Atrial Fibrillation

Atrial fibrillation is a common arrhythmia faced by clinicians. Rheumatic mitral valve disease is the commonest cause in India. Left ventricular systolic and/or diastolic dysfunction, myocardial infarction, sick sinus syndrome, thyrotoxicosis and pericardial constriction are other common causes. Electrocardiogram is necessary for diagnosis and planning of proper management.

**Keywords:** Atrial fibrillation, Electrocardiogram, Supraventricular tachycardia.

**Mechanism**

It is caused by multiple micro-reentry circuits with in the atria (Fig. 1) or a rapidly firing focus in the proximal portion of a pulmonary vein. This arrhythmia can also be produced by ‘focal’ mechanism rather than reentry. Atrial fibrillation promotes further atrial fibrillation due to electric remodeling of atria. Atrial fibrillation in absence of any known aetiology is labelled “lone atrial fibrillation”. Atrial fibrillation may be precipitated by an atrial premature beat (Fig. 2). In other cases atrial tachycardia, multifocal atrial tachycardia or atrial flutter may degenerate into atrial fibrillation. It also forms part of spectrum of “sick sinus syndrome” (Fig. 2B). In patients with sick sinus syndrome, management of atrial fibrillation with digoxin can produce longer pauses of bradycardia and electric cardioversion can produce long period of asystole. Atrial fibrillation may be paroxysmal (transient) (Fig. 2) and may terminate spontaneously. Digoxin causes more frequent and longer paroxysms by decreasing refractory period of atrial musculature.

**Atrial waves**

Atrial activity appears as small baseline undulations of changing amplitude, morphology and duration (Fig. 3A) at a rate varying between 350 to 600/minute. These are called fibrillatory waves (f waves). These waves should be differentiated from technical errors. Tremors produce vibrations localised to leads which include the limb having tremors. Electrical disturbance or improper earthing produces very fast and sharp vibrations in baseline. In both these conditions QRS is regular as opposed to irregularly irregular QRS in atrial fibrillation (Fig. 3B). When f waves are prominent, it is called coarse atrial fibrillation (Fig. 3C). When f waves are small or not visible, it is called fine atrial fibrillation (Fig. 3D). This is common with long standing atrial fibrillation. Usually f waves are prominent in lead II and lead V1.
Sometimes undulations are seen only in right precordial leads (Fig. 3D) and lead II may show relatively straight line without any undulations.

Atrioventricular conduction and ventricular rate

- In atrial fibrillation, large number of irregular atrial impulses (350 to 600/minute) try to cross A-V node to reach the ventricles. Normal A-V node can conduct only up to 180 impulses per minute due to its long refractory period. Therefore only some impulses are able to cross the A-V node. Other impulses penetrate the A-V node for varying distance but are unable to cross it. These impulses, however, do depolarise the A-V node for varying interval. This prevents penetration of subsequent impulses for variable interval resulting in irregular QRS. As atrial rate increases ventricular rate decreases because of increasing competition within the AV node. At very fast ventricular rates, irregularity may be difficult to detect due to short R-R interval. Carotid sinus massage can help by reducing ventricular rate. Increasing speed of electrocardiogram trace may also help.

- If A-V node is normal, ventricular rate as high as 200/minute may develop. Digoxin is helpful in controlling ventricular rate at rest by decreasing A-V conduction. It, however, poorly controls ventricular rate during exercise and in other conditions of high sympathetic drive. Concomitant use of beta blockers, verapamil or diltiazem is needed for adequate control of ventricular rate in such situations.

- Ventricular rate of more than 250/minute strongly suggests possibility of preexcitation with antegrade conduction over accessory pathway with short refractory period resulting in broad QRS (Fig. 4A). It may be difficult to differentiate from ventricular tachycardia. Intermittent irregularity and slow QRS upstroke indicating delta wave may help in differentiation. Digitalis and verapamil are contraindicated as they increase conduction over accessory pathway and can induce ventricular fibrillation. Electric cardioversion is needed. Thyrotoxicosis, anxiety, sympathetic stimulation, bronchodilators, cor pulmonale (Fig. 4B), hypoxia and inherently fast atrio-ventricular conduction can also cause fast ventricular rate. However, in these situations, ventricular rate is usually less than 200/minute.

- Regular rhythm in presence of atrial fibrillation
  - Rate around 65/minute suggests A-V dissociation with accelerated junctional rhythm. QRS is narrow if intraventricular conduction is normal. Patients with
sick sinus syndrome may have slower junctional escape rhythm (Fig. 5A).
- Rate around 45/minute with broad QRS suggests complete A-V block with escape rhythm arising from either ventricle (Fig. 5B). Possibility of digitalis toxicity should be considered whenever there is regular rhythm with atrial fibrillation. Continuation of Digitalis results in acceleration of junctional or ventricular rhythm (Fig. 5C).

QRS configuration
- Usually QRS is narrow due to normal intraventricular conduction.
- It may be affected by underlying disease e.g., rheumatic heart disease, hypertension, myocardial infarction, cardiomyopathy, electrolyte imbalance, bundle branch block or pericardial constriction (Fig. 6A and 6B). These conditions are also likely to affect ST segment and T waves.
- Irregular broad QRS (rate>250/minute) may be seen with antegrade conduction over accessory pathway or aberrant ventricular conduction (ventricular rate usually <200/minute)
- Regular broad QRS at slow rate is seen in complete A-V block with escape ventricular rhythm (Fig. 6B).
- Intermittent broad QRS may be seen due to aberrant conduction in a short cycle following a long cycle (Fig. 6C) or intermittent ventricular ectopics (Fig. 6D). Ventricular ectopics can also follow long-short cycle sequence.

Fig. 6(A): Atrial fibrillation with pre-existing left bundle branch block
(B): Atrial fibrillation with low voltage QRS due to pericardial constriction
(C): Atrial fibrillation with aberrant conduction
(D): Atrial fibrillation with ventricular premature beats

Fig. 7(A): Change in degree of aberrancy with change in preceding cycle length
(B): Atrial fibrillation with ventricular ectopic showing difference in initial QRS vector from narrow QRS
(C): Atrial fibrillation with ventricular premature beats showing biphasic QRS

Fig. 8(A): Atrial fibrillation with anterior myocardial infarction
(B): Atrial fibrillation with inferior myocardial infarction
(C): Atrial fibrillation with ST depression and short QTc due to digitalis overdose
(D): Atrial fibrillation with tall tented T waves due to hyperkalemia
but as opposed to aberrant conduction, ventricular ectopics have
- Consistent morphology uninfluenced by cycle length. Aberrancy changes with cycle length and heart rate (Fig. 7A).
- Initial QRS vector opposite to narrow QRS complex (Fig. 7B). In aberrant conduction initial vector of broad QRS is similar to narrow QRS.
- Tendency for compensatory pause4 (Fig. 7B) due to concealed retrograde conduction of ventricular extrasystolic impulse into the A-V node. This renders the A-V node refractory to ensuing impulses of atrial fibrillation. In aberrant conduction, there is no compensatory pause after broad QRS.
- Uniphasic or biphasic QRS complex in lead V1 (Fig. 7C) as opposed to triphasic rSR’ configuration in aberrant ventricular conduction (Fig. 6C)

ST segment and T wave changes
- These are influenced by underlying disease or intraventricular conduction defect (Fig. 8A,B,C)
- Digitalis overdose causes ST segment depression with shortening of QTc interval (Fig. 8D). Other ECG manifestations of digitalis overdose which may be seen in a case of atrial fibrillation include accelerated junctional rhythm, atrio-ventricular block with bradycardia and junctional or ventricular ectopics.
- Amiodarone can produce
  - Slow ventricular rate
  - QT prolongation.
- Hyperkalemia can cause tall tented T waves (Fig. 7E). Hypokalemia can also cause prolongation of QT.

References