Managemet of Atrial fibrillation

In critical ill patients

An Essay
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Abstract

The pathogenesis of AF is thought to involve an interaction between initiating triggers, and an abnormal atrial tissue substrate capable of maintaining the arrhythmia

AF is capable of inducing electrophysiologic changes that promote further AF. These include electrical, contractile and structural changes to the atria that have collectively become known as atrial remodelling.

AF may be immediately recognized by sensation of palpitations or by its hemodynamic or thromboembolic consequences or may remain asymptomatic for a period of unknown duration.

Key word: ECG- ACEIs-Fibrilation-Critical- ARBs

Dedication

I dedicate this work to my parents who gave me the strength , to my beloved wife and children who have been always by my side and gave me the greatest support all the time.

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ABBREVIATIONS

*ACC : American college of cardiology

*ACEIs: Angiotensin converting enzyme inhibitors

*ARBs : Angiotensin receptor blockers

*ADP : Adenosine diphosphate

*AF : Atrial Fibrillation

*AHA : American heart association

*APTT : Activated partial thromboplastin time

*ANP : Atrial natriuretic peptide

*AV : Atrioventricular

*BID : Twice daily

*BMI : Body mass index

*BNP : B type natriuretic peptide

*CAMP: Cyclic adenosine monophosphate

*CRP : C-reactive protein

*CHADS:Cardiac failure, hypertension, age, diabetes, stroke

*ESC : European society of cardiology

*ECG : Electrocardiogram

*EPS : Electrophysiological study

*ERP : Effective refractory period

*FDA : Food and drug administration

*HF : Heart failure

*HRV : Heart rate variability

*ICU : Intensive Care Unit

*INR : International normalized ratio

*LA : Left atrium

*LAA : Left atrial appendage

*LMWH: Low molecular weight heparin

*LV : Left ventricle

*MI : Myocardial infarction

*PV : Pulmonary veins

*PUFAs:Polyunsaturated fatty acids

*PT : Prothrombin time

*RA : Right atrium

*RV : Right ventricle

*SAN : Sinoatrial node

*SOFA: sequential organ failure assessment

*SVT : Supraventricular tachycardia

*TEE : Transesophageal echocardiography

*TTE : Transthoracic echocardiography

*TIA : Transient ischemic attacks

*UFH : Unfractionated heparin

*VF : Ventricular fibrillation

*VT : Ventricular tachycardia

*WK : Week

Introduction

Atrial fibrillation (AF) is a relatively common arrhythmia, atrial fibrillation was not truly described until 1874, when *Edmé Félix Alfred Vulpian* observed the irregular atrial electrical behavior in dog hearts.(1)

The irregular pulse associated with AF was first recorded in 1876 by *Carl Wilhelm Hermann Nothnagel* , stating that "In this form of arrhythmia the heartbeats follow each other in complete irregularity". (2)

Correlation of this arrhythmias with the loss of atrial contraction as reflected in the loss of *a waves* in the jugular venous pulse was made by *Sir James MacKenzie* in 1904.(3)

Atrial fibrillation occurs at epidemic levels worldwide, affecting 1 – 1.5% of the population in the developed world. It presents an economic burden to healthcare systems as it leads to more hospitalizations than any other arrhythmia (4, 5).

The prevalence of AF depends upon the population studied, as the risk increases with age and with underlying heart disease (6, 7, 8).

It is important clinically because affected patients may be at increased risk for longer I.C.U stays and higher inpatient mortality especially when accompanied by myocardial infarction (9)

Increased mortality in A.F (1.5 to 1.9-fold in the Framingham study) is caused by deterioration in hemodynamics due to:

- Increased heart rate.

- Loss of atrioventricular (AV) synchrony.

- Progressive dysfunction of the left atrium and left ventricle.

- Decreased stroke volume, decreased cardiac output and acute congestive heart failure.

- Cerebrovascular stroke and other embolic events from atrial thrombi (10, 11).

One of the complications of atrial fibrillation is cerebrovascular stroke witch is prevented by anticoagulant (warfarin) that has narrow therapeutic window caring risk of intracranial hemorrhage if the international normalized ratio (INR) increases above therapeutic levels. Alternately, lack of insufficient anticoagulation increases the risks of embolic strokes. These strokes are typically more severe and result in greater disability than strokes from other causes (4).

The pharmacological treatment of atrial fibrillation has been the subject of some controversy over the past few years, with some advocating rhythm control and others advocating rate control (12).

Definition and Classification

Anatomical and physiological consideration

Anatomy of the conductive system:

Sinus node

The sinoatrial (SA) node occupies a 1 cm^2 area on the lateral surface of the junction of the superior vena cava and right atrium near the crista terminalis. In rare cases, the SA node may be found medially along the ridge of the atrial-caval junction (13).

Internodal pathways

The spread of electrical activation from the sinus node extends toward the AV node via Purkinje-like pale cells in atrial muscle bundles. Anterior, medial, and posterior interatrial conduction pathways arise from the SA node. The anterior and medial pathways are located anterior and posterior to the foramen oval, the posterior pathway is situated caudal to the foramen oval (14).

Atrioventricular node

The AV node is situated directly on the right atrial side of the central fibrous body in the muscular portion of the AV septum, just superior and anterior to the ostium of the coronary sinus. Measuring approximately 0.1 cm X 0.3 cm X 0.6 cm, the node is flat and oval. (15)

His bundle and bundle branches

The AV node continues onto the His bundle via a course inferior to the commissure between the septal and anterior leaflets of the tricuspid valve. The bundle follows a course along the inferior border of the membranous septum and, near the aortic valve, gives off fibers that form the left bundle branch.

The His bundle is located on the left side of the ventricular septum in 80 % of patients. In the 20 % of patients in whom the bundle is on the right side of the septum, the His bundle is connected to the left bundle by a narrow stem.

The left bundle branch further subdivides into several smaller branches that begin at the ventricular septal surface and radiate around the left ventricle. The right bundle branch originates from the His bundle near the membranous septum and courses along the right ventricular septal surface, passing toward the base of the anterior papillary muscle (16).

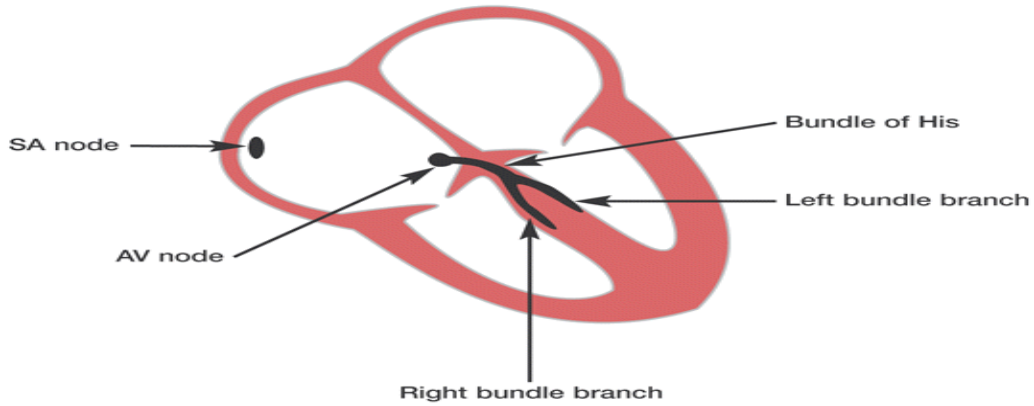

Fig 1 A diagrammatic representation of the electrical conductive system of the heart (17).