INTRODUCTION

Atrial fibrillation is a supraventricular arrhythmia. It is defined as rapid, uncoordinated atrial activity with a rapid, irregular ventricular response ***(Rosenthal et al., 2010)***.

Atrial fibrillation is the most common arrhythmia. It is a significant cause of morbidity such as stroke, thromboembolisms, and heart failure. There are medical conditions that are clearly the cause of some cases of atrial fibrillation, but often times the reason why atrial fibrillation happens is not known. Risk factors have been identified that increase the chances of developing atrial fibrillation. However, atrial fibrillation can also occur in healthy individuals when these risk factors are absent ***(Rosenthal et al., 2010)***.

Atrial fibrillation (AF) is the most common cardiac rhythm disorder and it affects an estimated 2.3 million adults in the United States, the majority of whom are over the age of 65 years ***(Feinberg et al., 1995)***.

Far from benign, AF can lead to stroke, tachycardia-induced cardiomyopathy, and congestive heart failure. AF accounts for about 15% of all strokes that occur each year in the United States ***(Hylek et al., 2001)***.

The number of patients with AF is increasing throughout the industrialized world as the population ages. In the United States, the prevalence of AF is expected to grow 2.5-fold to 5.6 million by 2050, and over half of those afflicted will be age 80 or older ***(Hylek et al., 2001)***.

DEFINITION

AF is a complex, progressive disorder that manifests in a variety of ways and is associated with a wide range of comorbidities. As a result there is no single treatment strategy that is optimal for all patients. Although drug therapy remains the most common first-line treatment, surgical methods for ablating AF have proven highly beneficial for patients who have failed drug therapy, including those with long-standing and/or continuous AF ***(Cox et al., 1991)***.

AIM OF THE WORK

This review describes the evolution of AF surgery, from Atrial isolation and "cut and sew" to ablative techniques, using novel energy delivery systems. We discuss its role both as a standalone and concomitant procedure and highlight the current indications and outcomes for the most common techniques described in the literature.

ETIOLOGY

There are many causes of atrial fibrillation. These can be conveniently divided into four basic categories: cardiac, medical/surgical, pharmacological, and genetic.

Genetic

There are rare forms of atrial fibrillation that are due to genetic single mutations ***(Elinor et al., 2003)***.

There is also increasing evidence that the risk of developing atrial fibrillation increases as much as 30% to 41% – even when risk factors have been accounted for – if one's first or second degree relatives have atrial fibrillation. This appears to be especially so for patients with lone atrial fibrillation ***(Marcus et al., 2008)***.

Pharmacological

There are several drugs that can cause atrial fibrillation. Ethanol – drinking alcohol – has long been known to be a cause of atrial fibrillation. Atrial fibrillation can occur as a result of chronic ethanol abuse and can occur acutely after binge drinking – the latter syndrome being called "holiday heart." ***(Ettinger, 1978)***.

It is not clear why ethanol causes atrial fibrillation. In acute cases, it may be that metabolic breakdown products of ethanol, e.g., acetylaldeyde, stimulate the release of

catecholamines from the myocardium. When it is chronically abused, ethanol may cause structural damage and metabolic derangements to the heart. In people who drink heavily, the risk for developing atrial fibrillation is approximately 1.6 times greater than among people who abstain or drink less ***(Falcone et al., 2009; Mukamal et al., 2005)***.

Stimulants such as cocaine, methamphetamine, and caffeine (in excess) may cause atrial fibrillation. It does not appear that moderate consumption of caffeine increases the risks of developing atrial fibrillation ***(Conen et al., 2010)***.

Cigarette smoking may increase the risk of developing trial fibrillation ***(Heeringa et al., 2008)***.

Medical/Surgical

There are many medical conditions that can cause atrial fibrillation. Atrial fibrillation occurs in approximately 15% of all patients with **hyperthyroidism**, and sub-clinical hyperthyroidism is associated with a three-fold increase in the incidence of atrial fibrillation ***(Bielecka-Dabrowa et al., 2009)***.

Diabetes has long been considered to be a risk factor for the development of atrial fibrillation. There is evidence for and against this association, but a recent study that controlled for other risk factors associated with atrial fibrillation indicated that for some people, diabetes can definitely increase the risk of developing atrial fibrillation ***(Nichols et al., 2009)***.

Because diabetes mellitus and obesity are increasing in prevalence and are associated with an elevated risk of AF, Fontes et al examined whether insulin resistance is an intermediate step for the development of AF. In a community-based cohort that included 279 patients who developed AF within 10 years of follow-up, no significant association was observed between insulin resistance and incident AF ***(Fontes et al., 2012).***

Atrial fibrillation is commonly seen (up to 76% incidence) after **subarachnoid hemorrhage** ***(Frontera et al., 2008)***.

Obstructive sleep apnea has also been strongly implicated as a risk factor for atrial fibrillation ***(Capampangan et al., 2010)***.

Obesity increases the risk of atrial fibrillation by approximately 49%, and the level of risk increases as body mass index increases ***(Crandall et al., 2009)***.

Atrial fibrillation is common as a postoperative event after **cardiac surgery**, and to a lesser extent, **pulmonary surgery**. Approximately 40% of all patients who have coronary artery bypass graft surgery or valvular surgery develop atrial fibrillation after the operation; if the two surgeries are combined this rate is approximately 60% ***(Jongnarangsin et al., 2008)***.

Advanced age, obesity, the metabolic syndrome, and cardiopulmonary disease are thought to increase the risk of post-operative atrial fibrillation ***(Echahidi et al., 2008)***.

It has been associated with an increase in morbidity and mortality and prolonged hospital stay ***(Echahidi et al., 2008)***.

The arrhythmia is usually transient, and preoperative treatment with amiodarone, beta-blockers, corticosteroids, sotalol, magnesium, ACE inhibitors, and some calcium channel blockers has been shown to reduce the incidence of post-operative atrial fibrillation ***(Burgess et al., 2006; Mayson et al., 2007)***.

Atrial fibrillation also commonly occurs during renal dialysis. However, it generally resolves spontaneously within several hours after the dialysis session has ended ***(Zebe et al., 2000)***.

Cardiac:

There are strong associations between cardiac disease and atrial fibrillation. Hypertension, congestive heart failure, valvular heart disease " e.g: mitral/tricuspid regurgitation or stenosis, mitral valve prolapse", and coronary artery disease are all important risk factors for the development of atrial fibrillation ***(Crandall et al., 2009)***.

Hypertension, especially, is considered to be the most reliable predictor of risk for atrial fibrillation ***(Go et al., 2009)***.

There is ongoing research that is attempting to discover the exact incidence and the relationship between these diseases and atrial fibrillation: one of the biggest questions seems to be whether or not some of the conditions are the cause of atrial fibrillation or an effect of the arrhythmia ***(Bouzas-Mosquera et al., 2010; Goto et al., 2008; Anter et al., 2009).***

PATHOPHYSIOLOGY

The Normal Heart Conducting System:

The cardiac impulse originates in the sinoatrial node (SA node), located in the right atrium and activates first the right atrium then the left atrium. The general direction of the atrial activation is inferiorly, to the left, and posteriorly. This causes the atria to contract and pump blood from the atria to the ventricles; it is recorded on an EKG as a P wave.

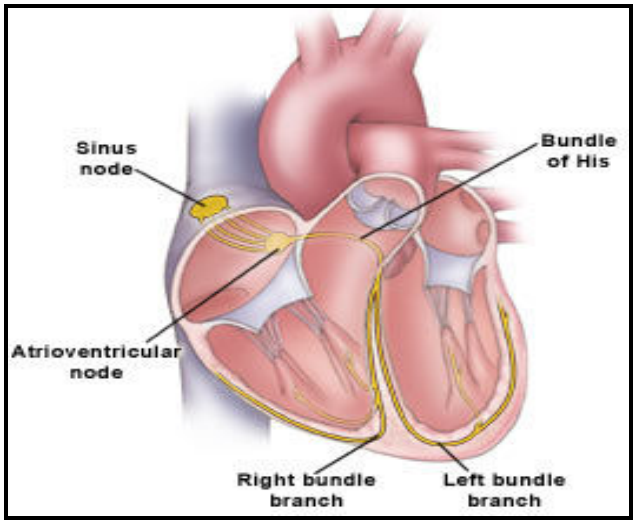

Figure (1): Noraml conducting pathway.

The atrial impulse is delayed in the atrioventricular node (AV node) to allow the ventricular chambers to fill, and is then conducted rapidly through the ventricles (the bundle of His, the right and left bundles, and the Purkinje fibers). This causes the

ventricles to pump blood out of the heart and to the body; it is recorded on an EKG as a QRS complex.

Recovery following the cardiac cycle, or repolarization, follows. This is recorded as a T wave (Baylor heart and vascular hospital center).

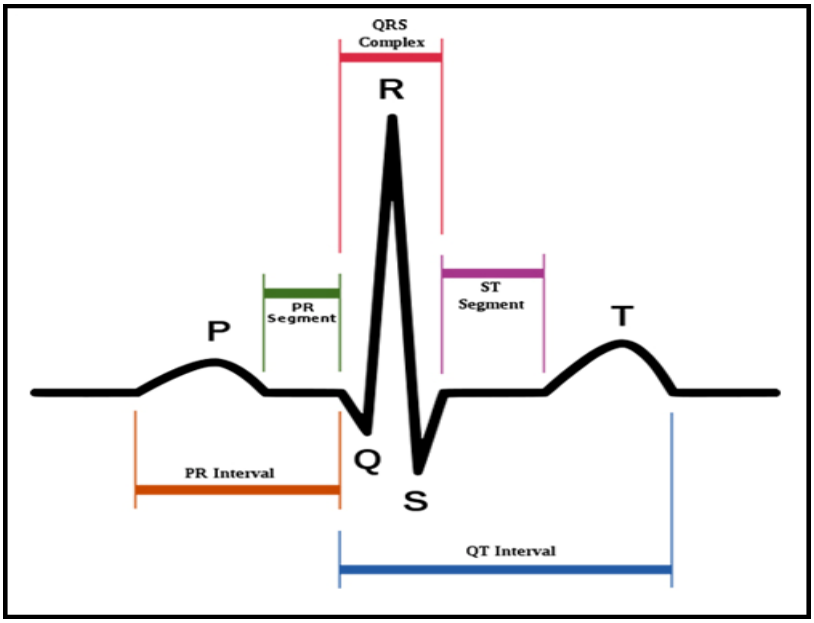

Figure (2): Normal ECG.

Atrial fibrillation (AF) shares strong associations with other cardiovascular diseases, such as heart failure, coronary artery disease (CAD), valvular heart disease, diabetes mellitus, and hypertension. These factors have been termed upstream risk factors, but the relationship between comorbid cardiovascular disease and AF is incompletely understood and more complex than this terminology implies. The exact mechanisms by which

cardiovascular risk factors predispose to AF are not understood fully but are under intense investigation. Catecholamine excess, hemodynamic stress, atrial ischemia, atrial inflammation, metabolic stress, and neurohumoral cascade activation are all purported to promote AF ***(Kannel et al., 1998).***

Although the precise mechanisms that cause atrial fibrillation are incompletely understood, ***AF appears to require both an initiating event and a permissive atrial substrate.*** Significant recent discoveries have highlighted the importance of focal pulmonary vein triggers, but alternative and nonmutually exclusive mechanisms have also been evaluated. These mechanisms include multiple wavelets, mother waves, fixed or moving rotors, and macro-reentrant circuits. In a given patient, multiple mechanisms may coexist at any given time. The automatic focus theory and the multiple wavelet hypothesis appear to have the best supporting data ***(Kannel et al., 1998)***.

Automatic focus

A focal origin of AF is supported by several experimental models showing that AF persists only in isolated regions of atrial myocardium. This theory has garnered considerable attention, as studies have demonstrated that a focal source of AF can be identified in humans and that isolation of this source can eliminate AF ***(Nakao et al., 2002)***.

The pulmonary veins appear to be the most frequent source of these automatic foci, but other foci have been demonstrated in several areas throughout the atria. Cardiac muscle in the pulmonary veins appears to have active electrical properties that are similar, but not identical, to those of atrial myocytes. Heterogeneity of electrical conduction around the pulmonary veins is theorized to promote reentry and sustained AF. Thus, pulmonary vein automatic triggers may provide the initiating event, and heterogeneity of conduction may provide the sustaining conditions in many patients with AF ***(Nakao et al., 2002)***.

Multiple wavelet::

The multiple wavelet hypothesis proposes that fractionation of wave fronts propagating through the atria results in self-perpetuating "daughter wavelets." In this model, the number of wavelets is determined by the refractory period, conduction velocity, and mass of atrial tissue. Increased atrial mass, shortened atrial refractory period, and delayed intra-atrial conduction increase the number of wavelets and promote sustained AF. This model is supported by data from patients with paroxysmal AF demonstrating that widespread distribution of abnormal atrial electrograms predicts progression to persistent AF ***(Nakao et al., 2002)***.

Intra-atrial conduction prolongation has also been shown to predict recurrence of AF. Together, these data highlight the

importance of atrial structural and electrical remodeling in the maintenance of AF-hence the phrase "atrial fibrillation begets atrial fibrillation ***(Akyürek et al., 2001)***.

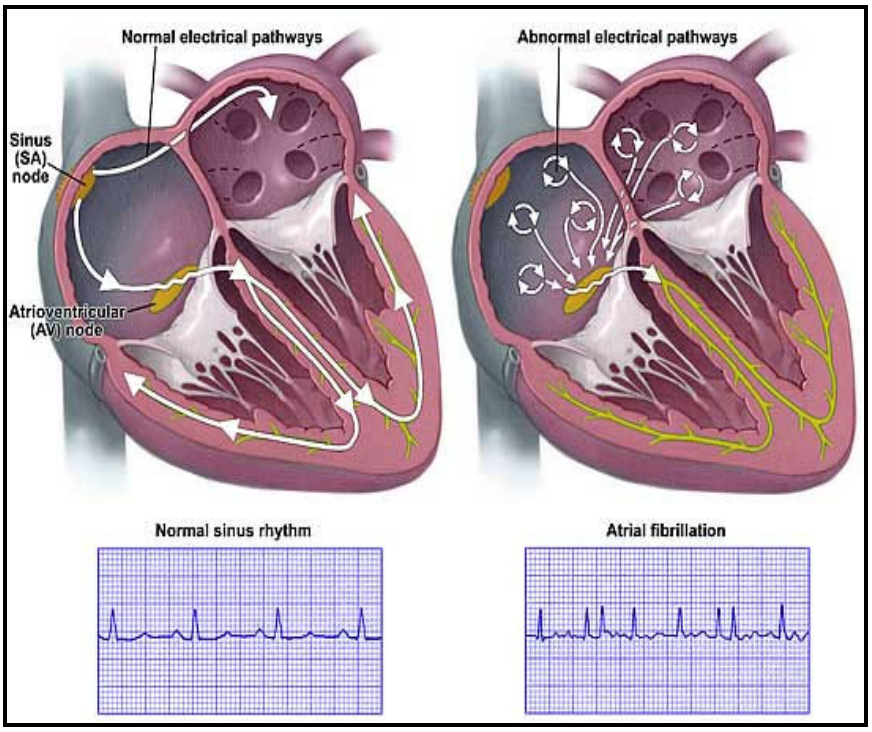

Figure (3): Normal and Abnormal conducting pathway.

EPIDEMIOLOGY

Atrial fibrillation affects more than 2.2 million persons in the United States. AF is strongly age-dependent, affecting 4% of individuals older than 60 years and 8% of persons older than 80 years. Approximately 25% of individuals aged 40 years and older will develop AF during their lifetime ***(Lloyd-Jones et al., 2004)***.

The prevalence of AF is 0.1% in persons younger than 55 years, 3.8% in persons 60 years or older, and 10% in persons 80 years or older. With the projected increase in the elderly population in the United States, the prevalence of AF is expected to more than double by the year 2050. AF is uncommon in childhood except after cardiac surgery ***(Abdel Latif et al., Jan 2004)***.

The incidence of AF is significantly higher in men than in women in all age groups. AF appears to be more common in whites than in blacks, with blacks have less than half the age-adjusted risk of developing AF.

In 10-15% of cases of AF, the disease occurs in the absence of comorbidities (lone atrial fibrillation). However, AF is often associated with other cardiovascular diseases, including hypertension; heart failure; diabetes-related heart disease; ischemic heart disease; and valvular, dilated, hypertrophic, restrictive, and congenital cardiomyopathies ***(Alonso et al., 2011)***.

The Atherosclerosis Risk in Communities (ARIC) Study suggests reduced kidney function and presence of albuminuria are strongly associated with AF ***(Alonso et al., 2011)***.

The rate of ischemic stroke in patients with nonrheumatic AF averages 5% a year, which is somewhere between 2 and 7 times the rate of stroke in patients without AF. The risk of stroke is not due solely to AF; it increases substantially in the presence of other cardiovascular diseases ***(Stöllberger et al., 2004)***.

The prevalence of stroke in patients younger than 60 years is less than 0.5%; however, in those older than 70 years, the prevalence doubles with each decade ***(Rathore et al., 2000)***.

The attributable risk of stroke from AF is estimated to be 1.5% for those aged 50-59 years, and it approaches 30% for those aged 80-89 years. Women are at a higher risk of stroke due to AF than men and some have suggested this may be due to undertreatment with warfarin. However, one study of patients 65 years or older with recently diagnosed AF found warfarin use played no part in the increased risk of stroke among female patients ***(Avgil Tsadok et al., 2012)***.