons. The body surface potential mapping was performed during the first days after admission; altogether in more than 90 patients. Patients with conduction disturbances, renal insufficiency and/or diabetes were excluded from this study.

The diagnosis of arterial hypertension was based on repeated clinical examinations when the systolic blood pressure was higher than 140 mmHg and/or the diastolic blood pressure was higher than 90 mmHg; the diagnosis was established at least 5 years before admission to hospital. All patients were treated according to the guidelines with combination therapy used in most patients (Mancia et al. 2007). Hypertension was controlled near to the target blood pressure only in the HT men subgroup ( $142 \pm 16 / 92 \pm 8$  mmHg); it was not well controlled in the HT women subgroup nor in the whole LVH group. The values of mean blood pressure measured a few minutes before the mapping examination are given in Table 1.

The left ventricular mass LVM [g] was calculated according to the formula

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

where IVSd is the thickness of the interventricular septum [cm], LVPWd is the thickness of the left ventricular posterior wall [cm], LVIDd is the diameter of the left ventricle [cm], all in diastole (Bulas et al. 1998). The left ventricular hypertrophy was based on the left ventricular mass index  $LVMI > 109 \text{ g/m}^2$  for women and  $LVMI > 134 \text{ g/m}^2$  for men, where

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

Body surface area was calculated according to Mosteller formula (Mosteller 1987)

$$BSA = \sqrt{\frac{m \cdot h}{3600}},$$

where  $m$  is the body mass [kg] and  $h$  is the body height [cm]. In the LVH group, 18 patients had concentric hypertrophy ( $RWT > 0.45$ ) and 8 had eccentric hypertrophy. RWT is the relative wall thickness

$$RWT = \frac{IVSDd + LVPWd}{LVIDd} .$$

Left atrial diameter was normal in 12/16 patients in HT group, in 13/26 patients in LVH group and in 7/15 patients in MI group. The remaining patients had the left atrium mildly or moderately abnormal (Lang et al. 2005). Right atrial diameter was normal in all subjects. Selected echocardiographic characteristics of all patients, all obtained in M-mode and measured by two investigators (co-authors), are given in Table 1.

The diagnosis myocardial infarction was based on medical history, standard electrocardiographic, on echocardiographic (ventricular wall motion) and on laboratory examinations. Seven patients had anterior and eight had inferior infarction, all of small size with preserved left ventricular function (the ejection fraction was 50 – 60 %). All patients underwent successful thrombolytic therapy.

#### Body surface potential mapping

Unipolar electrocardiograms were recorded in supine positions during normal expiration using the mapping system ProCardio with 24 leads after Barr (Barr et al. 1971; Rosík et al. 1997).

A linear baseline through the TP segments before and after the analysed heartbeat was used (Kozlíková 1990). The onset and the end of the P wave and the onset of the QRS complex were established manually from the root mean square signal of the map. The limiting points were set at the end of a sequence of decreasing values when starting from the middle of the P wave or from the middle of the QRS complex. The PQ segment was taken as the interval between the end of the

P wave and the QRS onset; the PQ interval involved both the P wave and the PQ segment. To ensure the reproducibility of data processing, all mapping registrations and signal processing were done by the same investigator (the author) and based on the same procedures and algorithms.

The isointegral maps (distributions of time integrals of voltage) were constructed for single beats. Then the mean maps were calculated for every subject ( $8 \pm 3$  beats depending on the heart rate) and evaluated. We analysed the values and positions of the map extrema – maximum, minimum, and the peak-to-peak amplitudes (maximum minus minimum), and compared the polarity of maps.

#### Statistical evaluation

To describe and to analyse the data statistically, we used averages and standard deviations. Analysis of variance with suitable post hoc tests was used to isolate differences for multiple comparisons. The polarity between pairs of maps was compared using Pearson's correlation coefficient. Each coefficient was tested whether it differs from zero to establish the statistical significance of correlation (Kozlíková and Martinka 2009). Extrema values and positions in pairs of maps were compared using paired t-test. Calculations were performed using the statistical package Statgraphics Plus (Statgraphics 1997). Differences were considered as statistically significant if  $p < 0.05$ .

#### Results

All electrocardiographic recordings were performed during normal sinus rhythm. The heart rate was comparable in all patient groups. Significantly higher heart rate was only in the MI group

against controls (Table 2). Nevertheless, the P wave and the PQ interval durations were significantly shorter in the control group than in the groups LVH and MI.

The individual single beat P wave maps displayed mostly a bipolar distribution (one maximum and one minimum) resembling the pattern types described earlier (Kozlíková 2007). The PQ segment maps were very often multipolar (3 or 4 extrema occurred), much flatter and visually often displaying the reversed polarity to the P wave maps. The mean maps of individual patients were smoother and their reversed polarity was visually preserved.

The group mean maps had bipolar distributions (Figure 1). The P wave maps displayed negativities in the upper part and positivities in the lower part of the chest. The polarity of the PQ segment maps was reversed and they were much flatter.

We found significantly higher maxima of the P wave in the group MI than in the groups HT and LVH. Because of multiple comparisons, differences concerning the values of next extrema were significant neither in the P wave maps nor in the maps of the PQ segment (Table 3). The maxima in the P wave maps were located mainly in the precordial area, the minima mainly parasternally right at the clavicular level or around the right shoulder (Figure 2). Although there were some shifts in positions of both extrema between different groups, the dominating locations were close each other.

The maxima in the single beat PQ segment maps were located approximately in reversed positions compared to the P wave maps (Figure 2). The minima of PQ segment maps were in average significantly closer to the sternum and shifted slightly upwards than the maxima in the P wave maps. The maxima of the PQ segment maps appeared almost in the same position than minima of the P wave maps, but some of them were found also on the upper half of the back.

The opposite (reversed) polarity of the atrial depolarisation and repolarisation was verified by the comparison of maps using the Pearson's correlation coefficient (Table 4). From all possible 83 comparisons, in 66 cases (80 %) was the correlation coefficient negative and significantly differed from zero. The most frequent significant negative correlations were found in the LVH



group. The weakest correlation was found in the MI group. Differences between groups were not significant.

## Discussion

The electrocardiographic body surface mapping is an above standard recording method that offers the most possible information about electrical activity of the heart obtained in a non-invasive way, but comparable to information available from invasive methods. The disadvantage that constrains the standard 12-lead electrocardiogram from providing a more comprehensive and transparent description of the electrical state of the heart, is the limited number of the used electrodes and the location of the chest electrodes in a small area compared to the whole chest. Although the electrodes of the unipolar leads, which are placed very close to the heart, give the best information about the events in their vicinity, this standard 6 chest electrode configuration causes that the most informative regions of the surface potential distribution, for example, local minima and maxima, are often missed by it as we found in this and previous studies. This can be critical in the case of low amplitude potentials such as the P wave or PQ segment (Ihara et al. 2006; Kornreich et al. 1989; Kozlíková 2007; Mirvis 1980; Spach et al. 1979; Stilli et al. 1983).

The duration of the P waves as well as of the PQ intervals in controls was shorter than in the published mapping data (Ihara et al. 2006; Kornreich et al. 1989). Nevertheless, prolonged P waves were found in patients with left ventricular hypertrophy against controls (Kornreich et al. 1989) and in patients with paroxysmal atrial fibrillation against controls when using high resolution electrocardiography recordings during sinus rhythm (Vranka et al. 2007). The differences concerning the controls could be due to different lead system (24 leads against 64 leads (Ihara et al.

2006) or 117 leads (Kornreich et al. 1989)) and slightly different criteria for the P wave onsets and ends used (established from the Frank VCG leads not map leads).

The duration of the P waves and the PQ intervals in this study was measured by applying the same method to all records. Therefore, the prolonged duration of these parts in patients can be explained neither by the higher age of the patients as was the age of the compared controls (Macfarlane and Veitch Lawrie 1989) nor by the heart rate (Table 2). It could be ascribed to dilated or hypertrophied atria, to the increased pressure in their cavities, to hypertension or the existence of atrial substrate for atrial fibrillation mainly in patients with ventricular myocardial infarction even in the case with no atrial involvement (Miyachu et al. 2003; Vranka et al. 2007).

Every mapping procedure has some specific advantages (Kittnar and Miček 2010). Isointegral maps can comprise phenomena lasting for longer time as they summarise in one map both the amplitudes and the time. Consequently, isointegral maps may suppress the noise in low amplitude signals as are the P wave and mainly the PQ segment.

There are no papers dealing with isointegral maps of the PQ segment, therefore, we can compare them only with isopotential maps. In all published papers, the locations of all maxima and some minima were found outside the regions examined by the standard chest leads. Amplitudes were much lower than in the corresponding P wave maps and the reversed polarity between the P wave and the PQ segment was described (Ihara et al. 2006; Kornreich et al. 1989; Mirvis 1980; Spach et al. 1979; Stilli et al. 1983). The PQ segment was never isoelectric as in our study. The description of potential distributions was only qualitative; no quantification of the reversed polarity such as correlation coefficients in this study was published yet.

Better correlation between the P wave and the PQ segment maps in the patients with arterial hypertension and left ventricular hypertrophy as compared to the controls was found. There was increased blood pressure in both groups of patients. To overcome it, the atria have to contract more strongly. As each myocardial contraction is preceded by its activation, this situation could probably

produce a better coordinated activation with less “noise” followed by a better coordinated repolarisation (smooth and not splitting wave fronts). The worst correlation in the MI group could be probably ascribed to the atrial substrate for atrial fibrillation that may influence both the atrial activation and recovery (Aldhoon et al. 2010; Vranka et al. 2007). Specialised studies are needed to confirm these assumptions.

The published data as well as our results suggest that the repolarisation spreads through the atrial walls in approximately the same order, as the depolarisation does. This is in agreement with experimental data demonstrating that the atrial regions that depolarize as first also repolarise as first (Spach et al. 1969).

To conclude, we found that the opposite polarity during the PQ segment to the P wave can be recorded when using body surface potential mapping – isointegral maps – and that these isointegral maps differ between the studied groups of patients. These differences are probably connected with diverse behaviour of the activation and recovery wave fronts due to several diagnoses. They are connected with prolonged P waves in patients.

We confirmed again that the extrema (potential amplitudes) occur in areas not examined by the standard electrocardiographic chest leads. This finding and the fact that the PQ segment is not isoelectric should be considered for diagnostic purposes.

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Table 1. Blood pressure and selected echocardiographic characteristics of patient groups.

Group	BPS [mmHg]	BPD [mmHg]	LVM [g]	LVMI [g·m <sup>-2</sup> ]	RWT	LAD [mm]
HT	145 ± 17	93 ± 7	212 ± 34 <sup>a</sup>	106 ± 11 <sup>a</sup>	0.42 ± 0.04	3.7 ± 0.4
LVH	157 ± 22	94 ± 14	317 ± 68	155 ± 30	0.49 ± 0.08 <sup>b</sup>	4.1 ± 0.5 <sup>c</sup>
MI	119 ± 12 <sup>d</sup>	79 ± 6 <sup>d</sup>	275 ± 83	134 ± 35	0.38 ± 0.10	3.4 ± 0.4

BPS: systolic blood pressure

BPD: diastolic blood pressure

HR: heart rate

LAD: left atrial diameter

<sup>a</sup> p < 0.05 group HT against groups LVH and MI

<sup>b</sup> p < 0.05 group LVH against groups HT and MI

<sup>c</sup> p < 0.05 group LVH against group MI

<sup>d</sup> p < 0.05 group MI against groups HT and LVH

Table 2. Heart rate and durations of the examined intervals.

Group	Hear rate [ $\text{min}^{-1}$ ]	Duration [ms]		
		P wave	PQ segment	PQ interval
C	$60 \pm 15$	$88 \pm 12$	$59 \pm 18$	$147 \pm 21$
HT	$68 \pm 21$	$95 \pm 14$	$73 \pm 28$	$167 \pm 38$
LVH	$65 \pm 26$	$96 \pm 9^*$	$72 \pm 29$	$168 \pm 31^*$
MI	$80 \pm 8^*$	$100 \pm 14^*$	$67 \pm 14$	$167 \pm 17^*$
Total	$67 \pm 19$	$94 \pm 13$	$67 \pm 24$	$161 \pm 29$

\*  $p < 0.05$  against group C



Table 3. The mean values and standard deviations of the map extrema in the studied groups.

Extreme	Maximum [mV·ms]	Minimum [mV·ms]	Peak-to-peak amplitude [mV·ms]
Group	P wave		
C	4.0 ± 1.1	- 4.0 ± 1.5	8.0 ± 2.5
HT	3.6 ± 0.8*	- 4.5 ± 1.5	8.1 ± 2.2
LVH	3.8 ± 0.8*	- 4.1 ± 1.3	8.0 ± 1.9
MI	5.0 ± 1.1	- 4.0 ± 1.5	8.0 ± 2.5
Group	PQ segment		
C	1.0 ± 0.7	- 1.7 ± 0.7	2.8 ± 1.4
HT	1.1 ± 0.6	- 2.2 ± 1.0	3.3 ± 1.4
LVH	1.1 ± 0.8	- 2.5 ± 1.5	3.6 ± 2.2
MI	0.9 ± 0.4	- 1.6 ± 0.7	2.5 ± 0.9

\* p < 0.05 against group MI

Table 4. Correlation coefficients from the comparison of the P wave and PQ segment maps.

Group	Correlation coefficient		
	All	Only negative	Different from zero
C	- 0.35 ± 0.29 (26; 100 %)	- 0.44 ± 0.22 (22; 85 %)	- 0.50 ± 0.17 (19; 73 %)
HT	- 0.53 ± 0.35 (16; 100 %)	- 0.57 ± 0.32 (15; 94 %)	- 0.65 ± 0.26 (13; 81 %)
LVH	- 0.46 ± 0.27 (26; 100 %)	- 0.53 ± 0.19 (23; 88 %)	- 0.53 ± 0.19 (23; 88 %)
MI	- 0.32 ± 0.29 (15; 100 %)	- 0.43 ± 0.21 (12; 80 %)	- 0.46 ± 0.19 (11; 73 %)
Total	- 0.41 ± 0.30 (83; 100 %)	- 0.49 ± 0.24 (72; 87 %)	- 0.53 ± 0.21 (66; 80 %)

average ± standard deviation (number of cases; percentage)

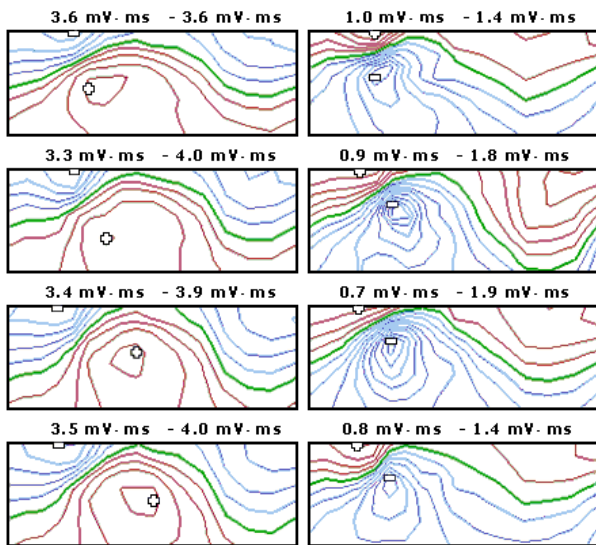


Figure 1. The mean isointegral maps of all studied groups.

The P wave maps (from top to bottom: C, HT, LVH, and MI) are in the left column (step between isointegral lines is 0.8 mV·ms) and the matching PQ segment maps are in the right column (step 0.2 mV·ms). The corresponding maximum and minimum are above each map. The left half of each rectangle displays the anterior chest, the right half the back.

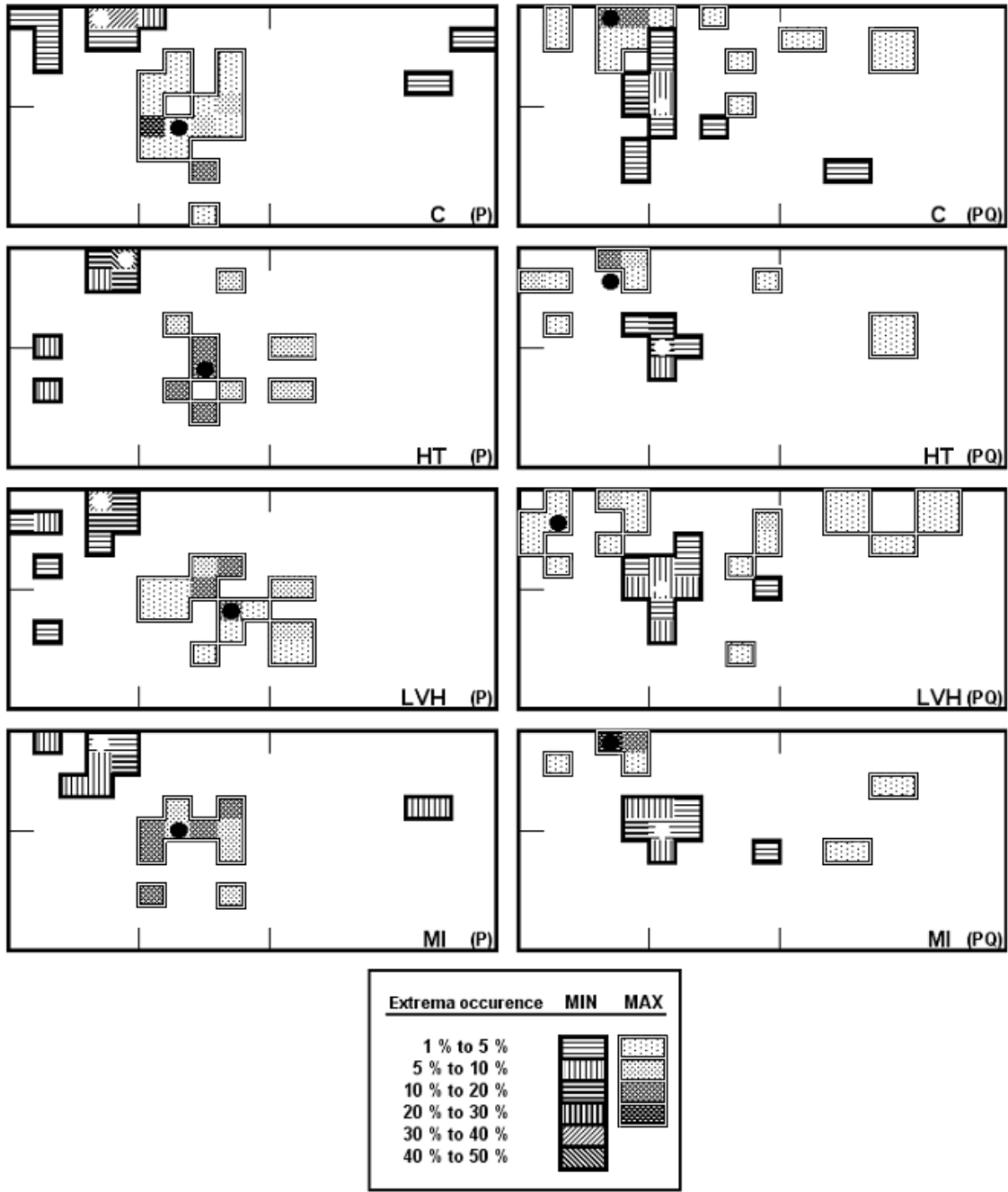


Figure 2. The positions of the P wave (left column) and the PQ segment (right column) extrema in individual maps.

The short vertical lines in every map depict the positions of the sternum and the left mid-axillary line, respectively. The short horizontal line depicts the approximate position of the 5<sup>th</sup> intercostal space. The white circles display the median position of the minimum, the black circles the median position of the maximum.