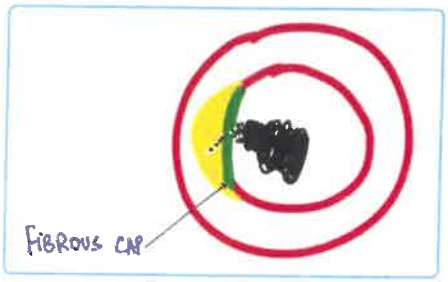
MEDICINE

1 CARDIOLOGY PART - 1

Myocardial Infarction

00:00:26

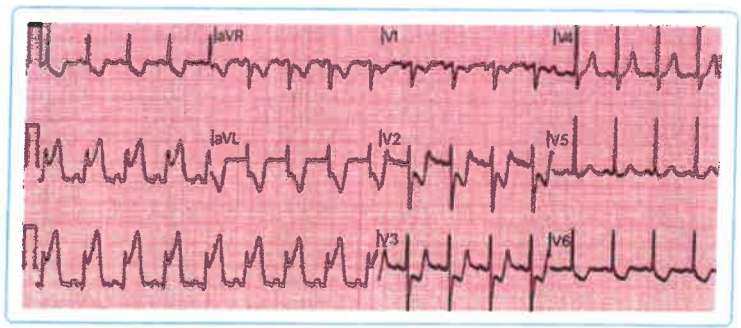


- Coronary artery disease contributes to the maximum amount of mortality in India (the diabetic capital of the world).
- Cut section of coronary artery showing atherosclerosis:
  - Yellow: Fat deposition i.e., lipid core in the tunica intima.
  - Green: The fibrous cap

Atherosclerosis
↓
Blood flowing at a fixed pressure through the coronary artery
↓
Plaque fissure
↓
Exposure of collagen
↓
Platelet plug formation
↓
Clot/Thrombus formation
↓
Occlude the entire lumen
↓
Muscle death/Myocardial necrosis

ECG Interpretation

00:04:30

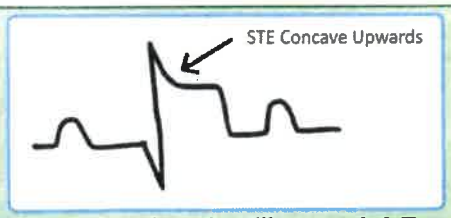


- ECG image with ST elevation in LEAD II, III, and aVF and reciprocal changes of ST depression in V1,2,3,4 indicating Inferior wall MI.
- In lead II:
  - P wave present, no Q wave
  - Characteristic ST elevation present - Tomb Stone Pattern (ST elevation is convex in an upward direction)
- In Inferior wall MI, the thrombus mainly presents in RCA

PYQ: FMGE 2019

Important Information

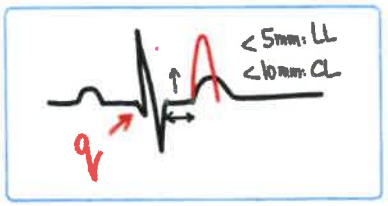
- If ST elevation is concave in an upward direction: Inflammation of the outermost layer of the heart - Acute Pericarditis.



- To differentiate MI from Acute pericarditis. Cardiac biomarkers like troponin I, Troponin T, and CPKMB are grossly elevated in MI and usually normal in Acute Pericarditis.

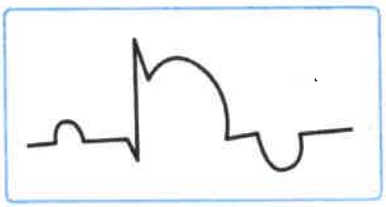
ECG Findings in MI 00:09:10

1. **Hyperacute T wave (Earliest ECG finding)**



- Normal size criteria of T wave:
  - Vertical height is <5mm in limb leads & <10 mm in chest leads
- More than normal size of T wave - Hyperacute T wave
- Indicates Myocardial Ischemia.
- Develops within seconds of infarction

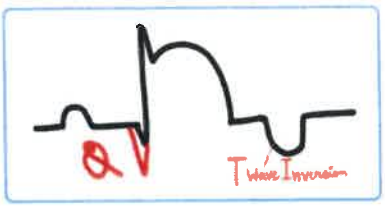
2. **ST elevation**



- Significant ST elevation:
  - For Male patients: The rise is ≥ 2mm
  - For female patients: The rise is ≥1.5mm
- Also called current of injury.
- Indicates Myocardial injury.
- Myocardial injury is best identified by cardiac biomarkers
- Develops within minutes of infarction

3. **T wave Inversion**
   - ST elevation is associated with a T wave inversion.

4. **Q waves/ Pathological Q waves**



- In a normal individual, the Q wave is within one small square
- Indicates Cell Death or Previous MI
- A deep Q wave persists for the whole life.
- Usually develops after >1 hr of infarction

Definition of Myocardial Injury and Infarction 00:18:20

Criteria for myocardial injury

- The term myocardial injury should be used when there is evidence of elevated cardiac troponin (cTn) levels with at least one value above the 99th percentile upper reference limit (URL). The myocardial injury is considered acute if there is a rise and/or fall of cTn values. [Harrison textbook]
- The normal value of Troponin I is 0.04 ng/dl
- MI is confirmed if there is at least a doubling of the Trop I value.
- It indicates that myocardial necrosis has already occurred.
- Troponin I value rise occurs in 3 to 4 hrs in contrast with CPKMB which will take about 4 to 6 hrs to rise
- ECG is done first but ECG alone should not be used as a diagnosis or conformation, cardiac biomarkers should also be checked for diagnosis of MI.
- Troponin I is more specific in diagnosis.
- Serial Trop I: If a patient presents early with definitive necrosis in the heart the values are repeated but the treatment is still initiated.

Criteria for Acute Myocardial Infarction (types 1, 2 and 3 MI)

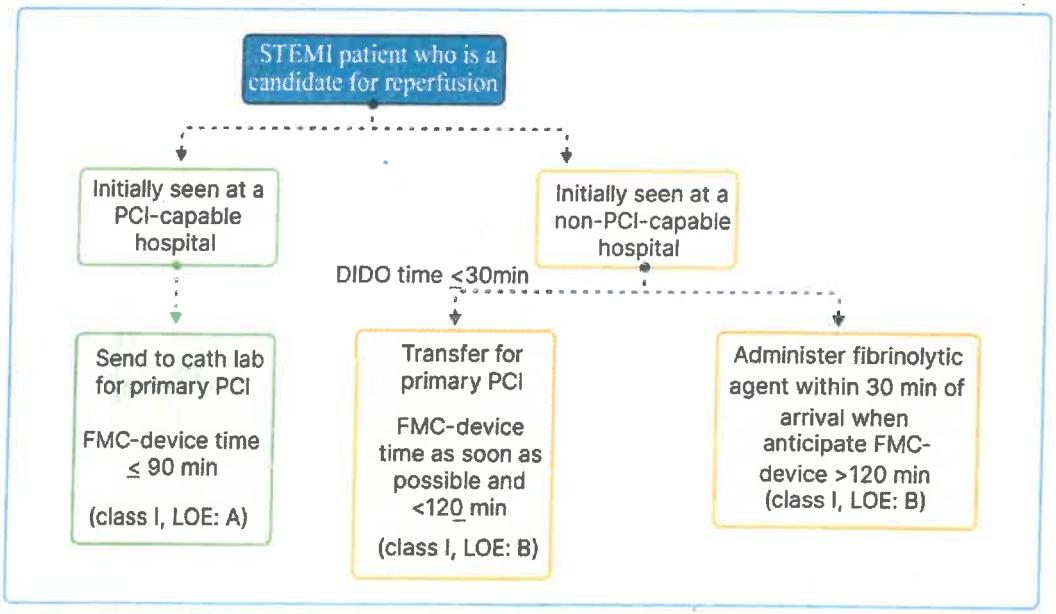
- The term myocardial infarction (MI) should be used when there is acute myocardial injury with clinical evidence of acute myocardial ischemia and with detection of a rise and/or fall of cTn values with at least one value above 99th percentile URL and at least one of the following:
  - Symptoms of myocardial ischemia
  - New ischemia electrocardiographic (ECG) changes
  - Development of pathologic Q waves
  - Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischemic etiology
  - Identification of a coronary thrombus by angiography or autopsy (not for types 2 or 3 MIs) [Harrison textbook]
- **Levine sign:** Diffuse chest pain > 20 minutes presenting with a clenched fist over the chest.
- **ECHO**: Reveals the presence of regional hypokinesia/stunned myocardium (sudden decrease in the contractility of the heart).
- Best test/ IOC/ gold standard diagnosis for MI: Elevated cardiac biomarkers with any one of the above 5 points

Important Information

- **Leading cause of death in DM - Increased incidence of cardiovascular mortality**
- **In DM due to neuropathy, there is Silent MI.**

STEMI- Management

00:27:30



PYQ: FMGE 2018

- **PCI-capable hospital with CATH lab facility:**
  - Coronary angiography is done by using femoral or radial artery access (preferred)
  - Guidewire is sent retrogradely towards the heart.
  - However, the contrast is unable to flow forward because of the coronary artery occlusion.
  - In such cases, a balloon angioplasty/ primary percutaneous intervention (PCI) is done.
  - Done <90 mins of a patient coming in contact with medical services - Door to balloon time/FMC (First medical contact) device time.
- **Non-PCI hospital without CATH lab facility:**
  - Thrombolysis is initiated in the shortest possible time.
  - Door-to-needle time: <30 mins

- **Mnemonic**: MOAN
  - Initially, the patient is given Dual Antiplatelet Therapy (DAPT): Aspirin + Ticagrelor
  - Anti-ischemia measure: Sublingual Nitro-glycerine (SLNTG)
    → Avoid if SBP is <90 mmHg - because nitrates are vasodilators and a further decrease in BP will compromise the coronary blood flow and renal blood flow
  - Morphine: Used when nitrates are given 3 times and there is no improvement.
  - O_2: If $SPO_2 < 93\%$
    → Example: Pulmonary edema seen in LV involvement
  - Metoprolol (β-blocker): Reduces O_2 consumption by decreasing heart rate and thus reduces the size of the infarct.
    → Avoided in bradycardia as it leads to hypotension.

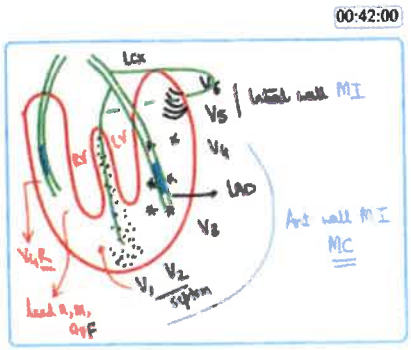
Important Information

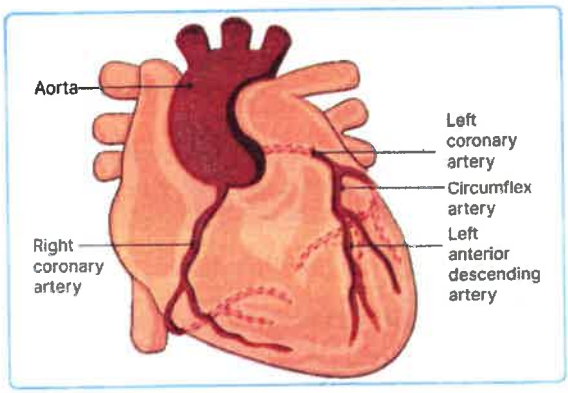
- The Upper limit of thrombolysis: <12 hrs
  - > 12 hrs the side effects are more than the benefit.
  - S/E: Bleeding
- The window period of acute ischemic stroke: 4.5 hrs
  - Thrombolysis is contraindicated in hemorrhagic stroke.
- Antidote of streptokinase toxicity: Epsilon Amino Caproic Acid (E.A.C.A)

- **STEMI:**
  - TOC: Primary PCI.
  - The clot is rich in Fibrin and RBCs.
  - Fibrinolytic drugs (Streptokinase, Alteplase, Reteplase, and Tenecteplase) will work.
- **NSTEMI:**
  - The clot is rich in platelets.
  - Antiplatelet/ antithrombotic drugs are used.
  - Thrombolysis is contraindicated in NSTEMI.

Infarct Localization

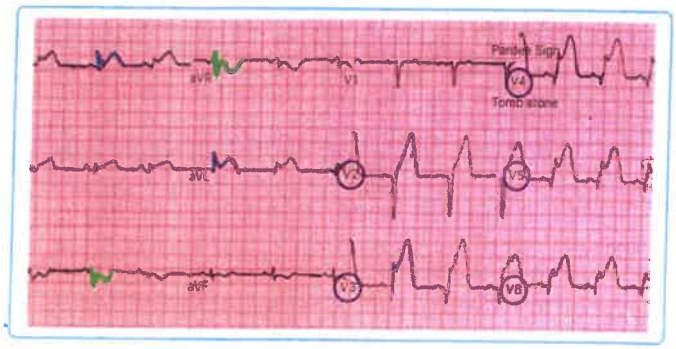
- Septum and some part of LV is studied by lead V1, V2 in ECG
- The major part of the LV is read by lead V3, and V4.
- Some part of LV facing the axilla is studied by lead V5, and V6 (lateral wall of the heart)
- Some part of RV is studied by V1
- Chest lead used to study RV - V4R (not used routinely)
- RV is studied by scanning lead II, III, aVF
- Anterior wall MI is most common because the commonest blood vessel of heart involved in atherosclerosis is LAD





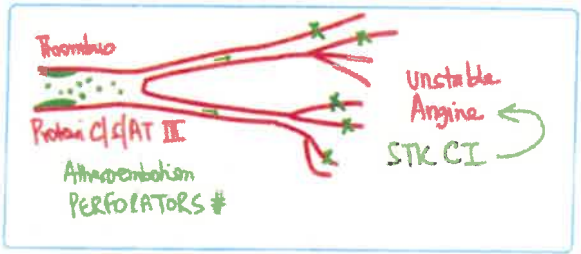
Blood Vessel	Surface	Leads
Left circumflex artery	Lateral wall MI	I, aVL, V5, V6
Septal branch artery	Septal MI	I, aVL, V1, V2
Left anterior descending	Anterior wall MI	I, aVL, V1-V4
Left main coronary artery	Extensive anterior wall MI	I, aVL, V1-V6
Posterior descending artery	Posterior wall MI	V1-V4= STD V7,V8,V9= STE
Left anterior descending	NSTEMI anterior wall	V1-V4= STD
Right coronary artery	Inferior wall MI	Lead II, III, avF

ECG Interpretation 00:56:12



- Tombstone pattern/ Pardee sign (Pardee waves refers to the symmetric inversion of T waves during an acute coronary syndrome or myocardial ischèmia.)
- Significant ST elevation present in V2, V3, V4, V5,V6, Lead I, aVL and reciprocal change ST elevation is present in Lead III, aVR
- The above findings indicate: Extensive anterior wall MI
- High risk of developing Cardiogenic shock, Pulmonary edema, and Sudden cardiac death
- Sudden cardiac death: Death within 1 hour of being seen or heard
  - It could be Ventricular fibrillation or Pulseless Ventricular Tachycardia (PVT) and need defibrillation

Non-ST Elevation-Acute Coronary Syndrome (NSTE-ACS)/ Unstable Angina 00:59:32



Unstable angina: Plaque fissure leading to thrombus formation

↓

Inbuilt anticlotting system
(Protein C, Protein S, Antithrombin III)

↓

Breaks the clot into fragments

↓

Fragments block the perforators

↓

Chest pain

- Therefore, Atheroembolism is seen in NSTEMI
- Thrombolysis/Streptokinase: Contraindicated in NSTEMI/unstable angina because it would further disseminate the fragments and worsen the symptoms

Important Information

- **Acute chest syndrome:** Seen in sickle cell anemia patients during sickling episodes → RBCs becomes sticky and occlude the coronary micro-circulation leading to chest pain.
- **Acute coronary syndrome (ACS):** The problem is atherosclerosis

Drugs Used

- **Anti-ischemia measure:** Initial approach
  - Sublingual Nitrates or buccal spray
  - β-blocker (Metoprolol): Decreases O_2 consumption of the heart, decrease HR, and reduces hypoxia.
  - Morphine: Given if there is no improvement with nitrates.
    - → Morphine also reduces pulmonary edema.
    - → Disadvantage: Stimulates vagus nerve and person can have a vomiting sensation.
- **Dual Antiplatelet drugs:** Aspirin + Clopidogrel / Prasugrel / Ticagrelor
  - Clopidogrel may not show good results, especially in Indian patients
  - P2Y12 inhibitors: Prasugrel / Ticagrelor
  - Commonly used nowadays: Aspirin + Ticagrelor
- **IV Antiplatelet Drugs (at the time of PCI):** Cangrelor, Eptifibatide, Tirofiban
- **Parenteral anticoagulation:** Low molecular weight heparin - Enoxaparin

PYQ: AIIMS 2019

ACS			
	STEMI	**NSTEMI**	**UA**
ECG	ST ↑	ST Ⓝ/↓	ST Ⓝ/↓
Trop I	x 2	x 2	Normal

01:13:52

Prinzmetal Angina

- Not a component of Acute coronary syndrome
- Common in Female
- Chest pain at rest
  - More in morning hours/Winter season - because prinzmetal angina is related to vasospasm
- Past medical history of Raynaud's phenomenon (fingers changing white → blue →red)
- ECG showing ST elevation: It is the only angina with ST elevation
  - Also known as Variant Angina
- Trop I is normal - vasospasm will not cause necrosis/death of cardiac muscles
- Rx:
  - In Emergency: Nitrates
  - To prevent episodes: CCBs (Amlodipine) are used as they prevent the spasm

Types of Myocardial Infarction

00:17:26

Types of MI	Description
Type-1: Spontaneous	• Spontaneous MI as a result of intraluminal thrombus as a consequence of either atherosclerotic disease or dissection
Type-2: Secondary to ischemic imbalance	• An imbalance between myocardial oxygen supply and demand that is not precipitated by coronary artery disease (but can occur in the presence of stable coronary artery disease) • Example: Cocaine overdosage, Severe Anemia
Type - 3: Resulting in death before Biomarker values	• Death occurring before biomarkers can be taken where the clinical presentation and ECG changes support a diagnosis of MI • Sudden death is due to tachyarrhythmias (V fibrillation, PVT) and Mobitz II heart block
Type 4A: Related to percutaneous coronary intervention	• A rise of cTn greater than 5 times the upper limit of normal in association with any of the symptoms of myocardial ischemia; new ECG changes; angiographic loss of a coronary artery or persistent slow/no-flow; new regional wall motion abnormality demonstrated on imaging
Type 4B: Related to stent thrombosis	• Detected at either angiography or autopsy with an associated biomarker rise • This is Latent stent thrombosis
Type 4C: Related to re-stenosis	• MI in the context of angiographically significant re-stenosis within the infarct artery
Type 5: Related to coronary artery bypass grafting (CABG)	• Associated with CABG and a biomarker rise 10 times the upper limit of normal in association with any of: new pathological Q waves or LBBB; new graft or coronary artery occlusion; new regional wall motion abnormality demonstrated on imaging.

Important Information

Mobitz II heart block

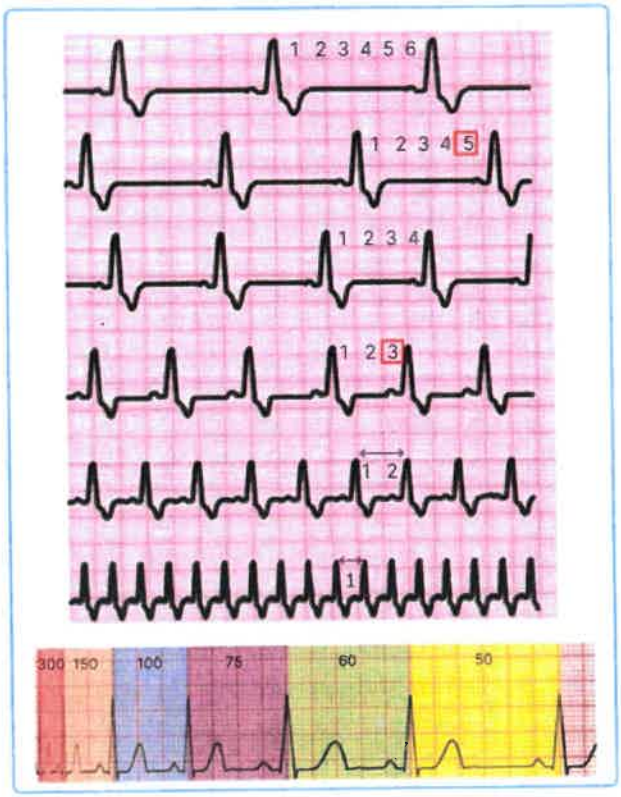
- Sudden death due to bradyarrhythmias after MI.
- Rx:
  - Atropine 1 mg thrice can be given according to AHA guidelines 2020
  - Main management: Trans Cutaneous Pacemaker

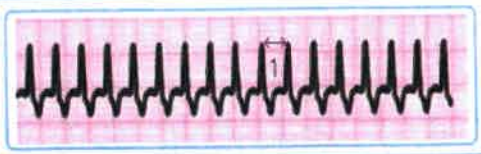
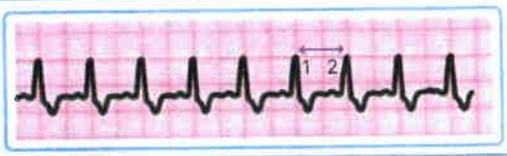
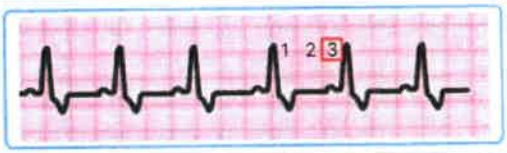
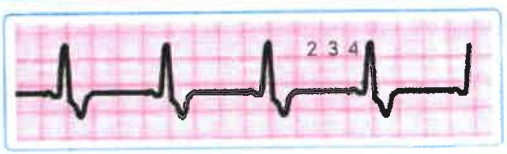
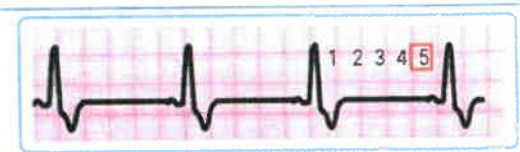
ECG

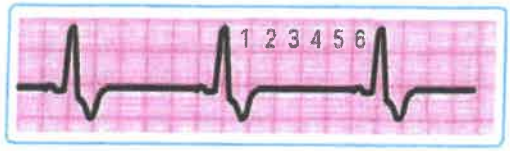
01:25:05

- Heart Rate = $\frac{300}{\text{R-R interval}}$
- R-R interval = No. of large squares
- Therefore, when the number of large squares increases, the Heart rate decrease

Example:



	• R-R interval: 1 large square • HR= 300/1 = 300.
	• R-R interval is composed only of 2 large squares • HR = 300/2 = 150 bpm. • Indicates Tachycardia.
	• R-R interval is composed of 3 large squares • HR = 300/3 = 100 bpm
	• R-R interval is composed of 4 large squares • HR = 300/4 = 75 bpm.
	• R-R interval is composed of 5 large squares • HR = 300/5 = 60 bpm.



- R-R interval is composed of 6 large squares
- HR = 300/6 = 50 bpm
- Indicates Bradycardia.
- A fit individual with bradycardia - High vagal tone might be the cause.

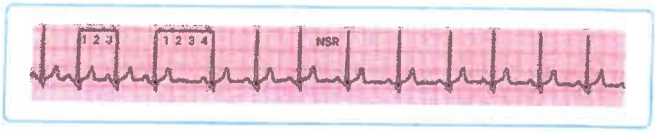
No. of Large Squares	Heart Rate
<3	>100 bpm (Tachycardia)
>5	<60 bpm (Bradycardia)

Important Information

- **Inferior wall MI**
  - Right ventricle is involved
  - Inferior wall MI with bradycardia:
    → Vagus nerve innervates the wall of the RV along with the conduction system
    → Damage to RV wall → Irritation of vagus nerve → Bradycardia, Vomiting, Hiccoughs
  - Only type of MI that can have a history of significant gastrointestinal complaints.
  - Only type of MI in which IV fluids are administered to maintain BP in case of bradycardia with hypotension.
- Lead used to calculate HR in a 12 lead ECG - Lead II/ Rhythm strip

Sinus Arrhythmia

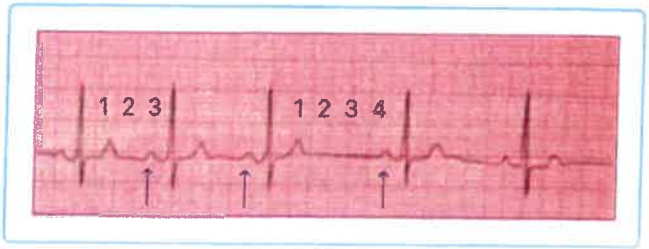
01:32:35



- Normal sinus rhythm with variable R-R interval that is not tachyarrhythmia is known as Sinus arrhythmia.
- Occurs as a result of vagal tone variation which is produced by the phases of respiration.

Q. An 18-year-old female patient presented with recurrent infections of the respiratory tract. ECG was given. What's the diagnosis?

PYQ: FMGE 2020



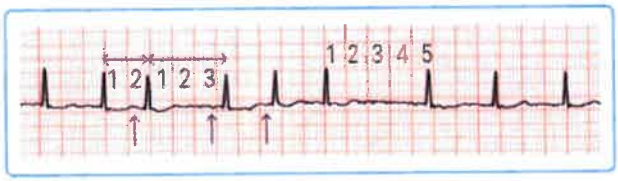
A. Wolf Parkinson White Syndrome
B. Atrial flutter
C. Atrial fibrillation
D. Sinus arrhythmia

Explanation

- ECG shows variable RR interval like in the above figure.
- Here HR is varies from 100 to 75bpm (this is not tachyarrhythmia: HR>100bpm)

- P wave with QRS complex present - normal sinus rhythm.
- Normal sinus rhythm + Varying HR = Sinus arrhythmia.

Atrial fibrillation 01:35:22



- An ECG showing both increasing and decreasing variations of HR with an absence of a P wave and the presence of twitching indicates Atrial Fibrillation.
- Due to atrial twitching, clots develop in the Left Atrial Appendage is seen.
  - Blockage in the brain causes stroke
  - Blockage in the mesenteric arteries causes infarction of bowel with lower GI bleeding presenting as very severe abdominal pain, and if not identified will lead to gangrene of the intestine
- There is a variable R-R interval
- The pulse in AF is often described as irregularly irregular.
- Atrial Fibrillation is the most common sustained arrhythmia

Important Information

PYQ: NEET PG 2021

- **Atrial Fibrillation**
  - M/c sustained arrhythmia in Hypertrophic cardiomyopathy.
  - M/c sustained arrhythmia in HTN.
  - M/c sustained arrhythmia in the > 65 yrs age group.
- **Holiday Heart Syndrome:** Sudden cardiac death in an alcoholic post-binge drinking.
  - Consumption of alcohol on a long-term basis leads to DCM and structural damage of the heart resulting in AF.
  - Post-binge drinking → inhibition of vagus nerve → Ectopic foci in AF increase HR (pathological tachycardia) → Decreased CO

Pathological Tachycardia

- All tachyarrhythmias will contribute to a decrease in blood pressure.

Heart beating disproportionately fast
↓
↓ Time for relaxation of heart/↓Diastole
↓
↓Filling of heart
↓
↓EDV
↓
↓SV

Treatment

R	Rate control	• First step • Most commonly used drugs - Esmolol (short-acting β-blocker)/ Verapamil (CCB)
A	Anticoagulants	• Rheumatic Heart Disease/ Mitral Stenosis/ Left Atrial appendage dilatation: Warfarin is used • Non-rheumatic AF: Newer Oral Anticoagulants (N.O.A.C)
C	Chemical cardioversion	• Use of certain drugs like amiodarone/ Ibutilide to shut down the ectopic focus in the left atrium
E	Electrical cardioversion	• Only used in case of failure of chemical cardioversion. • 200 joules biphasic of DC shock will be deployed

- The order of treatment should not be bypassed

CHA_2DS_2-VASc Score

01:52:20

- A clinical prediction rule for estimating the risk of stroke in people with non-rheumatic atrial fibrillation (AF).

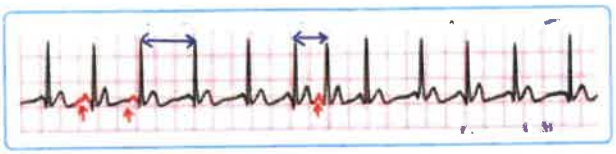
Letter	Component	Score
C	Congestive Heart Failure	1
H	Hypertension	1
A_2	Age ≥ 75 years	2
D	Diabetes	1
S_2	Stroke	2
V	Vascular disease	1
A	Age ≥ 65 years	1
Sc	Sex category, female	1

- Maximum total score = 9
- Score = 0 - no therapy is preferred
- Score = 1 - Aspirin or oral anticoagulation
- Score ≥ 2 - Oral anti-coagulation

Condition	Recommendation	
Nonvalvular atrial fibrillation	CHA_2DS_2-VASc score of 0	• Calculate CHA_2DS_2-VASc score • Aspirin or no antithrombotic
	CHA_2DS_2-VASc score of 1	Aspirin or OAC
	CHA_2DS_2-VASc score of ≥2	OAC
Rheumatic mitral valve disease	With atrial fibrillation, previous embolization, or atrial appendage thrombus, or left atrial diameter > 55 mm	OAC
	Embolization or appendage clot despite OAC	OAC plus aspirin despite OAC
Mitral valve prolapse	Asymptomatic	No therapy
	With otherwise cryptogenic stroke or TIA	Aspirin
	Atrial fibrillation	OAC

Multi-Focal Atrial Tachycardia

01:55:54

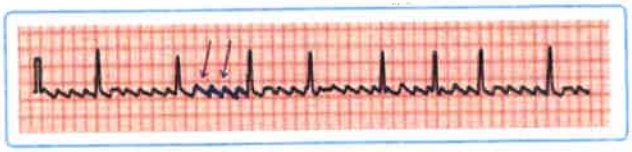


- Patient with COPD with recurrent episodes of palpitations and dizzy spells.
- P waves are present - variable morphology/height
  - ≥3 different varieties of P wave should be present
- Variable R-R interval (HR >100/min)
- Rx: DOC: Verapamil
- DC Shock is Contraindicated.

Atrial Flutter

PYQ: NEET PG 2022 01:59:52

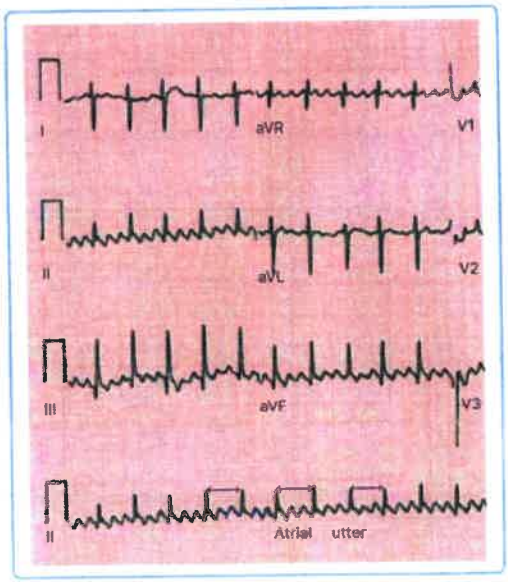
- Atrial flutter is a variant of Atrial fibrillation, only the site of heart involvement is different.
- Characteristic ECG finding: Saw Tooth Pattern.
- The macro re-entrant circuit around the cavotricuspid isthmus in the right side of the heart is responsible.
- Rx:
  - Rate control with esmolol
  - Anticoagulants
  - Ibutilide
  - If Ibutilide fails - Low-intensity DC shock with 25-50 J.



Important Information

- **Saw Tooth Waves in EEG are seen in REM sleep.**

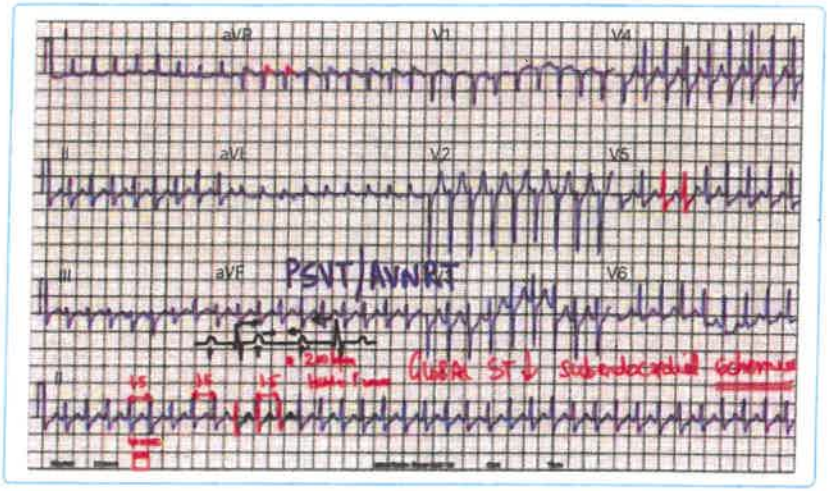
Q.



- R-R interval is not changing and Saw tooth pattern is seen indicating Atrial flutter

Paroxysmal Supraventricular Tachycardia

02:06:02



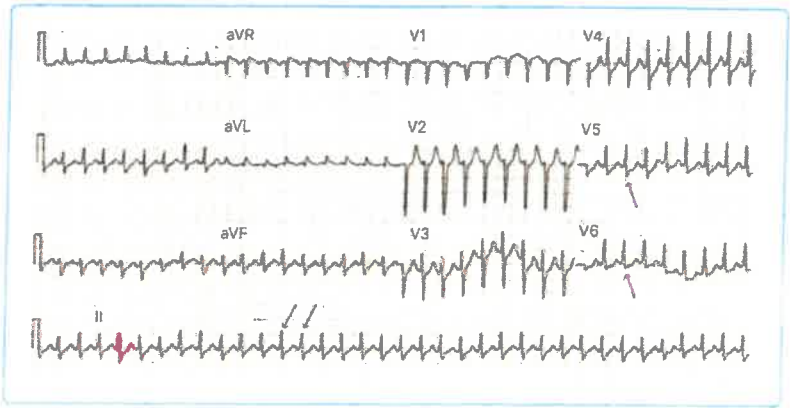
- Based on the HR in lead 2, the ECG shows tachyarrhythmia.
- Constant R-R interval
- QRS is on the lower side of the normal
- Global ST depression is present - Evidence of sub-endocardial ischemia.
- Merged P and T wave - **Hidden P wave**
- Also known as **AV nodal re-entrant tachycardia** (AVNRT) i.e., the narrow QRS complex tachycardia
- Rx:

PYQ: NEET PG 2019

02:35:05

Crashing Patient	SBP>90mmHg	For Prevention
Synchronized DC shock (Cardioversion)	• Carotid sinus massage or Facial ice pack (child) • Adenosine (S/E: Transient bronchospasm) • Synchronized DC shock: 120-200 J (is done only when adenosine fails to work)	Verapamil

Q. A diabetic patient presents with palpitations. BP on admission is 122/80 mm hg. Which of the following is the correct intervention?



A. Adenosine
B. Cardioversion
C. Amiodarone
D. PCI

PYQ: FMGE 2022

Explanation

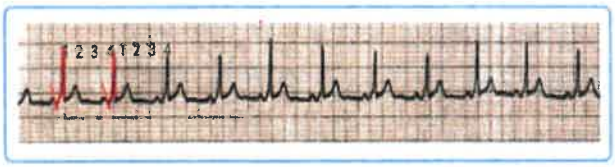
- In the given ECG a narrow QRS complex and ST depression along with a merged P and T wave is seen indicating PSVT.
- Since the BP is toward normal. Therefore, Adenosine is given.

Important Information

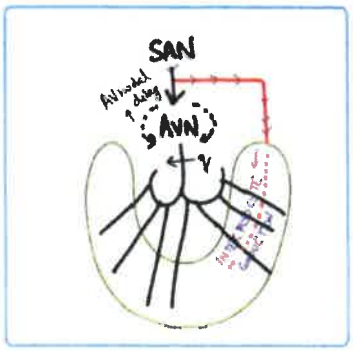
- **Cardioversion: Uses synchronized DC shock.**
- **Defibrillation: Uses non-synchronized DC shock, used for V. Fib and PVT**

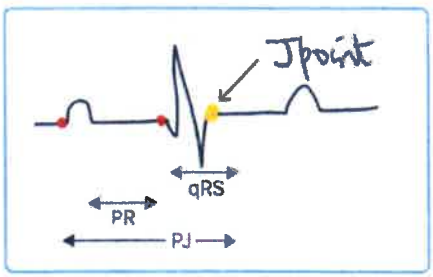
Wolf Parkinson White (WPW) Syndrome

PYQ: FMGE 2021 02:17:42



- There is change in the slope of the upswing of R wave - Delta waves.
- There are 2 conduction pathways in the heart:
  - Normal conduction system
  - Abnormal conduction pathway - Bundle of KENT
- In this figure, the black line indicates the normal conduction pathway, and the red line indicates the pathological pathway.
- In normal conduction pathway:
  - Current goes from SA node to the AV node and there is an AV nodal delay.
  - This pathway has its own self-limiting mechanisms
- In accessory pathway:
  - Fast conduction: It bypasses the normal pathway and the impulse from the SA node travels via the abnormal pathway/ bundle of Kent.
  - This pathway does not have any decremental mechanisms.
- Features: Palpitations, Recurrent syncopal attacks, and H/O sudden cardiac death in sibling
- ECG findings:
  - Short PR interval because of fast conduction in the heart (PR interval is inversely related to HR)
  - 'q' wave is absent - Since the current is not involving the AV nodal pathway and bundle of HIS
  - Change in the upswing of R wave - Delta waves
  - Broader RS complex:
    - → Delta wave contributes to a broader RS complex
    - → Intermyocyte conduction: The current travels through the myocytes (slower) instead of the Purkinje fibers (Fastest conducting fiber in the heart)
  - PJ interval remains normal
    - → PJ=(↓)PR+(↑)qRS
    - → The amount of PR reduction is equal to the increase in the RS complex





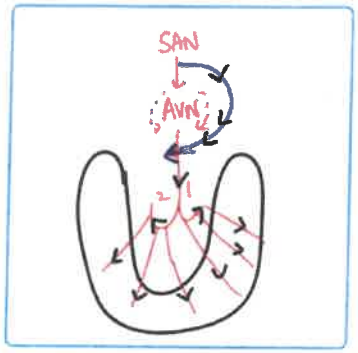
Important Information

- **Delta waves (slowest wave) in EEG is seen in Stage 3 of NREM sleep**

Lown Ganong Levine Syndrome (LGL)

PY Q: NEET PG 2021 02:28:22

- It is a pre-excitation syndrome of the heart.
- There is an alternate conduction system - The James bundle.
- The current from the SA node bypasses the AV node and reaches the bundle of HIS and Purkinje fibres.
- ECG findings:
  - Short PR interval
  - 'q' wave is present
  - Normal QRS complexes
  - PJ interval is short
    → PJ=(↓)PR+(n)qRS



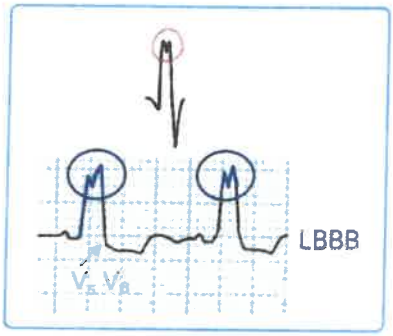
Treatment for WPW and LGL Syndrome

- **TOC:** Radiofrequency ablation of abnormal pathway.
- **DOC:**
  - In emergency: Procainamide
  - For prevention: Oral Flecainide (helps to slow the conduction in the accessory pathway)

QRS abnormalities

02:38:34

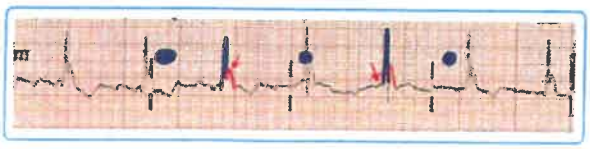
1. Narrow QRS complex tachycardia: Supra Ventricular Tachycardia
2. Wide qRS complex tachycardia: Ventricular Tachycardia/ Polymorphic VT
3. Osborn wave: Hypothermia (core temperature <35 degrees)
4. Rabbit ear appearance or M/W in the peak of qRS: Bundle branch block.
   - In LBBB: Seen in V5, V6.
   - In RBBB: Seen in V1, V2



Hypothermia

Core Body Temperature in Hypothermia	
32-35° Celsius	Mild hypothermia
28-32° Celsius	Moderate hypothermia
<28° Celsius	Severe hypothermia

- **ECG findings**
  - Prolonged PR interval - due to slowing of the heart in hypothermia.
  - An inverted P wave might be present - due to AV node firing the atria retrogradely
  - A deflection/notch in the downswing of R wave - Osborn wave



- Ideal site for checking core temperature: Pulmonary Artery (invasive)
- Preferred site for checking core temperature: Lower esophagus

MCQ

Q. Which of the following is not an adaptation at high altitude?

A. Pulmonary vasoconstriction
B. Increased heart rate
C. Respiratory acidosis
D. Increased cerebral blood flow

Solution

- In High altitudes: low PO_2 causes pulmonary vasoconstriction and cerebral vasodilatation
  - Hypoxia causes dilatation of all blood vessels in the body except pulmonary artery
- Cerebral vasodilatation leads to headache in this patient
- Hypoxia leads to tachycardia
- Since high altitude results in rapid respiration → CO_2 washout → it will cause respiratory alkalosis, not acidosis.
- Therefore, ANS: Respiratory acidosis

Q. Which of the following is not associated with central cyanosis?

A. Meth hemoglobinemia
B. Hypothermia
C. High altitude
D. Pulmonary AV malformation

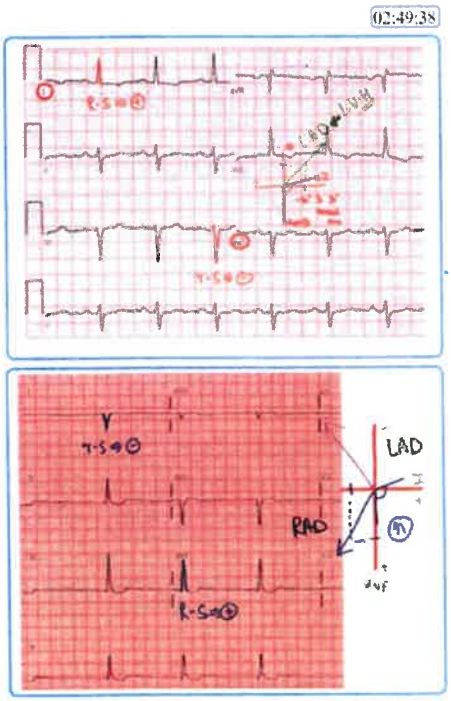
Solution:

- Meth hemoglobinemia: oxidized Hb ($Fe^{2+} \rightarrow Fe^{3+}$) leads to anemic hypoxia and cause central cyanosis
- **Hypothermia**: causes localized vasoconstriction that is associated with peripheral cyanosis, not central cyanosis.
- High altitude leads to hypoxic hypoxia and causes central cyanosis
- Pulmonary AV malformation: extracardiac shunt leads to mixing of arterial & venous blood and causes central cyanosis

Axis Deviation

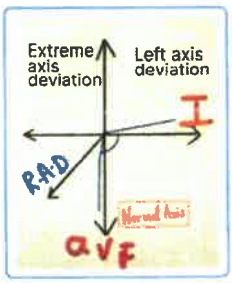
Solution:

- In a normal individual,
  - X-axis is lead I
  - Y-axis is lead aVF
- Normal axis of the heart ranges between -30 to +90 degrees
- **Left Axis deviation**
  - R wave is bigger than S wave therefore R wave – S wave gives +ve value in Lead I
  - R wave is smaller than S wave therefore R wave – S wave gives -ve value in Lead aVF
  - On vector analysis: It shows left axis deviation for the above ECG
  - Left axis deviation seen in left ventricular hypertrophy
- **Right Axis deviation**
  - R wave is smaller than S wave therefore R wave – S wave gives -ve value in Lead I
  - R wave is bigger than S wave therefore R wave – S wave gives +ve value in Lead aVF
  - On vector analysis: it shows right axis deviation for the above ECG
  - Right axis deviation seen in right ventricular hypertrophy
- **Extreme Axis deviation**
  - If in ECG, the R-S valve is negative in both Lead I and lead avF indicates extreme axis deviation
  - It is seen Ventricular Tachycardia



Q. MBBS student was asked to diagrammatically make the axis deviation of a patient. The following schemata shown represent.

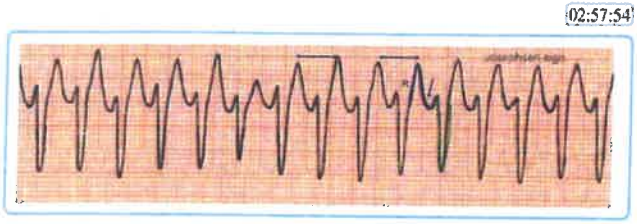
A. **Right ventricular hypertrophy**
B. Left ventricular hypertrophy
C. Biventricular hypertrophy
D. Normal axis in obese individual



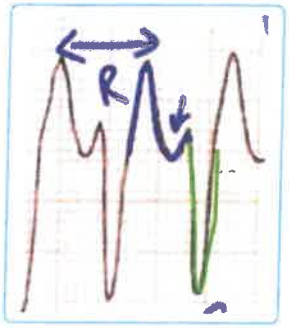
Solution

- This schematic diagram, shows Right axis deviation indicating Right ventricular hypertrophy

Ventricular Tachycardia



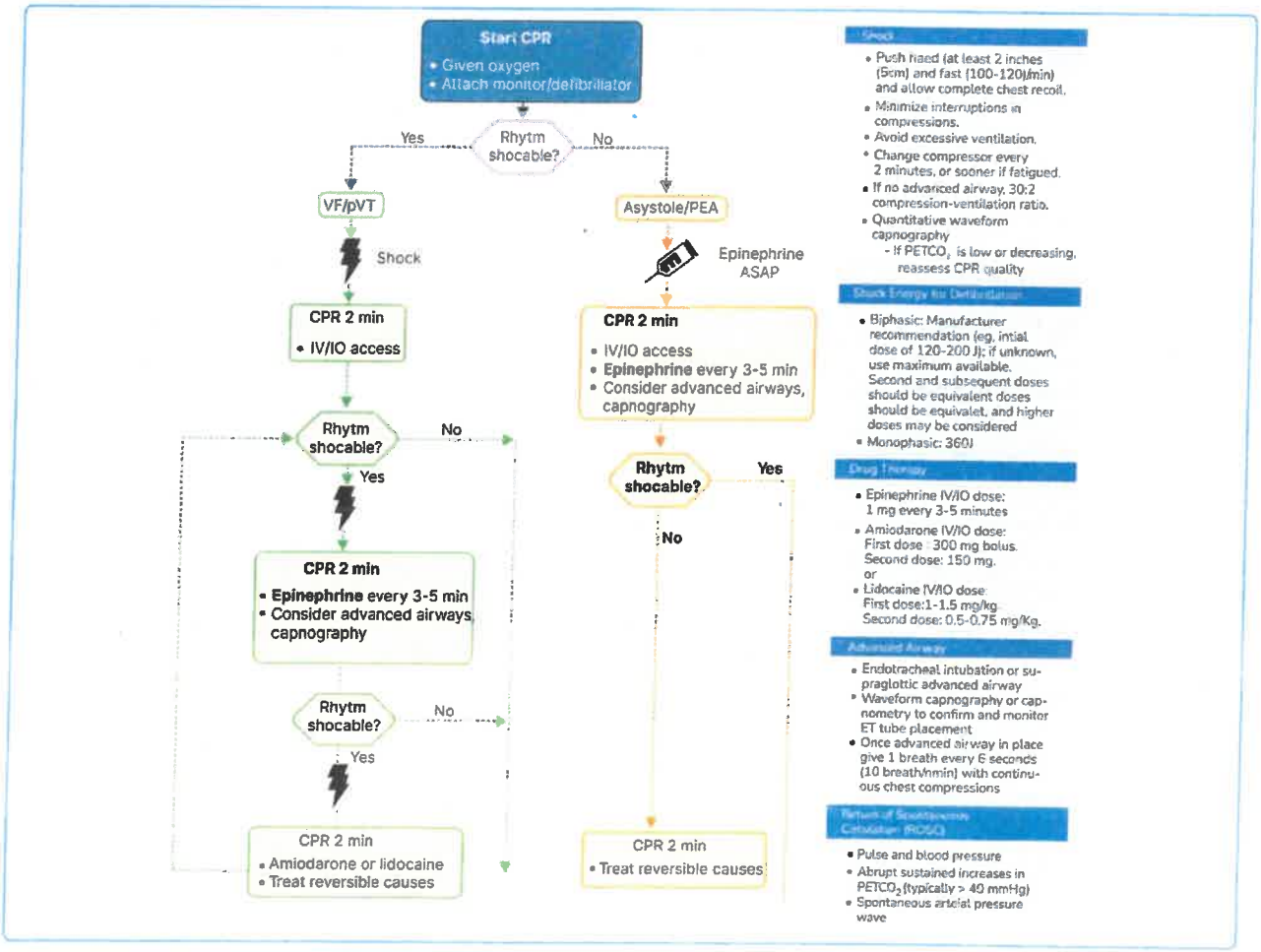
- HR ≃ 200 bpm
- Shortened R-R interval
- Broad qRS complex (Normal qRS complex is 2-2.5 small squares i.e., 80 to 100 milliseconds)
- Tachycardia with Broad qRS complex indicates VT
- All of the broad qRS complexes are monomorphic. Therefore, the given ECG shows Monomorphic VT
- **Josephson Sign:** A slight notch which is exclusively noted in VT



Treatment

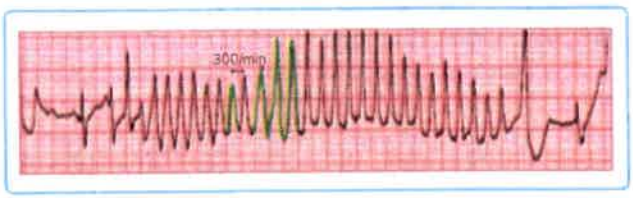
PYQ: NEET 2021, 2022, 2023

- Hemodynamically stable VT: Amiodarone.
- Hemodynamically unstable VT: DC shock of 200 J, followed by CPR until the normal rhythm is established.



Torsades de pointes/ Polymorphic VT

PYQ: FMGE 2021 03:06:27



- There is variation in size of the broad qRS complex - Polymorphic VT
- HR ≃ 300 bpm
- **Causes**
  1. Electrolyte imbalances (↓ Mg, ↓ K, etc.)
  2. Class Ia, Ic, and III of anti-arrhythmic drugs.
- **DOC:** $MgSO_4$

Electrical Alternans

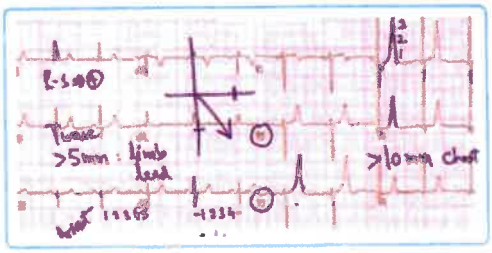
03:08:42

- Variable amplitude of the qRS complex
- HR ≃ 125 bpm
- Normal sinus rhythm with slight tachycardia
- Seen in Pericardial effusion
- It occurs due to the "swinging movement of the heart" in a bag of fluid surrounding the heart.

Important Information

- Unrecordable BP is present in 2 condition - Cardiac Tamponade & Tension Pneumothorax.
  - **Cardiac Tamponade:** Breath sounds - present
  - **Tension Pneumothorax:** Breath sounds - absent

Hyperkalaemia



- HR >60 bpm.
- Tall-tented T waves are present (>10mm in chest leads, >5mm in limb leads).
- In hyperkalemia, ECG is not reliable. It is just supportive evidence.
- Serum electrolytes - more reliable
- Increased R-R interval - K^+ slows the heart
- ST elevation is seen
- The amplitude of the P wave decreases and gradually disappears.
- PR interval increases and the broad QRS complex merges with T wave - the Sine wave pattern.
  - Occurs just before diastolic arrest
- Hyperkalemia can lead to death by causing diastolic arrest (K^+ slows the heart)

Hypokalemia

03:22:00

- T wave inversion - characteristic feature
  - In progressive cases, the T wave could even be absent.
- ST depression would occur.
- Pseudo-P pulmonale - the vertical height of P wave >2.5 mm in absence of pulmonary artery HTN
- Prominent U wave will be present

General rule for correction: To raise K^+ by 1 mEq/L, 200 mEq of KCl is required

- The lower limit of normal for K^+ = 3.5 mEq/L
- Threshold = 3.0 mEq/L
- From threshold (3.0mEq/L) to 3.5mEq/L - Oral potchlor mixed with water/coconut water/banana
- Correction up to to threshold - KCl in IV fluid

Q. A patient on Amphotericin-B presented with K^+ = 2.3 mEq/L. Calculate the correction required.

Solution:

- K^+ = 2.3 mEq/L
- To reach the threshold (3.0mEq/L), an increment of 0.7mEq/L is needed.
- Therefore, according to the general rule: 0.7 x 200 = 140mEq KCl to be added to the bottle of IV fluid over 24hrs
- Amphotericin-B is a nephrotoxic drug associated with hypokalemia

2 CARDIOLOGY PART-2

Hypokalemia

00:00:28

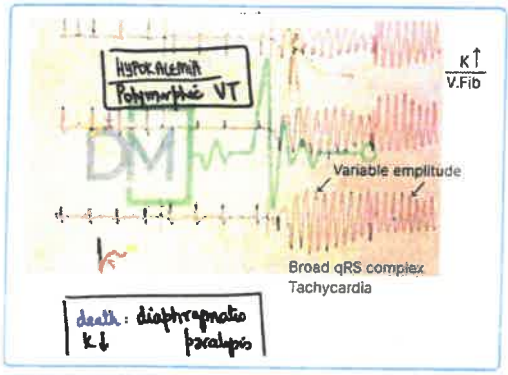
- ECG - Lead II findings

  - P wave slightly taller than normal
  - Visible ST segment depression (Yellow in inset image)
  - Concomitant T wave inversion (Green)
  - Presence of prominent U - wave (Purple)

Important Information

- **U wave can be found in the normal person's ECG due to delayed papillary muscle repolarisation.**

- Polymorphic VT can be seen in patients with hypokalemia
  - Broad QRS complex
  - R-R interval is reduced
  - Varying amplitude of QRS complexes
- Leading cause of death in hypokalemia - Diaphragmatic paralysis
- $MgSO_4$ is used therapeutically for the management of Torsades de Pointes which is seen with hypokalemia
- Hyperkalemia usually causes
  - Bradycardia
  - Diastolic arrest
  - Tachyarrhythmia seen in hyperkalemia is ventricular fibrillation



Drugs used in Management of Hyperkalemia

00:03:40

Chronic	Acute onset K↑
• Like in the case of grade 4 CKD 1. Patiromer 2. Sodium Zirconate 3. K bind - Sodium Polystrene sulphonate resin (oral or enema) o Side effect (Rare)-Colonic necrosis post enema component	1. Calcium gluconate or Calcium chloride (more efficacious) given IV 2. Insulin drip (Causes a redistribution of potassium) → Most effective drug → Supplemented with - Salbutamol / Albuterol (causes endogenous increase in insulin) 3. Furosemide - Kaliuria (Cause urinary loss of potassium) 4. Hemodialysis - Most effective method

- Calcium contracts so it antagonizes the effect of potassium which relaxes the heart
- Calcium carbonate (oral salt) is not used

Bradyarrhythmias

00:11:34

Sick Sinus Syndrome