34th Annual International Conference of the IEEE EMBS
San Diego, California USA, 28 August - 1 September, 2012

Wavelet Variability of SA node Originated P waves in Atrial Fibrillation and in Signals with Ectopic Beats

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***Abstract*— Atrial fibrillation (AF) is one of the most common cardiac arrhythmia [1]. Electrical properties of the atrial myocardium may be related to the appearance of this type of arrhythmia. However ectopic beats, occurring normally in healthy people, disturb cardiac rhythm. Those beats arise from fiber outside the region of SA node. With this work we aim at highlighting differences in the atrial activity between healthy subjects, healthy subjects presenting many ectopic events and patients with AF. The X-Y-Z leads of vectorcardiography recordings are considered. Wavelet-based parameters are extracted from a window which represents atrial activity originated from SA node and compared between signals of the three groups. Results show differences potentially related to the conduction system of the atrium between healthy people and people with AF, as well as between healthy people and people with ectopic events. No difference was found from the analysis of SA node beats between people with AF and healthy with ectopic events.**

I. Introduction

The study of Atrial Fibrillation (AF) has drawn the attention of the scientific community to a great extent, for two main reasons: a) AF is the most common cardiac arrhythmia in clinical practice, as it affects approximately 1 % of the general population and up to 8 % of subjects over the age of 80 years [2], and b) it is a complex and intriguing phenomenon. Actually, the origin and persistence of AF result from a complex and multiscale interaction between triggers, substrate, and modulating factors involved in atrial remodeling [3]. Due to this complexity, the pathophysiology and dynamic evolution of AF differs among patients, which makes the intervention and prognosis rather challenging.

AF has been studied at different scales, from sub-cellular to organ level. In this work, starting from a macroscopic view (ECG), with orthogonal leads providing information regarding conduction in X-Y-Z axes, effort is paid to look into potential differences that point to conduction problems, and could help form patient sub-groups with AF.

The questions tackled in this work are the following:

- Do AF-prone patients have bigger percentage non-SA node originating beats? A positive answer could link to an increased percentage of arrhythmiogenic triggering factors.
- Is there a difference in the PR-interval between normal subjects and those prone to AF? This would link to a prolonged atrioventricular conduction time, due to volume differences or path differences.
- Are the P-waves of SA-node beats similar on average between normal subjects and subjects prone to AF? This would show whether the dominant conduction pattern is similar, or whether there are recognizable components in AF-prone subjects, that distinguish it from normal atrial activity.
- Do the P-waves of SA-node beats present different dynamics, i.e. variable beat-to-beat morphologies? This issue would go beyond the dominant morphology and examine the stability, or in opposite, the temporal variability of the p-wave morphology with respect to normal or AF subjects.
- Does the presence of many events of ectopic beats induce differences concerning the P-waves of SA-node beats, as compared with those of healthy subjects in the absence of ectopic beats or AF subjects? In other words, would the conduction variation (as expressed by the frequency of non-SA beat initiation) be linked with diverse p-wave morphology?

In order to shed some light in these questions, we followed an approach for the definition of those beats which can be considered as originating from the SA-node. Additionally we defined a window of interest which encloses the atrial activity and we applied a continuous wavelet transform in order to extract specific features. Finally we applied a statistical analysis to investigate the above mentioned questions. The approaches followed, and preliminary results are presented in the following sections.

II. Methology

A. Data and Preprocessing

In this study X-Y-Z leads of vectorcardiography (VCG) recordings were used. The sample frequency of the signals was at 1000Hz. We used three age matched categories of both genders. The first category includes signals from healthy volunteers but which contains great number of ectopic beats (HE), the second one signals from subjects suffering from paroxysmal AF (A) respectively whereas signals from both categories derived from the 1st Cardiology Clinic of AHEPA University Hospital of Thessaloniki. The last category contains signals from healthy subjects, without any event of ectopic depolarization, obtained from the

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Physionet's PTB Diagnostic ECG database (H). The signals whose heart rate was greater than 90 bpm were excluded.

A preprocessing denoising procedure was applied in all the signals. This procedure includes the removal of the linear trend, a 50 Hz notch filter for the removal of the electric current interference and a high-pass Butterworth filter with 1Hz cut off frequency, for the removal of the dc parameter.

TABLE I. DISTRIBUTION OF THE ANALYZED BEATS WITHOUT TAKING INTO ACCOUNT THE ECTOPIC BEATS.

Category	*Number of signals*	*Mean Heart Rate*
HE	22	76±7
A	20	66±9
H	22	67±9

B. Localization of the SA node Derived Beats

The P wave of an ECG signal represents the atrial depolarization and the direction of the electrical signal from the SA node towards the AV node. In this work we do not localize the accurate time of the beginning and the end of the P wave, but we do analyze a segment of the ECG signal, referred as segP, that contains the P wave. We investigate possible morphological differences of these segments between the signals obtained from healthy subjects and subjects with AFIB only for those beats which activation is attributed to the SA node. The method for the definition of segP is described in [4], where medical experts marked the fiducial points of one beat in each signal and population mean and standard deviation of PQ (meanPQ and STDPQ) were extracted. The segP, which represents atrial activity to be later analyzed, extends from Q-250mesc to Q-(meanPQ+STDPQ). The segP duration was set equal to 220msec. Finally, segP was detrended in order to preserve the isoelectric line.

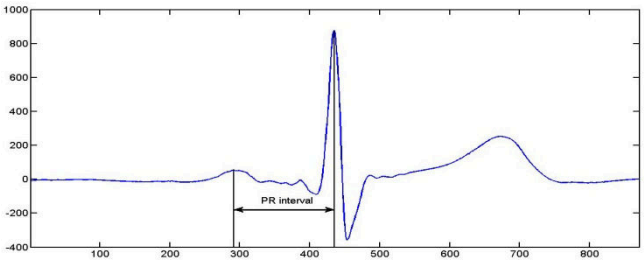

Figure 1. Definition of the PR interval.

The purpose of this work was the study of the P waves that are originated by the SA node i.e. excluding the atrial ectopic beats (AEB) and the non-SA originating beats (NSAB), both arising from fibers outside the region of the SA node. Firstly, the localization of AEBs was conducted, based on the following criterion: the RR intervals of an unstressed subject range between 500 and 1000 msec, while the RR interval of an ectopic beat is less than 450 msec, followed by an extended RR interval.

In both healthy and AF subjects, several NSABs may appear, and they are not indicative of pathology. Such arrhythmias are very minor and can be regarded as normal variants. However, studies suggest that more NSABs appear in AF subjects [5,6]**.** The localization of these beats requires the calculation of the time intervals within which normally a P wave, originated by SA node, must appear. The PR interval is estimated as the time between the peak of the P wave and the peak of the QRS complex. The distribution of the PR intervals averaging all the signals of H, HE, A groups, was proven to be normal (Fig.2). A PR interval that was out of the mean(PR)±2*std(PR) borders, here found as [67.41, 210.55msec], was characterized as NSAB, i.e. as derived from centers in the atrium other than SA node, and was therefore excluded from the analysis.

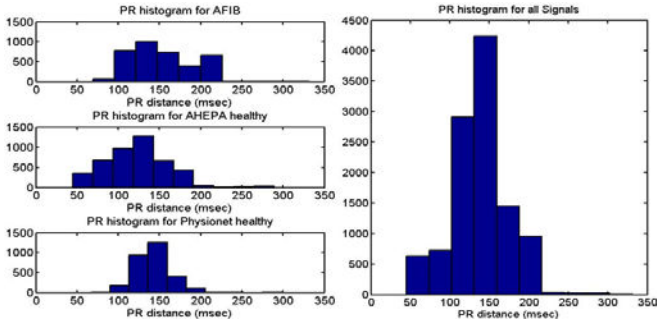

Figure 2. Histograms of the PR distance for all the signals'categories (left) and for all subjects included in the stady (righ).

As the P wave is easier to be detected in the X lead of the VCG, at this part of the analysis, for the identification of the beats, we observe only X lead (Fig. 3). Nevertheless, if a beat in the X lead is considered as NSAB, the appropriate beat is rejected from all the remaining lead, Y and Z, too.

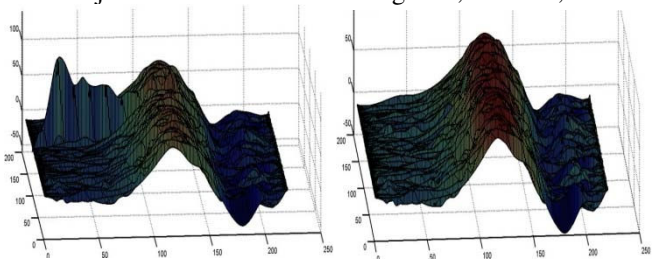

Figure 3. Depiction of all the windows of interest (left) and the SA node originated ones (righ) of a single signal, in X lead.

C. Wavelet Features and Statistical Analysis

Having detected the ECG segments which contain the SA node originated P waves, we apply a continuous wavelet transform in the window of interest. As proposed to the [4] the mother wavelet that performs better results is the wavelet of Morlet. The frequency range selected for the current analysis is of 6-100Hz (based on the central frequencies of the mother wavelet). The range of frequencies, and the corresponding scales, were divided to 4 bands, according to [4] and are referred in Table II.

TABLE II. WAVELET SCALE BANDS AND FREQUENCY CORRESPONDENCE

Scale Band	***Starting Frequency***	***Ending Frequency***
S1	6 Hz	16 Hz
S2	16 Hz	25 Hz
S3	25 Hz	50 Hz
S4	50 Hz	100 Hz

As a next step we compute the wavelet energy for each subject, for each scale band and each lead. We finally extract two parameters as described in detail in [4]. The first one is WBmeanE which represents the mean wavelet energy among beats within a scale band and the second one is the WBcvE for the coefficient of variation (cv) of energy among beats, which is the quotient of the standard deviation of the wavelet energy among beats to WBmeanE.

The outcome of the previous step is the computation of two features per band x four band x 3 leads. For the evaluation of the resulting features we adopted a statistical analysis based on one way t-test.

III. RESULTS

The consideration followed for the identification of the SA node originated beats verified the hypothesis of the existence of more arrhythmiogenic factors in the atrium cavity in signals derived from subjects with AF history compared to those from healthy subjects. As depicted in the Table III, the signals derived by subjects suffered by AFIB were proven to have greater percentage of NSABs compared to the signals derived by healthy subjects. Additionally, we observe the presence of many non NSABs in the signals obtained from healthy subjects in which there are plenty of AEBs. The percentage of NSABs is quite similar in HE and A groups, as depicted in Table III, where AEBs are not included.

TABLE III. DISTRIBUTION OF THE ANALYZED BEATS

Category	*SA beats*	*NSABs*	*Ratio of NSABs to SA beats*
HE	4152	303	7.2976
H	2874	10	0.3479
A	3389	255	7.5243

For the estimation of the PR interval, the PR histogram was calculated for all the categories of signals. The distribution of the PR interval for the H subjects was proved to be normal, with small standard deviation, unlike the one of the healthy subjects with many ectopic events whose standard deviation is higher. However, the distribution of the PR intervals in AF subjects was not proven to be normal. Additionally, the peak of the PR histogram was near 100-150 msec for the first two categories but in AF subjects a second peak was observed near 220msec (Fig. 2). This observation may be attributed to the presence of many NSABs, and also to the very fast depolarization of the atrium. This finding agrees with previous findings which show increased PR in AF risk subjects [7]. Additionally, this observation can be a hint of the presence of conduction path differences among healthy and AFIB subjects.

Beyond the differences in percentage of NSABs and AEBs, and in PR duration among the three groups, we evaluate the multiscale morphological characteristics of atrial activity in these groups. Hence, we analyze the WBmeanE and the WBcvE for statistical differences between the overmentioned groups. The results are depicted in Tables IV and V, respectively.

TABLE IV. STATISTICALLY SIGNIFICANT DIFFERENCES OF WBMEANE BETWEEN H AND A

Lead	Wavelet band	*ttest p*	*Mean of WBmeanE in H*	*Mean of WBmeanE in A*
X	1	$6.7969*10^{-5}$	$2.9888*10^{+6}$	$1.1526*10^{+6}$
X	2	0.0016	$7.6043*10^{+4}$	$2.8906*10^{+4}$
X	3	$6.2459*10^{-6}$	$1.8899*10^{+4}$	$4.5328*10^{+3}$
X	4	$4.3022*10^{-4}$	$2.3266*10^{+3}$	360.4321
Y	1	$6.5431*10^{-5}$	$5.8634*10^{+6}$	$1.4643*10^{+6}$
Y	2	$1.0640*10^{-5}$	$1.0895*10^{+5}$	$2.5796*10^{+4}$
Y	3	$7.1132*10^{-10}$	$4.2761*10^{+4}$	$5.7338*10^{+3}$
Y	4	$6.8250*10^{-7}$	$4.3504*10^{+3}$	595.6503
Z	3	$6.2947*10^{-5}$	$1.5450*10^{+4}$	$6.2553*10^{+3}$
Z	4	$2.3817*10^{-7}$	$2.0759*10^{+3}$	488.4514

The results depicted in Table IV show that in X and Y axis, mean energy is higher in H subjects than in A. Even though those results agree with findings from previous studies [8], yet it is characteristic that there are methodological differences concerning the analyzed frequencies as well as the window of interest.

TABLE V. STATISTICALLY SIGNIFICANT DIFFERENCES OF WBCVE BETWEEN H AND A

Lead	Wavelet band	*ttest p*	*Mean of WBcvE in H*	*Mean of WBcvE in A*
X	1	0.0155	0.1946	0.4951
X	2	0.0047	0.3141	1.2862
X	3	0.0014	0.3181	1.8518
X	4	$6.8964*10^{6}$	0.3016	2.2918
Y	1	0.0062	0.2043	0.5520
Y	2	$7.0439*10^{-4}$	0.2652	1.1345
Y	3	$1.1238*10^{-4}$	0.2385	1.8357
Y	4	$1.4024*10^{-5}$	0.2788	2.4183
Z	1	0.0244	0.1978	1.0653
Z	2	0.0036	0.2310	1.6751
Z	3	$2.6362*10^{-4}$	0.2317	2.1374
Z	4	$7.1753*10^{-6}$	0.2267	2.7320

However the analysis of WBcvE and WBmeanE between HE and A shows that there is no significant statistical difference. On the other hand the analysis of these features between H and He denotes that there is significant statistical difference shown in Table VI and VII.

TABLE VI. STATISTICALLY SIGNIFICANT DIFFERENCES OF WBMEANE BETWEEN H AND HE

Lead	Wavelet band	*ttest p*	*Mean of WBmeanE in H*	*Mean of WBmeanE in HE*
X	1	$1.4547*10^{-5}$	$2.9888*10^{+6}$	$9.6841*10^{+5}$

Lead	Wavelet band	*ttest p*	*Mean of WBmeanE in H*	*Mean of WBmeanE in HE*
X	2	$3.2863*10^{-4}$	$7.6043*10^{+4}$	$2.7661*10^{+4}$
X	3	$1.1167*10^{-6}$	$1.8899*10^{+4}$	$4.4471*10^{+3}$
X	4	$3.0624*10^{-4}$	$2.3266*10^{+3}$	444.0290
Y	1	$7.9715*10^{-5}$	$5.8634*10^{+6}$	$1.71*10^{+6}$
Y	2	$6.8369*10^{-6}$	$1.0895*10^{+5}$	$3.0787*10^{+4}$
Y	3	$1.0566*10^{-10}$	$4.2761*10^{+4}$	$5.7731*10^{+3}$
Y	4	$4.4918*10^{-7}$	$4.3504*10^{+3}$	774.9055

TABLE VII. STATISTICALLY SIGNIFICANT DIFFERENCES OF WBCVE BETWEEN H AND HE

Lead	Wavelet band	*ttest p*	*Mean of WBcvE in H*	*Mean of WBcvE in A*
X	1	$3.9710*10^{-4}$	0.1946	0.4053
X	2	0.0016	0.3141	0.9410
X	3	0.0033	0.3181	1.3561
X	4	$1.5612*10^{-4}$	0.3016	2.1375
Y	2	$1.1111*10^{-5}$	0.2652	1.1581
Y	3	0.0031	0.2385	1.1921
Y	4	$1.2873*10^{-5}$	0.2788	1.6039
Z	1	$5.2088*10^{-4}$	0.1978	0.6782
Z	2	$5.1051*10^{-4}$	0.2310	0.7595
Z	3	0.0012	0.2317	1.1327
Z	4	$3.4987*10^{-5}$	0.2267	1.7551

The analysis of the results concerning WBcvE as indicated above, coincide with the results of a previous study [2].This fact reveals the stability in time of the atrial signals derived from healthy people, as regards wavelet energy in all leads. The coefficient of variation in signals from people with AFIB, is two to even three times higher than in healthy ones, and especially in the leads X and Z, where statistically significant differences in all the scale bands are observed.

IV. DISCUSSION

This present study extends the methodology proposed by [4] aiming at the analysis of healthy and pathologic atrial activity.

Regarding the percentage of NSABs, we observed that the signals obtained from people with history of AFIB present greater number of beats that have not been activated by the stimulation of SA node but from other centers on the atrium fibers, which could link to an increased percentage of arrhythmiogenic triggering factors.

The distribution of the PR distance, excluding AEBs, presents differences between healthy and AF subjects, and is broader in AF, which could point at different or even variable conduction paths.

Having defined the window of interest that includes the P wave, we proceeded to the VCG analysis of the SA-originated beats, according to the criteria established, excluding AEBs and NSABs. The Morlet mother wavelet was used for the extraction of the wavelet parameters.

With the present analysis we can detect significant differences of the P-waves that come from the SA node of healthy people and people with AFIB, both concerning the coefficient of variation as well as the mean energy. The mean wavelet energy (WBmeanE) of X and Y axes is higher in H group, than in A and HE, in all wavelet scales, suggesting different dominant morphologies, and potentially different dominant conduction patterns. [3]. The high values of WBcvE observed on all the axes of the VCG in groups A and HE, expressing more variable beat-to-beat morphologies, indicated possible electrical instability of the beats deriving from SA node.

Furthermore, statistically significant differences were found after the analysis of atrial activity of the SA-originated beats, between healthy subjects HE and H (presenting or not plenty of ectopic beats). On the contrary no differences were found in the over mentioned beats among subjects with ectopic events and those with AF. Yet, study of the beats that do not come from SA node is required in order to understand in depth the abnormalities that may lead to the atrial arrhytmiogenesis.

V. CONCLUSION

The analysis of the atrial activity of the SA node originated beats denotes the presence of arrhythmiogenic substrate on the atrium which leads to different conduction patterns in AF-prone subjects and in healthy ones with great number of ectopis. Present analysis can help us in the direction of the better understanding of conduction mechanisms of the atrium, and potentially in the characterization of the AF-related substrate.

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