

## [ Original article ]

# Characteristics of the atrial repolarization phase of the ECG in paroxysmal atrial fibrillation patients and controls

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**Objective** The aim of this study is to characterize the observable segment of the atrial repolarization (Ta wave) of the standard ECG during sinus rhythm in paroxysmal atrial fibrillation (PAF) patients and controls.

**Methods** Ta and P waves were measured from signal-averaged recordings of a standard 12-lead ECG in 40 patients, 20 with PAF, but in SR at the time of recording, and 20 healthy controls. Wave amplitudes and morphologies were measured.

**Results** There were no significant differences in Ta amplitude between the PAF patients and controls. A subgroup analysis of patients on and off anti-arrhythmic drugs also showed no significant differences in Ta amplitudes. For both groups Ta wave had opposite polarity to the monophasic P wave. Biphasic P waves had Ta polarity opposite to the initial phase of the P wave. Ta wave amplitudes were largest in leads II (mean  $\pm$  SD,  $-25 \pm 16 \mu\text{V}$ ), V2 ( $-22 \pm 10 \mu\text{V}$ ), V3 ( $-21 \pm 10 \mu\text{V}$ ) and V4 ( $-20 \pm 8 \mu\text{V}$ ). A significant correlation was found between Ta and P wave amplitudes, leads recording larger P waves also had larger Ta waves (PAF group:  $r = 0.15$  ( $P = 0.02$ ) PAF vs  $r = 0.33$  ( $P = 0.002$ ) HC).

**Conclusion** No differences in the amplitude of the observable section of the atrial repolarization phase of the ECG could be observed between patients with PAF and controls. Ta wave correlates with the corresponding P wave in both amplitude and polarity.

**Keywords** Atrial repolarization – atrial depolarization – Ta wave – P wave – morphology – atrial fibrillation.

## INTRODUCTION

Atrial repolarization has received little attention to date and little has been published on the atrial repolarization phase on the electrocardiogram (ECG). This is probably due to the fact that much of this phase of the ECG is obscured by the large ventricular activity. The only observable part of atrial repolarization is during the short PQ segment of the ECG. The atrial repolarization phase is known as the atrial T wave or Ta wave. Its amplitude is small and signal averaging is necessary to derive accurate measurements<sup>1,2</sup>.

Given the importance of abnormal ventricular repolarization in the development of ventricular arrhythmias,

the study of the atrial repolarization and its association with atrial arrhythmia is timely. Analysis of the Ta wave during periods of sinus rhythm might contain important information about propensity to atrial arrhythmias, similar to that of the T wave in the case of some ventricular arrhythmias<sup>3</sup>. Intracardiac electrophysiology studies have already shown abnormalities of atrial repolarization in patients with paroxysmal atrial fibrillation<sup>4</sup>. These include, for example, a progressive shortening of the atrial refractory period<sup>5</sup>. If these changes persist during periods of sinus rhythm, abnormalities of atrial repolarization might be apparent on the Ta wave. Of course it is not possible to derive any Ta wave during atrial fibrillation itself since the continuous rapid activation of the atria means that there is no PQ segment on the ECG. One small previous study was unable to show any significant difference in Ta amplitude or polarity between patients with a history of AF and those without<sup>6</sup>. Others have shown that in patients with complete AV-block, where the full atrial repolarization phase may be observable (dependent upon AV synchronization) the Ta peak is usually located over 200 ms

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Received 25 May 2015; accepted for publication 2 June 2015.

after the onset of the *P* wave. This means that in patients with normal AV-conduction it is located typically in the subsequent QRS complex<sup>5</sup>. Therefore in normal sinus rhythm the end of the Ta wave is completely obscured by the large ventricular activity so atrial repolarization duration cannot be measured. However, the morphology of the Ta wave might yield diagnostic information but so far there have been few pioneering descriptions of the morphology of the Ta wave and its relationship to the *P* wave<sup>7,8</sup>. Therefore the aim of this study was to characterise atrial repolarization in terms of its amplitude and polarity from the short observable segment of the surface ECG. In particular relating the Ta wave to the corresponding atrial depolarisation phase, the *P* wave, in patients with paroxysmal atrial fibrillation (PAF) and controls without any arrhythmia or cardiac diagnoses.

## METHODS

### Patient population

The patient group comprised 20 subjects, all males, diagnosed with PAF, but all in SR at the time of data collection (PAF group). Patients were recruited from those referred to the Arrhythmia Services at the Freeman Hospital and listed for catheter ablation of AF for standard clinical indications. Median (range) duration of diagnosis of AF was 4.5 (1-15) years. Cardioactive medications of the PAF group were antiarrhythmic drugs, ACE inhibitors, antiplatelet drugs, anticoagulation therapy, beta blockers in 14 (70%), 5 (25%), 7 (35%), 5 (25%) and 7 (35%) patients, respectively. The healthy control group (HC group) comprised 20 males without arrhythmia or known heart disease. Both groups were similar in age (mean (range)) (HC vs PAF: 49 (42-62) vs 49 (44-58) years;  $P > 0.05$ ) and heart rate in SR (mean  $\pm$  SD) ( $64 \pm 11$  vs  $63 \pm 11$  beats per minute;  $P > 0.05$ ). No atrioventricular block of any degree was present in the subjects of both groups.

### Data acquisition and analysis

Twelve-lead ECG was recorded in all subjects for 600 s. ECG was sampled at 500 Hz with an amplitude resolution of less than  $5 \mu\text{V}$ . Recordings were transferred to a central database for subsequent off-line processing. Pre-processing of signals consists of 3 infinite impulse response elliptical filters: a high pass filter with cut-off frequency of 0.05 Hz, a low pass filter with cut-off frequency of 100 Hz and a notch filter to suppress main line interference.

A QRS detector was used to identify individual cardiac beats and detected R wave peaks were manually

verified by visual inspection. Ectopic beats were removed from the analysis. Knowing the locations of the R peak in each beat, a window containing the atrial beat was defined. The window was chosen such that it included a segment before onset of the *P* wave, the *P* wave and the observable Ta wave up to the onset of the Q wave. These features were captured by a window of 270 ms duration located to start 320 ms before the R peak in each beat.

For each lead the average atrial beat was computed from the collection of all beat windows. To minimise the effect of *P* and Ta wave morphology changes due to RR changes, only beats with RR intervals within 1% of the most common (mode) were used to generate the average atrial beat in each lead. As a further measure to ensure correct alignment of atrial beats, the beats were aligned by maximum correlation before calculating the average atrial beat.

On the resulting average beat an ‘isoelectric’ window of 80 ms duration positioned before the onset of the *P* wave was selected to define the baseline amplitude. The mean value in this window was subtracted from the average beat to remove the baseline offset. The standard deviation calculated in the same window was considered as an estimation of the noise on the signal.

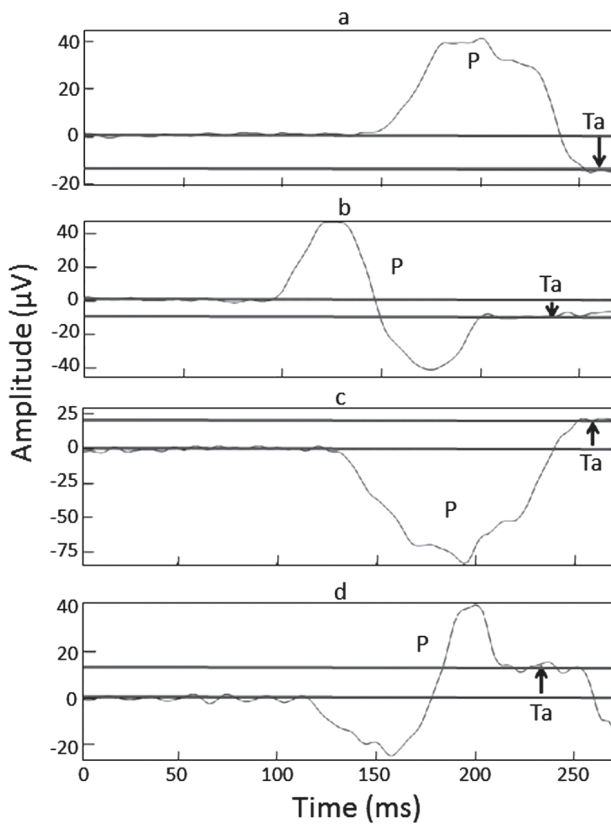
Each *P* wave was classified on the basis of its morphology as “up-right” (one positive phase), “biphasic up-down” (two phases, the first positive and the second negative), “down-right” (one negative phase), “biphasic down-up” (two phases, the first negative and the second positive).

The observable Ta wave is generally seen as a relatively flat waveform, offset from the baseline, directly after the end of the *P* wave. Hence, the Ta wave amplitude was estimated as the average amplitude in a window after the end of the *P* wave. This window was defined manually in the flattest segment before Q wave onset. Examples of the four *P* wave morphologies and Ta waves are shown in figure 1.

Ta waves were measured in all the 12 leads for all the subjects of the two groups. The same was performed for the *P* waves; their amplitude was measured as the peak for monophasic morphologies and as the difference between the positive and the negative peaks for biphasic morphologies.

### Statistical analysis

Statistical analysis of the data was performed in Matlab 7.6.0 and in SigmaPlot 8.0 (Systat Software Inc). The unpaired Student’s T-test with two tails was used to test for significant differences in the Ta wave amplitude between groups (PAF-HC; AADs on -AADs off), while to test differences between (i) the 12 leads and



**Fig. 1** Four examples of atrial depolarization (*P* wave) and repolarization (*Ta* wave) for different *P*-wave morphologies. (a) "Up-right" *P* wave and negative *Ta* wave, (b) "biphasic up-down" *P* wave and a negative *Ta* wave, (c) "down-right" *P* wave and positive *Ta* wave, (d) "biphasic down-up" *P* wave and positive *Ta* wave, here the onset of the QRS complex is visible at 250 ms.

(ii) morphology categories, the ANOVA test was used. Correlation between *Ta* wave amplitude and *P* wave amplitude was analysed with the Pearson's coefficient (*r*). The differences were considered significant with  $P < 0.05$ .

## RESULTS

### Ta wave amplitude

Table 1 shows the signed and absolute *Ta* wave amplitudes for all the leads in the PAF and HC groups. No significant differences in *Ta* amplitude were identified between the groups. A subgroup analysis of patients on and off anti-arrhythmic drugs also showed no significant differences in *Ta* amplitudes (table 2). However, this result needs to be treated cautiously due to the small number of subjects in this subgroup analysis.

Combining data from both the PAF and HC groups, *Ta* amplitude differed significantly between leads. Leads with the largest *Ta*-wave amplitude were II (mean  $\pm$  SD,  $-25 \pm 16 \mu\text{V}$ ), V2 ( $-22 \pm 10 \mu\text{V}$ ), V3 ( $-21 \pm 10 \mu\text{V}$ ), V4 ( $-20 \pm 8 \mu\text{V}$ ). Noise amplitude on the signal was of the order of a 1/10<sup>th</sup> of the *Ta* wave amplitude.

### Correlation between *Ta* wave amplitude and *P* wave amplitude

We assessed the correlation between the magnitude of atrial depolarization and repolarization phases using absolute amplitude values of *P* and *Ta* waves in all leads (figure 2). As expected there was a significant positive correlation in both groups – indicating that larger *Ta* waves are associated with larger *P* waves. However, the correlation was weaker for the PAF group than for the HC subjects [PAF:  $r = 0.15$  ( $P = 0.02$ ); HC:  $r = 0.33$  ( $P = 0.002$ )].

### Relationship between *Ta* wave polarity and *P* wave morphology

The majority of *P*-wave morphologies in leads I, II and the left precordial leads had "up-right" *P* waves (Table 3).

**Table 1** Actual and absolute *Ta* wave amplitude for PAF and HC groups among the 12 leads

| Lead           | Mean <i>Ta</i> amplitude [stdev] ( $\mu\text{V}$ ) |          | <i>P</i> value | Mean absolute <i>Ta</i> amplitude [stdev] ( $\mu\text{V}$ ) |          | <i>P</i> value |
|----------------|--|----------|----------------|---|----------|----------------|
|                | PAF group  | HC group |                | PAF group   | HC group |                |
| I              | -14 [8]  | -13 [6]  | 0.50           | 14 [8]  | 13 [6]   | 0.38           |
| II             | -22 [8]  | -27 [10] | 0.13           | 22 [8]  | 27 [10]  | 0.12           |
| III            | -8 [10]  | -11 [12] | 0.42           | 10 [8]  | 14 [7]   | 0.05           |
| aVr            | 19 [7]   | 20 [8]   | 0.81           | 19 [7]  | 20 [8]   | 0.61           |
| aVL            | -5 [7]   | -1 [6]   | 0.13           | 7 [5]   | 5 [3]    | 0.16           |
| aVf            | -15 [9]  | -19 [9]  | 0.11           | 15 [8]  | 19 [9]   | 0.17           |
| V <sub>1</sub> | -10 [11]   | -12 [14] | 0.56           | 13 [8]  | 14 [13]  | 0.48           |
| V <sub>2</sub> | -20 [16]   | -22 [11] | 0.61           | 24 [20]   | 22 [10]  | 0.56           |
| V <sub>3</sub> | -22 [17]   | -18 [12] | 0.43           | 26 [9]  | 19 [10]  | 0.05           |
| V <sub>4</sub> | -20 [11]   | -18 [11] | 0.51           | 22 [7]  | 18 [10]  | 0.19           |
| V <sub>5</sub> | -15 [9]  | -17 [8]  | 0.54           | 17 [5]  | 17 [8]   | 0.65           |
| V <sub>6</sub> | -12 [7]  | -15 [8]  | 0.29           | 13 [5]  | 15 [7]   | 0.34           |

**Table 2** Ta wave amplitude for PAF patients on (AADs on) and off (AADs off) antiarrhythmic drugs

| Lead | Mean actual Ta amplitude [stdev] ( $\mu\text{V}$ ) |          | P value |
|------|--|----------|---------|
|      | AADs on  | AADs off |         |
| I    | -14 [9]  | -14 [8]  | 0.88    |
| II   | -20 [7]  | -28 [9]  | 0.05    |
| III  | -6 [10]  | -12 [11] | 0.23    |
| aVr  | 18 [7]   | 22 [7]   | 0.25    |
| aVI  | -6 [7]   | -2 [6]   | 0.29    |
| aVf  | -12 [8]  | -20 [9]  | 0.07    |
| V1   | -10 [12]   | -9 [9]   | 0.86    |
| V2   | -19 [14]   | -21 [23] | 0.82    |
| V3   | -22 [13]   | -22 [26] | 0.99    |
| V4   | -18 [11]   | -26 [7]  | 0.11    |
| V5   | -14 [9]  | -20 [6]  | 0.14    |
| V6   | -11 [7]  | -16 [5]  | 0.15    |

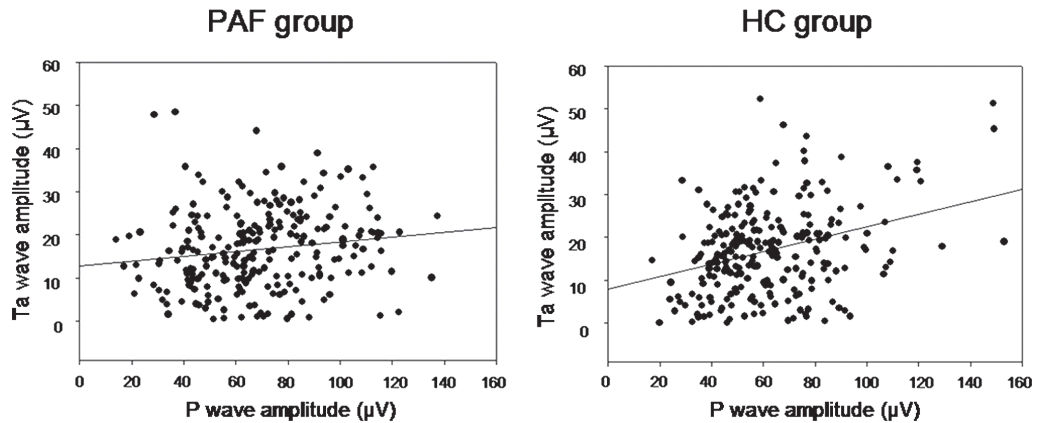
Lead aVr always had a “down-right” P wave morphology. In other leads morphologies were more variable.

Ta wave polarity and amplitude were significantly different among the four P wave morphology categories ( $P=0.003$ ) for PAF and HC groups (figure 3). “up-right” P waves had negative Ta waves in both groups (PAF:  $-18 \pm 13 \mu\text{V}$ ; HC:  $-19 \pm 11 \mu\text{V}$ ). “biphasic up-down” P waves also had negative Ta waves (PAF:  $-12 \pm 7 \mu\text{V}$ ; HC:  $-14 \pm 11 \mu\text{V}$ ). “Down-right” P waves had positive Ta waves for both groups (PAF:  $15 \pm 9 \mu\text{V}$ ; HC:  $17 \pm 9 \mu\text{V}$ ). “Biphasic down-up” P waves also had positive Ta waves (PAF:  $7 \pm 6 \mu\text{V}$ ; HC:  $4 \pm 3 \mu\text{V}$ ).

### DISCUSSION

Signal-averaged P wave is the most validated method of recording P-waves for detailed analysis<sup>9-12</sup>. In the present study we extend this methodology to allow

**Fig. 2** Relationship between absolute P wave amplitude and the absolute Ta wave amplitude for PAF and HC groups. Pearson's correlation was weaker for the PAF group ( $r=0.15$  ( $P=0.02$ )) than for the HC group ( $r=0.33$  ( $P=0.002$ )).

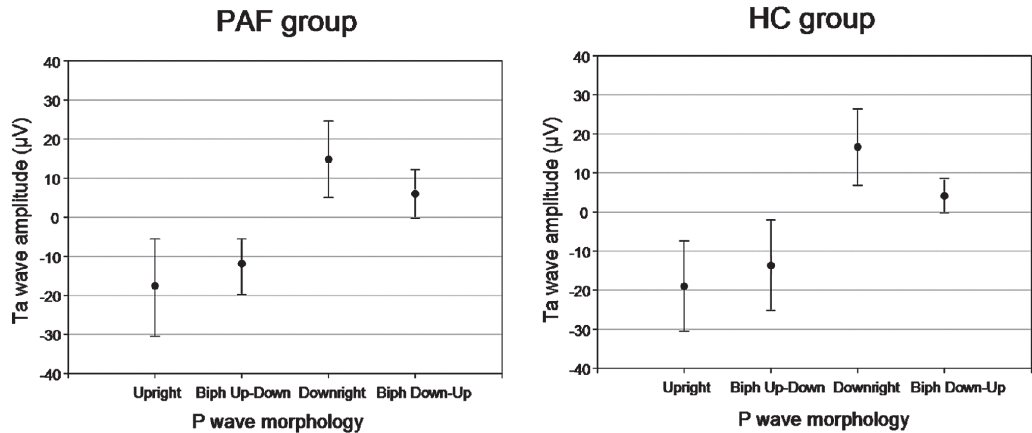


**Table 3** The distribution of P wave morphologies in the 12 leads for PAF and HC groups

| Lead           | Up-right |    | Biphasic up-down |    | Down-right |    | Biphasic down-up |    |
|----------------|----------|----|------------------|----|------------|----|------------------|----|
|                | PAF      | HC | PAF              | HC | PAF        | HC | PAF              | HC |
| I              | 20       | 20 | -                | -  | -          | -  | -                | -  |
| II             | 18       | 20 | 2                | -  | -          | -  | -                | -  |
| III            | 4        | 11 | 11               | 5  | 5          | 3  | -                | 1  |
| aVr            | -        | -  | -                | -  | 20         | 20 | -                | -  |
| aVI            | 17       | 10 | -                | 1  | -          | 2  | 3                | 7  |
| aVf            | 11       | 17 | 8                | 3  | 1          | -  | -                | -  |
| V <sub>1</sub> | -        | 4  | 17               | 15 | 3          | 1  | -                | -  |
| V <sub>2</sub> | 16       | 15 | 2                | 5  | 1          | -  | 1                | -  |
| V <sub>3</sub> | 18       | 19 | -                | -  | 1          | 1  | 1                | -  |
| V <sub>4</sub> | 18       | 20 | 1                | -  | -          | -  | 1                | -  |
| V <sub>5</sub> | 19       | 20 | -                | -  | -          | -  | 1                | -  |
| V <sub>6</sub> | 19       | 20 | -                | -  | -          | -  | 1                | -  |

The table specifies the number of subjects with each P wave morphology category in all leads. P wave morphologies are the following: “up-right” (one positive phase), “biphasic up-down” (two phases, the first positive and the second negative), “down-right” (one negative phase), “biphasic down-up” (two phases, the first negative and the second positive).

**Fig. 3** Relation between P wave morphology categories and Ta wave amplitude.



analysis of the Ta segment and provide insights into atrial repolarization from the surface ECG. Previous studies have reported differences in the duration and timing of the Ta wave peak between healthy subjects and patients with AF history<sup>5,10,13,14</sup>. In our study we focused on the amplitude and polarity of the Ta wave, observable in SR, and on its relationship to P wave morphology.

Leads II, V2, V3 and V4 had the largest Ta wave representation. Lead II would be expected to be the lead most parallel to the atrial repolarization ‘propagation’ vector, while the first left precordial leads are the nearest ones to the atria. Similar to the findings of others previously<sup>6</sup>, we found that the observable section of the atrial repolarization phase of the ECG in patients with PAF was similar to that in healthy controls. Moreover, we were not able to see any differences in amplitude between PAF patients in therapy with antiarrhythmic drugs from PAF patients without. Since in sinus rhythm we are only able to examine the observable section of atrial repolarization in the PQ interval of the ECG differences in atrial repolarization outside of this interval can only be investigated by invasive studies.

As expected, a positive correlation was found between the absolute value of Ta wave and P wave amplitudes. This was stronger in the HC group. The weaker correlation between magnitude of depolarization and repolarization phases in the PAF group might indicate that atrial repolarization is less organised with increased dispersion of atria repolarization in PAF patients.

A strong link was observed between P and Ta wave morphologies. Four distinct P-wave morphologies were identified, each having a consistent pattern of Ta wave polarities: negative Ta waves for “up-right” and “biphasic up-down” P waves; positive Ta waves for “down-right” and “biphasic down-up” P waves. The Ta wave invariably has the opposite polarity to the P wave and, in the case of biphasic P waves, the opposite polarity to the first phase of the P wave. This indicates that repolarization ‘propagation’ proceeds in a similar but opposite direction to depolarization in the atria and this does not change in PAF during SR. A limitation of our study was that the majority of the PAF patients received anti-arrhythmic medications and the effect of these on the Ta wave are unknown. Further analysis of atrial repolarization on a larger group of patients, stratified by anti-arrhythmic drugs, atrial dimensions and severity of arrhythmic burden is warranted.

## CONCLUSION

The observable segment of the atrial repolarization phase of the ECG exhibits a consistent morphological relationship with the atrial depolarization phase and this relationship is unaltered in PAF.

**CONFLICT OF INTEREST:** none.

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