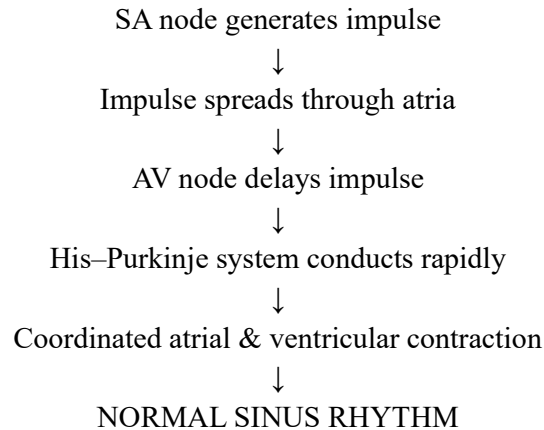


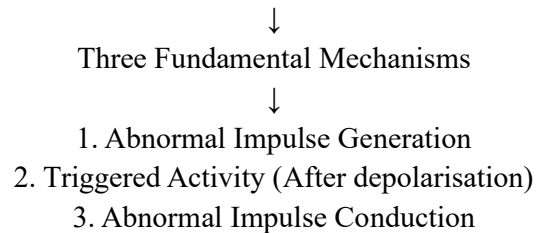
GENESIS OF CARDIAC ARRHYTHMIAS

1. NORMAL HEART RHYTHM (STARTING POINT)



Arrhythmias occur when **impulse generation**, **impulse conduction**, or **both** become abnormal.

DISTURBANCE IN ELECTRICAL ACTIVITY



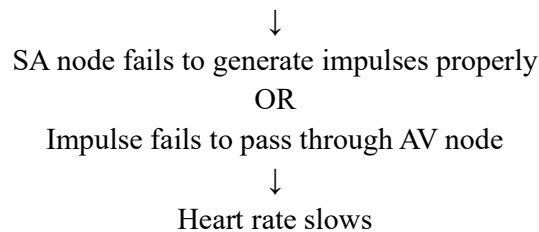
1. ABNORMAL IMPULSE GENERATION : *(Problem at the pacemaker level)*

Normally, **SA node** controls heart rate.

Arrhythmias occur when:

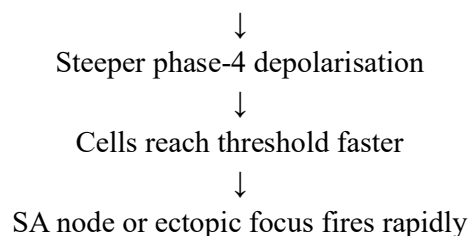
- SA node fires **too slowly**
- SA node fires **too fast**
- Other cells start acting as pacemakers

A. DECREASED AUTOMATICITY → SLOW RHYTHMS ⇒ Sinus bradycardia, AV Block, Asystole → These patients often need **pacemakers**, not drugs.



BRADYARRHYTHMIAS

B. INCREASED AUTOMATICITY → FAST RHYTHMS





TACHYARRHYTHMIAS + Ectopic beats

- **Where ectopic pacemakers arise** from Atria, AV node, Purkinje fibres, Ventricular myocardium
- **Common triggers** : Ischaemia, Hypokalaemia, Catecholamines, Digitalis toxicity

2. TRIGGERED ACTIVITY (*Impulse triggered after a normal beat*) Occurs when normal AP triggers extra abnormal depolarisations.

The problem is **not automaticity**, but **abnormal depolarisations occurring after an action potential**.

Normal action potential



Abnormal calcium influx into cardiac myocyte during or immediately after phase 3 repolarisation



After-depolarisations



Extra (triggered) beat



Arrhythmia (Seld sustained and depends on proceeding AP) Not self-initiated or spontaneous

A. DELAYED AFTER-DEPOLARISATIONS (DAD)

Occurs AFTER repolarisation (Phase 4)



Intracellular Ca^{2+} overload



Extra depolarisation reaches threshold



Triggered tachycardia

- Exacerbated in Digitalis toxicity, High sympathetic activity, Myocardial ischemia
- DADs are **rate-dependent** → worse at fast heart rates.

B. EARLY AFTER-DEPOLARISATIONS (EAD) => Prolong cardiac repolarisation phase

Occurs DURING repolarisation (Phase 2 or 3)



Action potential becomes prolonged



Reactivation of Na^+ / Ca^{2+} channels



Triggered beat



Polymorphic VT (Torsades de Pointes) associated with Prolonged QT → dangerous ventricular arrhythmias.

5. ABNORMAL IMPULSE CONDUCTION: (*Impulse formed normally but travels abnormally*)

Normal impulse generation



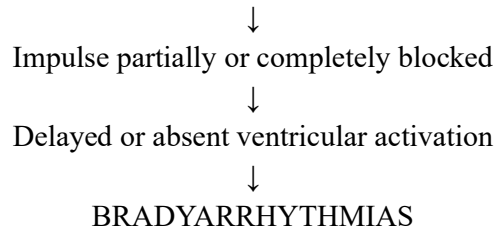
Abnormal impulse propagation



Three mechanisms

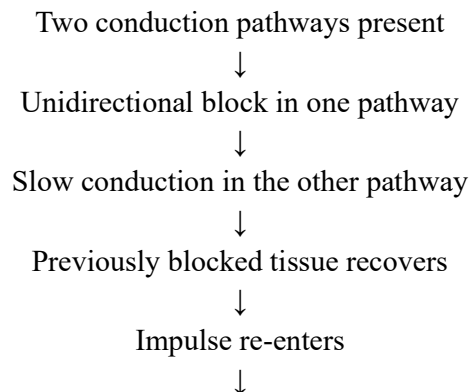
1. Conduction Block
2. Re-entry Phenomenon
3. Accessory tract pathways

A. CONDUCTION BLOCK → result in AV nodal block, Bundle branch block



B. RE-ENTRY PHENOMENON (*Most common mechanism – ~80–90%*) → *Observed in Atrial flutter, VT, AVNRT*

An impulse **keeps circulating in a loop** instead of dying out.



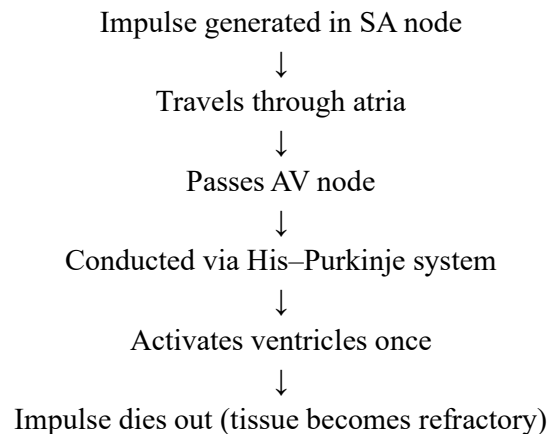
RE-ENTRY PHENOMENON (*Mechanism of Tachyarrhythmias*)

WHAT IS RE-ENTRY? (Core Concept)

Re-entry is a mechanism of arrhythmia in which a single electrical impulse repeatedly circulates in a closed loop, re-exciting the myocardium instead of extinguishing normally.

The impulse goes around in circles and keeps the heart beating fast.

NORMAL IMPULSE CONDUCTION (FOR CONTRAST) : Normal conduction is **one-way and self-terminating**.



CONDITIONS REQUIRED FOR RE-ENTRY : (*All three must be present*)

1. Two conduction pathways
2. Unidirectional block in one pathway

3. Slow conduction in the other pathway

Without **all three**, re-entry cannot occur.

HOW RE-ENTRY DEVELOPS (STEP-BY-STEP)

Step 1: Two Parallel Pathways Exist

- These may be:
 - Two AV nodal pathways (fast and slow)
 - Normal pathway + accessory pathway (Bundle of Kent)
 - Normal myocardium + damaged (ischemic) tissue

Pathway A – fast conduction, long refractory period

Pathway B – slow conduction, short refractory period

Step 2: Premature Impulse Occurs

- A premature atrial or ventricular beat arrives early.
 - Premature impulse reaches pathways
 - ↓
 - Pathway A still refractory → impulse blocked
 - Pathway B excitable → impulse conducts slowly

→ This creates a **unidirectional block**

Step 3: Impulse Travels Slowly Through Alternate Pathway

Impulse moves down slow pathway



Time delay allows Pathway A to recover excitability

Step 4: Retrograde Conduction (Re-entry)

Impulse reaches distal end



Travels backward (retrograde) through recovered Pathway A



Re-excites the original tissue

Step 5: Self-Sustaining Loop

Impulse keeps circulating



Repeated myocardial activation



SUSTAINED TACHYCARDIA

The impulse is now **trapped in a loop**, producing rapid heart rate.

C. ACCESSORY PATHWAYS (WPW SYNDROME)



Extra pathway (Bundle of Kent)



Impulse bypasses AV node



Very rapid ventricular activation



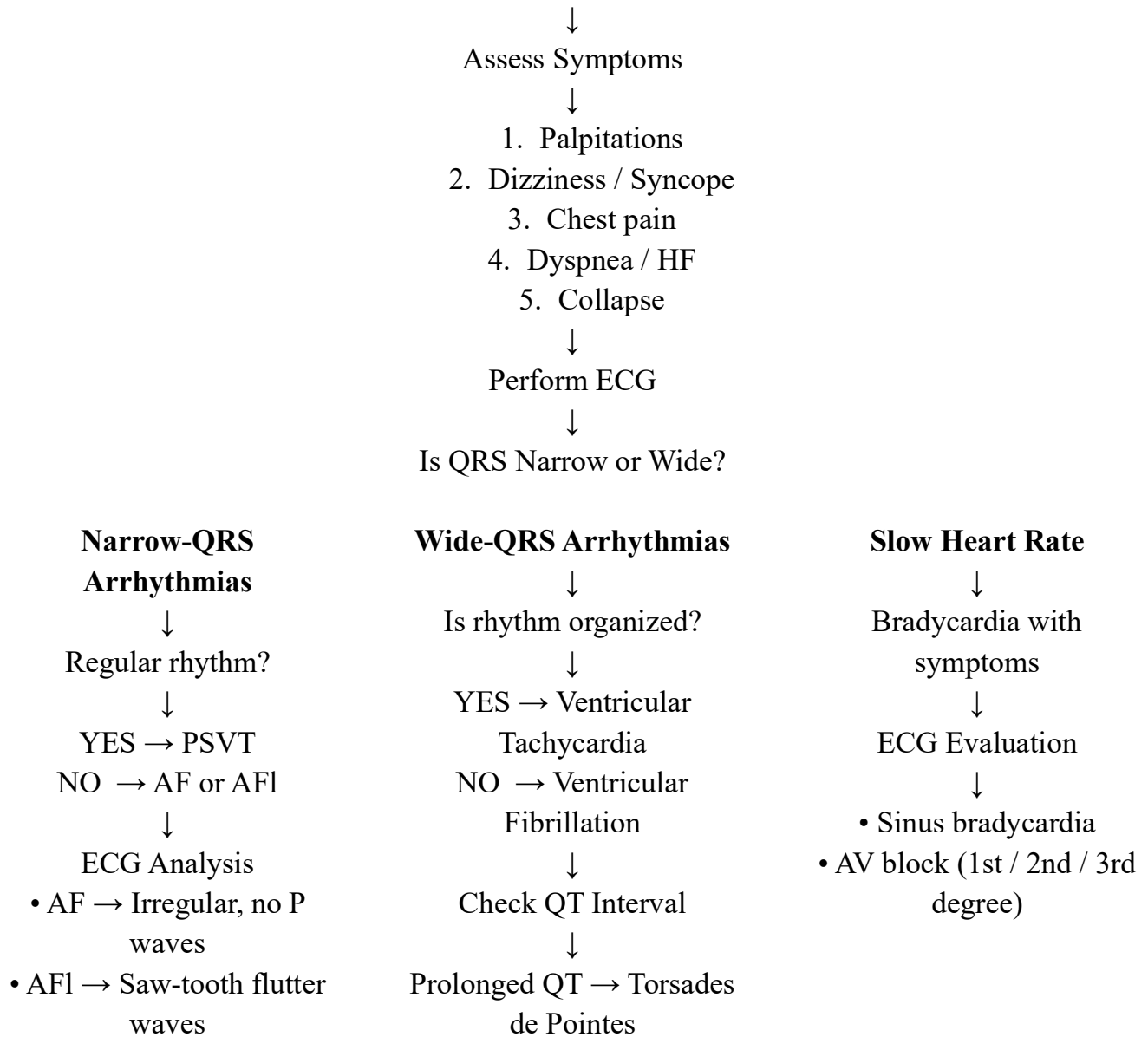
Re-entry circuit



Paroxysmal SVT

Arrhythmia	Clinical Presentation	ECG Features
Supraventricular Tachycardias (SVT – General)	Asymptomatic to palpitations, irregular pulse, dizziness, syncope, chest discomfort, choking/pressure sensation; may precipitate HF	Narrow QRS tachycardia (usually), rapid ventricular rate
Atrial Fibrillation (AF)	Palpitations, dyspnea, fatigue, chest pain; syncope uncommon; risk of embolic stroke due to atrial stasis	Irregularly irregular rhythm, no P waves, variable RR intervals
Atrial Flutter (AFL)	Similar to AF; severe symptoms in 1:1 conduction (≈ 300 bpm); worse in mitral stenosis, cardiomyopathy	Saw-tooth flutter waves, atrial rate ≈ 300 bpm, ventricular rate 150/100/75
Paroxysmal SVT (Re-entry)	Sudden onset & termination of palpitations; chest pressure or neck pounding; syncope if very fast	Regular narrow-QRS tachycardia; P waves hidden in QRS or T wave
Premature Ventricular Contractions (PVCs)	Often asymptomatic; palpitations or “missed beats” sensation	Early wide QRS complex followed by compensatory pause
Ventricular Tachycardia (VT)	Asymptomatic to dizziness, syncope, hypotension, collapse; severity $\uparrow$ with poor LV function	Wide-QRS tachycardia; monomorphic or polymorphic
Torsades de Pointes (TdP)	Dizziness, syncope, sudden death risk	Polymorphic VT with twisting QRS, prolonged QT
Ventricular Fibrillation (VF)	Immediate hemodynamic collapse, syncope, cardiac arrest	Chaotic, disorganized ECG with no identifiable QRS
Bradyarrhythmias	Dizziness, syncope, fatigue, confusion; worsening HF if LV dysfunction	Sinus bradycardia, AV block patterns
AV Block	Fatigue, syncope, hypotension	1st, 2nd, or 3rd-degree AV block on ECG

PATIENT WITH RHYTHM DISTURBANCE



A. Supraventricular Tachyarrhythmias

Supraventricular tachycardias produce a wide spectrum of symptoms, ranging from no symptoms to palpitations and irregular pulse, and in severe cases, dizziness, syncope, chest pain, or worsening heart failure. Many patients describe a choking or pressure sensation in the neck or chest, especially during re-entrant tachycardias due to simultaneous atrial and ventricular contraction.

B. Atrial Fibrillation & Atrial Flutter

AF and AFl usually do not cause syncope or hemodynamic collapse, except in cases of 1:1 atrial flutter or in patients with underlying heart disease. The most important complication of AF is arterial embolization, leading to ischemic stroke due to atrial stasis and thrombus formation.

On ECG:

- AF shows an *irregularly irregular rhythm with absent P waves*.
- AFl shows *organized atrial activity with flutter (saw-tooth) waves*.

C. Paroxysmal Supraventricular Tachycardia (PSVT)

PSVT typically presents as sudden episodes of rapid heart rate that start and stop abruptly. Symptoms include palpitations, dizziness, and chest pressure. In AV nodal re-entry, patients often complain of neck pounding, due to atrial contraction against a closed tricuspid valve. ECG usually shows a regular narrow-QRS tachycardia with hidden P waves.

D. Ventricular Arrhythmias

PVCs

PVCs are usually benign and asymptomatic. Patients feel palpitations due to the post-PVC compensatory pause, not the PVC itself.

Ventricular Tachycardia

VT symptoms depend on heart rate, duration, and LV function. Sustained VT may cause hypotension, syncope, or cardiac arrest, while nonsustained VT may be minimally symptomatic.

Ventricular Fibrillation

VF always results in loss of cardiac output, leading to syncope and sudden cardiac death unless immediate defibrillation is performed.

E. Bradyarrhythmias

Patients with bradyarrhythmias experience symptoms related to low cardiac output, including dizziness, syncope, fatigue, and confusion. If LV dysfunction is present, heart failure symptoms worsen. AV blocks are diagnosed and classified based on ECG findings.

Treatment

Goals:

1. Relieve symptoms
2. Prevent hemodynamic compromise
3. Prevent progression to more dangerous arrhythmias
4. Reduce morbidity and mortality
5. Avoid treatment-related harm (proarrhythmia)

Pharmacological Treatment

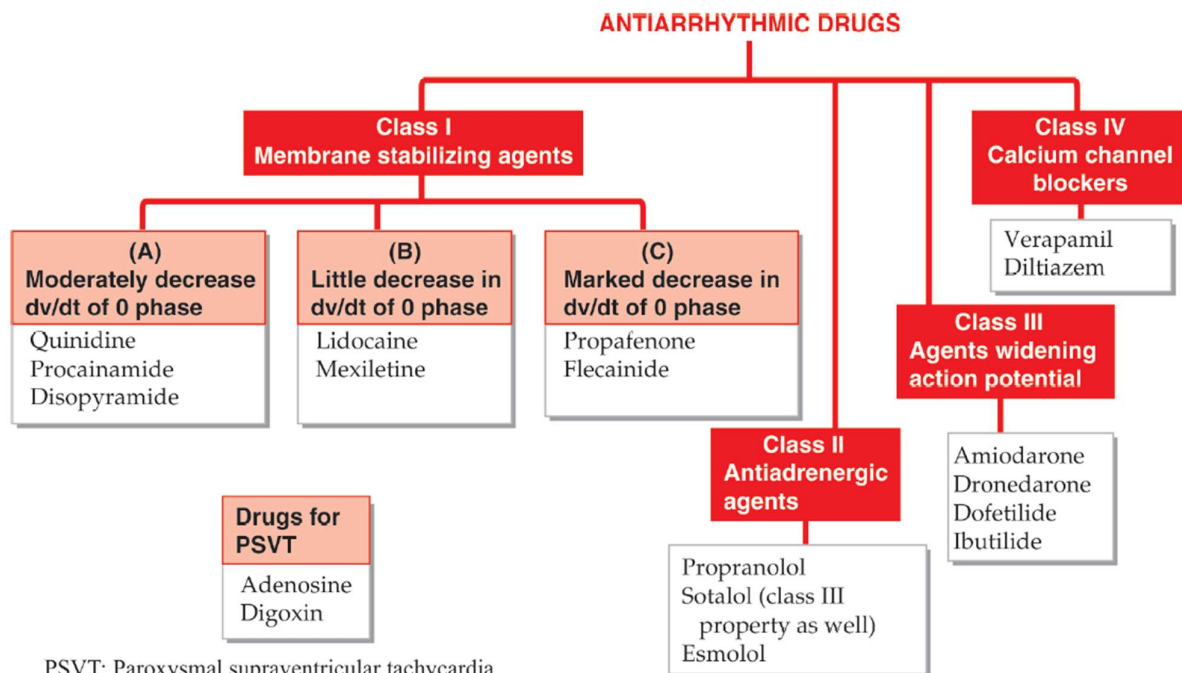
I. CLINICAL (DESCRIPTIVE) CLASSIFICATION

(Based on the type of arrhythmia treated)

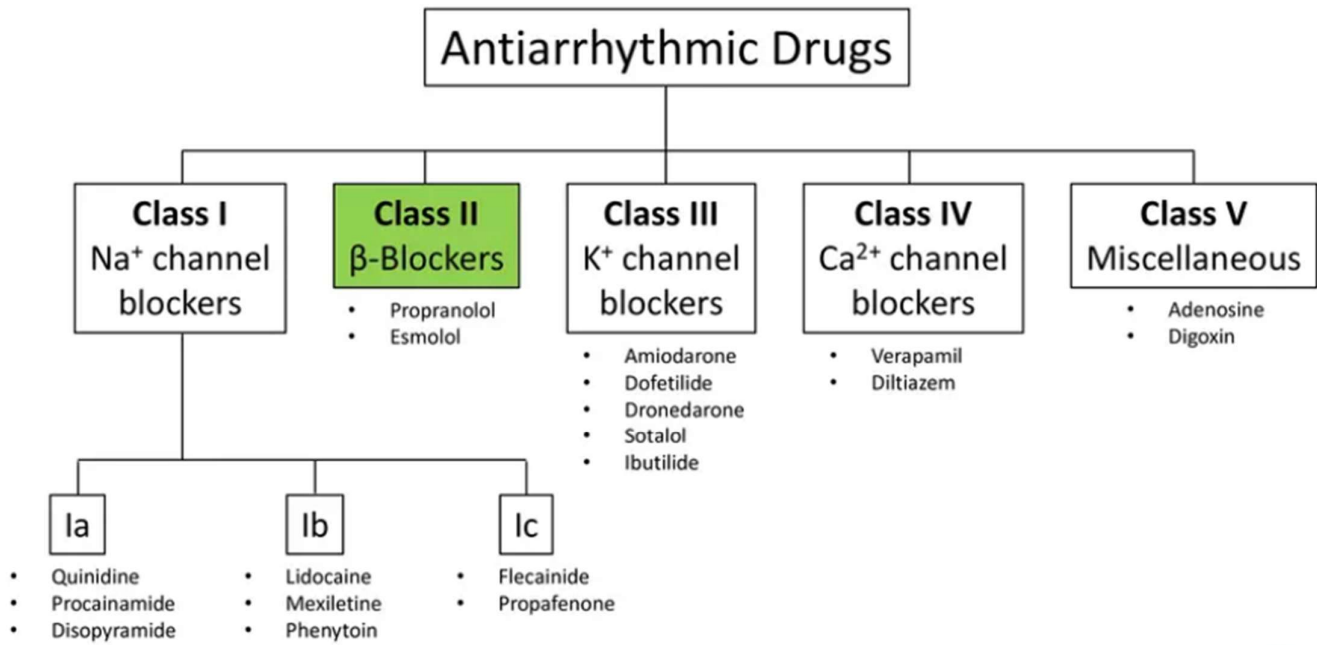
Category	Drugs	Clinical Use
Drugs used for supraventricular arrhythmias	Adenosine, Verapamil, Diltiazem	PSVT, AV nodal–dependent tachycardias, rate control in AF/AFl
Drugs used for ventricular arrhythmias	Lignocaine (Lidocaine), Mexiletine, Bretylium	Ventricular tachycardia, ventricular ectopy
Drugs used for both supraventricular and ventricular arrhythmias	Amiodarone, β -blockers, Disopyramide, Procainamide	AF, AFl, VT, mixed arrhythmias

Type of Arrhythmia	Goals of Treatment
Supraventricular Tachyarrhythmias (PSVT, AF, AFl, atrial tachycardia)	1. Relieve symptoms (palpitations, dizziness, chest discomfort) 2. Control ventricular rate 3. Restore sinus rhythm when appropriate 4. Prevent recurrence of tachyarrhythmia 5. Prevent hemodynamic compromise and ischemia 6. Improve functional status and quality of life
Atrial Fibrillation (AF)	1. Control ventricular rate 2. Prevent thromboembolism (stroke prevention) 3. Restore and maintain sinus rhythm in selected patients 4. Prevent recurrence of AF 5. Improve symptoms and exercise tolerance 6. Reduce risk of heart failure and mortality
Atrial Flutter (AFl)	1. Control ventricular rate 2. Restore sinus rhythm (cardioversion or ablation when appropriate) 3. Prevent recurrence 4. Prevent thromboembolic complications 5. Improve hemodynamics and symptoms
Paroxysmal Supraventricular Tachycardia (PSVT)	1. Terminate acute tachycardia episodes 2. Prevent recurrence of episodes 3. Reduce symptom burden (palpitations, dizziness, neck pressure) 4. Avoid hemodynamic compromise 5. Minimize need for invasive therapy
Ventricular Tachycardia (VT)	1. Terminate acute VT, especially if unstable 2. Prevent degeneration into ventricular fibrillation 3. Prevent sudden cardiac death 4. Reduce recurrence of VT 5. Improve survival and hemodynamic stability 6. Treat underlying causes (ischemia, structural heart disease)
Torsades de Pointes (TdP)	1. Terminate the arrhythmia promptly 2. Correct prolonged QT interval 3. Correct electrolyte abnormalities (especially hypokalemia, hypomagnesemia) 4. Discontinue QT-prolonging drugs 5. Prevent recurrence and sudden death

Ventricular Fibrillation (VF)	1. Immediate restoration of effective cardiac output 2. Prevent sudden cardiac death 3. Correct reversible causes (ischemia, electrolyte imbalance) 4. Prevent recurrence of VF 5. Maintain long-term survival
Bradyarrhythmias (General)(Sinus bradycardia, AV block)	1. Restore adequate heart rate 2. Improve cardiac output and organ perfusion 3. Relieve symptoms of hypotension (dizziness, syncope, fatigue) 4. Prevent syncope and sudden cardiac arrest 5. Prevent worsening of heart failure
Atrioventricular (AV) Block	1. Maintain adequate ventricular rate 2. Prevent syncope and hypotension 3. Improve quality of life 4. Prevent progression to complete heart block 5. Reduce mortality (often via pacing)



Vaughan Williams classification



Class I A		
Drug	Common Side Effects	Serious / Important Effects
Disopyramide	Dry mouth, constipation, urinary retention, blurred vision	TdP, heart failure (HF), ventricular arrhythmias
Procainamide	Hypotension	TdP, worsening HF, ventricular arrhythmias
Quinidine	GI upset (diarrhea, nausea, vomiting), cinchonism (tinnitus, headache)	TdP, hypotension, thrombocytopenia, hemolytic anemia
Class I B		
Lidocaine	Dizziness, sedation, confusion, paresthesia	Seizures, sinus arrest, conduction disturbances
Mexiletine	Dizziness, tremor, nausea, vomiting	Ventricular arrhythmias, conduction disturbances
Class I C		
Flecainide	Dizziness, blurred vision, headache	Worsening HF, ventricular arrhythmias
Propafenone	Dizziness, fatigue, metallic taste, nausea	Bradycardia, AV block, worsening HF
Class III		
Amiodarone	Tremor, insomnia, nausea, photosensitivity	Pulmonary fibrosis, thyroid dysfunction, liver toxicity, bradycardia
Dofetilide	Headache, dizziness	TdP
Dronedarone	Nausea, diarrhea, ↑ serum creatinine	Worsening HF, liver toxicity, TdP (rare)
Ibutilide	Headache	TdP, hypotension
Sotalol	Fatigue, dizziness, bradycardia	TdP, bronchospasm, worsening HF

A. Emergency / Acute (IV) Use
Ventricular Arrhythmias & AF Termination

Drug	Used For	Easy-to-Remember IV Dose
Amiodarone	Pulseless VT/VF	300 mg IV push, then 150 mg if needed → infusion 1 mg/min (6 h) → 0.5 mg/min
	Stable VT (pulse)	150 mg IV over 10 min → infusion
	AF termination	5 mg/kg IV over 30 min → infusion
Lidocaine	VT/VF	1–1.5 mg/kg IV bolus, repeat → max 3 mg/kg, then infusion
Procainamide	AF / Stable VT	15–18 mg/kg IV over 60 min → infusion
Ibutilide	AF termination	1 mg IV over 10 min, repeat once
Sotalol	VT / AF	75–150 mg IV over 5 h

Rate Control (SVT / AF)

Drug	Used For	Easy IV Dose
Diltiazem	PSVT, AF	0.25 mg/kg IV bolus → infusion 5–15 mg/h
Verapamil	PSVT, AF	2.5–5 mg IV, repeat → infusion 2.5–10 mg/h

B. Long-Term (Oral Maintenance Therapy)

Chronic Rhythm / Rate Control

Drug	Simple Oral Dose
Disopyramide	100–150 mg q6h OR 200–300 mg q12h (SR)
Quinidine	200–300 mg q6h
Mexiletine	200–300 mg q8h
Flecainide	50–200 mg q12h
Propafenone	150–300 mg q8h OR 225–425 mg q12h (SR)
Amiodarone	Loading: 400 mg 2–3×/day → Maintenance: 200–400 mg/day
Dofetilide	500 mcg q12h
Dronedarone	400 mg q12h with meals
Sotalol	80–160 mg q12h

Treatment of Atrial Fibrillation (AF) and Atrial Flutter

Management of AF is done in sequential steps, with three main goals:

1. Goals of Treatment in Atrial Fibrillation

- AF management follows three sequential goals:
 - Acute stabilization – control ventricular rate and relieve symptoms
 - Restoration of sinus rhythm – if clinically indicated
 - Prevention of complications – stroke, thromboembolism, recurrence
- Rate control and anticoagulation are essential even if rhythm control is planned.
- Choice of therapy depends on hemodynamic stability, AF duration, LV function, and stroke risk.

2. Immediate Assessment: Hemodynamic Status

a. Management of Hemodynamically Unstable AF

- Hemodynamic instability includes:
 - Severe hypotension
 - Angina
 - Pulmonary edema
 - Shock
- Immediate synchronized direct-current cardioversion (DCC) is indicated.
- Anticoagulation status should not delay cardioversion.
- DCC rapidly restores sinus rhythm and improves cardiac output.

b. Hemodynamically Stable AF

✓ Primary focus: Ventricular Rate Control

3. Ventricular Rate Control (First-Line Strategy) : Slow ventricular response and improve symptoms.

Why Rate Control?

- AF causes rapid ventricular response
- Leads to ↓ cardiac output, ischemia, HF
- Slowing AV nodal conduction improves symptoms

- Rate control is the initial strategy in stable patients.
- Drugs act by slowing AV nodal conduction.
- Target heart rate:
 - <80 bpm (strict control)
 - <110 bpm (lenient control)

AV Nodal Blocking Agents

Patient Condition	Preferred Drugs
Normal LV function (LVEF >40%)	IV β -blockers (metoprolol, propranolol, esmolol), diltiazem, verapamil

High adrenergic state	IV β -blockers (first choice)
LVEF $\leq 40\%$	Avoid diltiazem & verapamil; use β -blockers cautiously
Heart failure exacerbation	IV digoxin or IV amiodarone
Refractory or contraindicated to others	IV amiodarone

4. Reassessment After Rate Control

After adequate AV nodal blockade:

- ✓ Reassess symptoms
- ✓ Evaluate AF duration
- ✓ Decide on rhythm control vs rate control strategy

✓ Once ventricular rate is controlled: → **Assess whether sinus rhythm restoration is needed**

Rhythm control is preferred in:

- Younger patients
- First episode AF
- Highly symptomatic patients

AF causing HF or ischemia

A. Stroke Risk Assessment in AF (CHADS₂ Score)

- AF increases risk of **thromboembolic stroke**.
- CHADS₂ score assesses stroke risk.

Risk Factor	Score
CHF	1
Hypertension	1
Age >75 yrs	1
Diabetes	1
Prior stroke/TIA	2

CHADS ₂ Score	Stroke Risk	Recommendation
0	Low	No therapy preferred; aspirin optional
1	Intermediate	Oral anticoagulation preferred
≥ 2	High	Oral anticoagulation mandatory

B. Long-Term Antithrombotic Therapy Options

- Indicated to prevent **stroke and systemic embolism**.
- Options include:
 - Warfarin (INR 2–3)
 - Dabigatran
 - Rivaroxaban
 - Apixaban

Drug	Dose	Key Points
Warfarin	INR 2–3	Requires monitoring
Dabigatran	150 mg twice daily	CrCl >30 mL/min
Rivaroxaban	20 mg once daily	Moderate–high stroke risk
Apixaban	5 mg twice daily	Alternative to warfarin or aspirin

✓ **Nonvalvular AF:** All above are acceptable

✓ **Low-risk patients:** No therapy preferred over aspirin

- In **nonvalvular AF**, all above agents are effective.
- Aspirin is inferior to anticoagulants and reserved for low-risk patients.
- Dual antiplatelet therapy (aspirin + clopidogrel) is used only if anticoagulation is not feasible.

6. Anticoagulation Before Cardioversion

Based on Duration of AF

Duration of AF	Anticoagulation Strategy
<48 hours	No prior anticoagulation required; give IV heparin or LMWH at presentation
>48 hours or unknown	Anticoagulation for ≥ 3 weeks before cardioversion

Post-Cardioversion:

- ✓ Continue anticoagulation for **at least 4 weeks** (warfarin or dabigatran)
To Restoration of atrial contraction ↑ risk of thromboembolism

7. Methods to Restore Sinus Rhythm

- Rhythm control is considered after rate control.
- Indicated in:
 - Young patients
 - First-episode AF
 - Severe symptoms
 - AF causing HF or ischemia

A. Electrical Cardioversion (DCC)

Advantage	Disadvantage
Rapid, highly effective	Requires anesthesia
Higher success rate	Risk of sinus arrest, ventricular arrhythmias

B. Pharmacologic Cardioversion

Drug Class	Examples
Class III (IK blockers)	Ibutilide, dofetilide
Class Ic	Flecainide, propafenone
Class III	Amiodarone (oral or IV)

C. “Pill in the Pocket” Approach

- Single oral dose of **flecainide or propafenone** taken by patient during AF episode.
- Suitable only for selected patients:
 - No structural heart disease
 - No AV or sinus node dysfunction
 - No QT prolongation or Brugada syndrome
- Must be tested successfully in hospital before outpatient use.

Reduces hospital visits and improves patient autonomy

D. Long-Term Maintenance of Sinus Rhythm

- AF commonly recurs due to structural heart disease and atrial remodeling.
- Antiarrhythmic drugs used:
 - Class Ic: Flecainide, propafenone (avoid in structural heart disease)
 - Class III: Amiodarone (most effective)
- Quinidine maintains rhythm but increases mortality.
- Amiodarone requires monitoring for thyroid, lung, and liver toxicity.

Drug	Key Points
Quinidine	Maintains rhythm but ↑ mortality
Class Ic agents	Avoid in structural heart disease
Amiodarone	Most effective; risk of organ toxicity

PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA (PSVT) – TREATMENT

1. Principles of PSVT Management

- Treatment depends on:
 - Severity of symptoms
 - Hemodynamic stability
 - Frequency of episodes
- Management has two phases:
 - Termination of acute episode
 - Prevention of recurrence

2. Acute Management of PSVT (Stepwise Approach)

Step 1: Assess Symptom Severity

Clinical Status	Examples
Severe symptoms	Syncope, near syncope, angina, severe heart failure
Mild–moderate symptoms	Palpitations, dizziness, mild dyspnea

Step 2: Immediate Treatment Based on Severity

A. Hemodynamically Unstable / Severe Symptoms

✓ Synchronized Direct-Current Cardioversion (DCC)

- First-line therapy
- Rapid and highly effective
- Restores sinus rhythm immediately
 - Used when drug therapy may be too slow or unsafe

B. Hemodynamically Stable / Mild–Moderate Symptoms

✓ Non-Pharmacologic (First Attempt)

✓

Vagal Maneuver	Mechanism
Valsalva maneuver	↑ vagal tone → slows AV nodal conduction
Unilateral carotid sinus massage	Reflex AV nodal inhibition

Step 3: Pharmacologic Therapy (If Vagal Maneuvers Fail)

Drug Therapy for Acute PSVT (Based on QRS Width)

Drug Group	Mechanism	Examples
↑ Vagal tone	Indirect AV nodal inhibition	Digoxin
Slow Ca ²⁺ -dependent conduction	AV nodal blockade	Adenosine, β-blockers, verapamil, diltiazem
Fast Na ⁺ -dependent conduction	Suppress atrial/ventricular conduction	Quinidine, procainamide, disopyramide, flecainide

4. Adenosine – Drug of First Choice

Reasons:

- Very short half-life (seconds)
- Rapid onset and offset
- Safer if diagnosis is uncertain (wide QRS tachycardia may be VT)

5. Long-Term Prophylaxis of PSVT

When Is Long-Term Therapy Indicated?

- Frequent PSVT episodes requiring treatment
- Infrequent but severely symptomatic episodes

Methods for Long-Term Management

Strategy	Explanation
Antiarrhythmic drug therapy	Prevents recurrence
Monitoring	Holter monitor or event recorder
Electrophysiologic testing	Identifies arrhythmia mechanism

PREMATURE VENTRICULAR COMPLEXES (PVCs) – TREATMENT

1. Overview of PVC Management

- Treatment depends on:
 - Presence of structural heart disease
 - Risk of arrhythmic death
- PVCs are common and often benign.

2. Management in Apparently Healthy Individuals

- ✓ No drug therapy required
- Reason:
 - PVCs without heart disease carry little or no risk
 - Antiarrhythmic drugs may cause more harm than benefit

3. Management in High-Risk Patients

High-Risk Features

- Recent myocardial infarction
- Left ventricular dysfunction
- Frequent or complex PVCs

Preferred Therapy

✓ β -Blockers only

Reason	Explanation
Mortality benefit	Only drugs proven to reduce death
Antiarrhythmic effect	Reduce PVC frequency
Cardioprotective	Reduce sympathetic activity

VENTRICULAR TACHYCARDIA – TREATMENT

1. Ventricular Tachycardia (VT)

- VT is a rapid rhythm originating from the ventricles.
- It can be life-threatening due to reduced cardiac output and risk of degeneration into VF.
- Management depends on:
 - Hemodynamic stability
 - Presence of symptoms
 - Underlying cause

2. Acute Ventricular Tachycardia

Step 1: Assess Symptom Severity

Clinical Status	Examples
Severe symptoms	Hypotension, syncope, angina, acute HF
Mild or no symptoms	Palpitations, dizziness, stable BP

A. Acute VT with Severe Symptoms

✓ Immediate synchronized direct-current cardioversion (DCC)

Key points:

- Rapidly restores sinus rhythm
- Prevents progression to VF
- Correct precipitating factors (ischemia, electrolyte imbalance, drug toxicity)
- If VT is due to a transient cause (e.g., acute MI, digitalis toxicity), long-term antiarrhythmic therapy is not required once the cause is corrected.

B. Acute VT with Mild or No Symptoms

✓ Initial pharmacologic therapy

Drug	Comments
IV Procainamide	Effective for monomorphic VT
IV Amiodarone	Useful in structural heart disease
IV Sotalol	Class III effect
IV Lidocaine	Alternative agent

✓ Proceed to synchronized DCC if:

- Patient deteriorates
- VT degenerates into VF
- Drug therapy fails

3. Sustained Ventricular Tachycardia (Chronic / Recurrent)

Definition

- VT lasting **>30 seconds** or requiring intervention for termination.

Key Features

- Patients are at **very high risk of sudden cardiac death**.
- Trial-and-error drug testing is **unsafe and ineffective**.

Preferred Management Strategy

✓ Nondrug approaches are favored

Approach	Explanation
Implantable Cardioverter-Defibrillator (ICD)	Detects and terminates VT/VF automatically
Drug therapy alone	Not reliable for mortality reduction

ICD is the most effective method for preventing sudden death in sustained VT.

4. Ventricular Proarrhythmia

Definition

- Worsening or induction of arrhythmia caused by antiarrhythmic drugs.

Common Cause

- Class Ic drugs (flecainide, propafenone)

Typical Features

- Rapid, sustained monomorphic VT
- Sinusoidal QRS pattern
- Often resistant to cardioversion

Management

Treatment	Rationale
IV Lidocaine	Competes at sodium channel
Sodium bicarbonate	Reverses sodium channel blockade

These agents counteract excessive sodium channel inhibition.

5. Torsade de Pointes (TdP)

Definition

- Polymorphic VT associated with **QT interval prolongation**.

Acute Management of TdP

✓ Direct-current cardioversion (DCC)

- Required in most acute episodes
- TdP often recurs after DCC

Prevention of Recurrence

✓ **IV Magnesium sulfate – Drug of choice**

✓ If magnesium is ineffective:

- Increase heart rate to shorten QT interval

Method	Details
Temporary pacing	105–120 beats/min
Pharmacologic pacing	Isoproterenol or epinephrine infusion

Additional Measures

✓ Discontinue QT-prolonging drugs

✓ Correct electrolyte abnormalities:

- Hypokalemia
- Hypomagnesemia

Contraindicated drugs:

- Procainamide
- Other QT-prolonging agents

Lidocaine is **usually ineffective** in TdP.

6. Ventricular Fibrillation (VF)

Definition

- Chaotic ventricular activity with **no effective cardiac output**.

Management of VF / Pulseless VT

✓ Treat according to **American Heart Association (AHA) CPR guidelines**

Key steps include:

- Immediate defibrillation
- High-quality CPR
- Advanced cardiac life support (ACLS)

VF is a **medical emergency** requiring immediate intervention.

Treatment of Bradyarrhythmias

Bradyarrhythmias are rhythms with abnormally slow heart rate.

- 1) Common causes:
 - a) Sinus node dysfunction
 - b) Carotid sinus hypersensitivity
 - c) Vasovagal syncope
 - d) AV conduction blocks
- 2) Treatment depends on:
 - a) Presence of symptoms
 - b) Hemodynamic impact
 - c) Reversibility of cause

Treat symptoms, not just heart rate.

- a) **Sinus Node Dysfunction (Sick Sinus Syndrome):** Sinus node dysfunction refers to **inappropriate sinus bradycardia, sinus pauses, or sinoatrial block**, often associated with **bradycardia–tachycardia syndrome**.

Goals

- 2) Eliminate **symptomatic bradycardia**
- 3) Prevent **syncope and hypotension**
- 4) Manage associated tachyarrhythmias (e.g., AF)
 - a)

Clinical Features

- Symptomatic sinus bradycardia
- Sinus pauses
- Alternating bradycardia and tachycardia (e.g., AF)

Treatment Approach

Situation	Management
Asymptomatic bradycardia	No treatment required
Symptomatic bradycardia	Permanent pacemaker
Bradycardia–tachycardia syndrome	Pacemaker + manage tachyarrhythmia

- Drugs used for supraventricular tachycardias (β -blockers, CCBs, digoxin):
 - Can worsen bradycardia
 - Should be used only if a pacemaker is in place

Role of Permanent Pacemaker in Bradyarrhythmias

- Permanent ventricular pacemaker is the long-term treatment of choice for:
 - Symptomatic sinus node dysfunction
 - Symptomatic carotid sinus hypersensitivity
 - Chronic symptomatic AV block

Pacemaker prevents:

- Syncope
- Hypotension
- Sudden cardiac arrest due to extreme bradycardia

b) Carotid Sinus Hypersensitivity

- Exaggerated vagal response to carotid sinus stimulation
- Causes bradycardia, hypotension, or syncope

Treatment

Condition	Therapy
Symptomatic patients	Permanent pacemaker
Persistent symptoms	Add α -adrenergic agonist (midodrine)

Midodrine increases peripheral vascular tone and blood pressure.

C) Vasovagal Syncope

Mechanism

- **Trigger** → sympathetic surge → forceful ventricular contraction
- **Followed by reflex bradycardia and hypotension**

Drug Therapy for Vasovagal Syncope

Drug Class	Examples	Mechanism
β -blockers	Metoprolol	Blunts sympathetic surge
Mineralocorticoids	Fludrocortisone	Expands plasma volume
Anticholinergics	Scopolamine, dipyramide	Reduce vagal tone
α -agonists	Midodrine	↑ Peripheral resistance
Adenosine antagonists	Theophylline, dipyridamole	Increase heart rate
SSRIs	Sertraline, paroxetine	Modulate autonomic reflexes

ATRIOVENTRICULAR (AV) BLOCK – TREATMENT

- AV block is a **delay or interruption of impulse conduction** from atria to ventricles.
- **Types of AV Block (Clinical Relevance)**

Type	Severity
First degree	Usually benign
Mobitz I	Often transient
Mobitz II	Dangerous
Third degree (complete block)	Life-threatening

Acute Management of Symptomatic AV Block

Indications for Emergency Treatment

- Altered mental status
- Chest pain
- Hypotension
- Shock

Stepwise Acute Treatment

Step	Therapy	Dose / Notes
1	Atropine	0.5 mg IV every 3–5 min (max 3 mg)
2	Transcutaneous pacing	If atropine fails
3	Epinephrine infusion	2–10 mcg/min
4	Dopamine infusion	2–10 mcg/kg/min

Atropine, dopamine, and epinephrine are **often ineffective** in:

- Mobitz II block
- Infranodal or trifascicular block

Chronic Management of AV Block

Long-Term Therapy

Patient Type	Management
Symptomatic AV block	Permanent pacemaker
Asymptomatic AV block	Close monitoring