LETTERS TO THE EDITOR

doi:10.1093/eurheartj/ehp068
Online publish-ahead-of-print 17 March 2009

Central and peripheral vagal nerve involvement in atrial fibrillation

With interest we read the article by de Vos et al.1 about the characteristics, demographics, treatment, and outcome of patients with paroxysmal atrial fibrillation (AF), classified as vagal, adrenergic, or mixed. The study raises the following concerns.

If pathology of the vagal nerve is suspected to be involved in the pathogenesis of AF, all these patients need to be investigated by a neurologist. Since the tenth cranial nerve receives cerebral and spinal afferents, originates in the brainstem and carries motor, sensory, and parasympathetic fibres, the neurological investigation should be directed towards detection of innervation abnormalities of the striated muscles of the larynx, pharynx, tongue, and tensor veli palatini, for abnormalities of visceral sensory afferents from the larynx, trachea, oesophagus, thoracic and abdominal viscera, stretch receptors, and chemoreceptors from the aortic arch, and for sensory abnormalities behind the ear, external acoustic meatus, tympanic membrane, or the pharynx. Imaging studies should be carried out from the cerebrum, including the brainstem nuclei and the afferents of these nuclei.

Though AF is a cardiac disease, it may be triggered by extracardiac abnormalities, which significantly affect its function. This has been shown in animal experiments by stimulation of the left cervical vagal nerve.2 Also, in humans, AF has been reported to be triggered by implantation of a vagus nerve stimulator in a patient with partial seizures refractory to medical treatment,3 deglutition,4 reflux oesophagitis,5 colon diverticulitis,6 or ureterolithiasis.7 Thus, patients with suspected vagal AF should be investigated for abnormalities of the pharynx, tongue, larynx, or tensor veli palatini, of the thoracic and abdominal viscera. These investigations may have a therapeutic relevance since treatment of the triggering abnormality may reduce or even abolish AF. After treatment with proton pump inhibitors, the frequency of AF has been reported to decrease in patients with oesophagitis.5,8 Thus, it would be interesting to know how many patients with vagal AF in the Euro Heart Survey suffered from any of these disorders, and if treatment of these disorders had reduced the occurrence of AF and the event rate. It would also be interesting to know if there were differences concerning the rate of oral anticoagulated patients and rate of stroke or embolism between adrenergic and vagal AF. How to explain patients with detectable triggers more often had thyroid disease? Did they suffer from hyperthyroidism or hypothyroidism? How often was thyroid disease due to amiodarone therapy?

Overall, as mentioned by the authors in the limitations of the study, differentiation between vagal and adrenergic AF needs to be better delineated. Additionally, if vagal AF is assumed to exist, any pathology along the course of the vagus nerve needs to be excluded not to overlook a potential therapeutic intervention. It also needs to be re-evaluated whether the recommended therapeutic measures are applicable in the light of the present results, showing that patients with vagal AF require non-cardiac therapy for additional non-cardiac disease.

References

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Risk factors for myocardial infarction in women and men

The INTERHEART study demonstrated that women experience first myocardial infarction (MI) on average 9 years later than men even though similar risk factor associations with MI are present in women.1 Why do we find such a difference between males and females?

Many unanswered questions regarding sex differences remain. Oestrogen has cardioprotective effects against acute injury through a variety of complex mechanisms.2 Of interest, basal plasma epinephrine levels are lower in women than in men. This difference could reflect reduced synthesis, increased degradation, or reduced basal release. Stress activates early gene expression in both the central nervous system and the ventricular myocardium in rodent models,3 the myocardial changes in gene expression being mediated by activation of both α-adrenoceptors and β-adrenoceptors. Oestrogen reduces these changes in gene expression, protecting against the ventricular dysfunction observed in this rodent model of cardiomyopathy induced by chronic immobilization.3 Chronic but not

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