

# **Atrial Fibrillation After Cardiac Surgery**

**An Essay**

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general intensive care**

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بسم الله الرحمن الرحيم

قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا عَلَّمْتَنَا  
إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ

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# *List of contents*

<i>Introduction</i> .....	9
<i>Chapter I</i> : Pathophysiology and Mechanism.....	12
<i>Chapter II</i> : Features.....	31
<i>Chapter III</i> : Morbidity and Mortality.....	42
<i>Chapter IV</i> : Prevention.....	50
<i>Chapter V</i> : Treatment.....	61
<i>English summary</i> .....	78
<i>References</i> .....	81
<i>Arabic summary</i> .....	- -

## *List of abbreviations*

<b><i>ACC</i></b>	american college of cardiology
<b><i>ACEI</i></b>	angiotensin converting enzyme inhibitors
<b><i>AERP</i></b>	atrial effective refractory period
<b><i>AF</i></b>	atrial fibrillation
<b><i>AFFIRM</i></b>	atrial fibrillation follow-up investigation of rhythm managment
<b><i>AHA</i></b>	american heart association
<b><i>APD</i></b>	action potential duration
<b><i>a PTT</i></b>	activated partial thromboplastin time
<b><i>AVN</i></b>	atrio-ventricular node
<b><i>CABG</i></b>	coronary artery bypass graft
<b><i>CAD</i></b>	coronary artery disease
<b><i>CAMP</i></b>	cyclic adenosine monophosphate
<b><i>CPB</i></b>	cardio-pulmonary bypass
<b><i>CRP</i></b>	C-reactive protein
<b><i>CSA</i></b>	central sleep apnea
<b><i>DM</i></b>	diabetes mellitus
<b><i>ECG</i></b>	electro-cardiogram

<b><i>EF</i></b>	ejection fraction
<b><i>ERP</i></b>	effective refractory period
<b><i>ESC</i></b>	european society of cardiology
<b><i>ESRD</i></b>	end stage renal disease
<b><i>HCM</i></b>	hypertrophic cardiomyopathy
<b><i>HF</i></b>	heart failure
<b><i>HMG CoA</i></b>	hydroxyl-methyl-glutaryl coenzyme A
<b><i>HRV</i></b>	heart rate variability
<b><i>IABP</i></b>	intra aortic ballon pump
<b><i>I<sub>CaL</sub></i></b>	L-type Ca <sup>2+</sup> channels
<b><i>I<sub>K1</sub></i></b>	inward rectifier K <sup>+</sup> current channels
<b><i>I<sub>K,ACH</sub></i></b>	acetylcholine-activated K <sup>+</sup> current channels
<b><i>I<sub>Kur</sub></i></b>	ultra-rapid delayed rectifier K <sup>+</sup> current channels
<b><i>I<sub>SUS</sub></i></b>	sustained outward K <sup>+</sup> current
<b><i>I<sub>to</sub></i></b>	transient outward K <sup>+</sup> current channels
<b><i>INR</i></b>	international normalized ratio
<b><i>LA</i></b>	left atrium
<b><i>LAD</i></b>	left anterior descending
<b><i>LAF</i></b>	lone atrial fibrillation
<b><i>LIMA</i></b>	left internal mammary artery

<b><i>LVH</i></b>	left ventricular hypertrophy
<b><i>MI</i></b>	myocardial infarction
<b><i>NAC</i></b>	N-acetyl cystien
<b><i>NADPH</i></b>	nicotinamide adenine dinucleotide phosphate
<b><i>POAF</i></b>	post operative atrial fibrillation
<b><i>PUFA</i></b>	poly unsaturated fatty acid
<b><i>PV</i></b>	pulmonary vein
<b><i>RA</i></b>	right atrium
<b><i>RAP</i></b>	rapid atrial pacing
<b><i>RCA</i></b>	right coronary artery
<b><i>SAN</i></b>	sino-atrial node
<b><i>SVG</i></b>	saphenous vein graft
<b><i>SVT</i></b>	supra-ventricular tachycardia
<b><i>TEE</i></b>	trans oesophageal echo.
<b><i>TIA</i></b>	transient ischemic attack
<b><i>TTE</i></b>	trans thoracic echo.

## *List of figures*

*Figure (1)*.....the cardiac conduction system.....**13**

*Figure (2)*.....ECG of atrial fibrillation.....**39**

# ***Introduction***

# Introduction

Atrial fibrillation is the most common arrhythmia after coronary artery bypass graft with as many as 10% to 40% of all patients undergoing CABG. Although atrial fibrillation is a benign arrhythmia it may contribute to the morbidity, high cost and prolonged hospital stay ( *Hill et al ., 2002* ) .

Atrial fibrillation is the result of a fractionated atrial activity mainly due to shortening of atrial refractory period, which allows multiple wavelets pass through the atrial mass .If an obstacle in the conduction pathway exists, a subsequent phenomenon of reentry of the electrical activation can lead to the arrhythmia ( *Mathew et al ., 2004* ) .

The pathological mechanisms responsible for high incidence of atrial fibrillation after cardiac surgery in general and after coronary artery bypass surgery in particular remains unclear ( *Samantha Poli et al ., 2003* ) .

Although the pathogenesis of atrial fibrillation after open heart surgery is incompletely understood, stimuli and triggers such as preexisting structural changes of the atria related to hypertension, volume overload, age, atrial ischemia, electrolyte imbalances and pericardial lesions are thought to play a role in the pathogenesis of atrial fibrillation after coronary artery bypass graft ( *Johann Awer et al ., 2005* ) .

What is certain is that the incidence of atrial fibrillation far exceeds its reported prevalence in the general population and in patients with atherosclerotic coronary diseases .Several factors are associated with the development of atrial fibrillation after cardiac surgery. These factors can be classified as preoperative, intraoperative or postoperative.Older age has consistently predicted a higher incidence of postoperative atrial fibrillation. Incidence increases by at least 50% per decade of older age ( *Hravnak et al., 2002* ) .

## CHAPTER I

# ***PATHOPHYSIOLOGY & CAUSES***

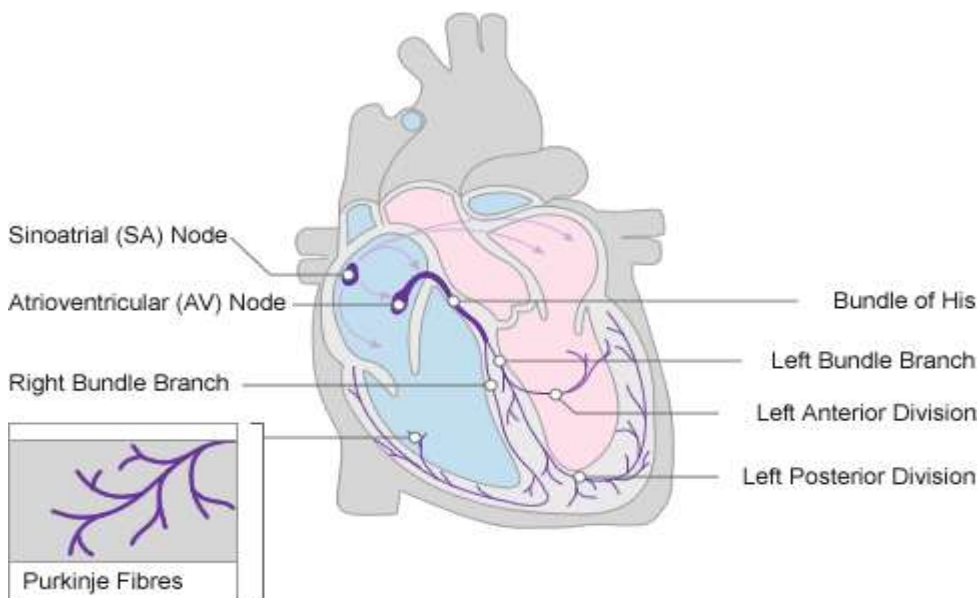
## *Cardiac Conduction System*

Going back to the analogy of the central heating system, the pump, pipes and radiators are of no use unless connected to a power supply. The pump needs electricity to work. The human heart has a similar need for a power source and also uses electricity. Thankfully we don't need to plug ourselves in to the mains, the heart is able to create it's own electrical impulses and control the route the impulses take via a specialised conduction pathway.

This pathway is made up of 5 elements:

1. The sino-atrial (SA) node
2. The atrio-ventricular (AV) node
3. The bundle of His
4. The left and right bundle branches
5. The Purkinje fibres ( *Schotten et al ., 2011* ).

The cardiac conduction system     *figure (1)*



( *Agur et al., 2005* )

The SA node releases electrical stimuli at a regular rate, the rate is dictated by the needs of the body. Each stimulus passes through the myocardial cells of the atria creating a wave of contraction which spreads rapidly through both atria. The heart is made up of around half a billion cells. The majority of the cells make up the ventricular walls. The rapidity of atrial contraction is such that around 100 million myocardial cells contract in less than one third of a second. So fast that it appears instantaneous. The electrical stimulus from the SA node eventually reaches the AV node and is delayed briefly so that the contracting atria have enough time to pump all the blood into the ventricles. Once the atria are empty of blood the valves between the atria and ventricles close. At this point the atria begin to refill and the electrical stimulus passes through the AV node and Bundle of His into the Bundle branches and Purkinje fibres ( *Banach et al ., 2006* ).

As the ventricles contract, the right ventricle pumps blood to the lungs where carbon dioxide is released and oxygen is absorbed, whilst the left ventricle pumps blood into the aorta from where it passes into the coronary and arterial circulation. At this point the ventricles are empty, the atria are full and the valves between them are closed. The SA node is about to release another electrical stimulus and the process is about to repeat itself. However, there is a 3rd section to this process. The SA node and AV node contain only one stimulus. Therefore every time the nodes release a stimulus they must recharge before they can do it again ( *Mathew et al ., 2004* ).

The SA node recharges while the atria are refilling, and the AV node recharges when the ventricles are refilling. In this way there is no need for a pause in heart function. Again, this process takes less than one third of a second. The times given for the 3 different stages are based on a heart rate of 60 bpm , or 1 beat per second. The term used for the release (discharge) of an electrical stimulus is "depolarization", and the term for recharging is "repolarization" ( *Auer et al ., 2005* ).

So, the 3 stages of a single heart beat are:

1. Atrial depolarization
2. Ventricular depolarization
3. Atrial and ventricular repolarization.

As the atria repolarise during ventricular contraction, there is no wave representing atrial repolarisation as it is buried in the QRS ( *Wellens ., 2003* ) .