

# Emergency surgery in a patient with Atrial fibrillation

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## Introduction

Atrial fibrillation is the most common sustained cardiac rhythm disturbance encountered in clinical practice and is often associated with organic or structural heart disease. Haemodynamic impairment and thromboembolic events increase the morbidity and mortality.

## Definition

Defined as a condition in which electrical impulses in the atria degenerate from their usual organized rhythm into a rapid chaotic pattern called Delirium cordis.

It is characterized by in coordinated atrial activation with consequent deterioration of atrial mechanical function usually associated with an irregular rapid ventricular response when AV node conduction is intact.

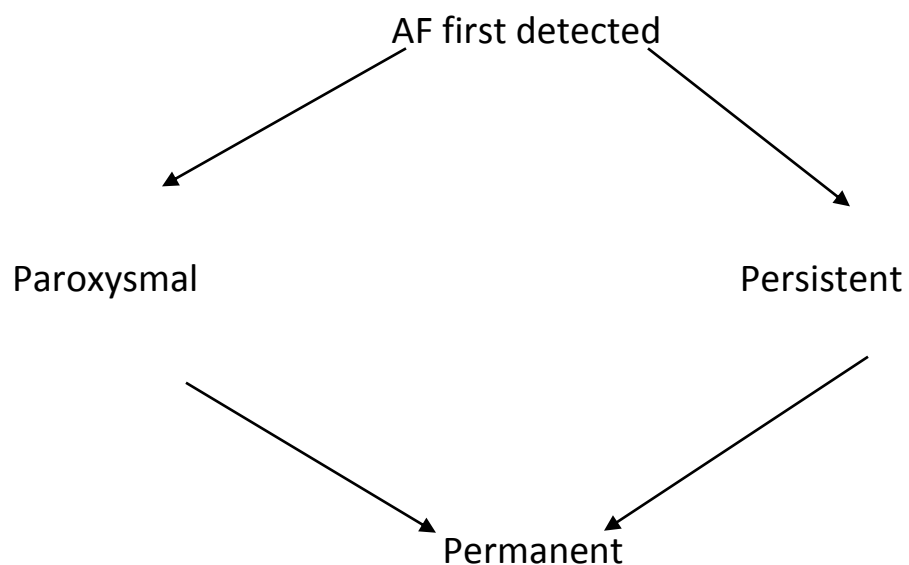
## Incidence

0.4\_1% of the general population and the incidence increases with age. Incidence is almost 4% in people aged more than 75 yrs. The incidence is more in male population.

## Classification

According to ACC/AHA/ESC guidelines on the basis of frequency of episodes and the ability to revert back to sinus rhythm atrial fibrillation which is not associated with reversible causes can be classified as follows.

1. Paroxysmal: When AF terminates spontaneously
2. Recurrent: When a patient has had two or more episodes
3. Persistent: Sustained without spontaneous termination
4. Permanent: AF of more than one year
5. Lone AF: AF without any clinical or echocardiographic evidence of cardiac disease in a patient of age less than 60 years.



## Aetiology of Atrial fibrillation

- Idiopathic
- *Acid base abnormalities*
- *Electrolyte abnormalities*
- Acute infections esp.pneumonia
- Alcohol intoxication
- Atrial septal defect
- *Manipulations during cardiac surgery*
- Atrial myxoma
- Cardiomyopathy
- Central venous catheter
- Drugs like digoxin,theophylline,caffeine and ondansetron
- Electroconvulsive therapy
- *Hypertension*
- *Hypovolemia*
- *Hypoxia*
- *Myocardial infarction*
- Pericardial disease
- Pleural disease
- Post pneumonectomy
- Pre excitation syndromes
- Pulmonary embolism
- *Rhumatic heart disease*
- Sick sinus Syndrome
- *Thyrotoxicosis*

## Pathophysiology

Structural abnormalities like patchy fibrosis with juxtaposition of normal and diseased atrial fibres .Fibrosis and fatty infiltration of SANode secondary to inflammatory and degenerative changes.

## **Mechanism of atrial fibrillation**

Enhanced automaticity in one or several rapidly depolarizing foci near superior pulmonary veins and reentry involving one or more circuits. AVnodal conduction is responsible for the rapid ventricular rate in AF. If conduction occurs over an accessory pathway during AF it can result in a very rapid ventricular response that can be fatal. Drugs like digitalis , calcium channel blockers and beta blockers which are given to slow the ventricular rate can sometimes enhance conduction across these accessory pathways resulting in ventricular fibrillation which can be fatal.

## **Haemodynamic consequences**

1. Loss of synchronous atrial mechanical activity ie loss of atrial kick.
2. Irregularity in ventricular response.
3. Inappropriately rapid heart rate reducing diastolic filling time there by reducing cardiac output. Persistently high heart rate results in myocardial energy depletion, ischemia and remodeling.
4. Systemic thromboembolism. Thrombus usually forms in the left atrial appendage. It is presumed that thrombus formation requires continuation of AF for 48hrs but thrombus formation is reported as early as 24 hrs. Resolution of thrombus occurs in most of the patients with anticoagulants for 4 weeks.

## **Clinical features**

Symptoms may range from an asymptomatic patient to a patient with embolic manifestations. Other symptoms include palpitation, syncope, chestpain, dyspnoea, fatigue, light headedness and polyuria.

Clinical signs will be an irregularly irregular pulse, JVP without an a wave, variation in the intensity of the first heart sound, signs of other cardiac abnormalities and signs of heart failure.

## Evaluation of patients

### History

- Presence and nature of symptoms
- Clinical type of AF
- Onset of the first symptomatic attack
- The frequency duration precipitating factors, modes of termination
- Response to drugs
- Presence of underlying heart disease or other reversible conditions.

### ECG

- To identify rhythm
- LV hypertrophy
- P wave duration and morphology of fibrillatory waves with a ragged baseline
- Preexcitation
- BBB
- Prior Myocardial infarction
- Other arrhythmias
- To follow the response to antiarrhythmic drugs



### Chest Radiograph

- Cardiomegaly
- Lung parenchyma
- Pulmonary vasculature

## **Echocardiography** To evaluate

- Valvular heart disease
- Left and right atrial size
- Left ventricular size and function
- Peak right ventricular pressure
- Left ventricular hypertrophy
- Left atrial thrombus
- Pericardial disease

## **Blood tests to evaluate thyroid function**

## **Coagulation profile**

## **Additional Tests**

- Exercise testing-to reproduce exercise induced AF and to rule out ischemia
- Holter monitoring-to document intermittent AF,to monitor rate control and to detect associated arrhythmias
- TEE - To identify a LA thrombus and to guide cardio version
- Electro physiological study-to classify mechanism,to identify predisposing arrhythmia and seeking sites for curative ablation.

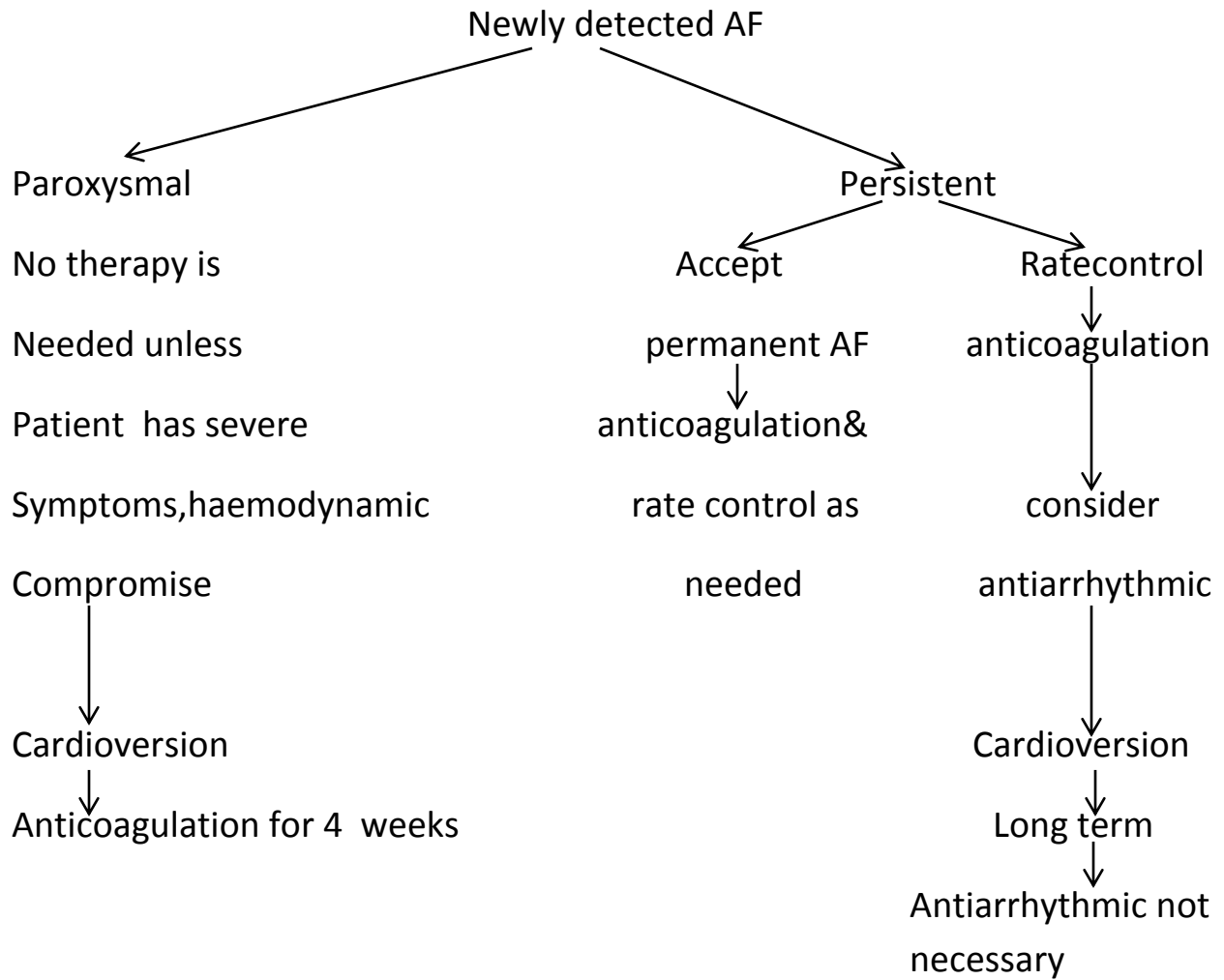
## **MANAGEMENT**

**GOALS** Control of ventricular rate

Restoration and maintenance of sinus

Rhythm

Prevention of thromboembolism



**Table 3 Non-pharmacological management of AF**

Rhythm control	Rate control	Stroke prevention
Device therapy  Atrial pacing (single or multisite)	Transcatheter AV junctional ablation and permanent pacemaker implantation	Percutaneous left atrial appendage transcathete occlusion (PLAATO)
Atrial defibrillators (stand-alone or with pacemaker function)	Radiofrequency transcatheter AV junction modification	
Ablation therapy  Operative (Maze procedure, Pulmonary vein isolation, His bundle ablation) Percutaneous transcatheter Techniques (pulmonary vein isolation, radiofrequency ablation of triggers or substrate)		

In an emergency scenario which can vary from an appendicectomy to a major laparotomy or thoracotomy involving major volume shifts a thorough clinical examination with a detailed history taking has to be done to confirm the diagnosis , presence of other coexisting diseases, extent of surgical pathology, haemodynamic status of the patient, history of drug therapy, previous cardioversion and previous anaesthetic exposure etc. During clinical examination all peripheral pulses should be examined and any absence of a peripheral pulse has to be documented. A thorough neurological examination has to be done and documented. Any history of transient ischemic attack or a stroke has to be properly documented.



Investigations like blood counts, blood biochemistry to know renal and hepatic status, thyroid function test, arterial blood gas analysis, serum electrolyte estimation, 12 lead ECG, Chest x-ray, an Echocardiogram, coagulation profile has to be done. If transthoracic echo fails to detect a left atrial appendage thrombus a trans esophageal echocardiogram can be done to evaluate the thrombus. An expert cardiologist help should be sought regarding management.

The reversible causes of atrial fibrillation like hypoxia, hypovolemia, acid base abnormalities like acidosis or alkalosis, electrolyte abnormalities like hypokalemia and hypomagnesaemia have to be corrected.

If after correcting all reversible causes AF persists and if the patient is haemodynamically unstable an emergency synchronized electrical cardioversion should be performed under IV sedation.

Mechanism of DC CV—Circuits sustaining reentry within atria are disrupted and a period of asystole follows during which the SA node is able to resume its role. But atria go to a stand still following a DC shock called stunning of atria and the mechanical activity takes 48 hrs to resume and complete after 3 weeks so thromboembolic events can occur after CV and anticoagulants has to be continued at least for 3—4 weeks after CV. Success is higher in recent onset AF and anterior-posterior paddle positions. Energy levels used will be 25 to 100J using biphasic wave forms.

Risks involved with cardioversion will be embolic events and cardiac arrhythmias esp. Brady arrhythmias.

## **Pharmacological cardio version**

Can be done as follows

Amiodarone—Can be used in patients with severe LV dysfunction. 150-300mg given over 10 to 120mts with BP monitoring. Maintenance infusion 0.5-1mg/mt. It is very expensive, produces severe venous irritation and has an extremely long half life—120 days.

Procainamide—10-15mg/kg given IV upto 50mg/min. Maintenance 2-4mg/mt. Blood levels should be followed closely.

Ibutilide—Has few extracardiac side effects but incidence of torsades de pontis is higher . 1mg IV bolus can repeat after 10 mts if no effect. Avoid use in patients with baseline prolongation of QT interval.

## **Control of ventricular rate**

Can be done in an emergency situation with

### ***Beta blockers***

Ideal in a patient with ischemic heart disease. Esmolol—500micro gm/kg over 1mt as bolus. Maintenance 25-300microgm/kg/mt titrated. Onset of action is instantaneous and effect goes off after 30mts of stopping the infusion.

Metoprolol—2.5-5mg over 2-3 mts repeat as needed. Onset of action 5mts and duration of action 30-60 mts.

### ***Calcium channel blockers***

Ideal in patients with pulmonary diseases.

Diltiazem—0.25mg/kg over 2mts,infusion 5-15mg/hr upto 24hrs Repeat bolus may be needed.Onset of action -5mts and duration of action will be 5-7 hrs

Verapamil—5-10mg bolus can be repeated in 15-30 minute interval to achieve goal. Onset of action is 5mts and duration of action will be 5-12-hrs.

### **Digoxin**

Ideal in the presense of heart failure 0.25-0.5 mg IV bolus . Maintenance infusion to a total dose of 1mg/24hrs. Onset of action 30mts.

### **Prevention of Thromboembolic manifestations**

In an emergency situation if the patient is already on an anticoagulant our primary concern will be to balance the bleeding complications with maintenance of anticoagulation in the perioperative period.

A patient with acute onset AF with spontaneous termination and aged less than 60 years without any risk factors no anticoagulant is necessary in the perioperative period.

If the patient who has atrial fibrillation of recent onset ie less than 48hrs with a haemodynamic compromise who needs immediate cardioversion needs to be started on heparin IV in the perioperative period and followed up with oral warfarin.

Patients with persistent atrial fibrillation will be on oral anticoagulants like warfarin,aspirin,clopidogrel and dabigatran.

If these patients have to be taken up for an emergency surgery the coagulation status of the patient has to be managed according to the drug the patient is taking.

If the drug is warfarin patient can be taken up after giving adequate fresh frozen plasma and small doses of Vit k intravenously with careful monitoring of prothrombin time and INR.

If the patient is taking a combination of aspirin and clopidogrel patient has to be managed with platelet concentrates and fresh blood.

If the drug is dabigatran fresh frozen plasma and fresh blood can be used. The effect of the drug goes off in 12 to 17 hrs unlike other anticoagulants.

## **Anaesthetic Management**

Anaesthetic technique has to be individualised and decided by the patient's haemodynamic status, surgical condition and coagulation status.

### **Goals of Anaesthesia would be to**

1. Maintain ventricular rate between 80—100/mt. A linient control of rate is sufficient.
2. Avoid hypoxia
3. Avoid hypotension
4. Avoid hypercarbia

Anxiolytic premedication is recommended as anxiety and stress are important precipitating factors for an episode of AF. Midazolam is the ideal agent for anxiolysis.

## **Monitors**

Pulse rate, O<sub>2</sub> saturation, blood pressure, ECG—lead 2, EtCO<sub>2</sub>, urine output and temperature. In a major procedure a central venous pressure monitoring to guide replacement of fluids, Transoesophageal echocardiogram, Arterial line to measure direct blood pressure. Care should be taken in using invasive monitoring devices in a patient who is taking anticoagulants.

## **Induction**

Agents of choice will be sleep dose of thiopentone, etomidate, coinduction-opioid with a benzodiazepine and propofol.

**Maintenance** with O<sub>2</sub>, N<sub>2</sub>O, Isoflurane which has an antifibrillatory action, fentanyl and vecuronium.

## **Post-operative Period**

At the end of the procedure the need for elective postoperative ventilation has to be decided upon the individual patient. If the patient is otherwise stable patient can be extubated after adequate reversal.

Patient has to be monitored in a high dependency unit in the postoperative period.

All peripheral pulses should be felt and the inference documented. Neurological status has to be evaluated for any focal deficit.

Post-operative pain relief has to be managed with opioids, IV Paracetamol and NSAIDs

Antiarrhythmic drugs have to be continued. Anticoagulants should be restarted.

Use of regional anesthesia is always debated in a patient who is on anticoagulants for fear of producing haematomas esp. in areas like epidural space. Advantages of a regional anaesthesia should be weighed against the potential complications. If the patient is not on any anticoagulants continuous regional techniques can be administered safely in procedures involving lower abdomen and peripheral limb surgeries.

## **Conclusion**

Atrial fibrillation is often considered a nuisance arrhythmia because it is not immediately life threatening but it is associated with morbidity and mortality. So a patient with atrial fibrillation has to be managed very carefully in the perioperative period.

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