A New Approach for Mapping Electrical Conduction in Ventricular Tachycardia

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Abstract

Detection of slow electrical conduction areas is crucial for providing an effective ablation therapy in ventricular tachycardia. To this aim local activations and their duration should be accurately identified. Currently mapping systems identify the precocity or lateness of a local activation with respect to a fixed reference without considering its duration. In this study we developed an automatic approach to compute local activation durations from electrograms (EGMs) and electrographic signals (ECGs). EGMs were acquired during both sinus rhythm and ventricular tachycardia with a commercial mapping catheter (Abbott Advisor HD Grid) in six patients. EGMs were band-pass filtered before processing and the analysis was based on the EGMs histogram and similarity techniques, only when a repeatable rhythm was detected in the ECGs the proposed approach was validated against 2846 *activations manually annotated (GS) by an expert electrophysiologist. The mean error in the computation of the activation durations over each signal for each patient was -0.1±1.8ms (GS activation duration: 54.4±9.3ms). The developed algorithm is accurate, and the 3D dynamic maps showing slow electrical conduction areas may represent a useful tool to be integrated with activation and voltage maps to plan and assist therapeutic interventions in ventricular arrhythmias.*

1. Introduction

Ventricular tachycardia (VT) is an arrhythmia caused by the irregular activation of the right and left ventricles. VT includes a broad spectrum of macro-reentrant tachycardias in which the wave front does not travel through the normal electric pathways. Common symptoms include chest pain, dizziness, palpitation, and shortness of breath [1].

Non sustained VT may go away on its own, without the patient notices; sustained episodes may lead to loss of consciousness or cardiac arrest. When VT is not tolerated, and the patient is unresponsive to antiarrhythmic drugs catheter ablation treatment should be considered [2].

To date, there are no clear guidelines for catheter ablation of macro-re-entrant tachycardia circuits. Mapping systems are necessary to gain insight on unusual electrical activations, and to localise focal sources and mechanisms [3].

To properly recognize slow electrical conduction areas in ventricular arrhythmias, local ventricular activations must be accurately identified and characterized. As of today, mapping systems identify the precocity or lateness of a local activation with respect to a fixed time reference.

Information about activation duration could indeed be crucial to reconstruct slow conduction patterns and areas; unfortunately, such information is not available in the mapping system commercially available; moreover, when the activation waves are following different pathways assessing activation and duration times may not be possible [4].

In this study we developed an automatic approach to compute local activation durations using both EGMs and ECGs acquired using the Advisor™ mapping catheters (Abbott©) from which patient-specific dynamic 3D maps showing slow electrical conduction areas were reconstructed.

2. Material and Methods

2.1. Clinical Data Acquisition

EGMs were acquired using Advisor™ mapping catheters (Abbott©), the HD Grid (HD) (Figure 1) in six patients during the ablation procedure performed in the Electrophysiology Lab at the Infermi Hospital in Rimini (Italy) using the EnSite Velocity™ Cardiac Mapping System (Abbott©).

EGMs were acquired when the catheter position was stable, for few seconds when the VT was not sustained or up to two minutes in case of sustained VTs.

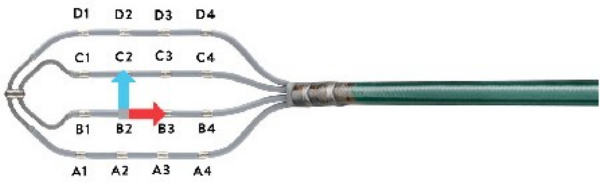

Figure 1 Advisor HD Grid Mapping catheter, where each electrode is labelled

2.2. Data Processing

Bipolar EGMs and ECGs sampled at 2KHz were exported from the mapping system and analyzed off-line.

The signals belonging to sustained VTs were divided in 10 seconds length.

ECGs were already filtered by the mapping system before the export.

EGMs were pass-band filtered (30 to 300Hz) applying a second order Butterworth filter. This range was selected to filter the signal from artifacts due to respiration and to attenuate high frequency noise.

From all the ECG derivations a sum signal was calculated (ECGsum); from this signal search windows for the EGMs were defined in order to exclude noise and artifacts such as the pacing signal. The histogram of the ECGsum was computed, then low and high threshold were defined as the 15 and 40 percentiles, and points nearer than 50ms were grouped in the same activation. The local maximum of the sum signal was used to center a 200ms search window, which was subsequently shifted to the left with a fixed offset based on the heart rate of the patient (Figure 2, black vertical lines).

The next step was the computation of the histogram for each EGM derivation of the HD Grid catheter, as shown in the Figure 3. Higher number of samples correspond to the baseline of the EGM; while the tails of the distribution correspond to the voltage values of the signal which are linked to local activations. Aiming at detecting only signal portions corresponding to activations, we selected a pair of thresholds based on the percentiles of the histogram to perform EGM segmentation.

Starting from the beginning of the EGM segment, if the value of the sample was included in the threshold range the sample was discharged; the next sample was then evaluated until a value out of the range was detected; this sample was considered part of an activation; to verify this hypothesis the next sample was evaluated and the time distance between them computed; in case this distance was greater than a cut-off value set to 30ms, then the two samples were assigned to two different activations. For the EGMs, cutoff thresholds already validated from previous studies were used.

The activation was then recognized as the portion of the signal from the first sample out of range and the last sample satisfying the cut-off constrain. This procedure was repeated for each detected activation until the end of the EGM.

The EGM segmentation was optimized increasing the duration of each activation by detecting the first sample of the EGM before and after the recognized activation in which the EGM second derivative changes its sign.

The activation duration is then computed as the time between these two points (Figure 2, red dashed lines). To correct potential errors due to different activation patterns,

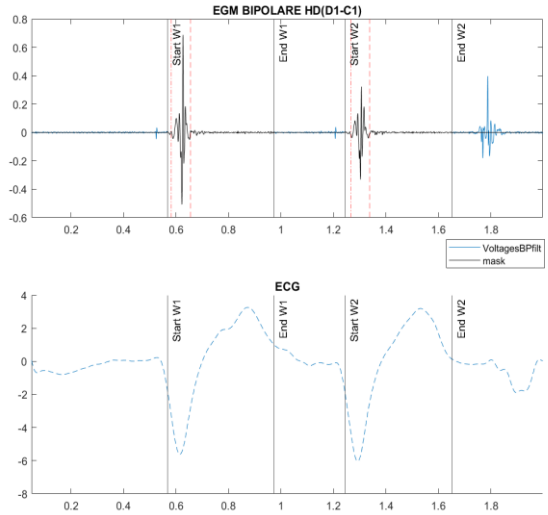

Figure 2 EGM and ECG signals showing both the windowing (black vertical lines) and the estimated duration (dashed red lines) of the algorithm.

an additional step based on the stability of the signal was added.

Each activation was assigned to a cluster based on the features derived from the signal, and the activation belonging to the most frequent cluster where detected.

Once activation duration was computed we designed a workflow to map this information on the patient-specific anatomy to provide a visual representation of activation duration. The patient-specific anatomical model of the atrium was exported from the mapping system together with the position of the mapping catheter during the acquisition of the EGMs. The mean duration of the detected activations was calculated; then, using information from the catheter's electrode position and geometry, the duration maps were obtained.

Patients in which more than one VT was detected, signals acquired at different times were used to increase the coverage of the maps over the anatomy. For each single acquisition session, a dynamic duration map was also computed, in this case the duration map shows the duration of each different ECG detected activation.

Data analysis was performed using MATLAB 2021b (The MathWorks® Inc.).

2.3 Validation

The developed approach was validated against (1) 454 activations from 20 EGM segments in 4 patients with un-sustained VT, and (2) 2392 activations from 14 EGM segments in 2 patients with sustained VT.

All these activations were manually annotated by an expert electrophysiologist (gold standard, GS).

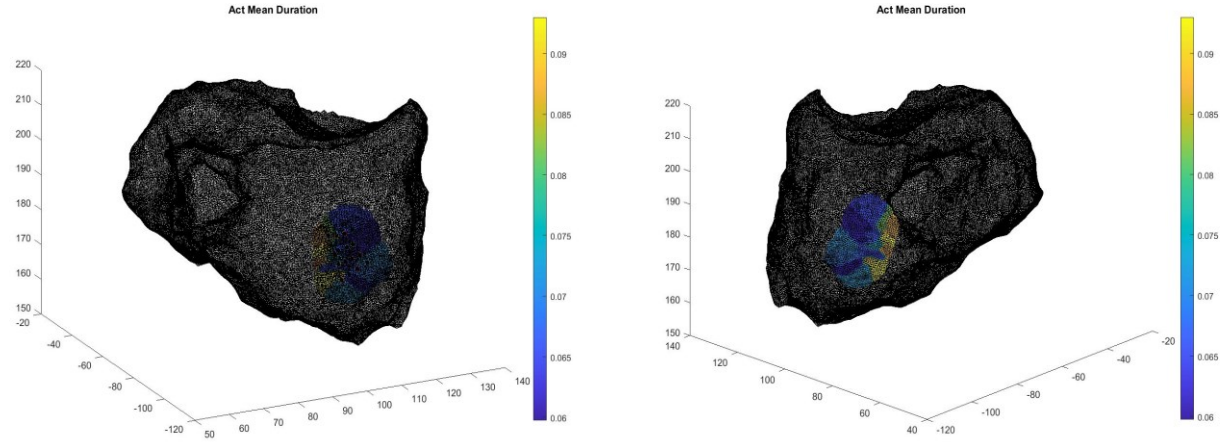

Figure 3. Mean activation duration map, back (left) and front (right) of the ventricle (duration maps are expressed in seconds, and the electrodes are visible as black dots).

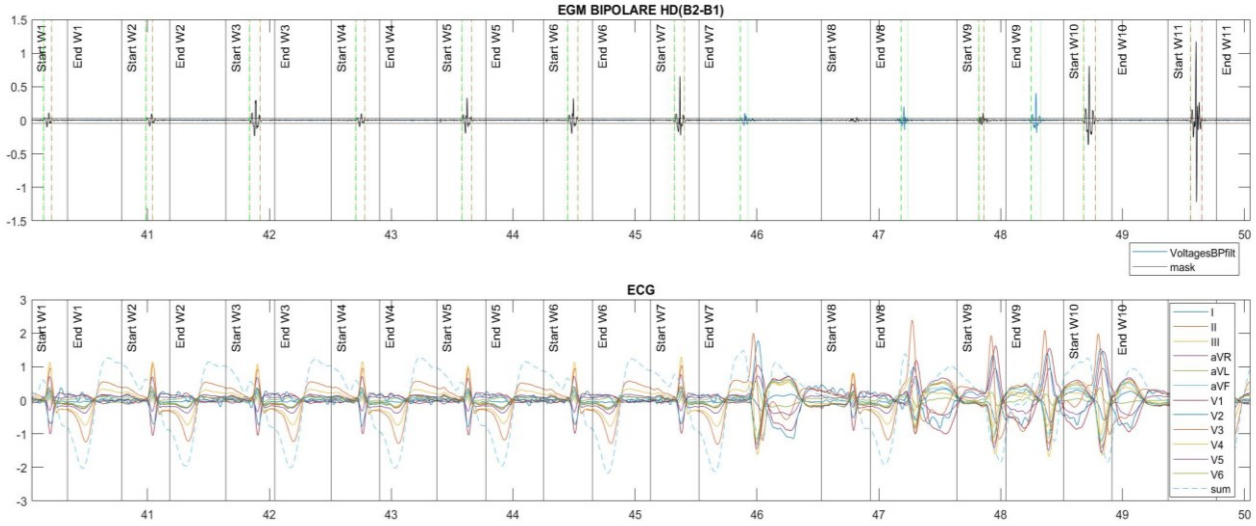

Figure 4 Ventricular EGM with the start of a VT, green (start) and red (end) dashed lines identifying the activations on the EGM. Black vertical lines are the corresponding search windows from the ECG.

3. Results

EGM analysis to compute activation duration is indeed very fast requiring less than 15 sec. for un-sustained VTs, and less than 1 minute in the case of sustained VTs with a 3-97% threshold, a sensibility of 95.5%, specificity of 99.3% and accuracy 97.2%.

The GS activation duration was *54.4±9.3ms*. Our analysis reported a mean error in the computation of the activation durations over each segment for each patient of -0.1±1.8ms ().

An example of the detected activations in sustained VT in Figure 4.

An example of the 3D map is shown in Figure 3. On the surface of the anatomical model of the ventricle obtained in one patient, the activation duration computed applying the proposed approach is mapped.

4. Discussion and Conclusion

In this study we developed an automated approach for local activation duration computation in VTs. The proposed technique was tested with data acquired with

Advisor HD Grid mapping catheter and validated against manual annotation in 2846 activations.

The performance of the algorithm was indeed promising, allowing a precise computation of activation duration.

Our approach has several limitations.

We analyzed data acquired in only 6 patients and increasing the data sample will be important to confirm our results and assess its generalizability.

Unfortunately, we are missing an objective reference annotation: our experience suggests manual annotation depends on the electrophysiology experience and the assessment of variability for the reference technique may help in better evaluating the performance of our approach.

Despite these limitations, preliminary results on biphasic and low amplitude EGMs were successful thanks to the ECGs windowing but further testing is required to confirm such promising development. In addition, the availability of a visual representation of 3D duration maps, both global and dynamic, could provide useful tools for understanding activation pathways and assist therapeutic interventions.

References

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