

Basic Life Support & Automated External Defibrillation

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Objectives

At the end of the lecture , participants should be able to demonstrate :

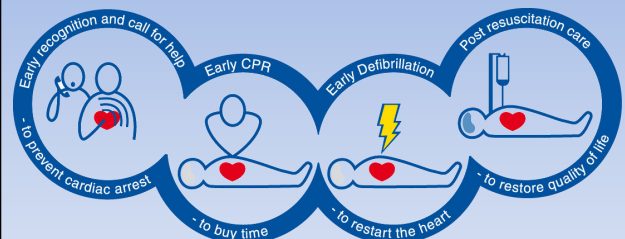
- How to assess the collapsed victim.
- How to perform chest compression and rescue breathing.
- How to operate an Automated External Defibrillator safely.
- How to place an unconscious breathing victim in the recovery position.

* REFERENCES : AHA AND EUROPEAN RESUSCITATION COUNCIL GUIDELINES

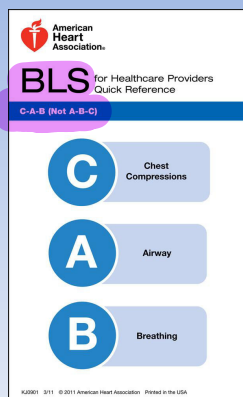
Background

- Survival to hospital discharge presently approximately 5-10%
- Bystander CPR is a vital intervention before arrival of emergency services
- Early resuscitation and prompt defibrillation (within 1-2 minutes) can result in >60% survival

Chain Of Survival

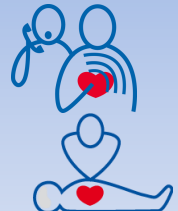


C-A-B



The three SSS's: safety, shake, shout

- Safety : Approach safely
- Shake : Check response
- Shout for help
- 30 chest compressions
- 2 rescue breaths



Approach Safely!

Scene

Rescuer

Victim

Bystanders

Check Response and Breathing



Call For Help

دو هار بجهنت امله مېر
بېبېلوي AED او اړخې اېښه اړا
موجود قبل ما اېډر CPR.



Check Carotid Pulses

look to see chest movements
*Look, Listen, Feel:
put your ear near the p's mouth to hear if feel any breaths
= palpates, palpation of sternal area (central area)
to feel the pulse with your fingers
to feel the pulse with your fingers



Start a High-Quality CPR

- Start compression within 10 seconds of recognition of cardiac arrest.
- Push Hard, Push Fast. (Rate of 100-120/30S) (5-6 cm depth, its ok to break ribs.)
- Allow complete chest recoil
- Minimize interruption (pauses can only be every cycle (2 min))
- Give effective breaths that make the chest rise.
- Avoid excessive ventilation.

30 Chest Compressions



Chest Compressions



- Place the heel of one hand in the centre of the chest
- Place other hand on top (Should be the dominant hand)
- Interlock fingers
- Compress the chest
 - Rate 100 min⁻¹ (100-120)
 - Depth 5-6 cm
 - Equal compression : relaxation
- When possible change CPR operator every 2 min

2 Rescue Breaths

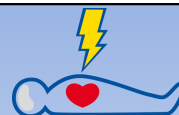
In AHA guidelines:
If you don't have
mouth piece don't do it,
due to infection
possibilities.



Continue CPR for 2 min

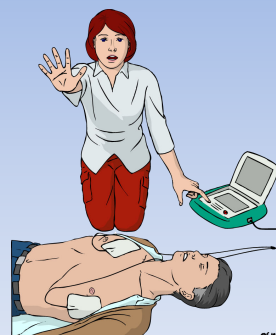


Defibrillation



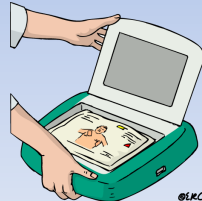
Attach AED

Follow voice prompts

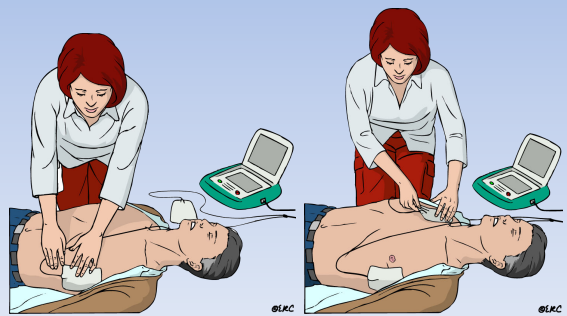


Switch on AED

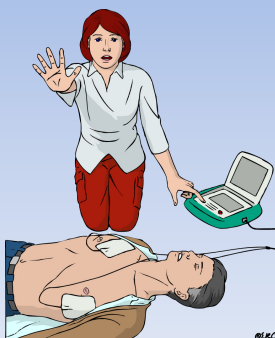
- Some AEDs will automatically switch themselves on when the lid is opened



Attach Pads to Victim's Bare Chest

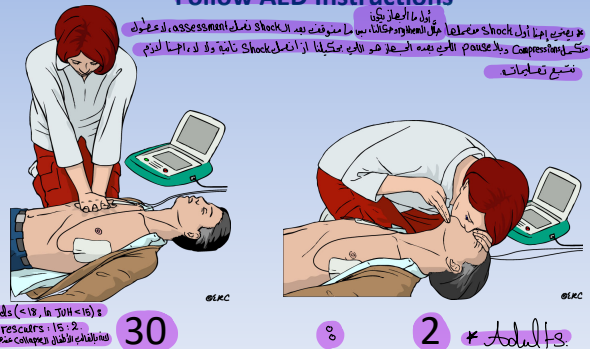


Shock Indicated



- Stand Clear
- Deliver Shock

Shock Delivered OR No Shock Advised Follow AED Instructions



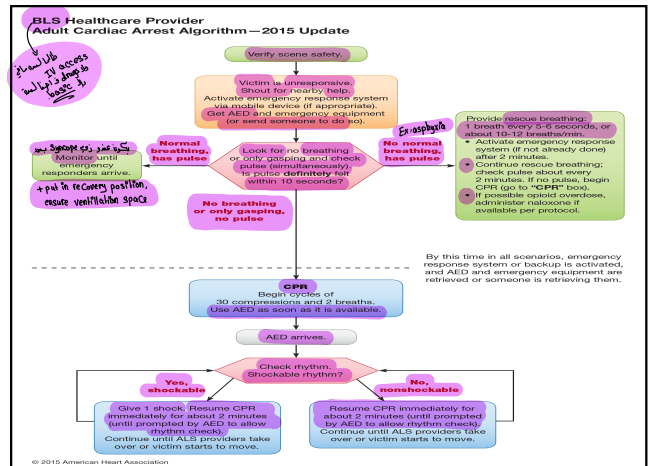
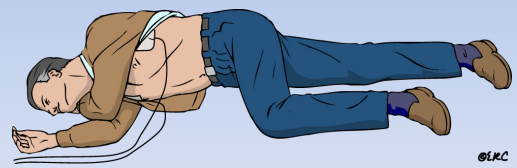
Manual Defibrillation 150 J

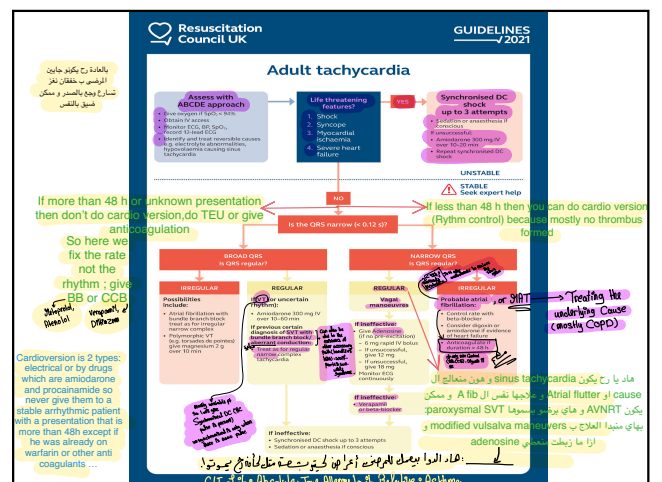
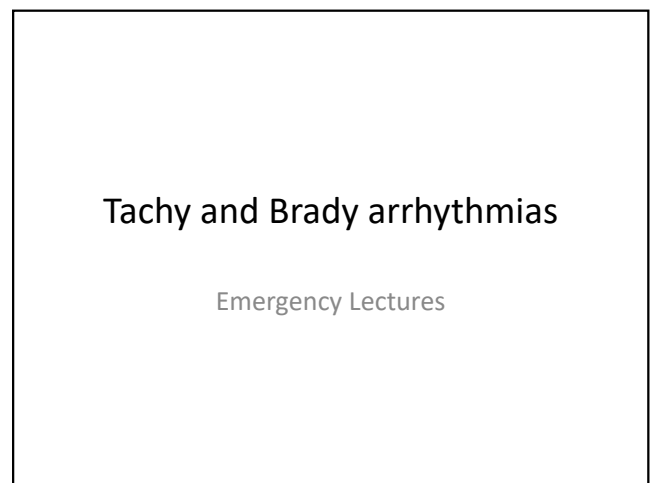
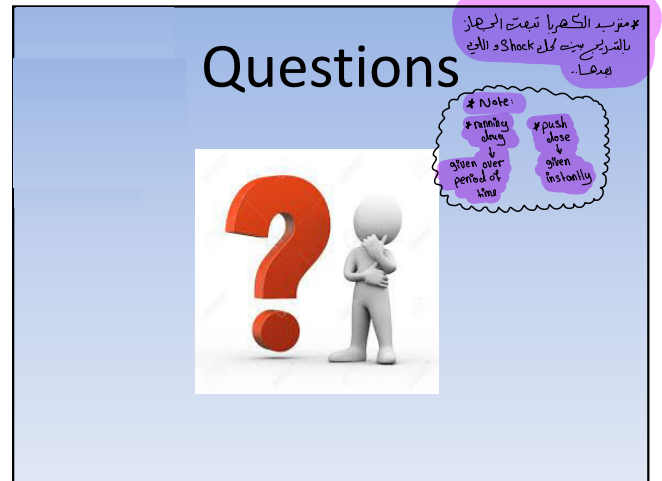
- 2 types → Biphasic (only this is currently used) : 120-200J for each pad
- Monophasic : 200-360J for each pad



If the victim starts to breathe NORMALLY place him in the Recovery Position

to decrease the risk of aspiration





Chest pain and Acute Coronary Syndrome

Emergency Medicine lectures

The differential diagnosis of central chest pain other than Ischemic chest pain

Types of Chest Pain

Musculo-skeletal MI, PE & aortic dissection are the serious ones that should be excluded

Pleuritic

Oesophageal

Pericarditis

Myocarditis

Aortic dissection

Cardiac-type chest pain

Typical description of cardiac type chest pain

Location	Duration	Character
<ul style="list-style-type: none"> Central Radiation Visceral type 	<ul style="list-style-type: none"> >15 minutes < 24 hours 	<ul style="list-style-type: none"> Not sharp Not stabbing Ache Burning Pressure Not movement or breathing related

Heart Score for major cardiac event

ما داخل ما نستخدق مع عشان نقرر اذا المريض يفضل ولا يروح هاد بس حكمي عن

The HEART Score for Chest Pain Patients in the ED		
History	<ul style="list-style-type: none"> Highly Suspicious Moderately Suspicious Slightly or Non-Suspicious 	<ul style="list-style-type: none"> 2 points 1 point 0 points
ECG	<ul style="list-style-type: none"> Significant ST-Depression Nonspecific Repolarization Normal 	<ul style="list-style-type: none"> 2 points 1 point 0 points
Age	<ul style="list-style-type: none"> ≥ 65 years > 45 - < 65 years ≤ 45 years 	<ul style="list-style-type: none"> 2 points 1 point 0 points
Risk Factors	<ul style="list-style-type: none"> ≥ 3 Risk Factors or History of CAD 1 or 2 Risk Factors No Risk Factors 	<ul style="list-style-type: none"> 2 points 1 point 0 points
Troponin	<ul style="list-style-type: none"> ≥ 3 x Normal Limit > 1 - < 3 x Normal Limit ≤ Normal Limit 	<ul style="list-style-type: none"> 2 points 1 point 0 points

Risk Factors: DM, current or recent (<one month) smoker, HTN, HLP, family history of CAD, & obesity

Score 0 - 3: 2.5% MACE over next 6 weeks → Discharge Home

Score 4 - 6: 20.3% MACE over next 6 weeks → Admit for Clinical Observation

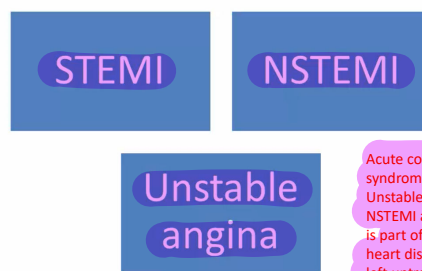
Score 7 - 10: 72.7% MACE over next 6 weeks → Early Invasive Strategies

Note:

The HEART score is a scoring system for patients presenting with chest pain at the emergency department.

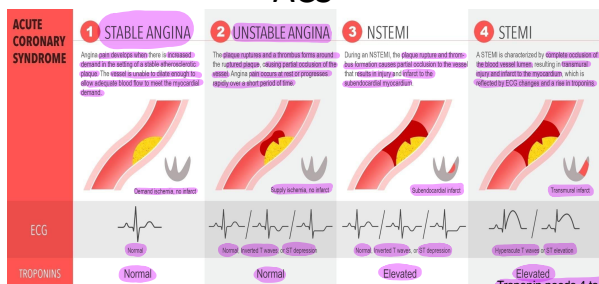
With the HEART score it is immediately clear which patient is eligible for discharge without additional tests or emergency invasive procedures should be done .

Acute Coronary Syndromes



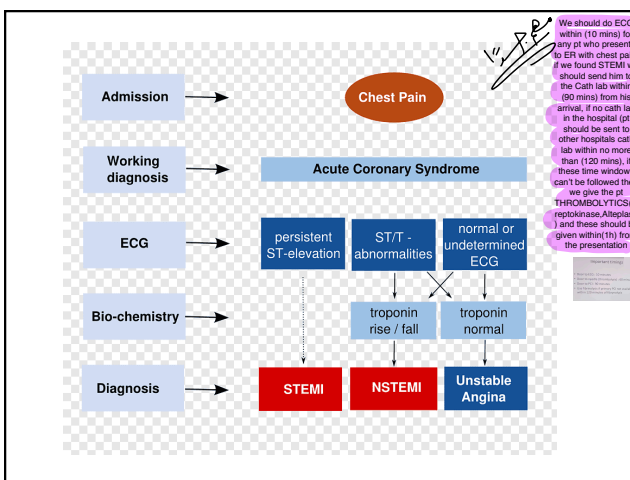
Acute coronary syndrome consists of : Unstable Angina , NSTEMI and STEMI . It is part of Ischemic heart diseases that if left untreated it will lead to acute cardiac event and death.

ACS





Acute coronary syndrome consists of : Unstable Angina , NSTEMI and STEMI

Stable Angina is part from Ischemic heart disease that is characterized by trivial central chest pain that last between 15-20 minutes , increased with exertion and relieved by rest or sublingual nitrates



UNSTABLE ANGINA CORE IM

PATHOPHYSIOLOGY	THROMBUS	CHEST PAIN SYMPTOMS
<p>Ruptured plaque with non-occlusive thrombus*</p> <p>*Occlusive thrombus would typically cause a full STEMI.</p> 	<ul style="list-style-type: none"> White Platelet-rich 	<ul style="list-style-type: none"> Acute chest pain With activity and rest
<p>Progressive mechanical obstruction</p> 	<ul style="list-style-type: none"> Red Fibrin-rich Same pathophysiology as stable angina. 	<ul style="list-style-type: none"> "Crescendoing angina" Chest pain worsens over days to weeks. Should not occur at rest

REMINDER UNSTABLE ANGINA = TROPONIN NEGATIVE
NSTEMI = TROPONIN POSITIVE

The two share the same pathophysiology and symptomatology - the difference is in the cardiac biomarkers!

Pre/In-hospital management of suspected ACS

Give the patient MONA

M: Morphin (pain management)

O : oxygen according to BTS protocol

N : Nitroglycerin for pain management

A : Anti-platelets (Aspirin)

Anti-thrombin

موننا صارت قديمة و اهم
اشياء بتغطي هي
Anti nitrates
platelets and anti
coagulants
الحاجة

If we suspect ACS

Do not routinely administer oxygen, but monitor oxygen saturation using pulse oximetry as soon as possible, ideally before hospital admission. Only offer supplemental oxygen to:

- people with oxygen saturation (SpO₂) of less than 94% who are not at risk of hypercapnic respiratory failure, aiming for SpO₂ of 94–98%
- people with chronic obstructive pulmonary disease who are at risk of hypercapnic respiratory failure, to achieve a target SpO₂ of 88–92% until blood gas analysis is available.

1.2.4 Assessment in hospital for people with a suspected acute coronary syndrome

1.2.4.1 Take a resting 12-lead ECG and a blood sample for troponin I or T measurement (see section 1.2.5) on arrival in hospital.

1.2.4.2 Carry out a physical examination to determine:

- haemodynamic status
- signs of complications, for example pulmonary oedema, cardiogenic shock and
- signs of non-coronary causes of acute chest pain, such as aortic dissection.

1.2.4.3 Take a detailed clinical history unless a STEMI is confirmed from the resting 12-lead ECG (that is, regional ST-segment elevation or presumed new LBBB). Record:

- the characteristics of the pain
- other associated symptoms
- any history of cardiovascular disease
- any cardiovascular risk factors and
- details of previous investigations or treatments for similar symptoms of chest pain.

1.2.5 Use of biochemical markers for diagnosis of an acute coronary syndrome

1.2.5.1 Take a blood sample for troponin I or T measurement on initial assessment in hospital. These are the preferred biochemical markers to diagnose acute MI.

1.2.5.2 Take a second blood sample for troponin I or T measurement 10–12 hours after the onset of symptoms.

1.2.6 Making a diagnosis

1.2.6.1 When diagnosing MI, use the universal definition of myocardial infarction^[2]. This is the detection of rise and/or fall of cardiac biomarkers (preferably troponin) with at least one value above the 99th percentile of the upper reference limit, together with evidence of myocardial ischaemia with at least one of the following:

- symptoms of ischaemia
- ECG changes indicative of new ischaemia (new ST-T changes or new LBBB)
- development of pathological Q wave changes in the ECG
- imaging evidence of new loss of viable myocardium or new regional wall motion abnormality^[3].

Anti-platelet and antithrombin therapy

Antiplatelet

*PL should crush them.

- Aspirin 300mg (unless allergic)
- Clopidogrel 300mg (unless very low risk)

Antithrombin

- Fondaparinux 2.5 mg sc
- Unfractionated heparin if PCI within 24 hours
- Reduce dose if significant bleeding risk
- Monitor clotting to guide dose if significant renal impairment (creatinine > 265 µmol/l)

STEMI management

~~if < 12 hours:~~

Aim for reperfusion as quickly as possible ✓

Primary PCI if possible ✓

Use fibrinolysis if Primary PCI not within 2 hours of possible fibrinolysis time ✓

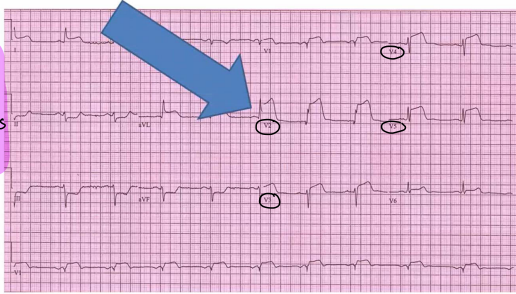
Give antithrombin with thrombolysis ✓

If MI or typical unstable angina we give dual anti platelets therapy

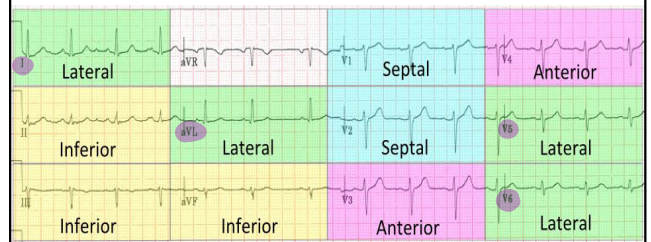
* Please remember that to say ST segment elevation you should have compared ST segment with TP segment not PR, 2 mill squares elevations at least on chest leads & 1 msq at least on limb leads

STEMI? ^{yes} Anterior MI

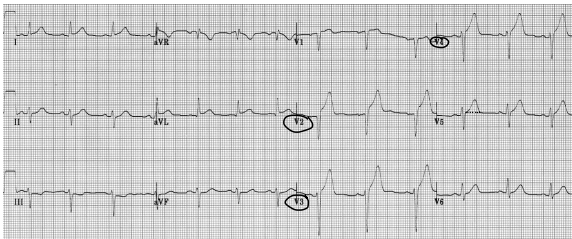
* please note the reciprocal changes (ST depressions on II, III & AVF)



Distribution of leads

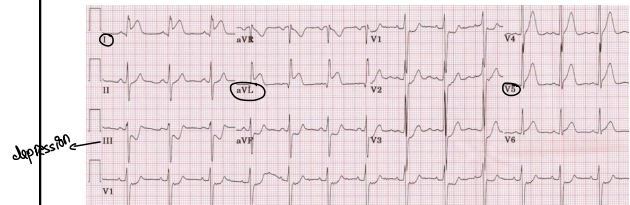


Anterior STEMI (Antero-Septal) - Dr Said-

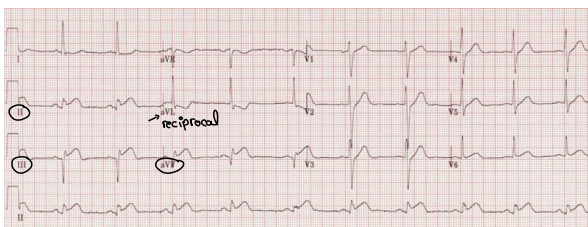


Lateral MI

with reciprocal changes on the inferior wall.

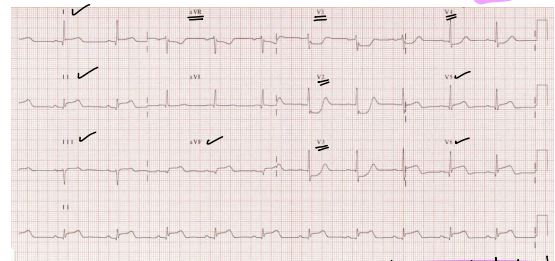


Inferior MI



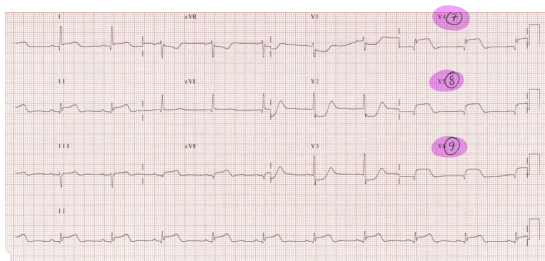
Posterior MI

* reciprocal changes will be on the Anterior leads (V1, V2, V3, V4)



Inferolateral STEMI. Posterior extension is suggested by:
* Horizontal ST depression in V1-3 which indicates the reciprocal changes
Tall, broad R waves (> 30ms) in V2-3
Dominant R wave (R/S ratio > 1) in V2
Upright T waves in V2-3

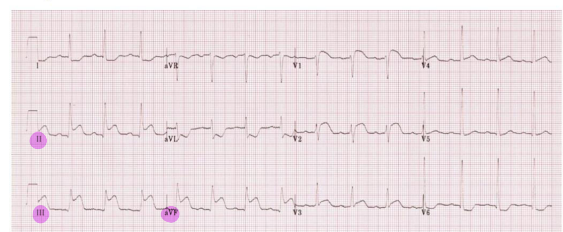
Posterior MI using posterior leads



Marked ST elevation in V7-9 with Q-wave formation confirms involvement of the posterior wall, making this an inferior-lateral-posterior STEMI (= big territory infarct!).

RV Wall MI

Example 1a

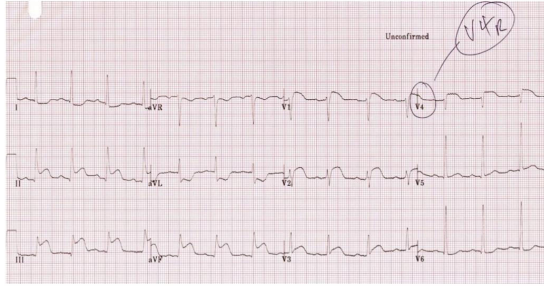


Inferior STEMI. Right ventricular infarction is suggested by:

- ST elevation in V1
- ST elevation in lead III > lead II

Example 1b

Repeat ECG of the same patient with V4R electrode position:

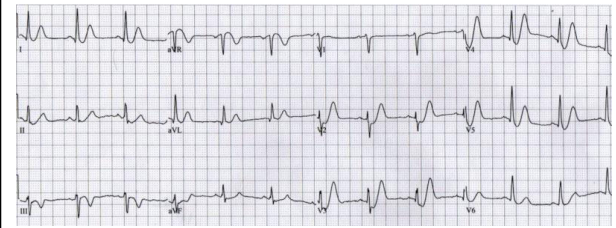


- There is ST elevation in V4R consistent with RV infarction

مثال 1ب

De Winter T Wave

مثال 1ب



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Note:

The de Winter ECG pattern is an **anterior STEMI equivalent** that presents *without* obvious ST segment elevation.

Diagnostic Criteria :

- Tall, prominent, symmetric T waves in the precordial leads
- Upsloping ST segment depression >1mm at the J-point in the precordial leads
- Absence of ST elevation in the precordial leads
- ST segment elevation (0.5mm-1mm) in aVR

"Normal" STEMI morphology may precede or follow the deWinter pattern

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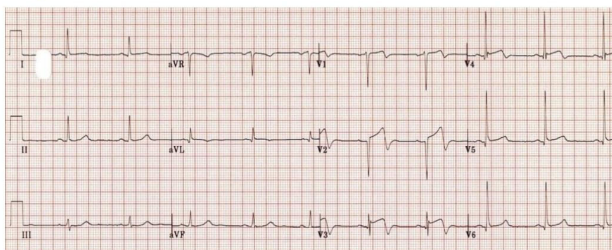
Wellens Syndrome

مثال 1ب

- Wellens syndrome is a pattern of **deeply inverted or biphasic T waves in V2-3**, which is highly specific for a **critical stenosis of the left anterior descending artery (LAD)**.
- Patients may be pain free by the time the ECG is taken and have normally or minimally elevated cardiac enzymes; however, they are at extremely **high risk for extensive anterior wall MI** within the next few days to weeks.
- Due to the critical LAD stenosis, these patients usually require invasive therapy; do poorly with medical management; and may suffer MI or cardiac arrest if inappropriately stress tested.

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Example 1

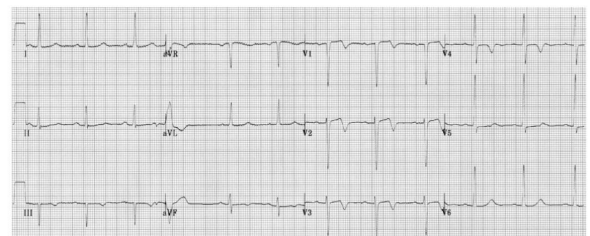


Wellens Syndrome (Type A Pattern)

- Biphasic precordial T waves with terminal negativity, most prominent in V2-3.
- Minor precordial ST elevation.
- Preserved R wave progression (R wave in V3 > 3mm)

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Example 2

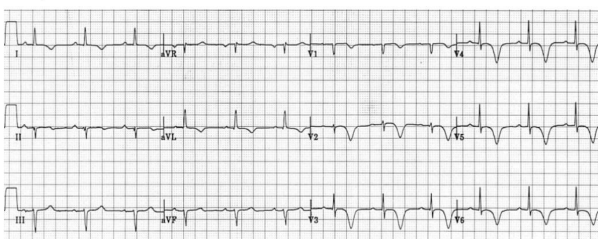


Wellens Syndrome (Type A Pattern)

- The biphasic T waves in V2-3 are characteristic of Wellens syndrome.

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Example 3



Wellens Syndrome (Type B Pattern)

- There are deep, symmetrical T wave inversions throughout the anterolateral leads (V1-6, I, aVL).

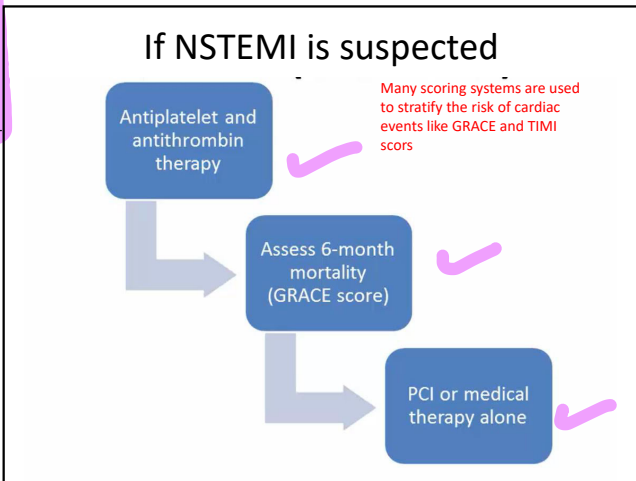
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If NSTEMI is suspected

- As soon as the diagnosis of unstable angina or NSTEMI is made, and aspirin and antithrombin therapy have been offered, formally **assess individual risk of future adverse cardiovascular events** using an established risk scoring system that predicts **6-month mortality** (for example, Global Registry of Acute Cardiac Events [GRACE]).

* STEMI → dual → Aspirin + preferably (Ticagrelor) → brisq®
Non-STEMI → clopidogrel → plavix®

* Also give:
- nitrates
- fluids
- then if no cath lab available
→ thrombolysis.



الفكرة انه هذول scores كلها ما بقدره يحكملي اذا اعطى admission للمريض ولا لا. بس بتبقى بال future risk

اما بخصوص اذا نروح المريض ولا ندخلهم في هاد يعتمد اذا كان واحد من ثلث اشياء:

- ECG changes أي STEMI
- troponin positive
- و حتى لو ما عنقو أي تغيرات لا عال

ECG nor troponin but he has very classic chest pain that came with rest and not being relieved with nitrates

TIMI UA/NSTEMI RISK SCORE

1) Age ≥ 65	1 point
2) ≥ 3 risk factors for CAD	1 point
3) Use of ASA (last 7 days)	1 point
4) Known CAD (prior stenosis $\geq 50\%$)	1 point
5) >1 episode rest angina in <24 h	1 point
6) ST-segment deviation	1 point
7) Elevated cardiac markers	1 point

Risk assessment (GRACE Score)

- Age
- Heart Rate
- Systolic Blood Pressure
- Creatinine
- Heart failure
- Cardiac arrest at presentation
- Cardiac enzyme elevation
- ST deviation

NSTEMI final management

Angiography

- Intermediate or higher risk
- Ischaemia returns
- Ischaemia on stress testing

Conservative

- Low risk

* Please note that my editing is according to Dr Ehab's mini-OSCE lectures to the second submedicine rotation group [October-November]. So regarding excluded topics, slides, or other.. you better check if its the same for your group too.. As the following lecture has several topics that are not required for the sake of mini-OSCE exam.



Traumas 2 types: ① penetrating: gunshot, knife stab... ② Blunt: fallen down, car accident...

Approach to trauma:
A: Airway + Spinal protection
B: Breathing
C: Circulation
D: Disability
E: Exposure & Events

AKA: The primary Survey.
• Adjuncts of primary Survey?
* Can include ECG
1- Vital Signs
2- Chest X-ray
3- pelvic X-ray
* Ultrasound can be used here too.

A: In head traumatic pts: no head-lift chin lift, only jaw thrust, then we put hard neck collar.

B: Assess breathing by: ① Inspection: Seeing symmetry of chest raising? ② palpation: feeling the raising? ③ percussion: nothing resonance on all intercostal spaces? ④ auscultation: good bilateral air entry?

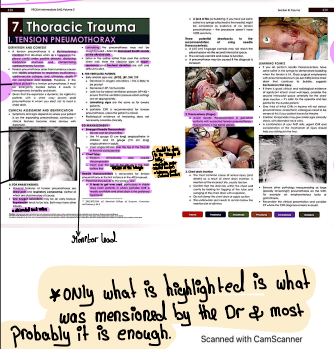
C: Insert 2 large-bore Canulas bilaterally
(give fluids if needed, you can give up to 2L for adults, ensure them to be warm in order not to induce hypothermia) [crystalloids: NL or RL]
- you can do FAST here which is using portable US. (can be used to check for Cardiac tamponade:
↳ diagnosed clinically by Beck's triad:
① muffled heart sounds ② distended neck veins ③ hypotension. but US can also help.

D:
• examine pupils
• Glasgow coma score.
• check the power of the extremities roughly, & any lateralization

E: • ask about events.
• Full Exposure + Log roll to pt. to see the back..
• PR to check for blood per rectum.
• X-rays of chest, pelvis.

diagnosed clinically & X-rays is somehow malpractice as mostly there is no need for this delay.

broncho-vascular markings can't be seen & lung is obviously collapsed on same side. Also diaphragm is pushed downward.



* ONLY what is highlighted is what was mentioned by the Dr & most probably it is enough.

* Secondary Survey: head to toe examination.

- After finishing primary Survey & stabilizing the pt.
- Adjuncts include: CT scans, MRI, barium studies,...

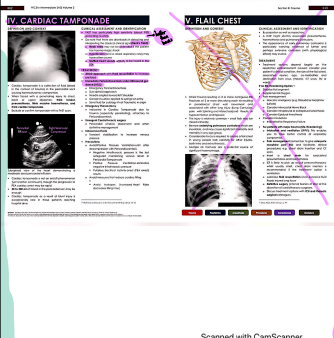
* In any hypotensive traumatic pt. we assume the shock to be hypovolemic until proved otherwise.

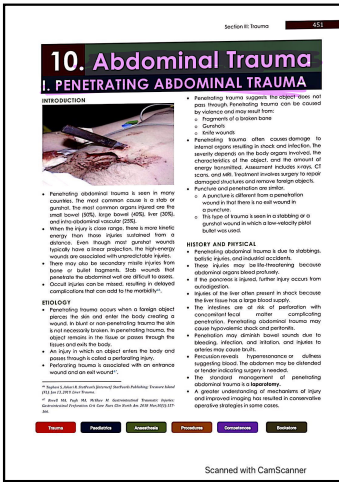
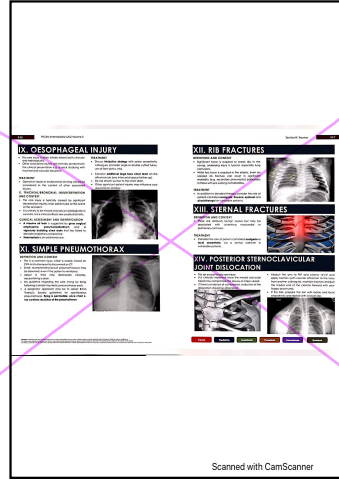
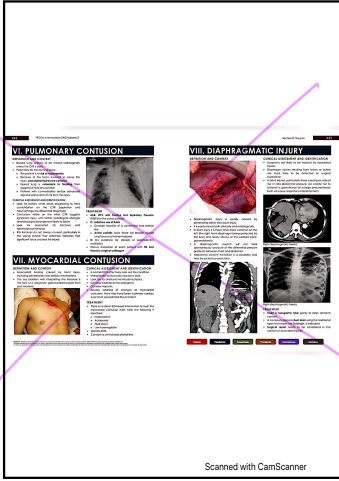
Stages of Hypovolemic Shock:

①	②	③	④
Normal Vital Signs HR <100 lost <750 ml	HR (100-120) normal BP lost 750-1500 ml	HR >120 hypertension lost 1500-2000 ml	HR >140 + hypertension lost >2000 ml
given fluids without the need for blood	given fluids but we do cross match in case pt. doesn't respond solely to fluids	given fluids if no response directly give blood	give blood & activate massive transfusion protocol (1:1:1) of all blood products



No role for needle thoracostomy here at all





از این جا و فلش سبز به لب ۱۱۱ شروع می شود و بالا می آید

ABCDE

نظمی و منظمی

در این جا به این شکل به این شکل به این شکل

به این شکل به این شکل به این شکل

به این شکل به این شکل به این شکل

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