

Cardiac Arrhythmias

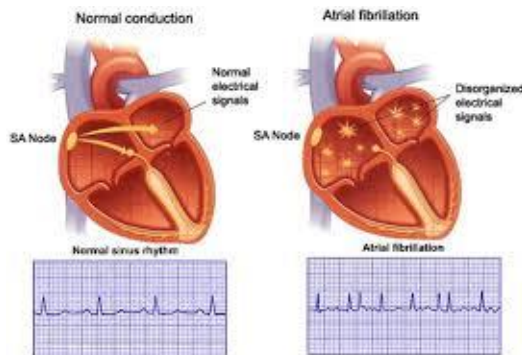
Dr. RAVINDRA GUNDELI (M.D.)
Shri Markendeya Solapur Sahakari Rignalaya
Solapur, India

Definition

Arrhythmias are disorders of impulse formation or disorders of impulse conduction or combination of both.

Mechanisms for arrhythmias in acute myocardial infarction. They include -

- Autonomic nervous system imbalances
- Electrolyte disturbances
- Ischaemia or necrosis
- Slowed conduction



Activation of receptors within atrial or ventricular myocardium by ischaemic or necrotic tissue may cause enhanced efferent sympathetic activity, increased concentrations of circulating catecholamines and local release of catecholamines from nerve

endings within the heart. The last phenomenon may also result from direct ischaemic damage of adrenergic neurons.

Experimental and clinical studies have suggested that electrolyte disturbance; elevated free fatty acid levels and oxygen-derived free radicals also contribute to development of arrhythmias. The severity of these abnormalities with size of infarction and perfusion status of infarct related artery, appear to determine a patient's risk for developing most serious rhythm disturbance - primary ventricular fibrillation.

Definite relationship between episodic and constant hypoxaemia, after acute myocardial infarction is also proved in study done by Dr. Galatius Jensen S, Hansen J, et al.

Classification:

Broadly classified as:

- 1) Bradyarrhythmias
- 2) Tachyarrhythmias

Otherwise classified as below:

Category	Arrhythmias
1) Electrical Instability	a)Ventricular premature beats b)Ventricular tachycardia c)Ventricular fibrillation d)Accelerated idioventricular rhythm e)Nonparoxysmal Av junctional tachycardia
2) Pump failure/ Excessive sympathetic stimulation	a)Sinus tachycardia b)Atrial fibrillation Or Atrial flutter c)Paroxysmal supraventricular tachycardia
3) Brady arrhythmias and conduction disturbances	a)Sinus bradycardia b)Junctional escape rhythm c)Atrioventricular block and intraventricular block

We will now consider each one briefly below.

Ventricular Arrhythmias:

1) Ventricular premature beats (VPBs)

It is believed that frequent Ventricular premature beats (more than five per minute), Ventricular premature beats with multiform configuration, early coupling (the 'R' on T phenomenon) and repetitive patterns in form of couples or salvos presaged ventricular fibrillation. These are like "warning arrhythmias" are present in many patients who develop ventricular fibrillation and now it is clear that it may do with patients who do not develop ventricular fibrillation also. Some reports have also been shown that primary ventricular fibrillation occurs without warning arrhythmias and may even develop in spite of suppression of warning arrhythmias.

Such heterogeneity nature of Ventricular premature beats in acute myocardial infarction studied by, Ronald F. Cawbell, Allan Murray et al.

These Ventricular premature beats result from depolarization that originate in ventricles and occur prior to next normally conducted sinus beats. Suppression of it outweighs potential benefits in most case of acute myocardial infarction.

They occur more than 80% of patients of acute myocardial infarction who were monitored in Coronary Care Units and some studies reported only 45%.

An increasing number of patients had shown 'R' on 'T' ventricular ectopic complexes in the 20 minutes before onset of ventricular fibrillation. This suggests that 'R' on 'T' types of Ventricular premature beats are in some way related to primary ventricular fibrillation.

On the contrary ventricular tachycardia was rarely initiated by an 'R' on 'T' types of Ventricular premature beats and its peak occurrence was at time when 'R' on T ventricular ectopic complexes were rare.

Thus it was concluded by end of study done by Dr. Ronald F. Cawbell, Allan Murray et al at university

department of Cardiology, Freeman Hospital, New Castle - upon - Type - that within first 12 hours from onset of symptoms of acute myocardial infarction, the initiation of primary ventricular fibrillation and ventricular tachycardia is different. Primary ventricular fibrillation is typically 'R' on T initiated where as almost all episodes of ventricular tachycardia are not.

2) Accelerated Idioventricular Rhvthm

It is commonly defined as a ventricular rhythm with a rate of 60 to 125 beats/min and frequently called 'slow ventricular tachycardia'.

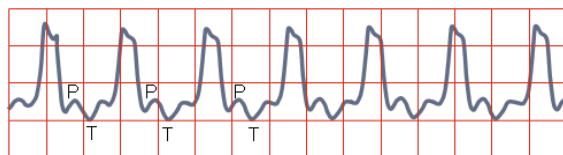
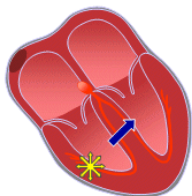
It occurs up to 20% of patients with acute myocardial infarction and occurs frequently during first 2 days with about equal frequency in anterior and inferior wall infarction and probably results from enhanced automaticity of perkinje fibres. Most episodes are of short duration and may terminate abruptly, slow gradually before termination or be overdriven by acceleration of basic cardiac rhythm. Variation in rate is common.

Accelerated idioventricular rhythms are thought not to affect prognosis in acute myocardial infarction and so most are left untreated except in rare patients with clear-cut haemodynamic compromise.

3) Ventricular Tachycardia

VENTRICULAR TACHYCARDIA

Impulses originate at ventricular pacemaker



Wide ventricular complexes. Rate > 120/min

It is most frequently encountered life threatening arrhythmia. Its prompt recognition and acute treatment is critical.

It is defined as series of three or more ventricular complexes occurring at a rate of 100 - 250 beats/min, where the origin of activation is within ventricle.

Typically QRS complexes are wide (usually > 120 milliseconds).

Also other types classified depending upon duration of ventricular tachycardia.

Sustained Ventricular tachycardia - refers to similar above described rhythm but lasting longer than 30 seconds or cause haemodynamic compromise that requires intervention.

Or

Non-sustained Ventricular tachycardia - lasting < 30 seconds without haemodynamic compromise.

Electrocardiogram appearance may be monomorphic or polymorphic. This may be important because former is more likely to be due to a myocardial scar and requires aggressive strategies to prevent its recurrence and later may be more responsive to measures directed against ischaemia.

Occurs up to 67% of patients in acute myocardial infarction in first 12 hours and hospital mortality with sustained ventricular tachycardia during first 48 hours following acute myocardial infarction is about 20%.

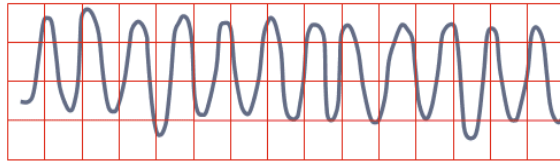
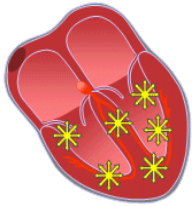
More common in patients, with transmural infarction and left ventricular dysfunction, in settings of acute anterior wall myocardial infarction. It again emphasizes the early monitoring of patients is beneficial to lower mortality in CCU.

One of important differentiating point already discussed was rarity of 'R' on T' types of Ventricular premature beats - its peak occurrence was significantly reduced in ventricular tachycardia.

4) Ventricular Fibrillation

VENTRICULAR FIBRILLATION

Chaotic ventricular depolarization



Rapid, wide irregular ventricular complexes

Ventricular fibrillation is the result of rapid, repetitive activation of ventricles from multiple coalescing and fractionating wave fronts of depolarization. It is associated with disorganized mechanical contraction, haemodynamic collapse and sudden death.

Electrocardiogram reveals irregular and rapid oscillations (250-400 beats/min) of highly variable amplitude without uniquely identifiable QRS complexes or T waves. The combination of metabolic and electrolyte abnormalities, as well as neurophysiological and neurohumoral changes on the susceptible ischaemic myocardium is evolving as a fundamental electrophysiological concept for the mechanism of initiation of potentially lethal arrhythmias like ventricular fibrillation and ventricular tachycardia.

This arrhythmia is the commonest remediable cause of death in acute myocardial infarction and therefore emphasizes prompt treatment.

It occurs in three settings in hospitalized patients as below:

a) **Primary Ventricular Fibrillation** occurs suddenly and unexpectedly in patients with no or few signs or symptoms of left ventricular failure.

Primary ventricular fibrillation occurred in up to 10% of patients' hospitalized patients with acute myocardial infarction. Approximately 60% of episodes occur within 4 hours and 80% within 12 hours of onset of symptoms.

b) **Secondary Ventricular Fibrillation.** On the other hand, it is often the final event of a progressive downhill course with left ventricular failure and cardiogenic shock.

c) **Late Ventricular Fibrillation.** So called late as it develops more than 48 hours following acute myocardial infarction and frequently but not exclusively occurs in patients with large infarcts and ventricular dysfunction.

Other than above mentioned the group of patients who can still develop ventricular fibrillation are with intraventricular conduction defects and anterior wall infarcts with associated other supraventricular arrhythmias (which are discussed further). Also patients with right ventricular infarction who require ventricular pacing are at higher risk for developing late in hospital ventricular fibrillation.

Outcome Prognosis

Effect of ventricular fibrillation on prognosis continues to be debated.

The MILIS study suggested that it does not have an adverse effect on hospital mortality whereas first GISSI trial suggested excess in hospital mortality due to primary ventricular fibrillation.

On the other hand secondary ventricular fibrillation entails a very poor prognosis in hospital mortality rate of 40 to 60%

Other studies reported incidences of in hospital mortality are 50% and 54% respectively for primary and secondary ventricular fibrillation.

Approximately 20% of patients with acute myocardial infarction with primary ventricular fibrillation suffer a recurrence and mean time of recurrence is 4 days. Recurrence rate is nearly 50%.

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