Spatial Modeling of the Wolff–Parkinson–White Syndrome Induced Ventricular Fibrillation

Sándor M Szilágyi[1], László Szilágyi[2], Constantin T Luca[3], Dragoş Cozma[3], Gabriel Ivănică[3], Călin Enăchescu[1]

[1] Petru Maior University, Tîrgu Mureş, Romania
[2] Sapientia University of Transylvania, Tîrgu Mureş, Romania
[3] Cardiology Center, Timişoara, Romania

Abstract

Aims: The goal of this study is to assess the influence of the accessory pathway's (AcP) location and its repolarization period on the incidence of ventricular fibrillation (VF), in order to develop a non-invasive method able to select the most endangered patients that suffer from Wolff–Parkinson–White (WPW) syndrome.

Methods: 12-lead ECG was recorded from 79 patients suffering from WPW (aged between 9 and 71 years) at the Cardiology Center of Timişoara (Romania), and the insertion place of AcP-s were determined using Arruda localization method. We developed a spatio-temporal computerized model of the whole heart. Using a high spatio-temporal resolution we modeled the paroxysm of atrial fibrillation (AF) that in the presence of an AcP caused a catastrophically rapid ventricular response with degeneration to VF.

Results: Irrespectively of the AcP location, in presence of low AcP repolarization period (under 200 ms) the VF was developed for all 79 cases (average simulation time of 2 minutes 17 seconds using five stimulations per second).

Conclusion: Low repolarization period is the most important danger to VF for patients suffering from WPW, while the connection place and the location of AcP has relatively reduced imperilment impact.

1. Introduction

The Wolff–Parkinson–White (WPW) syndrome is a serious disorder of the heart's electric conduction system that may produce pre-excitations. Most patients that suffer from WPW remain asymptomatic during their lives, but there exists a risk to produce deadly irregular ventricular excitation.

The WPW syndrome is characterized by the presence of one or more accessory pathways (AcP) (by-pass tract) between the atria and ventricles that may conduct parallel with the atrioventricular (AV) node – His bundle conduction pathway, but usually faster [1, 2].

The electric signals conducted by these AcP's may stimulate the ventricles earlier than normally, thus enforcing it to contract prematurely. An irregular ventricular contraction renders more difficult the heart's pumping function. Another source of danger consists in the altered ventricular depolarization and repolarization. As an accessory AV connection can conduct in both directions, the presence of these by-pass tracts may predispose to atria-ventricular re-entrant tachycardia. Moreover, in the setting of atrial fibrillation, the WPW syndrome may cause a catastrophically rapid ventricular response with degeneration to ventricular fibrillation (VF).

Electrocardiographically, the WPW syndrome can be characterized by a specific sinus rhythm pattern. Other specific features may appear, such as paroxysms of re-entry tachycardia (the incidence in the young adult population suffering from WPW, it is about 10% and rising with age to 30%), more rarely paroxysm of atrial fibrillation (20–30% of patients having the syndrome), or atrial flutter [3, 4].

The localization of the AcP has important role, as it is a non-invasive procedure and may help to select the most endangered patients [5]. The selected patients should be further investigated by a semi-invasive process to determine the repolarization period of the AcP. A low AcP repolarization period highly increases the chance of VF development.

The higher incidence of VF represents a high risk of live threat, as in developed countries VF is the leading reason of cardiac death. Despite decades of intensive research, the mechanisms responsible for VF are only partially discovered [6].

The main reason of slow progress represents the partially understood heart excitation and contraction

functioning. The dysfunction of electrical impulse propagation may develop cardiac arrhythmias that perturb pumping activity [7]. Despite significant progress in the visualization of the heart's electrical dynamics, many details of arrhythmias are still unknown. To properly understand the behavior of the cardiac conduction system and pumping function under pathological conditions, several long duration measurements have to be performed.

In the last decades several studies have suggested that in the presence of a functional obstacle in cardiac muscle, a depolarizing wave (DW) may form a reentry circuit. It is believed that these reentry waves develop cardiac fibrillation [8].

It is essential to correctly distinguish the onset of arrhythmias that may cause fibrillation on various heart structures, from those ones that do not favor these events [9]. Cardiac robustness depends on details such as the heart's size [10], geometry [11], mechanical [12] and electrical state [13], anisotropic fiber structure [14], and inhomogeneity [15].

The main goal of this paper is to assess the influence of the AcP's location and its repolarization period on the incidence of VF, in order to develop a non-invasive method able to select the most endangered patients that suffer from WPW syndrome.

The rest of the paper is organized as follows: Section 2 gives a detailed description of the cardiac excitation and contraction for normal and in presence of WPW. Section 3 presents and discusses several aspects of the WPW and the results of simulations. In Section 4, the conclusions are formulated.

2. Methods

We started our investigations from the results and considerations published in an earlier paper [16], where it has been concluded that the efficiency of the localization method – described in Arruda's work [17] – significantly depends on the location of the AcP. All measurements involved in the study were effectuated at the Cardiology Center of Timişoara.

The initial population of the research consisted of 85 patients having at least one AcP. From these patients 6 had more than one AcP, whose location could not be determined by Arruda localization method (it can be used to localize only one AcP). For each patient (aged between 9 and 71 years) that suffered from WPW, a 5-10 minutes long 12-lead ECG was recorded, and the insertion place of AcP-s were determined using Arruda [17] localization method.

The knowledge of the spatio-temporal propagation of the DW could have an important role in selecting the most endangered patients, but cannot be determined by non-invasive medical apparatus. This investigation can be realized indirectly by computerized simulation and visualization programs.

To enhance the available visualization performance, we developed a spatio-temporal computerized model of the whole heart that handles half millimeter sized compartments using 1μs time step.

The developed modeling tool can determine the electrical and mechanical properties of cardiac tissue. Each cardiac cell type has specific properties. In order to simulate various pathological phenomena, the modeling tool can handle inhomogeneous tissue, laminar sheets, and multiple excitation and speed differences for base-apex gradient.

To perform real-time simulation using a strong PC, but not a supercomputer, we had to use homogenous cellular compartments, where the size of compartments and the temporal resolution of the simulation may be modified adaptively. It is considered that the finest spatial and temporal resolution is needed in the depolarization waves' frontline due to the fast voltage rise caused by fast sodium current [9].

To enhance simulation speed, during simulation the compartments are be adaptively modified for each cardiac cell type. The state of each compartment is modeled separately, so the differences for normal and pathologic cases can be visualized permanently. This model allows describing the electrical and mechanical behavior of each compartment. The connections among compartments are a priori determined, so we can properly model the propagation of the depolarization wave and the mechanical contraction of the compartments.

Several higher level parameters are included in cardiac tissue modeling. The connection among compartments varies in both space and time. For example, ventricular muscle conducts the depolarization wave much slower than atria, but in the presence of cardiac muscle injury in the atria, the conduction speed may decrease drastically or even may drop to zero.

Several time- and state-dependent tissue-related parameters were involved in our model. These parameters greatly influence the anatomy-related tissue parameters, such as fiber direction, anisotropy, average depolarization period, laminar sheets and spontaneous cell inhomogeneity. The used component models enable us to determine the electrical excitation and mechanical contraction of the cardiac muscle, thus supporting the volumetric analysis for atria and ventricles.

In this study, the tissue level excitation mechanism is based on Fast's work [18], while the activation potential is based on Luo-Rudy II (LR) model [19-20]. In this stage, each tissue element works as a secondary generator element. These elements can generate a depolarization wave if the adjacent elements are repolarized; otherwise, the wave propagation is swooned [21].

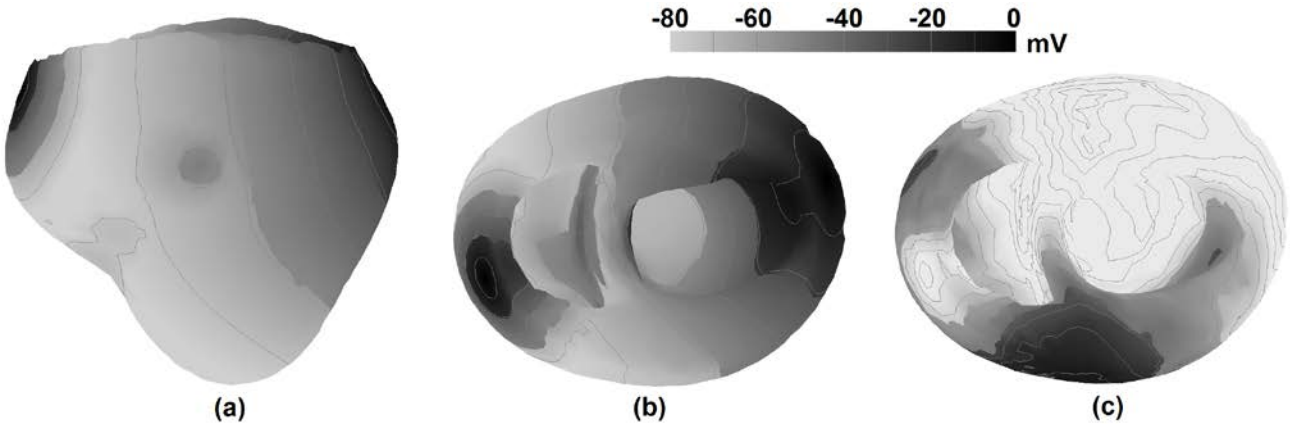

Figure 1. Surface potential of the ventricular tissue in presence of WPW syndrome is presented. Image (a) uses anterior view and present an excitation of a right lateral AcP. In image (b) and (c) we see upper view of ventricles, having right lateral AcP in (b) and right anterior AcP in (c).

In our simulation we applied half millimeter sized compartments and 1μs time step. This minimal spatio-temporal step can be increased if the compartment is not in the fast depolarizing phase. In the presence of irregular contraction the minimal spatio-temporal step was only slightly increased, while in normal situation the increment was considerably larger.

For the case of healthy cardiac functioning we employed the effect of muscle fiber direction (the ratio between longitudinal and transversal conductivity varies from 2 to 10), normal and minimal depolarization period (considered 80-250 ms), laminar sheet effect (in-sheet transversal conduction 2-5 times faster than trans-sheet conduction), and cell inhomogeneity (using conduction speed differences for base-apex gradient (5%-20%), transmural epicardial–endocardial gradient (5%-35%), left-right ventricular gradient (5%-15%)).

Using this high spatio-temporal resolution we modeled the paroxysm of atrial fibrillation (AF) that in the presence of an AcP caused a catastrophically rapid ventricular response with degeneration to VF. In our simulation the upper chambers made 250 to 500 regular and irregular contractions per minute while the ventricles were stimulated 83 to 350 times. The AcP-s were placed in 14 different locations, having a 150-500 ms repolarization period and conducting the excitation wave 20-50 ms faster than the normal atrioventricular (AV) node – His bundle pathway.

3. Results and discussion

Figure 1 visualizes the surface potential of the ventricular tissue in the presence of WPW syndrome.

Irrespectively of the AcP location, in the presence of low AcP repolarization period (under 200 ms), the VF was developed for all 79 cases (average simulation time of 2 minutes 17 seconds using five stimulations (ST) per second). We have to mention that in case of multiple AcP the VF may develop much easier. We consider that in the presence of long duration and fast excitation (as happens in case of atrial fibrillation), a rapidly repolarizationing AcP in short time may develop VF.

Table 1. The probability of VF occurrence in function of AcP insertation place and ST frequency. All values were determined for 2 minute simulated period.

Location and ST frequency	1	2	4	6
Endocardial AcP	0 %	0 %	9 %	60 %
Epicardial AcP	0 %	0 %	7 %	42 %
Intermediate AcP	0 %	0 %	7 %	45 %
Central AcP	0 %	0 %	6 %	39 %

The mass of prematurely excited ventricular tissue caused by AcP highly depends on the location and insertion place. The endocardium contains Purkinje cells that conduct the excitation up to 10 times faster than normal ventricular cell, so a large volume of cells can be prematurely excited.

Ventricular tissue inhomogeneity may further alter the depolarization-repolarization process, so it increases the appearance of VF.

In the presence of ischemia the low-conducting ventricular tissue deflected the DW and generated perturbations on the wave front; the strength of deflections are directly proportional with the size of low-conduction area, so a higher injured tissue size mean higher VF risk.

The low depolarization period represents a possible danger by increasing the time window, when a pathological excitation may occur.

Low DW conduction speed of the VT also increases the depolarization period. Both above mentioned factors

produce longer depolarization, so reentry waves may form more easily.

In case of AcP connected to endocardium the VF appeared 15% faster than for AcP connected to epicardium or to intermediate ventricular tissue. Central AcP locations with average repolarization time (greater than 270 ms) reduced the occurrence of VF by 7%.

4. Conclusion

Computerized simulation of cardiac tissue represents a non-invasive visualization tool that helps to understand the inner cardiac depolarization-repolarization process in normal and various pathological cases. An adequate simulation platform may help to select the most endangered patients by a non-invasive method that can enhance the efficiency of computerized health care.

Low repolarization period is the most important danger to VF for patients suffering from WPW, while the connection place and the location of AcP has relatively reduced imperilment impact.

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Address for correspondence:

Sándor Miklós Szilágyi
Str. Frantz Liszt nr. 8, 540068 Târgu Mureş, Romania
szsandor72@yahoo.com