## STRESS ECHOARDIOGRAPHY

**PROVIDED DATA**

1. *Ischemia*
2. *Pulmonary pressures during exercise stressor*
3. *Hemodynamic significance of valvular abnormalities*

**PROTOCOL**

| | |
|---|---|
| **EXERCISE STRESSOR** | • *Performed with supine or upright bicycle ergometry (allow continuous imaging) or with the treadmill protocol (requires poststress images within 90 seconds)* |
| **DOBUTAMINE** | • *Progressively infused (up to 40 µg/kg/min) to achieve 85% of age-predicted maximal heart rate +/- atropine if the target heart rate is not achieved* |

**LIMITATIONS**

1. *Reduced sensitivity in –*
   - *Baseline wall motion abnormalities*
   - *Baseline systolic dysfunction*
   - *Single-vessel disease*
2. *Sometimes suboptimal images but enhanced with microbubble study*

**TEST FINDINGS**

| AT REST | POST-STRESS | INTERPRETATION |
|---|---|---|
| *Normal* | *Normal* | *Normal* |
| *Normal* | *Wall motion abnormality* | *Stress-induced myocardial ischemia* |
| *Normal* | *LV dilation (global ↓ systolic LV function)* | *Small or distinct zone ischemia (possible balanced ischemia or multivessel CAD)* |
| *Regional wall motion abnormalities* | *Same regional wall motion abnormalities* | *Infarct or hibernating myocardium (chronic but potentially reversible ischemic dysfunction)* |

## MYOCARDIAL PERFUSION IMAGING (MPI)
## (NUCLEAR STRESS TESTING)

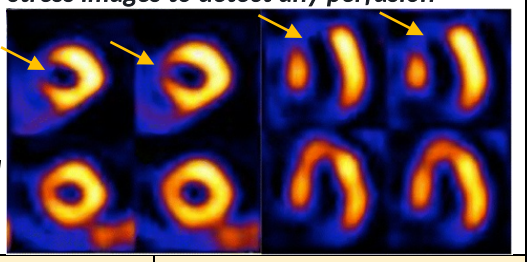
### ➢ SPECT (SINGLE-PHOTON EMISSION CT) MPI

**MECHANISM**

➡ *Detect myocardial ischemia based on the differences in myocardial blood flow with using injected radiotracer at rest & at peak exercise/vasodilation → myocardium will take radiotracer relative to blood flow → then rest images are compared with after stress images to detect any perfusion defects = flow-limiting CAD*

**SPECT MPI**

*Stress (upper rows) & Rest (lower rows) images with severe reversible perfusion defect the anteroseptal area of left ventricle*
*Source: Louise Nissen*



**RADIOTRACERS**

| | TECHNITIUM (Tc-99 m Sestamibi) | THALLIUM |
|---|---|---|
| **MECHANISM** | • *Technetium-based agents bind to the mitochondria → allowing for delayed imaging* | • *Thallium uptake requires active metabolism* |
| **ADVANTAGES** | • *Higher sensitivity & specificity (compared to Thallium) due to providing better images* | • *Assess myocardial viability* |

**PROVIDED DATA**

1. *Myocardial ischemia with localization*
2. *Gated images can provide left ventricular systolic function assessment*
3. *Quantify the extent & severity of disease (prognostic value)*
4. *Assessing high-risk features (worse prognosis) –*
   - *Multiple regions of hypoperfusion*
   - *Transient cavity dilatation/wall motion abnormalities*
   - *Reduction or lack of augmentation in poststress ejection fraction*

**TEST FINDINGS**

| AT REST | POST-STRESS | INTERPRETATION |
|---|---|---|
| *Normal* | *Normal* | *Normal* |
| *Normal* | *Perfusion defect* | *Stress-induced myocardial ischemia* |
| *Normal* | *LV dilation (global ↓ systolic LV function)* | *Small or distinct zone ischemia (possible balanced ischemia or multivessel CAD)* |
| *Perfusion defect* | *Same perfusion defect* | *Infarct or hibernating myocardium* |

# DIAGNOSTIC TESTING FOR CARDIAC ARRYTHMIAS

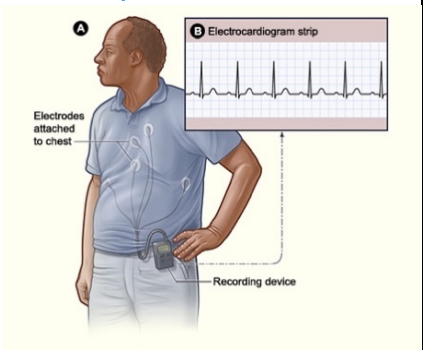
## AMBULATORY ECG MONTORING

**INDICATIONS**

1. *Diagnosing & monitoring arrhythmias –*
   - *A-fib*
   - *Atrial flutter*
   - *SVT*
   - *VT*
2. *Assess for A-fib in cryptogenic stroke*
3. *Episodic lightheadedness*
4. *Palpitations*
5. *Syncope*

**MODALITIES**

- *3 types based on the length of the recording time of heart rhythms (hours to years) so → the device choice will be based on the patient's frequency of symptoms*
- *Not for ischemic interpretation as ECGs waveform details may not render accurately*




**Holter Monitor**
*Source: NIH*

| CONTINUOUS AMBULATORY ECG (HOLTER) MONITORS | | EVENT MONITORS | LOOP RECORDERS |
|---|---|---|---|
| **TECHNIQUE** | | | |
| • *Holter monitor is attached to leads placed on the chest & continuously records the ECG over 24–48 hours up to 30 days for later analysis with correlation with the patient's diary of arrythmias symptoms*<br>• *Central monitoring service could be used for rapid analysis* | | • *Worn by patients at home to record infrequent symptomatic arrythmias that last >1–2 minutes over 2–4 weeks for later analysis along patient's long based on button activation*<br>• *Central monitoring service could be used for rapid analysis* | • *Wearable device (for 2–4 weeks) or implantable subcutaneous chip (for 1–3 years) with wireless access*<br>• *Patients activate the device (wearable edition) or heart-rate/rhythm activated (implantable edition)*<br>• *Onset of arrythmias is recorded* |
| **INDICATIONS** | | | |
| **SHORT-TERM HOLTER** | • *Asymptomatic or symptomatic for 24 & 48-h monitors* | *Used in infrequent symptoms (once or twice monthly)* | • *For symptomatic arrythmias that are even less frequent (once every 6 months or once per year)*<br>• *Syncope* |
| **LONG-TERM HOLTER** | • *Infrequent asymptomatic or symptomatic up to 30 days monitoring* | | |
| **LIMITATIONS** | | | |
| **SHORT-TERM HOLTER** | • *Not hopeful if infrequent arrythmias* | 1. *Symptomatic arrhythmias must be long enough for patient to activate the device*<br>2. *Arrhythmia onset is not recorded*<br>3. *Not useful for syncope or extremely brief arrhythmias* | • *ECG leads limit patient's activity (wearable edition)*<br>• *Minor invasive procedure (implantable edition)* |
| **LONG-TERM HOLTER** | • *Long use of adhesive leads attachment*<br>• *Limited in non-clinical significant arrythmias* | | |

## ELECTROPHYSIOLOGIC STUDY (EP)

| | |
|---|---|
| **INDICATIONS** | 1. *Inducing arrythmias to identify & clarify the mechanism of arrhythmia*<br>2. *Potential correction of arrythmias (catheter ablation)*<br>3. *Implantation of cardiac electronic device (pacemaker or cardioverter-defibrillator)* |
| **LIMITATIONS** | 1. *Risk of invasive procedure*<br>2. *Some arrhythmias may not be inducible (especially if the patient is sedated)* |

# 12- LEAD ELECTROCARDIOGRAM (ECG)

## ECG BASICS

### CONDUCTION PATHWAY

| | |
|---|---|
| **ATRIAL CONDUCTION** | ➡ *Mostly myocyte-to-myocyte conduction = slow conduction* |
| **VENTRICULAR CONDUCTION** | • *Specialized conduction system (His bundle & its left and right bundle branches) – much faster → larger mass in the ventricles is depolarized more quickly than the atria →*<br>1. *Left bundle branch → divides into anterior & posterior fascicles then → smaller branches (Purkinje fibers)*<br>2. *Right bundle branch (no fascicle branches) → transitions directly into Purkinje fiber* |

### PACEMAKER FIRING RATES

*All heart tissue is capable of depolarizing automatically → conduct their own pacemaker with different depolarization rates*

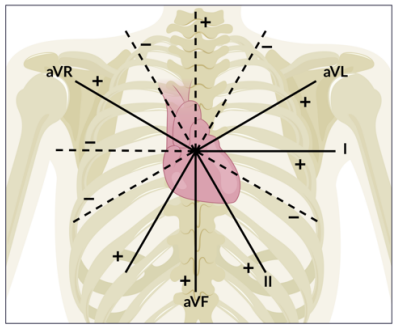
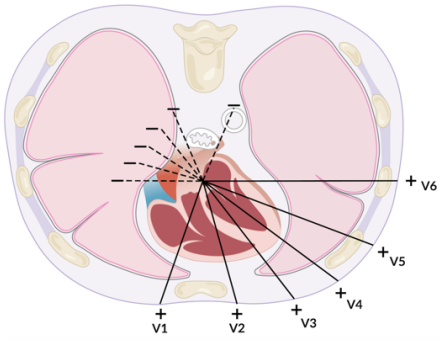
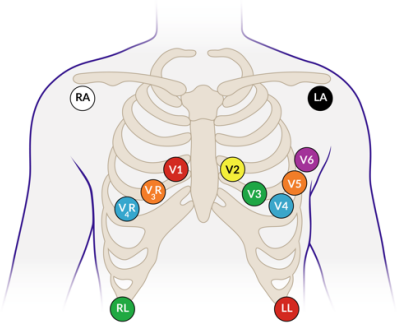
| | |
|---|---|
| **SINOATRIAL (SA) NODE** | • 60–100 *bpm (suppresses all other foci in the heart)* |
| **ATRIAL PACEMAKER (NON-SA-NODE)** | • 60–80 *bpm* |
| **AV JUNCTION PACEMAKER (HIS BUNDLE – NOT AV NODE)** | • 40–60 *bpm* |
| **VENTRICULAR PACEMAKER (IDIOVENTRICULAR RHYTHM)** | • 20–40 *bpm* |

### DIRECTION OF DEPOLARIZATION & REPOLARIZATION WAVE

| | |
|---|---|
| **POSITIVE LEAD TRACING** | • *If the wave of depolarization spreads toward the positive pole of that lead → positive deflection (P wave & QRS complex)*<br>• *If the wave of repolarization spreads away from the positive pole of that lead → positive deflection (T wave)* |
| **NEGATIVE LEAD TRACING** | • *If the wave of depolarization spreads away from the positive pole*<br>• *If the wave of repolarization spreads toward the positive pole of that lead* |
| **ZERO TRACING** | • *If the wave is maximal at 90° angle to the pole* |

### ECG LEADS TYPES

**(CREATING 3-DEIMENSTIONAL REFLECTION OF THE CARDIAC ELECTRICAL ACTIVITY)**

| FRONTAL PLANE (LIMB LEADS) | HORIZONTAL PLANE (PRECORDIAL LEADS) | RIGHT CARDIAC SIDE (V3R & V4R) |
|---|---|---|
| • *I – II – III & leads aVR – aVL – aVF (mathematically derived from I/II/III)*<br>• *For inferior-superior-left-right pathology (ST variations/Q waves in II – III & aVF = inferior area pathology)* | • *V1 – V2 – V3 – V4 – V5 – V6*<br>• *For anterior-posterior-lateral pathology* | • *Placed as the same as V3 & V4 on the right side of the chest*<br>• *Detect right-ventricular ischemia* |
|  |  |  |

| | |
|---|---|
| **ANTERIOR LEADS** | ➡ *V2 – V3 – V4* |
| **LEFT LATERAL LEADS** | ➡ *I – aVL – V5 – V6* |
| **INFERIOR LEADS** | ➡ *II – III – aVF* |
| **RIGHT VENTRICULAR LEADS** | ➡ *aVR – V1* |

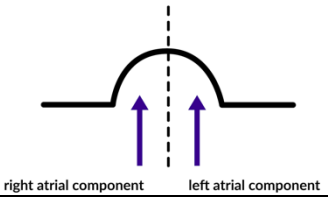
# ECG WAVEFORMS & SEGMENTS

## P WAVE

### NORMAL P WAVE

**PHYSIOLOGY**

➡ *Due to atrial depolarization*




**CRITERIA**

- *Height <2.5 mm*
- *Duration <120 ms (3 small squares)*
- *Normal axis 0° & +75°*

### P WAVE AXIS

**NORMAL**

➡ *Discussed under cardiac axis*

**ECTOPIC FOCUS**

- ***P-wave axis is superior** (from bottom to top) & **rightward** (opposite of the normal P-wave axis) causing –*

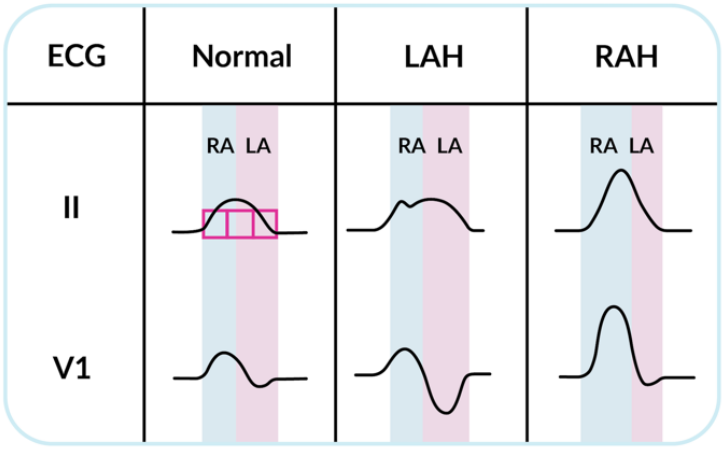
1. *Negative P waves on lead II*
2. *Inverted P wave in inferior leads*

**DEVIATION**

➡ *In chronic obstructive pulmonary disease → hyperexpanded lungs that causing more vertical cardiac position → right axis deviation of P wave (+90° P-wave axis)*

### ATRIAL ENLARGMENT

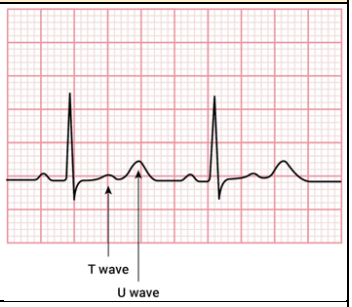
| LEAD II | LEAD V1 |
|---|---|
| **NORMAL P WAVE** | |
| *Positive P wave*<br>*As atrial depolarization traverses from the patient's right to left atria →*<br>• *Left/initial side of the P wave = right atrium*<br>• *Right/terminal side of the P wave = left atrium*<br>• *Mid-P wave = both* | *Positive/Biphasic P wave*<br>*Initially positive → negative*<br>*Due to depolarization wave through atria →*<br>• *Initially toward V1 in RA (initial part of P wave)*<br>• *Away from V1 in the LA (latter part of P wave)* |
| **RIGHT ATRIAL ENLARGEMENT (RAH)** | |
| • *Right atrial (initial) portion of the P wave is widened then → overlaps with left atrial (latter) portion of P wave*<br>• *P wave width stays normal <120 ms but ↑ P-wave amplitude in II & V1 (positive portion) → peaked* | |
| **LEFT ATRIAL ENLARGEMENT (LAH)** | |
| ➡ *Enlarged latter part of P wave → wide P wave with shortened or absent PR interval <120 ms + Notched P wave (usually in II) with interpeak distance of >40 ms (the most specific ECG finding)* | ➡ *Enlarged latter part of P wave → wide P wave with shortened or absent PR interval <120 ms + Notched P wave (usually in II) with interpeak distance of >40 ms (the most specific ECG finding)* |



## U WAVE

**MORPHOLOGY**

- *Not common finding & occurs just after T wave*
- *Usually small & best seen in V2–V3 → <1 mm rounded deflection in the same direction as T wave*



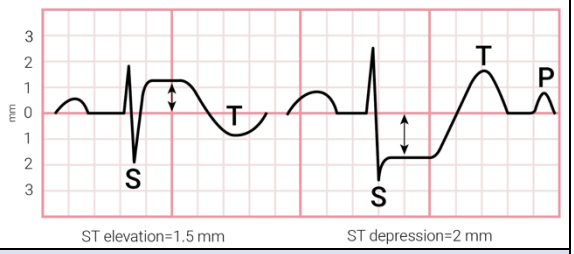

**CAUSES**

| | | |
|---|---|---|
| **PROMINENT U WAVE (↑ RISK OF POLYMORPHIC VT)** | • *Hypokalemia*<br>• *Amiodarone* | • *Bradycardia*<br>• *Digoxin* |
| **INVERTED U WAVE (SIGNIFICANT FINDING EVEN WITH NORMAL ECG)** | • *Ischemia & anterior/inferior MIs*<br>• *Hypertension* | • *Mitral or tricuspid valve disease*<br>• *RVH* |

## ST SEGMENT DEVIATION

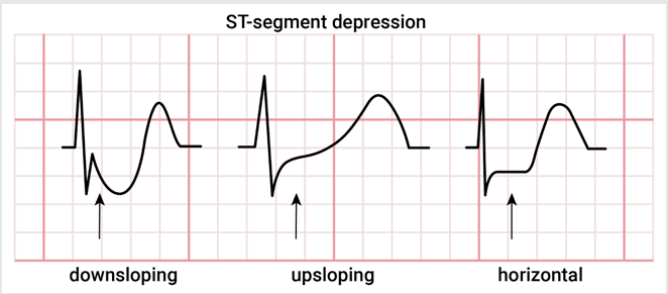
**ECG FINDINGS**

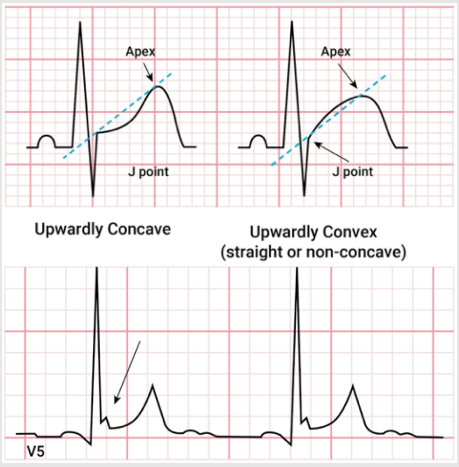
- *Evaluation of ST-segment deviation (elevation or depression – measured in millimeters) in relation to isoelectric line = preceding TP segment (end of the T wave to the beginning of the P wave)*
- *If the isoelectric line is not clear (artifact or labored breathing) → onset of Q wave is used*
- *The more deviated the ST segment (whether ST elevation or ST depression) → the more severe the ischemia & myocardial infarction*
- *The larger the number of leads showing ST-segment deviation (whether ST elevation or ST depression) → the larger the area of involved myocardial tissue*




### MORPHOLOGY

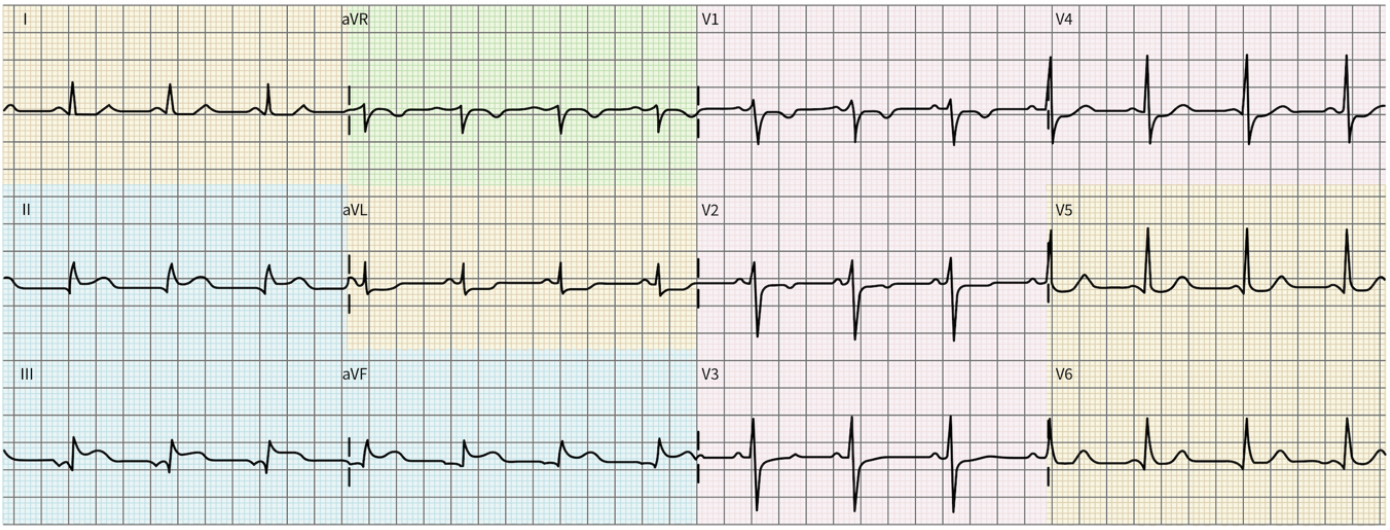
| ST – DEPRESSION | | | ST – ELEVATION | |
|---|---|---|---|---|
| **DOWNSLOPING** | **HORIZONTAL** | **UPSLOPING** | **CONCAVE UPWARD** | **CONVEX UPWARD** |
| *Indicative of ischemia or infarction* | | *Less specific for ischemia* | • *Pericarditis*<br>• *Early repolarization (normal variant)* | • *ST-elevation MI*<br>• *Coronary vasospasm* |




**Early Repolarization**

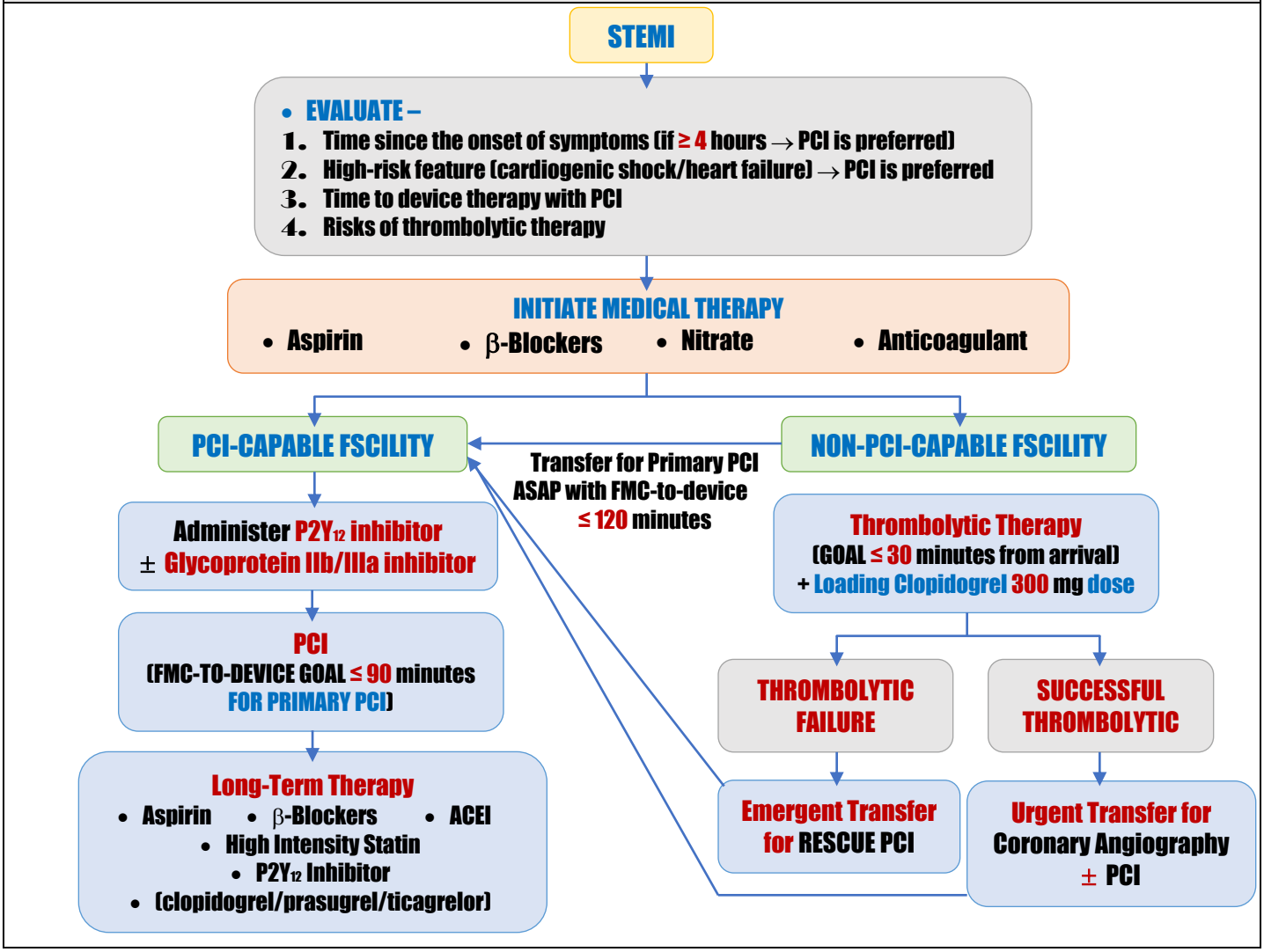
# STEMI LOCALIZATION

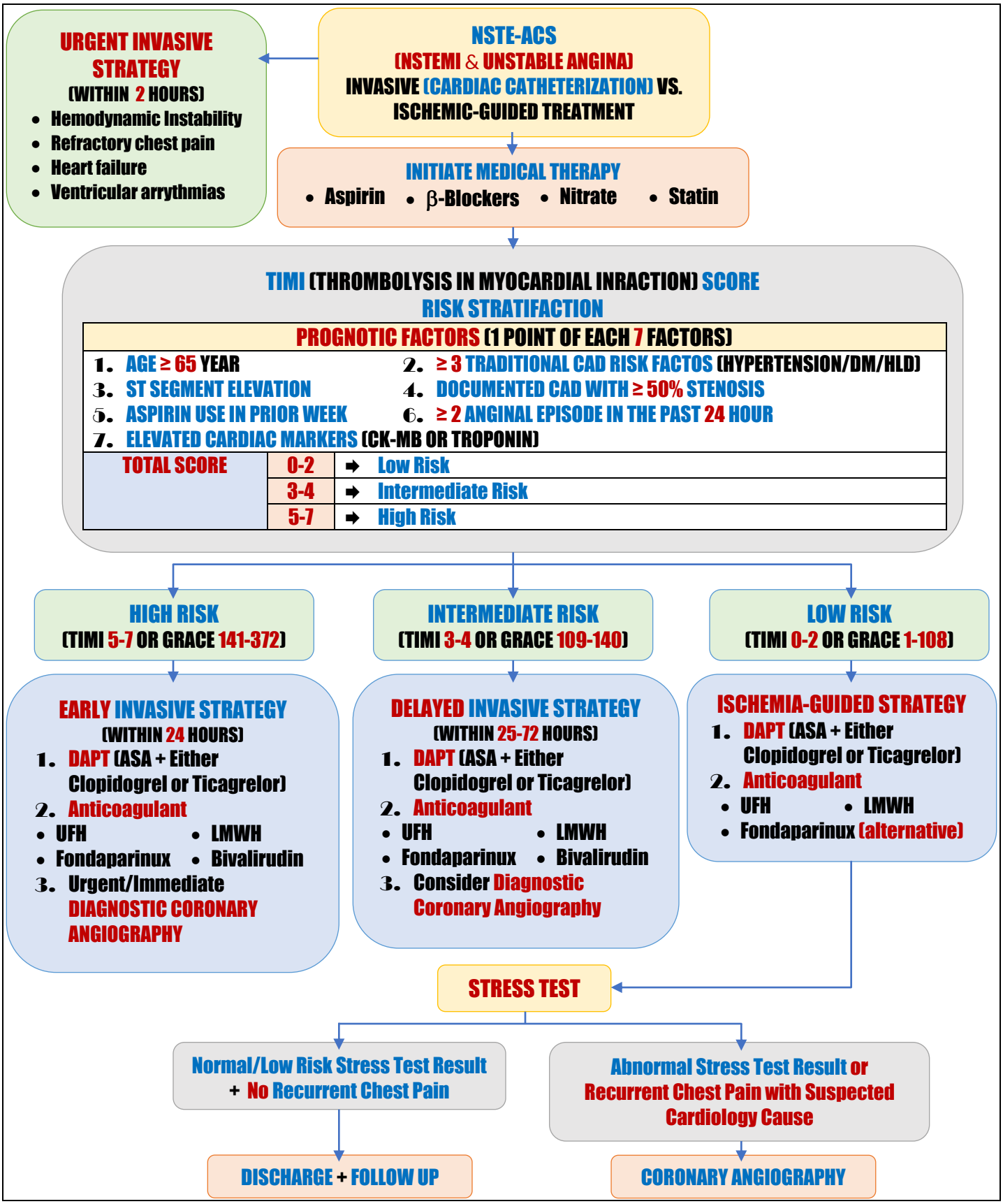



| LOCATION | LEADS | OCCLUDED ARTERIES |
|---|---|---|
| **NEW STEMI** | | |
| **SEPTAL MI** | *ST elevation in V1–V2* | **Proximal LAD** |
| **ANTERIOR MI** | *ST elevation in V3–V4* | **LAD** |
| **ANTEROSEPTAL MI** | *ST elevation in V1–V4* | |
| **LATERAL MI** | *ST elevation in I – aVL – &/or V5–V6* | **Circumflex artery** |
| **HIGH LATERAL MI** | *ST elevation in I and aVL* | |
| **ANTEROLATERAL MI** | *ST elevation in I – aVL – V2–V6* | |
| **INFERIOR MI** | *ST elevation in II – III – aVF* | **90% RCA**<br>**10% Circumflex artery** |
| **INFEROLATERAL MI** | *ST elevation in II – III – aVF +*<br>*I – aVL – &/or V5–V6* | |
| **APICAL MI** | *ST elevation in II – III – aVL*<br>*& any of V1–V4* | **Distal LAD**<br>**Circumflex artery** *or*<br>**RCA** |
| **POSTERIOR MI** | *Tall R in V1–V2 (mirror image of Q wave) with R:S ratio >1 in V1 or V2*<br>*ST depression in V1–V2 (mirror image of ST elevation)* | **RCA** *or*<br>**Circumflex artery** |
| **INFEROPOSTERIOR MI** | *ST elevation in II – III – aVF + tall R in V1–V2 (mirror image of Q wave)*<br>*ST depression in V1–V2 (mirror image of ST elevation)* | |
| **RV MI** | • *ST elevation in the right precordial leads – V1*<br>• *ST elevation in V4R to V6R is fairly sensitive & specific for RV infarction*<br>• *Diagnostic of RV infarction → ST elevation is greater in V4R than in V1* | |
| **OLD STEMI** | | |
| **OLD ANTERIOR MI** | ➡ *Q wave in V3–V4* | |
| **OLD INFERIOR MI** | ➡ *Q wave in II – III – aVF* | |
| **OLD POSTERIOR MI** | ➡ *Tall R in V1–V2 (mirror image of Q wave)* | |
| **OLD INFEROPOSTERIOR MI** | ➡ *Q wave in II – III – aVF + Tall R in V1–V2 (mirror image of Q wave) with R:S ratio >1 in V1 or V2* | |

| | | |
|---|---|---|
| **CHEST PAIN RELIEF** | **NITROGLYCERIN (TABLETS/SPRAY)** | ➡ *Administer sublingual up to 3 doses at 3–5 minute intervals*<br>➡ *IV NTG for persistent angina or hypertension*<br>**CONTRAINDICATION OF NITROGLYCERIN DUE TO RISK OF SEVERE HYPOTENSION –**<br>1. *Systolic BP <90 mmHg or 30 mmHg below baseline*<br>2. *Right ventricular infarction*<br>3. *Patient is on phosphodiesterase-5 inhibitor –*<br>➡ *Sildenafil – Vardenafil within 24 hours*<br>➡ *Tadalafil within 48 hours* |
| | **PAIN DRUGS** | ➡ *IV Morphine should be avoided if possible and reserved for high level of pain due to ↑ risk of death (showed in CRUSADE Initiative retrospective study)*<br>➡ *Stop all NSAIDs (except ASA) → ↑ risk of adverse cardiovascular events* |
| **HIGH RISK FEATURES** | • *Recognize & manage ventricular arrhythmias*<br>• *Recognize High-risk features –*<br>1. *Ongoing chest pain for >20 minutes*<br>2. *Signs of heart failure*<br>3. *Elevated cardiac enzymes (cTnI or cTnT)*<br>4. *Hypotension*<br>5. *New ST changes (any one of the following) –*<br>• *ST depression ≥0.5 mm*<br>• *ST elevation ≥ 2 mm in V1/ V2/V3*<br>• *ST elevation ≥ 1 mm in leads other than V1/V2/V3* | |

## MANAGEMENT APPROACH OF ACS




URGENT INVASIVE STRATEGY
(WITHIN 2 HOURS)
• Hemodynamic Instability
• Refractory chest pain
• Heart failure
• Ventricular arrythmias
NSTE-ACS
(NSTEMI & UNSTABLE ANGINA)
INVASIVE (CARDIAC CATHETERIZATION) VS.
ISCHEMIC-GUIDED TREATMENT
INITIATE MEDICAL THERAPY
• Aspirin • β-Blockers • Nitrate • Statin
TIMI (THROMBOLYSIS IN MYOCARDIAL INRACTION) SCORE
RISK STRATIFACTION
PROGNOTIC FACTORS (1 POINT OF EACH 7 FACTORS)
1. AGE ≥ 65 YEAR
2. ≥ 3 TRADITIONAL CAD RISK FACTOS (HYPERTENSION/DM/HLD)
3. ST SEGMENT ELEVATION
4. DOCUMENTED CAD WITH ≥ 50% STENOSIS
5. ASPIRIN USE IN PRIOR WEEK
6. ≥ 2 ANGINAL EPISODE IN THE PAST 24 HOUR
7. ELEVATED CARDIAC MARKERS (CK-MB OR TROPONIN)
TOTAL SCORE
0-2 ➡ Low Risk
3-4 ➡ Intermediate Risk
5-7 ➡ High Risk
HIGH RISK
(TIMI 5-7 OR GRACE 141-372)
INTERMEDIATE RISK
(TIMI 3-4 OR GRACE 109-140)
LOW RISK
(TIMI 0-2 OR GRACE 1-108)
EARLY INVASIVE STRATEGY
(WITHIN 24 HOURS)
1. DAPT (ASA + Either Clopidogrel or Ticagrelor)
2. Anticoagulant
• UFH • LMWH
• Fondaparinux • Bivalirudin
3. Urgent/Immediate DIAGNOSTIC CORONARY ANGIOGRAPHY
DELAYED INVASIVE STRATEGY
(WITHIN 25-72 HOURS)
1. DAPT (ASA + Either Clopidogrel or Ticagrelor)
2. Anticoagulant
• UFH • LMWH
• Fondaparinux • Bivalirudin
3. Consider Diagnostic Coronary Angiography
ISCHEMIA-GUIDED STRATEGY
1. DAPT (ASA + Either Clopidogrel or Ticagrelor)
2. Anticoagulant
• UFH • LMWH
• Fondaparinux (alternative)
STRESS TEST
Normal/Low Risk Stress Test Result + No Recurrent Chest Pain
Abnormal Stress Test Result or Recurrent Chest Pain with Suspected Cardiology Cause
DISCHARGE + FOLLOW UP
CORONARY ANGIOGRAPHY

# DYSLIPIDEMIA

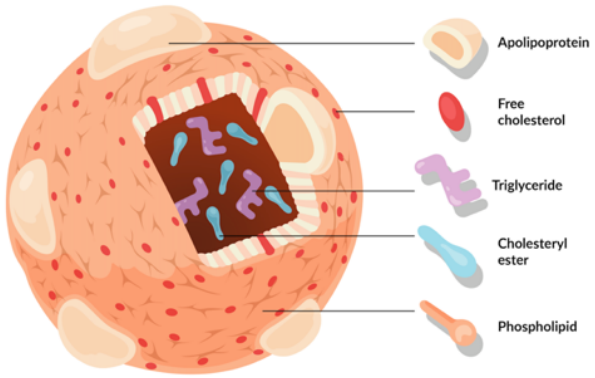
## CAD PREDICTORS

| | | |
|---|---|---|
| **HIGH LDL** | • *The single most important test because lipid-lowering therapy recommendations are based mostly on LDL level*<br>➡ *Calculated using –*<br>*LDL = total cholesterol – HDL – (triglycerides/5) Or total cholesterol – HDL – VLDL*<br>➡ *If lipid panel is obtained in nonfasting state (especially after fatty meal) → triglycerides levels will be elevated & calculated LDL will show underestimation of the true level* | |
| **LOW HDL** | ➡ *No associated link between low HDL & ASCVD has been confirmed (refer to HDL section)* | |
| **HIGH TRIGLYCERIDES** | **PATHOLOGY** | ➡ *Indicates –*<br>1. *Elevated chylomicrons*<br>2. *Elevated very-low-density lipoprotein*<br>3. *Elevated intermediate-density lipoprotein* |
| | **CVD RISK** | ➡ *↑ triglyceride levels (>150 mg/dL) are independently linked with ↑ ASCVD risk (but unclear if reducing triglyceride levels will reduce CVD risk)* |
| | **CAUSES** | 1. *Primary – familial hypertriglyceridemia (screen for FH if TG ≥500 mg/dL with no apparent cause)*<br>2. *Secondary –*<br>• *Diabetes mellitus*<br>• *Hypothyroidism*<br>• *Medications (steroid/protease inhibitors/estrogens)*<br>• *Concentrated sugar intake*<br>• *Excessive alcohol intake*<br>• *Obesity* |
| | **PRESENTATION** | ➡ *TG levels >500 – 1000 mg/dL causes acute pancreatitis (screen for TG in selected pancreatitis cases with no apparent causes as biliary/acholic abuse)* |

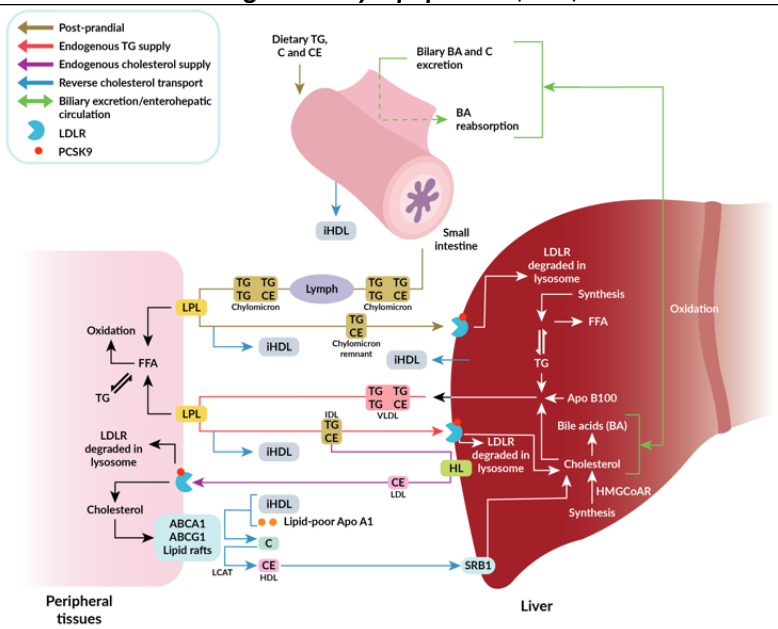
## LIPOPROTEINS PATHWAY

### LIPOPROTEINS

| | |
|---|---|
| **STRUCTURE** | ➡ *Contain hydrophobic core (triglycerides and/or cholesterol) – surrounded by hydrophilic phospholipid outer layer that facilitates transport through the serum* |
| **TYPES** | • *Chylomicrons*<br>• *Intermediate-Density Lipoprotein (IDL)*<br>• *Lipoprotein(a) [LP(a)]*<br>• *Very-Low-Density Lipoprotein (VLDL)*<br>• *Low-Density Lipoprotein (LDL)*<br>• *High-Density Lipoprotein (HDL)* |




**Lipoprotein Structure & Hepatic Pathway**



## MANAGEMENT OF DYSLIPIDEMIA IN SPECIAL POPULATIONS

### > 75 YEAR OLD

**1RY PREVENTION**

- ➡ *Moderate-intensity statin can be initiated (after discussion of the potential benefits with the patient) in no known ASCVD & LDL level of 70 – 189 mg/dL (the benefit in this population is less robust than in other higher-risk groups)*
- ➡ *If the decision is still uncertain to initiate statin therapy → use CAC score (no statin if CAC 0)*
- ➡ *It is reasonable not to start statin therapy in case of –*
  1. *Functional decline*
  2. *Frailty*
  3. *Multiple comorbid conditions*
  4. *Reduced life expectancy*

**2RY PREVENTION**

- ➡ *It is reasonable to continue statins if already tolerating therapy (moderate-intensity therapy is is preferable to high-intensity therapy in population)*

### ESRD ON HD

**GUIDELINES**

- *Not recommended to start statin in adults on dialysis for end-stage kidney disease*
- *It is reasonable to continue statin if already on it*

### WOMEN

**HIGH ASCVD RISK**

1. *Premature menopause*
2. *History of pregnancy-associated disorders (hypertension – preeclampsia – gestational diabetes)*

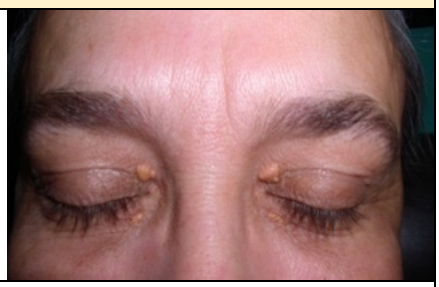
**PREGNANCY**

- ➡ *Statin contraindicated in pregnant patients*
- ➡ *Counseling women of childbearing age to use reliable contraception while on statin & stop statin therapy 1 – 2 months before pregnancy is attempted*

### XANTHOMAS

**PATHOLOGY**

- ➡ *Lipid deposits as yellow/orange/reddish/yellow-brown nodules/papules/plaques in the connective tissue of the skin/tendons/fasciae*
- ➡ *Linked to primary & secondary hyperlipidemias (the type of xanthoma closely correlates with the type of increased lipoprotein)*



Xanthelasma
Source: Klaus D. Peter

**TYPES**

1. *Eruptive*
2. *Plane (include xanthelasma)*
3. *Tuberous*
4. *Tendinous*
5. *Verruciform*

**CAUSES**

| | |
|---|---|
| ERUPTIVE | ➡ *Pathognomic of hypertriglyceridemia with high incidence of diabetes* |
| PLANE | ➡ *Linked to familial dyslipidemias & hematologic cancers* |
| XANTHELASMA | • *Occur without hyperlipidemia especially in older patients*<br>• *Linked with familial dyslipidemias in younger person*<br>• *Primary biliary cholangitis classic presentation (associated with marked hypercholesterolemia)* |
| TENDINOUS | ➡ *Linked to familial hypercholesterolemia* |

**PRESENTATION**

| | |
|---|---|
| ERUPTIVE | • *Cluster of small erythematous papules on the extensor surfaces of –*<br>1. *Arms* 2. *Legs* 3. *Buttocks* |
| PLANE | • *Yellow-to-red plaques in the skin folds of the neck & trunk* |
| XANTHELASMA | • *Type of plane xanthoma localized to the periorbital area (most commonly on the upper medial eyelid) as soft – nontender – nonpruritic plaques* |
| TUBEROUS | • *Yellow-orange or erythematous papules or nodules on the extensor extremities/joints* |
| TENDINOUS | • *Subcutaneous nodules on the extensor tendons (esp. on the hands/Achilles tendon)* |
| VERRUCIFORM | • *Verrucous papules in the oral cavity or on anogenital skin* |

**DIAGNOSIS**

- ➡ *Skin biopsy that shows lipid-laden macrophages in the dermis*

**TREATMENT**

- ➡ *Lesions usually resolve with carbohydrate and lipid management*

# HEART FAILURE

**DEFINITION**

➡ *Complex clinical syndrome due to any structural or functional cardiac disorder that impairs the ventricle ability to fill or eject blood → volume overload symptoms (↑ LV filling pressure) & ↓ CO*

**TYPES**

| SYSTOLIC DYSFUNCTION | | DIASTOLIC DYSFUNCTION |
|---|---|---|
| **HEART FAILURE WITH REDUCED EJECTION FRACTION (HFrEF)** | **HEART FAILURE WITH MILDLY REDUCED EJECTION FRACTION (HFmEF) (25% Of All Heart Failure Patients)** | **HEART FAILURE WITH PRESERVED EJECTION FRACTION (HFpEF)** |
| **LVEF ≤40%** | **LVEF 41% - 49%** | **LVEF ≥50%** |

**CAUSES**

**SYSTOLIC DYSFUNCTION**

1. *Coronary artery disease (CAD)*
2. *Hypertension*
3. *Obesity*
4. *Diabetes mellitus*
5. *Valvular heart disease*

**DIASTOLIC DYSFUNCTION**

1. *Hypertension (the most common)*
2. *Aging*
3. *Obesity*
4. *Diabetes mellitus*
5. *Atrial fibrillation*
6. *Coronary artery disease (CAD)*
7. *Amyloidosis (10%) – wild type or mutation in the transthyretin gene*

**PATHOLOGY**

**SYSTOLIC DYSFUNCTION**

➡ *Due to ↓ LVEF → reduced renal perfusion → activation of neurohormonal system -*

**NEUROHORMONAL ACTIVATION**

| Renin-Angiotensin-Aldosterone System (RAAS) | Sympathetic Nervous System |
|---|---|
| ↑ Renin → ↑ AG I → ↑ AG II → ↑ Aldosterone | • ↑ *Epinephrine & norepinephrine → Increase –* 1. *Heart rate* 2. *Contractility* 3. *Vascular resistance* |
| • *Angiotensin II → vasoconstriction → ↑ blood pressure & stimulates thirst*<br>• *Aldosterone → ↑ sodium resorption → ↑ fluid retention* | |

*Both mechanisms will lead to ↑ vasopressin → additional fluid retention & hyponatremia (one of poor prognosis HF signs)*

**INITIAL NEUROHORMONAL ACTIVATION**

➡ *It is adaptive response with maintaining effective CO & BP and blood pressure*

**CHRONIC NEUROHORMONAL ACTIVATION**

1. *Hemodynamic alterations by vasoconstriction & fluid overload*
2. *Structural & functional changes in the individual myocytes → eventually worsening global LV function & dilation (ventricular remodeling)*

**DIASTOLIC DYSFUNCTION**

➡ *Stiffened LV with abnormal relaxation during diastole → increase in LV preload (but normal LVEF)*

**EPIDMIOLOGY**

**AGE**

➡ *HF lifetime risk is 20% for Americans ≥40 years of age*

**RACE**

➡ *African American males have the highest HF risk & highest 5-year mortality rate*

**PRESENTATION**

**VOLUME OVERLOAD**

- *Weight gain*
- *Dyspnea (exertional → orthopnea → paroxysmal nocturnal dyspnea)*
- *Bendopnea (shortness of breath when leaning over) linked to ↑ filling pressures (especially with ↓ cardiac index)*

**LOW CO**

- *Hypotension*
- *Low pulse pressure*
- *Cool extremities*
- *Reduced cognition*
- *Worsening kidney or liver function (low CO or vascular congestion)*

**EXAMINATION**

- *Peripheral edema*
- *Abdominal distension/ascites*
- *Crackles/pleural effusion*
- *$S_3$*
- *↑ Central venous pressure*

**COMPLICATION**

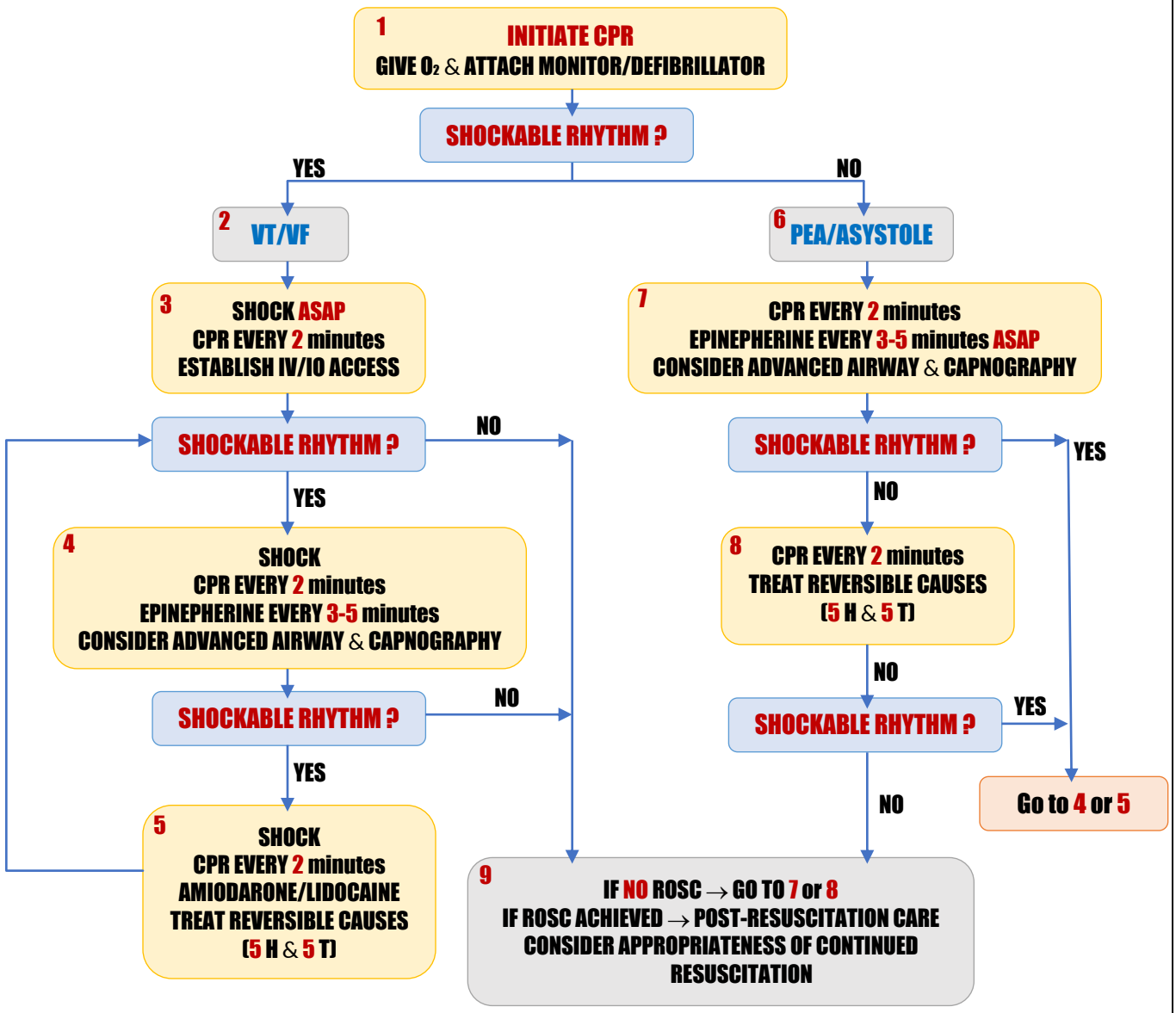
- ***Actual pump failure** (50% mortality rate)*
- ***Arrhythmias** (40% mortality rate)*

# SUDDEN CARDIAC ARREST (SCA)

| | |
|---|---|
| **DEFINITION** | ➡ *Fatal event or collapse within 1 hour of symptom onset without recent acute illness (if unwitnessed so SCD considered happened when the patient was known to be alive & well within the last 24 hours)* |
| **ANNUAL RISK** | ➡ *1:1000 in general population* |
| **CAUSES** | ➡ *VT & VF (most common)* |
| **RISK FACTORS** | • *Diminished LV function (but most SCD cases have normal LV function)*<br>• *LV hypertrophy*<br>• *Nonsustained ventricular arrhythmia*<br>• *Unexplained syncope*<br>• *Chronic kidney disease*<br>• *Heart failure*<br>• *Prior MI*<br>• *Sleep apnea* |

## ACUTE MANAGEMENT

*ACLS Protocol with Minimal Chest Compression Interruption + Defibrillator ASAP*
*(time to defibrillator is the most important factor to determine survival to hospital discharge)*



# VALVULAR HEART DISEASES (VHD)

## GENERAL PRINCIPLES

| | | |
|---|---|---|
| **DEFINITION** | ➡ *Consist of cardiac dysfunction due to structural or functional valve abnormalities due to failure of the valves to either effectively close (regurgitation) or effectively open (stenosis)* | |
| **INCIDENCE** | ➡ *3-6% in ≥65 year old* | |
| **PRESENTATION** | **MOST COMMON** | • *Exertional dyspnea* |
| | **OTHERS** | • *Angina* • *Palpitations* • *Syncope* • *Lower extremity edema* • *Ascites with ↑ girth* |

## CLINICAL SOUNDS OF HEART DISORDERS

| VALVULAR DISORDER | MURMURS | CLICKS | HEART SOUNDS | WAVEFORMS |
|---|---|---|---|---|
| **AORTIC STENOSIS (S)** | • *Systolic ejection murmur at right upper sternal border (RUSB)*<br>• *Mid-to-late peaking diamond-shaped = crescendo-decrescendo* | *Systolic ejection click if congenital or bicuspid* | • *Absent $S_2$ (occasional)*<br>• *S4*<br>• *Paradoxically split $S_2$*<br>• *Radiation to right clavicle/carotid/apex* | *Slowed carotid upstroke* |
| **CHRONIC AORTIC REGURGITATION (S or D)** | • *Occasional early systolic ejection murmur*<br>• *Diastolic –*<br>**a)** *High pitched decrescendo early to holodiastolic*<br>**b)** *Austin flint* | - | *$S_3$ if severe*<br>*No radiation* | *Corrigan or water-hammer pulse*<br>*(rapidly ↑ & falling)* |
| **ACUTE AORTIC REGURGITATION (D)** | *Short diastolic murmur* | - | *$S_3$ if severe* | *Thready pulse* |
| **MITRAL STENOSIS (D)** | *Diastolic rumble* | *Diastolic Opening snap (the only click that occurs in diastole)* | *$S_1$ is enhanced – sometimes snappy & silent if severe calcified*<br>*No radiation* | *Large left (pulmonary venous) a waves & y descent (on PCWP)* |
| **CHRONIC MITAL REGURGITATION (S)** | *Holosystolic constant murmur* | - | *$S_3$ if severe*<br>*$S_4$*<br>*Radiation to axilla/back & anterior precordium* | - |
| **MITRAL VALVE PROLAPSE + MURMUR (S)** | *Late systolic ejection murmur follows click* | *Midsystolic click (Click-murmur syndrome)* | - | - |
| **ACUTE MITRAL REGURGITATION (S)** | *Pansystolic decrescendo murmur at apex* | - | *$S_3$ if severe* | *Large left pulmonary venous v wave* |
| **TRICUSPID STENOSIS (D)** | *Diastolic at left lower sternal border (LLSB)* | - | -<br>*No radiation* | *Giant right jugular a wave* |
| **CHRONIC TR (S)** | *Systolic at left lower sternal border (LLSB) that ↑ with inspiration* | - | -<br>*Radiation to left upper sternal border* | *Large right jugular c & v wave* |
| **PULMONIC STENOSIS (S)** | *Systolic ejection murmur maximal at LUSB with* | *Systolic ejection click* | *Persistent/Wide split $S_2$*<br>*Radiation to Lt clavicle* | - |
| **PULMONIC REGURGITATION (D)** | *Diastolic decrescendo in LLSB* | - | *Loud P2 if PH*<br>*No radiation* | |

## BICUSPID AORTIC VALVE DISEASE

| | | | |
|---|---|---|---|
| **INCIDENCE** | **1-2%** | ➡ *% of bicuspid aortic valve in the general population* | |
| | **>30%** | ➡ *% of bicuspid aortic valve that found in >70 years with severe AS* | |
| **PATHOLOGY** | ➡ *Bicuspid morphology → causing abnormal shear forces → early valve degeneration →*<br>• *Stenosis in most patients (up to 75%)*<br>• *Pure regurgitation in lower percentage of patients (2%–10%)* | | |
| **PRESENTATION** | ➡ *Usually asymptomatic finding of systolic ejection murmur in adolescence or young adulthood → gradually progress to severe disease in 5th or 6th decade of life* | | |
| **DIAGNOSIS** | **TTE** | 1. *Diagnose bicuspid valvulopathy*<br>2. *Diagnose associated congenital aortopathy disorders –*<br>• *Aortic aneurysm* • *Aortic dissection* • *Aortic coarctation* | |
| | **IF SUBOPTIMAL TTE** | ➡ *Use CMR angiography or CT angiography* | |
| | **LIFELONG SURVEILLANCE** | • *Serial imaging to follow any diagnosed abnormality & the frequency based on*<br>1. *The nature of disorder (stenosis – regurgitation – aneurysm)*<br>2. *Severity of the abnormality*<br>3. *Age of the patient*<br>4. *Family history*<br>5. *Candidacy for surgery* | |
| | **SCREENING** | ➡ *Echocardiography screening for 1st-degree relatives (as bicuspid aortic valve is heritable abnormality) for bicuspid aortic valve & aortopathy* | |
| **TREATMENT** | **SURGICAL THERAPY** | **≥ 4.5 cm** | ➡ *Reasonable for surgical repair of ascending aorta if aortic dimension ≥4.5 cm + undergoing surgery for severe aortic stenosis or regurgitation in bicuspid valve* |
| | | **> 5.5 cm** | ➡ *Surgical repair of the ascending aorta or aortic sinuses if aortic dimension is >5.5 cm but no indication for stenotic or regurgitant aortic valve surgery* |
| | | **> 5 cm** | ➡ *Surgical repair of the ascending aorta is reasonable if dimension is >5 cm + additional risk factor for dissection –*<br>1. *Family history*<br>2. *Progression rate ≥0.5 cm/year* |
| | **MEDICAL THERAPY** | ➡ *No medical therapy slow aortic dilatation in aortopathy/bicuspid aortic valve* | |

## MITRAL STENOSIS (MS)

| | | |
|---|---|---|
| **PATHOLOGY** | ➡ *Fusion of the mitral commissures → valve calcification & abnormalities in the subvalvular apparatus in more advanced disease → slow progressive disease over decades with gradual left atrial (LA) enlargement & preservation of LV function* | |
| **CAUSES** | **MOST COMMON** | ➡ *Rheumatic heart disease (women > men with 4:1 ratio) – rare in U.S.* |
| | **OTHERS** | • *Parachute mitral valve* • *Chest irradiation*<br>• *Systemic lupus erythematosus* • *Rheumatoid arthritis*<br>• *Severe mitral annular calcification (common in elderly)* |
| **PRESENTATION** | **MANIFESTATION** | • *Fatigue (due to low CO)* • *Dyspnea (due to pulmonary congestion)*<br>• *Lower extremity edema (due to pulmonary hypertension with right-sided HF)* |
| | **COMPLICATION** | 1. *Systemic embolization (20–25% without anticoagulation)*<br>2. *Atrial fibrillation (50% in MS due to left atrial stretching and dilation)*<br>3. *Hemoptysis in severe cases (due to rupture of pulmonary bronchial vessels distended by pulmonary venous congestion)* |
| | **PRECIPITATING FACTORS** | 1. *Exercise (so MS symptoms as usually exertional) → shortens diastolic filling time → ↑ transvalvular flow & diastolic mitral gradient → worsening of LA hypertension*<br>2. *Pregnancy → ↑ blood volume & cardiac output* |
| | **CAUSE OF DEATH** | **60%** • *Heart failure*<br>**40%** • *Thromboembolism* |

# MYOCARDIAL DISEASE (CARDIOMYOPATHIES)

## TYPES

| | | |
|---|---|---|
| **STRUCTURAL HEART DISEASE + LV DYSFUNCTION** | | 1. *Ischemic heart disease* (ischemic cardiomyopathy)<br>2. *Hypertensive heart disease*<br>3. *Valvular heart disease* |
| **DILATED (NONISCHEMIC)** | **IDIOPATHIC** | 1. *Idiopathic dilated cardiomyopathy* (20–30%)<br>2. *Genetic dilated cardiomyopathy (AD)* |
| | **INFECTIONS** | 1. *Viral* – coxsackievirus B & HIV<br>2. *Protozoal – Chagas disease in* Latin America |
| | **DRUG/TOXIN** | 1. *Chemotherapy agents* –doxorubicin & daunorubicin<br>2. *Alcohol* 3. *Cocaine* |
| | **ENDOCRINE** | • *DM* • *Thyroid diseases* • *Acromegaly* |
| | **RHEUMATOLOGY** | *Connective tissue & inflammatory diseases* |
| | **STRESS** | 1. *Stress-induced cardiomyopathy*<br>2. *Tachycardia-induced cardiomyopathy* |
| | **OBGY** | ➡ *Peripartum cardiomyopathy* |
| | **OTHERS** | 1. *Infiltrative diseases* (sarcoidosis)<br>2. *Left ventricular noncompaction = increased LV trabeculae* |
| **HYPERTROPHIC** | | ➡ *Genetic disease → thickening of the heart muscle & heart failure with preserved EF* |
| **RESTRICTIVE** | | ➡ *Infiltrative diseases* –<br>1. *Amyloidosis* 2. *Hemochromatosis* 3. *Sarcoidosis* |

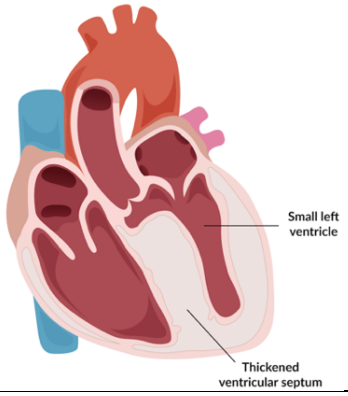
## HYPERTROPHIC CARDIOMYOPATHY (HCM)

| | |
|---|---|
| **INCIDENCE** | ➡ 1:500 *in all age groups (but increase incidence in U.S. in* 3rd – 4th *decades of life)* |
| **CAUSE** | ➡ *Autosomal dominant heritable disorder due to mutations in the genes of sarcomeric proteins coding → ↑ LV wall thickness with no loading conditions* (hypertension) *or other underlying causes* |

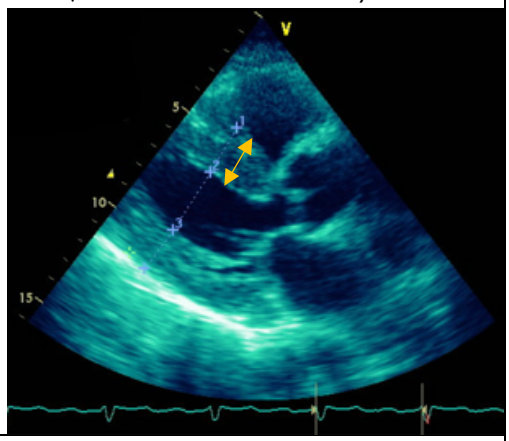
## PATHOLOGY

**MAIN**

- *Septal hypertrophy + left ventricle is thickened but not dilated* (with no other cardiac or systemic conditions [HTN/AS])

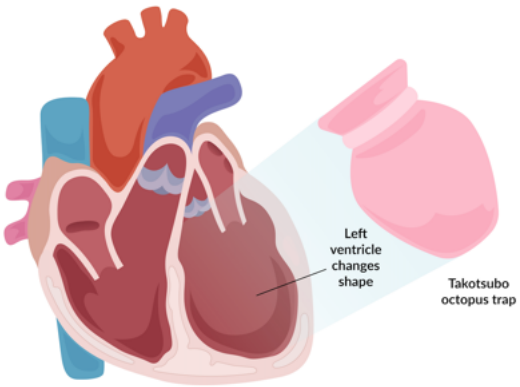


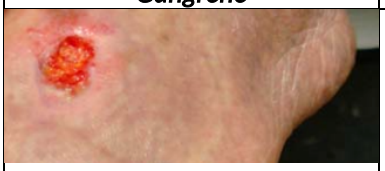
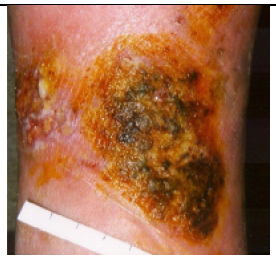
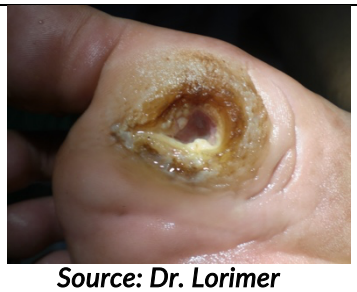
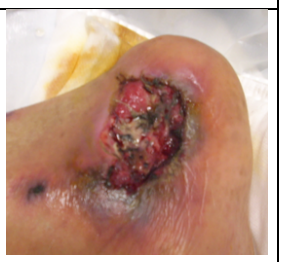

**HCM**
*Source: Hugo*



**CONSEQUENCES**

1. *Dynamic LV outflow tract obstruction (LVOT) = obstructive HCM due to asymmetric LV hypertrophy with prominent interventricular septal thickening*
2. *Diastolic dysfunction* (HFpEF) *due to –*
   - *↑ Chamber stiffness*
   - *Progressive fibrosis*
   - *Myocardial ischemia due to mismatch of coronary flow & LV mass*
3. *Myocardial ischemia*
4. *Systolic anterior motion of mitral valve → causing early to midsystolic LVOT obstruction → MR due to leaflet malcoaptation (eject-obstruct-leak triad)*
5. *High resting LV outflow gradient* (≥30 mmHg) *in* 25%

<table>
<tr><td rowspan="6">COMPLICATION</td><td colspan="3">AFIB</td></tr>
<tr><td>GUIDELINES</td><td colspan="2">• Use rate control + anticoagulation (regardless $CHA_2DS_2$-VASc score)<br>• Add rhythm control early as the AFib HCM patients remain symptomatic despite rate control & anticoagulation<br>• Add anticoagulation in subclinical AFib >24 hours detected by ambulatory ECG</td></tr>
<tr><td rowspan="3">AGENTS</td><td rowspan="2">NONPREGNANT</td><td>1st</td><td>➡ Direct oral anticoagulants</td></tr>
<tr><td>2nd</td><td>➡ Warfarin</td></tr>
<tr><td>PREGNANT</td><td colspan="2">➡ Low-molecular-weight heparin or warfarin (maximum dose <5 mg/day)</td></tr>
<tr><td>AVOID</td><td colspan="3">➡ Digoxin in AFib HCM due to positive inotropic effects that may worsen LVOT gradient</td></tr>
<tr><td colspan="4">DILATED CARDRIOMYOPATHY</td></tr>
<tr><td>PATHOLOGY</td><td colspan="3">➡ <5% of patients will progress to end-stage HCM = dilated cardiomyopathy + systolic dysfunction (LVEF <50%)</td></tr>
<tr><td>TREATMENT</td><td colspan="3">➡ Using guideline-directed therapy</td></tr>
<tr><td colspan="5">DILATED CARDIOMYOPATHY (DCM)</td></tr>
<tr><td>PATHOLOGY</td><td colspan="4">• Ventricular dilation + depressed myocardial contractility of one or both ventricles (EF <40%) with or without overt heart failure symptoms</td></tr>
<tr><td>INCIDENCE</td><td colspan="4">➡ Atrial & ventricular arrhythmias are common with increased risk for SCD<br>➡ African Americans → 3x higher risk for developing DCM than Caucasians</td></tr>
<tr><td>PROGNOSIS</td><td colspan="4">➡ 50% mortality at 5 years with symptomatic HF & DCM</td></tr>
<tr><td>TREATMENT</td><td colspan="4">➡ Heart failure due to dilated cardiomyopathy is treated as other causes of HF</td></tr>
<tr><td colspan="5">STRESS CARDIOMYOPATHY<br>(TAKOTSUBO CARDIOMYOPATHY – APICAL BALLOONING SYNDROME – BROKEN HEART SYNDROME)</td></tr>
<tr><td>PATHOLOGY</td><td colspan="4">➡ Thought to be due to reversible myocardial toxicity induced by very high catecholamine levels → reduced LVEF + elevated cardiac enzyme levels + signs of ischemia on ECG<br>  <br></td></tr>
<tr><td>CAUSES</td><td colspan="4">➡ Illicit by stressful physical or emotional event (loved one death)</td></tr>
<tr><td>PRESENTATION</td><td colspan="4">➡ Usually occurs in older women with sudden onset of chest pain suggesting ACS</td></tr>
<tr><td rowspan="4">DIAGNOSIS</td><td>TROPONIN</td><td colspan="3">• Elevated in all patients</td></tr>
<tr><td>ECG</td><td colspan="3">• Anterior ST segment elevations of acute myocardial infarction</td></tr>
<tr><td>ECHO</td><td colspan="3">• Wall motion abnormalities (typically apical dyskinesis or ballooning) that do not follow specific coronary artery territory with preservation of basal wall motion</td></tr>
<tr><td>CARDIAC CATH</td><td colspan="3">• Done as part of ACS & shows non-ischemic finding</td></tr>
<tr><td>TREATMENT</td><td colspan="4">➡ Supportive & treatment as other heart failure causes with recovery in most of patients over few weeks – months (most clinicians continue therapy for at least 1 year)</td></tr>
<tr><td>FOLLOW-UP</td><td colspan="4">➡ Repeat echocardiography in 3 - 6 months to assess recovery</td></tr>
</table>

| DDx | | | |
|---|---|---|---|
| **ARTERIAL ISCHEMIC ULCER** | **VENOUS STASIS ULCER** | **NEUROPATHIC DIABETIC ULCER** | **PRESSURE ULCER** |
| **ETIOLOGY** | | | |
| *Atherosclerosis*<br>*Vascular disease* | *Varicose veins*<br>*Prior DVT* | *DM*<br>*Trauma* | *Limited mobility* |
| **ULCER LOCATION** | | | |
| *Pressure points –*<br>*Toes*<br>*Feet*<br>*Lateral malleolus & tibia* | *Gaiter area – area between lower calf & medial malleolus* | *Planter aspect of foot*<br>*Tip of the toes* | *Bony prominence*<br>*Heel* |
| **ULCER DESCRIPTION** | | | |
| *Punched out & deep*<br>*Irregular in shape*<br>*Necrotic tissue + minimal exudate*<br>*Minimal granulation tissue* | *Shallow flat margins*<br>*Moderate/heavy exudate*<br>*Slough at the base + granulation tissue* | *Deep surrounded by callus* | *Deep & often macerated* |
| **LOWER EXTREMITY CONDITION** | | | |
| *Thin shiny skin*<br>*Reduced hair growth*<br>*Cooler skin*<br>*Pallor on leg elevation*<br>*Absent/weak pulses*<br>*Delayed capillary refill*<br>*Gangrene* | *Hemosiderin staining*<br>*Thickening & fibrosis*<br>*Eczematous & itchy skin*<br>*Limb edema*<br>*Normal capillary refill* | *Dry cracked insensate skin + callus* | *Atrophic skin*<br>*Loss of muscle mass* |
|  <br>*Source: J. Moore* |  <br>*Source: Prof. G.Hoffman* |  <br>*Source: Dr. Lorimer* |  <br>*Source: Ashashyou* |
| **CLAUDICATION**<br>**(Vascular Disease)** | | **PSEUDOCLAUDICATION**<br>**(Lumbar Spinal Stenosis)** | |
| **DISCOMFORT CHARACTER** | | | |
| • *Cramping* • *Tightness* • *Aching* • *Fatigue* | | ➡ *As claudication +*<br>• *Tingling* • *Burning* • *Numbness* • *Weakness* | |
| **DISCOMFORT LOCATION** | | | |
| • *Buttock* • *Hip* • *Thigh* • *Calf* • *Foot* | | *As claudication (mostly bilateral)* | |
| **EXERCISE-INDUCED** | | | |
| *Yes* | | *Variable* | |
| **WALKING DISTANCE BEFORE ONSET OF THE SYMPTOMS** | | | |
| *Consistent* | | *Variable* | |
| **DISCOMFORT WITH STANDING STILL** | | | |
| *No* | | *Yes* | |
| **RELIEF ACTIONS** | | | |
| *Stand or sit* | | *Sit or flexion at the waist (leaning on shopping cart)* | |
| **TIME TO RELIEF** | | | |
| *<5 minutes* | | *≤30 minutes* | |

## MANAGEMENT OF CARDIOVASCULAR DISEASES IN PREGNANCY

| | | |
|---|---|---|
| **MORTALITY** | • ↑ *Maternal mortality in U.S. (despite ↓ in other Western countries) over the prior 2 decades with the acquired cardiovascular disorders (cardiomyopathy – CAD – aortic disorders) are the most common cause of maternal mortality*<br>• *The leading cause of pregnancy-associated MI (commonly in 1st month postpartum) is spontaneous coronary artery dissection that is managed by conservative noninterventional therapy* | |
| **SPECIFIC DISORDERS** | **OBSTRUCTIVE VALVULAR DISORDER** | • *Women can be symptomatic during pregnancy due to ↑ blood volume and CO so proper intervention should be determined before pregnancy* |
| | **VALVULAR REGURGITATION** | • *Usually tolerated well during pregnancy* |
| | **OBSTRUCTIVE HCM** | • *Treat symptomatic obstructive HCM with nonvasodilating β-blockers + monitoring fetal growth* |
| | **MARFAN SYNDROME (↑ RISK OF AORTIC DISSECTION DURING PREGNANCY)** | • *Prepregnancy aortic repair is recommended with aortic diameter ≥4.5 cm*<br>• *Prepregnancy aortic repair/replacement if ascending aortic diameter >4.0 cm + Risk factors for dissection –*<br>1. *Rapid dilatation of the aorta*<br>2. *Personal or family history of aortic dissection* |
| | **ARRHYTHMIAS** | • *Most arrhythmias in pregnancy are benign so starting antiarrhythmic drugs is based on shared decision with the risks/benefits to the mother & fetus*<br>• *Most β-blockers (except atenolol) are safe to use in pregnancy & lactation* |
| **BIRTH ROUTE** | **VAGINAL DELIVERY INDICATIONS** | • *Generally preferred in patients with cardiovascular disease due to –*<br>1. *Lower blood loss*<br>2. *Quicker recovery*<br>3. *Lower risk for thrombosis than does cesarean delivery* |
| | **CESAREAN DELIVERY INDICATIONS** | 1. *In severe decompensated cardiovascular disease*<br>2. *In markedly dilated aorta*<br>3. *In patients on warfarin therapy (↓ risk of fetal intracranial hemorrhage as the fetus is fully anticoagulated)* |

## PERIPARTUM CARDIOMYOPATHY

| | | |
|---|---|---|
| **PATHOLOGY** | ➡ *Left ventricular systolic dysfunction (in prior healthy women) occur toward the end of pregnancy or in the 5 months after delivery in the absence of another identifiable cause* | |
| **RISK FACTORS** | • *Age >30 years*<br>• *Gestational hypertension*<br>• *Prior episode of peripartum cardiomyopathy* | • *Multiparity*<br>• *Preeclampsia* |
| | | • *Multifetal pregnancy*<br>• *Tocolytic therapy (terbutaline)* |
| **COMPLICATION** | 1. *Heart failure* | 2. *Thromboembolic events*<br>3. *Arrhythmias* |
| **TREATMENT** | **OUTCOME** | • *Full recovery in most patient within 6 months (EF is normalized or improved)*<br>• *Recurrent or more reduction of LVF in subsequent pregnancy* |
| | **SUBSQUENT PREGNANCIES** | ➡ *Recommend avoidance of pregnancy if prior episode of peripartum cardiomyopathy with persistent left ventricular dysfunction* |
| | **MEDICAL THERAPY** | • *β-blockers* • *Digoxin* • *Diuretics* • *Hydralazine* • *Nitrates* |
| | **AVOID** | ➡ *ACEIs/ARBs & aldosterone antagonists as they are teratogenic until after delivery* |
| | **ANTICOAGULATION – INDICATION** | ➡ *If LVEF <35% (due to high risk for thromboembolism)* |
| | **TIMING** | ➡ *8 wks but therapy can be discontinued sooner if EF normalizes* |
| | **AGENTS** | ➡ *UFH or warfarin based on teratogenicity & the time since delivery* |
| | **SEVERE REFRACTORY HEART FAILURE** | • *Treated in specialty center with advanced therapies –*<br>1. *Ventricular assist device placement*<br>2. *Advanced arrhythmia management*<br>3. *Heart transplantation* |