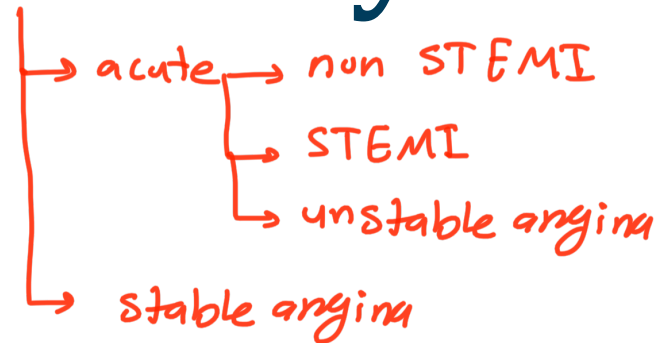


Coronary Artery Disease

the pt presented with chest pain related to exercise.

Jason Ryan, MD, MPH



* chest pain at rest [at night or morning] called:- **premenstrual**... presented in young female..

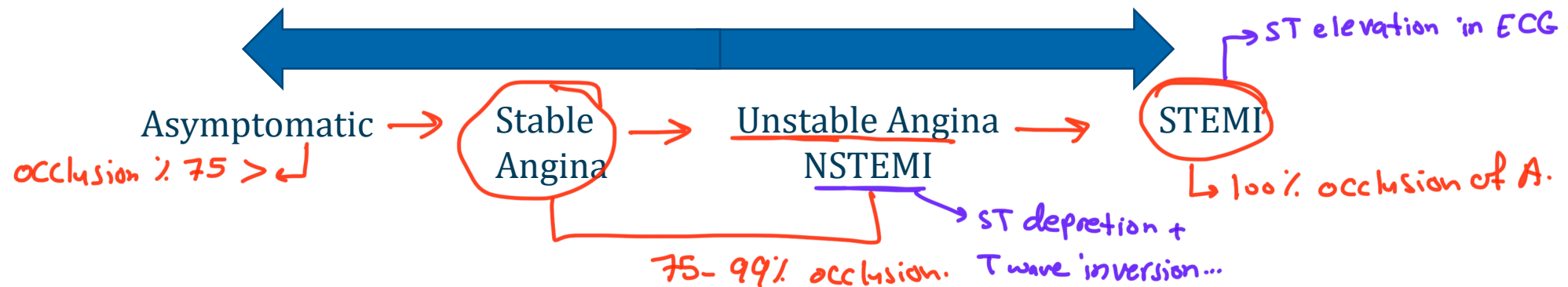
Coronary Artery Disease

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Freestocks.org

- Narrowing of coronary artery
- Caused by atherosclerosis *lead to*
- the pt may presented with:-*
 - ① Asymptomatic until ~75% artery lumen occluded
 - ② Chest pain (angina) *Symptomatic [90% > occlusion > 75%]*
- May also cause dyspnea, other symptoms *exertional increased with exercise...*



Major Risk Factors

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- ✓ • Diabetes
- ✓ • Chronic kidney disease
- ✓ • Hypertension
- ✓ • Hyperlipidemia (LDL) ^{بیچونی}
- ✓ • Age (M > 45, F > 55)
- ✓ • Family History of premature CAD (1° relative, M < 55, F < 65)
- ✓ • Smoking (quitting smoking → significantly ↓ risk)
- ✓ • Obesity, sedentary lifestyle

} CAD Eq. need Aspirin - statins...

CAD Equivalents

- ✓ Diabetes
 - ✓ CKD
- } the mcc of death in these CAD as MI
لہذا یہاں پر CAD - AM کے ساتھ CAD یعنی کا اور ایسے
اجزاء ملے...

رغ - ام - ایسے

smoking → ↑ risk of CAD - atherosclerosis - aneurysm.

Stable Angina

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→ not Acute CAD..
→ diagnosed clinically + confirmed by stress testing...

* Chest pain → Typical 3/3
→ A typical 2/3
→ non classical 1/3

• Plaque occluding $\geq 75\%$ or more of coronary artery

• Causes "typical" chest pain

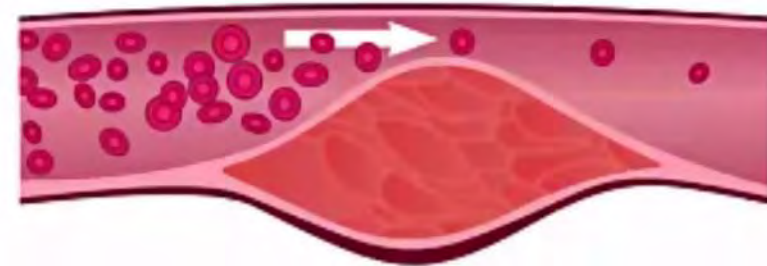
- ① Pressure-like chest pain [heaviness]
• ② Occur with exertion
• ③ Relieved by rest or nitroglycerine

• EKG at rest: normal

• Symptoms at rest: absent

• Diagnosis: symptoms or stress testing

• Stress testing key when diagnosis uncertain



* the pt of stable angina presented with chest pain increased with walking when we do ECG :- normal...
cardiac biomarker [enzymes] :- normal..
... rest ...

→ the perfusion = cardiac demand...

So we do stress testing : قلبی انجینجری مع جہاز red men اور خطیہ ادویہ :-

تیز سنار HR ← یسٹن خ attack of chest pain ← ECG changes ← stable angina

Stress Testing

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we ↑ the exertion → ↑ demand of Blood → ↓ perfusion → attack of chest pain.
تقویت الـ Blood → attack of chest pain.

to do stress testing..

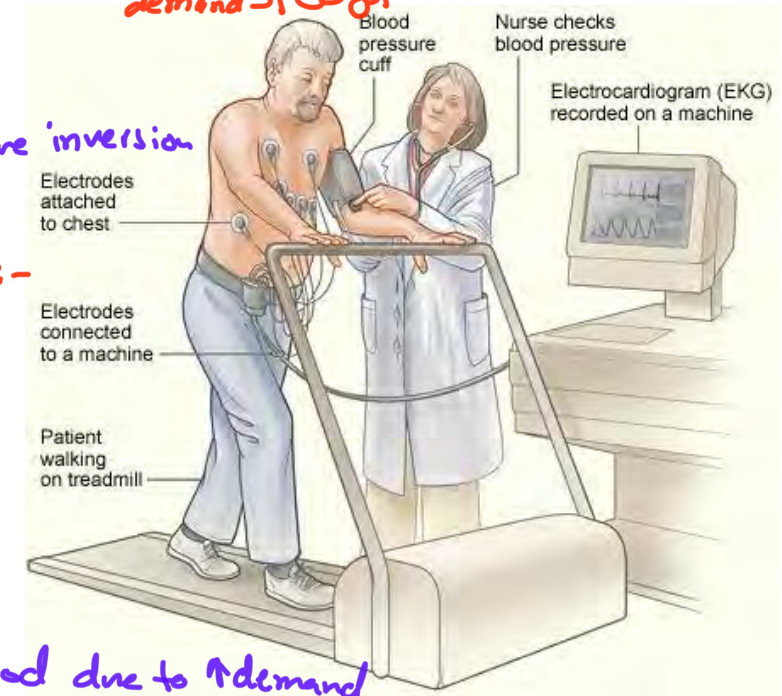
- Patient must be **asymptomatic**
- **Goal:** provoke ischemia and detect ischemia ⊕ to detect:-
- Provocation: exercise always preferred than drugs...
- **Detection:**

- EKG changes (**ST-depressions**) + **T wave inversion**...
- Nuclear imaging (usually Technetium)
- Echocardiography

کدام نعلام و دفعه بپرسه
به ایشان ...

when the exertion happen..

ST depression
T wave inversion

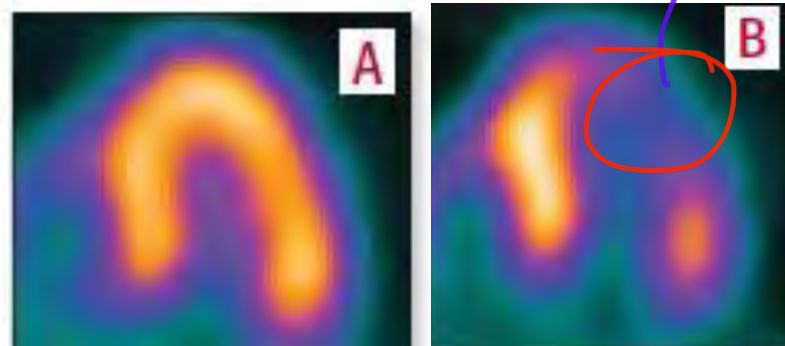


Wikipedia/Public Domain

Contraction بطل نیوا
منبع ...
hypo kinesis in
the hypo perfusion area..
as in inferior wall

حالة قه ما بتنتز یفانی
perfusion..

no blood due to ↑ demand
خالی بطل کافی الـ ریفت



at rest

at exertion..

* Pt with OA or Amputated or neurological dx
ما بقدره ما لجرا فما بقدره نعلام الـ Stress testing فبناطیم اذویج
رفع الـ HR.

Chemical stress testing...

Pharmacologic Stress Testing

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Pharmacologic nuclear (induce coronary steal)

- Regadenoson

- Dipyrimadole ✓

- Adenosine ✓

- Persantine

- Contraindication: reactive airway disease/wheezing

Dobutamine stress echocardiography

- Risk of arrhythmias

↑ HR
lead to hypokinesia in some area appear in echo...

note :- Adenosine, dipyrimadole contraindicated in COPD pt :- [Broncho constriction داء الربو]..

* Adenosine is drug of choice in supra ventricular tachycardia [SVT]...

→ if there is occlusion in LAD for ex. so pt presented with chest pain but pharmacological stress testing will be normal at rest normal

lead to coronary VD → ↑BS in other A. but not in LAD..

[LAD] i.e. LAD is A. is (occluded)
lead to coronary steal...

→ we give them CCB or verapamil

Stress Testing

for stable angina pt but if occlusion $< 75\%$ not appear any changes in stress test this called non-flow limiting...
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- Identifies ischemia due to "flow-limiting" stenosis (usually $> 75\%$)
- Non-flow-limiting lesions not detected if the occlusion $< 75\%$. but we predict there is occlusion
- Acute MI may occur despite negative stress test

دفعاً نظري بالقرعة..

Angiography or Catheterization are the choice if occlusion < 75% www.afratafreeh.com

وہ بہتر کن اگر occlusions



Stable Angina

Management

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- **Preventative therapy** (↓ risk of mortality and myocardial infarction) , ↓ morbidity..

• Aspirin ✓
• Statin ✓ } give them for all stable angina pts.

- Anti-angina therapy (goal: ↓ O₂ demand)
 - Beta-blockers or calcium-channel blockers (↓ heart rate/contractility) → ↓ cardiac demand
 - Nitroglycerine (↓ preload) → ↓ stress in the heart.

- Coronary stent implantation
- Coronary artery bypass grafting (CABG) surgery
- COURAGE Trial: medical therapy has similar outcomes to stent implantation
or PMS + anti Plt therapy...
or DES

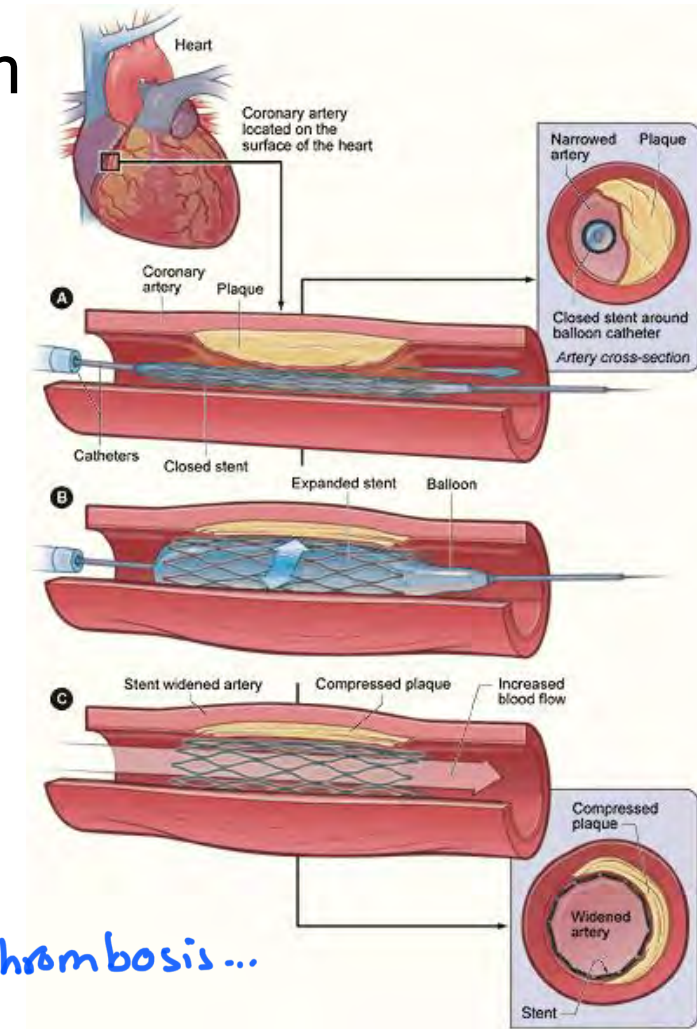
* 2 indication for statins:- ① 1^{ry} prevention.. [to prevent the vascular dz as in pt with ① DM + age > 40
② LDL > 390 ③ risk stratification of ACD → > 7.5 - 6% need statin...

② 2^{ry} prevention for any pt with A. occlusion as:- CVA - CAD - stable angina - peripheral artery dz - aortic aneurysm...

Coronary Stents

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- Percutaneous Coronary Intervention (PCI)
- About 600,000 stents/year implanted US



*Stent 2 types

→ **Peri metal stent [PMS] ...**

← خلكا الحالىين المريضه زي افيد
Aspirin + Statin...

→ **drug eluting stent [DES] ...**

→ with material prevent the occurring of thrombosis...

علیہ القابہ
الحق تعالیٰ

CABG

we do it if the LAD occluded 100% or if there is 3 vessel occlusion.

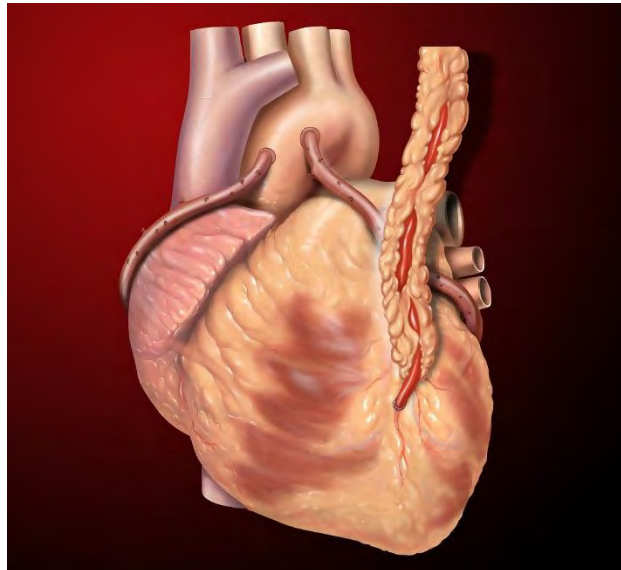
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Coronary Artery Bypass Grafting

* بنوع نجیب Stent A > و بنوع نزرعه ما بعد A ... occlusion

- "Bypass Surgery"
- Left Internal Mammary Artery (LIMA) Graft
- Saphenous (leg) Vein Grafts
- Radial (arm) Artery Grafts

↓
may be from 1st int. mammary A.. ^① ← first choice
or saphenous v. ^② or radial A.. ^③
[leg vein] 2nd choice ↳ 3rd choice [rare]



Patrick J. Lynch/Wikipedia

Stable Angina: Typical Case

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* 75-99% occlusion maybe
Stable angina ↙ ↘ non stable angina
non STEMI

* 100% occlusion → STEMI...

- 65-year-old man with ^{heaviness} chest pain while walking

- Relieved with rest

- Presents to ED:

- EKG normal

- Biomarkers normal

cardiac enzyme

- Stress test

- Walks on treadmill → chest pain, EKG changes

ST depression

T wave inversion

- Cardiac catheterization performed

- 90% LAD artery blockage

- Stent placed → angina resolved

need

Stent Complications

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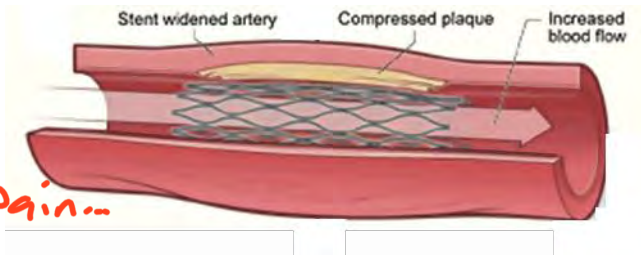
① Restenosis

to prevent it the Pt should take Aspirin-clonidrogrel for 1 year.

بعد ١٠ سنة دواء ريجل ماتي مع اسبرين

- Slow, steady growth of scar tissue over stent
- "Neo-intimal hyperplasia"
- Re-occlusion of vessel so presented with new MI [chest pain]
- Rarely life-threatening
- Slow, steady return of angina slow steady ↑ of pain...
- Most stents coated "drug-eluting stents"
 - Metal stent covered with polymer to prevent formation of scar tissue...
 - Polymer impregnated with drug to prevent tissue growth
 - Sirolimus → prevent the neo intimal hyperplasia...

DES



* Tacrolimus for cancer...

Stent Complications

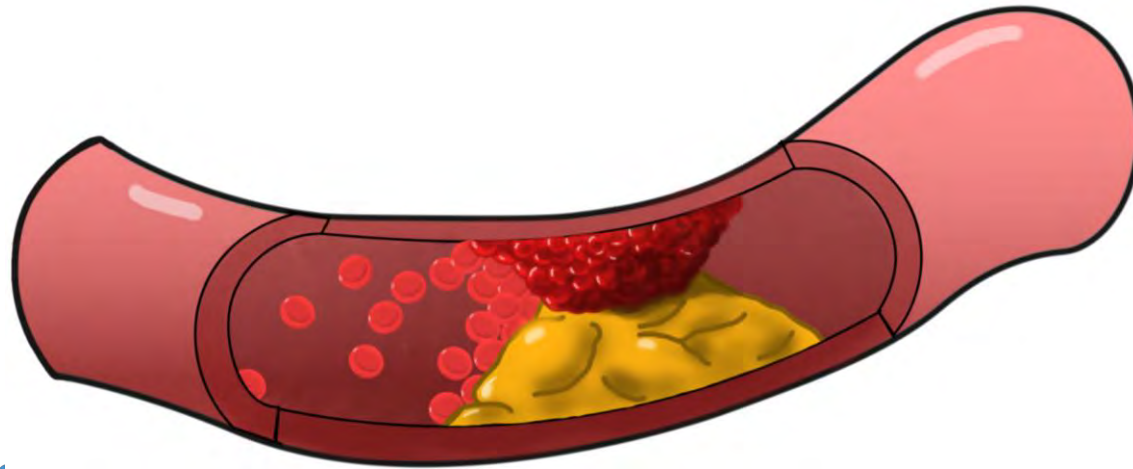
www.afratafreeh.com

② **Thrombosis** sudden onset closure of stent..

- Acute closure of stent
- Same as **STEMI**: life-threatening event
- Dual anti-platelet therapy for prevention
- Associated with **missed medication doses**

aspirin
clopidogrel

لما حاطاه ادوية ايس اس بى ايس اس بى ..



Stent Thrombosis Prevention

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for any pt with stent in 1st year...

- "Dual antiplatelet therapy"

- Typically one year of:

- Aspirin ✓

side effect: - !!

- Clopidogrel, prasugrel or ticagrelor (P2Y12 receptor antagonists) ✓

- After one year, stent metal no longer exposed to blood fibrotic changes ← stent

- Scar tissue and endothelial growth

- Risk of thrombosis is lower (but not zero)

- Most patients take aspirin only life long...

So we give the pt single therapy..

with statin → single therapy..
[2nd prevention]

* any pt presented with MI [angina] ask him for previous similar attack...

Variant (Prinzmetal) Angina

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→ Vasoocclusion...

↳ more in young female at night or early morning...

- Episodic vasoconstriction of coronary vessels

- Episodes usually at rest

- Midnight to early morning

→ complaint of heaviness chest pain at top rest...

- Sometimes symptoms improve with exertion

- Associated with smoking make VC

↳ as walking.

So should
stop smoking

- Diagnosis: usually based on history

* Ht :- CCB...

Variant (Prinzmetal) Angina

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Treatment

↳ there is ST elevation without vessel occlusion..

* CCB → dihydropyridine.
↳ nondihydropyridine.
↳ ↑ HR → Ischemia
as:- verapamil

- Quit smoking
- **Calcium channel blockers, nitrates**
 - Dihydropyridine CCB: amlodipine
 - Vasodilators
 - Dilate coronary arteries, oppose spasm
- Avoid propranolol as:- pheochromocytoma.
 - Nonselective beta blocker
 - Can cause unopposed alpha stimulation lead to VC
 - Symptoms may worsen

occur in the night or early morning...

worsen the symptoms

* Berger dz:- peripheral A. occlusion due to smoking
lead to tip necrosis → gangrene..



Acute Coronary Syndromes

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non-STEMI

non stable angina

STEMI

sudden cardiac death..

37

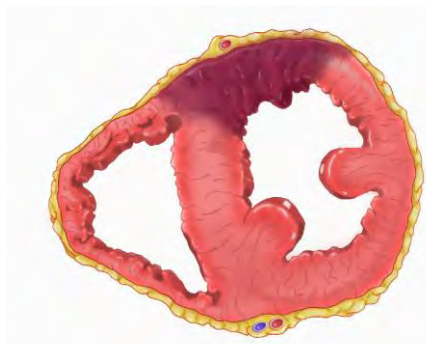
- Plaque rupture → thrombus formation
 - Subtotal occlusion Just in endocardium.. [subendocardial Ischemia]
 - Unstable angina ✓
 - Non-ST elevation myocardial infarction non STEMI ✓
 - Total occlusion (100%)
 - ST-elevation myocardial infarction (STEMI) ✓



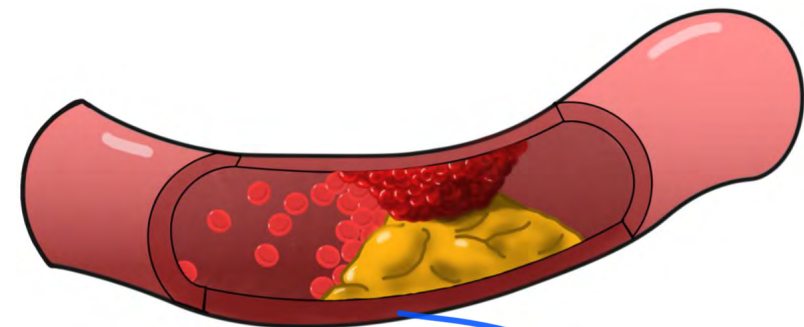
Subtotal Occlusion



Subendocardial Ischemia



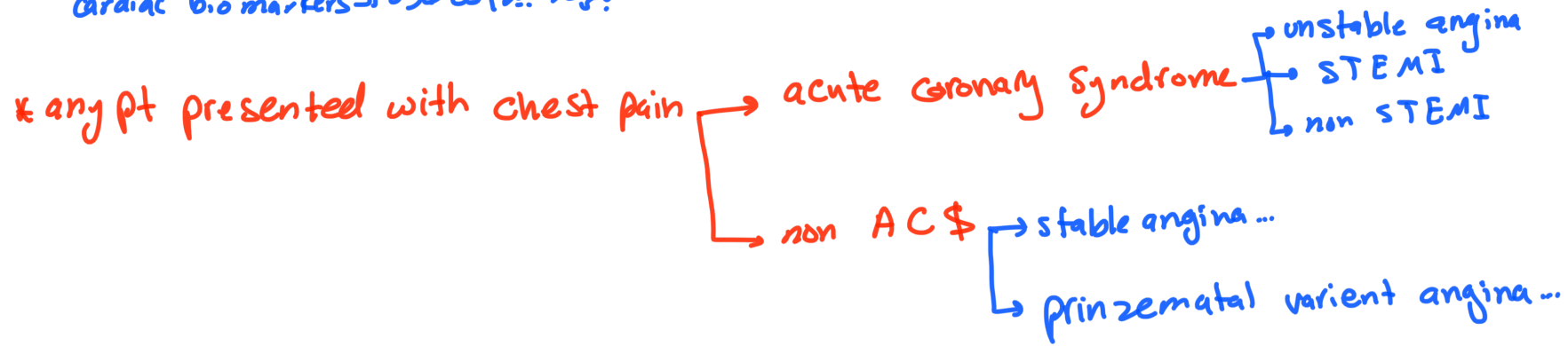
Transmural Ischemia



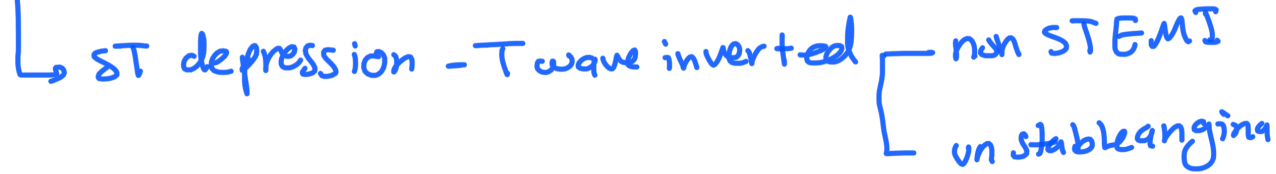
Total Occlusion

* Both unstable angina + non STEMI lead to ST depression + T wave inversion..

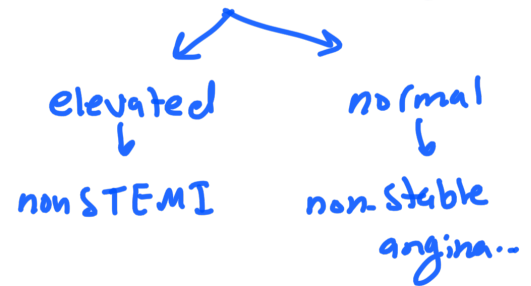
بنفسه بغيره مع قلال الـ cardiac bio markers



* ACS → ST elevation ACS → STEMI



so we do cardiac bioenzymes..



* non stable angina

- chest pain at rest
- denovo chest pain [sudden onset not gradual]
- after MI

Unstable Angina and NSTEMI

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- **Unstable angina**

at rest
post MI
crescendo
denovo [new onset angina less than month]...

- Ischemic symptoms occurring with increasing frequency or at rest

- Negative biomarkers [normal]...

- **NSTEMI**

- Positive biomarkers [elevated]...

- Ischemic EKG changes (other than ST-elevation) may occur in both

ST depression

T wave inversion...

any ST depression significant...

T-P wave ↓ بنقار
Segment

→ more than 1mm in peripheral leads

→ " " 2mm in chest leads

→ presented in 2 _____ leads

[lead 2 or 3 or 4 or 5 or 6 or 7]

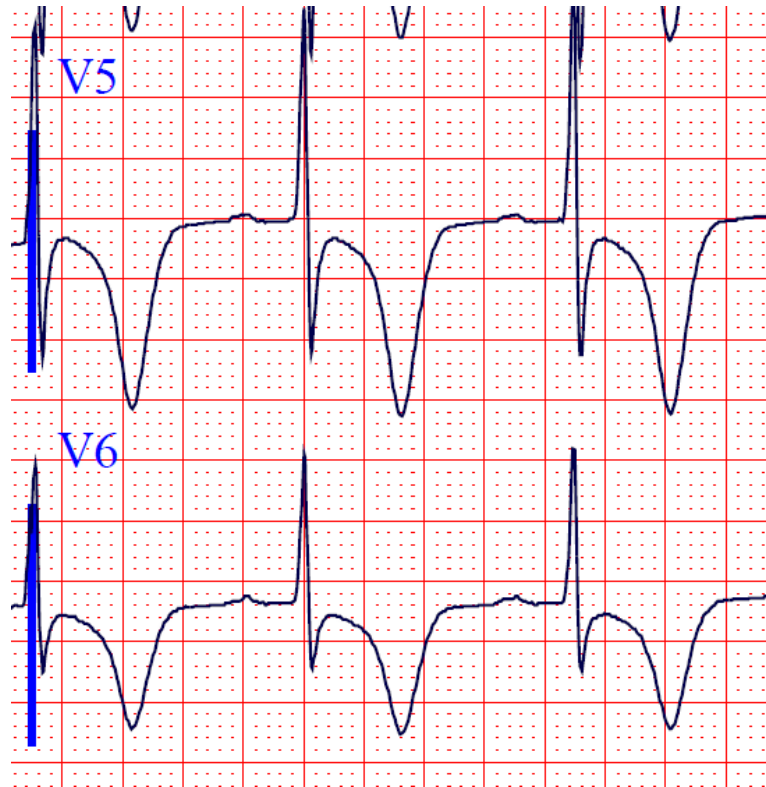
significant

UA/NSTEMI

ECG Changes

- ✓ • ST depressions
- ✓ • T-wave inversions

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* ALT > AST → liver dz ١٥٪ except Alcoholic liver dz + cirrhosis ⇒ AST > ALT [2:1]...

UA/NSTEMI

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Cardiac Biomarkers 1- myoglobin 2- LDH 3- troponin 4- CK-MB 5- AST

- Biomarkers spill into blood with cardiac injury
- Most common marker used: **Troponin I or T**

- Increase 2-4 hours after MI
- Stay elevated for weeks

• CK-MB also used

- Increase 4-6 hours after MI
- Normalize within 2-3 days
- Very good for re-infarction

④ LDH → not specific [بعضها يرفع]

⑤ AST → not specific
→ elevated in MI

→ AST/ALT → 2/1 in alcoholic liver dz or liver cirrhosis...
→ presented in mitochondria [damaged by alcohol]

* pt presented by chest pain 2 day ago...
1 troponin, normal CK-MB

But if it elevated it's ↓
mean reinfarction...

① myoglobin appear after 1 hour [not specific or sensitive] ألبومين - most sensitive

② troponin → I: mc marker use appear after 2-4 hours [most sensitive test] we ask about serial troponin test every 2 h...
→ T
... 1-2 week يرفع فيه so we use it just for diagnosis...

→ there is high ——— [x1000 time than troponin]

③ CK-MB appear after 1-2 h. and disappear after 2 day.



Boards&Beyond®
STUDY SMARTER

if it remain elevated after 2 days → indication for reinfarction...

* CK → MB: in heart قلب
→ MM: in brain
→ total

Treatment of UA/NSTEMI

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* any ACS need admission if STEMI need angioplasty
but if non-STEMI or stable angina need
Aspirin + Clopidogrel + β Blocker + heparin
└──────────┘
dual antiplt
therapy...

- Thrombotic and ischemic syndrome (like STEMI)
- No "ticking clock" (unlike STEMI)
 - Subtotal occlusion ✓
 - Some blood flow to distal myocardium ✓
 - No emergency angioplasty ✓
 - No benefit to thrombolysis ✓
- Aspirin
- Beta-blocker
- Heparin
- Angioplasty (non-emergent)

Typical UA/NSTEMI Course

unstable angina

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- Presents to ER with chest pain
 - Biomarkers elevated
 - Absence of ST-segment elevations
 - Medical Therapy
- there is ischemia
- ST depression - T-wave inversion or normal ECG...
- Obtain EKG in all chest pain patients
↳ maybe normal
- Give aspirin to chest pain patients with possible NSTEMI
- Aspirin
 - Metoprolol B blocker.
 - Heparin drip or clexan.
- clopidogrel +
- Admitted to cardiac floor
 - Hospital day 2 → angiography
 - 90% blockage of LAD → Stent
- not emergent.
- clexan preferred more than heparin... ولكن باين العيب جى
- * heparin preferred than clexan in :-
1. CKD [the clexan need close adjustment 'in renal failure']... LMWH
 2. passive pulmonary embolism [heparin here preferred more than LMWH]...

UA/NSTEMI Treatment

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Long-term therapy

- Goal ↓ mortality and recurrent infarction
 - Aspirin ✓
 - Statin ✓ + Plavix [clopidogrel] ← stent دكتور
- Beta-blocker for prevention of recurrent disease
 - Strong indication in STEMI
 - Weak indication NSTEMI/UA } → B-blocker
- Used for prevention in NSTEMI only

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STEMI

Jason Ryan, MD, MPH



STEMI

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ST-Elevation Myocardial Infarction

there is 100% occlusion in the A this called ^{صغرى الشريان} calbert A...

- Angina at rest with ST-segment elevation ^{MI}
- Biomarkers elevated after 4-6 hours

* Ischemic pain as MI make heaviness chest pain need morphine ...



* note:- T- P segment is Isoelectric line in the ECG ...

نقطة قبل ال J point

← ال T segment ال S segment ال J point

} see if there is elevation or depression

ST elevation can if $> 1m$ in kit lead
 $> 2m$ in chest "

* ST segment changes

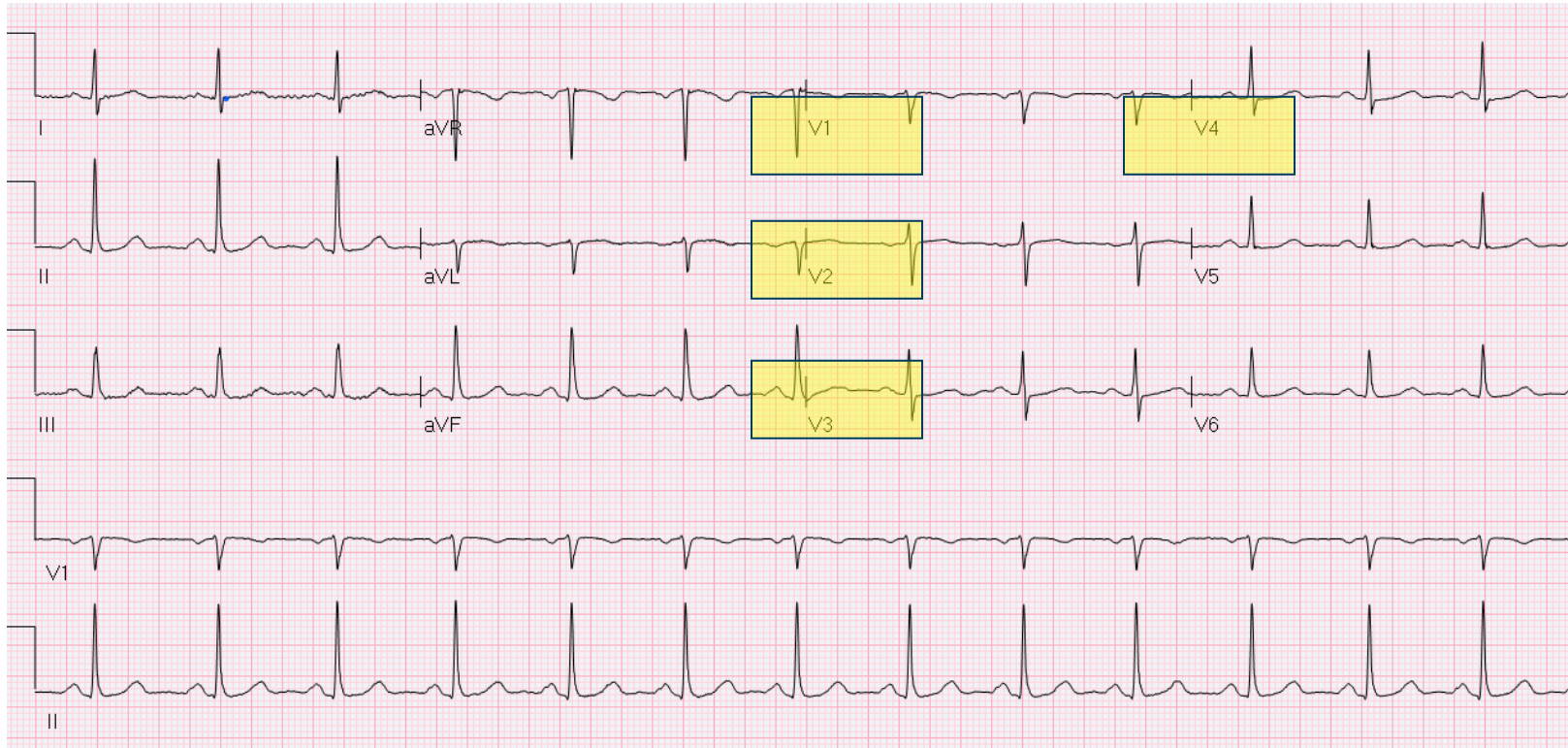
Leads go together

ST Elevations - Anterior

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* The ant. leads V1 - V2 - V3 - V4...
التي تقترن مع بعضها في الحائط الأمامي

Ant wall MI lead to cardiogenic shock and other bad complication...



* The lat. leads :-

high low..

aVL

V1

V5

V6

- When there is ST elevation in one site → there is ST-depression in other site... this called:- **disprocal**

changes...

- The ant. or lat. leads

has disprocal changes in inf. walls.
... في الجدران البعيدة

Leads go together

ST Elevations - Anterior

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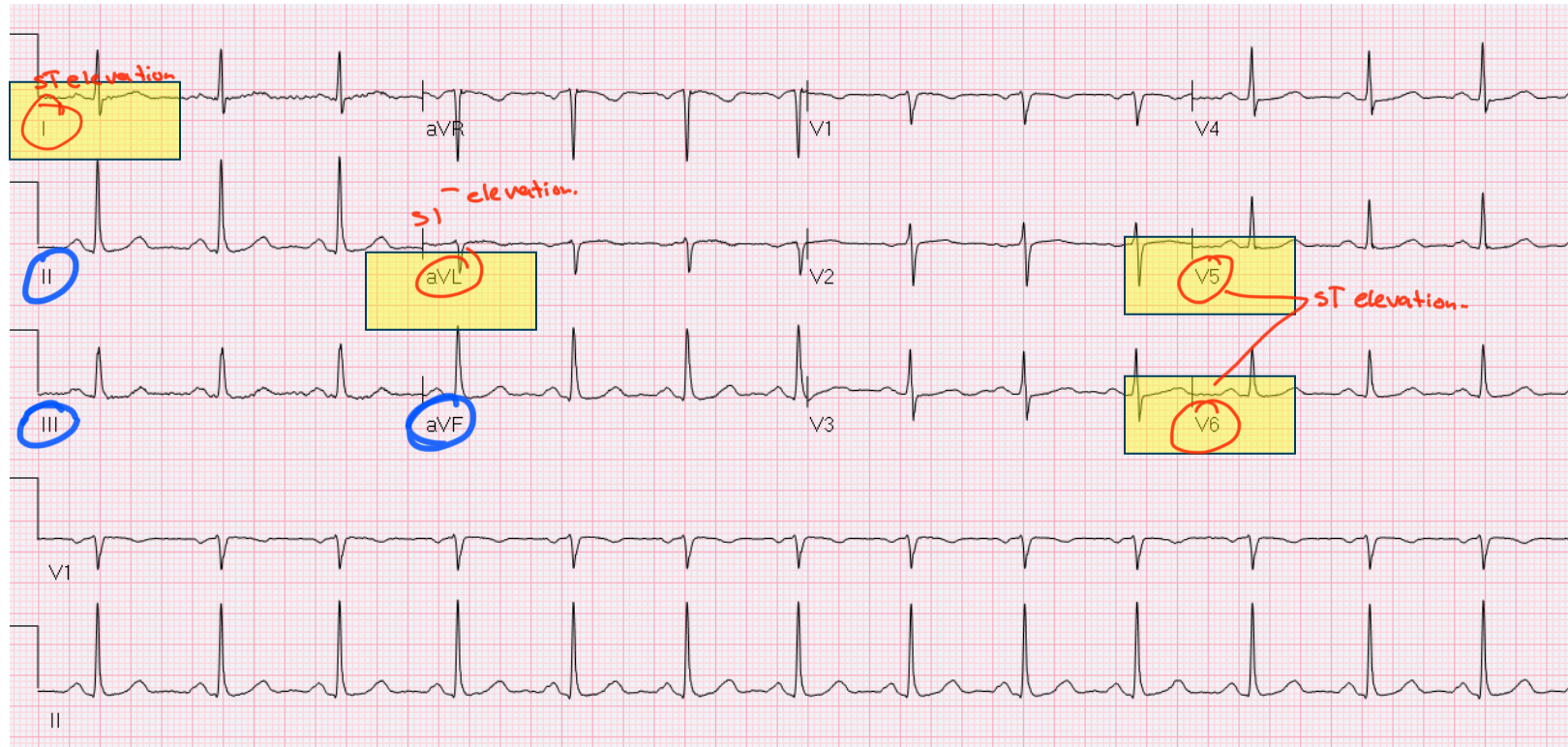


ST depression +
T wave inversion
in $\approx$ lead II, III
aVF...
which are reciprocal
changes of ant. MI

Leads go together

ST Elevations - Lateral

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[So this high lat. MI] + If circumflex occlusion..

↳ ⊕ there is mirrored inf lead [disproportional changes]..

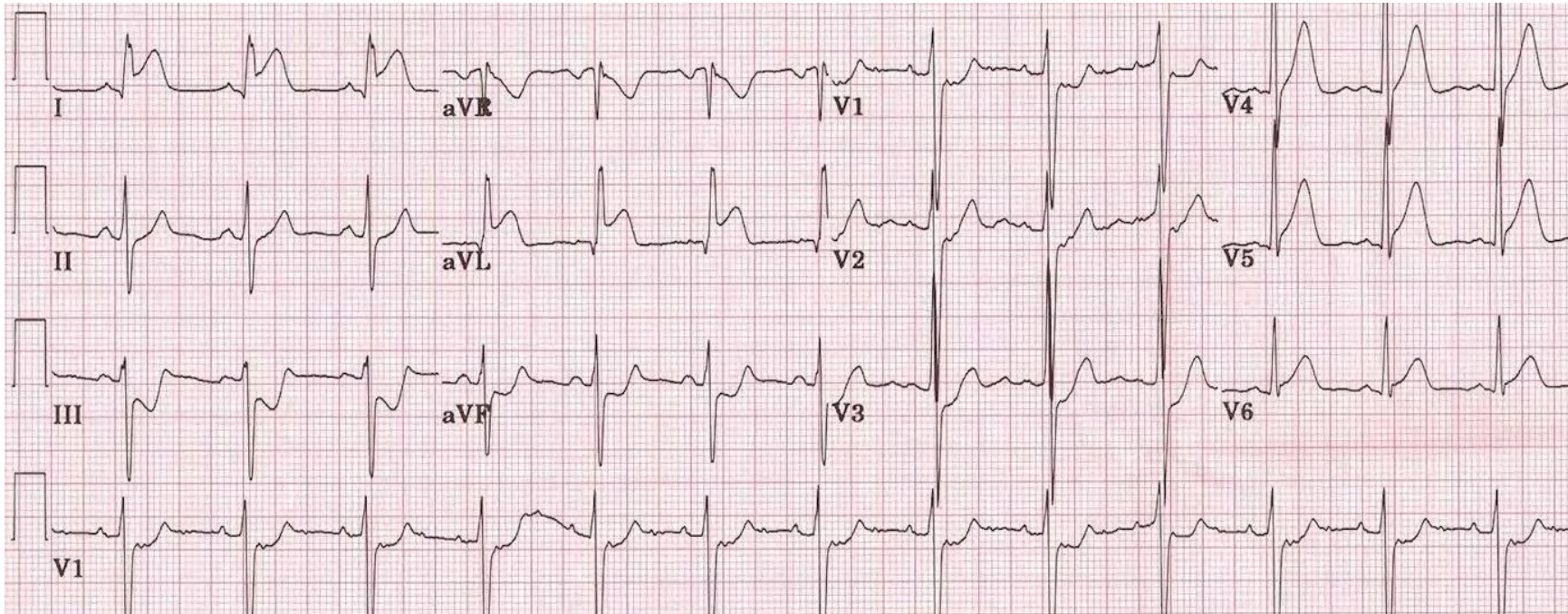
↳ lead II, III - aVF [ST depression - T wave inversion]..

Leads go together

ST Elevations - Lateral

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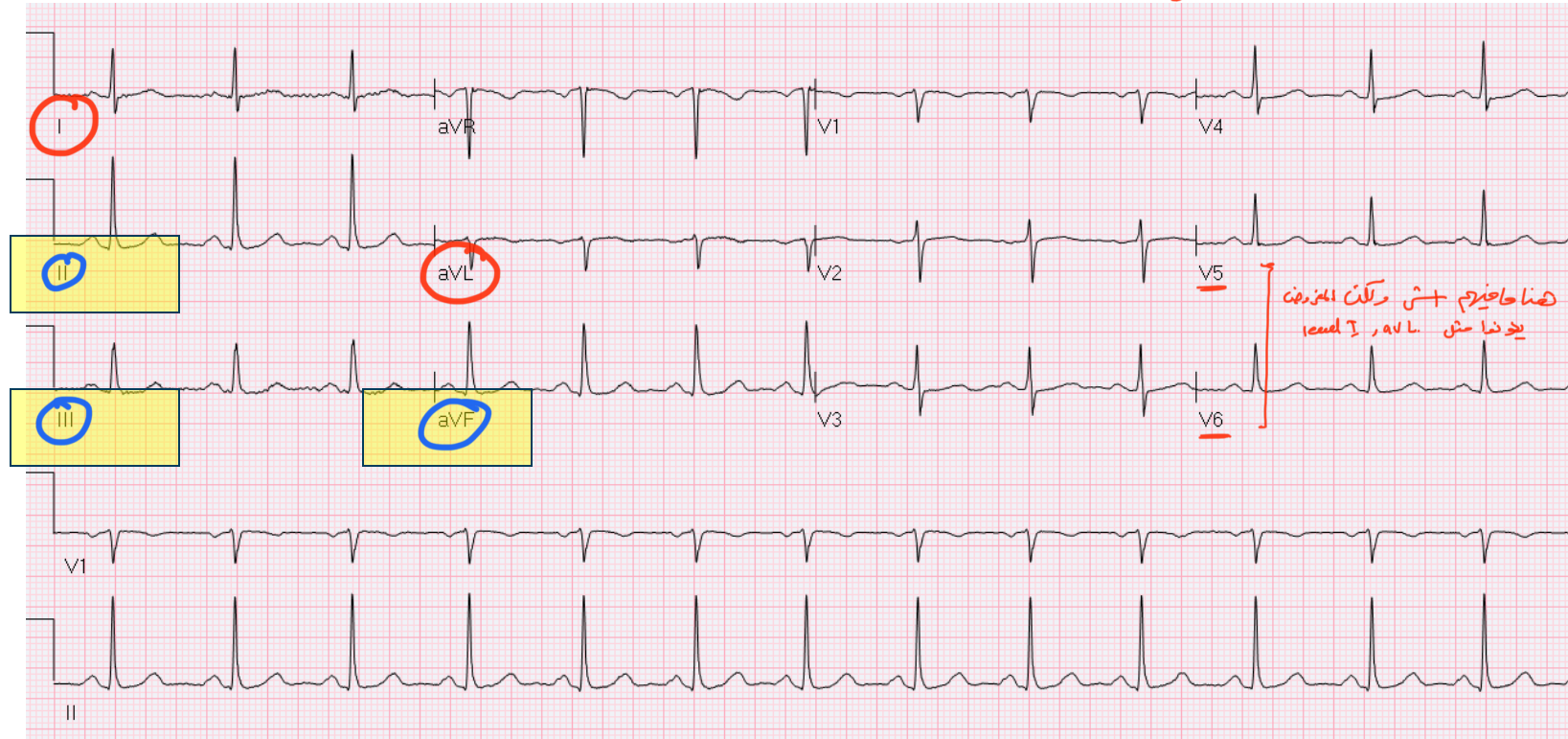
lead I
aVL
V6
V5
high lat.



Leads go together

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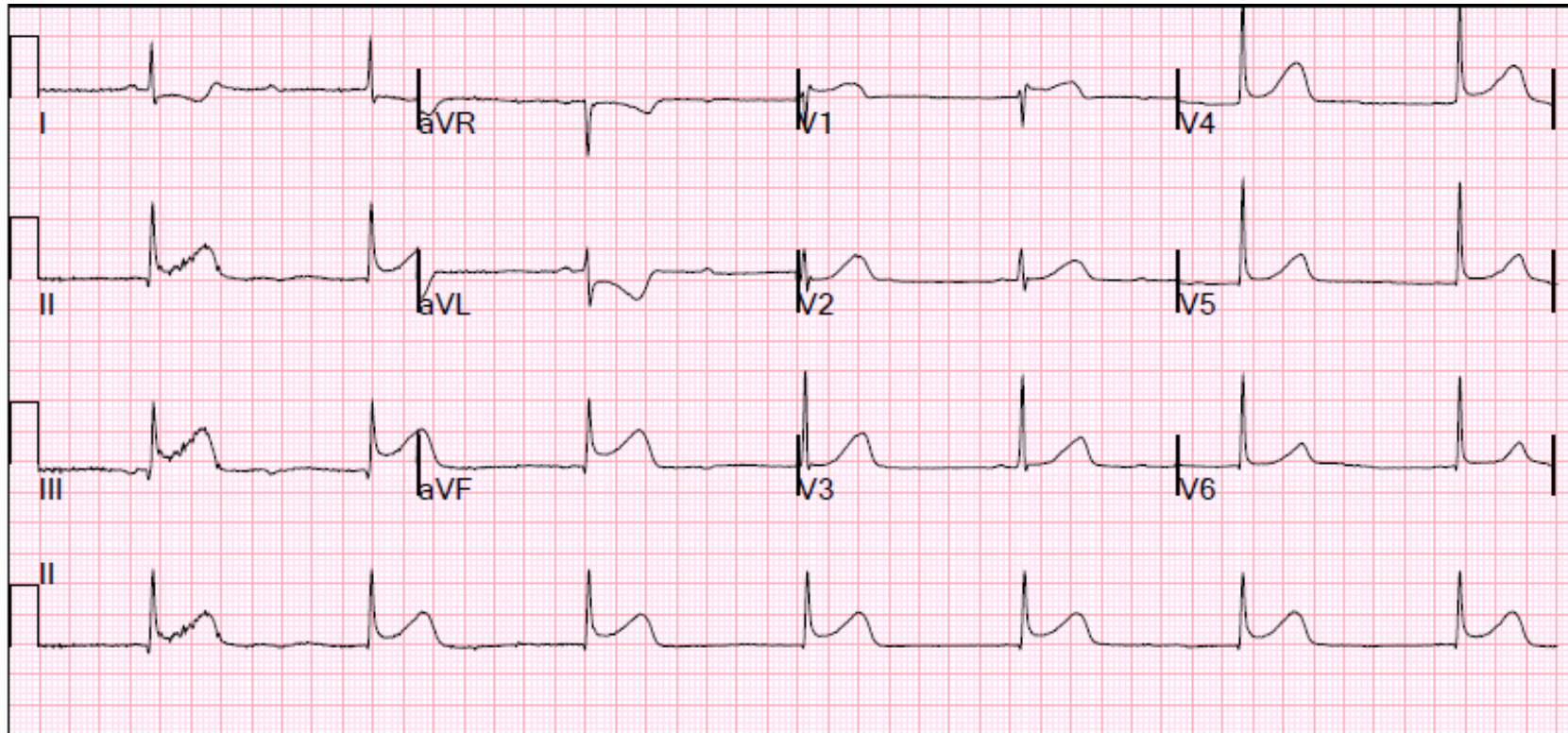
ST Elevations - Inferior in lead II, III, aVF + reciprocal changes in lead I, aVL



Leads go together

ST Elevations - Inferior

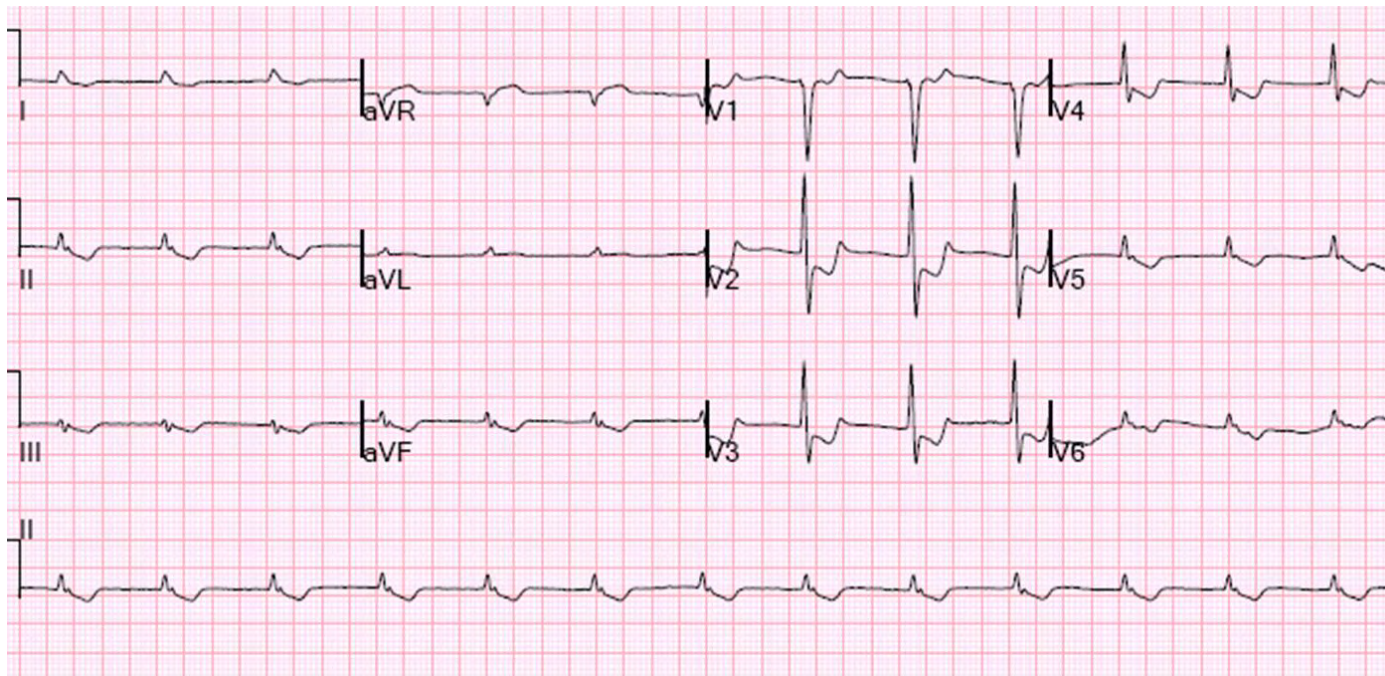
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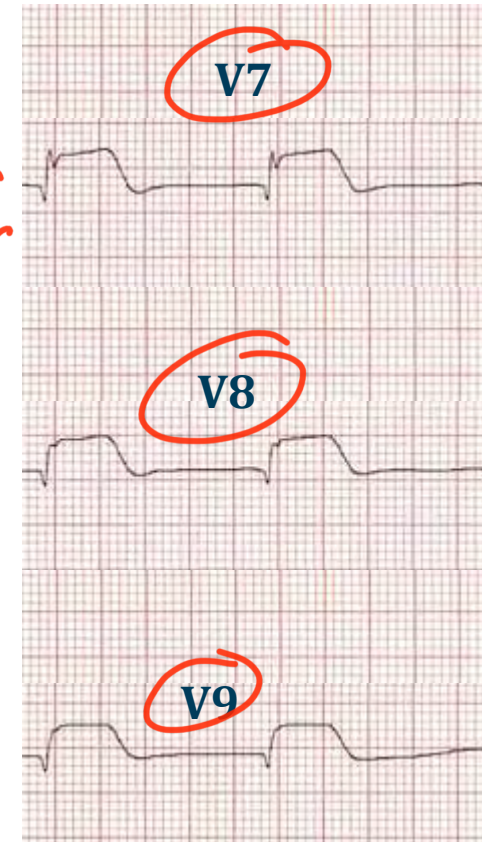
Posterior STEMI

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- Anterior ST depressions with standard leads
 - ST-elevation in posterior leads (V7-V9)
- diffuse + T wave inversion*
leads I, II, III, aVL, aVF
we put them in scapula to see ST elevation + V8



*in V7
V8
V9*



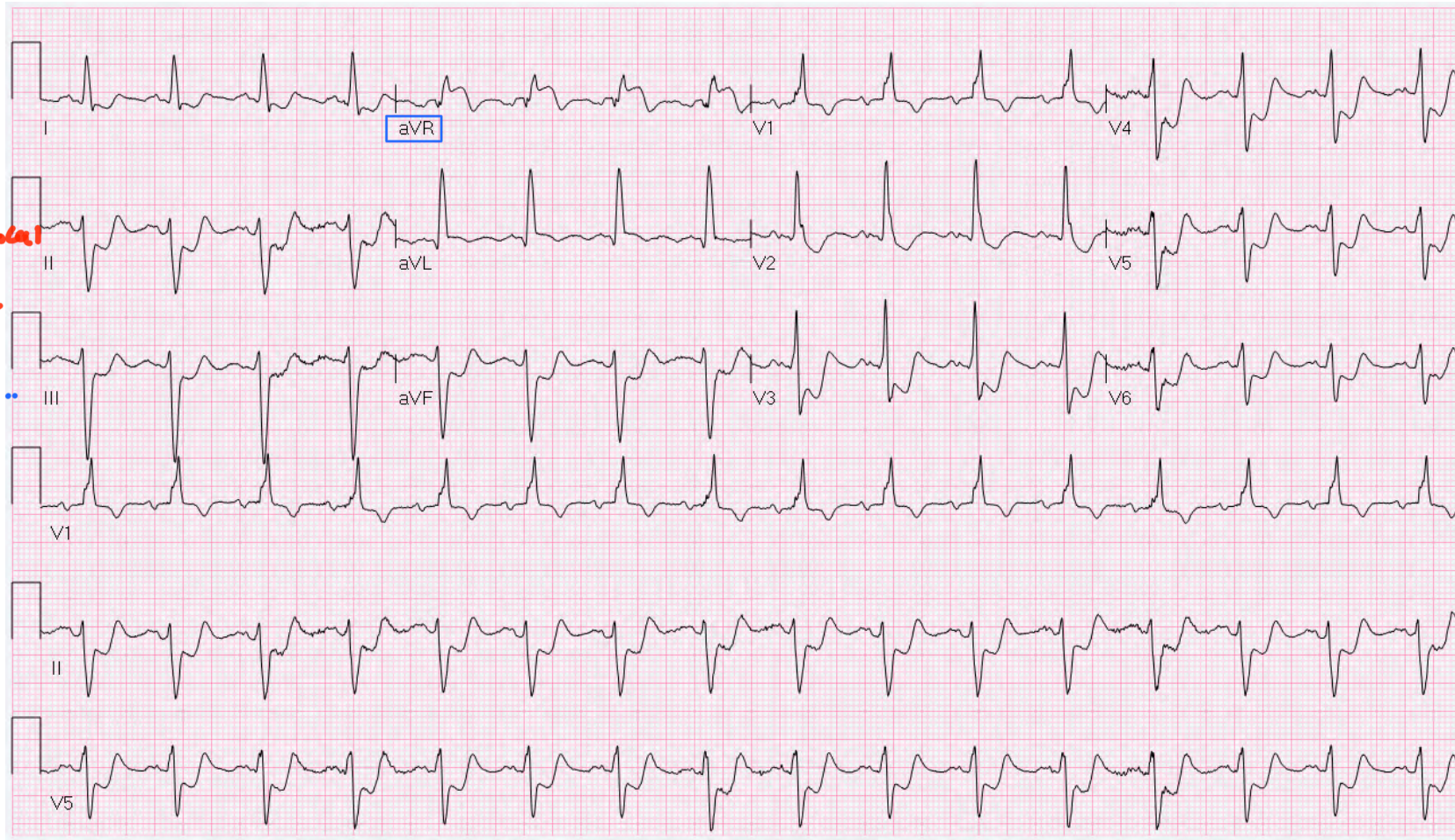
note:- T wave inversion may be normal in lead III, aVR.
but if it presented in aVL this indication for Ischemia...

Left Main Occlusion

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indication for LAD occlusion ←

* لا ملاحظة خاصة في aVR
if there is ST elev.
init alone → significant
فإنه في ارتفاع القوسية
يكون مع القوس



في البنية كده قهر ضخم كده
* missed leads in
aVR: ST elevation..
with diffuse dispersion
changes in all leads..
mid stenosis in LAD..

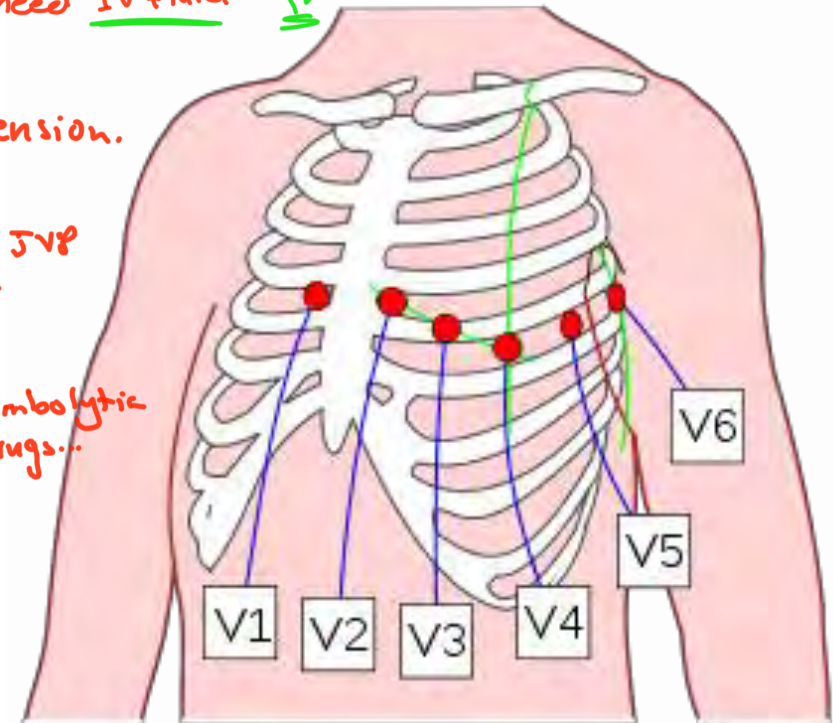
Special Complications static...

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Inferior MI R+ coronary A. occlusion which supply the SA - AV node this lead to Heart block - brady cardia - ↓BP.
 ↳ the epigastric pain come in this type of MI... [+ Hypotension]..

① **Right ventricular infarction** no preload → hypo tension so need IV fluid...

- Occurs in inferior STEMI (II, III, aVF)
- Loss of right ventricular contractility no preload → hypo tension.
- **Elevated jugular venous pressure**
- Decreased preload to left ventricle → **hypotension** + ↑ JVP
- Diagnosis: right-sided chest leads more than 8-9...
- Avoid nitroglycerine ↳ lead to ↓ pre load + hypotension... ↳ v4-5-6 → Rt Prime...
- Treatment: IV fluids (↑ preload) + treat MI → PCI or thrombolytic drugs...



* epigastric pain + hypotension → inf. MI until prove otherwise...

...inferior MI , low nitroglycerin - Aspirin ... MI ...

* normal JVP = 6-8 mmHg

Special Complications

Inferior MI

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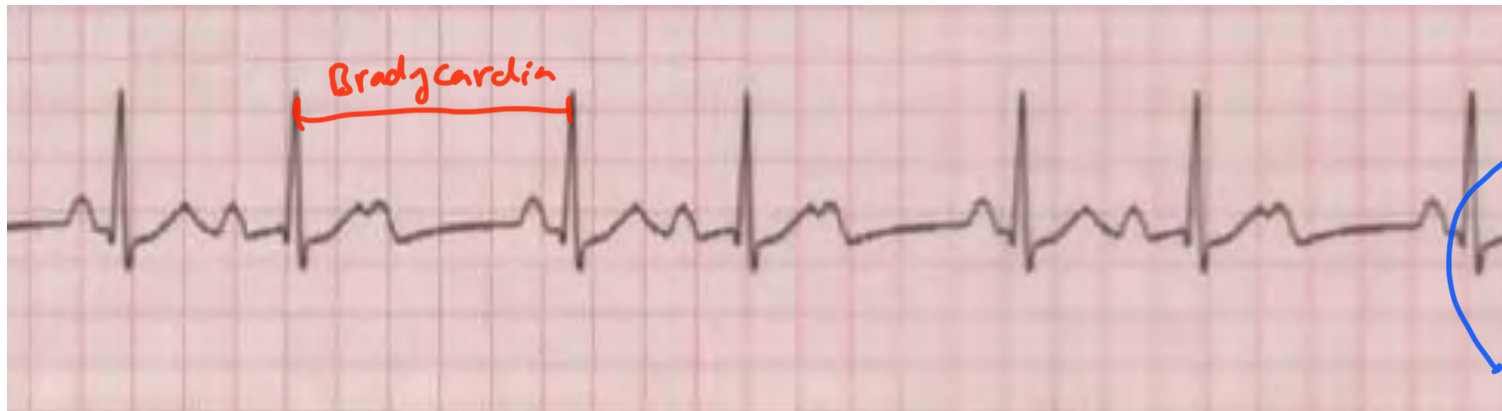
* Brady cardia + irregular HR → 2nd, 3rd degree heart block..

- 1st degree heart block:- regular heart rate
↳ At depend on symptom.

- 2nd degree heart block type
irregular HR.
I: fixed prolongation
QT interval
[benche pach]
II: no fixed
prolongation
... QT, فجأة يتوقف.

• Sinus bradycardia and heart block

- Inferior wall ischemia and hypotension
- RCA: SA node 60%, AV node 90%



stage III :- Complete H.b
..dA Atrium contraction
..dV ventricular " J
no association between P.wave
and QRS complex...

! Jegandec A wave ← 3rd degree أو TC valve contrac. مع Atrium contraction أو TC stenosis...
or due to complete AV block...

Special Complications

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Anterior MI

- **Cardiogenic shock** mc in Ant. MI [closed LAD] [any MI lead to cardiogenic shock but Ant. MI mcc of cardiogenic shock]
 - Usually occurs with large anterior STEMIs 2nd mcc of death in MI pt
 - Hypotension
 - Tachycardia
 - Pulmonary edema
 - Respiratory distress
- no contraction

* mcc of death in MI pt:-

- ① V. tag - V. F...
- ② Cardiogenic shock...

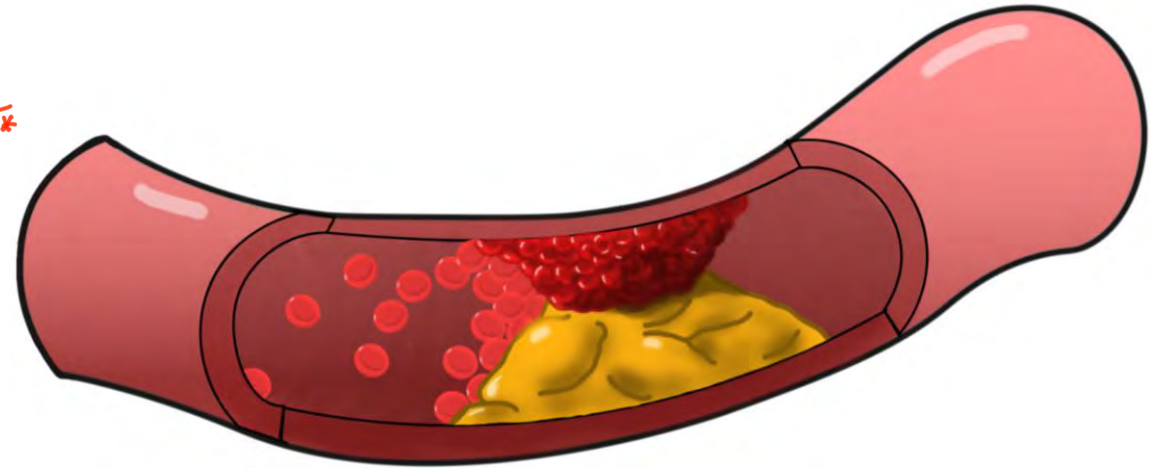
Treatment of STEMI

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PCI...
thrombolytic therapy...

- "Time is muscle" كل ما يكون لكثافة بروتين أقل يعني تلف عضلة أكبر muscle
- Coronary artery occluded by thrombus
- Longer ^{complete} occlusion → more muscle dies
 - More likely the patient may die if untreated
 - More heart failure symptoms
 - More future hospitalization for heart disease
- **Medical emergency**

↑ risk of HF and mortality ← thrombolytic or PCI قبل ما يغلق



Treatment of STEMI

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* if the pt presented to hospital with chest pain and we give him thrombolytic drug...
→ this mean the PCI not emergency...
thrombolytic → rescue PCI...

- Main objective is to open the artery
 - "Revascularization"
- Option 1: Emergency angioplasty PCI [Better]

- Mechanical opening of artery

- Stent placemnt

- Should be done < 90min if the PCI available in the hospital but if it not available we have 120 min. to make it in another hospital...

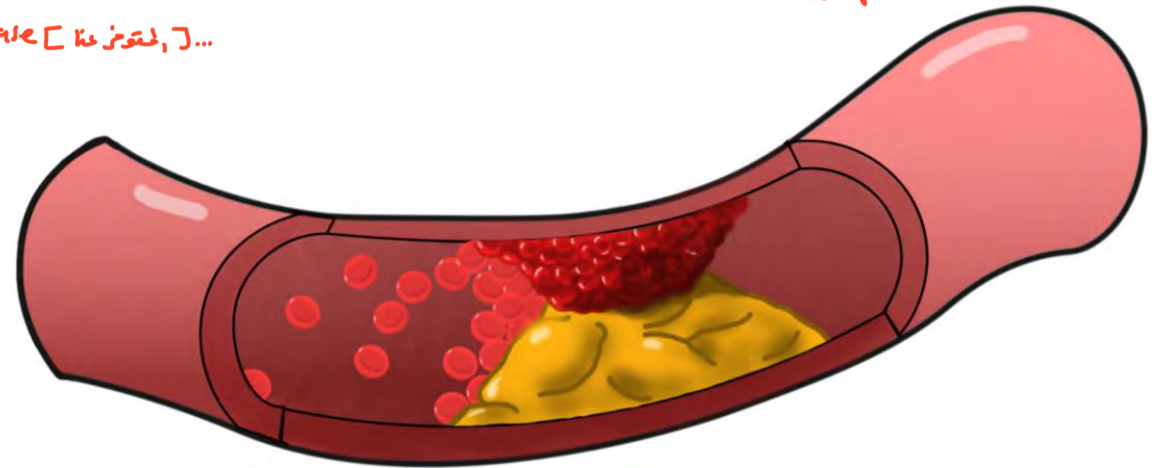
- Option 2: Thrombolysis as:- streptokinase or alteplase [تفريغ...]

- Alteplase, TPA

- Should be done < 30min ← optimal time الفعل والحد الأقصى 12 ساعة

- "Door to balloon" or "door to needle"

بعد 12 ساعة فمضغ انه مضغ PCI و thrombolytic...
مضغ مضغ Chest pain في البيت...



Treatment of STEMI

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- Time matters
 - Medical therapy is supportive
 - Given while working to open artery
- This is a **thrombotic problem** ✓
 - Aspirin to inhibit platelet aggregation
 - Heparin to inhibit clot formation
- This is also an **ischemic problem** ✓
 - Beta-blockers to reduce O2 demand
 - Nitrates to reduce O2 demand

Aspirin ← *is the 1st thrombolytic agent in PCI unless pt is allergic*
clopidogrel ←
statins ←
heparin ←
B. blockers ←

Cautions

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- **Beta-blockers** contraindicated in:-
 - **Inferior MI**
 - **Bradycardia and AV block** can develop or hypotension..
- **Nitrates**
 - Occlusion of RCA can cause **RV infarct** due to hypotension so we give them fluids to ↑ preload
 - RV infarction → ↓ preload
 - Nitrates ↓ preload → **hypotension**

Other STEMI Treatments

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- **Clopidogrel** ✓
 - ADP receptor blocker
 - Inhibits platelets
- **Eptifibatide** ✓
 - IIB/IIIA receptor blocker
 - Inhibits platelets aggregation..
- **Bivalirudin** ✓ as:- dabigatran - abacixiban
 - Direct thrombin inhibitor
 - Inhibits clot formation

→ the antidote is idarucizumab...
غير متوافر هنا //

→ HIT syndrome...
نتقال بعد الـ heparin في حين الـ HIT syndrome...

Typical STEMI Course

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- Arrival in ER with chest pain 5:42 pm
- EKG done 5:50 pm
 - STEMI identified
- Cardiac cath lab activated for emergent angioplasty
- Meds given in ER
 - Aspirin
 - Metoprolol
 - Nitro drip
 - Heparin bolus
- Transport to cath lab 6:15 pm
- Artery opened with balloon 6:42 pm
 - DTB time 60 minutes (ideal < 90min)

typical presentation.

Typical STEMI Course

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- Arrival in ER with chest pain 5:42 pm
 - EKG done 5:54 pm
 - STEMI identified ✓
 - Meds given in ER
 - Aspirin
 - Metoprolol
 - Nitro drip
 - Heparin bolus
 - tPA given based on weight 6:07 pm
 - IV push ✓
 - Door to needle time 25 min (ideal < 30)
- Thrombolytic...

Complications of Ischemia

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First 4 days

- Arrhythmia $\leftarrow$ v. tag. v. F } Shockable rhythm

* MI pt with AF $\rightarrow$ will die [fatal]...
 $\downarrow$
 msc of MI

$\hookrightarrow$ if the AF due to MI...!!

5 - 10 days

- damage in part of the muscle...
- Free wall rupture ✓
 - Tamponade ✓ due to free wall rupture
 - Papillary muscle rupture ✓
 - VSD (septal rupture) ✓

Weeks later

- Dressler's syndrome $\leftarrow$ [pericarditis]
- Aneurysm ✓
- LV Thrombus/CVA ✓
 $\hookrightarrow$ If ventricular

* MI pt presented with pulmonary edema after 6 days:-

So the dx is papillary muscle rupture...

hypotension - muffled heart sound - tachycardia after 6 day:-

The dx is free wall rupture - corp pulmonale...

* Shockable rhythm:-

① pulses v tag...

② VF...

The treatment:-
 dc shock + P.R..

* electrical activity + S-stone !!

there is pulse
 $\downarrow$

non shockable rhythm

tt:- CPR - epinephrine -
 amiodarone...

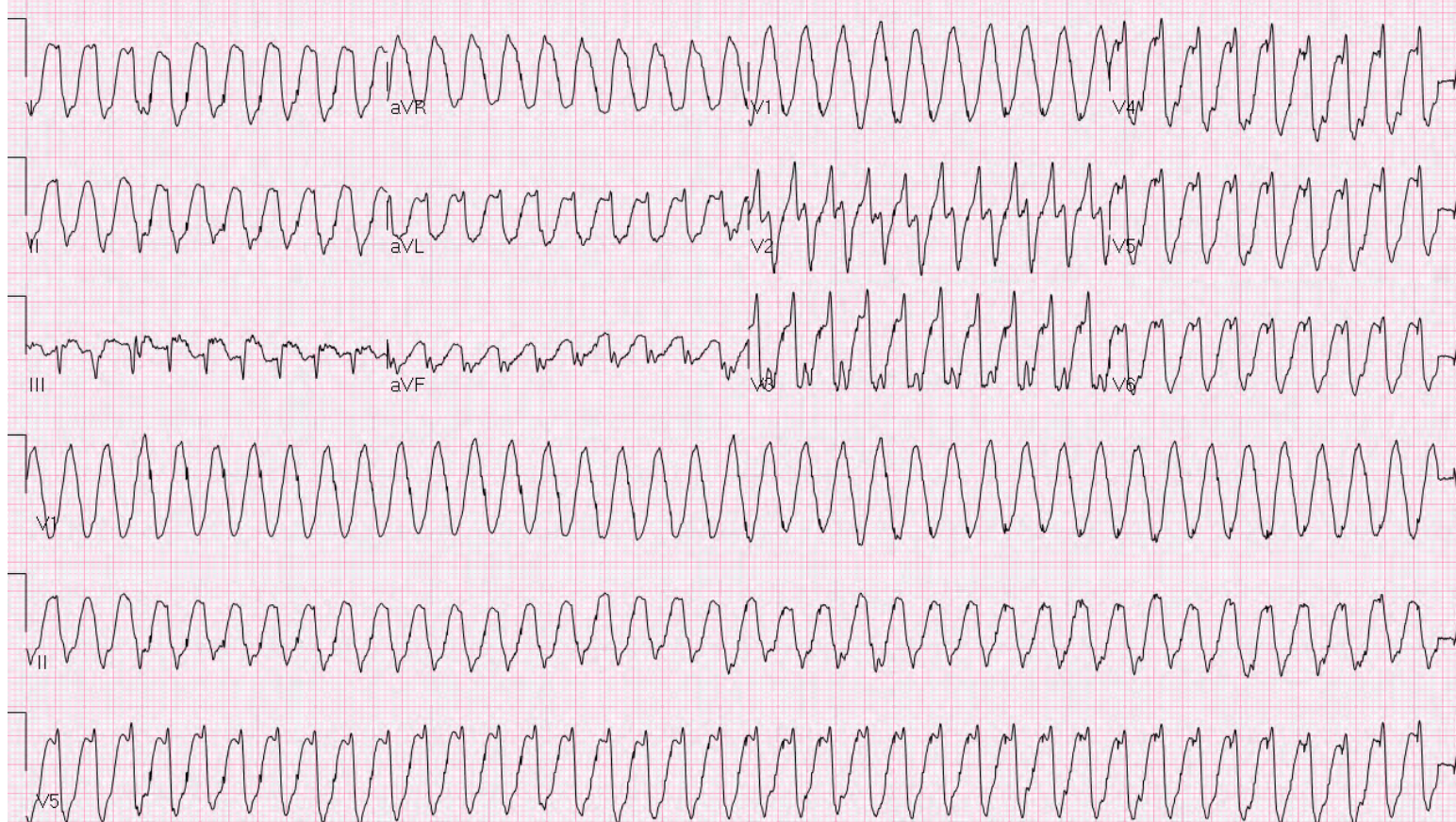
> chest pain increase with insp. and relieved with lying forward after 2 week the dx is dresser syndrome...

Cause of Death

0 – 4 days after MI

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monomorphic v. tag: - $\frac{\text{monomorphic}}{\text{tag}}$ $\left\{ \begin{array}{l} \text{if it with pulse} \rightarrow \text{ttt: drugs as: amiodarone...} \\ \text{if it without pulse} \rightarrow \text{ttt: dc. shock - PCR...} \end{array} \right.$



Sudden Cardiac Death

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- Most common cause among older patients: **myocardial infarction**
- Ventricular tachycardia → ventricular fibrillation → cardiac arrest

↳ J-tag →
سنة شدة blood يطلع

VF
no Blood (no contraction)



5-10 days after MI

* holosystolic murmur happen in:-

- ① mitral regurge.
- ② TC regurge..
- ③ VSD..

Transthoracic Echocardiogram

- **Free wall rupture** ✓
 - Usually fatal – sudden death
 - May lead to tamponade *Pericardium بليغ وبتحويته*
- **Papillary muscle rupture** ✓
 - Acute mitral regurgitation (holosystolic murmur)
 - Heart failure, respiratory distress *lead to pulmonary edema.*
 - More common inferior MIs
- **Septal rupture – VSD**
 - Loud, holosystolic murmur (thrill) *an systolic murmur...*
 - Hypotension right heart failure (↑ JVP, edema) *no pulmonary edema sympt*

* Cardiac tamponade → 50% of pt presented with triad:-
1- hypotension.. 2- Muffled heart sound.. 3- ↑JVP ...
in this case need pericardial synthesis



* PDA lead to machinery number...

Ventricular Aneurysm

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Weeks to months after MI

- More common anterior infarction
- Risk of thrombus → stroke, peripheral embolism
- Causes persistent ST elevations appear in the Echo..

↳ Just need anti coagulant...



Patrick J. Lynch, medical illustrator/Wikipedia

* Persistent ST elevation:-

Pt presented with ST elevation and we make PCI then after 2 week the pt presented with another ST elevation... + normal cardiac bio markers [no Ischemia]...

This indication for Ventricular aneurysm...

↳ late complication...

it's mean there is thrombus causing stagnation of the blood
↳ lead to CVA, acute mesenteric Ischemia, acute LI Ischemia... → presentation of the pt...

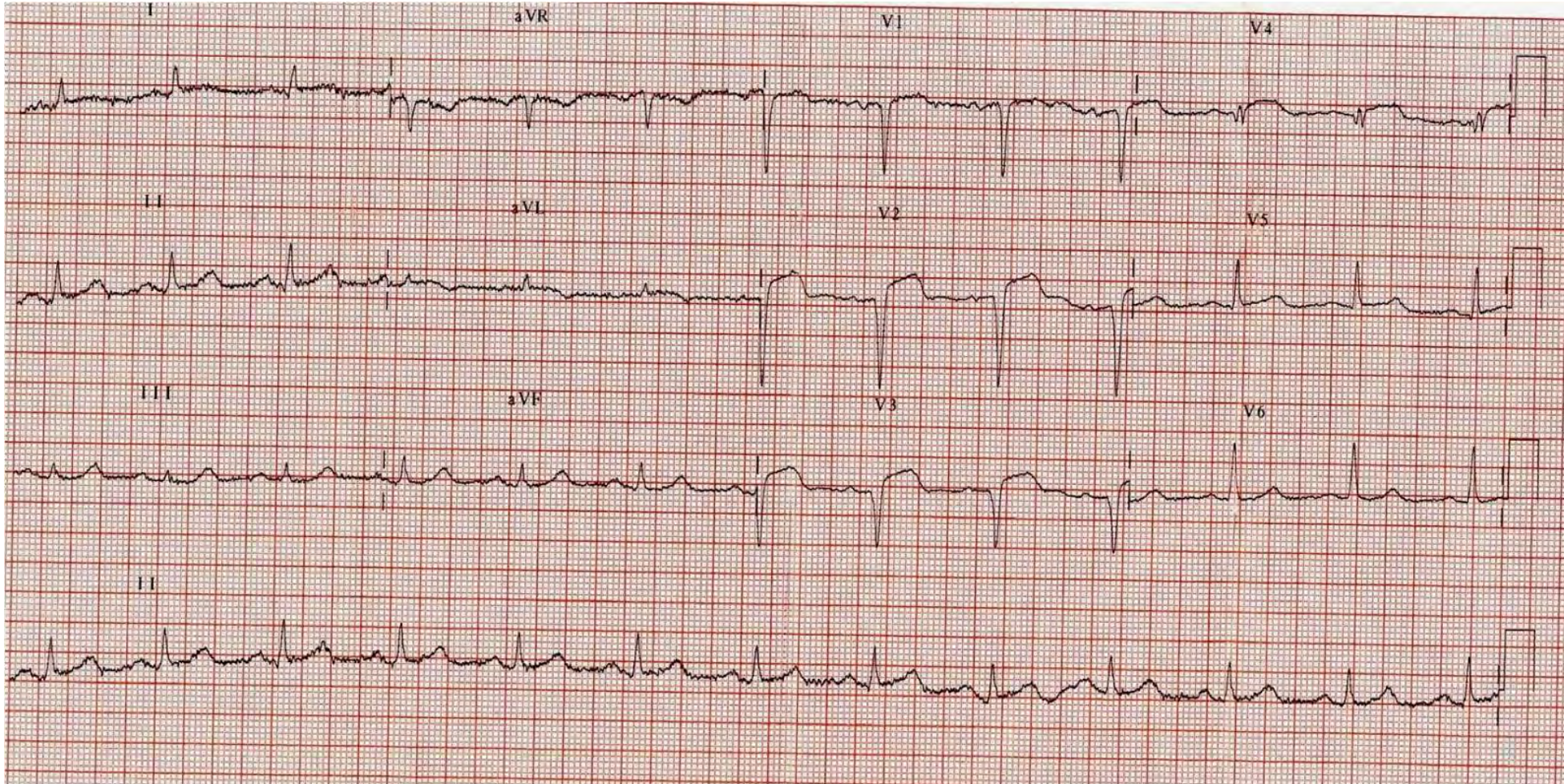
Ventricular Aneurysm

late complication
Weeks to months after MI [persistent ST elevation] ...

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* Q wave :- the 1st depressed wave in the QRS complex...

القياس الأقل من 1/4 R



Ventricular Pseudoaneurysm

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mean:- injury in the layer and the blood enter
دیوار به بیرون

1 to 2 weeks after MI

- Rupture contained by pericardium/scar tissue
- Not a true aneurysm
 - No endocardium or myocardium dilatation...
- May rupture
- Presents as chest pain or dyspnea
- Often seen in the inferior wall
- Occurs earlier (< 2 weeks) than true aneurysm

* pericarditis with MI → after 2-3 days of MI ... [fibrinous] ...
↓
lead to pericardial fluid ...
→ after 2-3 weeks of MI ... [autoimmune called Dressler's] ...

Dressler's Syndrome

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Weeks to months after MI

- Form of pericarditis
 - Chest pain ✓
 - Friction rub ✓
- Immune-mediated (details not known)
- Diagnosis: clinical the chest pain increased with insp. but relieved with lying forward دکتر
- Treatment: NSAIDs or steroids and colchicine [drug of choice]
 - ↳ pain reliever..
 - ↳ mc side effect :- diarrhea..

Fibrinous Pericarditis

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not need Ht ...

- Occurs days after MI
 - Sometimes called “post-MI” pericarditis
 - Not autoimmune
 - Extension of myocardial inflammation
- Dressler’s occurs weeks after MI *autoimmune...*
 - Sometimes called “post cardiac injury” pericarditis
- Rarely life-threatening
- Diagnosis: clinical

Evaluation of Chest Pain

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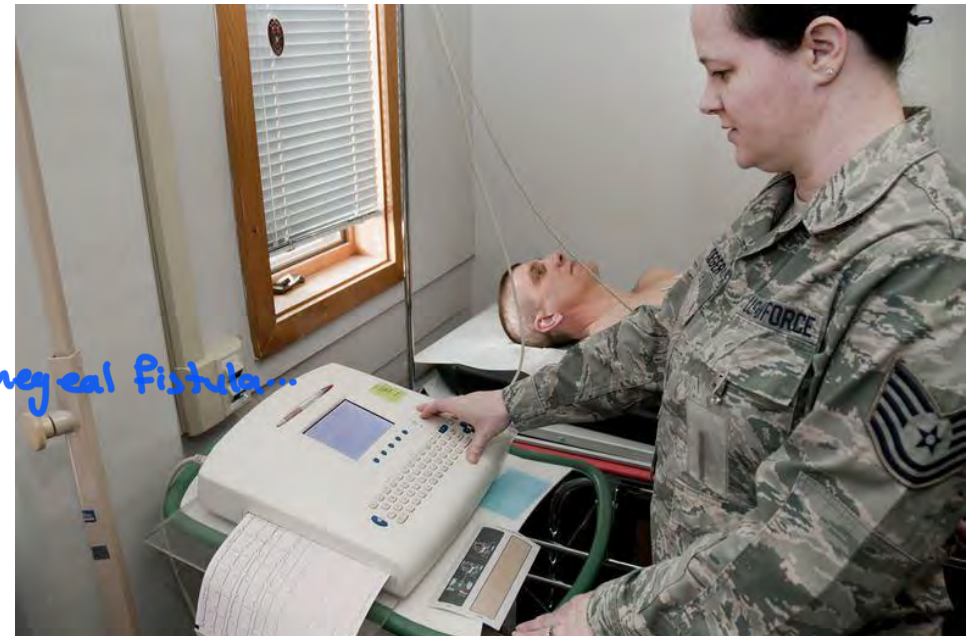
- Must consider: STEMI, aortic dissection and pulmonary embolism
- Best first test: EKG to evaluate for STEMI
- Additional testing considerations:

to exclude other cause..

- CT scan (PE or dissection)
- Cardiac biomarkers (NSTEMI)
- D-dimer

* life threatening chest pain:-

- MI
- esophageal rupture or tracheoesophageal fistula...
- tension pneumothorax
- massive PE
- aortic dissection



Public Domain

* The heart in Aortic regurg or stenosis make:- compensatory mechanisms to make the symptom acute

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↑ If ventricular complians... ^{due to}
via eccentric hypertrophy → ↑ Blood in ventricle
بب و رجاج

* pt with If ventricular systolic dysfx → HF..

* HF < ^{Systolic} diastolic OR * HF < ^{lF side} R side

Heart Failure I

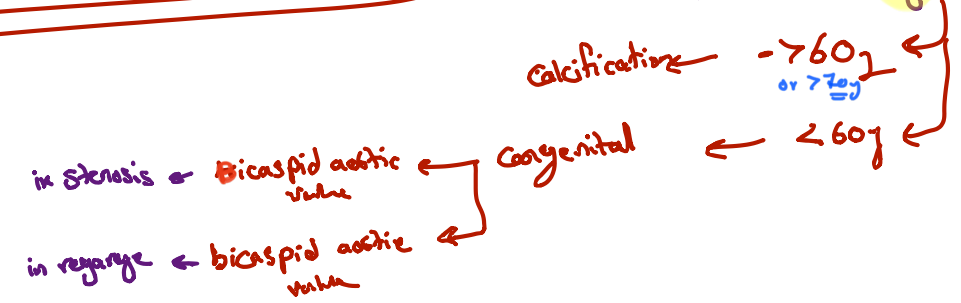
Jason Ryan, MD, MPH

* diastolic dys fx HF called heart failure with preserve ejection fraction [HFPEF]
normally:- 50-65

* HF with reduced ejection fraction [HFrEF]
↳ < 45 ↳ Systolic HF...



^{severe}
* aortic regurg and stenosis → most common cause of them depends on age



Heart Failure

Definition

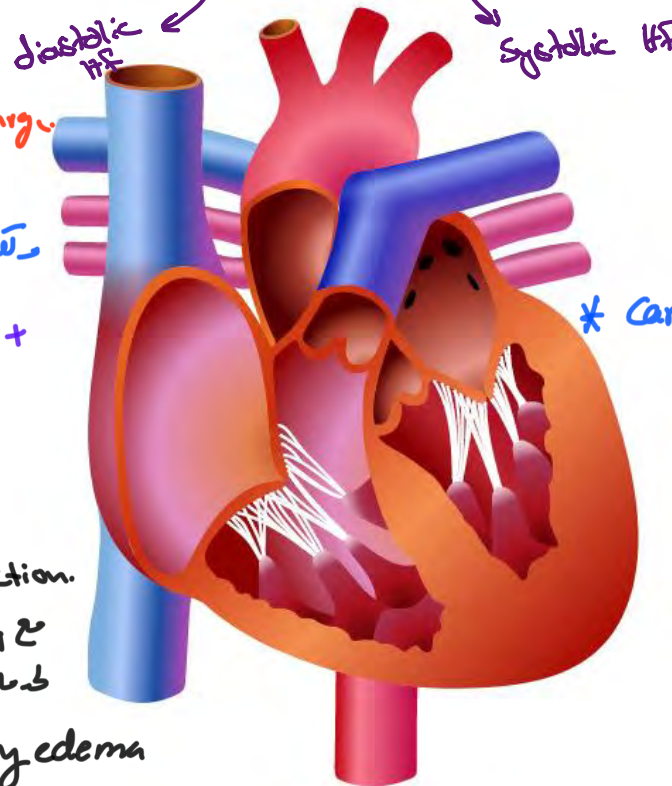
- Clinical syndrome
- Impaired ability of **left ventricle** to **fill or eject blood**

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mcc for it is previous MI [Ischemic Heart dz] $\Rightarrow$ 50% of causes...
 مبدء القلب بطل تاد يعجل contraction

Lt side HF $\rightarrow$ Symptoms: dyspnea, orthopnea, PND, night cough
 Rt side HF $\rightarrow$ Symptoms: up $\rightarrow$ $\uparrow$ JVP
 down $\rightarrow$ not neg liver, ascites, cardiac cirrhosis, LL edema "bilateral"
 Cirrhosis due to congestion in the liver... $\rightarrow$ pitting.

* Congestive heart failure combined...
 $\rightarrow$ Rt + Lt HF



* In valvular dyfx as:- Aortic regurge or mitral regurge.
 في مبدء القلب بطل تاد يعجل contraction
 مبدء مع جزر مبدء...

* The Aortic or mitral regurge + Ischemic heart dz + Cardiac myopathy has the same mechanism:-
 IP ventricular volume overload $\rightarrow$ IP.V. stretch...
 [$\uparrow$ Blood in IP.V.]
 $\downarrow$
 $\uparrow$ Stroke volume
 $\uparrow$ ejection fraction.

* Cardiac hypertrophy $\rightarrow$ eccentric in systolic HF...
 $\rightarrow$ concentric in diastolic HF...

eccentric hypertrophy $\Rightarrow$ chronic volume overload $\Rightarrow$ decompensating HF $\rightarrow$ lead to pulmonary edema

Heart Failure

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Types

Heart failure with reduced ejection fraction

↪ < 45

- HFrEF
- Systolic heart failure

Problem ejecting blood from left ventricle over load...

- Usually caused by a dilated cardiomyopathy

ischemia 50% "previous MI"
valvular heart disease
Cardiomyopathy
→ If ventricular
Volume over load
due to systolic dysfx

Heart failure with preserved ejection fraction

lead to stiff
ventricle

- HFpEF
- Diastolic heart failure

or diastolic dysfunction

Problem filling left ventricle with blood

↳ loss of lusitropy = loss of compliance = stiff ventricle...

مشاكل في قلب دم

بشكل دم يعني الدم
الراصد لا يتقبل...

ventricle متقار

lead to stiff

ventricle متقار

بشكل دم يعني الدم
الراصد لا يتقبل...

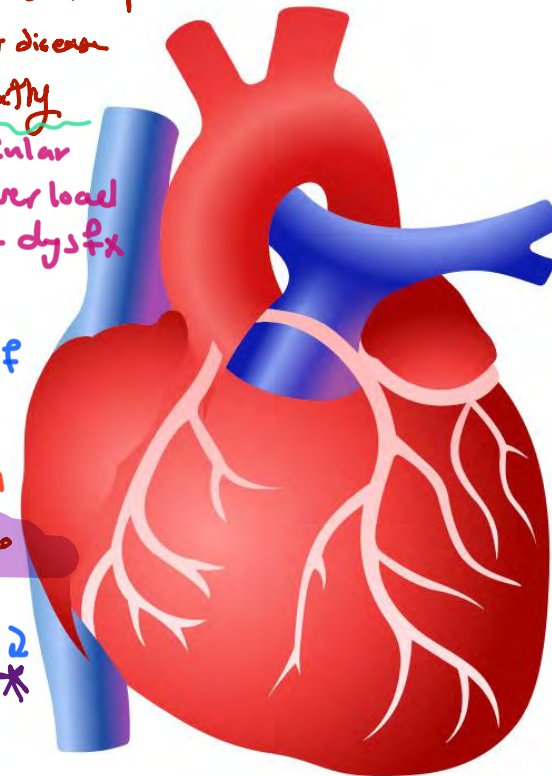
مشاكل في قلب دم

HFPEF

normal ejection fraction HF

بتصير في الكار بالمر نتيجة

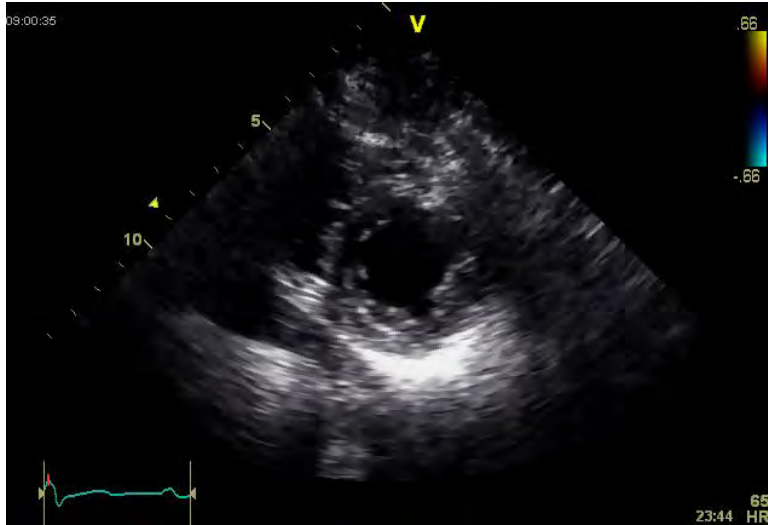
long standing HTN, diabetes



normal ejection fraction 50 - 65%

متن قادر یستقبل دم

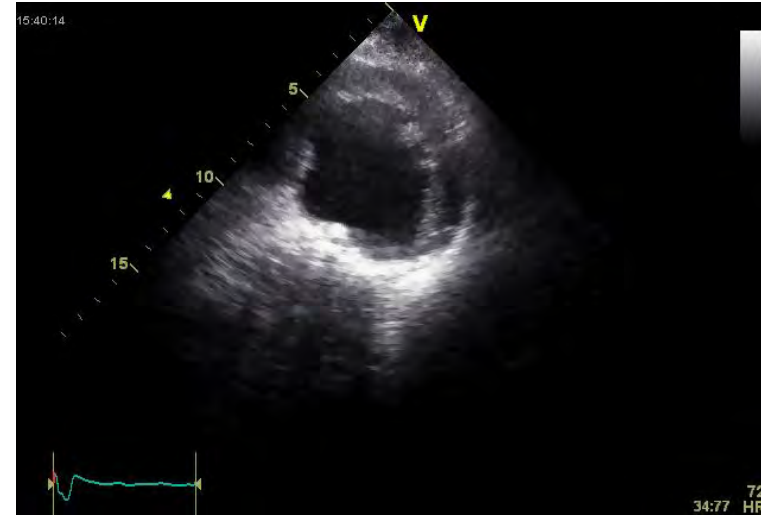
Diastolic Heart Failure



EF is normal (55-65%)

stiff ventricle متن قادر یستقبل مله بقه ریجی contraction

Systolic Heart Failure متن قادر یعمل ejection



Ejection fraction is reduced

There is dilated ventricle..

بہتر کلینکل

Volume Status

* HF sign:-
 ↑ central v.p.
 ↑ JVP.
 LL edema
 crepitation
 3rd space loss...

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* Euvolemic

* Hypervolemic

- Volume overloaded
- Wet

* Hypovolemic

- Volume depleted
- Dry
- Most heart failure symptoms from **volume overload**

3rd space loss
 LL edema

Crackles in lungs

↑ JVP [Rt atrial p + central venous p.]
 normally :- 6-8 ...

ascites and pleural effusion

3rd space loss

There is blunt in costo phrenic angle...
 X-ray, Clinical signs

~ 3000 cc

in Antro post. xray.

* but in lat xray it appear at 200-1500 cc

normal

2000

5000

10000

← Pleural effusion

← Pericardial

← Peritoneal

lead to retroperitoneal fluid...

1st space → fluid in cells [intracellular]

2nd " → " = interstitial

3rd " → " = site not participate in circulation

ascites

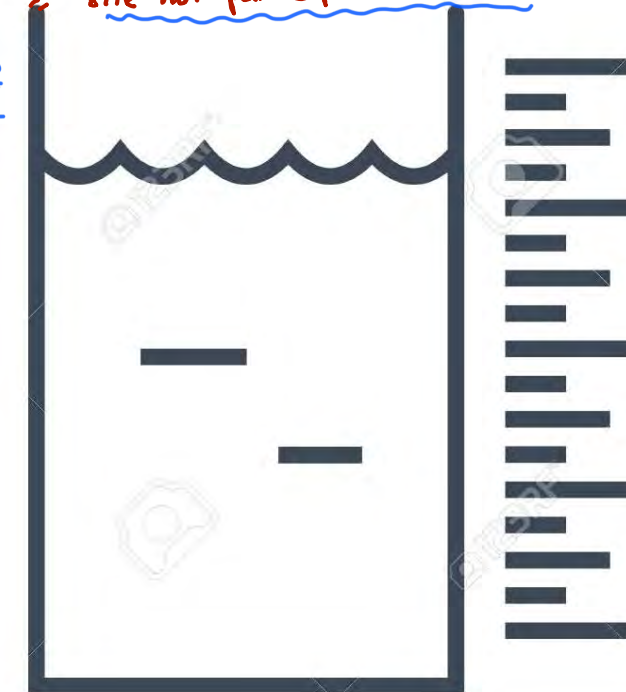
Sepsis ①

burn ②

pancreatitis ③

Cavities ④

Causes of Ascites:-



X-ray → anteroposterior
 → lateral

$$JVP = \underset{\substack{\text{Rt atrial pressure} \\ \rightarrow 5}}{\text{Rt atrial pressure}} + \underset{\substack{\text{Central veins pressure} \\ \rightarrow 1-3}}{\text{Central veins pressure}} = 6-8$$

Volume Status

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- Assessed by physical exam
 - Rales, edema, jugular venous pressure if elevated...

The volume state

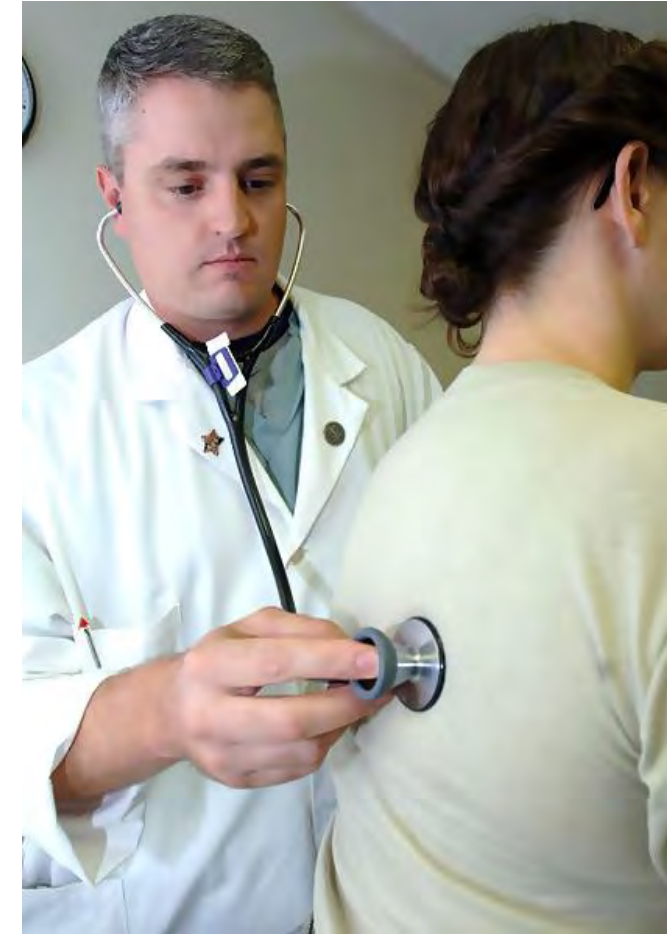
Unrelated to ejection fraction

- LVEF of 20% can be dry → severe systolic HF
 - LVEF of 70% can be wet → has edema "HFrEF"
- * Maybe normal ejection fraction but volume overload

hyper volemia wet patient overloaded

* reduced ejection fraction → the Heart pump متقاربة تفعل
pulmonary edema بزيادة في ضغط الدم في الرئة

لا تكون موجودة
لا تكون موجودة



Wikipedia/Public Domain

Heart Failure

Volume Overload Symptoms

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Normal

^{clinical}
Pulmonary Edema



- Dyspnea + night chronic cough...

* Orthopnea → when lying flat

* Paroxysmal nocturnal dyspnea → بعضى سم النظم خفونه

- Classic finding is rales → خرخرة [أنتس على رئة حليقة عاد ما تخرج بعد صدمة
فوضلة ارسلى الارجينين ...]

- Fluid filled alveoli "pop" open on inspiration

- Chest X-ray shows congestion

characteristic

white bilateral xray

→ pulmonary edema

شوي جناح بخاش appearance

له عين اى fluid يقع بشكل كبير حول heart
من خلاصة من علامات ال HF ...

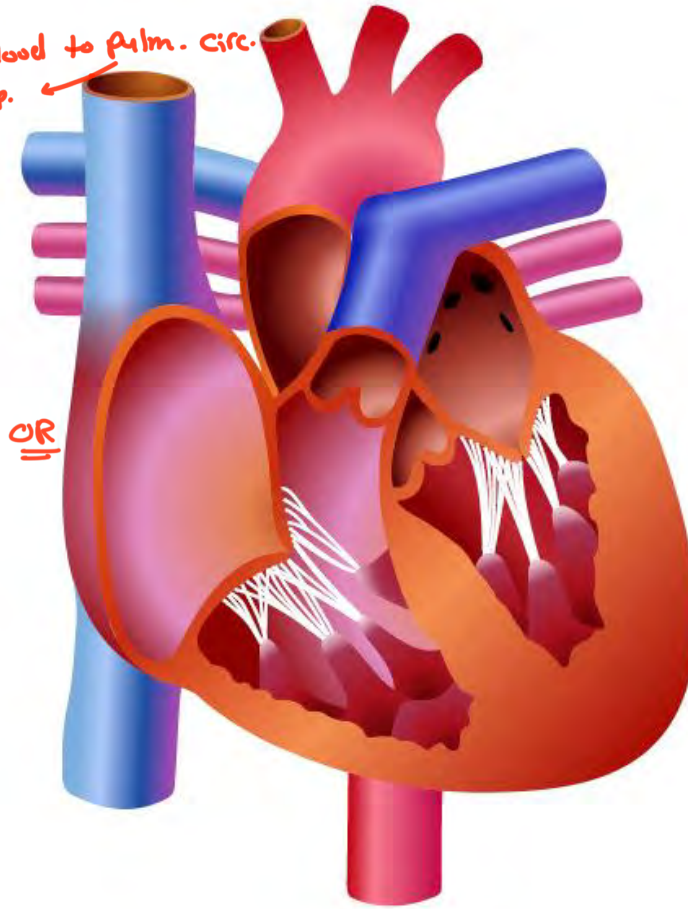
What are x-ray findings of HF?

- ① Bat wing appearance [white lung] → transudative → MC cause is HF
- ② pleural effusion → exudative → MC cause is infection
- ③ Kerley B line → خطوط بيضاء
- ④ Pulmonic cephalization → upper congestion > lower congestion in xray.
- ⑤ cardiomegaly

Increased Pressures

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- * Increased left atrial pressure → lead to back ward of the blood to pulm. circ.
- * Pulmonary capillary wedge pressure $\uparrow$ pulm. cap. wedge P. $\uparrow$ MPAP
- * Increased pulmonary artery pressure $\uparrow$ MPAP
- * Increased right atrial pressure



main pulmonary arterial pressure = $\boxed{93 \text{ mmHg}}$ (normally) $= D + \frac{1}{3} \text{ pulse pressure [systolic-diastolic]}$ OR $\frac{2D+S}{3}$

$80 + \frac{1}{3} (120-80) = 93$

pulmonary hypertension → MPAP > 25 mmHg at rest or > 30 mmHg at exercise

↳ class ↳ may be due to HF...

* pulse pressure → wide
← normally 40 → narrow...

* pt 85 years old presented to ER with BP: 180/60
So the pulse pressure = $180 - 60 = 120$ so it wide P.P...
normal in this age...

NYHA Classification

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Depend on symptom...
مراحيل يفرق حينا في

Class	Symptoms
I	Asymptomatic
II	Symptoms with moderate exertion
III	Symptoms with ^{minimal activity} <u>activates of daily living</u>
IV	Symptoms at rest

the ejection fraction 40% in Echo...

has history of heart disease

مع الحركة الكيفية بتعب

دوبوا راح عالجمام

need heart transplantation...

① S3 Gallop

Signs/Symptoms

indication for ventricular volume overload...

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murmurs { Diastolic → always pathologic as S₄
Systolic → pathologic
physiologic

- Classic finding associated with **increased LA pressure**
- Indicates **volume overload state**
- Normal finding in **children** pregnant women

→ Systolic HF

→ physio S3 presentation...



Heart Failure

Signs/Symptoms

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heart exam
a part of precordial examination

② Elevated jugular venous pressure (normal 6-8cmH₂O)

• Look for height of double bounce

• Hepatojugular reflux →

JVP المرفأ تا 45° بضغط على liver بنري

normal increase JVP [3]

Volume overload
HF
في كثير من صيغ المرض

seen not palpable

↓

JVP features:-

→ between 2 sternocleidomastoid m.

→ above 1-3 of sternum → أكثر من صيغ حالي
المرضى صلي راني

→ 6-8 mm Hg

→ JVP = 5 + CVP
Arterial P. CVP 1-3

ICU في ICU
CVP

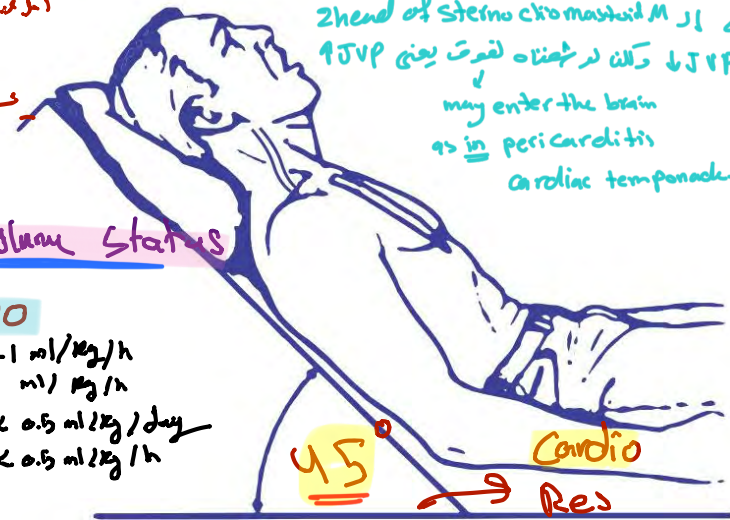
سؤال جرافة
المرضى

What is the best method to detect volume status to patients in ICU? urine output.

Urine output → anuric oliguric normal
المرضى

CVP in cardiac patients

* Normal VO
adult: 0.5-1 ml/kg/h
child: 1-2 ml/kg/h
- anuria < 0.5 ml/kg/day
- oliguria < 0.5 ml/kg/h



2 head of sternocleidomastoid M
JVP قد يكون
قد يدخل الدماغ
as in pericarditis
cardiac tamponade.

Jugular

Carotid

between 2 sternocleidomastoid m.

seen not palpable
has 2 waves (A, V)

palpable not seen
has 1 wave

* hepatojugular reflux:- بقاء المرفأ 45 درجة

deep palpation liver لا يضغط في JVP يزيد
↑ JVP ← Rt A ← hepatic vein ← blood

normally increased less than 3
but if increased more than 3 indication for volume overload in HF pt

15° → GI

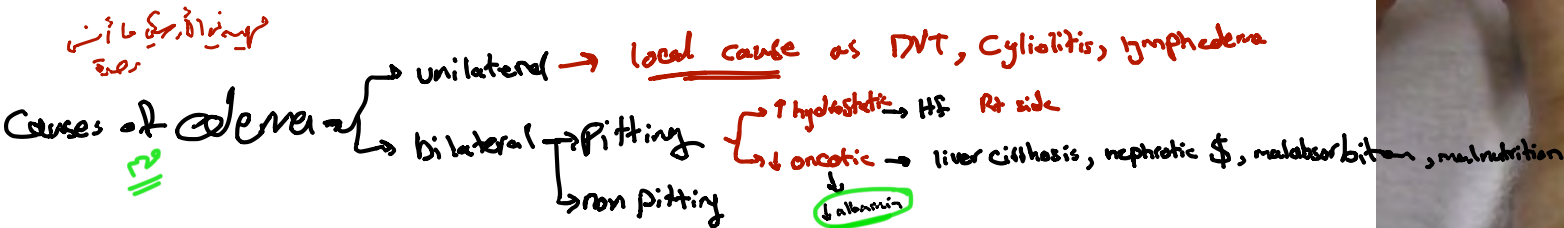
Heart Failure

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Signs/Symptoms

③ Lower extremity pitting edema ^{من نقرت باصبع}

- Increased capillary ↑ hydrostatic pressure
- Fluid leak from capillaries → tissues
- Gravity pulls fluid to lower extremities



James Heilman, MD/Wikipedia

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↳ but if it $> 1/4$ indicate for old Ischemia ...
[علا صدمة ولكن عند شدة دمومها]

- ECG



Q waves

- ## Transthoracic echocardiogram

- Used to distinguish HFpEF from HFrEF → ↓ E fraction
- Critical to determine therapy

- * Echo

Brain Natriuretic Peptide

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* HF pt + very ↑ BNP → poor prognosis...

* HF pt + hyponatremia → poor prognosis...

BNP

hormones

releases in volume overload states
→ HF...

- ANP (atrial natriuretic peptide) released by atrial myocytes
- BNP (brain natriuretic peptide) released by ventricles → If high in HF patient is a poor prognosis
- Serum levels rise with volume/pressure overload → in HF
- Both counter effects of RAAS system → HF due to RAAS system...
- BNP sometimes used for diagnosis in dyspnea
- Highly sensitive for diagnosis of heart failure (100% for HF)
- High levels seen in most cases of heart failure but also many other disorders
- Low levels strongly suggest other causes of dyspnea

HF with hyponatremia } الی بخونی
HF with high BNP } نی سریه

مقال
MRCB

* HF patient - , which of the following is the worst prognostic factor of this patient?

→ hyponatremia

clinical مرصه عنه dyspnea مشكاهه

بدی test لخواه یفرستی ایا res.p, Cardiac

ANP/BNP
↓
sensitive not specific
normal → HF
↑ → Cardiac → HF [normal BNP] (مستثنی بمر)
ves → pneumonia, COPD
normal (ممكنه)

RAAS

100% sensitive tests:-
ANA → SLE
D-dimer → PE
BNP → HF

used for exclusion



Right heart catheterization

Pulmonary Artery Catheterization

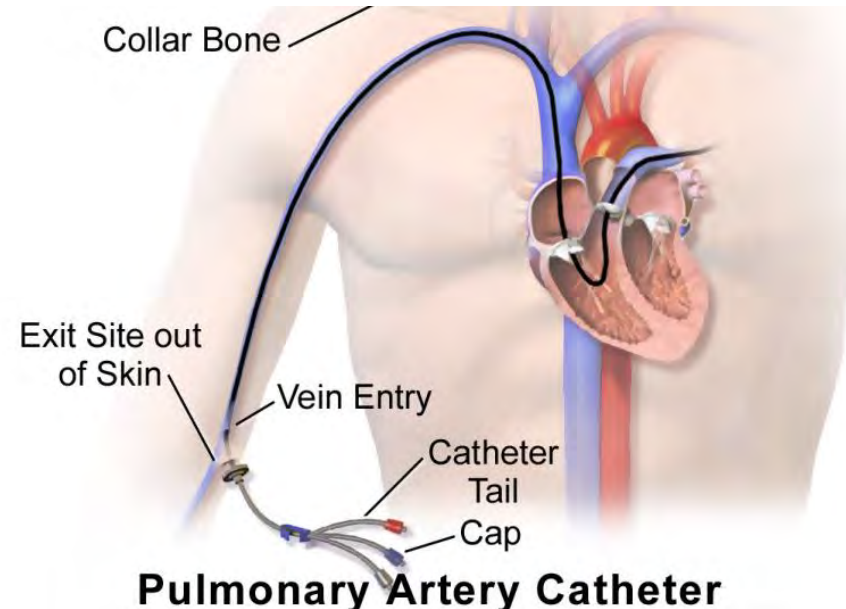
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enter from radial or femoral vein...
to reach Rt A and Rt V...

HF diagnosis modality of choice to diagnose the HF...

ما تكون Echo من وظيفتها :-

- Increased PCWP = left heart congestion/failure
 - PCWP = pulmonary capillary wedge pressure
- Increased RA, RVEDP = right heart congestion/failure
 - ↓ Rt atrial pressure
 - ↓ Rt ventricle in diastolic pressure



Pulmonary Artery Catheter

BruceBlaus

Heart Failure

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Diagnosis

- History ↗ Rt HF: edema - liver congestion - Ascites - etc
↘ Lt HF: dyspnea - orthopnea - PND - night cough..
- Physical examination hyper volemia - Clpitation..
- Chest X-ray 5 finding
- Transthoracic echocardiogram to confirm if it diastolic dysfx or systolic dysfx ⊕ to see the valves...
- EKG may appear in it:- old Ischemia [Q wave] - arrhythmia.
- BNP level to confirm if it cardiac or not..
- Right heart catheterization ✓

Sensitive

Cor Pulmonale

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* R+side HF due to
 → IP side HF..
 → lung dz..

- Isolated **right heart failure**
 - Hypertrophy ✓
 - Dilation ✓
 - Decreased contractility ✓
- Caused by long standing pulmonary hypertension
 - Lung disease (e.g., **COPD**)
 - Primary pulmonary hypertension
 - idiopathic



Cor Pulmonale

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↳ There is R + A enlargement due to R + side HF due to lung dz...

- Dyspnea with **right heart failure**

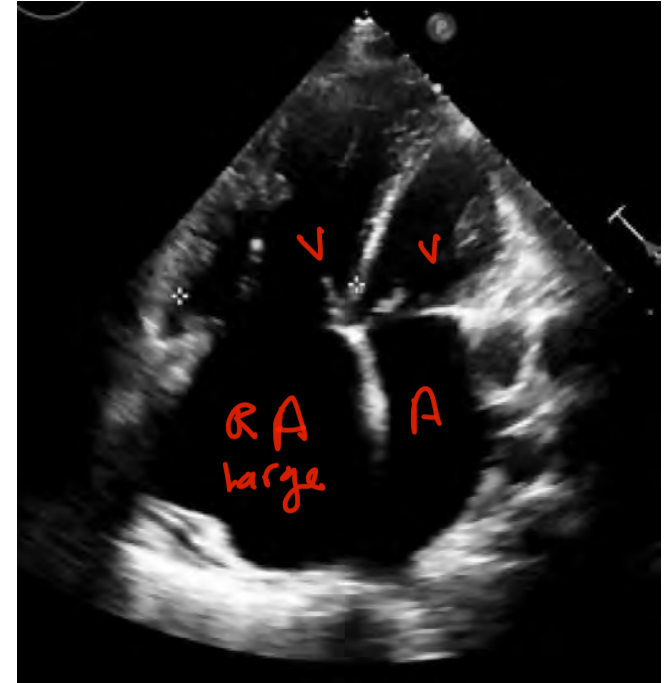
- Elevated jugular venous pressure ✓
- Lower extremity edema ✓
- Hepatomegaly ✓

- Absence of rales ✓

رئتين متضخمتين
due to lung dz...

- Treatment:

- Usually aimed at underlying disease (COPD, etc.)



* if there is rales → indication for L + side HF...

* pt with systolic dysfx [HFPEF] :- has bi Atrial enlargement but the ventricles normal...

High Output Heart Failure

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↳ ↑ HR or other cases as:- Anemia - thyroid dz ...etc

- Exact mechanism unclear
- ① • Decreased LV filling time
- Defining characteristic: **HIGH cardiac output**
 - Heart failure symptoms in absence of low output
 - ↑JVP, pulmonary edema

② • Heart in overdrive *منه* so appear the HF symptom...

↑ demand > supply ① Severe anemia Hb: 5, HR - stroke volume normal...

② Thyroid disease [hyperthyroidism → tachycardia]

③ Thiamine (B1) vitamin deficiency (beriberi) common in alcoholic pt...

④ A-V fistulas ⑤ Fever ⑥ RA ⑦ pregnancy ⑧ Bacteremia dz ⑨ Aortic regurg...

* $COP = \text{stroke volume} \times HR \dots$

* Beriberi → wet [cardiac]

→ dry [CNS]

↳ if it ↓ lead to sensory neuropathy



John Liu/Flickr

* ↑ HR → ↑ COP *منه* due to ↑ demand → ↑↑ HR :- filling لا وقت له → ↓ COP...

so there is impaired filling due to tachycardia lead to VF, AF and cardiac arrest

لوقت لا يطلع بيه الـ very ↑ HR

note:- when ^[B1] thiamine enter the cells lead to

- Acute:- wernicke encephalopathy [Confusion - nystagmus - Ataxia]...
- chronic:- korsakoff syndrome chronic form of wernicke + ^{بیش} Amnesia + ^{اضطراب بیاد} Confabulation... [re + n grade + type]

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* alcoholic pt presented to ER with confusion and syncope → we do glu stick :- we find the glucose:- 40 - 30...

طاولا، Pt ← alcoholic یعنی منبع غلucose بل غلucose First line ← Thiamine بیه دضه، ریاضه،
refeeling syndrome ⇒ ICU یا در منزل تاکن مثل بار ICU OR alcoholic pt + ↓ Glucose ...

Heart Failure II

↓ Glucose و thiamine و K⁺ + phosphate ...

Jason Ryan, MD, MPH ناله ال alcoholic pt دهنه ↓ glucose و thiamine و K⁺ + phosphate ...
دضه ال Confusion بیه در thiamine deficiency خلاصه جوتکه راجه غلucose ال thiamine اقلیل یه ضایل بارک یرض
↓ خلاصه رگده زندا اطیه بیه !! متعلیه Thiamine فانه ال glucose ...

* pt in ICU take normal saline for 3 days for example lead to ↓ glucose so the doctor give the pt glucose → هاد ضه افریه
یرض ال Confusion عطره بیه صدر ال refeeling syndrome و thiamine و K⁺ + phosphate ...
↓ دضه حله بیره



Boards & Beyond®
STUDY SMARTER

death ← resp muscle paralysis ل غلucose خنجا راجه یزدی ال

So in this & there is hypokalemia → arrhythmia
Maybe..

Heart Failure

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Treatment

- **Acute** heart failure *← pt presented to ER with dyspnea...*
 - Volume overloaded *we want to reduce this volume...*
 - Goal: **improve symptoms** of dyspnea, pulmonary congestion
- **Chronic** heart failure *in out pt*
 - Euvolemic
 - Goal: **prevention** of hospitalizations, mortality

Acute Heart Failure

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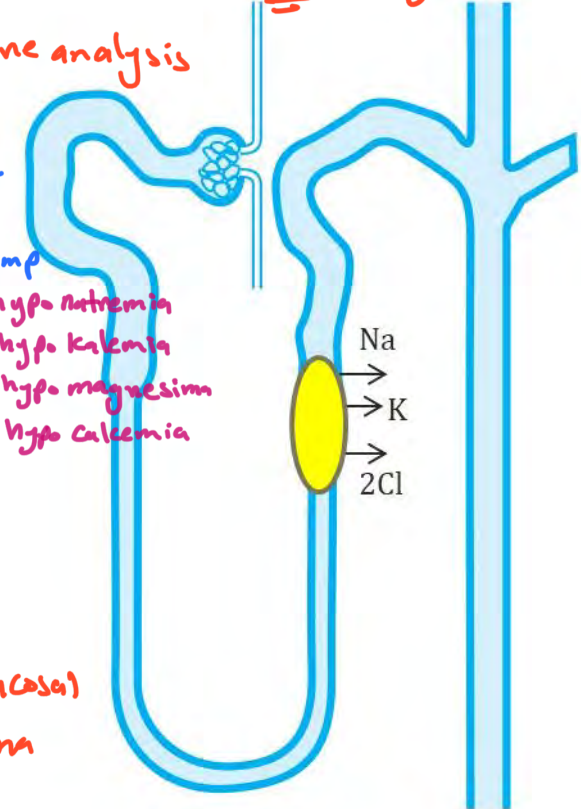
we give them

- O₂ [target O₂ saturation > 94%]
- sitting position...
- ECG to see if there is arrhythmia
- Chest xray to exclude the infection
- CBC, Chemistry to see if there is electrolyte dist.

urine analysis

mc

hypotatremia, acid



Goal: reduce left atrial pressure and remove fluid

- Improves pulmonary edema
- Most commonly used drug class: loop diuretics
 - Furosemide, torsemide [has sulfa group] + thiazide..
 - Increased urine output
 - Reduce intracardiac pressures
 - Improve pulmonary edema and lower extremity edema
 - Main adverse effect: hypokalemia
 - Other adverse effects: acute renal failure, hypotension
- Also cause some venous dilation: ↓ left atrial pressure

* all body mucosa has edema so the oral diuretic not effective in these pt due to GI mucosa
need IV diuretic as ↓ absorption of drug. ← edema
happens in high doses..

* if the pt has sulfa allergy give him → ethacrinic acid [loop diuretic doesn't have sulfa group]

Acute Heart Failure

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* The preload reducer drug is 1st choice in these pt...

Nitroglycerine

nitrate [nitroglycerine] → sublingual → ↓ preload → يتجنب الحرقية

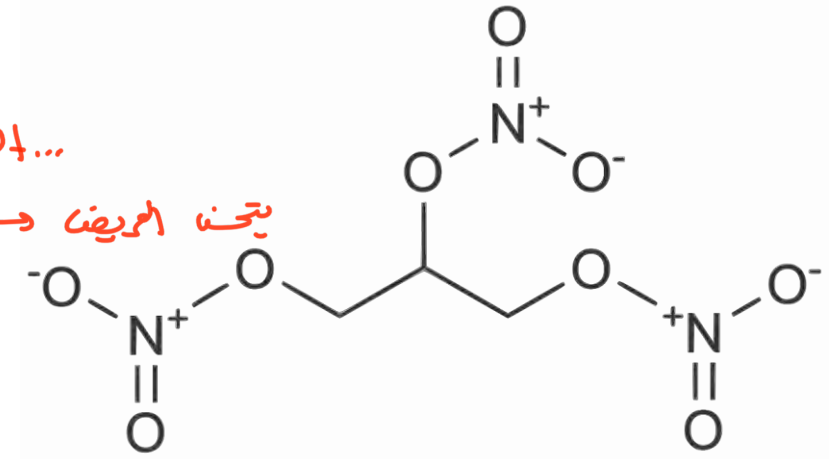
الغرض من إعطائه IV وoral أيضا بنعطيها
sublingual...

- Venous dilation → ↑ pool volume in venous system → ↓ preload.
- Reduces left atrial pressure ✓
- Improves dyspnea ✓

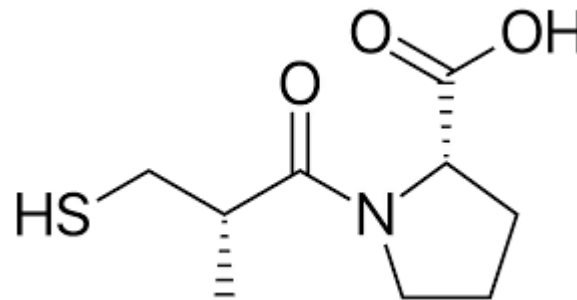
Afterload reducers

- Hydralazine, ACE-inhibitors ^{IV} _{oral}
- Increased cardiac output → improve renal perfusion → ↑ diuresis

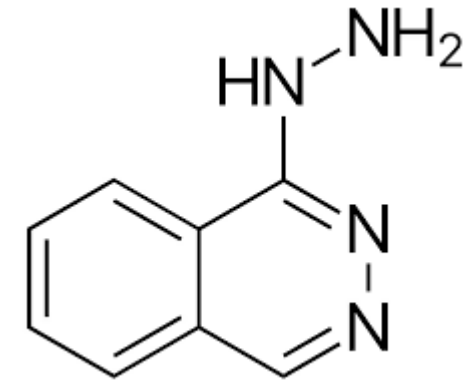
* in hypotensive pt not give nitroglycerine...



Nitroglycerine



Captopril



Hydralazine

Chronic Heart Failure

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- **HFpEF** *diastolic HF [dysfx]*
 - No specific therapies ✓
 - Control blood pressure, blood sugar
 - Chronic diuretics often used (furosemide)
- **HFrEF** *systolic HF..*
 - Many specific therapies shown to reduce hospitalizations and mortality
 - Treatments aimed at prevention of cardiomyopathy progression

Chronic Systolic Heart Failure

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- Goal: reduce hospitalizations and mortality

- **Guideline directed medical treatment (GDMT)**



- **Beta-blockers** (metoprolol, carvedilol, bisoprolol)

MBC

B1 Blocker

B1-B2 - α1 Blocker

B1 Blocker

- **ACE-inhibitors** (captopril, lisinopril) *via reduce thm after load.*

ARAB OR

Sub lingual

oral

- **Aldosterone antagonists** (spironolactone, eplerenone) *lead to hyperkalemia... [K⁺sparing agent]*

used in HF and ascites...

- **Neprilysin inhibitors** (sacubitril → increases ANP)

↳ androgen antagonist lead to gynecomastia...

- **Ivabradine** (inhibits SA node → ↓ heart rate) *but lead to ↑ contraction.. [-ve inotrope, +ve chronotrope]..*

↓ HR, ↓ HF pt 20%

- **Nitrates/hydralazine** (combination therapy)

- Most drugs disrupt chronic activation of SNS and RAAS

- Prevent cardiac remodeling (progression of LV dysfunction)

* ↑ ANP, BNP → indication for HF severity..

* we stop RAAS system via:- ACE inhibitor - Aldosterone antagonist - neprilysin inhibitor..



* **Digoxin** *Just give it in acute thm not respond to any other thm... not reduce mortality rate but reduce the hospitalization [morbidity]...*

mortality ↓ i.e. 5-2% long term ↓ 10% SW!

ICD

Implantable Cardiac Defibrillator

نفس shock للقلب

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* defibrillator
→ synchronise:- we use it in the AF - vtag [monomorphic] ^{pulse}
→ un synchronise:- we use it in cardiac arrest [pulseless vtag]..

in the hand

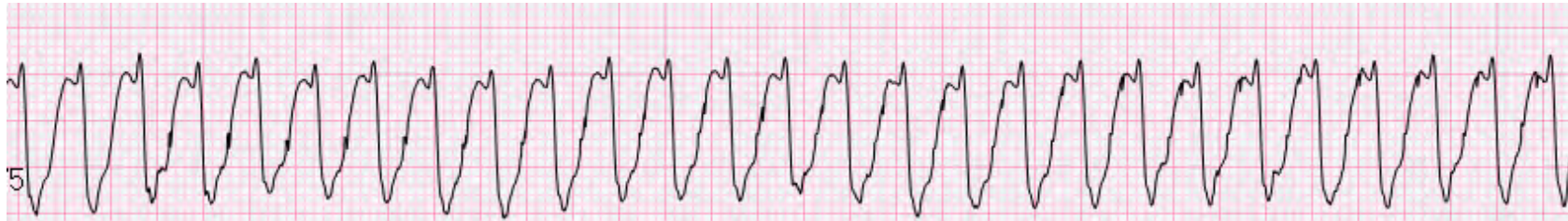
- Annual risk SCD $> 20\%$ some studies
- Most due to ventricular tachycardia

* The approach to cardiac arrest pt:- depend on electrical activity:-

① pulseless electrical activity + S. stole:-

non shockable rhythm → CPR - epinephrine
then amiodarone

② VF or pulseless v. tag:- Shockable rhythm $\leftarrow$ CPR rhythm



* any pt with $\downarrow$ EF < 35 or 30 need ICD...

* HF pt die from arrhythmia...
[shockable]

↓
so need ICD to give pulse when the attack happen..

* v tag
→ monomorphic :- no QRS complex
→ pulseless :- no pulse
→ خرابیت جاج [—]

ICD

Implantable Cardiac Defibrillator

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- Improve **mortality** in appropriate patients
- Indications:
 - Aborted sudden cardiac death HF + VF and enter ICD then improved so we put ICD as prophylaxis...
 - LVEF < 35% - 30



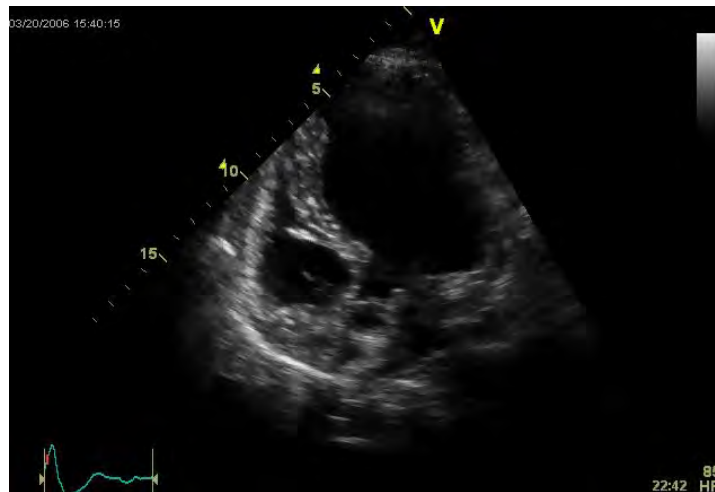
Biventricular Pacemakers

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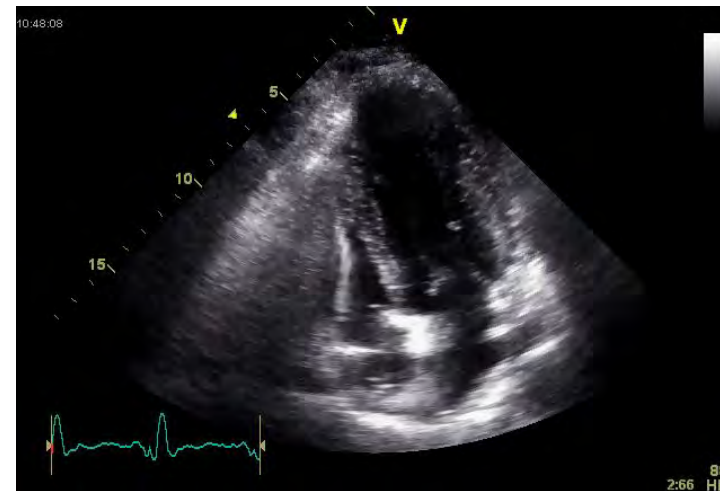
Cardiac Resynchronization Therapy (CRT)

→ we put pacemaker in both ventricles to prevent the cardiac arrest..

Out of Synchrony



After Pacemaker

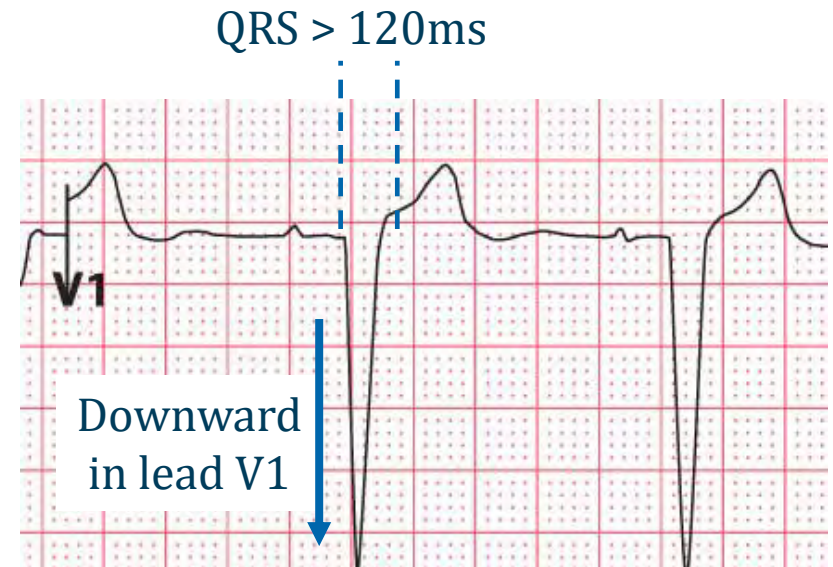
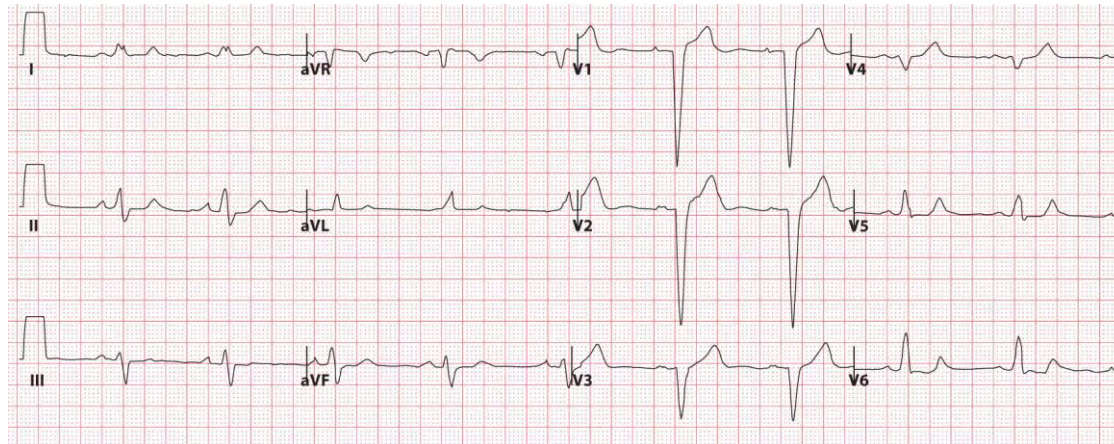
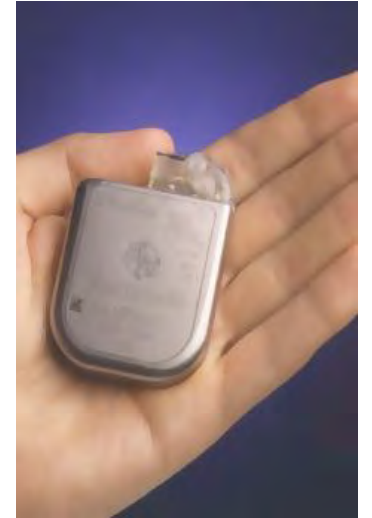


Biventricular Pacemakers

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Cardiac Resynchronization Therapy (CRT)

- Improve **mortality** in appropriate patients
- Indications:
 - **Left bundle branch block** (wide QRS; > 120ms)
 - **LVEF < 35%** - 30



Heart Failure Treatment Pathway

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Acute
Heart Failure

Rx

Furosemide
Nitroglycerine
Afterload reducers

Chronic
Heart Failure

Diastolic
Heart Failure

No specific therapy

Systolic
Heart Failure

Drugs ✓
morbedity - mortality ↓

ICD ✓

Bi-V Pacer ✓

فقط تخاف مع السج والفينه ديفينه

Advanced Heart Failure

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- Severe form of HFrEF
- Severely reduced cardiac output
 - Fatigue
 - Hypotension
 - Cool extremities
 - Confusion
 - Decreased appetite (cardiac cachexia)

Advanced Heart Failure

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Treatments

- **Inotropes (associated with ↑ mortality)**
 - Dobutamine ✓
 - Milrinone ✓
 - Digoxin (oral) ✓
- Left ventricular assist devices (LVADs)
- Heart transplantation

give it in PT with ↓ BP + HF

!!

Acute Exacerbations

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Causes

- #1: Dietary indiscretion
 - High salt intake
- #2: Poor medication compliance

* normal protein requirement daily :- $1\text{g}/\text{kg}$..
* normal K^+ requirement daily :- $0.5 - 1\text{ meq}$.
* normal Na^+ require $\rightarrow 1 - 2\text{ meq}$ daily



Acute Exacerbations

Causes

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A KI $\leftarrow$ ↓ renal perfusion $\leftarrow$ PG ↓ $\leftarrow$ قلة PG
* The NSAIDs contra indication:-

① HF...

② gastritis...

③ gastric ulcer...

- Infection/trauma/surgery
 - Activation of sympathetic nervous system
- Ischemia (rare)
 - Decreased cardiac output
- Arrhythmias (A fib)
- NSAIDs
 - Inhibit cyclooxygenase (COX) $\rightarrow$ ↓ prostaglandins
 - Prostaglandins maintain renal perfusion
 - Result: Less renal perfusion $\rightarrow$ salt/water retention

* تجنب مضادات الالتهاب غير الستيرويدية (NSAIDs) مثل ibuprofen + tramadol
في حالات renal colic + HF

Typical Acute Heart Failure Course

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- ER presentation:
 - Dyspnea, edema, sleeping in chair
- Admitted to hospital
 - Nitro drip to relieve dyspnea
 - IV Furosemide to remove fluid
- Hospital Day 2
 - Weight down 4 kg, feels better
 - Nitro drip stopped
 - Changed to oral furosemide *after relieved the bowel edema..*
- Hospital Day 3: Discharge

More Complex Heart Failure Course

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- ER presentation:

- Dyspnea, edema, sleeping in chair
- Known LVEF 10% *need ICD*

- Admitted to hospital

- Nitro drip to relieve dyspnea
- IV Furosemide to remove fluid

- Hospital Day 2

- Poor urine output, Cool extremities Cr rises 1.1 → 1.4 *if it increase 20% → AKI* *+ hypotension.*
- Dobutamine drip started: *دوبوتامين*

if stop it will die *||*
عنه، يمشي

More Complex Heart Failure Course

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- Hospital Day 3-5

- Good urine output
- Weight loss 4 kg
- Breathing improves

- Hospital Day 6

- Dobutamine stopped
- Furosemide drip stopped

↙ if we stop it and the symptoms relapse need life long dobutamine..

- Hospital Day 7

- Oral furosemide given

- Hospital Day 8: Discharge

Noninvasive Ventilation

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CPAP

- Positive pressure ventilation
- Administered through mask not ETT
- Used in acute respiratory failure type II
- Often effective for pulmonary edema
- Often avoids intubation
- Patient must be awake, alert, cooperative
- Classic case
 - HF patient in respiratory distress
 - Tachypnea, hypoxemia
 - NIV → improved respiratory status

endo tracheal intubation [NIV]
invasive method
so use CPAP

* CPAP :- Continuous pressure.

↳ used in HF and OSA [obst. sleep apnea]

↳ due to ↑ weight [obesity]

...hypoxia - cyanosis رصيد معمر بالليل



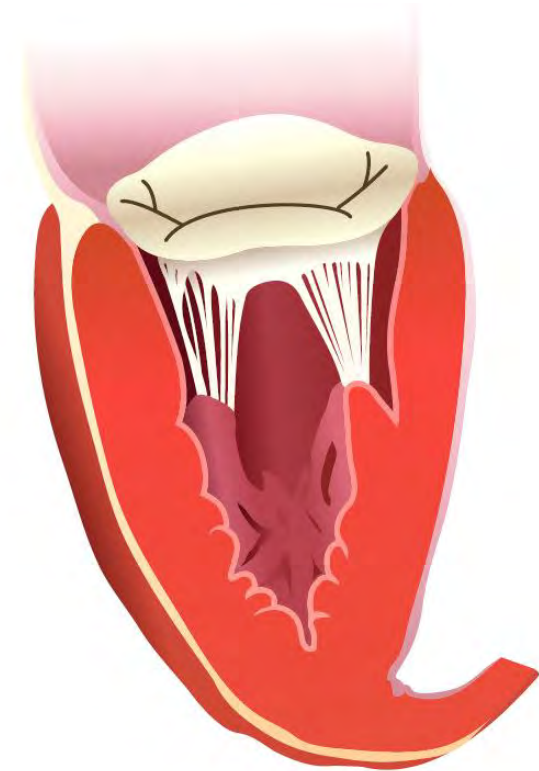
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* if CPAP failed the HF pt need intubation... !!

Functional Mitral Regurgitation

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- Caused by left ventricular dilatation
- Mitral valve leaflets pulled apart
- Can cause holosystolic murmur at cardiac apex
- May resolve with diuresis



* ADH lead to water retention → Hyponatremia.

Hyponatremia

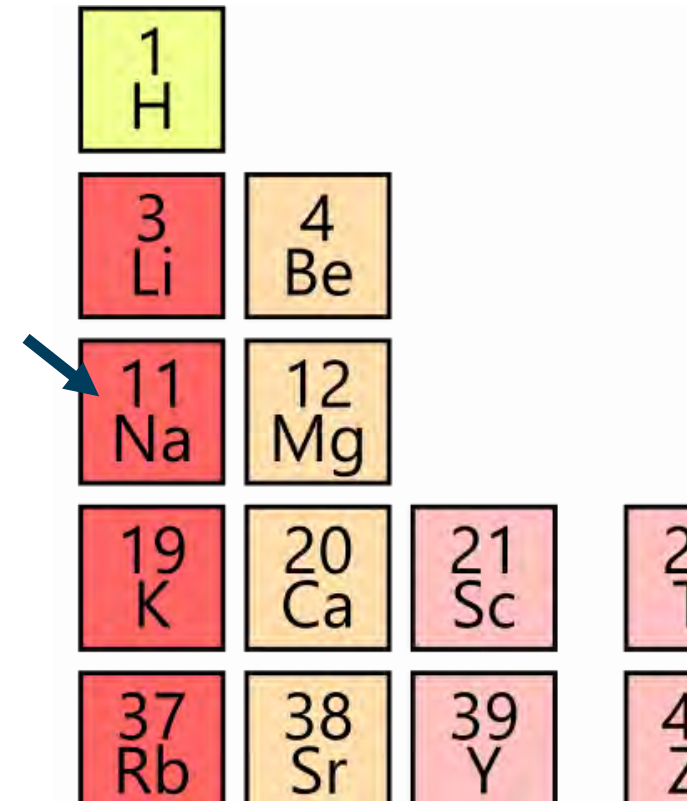
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↳ Most poor prognostic laboratory indicators...

- Occurs in severe heart failure
 - RAAS activation → ADH release → water retention
 - Poor renal perfusion → decreased water excretion
- Poor prognostic indicator**

↓ EF → ↓ kidney perfusion → activate RAAS system

... ↑ fluid ← Na⁺ - water retention



1 H			
3 Li	4 Be		
11 Na	12 Mg		
19 K	20 Ca	21 Sc	22 Ti
37 Rb	38 Sr	39 Y	40 Zr

لبسته تعب از valve بدو در آن شش از اخروی ..

Venous Stasis Edema^{LL}

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- Common in the elderly + edema may due to
- Caused by poor venous drainage
- Unrelated to heart
 - No dyspnea ✓
 - Normal lung exam ✓
 - Normal jugular venous pressure ✓
- Treatments:
 - Leg elevation ✓
 - Compression stockings ✓
 - Diuretics rarely work ✓
- May lead to skin ulcers/infection

heart
Kidney
liver

کاهش فشار خون و کاهش فشار لیبید و
venous stasis...



Drugs to Avoid in Heart Failure

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- **Metformin**

- May cause lactic acidosis in **CKD - HF...**

- **Thiazolidinediones (glitazones)**

- Pioglitazone, rosiglitazone pt with volume overload
...فقدت الوزن فارتفعت نسبة السوائل
- Cause fluid retention

- **Calcium channel blockers**

- Negative inotropes

- **NSAIDs** **lead to AFI...**

- Cause fluid retention

dihydropyridine
non dihydropyridine
lead to Bradycardia so
we don't use it in HF...



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Cardiomyopathy

Jason Ryan, MD, MPH

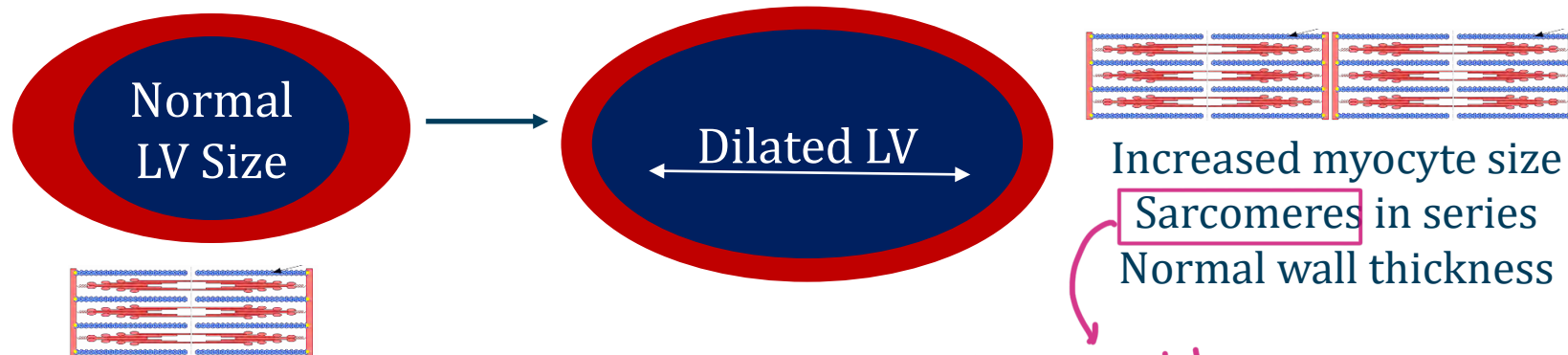


Dilated Cardiomyopathy

part of HF...

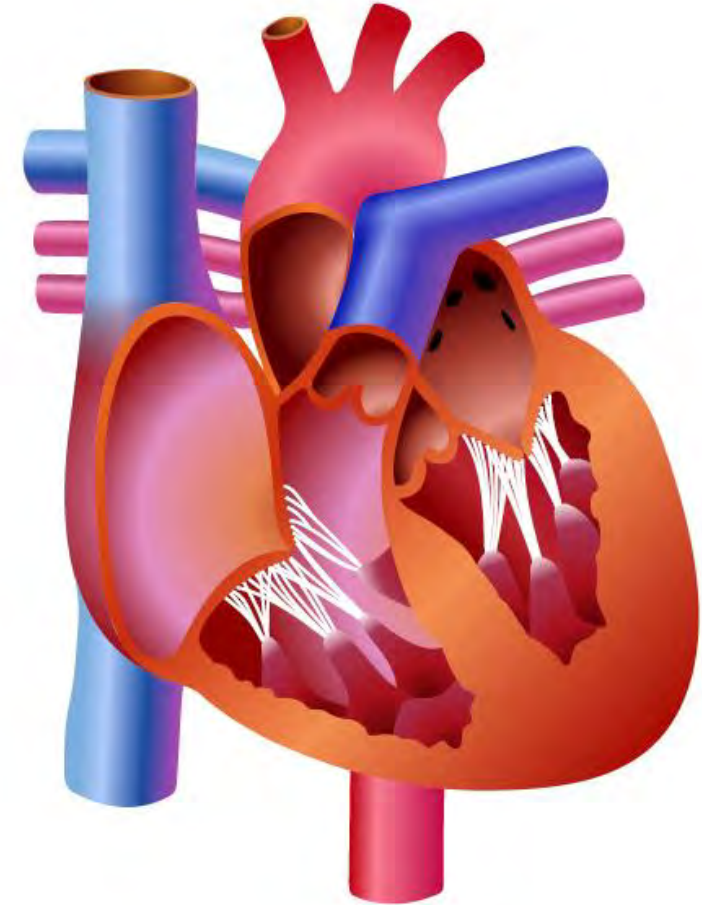
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- Systolic heart failure with LV cavity dilation with no hypertrophy in wall... so called eccentric hypertrophy due to volume overload...
- **“Eccentric” hypertrophy**
 - **Volume overload** (chronic retention of fluid in cavity)
 - Longer myocytes
 - Sarcomeres added in series



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- **Depend on volume status** ✓
- **Volume overload** → acute systolic heart failure
 - Dyspnea ✓
 - Pitting edema ✓
- **Severely reduced LVEF** <15%
 - Fatigue ✓
 - Cachexia ✓
 - Confusion ✓
 - anorexia due to mucosal edema + loss of satiety...



Dilated Cardiomyopathy

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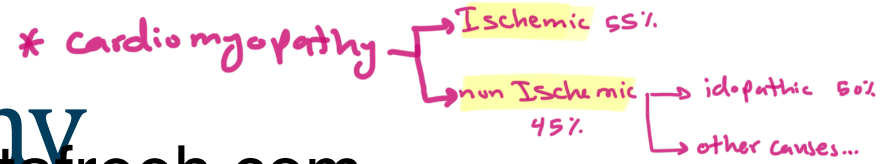
- Diagnosis: **Transthoracic echocardiography**
- **Treatment: Guideline directed medical treatment (GDMT)**
 - Beta-blockers
 - ACE inhibitors
 - Aldosterone antagonists
 - Neprilysin inhibitors
- Defibrillators ✓
- Bi-ventricular pacemakers ✓



Ejection fraction is reduced

Dilated Cardiomyopathy

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- Most common cause: ^{previous} myocardial infarction
 - Ischemic cardiomyopathy [fibrosis in muscle → ↓ contraction]
 - Myocytes replaced by scar tissue
 - Focal hypokinesis (e.g., anterior wall)
- Many causes of "non-ischemic" cardiomyopathy
 - About 50% idiopathic ✓
 - Many other causes ^{← viral} _{peripartum}
 - Global hypokinesis

* covid 19 may lead to [→] pericarditis
_→ myocarditis

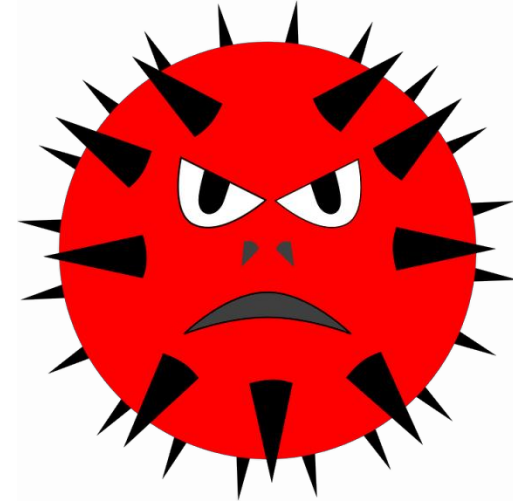
Nonischemic Cardiomyopathy

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Viral

- May follow upper respiratory infection
- Many associated viruses
 - **Coxsackie** mcc
 - **Influenza, adenovirus, others**
- No specific therapy for virus

* vn tt myo carditis lead to HF.



Pixabay/Public Domain

Nonischemic Cardiomyopathy

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Peripartum → risk for pulmonary edema + crepitation + rales → indication for peripartum cardiomyopathy.

3rd trimester

- Late in pregnancy or early post-pregnancy
- Exact cause unknown (likely multifactorial)
- Women often advised to avoid future pregnancy

* may due to volume overload or viral in pregnancy..

* pregnancy absolute contraindication in pulmonary HTN - severe VHD...

* pregnancy relative contraindication in cardiomyopathy...



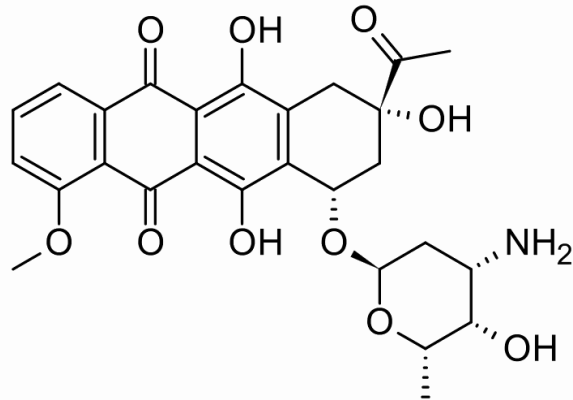
Øyvind Holmstad/Wikipedia

Nonischemic Cardiomyopathy

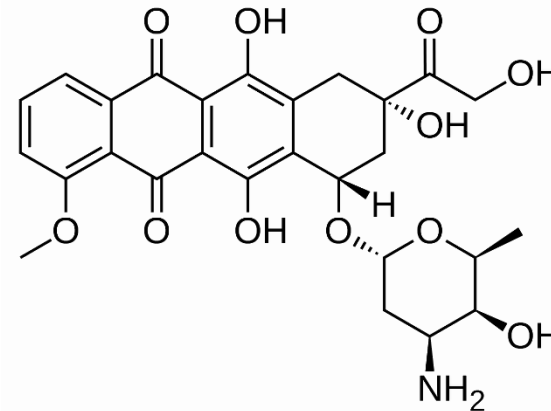
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Chemotherapy

- Usually after treatment with anthracyclines for lymphoma- leukemia- cancer pt..
 - Antitumor antibiotics
 - Doxorubicin and daunorubicin



Daunorubicin



Doxorubicin
(Adriamycin)

* thy myosine for contraction..

Nonischemic Cardiomyopathy

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Familial

- Mutations
 - Often sarcomere proteins
 - Beta-myosin heavy chain
 - Alpha-myosin heavy chain
 - Troponin
- Many autosomal dominant
- X-linked, autosomal recessive also described



Wikipedia/Public Domain

Nonischemic Cardiomyopathy

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Tachycardia-mediated

[recurrent tachycardia or AF]..

- Constant, rapid heart rate for weeks/months
- Leads to depression of LV systolic function
- **Reversible** with slower heart rate

غير معالجة السبب
tachycardia or AF



Nonischemic Cardiomyopathy

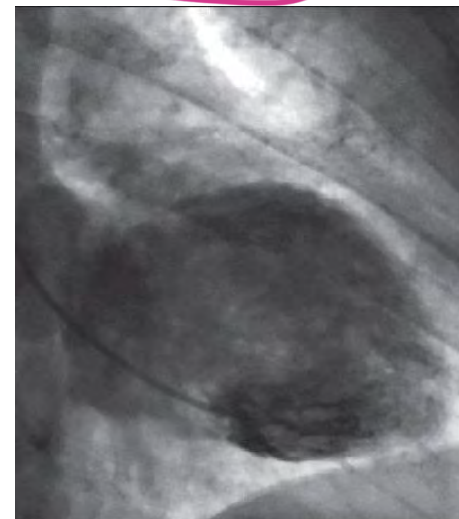
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Takotsubo/Apical ballooning

- **Stress-induced** cardiomyopathy
- Occurs after severe emotional distress and **high catecholamines**
- Markedly reduced LVEF
- Increase CK, MB, Troponin; EKG changes
- Looks like anterior MI (but no coronary disease)
- Usually **recovers 4-6 weeks**

* pt with chest pain + ↑ troponin + no coronary dz → takotsubo.
يعني بسبب stress شديده

Diastole



Systole



ضامة ممتدة

Alcohol

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- Chronic consumption can cause cardiomyopathy
- Believed to be due to toxic metabolites
- Can recover with cessation of alcohol



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Restrictive Cardiomyopathy

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lead to diastolic dys fx. low bi Atrium enlargement.

- abnormal sub. • Something “infiltrates” the myocardium
 - Amyloid protein (Amyloidosis) abnormal sub. enter the myocardium → myocardium infiltration...
- Heart cannot relax and fill ② Hemochromatosis...
- ③ Sarcoidosis..
- **SEVERE diastolic dysfunction**
 - Presents as diastolic heart failure
 - Treatment: diuretics
 - Also treat underlying cause

lead to dilated (more) restrictive

* when ↓ iron in hemochromatosis → the heart return normal...



MarkBuckawicki/Wikipedia

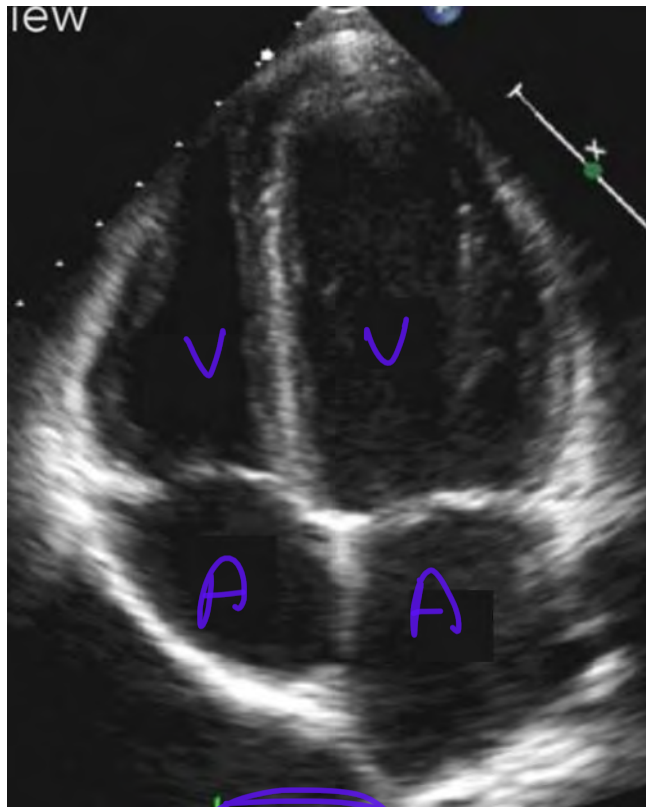
Restrictive Cardiomyopathy

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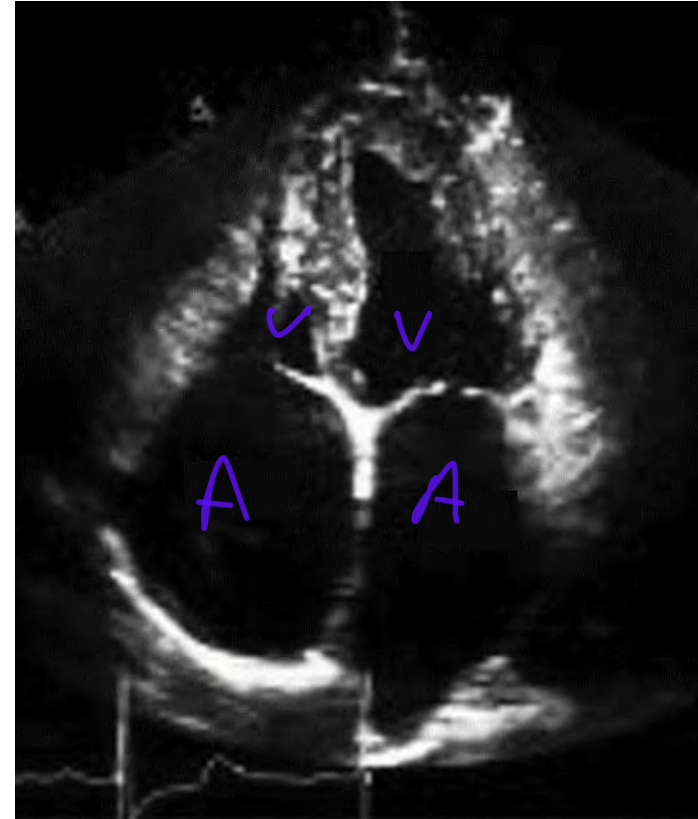
- **LVEF = normal**
- Restricted filling = $\uparrow$ atrial pressure
- **Dilated left and right atria**
- Classic imaging findings:
 - Normal left ventricular function/size ✓
 - Bi-atrial enlargement

Restrictive Cardiomyopathy

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Normal



Restrictive Cardiomyopathy
bi Atrial enlargement..

Restrictive Cardiomyopathy

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Clinical Features

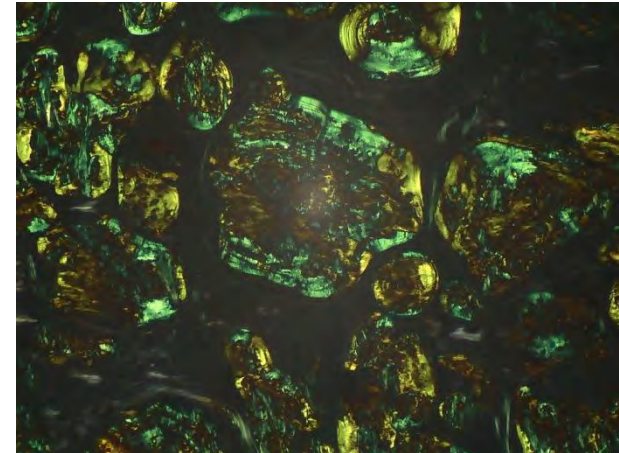
- **Dyspnea** ✓
- Prominent **right heart failure** ✓
 - Markedly elevated jugular venous pressure
 - Lower extremity edema
 - **Liver congestion**
 - May lead to cirrhosis

Restrictive Cardiomyopathy

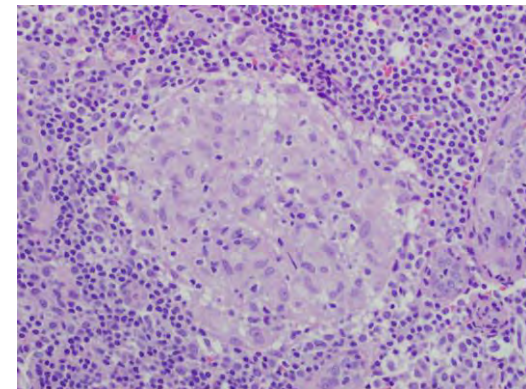
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Major Causes *any thing deposit in the heart*

- Amyloidosis ✓
- Sarcoidosis ✓
- Fabry disease *in children* (lysosomal storage disease) ✓
- Hemochromatosis (rare) ✓
- Post-radiation ✓
- Loeffler's syndrome (hypereosinophilic syndrome) ✓
- Endocardial fibroelastosis (babies) ✓



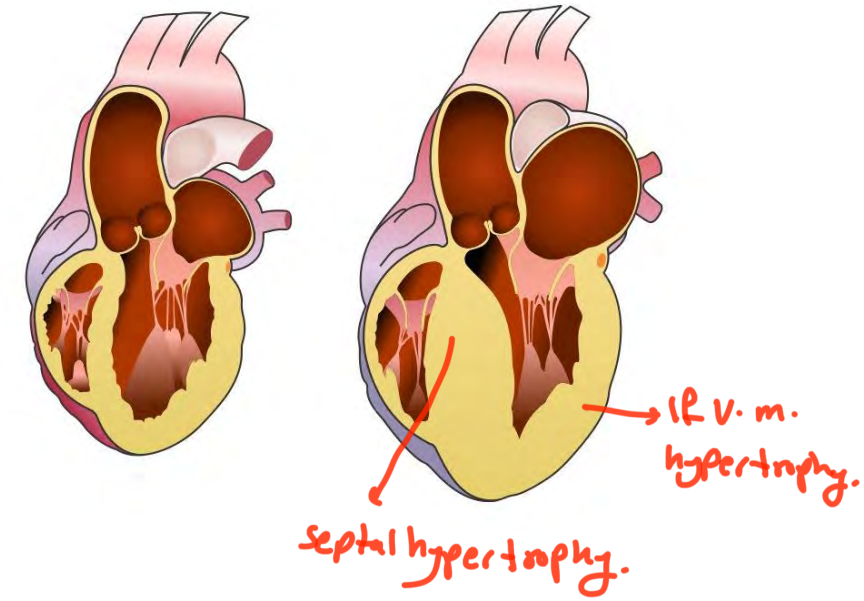
Ed Uthman, MD



Hypertrophic Cardiomyopathy [HCM] *→ mcc of cardiac arrest in young*

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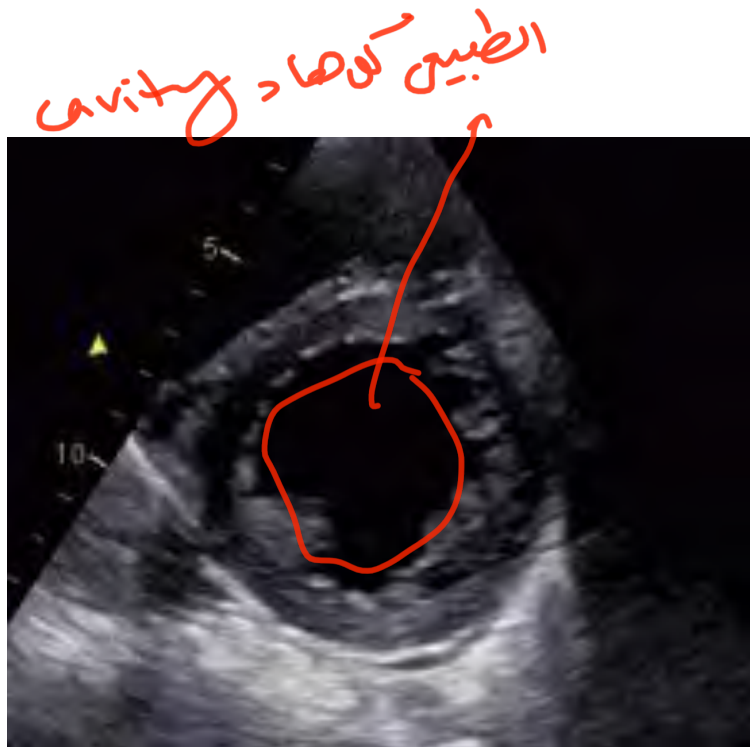
- Cardiomyopathy of **myocyte hypertrophy**
 - **Concentric** hypertrophy ✓
- Pathologic hypertrophy
 - Not due to stress
 - Absence of HTN or athlete's heart *← HOCM not HCM.*
- Diagnosis:
 - Echocardiography ✓
 - Genetic testing ✓
- LVEF normal with increased wall thickness
↳ especially in the septum...



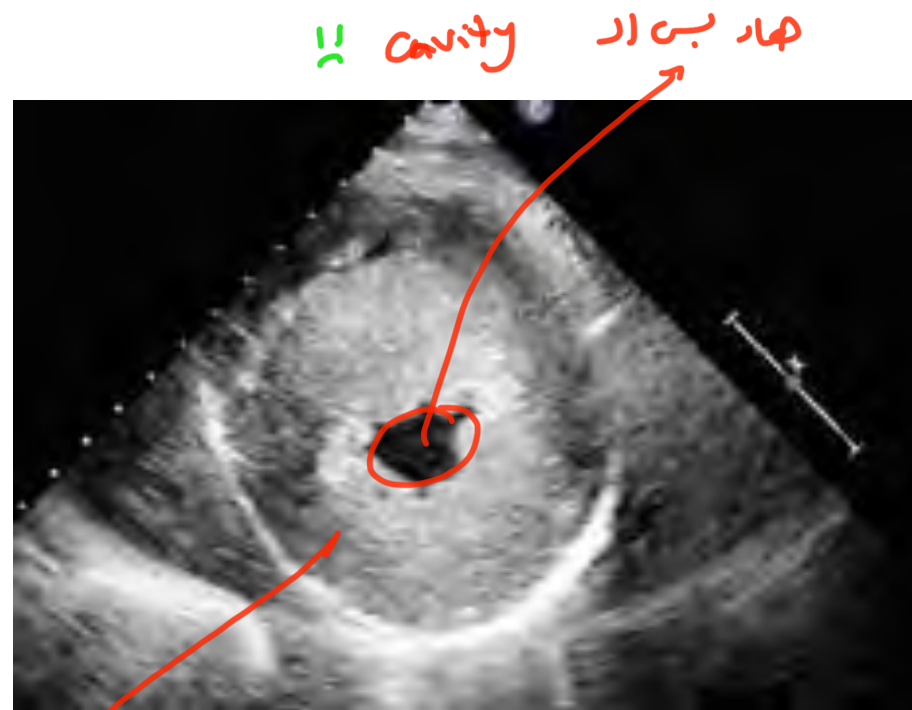
** 2nd mcc of cardiac arrest in young :- R + v. dysplasia...*

Hypertrophic Cardiomyopathy

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Normal



Hypertrophic Cardiomyopathy

حار بشار cavity

Handwritten red text below the image, with a red arrow pointing to the thickened septum.

Hypertrophic Cardiomyopathy

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- Genetic disorder caused by gene mutations
- About 50% cases familial (50% sporadic)
- **Autosomal dominant**
- Variable expression
 - Significant variation in severity of symptoms
 - Many variations in location/severity of hypertrophy



Wikipedia/Public Domain

Hypertrophic Cardiomyopathy

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Clinical Features

- Many patients asymptomatic
- Many symptoms from **LVOT obstruction**
 - Left ventricular outflow tract

* may present as first time as syncope or cardiac arrest or
mitral regurg...
↓
[systolic Ant. motion]

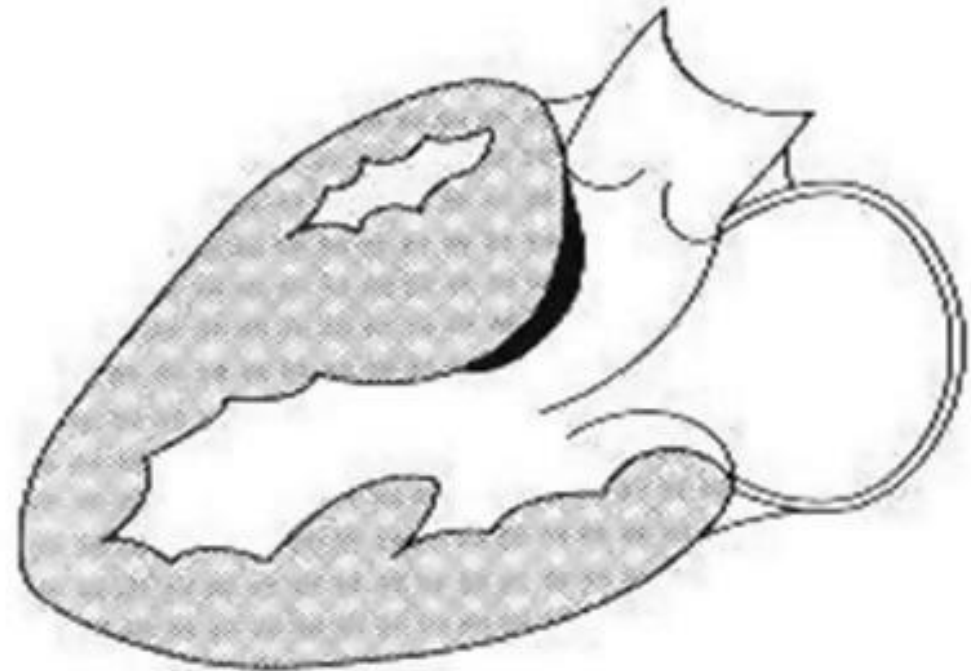


Hypertrophic Cardiomyopathy

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Clinical Features

- **Heart failure** ✓
 - Diastolic dysfunction
 - LVOT obstruction
- **Chest pain (angina)** ✓
 - Increased O₂ demand

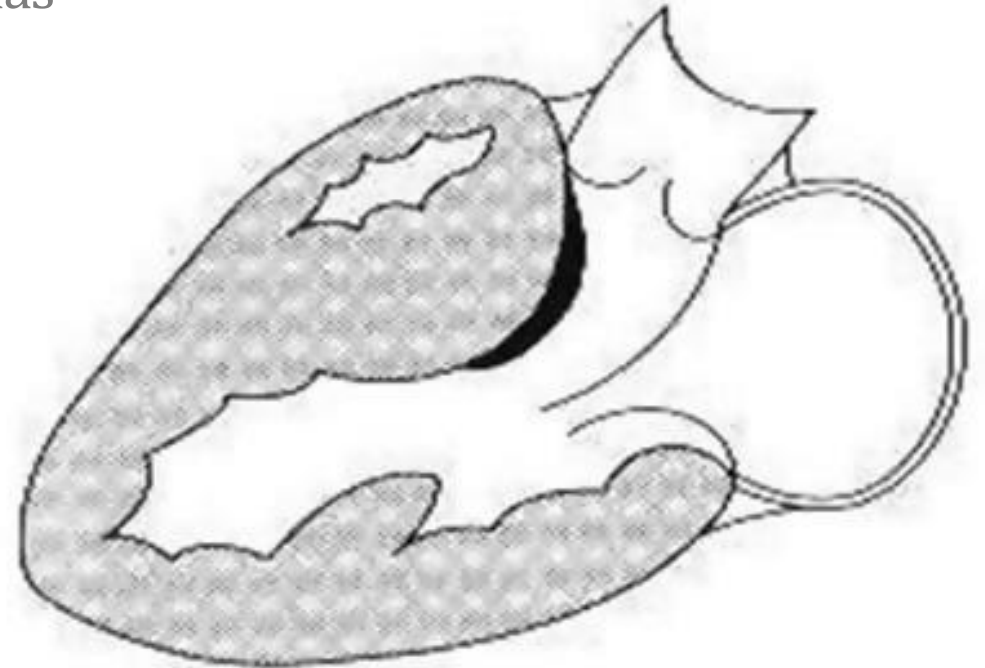


Hypertrophic Cardiomyopathy

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Clinical Features

- ✓ • **Sudden cardiac death**
 - Abnormal myocytes → ventricular arrhythmias
 - Most common cause SCD in young patients
- ✓ • **Syncope**
 - Arrhythmias may lead to syncope
 - LVOT obstruction
- ✓ • **Mitral regurgitation**
 - Systolic anterior motion (SAM)



Hypertrophic Cardiomyopathy

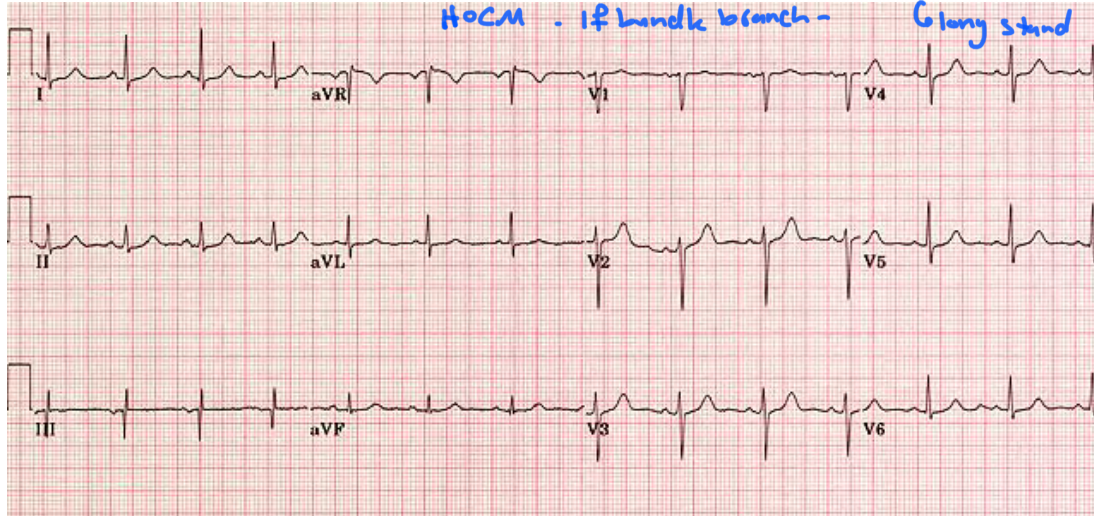
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Clinical Features

- EKG: Left ventricular hypertrophy (LVH)

muscle جدار بطين اذ.ف.ف

↳ due to ↑ P. in LV as Aortic stenosis - HTN .. etc
HOCM - 1st bundle branch - Glauy stand



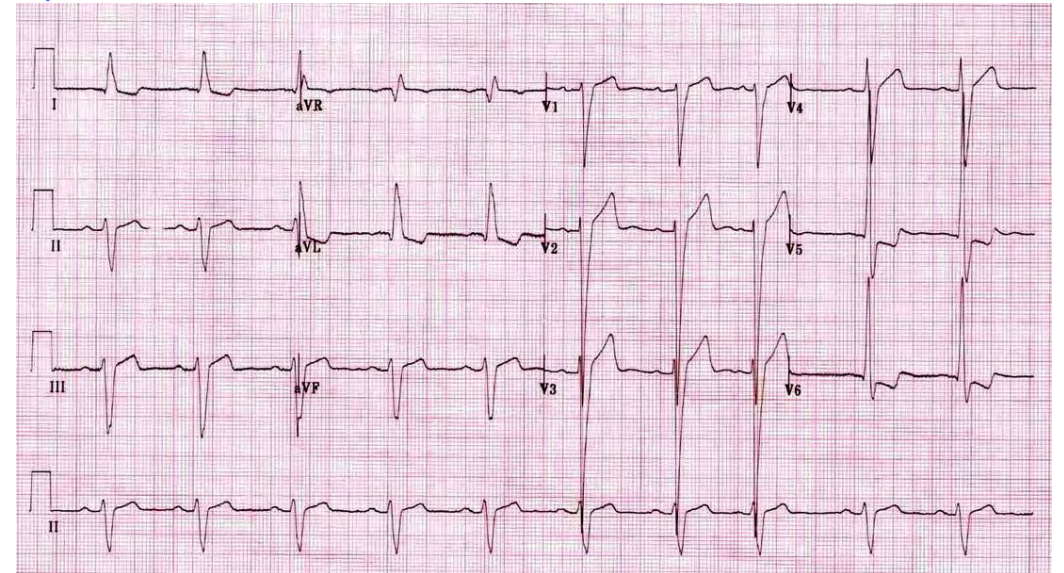
Normal

* If axial deviation:- lead I +ve, lead II -ve in QRS complex
* lead I, II → +ve QRS complex Normally.

large R wave in V5 - V6 due to LV. hypertrophy.

deep S wave in V1 - V2

← R wave in V5 - V6 / S wave in V1 - V2 في 5 مبرعات كبيرة



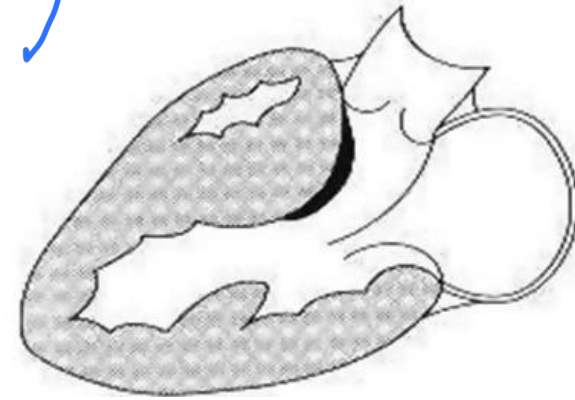
Hypertrophic Cardiomyopathy

Hypertrophic Cardiomyopathy

Clinical Features

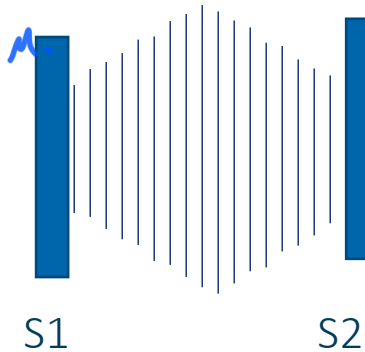
- ^{physio} ^{patho} **Systolic ejection murmur**
- Usually left lower sternal border
- Caused by outflow tract obstruction
- Sounds just like AS unless you do maneuvers
- Other associated abnormal heart sounds
 - S4
 - Holosystolic murmur of MR
 - Paradoxical split S2

* Aortic stenosis - HCM - Aortic sclerosis - pulmonary Stenosis → lead to ejection systolic murmur...



* Aortic stenosis murmur radiate to the neck...
So we hear it in R+ 2nd intercostal space..

* HCM murmur we hear it in LF 4th intercostal space..
↓
systolic M.



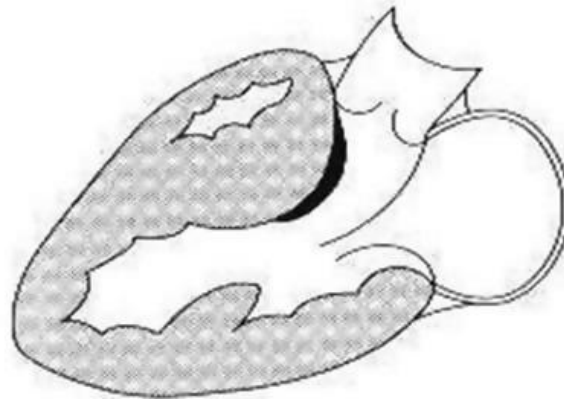
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Maneuvers

- For any HCM maneuver, think about size of LV

- ↑ preload • ↑ LV size → ↓ murmur ✓
- ↓ preload • ↓ LV size → ↑ murmur ✓



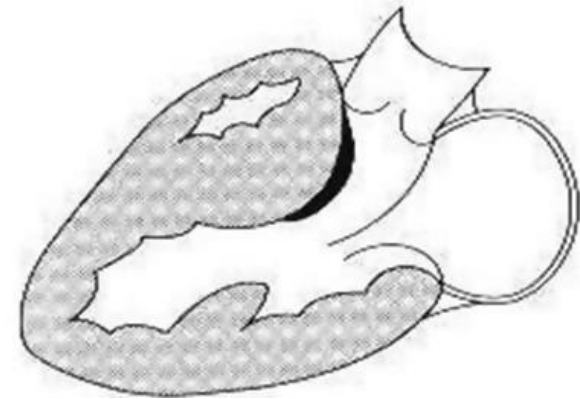
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Maneuvers

- **Valsalva** → ↓ preload → ↑ murmur of HOCM..
↓ الجهد - murmur
- Patient bears down as if having a bowel movement
- Or blows out against closed glottis
- Increase thoracic pressure → compression of veins → ↓ VR
- Less VR → Less preload → Smaller LV cavity
- Obstructing septum moves further into the outflow tract
- Murmur **INCREASES** in intensity

* the H of SVT :- blows out against closed glottis... [↓ preload]



Hypertrophic Cardiomyopathy

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Maneuvers

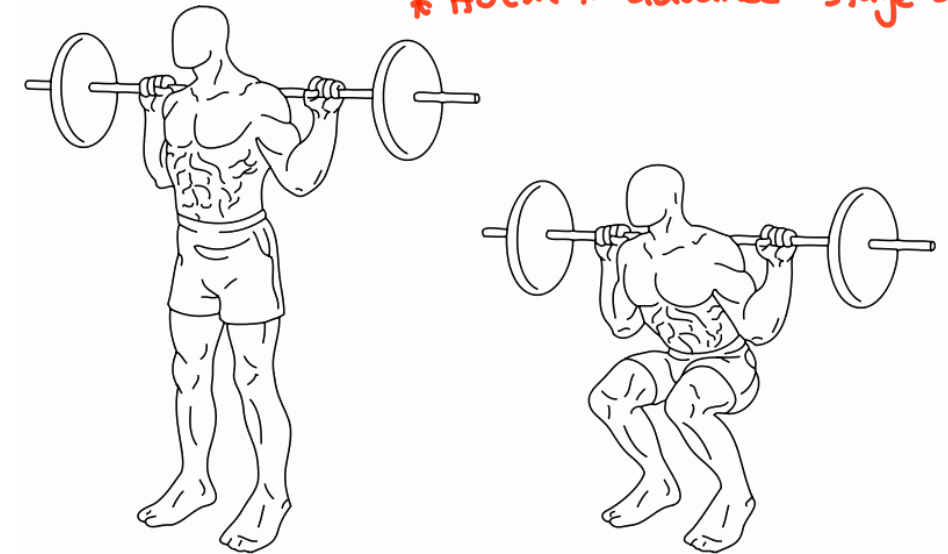
- **Squatting** $\uparrow$ pre load $\rightarrow \uparrow$ cavity $\rightarrow \downarrow$ murmur
 - Forces blood volume stored in legs to return to heart
 - Preload rises $\rightarrow$ size of LV increases $\rightarrow$ less obstruction
 - Murmur **DECREASES** in intensity
- **Raising the legs** $\uparrow$ pre load $\rightarrow \downarrow$ murmur...
 - Increases venous return
 - Murmur **DECREASES** in intensity
- **Standing** $\rightarrow \uparrow$ murmur of HOCM via $\downarrow$ pre load...
 - Opposite mechanism of leg raise
 - Murmur **INCREASES** in intensity

* HCM - MVP $\rightarrow \downarrow$ murmur of them by $\uparrow$ Pre load...
 جبر رفع رجل المريض $\rightarrow$ يرفع



[MVP: mitral valve prolapse]...

* HOCM :- advance stage of MVP.



Wikipedia

Aortic Stenosis

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- Both HCM and AS cause a systolic ejection murmur
- Opposite effects of maneuvers in aortic stenosis
 - Less preload → less flow → quieter AS murmur