CARDIAC ARRHYTHMIAS

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NORMAL CARDIAC RHYTHM

For normal cardiac rhythm, the following points should be considered:

- Heart rate should be 60 – 100 beats per minute.
- Every heart beat should be originating from SA node.
- All electrical impulses should propagate through normal conduction pathway.
- Cardiac impulses should pass with normal velocity
  In atrium impulses pass with moderate speed (1m/s)
  In AV node, impulses pass with very slow speed
  In purkinje fibers, impulses travel with fast speed

CARDIAC ARRHYTHMIAS

An irregularity in electrical system of heart is called arrhythmia, or heart rhythm disorder.

BRADY ARRHYTHMIA / BRADYCARDIA

When rate becomes less than 60 beats per minute, it is called brady arrhythmia.

Mild Brady Arrhythmia → Heart rate between 40 – 60 beats per minute

Moderate Brady Arrhytmia → Heart rate between 20 – 40 beats per minute

Severe Brady Arrhythmia → Heart rate less than 20 beats per minute

TACHY ARRHYTHMIA / TACHYCARDIA

When heart rate becomes more than 100 beats per minute, it is called tachy arrhythmia.

Simpla cardiac tachy arrhythmia → Heart rate between 100 – 150 beats per minute

Paroxysmal arrhythmia → Heart rate between 150 – 250 beats per minute

FLUTTERS

Heart rate between 250 – 350 beats per minute

The flutters may be in atrium as atrial flutters or in ventricles as ventricular flutters

FIBRILLATION

Heart rate above 350 beats per minute.

ARRHYTHMIAS IS DIFFERENT HEART LOCATIONS

Sinus Arrhythmias – Abnormal rhythms coming from SA node

Atrial Arrhythmias – Abnormal rhythms which are due to abnormal activity within the atrial myocardial tissue

Junctional or Nodal Arrhythmias – Abnormal rhythms related with AV node

Ventricular Arrhythmias – Abnormal rhythms originating due to abnormality of ventricles

The sinus, atrial and junctional tachy Arrhythmias are called **Supra ventricular Tachy Arrhythmias**.

MECHANISMS OF CARDIAC ARRHYTHMIAS

1. Increased automaticity
2. Triggered automaticity
3. Re-entry phenomenon (Circus movement)

INCREASED AUTOMATICITY

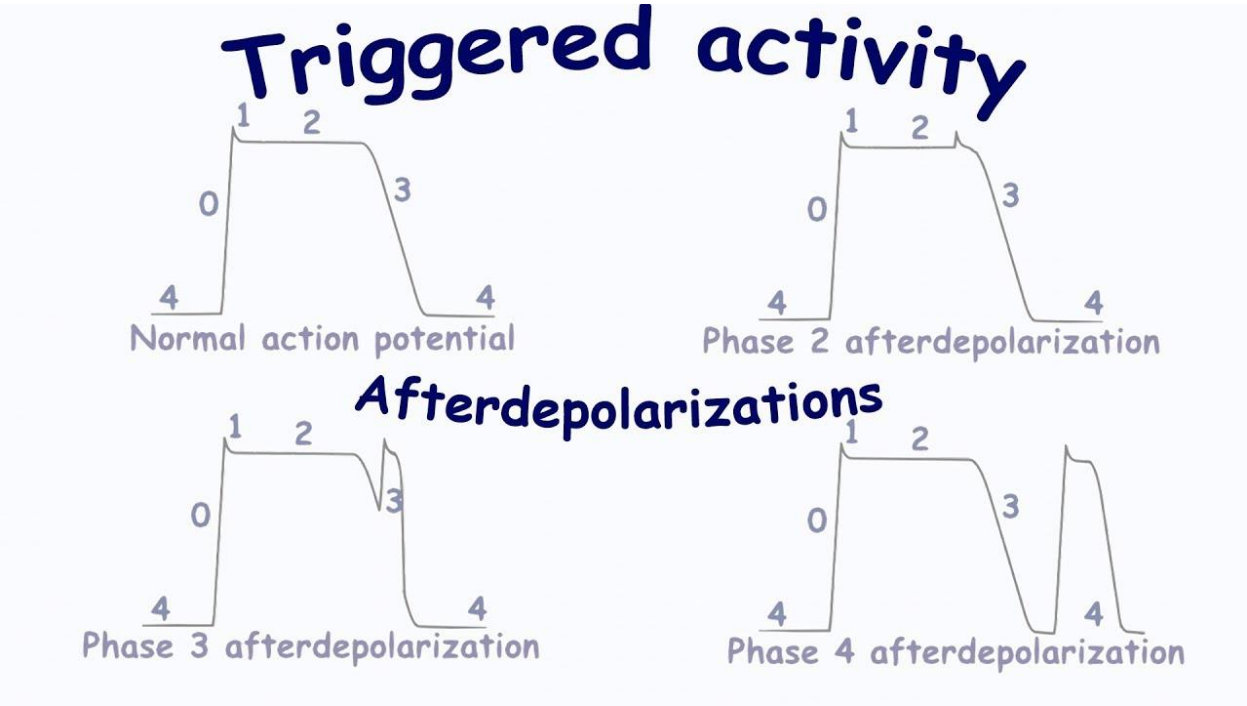
Enhanced normal automaticity accounts for the occurrence of sinus tachycardia, while abnormal automaticity may result in various atrial or ventricular arrhythmias

The automaticity in the sinoatrial node increases during physical exercise. The increased automaticity is a normal reaction since the cardiac output must increase during exercise. This is an example of normal (physiological) increase in automaticity.

TRIGGERED AUTOMATICITY

In triggered automaticity, after the spontaneous depolarization, there is triggered depolarization. This triggered depolarization may be early or late and called as **early afterdepolarization** (EAD) or delayed afterdepolarization (DAD) respectively.

Such tachy arrhythmias produced are called Triggered automaticity induced tachy arrhythmias.



Early afterdepolarizations (EADs) occur with abnormal depolarization during phase 2 or phase 3.

Delayed afterdepolarizations (DADs) begin during phase 4, after repolarization is completed but before another action potential would normally occur via the normal conduction systems of the heart. They are due to elevated cytosolic calcium concentrations, classically seen with digoxin toxicity. Delayed afterdepolarization is also seen in myocardial infarction.

The mechanisms which can produce delayed afterdepolarization include ischemia or injured ventricular cells or loading of cations e.g. catecholamines into these cells. In such cases the cationic load will cause fluctuations in resting membrane potential and when such fluctuating potentials touches the threshold, it produces unwanted triggered autonomic events called as triggered tachy arrhythmias.

RE-ENTRY (CIRCUS MOVEMENT)

The re-entrant tachycardia arises from circular electrical pathways, often initiated by a blocked impulse.

Re-entrant tachycardia is defined as continuous repetitive propagation of an excitatory wave, traveling in a circular path, returning to its site of origin to reactivate that site.

Normally, impulses propagate in synchronized manners. But here one impulse re-enters and re-excites areas of heart more than once.

SINUS ARRHYTHMIAS AND ECG PATTERNS

1. PHYSIOLOGICAL SINUS ARRHYTHMIAS

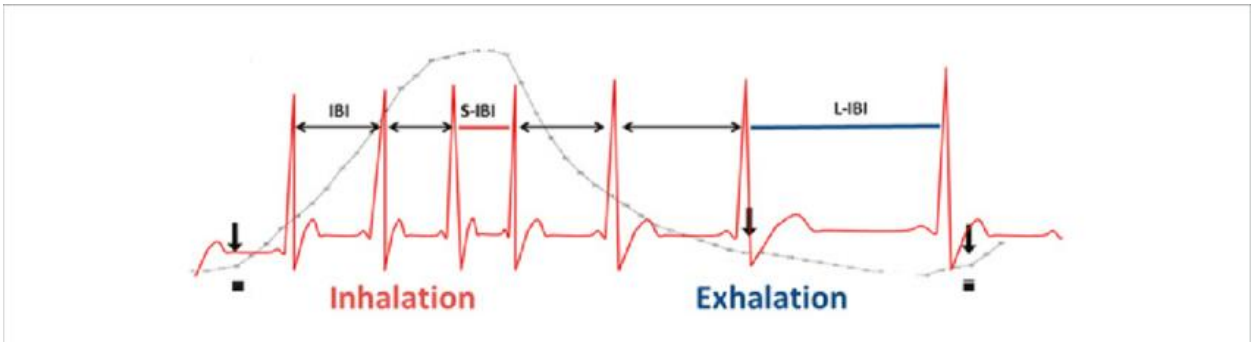
Heart is under the influence of autonomic nervous system and one of the major parasympathetic outflow is vagus nerve that controls the automaticity of SA node and AV node.

Vagus nerve has inhibitory effect on SA node. Hence whenever vagus nerve is stimulated, SA node is inhibited and heart rate goes down.

Vagus activity fluctuates during respiratory cycle. During inspiratory phase, vagus nerve is inhibited and heart rate goes slightly up. During expiratory phase, vagus nerve is stimulated and heart rate goes slightly down.

Due to any reason if vagus nerve is not functional e.g. in autonomic neuropathy, in such case heart rate will not fluctuate during respiratory cycle. A transplanted heart is not under vagus influence so transplanted heart will not undergo physiological sinus arrhythmia.

ECG PATTERN OF PHYSIOLOGIC SINUS ARRHYTHMIA



During inspiration, R to R distance decrease.

During expiration, R to R distance increases.

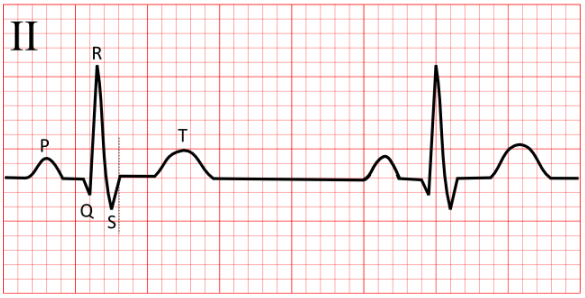
2. SINUS TACHYCARDIA

Increased activity in sinus is seen in exercise, fever or thyrotoxicosis.

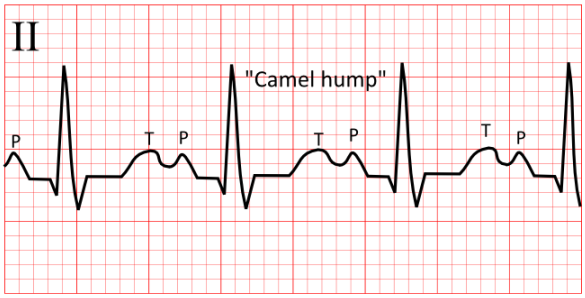
Excessive stimulation of SA node cause SA node to generate depolarization more frequently and heart rate goes up.

In ECG pattern, P waves are generated more frequently in a given time than normal.

Sinus rhythm



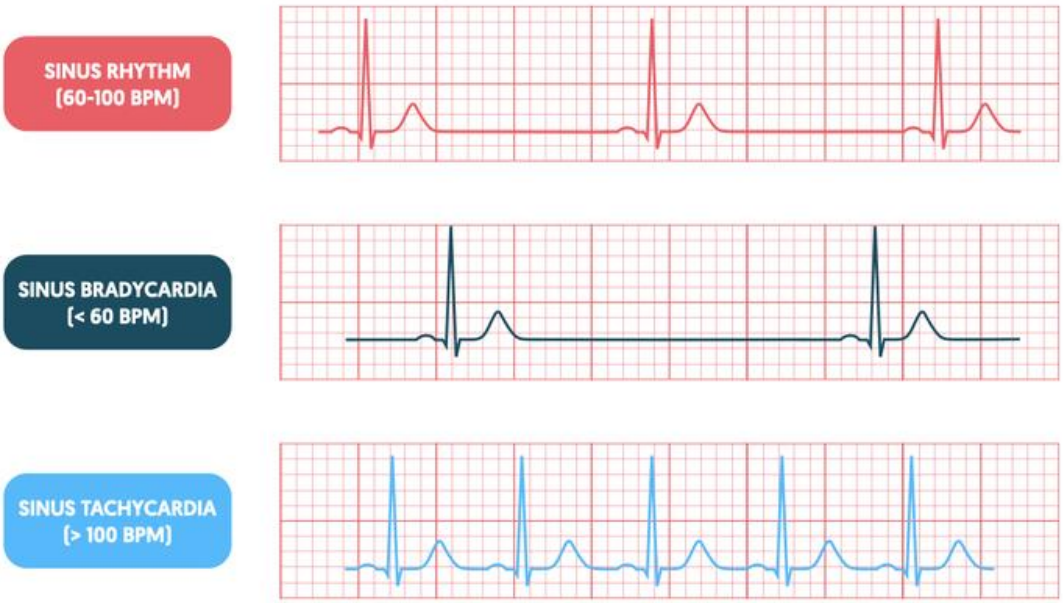
Sinus tachycardia



3. SINUS BRADYCARDIA

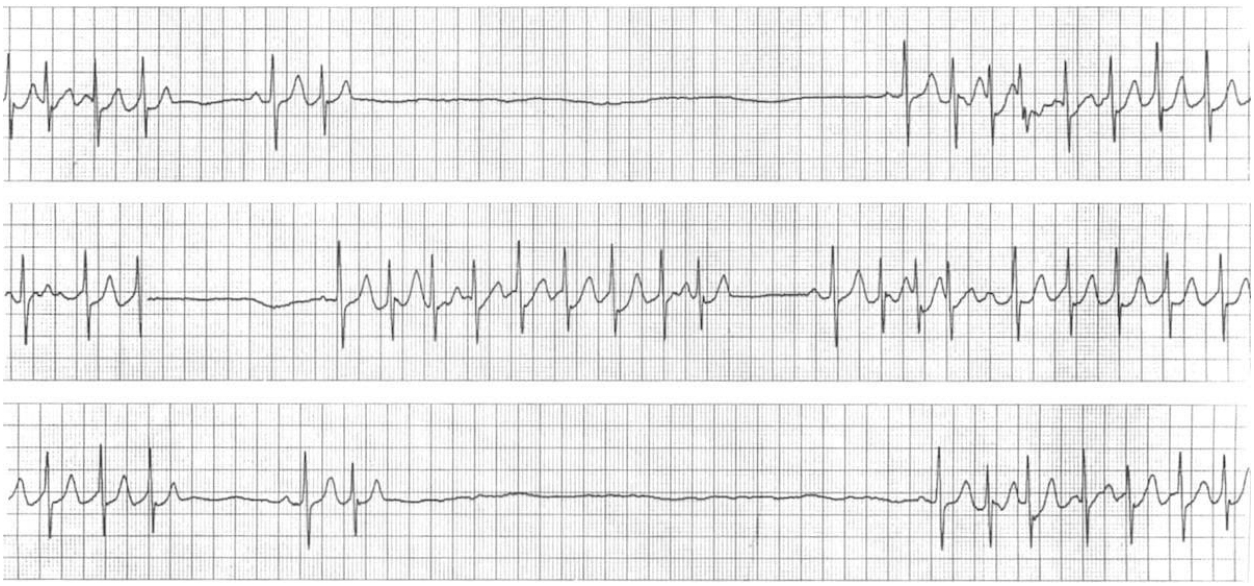
In sinus bradycardia, SA node is inhibited and automaticity of SA node is slowed down. Bradycardia is seen in athletes, hypothyroidism and cholestatic or obstructive jaundice.

In ECG pattern of bradycardia, P waves appear with lesser frequency in a given time. In sinus bradycardia, R to R distance increase.



4. SINUS TACHY BRADY SYNDROME (SICK SINUS SYNDROME)

In this syndrome, sometimes SA node fires faster than normal (tachycardia) and other times fires slower than normal (bradycardia).

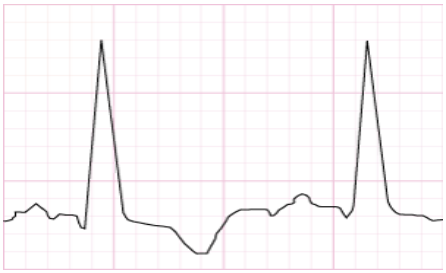


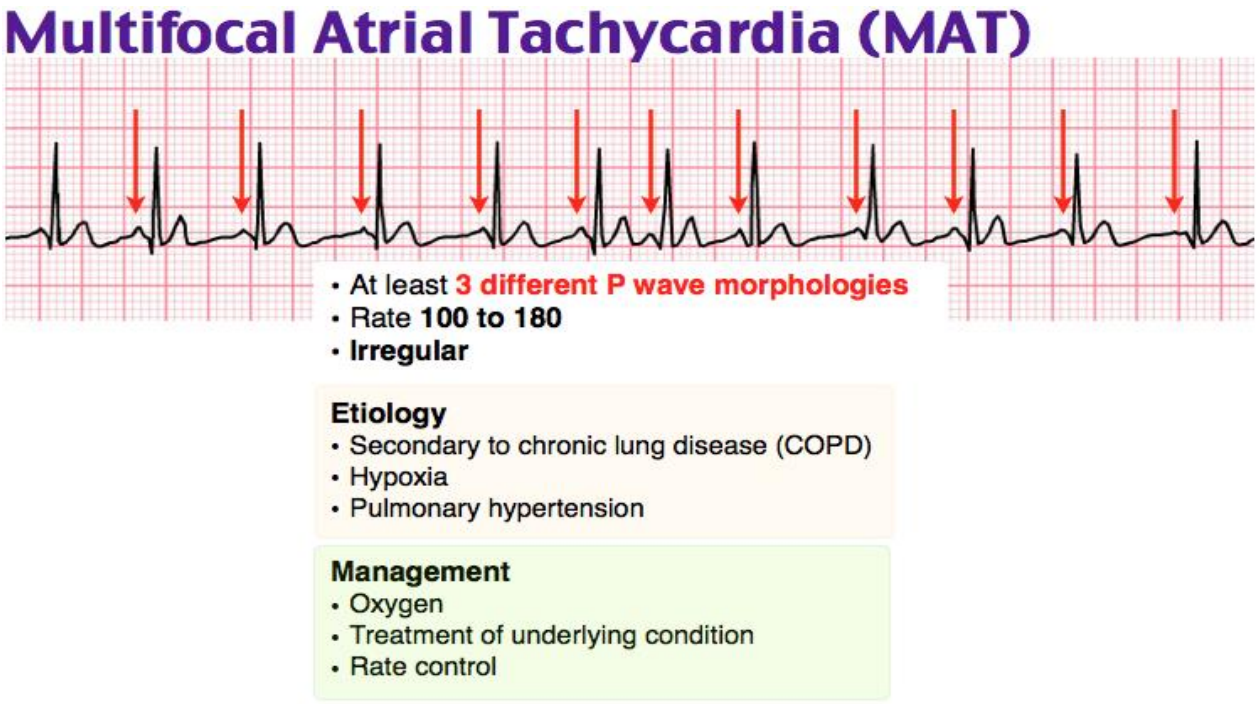
ATRIAL TACHY ARRHYTHMIAS AND ECG PATTERNS

1. ATRIAL TACHYCARDIA

In this case, atria acts as pacemaker and produces electrical activity at the rate of 120 – 250 beats per minute. Hence atrial activity is more as compared to ventricular activity.

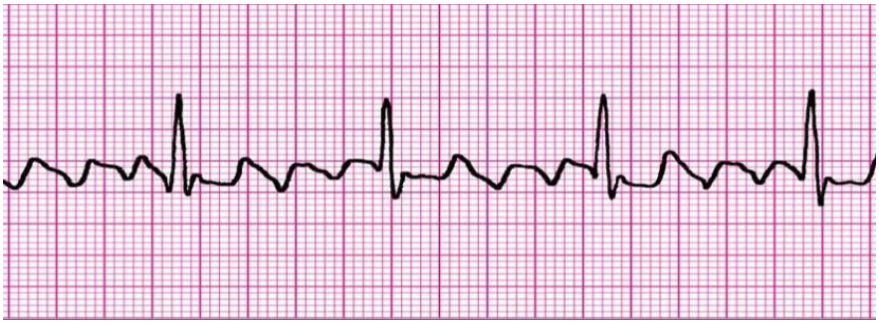
The ECG pattern shows well defined multiple P waves followed by one QRST complex.





2. ATRIAL FLUTTER

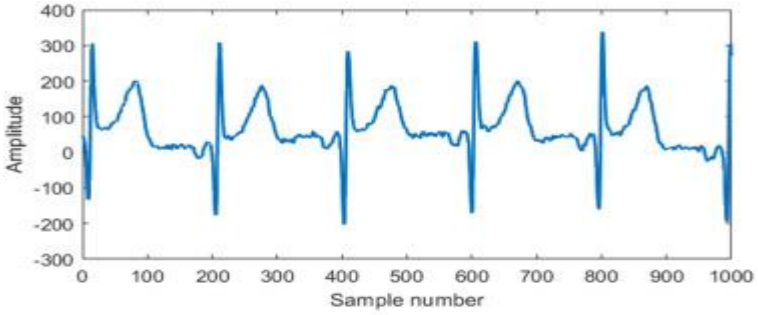
Special types of waves which are not well recognized P waves and are called Flutter waves (represented by 'F') followed by QRST complex.



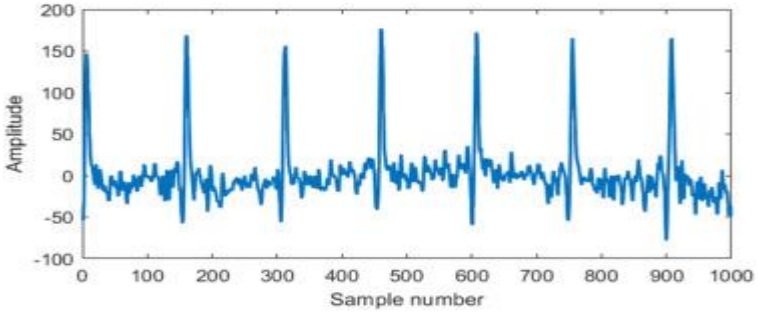
3. ATRIAL FIBRILLATION (AFib)

Very fast rhythm in atrium due to firing of multiple ectopic foci present in atrium.

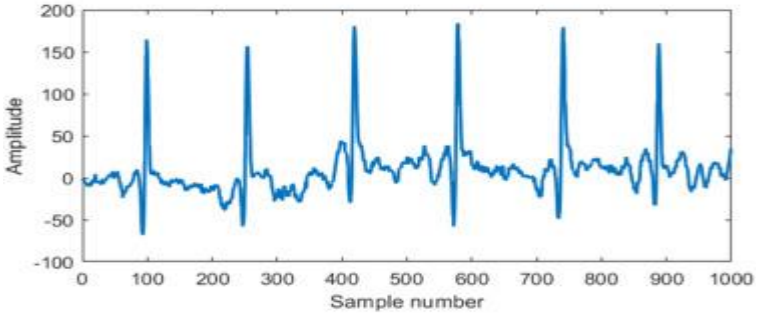
The small multiple waves produced in ECG are called 'f' waves. The smaller fibrillary waves are followed by QRST complex on ECG.



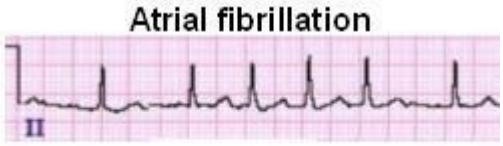
(a) Normal sinus rhythm

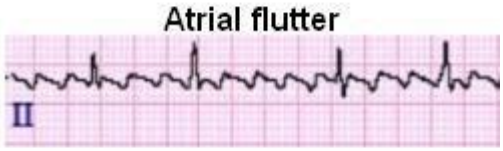


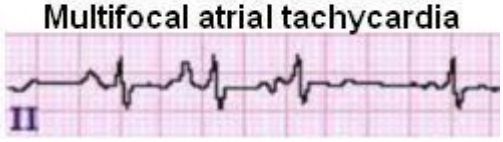
(b) Atrial fibrillation



(c) Atrial flutter







When a patient is having tachy arrhythmia, it should be made sure that these tachy arrhythmias should not transmit rapidly to the ventricles as it can in turn cause, ventricular tachy arrhythmia which may be life-threatening.

To manage such patients, AV node is slowed down which will allow lesser impulse to transfer from atria to ventricles. Drugs which slow AV nodes include:

1. Calcium channel blockers
2. Beta blockers
3. Digitalis which is a positive ionotropic drug used in cardiac failure. Digitalis can also increase vagus activity which in turn slows down AV nodal activity.

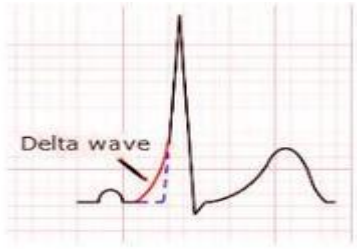
JUNCTIONAL TACHYCARDIA

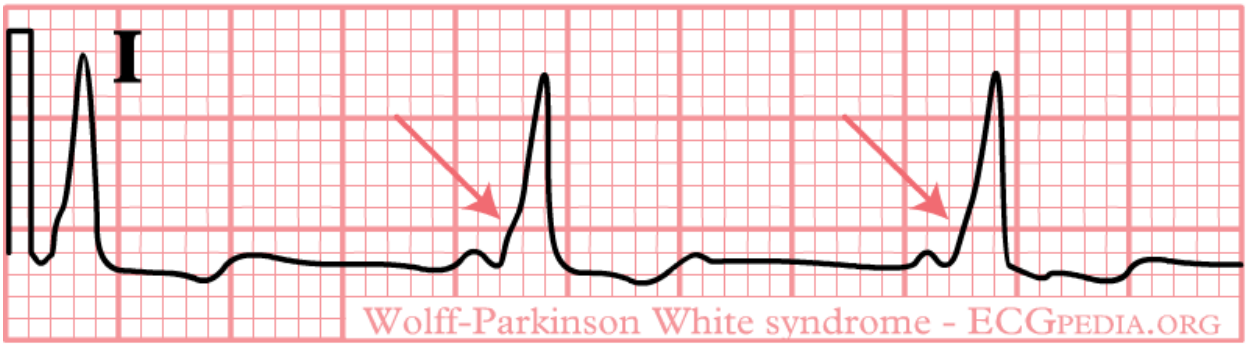
1. WOLF PARKINSON WHITE SYNDROME (WPW)

WPW is an electrical abnormality in the heart that may be associated with supraventricular tachycardia. In people with WPW, along with normal conduction pathway, an extra pathway called **Bundle of Kent** is present. This accessory pathway communicates between atria and ventricles. This accessory pathway does not share the rate-slowing properties of the AV node, and may conduct electrical activity at a significant higher rate than AV node.

Normally, patients with WPW can have a normal heart rate. When current is passing through some area, that area is refractory for re-entry i.e. resistant to the phenomenon of re-entry. In normal conditions when SA node fires, SA nodal current goes to atrial current and atria gives impulse to bundle of Kent as well as impulse to AV node and both these impulses cannot re-enter into each other's area.

When some irritable focus is developed in atria in patients with WPW due to consumption of caffeine or exercise or if the patient becomes emotionally excited, dangerous junctional tachycardia is produced in such patients. The current conduct through the pathway (AV node or Bundle of Kent), which is near to this irritable focus. In such case the current is conducted only through one pathway and not through the other. The impulse going through one pathway may re-enter from the other pathway and by the time it reaches back to the initial point, it is ready to re-excite that area (as it is out of refractory period by the time) and re-entry begins. This circus movement causes junctional tachycardia and ventricles beat every 0.4s or 0.3s.





2. INTRA-NODAL RE-ENTRANT PHENOMENON

When there is a problem with AV node conduction velocity such that one pathway conducts current fast while other pathway conducts current slower. In such case, by chance some of the current from the fast conduction pathway will re-enter into AV node and is called intra-nodal re-entrant phenomenon.

If this re-entrant current is completing cycle every 0.3s, it will shoot down current every 0.3s to the ventricles and heart rate will increase to about 180 – 200.

The classical method to control junctional tachycardia is to massage carotid sinus of the patient on one side. This will stimulate strong vagus outflow which inhibits AV node and re-entrant phenomenon breaks down and normal sinus rhythm begins.

VENTRICULAR TACHY ARRHYTHMIA

One of the common cause of ventricular tachy arrhythmia is irritable foci in the ventricles.ventricular tachycardia most often occurs when the heart muscle has been damaged and scar tissue creates abnormal electrical pathways in the ventricles.

PREMATURE VENTRICULAR COMPLEX

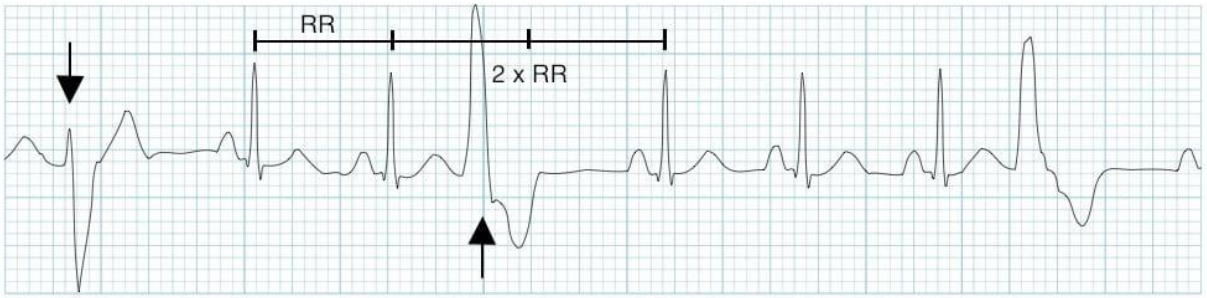
A premature ventricular complex (PVC) is a premature beat arising from an ectopic focus within the ventricles. These are Broad QRS complex (≥ 120 ms) with abnormal morphology and occur Prematurely i.e. occurs earlier than would be expected for the next sinus impulse.

Ectopic firing of a focus within the ventricles bypasses the His-Purkinje system and depolarises the ventricles directly. This disrupts the normal sequence of cardiac activation, leading to asynchronous activation of the two ventricles. The consequent interventricular conduction delay produces QRS complexes with prolonged duration and abnormal morphology.

PVCs may be either:

- **Unifocal** — arising from a single ectopic focus; each PVC is identical
- **Multifocal** — arising from two or more ectopic foci; multiple QRS morphologies

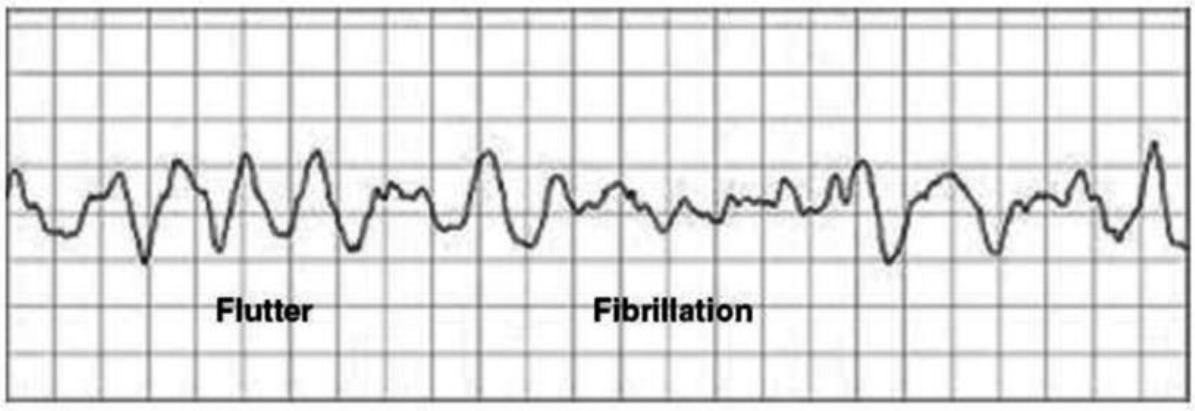
PVCs are a normal electrophysiological phenomenon not usually requiring investigation or treatment. Frequent PVCs may cause palpitations and a sense of the heart "skipping a beat" and may require medical attention.

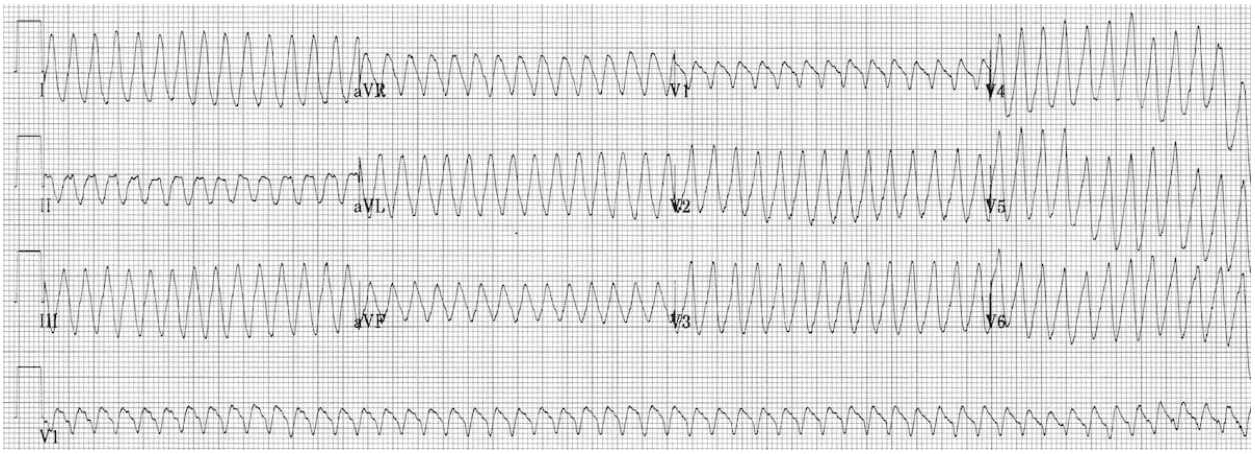


In this ECG pattern, the downward dip shows PVCs. These are PVCs of two different morphologies.

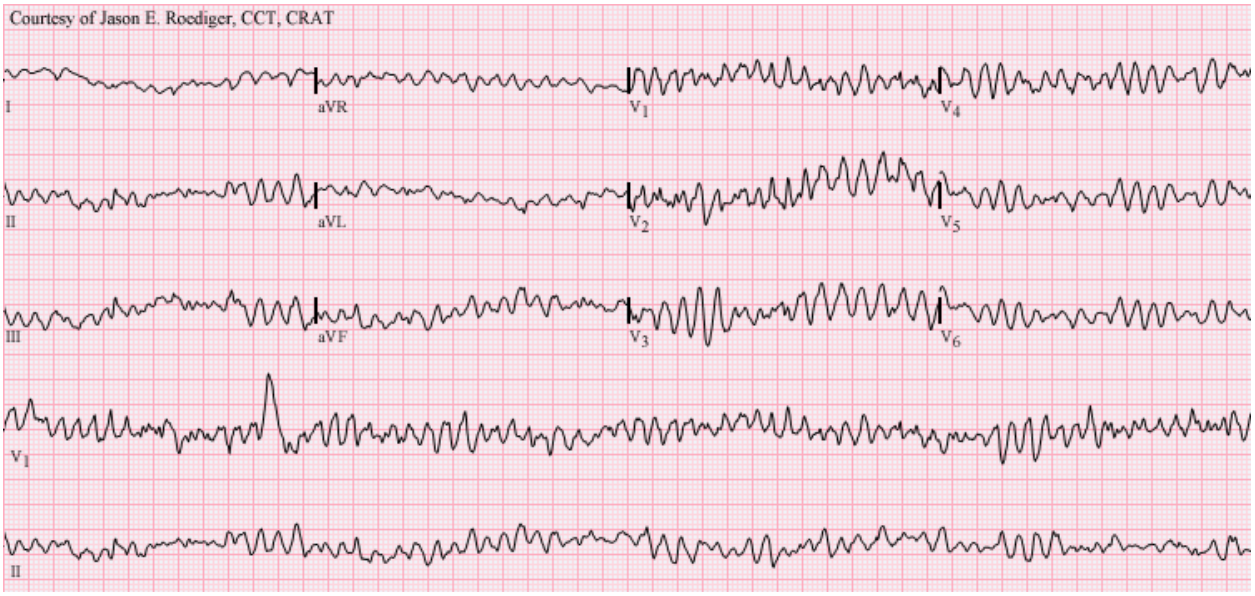
VENTRICULAR FLUTTER AND FIBRILLATION

In ventricular flutter, one or two ectopic foci fires rapidly while in ventricular fibrillation, many ectopic foci fires rapidly. If ventricular fibrillation is not controlled rapidly, it leads to death.





ABOVE IS EKG OF VENTRICULAR FLUTTER



Above is EKG of ventricular fibrillation