Arrhythmia and ECG abnormality

Objectives:

1. To be able to approach patients with symptomatic bradycardia.
2. To be able to approach patients with sinus tachycardia.
3. Define the atrial fibrillation and its complications and be able to initiate therapy.
4. Identify ventricular arrhythmias based on ECG and initiate a management plan.
5. Identify atrial fibrillation and heart blocks on ECG.

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Editing File

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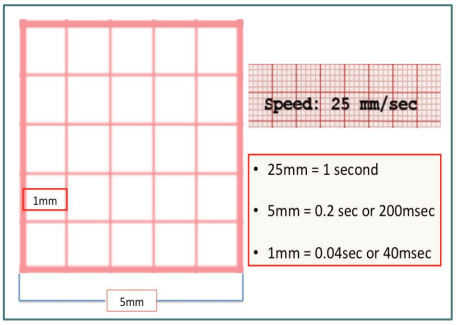
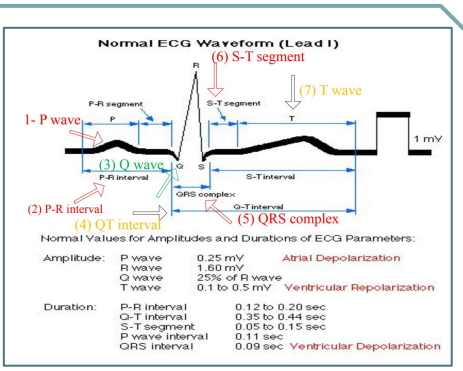
ECG Introduction

- **What is good about the ECG?**
  - Fast.
  - Cheap.
  - Non-invasive.
- Every **big square** contains **5 small squares.**
- Every small square is 1mm which **equals 0.04sec.**
  - So **ONE** big square equals **0.2sec.**



1. **P wave:** Atrial depolarization.
2. **PR interval:** Time of travel from SA node ⟶ AV node.
   - Normally it is **3-5 small squares.**
   - Prolonged in AV blocks.
3. Q wave.
4. QT interval.
5. QRS Complex.
6. ST segment.
7. T wave.



ECG Interpretation Approach

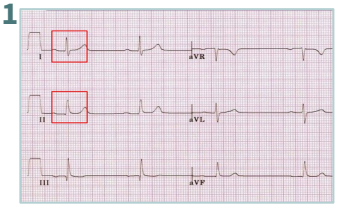
- This ECG interpretation approach to help to diagnose some common conditions. It is important to note that there are many other helpful approaches to interpret ECG and there are Many conditions not covered in this approach.
- The first thing you do is checking the patient's name. **Then look at the:**

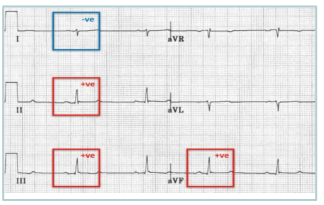
1. **Rhythm[1]:**
   - Check the R-R intervals if it is constant or not, to decide whether it is regular or irregular.
   - There are many causes of **irregular rhythm**, but the **most important** and common are:
     A. **Atrial fibrillation** (Irregular rhythm + absent P wave)
     B. Atrial flutter (Sawtooth pattern).
     C. Second degree heart block Type 1 (mobitz l) (**Progressive** PR prolongation then sudden beat drop).
     D. Second degree heart block Type 2 (mobitz ll) (**Fixed** PR prolongation + sudden beat drop).
     E. Sinus arrhythmia:
        - Common in pediatrics.
        - They just have irregular rhythm, NO beat drop nor P wave absence.

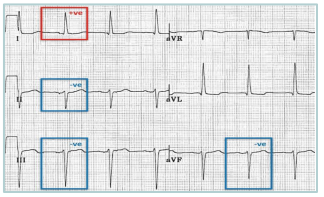
1. Distance between consecutive P waves and consecutive QRS complexes should be the same.
   - If the distance of the R-R intervals and P-P intervals is the same the rhythm is regular.
   - If the distance differs, the rhythm is irregular.

ECG Interpretation Approach Cont..

2. **Rate:**
   - **If regular rhythm:** Calculate big square between R-R
     - If > 5 big square (Bradycardia).
     - If < 3 big square (Tachycardia).
     - If between 3 and 5 big squares (Normal heart rate).
     - In standard ECG: Calculate the number of QRS complex in ECG and multiply by 6.
   - **The methods working for any ECG** (Eg. irregular rhythm)**:**
     - Calculate 30 large boxes, then count the number of QRS complex in these boxes.
       - Number of QRS complexes in 30 large boxes **X** 10 = HR
3. **Axis:** (Not Imp.)
   - Check **Lead I & II**:
     - If **both** are **positive** (pointing upward) = Normal axis[1]
     - If **lead I** is **Negative** & **lead II** is **Positive** = Right axis deviation[2]
       - They look at each other othe in ECG (اصحاب **اليمين** على سرر **متقابلين**)
     - If **lead I** is **Positive** & **lead II** is **Negative** = Left axis deviation[3]

1 

2 

3 

4. **P wave:**
   - Check for the P wave if it present or not.
   - What is the differential diagnosis of **ABSENT P Wave?**
     - **Atrial fibrillation:** Absent P Wave + Irregular rhythm.
     - **Supraventricular tachycardia (SVT):** Absent P Wave + Regular **Narrow QRS** complex tachycardia.
     - **Ventricular tachycardia (V tach):** Absent P Wave + Regular **WIDE QRS** complex tachycardia.
       - Any wide QRS complex tachycardia is considered VTuntil proven otherwise.
     - **Ventricular Fibrillation (V Fib):** An ECG finding of a rapid grossly irregular ventricular rhythm with marked variability in QRS cycle length, morphology, and amplitude.
5. **PR Interval:**
   - If the PR interval is **prolonged** {>0.2 sec (> 200 ms) (>5 small boxes)}, think about AV block (1st, 2nd, 3rd) & Hyperkalemia.
   - If the PR interval is **short** {<0.120 sec (120 ms) (< 3 small squares)}, think about **WPW** (if the duration is short always think of WPW regardless of the **delta wave**).
   - Remember 3rd degree heart block causing Variable P-R Interval length, So it will cause short and prolonged P-R interval.

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Heart Block (AV Block)

1. **First degree heart block: Regular rhythm**
   - The electrical impulses pass, but in a lower velocity.
     - **Finding: FIXED prolonged PR interval**
2. **Second degree heart block: Irregular rhythm**
   - **Type I (Mobitz I): Progressive prolongation** of P-R interval then **sudden QRS drop.**
   - **Type II (Mobitz II): Fixed** P-R interval (Prolonged or normal) + **Sudden QRS drop.**
3. **Third degree heart block (**Complete heart block**):**
   - **Regular rhythm** (because R-R is fixed).
   - When there is complete heart block, the AV node start to generate its own impulses (which are lower than SA node) so the patient will have **slow heart beats (lower side of normal).**
   - **P-P interval is fixed** (Because SA node can generate the P wave but not QRS complex due to the complete block).
   - **R-R interval is fixed** (AV node is generating the QRS complexes independently of the SA node)
   - **P-R Interval is variable** (Short, normal or prolonged).
   - **QRS complex might be wide (because it is from the AV node).**
     - **Wide QRS is usually dangerous DDX:**
       - 3rd degree block.
       - V Tach.
       - Hyperkalemia.
       - BBB.
       - WPW.

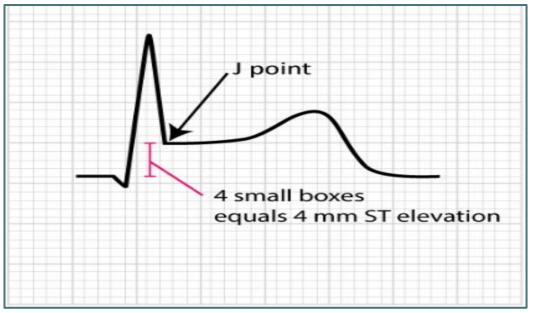
J point

J point

What's the j point?
- The connection between the S and T.

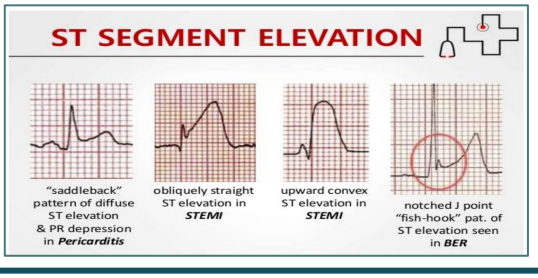
What's the isoelectric line?
- It's the segment between the T and P waves.

How can i know if the ST elevation is of clinical significance?
1. Compare the j point with the isoelectric line:
   - ≥ 1mm (one small square) is of clinical significance.
2. ST elevation in two or more consecutive leads in the same anatomy e.g. anterior, posterior, lateral.



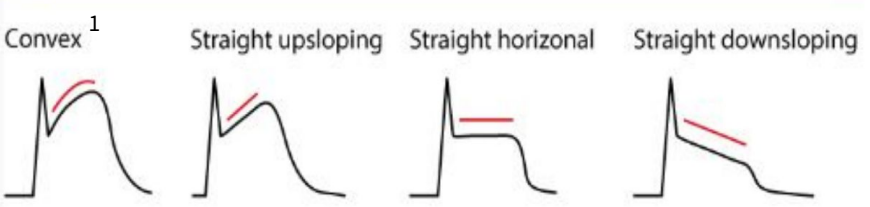
ST Elevation

What are the Most Important causes of ST elevation in ECG?
- **MI**
- **Acute pericarditis**
- LBBB
- Benign early repolarization (very common in young)



ST Elevation Cont'

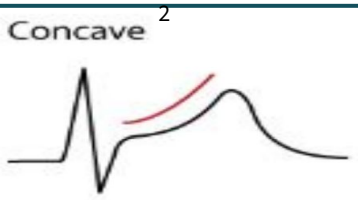
Characteristics of ST elevation caused by ischemia



ST-segment elevations caused by ischemia typically displays a convex or straight ST-segment. Such ST-segment elevations in presence of chest discomfort are strongly suggestive of transmural myocardial ischemia. Note that the straight downsloping variant is unusual.

What are the major ECG changes we can see in ischemia?

- ST elevation, ST depression, hyperacute T wave, T wave inversion, pathological Q wave.
- T wave changes can be due to ischemia but they are not specific.



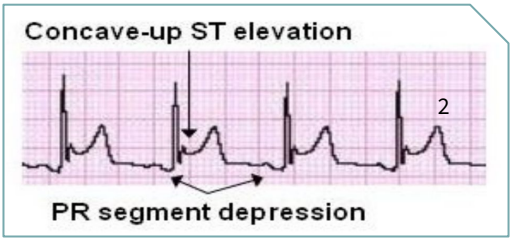
Non-ischemic ST-segment elevations are extremely common in all populations. They are characterized by a concave ST-segment and a greater distance between the J point and the T wave apex.

Typical non- ischemic ST-segment elevation

Acute Pericarditis

Acute Pericarditis

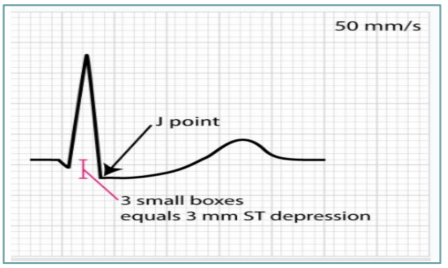
- **Usually diffuse ST elevation** and not in one anatomy.
- Can be associated with **PR depression (except in aVR it will be elevated)**
  - How to know if PR segment is depressed/elevated? compare it to isoelectric line (TP segment)
- No reciprocal changes
- The morphology of the ST segment



ST Depression

ST segment depression

- **≥ 0.05** mV (or 0.5 mm) in leads **V2 and V3.**
- **≥ 0.1** mV in all other leads.
- What's the **first** differential diagnosis of ST depression?
  - reciprocal change from ST elevation.



1. Convexity looks like a sad face (Sad= Bad), ischemic. ☹
2. Concavity looks like a happy face (Happy= Good), Non ischemic. ☺

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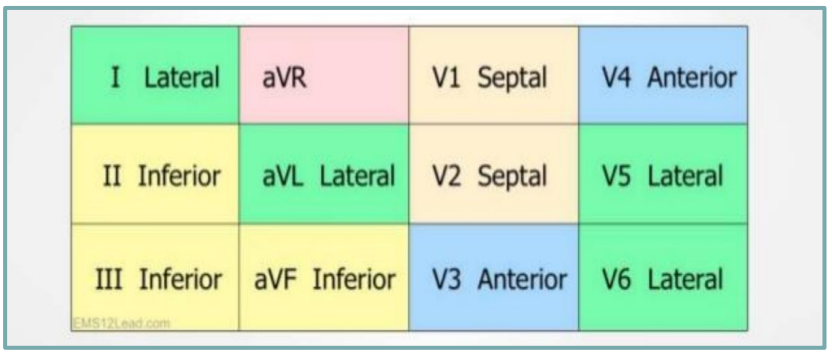
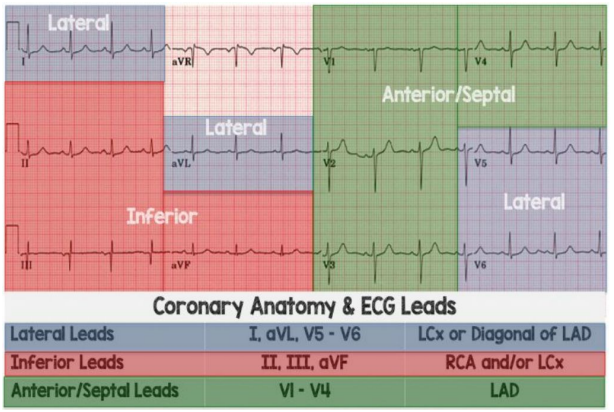
Lead Perspectives



Lateral	Inferior	Anterior	Septal
I, aVL, V5, V6	II, III, aVF	V3, V4	V1, V2



Reciprocal Changes

- **Reciprocal change is like a mirror, it happens due to ST elevation.**
- The inferior leads are mirrors to the lateral leads and vise versa.
- The anterior leads are mirrors to the posterior leads and vise versa
- **Each lead looks at the heart from a different view.**
  - **II, III, aVF looks at the heart inferiorly.**
  - **I, aVL, V5, V6 looks at the heart laterally.**
  - **V1, V2, V3, V4 looks at the heart anteriorly.**
- **Now let's say someone has MI. you'll have ST elevation on the leads looking at the affected part and ST depression on the opposite leads (think of it as if the ST elevation is dragging the electricity from the opposite leads and causing ST depression). So which lead is opposite to which?**
  - **lateral leads are opposite to inferior leads and vice versa.**
  - **Anterior leads are opposite to posterior leads and vice versa but you need 15-ECG leads to look at the heart posteriorly rather than the regular 12-ECG leads.**
- **Examples:**
  - If there is ST elevation in the lateral leads, **there might be** reciprocal changes (ST depression) in the inferior leads.
- **Can ST elevation be a reciprocal change to ST depression?**
  - No.
- **Please note:**
  - **Reciprocal changes doesn't always occur.**
  - **There are other causes for ST depression such as NSTEMI and LVH with repolarization abnormality.**
  -

Introduction

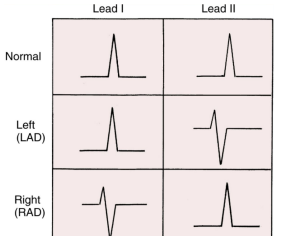
CARDIAC ARRHYTHMIAS

- An abnormality of cardiac rhythm is called a cardiac arrhythmia. Arrhythmia may cause sudden death, syncope, dizziness, palpitations or no symptoms at all.
- There are two main types of arrhythmia:
- Bradycardia: the heart rate is slow (<60 beats/min). Slower heart rates are more likely to cause symptomatic arrhythmias.
- Tachycardia: the heart rate is fast (>100 beats/min). Tachycardias are more likely to be symptomatic when the arrhythmia is fast and sustained.
- They are subdivided into supraventricular tachycardias (SVTs), which arise Cardiac arrhythmias from the atrium or the atrioventricular junction, and ventricular tachycardias, which arise from the ventricles.

Sinus rhythms

- Sinus rhythm: In lead 2 : **positive** P wave is **followed** by QRS complex.
- The normal cardiac pacemaker is the sinus node with the rate of sinus node discharge under control of the autonomic nervous system with parasympathetic predominating (resulting in slowing of the spontaneous discharge rate).
- Sinus arrhythmia is : Fluctuations of autonomic tone result in phasic changes in the sinus discharge rate. During inspiration, parasympathetic tone falls and the heart rate quickens, and on expiration the heart rate falls. This variation is normal, particularly in children and young adults, and typically results in predictable irregularities of the pulse.
- Sinus tachycardia is a physiological response during exercise and excitement. It also occurs with fever, pain, anaemia, heart failure, thyrotoxicosis, acute pulmonary embolism, hypovolaemia and drugs (e.g. catecholamines and atropine). Treatment is aimed at correction of the underlying cause. If necessary, β-blockers may be used to slow the sinus rate, e.g. in hyperthyroidism.

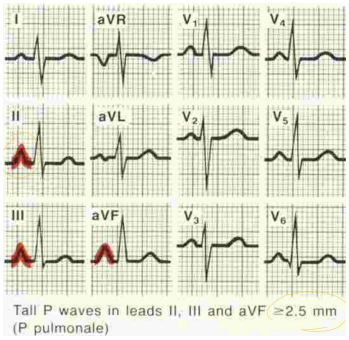
- Sinus bradycardia is normal during sleep and in well-trained athletes



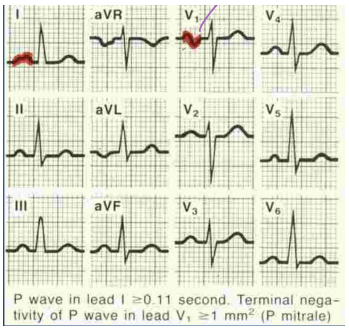
- Dr notes :

Right axis deviation causes : pulmonary HTN , Cor palmonale, PE , mitral stenosis .

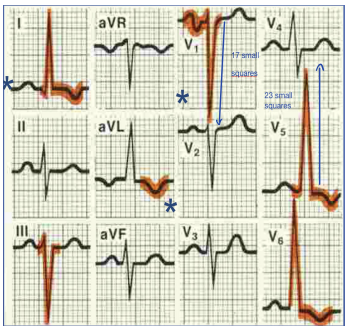
Left axis deviation causes : LVH , LBBB : QRS will be wide , Aortic stenosis.



Right atrial enlargement: Tall P wave > 2.5 mm



Left atrial enlargement: wide P wave > 2.5 mm

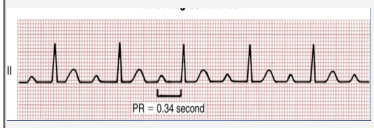
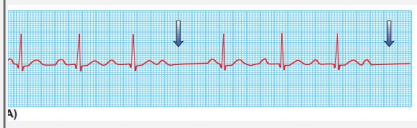
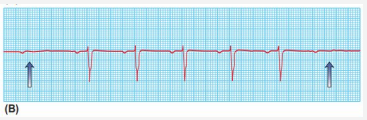
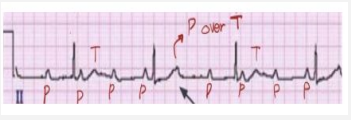


The summation of S wave in v1 (17) + R wave in V5 (23) = 40, if more than 35 (LVH)

To be able to approach patients with symptomatic bradycardia.

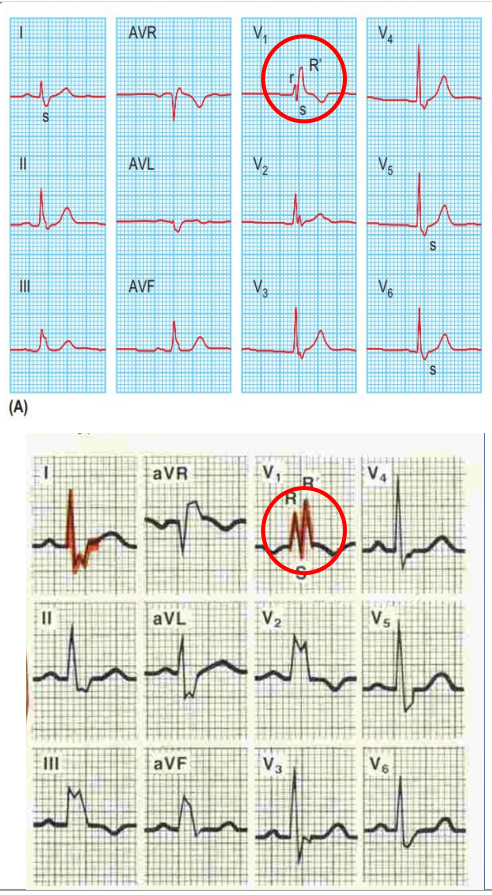
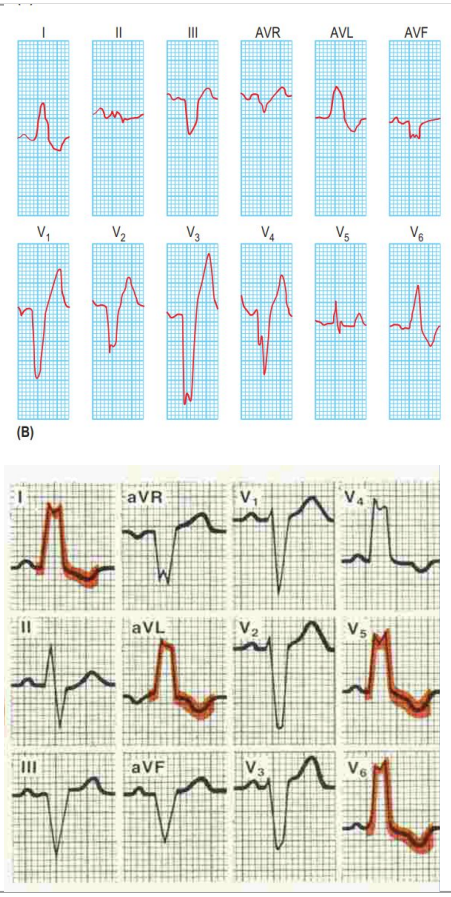
Heart block

Heart block		common causes of heart block	Management Of Brady arrhythmia
Atrioventricular block : Block in either the AV node **or** the His bundle results in AV block	**Bundle branch block :** block lower in the conduction system	CAD	**Stable patient:** Atropine
1st degree	LBBB	Cardiomyopathy	**Unstable:** Pace
2nd degree type 1 2nd degree type 2	RBBB	Fibrosis of the conducting tissue	
3ed degree			

1st degree	2nd degree type 1	2nd degree type 2	3rd degree
Delayed AV conduction and is reflected by a prolonged PR interval (>0.22 s) on the ECG	Progressive PR interval prolongation until a P wave fails to conduct, (drop QRS after progressive PR prolongation).	A block at an infra-nodal level so the QRS is widened and QRS complexes are dropped without PR prolongation	Complete dissociation between atrial and ventricular activity; P waves and QRS complexes occur independently of one another and ventricular contractions
			

To be able to approach patients with symptomatic bradycardia.

Bundle branch block know them by the pattern

RBBB rabbit ear pattern in V1	LBBB wide QRS
There is a sequential spread of an impulse (i.e. first the left ventricle and then the right) resulting in a secondary R wave (RSR0) in V1 and a slurred S wave in V5 and V6	Left bundle branch block (LBBB – the opposite occurs with an RSR0 pattern in the left ventricular leads (I, AVL, V4–V6) and deep slurred S waves in V1 and V2.
Occurs in normal healthy individuals, Pulmonary embolism, Right ventricular hypertrophy, Ischaemic heart disease and Congenital heart disease, e.g. atrial and ventricular septal defect and Fallot's tetralogy.	Indicates underlying cardiac pathology and occurs in aortic stenosis, hypertension, severe coronary artery disease and following cardiac surgery
	

To be able to approach patients with sinus tachycardia.

Sinus tachycardia

It's a physiological response during exercise and excitement. It also occurs with:

- Fever
- Pain
- Anaemia
- Heart failure
- Thyrotoxicosis
- Acute pulmonary embolism
- Hypovolaemia and drugs (e.g. catecholamines and atropine)
- Treatment is aimed at correction of the underlying cause. If necessary, β-blockers may be used to slow the sinus rate, e.g. in hyperthyroidism.

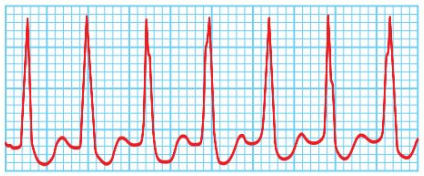
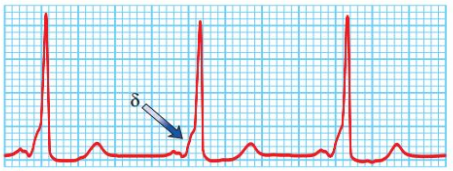
Box 30.14 Causes of supraventricular tachycardia (SVT)

Tachycardia	ECG features	Comment
Sinus tachycardia	P wave morphology similar to sinus rhythm; p waves always precede QRS	Need to determine underlying cause
Atrioventricular nodal re-entrant tachycardia (AVNRT)	No visible P wave, or inverted P wave immediately before or after QRS complex	Most common cause of palpitations in patients with normal hearts
Atrioventricular re-entrant tachycardia (AVRT) complexes	P wave visible between QRS and T wave	Due to an accessory pathway; if pathway conducts in both directions, ECG during sinus rhythm may be pre-excited
Atrial fibrillation	'Irregularly irregular' RR intervals and absence of organized atrial activity	Most common tachycardia in patients >65 years
Atrial flutter	Visible flutter waves at 300 b.p.m. (sawtooth appearance), usually with 2:1 AV conduction	Suspect in any patient with regular SVT at 150 b.p.m.
Atrial tachycardia	Organized atrial activity with P wave morphology different from sinus rhythm preceding QRS	Usually occurs in patients with structural heart disease or following extensive ablation within atria
Multifocal atrial tachycardia	Multiple P wave morphologies (≥3) and irregular RR intervals	Rare arrhythmia; most commonly associated with significant chronic lung disease
Accelerated junctional tachycardia	ECG similar to that in AVNRT	Rare in adults

To be able to approach patients with sinus tachycardia.

Supraventricular tachycardia

These are usually regular narrow-complex tachycardias and are characterised by a re-entry circuit or automatic focus involving the atria. Types of SVT :

Atrioventricular nodal re-entry tachycardia (AVNRT)	Atrioventricular re-entry tachycardia (AVRT)
AVNRT is the most common type of SVT and is twice as common in women as in men. It is due to the presence of a 'ring' of conducting pathway in the AV node	AVRT is due to the presence of an accessory pathway that connects the atria and ventricles and is capable of antegrade or retrograde conduction or both
The QRS complexes are narrow and the P waves cannot be seen	**Best example is WPW** : The early depolarization of part of the ventricle leads to a shortened PR interval and a slurred start to the QRS (delta wave). The QRS is narrow
 (A)	 (C)

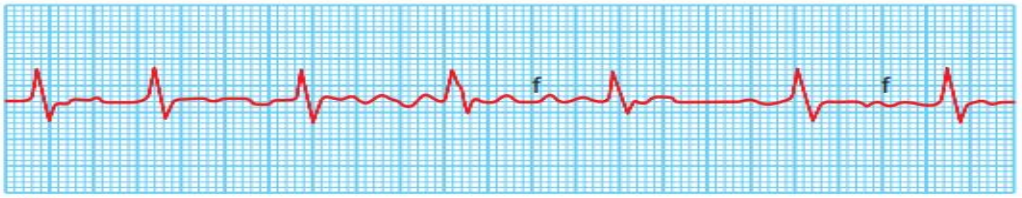
Signs and symptoms	The usual history is of rapid regular palpitations, usually with abrupt onset and sudden termination. Other symptoms are dizziness, dyspnoea, central chest pain and syncope. Exertion, coffee, tea or alcohol may aggravate the arrhythmia.

Management

Acute		Long term management
Stable	**Unstable**	Radiofrequency ablation of the accessory pathway via a cardiac catheter
1- Increase vagal stimulation of the sinus node by the Valsalva manoeuvre or right carotid sinus massage 2- Adenosine is a very short-acting AV nodal-blocking drug that will terminate most junctional tachycardias. Other treatments are intravenous verapamil or β-blockers, e.g. metoprolol. Verapamil is contraindicated with β-blockers,	Emergency cardioversion is required in patients whose arrhythmia is accompanied by adverse symptoms and signs	

Define the atrial fibrillation and its complications and be able to initiate therapy.

- AF is the most common arrhythmia and occurs in 15% of patients over 75 years of age. It also occurs, particularly in a paroxysmal form (stopping spontaneously within 7 days), in younger patients. Atrial activity is chaotic and mechanically ineffective. The AV node conducts a proportion of the atrial impulses to produce an irregular ventricular response – giving rise to an irregularly irregular pulse.
- ECG : There are no clear P waves + irregular fast rhythm, only a fine oscillation of the baseline (so-called fibrillation or f waves).



(B)

Complication	General Info
Death	AF Increases mortality 1.5 - 3.5 folds, due to sudden death, HF, comorbidities or stroke.
Stroke	20-30% of all ischemic strokes (preventable) and 10% of cryptogenic strokes are due to AF. Mainly due to cardioembolic or related to comorbid vascular atheroma.
Hospitalizations	10-40% of AF are hospitalized every year. For AF management, related to HF or MI or AF related symptoms also for treatment of associated complications.
Quality of life	More than 60% of patients. It's related to AF burden, comorbidities, psychological functioning and medication, distressed personality type.
Left ventricular dysfunction (LVD) & HF	LVD is found in 20-30% of all AF patients. Due to excessive ventricular rate, irregular ventricular contractions, a primary underlying cause of AF.
Cognitive decline and vascular dementia	HR 1.4/1.6 (irrespective of stroke history). Due to Brain matter lesions, inflammation or hypoperfusion and micro-embolism.
Depression	In 16-20% of patients (even suicidal ideation). Due to severe symptoms, decreased QoL and drug side effects.

Define the atrial fibrillation and its complications and be able to initiate therapy.

Management

First treat the underlying cause then to initiate treatment for the arrhythmia you have to determine if the patient is hemodynamically stable or not.How? If there's chest pain, shortness of breath, altered mental status (confusion), or a systolic BP < 90, then the patient is considered unstable. If they're unstable use electricity. If instead the patient has symptoms, but not any one of those listed above, the patient is stable. A patient who is stable has time to fix the rhythm. They're not going to die at this moment; pharmacotherapy can be used.

3rd year medicine team

Hemodynamic stability — ABCD

Stable patients

1) Rate control (BB or Ca++ blockers or Digoxin)
2) Rhythm control (Cardioversion)
3) Anticoagulation

Unstable patients

1) Do synchronized **electrical Cardioversion (Shock)** immediately.

1) Rate control

- Target heart rate for AF patients is < 110 bpm during activity and 60-80 bpm during rest.
- **Drugs:** (BB and CCB have equal efficacy, choice depends on the pt situation, digoxin has lowest efficacy)
  1. **Beta-blockers:** Atenolol, Metoprolol (Used in cases of Graves disease, CHF, IHD, HTN)
  2. **Ca2+ - channel blockers:** Verapamil , diltiazem (Used if patient is Asthmatic)
  3. **Digoxin**: (Used if patient is hypotensive or suffering from LV systolic dysfunction)
  4. **Pace and ablate strategy**[1]

2) Rhythm control

Click here for a summary of all antiarrhythmics (EXTRA!!)

- Electrical Direct current (DC) cardioversion (More effective) or pharmacological cardioversion may be used.
- Likelihood of successful cardioversion decreases with the duration of AF:
  - Pharmacological cardioversion is most effective when initiated within 7 days of AF onset.
- It's advocated for younger, symptomatic and physically active patients.
- **Pharmacologic cardioversion use depends on the presence or absence of of underlying heart disease:** (It's mainly used if electrical cardioversion fails or not feasible)
  - **A. Absent:** Class IC antiarrhythmics e.g. **IV Flecainide,** Propafenone Or Class IA e.g. Procainamide
  - **B. Present:** Class III antiarrhythmics e.g. **IV Amiodarone**[2] Or Dronedarone (alternative)
- Others: Ablation[3], Surgery (MAZE)
- **AF for <48hr → Cardiovert immediately** either pharmacologically or **electrically** (or both) then anticoagulate for 4 wks
- ★ **AF for ≥48hr (or unknown)** → Either do **TEE** to check atrial thrombus then cardiovert (If no thrombus) **Or Give Anticoagulants for ≥3wks → Cardiovert → Give Anticoagulants again for at least 4wks.**

Why do we give anticoagulants before cardioversion? **Because LAA thrombus can dislodge and cause stroke**
Why do we give anticoagulants after cardioversion? **Because cardioversion may lead to atrial stunning → Blood stasis → Thrombus formation.**

3) Stroke prevention

- ★ Patient with **AF related Moderate or severe mitral stenosis or the presence of a mechanical heart valve should always be anticoagulated (With Warfarin)** because the risk is so high. In other patients, **CHA_2DS_2-VASc score** is used to assess the risk of stroke.
- **New Oral AntiCoagulants (NOAC) "First line treatment":**[4]
  - Dabigatran, Apixaban, Rivaroxaban, Edoxaban
- **Warfarin**[5] **(Coumadin®) "used in cases of Mitral stenosis or if pt has metal valve"**
- WATCHMAN® device or surgery: For removal/isolation of left atrial appendage.

Define the atrial fibrillation and its complications and be able to initiate therapy.

Management - cont

CHA_2DS_2-VASc scoring system

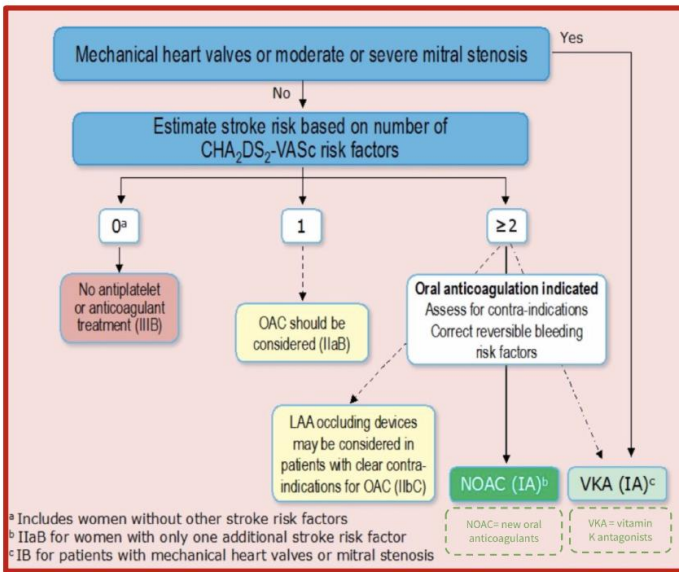
- A scoring system used to identify which patient is at high risk of thromboembolic complications and will benefit from anticoagulation therapy.

16.23 CHA_2DS_2-VASc stroke risk scoring system for non-valvular atrial fibrillation

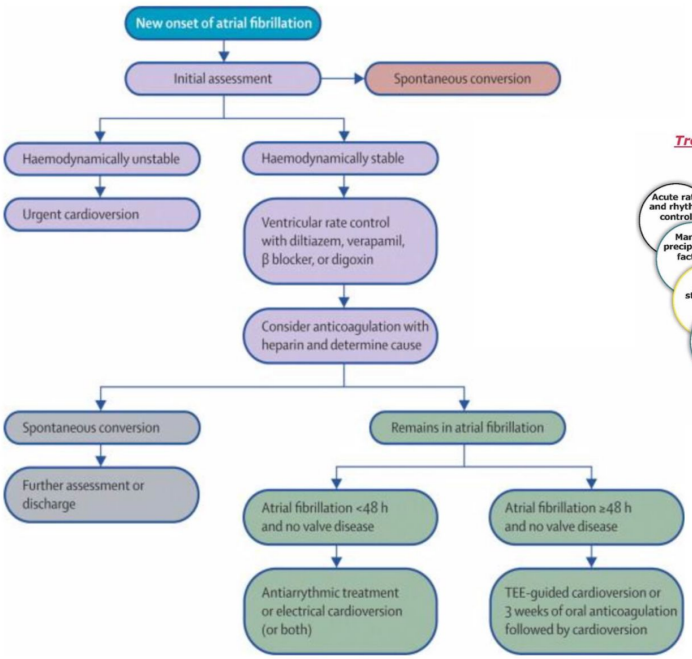
	Parameter	Score
C	Congestive heart failure	1 point
H	Hypertension history	1 point
A_2	Age ≥75 years	2 points
D	Diabetes mellitus	1 point
S_2	Previous stroke or transient ischemic attack (TIA)	2 points
V	Vascular disease	1 point
A	Age 65–74 years	1 point
Sc	Sex category female	1 point
	Maximum total score	9 points

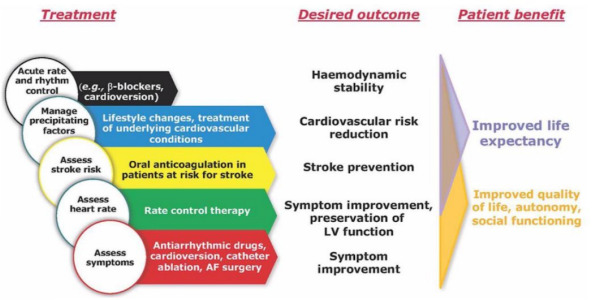
Annual stroke risk
0 points = 0% (no prophylaxis required)
1 point = 1.3% (oral anticoagulant recommended in males only)
2+ points = > 2.2% (oral anticoagulant recommended)

0 → Low risk (No need for anticoagulants)
1→ Moderate risk (the need of anticoagulants is controversial)
2 or more → High risk (Requires anticoagulation)



AF Management Summary

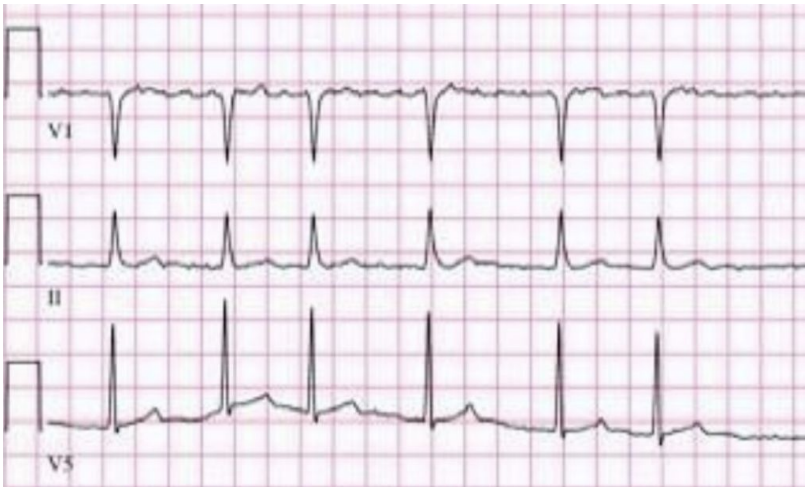


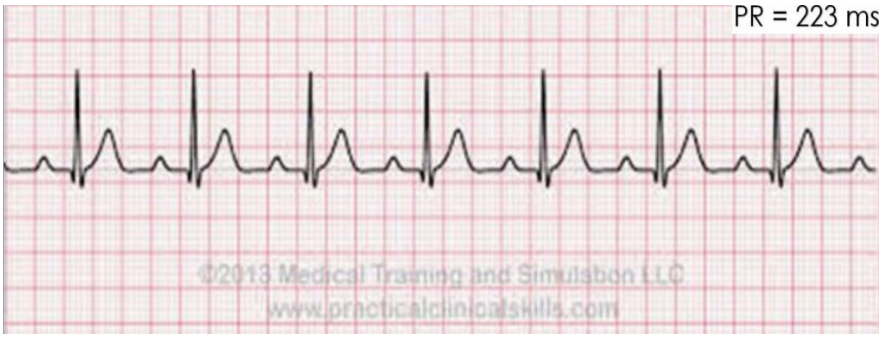


Identify atrial fibrillation and heart blocks on ECG.

Character on ECG:
- Irregular rhythm
- Absent P wave

What's the diagnosis:
most likely **Atrial fibrillation**



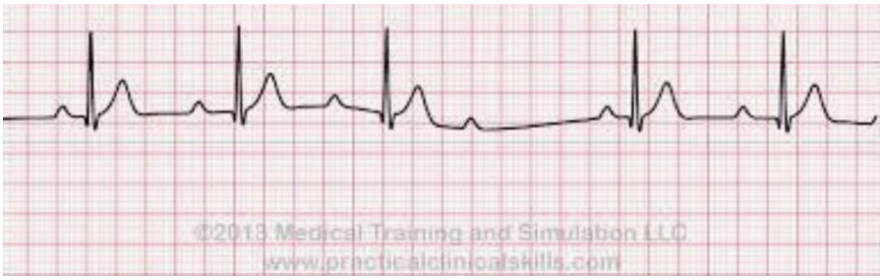


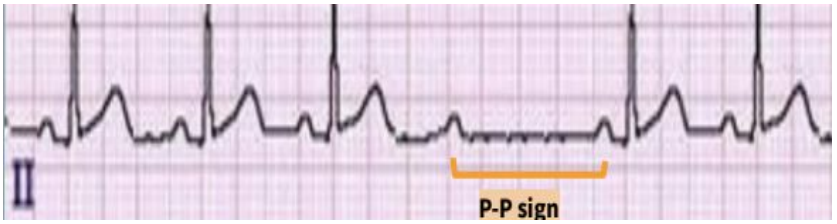
Character on ECG:
- Fixed Prolonged PR Interval (normal: 120ms)

What's the diagnosis:
1st degree heart block

Character on ECG:
- Irregular rhythm
- Progressive prolongation of PR interval
- QRS complex drop

What's the diagnosis:
2nd degree heart block type 1 (Mobitz I)



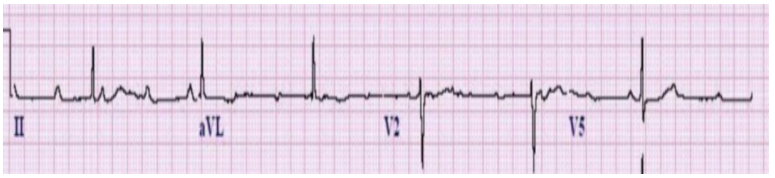


Character on ECG:
- Irregular rhythm
- Fixed PR interval followed by QRS complex drop

What's the diagnosis:
Second degree heart block Type 2 (mobitz ll)

Character on ECG:
- Bradycardia
- Variable PR Interval

What's the diagnosis:
3rd degree heart block.



Identify ventricular arrhythmias based on ECG and initiate a management plan.

Ventricular tachyarrhythmias

1) Ventricular ectopic premature beats (extrasystoles)

These are asymptomatic or patients complain of extra beats, missed beats or heavy beats.
The ectopic electrical activity is not conducted to the ventricles through the normal conducting tissue and thus the QRS complex on the ECG is widened, with a bizarre configuration
Happens due to hypoxia, hyperthyroidism, electrolyte abnormalities

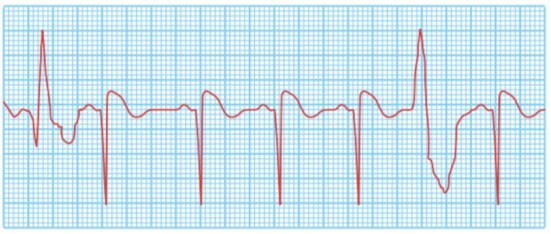


Fig. 10.12 A rhythm strip demonstrating two ventricular ectopic beats of different morphology (multimorphological).

2) Sustained ventricular tachycardia

The ECG in sustained VT (>30 s) shows a regular rapid ventricular rhythm with broad abnormal (wide)QRS complexes.(>=3 consecutive premature ventricular beats) all wide QRS is VT until proven otherwise and if the patient is unstable shock him immediately

- ➢ Monomorphic VT: (the most common); single QRS morphology
- ➢ Polymorphics VT: multiple QRS morphologies

AV dissociation (P waves maybe not discernible)
Patient with structural heart disease or prior MI and show with wide QRS consider mostly VT

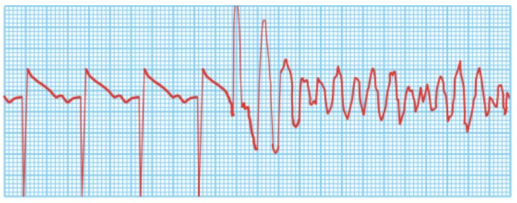


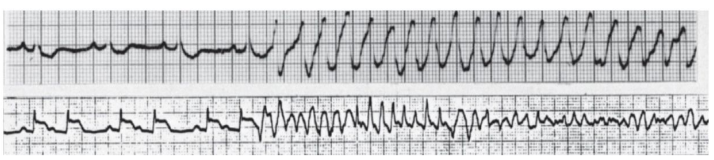
Fig. 10.13 A rhythm strip demonstrating four beats of sinus rhythm followed by a ventricular ectopic beat that initiates ventricular fibrillation. The ST segment is elevated owing to acute myocardial infarction.

3) Ventricular fibrillation

VF is a very rapid and irregular ventricular activation with no mechanical effect and hence no cardiac output. The patient is pulseless and becomes rapidly unconscious, and respiration ceases
Characters on ECG: No P waves, No QRS waves with irregular rhythm with >300 bpm
Causes

1) Ischemic Heart disease (the most common)
2) Antiarrhythmic drugs
3) MI
4) Structural heart disease

Treatment is immediate defibrillation and CRP



4) Long QT syndrome

Ventricular repolarization (QT interval) is greatly prolonged Symptoms are palpitations and syncope, as a result of a polymorphic VT (torsade de pointes, rapid irregular sharp QRS complexes that continuously change from an upright to an inverted position on the ECG)
causes:

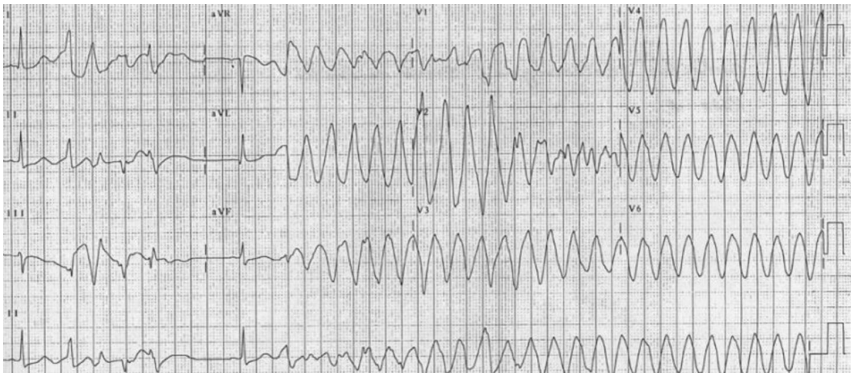
- ➢ **Congenital** (mutations in sodium and potassium-channel genes)
- ➢ **Electrolyte disturbances** (hypokalaemia, hypocalcaemia, hypomagnesaemia)
- ➢ **Drugs** (e.g. tricyclic antidepressants, phenothiazines and macrolide antibiotics).

Identify ventricular arrhythmias based on ECG and initiate a management plan.

Ventricular tachyarrhythmias

5) Torsade de pointes tachycardia

Polymorphic Ventricular tachycardia with QRS complex that appear to twist around the isoelectric line
Associated with Long QT syndrome, proarrhythmic drugs and electrolyte abnormalities (hypokalemia)



Identify ventricular arrhythmias based on ECG and initiate a management plan.

Management:

Ventricular ectopic premature beats (extrasystoles)

- Most patients don't require any treatment
- Treat underlying disease (CAD, myocarditis)
- Only treat frequent and significantly symptomatics
- Antiarrhythmic therapy
- Catheter ablation if antiarrhythmic therapy fails

Ventricular tachycardia

- **Treat underlying cause**
- **Ongoing or sustained VT**
  - Hemodynamically stable patients with mild symptoms and systolic BP.90 => IV amiodarone
  - Hemodynamically unstable patients or patients with severe symptoms => Immediate synchronous DC cardioversion, follow with IV amiodarone to maintain sinus rhythm
  - ICD placement
- **Non sustained VT or resolved Sustained VT:**
  - No underlying Heart disease and asymptomatic => Don't treat
  - Have underlying heart disease (recent MI, evidence of Left ventricular dysfunction) or symptomatic => order and electrophysiology study > if it show inducible or sustained VT => ICD placement
  - Pharmacologic therapy is Second line treatment
- **Long term management of VT patients**
  - Antiarrhythmics with device therapy (ICD); to minimize the symptoms, risk of recurrence and risk of sudden cardiac death, also Ablation of arrhythmogenic foci is potentially curative
  - For Antiarrhythmic consider B-blocker as 1st line therapy (reduce the risk of sudden cardiac death)
  - Indication of ICD: Expected survival >1y, Recurrent VT despite treatment of reversible causes
  - Indication of ablation is recurrent VT despote optimal therapy, ANtiarrhythmics are not tolerated, patient preference

Torsades de pointes

- Administer IV magnesium
- Avoid amiodarone , procainamide and sotalol
- Identify and treat the underlying cause

Ventricular Fibrillation

- **Resuscitation for V-fib**: ACLS, refractory V-fib consider administration of Lidocaine, procainamide or magnesium
- **Post -resuscitation care:**
  - Intensive care monitoring
  - Maintain application of antiarrhythmics (IV amiodarone or IV lidocaine)
  - Admission of B-blocker
  - Treat underlying causes
  - ICD in patient without readily reversible or treatable cause, Or high risk of recurrence

Wide complex tachyarrhythmias (WCT) doctor slides

It has the QRS >0.12s and HR>100

Causes:

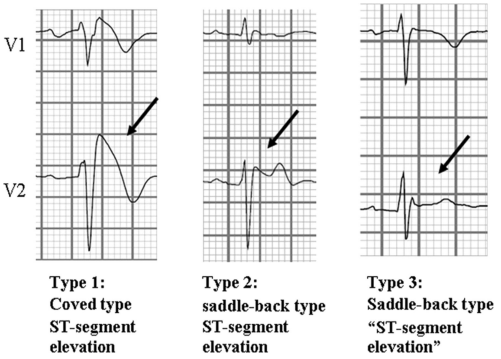
Cardiomyopathy (IHD,DCM)

Idiopathic VT

Inherited Arrhythmias

Malfunction of heart channels leading to dysregulation of (Ca+, K+, Na+) show on ECG as:

- Long QT (Downregulation of K+)
- Short QT (Upregulation of Ca+)
- Brugada syndrome (Na+ channel block)
  - Type 1 (diagnostic): Coved ST elevation >= 2 mm with -ne T wave, increase sensitivity by moving V2/V3 from 4th to 2nd/ 3rd intercostal space
  - Type 2: Saddleback ST elevation >= 2mm w/ST trough >= 1mm, +ve biphasic T wave
  - Type 3: Coved/ saddle ST elevation >= 2mm without ST trough < 1 mm
- Catecholamine polymorphic VT
- Arrhythmogenic Right Ventricular Dysplasia (ARVD)
- Hypertrophic cardiomyopathy



Approach for WCT patients

1 Hemodynamically status
- Unstable > ACLS
- Stable > Go to step 2

2 Find the underlying cause
- VT (mostly)
- SVT
- AF with BBB or with WPW
- Paced rhythm
- Sinus tachycardia with BBB

3 EKG criteria
- QRS; duration, axis and concordance
- AV dissociation
- Fusion and capture beat
- Specific pattern in V1
- Absent RS in precordial leads

Click here for more info

Step 1: General Principles

The Step 2 exam will ask to either **identify the rhythm** or **choose an intervention**. In order to identify the rhythm, follow these simple principles. 1) Determine the **rate**: **tachycardia** is **> 100**, **bradycardia < 60**. 2) Determine the **QRS complex**: **wide** is > **.12msec** and means it's a **ventricular** rhythm while **narrow** is < **.12msec** and means it's an **atrial** rhythm. These two things will give you 80% of the answers on the test. The third and final decision is if the rhythm is **regular** or **irregular**. Of course, to determine any of this an **ECG**, preferably a **12-lead**, is needed.

With the ECG ask if there's an arrhythmia or not. Note that there are two, maybe three, rhythms that aren't arrhythmias. **Normal Sinus Rhythm** is what everyone should be in. **Sinus tachycardia** is typically a normal, physiologic response to an underlying stressor. **Sinus bradycardia** may be a normal rhythm in a competitive athlete, though they usually do not appear in a vignette or in the hospital as an "arrhythmia."

Step 2: Symptoms or No Symptoms

Ask, "are there symptoms?" An arrhythmia without any symptoms does not warrant attention. Simply: if there are **no symptoms** then **do nothing**. "Nothing" means routine care: IV, O_2, and Monitor. Likely, this will be a question about rhythm identification.

Step 3: Stable vs Unstable

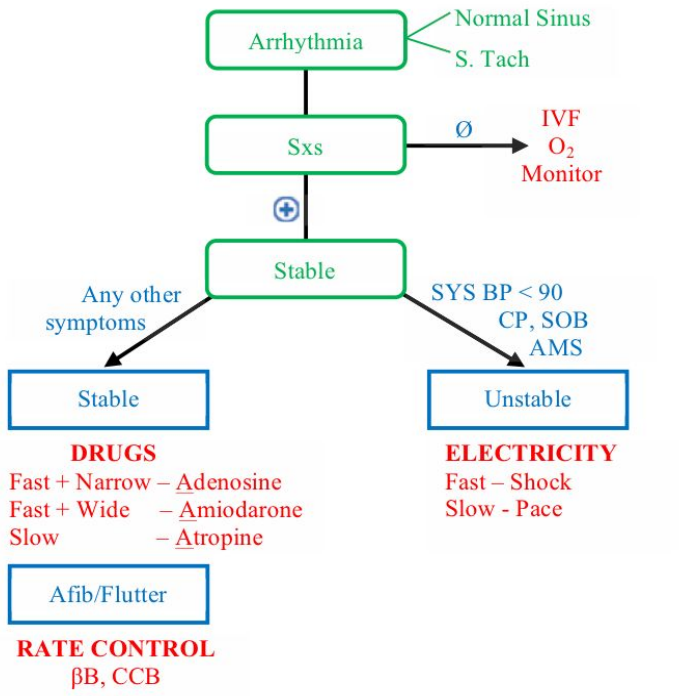
If the patient has symptoms decide whether there's time to stay and play or if definitive therapy is needed right now. Stability is a product of your own comfort. But for a test, if there's **chest pain**, **shortness of breath**, **altered mental status**, or a **systolic BP < 90**, then the patient is considered **unstable**. If they're unstable use **electricity**.

If instead the patient has symptoms, but not any one of those listed above, the patient is **stable**. A patient who is stable has time to fix the rhythm. They're not going to die at this moment; **pharmacotherapy** can be used.

Step 4: Choose an intervention

If you've chosen **unstable/electricity** only one question needs to be asked - fast of slow. If the rhythm is **fast + unstable** then **shock**. If the rhythm is **slow + unstable** then **pace**.

If you've chosen **stable/electricity** it's a slightly more difficult task. For stable rhythms, there are three, maybe four, options. 1 - If the rhythm is **fast + narrow + stable** use **adenosine**. 2 - If the rhythm's **fast + wide + stable** use **amiodarone**. 3 - If the rhythm's **slow + stable** use **atropine** (epi drips can also be used in the new ACLS roll out). 4 - If the rhythm's **Afib/Aflutter** (note this is the only rhythm that actually had to be identified to do the right intervention), **rate control** is preferred. If they were unstable shock them since afib usually presents as a tachycardia. By "rate control" we mean **Beta Blockers** or **Calcium Channel Blockers**.



Tachy Rhythms
- Sinus Tachycardia
- Supraventricular Tachycardia
- Multifocal Atrial Tachycardia
- Afib
- Aflutter
- Vtach
- Vfib
- Torsades

Atrial Narrow

Ventricular Wide

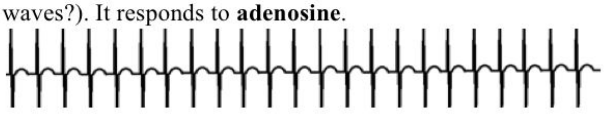
Brady Rhythms
- Sinus Bradycardia
- 1^o Block
- 2^o Block
- 3^o Block
- Junctional
- Idioventricular

Varying degree of PR intervals

Intervention	*Heart Rate*	*QRS Complex*	*Stability*
Pacer	Brady	Any	Unstable
Cardioversion	Tachy	Any	Unstable
Atropine	Brady	Any	Stable
Adenosine	Tachy	Narrow	Stable
Amiodarone	Tachy	Wide	Stable
Rate Control	Tachy	Afib/Flutter	Stable

"Rate Control" = Verapamil / Diltiazem, Metoprolol

Supraventricular tachycardia is an **aberrant reentry** that bypasses the SA node. It's **narrow** (atrial), **fast** (tachycardia), and will be distinguished from a sinus tachycardia by a **resting heart rate > 150** + the loss of **p-waves** (can you tell p-waves from t-waves?). It responds to **adenosine**.

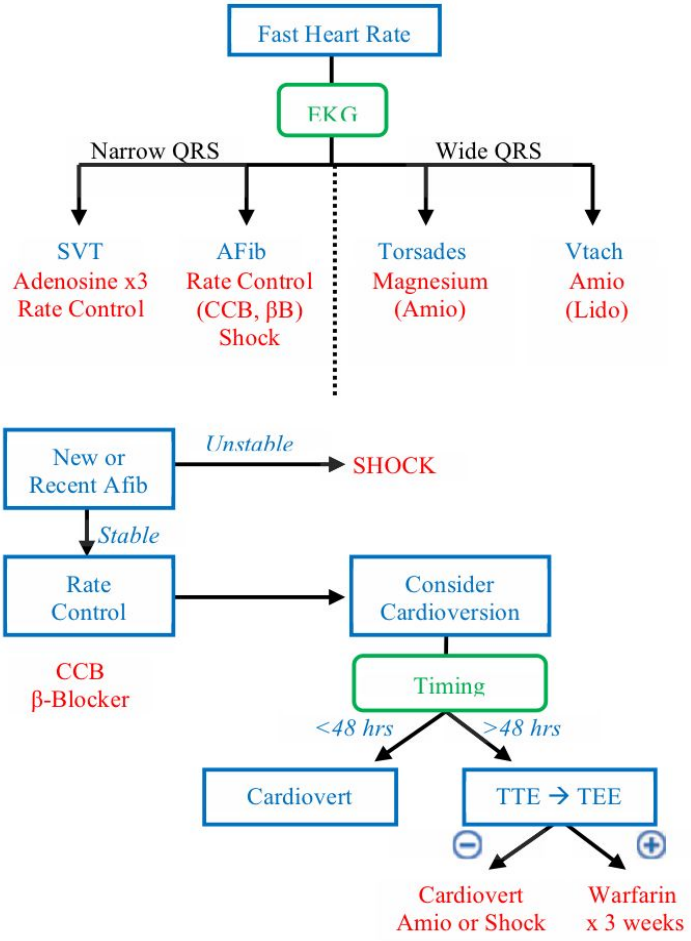


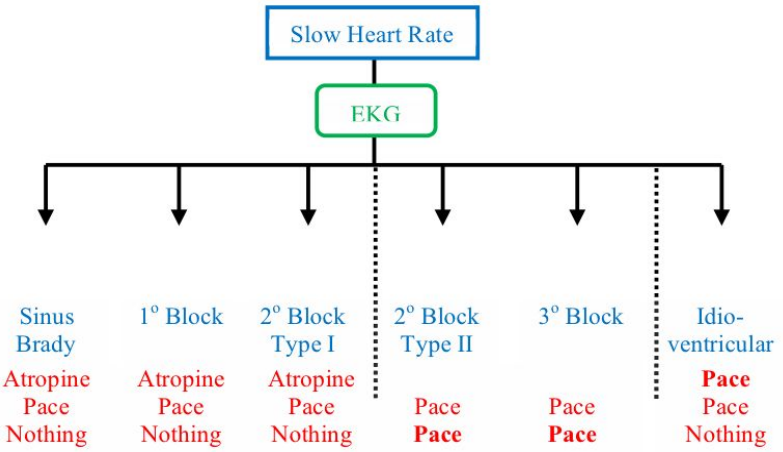
Ventricular Tachycardia is a **wide complex** and **regular** tachycardia. Look for the "tombstones." Since it's ventricular there are **no paves** at all - just the **QRS complexes.** It responds to **amiodarone** (newer/better) or **lidocaine** (older/cheaper)

Atrial Fibrillation can be identified by a **narrow complex** tachycardia with a **chaotic background, absent p-waves,** and an **irregularly irregular** R-R interval. It has a special treatment algorithm. In the acute setting (ACLS in a nutshell) simply decide between shock and rate control. **Rate control** is just as good as **rhythm control** (cardioversion). But, you have to weigh risks and benefits in each patient. If the goal is **rhythm control** (cardioversion) it's necessary to determine **how long** the Afib's been present. Simply cardioverting an Afib that's lasted > 48 hrs runs the risk of throwing an **embolism** (and a stroke). If < 48hours cardioversion is ok. But if it's been present **> 48 hours** the patient needs to go on **warfarin** for four weeks. At the end of four weeks, the **TEE** is done. If no clot is found, cardioversion is done and the patient remains on warfarin for another 4 weeks. If you decide to do **rate control** (beta blockers and calcium channel blockers) **anticoagulation** may still be needed. Decide this using the **CHADS2 score**. The higher the score the higher the risk of embolism and the more likely the patient is to benefit from **warfarin** (2+ CHADS2). Now, the Xa- or Thrombin-inhibitors can be used instead (1+ CHADS2). Examples include **apixiban** or **dabigatran**.

Sinus bradycardia is simply a slow normal sinus rhythm. The blocks are a worsening of that normal bradycardia. Almost everything responds to **Atropine** until it gets really bad - then **only pacing will do**.

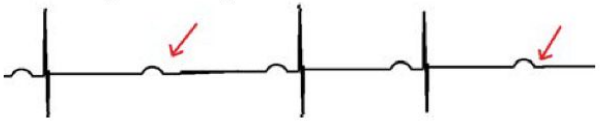
1° AV Block is characterized by a **regularly prolonged PR interval**. There's no change in the interval between beats, but each is prolonged. There are no dropped beats.



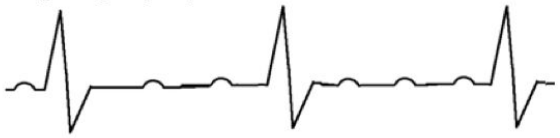


2° AV Block Type I is a normal rhythm with a constantly **prolonging PR interval** with each beat, until a QRS complex is **finally dropped**. The signal comes from the atria so there is a narrow QRS complex.

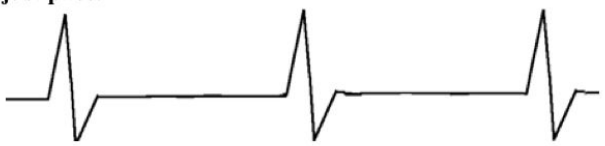
2° AV Block Type II has a **normal PR interval** but simply **drops QRSs** randomly. The signal comes from the atria so the QRS complexes are narrow. This is the most severe a rhythm can be before atropine no longer works.



3° AV Block. There's total **AV node dissociation.** The **Ps march out** (regular interval between P waves) and the **QRSs march out** (regular interval between QRS complexes). At times, the P waves may seem lost or dropped; the QRS complex occurs at the same time and obscures the p wave. Because the impulse comes from the ventricles it's a wide QRS complex. In general, **avoid atropine** (just pace). This is controversial.



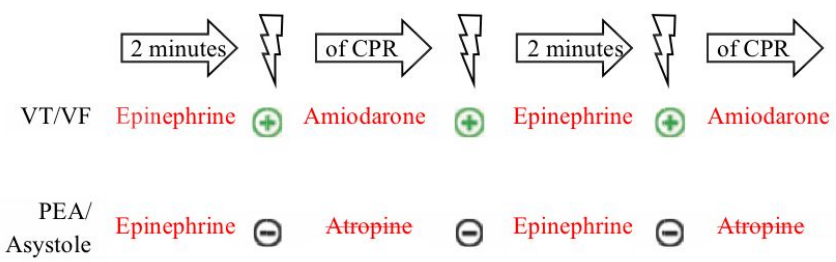
Idioventricular Rhythm is a rhythm without atrial activity. Only the ventricles are contracting, only the ventricles have electrical activity. It looks like a 3° block, but without p waves. **Avoid atropine** (it won't work), as there is no atrial conduction at all, so **just pace**.



This is not every rhythm you could see, but it's way more than you need to be prepared for the USMLE. You'll see a rhythm, MAYBE two on the test. MAYBE.

CARDIAC ARREST

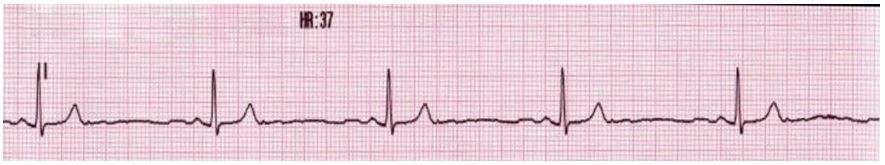
When dead, remember 1 thing: compressions. Everything is based around 2 minutes of CPR. 2 minutes of CPR, check a pulse, check a rhythm, shock if indicated. Shock is indicated only in Vtach/Vifb arrest. Always start with Epi. Only in VT/VF can you shock, and so too only in VT/VF can antiarrhythmics be used. That's it. This is almost never tested on Step 2 but is here for completeness.



Lecture Quiz

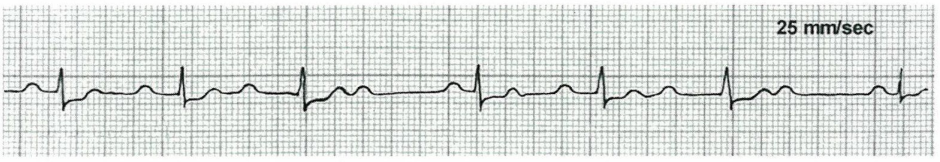
1:C / 2: C/

Q1: A 59-year-old woman is brought to the emergency room after collapsing at home. She had been sitting on her couch reading, when she started feeling lightheaded and lost consciousness. According to her husband, she was unconscious for approximately 30 seconds. Since regaining consciousness, she has continued to be lightheaded and dizzy. She has not had palpitations. Her only medication is simvastatin for hyperlipidemia. Her pulse is 37/min, respirations are 18/min, and blood pressure is 92/55 mm Hg. An ECG is shown. Which of the following is the most appropriate next step in management?



A. Administration of dopamine
B. Fluid repletion with crystalloids
C. Administration of atropine
D. Administration of epinephrine

Q2: A 45-year-old man comes to the physician for a routine health maintenance examination. He feels well. He underwent appendectomy at the age of 25 years. He has a history of hypercholesterolemia that is well controlled with atorvastatin. He is an avid marathon runner and runs 8 miles per day four times a week. His father died of myocardial infarction at the age of 42 years. The patient does not smoke or drink alcohol. His vital signs are within normal limits. Cardiopulmonary examination shows no abnormalities. His abdomen is soft and nontender with a surgical scar in the right lower quadrant. Laboratory studies are within normal limits. An ECG is shown. Which of the following is the most likely diagnosis?



A. Myocardial infarction
B. Mobitz type II AV block
C. Mobitz type I AV block
D. First-degree AV block