P-Pulmonale and New-Onset Atrial Fibrillation

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Atrial fibrillation is the most common arrhythmia encountered in daily clinical practice and is associated with increased morbidity and mortality. Although the prevalence of AF in Japan is estimated to be approximately two-thirds that in the USA, it is increasing, and anticoagulant therapy is mandatory to avoid ischemic stroke and systemic embolization. Furthermore, to prevent AF, improved knowledge of the risks for new-onset AF is required.

In this issue of the Journal, Hayashi et al attempt to determine the risk factors for new-onset AF using the classical ECG parameter, P-pulmonale. Here, we revisit P-pulmonale and the clinical predictors for new-onset AF.

P-Pulmonale and Its Significance

P-pulmonale is an ECG finding that is defined as a P-wave amplitude ≥2.5 mV in the inferior leads II, III, and aVF. The width of P is normal (≤0.10 s), and the frontal axis points toward ≥+75°. P in V1 peaks at ≥1.5–2.5 mV. P-pulmonale can be observed in patients with right-sided pressure overload (Figure 1), and in 302 patients with COLD, it was one of the most commonly observed ECG patterns. However, P-pulmonale patients may show no significant increase in right atrial volume or wall thickness compared with normal hearts at autopsy. In cases of volume overload of the right atrium, such as atrial septal defect, P-pulmonale can be detected in only 6% patients with right atrial enlargement. Furthermore, in 49% of cases, P-pulmonale is associated with overload of the right atrium, and in 36% of cases, it is associated with overload of the left atrium.

Therefore, P-pulmonale has limited value for the diagnosis of overload of the right atrium, and may be associated with left-sided cardiovascular disease, as demonstrated by Hayashi et al. Although left-sided heart failure can cause pulmonary hypertension that subsequently leads to right-sided heart failure, it is unlikely that P-pulmonale was responsible for the overload of the right atrium or right side-heart failure in their study.

Predictors of New-Onset AF

Age, male sex, overt cardiovascular diseases, and diabetes are associated with new AF, for which cardiac failure and rheumatic heart disease are the most powerful predictive precursors, with relative risks in excess of 6-fold. In subsequent echocardiography studies, left atrial size, left ventricular fractional shortening and the sum of the septal and left ventricular posterior wall thicknesses have been found to be risk factors for new onset of AF.

Obesity is an important, potentially modifiable risk factor for AF. The obesity-AF association appears to be partially mediated by diabetes mellitus (DM), and DM has been established as an independent risk for new-onset AF with an odds ratio of 2.13. We studied 28,449 participants who had no baseline AF and during a mean follow-up of 4.5 years, AF developed in 265 subjects. Obesity, diabetes mellitus and hypertension were risk factors for AF development, and AF and metabolic syndrome were significantly linked. Some ECG findings, including evidence of left ventricular hypertrophy, ST-segment abnormality, and high-frequency premature complexes, were revealed to be risk factors for new-onset AF.

ECG atrial parameters may also be risk factors for AF development. To date, P duration, including the width of the filtered P wave and prolonged conduction time in the atrium, have been confirmed as predictors of new-onset AF. Hayashi et al. studied 28,449 participants who had no baseline AF and during a mean follow-up of 4.5 years, AF developed in 265 subjects. Obesity, diabetes mellitus and hypertension were risk factors for new-onset AF.

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et al\(^4\) studied 591 patients with P-pulmonale and 308,391 ECG recordings. AF occurred in 61 patients (AF group) but not in the remaining 530 patients (non-AF group). They are the first to demonstrate that P-wave duration and PQ interval are risk factors for new AF. P-wave duration was highly correlated with PQ interval $>150$ ms, and the latter was a predictor of poor long-term outcome. However, the effect of cardiovascular disease associated with P-pulmonale on their long-term outcomes must be clarified.

**Underlying Mechanism of AF Induction**

Most risks for new-onset AF are considered to result from overload of a dilated left atrium and/or pressure, because reduced cardiac function, hypertrophy, fibrosis, and myocardial ischemia result in increased intracardiac diastolic pressure (Figure 2). Obesity is known to cause dilatation and inflammation of the left atrium. DM is a risk factor for ischemic heart disease. ECG parameters, such as LVH, ST-T changes and premature contraction, would reflect or result in reduced function and compliance. There is increased interest in P-wave analysis, and the feasibility of using surface ECG to detect the presence of an abnormal electrophysiological substrate in the atrial myocardium is becoming possible. Detailed analysis of the P wave with a hardware setup that enables high-quality signal acquisition of the P wave will make it possible to analyze P-wave morphology in relation to AF onset, as performed by Hayashi et al.\(^4\)

The relationship between P duration and PQ interval and the problem of AF detection and progression could not be addressed because of limited space.

**Disclosures**

None.

**References**


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**Figure 2.** Risk factors for new-onset atrial fibrillation (AF). In addition to the primary risk factors (eg, age and male sex), cardiovascular disease is a known risk factor because it is associated with increased diastolic pressure and triggers pressure overload of the atrium. Diabetes mellitus (DM) or obesity can be the cause of inflammation of oxidative stress in the atrium. Among the parameters, left ventricular hypertrophy (LVH), ST-T changes or relatively frequent premature contractions are risk factors, because they represent reduced ventricle compliance. Prolonged P duration or intra-atrial conduction time are more serious risk factors for AF. The presence of P pulmonale is a risk factor that is confirmed in the this issue of the Journal. HF, heart failure; HT, hypertension; VPC, ventricular premature contractions.