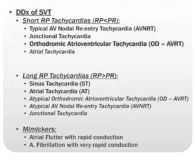
- How to calculate HR? → Regular: 300 / R-R intervals large square vs irregular: QRS in large 30 boxes * 10
- QRS narrow: rhythm from AV node and above vs wide: Below AV (Abnormal conduction)
- Note: when looking at p waves look specifically at lead 2 and V_1
- How to know if you have normal cardiac axis: 2 ways: 1 lead I and AVR → if lead I is +ve and AVR opposite looking at V_2 if true then this is normal ✓ if negative then abnormal 2 look at lead I+2+3

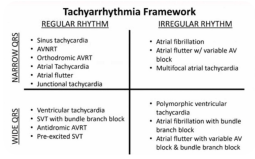
Axis	Lead I	Lead II	Lead III
Normal	Positive	Positive	Positive
LAD	Positive	Negative	Negative
RAD	Negative	Positive	Positive
Extreme	Negative	Negative	Negative

- Amplitude → Low: if limb lead <0.5 or precordial lead <1.0
  causes of low: Hypothyroid, Amyloidosis, pericardial effusion

Component	Amplitude (mV)
P wave	0.2
QRS	1.0
T wave	0.2 - 0.3

  - High → Atrial: 1) Rt (also called p. pulmonale) → peaked, pointy p wave + >2.5mm amplitude
    2) Lt (also called p. mitral) → wide p wave >0.12 + Biphasic p wave + -ve p wave in V_1
  - High → Ventricles:
    1) Lt: if: A) R in AVL >11 mm or B) S in V3 + R in AVL >24 in men or >20 in women
    2) Rt: if: A) in lead V_1 R> 6mm or (R/S ratio is >1 and negative T wave) B) R in AVR > Q in AVR or R in AVA ≥ 5mm
- PR interval (normal is 0.12 - 20) if longer then this is a block and if shorter this is an accessory pathway
- QRS (normal is less than 0.12) if more (around 0.16) then this is a ventricular cause
- QT interval: if more than 460 in male or 470 in females → this is torsade de pointes
- T wave inversion? this is bad prognosis indicative of: 1) proximal LAD occlusion 2) intracranial hemorrhage
- Hyperacute T wave? Seen in 1) Hyperkalemia 2) STEMI
- U- wave: Seen in 1) Digoxin 2) Hypothermia





- What are the mechanisms of Arrythmias?
  1) Automaticity: Hyperdepolarization or Sympathetic System activation (Tx: B- blockers) 2) Triggered Activity:
  A) Early afterdepolarization (prolonged QT intervales) or torsade } happens in early phase 3? B) Late afterdepolarization (Digoxin toxicity) } happens in late phase 3 or early 4?
  3) Re-entery: Macro: Like A. flutter or Micro: A fib

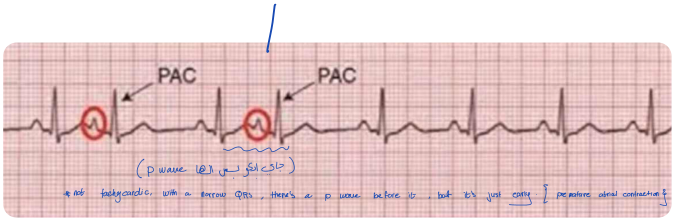
MC of Arrythmias

Premature Complexes

Premature complexes = **extra beats** that occur earlier than expected in the normal heart rhythm. These beats can arise from **atria**, **AV junction**, or **ventricles**.

They are caused by **increased automaticity** (spontaneous firing) or **triggered activity** (afterdepolarizations), often linked to things that stimulate the **sympathetic system**, like:

- Exercise 🏃
- Stress or catecholamine release
- Medications (stimulants, digitalis)
- Ischemia or infarction
- Hypoxia
- Structural heart disease (especially for ventricular)



🔶 1. Atrial Premature Contractions (PACs)

ECG Features:

- **Narrow QRS**
- A **P wave** appears *before* the QRS, but **earlier than normal**.
- A **pause** follows before the next beat.
- QRS is **normal** because the ventricles conduct normally.

Why? The atria fire early → impulse travels normally through AV node and His-Purkinje → normal QRS.

Symptoms:

- Mostly **asymptomatic** (people don't feel them).
- May cause **palpitations** in some.

Treatment:

- **Observe only** if there are no symptoms.
- If **symptomatic**, give:
- **Beta-blockers**
- **Treat underlying cause** (e.g., anxiety, caffeine, electrolyte imbalance).

🔶 2. Ventricular Premature Contractions (PVCs)

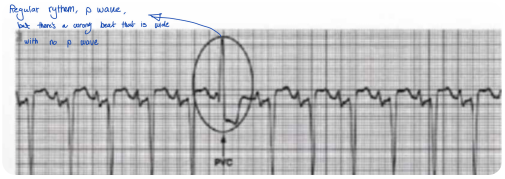
ECG Features:

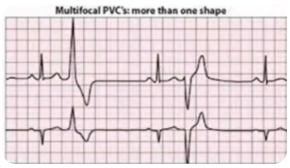
- **Wide QRS complex** (because conduction is through muscle, not the fast His-Purkinje system).
- **No visible P wave** before the QRS (the atria didn't fire or it's buried inside the wide QRS).

Why? The impulse originates **from the ventricle**, bypassing the normal conduction system → slow conduction → wide QRS.

Symptoms:

- May be felt as **"skipped beats" or "thumps"**.
- Often **benign** in healthy hearts.





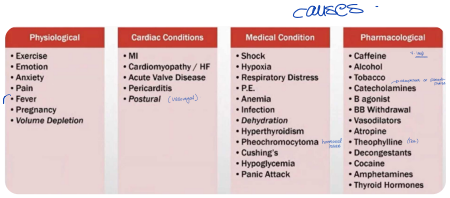
Treatment:

- If asymptomatic: **Reassurance**.
- If symptomatic or frequent:
- **Beta-blockers**
- Investigate for **structural heart disease**
- **Ablation** (rare cases)

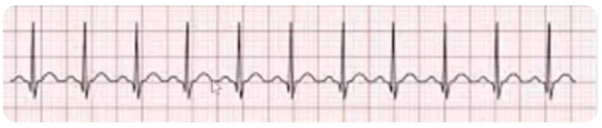
📌 **Summary Table:**

Type	Origin	QRS Width	P Wave	Treatment
PAC	Atria	Narrow	Present (early)	Observe / Beta-blockers
PVC	Ventricle	Wide	Absent or hidden	Observe / Beta-blockers / Treat cause

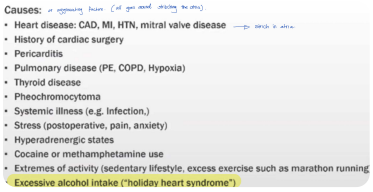
1- Sinus Tachycardia



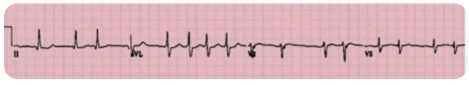
- **What it is:** Your normal pacemaker (the sinus node) just speeds up.
- **ECG look:**
- **P wave** (tiny bump) → **QRS** (big spike) → **T wave** sequence all normal, but closer together.
- **Rate:** 100–180 bpm.
- **Regular** spacing.
- **How to spot it:** Normal shape, narrow QRS (<120 ms), but rate >100 bpm.
- **Why it happens:** Fever, pain, dehydration, anxiety, thyroid problem, etc.
- **Treatment:**
- **Fix the cause** (give fluids, reduce fever, treat pain).
- If no clear cause and still too fast → **β-blocker** or **ivabradine**.
- Ablation is almost never needed.



2- Atrial Fibrillation (AFib)



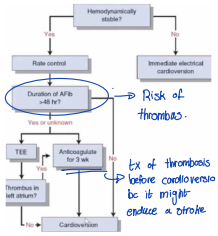
- **What it is:** Atria quiver ("fibrillate") instead of a nice squeeze.
- **ECG look:**
- **No P waves**—just a wiggly baseline.
- **Irregularly irregular** QRS timing.
- Narrow QRS.
- **How to spot it:** Totally random spacing, no P waves.
- **Why it happens:** High blood pressure, heart disease, alcohol binge, lung disease, etc.
- **Treatment:**
- **If unstable** (low BP, chest pain) or a fib less than 48h or young patient or rate is not controlled or symptomatic → **immediate cardioversion** (shock).
- **If stable** → slow the rate with **β-blockers**, **diltiazem/verapamil**, or **digoxin** THEN anticoagulant (warfarin or DOAC) only if (a fib more than 48 hours Or based on your **CHA_2DS_2-VASc** stroke risk.)



- **Options**:
- **Warfarin** (monitor INR 2.0–3.0)
- **DOACs** (dabigatran, rivaroxaban, apixaban) – *only for non-valvular AF; avoid with mechanical valves or severe mitral stenosis.*

CHA_2DS_2-VASc Score for Stroke Risk

Factor	Points
Congestive heart failure or LV dysfunction	1
Hypertension (high blood pressure)	1
A_2ge ≥ 75	2
Diabetes mellitus	1
S_2troke/TIA/thromboembolism history	2
Vascular disease (prior MI, PAD, aortic plaque)	1
Age 65–74	1
Sc Female sex	1
Total possible	**0–9**



- **Score 0** (male) or **1** (female only): low risk → **no anticoagulation** or consider aspirin.
- **Score 1** (male) or **2** (female): intermediate → consider anticoagulation based on bleeding risk and patient preference.
- **Score ≥ 2** (male) or **≥ 3** (female): high risk → **anticoagulation recommended**

Consider Bleeding Risk: (HAS-BLED Score)

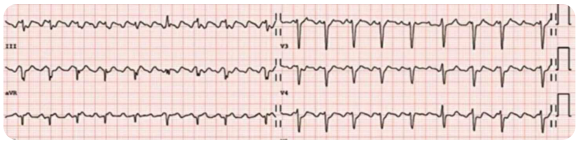
HAS-BLED assesses bleeding risk:

HAS-BLED criteria	Points

H Hypertension (SBP >160 mmHg)	1
A Abnormal renal or liver function	1 or 2
S Stroke history	1
B Bleeding history or predisposition	1
L Labile INR (unstable warfarin control)	1
E Elderly (>65 yrs)	1
D Drugs/alcohol (NSAIDs, antiplatelets, alcohol use)	1 or 2

- **Score ≥ 3:** high bleeding risk → consider carefully (does not exclude anticoagulation but requires caution).
- **Score < 3:** lower bleeding risk → safer to anticoagulate.

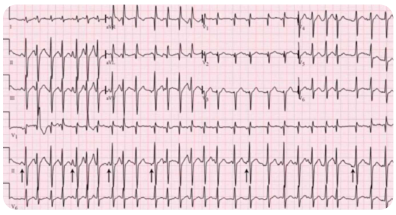
3- Atrial Flutter



- **What it is:** One big re-entry loop in the atria (usually around the tricuspid valve).
- **ECG look:**
- Tiny **"sawtooth"** waves (flutter waves) instead of P waves.
- Often a **2:1 block** → about **150 bpm** (two flutters for every QRS).
- Narrow QRS.
- **How to spot it:** Sawtooth pattern vs AFib's chaotic baseline.
- **Treatment:** Same as AFib—rate control, anticoagulation, consider ablation.

Workbook point	What it means in plain language
One focus in the cavotricuspid isthmus (CTI)	Unlike AF (many chaotic foci), typical flutter is a single **macro-re-entry loop** that circles the right atrium between the *tricuspid annulus* and the *inferior vena cava*.
Regular rhythm, saw-tooth waves	On ECG the F-waves march along at ≈ 300 bpm, producing the classic **"saw-tooth" baseline**; the ventricular response is often exactly half that (2 : 1 block → ~150 bpm).
Best seen in leads II, III, aVF	The negative F-waves in the inferior leads make the saw-teeth obvious; V1 often shows positive flutter waves.
Treatment same as AF	Stroke risk and first-line therapy (**rate control + anticoagulation**) mirror AF.

4- Multifocal Atrial Tachycardia (MAT)



What the *Medical Workbook 3* says

- **ECG** looks "the same as A-fib but P-waves have ≥ 3 different morphologies"
- **Cause**: "Hypoxia in severe pulmonary diseases such as COPD"
- **Treatment**:
- **First-line** → oxygenation
- If that fails → "CCB, β-blockers, digoxin, amiodarone"
- Cardioversion "believed … ineffective"

Readable breakdown & clinical pearls

- **Definition** — chaotic firing from ≥ 3 distinct atrial sites → wandering atrial pacemaker that speeds up (> 100 bpm).
- **Key ECG criteria:**

1. Rate **> 100 bpm** (if ≤ 100 it's called *wandering atrial pacemaker*).
2. **Irregular rhythm** (variable R-R like AF).
3. **≥ 3 P-wave shapes** in the same lead (often II, V1).
4. **Variable PR intervals** (each focus conducts differently).
5. **Narrow QRS** (supraventricular origin).

- **Classic setting:** elderly patient with **COPD exacerbation or hypoxaemia**—think of MAT whenever you see "irregular tachycardia + wheezes & oxygen mask."
- **Pathophysiology:** low O_2 → atrial irritability + catecholamine surge → multiple ectopic foci.
- **Step-wise management:**

1. **Correct hypoxia** (high-flow O_2, treat COPD/pneumonia, normalize electrolytes).
2. **Rate control** if still rapid:

- **Verapamil or diltiazem** (best evidence).
- **Metoprolol** (if no bronchospasm).
- **Digoxin or amiodarone**—second-line in refractory cases.

3. **Electrical cardioversion rarely works** (circuit isn't a single loop).
4. **Ablation** plays no role; the foci are multiple and mobile.

- **Prognosis:** usually resolves once the underlying pulmonary trigger is fixed; mortality relates to lung disease, not the arrhythmia itself.
- **How to tell MAT from AF or A-flutter:**

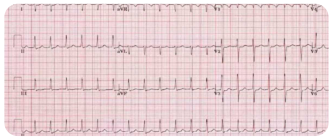
• Feature	• MAT	• AF	• A-flutter
• P-waves	• ≥ 3 shapes present	• Absent (f-waves)	• Saw-tooth F-waves

• Rhythm	• Irregular	• Irregular	• Usually regular (2:1 or 3:1)
• Ventricular rate	• 100–120 (variable)	• 90–170	• Often exactly 150 (2:1)

5- supra ventricular tachycardia

What is "Supraventricular Tachycardia" (SVT)?

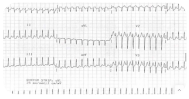
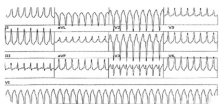
- Umbrella term for **rapid rhythms that start above the ventricles** (atria or AV node) and **use the normal His–Purkinje system**, so the QRS is **narrow (< 120 ms)**.
- Two major re-entry flavours are highlighted in your workbook:
- **AVNRT** (AV-nodal re-entrant tachycardia)
- **AVRT** (AV-re-entrant tachycardia) ⇒ • *orthodromic* • *antidromic*



AV-Nodal Re-entrant Tachycardia (AVNRT)

- **Pathophysiology**: the AV node houses two functional pathways:
- **Slow pathway** → repolarises quickly.
- **Fast pathway** → depolarises quickly. A premature atrial beat can block in the fast limb, dive down the slow limb, then race **retrograde** up the fast limb → a **tight micro-circuit** your workbook calls "AV node re-entery circuit"
- **ECG**: regular rate 150-250 bpm, **no obvious P-waves** (buried in or just after QRS), narrow complexes.
- **Acute treatment** ("block AV node"):
- **Vagal manoeuvres** (Valsalva, carotid massage).
- **IV adenosine** (stops AV nodal conduction for a few seconds).
- If adenosine fails or is contraindicated → **diltiazem / verapamil** or **β-blocker**.
- **Synchronized cardioversion** if haemodynamically unstable.
- **Prevention**: daily **β-blocker or non-DHP CCB**; **radio-frequency ablation of the slow pathway** has >95 % cure rate

AV-Re-entrant Tachycardia (AVRT)

Sub-type (workbook label)	Conduction loop	QRS	Key ECG clue	First-line acute Rx
B Orthodromic AURT 	**Down AV node → up accessory pathway**	**Narrow**	Looks like AVNRT but RP' interval often longer	**Same as AVNRT**: vagal → adenosine → CCB/BB Medical Workbook 3Medical Workbook 3
c Antidromic AURT 	**Down accessory pathway → up AV node**	**Wide**(ventricles pre-excited)	Regular wide-complex tachycardia	**Treat like WPW**: **IV procainamide** or **ibutilide**; avoid AV-nodal blockers Medical Workbook 3Medical Workbook 3

Remember: Antidromic AVRT is essentially **"SVT with a WPW QRS"**—blocking the AV node alone can precipitate ventricular fibrillation, so **never give adenosine, verapamil, β-blocker or digoxin** in that setting.

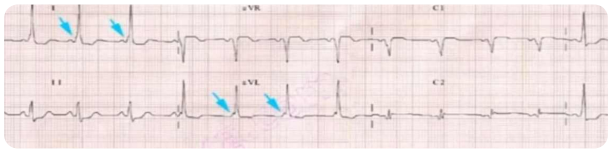
Step-by-step bedside algorithm

1. **Assess stability** – shock immediately if hypotensive, chest-pain, heart failure.
2. **Regular, narrow QRS, no P-waves?** → think **AVNRT or orthodromic AVRT**.
3. **Try vagal manoeuvre** (success rate ~25 %).
4. **Give 6 mg IV adenosine** (follow with 20 ml flush).
   - If terminates → AVNRT/AVRT confirmed.
   - If transient slowing reveals saw-teeth or flutter waves → treat as flutter.
5. **Second-line**: diltiazem 0.25 mg/kg IV or β-blocker.
6. **Wide-complex but regular?** → give **procainamide 15 mg/kg IV over 30 min** (suspect antidromic AVRT).
7. **Plan for EP referral** – ablation offers definitive cure in most symptomatic cases.

How to remember the workbook's key lines

- "**Regular multiple narrow complex with no P-waves like AFib but regular pattern**" → that is the textbook ECG description of AVNRT / orthodromic AVRT.
- "**Slow pathway has fast repolarization … fast pathway has fast depolarization**" → the dual-pathway physiology.
- "**Block AV node … vagal manoeuvres … IV adenosine … CCB and BB … cardioversion**" → the exact therapeutic staircase.
- "**Preventive … CCB and BB or ablation of the slow pathway**" → chronic management.
- "**QRS is wide … opposite direction … same tx as WPW**" → antidromic AVRT needs WPW protocol.

6-What is Wolff–Parkinson–White (WPW) syndrome?



- **Accessory "shortcut" (Bundle of Kent)** bridges atria ↔ ventricles, bypassing the AV-node brake.
- Ventricles can be **pre-excited** (partly depolarised early), creating the classic ECG pattern.
- This shortcut also sets up a **macro-re-entry loop → AV re-entrant tachycardia (AVRT)** and raises the risk of fast atrial arrhythmias degenerating into VF.

ECG triad

Workbook bullet	How it appears	Why it happens
1 Delta wave	Slurred up-stroke at QRS onset	Accessory pathway activates ventricle early
2 Short PR	≤ 120 ms	AV-node delay bypassed
3 Wide QRS	≥ 120 ms with fusion of early (Kent) + late (His) depolarisation	Mixed conduction pathways

Tip: delta is easiest to see in leads V2–V4 and limb lead I.

Rhythm problems you'll meet

Scenario	ECG look	Immediate danger	First-line treatment
Orthodromic AVRT (down AV-node, up Kent)	Narrow QRS, 180–250 bpm	Usually stable	Same "SVT ladder": **vagal → IV adenosine** → CCB/β-blocker → cardioversion
Antidromic AVRT (down Kent, up AV-node)	**Wide** regular QRS tachycardia	Can mimic VT	**IV procainamide** (workbook) or **ibutilide**; *avoid AV-nodal blockers*
AF + WPW	Irregular, **wide** fast (200–300 bpm)	May → VF in seconds	**Procainamide** or **ibutilide**, urgent **DC shock** if unstable

Never give β-blocker, diltiazem, verapamil or digoxin when pre-excited AF is suspected — exactly as your workbook warns Medical Workbook 3Medical Workbook 3.

Definitive cure

- **Radio-frequency ablation of the Kent bundle** (success > 95 %, complication < 1 %).
-

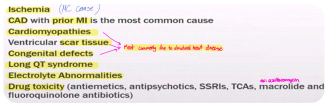
Quick decision algorithm (bedside)

1. **Identify pre-excitation** (delta wave or wide regular tachycardia in known WPW).
2. **Check haemodynamics** – if BP low, chest-pain, shock → **synchronised DC cardioversion** immediately.
3. **Stable + narrow QRS SVT** → treat like AVNRT (vagal → adenosine).
4. **Stable + wide QRS or irregular wide** → use **procainamide** 15 mg/kg IV over 30 min (or ibutilide 1 mg IV).
5. **Plan EP referral** for ablation at first opportunity.

Exam & ward pearls

- **Up to 60 %** of WPW patients develop AVRT; 10–20 % experience AF with WPW.
- **Sudden-death risk** is tied to how fast the accessory pathway can conduct (< 250 ms): EP study answers this.
- **"Latent WPW"**: baseline ECG normal, but pathway unmasked during tachycardia.
- **Post-ablation OAC** not routinely required unless CHA_2DS_2-VASc ≥ 1 ♂ / 2 ♀ for other reasons.

7- ventricular tachycardia

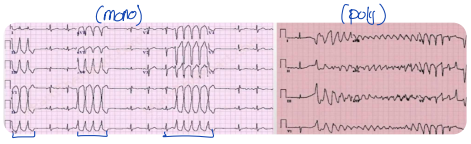


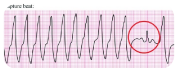
ventricular tachycardia: Mainly due to structural cause, other causes include:

- Note that VT are devided into sustained (>30min) and non sustained (<30 min) } the difference is the duration?
- Note: polymorphic (① prolonged QT ② ↓ Mg^+)

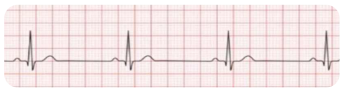
sustained → polymorphic (Torsade de points), monomorphic (if pulseless: this a cardiac arrest)
non sustained → polymorphic, monomorphic

- How to treat it: [1] if Stable: (A) Acute management: tx the underlying cause then use IV amiodarone and consider cardioversion (B) preventive: to prevent from cardiac arrest → consider implantation "ICD" and "EPS" [2] if unstable: code blue (worst tachy): use defibrillators.
- On ECG: VT association: ① width of QRS >160 ② undeterminate axis ③ AV association ④ capture beats (sudden and narrow QRS among wide ones)





Bradyarrhythmias

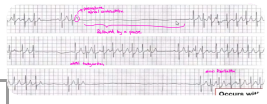


1. Sinus bradycardia When the SA-node fires slowly (< 60 bpm) yet the P-QRS-T pattern remains perfectly normal.

- **Why it happens:** higher vagal tone during sleep or in athletes, acute inferior-wall ischaemia that supplies the SA-node, infiltrative or congenital heart disease, and rate-slowing drugs such as β-blockers, digoxin or non-dihydropyridine Ca-channel blockers.
- **When to worry:** fatigue, dizziness, angina or exercise intolerance suggest the cardiac output has become inadequate.
- **What to do:**
- **Asymptomatic** → watchful waiting.
- **Symptomatic** → correct triggers (stop offending drugs, treat ischaemia or electrolytes), then give IV atropine 0.5 mg; if ineffective, start a β-agonist infusion (isoprenaline, adrenaline or dopamine). Persistent instability calls for temporary pacing.

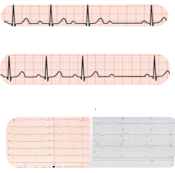
2. Sick-sinus (brady-tachy) syndrome Degeneration or fibrosis of the SA-node produces erratic pauses that may alternate with bursts of atrial tachycardia or AF ("brady-tachy"). Most patients are elderly or have long-standing sinus bradycardia.

- **Management pearls:** dual-chamber pacemaker is definitive. Rate-controlling drugs for the tachy phases (e.g., β-blockers) are added **after** pacing is in place to avoid excessive slowing.



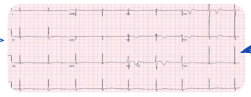
3. Atrioventricular (AV) block – a traffic-jam between atria and ventricles. Medical Workbook 3Medical Workbook 3

Block	ECG hallmark	Typical site	Immediate care	Long-term
1st-degree	PR > 200 ms, every beat conducts	AV-node	Atropine, b agonist , pacemaker	none
2nd-degree Mobitz I (Wenckebach)	PR lengthens until a QRS drops	AV-node	Atropine, b agonist , pacemaker	rarely pacing
2nd-degree Mobitz II	Constant PR, random dropped QRS	infra-His bundle	Pacemaker	**permanent pacemaker**
3rd-degree (complete)	P-and-QRS march independently	any level	*Pacemaker*	**permanent pacemaker**



Additional clinical clues: 1- if a patient with chronic a fib suddenly developed a complete heart block -> digoxin toxicity

2- irregular irregular then regular irregular -> look at p wave if found -> sinus if not found -> complete heart block

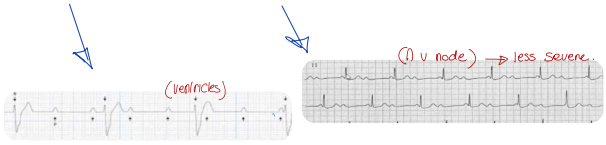


Note : in complete heart block p>QRS. Vs v. Tach , QRS>p

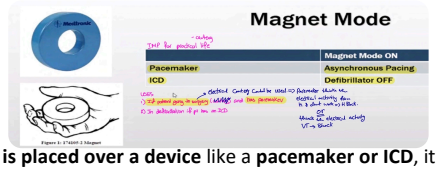
Note : does 3rd degree have wide or narrow QRS ? It depends wether its from the ventricle (wide) or close to AV node (narrow)

Quick takeaway

- **Slow but stable?**—observe.
- **Slow + symptoms?**—remove causes → atropine → β-agonist → pace.
- **SA-node worn out?**—pacemaker, then tame tachy bursts.
- **Any Mobitz II or complete block?**—think pacing first, questions later.



Devices :



1. Magnet Mode

What's a Magnet Mode?

- **When a magnet is placed over a device** like a **pacemaker or ICD**, it changes how the device behaves temporarily.

Effect on Devices:

Device	Magnet Mode ON
Pacemaker	**Asynchronous Pacing**
ICD	**Defibrillator OFF**

- **Asynchronous pacing** means the pacemaker paces without sensing the heart's intrinsic activity.
- **Defibrillator OFF** means the ICD will not deliver shocks even if dangerous rhythms are present.

Why use it?

- **During surgeries**: Especially when using **electrical cautery**, because electromagnetic interference might confuse the pacemaker or ICD.
- **To avoid inappropriate shocks** during surgery or manipulation.

Real-life example:

- If the ICD is ON during surgery and electrical cautery is used → it might interpret it as VT/VF → deliver unnecessary shock.
- Putting a magnet over it will stop this.

2. Cardiac Resynchronization Therapy (CRT)

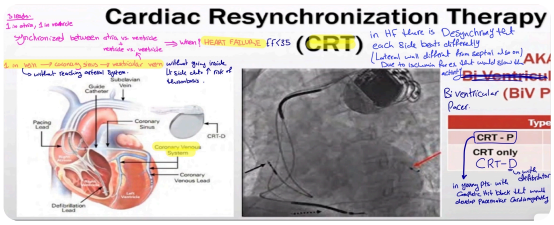
What is CRT?

- Also called **BiV Pacing**.
- Used in **Heart Failure** with **EF < 35%** and **dyssynchrony** between ventricles.

Why do we need it?

- In HF, the heart's sides don't beat in sync (esp. LBBB).
- CRT helps both ventricles contract together → more efficient pumping.

Leads:



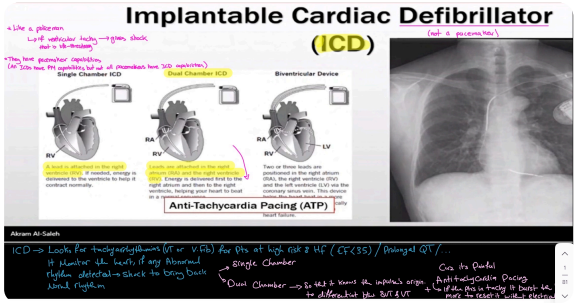
- 3 leads:

1. Right atrium
2. Right ventricle
3. Left ventricle via **coronary sinus** (not artery!)

Types:

- **CRT-P**: Only pacing
- **CRT-D**: CRT + **defibrillator** (used in young patients or those at higher risk)

3. Implantable Cardioverter Defibrillator (ICD)



What's an ICD?

- Like a **policeman**.
- Detects dangerous arrhythmias like **VT/VF** → gives shock to save life.

Function:

- **Monitors rhythm** all the time.
- If life-threatening tachyarrhythmia is detected → **shock** delivered.

Indications:

- EF < 35%
- Prior VT/VF
- Prolonged QT

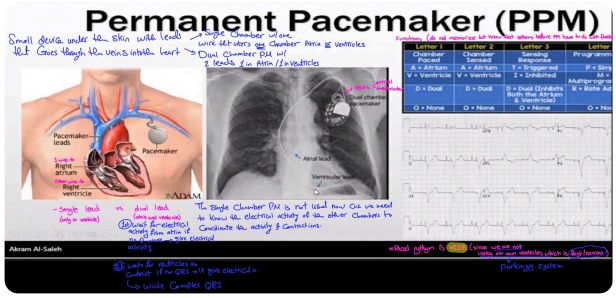
Types:

- **Single Chamber**: Lead in RV
- **Dual Chamber**: RA + RV → helps identify rhythm origin (e.g., differentiate SVT vs. VT)
- **Biventricular**: Used with CRT

Extra:

- Has **pacemaker capability** too (not all pacemakers have ICD though)
- **ATP (Anti-Tachycardia Pacing)**: Instead of shock, tries fast pacing to terminate VT.

4. Permanent Pacemaker (PPM)



What is it?

- A device under the skin with **wires to the heart**.
- Sends electrical impulses when the heart is too slow or blocked.

Types:

- **Single chamber**: One wire to RV.
- **Dual chamber**: Two wires (RA + RV) → better coordination of heart beats.

How it works:

- Senses atrial activity → waits → if no ventricular response, it paces.
- Produces **wide QRS** because impulse bypasses natural conduction system.

ECG Clue:

- Wide rhythm = pacemaker is working (not using Purkinje system)

NBG Code (3-5 Letters):

- Tells which chambers are paced, sensed, and how device responds.
- Don't memorize, just know it defines **function** of pacemaker.

5. Temporary Pacing

Two Types:

1. **Transcutaneous** (external)

- Pads on skin (anterior-posterior)
- Used in **emergency** (HR < 30s, complete heart block)
- **Very painful** and **not reliable**
- For **temporary** use only

2. **Transvenous (TVP)**:

- Wire inserted into vein (femoral or internal jugular)
- Wire enters RV and paces heart
- Used when patient can't move or for longer support
- **Risks**: Infection, heart perforation

When to use?

- In **MI**-induced blocks
- Before placing permanent pacemaker