P Wave Analysis in the Era of Atrial Fibrillation Ablation

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KEYWORDS

• Atrial fibrillation • Atrial remodeling • Holter recording • P wave

KEY POINTS

- Atrial fibrillation (AF) is a complex arrhythmia not yet completely understood.
- The role of surface electrocardiogram (ECG) is only apparently limited by the disorganized nature of AF.
- Attentive analysis of the ECG can greatly help in the diagnosis and management of AF.
- Electrocardiographic techniques useful in characterizing and managing AF include Holter monitoring and frequency domain analysis of atrial electrograms.

INTRODUCTION

Atrial fibrillation (AF), affecting 1% to 2% of general population, is the most common sustained arrhythmia. It is also related to increased hospitalizations and mortality, causing a significant increase in health care financial resources spent for AF treatment. Its prevalence and incidence of associated morbidity dramatically increases with age.[1] The usual role of the electrocardiogram (ECG) in the management of AF is to diagnose this arrhythmia, to monitor the effects of antiarrhythmic drugs (AADs), and to assess the ventricular response during episodes of AF. In a patient complaining of palpitations, AF episodes can be suspected by simple pulse palpation by the patient or relatives, or by documenting heart rate by using modern sphygmomanometers that are able to identify irregular cardiac rhythms. The suspicion of AF can then be confirmed by a 12-lead ECG, which can differentiate between AF and other irregular rhythms.

More recently, single-lead tracings can be obtained using small tools attached to a smartphone, allowing low-cost continuous monitoring of patients with arrhythmias.

Despite its obvious limitations, surface ECG and its applications remains a very valuable tool, not only in the diagnosis of AF but also in the definition of its proarrhythmic substrate and response to therapy.

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THEORY OF ATRIAL FIBRILLATION, TRIGGERS, AND SUBSTRATE (FOCAL ATRIAL TACHYCARDIA, DISPERSION OF REPOLARIZATION, FIBRILLATORY CONDUCTION, ROTORS)

Despite many years of intense research, the mechanism of AF remains imperfectly understood. This is in part because most of the relevant observations have been made in experimentally induced AF in animal models and extrapolated to clinical human AF. There is general agreement that the term AF includes different types of arrhythmia that are clinically defined as paroxysmal, persistent, and permanent. Behind this subdivision, which is based on the duration of AF and its spontaneous ability to return to sinus rhythm (SR), is the fundamental concept of electrical remodeling induced by repetitive of bouts of AF on cardiac tissue.

The term remodeling refers to several changes induced by periods of fast electrical stimulation, such as that observed during paroxysms of AF, on ionic channels expressions, cellular metabolism, and interstitial cardiac tissue. These alterations include downregulation of sodium and calcium channels, and the potassium channel (Transient Outward Potassium Current).

These changes lead to a shortening of the atrial refractory period, loss of physiologic adaptation to increased rates, and a decrease of conduction velocity. The consequences of these changes are an increased inducibility and duration of periods of induced AF, leading to the concept of AF begetting AF.[2]

Finally, the profound electrophysiological abnormalities induced by rapid atrial stimulation in animal models lead to a higher incidence of spontaneous tachycardia often initiated by unstable Ca currents leading to early after depolarizations. This appears to be particularly frequent in myocytes within the pulmonary veins (PVs), Marshall veins, and (possibly) thoracic veins.

Longer periods of AF induce changes in atrial cellular substructure, including accumulation of glycogen, loss of myofibrils, fragmentation of sarcoplasmic reticulum, dispersion of nuclear chromatin, and (more generally) loss of muscle mass. The effects of these functional and structural abnormalities have a wide impact on the propagation of the electrical impulse and atrial mechanics. In humans, a clear relationship between atrial dilatation and AF is commonly observed. Although atrial dilation often precedes AF, atrial diameter has been observed to increase as consequence of this arrhythmia. The enlargement is accompanied by impaired contractility, observed after a few minutes of experimental AF and persisting, in humans, for weeks or even months in atria exposed to prolonged periods of AF.[3]

The physiologic anisotropy and dispersion of atrial conduction is greatly enhanced by the electrophysiological changes induced by even a short burst of AF, becoming greatly altered by the appearance of diffuse fibrosis secondary to more sustained periods of this tachycardia.

The current understanding of induction and maintenance of AF is based on these fundamental clinical and experimental observations. The concept of triggers as fast bursts of automatic atrial tachycardia (AT) inducing atrial remodeling and altering the substrate to increase spontaneous discharges and perpetuate longer periods of AF has received considerable clinical support, and has served well in guiding modern ablative strategies.

The triggering of AF by AT had been reported long before a seminal observation localized the discharging focus to 1 of the PV and eliminated recurrence of AF by ablating the arrhythmogenic area.[4] This initial and the ensuing confirmatory reports helped establish the PVs and, more generally, the left atrium (LA) as the source of most AF triggers.

Having identified triggers and the effects of their repetitive discharges on atrial tissue, it remains to clarify the electrophysiological mechanism leading from a fast regular AT to a chaotic rhythm such as AF. There is general agreement that the irregular propagation of AF is greatly facilitated by the presence of localized areas of tissue anisotropy where a rapid single activation wave-front turns into fibrillatory conduction. Regions with variable conduction velocities and refractoriness due to heterogeneity in fiber orientation and thickness, such as the pectinate muscle, the crista terminalis (CT), and the posterior LA wall are incapable of maintaining 1 to 1 conduction at a high frequency of stimulation. In these circumstances, the uniform front of activation breaks into reentrant wavelets, causing AF. By inducing the changes previously described, any conditions that induce atrial remodeling, from bursts of rapid stimulation to disease-increasing atrial volume or pressure load, will increase the physiologic anisotropy present in normal atria.

There is, therefore, a shift in arrhythmia mechanism from regular, which is initiated and maintained on bursts of fast automatic AT, to a perpetuation of self-sustained chaotic tachycardia.

Although abnormal automaticity can explain the mechanism of triggers, the mechanism of degeneration and maintenance of AF is not fully understood.

There is general agreement that reentry is the basic mechanism of AF. This is not the anatomically determined reentry observed in atrial flutter