Atrial Fibrillation Catheter Ablation

Dr. Yui-chi So
MRCP (UK) MRCP (Ireland) FHKCP FHKAM
Specialist in Cardiology

Introduction

In China Mainland, a cross-sectional survey of AF conducted from 2005 to 2006 including 19,368 participants (8,636 men, 10,732 women) aged >= 35 years showed that the prevalence of AF in Chinese adults was 0.73% (0.74% in men and 0.72% in women). The AF incidence was 0.43% in men and 0.44% in women with age <60 years old, and was 1.83% in men and 1.92% in women for age >= 60 years old. AF prevalence was estimated to be 0.41% around 5.3 million patients in the Mainland.

In the United States, AF prevalence was estimated to be 2.3 million. Between 1980 and 1999, AF hospitalisations increased 80% for patients aged 45-65 and doubled for patients 65 yrs or older. The ageing of the population alone is expected to raise the number of AF from 2 million in 1995 to more than 3 million by 2020 and 5.6 million by 2050.

Framingham data showed that at age 40, there is a risk of 1/4 to develop AF. 1.3% of individuals in the elderly population in Hong Kong had AF.

Paroxysmal AF

Defined as an AF episode which spontaneously terminates within 7 days.

Persistent AF

Defined as an AF episode which lasts for more than 7 days or requires cardioversion.

Permanent AF

An AF episode which fails to terminate with cardioversion or terminates and relapses within 24 hours.

Epidemiology

In China, the percentage of AF of first diagnosed were 30.9%; paroxysmal 33%, persistent 7.2% and permanent 28.9% respectively.

The recurrence rate of AF is around 49-90%. In the Stroke Prevention trial, the independent predictors of recurrence were left atrial enlargement and a history of myocardial infarction. Around 18-33% of patients will develop permanent AF. Old age and AF at presentation predicted transition to permanent AF.

However, many PAF episodes are asymptomatic. A transtelphone ECG monitor found that asymptomatic PAF was 12 x more frequent than symptomatic ones.

Aetiology

1. Idiopathic (lone AF)
2. Increased LA pressure
3. Ischaemia
4. Inflammatory
5. Age related (fibrosis and amyloid)
6. Alcohol
7. Increased sympathetic activity such as thyrotoxicosis, anxiety, exercise
8. Increased parasympathetic activity such as during sleep
9. Congenital heart disease such as ASD
10. Neurogenic such as Subarachnoid haemorrhage
11. Familial
12. Sick sinus syndrome

Foci of AF

a) Pulmonary vein(PV) ectopic beat for AF initiation:

There are myocardial sleeves in the embryonic development of pulmonary veins which give rise to abnormal automaticity. Many evidences show that there is dilation of PV ostia in patients with AF. These demonstrate that haemodynamic factors and stretch mechanisms may account for PV ectopic beats. Haissaguerre first studied the mechanism of spontaneous onset of AF was due to PV ectopic triggers. Investigators also demonstrated that PV activity may also have a role in maintaining AF too.

PV ablation

1. Focal ablation- The PV potential which initiates AF had high frequency spikes. Therefore, it is logical to directly ablate the PV potential. However, the recurrence rate is high even after ablation of the PV potential at OT site. Moreover, it will cause PV stenosis. Inconsistent inducibility, multiple and new foci of triggers are among the failure reasons for AF initiation.
2. Segmental ablation- There is an extension of left atrium muscle to the PV. Therefore, ablation at the ostium of PV using a Lasso catheter can easily identify the breakthrough sites from left atrium (LA) to PV. Pappone using the Carto system (3D mapping system) applied circumferential ablation of PV orifice. He reported that there was more than 80% successful rate.

3. Linear ablation- The left atrial wall especially the left posterior left atrium is involved in the initiation and maintenance of AF. Therefore, ablation of the left atrial wall, PV ostial atrial tissues and mitral annulus atrial tissues are necessary in treating chronic AF. The noninducibility of AF as end point for linear ablation in Bordeaux group increased the successful rate of AF ablation.

b) Other Thoracic Foci of AF
SA node derives from sinus venosus embryologically. However, there are other areas of thoracic veins which are also remnants of sinus venosus such as SVC, coronary sinus, etc.

1. Superior vena cava (SVC) - The junction between SVC and right atrium contains myocytes that has pacemaker activity. If there is enhanced automaticity this will play as a trigger for AF. Clinical study confirmed that there is a layer of myocardial tissue on the dorsal surface of SVC.

2. Coronary sinus (CS) - In animal studies, there is automatic rhythmic activity triggered by catecholamines. Clinically, we prove that there are a lot of fractionated potentials in CS.

3. Crista Terminalis - Hogan found that there are atrial fibres all along the border of crista terminalis which has spontaneous discharge. This may also account for initiation of AF.

4. Ligament of Marshall- It is the embryonic sinus venosus and left cardinal vein running between the superior and inferior left pulmonary veins. It is found that there is atrial musculature which runs in from coronary sinus. This musculature has also been found to have triggered activity.

5. Left sided posterior atrial wall- For diseased atria, the musculature is hypopolarised and can produce ectopics which may trigger AF. The mechanism may be slow depolarisation of phase 4 or delayed after depolarisation triggers after isoprenaline.

Non-PV ectopics account for around 25% of patients who have recurrences of AF. 15-25% of patients actually have non-PV triggers.

Complex Fractionated Atrial Electrograms CFAE

Another approach of ablating AF suggested by Professor Nademanee is to ablate the Complex Fractionated atrial electrograms (CFAE):

Defined: 2 deflections or more with fluctuating baseline or atrial electrograms with a very short cycle length (< 120 msec).

It was thought that by ablating the CFAE, the ganglionic plexi (GP) will be modified. Therefore, the maintenance substrate of AF was also modified too.
Post Ablation Management

We start anticoagulation after 4 hours of sheath removal. Nowadays we usually use the LMWH. Oral warfarin is also started at the same time. Anticoagulation will be stopped after 3 months treatment if there is no more AF recurrence. Anti-arrhythmic drugs are also prescribed for 1-3 months.

Breakthrough attacks of AF and atrial tachyarrhythmia are quite common within the 1st month. Therefore, we can only label success or not after at least 1 month’s time.

Complications

- Pericardial effusion 0.1%
- Stroke 0.03%
- TIA 0.2%
- Cardiac tamponade 0.1%
- Severe PV stenosis 1%
- Phrenic nerve palsy 0.5%
- Atrio-oesophageal fistula 0.05%
- Atrial flutter or tachyarrhythmia 5-10%
- Death <0.1%

Successful Rate of AF Ablation

Nowaday, we use different approaches of AF ablation and we can report the successful rate at around 80%-90% for 1 year. It also depends on the age of the patient (>70 yrs old); underlying heart disease etc.

References

1. HK-IN-PACE 10 th Annual Scientific meeting:- AF presentations by Professor Zhang Shu.
2. Effects of rapid atrial pacing on the arrhythmogenic activity of single cardiomyocytes from PV:- Chen VJ, Chen SA, Chen YC. Circulation 2001;104:2849-2854
14. AF originating from persistent left SVC. Hsu LF, Jais p, Keane D. Circulation 2004;109:829-832