Impact of the Heart Rate on Normal STT Integral Body Surface Potential Maps

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Abstract. *We suggested a method for localization of ischemic lesions from difference integral body surface potential maps (IBSPMs) computed by subtraction of IBSPM without manifestation of ischemia from the IBSPM during ischemia that can be obtained at rest and during the stress test. In this work the possible changes in IBSPMs of healthy subjects with increasing HR were studied. BSPMs were measured during the stress test in 12 subjects (age 30-53). During the test, the HR of each subject increased from the rest value up to the value higher than 85% of the value (220-age) at the top of the load. The IBSPMs were computed for 5 HR values up to the highest one. Correlation and power of IBSPMs at subsequent values of the HR were evaluated. The correlation coefficient between the IBSPM at rest and IBSPM at the top HR was higher than 70%. The root-mean-square value of the IBSPM at the top HR varied from 43% to 128% of the root-mean-square value of the IBSPM at rest. The existence of remarkable differences in STT IBSPMs at rest and at stress in healthy subjects should be considered when the IBSPMs during the stress test are evaluated for ischemic patients.*

Keywords: STT integral body surface potential maps, stress test, heart rate, local repolarization changes

1. Introduction

Local repolarization changes (e.g. during ischemia) are reflected in integral body surface potential maps (IBSPMs) of the STT interval [1]. In previous simulation study we suggested a method for identification and localization of local ischemic lesions from difference IBSPMs using the inhomogeneous torso model and the geometrical model of heart ventricles [2], [3]. The activation propagation was simulated by cellular automaton and ischemic lesions were modelled by shortening the action potential duration by 20% in selected areas of the ventricles. The difference IBSPM was computed by subtraction of IBSPM without manifestation of ischemia from the IBSPM during ischemia. It was supposed that for real patients such data can be obtained from ECG measurements performed before and during the stress test. However, in the simulation study all changes in IBSPMs were considered as the effect of repolarization changes. During a real stress test the HR considerably increases, so the question is how to compute the difference IBSPM correctly if each member of the subtraction is computed for significantly different HRs, thus for a different time interval.

The aim of this work was to study the behavior of IBSPMs in dependence on the HR for subjects with no heart disease history, whether we can suppose, that the changes in IBSPM during the stress test reflect only pathological repolarization changes or there are some changes that can be considered physiological.

2. Subject and Methods

Multichannel ECG recordings with 64 leads were performed during the stress test on 12 volunteers (age 30-53) with no heart disease history in Medical University Warsaw as it is described also in [4]. BSPMs from 64 measured leads were computed. Simultaneously, the 12-lead ECG was measured to evaluate the measured data in a standard way. During the test,

the HR of each subject increased from the basal value at rest up to the value higher than 85% of the value 220-age at the top of the load.

The IBSPMs from STT time interval were computed for each subject at the basal and at the highest HR as well as for three HR values in between (together in 5 levels of HR). The IBSPMs at each HR level were computed from signals averaged over intervals of 10 seconds [4]. The fiducial points Q, J and the end of T wave were set manually. To compensate for the changes in STT interval duration with increasing HR, the IBSPMs were normalized by dividing them by the length of the corresponding STT time interval. Then at each level of the HR the root-mean-square (RMS) signal of the normalized IBSPM was computed according the Eq. 1 as well as its correlation with the normalized IBSPM measured at rest (at the beginning of the exercise). The differences between normalized IBSPMs at observed levels of the HR were evaluated for each subject.

$$RMS = \sqrt{\frac{\sum_{i=1}^{n} x_i^2}{n}} \qquad (1)$$

Where x_i STT integral of the measured ECG signal in the lead i
n number of measured leads

Although the multichannel measurements of ECG are usually interpolated and visualized in regular rectangular grid (Fig. 2), in this work the evaluated parameters of IBSPMs were computed only from the measured signals (not from interpolated map values). IBSPM was handled as a multidimensional vector.

3. Results

The increasing HR shortened the QRST interval significantly from 100% to 66.3 ± 4.6%. The shortening of the STT interval was more dramatic – from 100% to 58.4 ± 5.9%, while the QRS interval shortened slightly from 100% to 92.7 ± 7.4%. All next parameters were computed for the normalized IBSPMs.

The mean value of the correlation coefficient between the IBSPM at rest and IBSPM at the top of HR was 88.1 ± 9.5% (70% – 98%) (Fig. 1).

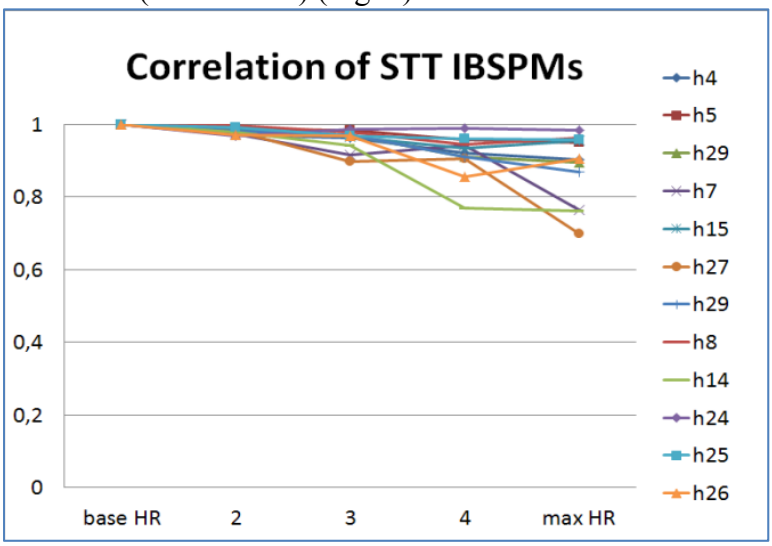

Fig. 1. Correlation coefficient between the IBSPM at rest and IBSPMs computed at 4 values of HR up to the maximal reached value of HR at the top of the load for 12 measured subjects.

The RMS value of the IBSPM at the top HR varied more significantly for different subjects from 43% to 128 % (mean 79.7 ± 36.4%) of the RMS value of the IBSPM at rest (Fig. 2).

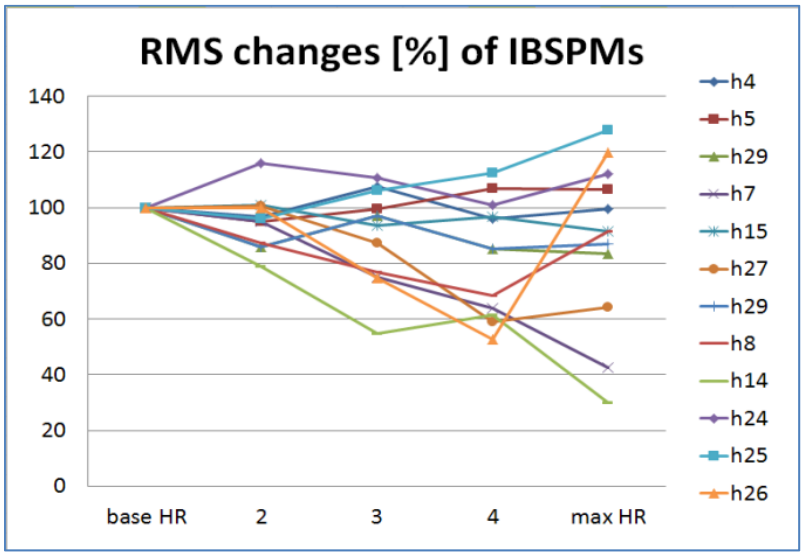

Fig. 2. Percentage of RMS values of IBSPMs for 4 values of HR in comparison to the RMS value of IBSPM at the beginning of the exercise.

In Fig. 3 the examples of changes in IBSPMs during the stress test are depicted.

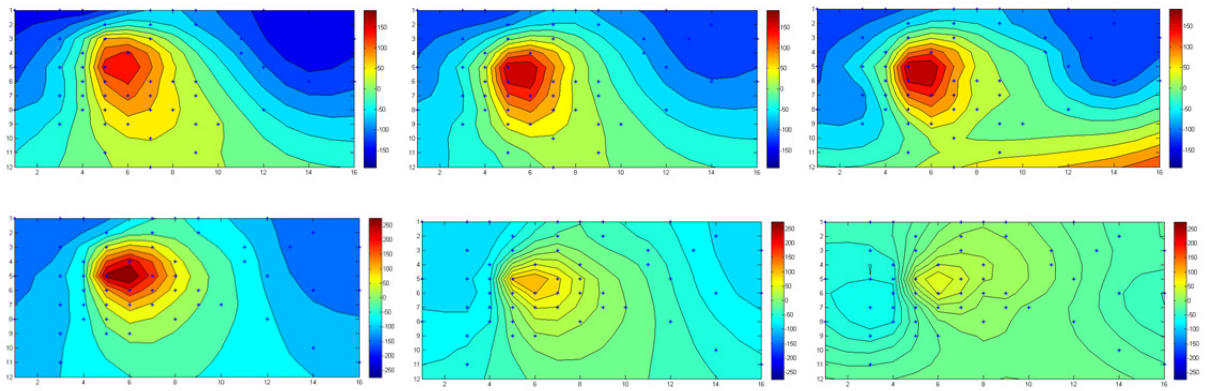

Fig. 3. IBSPMs at the beginning of the exercise (left), at the 3rd level of HR (middle) and at maximal value of HR (right) for the subject h15 with minimal changes of observed parameters (top) and for the subject h14 with maximal changes of observed parameters (bottom). Asterisks mark the measured points.

4. Discussion and Conclusions

Remarkable differences in STT IBSPMs at rest and during the stress test were observed in healthy subjects, so the hypothesis that all changes in STT integral BSPMs during the stress test can be considered pathological was not confirmed.

We can suppose that there are additional physiological factors influencing the ECG signal during increasing the HR that reflected in RMS signal of BSPMs.

The measured signals for higher HR were very noisy because of the presence of myopotentials and moving artefacts. In some cases the signal was completely damaged and was excluded from evaluation. If the number of "bad" electrodes varied in different levels of HR for one particular subject, the final evaluation and comparison of observed parameters was made only for leads that had produced acceptable signal in all HR levels. Better quality of signals could be obtained if the stress is evoked by pharmacological or mental load [6].

The limitation of the study was small number of measured subjects and also the age of subjects (30 – 50 years). The results from younger population would be desirable.

For ischemic patients, similar changes in IBSPMs that were observed in healthy subjects during the stress test can be associated with repolarization changes. From the obtained results it implies that for ischemic patients we cannot assign all changes in IBSPM during the stress test to the pathological repolarization changes and it is questionable whether the simple difference IBSPM computed from the IBSPM at rest and at the top of the load can be used as input data for inverse localization of possible ischemic lesions. Additional properties of IBSPMs should be searched for distinguishing between normal physiological changes and pathological repolarization changes during the stress test.

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