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Electrophysiological abnormalities in patients with paroxysmal atrial fibrillation in the absence of overt structural heart disease

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ABSTRACT

Purpose: The aim of the present study was to define the atrial electrical substrate in patients with paroxysmal atrial fibrillation (AF) occurring in the absence of overt structural heart disease and to assess if electrophysiological parameters could predict AF recurrence after radiofrequency ablation in this population.

Methods and results: 45 consecutive patients (39 male, age 59 ± 10 years) with paroxysmal AF and without overt structural heart disease, referred for radiofrequency catheter ablation, were prospectively enrolled. A cohort of 12 age-matched patients without a history of AF, served as a control group. Atrial electrical substrate was assessed by P-wave signal-averaging, intracardiac conduction delays and refractory periods. Total P wave duration during signal-averaging was longer in patients with paroxysmal AF than in controls (140 ± 19 ms vs 123 ± 13 ms, $p = 0.004$). Patients with paroxysmal AF showed an increase in right intra-atrial (40.2 ± 11.3 ms vs 31.7 ± 11.8 ms, $p = 0.02$) and inter-atrial conduction delays (87.93 ± 22.0 ms vs 65.3 ± 15.6 ms, $p = 0.001$) in sinus rhythm. Refractory periods in the right atrium were longer in patients with paroxysmal AF (265 ± 44 ms vs 222 ± 32 ms, $p = 0.002$). After ablation, 22 patients had AF recurrence but showed no differences in electrophysiological parameters compared to patients without recurrence.

Conclusion: Electrophysiological abnormalities are present in patients with paroxysmal AF without overt structural heart disease. Neither signal-averaged P-wave duration nor intracardiac atrial electrophysiology could predict arrhythmia recurrence after pulmonary vein isolation.

1. Introduction

Atrial fibrillation (AF) is the most common sustained arrhythmia encountered in clinical practice. Although this arrhythmia is primarily seen in the elderly or in patients with heart disease, it is not exceptional to diagnose AF in patients without any overt structural heart disease or in relatively young patients [1]. This observation has lead Evans and Swann in 1954 to introduce the term "lone" atrial fibrillation, which has been used for decades by clinicians [2]. However, it has been recommended recently that the use of terms such as "idiopathic AF" or "lone AF" be avoided, because there has been a huge progress in the understanding of the pathophysiology of AF in the last 20 years; many causes of AF have been highlighted (obesity, sleep-apnea-syndrome, alcohol, vagal or adrenergic influences, excessive sporting activities, family history, genetics etc [3,4]), and abnormal electrical and anatomical substrates have been identified [5–9], including occult myocardial diseases as proven by atrial or ventricular biopsies [10,11].

In the present study we used signal-averaged P-wave analysis and intracardiac recordings to define the atrial electrical substrate of patients with paroxysmal AF occurring in the absence of overt structural heart disease and submitted to pulmonary vein isolation. We also tried to define if electrophysiological parameters could be predictive for arrhythmia recurrences after ablation in these patients.

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2. Methods

2.1. Patients

After informed consent, 45 consecutive patients (39 male, 6 female, mean age 59 ± 10 years) with paroxysmal AF referred for *de novo* radiofrequency catheter ablation (pulmonary vein isolation) were prospectively enrolled. Patients were considered eligible for catheter ablation if they had documented symptomatic AF, at least one failed antiarrhythmic drug and no history of coronary artery disease, heart failure, diabetes or pulmonary disease, with a normal physical examination, resting 12-lead ECG, transthoracic and transoesophageal echocardiogram (mild mitral regurgitation and mild atrial dilatation were not excluded). Patients were treated either with uninterrupted acenocoumarol (target INR of 2.0–3.0) or non-vitamin K oral anticoagulants (interrupted the day before the procedure). All patients underwent transoesophageal echocardiography immediately before the ablation procedure to exclude left atrial thrombi. All antiarrhythmic drugs were interrupted since at least 5 half-lives before ablation. A control group of 12 aged-matched without any history of AF, structural heart disease or hypertension and submitted to an electrophysiological study for syncope or supraventricular tachycardia was included in the study for comparison (Table 1).

2.2. Radiofrequency (RF) procedure

The procedure was performed in a fasting state under light sedation with midazolam and/or fentanyl. Only the right femoral vein was used for insertion of catheters, and in the absence of a patent foramen ovale a single transseptal puncture was performed using the Brockenbrough technique and a long sheath (SL0, St Jude Medical, St Paul, MN, USA). For the conventional point-by-point approach (n = 14), 3 catheters were inserted: one duodecapolar lasso catheter for pulmonary vein (PV) recording (introduced through the SL0 long sheath and positioned at the ostium of each PV sequentially), one 4-mm irrigated-tip ablation catheter for segmental isolation, and one decapolar (2 mm spacing) steerable catheter in the distal coronary sinus. For circular irrigated radiofrequency ablation (nMARQ, Biosense Webster, n = 31), only 2 catheters were inserted: one catheter for mapping and ablation in the left atrium (nMARQ catheter) and one decapolar (2 mm spacing) steerable catheter in the distal coronary sinus. The technique has been described in detail previously [12]. After transseptal puncture, a bolus of intravenous heparin was administered to aim for an ACT of 250–350 s and 3D electro-anatomical mapping using the CARTO3 system (Biosense Webster) was performed in all cases. With the point-by-point approach, ostial segmental isolation of all 4 PVs was performed using the ablation catheter under the guidance of the circumferential mapping catheter in the PV (maximum power 30–35 Watts; maximal temperature 48°; duration of the RF application 60 s). For circular irrigated nMARQ RF ablation, maximal power was 15 Watts and RF current was applied during 40 s at each site. The endpoint of RF application was complete PV isolation, demonstrated by the absence of PV potentials during sinus rhythm or coronary sinus pacing. Reconfirmation of PV isolation was performed 30 min after ablation for each PV. Patients were followed with continuous ECG monitoring for 24 h and were discharged from the hospital the day after the procedure. Patients were prospectively followed for up to 18 months or until recurrences occurred. Recurrences were defined as documented AF (duration > 30 s) occurring after a blanking period of 2 months after the ablation procedure. To confirm AF recurrence, serial 12-lead ECG recordings were obtained as well as at least one 24-h Holter ECG or one 7-days loop recording at 3–6 months post-procedure. Anticoagulation was maintained for 3 months in the absence of recurrence and longer in the presence of recurrences.

2.3. Intracardiac recordings

Intra-atrial conduction (right atrial conduction) was evaluated by measuring the interval between the atrial component in the high right atrium (HRA) and the atrial component recorded with the His bundle electrode(HBE) or with the proximal pole of the decapolar catheter-electrode positioned inside the coronary sinus (pCS), during sinus rhythm and during right atrial pacing (at 600 ms cycle length). Inter-atrial conduction was evaluated by measuring the interval between the atrial component in the high right atrium (HRA) and the atrial component recorded with the distal pole of the decapolar catheter-electrode positioned inside the coronary sinus (dCS), during sinus rhythm and during right atrial pacing (at 600 ms cycle length). The effective refractory period (ERP) of the right and the left atrium were determined by extra-stimulation during continuous pacing at 600 ms, at twice the diastolic threshold voltage and with a pulsewidth of 2 ms.

Table 1
Clinical and electrophysiological characteristics in the control group and in patients with paroxysmal atrial fibrillation (AF) in the absence of structural heart disease.

	Control group n = 12	Paroxysmal AF n = 45	p value
Age (years)	58.6 ± 12.3	58.8 ± 9.9	0.32
Left atrial diameter (cm)	33.8 ± 3.4	38.9 ± 4.3	0.0003*
Left atrial surface (cm^2)	15.8 ± 1.5	19.9 ± 2.8	<0.0001*
Left ventricular ejection fraction (%)	70.4 ± 4.0	67.1 ± 3.7	0.008*
P wave duration (ms)	122.8 ± 12.8	140.3 ± 19.3	0.004*
RMS 20 (μV)	3.50 ± 2.61	3.95 ± 2.64	0.59
RMS 30 (μV)	4.75 ± 3.04	4.68 ± 2.77	0.94
RMS 40 (μV)	5.25 ± 3.16	5.24 ± 2.74	0.99
P wave integral (μVs)	525 ± 161	646 ± 260	0.13
HRA-HBE (ms)	31.7 ± 11.8	40.2 ± 11.3	0.02*
HRA-pCS during sinus rhythm (ms)	54.3 ± 14.9	63.9 ± 17.4	0.08
HRA-pCS during pacing 600 ms (ms)	76.2 ± 13.8	88.9 ± 18.9	0.03*
HRA-dCS during sinus rhythm (ms)	65.3 ± 15.6	87.9 ± 22.0	0.001*
HRA-dCS during pacing 600 ms (ms)	92.8 ± 16.6	118.4 ± 22.2	0.0005*
Right atrial ERP (ms)	221.7 ± 31.9	265.0 ± 43.7	0.002*
Left atrial ERP (ms)	257.5 ± 33.9	271.8 ± 26.9	0.12

dCS = distal coronary sinus; ERP = effective refractory period; FRP = functional refractory period; HBE = atrial electrogram on the His bundle electrode; HRA = high right atrium; pCS = proximal coronary sinus; ms = milliseconds; RMS = root mean square; μV = microvolt. * = statistically significant.