The Association of Chronic Atrial Fibrillation with Right Atrial Dilatation and Left Ventricular Dysfunction in the Elderly

Thesis

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LIST OF ABBREVIATION

ACC : American college of cardiology.

AF : Atrial fibrillation

AHA : American heart association.

ASE : American society of echocarchiography.

AT-1 : Angiotensin type I.

AT-2 : Angiotensin type II.

AV Conduction : Atrio-ventricular conduction

AV Node : Atrio ventricular node.

CHARM trial : Candesartan in heart failure

assessment of reduction in mortality.

CHF : Congestive heart failure

DIAMOND : Danish – investigations of arrhythmia

and mortality on dofetilide.

DIG : Digitalis investigation group.

ECG : Eletrcocerdiogram.

EDV : End diatolic volume.

ESV : End systolic volume.

LA : Left atrium.

LC : Light chain.

LC-1 : Essential light chain.

LC-2 : Regulatory light chain.

LIST OF ABBREVIATION (Cont...)

LV : Left ventricle.

LVEDd : Left ventricular end diastolic diameters.

LVEF : Left ventricular ejection fraction.

LVESD : Left ventricular end systolic diameter.

LVFS : Left ventricular fraction shortening.

MHC : Myosin heavy chancel molecule.

NYHA : New York heart association.

RA : Right atrium.

RV : Right ventricle.

SA Node : Sino-atrial node.

SOLVD : Studies of left ventricular dysfunction.

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INTRODUCTION

espite an extensive literature on atrial fibrillation since it was first documented by Sir James Mackenzie almost a century ago, certain questions still remain regarding the mechanical of fibrillation. consequences atrial Clinicoechocardiographic studies have demonstrated that left atrial enlargement is very common in patients with atrial fibrillation, and is usually associated with clinical deterioration (Barker et al., 1993).

Although left atrial dilatation is generally considered to be the cause of atrial fibrillation, it could be the consequence of atrial fibrillation in the absence of mitral valve disease, as suggested by longitudinal studies. If this is true, atrial fibrillation should produce similar effects on the right atrium (*Dreifus et al.*, 1982).

AIM OF THE WORK

The aim of this study is to assess the prevalence of right atrial dilatation in chronic atrial fibrillation and to evaluate the left ventricular functions by echocardiography.

Definition of Atrial Fibrillation

Introduction

Atrial fibrillation (AF), the most common sustained cardiac rhythm disturbance, is increasing in prevalence as the population ages. Although it is often associated with heart disease, AF occurs in many patients with no detectable disease. Hemodynamic impairment and thromboembolic events result in significant morbidity, mortality, and cost. (Fuster et al., 2001).

Definition and electrocardiographic patterns A. Atrial fibrillation

Atrial Fibrillation is a supraventricular tachyarrhythmia characterized by uncoordinated atrial activation with consequent deterioration of atrial mechanical function. On the electrocardiogram (ECG), AF is described by the replacement of consistent P waves by rapid oscillations or fibrillatory waves that vary in size, shape, and timing, associated with an irregular, frequently rapid ventricular response when atrioventricular (AV) conduction is intact (Bellet et al., 1971)

The ventricular response to AF depends electrophysiological properties of the AV node, the level of vagal and sympathetic tone, and the action of drugs. Regular RR intervals are possible in the presence of AV block or interference by ventricular or junctional tachycardia. A rapid, irregular, sustained, wide-QRScomplex tachycardia strongly suggests conduction over an accessory pathway or AF with underlying bundle-branch block. Extremely rapid rates (over 200 bpm) suggest the presence of an accessory pathway (Prystowsky et al., 1998)

B. Related arrhythmias

AF can be isolated or associated with other arrhythmias, often atrial flutter or atrial tachycardia. Atrial flutter can arise during treatment with antiarrhythmic agents prescribed to prevent recurrent AF. Atrial flutter is more organized than AF, with a saw-tooth pattern of regular atrial activation called flutter (f) waves on the ECG, particularly visible in leads II, III, and aVF. Untreated, the atrial rate typically ranges from 240 to 320 beats per minute (bpm), with f waves inverted in ECG leads II, III, and aVF and upright in lead V₁. The wave of activation in the right atrium (RA) may be reversed, resulting in f waves that are upright in leads II, III, and aVF and

inverted in lead V₁. Two-to-one AV block is common, producing a ventricular rate of 120 to 160 bpm. Atrial flutter can degenerate into AF. AF can initiate atrial flutter, or the ECG pattern can alternate between atrial flutter and AF, reflecting changing atrial activation. Other atrial tachycardias, as well as AV reentrant tachycardias and AV nodal reentrant tachycardias, can also trigger AF. In other atrial tachycardias, P waves are readily identified and are separated by an isoelectric baseline in 1 or more ECG leads. The morphology of the P waves can help localize the origin of atrial tachycardias. A unique type of atrial tachycardia originates in the pulmonary veins, is typically more rapid than 250 bpm, and often degenerates into AF. Intracardiac mapping can help differentiate the various atrial arrhythmias (Jais et al., *1997)*.

Historical background

the diagnosis of atrial fibrillation Because requires measurement of the electrical activity of the heart, it was not truly described until 1874, when Edmé Félix Alfred Vulpian observed the irregular atrial electrical behavior that he termed "fremissement fibrillaire" in dog hearts (Vulpian et al., 1874).

In the mid-eighteenth century, Jean-Baptiste de Sénac made note of dilated, irritated atria in people with mitral stenosis (McMichael et al., 1992)

The irregular pulse associated with AF was first recorded in 1876 by Carl Wilhelm Hermann Nothnagel and termed "delirium cordis", stating that "In this form of arrhythmia the heartbeats follow each other in complete irregularity. At the same time, the height and tension of the individual pulse waves are continuously changing" (Nothnagel et al., 1876).

Correlation of delirium cordis with the loss of atrial contraction as reflected in the loss of waves in the jugular venous pulse was made by Sir James MacKenzie in 1904 (MacKenzie et al., 1904).

Willem Einthoven published the first electrocardiogram showing AF in 1906 (*Einthoven et al., 1906*).

The connection between the anatomic and electrical manifestations of AF and the irregular pulse was made in 1909 by Carl Julius Rothberger, Heinrich Winterberg, and Sir Thomas Lewis (Rothberger et al., 1909).