ORIGINAL ARTICLE

A Simple Numerical Body Surface Mapping Parameter Signifies Successful Percutaneous Coronary Artery Intervention

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Background: In coronary artery disease (CAD), body surface potential mapping (BSPM) may reveal minor electrical potential changes appearing in the depolarization phase even if pathological changes are absent on the conventional 12-lead ECG. We hypothesized that a simple BSPM parameter, Max/Min signifies successful percutaneous coronary intervention (PCI).

Methods: Ninety-two adult Caucasian patients with stable CAD and positive exercise test underwent coronary angiography. Seventy patients (age, 59 ± 8 ; 46 males) were revascularized by PCI (left anterior descending [LAD] in 38, right [RCA] in 17 and left circumflex [LCX] coronary artery in 15). Control groups contained 22 patients (age, 60 ± 8 ; 14 males) without intervention and 35 healthy subjects (age, 58 ± 2 ; 15 males). Left ventricular ejection fraction (LVEF, transthoracic echocardiography) and Max/Min BSPM parameter (63-lead Montreal system) were evaluated before and 4–40 days following coronary angiography. Max/Min was defined by the ratio of the highest maximum to the deepest minimum potential of all leads recorded by BSPM.

Results: Before PCI, Max/Min value of patients with LAD lesion (0.83 [0.74; 0.93]) was significantly lower while that with RCA lesion (1.63 [1.35; 1.99]) was significantly higher than that of healthy group (1.01 [0.970; 1.13]) ($P < 0.05$) and LVEF was significantly lower in LAD lesion (46.50% [43.00; 51.00]) than in the healthy group (55.00% [50.00; 58.75]) ($P < 0.01$). Max/Min value significantly increased from 0.83 [0.74; 0.93] to 0.92 [0.82; 0.99] ($P < 0.01$) following LAD PCI while significantly decreased from 1.63 [1.35; 1.98] to 1.35 [1.21; 1.43] ($P < 0.01$) post-RCA PCI. It did not vary significantly, however, either following LCX PCI or without intervention. LVEF significantly increased (from 46.50% [43.00; 51.00] to 49.00% [46.00; 51.00]) only after LAD PCI.

Conclusion: Max/Min parameter is suitable to follow patients after LAD and RCA PCI.

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Despite many preventive lifestyle and pharmacological interventions, ischemic heart disease (IHD) remained the leading cause of mortality in the developed Western world.¹ The desired decline in coronary artery disease (CAD) mortality necessitates the accurate diagnosis and assessment

of the ongoing pathological processes. Based on these considerations, medical, surgical or interventional treatment needs to be tailored for each individual patient. Even today, the 12-lead electrocardiogram (ECG) is an important and central tool in establishing the diagnosis of myocardial

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ischemia or infarction.² Patients with clinically significant CAD, however, may have frequently normal resting ECG.³ Acute coronary syndrome (ACS) may trigger ECG changes in its acute phase, nevertheless pathologic changes may be absent on the conventional 12 lead ECG in the more advanced stages of ACS. Usually, stable CAD does not trigger pathological resting electrocardiogram.³ Ischemia-induced electrophysiological alterations could be characterized often by simply minor potential changes.

Body surface potential mapping (BSPM) is a much more sensitive method.⁴ One of the unique clinical values of BSPM is its ability to reveal minor electrical potential losses appearing in the depolarization phase even if pathological changes are absent on the resting ECG.⁴⁻⁷ In addition, its usefulness to detect electrophysiological changes accompanying percutaneous coronary intervention (PCI) has also been described.⁸ Although CAD can be recognized more readily by ST-T changes (even with 12-lead ECG), these repolarization signs are varying.^{9,10} Investigation of the electric activity of the P wave with BSPM also enables the detection of CAD without exercise.¹¹

A significant shortcoming of BSPM is, however, that human expertise is required to interpret the maps. In order to enable a computer-based diagnosis, a new quantitative, powerful BSPM parameter, maximum to minimum ratio (Max/Min) has been introduced.⁷ Max/Min is defined by the ratio of the highest maximum to the deepest minimum potential of all leads recorded by BSPM (Fig. 1). In normal subjects, the Max/Min value equals to about 1.0. Max/Min is suitable to detect electrophysiological alterations during the depolarization period. It has been shown, that Max/Min indicates anterior (Max/Min $\leq$ 0.6) as well as posterior (Max/Min $\geq$ 1.25) Q-wave myocardial infarctions with good sensitivity and specificity.⁷ Max/Min parameter is able to differentiate anterior and posterior electrical potential losses because electrical events in the anterior heart region triggers higher potentials on the body surface due to eccentric position of the heart⁷ (Fig. 2). Max/Min parameter indicating significant anterior potential loss may be associated with impaired systolic left ventricular function.¹²

The aim of this study was to investigate the modification of Max/Min parameter following PCI in stable CAD. We hypothesized that Max/Min

signifies successful revascularization of major coronary arteries following PCI. We also hypothesized that alteration of this parameter depends on the coronary artery branch affected by PCI.

PATIENTS AND METHODS

The Institutional Review Board accepted the protocol of the study. Experimental subjects gave informed consent. Ninety-two adult Caucasian patients with stable CAD and positive exercise test referred to coronary angiography were enrolled consecutively. Seventy of patients (age, 59 ± 8 ; 46 males) were revascularized by PCI (left anterior descending [LAD] in 38, right [RCA] in 17 and left circumflex [LCX] coronary artery in 15). In multivessel coronary disease only the culprit lesion was revascularized.

Successful revascularization was defined as TIMI-3 flow of the distal artery following PCI. There was no intervention in 22 (age, 60 ± 8 ; 14 males) patients (control group for PCI). Thirty-five (35) healthy subjects (age, 58 ± 2 ; 15 males) were also investigated (controls for coronary angiography).

All clinical examinations were performed on patients with clinically stable state, without acute ST-T changes. No patient had a cardiac disorder other than ischemic heart disease. Exclusion criteria were atrial fibrillation, intraventricular conduction disturbances, and signs of ventricular hypertrophy/strain on the ECG.

BODY SURFACE POTENTIAL MAPPING AND ECHOCARDIOGRAPHY

Patients were examined with BSPM and transthoracic echocardiography (TTE) before and 4-40 days after coronary angiography. Max/Min value was evaluated in resting state by the 63-lead Montreal system BSMP (63 unipolar leads, amplifier: 0.05-200 Hz, signal averaging 52 seconds).¹³ Echocardiographic left ventricular ejection fraction (LVEF) was determined by Teichholz method.

STATISTICAL ANALYSIS

Statistical analysis was carried out using Sigma-Stat 2.03 (SPSS Inc., Chicago, IL, USA). One-way analysis of variance was used to compare mean of

Figure 1. Definition of the Max/Min value during depolarization. Maximum/minimum diagram is derived from the maximum and minimum potential values on the BSPM isopotential maps as a function of time. Max/Min is defined by the ratio of the highest to the deepest values on this diagram.

age of different groups, since these data sets passed both the normality and the equality of variances tests. Results of age data are reported as mean $\pm$ SD. Normal distribution of data and equality of variances were also tested on Max/Min and LVEF data set. These data failed at least one of these tests and therefore the results are reported as median with 25th and 75th percentiles shown in brackets, rather than as mean $\pm$ SD. In addition, nonparametric tests were used to analyze further these data.

Kruskal-Wallis one way analysis of variance on ranks was used to check the null hypothesis whether the median Max/Min value or the median LVEF of different treatment groups before PCI are not statistically different. An overall significance ($P < 0.001$) was established for rejecting the null hypotheses either for Max/Min or LVEF that the five treatment groups (LAD, LCX, RCA, without intervention, healthy) were not different.

Accordingly, pairwise differences between the groups were revealed by using Tukey's method of multiple comparisons. Wilcoxon signed-rank test with Bonferroni correction was used to test either Max/Min parameter or LVEF before and following coronary intervention. The null hypothesis was rejected with $P < 0.05$.

RESULTS

There was no statistical difference ($P = \text{NS}$) between the mean of age of different groups.

Before PCI, Max/Min value of patients with LAD lesion (0.83 [0.74; 0.93]) was significantly lower while RCA lesion (1.63 [1.35; 1.99]) was significantly higher than that of healthy group (1.01 [0.970; 1.13]) ($P < 0.05$). LVEF was significantly lower in the LAD (46.50 [43.00; 51.00]) than in the healthy group (55.00 [50.00; 58.75]) prior to PCI ($P < 0.01$).

Figure 2. Explanation of Max/Min value evolution in case of potential loss with different localizations. Summation of positive and negative potentials on the epicardial surface of the heart is always zero. In case of no potential loss, the summation of potentials on the body surface is also zero, therefore the Max/Min value is approximately 1. In case of anterior potential loss, however, the potential on the anterior epicardial surface is less than that on the posterior wall. In this case, owing to the eccentric position of the heart in the thorax, this negative potential value dominates the body surface, and the Max/Min value will be less than 1. In contradistinction, an inferior-posterior potential loss leads to a compensatory positive potential value originating on the anterior epicardial surface. On the body surface, this will result in Max/Min value greater than 1.

Max/Min value significantly increased from 0.83 [0.74; 0.93] to 0.92 [0.82; 0.99] ($P < 0.01$) following LAD PCI while significantly decreased from 1.63 [1.35; 1.98] to 1.35 [1.21; 1.43] ($P < 0.01$) post-RCA PCI. Max/Min value did not vary significantly either following LCX PCI (from 1.22 [0.99; 1.34] to 1.08 [0.87; 1.32], $P = \text{NS}$) or without intervention (from 0.80 [0.68; 1.17] to 0.80 [0.69; 1.12], $P = \text{NS}$). LVEF significantly increased from 46.50% [43.00; 51.00] to 49.00% [46.00; 51.00] following LAD PCI but did not following PCI of either RCA or LCX (Table 1, Figs. 3 and 4).

DISCUSSION

Data of the study population showed that stable CAD with significant coronary artery stenosis

precipitated pathologic Max/Min value. LAD lesion decreased, LCX lesion did not influence while RCA lesion increased Max/Min value relatively to the normal population. It was demonstrated that significant LAD stenosis was connected to the decrease of the LVEF.

Our original hypotheses have been verified, that is, Max/Min signified successful revascularization of major coronary arteries and alteration of this parameter depended on the coronary artery branch affected by PCI. Max/Min value was increased by LAD PCI while decreased by RCA PCI. Max/Min value was influenced neither by LCX PCI nor without intervention.

These results could be explained by the following.

It is well known that the magnitude of left ventricular systolic dysfunction is much more serious

Table 1. Change of the Max/Min Body Surface Mapping Parameter and the Left Ventricular Ejection Fraction (LVEF) in Ischemic Heart Disease Due to Percutaneous Coronary Intervention (PCI)

Coronary artery PCI	n	Max/Min Value			LVEF (%)		
		Before PCI	After PCI	P Value	Before PCI%	After PCI%	P Value
LAD	38	0.83 [0.74; 0.93]	0.92 [0.82; 0.99]	<0.01	46.50 [43.00; 51.00]	49.00 [46.00; 51.00]	<0.005
RCA	17	1.63 [1.35; 1.98]	1.35 [1.21; 1.43]	<0.01	55.00 [50.70; 59.00]	55.00 [50.70; 59.00]	NS
LCX	15	1.22 [0.99; 1.34]	1.08 [0.87; 1.32]	NS	56.00 [49.75; 58.00]	56.00 [54.25; 59.50]	NS
Without PCI	22	0.80 [0.68; 1.17]	0.80 [0.69; 1.12]	NS	50.50 [48.00; 59.00]	53.50 [49.00; 59.50]	NS
Healthy	35	1.01 [0.97; 1.13]	–	–	55.00 [50.00; 58.75]	–	–

The results are reported as median with 25th and 75th percentiles shown in brackets.

PCI = percutaneous coronary intervention; Max/Min value = maximum to minimum ratio; LVEF = left ventricular ejection fraction; LAD = left anterior descending coronary artery; RCA = right coronary artery; LCX = left circumflex coronary artery; NS = not significant.

Figure 3. Median (25; 75 percentile) Max/Min values of the different treatment groups before and after PCI. Before PCI, median Max/Min of the LAD group is significantly lower while that of RCA is significantly higher than that of healthy group. The change of Max/Min by PCI is significant in the LAD and RCA groups, in addition, trends toward normalization.

Figure 4. Median (25, 75 percentiles) LVEF values of the study groups before and after PCI. Before PCI, LVEF of the LAD group was significantly lower than that of healthy group. Following PCI, LAD group demonstrated a significant improvement of LV systolic function.

after anterior transmural myocardial infarction¹⁴ than in other localizations. Our results can draw the attention that this basic observation can be extended to an earlier phase of myocardial

ischemia, that is, prior to myocardial infarction. Increasing Max/Min values indicated an augmentation of the electrical activity of the anterior wall after LAD PCI, while decreasing Max/Min

values referred to decreased electrical activity of the anterior wall (due to the increased electrical activity of the posterior wall) after RCA PCI. LCX PCI has not modified Max/Min value significantly. This may be related to the fact that LCX can supply both the anterior and the posterior regions of the left ventricle, therefore successful PCI may cause increase in the electric potentials both of the anterior and the posterior region. These effects oppose each-other, and this balanced outcome may lead to an unchanged Max/Min value.

The pathologic alteration of the Max/Min value before PCI could be the hibernation of the myocardium produced by CAD causing minor electric potential changes¹⁵ during depolarization. It had been described earlier that R/S ratio of the QRS complex can change during PTCA. This phenomenon was explained by peris ischemic/intraischemic block.^{16,17} Our results may support the theory of the electrical viability, that is, the alteration of the Max/Min value toward normalization following successful LAD and RCA PCI. This process may also be associated with the restoration of the hibernated myocardium by the successful PCI. The accompanying metabolic restoration may also promote of electric and mechanical recovery, and may indicate the adjustment of Max/Min value toward normal direction, and the partial/total restoration of the LVEF, respectively.

Our results certified that BSPM parameter, Max/Min is a useful diagnostic tool for the assessment of patients with CAD without pathological resting electrocardiograms, even in stable and symptomless stage. It has been already known that Max/Min is a feasible parameter to detect myocardial infarctions with minor potential losses.¹² Novelty of the present observation is, however, that Max/Min parameter is also eligible for the follow-up of minor potential changes with transient or variable magnitude presumably due to hibernating myocardium. Consequently, the usefulness of BSPM has been confirmed in a novel diagnostic area. In addition, the above detailed application of the Max/Min parameter does not require special qualification such as the interpretation of isopotential maps since this parameter can be determined in an automated manner. It needs an uncomplicated maximum and minimum finding algorithm in an array of electric potential data and a simple calculation of a ratio. If the need of the development of a

computerized BSPM evaluation system comes up, the involvement of this parameter seems to be inevitable.

Concerning our results, we can bring up that monitoring of Max/Min parameter would be suitable to follow up of patients after PCI of LAD and RCA and this parameter may be able to indicate a restenosis noninvasively. To support this presumption, however, needs further investigation.

Limitations of the Study

- (1) The maximal potential during depolarization affecting the Max/Min value appears on the anterior-lateral surface of the chest surface. The maximal potential measured by the BSPM electrode may be less than the true value in women due to the potential decreasing (shadow) effect of the breast.
- (2) Only the culprit lesion was revascularized during PCI, however, the study population contained patients with two-vessel or multivessel coronary disease, as well. This fact has not influenced the direction or the magnitude of the change of Max/Min value after PCI. Concomitant LAD and RCA lesion, however, may have generated a Max/Min value closer to normal (1.0) than in the case of a single LAD or RCA lesion.
- (3) Wall motion abnormalities of the inferior or anterolateral wall, which do not affect LVEF determined by the quantification method, has been used in this study which may have led to biases. This may correlate with the finding that RCA or LCX lesion has not influenced the LVEF before PCI, in addition, neither PCI of RCA nor of LCX have improved the LVEF in the current study.

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REFERENCES

1. Go AS, Mozaffarian D, Roger VL, et al. Heart disease and stroke statistics—2014 update: A report from the American heart association. *Circulation* 2014;129:e28–e292.
2. Mirvis D, Goldberger AL. Electrocardiography. In: Mann DL (Ed). *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine*, 10th ed, Philadelphia: Elsevier/Saunders, 2014.
3. Martinez-Rios MA, Bruto Da Costa BC, Cecena-Seldner FA, et al. Normal electrocardiogram in the presence of severe coronary artery disease. *Am J Cardiol* 1970;25:320–324.

4. Green LS, Lux RL, Haws CW. Detection and localization of coronary artery disease with body surface mapping in patients with normal electrocardiograms. *Circulation* 1987;76:1290-1297.
5. DeAmbroggi L, Bertoni T, Breggi ML, et al. Diagnostic value of body surface potential mapping in old anterior non-Q myocardial infarction. *J Electrocardiol* 1988;21:321-329.
6. Horan LG, Sridharan MR, Flowers NC, et al. A basis for determining body surface potential patterns attributable to single-site coronary arterial occlusion. *J Electrocardiol* 1989;22(Suppl):99-106.
7. Medvegy M, Preda I, Savard P, et al. New body surface isopotential map evaluation method to detect minor potential losses in non-Q-wave myocardial infarction. *Circulation* 2000;101:1115-1121.
8. Preda I, Nadeau R, Savard P, et al. QRS alterations in body surface potential distributions during percutaneous transluminal coronary angioplasty in single-vessel disease. *J Electrocardiol* 1994;27:311-322.
9. Tseng YZ, Hsu KL, Chiang FT, et al. Characteristic findings of body surface potential map during ventricular repolarization in patients with coronary heart disease. *Jpn Heart J* 1999;40:391-404.
10. Wung SF, Drew B. Comparison of 18-lead ECG and selected body surface potential mapping leads in determining maximally deviated ST lead and efficacy in detecting acute myocardial ischemia during coronary occlusion. *J Electrocardiol* 1999;32:30-37.
11. Bauernfeind T, Preda I, Szakolczai K, et al. Diagnostic value of the left atrial electrical potentials detected by body surface potential mapping in the prediction of coronary artery disease. *Int J Cardiol* 2011;150:315-318.
12. Simonyi G, Szucs E, Bauernfeind T, et al. Judge of the impaired left ventricular systolic function from body surface mapping parameters. Abstract in 33rd International Congress on Electrophysiology; 2007.
13. Savard P, Ackaoui A, Gulrajani RM, et al. Localization of cardiac ectopic activity in man by a single moving dipole. Comparison of different computation techniques. *J Electrocardiol* 1985;18:211-221.
14. Pirolo JS, Hutchins GM, Moore GW. Infarct expansion: Pathologic analysis of 204 patients with a single myocardial infarct. *J Am Coll Cardiol* 1986;7:349-354.
15. Koch K-C, vom Dahl J, Wenderdel M, et al. Myocardial viability assessment by endocardial electroanatomic mapping: Comparison with metabolic imaging and functional recovery after coronary revascularization. *J Am Coll Cardiol* 2001;38:91-98.
16. Wagner GS, Selvester RH, Wagner NB, Krucoff MW. QRS changes during acute ischemia induced by balloon occlusion of the LAD artery. *J Electrocardiol* 1988;21(Suppl.):S18-19.
17. Wagner NB, Sevilla DC, Krucoff MW, et al. Transient alterations of the QRS complex and ST segment during percutaneous transluminal balloon angioplasty of the right and left circumflex coronary arteries. *Am J Cardiol* 1989; 63(17):1208-1213.
18. Medvegy M, Simonyi G, Szucs E, et al. The judge of the efficacy of percutaneous coronary artery intervention by a simple body surface potential mapping parameter. Abstract. *Circulation* 2010;122:e21.