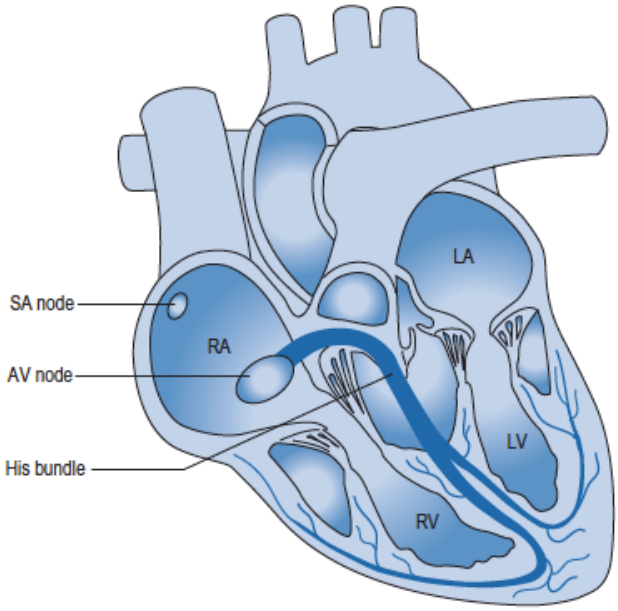
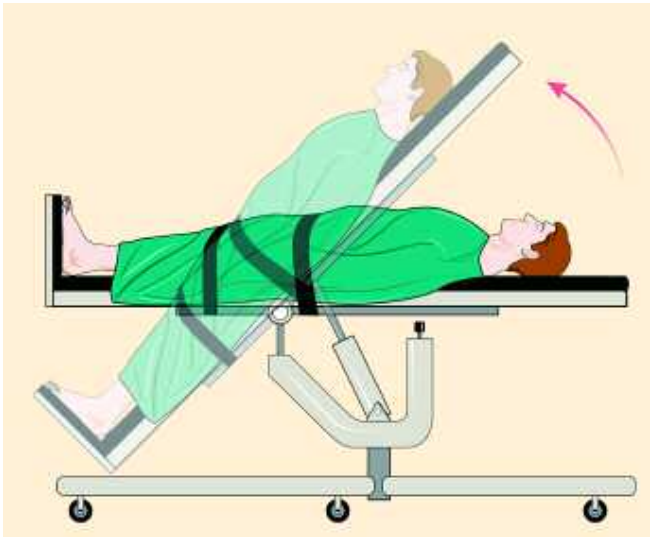
Arrhythmia

Arrhythmia or dysrhythmia is loss of cardiac rhythm, especially irregularity of heartbeat.

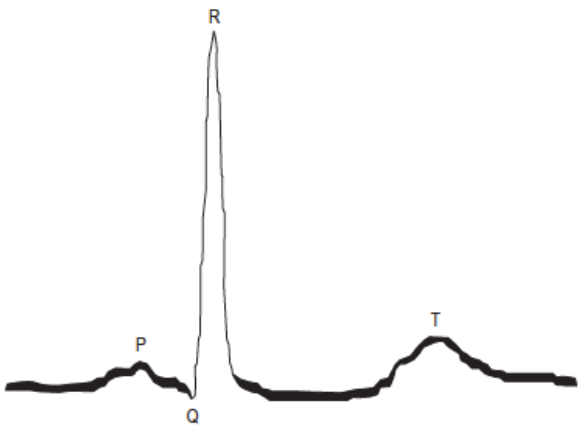
Diagnosis

- ***Electrocardiogram*** (ECG) is the cornerstone of diagnosis for cardiac rhythm disturbances.
- Cardiac ***auscultation*** can reveal the irregularly irregular pulse characteristic of AF.
- Diagnosis of carotid sinus hypersensitivity can be confirmed by performing ***carotid sinus massage*** with ECG and ***blood pressure monitoring***.
- Vasovagal syncope can be diagnosed using the ***upright body-tilt test***.





Upright body-tilt test



Normal ECG

Supraventricular arrhythmias

Common supraventricular tachycardias requiring drug treatment are atrial fibrillation (AF), atrial flutter and paroxysmal supraventricular tachycardia (PSVT).
Other arrhythmias do not require drug therapy such as premature atrial complexes, sinus arrhythmia and sinus tachycardia.

Supraventricular tachycardias may cause clinical manifestations ranging from no symptoms to minor palpitations or irregular pulse to severe and even life-threatening symptoms. Patients may experience dizziness or acute syncopal episodes, symptoms of HF, anginal chest pain, or a choking or pressure sensation during the tachycardia episode.

- **Atrial Fibrillation (AF)**

Atrial fibrillation has extremely rapid (400–600 atrial beats/min) and disorganized atrial activation. There is loss of atrial contraction (atrial kick), and supraventricular impulses penetrate the atrioventricular (AV) conduction system to variable degrees, resulting in irregular ventricular activation and irregularly irregular pulse (120–180 beats/min).

- **Atrial Flutter**

Atrial flutter has rapid (270–330 atrial beats/min) but regular atrial activation. Ventricular response usually has a regular pattern and a pulse of 300 beats/min. This arrhythmia occurs less frequently than atrial fibrillation but has similar precipitating factors, consequences and drug therapy.

- **Paroxysmal supraventricular tachycardia (PSVT)**

PSVT arising by reentrant mechanisms includes arrhythmias caused by AV nodal reentry, AV reentry incorporating an anomalous AV pathway, sinoatrial (SA) nodal reentry and intraatrial reentry. This type is not common.

Ventricular arrhythmias

• Premature Ventricular Complexes

Premature ventricular complexes (PVCs) can occur in patients with or without heart disease. PVCs often cause no symptoms or only mild palpitations.

• Ventricular Tachycardia

Ventricular tachycardia (VT) is defined by three or more repetitive premature ventricular complexes occurring at a rate greater than 100 beats/min. It is may result acutely from:

- Severe electrolyte abnormalities (hypokalemia or hypomagnesemia)
- Hypoxia
- Drug toxicity (e.g., digoxin)
- Acute myocardial infarction (MI)
- Ischemia complicated by heart failure (HF).

- ✓ ***Sustained VT*** is that which requires intervention to restore a stable rhythm or persists a relatively long time (usually >30sec.)
- ✓ ***Nonsustained VT*** self-terminates after a brief duration (usually <30sec.).
- ✓ ***Incessant VT*** refers to VT occurring more frequently than sinus rhythm, so that VT becomes the dominant rhythm.
- ✓ ***Monomorphic VT*** has a consistent QRS configuration.
- ✓ ***Polymorphic VT*** has varying QRS complexes.
- ✓ ***Torsade de pointes*** (TdP) is a polymorphic VT in which the QRS complexes appear to undulate around a central axis.

The presentation of VT may vary from totally asymptomatic to pulseless hemodynamic collapse.

• Ventricular Proarrhythmia

Proarrhythmia refers to the development of a significant new arrhythmia, such as VT, ventricular fibrillation (VF), or TdP, or worsening of an existing arrhythmia. Proarrhythmia results from the same mechanisms that cause other arrhythmias or from an alteration in the underlying substrate due to the antiarrhythmic agent. Consequences of proarrhythmia range from no symptoms to worsening of symptoms to sudden death.

• *Torsade de pointes*

Torsade de pointes (TdP) is a rapid form of polymorphic VT associated with evidence of delayed ventricular repolarization due to blockade of potassium conductance. TdP may be hereditary or acquired.

- **Ventricular Fibrillation**

VF is electrical anarchy of the ventricle resulting in no cardiac output and cardiovascular collapse. Sudden cardiac death occurs most commonly in patients with coronary artery disease and those with LV dysfunction.
VF associated with acute MI may be classified as

(1) Primary: uncomplicated MI not associated with HF
(2) Secondary or complicated: MI complicated by HF.

VF results in hemodynamic collapse, syncope and cardiac arrest.

Bradyarrhythmias

- ***Sinus bradyarrhythmias***

Sinus bradyarrhythmias (heart rate <60 beats/min) are common especially in young and athletically active individuals. They are usually asymptomatic and do not require intervention.
However, some patients have ***sinus node dysfunction*** (***sick sinus syndrome***) because of underlying organic heart disease and the normal aging process, which attenuates SA nodal function.
Alternating bradyarrhythmias and tachyarrhythmias are referred to as the ***tachy–brady syndrome***.

- ***AV block***

AV block or conduction delay may occur in any area of the AV conduction system. AV block may be found in patients without underlying heart disease (e.g., trained athletes) or during sleep when vagal tone is high.
It may be transient when the underlying etiology is reversible (e.g., myocarditis, myocardial ischemia, after cardiovascular surgery, or during drug therapy).
β-Blockers, digoxin or nondihydropyridine calcium antagonists may cause AV block, primarily in the AV nodal area.

Class I antiarrhythmics may exacerbate conduction delays below the level of the AV node. AV block may be irreversible if the cause is acute MI, rare degenerative diseases, primary myocardial disease or congenital heart disease.

Patients with bradyarrhythmias experience symptoms associated with hypotension, such as dizziness, syncope, fatigue, and confusion. If LV dysfunction exists, patients may experience worsening HF symptoms.

Antiarrhythmic drugs (the Vaughan Williams classification)

Class	Drug	Conduction Velocity[a]	Refractory Period	Automaticity	Ion Block
Ia	Quinidine Procainamide Disopyramide	↓	↑	↓	Sodium (intermediate) Potassium
Ib	Lidocaine Mexiletine	0/↓	↓	↓	Sodium (fast on/off)
Ic	Flecainide Propafenone[b]	↓↓	0	↓	Sodium (slow on/off)
II[c]	β-Blockers	↓	↑	↓	Calcium (indirect)
III	Amiodarone[d] Dofetilide Dronedarone[d] Sotalol[b] Ibutilide	0	↑↑	0	Potassium
IV[c]	Verapamil Diltiazem	↓	↑	↓	Calcium

➢ Class I

Class I drugs are sodium channel blockers. Antiarrhythmic sodium channel receptor principles account for drug combinations that are:

- additive (e.g., quinidine and mexiletine)
- antagonistic (e.g., flecainide and lidocaine)
- potential antidotes to excess sodium channel blockade (sodium bicarbonate).

✓ Class Ia

Class Ia drugs slow conduction velocity, prolong refractoriness, and decrease the automatic properties of sodium-dependent (normal and diseased) conduction tissue. Class Ia drugs are effective for both supraventricular and ventricular arrhythmias.

- E.g. Quinidine, procainamide and disopyramide

✓ Class Ib

Class Ib drugs probably act similarly to class Ia drugs, except that class Ib agents are considerably more effective in ventricular than supraventricular arrhythmias.

- E.g. Lidocaine and mexiletine

✓ Class Ic

Class Ic drugs slow conduction velocity while leaving refractoriness relatively unaltered. Although effective for both ventricular and supraventricular arrhythmias, their use for ventricular arrhythmias has been limited by the risk of proarrhythmia.

- E.g. Flecainide and propafenone

➢ **Class II**

Class II drugs include β-adrenergic antagonists; effects result from antiadrenergic actions. β-Blockers are most useful in tachycardias in which nodal tissues are abnormally automatic or are a portion of a reentrant loop. These agents are also helpful in slowing ventricular response in atrial tachycardias (e.g. AF) by effects on the AV node.

- E.g. *propranolol*, *metoprolol* and *esmolol*

➢ **Class III**

Class III drugs prolong refractoriness in atrial and ventricular tissue and include very different drugs that share the common effect of delaying repolarization by blocking potassium channels.

- Amiodarone and sotalol are effective in most supraventricular and ventricular tachycardias.
- Dronedarone, ibutilide, and dofetilide are indicated only for treatment of supraventricular arrhythmias.

➢ **Class IV**

Class IV drugs inhibit calcium entry into cells, which slows conduction, prolongs refractoriness, and decreases SA and AV nodal automaticity.
Calcium channel antagonists are effective for automatic or reentrant tachycardias that arise from or use the SA or AV nodes.

- E.g. Verapamil and diltiazem

➢ **Others / Class V**

Class V include other agents with by other or unknown mechanisms of actions, such as adenosine, digoxin and magnesium sulfate.

- ***Adenosine*** is indicated for rapid reversion to sinus rhythm of paroxysmal supraventricular tachycardias.
- ***Digoxin*** is a cardiac glycoside that increases the force of myocardial contraction and reduces conductivity within the atrioventricular (AV) node.
- Intravenous infusion of ***magnesium sulfate*** is usually effective in the management of uncontrolled torsade de pointes that can progress to ventricular fibrillation and sometimes death.