INTERNAL MEDICINE

2019

A short meander around electrocardiography, clinical neurology and clinical examinations.

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Introduction

I have been a physician for 38 years now. Towards the end of my career I have become a Senior Associate Professor of Aimst University, Bedong, Kedah and teach final year medical students, just before graduating to become functional doctors.

I was both impressed with their enthusiasm and keenness to learn, and an awareness of large gaps in understanding, especially Internal medicine; general principles, and ECGs and neurology.

In a moment of enthusiasm I started writing this book, mainly directing it towards the first two years of the three clinical years of training.

A group of newly graduated doctors assisted me carrying all the laborious chores and pushing me to finish.
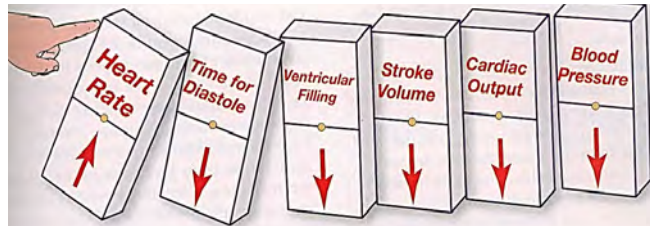I am extremely grateful to
Dr Cornelius Lee Chun Yin
Dr Dharshini Saminathan
Dr Tan Wan Ying
Dr Ang Yee Quan

My sincere wish is that medical students find this book an entrance to understanding the basics of Internal Medicine

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Ventricular Tachycardia

Facts:
- Tachycardia: Heart rate > 100 bpm
- Tachycardia is 'bad' because as the heart rate goes up, the systole cycle time remains the same, but diastole cycle time is reduced. This results in reduced time for ventricular filling, decreased stroke volume and subsequently decreased cardiac output.
- Often, a premature ventricular contraction (PVC) (R on T phenomenon on ECG) sets of VT. Many VT occur when there is presence of R on T phenomenon.
- The sequence of events:
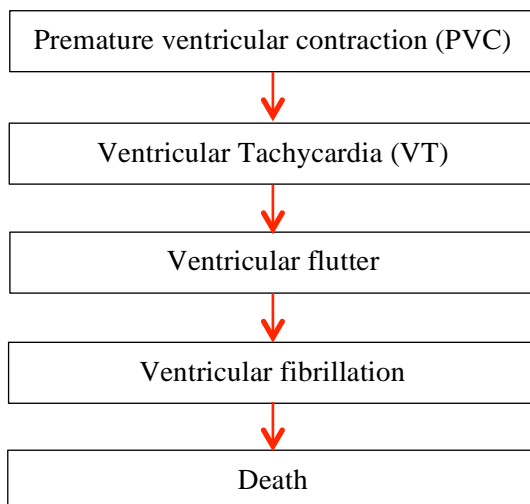
VENTRICULAR TACHYCARDIA
Can often deteriorate into

Ventricular FLUTTER
&
Ventricular FIBRILLATION

VT rapidly becomes VF
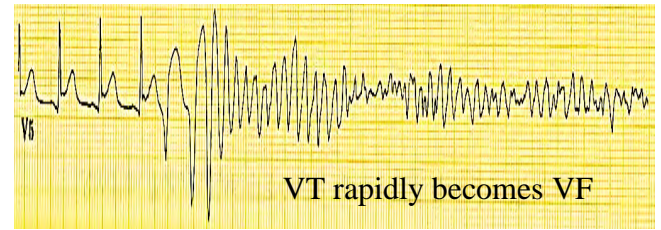
Resuscitate the patient by defibrillating (asynchronously) as there are no T waves in VT. If synchronized, the machine will never defibrillate because the T wave is never found.

Ventricular Tachycardia (VT) can be classified as:

1. **Primary**: Occurring within 24 hours of Acute Myocardial Infarction (AMI).(often first few minutes or hours)

2. **Secondary**: Occurs with underlying damaged heart (after months of AMI). The fibrous tissue after the infarct may throw VT all the time. Treatment is Intracardiac defibrillator (ICD)

Ventricular Tachycardia (VT) can also be classified as:

1. **Non-sustained**: Lasting <30s

2. **Sustained**: Lasting >30s or <30s on active treatment.
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The defibrillator / DC cardioversion

Settings:
1. **Synchronized**: Machine scans for T waves and avoids shocking it.
2. **Non-synchronized**: Machine fires regardless of T wave (does NOT scan for T waves).

Synchronized (Cardioversion) indicated for:
1. Atrial fibrillation (AF)
2. Atrial Flutter
3. Supraventricular tachycardia (SVT)

Non-synchronized (Defibrillation) indicated for:
1. Ventricular tachycardia (VT)
2. Ventricular fibrillation
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Supraventricular Tachycardia (SVT)
VS
Ventricular Tachycardia (VT)

- Most supraventricular tachycardia (SVT) appears as a NARROW QRS complex tachycardia on ECG.
- Some SVT presents with aberration, appearing on ECG as a WIDE QRS complex tachycardia.
- Ventricular tachycardia (VT) appears as a **WIDE QRS complex tachycardia** (WCT) on ECG. & 80 % of WCT are VT

Figure: Atrial flutter. An example of supraventricular tachycardia (SVT). Note the narrow QRS complex tachycardia.

Figure: 2:1 Atrial flutter. An example of supraventricular tachycardia (SVT). Note the narrow QRS complex tachycardia.
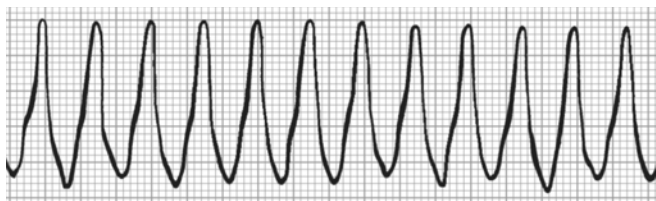

Figure: Ventricular tachycardia (VT). Note the wide QRS complex tachycardia.
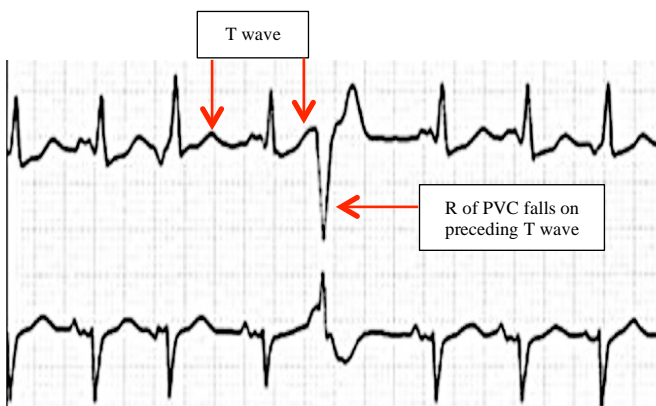
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R on T phenomenon

Figure: R on T phenomenon, suggesting a premature ventricular contraction (PVC). 'R' wave of an early premature beat falling on the peak of the T wave of the preceding beat, causing ventricular tachycardia.

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Case study
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It is 7pm and you are on duty at the casualty department. A patient presents to you with chest pain 1 hour ago, then palpitations and tachycardia 30 minutes ago. An ECG was done and revealed wide QRS complex. What is your diagnosis?

Answer: Ventricular Tachycardia (VT).

Explanation:
The sequence of symptoms for VT:
1st - Chest pain
2nd - Palpitations with Tachycardia
3rd - wide QRS Tachycardia
The probability of VT is very high in this situation

If palpitations appear first <u>then</u> chest pain <u>or</u> both occurring at the same time, you cannot diagnose VT "<u>only on history</u>" unless an ECG is done.

Question: How would you manage this patient?

Answer:
1. Gain IV access as soon as possible.
2. If BP stable, give IV amiodarone.
3. If BP unstable and dropping fast, drop the ECG (treat as VT regardless of ECG), Non-synchronized defibrillation 200 joules.

*** Note that blood pressure cannot be used to diagnose SVT or VT, as both these conditions can manifests as hypertension or hypotension. Stabilizing the patient's BP is a priority. A patient with stable BP gives more time for a doctor to treat. Therefore if BP dropping fast, drop the ECG and start resuscitating!
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Diagnosis of VT can be made if

1. ≥ 3 PVC occurring together in a row. (Non-sustained VT).

Or

2. Wide QRS ≥ 4 small squares tachycardia

Extra:
- If QRS complex > 160 msec (>4 sm sq), diagnose VT.
- If QRS complex < 140 msec (<3 sm sq), probably SVT.

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The concept of ventricular tachycardia

1. AV dissociation
2. Fusion beats
3. Capture beats
4. Concordance
5. Extreme Right Axis Deviation
6. Others (Josephson sign, Brugada sign)

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Atrio-ventricular (AV) dissociation

In a normal sinus rhythm there is a 1:1 P:QRS relationship. '*(atrio-ventricular __association__)*'
Let's say you disconnect the conducting system between the atria and the ventricles. Atrial sinus beats continue (constant rate of P waves) (Figure 41), and ventricles show beats of wide QRS complex – slower conduction.
The ECG,when recorded, will show the P waves and QRS waves without a '*P-QRS*' relationship. The result is an *atrioventricular (AV) dissociation*.
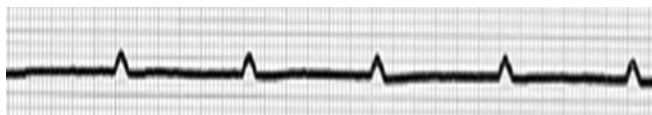
Figure: Example of isolated atrial activity, with only P waves. (NO QRS)
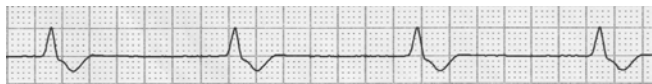
Figure: Example of isolated ventricular activity, with only wide QRS complexes. (NO Ps)

Figure: Example of AV dissociation. The ECG strip shows superimposed isolated atrial and ventricular electrical activity *WITHOUT a* fixed '*P-QRS*' *relationship.*

As a result of AV dissociation in VT, there are P on T waves / QRS complex appearing on the ECG as a notch on the T wave/ QRS complex. It distorts the appearance of the waves. In an ECG of wide QRS complex, if you can find one proof of P wave notching, then there is AV dissociation.

Figure: Ventricular Tachycardia. Note the notches (red arrows) on the wide QRS complexes.

*** Note: Sometimes the P wave notches can't be appreciated, in that case, keep in mind that practically ***ONLY VT can appear in ECG with QRS complex > 4 sm sq***. This enables diagnosis of VT without the P wave notches.

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Fusion beats

Sometimes 2 currents can start simultaneously
- A: From the ventricles, a slow ventricular conduction results in a wide QRS complex.
- B: From the SA node down the normal conduction system, a fast current having a ventricular conduction causes narrow QRS complex.
- When both A and B currents meet, the resultant wave is a fusion wave.
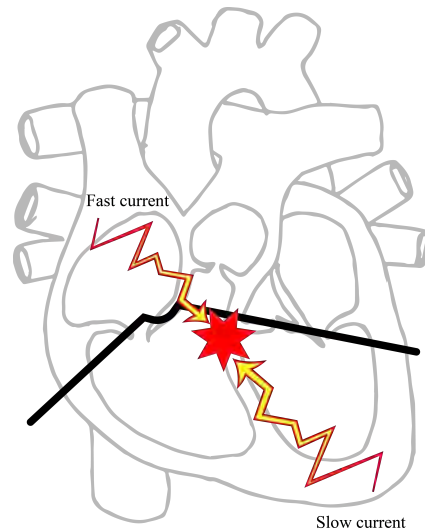- Only found in 5% of VT cases. 95% of VT cases won't have this.

Fast current

Slow current

Figure: Illustration of the fast current (narrow QRS) and slow current (wide QRS) meeting, resulting in a fusion beat.
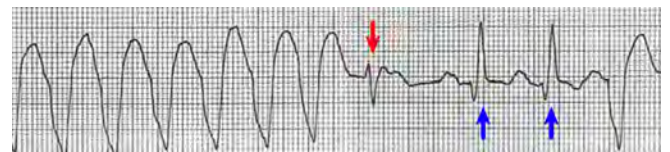
Figure: VT with captured fusion beat (red arrow) and captured beats (blue arrows).

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Capture beats

Occurs in 5% of VT patients. It happens when the normal impulse
(From SA ➔ AV ➔ His bundle ➔ Purkinje fibres) "captures" the ventricles and stimulate them and produce a normal SINUS heartbeat in the middle of the VT.
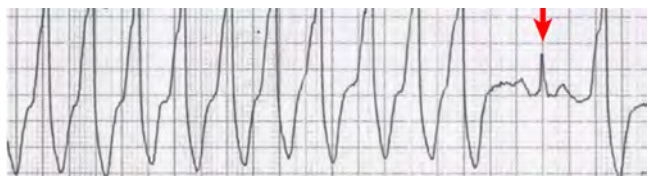

Figure: Ventricular tachycardia with capture beat (red arrow). It occurs in VT when the heart manages to capture a beat. Diagnose as VT when at least 1 seen.

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Concordance

In ventricular tachycardia, there are 2 types of concordances.
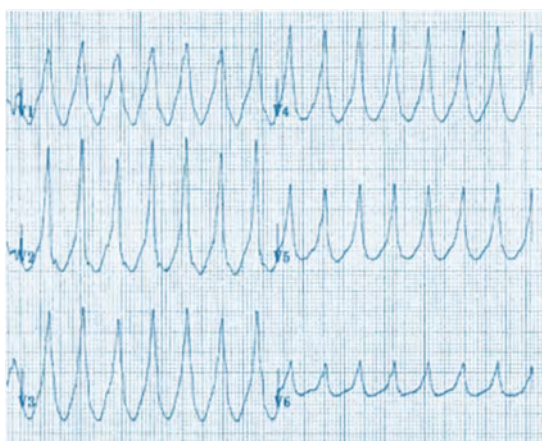1. Positive concordance
2. Negative concordance

Figure: VT with positive concordance. All R complexes in leads V1-V6 are upright. Diagnose VT.
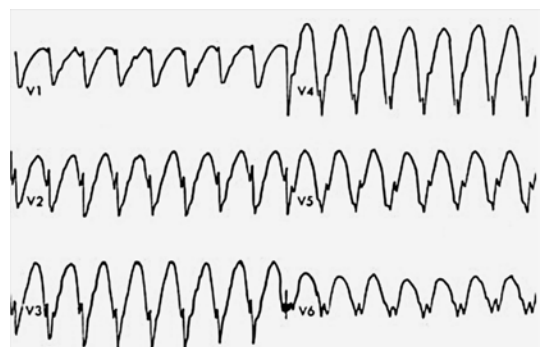

Figure: VT with negative concordance. All QS complexes in leads V1-V6 are downwards. Diagnose VT.

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Extreme Right Axis Deviation
(*Refer to cardiac axis explanation earlier)

Extreme right axis deviation:

Lead I & Lead aVF
NEGATIVE
(**Diagnose VT**)

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Brugada's and Josephson's sign in the diagnosis of Ventricular Tachycardia (VT)

In 1991, the Spanish Brugada brothers published a mathematical paper about the diagnosis of VT in the field of cardiology. Brugada's sign can be used to diagnose VT when there is no signs of capture beats.
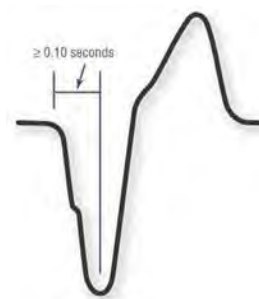

Figure 50: Brugada's sign. Diagnosis of VT is made when the time period between R wave and the deepest point of S wave ≥ 10 ms (0.10 s or 2.5 sm sq).
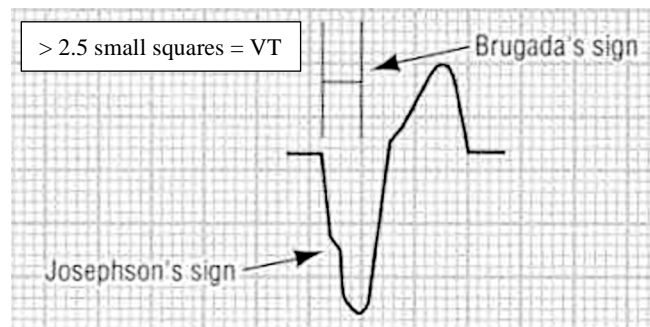

Figure 51: Josephson's sign. Note the notch in the QRS complex (arrow).
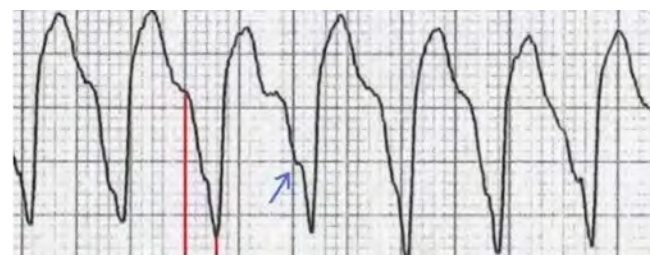

Figure 51: Brugada's sign (red lines) and Josephson's sign (blue arrow).

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Myocardial Infarction (MI)

Facts:
- About 5-10% of MI patients *may* die at admission and most die in the first few hours, usually *electrical* death [VT].
- 10% of MI patients re-infarct within 12 months post-treatment.
- If an MI patient dies within the first 24 hours, the mechanism of death is **ventricular tachycardia (VT)**, progressing to **ventricular fibrillation (VF)**. 50% of the patients with MI, die from VT and VF during the first 24 hours.
- If an MI patient dies later during admission, the most likely cause of death is **cardiogenic shock**.
- In a 47-year-old gentleman with underlying MI risk factors, the left anterior descending artery is most commonly blocked (ECG: ST elevation at leads V_1-V_4). They are more likely to die than a 67-year-old counterpart with the same block→ he may have developed *collaterals*.
(*This is why the LAD artery is also called the widow maker artery*). The 67-year-old patient may have more collateral vessels.

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Infarct, Reciprocal leads and its Significance
"Same part, different view."

ECG appearance	
Infarct	**Reciprocal**
ST-elevation	ST-depression
Inverted T	(Tall) Upright T

The absence of reciprocal changes does not mean anything. The **presence** of reciprocal changes only **confirms** the diagnosis.

Eg: ECG if inferior MI shows ST-elevation in Lead II, III & aVF, and reciprocal changes in Lead I & aVL.

SITE	FACING	RECIPROCAL
INFERIOR	II, III, aVF	I, aVL
HIGH LATERAL	I, aVL	II, III, aVF
ANTERIOR	V1, V2, V3, V4	NONE
POSTERIOR	NONE	V1, V2, V3, V4

EMS12Lead.com

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Just for completeness:

SITE	FACING	RECIPROCAL
SEPTAL	V1, V2	NONE
ANTERIOR	V3, V4	NONE
ANTEROSEPTAL	V1, V2, V3, V4	NONE
LATERAL	I, aVL, V5, V6	II, III, aVF
ANTEROLATERAL	I, aVL, V3, V4, V5, V6	II, III, aVF
INFERIOR	II, III, aVF	I, aVL
POSTERIOR	NONE	V1, V2, V3, V4

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Question
State the 4 areas of MI and their corresponding ECF leads to diagnose them.
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Answer:

No.	Surface (of MI)	ECG leads
1.	Anterior (antero-septal)	$V_1 - V_4$.
2.	Inferior	II, III, aVF
3.	Lateral	I, aVL, V_5–V_6
4.	Posterior	V_7, V_8, V_9

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Correlation of ECG Leads with their heart surfaces and its supplying arteries.

Surface	Artery	Leads
Lateral	Circumflex	I, aVL, V_5, V_6
Inferior	Right Coronary	II, III, aVF
Anterior	Right Coronary	V_1, V_2
Septal	Left descending anterior	V_3, V_4

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The J point
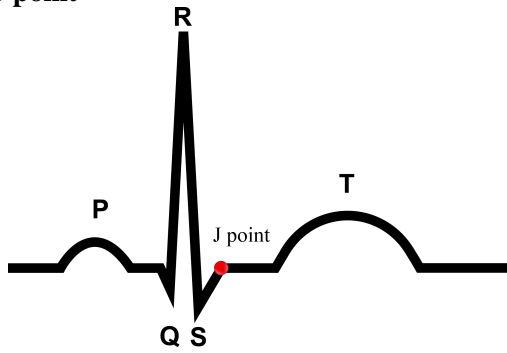
(Junction) J point [red dot].

Significance: Repolarization starts at the J point and continues until the T wave. It is heightened in ST-elevation.

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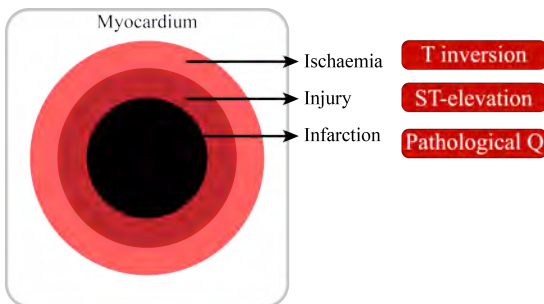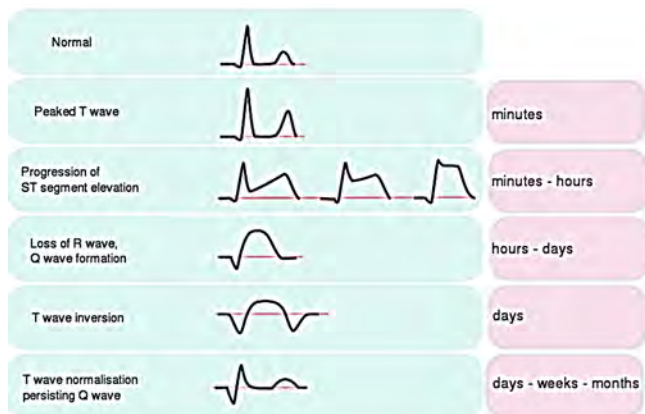**The pathological changes in MI and their corresponding ECG changes.**

Illustration of the pathological changes in MI and their corresponding ECG changes.

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Evolution of MI and corresponding ECG changes

Description:

| No.	Time	Changes
1.	-	• Normal
2.	Minutes	• (1st sign of MI) Localized tall T wave
3.	Minutes – hours	• ST segment starts elevating
4.	Hours – days	• Q starts forming • Loss of R wave (equivalent to Q wave formation) *Note: Normally, tallest T wave seen at Lead V_5.*
5.	Days – weeks	• T inversion • Pathological Q eave • Loss of R wave
6.	Weeks later	• Generally normalized • Persistent pathological Q wave

Clinical significance: A patient with MI may present to you in any of the phases mentioned above.
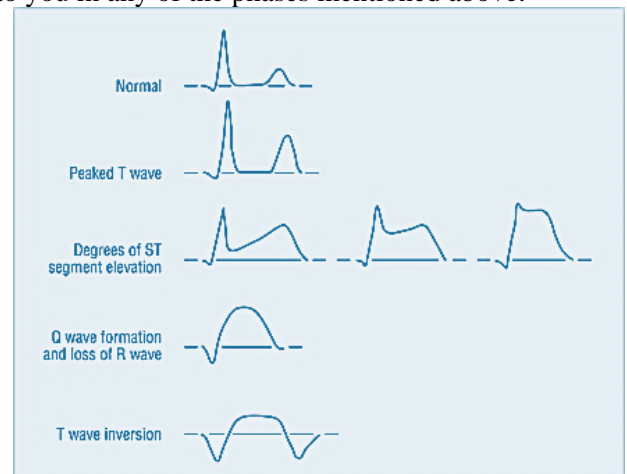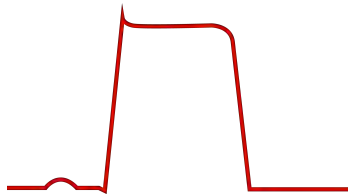

Evolution of MI and corresponding ECG changes
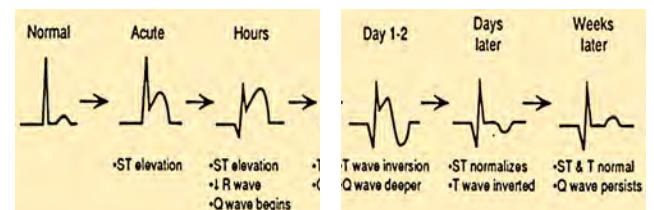
The ECG changes in the evolution of MI.
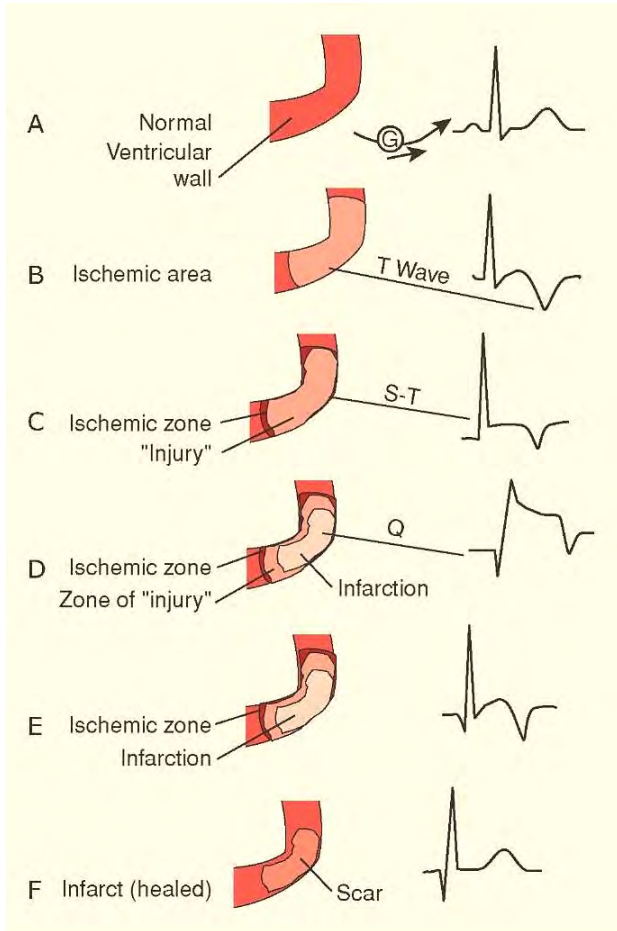
The ECG changes in the evolution of MI.

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The Tombstone appearance (in MI)

Tombstone appearance in the ECG of MI patients. It is the combination of QRS complex, ST elevation and the T wave, producing a single monophasic deflection
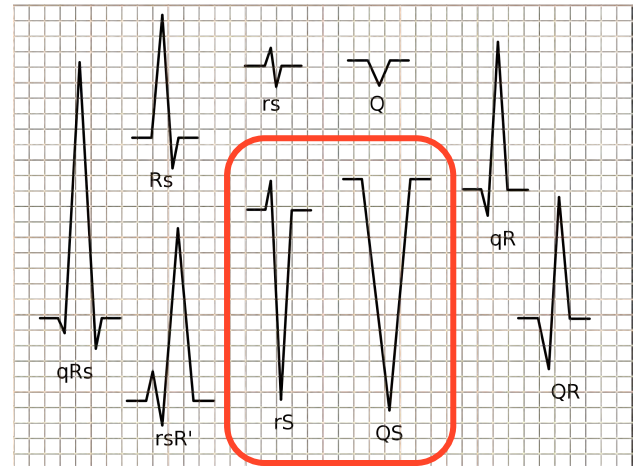
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The pathological Q wave ("Q")

Pathological Q waves are defined (on ECG) as **width > 0.03s**, which is almost the width of 1 small square (0.04s), and ± depth > R.

It is wise to compare the pathological Q waves ("Q") and physiological Q waves for further confirmation.

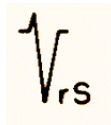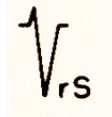**The physiological (normal) Q waves**

In a healthy adult, Leads I & aVL have physiological (normal) Q waves [< 0.03s].

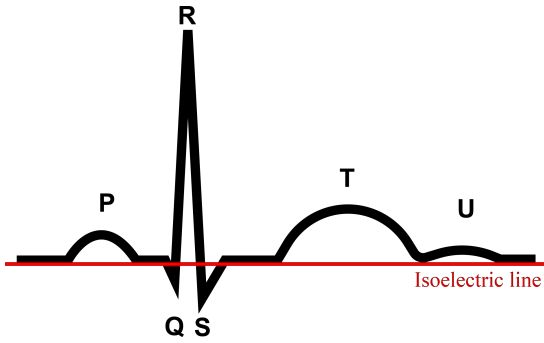_____

The rS and QS complexes

The names for the different QRS waveforms. Note the waveform of 'rS' and 'QS' complex (enclosed in red box).

In Lead V_1, an 'rS' pattern seen is or 'QS' normal.

Leads V_2, V_3 etc would normally show 'rS' pattern.

However, it is considered pathological if the 'QS' pattern appears in Leads V_2, V_3 etc.
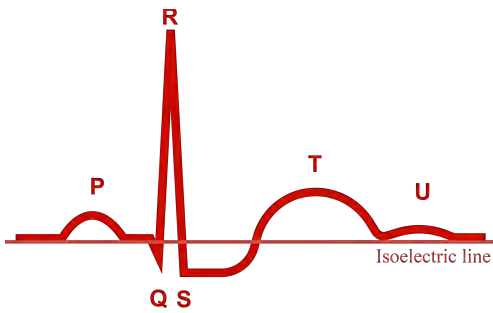
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The isoelectric line
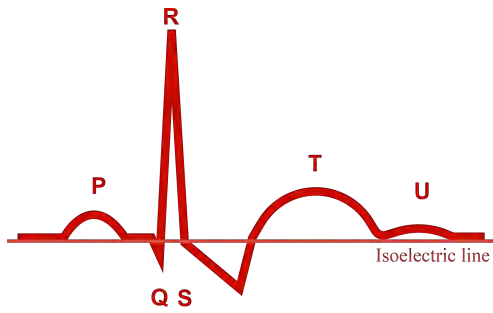

The isoelectric line. (red line in the diagram)

Deviations from the isoelectric line are termed:
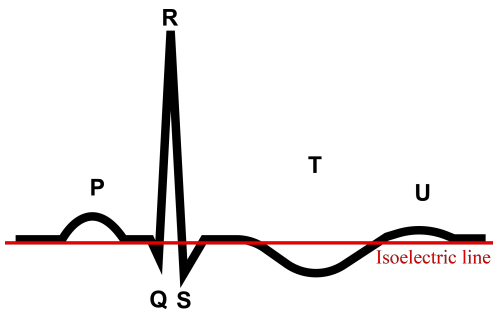1. Elevation
2. Depression

The following are examples of ECGs of unstable angina/ non-ST elevated MI:

Horizontal ST-depression from isoelectric line.

Downslope ST-depression from isoelectric line


T wave inversion

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The 3 types of tall T waves

	Tall, sharp T wave. Suggestive of hyperkalaemia.
	Tall, broad T wave. Suggestive of myocardial infarction.
	Tall, asymmetrical, broad based T wave. This is a common (normal variant) that is sometimes seen in healthy individuals. The T wave is rounded, its sides are not symmetrical, and it has a broad base. This is sometimes a finding in ECGs of Sabahans and Sarawakians.

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Peaked T waves
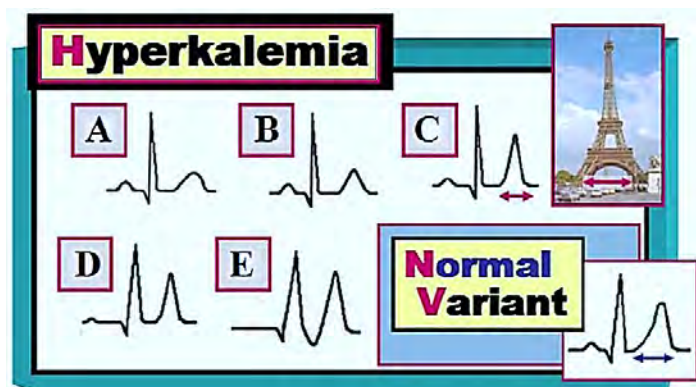
Shows ECG changes of hyperkalaemia and a normal variant of peaked T waves.

A. Normal ST-T wave
B. Peaking T wave (earliest change of hyperkalaemia)
C. T wave becoming taller and more peaked (K+ ~ 7-8mEq/L); It almost looks like the Eiffel Tower (Tall, peaked with a narrow base), in contrast to the T wave that is sometimes seen in healthy individuals (lower right box) in which it is asymmetrical and has a broad base.
D. P wave amplitude decreases, PR interval lengthens, QRS widens. (K+ >8mEq/L).
E. Disappearance of P wave (Sinoventricular rhythm) and the QRS becomes sinusoid. (K+ > 10mEq/L). Ventricular fibrillation usually follows.
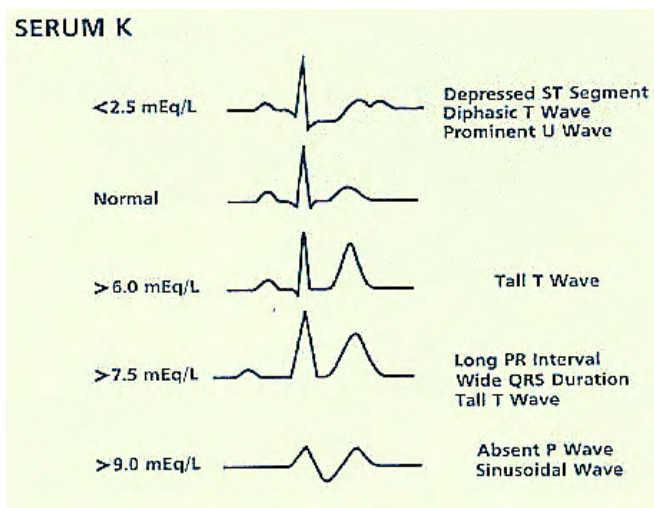
Shows ECG changes of hyperkalaemia.

Final point: All that produces tall, *peaked* T waves is *not* necessarily hyperkalaemia! Instead, the findings of T wave peaking should prompt one to consider a differential diagnosis of 3 *possible* causes – with the *clinical* situation (*as well as specific ECG features*) providing *KEY* clues as to which of the 3 is *likely* to be present.

1. **Hyperkalemia** - *Suspect* as the cause of T wave peaking when the *clinical* setting can produce hyperkalemia (*ie, renal failure, volume depletion, acidosis, potassium-retaining drugs*) - and - **when T waves are tall, pointed with steep ascent and near equally steep descent with a narrow base.**
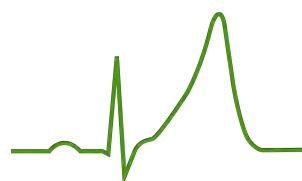

2. **Normal (Repolarization) Variant** - *Suspect* when T wave peaking has an *asymmetrical* ascent and descent and a *broader* base - especially IF the patient is *otherwise* healthy and *without* any apparent reason to have hyperkalemia.


3. **Myocardial ischemia -** *(in the area of the left ventricular posterior wall)* may sometimes present with the ECG finding of tall, *peaked* T waves in the *anterior* leads. Be aware that *ischemia* is a possible cause of T wave *peaking* in leads V_1, V_2, V_3 in a patient with known (or *suspected*) coronary artery disease & presents with chest pain - especially if there is *other* evidence on the tracing to suggest ischemia or infarction (*ie, inferior T wave inversion or ST depression*).

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The non-standard leads

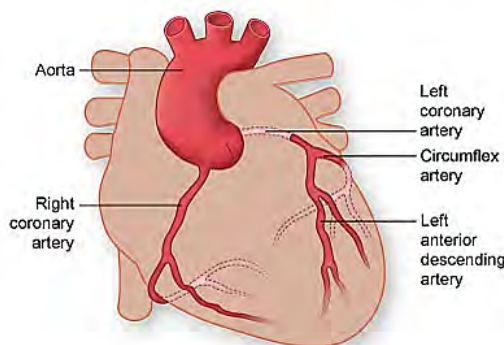| Non-standard leads	
Right ventricle (Right sided chest leads)	$V_1, V_2, V_3R - V_6R$
Posterior wall (Posterior leads)	V_7, V_8, V_9
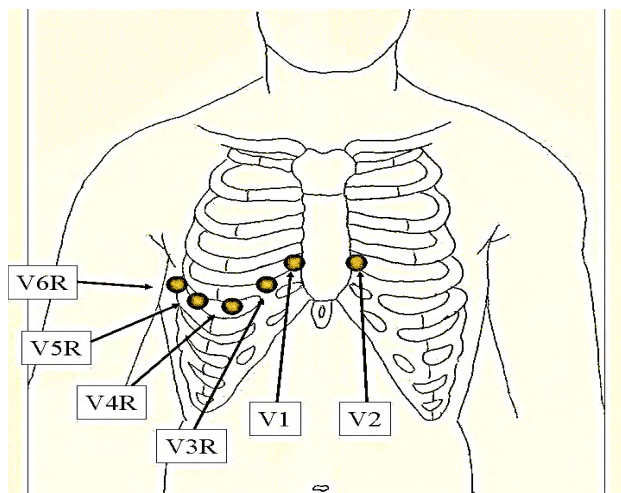
Right-sided chest leads
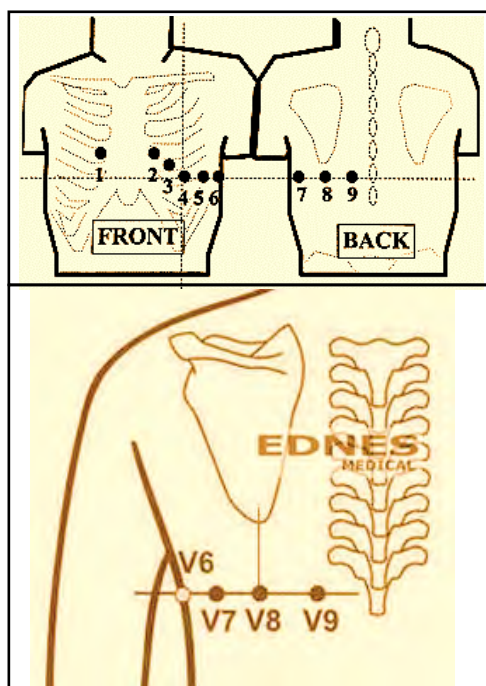
Posterior chest leads (with anterior leads).

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The Right Coronary Artery (RCA)

Note the right coronary artery (RCA). It usually supplies[more than 60% of the time; left circumflex other times] the right ventricular wall, the posterior part, and the inferior wall of the heart.

Clinical significance: 40% of patients with inferior STEMI (ST-elevation at Leads II, III & aVF) have right ventricular wall infarcts. If inferior infarct is diagnosed, place right-sided and posterior chest leads to detect the transient changes. It is 99% diagnostic.

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★★★★★
Right Ventricular Infarction
★★★★★

The right ventricle is $\frac{1}{6}$th the thickness of the left ventricular wall. The magnitude of current in the right ventricle is also smaller than that of the left ventricle.

"Patient with typical history of MI, **hypotensive**, raised JVP, lung bases clear. Clinically diagnosed as right ventricular infarction!"

"On ECG, focus on V_1. If V_1 shows ST elevation, diagnose right ventricular infarction."

"Usually when ST-elevation at Lead III > Lead II, there is a right ventricular infarction."

ECG shows:
1. Inferior infarct: ST-elevation at Lead II, III & aVF.
2. Right-sided ECG shows **ST-elevation from Lead V_1, V_2 & V_4R.**

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Treatment of Right Ventricular Infarction
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Commonly, volume replacement via intravenous fluids is indicated in hypovolaemia and/or hypotension. It is, however, contraindicated in (heart) pump failure.

Contrary to that, right ventricular infarction is the only 'pump disorder' that you treat by giving IV fluids and NOT ionotropes! (Frank-Starling's Law)

Treatment:
1. IV 200cc normal saline, up to 1L.
2. Continuously monitor lung bases for crackles.

Explanation: Frank-Starling's Law of the heart states that stroke volume increases in response to increase blood volume filling the heart (end diastolic volume) when all other factors remain constant. In right ventricular infarction, the administration of intravenous fluids increases the blood volume, which stretch the right ventricular muscles, and subsequently increase the force of contraction and stroke volume, keeping the right heart pumping.

_____

★★★★★
Posterior Myocardial Infarction
★★★★★

"If a patient is diagnosed with inferior STEMI, perform a right and posterior ECG. Be prepared to waste ECG paper…" [+ve in about 40%]

Anterior Leads V_1 and V_2 are reciprocal leads for posterior leads.

A patient with typical history suggesting myocardial infarction, but ECG shows:
- Leads V_1, V_2 & V_3 showing ST-depression with upright T wave.

At this point, a posterior ECG should be done. The ECG of posterior Leads show:
- Leads V_7, V_8 & V_9 showing ST-elevation.

Explanation: In a case of posterior MI, the Leads V_7, V_8 & V_9 directly show the surface of the infarct, whereas the reciprocals are V_1, V_2 & V_3.

| SITE	FACING	RECIPROCAL
INFERIOR	II, III, aVF	I, aVL
HIGH LATERAL	I, aVL	II, III, aVF
ANTERIOR	V1, V2, V3, V4	NONE
POSTERIOR	NONE	V1, V2, V3, V4

_____

★★★★★
Case Study
★★★★★

It is Friday night, and you're the only attending doctor in the ward. State the 3 possible different presentations of MI patients.

Answer:
1. (**Rarest and most dangerous. Pace immediately**) Hypotensive + severe Bradycardia: Suggestive of complete heart block.
2. Hypotensive + Tachycardia: Suggestive of cardiogenic shock
3. Normotensive and normal heart rate.

"Everybody got into trouble in the first 2 scenarios, because they got used to the 3rd. They never saw it coming."

_____

The different types of cardiac disorders seen in CCU patients.

_____
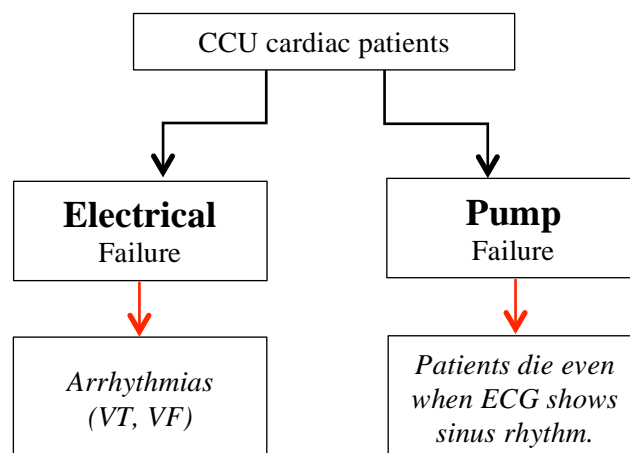
KILLIP Classification of Cardiac Failure

KILLIP class	Clinical features
I	No signs of LV failure
II	S3 gallop, bi-basal crackles
III	Acute pulmonary edema
IV	Cardiogenic shock

_____ END _____

Relevant Questions

1. List the risk factors for myocardial infarction.
   - Smoking
   - Hypertension
   - Hypercholesterolaemia
   - Diabetes Mellitus
   - Obesity
   - Occupation
   _____

2. State the accepted (target) blood pressure for a patient with underlying diabetes mellitus.
   - $\frac{130}{80}$ mmHg
   _____

3. State the target LDL level for a patient with established underlying heart disease.
   - 1.8 mmol/L

   Note: *Patient should be on lifelong statins and aspirin.*
   _____

4. State the 2 ECG criteria for diagnosing myocardial infarction? (Also the indication for streptokinase)
   - ST-elevation
   - **Newly** diagnosed/ presumed LBBB
   Note: The keyword is '**NEWLY'**.
   _____

5. State the side effect of administrating streptokinase.
   - Hypotension

   Note: *When the patient becomes hypotensive after streptokinase administration, streptokinase should be discontinued.*
   _____

6. State the absolute contraindication for streptokinase.
   - (life-long contraindication) History of intracranial haemorrhage.
   - < 3 months post surgery.
   - < 3months of ischaemic stroke
     [*see CPG AMI 2014*]
   _____

7. Medications and their indicated situations.

No.	Medication	Indications
1	ACE inhibitors	DM + HTN
2	β-blockers	Angina + HTN
3	CCB	COPD/ Asthma
4	Diuretics	Isolate systolic HTN

_____
8. State the relative contraindication for streptokinase.
   - History of stroke
   - High systolic blood pressure exceeding 180mmHg (systolic BP > 180 mmHg).

   Note: *If these patients present with systolic BP > 180 mmHg, give IV nitrates. Chest pain may be reduced along with the blood pressure.*
   _____

9. Why is intramuscular (IM) morphine contraindicated in a patient with myocardial infarction?
   - The patient would have already been on anti-platelets and anticoagulants. IM morphine may adversely cause a hematoma at the injection site.
   _____

10. After the administration of nitrates, other than dropping blood pressure, what else can it aggravate?
    - Tachycardia may worsen.
    _____

11. List contraindications of β-blockers.
    - **A**sthma
    - **B**radycardia,
    - **B**locks (3rd degree heart block)
    - **C**OPD
    - **D**ecompensated heart failure (bi-basal crackles heard on auscultation)
    - Peripheral vascular disease
    _____

12. When are ACE inhibitors indicated in a patient with heart failure?
    - When the heart's ejection fraction < 40%, even if heart is not in failure.
    _____

13. List 3 medications that prolong the life of a patient with heart failure.
    Mnemonic: **ABS** – Anti-lock-braking system
    - **A**CE inhibitors
    - **β**-blockers
    - **S**pirinolactone
    _____ END _____ _____

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Heart Blocks

The absolute normal about sinus rhythm:
Every P wave is followed by a QRS complex; Every QRS complex is preceded by a P wave.
(look at Lead II)

_____

The severity of heart blocks in sequence
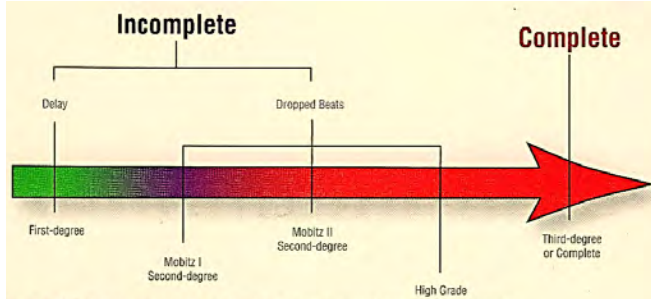
Less dangerous ⟷ **Very serious**

Figure : The severity of heart blocks in sequence (From less dangerous to very serious)

- 1st degree heart block

- 2nd degree heart block: Mobitz I
- 2nd degree heart block: Mobitz II

- High grade/ Advanced heart block
- 3rd degree heart block

_____

1st degree heart block

"1st degree heart blocks are 'harmless' and requires no treatment, unless they progress into 2nd degree or higher heart blocks. The patient becomes very ill."

Figure 73: 1st degree heart block ECG showing prolonged P-R interval.

ECG diagnosis:
1. Prolonged P-R interval.
   (>0.20s / >5 sm sq)

_____

2nd degree heart block

"Some P waves but no QRS complex"

A cardiac cycle with A P wave with no QRS complex *after it* is termed a *dropped beat*.

Mobitz type I, (also called Wenckebach's phenomenon. Karel Wenckebach first described it by feeling the carotid pulse.)
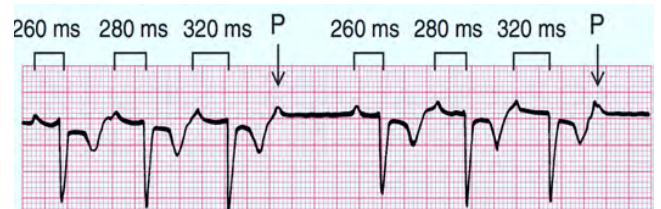
Figure 74: Mobitz type I 2nd degree heart block, showing progressive lengthening of P-R interval preceding a dropped beat.

Mechanism

Figure : Sinus beats A, B and C. Sinus beat A passes through the AV node normally.
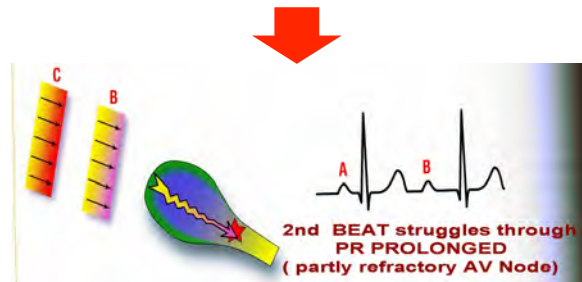
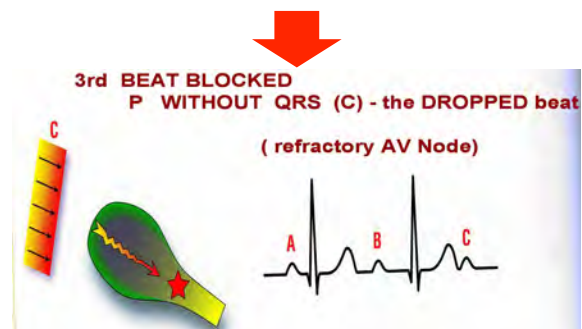Figure : Sinus beat B struggles through the AV node, resulting in a prolonged PR interval.

Figure : Sinus beat C completely blocked, as a result no current passes through.

- **Normal AV node**

- **(2) Partly refractory** AV node

- **(3) Completely refractory** AV node

- **(4) Dropped Beat**

2:1 type 2nd degree heart block (Emergency)

"Constant PR interval,
Constant pattern of: 2P – 1QRS – 2P – 1QRS"

Figure: 2:1 type 2nd degree heart block. Note 2 P waves precede a QRS complex.

ECG diagnosis:
1. Progressive lengthening of P-R interval, followed by a dropped beat.

Note: In the CCU setting, an inferior infarct can cause this. They usually recover without treatment.

On ECG, if noted "Group – gap – group" suspect Mobitz type I"
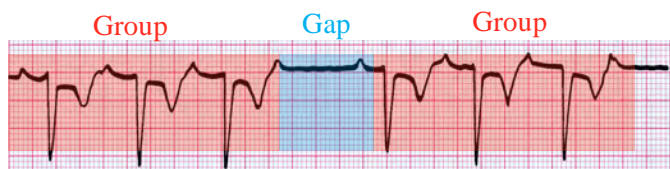
Figure : **Group (red) – Gap (blue) – Group (red)** pattern on ECG. Suggesting a Mobitz type I second degree heart block.
_____

Figure : 2:1 type 2nd degree heart block.

ECG diagnosis:
1. Constant PR interval in conducted beats
2. 2 P waves followed by 1 QRS complex.
_____

High grade / Advanced heart block

EMERGENCY! Pace them!

"Severity between 2nd and 3rd degree heart block"

Figure : High grade / Advanced heart block. The highlighted portion shows non-conducted P waves. Note that there is more than 1 P wave before a QRS complex; at least 2 consecutive P waves are blocked. *"Panic with 2 Ps"*
_____

Mobitz type II (Life threatening)
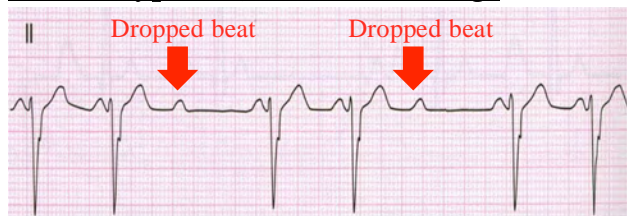
Figure : Mobitz type II phenomenon.

ECG diagnosis :
1. P-R interval **constant**, followed by a dropped beat. (**'P' WITHOUT a QRS**)

Note: Notice the P-R interval is constant preceding a dropped beat. This may progress to a complete heart block. The patient should be paced immediately.

3rd degree heart block

EMERGENCY! Pace them!

"Complete AV dissociation

$$P:QRS \neq 1:1$$

*A **wide QRS complex** with **slow R -R** rate,
with **normal, P** at a **fast P- P rate.**"*

"The problem with 3rd degree heart blocks is that it is easy to diagnose, but difficult to localize."

Possible sites of block include
- the His bundle, both RBB & LBB,
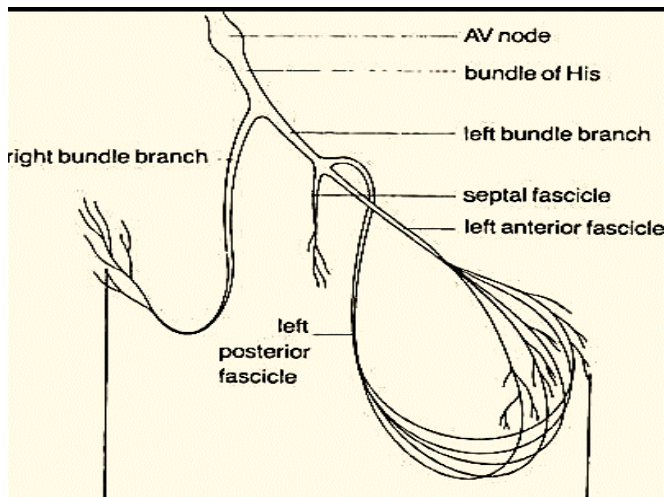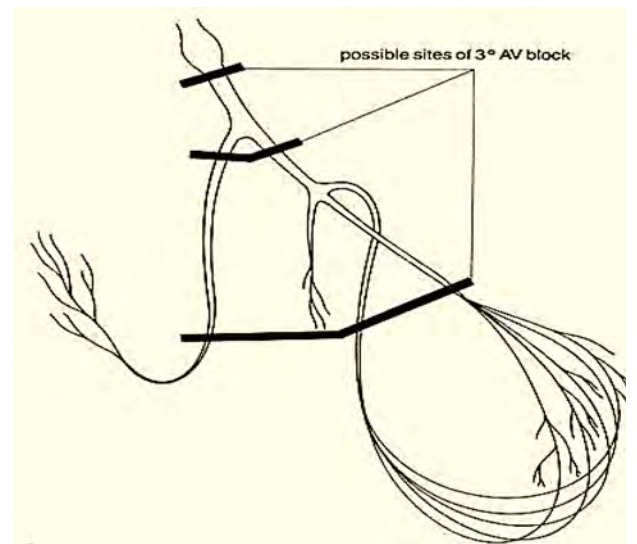- RBBB with blocked Left Anterior fascicle, Left Posterior fascicle.

Figure: The ventricular conduction system,
Below the Bundle of His, the conduction system divides into right and left bundle branches.
The right bundle branch remains intact, whereas the ***left divides into three separate fascicles***, namely the
> (1) Septal fascicle
> (2) Left Anterior fascicle
> (3) Left Posterior fascicle
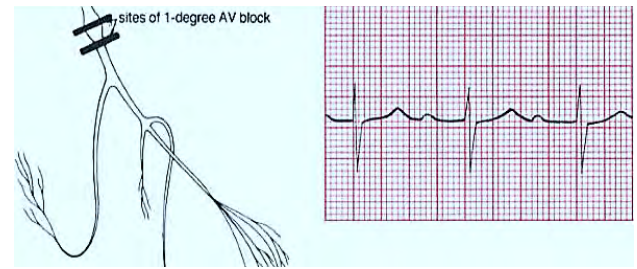
Figure : 1st degree AV block.

Figure : Mobitz type I block
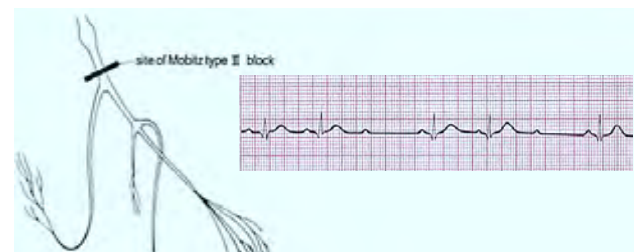
Figure : Mobitz type II block

Figure: 3rd degree heart block
Possible sites of block include
- the His bundle,
- both RBB & LBB,
- RBBB with blocked Left Anterior fascicle, Left Posterior fascicle.
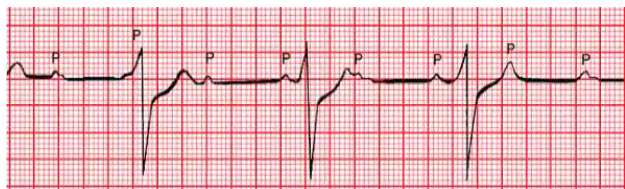
Figure: Third-degree AV block.
The P waves appear at regular intervals, as do the QRS complexes, but they have nothing to do with one another. The QRS complexes are wide, due to a ventricular origin.

Hypoxia and **acidosis** can fasten the death of a complete heart block patient. Treat with oxygen, bicarbonate ($CaCO_3$).
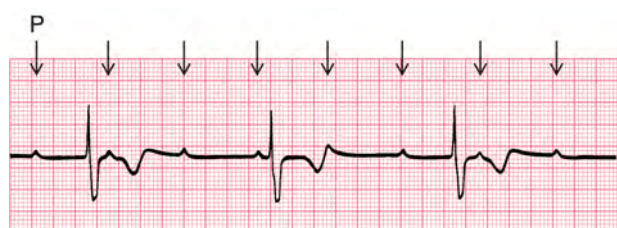

Figure: 3rd degree heart block. ECG showing complete AV dissociation, wide QRS complex with slow R -R rate, and fast P -P rate.

ECG diagnosis:
1. Complete AV dissociation
2. Wide QRS complex, slow rate
3. Fast P wave rate.

Note: The patient will likely die if pacing is not done immediately.

———————————————————————

★★★★★
Question:
How would you differentiate complete heart block from ventricular tachycardia?
★★★★★

Answer:

3rd degree heart block	Ventricular tachycardia
P rate > QRS rate (*More P than QRS*)	QRS rate > P rate (*More QRS than P*)
P-P and R-R interval equal, but…	
R-R > P-P interval	P-P > R-R interval

———————————————————————

★★★★★
Question:
List differential diagnoses for a non-conducted P wave
★★★★★
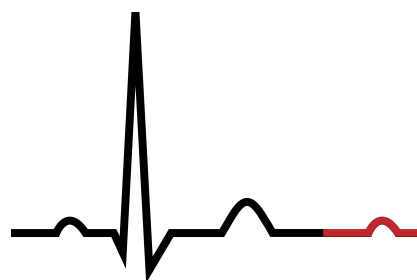
Figure 80: ECG showing non-conducted P wave (red).

Answer:
1. **Non conducted Atrial ectopic**
2. **Mobitz Type I** heart block
3. **Mobitz Type II** heart block
4. **High grade / Advanced** heart block.
5. **3rd degree** heart block

————————END————————

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Supraventricular Tachycardia (SVT)

Supraventricular Tachycardia (SVT)

*"Any tachycardia [**heart rate >100/minute**] resulting from a supraventricular current origin."*
➔ *[= __AT or ABOVE__ the AV Node]*

Supraventricular current may originate from:
1. Sino-atrial node
2. Atrio-ventricular node
3. Atrial tissue (muscle)

However, strictly based on the definition alone, the term 'supraventricular tachycardia' is an umbrella covering both physiological and pathological tachycardia.

Example: A healthy person experiencing tachycardia after jogging is technically a supraventricular tachycardia.

Example: A frail elderly experiencing an atrial fibrillation is also a supraventricular tachycardia.

It should be kept in mind that when some books mention it, they only refer to some disorders.

_____

Revision question: Why is tachycardia bad for us?
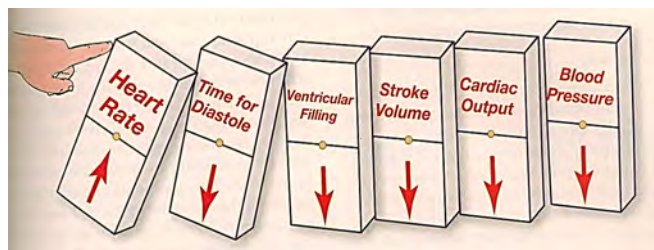
Answer: As the heart rate increases, the diastolic phase of the cardiac cycle shortens, while the systolic phase remains constant. The shortened diastolic phase reduces filling time and subsequently reduces ventricular filling volume. This reduces stroke volume and thus decreased cardiac output & BP

_____
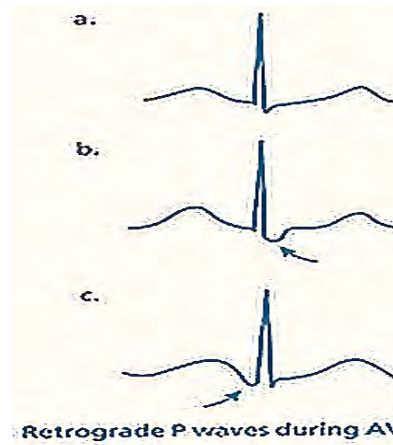
The possible locations of P wave on ECG in SVT

The P wave can appear on ECG:
a) During QRS complex
b) After QRS complex
c) Before QRS complex

_____

Revision question: Define narrow and wide QRS complex on ECG.

Answer:
- Narrow QRS complex : < 3 sm sq
- Wide QRS complex   : > 3 sm sq

*** Note**: 'sm sq' stands for small squares.*
_____

Revision question: Describe the ECG appearance of low atrial ectopic currents in the context of its distance from the AV node.

Answer:
(P-wave occurring **before** QRS complex)
The current first flows "backwards" into the atria and reaches the atrial tissue 1st before reaching the ventricles , which causes an inverted P wave. (as seen in Leads II, III, aVF)
(Retrograde P-wave)

SA node firing
NORMALLY
And producing a
"normal SINUS" beat.
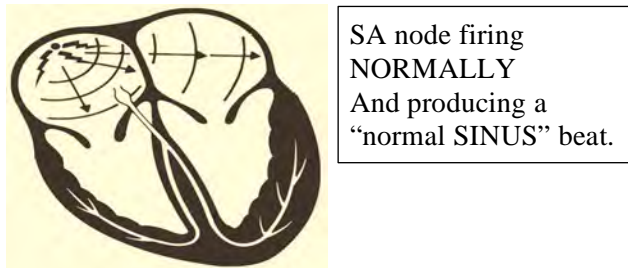
AV firing to produce a
NODAL / JUNCTIONAL beat
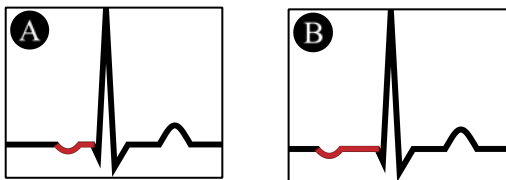
Figure 81: Figure A (left) illustrates an ECG of an atrial current originating near the AV node. Figure B (right) shows an atrial current originating further away from the AV node.

"The further the atrial current (inverted P) from the AV node, the longer the P-R interval; the closer the atrial current to the AV node, the shorter the P-R interval."
_____

Revision question: Describe the ECG appearance of a nodal/ junctional rhythm.

Answer:
P-wave occurring **during** QRS complex
g("buried" inside QRS -NOT ABSENT)
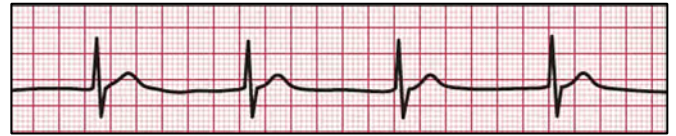
Figure 82: Nodal/ junctional rhythm.

ECG showing (Figure 82):
• Narrow QRS complex
• *No visible P wave*.
  (The P wave is hidden in the QRS complex, as both atria and ventricle receive current at the same time)
"The P is buried inside the QRS."
This rhythm may be seen in post-MI patients if **increased vagal tone** *suppresses SA node &*
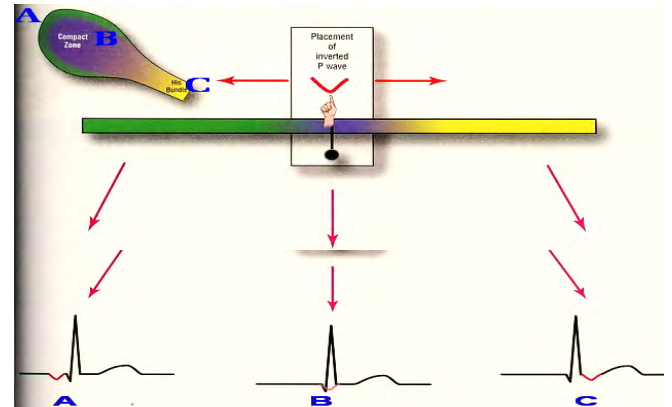**AV node becomes a 'temporary pacemaker'**
_____

Revision question: Describe the ECG appearance of an ectopic current *originating below the AV node, but above the His bundle.*
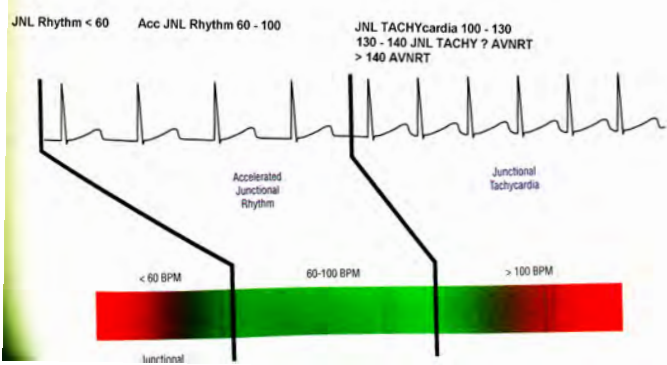
Answer:
(P-wave occurring **after** QRS complex)

Figure 83: ECG shows a late inverted P-wave **(Retrograde P-wave)** occurring *after* QRS complex, but before the T wave.
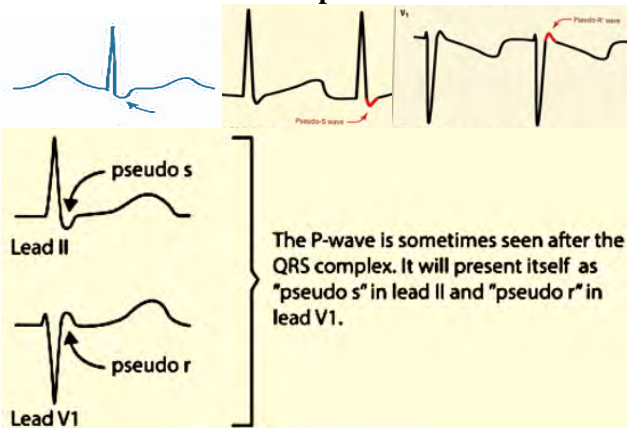

_____

The proper terminology for SVTs

No.	Rate	Terminology
1.	< 60	Nodal / Junctional rhythm
2.	60 – 100	Accelerated junctional rhythm
3.	100 – 140	Junctional tachycardia
4.	> 140	AVNRT

* AVNRT: Atrio-ventricular nodal re-entrant
       Tachycardia

_____

Pseudo-S and pseudo-r waves

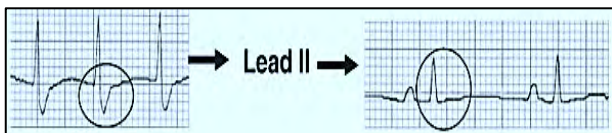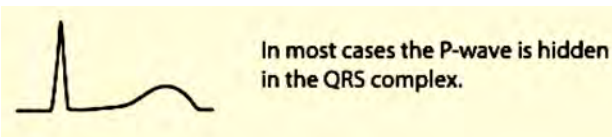Pseudo-S and pseudo-R' waves are actually retrograde P-waves.

Figure 84: ECG of lead II showing pseudo-S wave during AVNRT (left) disappearing on sinus rhythm (right).
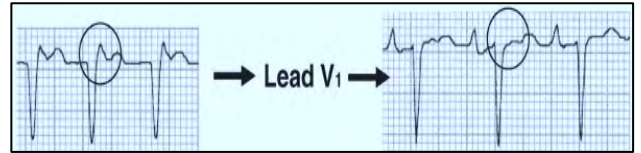
Figure 85: ECG of lead V₁ showing pseudo-R' wave during AVNRT (left) disappearing on sinus rhythm (right).
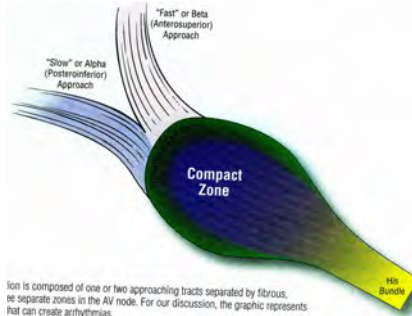
Clinical significance:

• When heart rate > 140bpm, and
• Pseudo-S and/or pseudo-r waves may be seen.
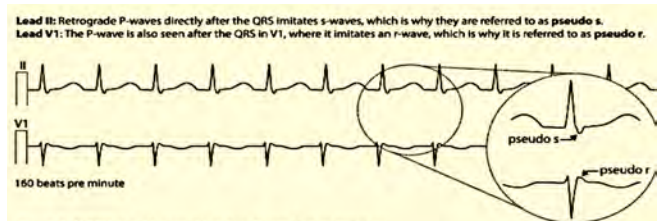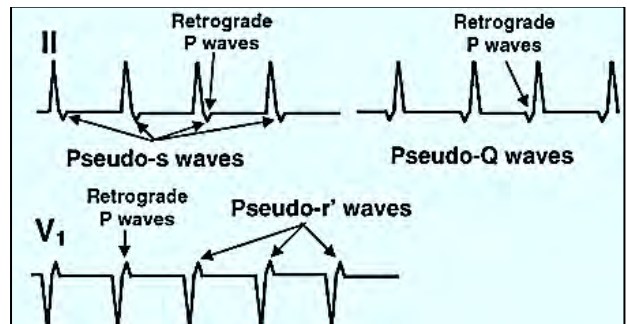

The diagnosis is **AVNRT**.

Figure 86: Pseudo-S and pseudo-r waves in AVNRT. In AVNRT, the retrograde P waves may emerge at the beginning or terminal portion of the QRS complexes, and can be mistaken for pseudo-Q or pseudo-S waves in leads II, III, and aVF, or as pseudo r' waves in V₁. These pseudo-Q and pseudo-S waves in lead II and pseudo-r' in V₁ should resolve upon conversion of the tachycardia to normal sinus rhythm.

_____

The mechanism of AVNRT

Some people, instead of having only one tract, they have 2 tracts going from the SA node to the AV node. The tracts are:

1. Slow α (with short refractory period)
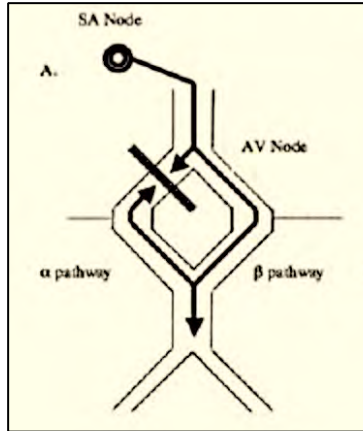2. Fast β (with long refractory period)

Figure 87: Normal cardiac action potential from SA node travels down the atrial tissue to the AV node. In susceptible individuals with 2 tracts (α and β), the current travels down both pathways simultaneously.

The fast β pathway conducts current faster down the His bundle, and at the same time, travelling back up the slow α pathway.
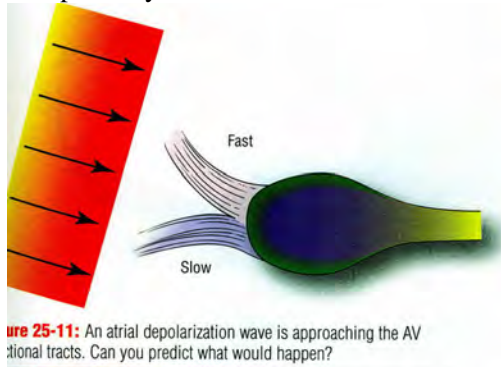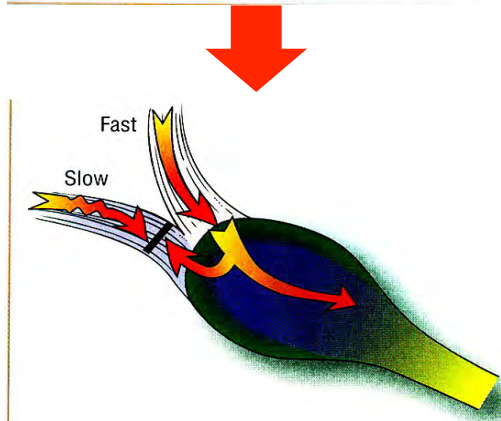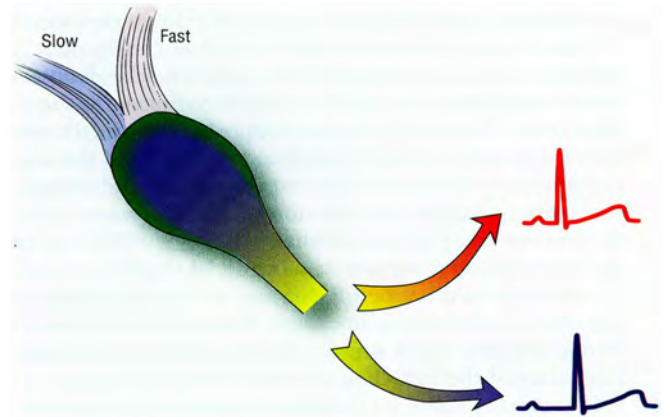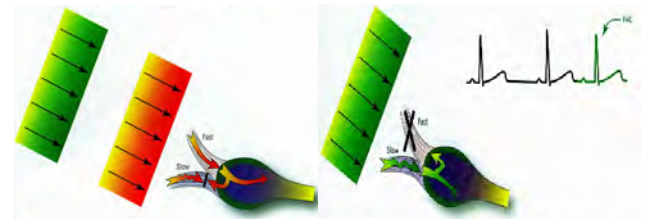

ure 25-11: An atrial depolarization wave is approaching the AV ctional tracts. Can you predict what would happen?


The 2 currents collide in the slow α pathway and cancel out each other. These occurrences are not a problem.

The 2 ECGs show DIFFERENT P-R intervals ➔ fast β & slow α currents through AV node

Rarely, immediately after a sinus beat, a premature atrial contraction (PAC) occurs. As the fast β tract is still refractory from the sinus beat, the impulse from the PAC travels down the excitable (non-refractory) slow α tract, down the His bundle, thus causing the heart to beat prematurely. This happens 99.9% of the time.
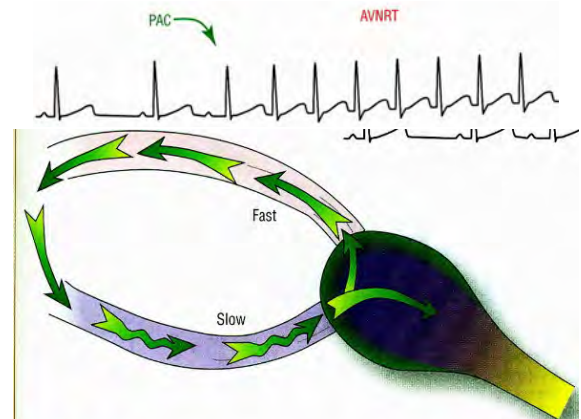
However, very occasionally, the problem arises when the current of a perfectly timed PAC travels down the slow α tract, and then carries the current to the fast β tract just in time when the fast β tract becomes excitable (non-refractory). The current from the fast tract re-enters the slow tract and a loop of current is formed, resulting in a **slow-fast type** 'A-V Nodal Reentrant Tachycardia'➔ AVNRT. See above and (Figure 89)

The commonest SVT in the E-D is the **slow-fast type** AVNRT.
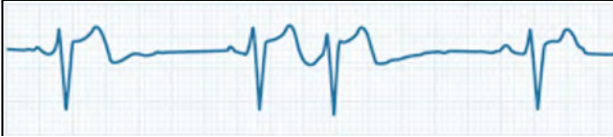The rare variant of SVT is the **fast-slow type** AVNRT (Figure 90).

Figure 88: Premature atrial contraction (PAC) after 2 sinus beats. The P-wave is buried in the preceding T-wave.
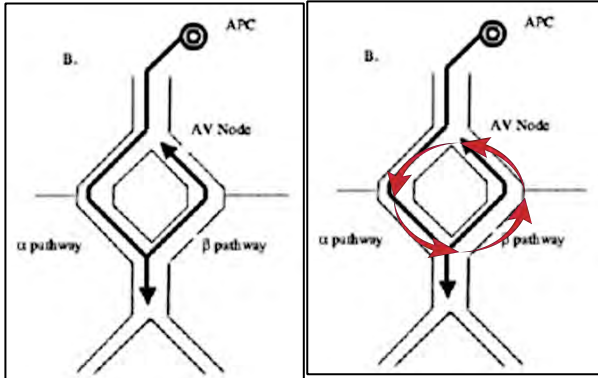

Figure 89: **Slow-fast type AVNRT**. The problem arises when the slow α tract carries the current of a perfectly timed PAC to the fast β tract just in time when it is excitable (non-refractory). The current then re-enters the slow pathway and a loop of current is formed, causing the slow-fast type AVNRT. This is the commonest type of SVT in the casualty department.
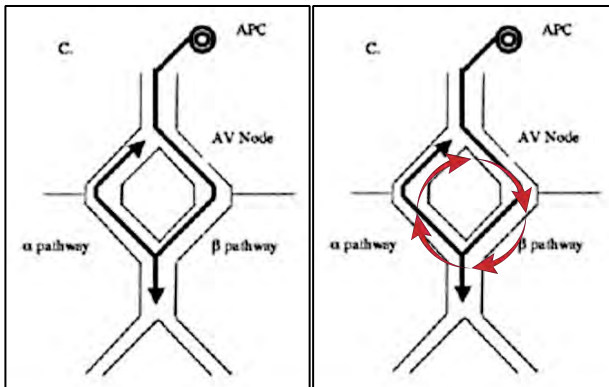

Figure 90: **Fast-slow type AVNRT** has a similar mechanism to the slow-fast type. The only difference is that the loop current travels in the opposite direction. This is an extremely rare type of SVT.

_____

Treatment for AVNRT

If blood pressure stable:
- Adenosine or
- Amiodarone or
- Lidocaine

If blood pressure unstable:
- *Synchronous* DC cardioversion

_____

ECG criteria for slow-fast type AVNRT:
1. Heart rate >140 bpm
2. P-waves often hidden and embedded in QRS complexes.
3. Pseudo-r may be seen in lead V1.
4. Pseudo-S may be seen in leads II, III or aVF.
5.

ECG criteria for fast-slow type AVNRT:
1. Heart rate > 140 bpm
2. Visible P-waves between QRS complexes and T-waves. (QRS-P-T)

_____END_____