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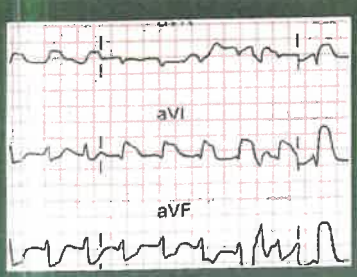
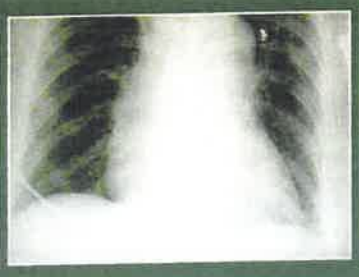
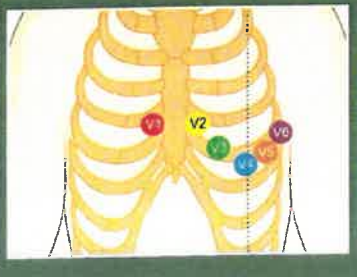
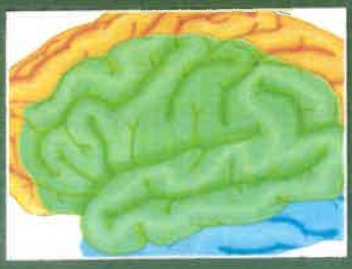
# Cerebellum Medicine

## For the Students
## By the Teachers





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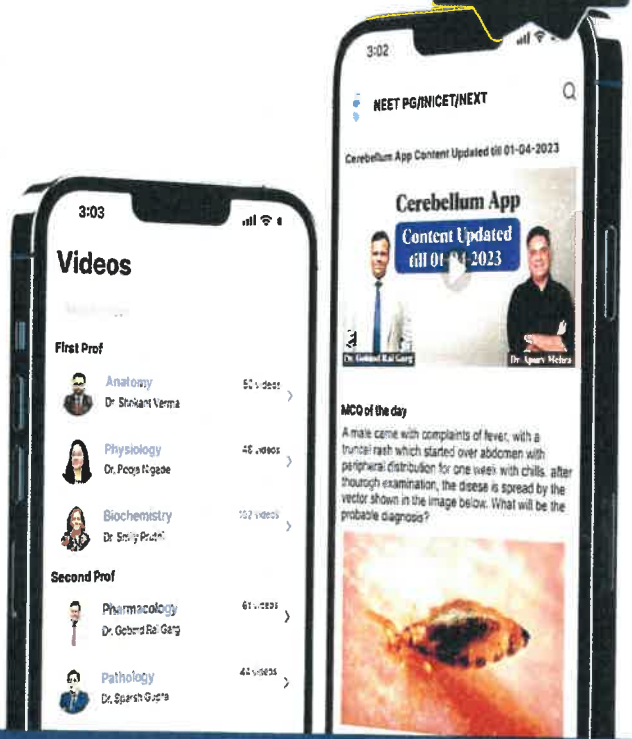


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# Table of Content

**Chapters LEAST IMPORTANT for FMGE aspirants:**

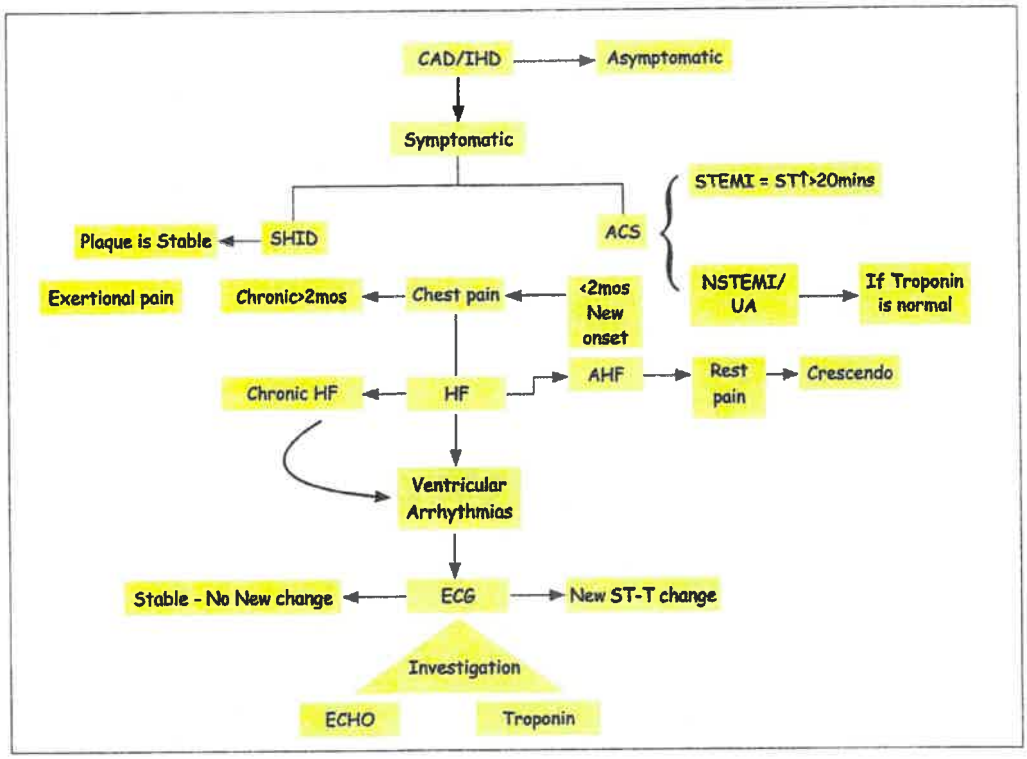
**Cardiology:** Stable Ischemic Heart Disease (except basics like indications of CABG, PCI)
**Endocrinology:** Genetic Disorders (MEON's & Autoimmune polyglandular Syndromes)
**Neurology:** Movement Disorders other than Parkinson's Disease
**Pulmonary and Critical Care:** Complications of Mechanical Ventilation
**Nephrology:** Rare types of AKI and Details of Renal Transplantation
**Rheumatology:** Infectious Arthritis, Adult onset Still's Disease, Cryoglobulinemia and Amyloidosis
**Medical Oncology:** Cancer Biology and Radiation Oncology
**Hematology and transfusion:** Details of Blood Products & Platelet Function defects
**Gastroenterology:** Details of IBD treatment, Metabolic Liver diseases except Wilson disease

Section 1

# Cardiology

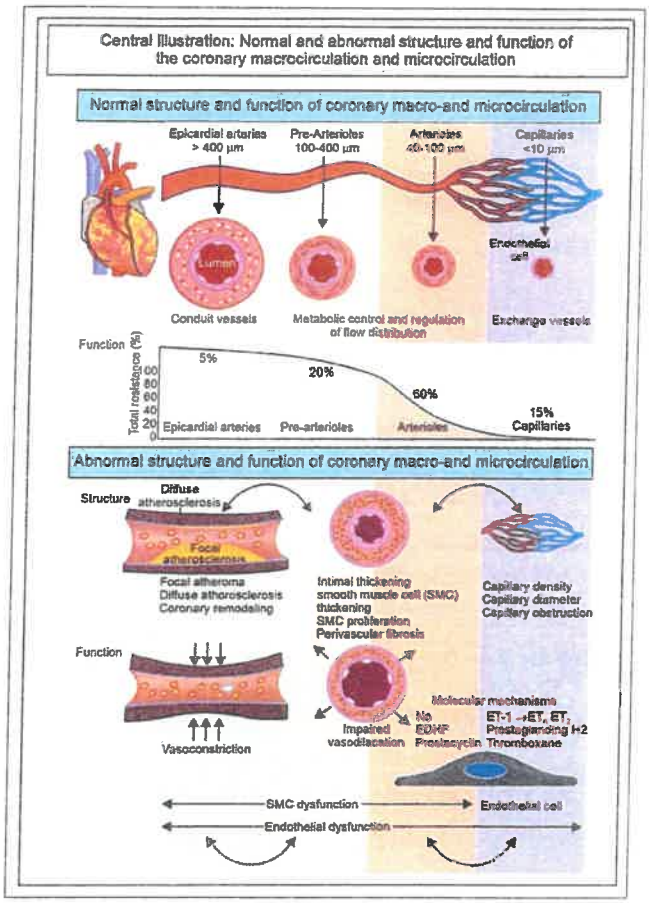
# 1.1 Chapter

# STABLE ISCHEMIC HEART DISEASE (SIHD) - DIAGNOSIS




## Coronary Blood Flow - Basics

- CBF contributes to ~ 5% of the total cardiac output and may ↑ **up to 5x with exercise** (↑ Workload)
  - Normally, the coronary microcirculatory resistance constitutes the only resistance to myocardial flow & the epicardial vessels (LMCA, LAD, LCX, RCA) are just conductance vessels
  - Epicardial vessels don't offer any sort of resistance and are diseased which are looked up on the Angiography and not the microvasculature.
  - **95% of the resistance is offered by the microvasculature to the coronary blood flow** and these vessels are adaptive in physiological as well as pathological conditions.
  - With ≥ 70% stenosis, the trans-stenotic flow drops during exertion & with ≥ 90% stenosis, the trans-stenotic flow drops at rest.
  - ↑Resistance in the epicardial vessels ↓ the flow to the microvasculature due to which the pressure ↓
  - Collaterals are formed due to the Pressure Gradient, i.e., pressure difference between the diseased and the normal vessels
  - > 80% of left coronary flow & ~ 50% of right coronary flow occurs in diastole due to differential systolic compression of microvasculature



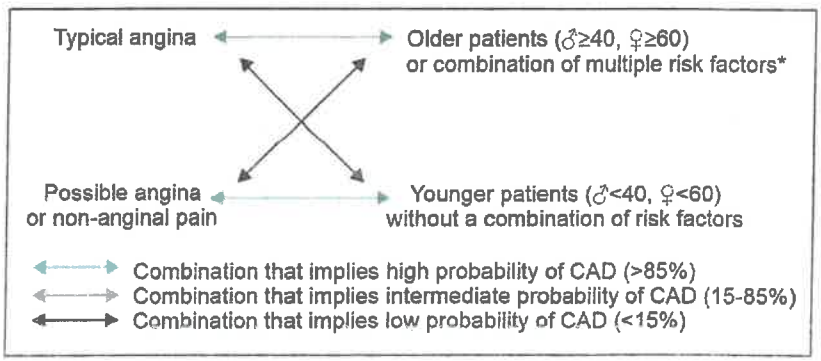

- **STEAL PHENOMENA** – Non diseased vessels are going to get more blood and diseased vessels will have less blood flow.
  - There are 2 types of STEALS, **Coronary Steal** and **Collateral Steal**.
  - During Exercise, dilation of the microvasculature (the diseased vessels which are maximally dilated won't dilate further but the normal vessels will dilate) → Resistance increases which will lead to increase blood flow to the normal vasculature and in contrast the flow is reduced in the collaterals as well as the stenotic vessels.
  - **Drugs which can induce STEAL phenomena are:**
    - Adenosine
    - Dipyridamole
    - Hydralazine (C/I in CAD)
    - Nitroprusside (arteriolar vasodilator)
    - Volatile General Anesthetics

Therefore, during exercise or adenosine infusion, extensive microvascular dilatation occurs and requiring an extensive increase in flow to fill the dilated circulation (since the flow cannot increase across a flow-limiting stenosis, ischemia occurs)

- **Angina features:**
  - Discomfort in chest **(Levine sign)**, jaw, shoulder, back, arm, epigastrium
  - Aggravated by exertion and / or emotional stress
  - Relieved by rest and / or NTG (< 30s to 5 min)
    - **3/3 features** = typical angina, 2/3 features = atypical angina, 0 or 1 feature = noncardiac
    - Noncardiac chest pain: **3P's = Pleuritic, Positional and Palpable**
- **CCS Angina classification:**

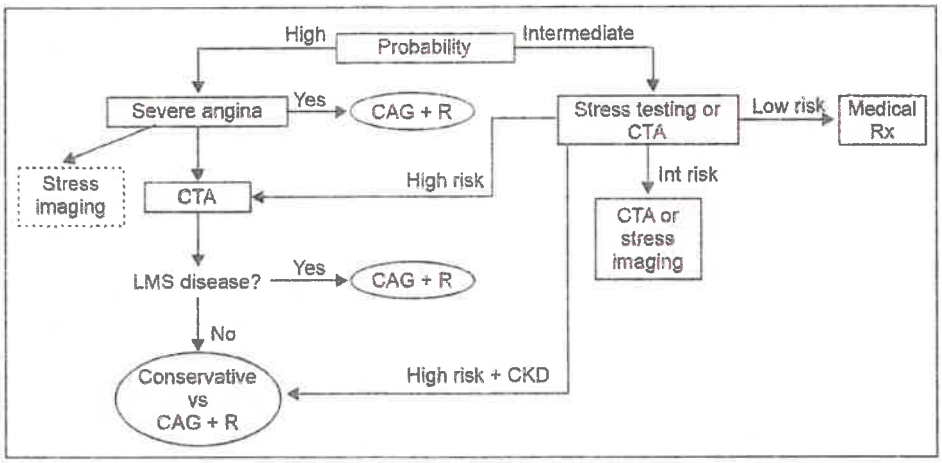
| | |
|---|---|
| I | No angina with ordinary activity. Angina only with strenuous, rapid, or prolonged exertion |
| II | Slight limitation of ordinary activity (Angina after > 2 blocks / > 1 flight of stairs) |
| III | Marked limitation of ordinary activity (Angina after 1-2 blocks / < 1 flight of stairs) |
| IV | Inability to carry out any physical activity without angina or angina occurring at rest |

- **Risk Stratification:**



## Risk Factors: DM/dyslipidemia/HTN/Active smoking

- Algorithm for Evaluation of Chronic Chest Pain



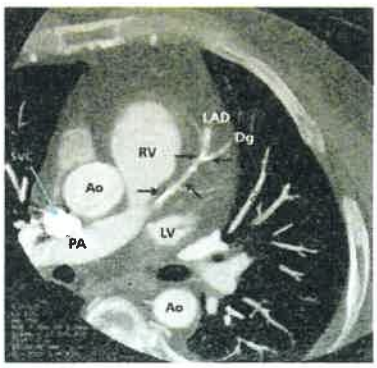

- CCTA vs Stress Testing/Imaging

| | |
|---|---|
| **Pretest likelihood of CAD** | Low → no testing necessary → option for CAC for ASCVD risk stratification |
| | Intermediate high → younger patient (< 65 y of age) OR less obstruction CAD suspected → CCTA favored |
| | Intermediate high → older patient (> 65 y of age) OR more obstructive CAD suspected → stress testing favored |

| | **Favors use of CCTA** | **Favors use of stress imaging** |
|---|---|---|
| Goal | • Rule Out Obstructive CAD<br>• Detect Nonobstructive CAD | • Ischemia-guided management |
| Availability of expertise | • High-quality imaging and expert interpretation routinely available | |
| Likelihood of obstructive CAD | • Age < 65 y | • Age > 65 y |
| Prior test results | • Prior functional study inconclusive | • Prior CCTA inconclusive |
| Other compelling indication | • Anomalous coronary arteries<br>• Required evaluations of aorta or pulmonary arteries | • suspected scar (especially if pet or stress CMR available)<br>• suspected coronary microvascular dysfunctional (when PET or CMR available) |

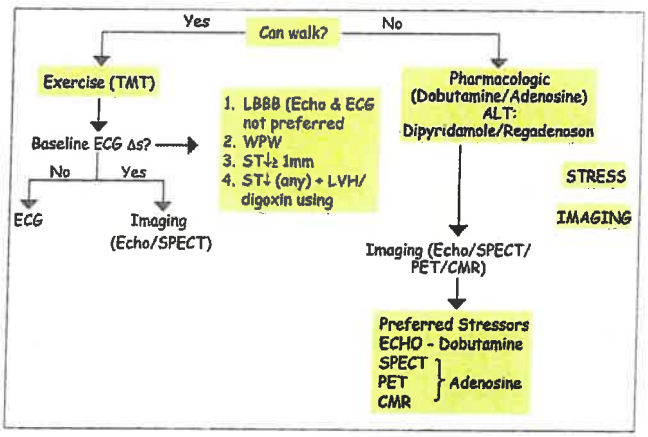
**Note:** Myocardial viability - FDG-PET is done (t1/2 = 110mins/2hrs)

- CCTA



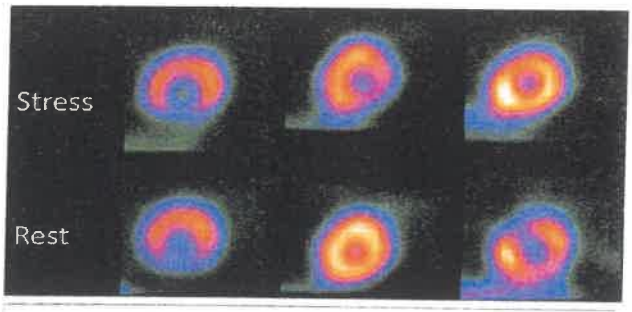
- Stress Testing - Indications:
  - To diagnose obstructive CAD
  - Evaluate change in clinical status in a known CAD
  - Risk stratification after ACS
  - Evaluate exercise tolerance
  - Localize ischemia
- Stress Testing - Contraindications:

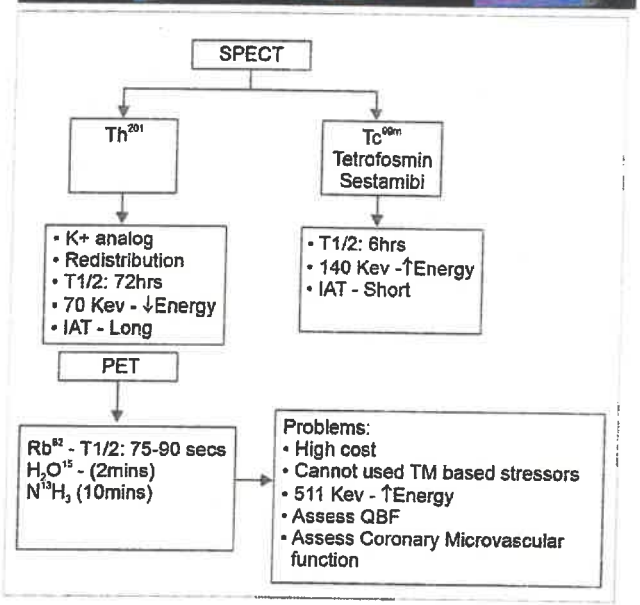
| Absolute | Relative |
|---|---|
| Acute MI < 48 h | LMCA disease |
| High-risk UA | Moderate valvular stenosis (AS), |
| Acute PE | Severe HTN |
| Severe AS | HCM |
| Uncontrolled HF | High-degree AVB |
| Uncontrolled arrhythmias | Severe electrolyte abnormalities |
| Myopericarditis | |
| Acute AoD | |




**Choosing a stress test depends on:**

- LBBB
- Ability of a patient to exercise
- Imaging modality

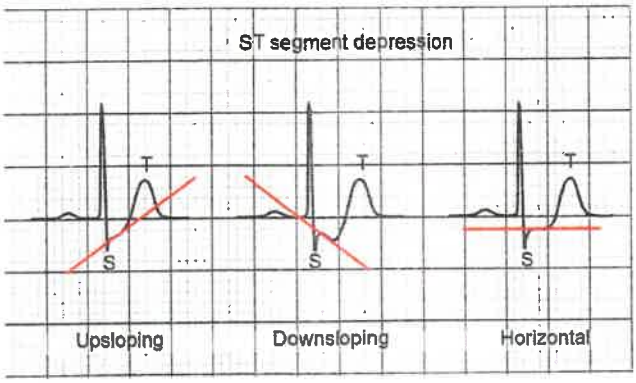



| Stress testing Information | | | | | |
|---|---|---|---|---|---|
| | ECG | Stress echocardiography | SPECT MPI | PET MPI | Stress CMR MPI |
| Patient capable of exercise | ✓ | ✓ | ✓ | × | × |
| Pharmacologic stress indicated | × | ✓ | ✓ | ✓ | ✓ |
| Quantitative flow | × | × | × | ✓ | ✓ |
| LV dysfunction / scar | × | ✓ | ✓ | ✓ | ✓ |

Stable Ischemic Heart Disease (SHID) - Diagnosis

- Treadmill ECG:




- **Target & Protocol for Exercise Stress**
  - Reach Target HR, which is 85% of the maximal HR (=85% of [220 – age]) PLUS achieve a good workload ≥6-7 METs (≥ 5 minutes of the Bruce protocol)
  - (OR) Attain high-risk criteria before this goal
- **Standard Bruce Protocol**

| Stage | Speed mph (kph) | Inclination (°) | Duration (min) | MET |
|---|---|---|---|---|
| 1 | 1.7 (2.7) | 10 | 3 | 5 |
| 2 | 2.5 (4.0) | 12 | 3 | 7 |
| 3 | 3.4 (5.4) | 14 | 3 | 10 |
| 4 | 4.2 (6.7) | 16 | 3 | 13 |
| 5 | 5.0 (8.0) | 18 | 3 | 16 |
| 6 | 5.5 (8.8) | 20 | 3 | 18 |
| 7 | 6.0 (9.6) | 22 | 3 | 20 |

## Duke's Treadmill Score = Angina Index –

- 0: no angina
- 1: Non-limiting Angina
- 2: Limiting Angina

| Level | Score | Management |
|---|---|---|
| Low | ≥ +5 | Medical Treatment |
| Intermediate | +4 to -10 | Stress imaging (SPECT)/ CCTA |
| High | ≤ -11 | CCTA/medical Rx/ CAG-R |

## Risk Stratification After a Stress Test

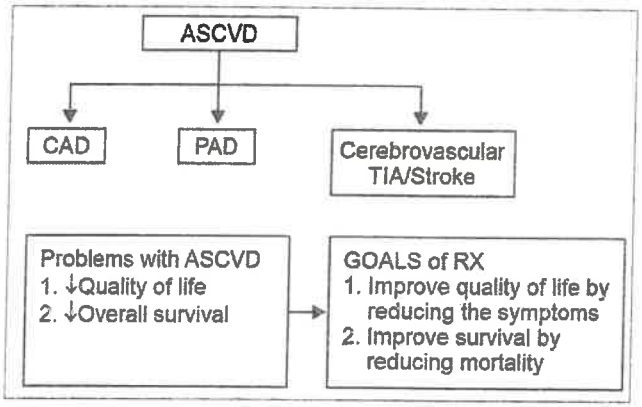
- **High risk features:** (Annual rate of death > 3%)
  - **Physiologic:** ↓ or fail to ↑ BP by > 20/10mm Hg, Angina @ < 4 METS, Chronotropic incompetence/ vagal failure
  - **ECG:** DTS ≤-11, ST ↓ ≥2 mm **(OR)** ≥1 mm in stage 1 **(OR)** in ≥5 leads **(OR)** ≥5 min in recovery, ST ↑, VT
  - **Echo/MPI:** Large/multiple (≥2) reversible perfusion defects, ≥ 3 regions of WMA in Echo.

# 1.2 Chapter | STABLE ISCHEMIC HEART DISEASE (SIHD) MANAGEMENT

## Basic Terms

**ASCVD/CV Disease – Atherosclerotic Cardiovascular Disease (Macrovascular)**

- MACE – Major Adverse Cardiac Events
  - Death due to some CV problem
  - Non-Fatal MI
  - Non-fatal Stroke – UA/ HF




## Management of CCS/SIHD

| ↓ Mortality | ↓ Symptoms |
|---|---|
| ASA (clopidogrel if ASA allergy – 75mg/d)<br>Dose – 75mg/day (81mg/d) | 1st choice: βBs (Alternatives ND-CCBs) + SA Nitrates PRN |
| Statins ± ezetimibe/PCSK9i (to keep LDL < 70)<br>High intensity statins – use Max tolerance dose<br>Rosuvastatin – 20 – 40 mg/d<br>Atorvastatin – 40 – 80 mg/d<br>PCSK9i – Evolocumab, Alirocumab | 2nd choice: D-CCBs, LA Nitrates, Ranolazine, Nicorandil, Ivabradine, trimetazidine, EECP |
| HTN control (ARBs + ACEi preferred especially if EF < 50%, DM, CKD) | Consider Revascularization – for refractory angina |
| DM control (preferably SGLTi/GLP1RA) | |
| Smoking cessation (50% ↓ in risk of MI and stroke < 1 year)<br>Risk ~ never smokers in 3-5 years | |

- **Antianginals:**
  - α1 – vasoconstriction
  - β1 – ↑ Inotropicity /↑Chronotropicity
  - β2 – vasodilation/ bronchodilation
- **β-Blockers –**
  - 1st Gen Non – selective = Propranolol / Sotalol (AAD)/ Nadolol/ Timolol/Pindolol (ISA)
  - 2nd gen Selective β1 –
    - Atenolol
    - Metoprolol
    - Esmolol
    - Bisoprolol
    - Acebutolol
  - 3rd Gen – Vasodilator β-blockers

| Non-Selective | Selective |
|---|---|
| Labetalol | Nebivolol (↑ NO release) |
| Carvedilol (Additional α1 blockage) | Betaxalol (CCB) |
| | Celiprolol (β2 +) (ISA) |

## Contraindications of β-blockers

1. Severe Reactive Airway Disease (BA/COPD)
2. Avoid in acute HF (pulmonary edema)
3. Hemodynamic instability
4. Low HR (HR < 60 - initiation not recommended, HR < 40 withhold β-blockers)
5. 2°/3° AV block
6. PR interval > 0.24secs

## Side effects of β-blockers

**Cardiac**

1. Severe bradycardia ↓HR
2. ↓CO →↑HF
3. AV nodal block
   - These effects are synergistic if β-blockers are used along with Non-dihydropyridine CCBs i.e., verapamil and diltiazem.

**Extracardiac**

1. Exacerbations of BA/COPD
2. Exacerbate some vascular problems - Raynaud's/ PAD
3. Metabolic S/E - Hyperkalemia (↑ risk with renal failure) / hyperglycemia (more in prediabetics) / hypertriglyceridemia

**Not due to β receptor blockage**

1. GI Intolerance
2. Vivid dreams
3. Depression
4. Sexual dysfunction

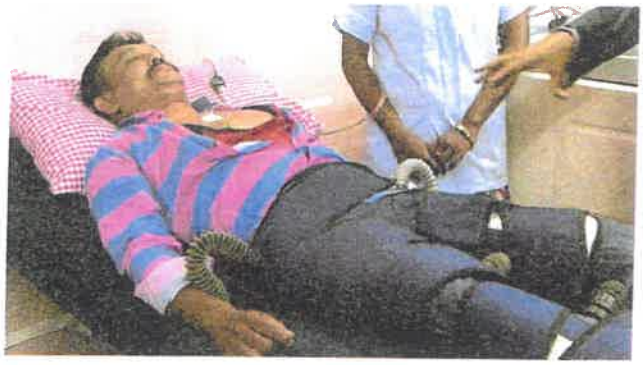
## Calcium Channel Blockers

| ND-CCBs | D-CCBs |
|---|---|
| Verapamil/ Diltiazem | DIPINES |
| Poor vasodilators | Potent vasodilators - cause reflect tachycardia (more with short acting drugs - Nifedipine) |
| Act on the heart - negative inotropism, Chronotropism and AVN blockade (Antiarrhythmic drugs - Class IV) | X |
| Side effects - Headache, peripheral edema, gingival hyperplasia | |

- **Long-Acting Nitrates -**
  - ISDN
  - ISMN
  - E. Tetranitrate
  - Penta Tetranitrate
    - One should provide adequate Nitrate free intervals of at least 12-14hrs/day to prevent the development of tolerance.
- **Ranolazine - Dual Effect**
  - Inhibit INA
  - Inhibit PFOX - to inhibit the beta-oxidation process, Ranolazine inhibits LC3KAT thereby forcing the heart to use more glucose in which less ATP will be consumed. Therefore, causing relief of Angina.
  - Act as an anti-anginal drugs, cause slight ↑QTc. Reduce HbA1c- approx. 1% in diabetics (only seen in patients with HbA1c >8%)
  - Side effects- headache, flushing and constipation (MC)
- **Nicorandil -**
  - Nitrate like action - ↓preload
  - K+ channel opening ability
  - Most important Side Effects - mucosal ulcers - oral and anal
- **Ivabradine -**
  - Funny current inhibitor exclusively in the SA node - reducing the HR overall

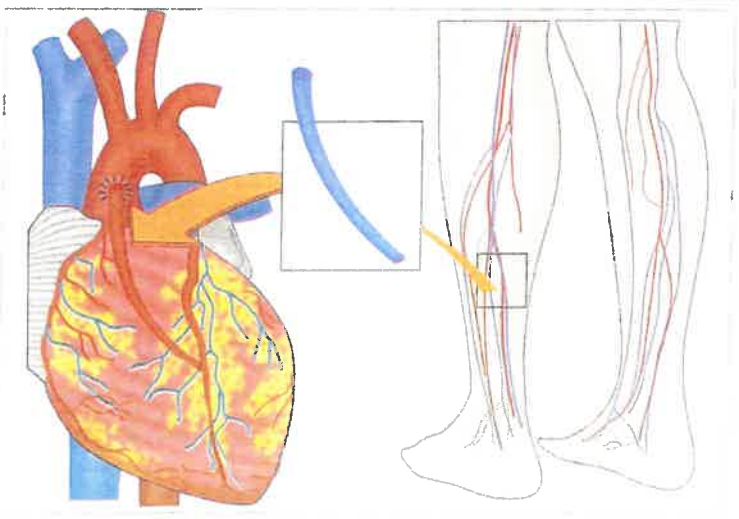
- Side Effects - Bradycardia, visual - luminous phenomenon
- At the time of initiation - HR >70 + Sinus rhythm

- **Trimetazidine -**
  - PFOX inhibition
  - Side effects - movement disorders (like chorea, parkinsonism)
- **Others: EECP -**
  - Indication - refractory angina
  - Wrap pneumatic cuffs around the legs and inflate sequentially during diastole resulting in ↑ in diastolic pressure →↑CBF
  - Long term effect is by VEGF - cause neovascularization and angiogenesis
  - Sessions - 1hr/day x 5d/week x 7 weeks = 35 sessions/ 35hrs

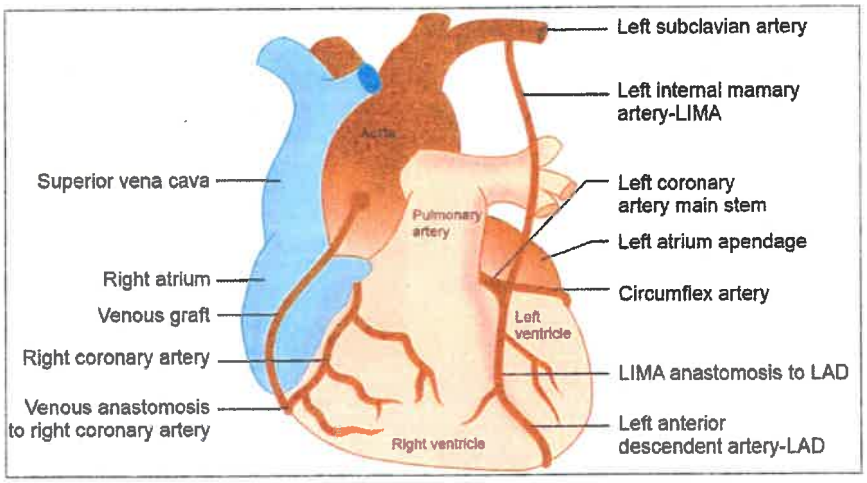


- **Revascularization:**
- **Indications:**
  - LMS disease- regardless of angina→ Absolute indication for CABG - improves survival
  - Severe/refractory angina (CABG vs PCI), CABG if -
    - 3VD PLUS DM/SYNTAX > 22
    - 2VD with proximal LAD PLUS DM/ SYNTAX > 22
- **CABG Grafts:**

| Types of Graft | Name | Patency | Atherosclerosis | Comments |
|---|---|---|---|---|
| **SVGs** | Free | Low (only 50% patent at 10 years) | Yes | ~10% occlusion rates in 1st month<br>Can develop Saphenous neuralgia |
| **LIMA** | In situ | High (90% at 10 years) | No | Most remain patent for life!<br>LIMA does NOT develop atherosclerosis |
| **RIMA** | Free | High | No | Risk of sternal wound infection |
| Radial<br>Free | | Average | Yes | High rates of vasospasm and can develop atherosclerosis |

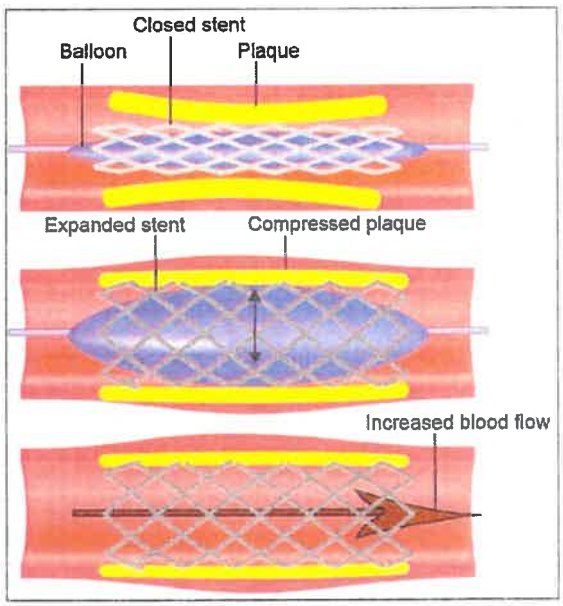


Free SVG Graft




**LIMA in Situ Graft**

## Stent & Types




1st a wire is pushed through the stenotic lesion and then a balloon is placed via a catheter in that area.

By inflating the balloon, the stent expands and pushes the plaques to the sides.

Once the stent is expanded, it will consolidate the area so that the vessel won't go under re-stenosis, so the blood flow will be restored and be patent for a long period of time.

## 2 types of Stent -

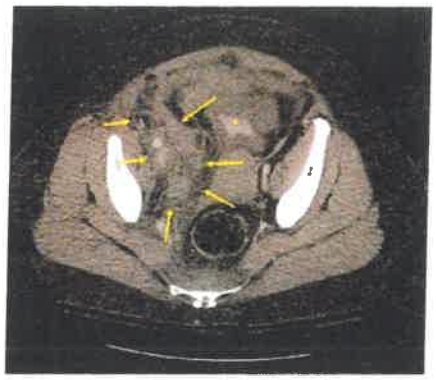
| BMS | Drug regulatory stent |
|---|---|
| No drug coating | Drug coating with mTORi = Sirolimus/ Everolimus/ Paclitaxel |
| **Advantage** -<br>Endothelium will proliferate and cover the stent<br>Faster endothelization - risk of thrombosis if for a short period of time, so can use short DAPT - Aspirin + P2Y12ri (Clopidogrel/ Prasugrel/ Ticagrelor) for > 1month. After 1 month, monotherapy can be given i.e., Aspirin.<br>**Disadvantage** - BMS stent can continuously stimulate the endothelium which can aggressively cause neointimal proliferation resulting in high rates of restenosis. | These stents are coated with drugs like sirolimus which will inhibit the endothelial proliferation.<br>Slow endothelisation - use of longer DAPT for a period of >6mos and later continue with monotherapy.<br>Longer duration of DAPT - lower rates of Restenosis |

### Post PCI complications

| Complication | Features | Rx |
|---|---|---|
| Vascular access complications | | |
| Groin hematoma | Groin swelling | Most resolve with manual compression (consider AC reversal) |
| Retroperitoneal bleed | ↓ Hct ± back pain, ↑ HR & ↓ BP (late). CT abdomen is suspected | ABC's + reverse/stop AC |
| Vascular damage | | |
| Pseudoaneurysm | triad of pain, expansile mass, systolic bruit. Diagnosed with US. | Treat if painful Rx or size >2 cm Options - directed compression, thrombin injection for surgical repair |
| AV fistula | Continuous bruit. (US can Diagnose) | surgical repair if large or symptomatic |
| LL ischemia (emboli, dissection, clot) | Classic ALI (6Ps) Urgent Angio (CTA/DSA) if suspected | Percutaneous or surgical repair |

## PCI - other complications

- Peri-PCI MI - type 4A MI (troponin ↑≥5x URL)
- CI-AKI
- Cholesterol emboli syndrome (intact pulses)
- In stent thrombosis - type 4B
- In-stent restenosis



### Preop management of antiplatelets

| Agent | D/C Prior to Surgery |
|---|---|
| Clopidogrel | 5 days |
| Prasugrel | 7 days |
| Ticagrelor | 5 days |
| 2B/3A | 3 hours |
| Enoxaparin | 12-24 hours |
| Fondaparinux | 24 hours |
| Bivalirudin | 4 hours |

## Note on vasospastic angina

- Most are smokers, relatively young and have resting angina (may be severe)
- Criteria:
  - Resting angina (typically at night & lasts 5-15 mins) & nitrate responsive
  - Transient ST Δ (ST↑ or ST↓)
  - Coronary artery spasm on CAG (>90% constriction) - can do optional provocative testing with Ach/ergonovine/ hyperventilation
- Ix:ECG and angiography (invasive or CTA) to r/o CAD
- Rx:CCBs (NDHP) & Nitrates
- Low dose ASA can be used of ASCVD (but carefully used since it can ↓ PGI2 production at high doses & worsen vasospasm)
- Drugs to be Avoided:non-selective βBs, triptans and 5-FU
- Long term prognosis is good (survival ~ 95% at 5 years).

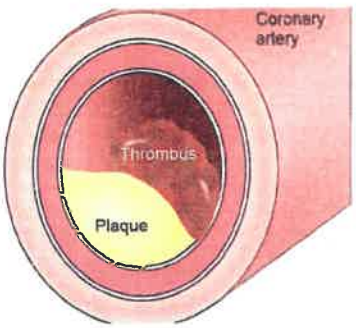
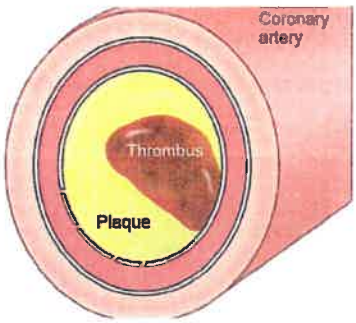
# 1.3 Chapter

# ACUTE CORONARY SYNDROMES

## ACS - Basics

Reasons for the instability of the plaque

- Plaque erosion (< 1/3rd)
- Plaque rupture (most common - >2/3rd) → thrombus formation → Acute ischemia → ACS
- Types of ACS: -

| Spectrum | UA | NSTEMI | STEMI |
|---|---|---|---|
| **Coronary thrombosis** | Subtotal occlusion - some flow   Multifocal & Subendocardial Ischemia | | Total Occlusion - No Flow   Focal & Transmural Ischemia |
| **History** | New onset angina, resting or crescendo angina | | Angina at rest |
| **Patient phenotype** | Relatively old with multiple comorbidities | | Relatively young with limited comorbidities |
| **Disease** | Typically, multivessel disease in most, ↑ collaterals | | Single culprit vessel, ↓ collateral formation |