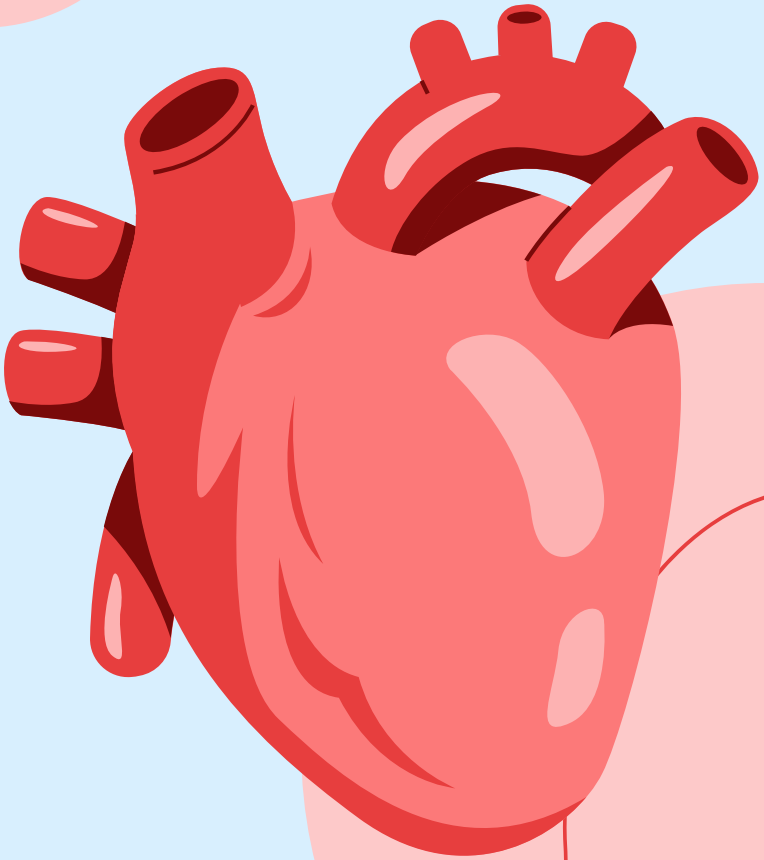
Atrial fibrillation



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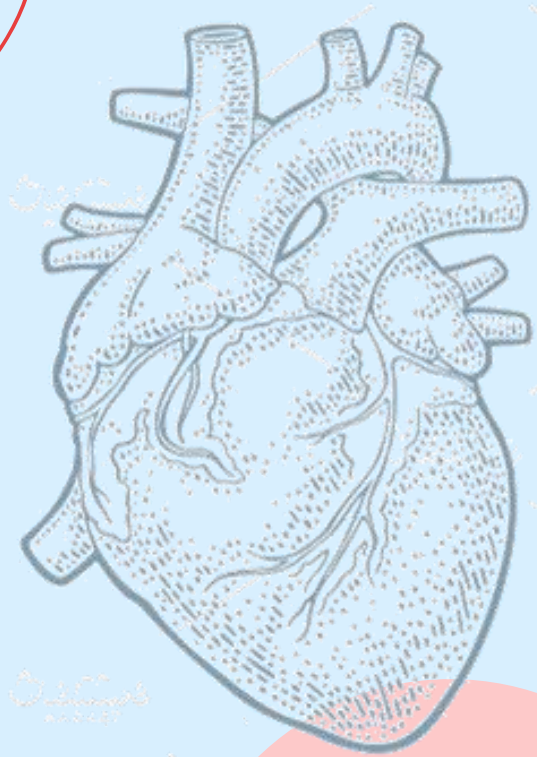
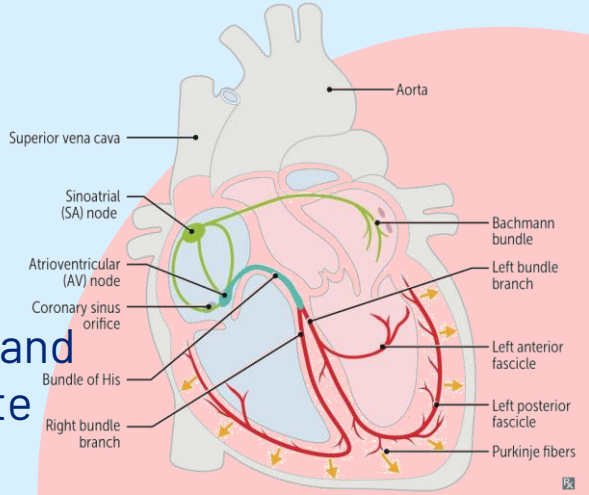


01

Normal physiology

Conductive system of the heart

- The heart generates its own electrical impulses due to pacemaker cell current “funny current” which is the spontaneous flow of Na.
- Cardiac automaticity means that pacemaker cells don’t require stimulation to initiate action potential.
- The cardiac conductive system is a collection of nodes and specialized conduction cells, that initiate and coordinate contraction of the heart muscle.
- The conduction system is made up of non-contractile myocardial cells, that specialize in initiating and conducting the cardiac impulses.



The conduction pathway

SA node → AV node → bundle of His → right and left bundle branches → Purkinje fibers

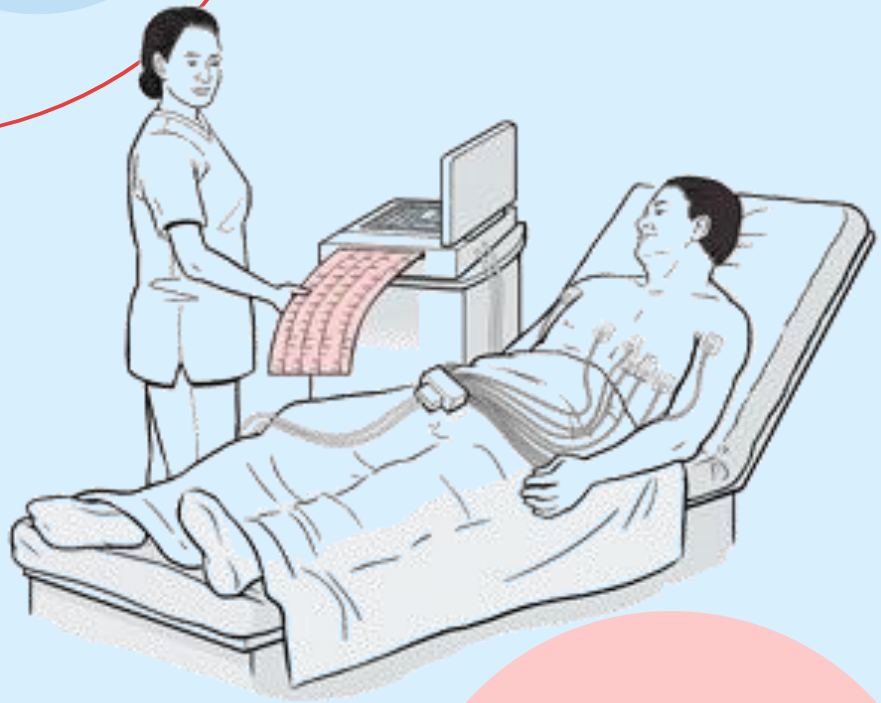
SA node → atrium

Speed of conduction:

1. His
2. Purkinje
3. Atrium
4. Ventricles
5. AV node

Mnemonic :
He
Parks
At
Venture
Avenue

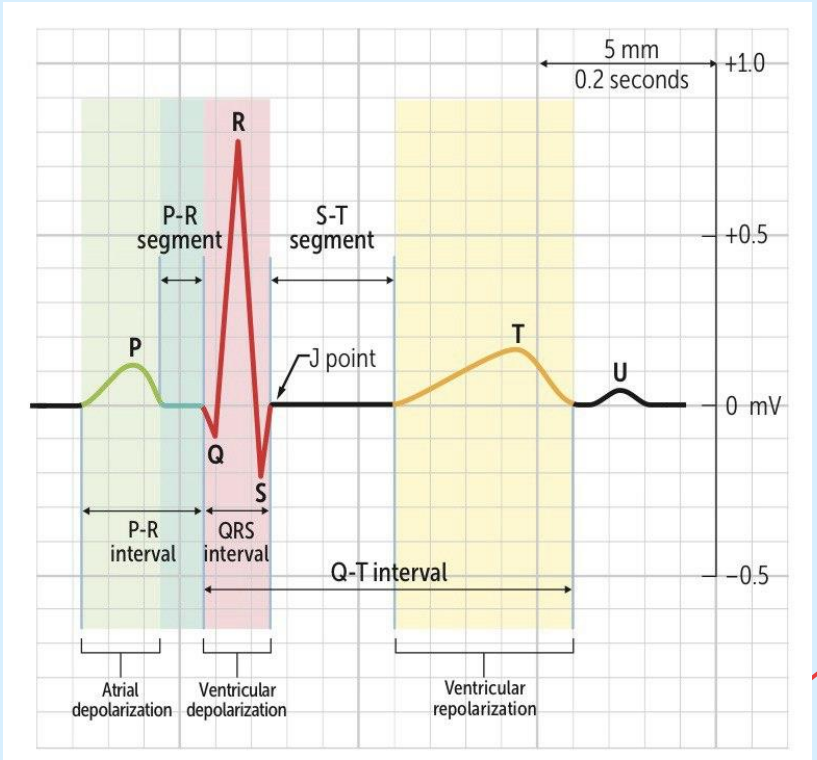
Nodal cells	Location	Function	Rate
SA node	Near the entrance of SVC into right atrium	Pacemaker of the heart • Sets the sinus rhythm and rate	60-100 bpm
AV node	At the top of the atrioventricular septum Superior and medial to entrance of coronary sinus into right atrium	Receives impulses from SA node Conducts impulses very slowly (0.1s or 100ms delay) • This delay prevents simultaneous atria and ventricular contraction	40-60 bpm
Bundle of His	Inferior to AV node in the membranous part of interventricular septum	Receives impulses from AV node	25-40 bpm
Bundle branches	Right and left bundle branches span along the length of the interventricular septum until the apex	Receives impulses from bundle of His	25-40 bpm
Purkinje fibers	Terminal conducting fibers located in the subendocardial layer of myocardium in right and left ventricles	Receives impulses from bundle branches	25-40 bpm

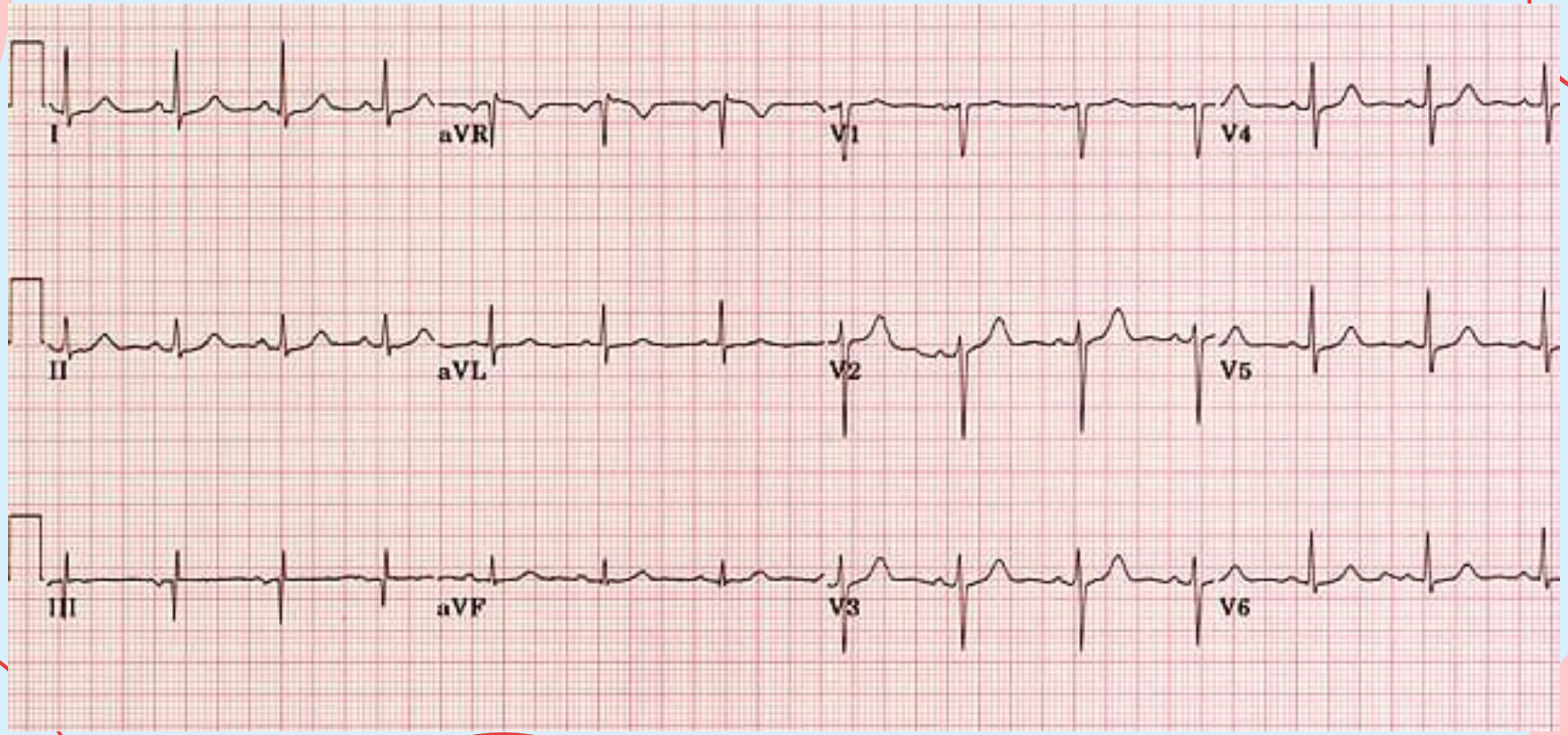


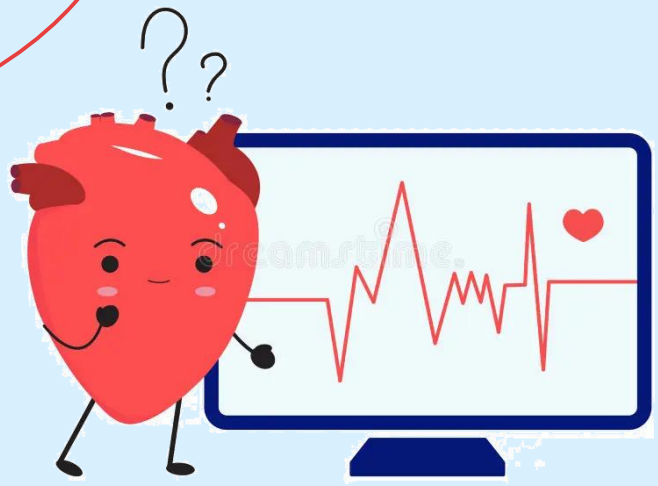
02

Electrocardiogram

Question	Answer	Diagnosis
1. Rhythm	Criteria for sinus rhythm: 1. Are the P waves positive in I and II? 2. Is there a QRS complex after each P wave? 3. Are the PR intervals constant? 4. Are the RR intervals constant?	sinus rhythm or no sinus rhythm?
2. Heart rate	Estimate heart rate: 300/number of large boxes between two QRS complexes	heart rate in beats per min
3. P waves	a) Large P-wave amplitude (>2.5 mm in II, III, or aVF)	right atrial enlargement
	b) Prolonged negative part of P wave in V1 (1 mm) and P wave with 2 peaks in II, P-wave duration >0.12 s	left atrial enlargement
4. PR interval	a) >0.2 s (if PR interval constant for all beats and each P wave is followed by a QRS complex)	I° AV block
	b) <0.12 s and QRS complex normal	LGL syndrome
	c) <0.12 s and visible delta wave	WPW syndrome
5. QRS axis	Determine the axis according to leads I, II, and aVF	normal axis left axis deviation right axis deviation northwest axis
6. QRS duration	a) ≥0.12 s (always think of WPW syndrome as a differential)	complete bundle branch block
	b) >0.1 s and <0.12 s with typical bundle branch block appearance (notching)	incomplete bundle branch block
7. Rotation	Rotation is defined according to the heart's transition zone. Normally the transition zone is located at V4, which means that right ventricular myocardium is located at V1–V3 and left ventricular myocardium is at V5–V6.	transition zone at V5–V6: clockwise rotation transition zone at V1–V3: counterclockwise rotation NOTE: don't evaluate rotation in the setting of myocardial infarction, WPW syndrome, or bundle branch block
8. QRS amplitude	a) QRS amplitude <0.5 mV in all standard leads	low voltage
	b) Positive criteria for left ventricular hypertrophy	left ventricular hypertrophy
	c) Positive criteria for right ventricular hypertrophy	right ventricular hypertrophy
9. QRS infarction signs	abnormal Q waves, QS waves, missing R-wave progression	myocardial infarction; localization according to affected leads




I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6



03

Arrhythmias

Arrhythmias

- An arrhythmia is any change (abnormality) in the normal cardiac rate or rhythm.
- An arrhythmia might be a result from physical conditions such as a heart defect or as a response to infections, fever and medications.
- It could be either symptomatic or asymptomatic.
- If its symptomatic it could cause chest pain, weakness, palpitations, syncope, sweating, slow or fast heart rate and SOB.

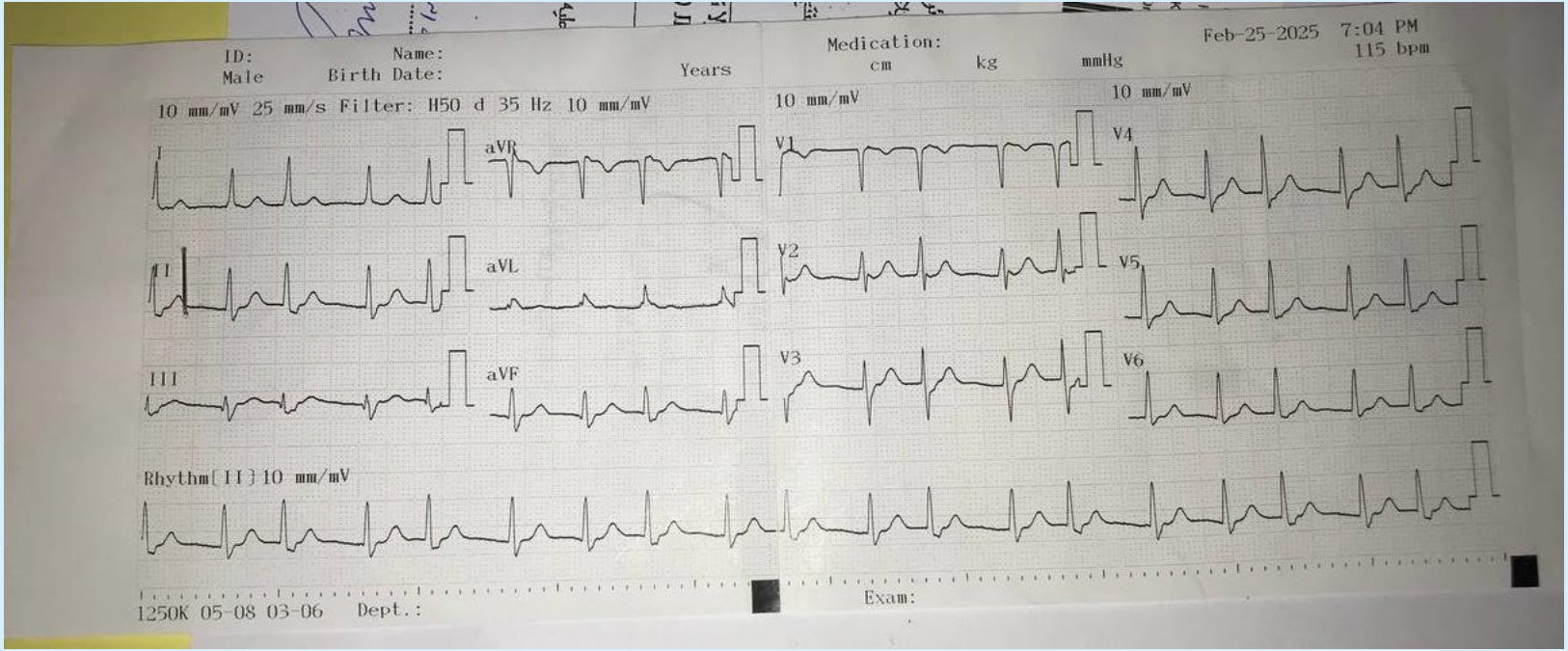
Arrhythmias are divided to brady-arrythmias and tachy-arrhythmias
Tachy-arrhythmias are then divided into narrow or wide QRS

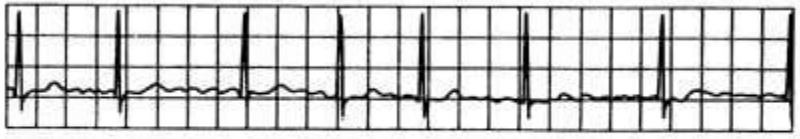
Wide QRS: Ventricular tachycardia, ventricular fibrillation and WPW syndrome.
Narrow QRS: sinus tachycardia, atrial fibrillation, atrial flutter and PACs.

- **Atrial fibrillation findings on ECG:**

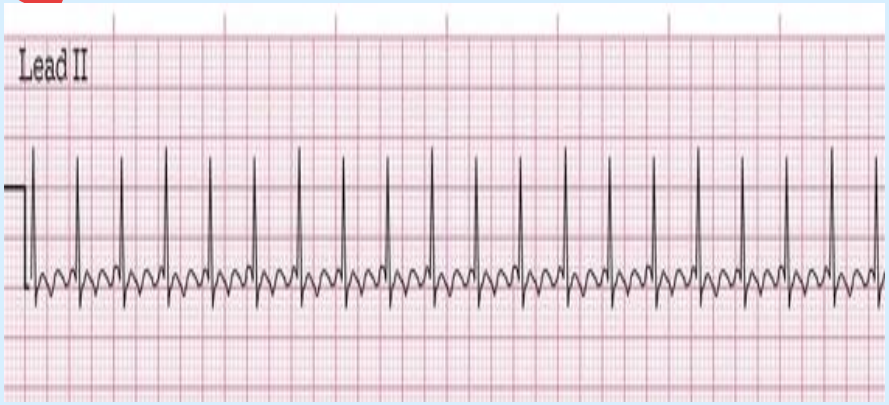
1. Absent P wave
- flat (more ectopic focus)
- small irregular fibrillatory waves

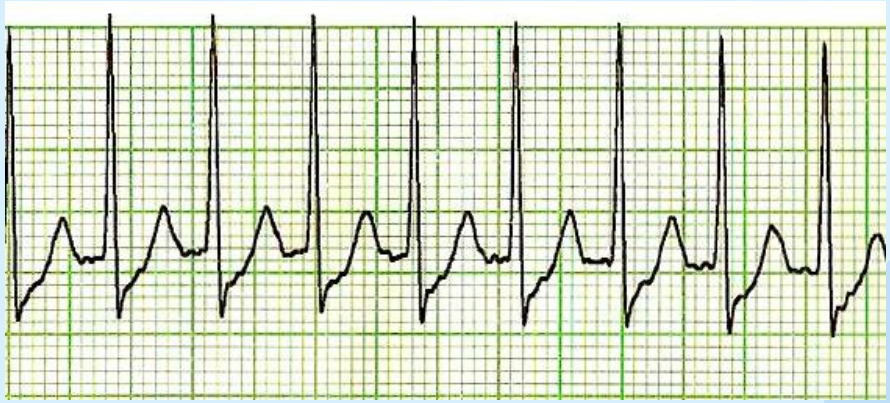
2. Irregularly irregular rhythm (variable R-R interval)

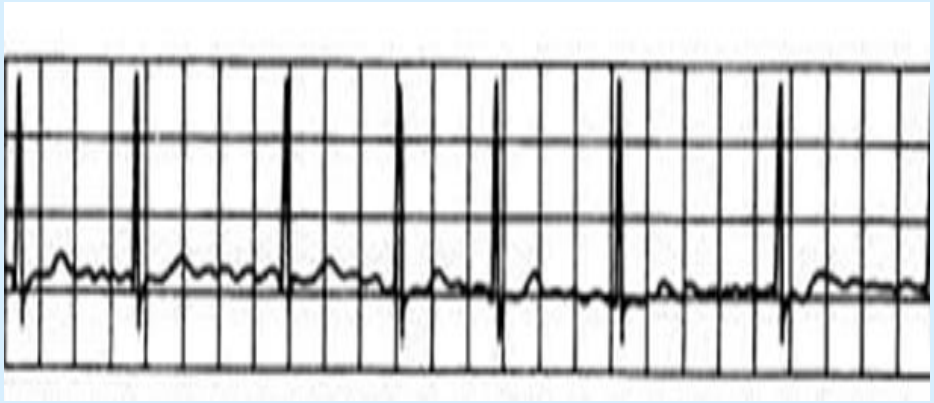




Atrial fibrillation

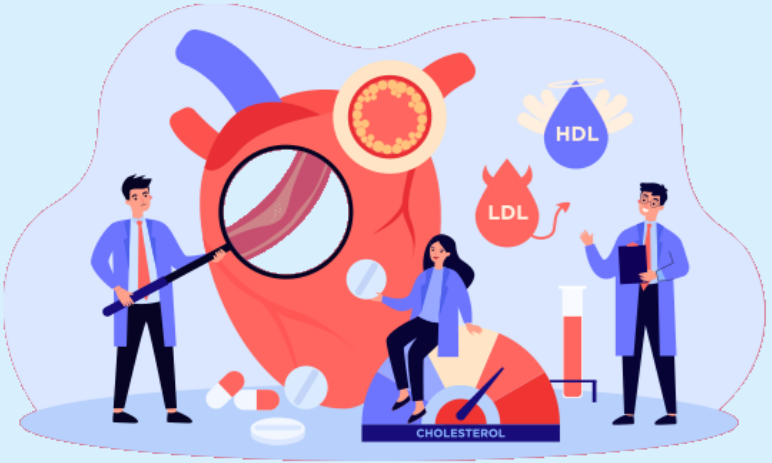

Lead II





04

Pathophysiology



Pathophysiology

- Atrial fibrillation (A-fib) is a supraventricular arrhythmia that originates above the ventricles, where multiple foci in the atria fire continuously in a chaotic pattern, causing a totally irregular, rapid ventricular rate.

- **Fibrillation:** all muscle fibers contracting at different time resulting in quivering twitching movement.

Epidemiology

- Atrial fibrillation is the most common cardiac arrhythmia.
- Ectopic foci of electrical activity are most commonly present around the pulmonary veins.
- AF is strongly age-dependent, approximately 25% of individuals aged 40 years and older will develop AF during their lifetime, 4% of individuals older than 60 years, and 8% of persons older than 80 years.
- The prevalence of AF is 10% in persons 80 years or older.
- The incidence of AF is significantly higher in men than in women in all age groups.

Causes of AFib

1. Cardiac causes:

- Congestive heart failure (CHF)
- Dilated cardiomyopathy
- Coronary artery disease / myocardial infarction
- Hypertension
- Rheumatic heart disease
- Valvular heart disease including mitral stenosis and regurgitation (less common)

2. Non cardiac causes:

- Lung pathologies
- Thyrotoxicosis of hyperthyroidism
- Pheochromocytoma
- Surgical procedures
- Drugs (cocaine, methamphetamines)
- Electrolyte abnormalities
- Holiday heart syndrome
- Sepsis

Risk factors

Sleep apnea

Advanced ages

Hypertension

CVS disorders

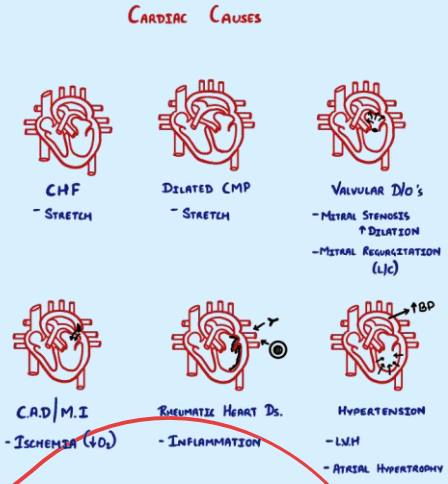
Obesity

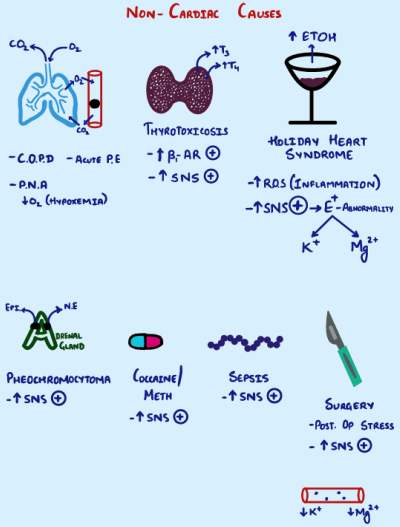
Diabetes mellitus

Smoking

- *Mechanisms by which AFib occurs:*

1. Stretching of the myocardium
2. Ischemia: decreased oxygen
3. Inflammation
4. Increase in sympathetic nervous system (SNS)
5. Electrolyte imbalance





Classification

Hemodynamic stability

1. Unstable Afib (e.g. chest pain, altered mental status, acute pulmonary edema, hypotension, or cardiogenic shock)
2. Stable Afib

Ventricular rate

1. Tachycardic Afib >100-110 bpm
2. Bradycardic Afib or slow Afib <60 bpm

Onset and duration

1. **Paroxysmal AF:** episodes of AF that terminate spontaneously within 7 days (most episodes last less than 24 hrs)

2. **Persistent AF:** episodes of AF that last more than 7 days and may require either pharmacologic or electrical intervention to terminate

3. **Long standing persistent AF:** AF that persisted for more than 12 months, either because cardioversion has failed or hasn't been attempted

4. **Permanent AF:** when both patient and clinician have decided to abort any further restoration strategies after shared clinical decision making.

Clinical manifestations

Asymptomatic atrial fibrillation
Its possible for a patient to be completely asymptomatic

1. Heart rhythm: no visible p waves, irregular rhythm, variations in RR intervals, variations HR can be fast or slow.

- **common symptoms:**

-Palpitations
-Shortness of breath
-Fatigue
-Episodes of syncope

2. Cardiogenic shock: the HR is so fast → there's not enough time for the ventricles to fill → lowers EDV → lowers SV → lowers CO

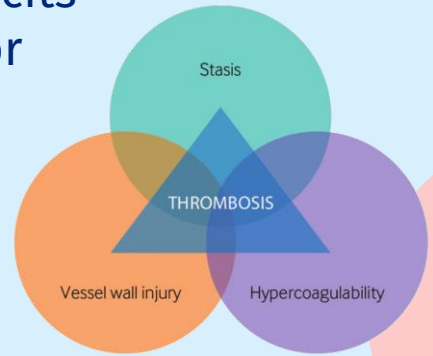
3. Emboli :

Atrial fibrillation → inadequate atrial contraction → blood volume stays in the atria → stasis → clot formation

- **Virchow's triad:** stasis, endothelial injury and hypercoagulability increases the risk of a clot

Clot becomes emboli

a. Brain: causes stroke, may present as neurological deficits
b. Spleen: may present as pain in LUQ and show issues or abnormalities in the CBC
c. Kidney: may cause an increase in BUN and creatinine
d. Gut: may present as abdominal pain



05

Diagnosis and treatment

Diagnosis flowchart

Atrial fibrillation is diagnosed based on clinical presentation, ECG findings, and additional tests to determine the underlying cause

Patient symptoms

- Palpitations
- Chest pain
- Dyspnea
- Fatigue
- Syncope

Asymptomatic pts. Are sometimes diagnosed incidentally on an ECG.

ECG

a **12 lead ECG** is the **gold standard test** for diagnosing AF
It can detect the characteristic irregularly irregular rhythm, the absence of p waves
variable RR intervals
narrow QRS complex

Laboratory tests

can be done to identify any underlying conditions that could affect management
Electrolytes → check for imbalances
TSH, free T4 → to rule out hyperthyroidism which is a common AF trigger
Troponin and BNP →to evaluate cardiac stress or HF.

Other investigations

TEE or **TTE** to assess left atrial size, LV function, valvular disease or clots.
Also done to check for left atrial thrombus before cardioversion.
Chest xray to identify pulmonary causes (PE, pneumonia)
Holter monitor used in paroxysmal or intermittent AF if an ecg is inconclusive.

1. Rate control

Ideally <110 bpm

The heart rate can be controlled using drugs that slow down AV node conduction:

1. **Beta blockers (class II antiarrhythmics)**
- first line
- Usually B1 selective agents such as metoprolol, atenolol.
- Decreases the slope of phase 4 of pacemaker AP, it also prolongs phase 3.

2. Calcium channel blockers (class IV antiarrhythmics)
-such as verapamil, diltiazem.
- Act on phase 0, slow rise in AP and also prolong phase 3.
That's why beta blockers and CCB can lead to AV block.

3. Digoxin → increases the parasympathetic tone to the heart.

2. Rhythm control

Restoration of normal sinus rhythm
This is done by cardioversion

- **Electrical cardioversion:** delivering synchronized shock at the time of QRS.
we administer anesthesia then deliver the electrical shock to the chest.
All myocytes depolarize, the SA node being the first to repolarize and depolarize.

- Chemical cardioversion: administration of an antiarrhythmic
Often Ibutilide (class III antiarrhythmic)
Less commonly used due to drug toxicity

Risk of stroke

Both chemical and electrical cardioversion can cause stroke

It takes 48 hours for a thrombus to form, thus

If symptoms appeared <48 hrs → cardioversion safe

If >48 hrs or we're unsure → Transesophageal echocardiogram to exclude the presence of a thrombus.
Or we can give an anticoagulant for 3 weeks → cardioversion.

EXCEPTION!! in a hemodynamically unstable patient (hypotensive or in shock) we perform emergent cardioversion.

- **Antiarrhythmic medications:**

Administered before/after cardioversion

Class I drugs :
Flecainide, propafenone
For pts without structural heart diseases

Class III drugs :
Amiodarone, sotalol, dofetilide
For pts with structural heart diseases

Amiodarone is restricted to pts. In whom other measures fail

3. Anticoagulation

- **Warfarin**

- Requires regular INR monitoring
- Goal INR 2-3

- **Rivaroxaban, Apixaban**

- Factor X inhibitors

- **Dabigatran**

- Direct thrombin inhibitor (factor IIa)

- **Aspirin**

- Less effective
- Less risk of bleeding
- Only used if risk of stroke is low

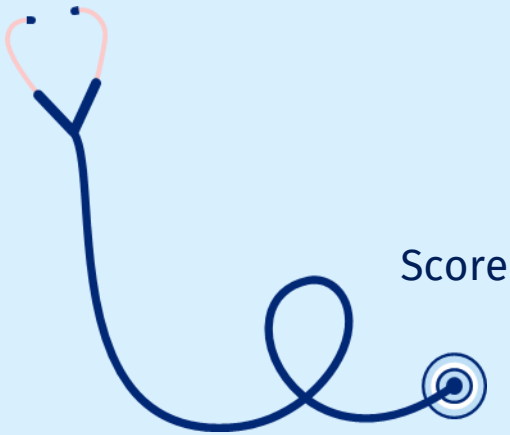
Anticoagulation must be administered whether AF persists or sinus rhythm is restored

Risk of stroke

CHADS VASC Score

- **C**HF (1 point)
- **H**TN (1 point)
- **A**ge 65-75 (1 point)
- **A**ge >75 (2 points)
- **D**iabetes (1 point)
- **S**troke (2 points)
- Female (1 point)
- **Vasc**ular disease (1 point)

Score 2 or more → **warfarin** or **other anticoagulant**
Score 0-1 → **aspirin**



4. Surgical therapy

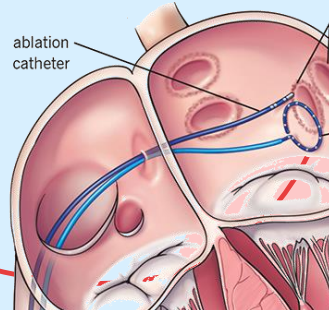
Pulmonary vein isolation (PVI):

Is a catheter ablation procedure used to treat atrial fibrillation by isolating the pulmonary veins from the rest of the atrium.

The pulmonary veins are a major source of AF triggers due to abnormal automaticity or micro-reentry circuits.

We isolate the veins by burning the myocytes around them and that will create scar tissue that doesn't conduct electricity, thus it eliminates the AF episodes.

Effective in paroxysmal AF as there are few ectopic foci unlike persistent AF.



Thank you

Resources

- Davidson’s principles and practice of medicine
- Boards and beyond
- First aid for the USMLE step 1
- Osmosis
- Ninja nerd