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3D HEART SIMULATION AND ANALYSIS

Sándor Miklós SZILÁGYI

Faculty of Technical and Human Science
Sapientia – Hungarian Science University of Transylvania
Tirgu Mures (Marosvásárhely), Romania
e-mail: szs@ms.sapientia.ro

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Abstract

This paper presents a new way to solve the inverse problem of electrocardiography in terms of heart model parameters. The developed event estimation and recognition method is based on an optimization system of heart model parameters. An ANN-based preliminary ECG analyzer system has been created to reduce the searching space of the optimization algorithm. The optimal model parameters were determined by minimizing the objective functions, as relations of the observed and model-generated body surface ECGs. The final evaluation results, validated by physicians were about 86% correct. Starting from the fact that input ECGs contained various malfunction cases, such as Wolff-Parkinson-White (WPW) syndrome, atrial and ventricular fibrillation, these results suggest that this approach provides a robust inverse solution, circumventing most of the difficulties of the ECG inverse problem.

Keywords: Heart modelling, inverse solutions, pathological case analysis, 3D simulation.

Introduction

The most important health problem affecting large groups of people is related to the malfunction of the heart, usually caused by heart attack, rhythm disturbances and pathological degenerations. One of the main goals of health study is to predict these kind of tragic events, and by identifying the patients situated in the most dangerous states, it is possible to apply a preventing therapy.

Computerized ECG analysing systems, most of which are based on recognition and clustering algorithms, have to recognize any potentially dangerous arhythmia with at most a few seconds delay. Recent signal processing techniques apply the mixture of time-frequency analysis (Fourier, wavelet, cosine transforms), sample-based (neural networks) and parameter estimation (regressive models).

Unfortunately, at the moment the main obstacle is that there are too many possible ECG waveforms that can differ a lot from patient to patient. Because a knowledge-based database cannot cover all cases, the precision of the analysis may decrease in the case of rare waveforms.

The application of large databases can cause the improvement of the precision, but the following disadvantages are still held:

1. It is impossible to store all possible wave forms;

2. If the size of the database grows, the clustering algorithm becomes more and more complicated, it will be hard to develop and maintain;
3. Real-time learning is impossible during the clinical practice at the moment (PC's are still too slow). The algorithm might take wrong decisions in case of unknown waveforms.

These disadvantages lead to the idea, that the development of a heart-model-based system is necessary.

Creating a heart model is basically practical [15] due to the fast that the computer, when applying traditional signal processing algorithms, recognizes lots of waves, but it does not really 'understand' what is happening. This can be avoided only if the computer knows the origin, the formation of the ECG signal [4]. During signal processing, if the traditional algorithm finds an unrecognizable waveform, the model-based approach is activated, which tries to estimate the causes of the encountered phenomenon (e.g. quick recognition of ventricular fibrillation) [7].

The main goal of the inverse problem of ECG is to characterize and reconstruct cardiac electrical events from measurements. In contrast to the forward problem of the electrocardiography, the inverse problem does not possess a mathematically unique solution. Another not easily by-passable problem is its ill-posed nature whereby the desired inverse solution is unstable and may oscillate widely with the slightest perturbation.

Body surface potential mapping (BSPM) was developed to allow an almost complete data acquisition from the body surface. BSPM may have a great advantage over the standard 12-lead system in different situations due to deeper accessible information. MIRVIS [5] has shown some cases of BSPM recordings that clearly demonstrate the inadequacies of the standard ECG lead sets in a variety of pathologies. As we know more about the depolarization – repolarization mechanism, we can understand in a better way the internal function of the heart.

This paper presents an event recognition study performed with ECG signal analysis and 3D heart model. The main purpose is to evaluate the strengths and weaknesses of each method, and to analyse the co-operation efficiency in malfunction diagnosis.

1. Materials and Methods

1.1. Study Records

The first signal resource was a 32-electrode measurement (BSPM) database (sampled at 1000 Hz with 12-bit resolution) obtained from the Research Institute for Technical Physics and Materials Science (MTA-MFA) of Budapest.

These registrations contain various malfunction cases as WPW syndrome, atrial and ventricular fibrillation, flutter. These measurements were collected using Lux-32a [3] electrode placement, as presented in *Fig. 1*.

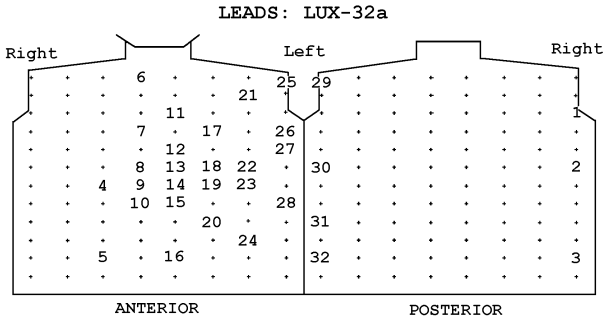

Fig. 1. Lux-32a electrode placement.

From this 32-lead electrode subset a total of 192 leads were created by estimation of missing channel data. This process is based on a statistical procedure focused to allow a low estimation error for the 160 unmeasured leads.

In the second stage of the study we used 12-lead ECG registrations from our database. These signals were sampled at 500-1000 Hz with 12-bit resolution.

1.2. The Approach of ECG Inverse Problem

In contrast to methods that directly solve the matrix equation linking electrical sources with electrical potential fields to estimate ECG inverse solution, our approach indirectly obtains the solvent in terms of heart model parameters. The schematic diagram of the method is presented in *Fig. 2*.

The preliminary ECG analyser system (PAS) is based on detailed human anatomical and functional a priori knowledge, developed using an ANN, tested and validated by physicians in clinical environment. In this study the PAS was used to obtain initial information on the site of origin of cardiac activation. The output of the ANN provides the initial heart model parameters. Then the BSPMs or 12-lead ECGs were simulated by the ECG generator unit, and the objective functions that assess the similarity between the measured and simulated signals were determined.

The heart model parameters are adjusted with the aid of optimization algorithms or eventually physicians. The simulation procedure is performed until the objective functions satisfy the given convergence criteria. Finally the parameters are validated.

1.3. ANN-based Preliminary ECG Analyser System

Application of a priori knowledge to reduce the searching space of heart model parameters is quite important. The PAS was developed to determine roughly the

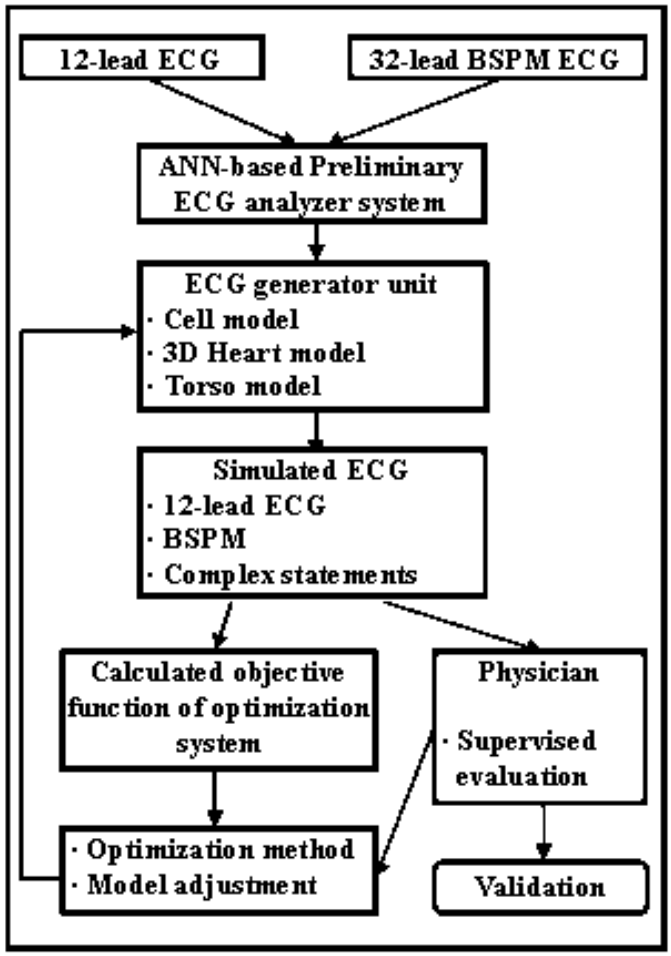

Fig. 2. Schematic diagram of the heart-model-based ECG analyser method.

cardiac status and state, which was than used to initialize the model parameters and decrease the searching space for the optimization system.

In the present study the PAS was developed based on a three-layer feed-forward ANN as shown in *Fig. 3*. This ANN is capable of mapping the nonlinear input-output relationship, to a desired degree of accuracy. An adaptively weighted coefficient algorithm was used to train the ANN. The number of neurons in the input layer was set to 192, corresponding to the number of body surface electrodes used in the present simulation study. The hidden layer had 125 neurons that were determined heuristically. The output layer had 512 neurons, which corresponded to 32 ventricular myocardial segments of computer heart model: *MS-1* to *MS-32*.

Sixteen cardiac cellular units were selected for each of 32 myocardial segments in the ventricles, and each of these 16 x 32 = 512 sites was then paced in the forward simulation using the computer heart-torso model, generating the dataset for training the ANN.

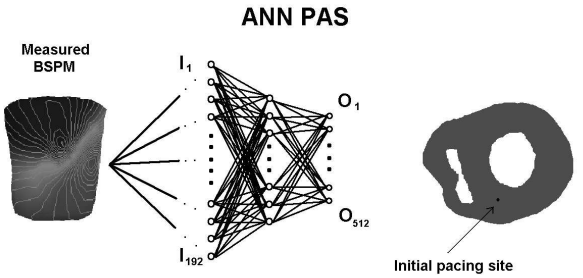

Fig. 3. The block diagram of the preliminary analysis system based on a three-layer feed forward ANN. The input to the PAS is the BSPMs at 192 body surface electrodes over time, and the output from the PAS is the region inside myocardium, which provides the initial pacing site in the parameter optimization. The input, hidden and output layers of the ANN have 192, 125, and 512 neurons, respectively.

1.4. The Structure of the Combined Heart Model

Based on a simulation and a measured ECG signal, the estimation of the internal state of the human body is a considerably complicated task, requires the solution of several theoretical and practical problems. Theoretically the model should contain all those factors that should be handled considerately during implementation, but that is practically impossible. If the required information is not available, the values of the unknown parameters should be determined empirically. As the amount of available information grows, the model can become more and more sophisticated. The theoretical model determines the internal structure of the human body on three levels.

1.4.1. Level of the Myocardial Cell

The ion channels (Na+, K+, Ca++, Mg++, etc.), pumps, and transporters are included in this model-layer as they determine the function of each cell. Unfortunately, the precise mechanism of these units is not known [15]. When we study the behaviour of a simple myocardial cell, it is likely to consider the character of the currents, which appear during depolarization.

Let I_m be the sum of the currents that flow through the cell membrane of unitary surface. Then

$$I_m = I_{cap} + I_{ion} + I_{ext} = C_m \cdot dV_m/dt + \sum (I_{ch} + I_{transp} + I_{pump}) + \sum I_{env} \quad (1)$$

where I_{cap} is the capacitive (due to potential difference from the cell membrane surfaces), I_{ion} the ionic (sum of currents generated by channels, pumps and transporters), and I_{ext} the current coming from the external environment. C_m is the electrical capacity of a cell membrane with unitary surface, V_m is the potential difference between the two sides of the membrane.

1.4.2. Anatomy of the Heart and Torso.

There are many possible different heart structures. To describe various representative cases we studied our breast MRI records (42 examples) and some CT images. These samples allowed us to construct a morphological heart structure for simulation.

The geometry model of the heart and torso was constructed by tetra meshes. The torso, lung, endo- and epicardial surfaces were divided into 2344, 3834, 7780 and 8942 tetrahedrons. The heart model could have a spatial resolution of 0.5 mm that means more than one million individual compartments at highest decomposition. For a better simulation result we choose a time-slice between 0.1ms and 2ms over the whole ventricular excitation cycle.

1.4.3. Position of the Surface Electrodes

The structure of the chest, its position in space, the relative position and distance of the compartments with respect to the electrodes, and the electrical behaviour of the chest's contents must be known.

As the model has to take into consideration extremely numerous parameter values, the problem cannot be solved in a deterministically way (we have much more unknown values then known equations). That is why a stochastically method (genetic algorithm, adaptive neural networks and fuzzy systems) should be applied to determine the values of the parameters [7, 8].

1.5. Mathematical Description of the Model

The heart is represented by a set of finite homogenous elements, called compartments. Since their size is obviously much larger than that of actual biological cells, these units effectively represent small cubic groups of biological cells, and must

capture their macroscopic behaviour rather than the microscopic behaviour of individual cells. Microscopic inter/intracellular interactions (such as ionic flow across membrane boundaries) are thus not modelled.

Compartment connectedness may be defined as the set of rules that establish which units are considered to be directly connected to a given one for the purposes of electrophysiological simulation, such that myocardial activation may be directly propagated between them. These rules establish what is called a neighbourhood for each unit.

The cubic compartments are arranged in a 3-D rectangular grid, so each unit has six locations with which it shares a common cubic face (mono-axial neighbours), twelve locations with which it shares only an edge (biaxial neighbours), and eight locations with which is shares only a corner vertex (tri-axial neighbours).

The neighbourhood size for a given preparation is configurable from the following choices:

- 18-element neighbourhood Activation may be conducted to up to six axial neighbours and twelve biaxial neighbours.
- 26-element neighbourhood Activation may be conducted to up to six axial neighbours, twelve biaxial neighbours, and eight tri-axial neighbours.

Each compartment was considered homogenous, constructed by only one type of tissue with well-defined properties, such as: cell type, cell state, cell activation potential (AP) function. The type of cells determines the electrical propagation properties, but no additional considerations were taken in, such as tissue fiber torsion and so on. The environmental parameters such as 4D position (x, y, z spatial coordinates and time), conduction speed of stimulus, weight and connection with neighbour structures, localize each unit.

Because the main ion channels situated inside the cells have a quite complicated behaviour (with lot of unknown parameters), the activation potential function of the compartment was considered as basic input parameter (we determine an AP function with static shape for each cell type and state). Due to contractions of the heart, respiration, and other disturbing phenomena, the position of compartments was considered time-varying. The following mathematical expressions that describe compartment behaviour are time variant.

Let V_K be the potential of an arbitrary compartment

$$K \quad V_K(t) = AP(T, S, t - \tau_K),$$

where τ_K is the time the stimulus needs to reach the compartment K. The activation potential function that varies from cell type T and state S, has a short delay τ_K due to activation propagation until compartment K.

If we study the measured potential E_j, generated by compartment K_i then:

$$E_{j,K_i}(t) = V_{K_i}(t) \cdot R_{E_j,K_i}(t) - E_{GND,K_i}(t) \tag{2}$$

where, $R_{E_j,K_i}(t)$ represents in time the resistance from compartment K_i to electrode E_j. Using bipolar electrodes, the value measured on the reference electrode E_{GND} will be subtracted.

As all compartments have an accession to the measured potential on each electrode, the measured voltage on electrode E_j will become the sum of each $E_{E,K_i}(t)$ generated by compartment K_i:

$$E_j(t) = \sum_{i=0}^{N-1} \left[V_{K_i}(t) * R_{E_j,K_i}(t) - E_{GND,K_i}(t) \right], \tag{3}$$

where $N$is the number of compartments.

These equations determine the measured electrical potentials and the inner mechanism in the heart. During the simulation, these voltages were determined for each compartment and electrode for every time-slice (especially between 0.1ms and 2ms). In the following it is presented, how simulations were antagonized by different environmental factors (muscle fiber elasticity, distortion, different structural abnormalities, etc.). Although these disturbing factors limit the overall performance of the simulation, with pragmatic algorithm organization these effects could be minimized.

1.6. Applied Tools and Mathematical Methods

At the moment, testing the theoretical model is possible only if the values of unknown variables are determined empirically. This compromise must be taken in all cases, where the estimation error is greater then the statistical error occurring when the empirically determined values are applied.

In order to speed up the calculations, a relatively small number of compartments have been applied (18779). In the case of this resolution (considered quite low for such simulations) it is not suitable to consider, that the compartments have the same size and identical shape, so a space-window has been introduced for each compartment. This window is an arbitrarily sized rectangular prism determined by spatial variables x, y, z and time variable t.

The internal structure of each compartment is considered homogenous, with equal density, so the weight is directly proportional with the volume (determined in case of rest state).

2. Results

During simulation, a parameter classification algorithm was applied to distinguish the normal QRS complexes from the abnormal ones, in order to determine the specific differences between the normal and abnormal parameter values.

Fig. 4 presents a cell activation simulation that was effectuated and visualized after 200ms of the rises of sinoatrial node excitation. In the right part of the image the activation time of cell is visualized. Each slice has 10mm distance from other, so totally a five centimeter wide ventricular tissue is visualized.

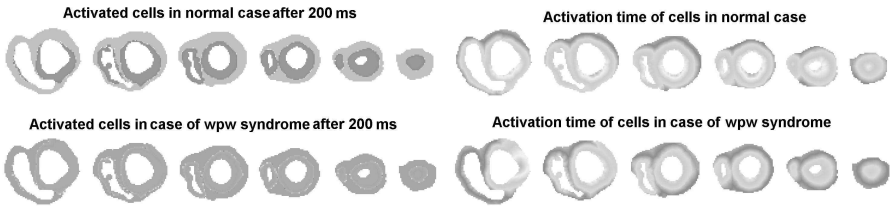

Fig. 4. The simulated depolarization in normal and abnormal case (bypass tract)

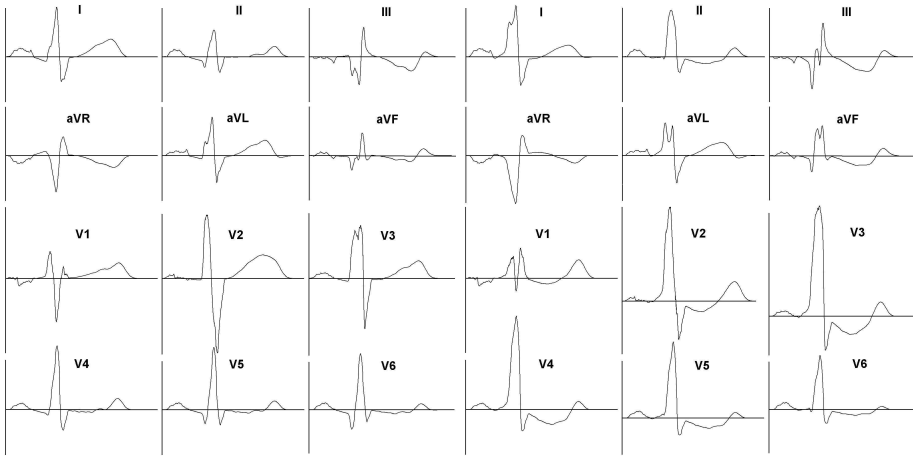

Fig. 5. The simulated ECG signal in normal and abnormal case (bypass tract)

For normal cases the detection ratio is practically 100%. During the simulation by using the initial parameter set for a normal and abnormal situation, we obtained the signals presented in *Fig. 5*.

Fig. 6 represents the simulated surface potentials t=41ms after the start moment of ventricular depolarization.

Table 1 shows the correctness of simulation for different cases. The evaluation of the simulated results was made by physicians. The performance was determined as the ratio of correct and total decisions.

3. Discussion

As *Table 1* showed that the 3D heart simulation [9, 10] could deal in most cases, such as WPW (Wolf Parkinson White) syndrome, pre-excitations, and tissue activation modelling. The cases of ectopic beats and triggered events represent the weak points of the simulation model.

The application in practice of the model has several obstacles, which can be

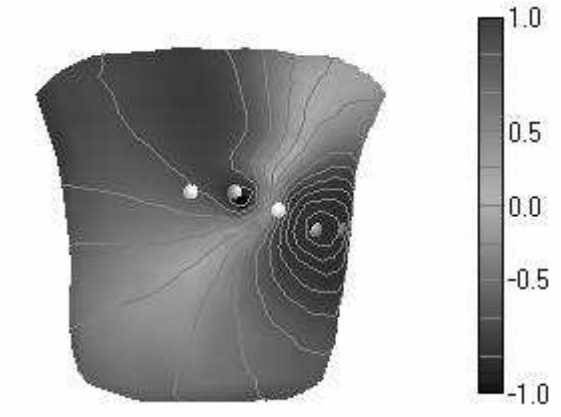

Fig. 6. The simulated surface potentials

Table 1. Simulation performance for various pathological and normal cases

Pathological case	Decision number	Failed decisions	Performance
Normal	44	1	97.72%
Ectopic beat	21	4	80.95%
WPW syndrome	14	2	85.71%
Atrial flutter	22	2	90.90%
Atrial fibrillation	18	2	88.88%
Ventricular fibrillation	19	1	94.73%
Re-entry mechanisms	19	3	84.21%
Triggered activity	36	5	86.11%
Aberrant ventricular conduction	21	3	85.71%

classified into the following groups:

- Effects of internal and external perturbations (such as environment, sympathetic and parasympathetic despondence);
- Lack of information on elements of the model;
- Lack of technical background.

3.1. The Deficiencies of the Model

Several problems could be conceptualized, but the most important disturbing deficiencies are:

- The processes performed inside the cells are not well known, the behaviour of the studied components cannot be determined with an acceptable precision;

- In critical cases, if a group of cells does not get the necessary food, it changes its behaviour. A model created to simulate the normal behaviour of the cell will not simulate it correctly in abnormal case;
- Because the structure of the heart differs from patient to patient, this structure is not known a priori, it has to be determined in real-time, based on the available information;
- The determination of the torso's structure introduces the same problem. It is hard to determine the electrical conductivity and precise position of its elements.

3.2. Perturbation Phenomena

In case of human system identification the most important disturbing phenomena are:

- It is known, that respiration makes the heart change its shape and position. Although the motion of the heart can be tracked, it is not possible to determine the amplitude of the motion from the ECG;
- The continuous motion and displacement involve very hard problems. Because the motion has an effect on the behaviour of all internal elements, the behaviour of the heart will also be modified. The model has to follow the changes of the cell properties. For example: a resting man suddenly jumps out of the bed. The controlling mechanisms start their adjustment, the values of model parameters will change;
- Fever and respiration frequency can also cause alterations;
- External events (the patient senses something annoying or pleasant) change the dependence between the previously measured signals and the determined parameters. This is one of the causes why the perfect simulation of a human body is impossible. These factors strongly influence the behaviour of the SA node. As the mechanism is not precisely known, the values of the model parameters cannot be determined in a deductive way.

4. Conclusions

Regarding the fact, that computerized ECG [11, 12] diagnostics refer to several medical and technical problems, at the moment it cannot be applied as a stand-alone system [1, 2, 6, 13, 14]. The short-term solution is the application of fuzzy systems and systems based on multi-agents that make it possible, based on empirical information, to accomplish an adaptive advising system based on continuous transitions.

If a hybrid system (neuronal-fuzzy and model-based approach, simultaneously) is built, it may become possible to learn the model via the knowledge of the traditional advising system, which, after a suitable learning process, will be able to replace gradually the old system.

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References

[1] BENYÓ, Z.– CZINEGE, L., Computer Analysis of Dynamic Systems with Application in Physiology, *Proc. World Congress of IMACS on Scientific Computation, Modeling and Applied Mathematics, Berlin, III.*, 1997, pp. 663–668.

[2] BOERSMA, L.– MORAN, E. G.– MONT, L.– BRUGADA, J., Accessory Pathway Localization by QRS Polarity in Children with Wolff-Parkinson-White Syndrome,*Journal Cardiovasc Electrophysiol,* **13/12** (2002), pp. 1222–1226.

[3] LUX, R. L. – SMITH, C. R. – WYATT, R. F.– ABILDSKOV, J. A., Limited Lead Selection for Estimation of Body Surface Potential Maps in Electrocardiography, *IEEE Trans. BME*, **25** (1978), pp. 270–276.

[4] MACLEOD, R.S.– BROOKS, D. H., Recent Progress in Inverse Problems in Electrocardiology, *IEEE Eng. in Med. and Biology*, (1998), pp. 73–83.

[5] MIRVIS, D. M., Validation of Body Surface Electrocardiographic Mapping, *Kluwer Academic Publisher*, Boston-Dordrecht-London, 1988, pp. 63–74.

[6] SHAHIDI, A. V.– SAVARD, P.– NADEAU, R., Forward and Inverse Problems of Electrocardiography, Modeling and Recovery of Epicardial Potentials in Humans, *IEEE Trans. Biomed. Eng.*, **41/3** (1994), pp. 249–256.

[7] SZILÁGYI, S. M., Event Recognition, Separation and Classification from ECG Recordings, *Proc. IEEE Conference on Engineering in Medicine and Biology, Hong Kong*, **20.**, 1998, pp. 236–239

[8] SZILÁGYI, S.M., Non-Linear Adaptive Prediction Based ECG Signal Filtering, *Proc. IEEE Conference on Engineering in Medicine and Biology, Atlanta*, **21.**,1999, p. 286.

[9] SZILÁGYI, S. M.– BENYÓ, Z. – DÁVID, L., Heart Model Based ECG Signal Processing, Modelling and Control in Biomedical Systems, *Proc. 5th IFAC Symposium on Modelling and Control in Biomedical Systems*, Melbourne, 2003, pp. 213–217.

[10] SZILÁGYI, S. M. – BENYÓ, Z. – DÁVID, L., WPW Syndrome Identification And Classification Using ECG Analysis, *Proc. World Congress on Medical Physics and Biomedical Engineering*, Sydney, 2003, 4423.pdf.

[11] SZILÁGYI, S. M., The Limits of Heart-Model-Based Computerized ECG Diagnosis, *Proc. World Congress on Medical Physics and Biomedical Engineering, Chicago*, **20** 2000, pp. 1913–1916.

[12] SZILÁGYI, S. M. – SZILÁGYI, L. – FRIGY, A.– INCZE, A., Holter Telemetry in the Study of Heart Rate Variability, *Romanian Heart Journal*, **2/6** (1996), p. 143.

[13] SZILÁGYI, S. M. – SZILÁGYI, L. – DÁVID, L., ECG Signal Compression Using Adaptive Prediction, *Proc. IEEE Conference on Engineering in Medicine and Biology*, Chicago, **19**, 1997, pp. 101–104.

[14] SZILÁGYI, S. M. – SZILÁGYI, L. – DÁVID, L., Comparison between neural-network-based adaptive filtering and wavelet transform for ECG characteristic points detection, *Proc. IEEE Conference on Engineering in Medicine and Biology*, **19**, 1997, pp. 272–274.

[15] THAKER, N. V. – FERRERO, J. M., Electrophysiologic Models of Heart Cells and Cell Networks, *IEEE Eng. in Med. and Biology*, (1998), pp. 73–83.