# Atrial Signal Amplitude in Healthy Subjects and Patients with Atrial Fibrillation from the 12-Lead ECG

P Langley, A Murray

University of Newcastle upon Tyne, Newcastle upon Tyne, UK

## Abstract

*The atrial electrical signal or P wave is becoming more widely studied because of the large incidence of atrial arrhythmias. There are few reports quantifying the spatial distribution of atrial signal amplitude in the 12-lead ECG. Our aims were to quantify the spatial distribution of atrial signal amplitudes in groups of healthy subjects and patients with atrial fibrillation, and to develop a simple model of atrial depolarisation based on a single dipole. Amplitude was significantly smaller ($p < 0.05$) in leads I, II, $aV_R$, $aV_F$, V5 and V6, and significantly larger in lead V1 in atrial fibrillation compared with those of the healthy group. A single dipole model was able to qualitatively simulate the body surface atrial electrical signal in specific subjects from these groups.*

## 1. Introduction

Atrial arrhythmias are the most common arrhythmia seen in clinical practice [1]. The P wave is the characteristic feature of the ECG corresponding to atrial depolarisation.

P wave amplitude has been proposed as a marker for predicting recurrence of atrial fibrillation (AF) after treatment [2], and for identifying pulmonary vein origin of atrial premature complexes [3, 4].

Relative to the ventricular components of the ECG, namely QRST waves, the atrial components are very small, making analysis of the P wave difficult. Multiple P wave signal averaging is often used to remove noise. Few studies have investigated the optimal recording sites for analysing the atrial signal or compared the differences between atrial signal amplitude in patients and healthy subjects.

The aim was to quantify differences in the spatial distributions of atrial signal amplitude in healthy subjects and patients with atrial fibrillation in the 12-lead ECG. Also, we have begun to investigate how well a single dipole model is able to simulate the body surface P and atrial fibrillation waveforms.

## 2. Methods

### 2.1. Data collection

Each 12-lead ECG of 25 subjects without heart disease and 25 patients with atrial fibrillation were recorded for 5 minutes and saved to a computer file at a sample rate of 500 Hz and amplitude resolution of 4.9 μV. For the ECGs of healthy subjects, signal averaging of beats was used to derive a single noise free P wave for each lead and the peak-to-peak amplitude was measured. In atrial fibrillation the rapid atrial activations prevent the use of signal averaging so the peak-to-peak amplitude of ECG lead sections containing only atrial activations were determined at each beat. A single value of atrial signal amplitude for each lead in these patients was then calculated as the median amplitude of these sections. Mean and standard deviation amplitude were calculated across subjects/patients for each lead. Differences in amplitudes of each lead between healthy and atrial fibrillation groups were assessed using the two sample t-test.

### 2.2. Single dipole model

We explored the possibility of simulating the body surface atrial electrical activity using a single dipole model. The model consisted of a dipole with fixed base and variable amplitude and orientation, placed within a cylindrical torso at the approximate anatomical location of the heart within a representation of a human torso. The cylindrical torso was 50 units in height with major axis diameter of 17 units and minor axis diameter of 13 units, and comprised 196 rectangular elements. The base of the dipole was located 2 units forwards (x), 5 units to the left (y) and 12 units up (z) from the centre of the cylinder. The model is illustrated in figure 1. The time varying dipole source was defined by a vector in Cartesian coordinates. The x, y and z components of the dipole source were determined from leads V2, I and $-aV_F$ of the 12-lead ECG. These were obtained from the ECGs of a single healthy subject, to simulate P waves, and a patient with atrial fibrillation to simulate atrial fibrillation waves. The potential of each element on the torso surface was

0276–6547/04 $20.00 © 2004 IEEE

Computers in Cardiology 2004;31:421–424.

calculated as

$$\Phi = l \cos\theta$$

Where $l$ is the magnitude of the dipole vector and $\theta$ is the angle between the dipole vector and the line joining the base of the dipole with the surface element.

We visually compared the real and simulated waveforms.

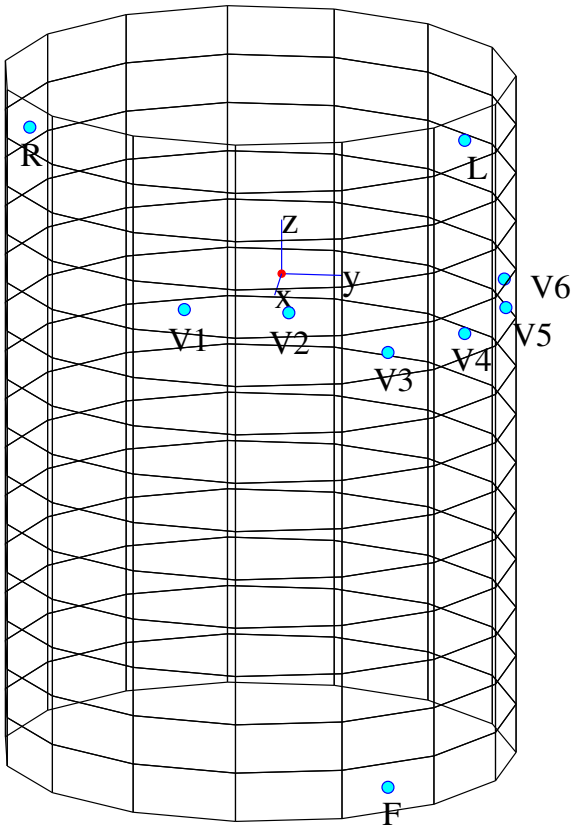


Figure 1. Single dipole model. Illustrates the base of the dipole and associated coordinate system located within a cylindrical torso on which the electrode positions for the 12-lead ECG are indicated.

## 3. Results

### 3.1. Atrial signal amplitude

Mean (sd) P wave amplitudes (μV) in healthy subjects were 94 (23) I, 126 (32) II, 75 (24) III, 108 (22) $aV_R$, 58 (15) $aV_L$, 90 (30) $aV_F$, 86 (22) V1, 89 (29) V2, 87 (24) V3, 81 (20) V4, 75 (17) V5 and 70 (16) V6.

For the patients with atrial fibrillation the amplitudes were 54 (17) I, 75 (26) II, 78 (24) III, 54 (17) $aV_R$, 58 (16) $aV_L$, 71 (24) $aV_F$, 110 (45) V1, 97 (38) V2, 91 (43) V3, 72 (26) V4, 60 (24) V5 and 50 (20) V6.

Amplitude was significantly smaller ($p < 0.05$) in leads I, II, $aV_R$, $aV_F$, V5 and V6, and significantly larger in lead V1 for the atrial fibrillation group. Figure 2 illustrates the spatial distribution of the atrial signal amplitudes in the 12-lead ECGs.

### 3.2. Single dipole model

Figure 3 shows the atrial signal vector loops for a healthy individual and a patient with atrial fibrillation. These vector loops defined the vector dipole for the single dipole model of these subjects. Figure 4 shows the simulated P waves against those of the normal subject and figure 5 those for the patient with atrial fibrillation.

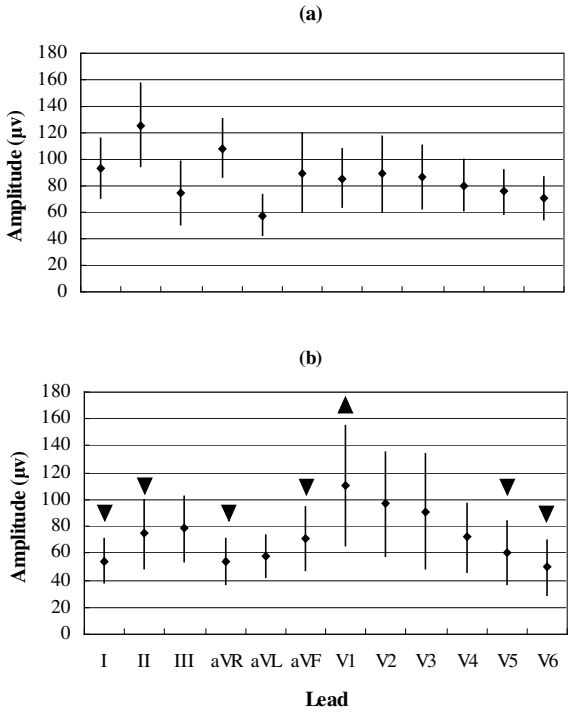


Figure 2. Atrial signal amplitude in (a) healthy subjects and (b) atrial fibrillation patients. Down arrow indicates significant decrease, up arrow indicates significant increase compared to healthy subjects.

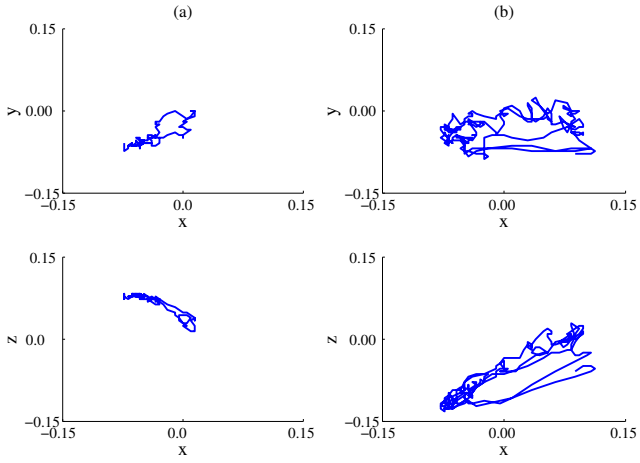


Figure 3. Vector loops in x,y and x,z planes for column (a) a healthy subject and (b) atrial fibrillation patient.

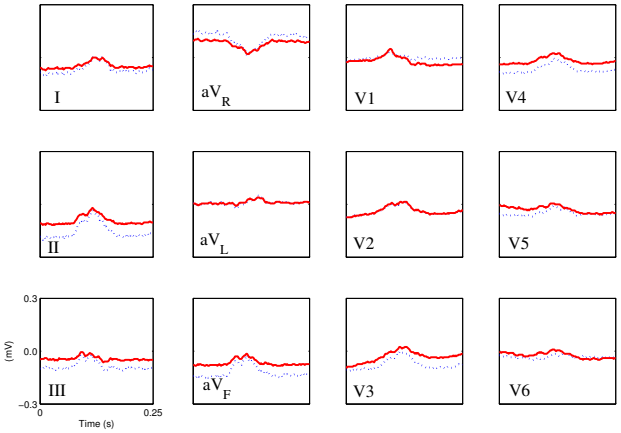


Figure 4. P waves from a healthy subject (solid lines) and simulated P waves using the single dipole model (dotted line).

## 4. Discussion and conclusions

The spatial distribution of amplitudes across the 12-lead ECG shows large differences in healthy and diseased groups. In atrial fibrillation lead V1 has been shown to have the largest amplitude atrial signal which confirms our observations in clinical practice. We have also shown that there are significant decreases in atrial signal amplitude in limb leads I, II and $aV_R$ and precordial leads V5 and V6.

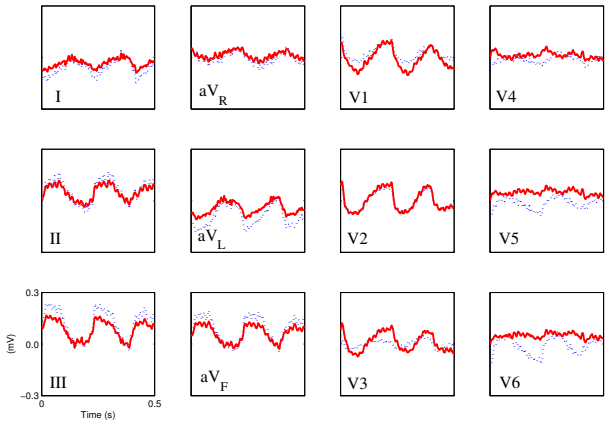


Figure 5. Atrial waves from a patient with atrial fibrillation (solid lines) and simulated waves using the single dipole model (dotted line).

There are many things which can influence lead amplitude, including body size and electrode positioning. Patients with atrial fibrillation often have enlarged atria which could lead to increased electrical amplitude on the body surface. It is likely that the most dominant effect seen by our analysis is the different orientations of propagation across the atria in the healthy subjects and atrial fibrillation patients. Figure 3 which shows the vector loops for a healthy subject and a patient with atrial fibrillation illustrates the different orientations of the atrial propagation. In the healthy subject the vector loop was positive in x, y and negative in x,z indicating propagation was downward and outward from the subject's right side to left side. For the patient with atrial fibrillation propagation was more disperse, but overall showed a strong positive x,z relationship indicating upward propagation.

The single dipole model simulation of P waves in the healthy subject showed good agreement with the 12-lead ECG. For the simulation of the atrial signal in atrial fibrillation the model showed good agreement for all leads except leads V5 and V6. Further work is needed to establish the sensitivity of the model to variations in dipole location and to different torso geometries. From the model the potentials over the full torso surface are calculated and the model may be useful for assessing the optimum recording sites for analysis of the atrial signal. With the incorporation of a further dipole source to model the ventricular activity, the model will be able to simulate the full cardiac cycle and would be useful for testing algorithms for extracting the atrial signal from the 12-lead ECG.

## Acknowledgements

P Langley is supported by a grant from the UK Engineering and Physical Sciences Research Council. Thanks to Susan King for data collection.

## References

[1] Fuster V, Rydén LE, Asinger RW, Cannom DS, Crijns HJ, Frye RL, et al. ACC/AHA/ESC guidelines for the management of patients with atrial fibrillation: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines and Policy Conferences (Committee to Develop Guidelines for the Management of Patients with Atrial Fibrillation). Eur Heart J 2001;22:1852-923.

[2] Gorenek B, Birdane A, Kudaiberdieva G, Goktekin O, Cavusoglu Y, Unalir A, et al. P wave amplitude and duration may predict immediate recurrence of atrial fibrillation after internal cardioversion. Ann Noninvasive Electocardiol 2003;8:215-8.

[3] Yamane T, Shah DC, Peng JT, Jais P, Hocini M, Deisenhofer I, et al. Morphological characteristics of P waves during selective pulmonary vein pacing. J Am Coll Cardiol 2001;38:1505-10.

[4] Rajawat YS, Gerstenfeld EP, Patel VV, Dixit S, Callans DJ, Marchlinski FE. ECG criteria for localizing the pulmonary vein origin of spontaneous atrial premature complexes: validation using intracardiac recordings. Pacing Clin Electrophysiol 2004; 27: 182-8.

Address for correspondence

Philip Langley
Medical Physics Dept.
Freeman Hospital
Newcastle upon Tyne
NE7 7DN, UK
philip.langley@ncl.ac.uk