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NEW ASPECTS IN ATRIAL FIBRILLATION

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ÖZET

ATRİAL FİBRİLASYONDA YENİ GÖRÜŞLER

Atrial fibrilasyon insanlarda en sık görülen kardiak aritmilerden biridir ve sebebi halen tam olarak anlaşılamamıştır. Bu makalede atrial fibrilasyonun sebebi ve kesin tedavi seçeneklerinin bazı çözüm yollarını gözden geçirdik.

***Anahtar kelimeler** : Atrial fibrilasyon, aritmi, tedavi*

SUMMARY

Atrial fibrillation, one of the most common cardiac arrhytmias in human, is maintained by mechanisms not yet fully understood. In this article, we reviewed many of these new avenues for unravelling the nature of the atrial fibrillation and the consequent possibilities for radical treatment.

***Key words :** Atrial fibrillation, arrhytmia, treatment*

INTRODUCTION

Atrial fibrillation (AF) is easily and frequently diagnosed in clinical medicine by electrocardiogram (1). The overall prevalence of AF in industrialized countries is about 0.4 to 0.9%, and the incidence is heavily age-dependent rising about 0.1% to 0.2% per year over the age range 40-90 years (1,2). The prognostic significance of AF is due to an almost doubled mortality rate compared with patients in sinus rhythm, and to the well-established increased risk of thromboembolic complications, mainly stroke (3).

ELECTROPHYSIOLOGY

Abnormalities in atrial electrophysiology and structure are important for the clinical development of AF. Chronic AF is often preceded by episodes of paroxysmal AF (4). Cox et al. (5) documented multiple wavelets wandering around both natural anatomical obstacles and functional arcs of conduction block, while in some cases the wavelets seemed to be offsprings of a single reentrant circuit. In Cox study no evidence for micro-reentry or focal automaticity was found and it was concluded that AF was maintained on the basis of macro-reentry. The increased intra-atrial pressure and higher oxygen consumption during fibrillation may explain changes in the atrial myocardium and progressive fibrosis (6).

CLINIC

Development of AF is associated with loss of AV synchrony and irregular heart rate. (7). When studying patients with paroxysmal attacs

of AF, they reported that two opposite patterns and mechanisms could be identified (vagal and sympathetic) and they often interacted (8,9). Vagal and adrenergic paroxysmal AF are easy to identify provided one carefully considers the clinical history and pays attention to heart rate changes prior to the arrhytmia onset (10) (Table I.).

Table I. Clinical differences between vagally mediated and adrenergically mediated atrial fibrillation.

Vagally mediated AF	Adrenergic AF
Male predominance, age 30-50	No sex or age predominance
Absence of structural heart dis.	Any cardiovascular disease
Attacs at night, never in the morn-	Attacs occuring at daytime,
ning favored by rest, alcohol, digestion	favored by stress, exercise
Mixed picture of atrial flutter/AF	Mixed picture of atrial tachycardia/AF
Vagal maneuvers may induce AF	Cathecolamines may induce AF
B-blockers/digoxin	B-blockade±digoxin:indicated±type
Contraindicated type IA/IC(except pro-	IA/IC drugs (propaf.) ±amiodarone
pafenon) and /or amiodarone	
Atrial pacing may be useful	No indication for atrial pacing

THEURAPEUTIC CONSIDERATION

The advent of shock treatment using direct electric current to restore sinus rhythm in patients with atrial fibrillation created a completely new situation for the treatment of this disorder (11). It was no longer necessary to rely upon the uncertain effect of quinidine in large and increasing doses for restoration of normal heart beat (11). For a long time, however, quinidine, has been the mainstay of therapy for keeping the sinus rhytm, once restored through the DC chock (12). Other newer antiarrhythmic drugs, and in particular amiodarone, have been advocated in this situation (13).

Oral theophylline as an effective therapy in most patients with AF and a slow ventricular response and suggest that initial dosage of the drug should be 500 to 600 mg daily; it can be then slightly decreased or increased according to the clinical course. Serum theophylline levels should be >5 ng/ml; with lower values, the effects of the drug on heart rate unreliable; furthermore, to prevent side effects of the drug, the serum concentration should not be >15 ng/mL (14).

The best and most appropriate stroke prevention in AF remains a challenge, but so far clear guidelines for the use of warfarin have emerged. Every patient is subjected to an individual risk factor evaluation, and the well-established absolute and relative contraindications for warfarin observed, and a modestly aggressive warfarin regimen is used, i.e., target INR 2.0-3.0, and finally, the major bleeding complications can be kept as low as in the reported trials, under 1.3% per year (1). In nonvalvular AF are found high embolization risk, especially in patients with hypertension, diabetes mellitus, prior stroke or transient ischemic attack (1). In all other heart disease are possible risk factors for embolic risk.

Not surprisingly, beta-blocking drugs are not useful in treating most cases of paroxysmal idiopathic AF, while by contrast, patients its heart disease do benefit from beta-blockers and or digoxin (10). This notion is important because it has positive as well as negative implications. In other words, beta-blockers or digoxin are not just indicated in vagal atrial fibrillation, but they are absolutely contraindicated; they tend to precipitate the arrhytmhmia and to diminish the efficacy of traditional antiarrhythmic treatment (10).

In conclusion, we believe that AF is still an important health problem and new therapeutic strategies should be investigated.

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