Non-invasive assessment of magnitude and dispersion of atrial cycle length during chronic atrial fibrillation in man

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Aims Atrial fibrillation cycle lengths can be assessed from right precordial ECG leads and the unipolar oesophageal ECG using a non-invasive method called Frequency Analysis of Fibrillatory ECG. The purpose of this report is to present the results from application of this method in a large group of patients with long-term atrial fibrillation and to examine the differences between patients with 'coarse' and 'fine' atrial fibrillation.

Methods and Results Simultaneous 15 min recordings from V1, V2 and an oesophageal lead at a position behind the posterior atrium were obtained in 28 patients, aged 41 to 78 years, with long-term (>1 month) atrial fibrillation. In each lead, using the time averaging technique, the QRST complexes were suppressed. Thereafter, the frequency distribution of the residual ECG was estimated by means of Fast Fourier Transform. In the 3–12 Hz range of each lead, the dominant atrial cycle length, the power maximum and the spectral width were calculated.

In 26 patients (93%), frequency spectra in the 3–12 Hz range could be obtained. The dominant atrial cycle length ranged from 120 to 175 ms, mean 150 ± 16 (SD) ms in V1, and from 120 to 190 ms, mean 150 ± 16 in an oesophageal lead (ns). The absolute difference in the dominant atrial cycle length between V1 and the oesophageal lead was 10.4 ± 7.7 ms. There was no significant difference in the dominant atrial cycle length in V1 between patients with coarse and fine atrial fibrillation. The power maximum in V1 was significantly greater in patients with coarse compared to fine atrial fibrillation (P=0.01). The spectral widths ranged from 10 to 55 ms and demonstrated significantly higher mean values in lead V2 compared to V1 (P=0.001). Compared to V1, the mean values tended to be smaller in the oesophageal lead (P=0.05).

Conclusions Using the Frequency Analysis of Fibrillatory ECG method, the dominant atrial cycle length, power maximum and spectral width can be estimated from the frequency spectra in the majority of patients with atrial fibrillation. Spatial dispersion of the dominant atrial cycle length occurs in some patients and may be an important proarrhythmic marker. The distinction between coarse and fine atrial fibrillation cannot be used as a marker of the atrial cycle length.

Key Words: Atrial cycle length, atrial fibrillation, oesophageal ECG, non-invasive, precordial leads, spectral analysis.

Introduction

Atrial fibrillation is diagnosed from the surface ECG and is characterized by irregular RR intervals, the absence of P waves and the occurrence of fibrillatory activity (f-waves) throughout the cardiac cycle. The fibrillatory activity has been divided into ‘coarse’ and ‘fine’ based upon the amplitude of the f-waves in lead V1[1–3], but otherwise information about the fibrillation extracted from the surface ECG has been limited[4–6]. The classification according to the coarseness of the fibrillatory waves has been of limited clinical value[7] and the electrophysiological differences have not been explained.

Assessments of atrial cycle lengths and the dispersion of this parameter in different parts of the atria have solely been performed from intra-operative or invasive endocardial ECG recordings[8–18]. In some of these studies, the local atrial fibrillation cycle length has been found to correlate to the local atrial refactoriness[8,12,15].


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Based on the hypothesis that f-waves contain information about atrial cycle lengths, we recently introduced a non-invasive method. Time averaging and spectral analysis were used to assess the atrial fibrillation cycle length from the surface ECG and the oesophageal ECG\(^\text{[19]}\). The method is called Frequency Analysis of Fibrillatory ECG. We showed\(^\text{[19]}\) that in patients with paroxysmal atrial fibrillation, surface lead V\(_1\) during induced atrial fibrillation reflects right atrial free wall activity, whereas the f-waves obtained from an oesophageal lead reflect both left and right posterior atrial activity.

This paper presents the results of applying the Frequency Analysis of Fibrillatory ECG method in surface leads V\(_1\)/V\(_2\) and unipolar oesophageal ECGs in a large group of patients with chronic atrial fibrillation and assesses intra-individual dispersion of the atrial cycle lengths. A further goal was to examine differences between patients with coarse and those with fine atrial fibrillation and finally to discuss clinical implications of this method.

**Methods**

**Study population**

The 28 study patients (19 males and 10 females) ranged in age from 41 to 78 years (mean 67). They were referred to our department for ambulant direct current conversion of long-term atrial fibrillation. The duration of atrial fibrillation since the last successful direct current conversion or last documented ECG with sinus rhythm was between 1 and 6 months in 12 patients, between 6 and 12 months in nine patients, exceeded 12 months in six patients and was uncertain in one patient. Sixteen patients had not previously undergone direct current conversion. The mean number of earlier direct current conversions was 1·4 (range 0–13). Six patients were not on antiarrhythmic medication at the time of study. Twenty-two of the patients were treated with antiarrhythmic drugs: digoxin eight; sotalol five; verapamil one; digoxin+sotalol two; digoxin+verapamil four and digoxin+a beta-blocking agent two. Each patient underwent clinical evaluation, exercise testing, two-dimensional transthoracic echocardiography and thyroid hormone and serum potassium analysis.

**Initial electrocardiographic recordings**

In each patient, a standard 12-lead surface ECG was obtained (frequency response 0·05–150 Hz). Heart rate and the presence of a bundle branch block were noted. Based on the amplitude of the fibrillatory activity in lead V\(_1\), atrial fibrillation was classified as coarse (f-waves $\geq 0·1$ mV) or fine (<0·1 mV)\(^\text{[3,4]}\). Coarse atrial fibrillation was present if any fibrillatory wave in the ECG tracing exceeded or was equal to 0·1 mV.

For the oesophageal ECG recording, a bipolar Medtronic oesophageal electrode 6992A (interspacing 15 mm) was introduced transnasally and passed to a position behind the left ventricle. Bipolar recordings and approximate unipolar oesophageal ECG were obtained by connecting the electrode poles to the extremity leads for the right and left arm on the ECG recorder. The oesophageal electrode was carefully withdrawn until the atrial fibrillatory activity on the bipolar electrogram was a maximum amplitude and the ventricular electrograms of decreasing amplitude (Fig. 1). At this position, the electrode poles were considered to be the closest to the atria. Thereafter, each electrode pole, proximal and distal, was connected in unipolar fashion, with the Wilson Central Terminal as a reference, and data acquisition was performed as described below.

**Data acquisition and analysis**

The ECGs (V\(_1\), V\(_2\), proximal and distal oesophageal recordings) were digitized with a sampling rate of 1 kHz and an amplitude resolution of 0·6 $\mu$V using a 16 bit A/D conversion (equipment supplied by Siemens-Elema AB, Sweden). Each recording was of 15 min duration. The Frequency Analysis of Fibrillatory ECG method first suppresses the QRST complex in the ECG lead and thereafter estimates the frequency spectrum of the residual ECG using Fast Fourier Transform (Fig. 2). The Frequency Analysis of Fibrillatory ECG method is described in detail elsewhere\(^\text{[19]}\).

In each of the four leads four measures were derived (Fig. 2):

1. The distribution was in the 3–12 Hz range, and named unimodal if one frequency component was present and multimodal if two or more frequency components were present. The peak magnitude had to be a local maximum and be at least 50% of the maximum amplitude in the 3–12 Hz interval to be regarded as a frequency component.

2. The peak frequency of the spectral component or components was converted to a cycle length and named ‘dominant atrial cycle length’. The dominant atrial cycle lengths were rounded off to the nearest multiple of 5 ms.

3. The leads V\(_1\), V\(_2\), proximal and distal were in the amplitudes of the 3–12 Hz components in the power spectra (the so-called ‘power maxima’). The unit for the power maximum is $\mu$V$^2$. Hz$^{-1}$.

4. The width of the curve of the frequency component in the 3–12 Hz domain (the so-called ‘spectral width’) was defined as the difference in cycle lengths at the 75% level of amplitude interceptions. The spectral width was not calculated for multimodal frequency distributions. The minimal proximal and distal values were chosen (ESOmin) to compare the spectral widths of the oesophageal leads with those of the precordial leads.

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Statistical analysis

Dominant atrial cycle length values and the spectral width for each of the recordings (V₁, V₂, distal and proximal oesophageal recordings) are expressed as mean values ± standard deviations. For comparison between different leads, a paired t-test was used. A value of \( P < 0.05 \) was considered significant.

Results

Four patients had ischaemic heart disease, four valvular disease (aortic stenosis two; severe mitral insufficiency one; mitral valve prolapse one), three patients hypertensive heart disease, three patients dilated cardiomyopathy and 14 patients had no known heart disease. The mean echocardiographic left atrial dimension was 46 mm (range 38–55 mm). Left ventricular function was normal in 20 patients, moderately depressed in six and severely depressed in two patients. All patients had thyroid hormone and potassium levels within normal limits.

In 26 out of 28 patients (93%) frequency spectra in the 3–12 Hz domain could be obtained. In two patients, the method failed to detect a maximum in the 3–12 Hz range. One of these patients had permanent left bundle branch block. In the other patient the recording quality was poor, i.e. low signal to noise ratio. The 26 patients are the subject of the following presentation. Nineteen of the patients had coarse and seven patients fine atrial fibrillation. Frequency spectra with the identifiable 3–12 Hz component could be obtained in all patients from V₁, in 21 out of 26 patients from V₂ and in all patients from the oesophagus. In three patients, the proximal oesophageal recording could not be obtained. Two patients had multimodal spectra, one patient in lead V₁ and the other patient in lead V₂. The following results are summarized in Table 1.

The dominant atrial cycle length

The dominant atrial cycle length in lead V₁ was 154 ± 16 ms (mean ± SD), range 120–175 ms; in V₂ it was 154 ± 17 ms (ns), range 115–175 ms (Fig. 3(a)). In the proximal and distal oesophageal leads, the dominant atrial cycle length was 150 ± 16 ms, range 120–185 ms, and 152–19 ms (ns), range 120–190 ms (Fig. 3(b)) respectively. In five patients there were differences in the dominant atrial cycle length between the proximal and distal oesophageal recordings: 15, 10, 10, 5 and 5 ms, respectively. Otherwise the dominant atrial cycle lengths were identical. The absolute difference in dominant atrial cycle length between V₁ and distal oesophageal
The recording was 10.4 ± 7.7 ms (mean ± SD), range 0–30 ms. This did not correlate to the left atrial diameter (not shown).

There was no significant difference between the dominant atrial cycle length in V₁ in patients with coarse and those with fine atrial fibrillation. There was no correlation between the dominant atrial cycle length in the oesophageal recordings and the left atrial dimensions obtained at echocardiography (not shown).

**Power maximum in the 3–12 Hz interval**

The mean values with standard deviations of the power maxima (in $10^4 \times \mu V^2/Hz$) were 6.5 (± 5.4) for lead V₁,

Table 1  *The dominant atrial cycle length (DACL), power maximum and spectral width*

<table>
<thead>
<tr>
<th></th>
<th>DACL (ms)</th>
<th>Power maximum ($10^4 \mu V^2 \times Hz^{-1}$)</th>
<th>Spectral width (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>V₁</td>
<td>154 ± 16</td>
<td>6.5 ± 5.4 (1.1–26)</td>
<td>24 ± 9 (10–50)</td>
</tr>
<tr>
<td>V₂</td>
<td>154 ± 17</td>
<td>5.5 ± 3.2 (1.3–12)</td>
<td>35 ± 13 (15–55)</td>
</tr>
<tr>
<td>ESOprox</td>
<td>150 ± 16</td>
<td>21.0 ± 15.2 (1.8–55)</td>
<td>20 ± 8 (10–35)</td>
</tr>
<tr>
<td>ESOdist</td>
<td>152 ± 19</td>
<td>25.6 ± 21.4 (3.8–78)</td>
<td>22 ± 12 (10–50)</td>
</tr>
<tr>
<td>ESOmin</td>
<td>—</td>
<td>—</td>
<td>19 ± 9 (10–30)</td>
</tr>
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Mean values and standard deviations, ranges in parentheses.

ESOprox and ESOdist = proximal and distal oesophageal recording; ESOmin = minimum values of ESOprox and ESOdist.
5.5 (± 3.2) for lead V₂, 21.0 (± 15.2) for proximal and 25.6 (± 21.4) for distal oesophageal recording. The power maxima of the oesophageal leads were significantly higher compared to leads V₁ and V₂ (P<0.0001 for all comparisons). No significant difference was found between V₁ and V₂. In lead V₁, the power maximum in patients with coarse atrial fibrillation was 8.1 ± 5.5 μV² × Hz⁻¹ (mean ± SD) and in patients with fine atrial fibrillation 2.2 ± 1.0 μV² × Hz. The difference is significant (P=0.01).

**Spectral width**

The spectral width in lead V₁ was 24 ± 9 ms (mean ± SD), range 10–50 ms; in V₂ the spectral width

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**Figure 3** Dominating atrial cycle lengths (DACL) from leads V₁, V₂ and an oesophageal lead. The ongoing antiarrhythmic drug treatment is depicted. (a) V₁ vs V₂ (ns). (b) V₁ vs ESOdist (ns). ESOdist = the distal oesophageal recording.
was 35 ± 13 ms, range 15–55 ms. In the oesophageal leads, the proximal spectral width was 20 ± 8 ms, range 10–35 ms, and the distal 22 ± 12 ms, range 10–50 ms. The spectral widths in V₁, V₂ and ESOmin are compared in Fig. 4(a) and (b). There was a tendency towards smaller spectral width values in oesophageal compared to V₁ ($P<0.05$) and a significantly larger spectral width in lead V₂ compared to V₁ and ESOmin, $P<0.001$.
Discussion

Our results show that the dominant atrial cycle lengths can be estimated using the Frequency of Analysis of Fibrillatory ECG method in the majority of patients with chronic atrial fibrillation. We have presented the range of dominant atrial cycle lengths in Frequency of Analysis of Fibrillatory ECG from V1, V2 and the oesophagus, and can detect no systematic differences between the leads although intra-individual differences were seen. There were no significant differences in dominant atrial cycle lengths when patients with coarse and fine atrial fibrillations were compared. Further, we have presented two other non-invasive parameters, the spectral width and the power maximum in the 3–12 Hz range, and shown that these parameters vary according to the site of recording.

**The dominant atrial cycle length**

In the initial study from our institution, we found a close correlation between the dominant atrial cycle length and the mean of the manually measured atrial cycle lengths of the ECG recordings[19]. Further, we showed close agreement between the dominant atrial cycle length from the Frequency of Analysis of Fibrillatory ECG in lead V1 and the spatial mean of intracardiac recordings from the right atrium. However, the agreement was less convincing when the oesophageal and left atrial ECGs were compared, assuming that the coronary sinus is representative of the anatomically corresponding parts of the left atrium. It was concluded, also based on anatomical considerations from magnetic resonance imaging scanning, that the unipolar oesophageal atrial ECGs probably receive contributions from the posterior right and left atrium as well as the inter-atrial septum. We therefore find it reasonable that the dominant atrial cycle lengths from leads V1/V2 and the oesophagus in this study reflect the spatial mean atrial cycle lengths in different parts of the right and left atria.

Few studies have attempted to estimate the atrial fibrillation cycle length from the surface ECG[5-6]. In these studies, the primary purpose was to develop a computer-based algorithm to discriminate between atrial fibrillation and other non-atrial fibrillation rhythms[5]. The authors noted a median frequency in the power spectrum of lead V1 in patients with induced atrial fibrillation corresponding to cycle lengths 179 ± 91 ms (mean ± SD)[6].

Several groups have studied local atrial cycle lengths invasively during atrial fibrillation. The studied arrhythmia was pacing-induced atrial fibrillation[8–10,12,14,15,18,20–22] or chronic atrial fibrillation[13,17]. Both animal[8,15,21,22] and human studies[9,10,12,14,17,18,20] have been performed either with endocardial catheters[9,12,14,20] or with electrode arrays placed intra-operatively on the epicardium[8,10,13,17,18,21,22].

In one study, 16 patients with chronic atrial fibrillation were investigated intra-operatively by means of electrode arrays in the right atrium[17]. A spectrum of activation patterns was seen from the disorganised to the more organised. Median atrial cycle lengths were between 146 and 165 ms, with local cycle lengths ranging from 110 to 281 ms. In another human study, pacing-induced atrial fibrillation was investigated intra-operatively in 25 patients with the Wolff–Parkinson–White syndrome[11]. The median cycle lengths varied between 136 and 174 ms and were related to the type (I, II, III) of atrial fibrillation, i.e. the degree of disorganisation reflected by the number of recorded wavelets. The cycle lengths were significantly shorter in patients with three or more wavelets. No significant difference was seen between the right and left atrium.

Catheter-based measurements of local atrial cycle lengths were performed in a study in patients with paroxysmal atrial fibrillation[12]. Mean atrial cycle lengths decreased in both atria over time in patients with long episodes of atrial fibrillation, from 182 to 161 ms in the low right atrium. The investigators found a significant correlation between mean atrial cycle length and the functional refractory period.

Due to the possible existence of excitable gaps during atrial fibrillation, it has been questioned whether the atrial cycle lengths actually reflect the refractoriness of the relevant part of the atrium[23]. The dominant atrial cycle length values in our study may therefore correlate well to the mean atrial cycle length, but this parameter should not be considered synonymous with functional refractoriness.

We could not show any significant difference between the dominant atrial cycle length from lead V1 and from the oesophagus when comparing mean values, although intra-individual differences were seen. This is in agreement with human studies[12] and differs from animal studies, which have shown a shorter refractory period in the left atrium compared to the right atrium[24]. The differences between the dominant atrial cycle length in V1 and the oesophageal recording seen in some of our patients may, however, reflect a spatial dispersion in refractoriness in different parts of the atria, which may be a proarrhythmic factor[10,25]. As we found a considerable variability in the dominant atrial cycle length in this study group, it remains to be verified if patients with low dominant atrial cycle length values have a greater tendency to perpetuation of atrial fibrillation, i.e. whether the dominant atrial cycle length may serve as a proarrhythmic marker.

Coarse and fine atrial fibrillation — relation to dominant atrial cycle length and power maximum

In our material, there were no significant differences between dominant atrial cycle length values in patients with coarse and those with fine atrial fibrillation. One would expect larger dominant atrial cycle length values in coarse compared to fine atrial fibrillation provided
Atrial cycle length in atrial fibrillation

The width of the frequency spectrum showed significant differences between coarse and fine atrial fibrillation have been investigated by several authors, but the information concerning the electrophysiology is limited[1,2,4,26]. In one study, invasive electrophysiological recordings were performed in patients with coarse atrial fibrillation or fibrillatory flutter[4]. No patient with fine atrial fibrillation was reported. Recordings were made bi-atrially from the right atrial endocardium and from the left atrium by means of a coronary sinus catheter or an oesophageal electrode. During coarse fibrillation, atrial cycle lengths shorter than 140 ms were seen with periods of more regular activity. However, these periods did not always coincide with the coarseness in the ECG. The definitions of coarse and fine atrial fibrillation are arbitrary and in patients categorised as having coarse atrial fibrillation the coarseness can interchange with periods of fine fibrillatory wavelets. Further, the amplitude of the fibrillatory waves depends theoretically on several factors (atrial myocardial tissue mass, impedance between the recording electrode and the atria and directionality and cancellation of wave fronts). The lack of correlation between type of atrial fibrillation and dominant atrial cycle length is therefore not surprising.

The power maximum in the 3–12 Hz range was significantly larger in the oesophageal lead compared to leads V1 and V2. These differences in power maximum reflect another measurement of the same phenomenon, i.e. the amplitude of the fibrillatory waves, and do not add information per se to the pathophysiology.

The spectral width

The width of the frequency spectrum showed significant variations in the different recordings. Whereas the dominant atrial cycle length correlates to the atrial cycle lengths, as documented by our earlier studies, we have not investigated factors which may influence the spectral width in detail and care should be taken in the interpretations. Similar variations in cycle lengths with similar curve morphologies are seen in invasive local atrial recordings[10,11,15]. In these recordings, the long cycle lengths can be explained by the considerable variation in local activation time due to excitable gaps, and short cycle lengths can be due to temporal overlap of different reentry waves as well as the presence of continuously reentering impulses in localized areas. Besides these local phenomena, far field factors must also be taken into account, as we found significantly larger spectral widths in lead V2 compared to V1 and a tendency toward narrower spectra in ESOmin compared to V1. This may be explained by the more remote distance from the atria in V2 compared to V1 and V2 compared to the oesophagus. One may argue that the use of the minimum value

from the two oesophageal recordings favours a priori this hypothesis. However, in contrast to the fixed position of leads V1 and V2, the exact positions of the oesophageal electrode poles were not known.

Other factors may also contribute to the size of the spectral width. Estimation of the frequency spectrum can be achieved using different techniques, which may influence the appearance of the frequency distribution[19]. Further, it should be recognised that our method of calculating the spectral width tends to give larger values in cases of short compared to long dominant atrial cycle length values, due to the transition from frequency measure to cycle length.

More studies are needed to characterize factors which influence the spectral width. Correlations to intracardiac frequency spectra, as well as possible temporal variations and the effect of interventions on this parameter, are relevant subjects for future investigations.

Limitations

A major limitation of this study is that our recordings have been obtained during ongoing antiarrhythmic drug treatment. It was not the purpose of the study to evaluate the effect of these drugs on the dominant atrial cycle length. A prolonging effect on the atrial refractory period is well described for class I and III drugs, but even so-called frequency modulating drugs have been shown to affect the atrial cycle lengths during atrial fibrillation[27,28].

Conclusions

We have described the application of a new non-invasive method called Frequency of Analysis of Fibrillatory ECG to assess the dominant atrial fibrillation cycle length in a relatively large group of patients with long-term atrial fibrillation. The dominant atrial cycle length could be obtained in the vast majority of patients from leads V1, V2 and the oesophagus and showed wide inter-individual variability in our patient group. A spatial dispersion in dominant atrial cycle length could be shown in some of our patients and may be of proarrhythmic importance.

The distinction between coarse and fine atrial fibrillation could not be explained by differences in the dominant atrial cycle length. Finally, we have introduced two now parameters, the power maximum and the spectral width obtained by the Frequency of Analysis of Fibrillatory ECG method, and discussed their importance.

References