Cardiac Arrhythmias

defined as a disturbance of the electrical rhythm of the heart.

heart rate of more than 100/min is called tachycardia.
heart rate of less than 60/min is called bradycardia

* Types

There are five main types of arrhythmias, described by speed of heart rate they cause, and where they begin in the heart.

1. Tachycardia

this fast heart rhythm causes a heart rate of more than 100 beats per minute.
it is usually due to an ↑ed in sympathetic activity.

Sinus tachycardia does not require treatment.

But in some cases β - blockers, CCB, Ivabradine are prescribed.

2. Bradycardia

this slow heart rhythm causes a heart rate of less than 60 beats per min.

if bradycardia is asymptomatic, then no treatment is required

Symptomatic bradycardia occur during MI and can be treated with atropin (0.6 - 1.2 mg).
Patient with persistent symptoms of sinus bradycardia should be considered for pacemaker implantation.

3. Premature heartbeat

a premature or extra, beat is common, usually harmless type of arrhythmia that typically does not cause symptoms.

People who experience an occasional extra heart beat do not need treatment.

4. Supraventricular Tachycardia
   - Supra: from above the ventricles
   - Tachycardia: fast heart rate

∴ comes from atria

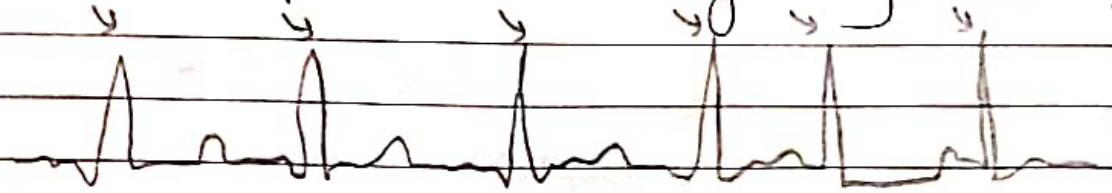
* most common cause of the SVT is when electrical signal re-entering atria from ventricles
once signal is back in atria it travels back to AV node causing another ventricular contraction
↓
Cause self-perpetuating electrical loop (without an end point)

* SVT causes narrow complex tachycardia
  - duration of QRS complex is < 0.12 sec (3 sq)
  - 4 differentials of NCT: sinus tach, SVT, atrial fibrillation, atrial flutter

- Types of SVT depending on the source of electrical signals.

a. Atrial fibrillation :

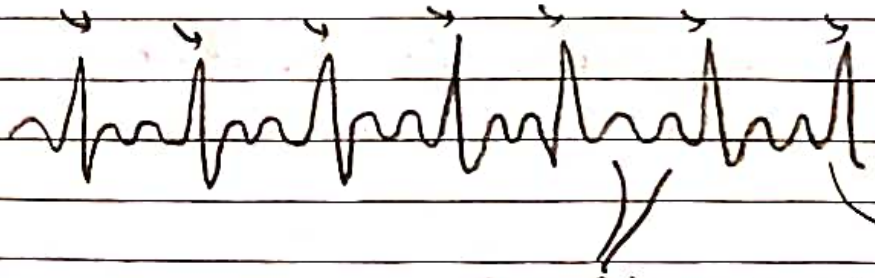
QRS complex → irregularly irregular



Commonly, AF is classified as paroxysmal, persistent or permanent.

b. Atrial flutter :

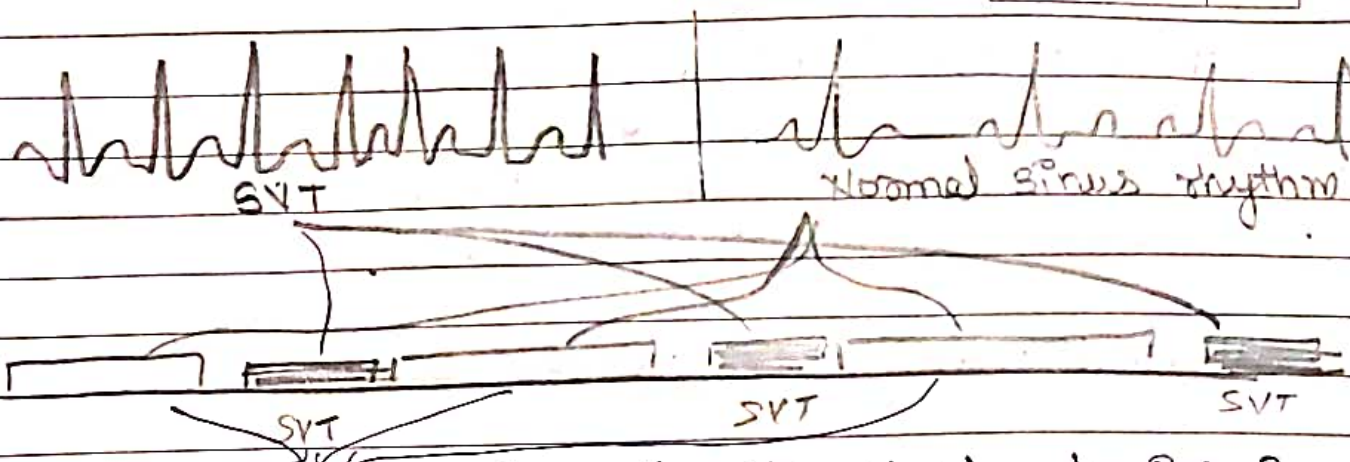
300 bpm, sawtooth pattern
QRS regular.



2 atrial contraction : 1 ventricular contraction

c. Paroxysmal supraventricular tachycardia.

It describes a situation in which SVT occurs in same patient over time in between which there are period of normal rhythms.

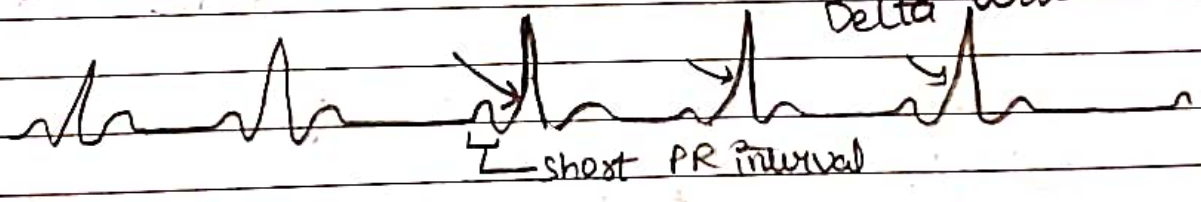


Rapid, regular heartbeat that begins & ends suddenly

d. Atrioventricular Re-entrant tachycardia
(Wolf - parkinson white)

Accessory pathway, refers to an additional electrical pathway betⁿ atria and ventricals.

Additional pathway from ventricals



5. Ventricular arrhythmias:-

Tachycardias that begin in lower chambers of the heart.

a. Ventricular tachycardia:

Rapid, regular heartb that can last for just few sec.
this increases risk of ventricular fibrillation.
Iv amiodarone may be given, IV lido cain for Left V fu
β-blockers are also effective

b) Ventricular fibrillation:

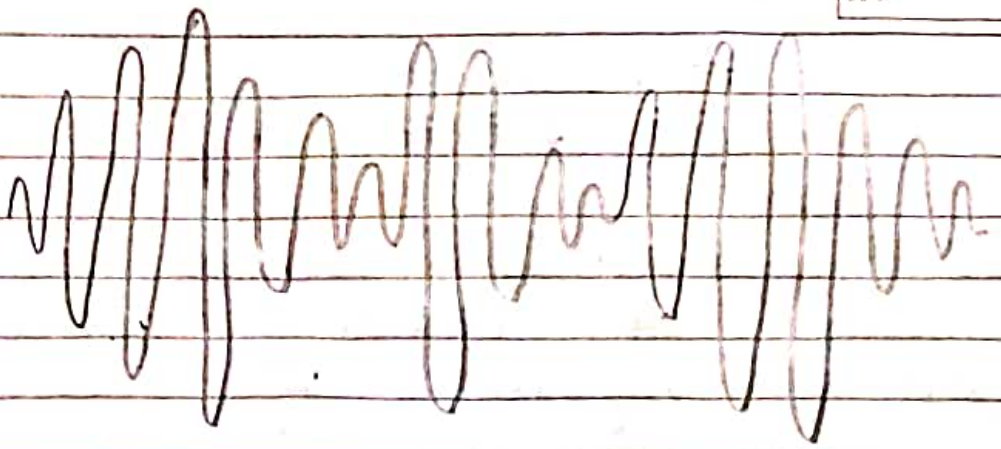
Rapid, irregular hb that causes ventricles to quiver ineffectively instead of pumping blood.
This can lead to cardiac arrest.

0 – regularity 

No P wave no PR interval No QRS complex

c) Torsades de pointes

Torsade de pointes is a peculiar coarse, wefred form of very raped ventricular rhythm.



Treatment - Magnesium, withdrawal of offending agent & Maintain electrolytic balance

Drugs & Toxin causing TdP:

- Procainamide, Quinidine (anti-arrhythmic)
- Erythromycin, ofloxacin (anti-biotic)
- Fluoxetine, Haloperidol, Lithium
  (anti-depp) (anti-psyco) (bipolar disorder)
  Tricyclic anti depressant.
- Organophosphate insecticides.

Pathogenesis

Cardiac arrhythmia usually occurs as result of pathology affecting the conduction system of heart.

Cardiac cycle is normally initiated by an electrical discharge from the SA node. Atria and ventricles then are activated sequentially, as electrical depolarisation passes through specialized conducting tissues.

Three major underlying mechanisms have been identified:
a. Increased automaticity
b. Re-entry
c. Triggered activity

a. Increased Automaticity:

↑sed automaticity occur in cells that normally have spontaneous diastolic depolarization. ie., — the sinus and AV node and His-Purkinje sys.

↳ This automatic behaviour may occur in sites that lack spontaneous pacemaker activity
Eg: depolarization of ventricular cells (eg by ischemia) may produce

such "abnormal" automaticity.

b. Re-entry
Tachycardia is initiated by an ectopic (premature) beat and sustained by a re-entry circuit
Re-entry occurs when there are two anatomical pathways with diff conducting properties

c. Triggered activity
In some conditions, a normal cardiac action potential may be interrupted or followed by an abnormal depolarization.
↓
If this abnormal depolarization reaches threshold, it may, in turn give rise to secondary upstroke and create abnormal rhythms.
↳ (Arrhythmias may be supraventricular (Sinus, atrial or junctional) or ventricular in origin.)

Bradycardia may be due to reduced automaticity of the SA node or abnormalities of conduction through AV node.
If the sinus node becomes slow, any part of the conducting sys may play the role of pacemaker. This is known as escape rhythm and may arise from AV node or His bundle (junctional rhythm) or in Purkinje (idioventricular rhythm)

Anti- Arrhythmic agents

Class I

Na^+ channel blockers
(membrane stabilizing agents)

1A
moderately decrease phase 0 depolarn
Quinidine
Procainamide
Disopyramide

1B
minimal effect on phase 0 depn
Lidocaine
Mexiletine

1C
marked decrease in phase 0 depn
Propafenone
Flecainide

Class II β- adrenergic blockers
(Anti- adrenergic agents)

Propranolol
Esmolol

Class III

drugs that prolong duration of action potential

Amiodarone
Dronedarone
Sotalol
Dofetilide
Ibutilide

Class IV

Calcium channel blockers

Verapamil
Diltiazem

Drugs for Proximal supraventricular tachycardia (PSVT)

Adenosine
Digoxin
Atropin
Ivabradine

Pharmacology of Class I:

The primary action of drugs in this class is to limit the conductance of Na^+ across the membrane — a local anaesthetic action.

Class Ia

Drugs — Quinidine
Procainamide
Disopyramide

*Quinidine:

(a) Mechanism of action:

It blocks Na^+ channels in the open state → decreases automaticity
decreases excitability
decreases conduction
decreases rate of phase 0 depolarization

It blocks K^+ channels →
increases duration of action potential
blocks re-entry of impulse

ECG : It prolongs QRS complex and QT interval.

(b) Pharmacokinetics

Absorbed in GI tract
Bound to plasma proteins
Metabolized in liver
Excreated 20% unchanged in urine

(c) Adverse effects

- Diarrhoea
- Fall in BP
- Torsades de pointes
- Hepatitis & fever (rare)
- Procaine syndrome — 'Cinchonism'
  ↓ causes
  deafness, headache, blurring of vision, confusion, psychoses.

(d) uses

It has a broad spectrum of anti-arrhythmic activity.

useful in maintaining normal sinus rhythm
↓
in atrial fibrillation / atrial flutter
occasionally in ventricular tachycardia

(e) Dose 100 - 200mg TDS

Class 1b

Drugs — Lidocaine
Mexiletine

★ Lidocaine :

(a) Mechanism of action

Lidocaine
↓
diffuses through cell memb
↓
Binds to voltage gated Na^+ channels
↓
Blocks Na^+ entry into the cells
↓
Raises the threshold for conduction
↓
No generation & conduction of action potential
↓
Local anesthesia

(b) Pharmacokinetics

Orally - Inactive
IV - last for 10 - 20 min
Metabolized - liver (blood)
Excreated - urine

$t_{1/2}$ - 8 min

(c) Adverse effect

- Main toxicity is related to neurological effect
  Drowsiness
  Nausea
  Blurred vision

- Cardiac depression } High dose
- hypotension

(d) uses

It is safe if given by slow IV inj.

Given in MI to prevent ventricular fibrillation

Generally use in emergency treatment of ventricular tachycardia.

Not used in atrial arrhythmia.

(e) Dose

- 20 - 40 mg IV every 10 - 20 min
- 1 - 3 mg/min IV

Class 1c

Drugs — Propafenone
Flecainide

(a) Mechanism of action

Propafenone

↓

Slows influx of Na^+ in cardiac cells

↓

This causes depress impulse transmission

↓

Has very good effect on His Purkinje

↓

Helps to decrease WPW syndrome

(b) Pharmacokinetics

Absorbed - orally
Metabolism - first pass
Excreted - through urine

$t_{1/2}$ - Variable

(c) Adverse effects

Constipation
Bradycardia - bronchospasm
Blurred vision
Nausea
Vomiting
Bitter taste (metallic)
Asthma
Increase risk of sudden death

(d) Uses

Mainly used to maintain sinus rhythm in patient having AF

used for supraventricular tachycardia
also for ventricular arrhythmias

(e) Dose

150 - 300 mg TDS