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Mapping and ablation of atrial tachyarrhythmias : from signal to substrate

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chapter 6

Degree of Fractionation of Atrial Fibrillation Electrograms during Acute and Chronic AF

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Abstract

Introduction: Fibrillatory conduction is associated with a different degree of fractionation of the local unipolar electrogram. The goal of the present study was to evaluate whether classification of fibrillation potentials according to their degree of fractionation and fractionation duration is useful for characterizing atrial fibrillation (AF).

Methods: Epicardial mapping of the right atrial free wall (244 unipolar electrodes, diameter 0.3 mm) was performed during cardiac surgery in patients ($n = 22$, 32 ± 11 yrs) without a history of AF (AF induced by rapid pacing, AAF) and in patients with chronic AF ($n = 15$, 66 ± 9 yrs, CAF) and valvular heart disease. Fibrillation potentials were classified according to the number of components (1-10) and total duration of each fractionated electrogram was determined by measuring the time between the first and last deflection. Fractionation fingerprints demonstrating the degree of fractionation and the relative frequency distribution of fractionation duration of all fibrillation potentials recorded within the entire mapping area during 8 seconds were constructed for each patient. Fractionation maps visualized fractionation and fractionation duration/potential at each electrode.

Results: In all patients, most fibrillation potentials were non-fractionated (AAF: $83 \pm 7\%$, CAF: $61 \pm 10\%$). Comparing AAF and CAF, the incidence of long double and fractionated potentials was higher ($8 \pm 9\%$ versus $2 \pm 5\%$, $p = 0.01$) and the fractionation duration was longer (long double potentials: 17 ± 3 ms versus 13 ± 4 ms, $p = 0.0005$, fractionated potentials: 61 ± 22 ms versus 48 ± 19 ms, $p = 0.003$). The averaged fractionation duration/potential during CAF was significantly longer than during AAF (CAF 28 ± 6 (13-36) ms/potential versus 19 ± 14 (0.62-65) ms/potential, $p = 0.01$). Fractionation was related with ageing ($r = 0.78$, $p = 0.001$), left atrial dilatation ($r = 0.68$, $p = 0.001$), a shorter excitable period ($r = 0.82$, $p = 0.001$) and conduction anisotropy ($r = 0.55$, $p = 0.001$).

Conclusion: Acute and chronic AF in the right atrium differs degree of fractionation and fractionation duration. Fractionation mapping may be useful to distinguish different types of AF and to identify the substrate of fibrillatory conduction.

Introduction

Fractionated electrograms are extracellular waveforms containing multiple, distinct deflections reflecting local asynchronous activation of myocardium surrounding the recording electrode.^{1;2} Local asynchronous activation can be the result of a spatial dispersion in refractory periods or non-uniform tissue anisotropy for instance due to fibrosis.³ Increased deposition of fibrous tissue in the atria occurs with ageing, hypertrophy and atrial dilatation.^{4;5}

High density mapping during electrically induced AF in humans has shown that there is a relation between the degree of fractionation in the right atrium and specific spatial patterns of activation.³ Double potentials were recorded along lines of conduction block whereas fractionated electrograms with 3 or more deflections were recorded at pivot points or areas of slow conduction. Variation in the degree of fractionation of electrograms in different parts of the atria has been demonstrated by several endocardial mapping studies performed in patients with AF.^{6;7}

Recently, it has been shown that ablation of areas with fractionated electrograms may eliminate AF suggesting that fractionated electrograms are indicative for atrial regions perpetuating AF.⁸ Hence, an electrogram-guided selective ablation procedure could be an alternative treatment of AF.

The aim of this study was to evaluate whether characterization of fibrillation potentials according to their degree and duration of fractionation is useful for classification of AF. For this purpose, epicardial high density mapping was performed during induced atrial fibrillation (acute AF, AAF) in patients with normal atria and during chronic AF (CAF) in patients with valvular heart disease. Differences in the degree of fractionation of right atrial fibrillation potentials recorded during AAF and CAF were analyzed.

Methods

Study Population

Recordings of AAF were obtained from patients ($n = 22$, 14 male, age 32 ± 11 yrs) who underwent open chest surgery for interruption of an accessory pathway. None of these patients had congestive heart failure, valvular or coronary artery disease. Anti-arrhythmic drugs were not used at the moment of cardiac surgery. Absence of atrial enlargement was confirmed by echocardiographic examination in all patients. Thus, AF in this patient group represented AF in non-remodelled, non-dilated atria. A more detailed description of this study population has been given previously.³

Recordings of CAF ($n = 15$, 8 male, age 66 ± 9 yrs) were acquired from patients during cardiac surgery for valvular heart disease (mitral valve disease: 13, aortic valve disease: 2). Coronary artery disease was present in 5 patients. Anti-arrhythmic drugs were continued by 7 patients (sotalol: 6, verapamil: 1). Left atrial diameter and left ventricular ejection fraction measured by echocardiography were respectively 57 ± 8 mm and $52 \pm 13\%$. AF duration ranged from one to nine years.

Mapping of Atrial Fibrillation

In all patients, epicardial mapping of the atria was performed before cannulation for cardiopulmonary bypass. If patients were in sinus rhythm at the onset of the mapping procedure, AF was induced by programmed electrical stimulation through electrodes sutured to the right atrial appendage.

A spoon shaped mapping array (244 unipolar electrodes, diameter 0.3 mm, inter-electrode distance 2.25 mm) was used for epicardial mapping of the right atrial free wall (RAFW). A silver plate positioned inside the thoracic cavity served as indifferent electrode. This mapping device was positioned in the middle of the right atrial free wall, thereby covering an area of 3.6x3.6 cm. Eight seconds of AF were analyzed, thereby excluding the first and last 12 seconds of each episode of induced AF.

Unipolar fibrillation electrograms were simultaneously recorded with a surface ECG and stored in a computer for off-line analysis after amplification (gain 1000), filtering (bandwidth 1-500 Hz), sampling (1KHz) and analogue to digital conversion (12 bits).

Atrial Fibrillation Cycle Length

The steepest negative deflection of each fibrillation potential was marked automatically. At each electrode, fibrillation intervals were assessed by measuring the time between activations by consecutive fibrillation waves. The averaged median atrial fibrillation cycle length (AFCL) was calculated from all fibrillation intervals measured during the entire recording time. All fibrillation intervals were analyzed in relation to spatial patterns of activation as described in chapter 2. The excitable period was approximated by calculating the difference between the median ACFL and the 5th percentile of the AFCL. Median

AFCL and the estimated excitable period were related with the incidence of fractionated potentials.⁹

Construction of fibrillation maps

A fibrillation map was constructed by creating time windows. A time window started at the moment that the first fibrillation wave entered the mapping area. The width of the window enclosed the time period during which all electrodes were once activated. When successive time windows were not separated by electrically silent periods, overlapping time windows were constructed. In this case, a new time window was created each time when the next entering fibrillation wave activated one of the electrodes for a second time.

Anisotropic Conduction

Local conduction velocity vectors were constructed in areas of 3x3 electrodes (4.5 x 4.5 mm). An ellipse was fitted through all local conduction velocity vectors obtained during 8 seconds of AF. Conduction in perpendicular directions with the highest and lowest velocity represents respectively the 'longitudinal' and 'transverse' conduction velocity. The ratio between the 'longitudinal' and 'transverse' conduction velocity were used to assess the degree of anisotropic conduction.¹⁰

Characteristics of Fractionation

All fibrillation electrograms were classified according to their degree of fractionation, ranging from 2 to 10 or more deflections. Examples of unipolar fibrillation electrograms with different degrees of fractionation are shown in Figure 1. The degree of fractionation was determined by counting the components of each fibrillation electrogram.

In case of a single potential, the fibrillation electrogram consisted of one rapid negative deflection, preceded by a positive deflection of varying amplitude. When fibrillation potentials contain multiple components, only components with an amplitude of at least 25% of the amplitude of the largest deflection of the fibrillation electrogram were taken into account. Electrograms consisting of 2 consecutive negative deflections were subdivided in short double potentials (time interval between the 2 components ≤ 10 ms) or long double potentials in which the 2 components were separated by more than 10 ms. This subdivision is based on the assumption that electrograms with a time interval of ≤ 10 ms between the 2 components are not indicative for conduction abnormalities.³

A relative short fibrillation interval between consecutive fibrillation potentials can be a true short fibrillation interval between two single potentials or a double potential with a long interval between the two different components. To discriminate between true short fibrillation intervals and long double potentials all electrograms were analyzed in relation to the corresponding fibrillation map. In case of two single potentials representing a true short fibrillation interval both potentials represent local depolarization and none of the potentials are far-field potentials. These two potentials are the result of activation of the

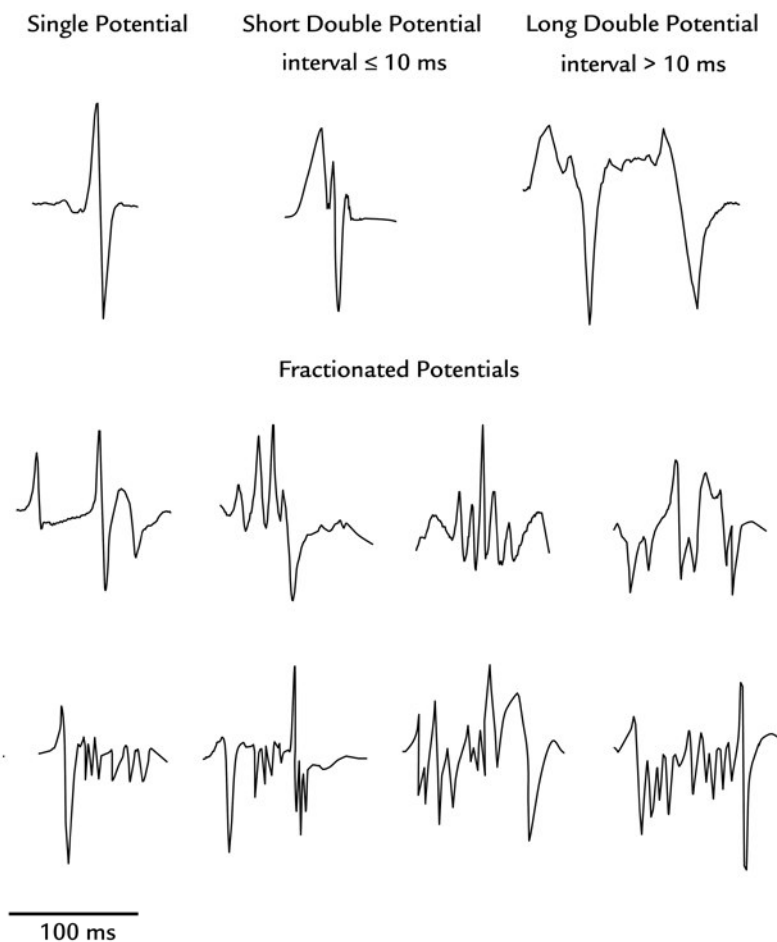


Figure 1.

Examples of unipolar fibrillation electrograms. All fibrillation electrograms were classified as single, short double potentials (interval between the 2 components ≤ 10 ms), long double potentials (interval > 10 ms) or fractionated potentials (≥ 3 deflections).

electrode twice by a passing fibrillation wave which conducted to the surrounding area. If two successive fibrillation potentials represent a long double potential, they are recorded from the border of two wavefronts separated by a line of conduction block and indicate local dissociation in conduction of fibrillation waves. Each component is the result of a fibrillation wave activating either site of the line of conduction block. A more detailed description of discriminating true short fibrillation intervals from double potentials has been given in chapter 2 of this thesis.

Fractionated potentials contain 3 or more components and represent slow conduction or local dissociation of conduction of fibrillation waves.

Series of multiple deflections bridging more than one atrial fibrillation cycle length are defined as continuous electrical activity. The electrogram in the upper part of Figure 2 shows an example of an episode of continuous electrical activity with a duration of 202 ms. Isochronal maps in the lower panel show the fibrillation waves activating the mapping area within this period. Multiple wavelets activate the mapping area asynchronously and give rise to several consecutive deflections at the electrode indicated by a grey circle. Thus, continuous electrical activity represents local asynchronous activation and not circulating electrical activity. The relative incidence of each electrogram type was determined for each patient. In case of continuous electrical activity, the duration of each episode was measured.

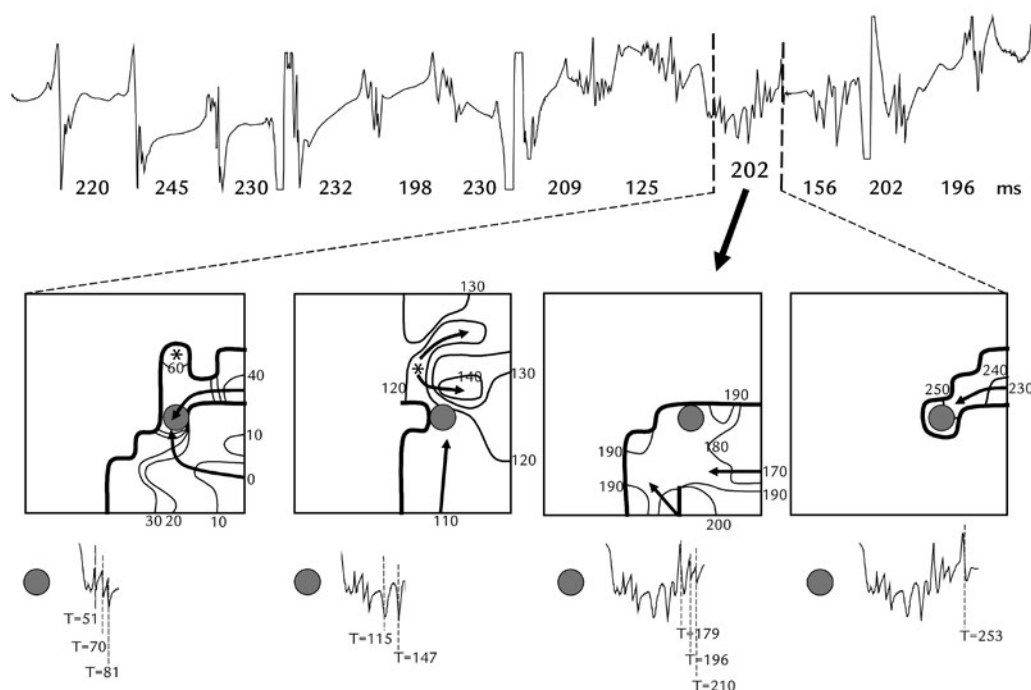


Figure 2.

Upper panel: unipolar recording with an episode of continuous electrical activity (202 ms). *Lower panel:* isochronal map demonstrating multiple fibrillation waves activating the mapping area giving rise to series consecutive deflections at the electrode indicated by the grey circle.

Fractionation Duration

Examples of measurements of fractionation duration of unipolar fractionated potentials are shown in Figure 3. Fractionation duration is determined by measuring the time between the steepest negative deflection of the first and last component. Fractionation duration is determined in order to assess the degree of asynchronous activation of myocardium surrounding the electrode.

Statistical Analysis

All data are expressed as mean $\pm$ standard deviation. Pearson's correlation coefficient (r) and (in) dependent student's T-tests were used to validate statistical significance of the data acquired. A probability level of 5% was considered statistically significant.

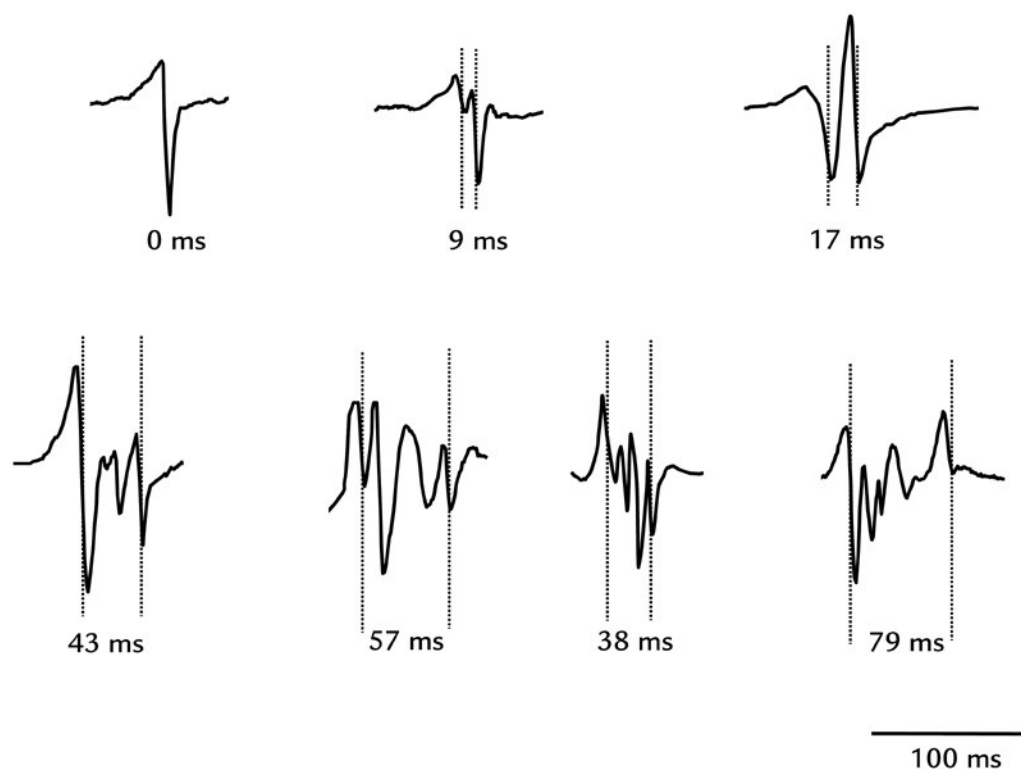


Figure 3.

Measurements of fractionation duration. Fractionation duration of a fibrillation potential is determined by measuring the interval between the steepest negative deflection of the first and last component.

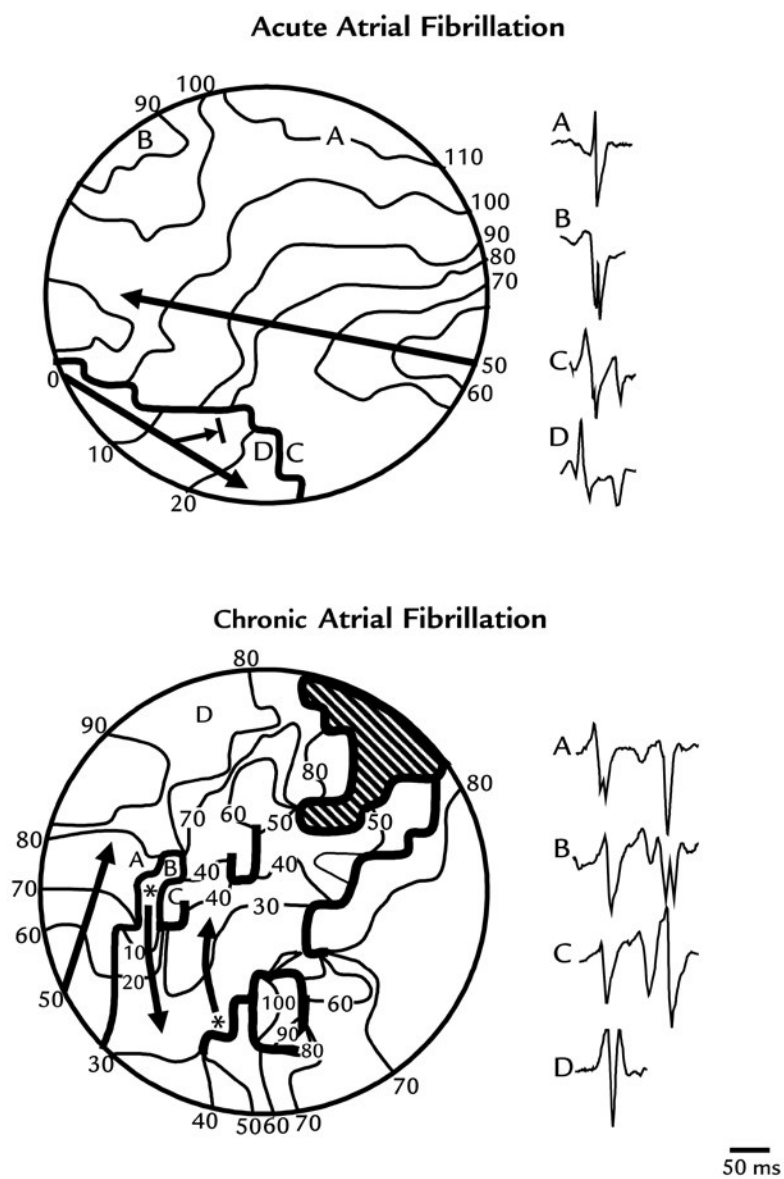


Figure 4.

Isochronal map of the right atrial free wall constructed during AAF and CAF (isochrones are drawn at 10 ms intervals, epicardial breakthroughs are indicated by an asterisk and areas of conduction block are represented by thick black lines). Fibrillation potentials recorded at different sites in the mapping area are shown outside the map.

Upper panel: two fibrillation waves enter the mapping area from opposite direction and fuse in the middle. Long double potentials are recorded along the line of conduction block. Fibrillation potentials recorded in this map are mainly single and short double potentials. *Lower panel:* multiple, simultaneously propagating wavelets encounter several areas of conduction block. Fractionated electrograms are recorded along the various lines of conduction block.

Results

Incidence of Fractionation: Acute versus Chronic AF

Figure 4 shows isochronal maps of the right atrial free wall constructed during AAF (upper panel) and CAF (lower panel). Electrograms recorded at several sites within the mapping area are shown outside the map.

During AAF, 2 fibrillation waves propagated across the mapping area. Most fibrillation potentials were either single or short double potentials (A-B). Long double potentials were recorded along the line of conduction block (C-D, thick black line). During CAF, the activation pattern was more complex due to epicardial breakthroughs (*) and the presence of several, long lines of conduction block giving rise to fractionation of electrograms. The multiple components represented activation from different sites (A-C).

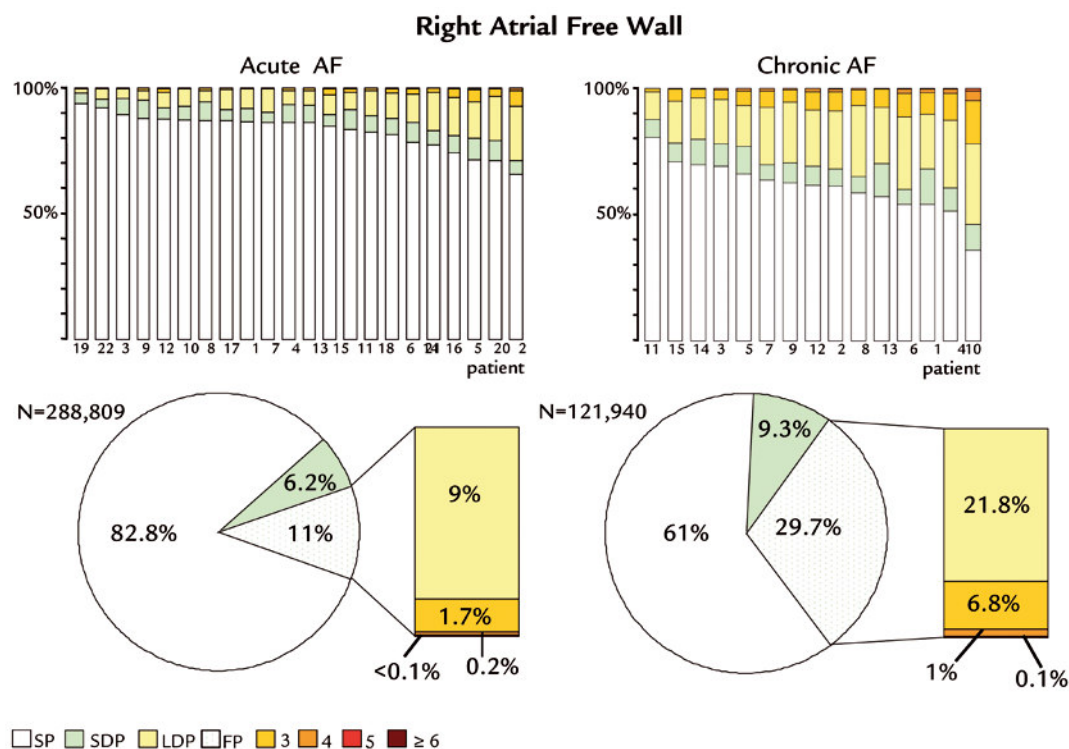


Figure 5.

Upper panel: the incidence of single, double and fractionated potentials recorded during 8 seconds of AF at the right atrial free wall for each AAF and CAF patient separately. Patients are ranked according to an increase in the degree of fractionation. During both AAF and CAF, there is an inter-individual variation in the degree of fractionation. *Lower panel:* quantification of fractionation during AAF and CAF. CAF is associated with a higher degree of fractionation. However, most fibrillation potentials are still single potentials.

SP = single potentials, SDP = short double potentials, LDP = long double potentials, FP = fractionated potentials consisting of 3, 4, 5 or ≥ 6 components.

The incidence of single, short double, long double and fractionated potentials for each AAF and CAF patient is shown in the upper panel of Figure 5. Patients are ranked according to an increase in the number of fractionated potentials. In both the AAF and CAF group, there was a considerable inter-individual variation in the degree of fractionation. During AAF, (median AFCL (156 ± 26 (122-215) ms), 288,809 unipolar fibrillation potentials recorded from the right atrial free wall of 22 AAF patients were classified. In each AAF patient, most fibrillation electrograms consisted of single potentials, varying from 66% to 94%, ($83 \pm 7\%$, Table 1). Double potentials comprised $15 \pm 6\%$ of the fibrillation potentials; long double potentials were more frequently recorded than short double potentials ($9 \pm 5\%$ versus $6 \pm 1\%$). Fractionated potentials mainly consisted of 3 components ($2 \pm 2\%$); fractionated potentials containing more than 3 components were rare (4 components: $0.20 \pm 0.29\%$, 5 components: $0.02 \pm 0.04\%$, $p = 0.01$). The highest

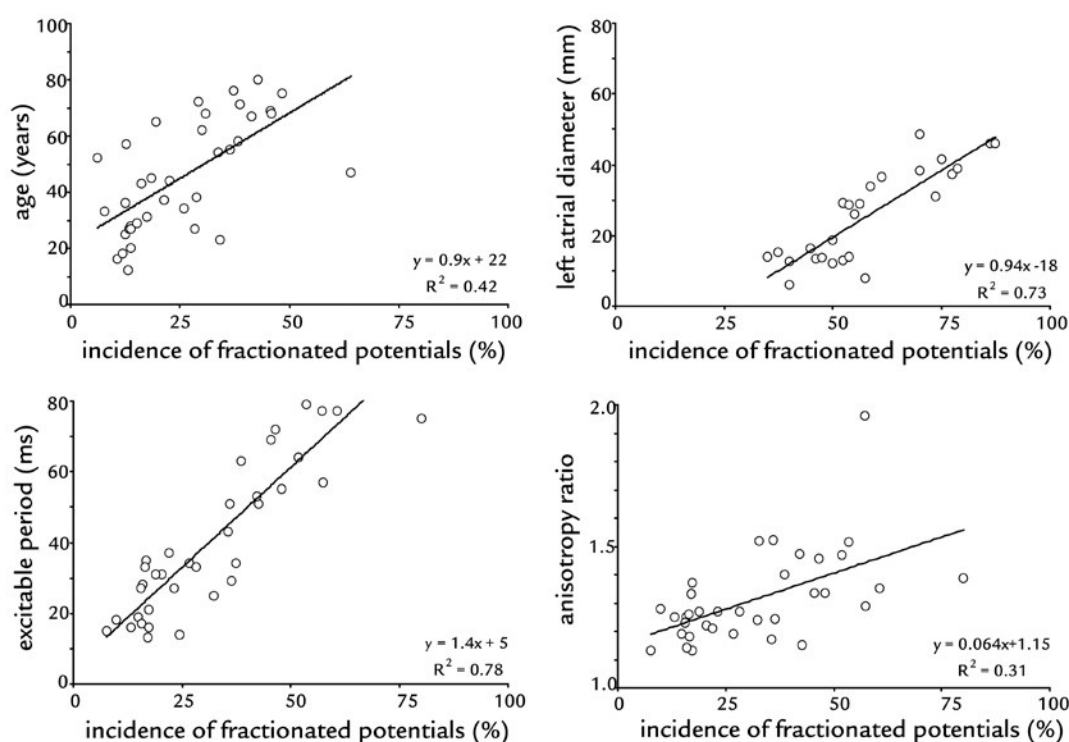


Figure 6.

A higher incidence of fractionated fibrillation potentials was related with ageing ($r = 0.78$, $p = 0.001$), left atrial dilatation ($r = 0.68$, $p = 0.001$), a shorter excitable period ($r = 0.82$, $p = 0.001$) and conduction anisotropy ($r = 0.55$, $p = 0.001$).

degree of fractionation observed during AAF was fibrillation potentials consisting of 5 deflections. During CAF, (median AFCL (190 ± 34 (137-252) ms), most fibrillation potentials ($n = 121,940$) were also single potentials (61 ± 10 (36-80)%). Only in one patient (no. 10), the majority of the fibrillation potentials were fractionated.

Similar to the AAF patients, long double potentials were more frequently recorded than short double potentials ($22 \pm 6\%$ versus $9 \pm 2\%$, $p < 0.00$). Also, the incidence of fractionated potentials was relative low (3 components: $7 \pm 4\%$, 4 components: $1 \pm 1\%$, 5 components: $0.15 \pm 0.22\%$). In two CAF patients, only one fractionated potential with 7 components was found. The lower panel summarizes the degree of fractionation of right atrial fibrillation potentials for the entire AAF and CAF group. Comparing AAF and CAF patients, there was a higher incidence of long double and fractionated potentials in the CAF patients ($8 \pm 9\%$ versus $2 \pm 5\%$, $p = 0.001$). Interestingly, continuous electrical activity was not observed during AAF and CAF. Fractionation of fibrillation potentials was associated with ageing and left atrial dilatation (Figure 6, age: $r = 0.65$, $p = 0.001$, left atrial diameter, $r = 0.86$, $p = 0.001$). There was no relation between the degree of fractionation and median AFCL ($r = 0.23$, $p = 0.17$). The estimated excitable period (P50th-P5th) in the AAF group was 28 ± 11 ms and in the CAF group 60 ± 21 ms ($p < 0.001$). There was a higher incidence of fractionated potentials in patients with a shorter excitable period ($r = 0.88$, $p = 0.001$) and a higher incidence of fractionated potentials was also associated with a higher degree of conduction anisotropy ($r = 0.55$, $p = 0.001$).

To study temporal variation in fragmentation of fibrillation potentials, recordings were subdivided into 3 episodes of 4 seconds. There was no difference in the degree of fragmentation between 3 different episodes of 4 seconds ($p = 0.85$) or between episodes of 4 and 8 seconds ($p = 0.87$).

Fractionation Duration

The cumulative frequency distribution of the fractionation duration of long double and fractionated potentials consisting of 3, 4, 5 or more components for all AAF and CAF patients is shown in Figure 7. The fractionation duration of long double potentials recorded during CAF was significantly longer than the fractionation duration of long double potentials recorded during AAF (13 ± 4 ms versus 17 ± 3 ms, $p = 0.0005$ ms). The fractionation duration of fibrillation potentials containing 3, 4 or ≥ 5 components was also longer during CAF compared to AAF (≥ 3 components: 61 ± 22 ms versus 48 ± 19 ms, $p = 0.003$). For each patient, fractionation duration of all fibrillation potentials recorded during 8 seconds were summated and divided by the number of fibrillation potentials, resulting in the averaged fractionation duration/potential (Table 1 and 2). The averaged fractionation duration/potential during CAF was significantly longer than during AAF (CAF 28 ± 6 (13-36) ms/potential versus 19 ± 14 (0.62-65.95) ms/potential, $p = 0.01$). A higher incidence of fractionated potentials is associated with a longer fractionation duration/potential ($r = 0.5$, $p < 0.001$). The degree of fractionation and the relative frequency dis-

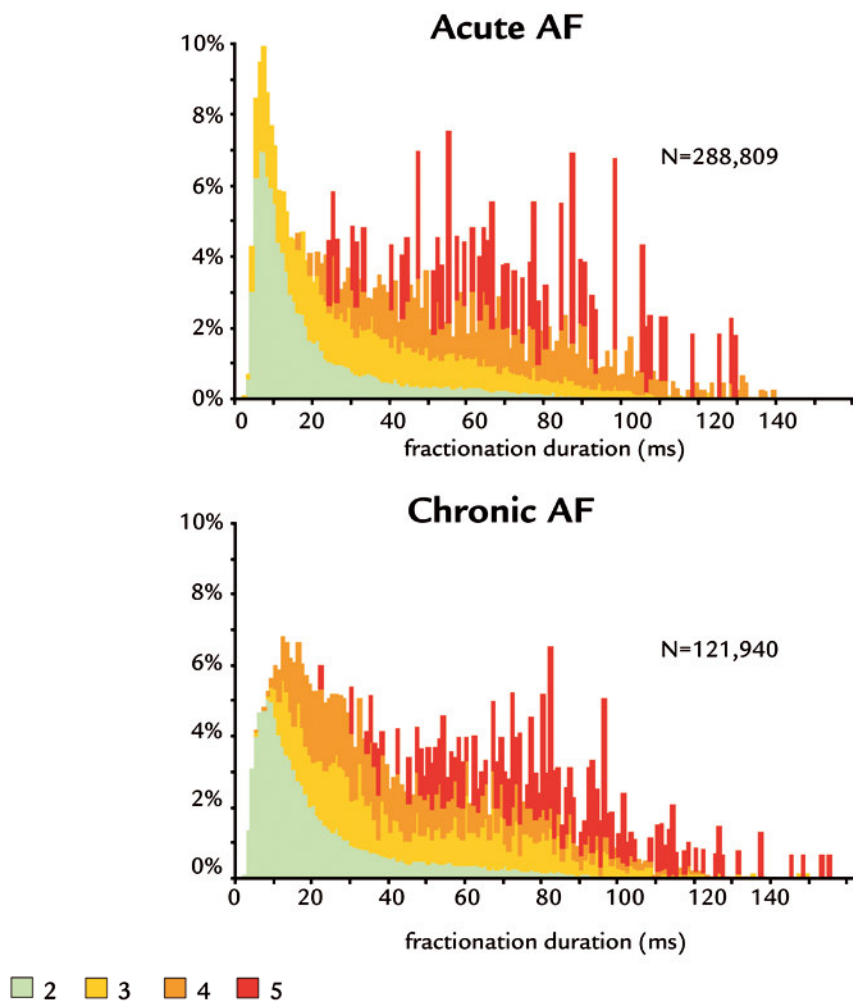


Figure 7.

Cumulative frequency distribution of local fractionation duration of long double and fractionated potentials recorded during AAF and CAF.

tribution of fractionation duration of all fibrillation potentials recorded within the entire mapping area during 8 seconds of AF are summarized in a *fractionation fingerprint* (Figure 8-10).

Fractionation Mapping

Fractionated fibrillation potentials were recorded by 80 ± 25 (28-100%) of the electrodes during CAF and by 57 ± 34 (8-94%) during AAF ($p < 0.001$). The highest incidence of both long double and fractionated potentials observed at a recording site of the mapping array during AAF ranged from 20 to 59 (38 ± 13)% and during CAF from 32 to 89 (63 ± 15)%.

Acute Atrial Fibrillation

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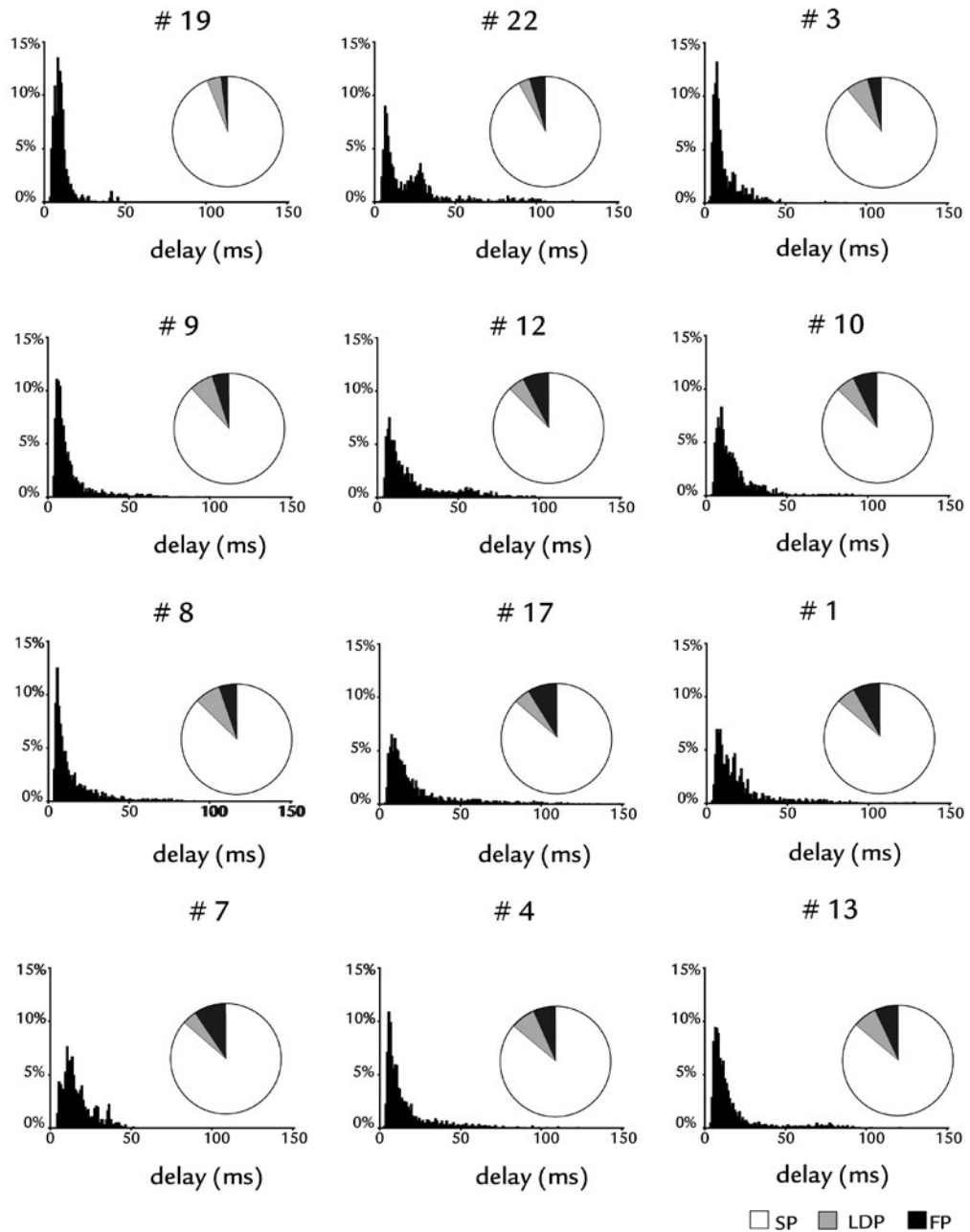
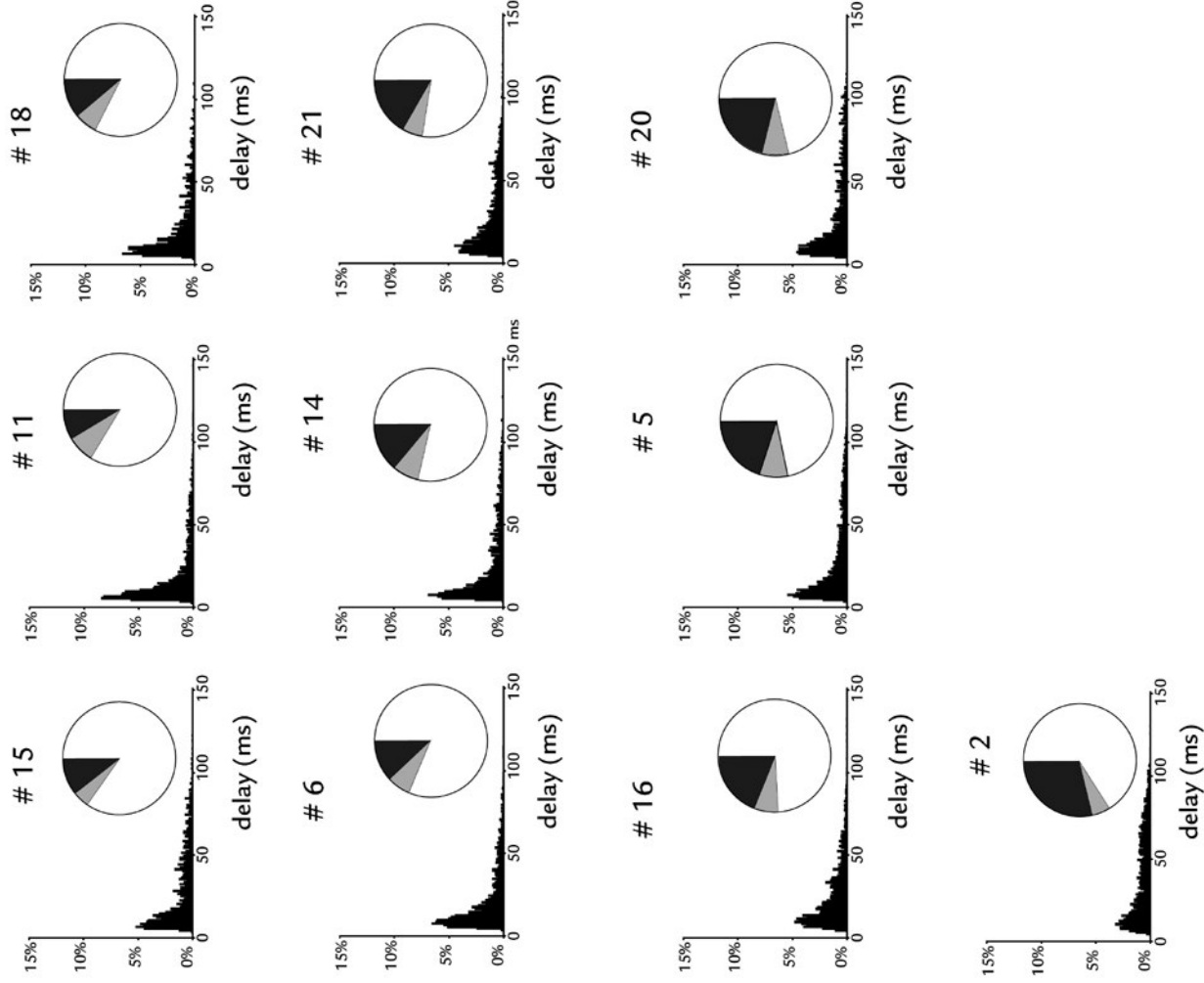


Figure 8-9.

Fractionation fingerprints of each AAF patient summarizing the incidence and fractionation duration of all fractionated potentials constructed.

SP = single potentials and short double potentials, LDP = long double potentials, FP = fractionated potentials.

Acute Atrial Fibrillation



Chronic Atrial Fibrillation

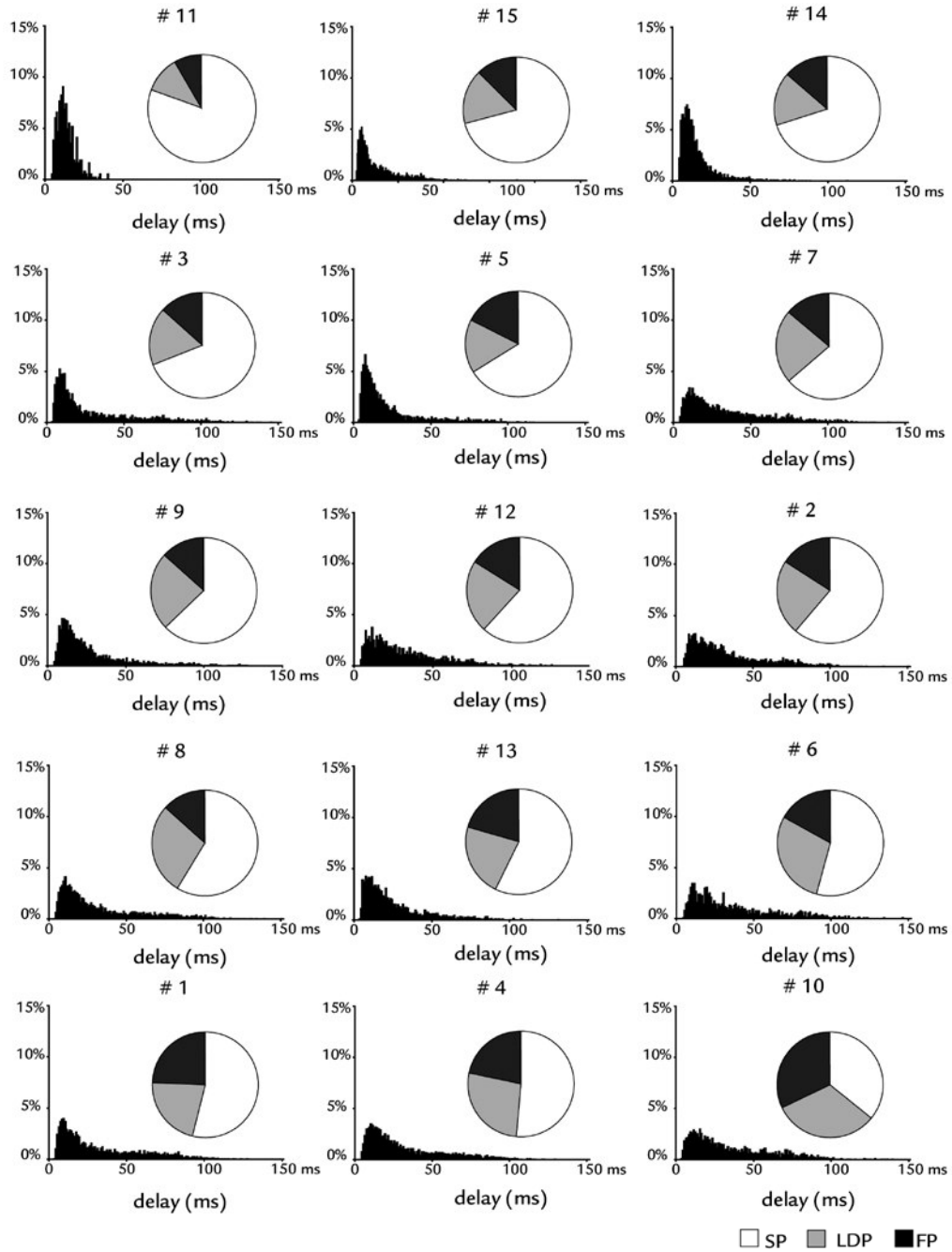


Figure 10.

Fractionation fingerprints of each CAF patient summarizing the incidence and fractionation duration of all fractionated potentials constructed.

SP = single potentials and short double potentials, LDP = long double potentials, FP = fractionated potentials.

Figure 11 and 12 show representative examples of respectively fractionation and fractionation duration/potential maps obtained from 4 AAF and CAF patients.

A fractionation map shows the incidence of long double and fractionated potentials at each electrode during 8 seconds of AF and a fractionation duration map shows the averaged fractionation duration/potential for each electrode. Fractionated potentials were recorded by a large number of electrodes throughout the mapping area though some sites had a higher degree of fractionation and a longer fractionation duration/potential than other sites. Comparing the AAF and CAF patients, there is not only a higher incidence of fractionated electrograms per electrode in the CAF patients, but fractionated electrograms with a longer fractionation duration were also recorded by a larger number of electrodes.

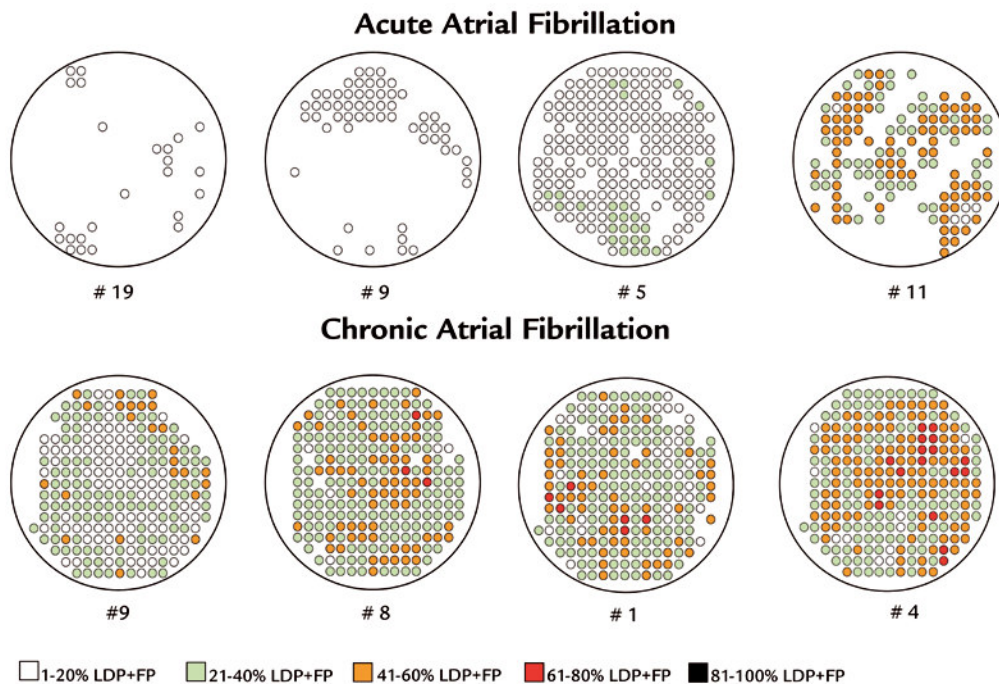


Figure 11.

Representative examples of color-coded fractionation maps obtained from 4 AAF and CAF patients demonstrating regional differences in the degree of fractionation.

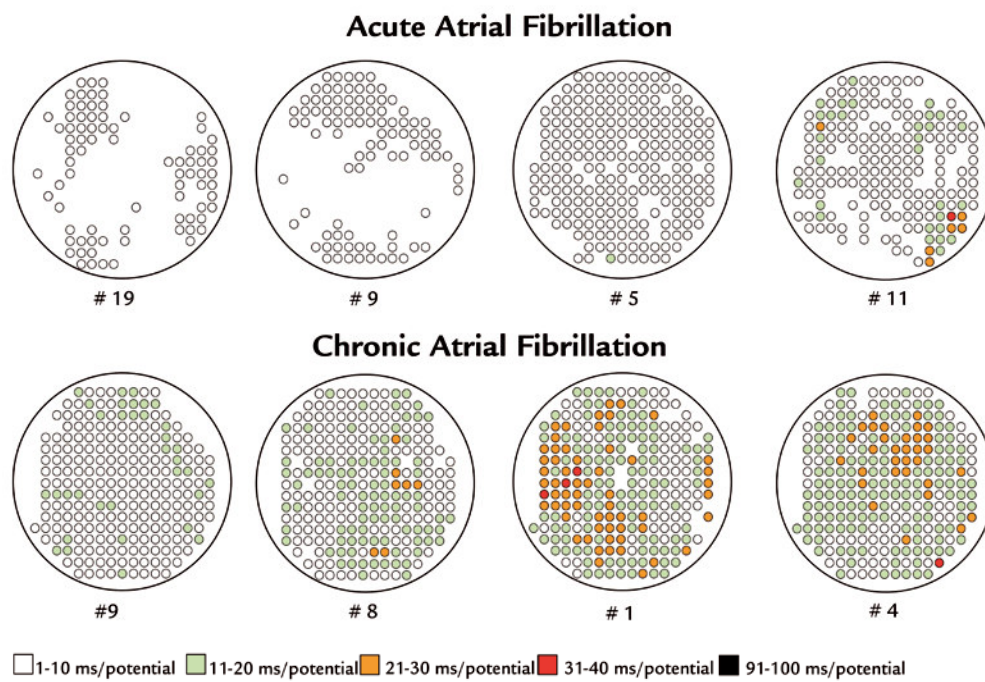


Figure 12.

Representative examples of color-coded fractionation duration maps obtained from 4 AAF and CAF patients demonstrating regional differences in fractionation duration.

Table 1. Classification of unipolar fibrillation potentials recorded from the right atrial free wall during AAF.

pt no.	S (%)	DP ≤ 10 ms	DP > 10 ms	fractionated potentials (%)			duration/ potential (ms)
				3	4	> 5	
19	93.8	4.1	1.9	0.1	0.0	0.0	0.62
22	92.1	3.3	4.3	0.3	0.0	0.0	1.76
3	89.3	6.6	3.8	0.3	0.0	0.0	12.45
9	88.1	7.1	3.9	0.8	0.1	0.0	13.21
12	87.5	4.6	6.3	1.4	0.2	0.0	22.42
10	87.3	5.4	6.8	0.4	0.0	0.0	15.36
8	87.1	7.5	4.5	0.8	0.0	0.0	15.12
17	86.8	4.5	8.0	0.7	0.0	0.0	3.05
1	86.6	5.3	7.6	0.5	0.0	0.0	20.20
7	86.2	4.2	9.3	0.2	0.0	0.0	16.71
4	86.2	7.3	5.4	1.1	0.0	0.0	15.82
13	86.1	7.0	5.9	1.0	0.1	0.0	16.60
15	84.8	4.7	7.6	2.4	0.4	0.0	29.16
11	83.6	7.8	7.0	1.4	0.2	0.0	19.77
18	82.4	6.6	9.9	1.0	0.1	0.0	17.15
6	81.4	6.7	9.7	1.9	0.2	0.0	21.39
14	78.5	7.7	11.3	2.3	0.2	0.0	5.43
21	77.4	5.8	15.0	1.7	0.1	0.0	65.95
16	74.1	7.0	15.0	3.4	0.5	0.0	23.95
5	71.5	8.5	14.4	4.8	0.6	0.1	28.96
20	71.1	7.8	17.7	3.2	0.2	0.1	9.06
2	65.8	5.4	21.6	6.3	0.9	0.1	38.29
	83 ± 7	6 ± 1	9 ± 5	2 ± 2	0.20 ± 0.29	0.02 ± 0.04	19 ± 14

S = single potentials, DP = double potentials.

3/4/5: fractionated potentials containing respectively 3, 4 or ≥ 5 deflections.

Table 2. Classification of unipolar fibrillation potentials recorded from the right atrial free wall during CAF.

pt no.	S (%)	DP ≤ 10 ms	DP > 10 ms	Fractionated Potentials (%)			duration/ potential (ms)
				3	4	> 5	
11	80.46	7.09	11.22	1.13	0.11	0.00	1.33
15	70.81	7.61	16.58	4.67	0.31	0.02	24.02
14	69.96	9.91	16.46	3.41	0.25	0.01	13.26
3	69.01	9.11	17.54	3.61	0.62	0.10	30.52
5	66.23	10.79	16.25	5.83	0.82	0.08	27.48
7	63.58	6.44	22.44	6.58	0.93	0.03	34.98
9	62.76	7.87	24.00	4.75	0.58	0.04	29.55
12	61.58	7.58	22.44	7.20	1.11	0.09	30.21
2	61.22	7.04	22.92	7.28	1.32	0.22	24.76
8	58.50	6.64	28.16	6.13	0.53	0.04	30.64
13	57.11	13.24	22.27	7.03	0.31	0.04	23.57
6	54.20	5.79	28.86	9.20	1.72	0.23	36.26
1	53.99	13.99	21.65	8.82	1.37	0.18	32.83
4	51.46	9.06	26.78	10.48	1.90	0.31	32.57
10	35.94	10.13	31.90	17.08	4.08	0.87	34.74
	61 ± 10	9 ± 2	22 ± 6	7 ± 4	1 ± 1	0.15 ± 0.22	28 ± 6

S = single potentials, DP = double potentials.

3/4/5: fractionated potentials containing respectively 3, 4 or ≥5 deflections.

Discussion

In this study, fractionation of fibrillation electrograms recorded during AAF and CAF was characterized by classifying fibrillation potentials according to the number of components and by measuring the duration of each fractionated fibrillation potential.

The key finding of this study is that despite a considerable inter-individual variation and some overlap between the AAF and CAF group, there was a higher incidence of fractionated fibrillation potentials in the CAF patients. Fractionation was associated with ageing, left atrial dilatation, conduction anisotropy and a shorter excitable period.

During AF, multiple, simultaneously circulating wavelets excite the atria. A fibrillation wave may encounter atrial tissue which is partially or totally refractory due to previous excitation by another wave.¹¹ This results in slowing of conduction, conduction block and turning of fibrillation waves around lines of conduction block. All these conduction abnormalities give rise to local asynchronous activation of the myocardium and thus to fractionation of electrograms.

There was no relation between the fibrillatory frequency and fractionation. However, the degree of fractionation was associated with the estimated excitable period (P50-P5).

A recently published study from our institution demonstrated that prolongation of AFCL by infusion of a class I drug was associated with a significant decrease in the incidence of fractionation electrograms.¹² Class I drugs widen the excitable gap by prolonging AF cycle length more than atrial refractoriness. It was postulated that widening of the excitable gap promotes uniform conduction of fibrillation waves thereby decreasing fractionation. This hypothesis is supported by the results of the present study in which we demonstrated that there was a higher incidence of fractionated potentials in patients with a shorter estimated excitable period.

The CAF group consisted of older patients with dilated atria and valvular heart disease. It can therefore be expected that fractionation of fibrillation electrograms in these patients is also influenced by anisotropic properties of atrial tissue. Non-uniform anisotropy of atrial tissue can be due to an increased deposition of fibrous tissue which occurs with ageing, valvular heart disease and atrial dilatation.^{1;2;4;13-15} The resulting electrical uncoupling gives rise to discontinuous conduction and hence to prolonged, fractionated electrograms.

Possible Clinical Applications of Fractionation Mapping

Several studies have emphasized the possible role of areas of fractionated electrograms in perpetuation of AF.^{3;6-8;16-26} So far, most studies analyzed electrograms recorded during sinus rhythm.²²⁻²⁶ Endocardial mapping of the right atrium during sinus rhythm in patients with atrial dysarrhythmias showed that subjects from whom a large number of fractionated atrial electrograms were recorded, were more susceptible to developing AF. Based on the hypothesis that areas of either structurally or functionally abnormal atrial tissue

perpetuate the fibrillatory process, identification and location of such diseased areas are crucial for local ablation therapy of AF. Nademanee et al. recently indeed showed that ablation of areas of fractionated electrograms eliminated AF.⁸

In this study, the degree of fractionation and fractionation duration of right atrial fibrillation potentials was measured with small electrodes (0.3 mm) with an inter-electrode distance of 2.25 mm. Fractionation fingerprints constructed in this way differed between AAF and CAF patients, suggesting that fractionation fingerprints can be used to distinguish different types of AF. Simultaneously recording of fibrillation potentials from multiple sites allowed assessment of the spatial variation in the degree of fractionation and fractionation duration.

Fractionation (duration) maps revealed a spatial dispersion in the incidence of fractionated fibrillation potentials and fractionation duration in all patients. So far, it is unknown whether these regional differences in fractionation and fractionation duration are due to physiological differences in atrial architecture and conduction or whether they represent pathologically altered myocardium. Electro-anatomical mapping studies are required to further develop fractionation mapping. If there is a correlation between atrial architecture (interstitial fibrosis) and fractionation, then fractionation mapping might become a useful tool to diagnose the underlying electro-pathological substrate of AF and to guide selective ablative therapies.

Study Limitations

In this study, high density mapping was limited to the right atrial free wall. As the goal of this study was to evaluate whether characterization of fibrillation potentials according to their degree and duration of fractionation is useful for classification of fractionation, we did not analyze intra-atrial variation in fractionation of fibrillation potentials. Further studies are therefore required to assess intra-atrial differences in fractionation of fibrillation potentials.

Conclusion

Fibrillation potentials differ between AAF and CAF patients with respect to the degree of fractionation and fractionation duration. Fractionation is associated with ageing, left atrial dilatation, conduction anisotropy and a shorter excitable period. Fractionation fingerprints classified fractionation by characterizing fibrillation potentials according to their degree and duration of fractionation. Fractionation and fractionation duration maps visualized regional differences in fractionation and fractionation duration. Hence, fractionation fingerprints and fractionation (duration) maps may become a useful tool to identify the electro-pathological substrate of AF and to guide selective ablative therapies.

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