

Interactive Simulation of Multiple Beats: A New Feature of ECGSIM

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Abstract

ECGSIM is an interactive open source software package aimed at supporting ECG education and research. In this package, the user can change the local transmembrane potential interactively the resulting ECG changes are simulated and displayed instantaneously. In the previous versions of ECGSIM only a single heartbeat can be simulated, limiting its possibilities in inspecting changes in ECG morphology over time.

This contribution introduces a new version in which changes in a sequence of heart beats can be displayed. The new feature is demonstrated in a Brugada patient.

The ECG of this patient has been recorded during the administration of increased doses of Ajmaline a sodium channel blocker that evokes the typical Brugada pattern in the ECG. The resulting changes in cardiac activity and corresponding ECG are shown in ECGSIM.

1. Introduction

Teaching the relation between the electric activity of the heart and the resulting ECG signal waveforms is difficult if just based on ECG textbooks. To support the understanding of the ECG *ECGSIM* has been developed and made freely available [1]. Within *ECGSIM* the user can interactively change the local transmembrane potentials on the heart surface and observe instantaneously the changes in the ECG signals anywhere on the body surface.

In the latest version of *ECGSIM* (3.0) a new feature has been added which enables the creation of multiple heart beats. Thus a rhythm strip can be created, potentially very useful in ECG education. This feature also offers the possibility to show the changes in ECG signals morphology under different circumstances, e.g. during progressing ischemia.

In this paper the new capabilities of *ECGSIM* are

described. The new functionality is explained by one of the existing *ECGSIM* cases and a new Brugada patient *ECGSIM* case file.

2. Materials

Healthy subject data

The data of a young healthy male are used to show the new functionality of *ECGSIM*. These data have been described previously [2, 3].

Brugada patient data

Body surface potentials (65-lead ECG) were recorded on a Brugada patient, a 49-year male. For this patient, MRI-based geometry data were recorded, from which the individualized volume conductor model was constructed, incorporating the effect of major inhomogeneities in the conductive properties of the thorax, i.e. the lungs, the blood-filled cavities and the myocardium on the transfer between the electric sources and body surface potentials. The geometries of the ventricles were reconstructed from the MRI data at peak QRS.

Continuous ECG data was recorded during infusion of a sodium channel blocker (Ajmaline)[1] in 10 bolus infusions of 10 mg, administered at approximately 1-min intervals (see Table 1) [2]. For each bolus administration representative ECG beats were selected for further analysis.

Table 1. The beats selected for analysis and the time between the administration of the next bolus of Ajmaline.

Beat #	time between 2 boluses [s]	Beat #	time between 2 boluses [s]
0	baseline		
1	60	6	70
2	70	7	55
3	50	8	65
4	70	9	65
5	90	10	45



Figure 1. Screen shot of ECGSIM showing multiple heart beats for the normal young male. The atria are activated from the left superior pulmonary vein using the previously described method to create activation waves [3]. The atrial rhythm was fixed 300 ms mimicking an atrial flutter. The ventricular intervals were set to 700 and 720 ms respectively. These coupling intervals may be set by adding either an atrial or a ventricular waveform, as indicated by the arrows.

3. Methods

ECGSIM is based on a biophysical model that links the local transmembrane potential (TMP) at the surface bounding the myocardial tissue to the ECG on the body surface. It includes atrial, ventricular and torso geometries, reconstructed from human magnetic resonance images [3,4].

The source model used is the equivalent double layer (EDL). This expresses the entire electrical activity within the atria or ventricles by means of a double layer source situated on the closed surface bounding either the atrial or the ventricular myocardium. For any position on the surface (node), the time course of the local EDL source strength is taken to be proportional to the TMP, $V_m(t)$, of the nearby myocytes [5, 6]. Six parameters are used to describe the TMP analytically. Any of these, including the local timing of local depolarization, can be changed interactively.

Simulation of multiple atrial or ventricular waveforms

The original *normal young male* case was used to create the ECG shown in figure 1. The new tab 'AV conduction' allows the user to add or delete either atrial or ventricular waveforms (activation and repolarization) to the ECG. In the example 6 atrial waveforms were

added at 300 ms after the previous activation. For the ventricles 2 more waveforms were added at 700 and 720 ms, respectively. The rhythm mimics an atrial flutter originating from the left pulmonary vein with a 2:1 AV conduction to the ventricles.

The TMP parameters on the heart of a certain ECG waveform can be changed after moving the time marker (yellow line in the ECG panel) to that specific part of the ECG.

Simulation of Brugada-type ECG

To be able to create an ECGsim case matching the recorded ECG for the Brugada patient the corresponding local parameter setting had to be specified. As a sodium channel blocker was administered to the patient, a global reduction in the propagation velocity can be anticipated, with a more pronounced reduction in the right ventricular outflow tract (RVOT) area [6]. The ST segment of the ECG leads over the RVOT shows major shifts during increased levels of the sodium channel blocker (figure 2).

A non-linear parameter estimation procedure was used to find the source parameters from the measured ECGs [2]. For each ventricular ECG beat the same initial activation estimate was used. The method used for obtaining the initial estimate is described in detail in van Dam et al [2].

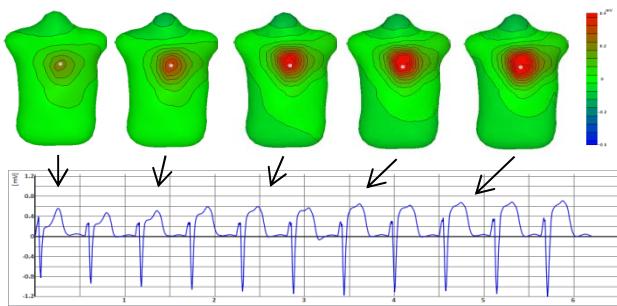


Figure 2. Bottom panel: The 11 ECG beats with increased levels of Ajmaline. The first beat shown is at baseline, every next beat is after the infusion of 10 mg Ajmaline. Upper panel: The body surface potential distributions at the J points of each second beat (arrows). The elevation of the ST segment over the RVOT is clearly visible as an increased positive area (red zone)

Additionally the TMP amplitude distribution on the heart surface was derived from the amplitude at the user defined J-point. The underlying reason for reducing the TMP amplitude is the notion that the capacity of the cardiac cells to depolarize is reduced, as the effect of the sodium channel blockers shows. In *ECGSIM* this effect can be modeled by reducing the local TMP amplitude.

Subsequently, using the ST segment derived local reduction of the TMP amplitude, the initial depolarization and repolarization estimates were optimized such that the resulting simulated ECGs matched the measured ECG.

3. Results

The results of the TMP amplitude estimate obtained by the inverse procedure from the 11 ECG beats are shown in figure 3. Note that the TMP amplitude is already somewhat reduced near the pulmonary valve prior to the start of Ajmaline infusion (< 5%). However, the estimated TMP amplitude drops up by 25% at the latest doses.

The depolarization times for each beat are shown in figure 4. In this figure the excessive local delays in the activation times are clearly visible in the RVOT region. The additional delay in activation time due to the increased dose in Ajmaline reaches up to 100 ms.

Within the *ECGSIM* program this delay can easily be inspected by selecting one of the 11 signals and the activation times on the heart surface. An example of the ECG as can be viewed within *ECGSIM* is shown in Figure 2. The corresponding body surface potentials are displayed by moving the time cursor to the selected point on the ECG. The estimated TMP parameters used may be viewed on the heart surface.

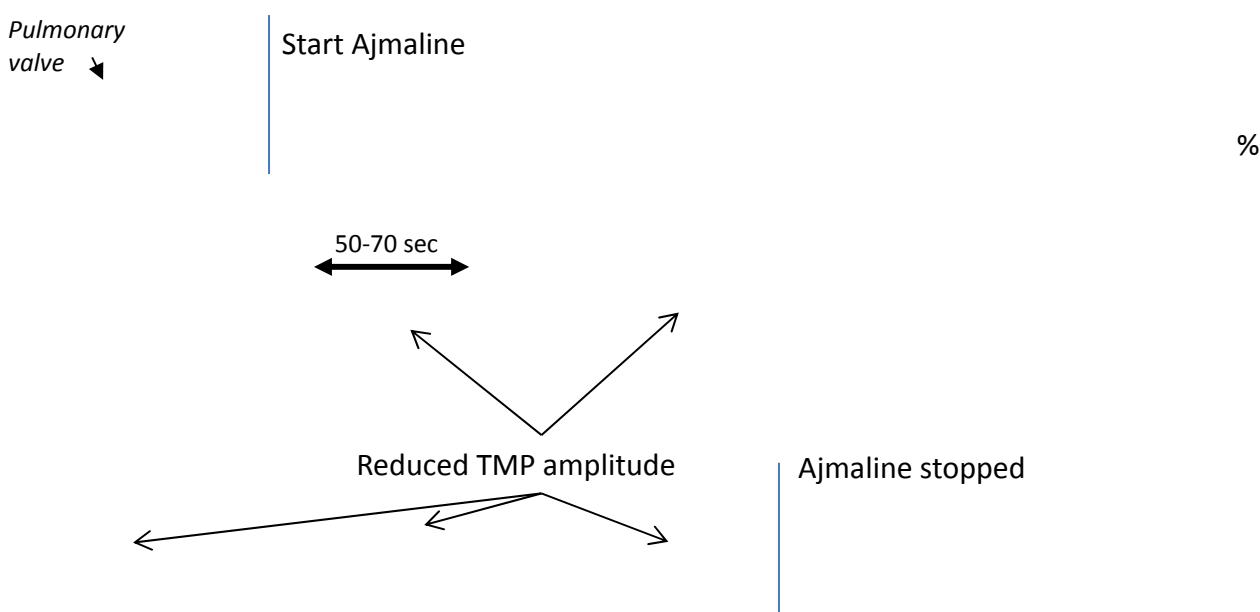


Figure 3. The reduction in transmembrane potential (TMP) amplitude of the 11 ECG beats following the administration of Ajmaline. An increased level of Ajmaline reduced the local TMP amplitude up to 25% in the right ventricular outflow tract area region. Lines are drawn at every 2.5%.

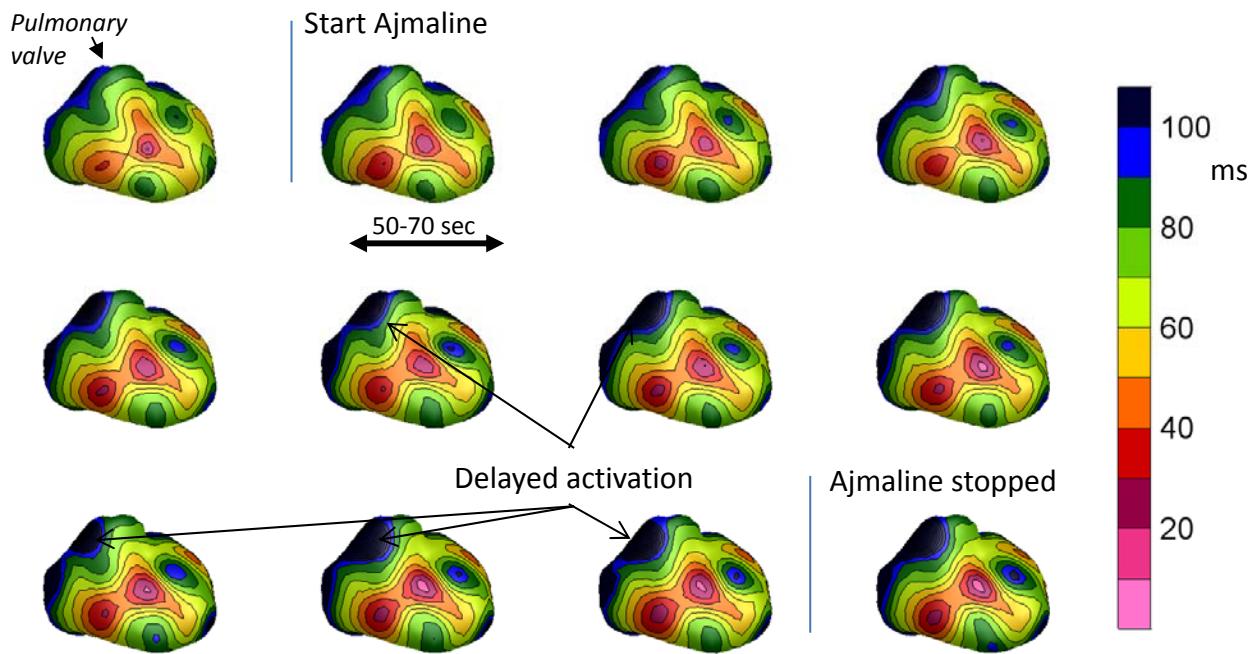


Figure 4. The activation times of the 11 ECG beats after administration of Ajmaline. A remarkable local activation delay was estimated in the RVOT area at increased levels of Ajmaline. Isochrones are drawn every 10 ms.

4. Discussion

The educational possibilities of *ECGSIM* are considerably extended by the new feature to create rhythm strips. Its potential is demonstrated by the simulation of atrial tachycardia and the typical changes in the ECG of a Brugada patient elicited by the administration of Ajmaline. As before, the clinical data used for the new cases will be made available at the ECGsim website (www.ecgsim.org). We feel confident that these new applications will increase even further the interest in ECGSIM and stimulate further the present collaboration with other educators and researchers in identifying new applications.

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