Left atrial conduction along the coronary sinus during ectopic atrial tachycardia and atrial fibrillation — a study using correlation function analysis.

Short title: Conduction along the coronary sinus

Jonas Carlson[1], MSc; Susana Santos[2], MSc; Pyotr G Platonov[1], MD, PhD; Ole Kongstad Rasmussen[1], MD; Rolf Johansson[2], MD, PhD; S Bertil Olsson[1], MD, PhD;

[1]Department of Cardiology, Lund University; [2]Department of Automatic Control, Lund Institute of Technology, Lund University

Corresponding author:

Jonas Carlson
Department of Cardiology
University Hospital
SE - 221 85 LUND
Sweden

email: Jonas.Carlson@kard.lu.se
fax: +46 46 157857

Abstract

Introduction: Correlation function analysis was applied to endocardial electrograms to investigate conduction patterns along the coronary sinus (CS) during sinus rhythm (SR) and atrial tachycardias.

Methods and Results: Eighteen recordings were obtained from 14 patients suffering from supraventricular tachycardias. Five atrial fibrillation (AF) recordings were compared to 10 SR recordings and 3 ectopic atrial tachycardia (EAT) recordings. The maximum correlation coefficient was used to assess similarity between signals, i.e., if they originate from the same wavefront. The cumulative time delay, calculated as pairwise summation of interelectrode time delays was used as an indicator of activation sequence along CS. Method validation using SR showed right to left conduction with high correlations in 8 of 10 recordings indicating one single wavefront. EAT recordings showed consistent left to right conduction with left atrial foci and right to left with right atrial focus and lower correlations than SR. All 5 AF recordings showed predominantly left to right conduction direction, also with correlations lower than SR.

Conclusion: 1) Correlation function analysis can be used to assess agreement between signals and direction of activation spread. 2) Due to the position of CS, the results can be used to derive mechanisms of interatrial conduction. 3) Consistency in electrical activity propagation along CS is common in AF.

Key words: Unipolar electrograms, Correlation function analysis, Coronary sinus, Atrial fibrillation, Preferential conduction

Introduction

Atrial fibrillation (AF) has a well-known electrocardiographic appearance, with a constantly but variably undulating signal of low amplitude. This pattern is caused by the coexistence of multiple and seemingly independent, more or less complete excitation wavelets involving the entire atrial myocardium [1, 2].

Neither eyeball inspection of the body surface leads, nor multiple lead exploration of epicardial excitation during few seconds [1] reveal any systematic excitation patterns during AF. However, using different mathematical signal handling techniques, it becomes apparent that the spread of the excitation during AF follows distinct patterns within the right atrium (RA) [2, 3]. Similar information on possible systematic spread of fibrillatory excitation within the left atrium (LA) is lacking, however.

Both anatomical [4, 5, 6] and functional [7, 8] studies reveal two separate major muscular connections between the atria - one anteriorly, the so-called Bachmann bundle [9], and one at the rear part of the atria. Both these connections are mostly made up of several individual fibres [5]. The two atria have usually been regarded as one functional global structure, where the fibrillatory waves may travel equally easy between the atria as within one single atrium. Recent studies have however revealed that deterioration of conduction between the atria is a factor predisposing to AF. Many patients with paroxysmal AF have thus signs of impaired conduction at the rear connection between the atria, as evidenced by non-invasive [10] as well as invasive [11] methods.

The variability of atrial excitation during AF necessitates analysis of longer periods of excitation data in order to reveal any systematic conduction patterns. Correlation function analysis provides a mean to evaluate such sig-

nals. We have applied this method to observe the conduction pattern along the great cardiac vein, Coronary Sinus (CS), running along the rear part of LA, emptying in the infero-posterior part of the interatrial septum, i.e. within RA.

The purpose of this paper is: 1) to present and validate the method, and 2) to identify any possible systematic conduction pattern in LA during paroxysmal AF of recent onset.

Method

Patients

Recordings from 14 patients admitted for clinical electrophysiological examinations were used. Six patients suffered from paroxysmal atrial fibrillation (PAF), three ectopic atrial tachycardia (EAT), two AV re-entrant tachycardia (AVRT) due to an accessory pathway, and two AV-nodal re-entrant tachycardia (AVNRT). All analyses were performed retrospectively on material collected on clinical indication.

In all, 18 recordings were obtained from the 14 patients. Of these recordings, five were made during AF in patients with PAF (one induced), three during EAT in patients with EAT, and ten during SR. Of the SR recordings, 4 were made on patients with PAF, 2 AVRT, 2 AVNRT, and 2 on patients with EAT.

In the three patients with EAT, endocardial mapping before the EAT recording showed that two patients had an ectopic focus in LA and one had a focus in RA.

In two patients with EAT and two with PAF, recordings were made both during SR and atrial tachycardia.

Recordings and data acquisition

Ten unipolar endocardial electrograms and a standard 12-lead ECG were acquired simultaneously using a BARD Cardiac Mapping System (BARD LabSystem DUO; Bard Electrophysiology; Billerica, MA; USA)

The electrograms were acquired using a 10-pole catheter (Dynamic XT 201-101; Bard Electrophysiology, Lowell, MA; USA) placed in CS, with pole 10 in the CS orifice and pole 1 (the tip of the catheter) in distal CS. The

use of the catheter was always motivated by the clinincal procedure. The electrodes on the catheter are 1 mm wide and organized in pairs with 5 mm space between the pairs and 2 mm between the electrodes in a pair. Thus, a distance of 40 mm is covered by the electrode.

The signals were sampled using 16-bit resolution and a sampling frequency of 1 kHz per channel. All recordings were at least 60 seconds in length. The ten electrograms and lead V1 of the surface ECG were transferred to a GNU/Linux workstation where all subsequent analyses were made using custom software written in MATLAB R12 (The MathWorks, Inc.; Natick, MA; USA).

In one AF recording, the signal from electrode 10 was excluded due to noise, and in another, the signal from electrode 1 was excluded due to saturation of the signal.

Removal of QRS complexes by linear interpolation

The electrograms showed both the atrial and ventricular activity. The QRS complexes were of much higher amplitude than the signal originating from the atria, which would affect the analysis if left without attention. To avoid this drawback, lead V1 of the surface ECG was used to identify the QRS complexes, aiming to remove them.

The onset and end of each QRS complex were identified automatically and the ventricular signal was removed from all electrograms by doing a linear interpolation between these points.

Correlation function analysis

To define 'similarity' between two electrograms, correlation function analysis was used. The correlation function between two signals is defined as

$$\rho(\tau) = \frac{C_{xy}(\tau)}{\sqrt{|C_{xx}(\tau)|}\sqrt{|C_{yy}(\tau)|}}, \tau = -n, \ldots, -1, 0, 1, \ldots, n$$

where τ is the time delay in milliseconds. Since the sampling frequency is 1 kHz, time shift will be done sample-wise. In this study, the maximum time delay analyzed, n, was 20 ms.

The sample covariances are defined as

$$C_{xy}(\tau) = \frac{1}{N-\tau} \sum_{i=1}^{N-\tau} (x_{i+\tau} - \bar{x}_{1+\tau,N})(y_i - \bar{y}_{1,N-\tau})^T$$

and the sample means as

$$\bar{x}_{1,N-\tau} = \frac{1}{N-\tau} \sum_{i=1}^{N-\tau} x_i$$

$$\bar{x}_{1+\tau,N} = \frac{1}{N-\tau} \sum_{i=1+\tau}^{N} x_i$$

where N is the total number of samples in an individual recording.

The correlation function was calculated for each adjacent pair of electrodes to find their $\tau_{\max}$ and $\rho_{\max}$, i.e., the time delay resulting in the highest correlation. Figure 1 shows an example of the result of the analysis.

Excitation direction

The excitation direction can be inferred from the sign of $\tau_{\max}$. With our definition of electrode-numbering, a positive time shift indicates propagation from the pole with lower number to the one with higher (i.e., left to right, or, from distal CS towards its orifice), whereas a negative time shift denotes

propagation from higher to lower numbered poles (i.e., right to left, or, from CS orifice towards its distal part).

To facilitate interpretation of the values of each $\tau_{\max}$, a graph of cumulative time delays was created by cumulative addition of the values of $\tau_{\max}$ from electrodes 1-2 towards electrodes 9-10 (Figure 1). In the case of uniform conduction from right to left, all values of $\tau_{\max}$ would be negative, thus resulting in a line pointing downwards. Consequently, conduction from left to right would result in a line pointing upwards.

Reproducibility

As stated above, 60-second long recordings are used for the correlation function analysis. To evaluate how long recording would suffice to reproduce the same excitation direction pattern in AF, the algorithm was applied to smaller segments of the whole recording. In all AF recordings, segments of lengths 5, 10, 15, and 20 seconds were used, 10 of each with randomly chosen position within the 60 s recording.

Results

Removal of QRS complexes

Removing the QRS complexes by linear interpolation also removed part of the electrograms. Of the entire recordings, between 8 and 24% (median 16%) of the SR recordings, 13-25% (16%) of the EAT recordings, and 15-26% (19%) of the AF recordings were "blinded".

Sinus rhythm

Variability of signal correlation

The eight recordings made during SR from the patients with PAF and AVRT all demonstrated a uniform signal correlation pattern with high values (10-percentile 0.76, 90-percentile 0.99), suggesting that CS was activated by a single wavefront.

The two recordings from the patients with AVNRT showed marked irregularity of signal correlation in proximal CS, most pronounced in signals from electrode pairs 5-6, and 9-10 (where $\rho_{\max}$ was between 0.47 and 0.73). See figure 2.

Direction of excitation

In 8 of 10 SR recordings, a right-to-left pattern is seen. The recordings from the 2 patients with AVRT differed from this. Both showed a more complicated pattern, indicating right-to-left conduction at the CS ostium and left-to-right at the distal end but with the area of signal collision slightly differing. See figure 2.

Ectopic atrial tachycardia

Variability of signal correlation

Of the three recordings made during EAT, one showed, with exclusion of the most proximal part of CS, high signal correlations suggesting one single wavefront propagating along CS (values between 0.90 and 0.99 except between electrodes 8 and 9 where $\rho_{\max}$ was 0.75). The other two had markedly lower and more variable values of correlations (values between 0.39 and 0.98). See figure 3.

Direction of excitation

The two EAT recordings from the patients with arrhythmia foci located in LA showed, except for the most distal part of the catheter, a distinct left-to-right conduction pattern. The recording made from the patient with focus in RA showed a consequent right-to-left conduction pattern. See figure 3.

Atrial fibrillation

Variability of signal correlation

Correlations between signals were, compared to SR, lower in the 4 recordings made during spontaneous paroxysmal AF (10-percentile 0.64, 90-percentile 0.95).

The recording made during induced AF showed values of $\rho_{\max}$ which were of lower variability compared to the results obtained from spontaneous AF (values between 0.72 and 0.85). See figure 4.

Direction of excitation

From all 5 recordings made during AF, left-to-right direction of conduction was seen except for the most distal part of the catheter in the recordings of spontaneous AF. The recording made during induced AF showed consistent left-to-right conduction in all calculations. See figure 4.

Fibrillatory rate

Mean fibrillatory interval, calculated from the atrial electrograms after QRS removal, ranged in the individual patients between 170 and 285 ms.

Multiple recordings from the same patient

In two of the patients with EAT, recordings were available both during atrial tachycardia and during SR (after radiofrequency ablation of the focus). The two SR recordings both showed an increased and less variable correlation (EAT range 0.39 to 0.98 and SR range 0.71 to 0.98 with one outlier at 0.38 at the tip of the catheter in the SR recording from the patient with LA focus).

During atrial tachycardia, the direction of excitation was in the LA case left-to-right and in the RA case right-to-left. After ablation, both SR recordings showed right-to-left direction.

In two patients with PAF, recordings made during AF and SR (after ablation) were available. In both AF and SR recordings, $\rho_{\max}$ was betweem 0.66 and 0.99 (with one outlier at 0.21 in one AF recording). The direction of excitation was left-to-right during AF in both patients. In the SR recordings made after ablation however, the direction changed to right-to-left in both cases.

Reproducibility

For all 5 AF recordings, it was seen that 5 or 10 seconds long segments would not produce an unambiguous result when interpretating the excitation direction. When 15 or 20 seconds long segments were used, the interpretation of excitation direction was the same in all 10 randomly positioned segments. Figure 5 shows a boxplot of the results from one AF recording.

Signal variability due to the catheter

The values of $\rho_{\max}$ in the pairwise calculations showed an up-and-down behaviour when plotted, most prominent in the EAT and AF recordings (Figures 3 and 4). This is an effect of the electrode spacing on the catheter not being equal, and the values of $\rho_{\max}$ calculated from signals from electrodes with 2 mm distances are higher than those calculated from signals from 5 mm spaced electrodes.

Discussion

Correlation function analysis

The method used in this study, correlation function analysis, is a mathematically simple way of finding the degree of similarity and time delay between two signal sequences. Using the correlation instead of the covariance has the advantage of the former being defined as having values $-1 \leq \rho \leq 1$, thus enabling a simpler comparison between two analyses and also a result to be 'perfect' if the value 1 is achieved.

Method validation

The validity of the method was evaluated by using the recordings made during SR. The general right-to-left direction (i.e., from the CS ostium to the distal part of CS) of impulse propagation obtained in 8 of 10 SR recordings in our study agrees with what would be expected from previous studies [7, 10, 11]. Furthermore, high values of the correlation coefficients show high consistency in propagation, as would be expected during sinus rhythm.

Further evaluation of the method was made using the results of the EAT recordings, since the focal origin of the tachycardia was known. The two patients with foci in LA had dominant left-to-right conduction and the patient with focus in RA had right-to-left, as expected. The recordings made on two of the patients with EAT after ablation of the foci, showed that both had a right-to-left propagation as would be expected during SR, indicating that the excitation of the atria originates from RA, i.e. the sinus node, and normal rhythm has been recovered.

Direction and speed of the conduction spread: anatomical considerations

Although the CS follows the curvature of LA, calculations based on signals from a catheter have to be treated as one-dimensional. This means that no exact calculations of conduction velocity can be made, since we do not know the angle at which the wavefront hits the catheter and this would obviously affect the result. In our material, rough estimates of the velocity show values between 1 and 2 m/s which is higher than would be expected from previous results where a normal velocity during AF was found to be 60 cm/s [2]. Using this value, the angle between the catheter and the wavefront can be estimated to be between 50 and 70 degrees.

It should be noted that the fibrillatory cycle length in the present atrial electrograms are of the same magnitude as those earlier documented to occur during periods of preferential conduction pattern in RA [2].

We have chosen to calculate the correlation function for a maximum time delay of ± 20 ms. With an electrode spacing of 6 mm, this means that we will find waves propagating faster than 30 cm/s. This is below what would be expected during AF [2] and was shown to be enough since the maximum time delay found in any calculation of $\tau_{\max}$ was 10 ms which means a velocity of 60 cm/s (the distance between the center of two electrodes being 6 mm).

Variability of the coronary sinus activation patterns during sinus rhythm

In several patients, correlation function analysis of the SR recordings revealed patterns deviating from the expected signal behavior both in terms of the signal correlation and the direction of the activation spread.

In the SR recordings from the patients with AVNRT, an irregular pattern could be seen in the correlation values. The recordings were made after these patients underwent radiofrequency catheter ablation with the curative lesion located in the vicinity of the CS ostium. A resulting effect on the conduction properties in this area may explain this finding.

The SR recordings from patients with AVRT showed a deviant left-to-right pattern (i.e., from the distal part of CS towards the CS ostium) which could not be readily explained. Our group has earlier reported a similar finding in patients with lone paroxysmal AF while the vast majority of the study subjects showed uniform right-to-left conduction along the coronary sinus during SR [11]. Recent studies of the left atrial activation during SR using non-contact mapping showed that such activation pattern could be an even more common phenomenon in patients with paroxysmal AF [12].

Atypical origin of the sinus impulse [13] or misbalance between the anterior and posterior interatrial conductive routes due to variability in either anatomy or function could underlie these findings. Several groups have recently extensively studied the variability of the anatomy of the posterior interatrial connections, which could explain differences in the left atrial activation pattern [5, 14, 15].

Consistent left atrial activation pattern during AF

The recording made during induced AF showed very regular, although low, values of correlation coefficients, and the predominant direction of conduction was very clearly left-to-right. This suggests that in this patient, one repetitive activation pattern exists along the CS, as part of AF. The observation of relatively stable, though irregular, activation sequences has earlier been repeatedly reported [16, 17, 18]. However, this behaviour is more typical for

the first cycles of AF.

The recordings made during spontaneous AF show irregular and low values of correlation with a very pronounced up-and-down behaviour (Figure 4), illustrating a complex signal which alters its appearance over short distances. All four recordings showed a very clear dominant left-to-right conduction pattern. It is tempting to suggest that this excitation direction is compatible with a concentric spread from the left upper pulmonary vein. The uniformity of this finding could however not be readily explained at this point in such a limited material. Alternatively, preexisting anomalies in interatrial connections, hampering right-to-left conduction across the interatrial septum in its posterior portion could be a possible interpretation of this sequence of CS activation. Despite the progress of endocardial mapping technologies, allowing three-dimensional reconstructions of the atrial activation sequences, their use in the AF settings up until now has been limited to the identification and ablation of the arrhythmogenic foci driving AF.

The position of CS allows evaluation of the conduction between the two atria. Unfortunately, the exact positioning of the catheter is not known and therefore we cannot say which, if any, of the electrodes record electrograms from RA. However, we may still be able to extract information on how the impulses move at the interatrial passage. During SR, it was seen that the impulse moved from right to left, probably utilizing the interatrial connections at the rear part of the atria. In the AF-recordings, conduction was predominantly left to right, indicating that during this arrhythmia there was no right to left conduction in these connections. Whether there was a left to right conduction, utilizing these connections, is of course not possible to answer, since the position of the catheter does not allow us to follow the signal into the right atrium.

Reproducibility

During AF, 15 seconds long, randomly selected segments would produce a plot of cumulative time delay leading to the same interpretation as the whole 60 s recording, whereas 10 seconds long segments would not be enough to produce reliable results. Included in these 15 seconds are the intervals removed due to ventricular disturbance, which amount to between 15 and 20 percent of the recording.

Limitations of the study

- The number of recordings during AF is small. In spite of this, the findings are encouragingly uniform.
- Electrodes within the CS record a composite signal where the major contributors are the ventricles and LA. However, there also may be a contribution from the muscular sleeve of CS itself, but because the mass of this sleeve is markedly smaller than the myocardial mass of the ventricles or LA, it is unlikely to affect the analysis.
- The spatial resolution of the electrodes allows neither three-dimensional reconstruction of the atrial activation, nor calculation of conduction velocity. Still, we have shown the feasibility of a method which could easily be expanded to cover more advanced ways of achieving endocardial data.

Conclusion

This pilot study shows that application of correlation function analysis to endocardially obtained unipolar electrograms is feasibile and allows assessment of the agreement between signals and determination of the direction of the activation spread. Validation was made during regular rhythms such as SR and EAT with known location of the ectopic foci.

This study also demonstrated that consistency in the electrical activity propagating along CS is common in AF.

The results can, due to the position of the CS, be used to derive mechanisms of impulse conduction between RA and LA. To the best of our knowledge, this has not been reported earlier.

References

[1] K. T. S. Konings, C. J. H. J. Kirchhof, J. R. L. M. Smeets, H. J. J. Wellens, O. C. Penn, and M. A. Allessie. High-density mapping of electrically induced atrial fibrillation in humans. *Circulation*, 89(4):1665–1680, April 1994.

[2] M. Holm, R. Johansson, J. Brandt, C. Lührs, and S. B. Olsson. Epicardial right free wall mapping in chronic atrial fibrillation, documentation of repetitive activation with a focal spread — a hitherto unrecognized phenomenon in man. *Europ. Heart. J.*, 18(2):290–310, February 1997.

[3] E.P. Gerstenfeld, A.V. Sahakian, and S. Swiryn. Evidence for transient linking of atrial excitation during atrial fibrillation in humans. *Circulation*, 86:375–82, 1992.

[4] J. M. Bourgery. *Traité complet de l'anatomie de l'homme.* Delanay, C. A., Paris, 1831–1854.

[5] D. Kozłowski, R. Kamiński, G. Piwko, M. Gawrysiak, A. Owerczuk, G. Piszczatowska, S. Budrejko, M. Kozłowska, B. Duda, and M. Grzybiak. Preliminary study of external interatrial muscle fascicles. *Folia Morphol.*, 61(2):97–101, 2002.

[6] S.Y. Ho, R.H. Anderson, and D. Sanchez-Quintana. Atrial structure and fibres: morphologic bases of atrial conduction. *Cardiovasc Res*, 54(2):325–36, May 2002.

[7] F. Roithinger, J. Cheng, A. SippensGroenewegen, J. L. Randall, L. A. Saxon, M. M. Scheinman, and M. D. Lesh. Use of electroanatomical

mapping to delineate transseptal atrial conduction in humans. *Circulation*, 100:1791–1797, 1999.

[8] D. C. Shah, M. Haïssaguerre, P. Jaïs, A. Takahashi, M. Hocini, and J. Clémenty. Dual input right to left atrial activation correlating with p wave morphology. In *PACE, NASPE Abstracts & Program*, 1999. Abstract 528.

[9] G. Bachmann. The inter-auricular time interval. *Am. J. Physiol.*, 41:309–20, 1916.

[10] P. G. Platonov, J. Carlson, M. P. Ingemansson, A. Roijer, A. Hansson, L. V. Chireikin, and S. B. Olsson. Detection of inter-atrial conduction defects with unfiltered signal-averaged p-wave ecg in patients with lone atrial fibrillation. *Europace*, 2:32–41, January 2000.

[11] P. G. Platonov, S. Yuan, E. Hertervig, O. Kongstad, A. Roijer, A. B. Vygovsky, L. V. Chireikin, and S. B. Olsson. Further evidence of localized posterior interatrial conduction delay in lone paroxysmal atrial fibrillation. *Europace*, 3:100–107, April 2001.

[12] V. Markides, R.J. Schilling, S.Y. Ho, A.W. Chow, D.W. Davies, and N.S. Peters. Characterization of left atrial activation in the intact human heart. *Circulation*, 107(5):733–9, February 2003.

[13] J.P. Boineau, T.E. Canavan, R.B. Schuessler, M.E. Cain, P.B. Corr, and J.L. Cox. Demonstration of a widely distributed atrial pacemaker complex in the human heart. *Circulation*, 77(6):1221–37, 1988.

[14] P.G. Platonov, L.B. Mitrofanova, L.V. Chireikin, and S.B. Olsson. Morphology of inter-atrial conduction routes in patients with atrial fibrillation. *Europace*, 4:183–92, 2002.

[15] S.Y. Ho, D. Sanchez-Quintana, J.A. Cabrera, and R.H. Anderson. Anatomy of the left atrium: implications for radiofrequency ablation of atrial fibrillation. *J Cardiovasc Electrophysiol*, 10(11):1525–33, 1999.

[16] S. Saksena, I. Giorgberidze, R. Mehra, M. Hill, A. Prakash, R.B. Krol, and P. Mathew. Electrophysiology and endocardial mapping of induced atrial fibrillation in patients with spontaneous atrial fibrillation. *Am J Cardiol*, 83(2):187–93, 1999.

[17] M. Adam, D. Fischetti, and A.S. Montenero. A typical pattern of activation in the right atrium during paroxysmal atrial fibrillation: the washing-machine phenomenon. *Cardiologia*, 44(1):63–8, 1999.

[18] L. Calo, R: Riccardi, M. Scaglione, F. Lamberti, R. Massa, L. Garberoglio, R. Antolini, and F. Gaita. Reproducibility of spontaneous initiations of atrial fibrillation. *G Ital Cardiol*, 29(4):435–440, 1999.

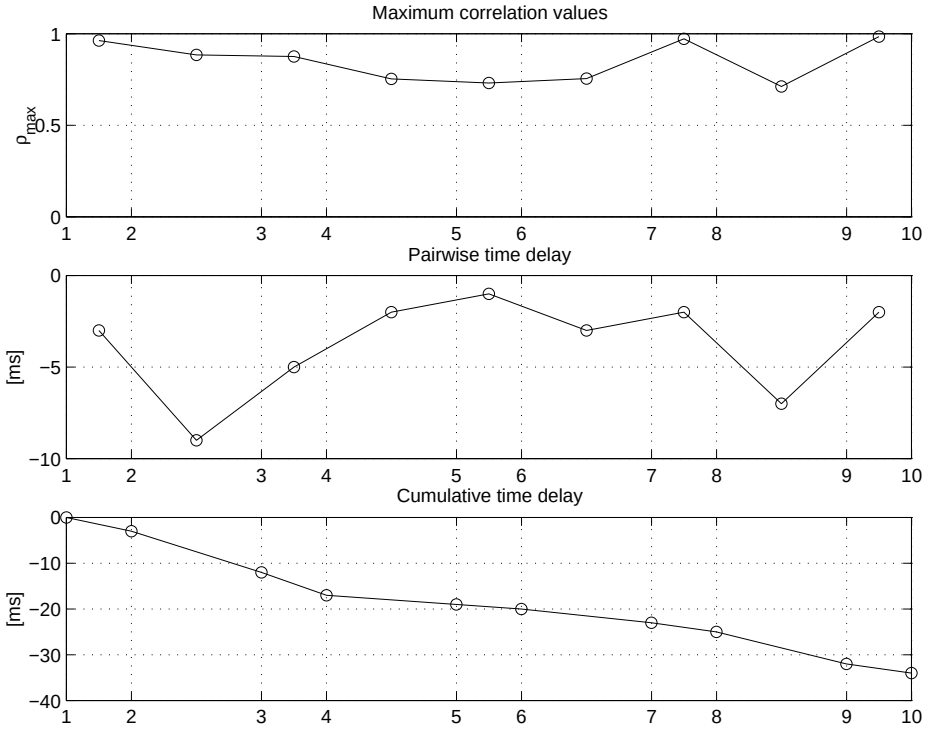

Figure 1: Example output of the correlation function analysis. The numbers on the x-axes refer to electrodes on the catheter, and the spacing of the gridlines reflect the positions of the electrodes. Top: $\rho_{\max}$ for each adjacent pair of electrodes. Middle: corresponding $\tau_{\max}$. Bottom: cumulative pairwise time difference, in this case indicating right-to-left conduction.

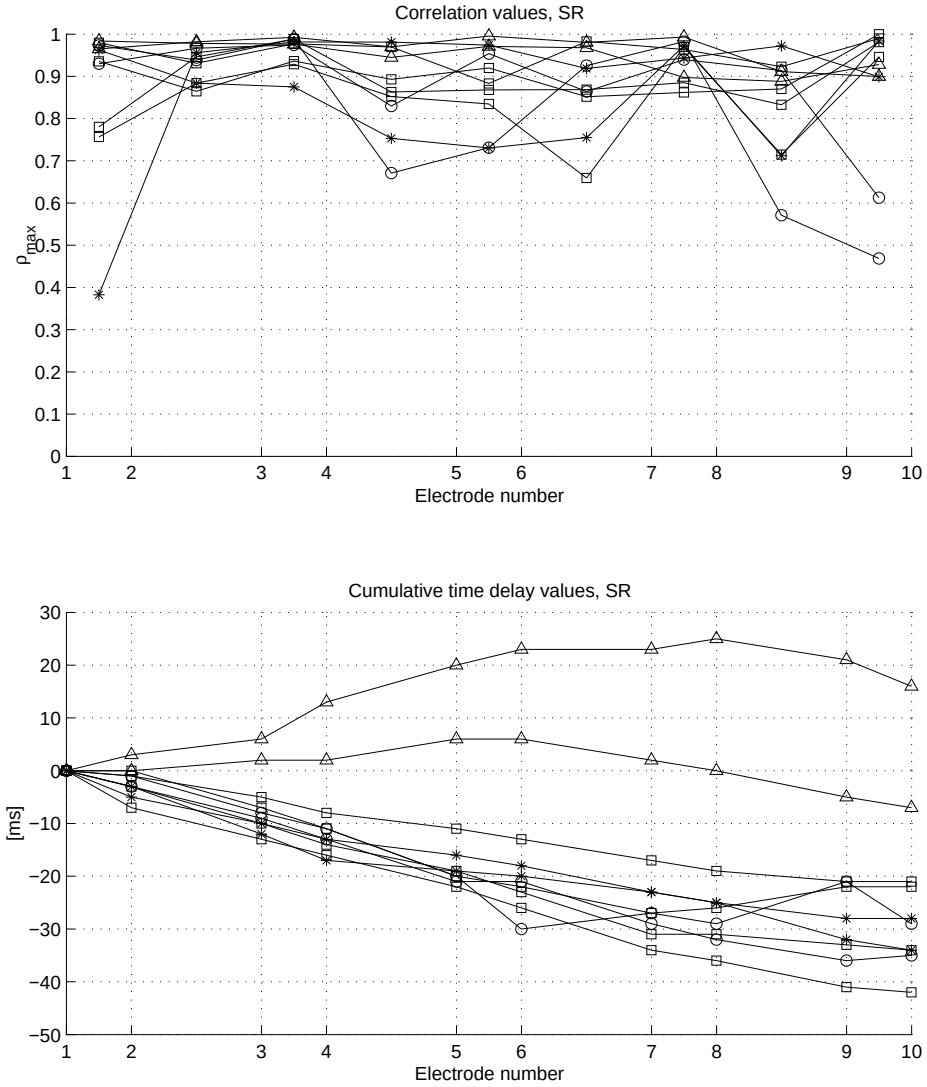

Figure 2: Correlation coefficients (top) and cumulative time delays (bottom) of sinus rhythm recordings. □: paroxysmal atrial fibrillation, △: AV re-entrant tachycardia, ◯: AV-nodal re-entrant tachycardia, ⋆: ectopic atrial tachycardia

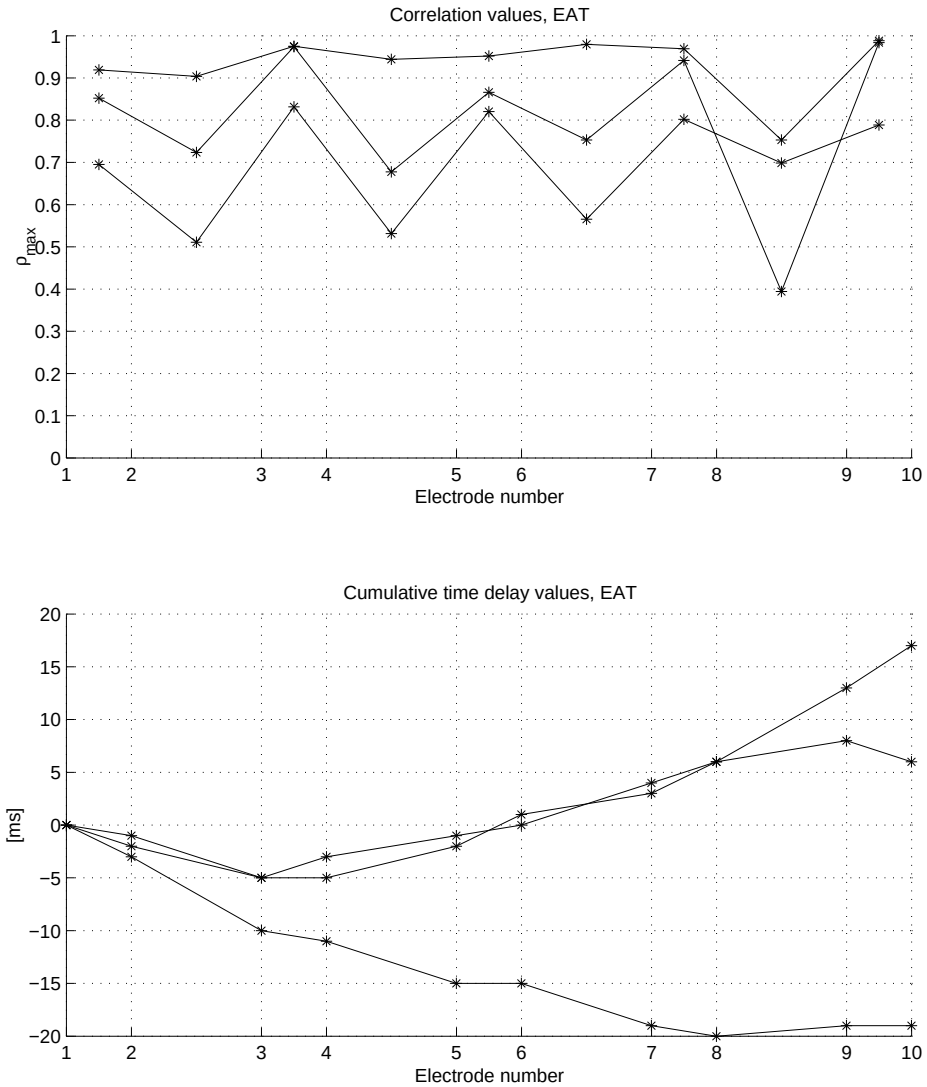

Figure 3: Correlation coefficients (top) and cumulative time delays (bottom) of ectopic atrial tachycardia recordings.

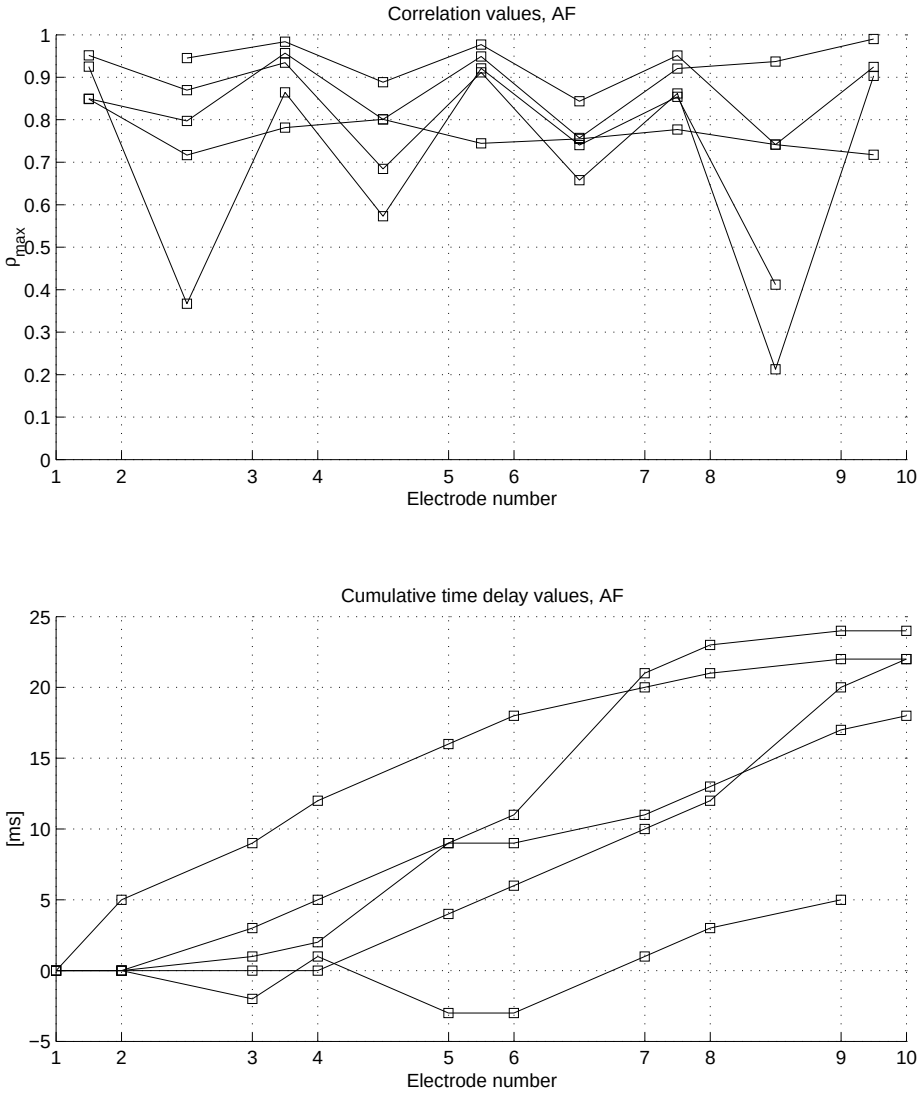

Figure 4: Correlation coefficients (top) and cumulative time delays (bottom) of atrial fibrillation recordings.

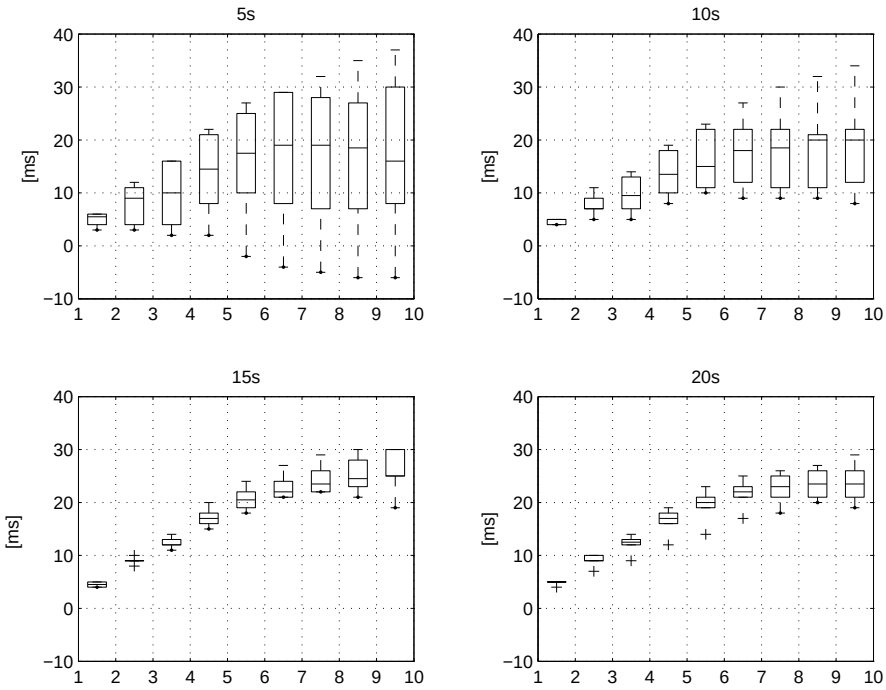

Figure 5: Boxplots of the calculations of cumulative time delays when using 10 randomly positioned segments of an AF recording. The boxplot shows median, upper and lower quartile, range, and outliers (+). Values on the x-axes denote electrode number. Top left: segment length 5 s, top right: 10 s, bottom left: 15 s, and bottom right: 20 s.