

ATRIAL ACTIVATION PATTERN FROM SURFACE LAPLACIAN ELECTROCARDIOGRAMS OF HUMANS

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Abstract: The goal was to determine whether atrial activation patterns could be obtained with our Laplacian electrocardiogram (LECG) system. Body surface maps from humans were generated depicting such patterns. Unprocessed atrial LECG activity coincided with P-waves in the Lead II ECG (L2). Partial atrial flutter activation patterns were also constructed. The system promises to reveal underlying cardiac activation patterns, including those of the atria, which are far less prominent than those from the ventricles.

INTRODUCTION

Cardiovascular disease is the number one cause of death in the United States. Atrial arrhythmias are most prevalent and often lead to disability, heart failure and stroke. There is need for a reliable non-invasive technique to predict an individual's risk for heart failure, localize accessory pathways and aid in pharmacological assessment.

Cardiac activation is a spatial and temporal function that can be sensed on the body surface. The 12-lead ECG yields limited spatial information about the ventricles and even less about the atria. To understand and treat certain arrhythmias, further diagnostic techniques are necessary, such as invasive electrophysiology (EP). Non-invasively derived spatial information from body surface potentials may reduce the time and risks for invasive EP exploration. Such a non-invasive system must have high spatial and temporal resolution to determine the origin of an arrhythmia.

The surface Laplacian, a non-invasive imaging technique, was first proposed by Hjorth[1] for EEG studies, and later by He and Cohen for Laplacian ECG (LECG)[2]. Tarjan et al.[3] introduced our concentric LECG sensor. Rasquinha[4] identified arrhythmias with the sequence of delays from the moments of activation (MOA) by using animal data. Active LECG sensors were developed by Lu et al.[5]. The sensor is shown in Fig. 1.

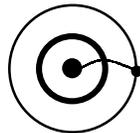


Fig. 1: The electrode configuration of the Laplacian ECG sensor.

METHODS

Our data were obtained from consenting healthy and heart diseased subjects. Over 70 subjects have been recorded to

date. For this paper atrial activity from young healthy (Healthy) and atrial flutter (Aflutter) subjects will be presented. The Healthy data were recorded on a regular grid at 35 locations as depicted in Fig. 2. Seven LECG channels and the L2 ECG were recorded and digitized simultaneously. The atrial flutter data were recorded from a non-uniform array. The data acquisition was controlled with a program written in LabView[®] (National Instruments). The LECG signals were bandpass filtered from 5 to 500Hz, amplified by 1120, digitized at 1KHz (1 ms resolution) for a length of 30 seconds or 30K samples. Digital filtering was accomplished by adapting Bertrand's filter[6], a sub-average spectral analysis adaptive Wiener Class III filter.

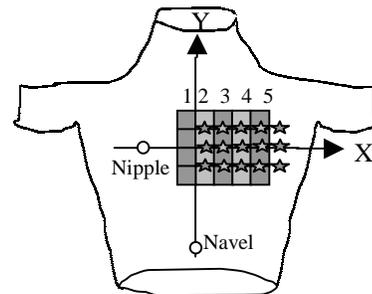


Fig. 2: Recording sites of the Healthy subjects. Seven sensors per recording, four placed in one of the five labeled columns and three placed at the adjacent stars.

The activation patterns are based on the MOAs, which we define as the instant the depolarization wavefront crosses the line normal to the axis of the sensor. At the MOA, the LECG rapidly slews between opposite extrema. For atrial flutter maps, the time of activation at a specific sensor is referenced to the atrial flutter peak in L2 for that cycle. The LECG was aligned to this atrial flutter peak and ensemble averaged.

RESULTS

Atrial-waves were evident in some of the unprocessed recordings. Fig. 3 shows 4.4 seconds from the unprocessed LECG (top) and L2 (bottom) for Healthy subject MA. The sensor was located at (x=0.0cm; y=1.8cm). The signal to noise ratio for the LECG is 10. The arrows point to the unprocessed atrial-waves and L2 P-waves. A second recording (not shown) from the same sites a few minutes later revealed similar atrial-waves. This emphasizes the stationary nature of the P-R coupling; it is not a transient relationship. The global P-wave in L2 is present before the QRS and the LECG atrial-waves are synchronized with the L2 P-waves.

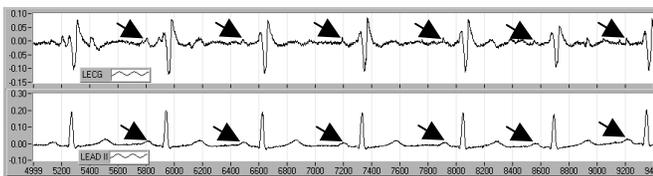


Fig. 3: Unprocessed recording from Healthy subject MA.

Figure 4 shows the LECG waves of Fig. 3 after Wiener Class III filtering. Again, a pronounced peak is evident in the top panel at approximately 260ms, -140ms with respect to the L2 R-wave peak (L2 R) and coincides with the P-wave in the bottom panel.

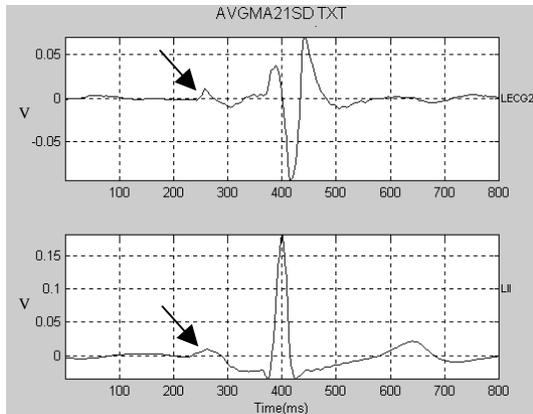


Fig. 4: The Wiener Class III filtered LECG of Fig. 3. Top: An atrial-wave at about 140ms before the L2 R.

As expected, the atrial flutter subjects did not show unusual ventricular depolarization patterns in their body surface isochronal MOA maps when synchronized to the L2 R. Atrial flutter was evident in some of the unprocessed recordings as shown in Fig. 5. Panel H (bottom right) shows atrial flutter in L2. Panel C (8.7; 1.3), D (7.7; -6.3) and G (10.2; -3.2) also show atrial flutter.

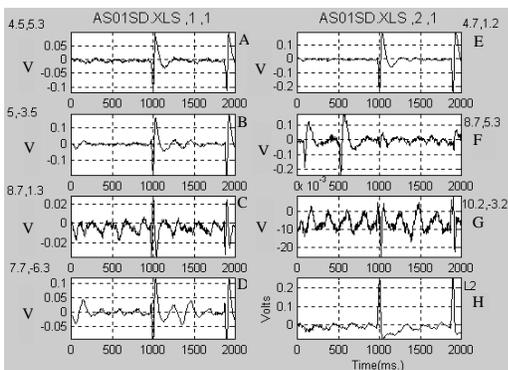


Fig. 5: Unprocessed recordings from Aflutter subject AS during atrial flutter.

In Fig. 6 the longer arrows illustrate two areas where activation spreads quickly during atrial flutter: at (7; 4), the contour originates at $t = -60$ ms; the (12; -8) neighborhood is activated at about $t = -40$ ms.

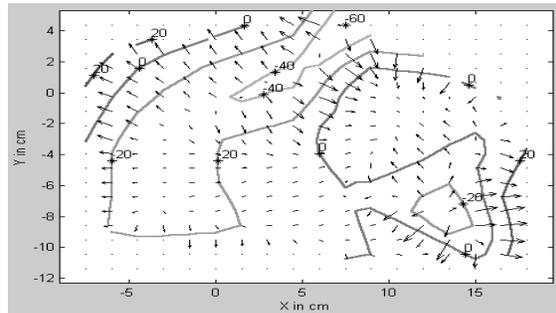


Fig. 6: A spatial portion of the atrial flutter cycle of Aflutter subject AS.

DISCUSSION

This new LECG sensor system was able to detect atrial activity as demonstrated in Fig. 3 and 4. With the atrial activity manifested in multiple LECG channels, detection of atrial activation patterns may be possible to aid diagnosis. The atrial flutter map shows an area on the body surface where the atrial activation pattern was detected. With future improvements, we expect to map a complete flutter cycle with sufficient spatial resolution to show the point of re-entry if it is within the mapped region.

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