Estimation of myocardial conduction velocity using a coronary sinus catheter

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Abstract—This paper deals with the measurement of the conduction velocity of the depolarization wave through the atrial myocardium. With the increasing number of electrophysiological procedures performed on people suffering from cardiac arrhythmias, the number of relapses is gradually rising. A reliable tool to predict these relapses is still being sought. One parameter for prediction could be the conduction velocity of the depolarization wave, which could reflect the degree of myocardial damage.

Data from pediatric patients were used to measure it. With knowledge of the electrode placement on the intracardiac catheter and detection of atrial activity in the measured leads, the average myocardial conduction velocity along the catheter was calculated ($v = 143.77 \pm 21.08$ cm/s).

This method brings simplicity and time saving to the whole process of measuring line speed. The results of this method should also be tested on an adult population where relevant models exist for comparison.

Index Terms—Intracardiac electrogram, Atria, Conduction velocity, Depolarization wave, Coronary sinus catheter

I. INTRODUCTION

In recent years, there have been major developments in the field of electrophysiology. With the advent of more advanced diagnostics and the miniaturization of catheters used for atrial and ventricular ablation, the number of patients undergoing this treatment, which is a full-fledged alternative to long-established drug therapy, is increasing [1]. As the number of patients ablated increases, the number of arrhythmia recurrences due to patients previously undergoing the procedure also increases. Reliable prediction of patient relapses is one of the current challenges. To date, there is no 100% reliable parameter that directly relates to the likelihood of arrhythmia relapse [2].

Myocardial conduction velocity could be one predictor of future relapses. The basic hypothesis is that in intact myocardium the conduction velocity of the depolarizing wave is higher than in myocardium affected by arrhythmias, ischemia or other degenerative influences [3]. The aim of this study is to develop and present a reliable method that will use the information commonly available from catheters to calculate conduction velocity in electrophysiological operations. Previously published work used whole atrial mapping or cellular models to calculate conduction velocity ([4], [5], [6], [7]). Despite higher accuracy, these methods are disproportionately more challenging. The method presented in this work creates a competitive model that allows real-time measurements during electrophysiological procedure.

II. METHODS

A. Data

Records from twenty patients from the previously measured database [8] were selected for this study. The patients were aged 12-17 years. They underwent electrophysiological procedure at the Children’s Hospital, University Hospital Brno, Brno, Czech Republic. The measurements were performed with the approval of the ethics committee. During the procedure, in addition to a 12-lead surface ECG, they had a 5-lead intracardiac ECG measured using a catheter inserted into the coronary sinus. The advantage of this database is that the atrial activity is annotated. Recordings, where only sinus rhythm was present, were used for this work. The recordings are therefore free of other arrhythmias or stimulation artifacts.

A 10-polar catheter was used for signal acquisition along with a St. Jude WorkMate 4.2 EP system with a sampling rate of 2000 Hz and a voltage resolution of 78 nV/LSB. The system incorporates a notch filter with a cutoff frequency of 50 Hz and an upper pass filter with a cutoff frequency of 0.1 Hz. The catheter used has electrodes arranged in a 2-8-2 mm configuration and the width of one electrode is 1 mm as can be seen in figure 1. As mentioned earlier, the catheter was placed in the coronary sinus whose orifice is located in the right atrium (see figure 2). This vein runs in the sulcus coronarius and runs around the perimeter of the left atrium. Due to this anatomical position, the depolarizing wave propagates along the catheter from its proximal to its distal end.

The electrodes placed on the catheter form five pairs from which bipolar leads are measured. These are designated in the records as cs1-2, cs3-4, cs5-6, cs7-8, cs9-10. The electrodes are numbered from the distal end of the catheter and the order of the leads and their spacing will be crucial for subsequent
analysis and calculation of the conduction velocity of the depolarizing wave through the atrial myocardium.

The signal from the catheters measured during physiological sinus rhythm can be seen in the figure 3. The signal can be divided into three parts. The first is the isoelectric line, which is present for most of the cardiac cycle. It begins approximately with the end of the S wave in the surface ECG, continues during the T wave, and ends during the P wave. During the P wave, high-frequency activity can be found in the intracardiac leads during the surface P wave. This corresponds to the course of the depolarization wave. This is followed by a low-frequency wave corresponding to the depolarization of the ventricles. This wave may not be present in all intracardiac leads. Moreover, its frequency composition may overlap with atrial activity and therefore its filtering may be difficult.

B. Atrial activity Detection

The main aim of the study is to determine the velocity of the depolarization wave propagating along the atria along the catheter placed in the coronary sinus. The aim of the work with the measured signal will be mainly to correctly localize the peaks of high-frequency activity in the five leads measured by this catheter. The signal analysis is based on two important facts:

1) The depolarization wave appears in the leads gradually from the proximal end of the catheter to its distal part.
2) The character of the signal during ventricular activity has a frequency maximum below 50 Hz.

The first step in the processing of intracardiac signals is filtering in order to highlight the atrial component and to suppress as much as possible the ventricular activity or possible noise caused most often by electric network noise. For filtering, a wavelet transform was chosen to divide the signal spectrum into a predefined number of octave bands [10].

To suppress the low frequency components, it is sufficient to obtain the first band in which the frequencies from $f_s/4$ to $f_s/2$ are captured. This step reliably removes from the signal most of the chamber activity that may be present in the signals. However, depending on how far in the coronary sinus the catheter is inserted, it is possible that the distal end is already too close to the left ventricle and therefore high frequency ventricular activity may be present in the signal. Therefore, after filtering, two peaks may occur in the signal close together. Their resolution will be subsequently addressed.

The next step was to create the envelope of the filtered signal. Using the Hilbert transform, an analytical signal was obtained whose magnitude is the envelope of the signal [11]. This was then smoothed with a low-pass filter to remove as many local outliers as possible. In this way, an envelope was created for each of the five signals. The result of this step is shown in the figure 4.

From the generated envelope, it is already possible to detect local maxima in each signal. Since the depolarizing wave propagates along the catheter from its proximal end, it is possible to start with peak detection in lead cs9-10 and adjust the detection in subsequent leads. Two conditions have been chosen that the peak must satisfy to be detected:

1) It must be greater than one-sixth of the maximum value taken by the signal envelope.
Intracardiac signal procedure. From top to bottom: a) scaling the voltage values of the raw signal to an interval from 0 to 1, b) filtering using a wavelet transform in order to highlight the high-frequency component of the signal, c) creation of an envelope of filtered signal (blue) from which the peaks (red lines) were detected.

2) Two adjacent peaks must be at least 0.45 s apart.

The first condition is based on empirical adjusting of the detection threshold. The second condition is based on the fact that the heart rate in children during sinus rhythm may be higher than in adults, but still should not exceed 130 bpm.

From the set of detected positions, those whose distance from two adjacent positions was less than one and a half times the standard deviation of the differences subtracted from the mean value of the differences were removed. This reduced the number of false positive detections that could bias the final conduction velocity calculation.

In subsequent leads, the detection of envelope peaks was modified so that in each subsequent lead the peak was searched for in a local neighborhood defined by the position of the previous peak. The neighborhood was defined from -15 to +35 milliseconds from the previous peak. Again, a local envelope maximum was found in this region. The neighborhood also considers the short signal segment before the preceding peak for the reason that cardiac tissue is not an isotropic environment for the propagation of the depolarizing wave and thus deviations from the expected propagation direction may occasionally occur.

C. Velocity Estimation

The position of atrial activity across the five leads in the intracardiac ECG detected in the previous section, together with the knowledge of the electrode placement on the catheter, is used for the final calculation of the conduction velocity of the depolarizing wave through the myocardium. Despite the possible presence of anatomical variations, we will assume that the catheter runs around the surface of the atria, with all electrodes insistent on the myocardium. Because of this, we can consider the distances of the leads on the catheter to correspond to the distance between the measured sites on the atrial wall.

One position obtained from each lead was always used to calculate the velocity. The position from catheter cs9-10 was marked as point [0,0]. The other leads were followed one centimeter at the lead. From the five points plotted on the distance-time graph, the coefficients of the line were obtained by linear regression. The slope of this line corresponds to the propagation velocity of the depolarizing wave in cm/s. This procedure was repeated for each cardiac cycle within a single signal. The resulting velocity for each patient is given by the
average of the all velocities. An example for one cardiac cycle is shown in the figure 5.

III. RESULTS

The outcome of the entire conduction velocity measurement depends on the accuracy of the detection of atrial activity in the intracardiac recordings. This was assessed using standard parameters such as sensitivity (true positive rate, TPR), positive predictive value (PPV) or F1 score [12]. The calculation of these parameters is summarized in equations (1), (2) and (3). As a ground truth detection, the position annotations from the database were taken.

\[
TPR = \frac{TP}{TP + FN}, \quad (1)
\]

\[
PPV = \frac{TP}{TP + FP}, \quad (2)
\]

\[
F1_{score} = \frac{2 * TP}{2 * TP + FN + FP}, \quad (3)
\]

where true positive (TP) is the number of correctly detected atrial activity episodes, false positive (FP) is the number of detected atrial activity episodes not presented in the signal and false negative (FN) is the number of atrial activity episodes present in the signal, but not detected by the algorithm.

Table I summarizes these calculated parameters. As can be seen, the chosen procedure has a relatively high sensitivity and a 100% positive predictive value. Thus, almost all episodes present in the signals were detected correctly. The algorithm did not detect any false positive episodes. False negative detections could be further eliminated by adjusting the detection thresholds.

<table>
<thead>
<tr>
<th>Attribute</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of true positive</td>
<td>228</td>
</tr>
<tr>
<td>Number of false positive</td>
<td>0</td>
</tr>
<tr>
<td>Number of false negative</td>
<td>21</td>
</tr>
<tr>
<td>True positive rate [%]</td>
<td>91.57</td>
</tr>
<tr>
<td>Positive predictive value [%]</td>
<td>100.00</td>
</tr>
<tr>
<td>F1 score [%]</td>
<td>95.60</td>
</tr>
</tbody>
</table>

For each of the twenty patients tested, the conduction velocity of the depolarizing wave through the myocardium was calculated. These results are summarized in table II. Because of the variability in heart rate, the number of cardiac cycles from which the velocity was calculated was also different for each patient. The table also captures other descriptive statistics such as the mean and the standard deviation of the velocities.

The calculated velocities have a normal distribution (143.77 ± 21.08 cm/s), as can be seen in figure 6, where the result of the Shapiro-Wilk test is mentioned. Along with this, the result of the student’s one sample t-test against the velocity value found in the available literature (52.7 ± 25.3 cm/s [5], 91.0 ± 44 cm/s [13]) was significantly different. The result of [14] showed that the conduction velocity can reach up to 150 cm/s. However, this work focused on the His bundle, not the atrial myocardium.

On the other hand, in papers [4] and [15] the conduction velocity in some patient groups reached up to 150 cm/s ± 50 cm/s. Such conduction velocities are already close enough to the values measured in the presented way (p = 0.366).

![Quantile-quantile plot of conduction velocities within the measured dataset. The data have a normal distribution, which was also verified using the Shapiro-Wilk's test (W = 0.96, p-value = 0.7).](image)

IV. CONCLUSION AND DISCUSSION

In this paper, a new method for measuring the conduction velocity of the depolarizing wave through the atrial musculature is described. The main advantage of this method lies in the use of data measured with a catheter placed in the coronary sinus. The method is simple, fast and uses signals that are commonly measured. This catheter is commonly used in electrophysiological procedures aimed at removing foci of cardiac arrhythmias by applying radiofrequency energy. This work is also unique because of the data used, which comes from the child population. However, this fact is also a weakness of the work because to the best of our knowledge no similar study has been conducted in children, so the measured conduction velocity can only be compared to results on the adult population.
Compared to conduction velocity measured on hearts from adults, the rate measured by this method is significantly higher compared to some of the literature. This could be due to several factors:

1) Age of the measured patients. The heart, like all organs, is subject to more wear and tear with increasing age. Taking this fact into account, one may consider the possible higher myocardial conduction velocity in children compared to adults who already have a heart affected to some extent. This hypothesis is also supported by the fact that experiments on isolated hearts have shown that as the ischaemia-affected cardiac wall is progressively remodelled, the conduction velocity through the affected area also decreases. To clearly confirm or refute this hypothesis, a group of adult patients undergoing electrophysiological procedures would also need to be tested. This data is not currently available. If the hypothesis were verified, conduction velocity could be considered as a marker of myocardial damage.

2) Number of patients studied. The number of patients whose data was used for this work is limited by the size of the available database and the fact that it is primarily a database of arrhythmias. Pure sinus rhythm records are also present but are not the main domain. Thus, it is possible that the twenty patients selected do not adequately capture the variability in the entire pediatric population. An option to broaden the patient selection could be to select certain types of regular arrhythmias (such as atrioventricular nodal tachycardia, atrioventricular tachycardia, atrial flutter, etc.) and adjust the peak detection parameters for these specific cases. Another option would be to include recordings with paced rhythms. However, these recordings are problematic for two reasons. The first is that the stimulation is often performed with the catheter being used. While this pinpoints the exact moment in time when the wave is generated, it also creates an artifact that is reflected in other leads and can complicate detection in them. The second problem is that any electrode on the catheter can theoretically be used for stimulation. If electrodes other than the outermost electrodes are used for stimulation, the depolarizing wave would propagate to both ends of the catheter, reducing the accuracy of the resulting velocity measurement.

In the two papers mentioned, the conduction velocity was close to the measured value presented in this paper. This was mainly in the control groups of patients. The children whose recordings were used for this work could also be considered as otherwise healthy control subjects, because the main reason for performing the electrophysiological procedure was structural malformations, especially accessory pathways leading to WPW syndrome. If we focus on the atrial myocardium itself, it can be assumed that the structural damage to it was not very high given the young age.

The reason for the relatively high variability in velocities could be anisotropy of cardiac tissue. The cardiac musculature does not form a unit in which the conduction velocity is the same in each direction. It is therefore possible that the average propagation velocity will be lower, but equally there may be areas of muscle where conduction will be faster. This hypothesis could be tested by measuring with multipolar catheters that would map both atria and then calculate specific velocity values in different directions.

The age of the patients, number of patients and high variability caused by anisotropy of cardiac tissue are probably the biggest issues that would lead to a refinement or at least verification of the correctness of the method used. The hypothesis that conduction velocity could be used as a predictive factor to determine arrhythmia relapse in patients cannot be reliably confirmed. However, there is certainly room for further investigation of this idea.

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REFERENCES


