EDITORIAL

Atrial Electrical Activity and Atrial Fibrillation

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The sinus node is the origin of the atrial electrical activity in the normal heart, and depolarization proceeds in an organized fashion by cell-to-cell transmission and through specialized conducting pathways from the right to the left atrium via internodal tracts. Ectopic foci exist in various locations in atrial tissue, and recent studies have highlighted the presence of ectopic foci in and around the origin of the orifice of pulmonary veins that open into the left atrium. Atrial depolarization generates the atrial P wave that is often manifest as a double-hump configuration reflecting early right atrial depolarization and late left atrial depolarization. This configuration can usually be seen in lead II. In lead V_1, the initial right atrial portion of the P wave has an upright configuration, and the terminal left atrial portion may be evident as an inverted wave, especially if the left atrium is enlarged. Atrial repolarization is usually not evident and may occur in the PR segment or in the early portion of the ventricular QRS complex. Alterations in the configuration of the atrial P wave due to intraatrial conduction disturbances have attracted considerable attention since these alterations may be a harbinger of atrial fibrillation.

A spectrum of atrial arrhythmias exist, and these include sinus node dysfunction with sinus bradycardia, sinus node arrest, and the atrial bradycardia-tachycardia syndrome. Most regular paroxysmal supraventricular tachycardias involve atrial-ventricular nodal reentry circuits. Atrial flutter almost always involves a discrete macro-reentry circuit. Atrial fibrillation is the most frequently sustained atrial tachyarrhythmia. It can occur as an isolated event termed "lone atrial fibrillation," but is more likely to be a consequence of underlying heart disease, atrial dilatation, or simply related to the aging process with a progressively increasing frequency of this rhythm disorder with advancing age past 60 years.

The causes, consequences, and treatment of atrial fibrillation have attracted considerable attention in the past decade. It is now generally accepted that atrial fibrillation is a reentrant tachycardia resulting from random intraatrial circuits that become fractionated around areas of refractory tissue—a pathophysiologic process that maintains the arrhythmia. Atrial fibrillation is frequently initiated by ectopic activity arising from the pulmonary vein region. Atrial fibrillation complicates the underlying heart condition and contributes to the development of atrial blood clots and systemic emboli. Management of atrial fibrillation involves rate control, anticoagulation to prevent emboli, pharmacologic or electrical conversion in selected cases,[1] elimination of the reentrant circuits by surgical or ablation-type maze procedures, or elimination of the focal activity in the pulmonary vein regions by ablation.[2]

When atrial fibrillation persists, remodeling takes place resulting in atrial dilatation and stretch, with secondary down regulation in several of the ion channels that contributes to reduction in atrial conduction velocity and prolongation in atrial repolarization duration. These secondary changes increase the likelihood that atrial fibrillation will persist. It has been said that atrial fibrillation begets atrial fibrillation. Thus, early diagnosis of atrial fibrillation, termination of atrial fibrillation before secondary remodeling takes place, and elimination of the initiating focus and/or reentrant circuits by ablation are important approaches if sinus rhythm is to be maintained as long as possible.

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Can we identify patients at high risk for atrial fibrillation? In this issue of the *Annals,* there are two articles relating P wave parameters recorded on the 12-lead ECG to the risk of atrial fibrillation.[3,4] These articles provide useful insight into altered atrial electrical activity that is part of the substrate for atrial fibrillation. Signal averaging of the P wave with high-frequency analysis may provide more precise and accurate quantification of the P wave than measurements obtained from the 12-lead ECG.

P wave signal averaging requires considerable sophistication, and the methodology has been well described by Dorbala and Steinberg.[5] A three-dimensional X-, Y-, and Z-lead system is used. P wave signals from each lead are amplified and digitized at sampling frequencies of 1000 to 2000 times per second. A template beat is selected. Each incoming digitized beat is aligned to the selected template beat and accepted or rejected depending on the degree of correlation between the two beats. Approximately 200–500 beats are averaged to complete the signal-averaging process, and the averaged P wave is then filtered. The primary parameters analyzed include the filtered P wave duration, terminal P wave duration, and terminal root mean square (RMS) voltage. The filtered P wave duration is generally considered the most useful parameter for predicting atrial fibrillation.

Identification of patients at high risk for intermittent or chronic atrial fibrillation can lead to more appropriate prophylactic therapy. To date, there has been no clinical trial to document the efficacy of this proposed approach. Recent advances in catheter ablation techniques when coupled with noninvasive techniques to better identify at-risk patients with abnormal atrial electrical activity should lead to a reduction in atrial fibrillation and its consequences.

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