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Dependence of the Vulnerability Index on the Heart Cycle Length

J. MARTINKA, K. KOZLÍKOVÁ

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

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Summary

Index of vulnerability is a parameter based on ventricular gradient evaluating the risk of arrhythmia development. The index is derived from isointegral maps of the QT interval. Individual characteristics of isointegral maps are influenced by different factors, which contribute to the relatively high variability among measured parameters of maps in measured subjects. While several electrocardiographic indexes have been introduced, there are only few studies of their dependence on heart rate. In this study we set out to establish the dependence of vulnerability index on the RR interval or heart rate in healthy population. A positive linear correlation between RR intervals and mean and minimum values of vulnerability indexes was found.

Key words

Body surface potential • Heart rate • Ventricular gradient • Isointegral map • Vulnerability index • RR interval

Introduction

Heart rate can influence the risk of arrhythmia development since it probably influences the dispersion of duration of the action potential (APD) across the heart. Experimental studies confirmed a significant increase with cycle length prolongation for transmural dispersion (Antzelevitch *et al.* 1999).

Electrocardiographic body surface mapping offers the possibility for evaluation of the risk of development of arrhythmia. It can be simply the number of extrema in the QRST isointegral map (IIM, Gardner *et al.* 1986, Martinka 2006) or more sophisticated, vulnerability index based on comparison of IIM QRST of a test subject with IIM QRS and IIM ST-T of a control (Urie *et al*. 1978). We wanted to find out whether there is any correlation between the vulnerability index changes and the heart rate of the examined person.

Methods

We constructed IIM QRS, IIM ST-T and IIM QRST for 106 young people of 18–25 years of age (47 women, 59 men). None had a history of cardiovascular disease and they had ECG without pathological changes. We calculated the vulnerability indexes for each subject as a test, while all other subjects served as controls.

Data for body surface mapping were registered using the limited 24-lead system after Barr (Barr *et al*. 1971) and processed using the ProCardio mapping

Fax +420 241 062 164
E-mail: physres@biomed.cas.cz http://www.biomed.cas.cz/physiolres

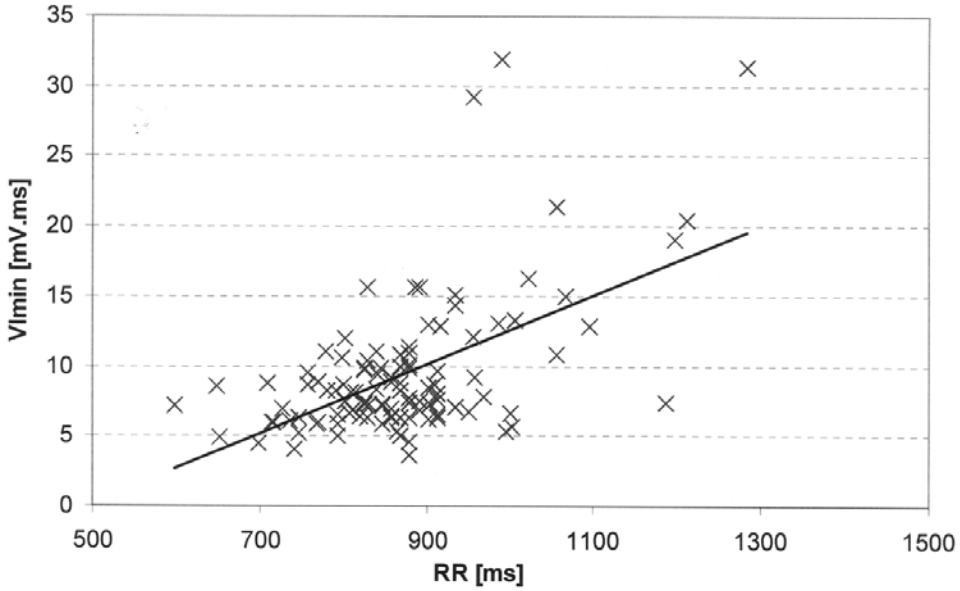

Fig. 1. Linear dependence of VI_{min} on the RR interval

system (Rosík *et al*. 1997). Data were registered in supine position. Linear baseline was taken through TP segments. The onset and offset of the QRS complex and the T wave was established manually from the root mean square signal (Kozlíková 1990). Isointegral maps were constructed as distributions of potential time integrals over chosen time intervals.

The vulnerability index is constructed using IIM QRST as follows: IIM QRST of the subject to be tested is compared with all subjects of the control group and the best matching map is determined (the map with the smallest total QRST area difference of all leads). The difference between the test map and the best match from the control group is shown as the "vulnerability map" according to the following equation for each point of the map:

$$V = IIM\ QRST_{test} - (IIM\ QRS_{control} + \alpha \cdot IIM\ ST\text{-}T_{control}) \quad (1)$$

where α is determined by minimizing the squared difference:

$$d(V^2)/d\alpha = 0 \quad (2)$$

Coefficient α has to fulfil the condition:

$$-1 \leq \alpha \leq 1 \quad (3)$$

The vulnerability index VI is calculated as the square root of the sum of the squares of all values contained in the vulnerability map

$$VI = (\Sigma Vi2 / n)^{1/2} \quad (4)$$

where V_i is the value of vulnerability map in the i-th point, i = 1, 2, …, n.

In an ideal situation, the same subject may serve as a test (under arrhythmic conditions) and as a control (under physiological conditions). In practice, this is usually not possible. Therefore, the best matching map of a different control subject serves for comparison. In this study we used the minimum vulnerability index (VI_{min}), which is the lowest value of VI obtained from all comparisons of the tested subjects with control subjects, and mean vulnerability index (VI_{mean}), which is the average value of all comparisons of the tested subjects with control subjects. Each comparison that was in compliance with equations (1) and (2) was considered.

Therefore, the aim of this study was to find out whether there exists any correlation between the vulnerability index and the RR interval duration representing the heart rate.

Results

The average value ± standard deviation of the RR interval was 872 ± 114 ms (the corresponding heart rate was 69.9 ± 8.7 bpm). The average value of the VI_{min} was 9.45 ± 5.00 mV.ms, for the VI_{mean} it was 15.81 ± 5.15 mV.ms.

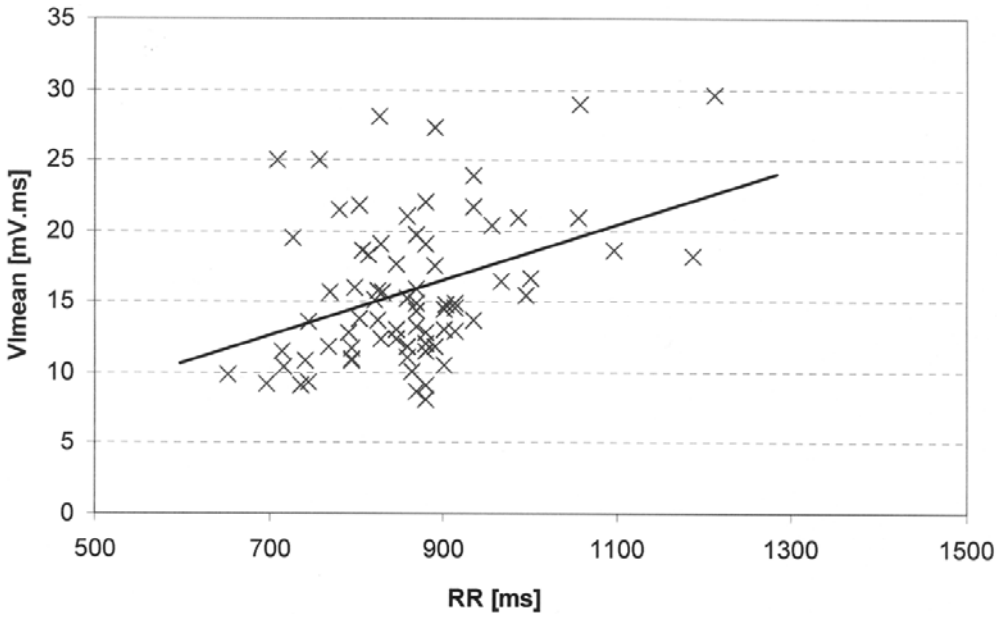

Fig. 2. Linear dependence of VI_{mean} on the RR interval

The vulnerability index in both cases increased with increasing value of the RR interval. We found statistically significant linear correlations between VI_{min} and RR (Fig. 1)

$VI_{min} = -12.12 + 0.025 \times RR$

with correlation coefficient $r = 0.562$

and between VI_{mean} and RR (Fig. 2).

$VI_{mean} = - 1.0049 + 0.0195 \times RR$

with correlation coefficient $r = 0.386$. A better fit for VI_{mean} was the exponential dependence

$VI_{mean} = exp(1.671 + 0.0012 \times RR)$

with correlation coefficient $r = 0.392$ (no statistically significant difference against the linear dependence).

Discussion

The value of VI depends only on the repolarisation sequence and does not depend on the depolarisation sequence (Urie *et al.* 1978). Previous studies showed that the dependence of QT interval on the RR interval is linear (Smetana *et al*. 2004). If the heart rate was influenced only by the action of the sinoatrial node, it would not necessarily cause any increase of the ventricular gradient. However, if a lower heart rate increases the dispersion of action potential duration across the heart, it can also change the repolarization sequence, which can increase the risk of arrhythmia development. It was found that the prolongation of the cycle length was linked to the occurrence of Torsade de pointes as well as to the QT interval prolongation at slow heart rates (Sicouri and Antzelevitch 1991), which is in accordance with our results.

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Corresponding author

K. Kozlíková, Institute of Medical Physics, Biophysics, Informatics and Telemedicine, Faculty of Medicine, Comenius University, Sasinkova 2, 813 72 Bratislava, Slovakia. E-mail: katarina.kozlikova@fmed.uniba.sk.