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Influence of individual torso geometry on inverse solution to 2 dipoles ☆

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Abstract

Background: The purpose of this study was to observe the influence of variety in individual torso geometries on the results of inverse solution to 2 dipoles.
Methods: The inverse solution to 2 dipoles was computed from the measured data on 8 patients using either standard torso with various shapes and sizes of the heart and lungs in it or using various outer torso geometries with the same inhomogeneities. The vertical position of the heart relative to the fourth intercostal level was kept constant in all models. The results were compared with the reference solution computed in standard torso.
Results: The inverse solution was influenced in 4 of 8 cases by changes of torso geometry and only in 1 of 8 cases by changes of internal inhomogeneities.
Conclusions: The use of individual torso geometry with the knowledge of the true heart position is very important for correct inverse results.

Keywords: Ischemic lesions; Inverse solution; Individual geometry of torso

Introduction

Coronary artery disease (CAD) is one of the most frequent cardiac problems. In early stages, it can be recognized only during physical or mental exercise and when it presents in a form of a subjective chest pain or objective changes in ST segment of measured electrocardiographic (ECG) signals. Therefore, the ECG stress test is generally used for the detection of possible presence of ischemic lesions. It was shown that the use of multiple leads measurements—body surface potential mapping (BSPM)—gives more information about the cardiac electrical generator than 12-lead ECG.[1,2] Ischemia causes significant changes in myocytes action potential (AP) shape and amplitude,[3] which, in turn, influence BSPMs. Simulation studies showed that the localization of the ischemic changes can be obtained from the difference QRST integral BSPMs,[4] computed by subtraction of QRST integral BSPM, which is computed at the manifestation of ischemia, from the QRST integral BSPM, which is computed during normal activation. A method for identification of 1 ischemic lesion from the difference QRST integral map using the inverse solution to 1 dipole was proposed in Tysler et al.[5] Analogically to this approach, a method for searching 2 ischemic lesions by an inverse solution to 2 dipoles was developed and applied to real measurements of BSPMs in 8 patients with CAD.[6] The inverse solution was computed in inhomogeneous torso model adapted from the Dalhousie torso model[7] (standard model). The aim of the present study was to study the influence of the geometry variability of individual torso surface and internal torso inhomogeneities on the results of the inverse solution.

Materials and methods

Inverse solution

The BSPM can be represented by the vector of body surface potentials $m(t)$ at the time instant t in selected points on torso surface. It can be computed as the product of the time-independent transfer matrix A and the equivalent multiple dipole heart generator $g(t)$:

$$m(t) = A.g(t) \tag{1}$$

The transfer matrix A represents distribution of conductivity and geometry of the torso volume conductor; $g(t)$

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represents equivalent multiple dipole electrical generator of the heart, in a particular time t of heart activation. The QRST integral of BSPM is computed as

$$p = \int_{QRST} m(t)dt = \int_{QRST} A.g(t)dt = A. \int_{QRST} g(t)dt = A.s \quad (2)$$

where s represents the time integral of dipole moments of modeled multiple current dipoles.

The computation of difference integral map (DIM) Δp by subtracting the normal integral map p_n from the integral map at ischemia p_i is then equivalent to the computation of an integral map for a difference integral multiple dipole generator Δs

$$\Delta p = p_i - p_n = A.s_i - A.s_n = A.(s_i - s_n) = A.\Delta s \quad (3)$$

where s_n and s_i are integral multiple dipole sources in the normal and ischemic myocardium correspondingly. We can assume that Δs reflects integral multiple dipole generators representing only the changes of the electrical activity in ischemic lesion.

The difference QRST integral maps were used as input data for identification of ischemic lesions. In the inverse solution, a pair of dipoles as an equivalent generator (EG) of the modeled ischemic lesions was identified from computed DIM. The inverse solution was calculated for all possible positions of pairs of dipoles located in predefined points evenly spaced within the modeled ventricular myocardium (N dipoles, $N.[N\text{-}1]/2$ dipole pairs). The solution was found using singular value decomposition of submatrix A_{sub} of the transfer matrix A. The submatrix A_{sub} was made of columns of matrix A corresponding to predefined position of the selected dipole pair. The EG (integral dipole moments for a pair of dipoles) was then computed as

$$EG = A^{+}_{sub}.\Delta p \quad (4)$$

where A^{+}_{sub} is a pseudoinverse of the corresponding submatrix.

As the transfer submatrix for each pair of dipoles was strongly overdetermined, a unique solution in the sense of minimum least-squares criterion was always obtained. To choose the best representative EG, the criterion of the minimum of the root mean square difference (RMSDIF) between the original DIM Δp and the map r generated by the inversely estimated EG was used.

$$RMSDIF = \frac{\sqrt{\sum_i (\Delta p_i - r_i)^2}}{\sqrt{\sum_i \Delta p_i^2}} \quad (5)$$

where i is the index of vectors Δp and r.

Identification of ischemic lesions

Inversely estimated EG was chosen according the criterion of global minimum of RMSDIF. However, there were several results (EGs) with RMSDIF varying very slightly from the global minimal value. Therefore, in addition, the results with RMSDIF within 1% difference from the best solution were analyzed to watch the stability of the solution.

The modified K-means clustering method based on Euclidean distance between the dipoles was applied on all analyzed dipoles to divide them into 2 clusters. The iterative algorithm started using the dipole positions of dipole pair with the smallest RMSDIF value as the initial positions of the cluster centers. The dipoles from the next analyzed dipole pair were then assigned to the cluster with the nearest cluster center. Because the dipoles from 1 pair should represent different ischemic lesions, they should belong to different clusters. If both dipoles were assigned to the same cluster, the pair was excluded from evaluation for the actual iteration step. At the end of every iteration step, the new cluster centers were recalculated from assigned dipoles, and the next iteration step was started. If no more changes occurred during the iteration step, the algorithm finished dividing dipoles into 2 clusters. Because the result of K-means clustering iterative procedure is, in general, dependent on starting points, all analyzed pairs were, step by step, used as starting centers of desired clusters, and the results were compared. If the results differed for different starting points, the best solution was chosen that had the least number of analyzed dipole pairs excluded from created clusters. The final gravity center of each cluster was then calculated, and the mean dipole moment computed from all dipoles in the cluster was used to represent the ischemic lesion.

Torso geometry

We obtained the geometrical data from Prof A. van Oosterom with permission of Dr R. Hoekema.[8] The data of 25 realistically shaped torso models (15 men and 10 women) reconstructed from magnetic resonance images were used in the study. The models of the heart, lungs, and ventricular cavities were also included in each individual torso model. The positions of 64 leads of the Amsterdam leads system were assigned to each torso. The lead V_2 was positioned on each subject with respect to the fourth intercostal level. In the present study, the influence of the variability of the individual torso model in comparison with standard model configuration on the results of the inverse solution to 2 dipoles was investigated.

Real ECG data

The proposed method for identification of small ischemic lesions was applied to real measured data from 8 patients (men in age from 38 to 66 years; mean, 56 years) with CAD who underwent stress test. The high-resolution ECG signals were recorded from 64 leads on the torso surface at rest and during exercise.[9] The patients underwent the stress test on supine ergometer with stepwise increasing load from 25 to 150 W.

The BSPMs were computed from averaged signals[10]; the 2 points for linear baseline correction were set by point Q and at the end of U wave. All fiducial points were determined manually from root mean square signal computed from all measured leads in all sampling time instants. Difference QRST integral maps were computed by subtraction of the integral maps measured at rest from the integral maps measured at load 75 W. The heart rate during exercise increases considerably; thus, the length of QRST interval