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Editorial

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The influence of hyperthyroidism in the electrophysiological properties of the atrial muscle in paroxysmal atrial fibrillation

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Editorial

Paroxysmal atrial fibrillation (PAF) is a common arrhythmia frequently seen in the clinical setting, and is well known to be associated with various pathological cardiac entities.[1–5] These cardiac conditions include coronary heart disease, cardiomyopathy, pericardial disease, valvular heart disease, arterial hypertension, congestive heart failure, Wolff-Parkinson-White syndrome, and sinus node dysfunction.[6–9] PAF is also associated with certain non-cardiac conditions, namely, aging, autonomic tones, thyroid function, acute alcohol intoxication, metabolic or electrolyte disturbances, and drugs, are also affecting initiation, maintenance, and termination of PAF.[6,7] On the other hand, lone or idiopathic PAF has no identifiable underlying cause, and can occur any time for no apparent reason.[10,11] Although these underlying causes may modify the electrophysiological properties of the atrium, there has been little information on the relationship between the atrial electrophysiological properties and predisposing conditions for PAF.

When a premature atrial depolarization encounters a period of incomplete recovery of excitability its conduction velocity diminishes. The conduction velocity and the effective refractory period (ERP) are the determinants in the genesis of reentrant arrhythmias according to the wavelength theory.[12–14] The wavelength is calculated by multiplying the refractory period by conduction velocity, and it is considered as the distance traveled by the depolarization wave during the duration of refractoriness. If the atrial wavelength is long, reentry may not be maintained and the episode of PAF may terminate spontaneously. On the other hand, if the atrial wavelength is relatively short because of either a short refractory period, depressed conduction, or both, then a greater number of wave fronts can circulate through the atrium and atrial fibrillation may be sustained. Therefore, prolonged atrial conduction delay and/or shortened ERP could be expected to increase the propensity to develop episodes of PAF.[12–14] The atrial vulnerability of the atrial myocardium in patients with PAF can be easily exposed in the electrophysiology laboratory. Abnormal responses of the atrial muscle can be elicited by programmed atrial stimulation during the electrophysiological study.[15–18] Certain parameters of atrial vulnerability, namely, repetitive atrial firing, fragmented atrial activity, and intraatrial conduction delay (IACD) can be induced by atrial stimulation. These electrophysiological parameters of atrial vulnerability are more frequently induced in patients with PAF than in those patients without it.[1–5]

Shorter atrial ERP has been also shown to be of electrophysiological significance in the genesis of PAF. On the other hand, the slowing in conduction of the atrial impulses may set the background for reentry to occur.[1–5] Although an increase of 20ms or more in the atrial conduction time in response to early extra-stimuli appears to be a physiological response of the normal atrium, patients with PAF show longer IACD zones and maximum IACDs than control subjects without atrial arrhythmias.[15–18] Thus, the IACD zone and maximum IACD are believed to be good indices of a tendency to develop AF. We have previously shown that these indices of atrial vulnerability were greater in patients with hyperthyroidism and PAF than in control subjects.[19,20] The greater atrial IACD and shorter atrial ERP resulted in shortening of the atrial wavelength, which predisposed to development of PAF.[19–22] The mean atrial ERP in patients with hyperthyroidism (187±7 ms) was significantly shorter than that of controls (215±29ms, $p<0.01$). The mean IACD zone in patients with hyperthyroidism (63±57ms) was significantly greater than that of controls (34±22ms) ($p<0.01$).[19] The mean maximum IACD in patients with hyperthyroidism (64±37 msec) was also significantly greater than that of controls (43±20msec) ($p<0.01$).[19] Therefore, the increase of atrial IACD and the shortening of atrial ERP may contribute to develop PAF in patients with hyperthyroidism. With atrial endocardial mapping, we have previously reported a significantly greater extension of abnormal endocardial electrograms within the right atrium during sinus rhythm in patients with PAF.[1–5] These abnormal atrial electrograms were believed to reflect areas of altered anatomy with fragmented, anisotropic, and slow conduction that renders patients with this electrophysiological abnormality vulnerable to develop AF. We have also showed that the area of diseased atrial muscle in the PAF patients with sinus node dysfunction was more extensive than in those without.[1] In clinical studies, the duration of the monophasic action potential has been suggested to predict recurrence of AF after electrical cardioversion.[23–25] Patients with persistently short monophasic action potential duration during sinus rhythm had a higher incidence of recurrent AF than patients with normal action potential duration.[23–27] Therefore, any condition which shortens atrial ERP and action potential duration will be arrhythmogenic. Previous experimental studies suggested that changes in the thyroid state could modulate the repolarization and duration of the cardiac

action potential.[24,25] Thus, a shortening of the atrial ERP is expected to increase the propensity for PAF in patients with hyperthyroidism despite having or not an organic substrate in the atrial muscle.

Because the atrial ERP was shorter in patients with hyperthyroidism than in controls, earlier atrial premature beats with coupling intervals close to their shortened atrial ERP could facilitate atrial IACD.[19] In patients with hyperthyroidism, shortening of the atrial wavelength due to a slow atrial conduction velocity with short atrial ERP seems to predispose to development of intra-atrial reentry and, thus, clinical episodes of PAF.

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Conflicts of interest

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