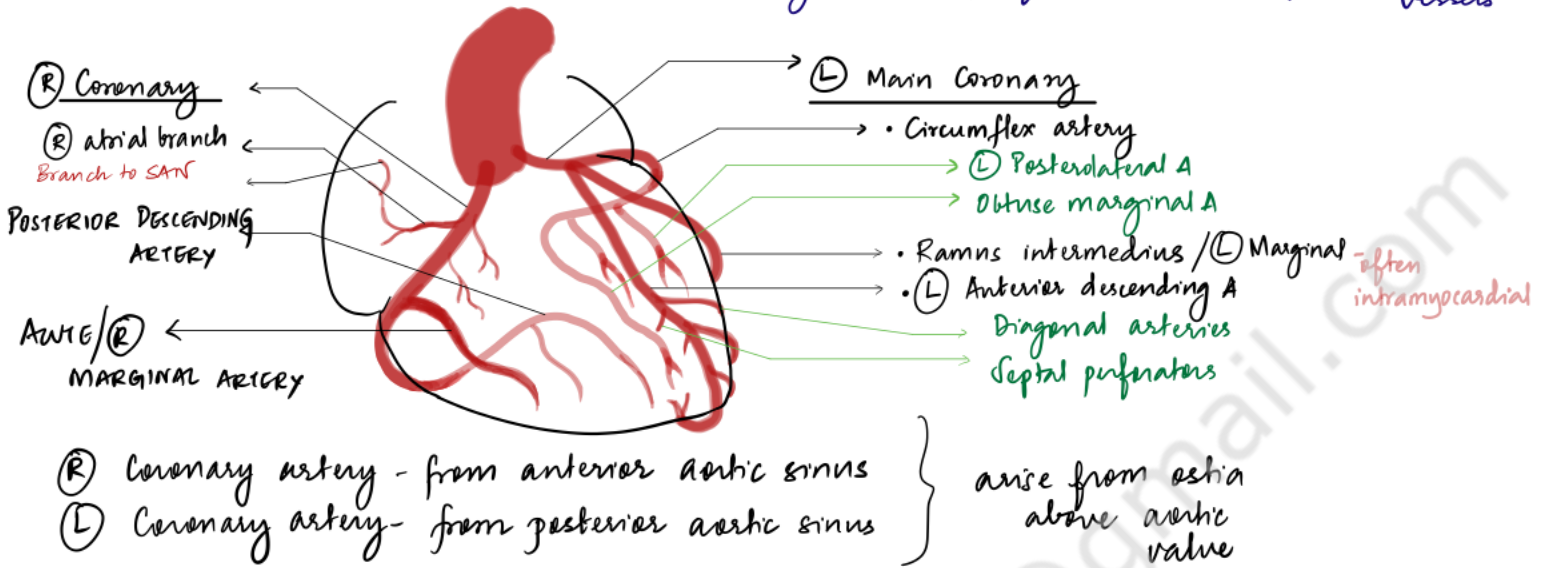


# CORONARY ARTERY DISEASE

CORONARY CIRCULATION

- Epicardial conductance vessels
- Intramyocardial perforator vessels / resistance vessels



3 VESSELS = LAD, RCA, CIRCUMFLEX (CX)

(L) Main Coronary / Left main stem disease - WORST PROGNOSIS out Sp  
(2-3cm)

Inaccessible at its origin

Grafts are anastomosed to its branches (LAD/OM)

LAD - most frequently diseased / most frequently bypassed during CABG

→ > 50% of LV MASS + interventricular septum

## CORONARY DOMINANCE

- Based on which coronary artery gives off Posterior Descending A

90% - RCA → PDA ⇒ (R) Dominance

## VENOUS DRAINAGE - 3 systems

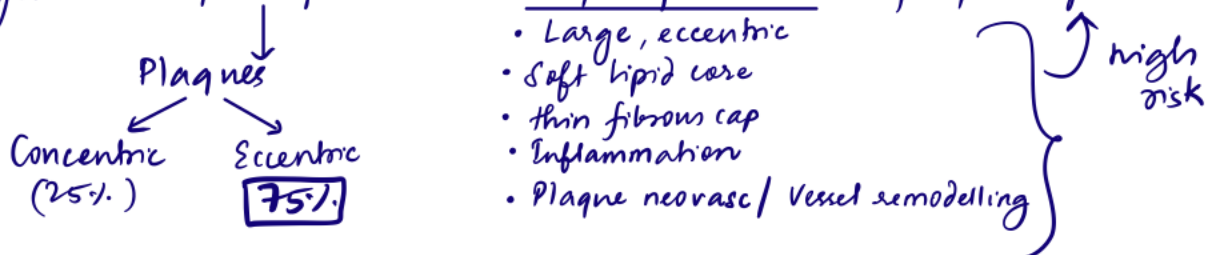
- 1) Coronary sinus & its tributaries → mainly drains LV
  - 2) Anterior (R) ventricular veins → RA
  - 3) Thebesian veins → RA + RV
- 85% coronary venous blood
- opens into RA

## CORONARY ARTERY DISEASE - Coronary atherosclerosis

Risk factors: Dyslipidemia, Smoking, HTN, T2DM etc.

Affects EPICARDIAL CONDUCTANCE VESSELS > INTRAMYOCARDIAL VESSELS

Pathogenesis: Lipid deposition → Plaque formation → plaque rupture → thrombosis



# IHD Clinical Manifestations

## STABLE ANGINA

## ACUTE CORONARY SYNDROME

- UNSTABLE ANGINA
- NSTEMI
- STEMI

## Evaluation of IHD

### NON-INVASIVE

- 1) Resting ECG - 12 lead
- 2) Troponins & Cardiac isoenzymes
- 3) Exercise tolerance testing (ETT) - TMT / Pharmacological testing
- 4) Echo - LVEF, RWMA, valvular defects, LV aneurysm, LV thrombus  
SPECT -  $Tc99m$
- 5) Radionuclide studies - Myocardial Perfusion scan - Thallium 201
- 4) Cardiac MRI  $\pm$  Gadolinium - evaluation of 'ischemic burden'
- 5) Cardiac CT - Proximal CAD  
Degree of Coronary artery calcification (Calcium score)

### INVASIVE

- 1) Coronary angiography - 'Gold standard'

Extent - one vessel / 2-vessel / 3-vessel

Severity - % luminal reduction ( $>70\%$  - significant;  $50\%$  for left main & proximal LAD)

Location

Distal coronaries - quality, size, collaterals

Functional Flow Reserve (FFR) - additional data about physiological effects of stenosis by passing a sensor guidewire into vessels - injection of adenosine to induce hyperemia

$FFR < 0.75 \rightarrow$  Ischemic lesion

- 2) Intravascular Ultrasound

- 3) Hybrid imaging - Combined CT Angio + PET / SPECT

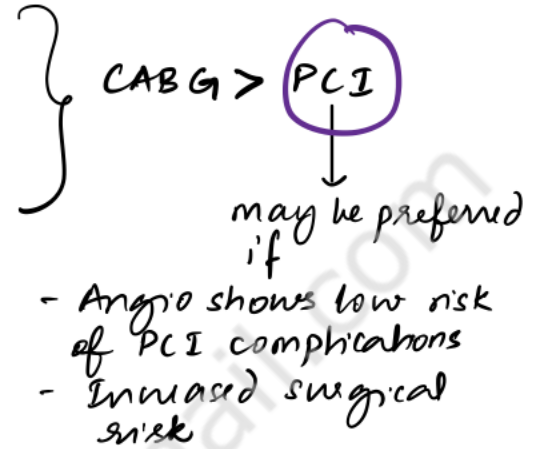
# CORONARY ARTERY REVASCULARIZATION < CABG PCI

## INDICATIONS FOR CABG - in Stable IHD

- 1) Left main vessel disease (CABG - Gold std)
- 2) 3 vessel disease ± proximal LAD involvement  
CABG ≈ PCI
- 3) 2 vessel disease + proximal LAD involvement  
especially in T<sub>2</sub>DM

Completeness of revascularisation is  
BETTER with CABG

- 4) Sudden Cardiac Death Survivor → CABG = PCI  
(Post MI arrhythmia)



## Revascularization in Acute Coronary Syndrome

- PCI has superseded Emergency CABG
- CABG is not usually performed in isolation - combined with an operation to treat a specific complication
  - Refractory post infarction angina
  - Papillary muscle rupture ± MR
  - Infarction VSD (septal rupture)
- Acute failure of PCI

## PERCUTANEOUS CORONARY INTERVENTION

Percutaneous access - femoral A / Radial A puncture - Seldinger technique

Guidewire → Sheath introducer → Guiding catheter advanced to coronary A → Anticoag advanced beyond blockage → Angiogram by injecting dye - tip of catheter advanced beyond blockage → Balloon is inflated, pull back

- Stent (Bare Metal Stent / Drug eluting stent) deployed

Paclitaxel, Sirolimus  
Everolimus

Primary PCI - done in ACS on emergency basis

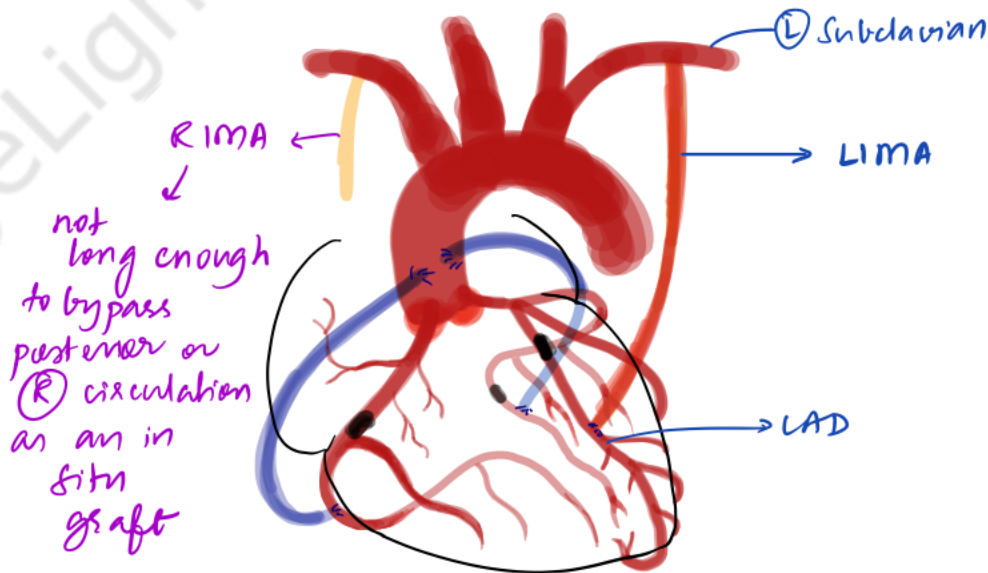
# CORONARY ARTERY BYPASS GRAFTING

## A- CHOICE OF CONDUITS

- 1) **Internal thoracic artery** - highest patency rates 98% @ 5y  
85-90% @ 10y  
(mammary)  
Proximally left attached to the subclavian (N)  
Distally anastomosed to the target coronary vessel  
Generally used to bypass the LAD (anterior circulation)  

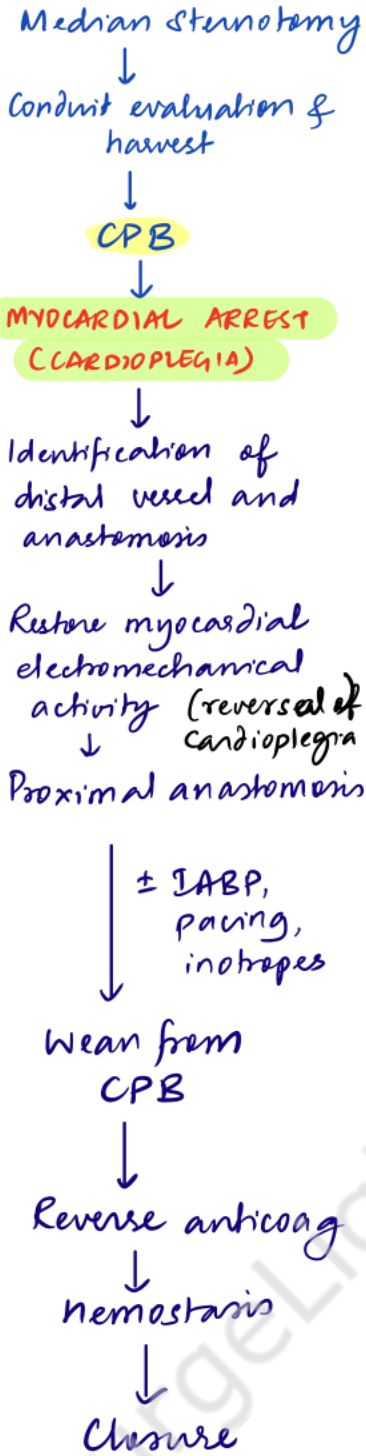
Ⓛ > B/L  
'LIMA' 'BIMA'

RIMA - closely related to phrenic N
- 2) **Greater saphenous vein** - 90% patency at 1y (60-70% at 10y)  
harvest technique  
open Endoscopic  
ensure minimal manipulation during harvest
- 3) **RADIAL ARTERY** (after confirming satisfactory ulnar collateral flow)  
open Endoscopic harvest → better patency than GSV  
RCA & Cx arteries → commonly bypassed using GSV & RA
  - can be made into composite T & Y grafts
  - can be sewn sequentially
- 4) Rarely used conduits - SSV  
Gastroepiploic artery  
Cephalic vein

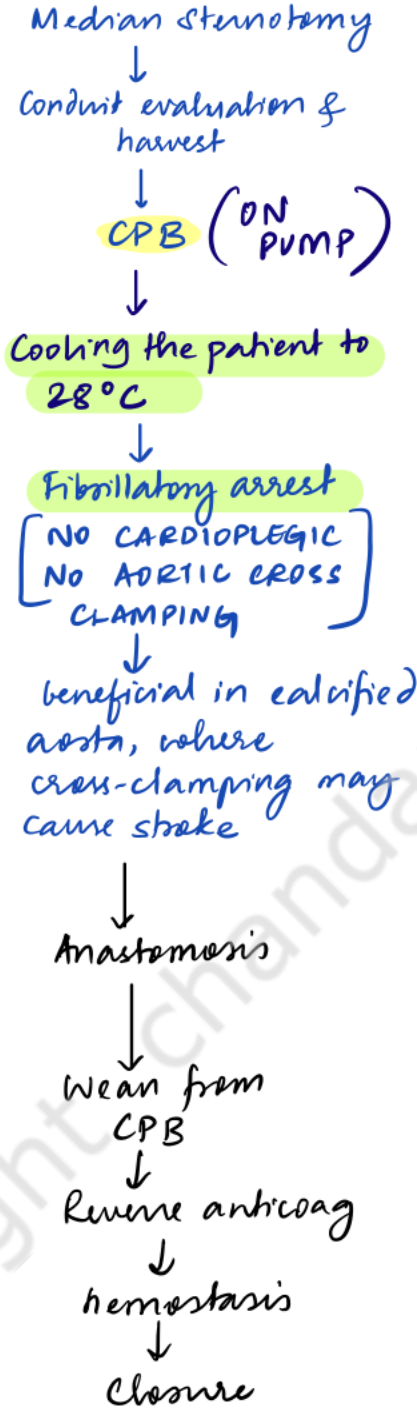


# APPROACHES TO CABG

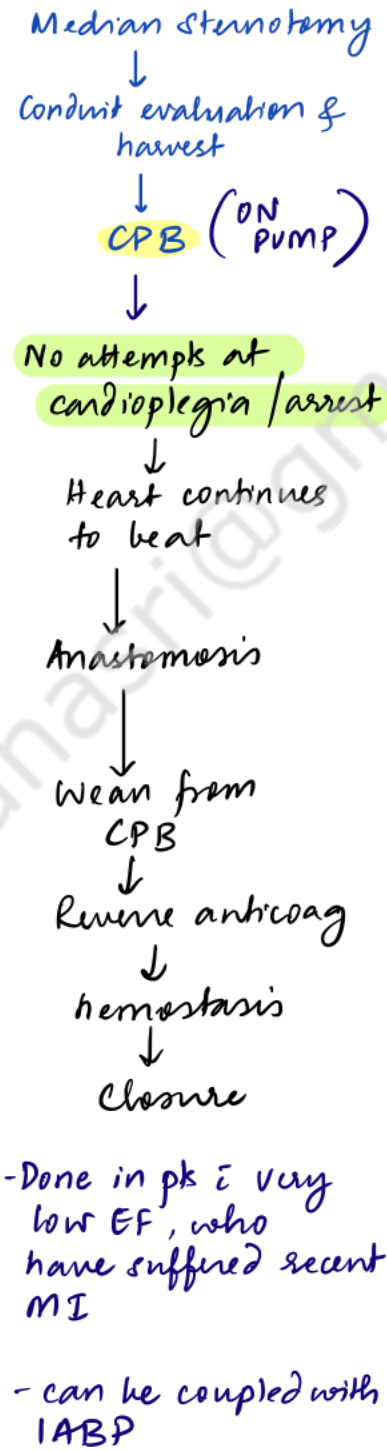
## CLASSICAL CABG



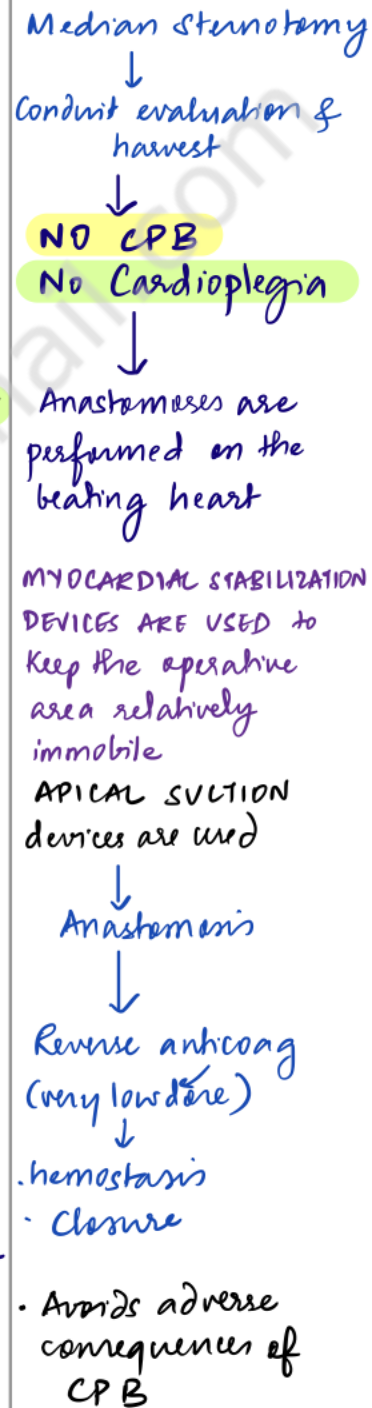
## CABG & HYPOTHERMIC FIBRILLATORY ARREST



## ON-PUMP BEATING HEART BYPASS



## OFF-PUMP CABG



CARDIOPLEGIC ✓

• NO CARDIOPLEGIC  
• HYPOTHERMIC ARREST

NO ARREST

CARDIOPULMONARY BYPASS ✓

NO  
CPB!

## NOVEL TECHNIQUES

- MIDCAB - Minimally invasive direct Coronary artery bypass

- extension of off pump CABG
- ⊕ anterior ministhrothotomy

↓  
mobilisation of LIMA

↓  
direct in-situ anastomosis to LAD / diagonal branches

→ for single vessel disease mainly

- Totally Endoscopic Coronary Artery Bypass (TECAB)

- Robotic assisted CABG

- Hybrid Coronary revascularization

PCI + MIDCAB / TECAB

- Transmyocardial Laser revascularisation

CO<sub>2</sub> / Holmium: Yttrium Al. garnet laser

↓  
create multiple transmural channels

(1mm wide)

↪ conduits for direct myocardial perfusion by ventricular blood

↪ angiogenesis

## COMPLICATIONS OF CABG

- 1) Bleeding

- early post op cardiac tamponade

- 2) Arrhythmias → R - drugs / cardioversion / pacing

- 3) Myocardial dysfunction

- ischemia/reperfusion injury - R - oxygenation  
inotropes

- 4) Stroke - Embolic

correcting ABG

- 5) Wound infection

pacing  
IABP

# CARDIOPULMONARY BYPASS

Gibton, 1953

CPB is the establishment of extracorporeal oxygenation and perfusion of the body

- All the venous blood returning to the heart

↓  
Diverted to a heart lung machine

↓  
Passed through an oxygenator

↓  
- Oxygenated blood is returned to the body in a controlled, pressurized manner

## STEPS

### • VENOUS CANNULATION

- RA + IVC
- SVC, IVC separate cannulation → RA surgeries
- Femoral vein → thoracic aorta procedures, minimally invasive procedures

• Blood is drained via gravity into a venous reservoir - Blood from cardiotomy suction pump can also be added

• Passed through heat exchanger & oxygenator

• Oxygenated blood is passed via centrifugal pump and reintroduced into body via arterial line  
↳ 2000-5000 rpm

### • ARTERIAL CANNULATION

Ascending aorta → avoided in aortic dissection (inadvisable)  
Femoral artery → aortic root surgery (impractical)  
Axillary artery → severe adhesions (impossible)

• A parallel circuit draws blood from the oxygenator outlet → mixes it with cardioplegic solution (4°C) in the ratio 4:1

• Cardioplegic is delivered to protect ischemic myocardium from cell death

- Anterograde → into aortic root → coronary artery → Myocardium
- Retrograde → Coronary sinus → Myocardium

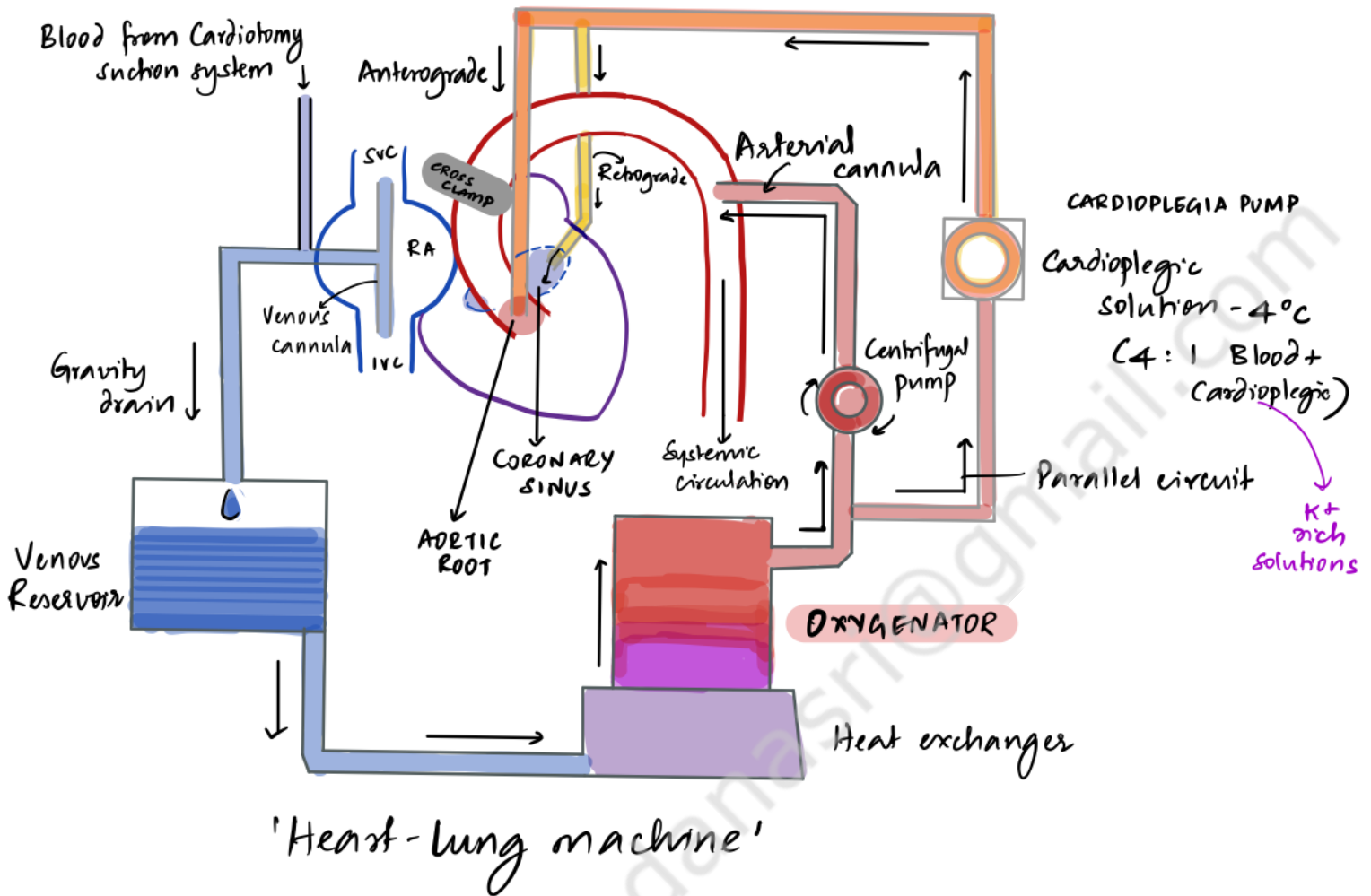
↳ better in CAD

- Additional - Anticoagulation - Heparinization (Reversal i Protamine SO<sub>4</sub>)  
Priming solution - Balanced salt solution/plasma/colloid  
Blood & blood products etc.

- SYSTEMIC HYPOTHERMIA - to reduce metabolic demand  
- for neuroprotection

↳ O<sub>2</sub> consumption falls by 50% for every 10°C ↓ in temperature

# CPB



## Uses

- For CABG
- Valvular repair / replacement
- Septal surgery
- Congenital heart disease surgery
- Transplantation - heart, lung, liver
- Aneurysm repair
- Pulmonary thrombectomy / thromboendarterectomy

## Adverse effects of CPB

- 1) Complement activation → systemic inflammation
- 2) Heparinization → Heparin Induced Thrombocytopenia
- 3) Hypoperfusion, embolization → Oxygenator failure
- 4) Aortic dissection - cannulation



# CARDIAC ARREST & CARDIOPULMONARY RESUSCITATION

CARDIOVASCULAR COLLAPSE	CARDIAC ARREST	SUDDEN CARDIAC DEATH
<p>Sudden loss of effective circulation due to <b>CARDIAC/PERIPHERAL VASULAR FACTORS</b> that may reverse spontaneously (Neurogenic/vasovagal syncope) or may require intervention</p> <p>Broad term - includes both cardiac arrest &amp; transient events that can revert spontaneously</p> <p>- presents as 'SYNCOPE'</p> <p>Severe hypotension → cerebral hypoperfusion</p> <p>DI- Arrhythmia Myocardial/valvular dysfunction Loss of vascular tone Acute complete disruption of venous return Tension pneumothorax Cardiac tamponade</p>	<p><b>ABRUPT CESSATION</b> of cardiac function resulting in loss of effective circulation</p> <p>- Reversible by <b>PROMPT EMERGENCY MEDICAL INTERVENTION</b></p> <p>↓ leads to death otherwise</p> <p>DI+</p> <ul style="list-style-type: none"><li>- Ventricular fibrillation</li><li>- Ventricular tachycardia</li><li>- Asystole</li><li>- Bradycardia</li><li>- Pulsless electrical activity</li><li>- Non cardiac mechanical factors</li><li>- <b>PULMONARY EMBOLISM</b></li></ul>	<p>Sudden <b>UNEXPECTED</b> death attributed to Cardiac arrest, which, if witnessed occurs within <b>1 hr</b> of symptom onset</p> <p>Subject documented to be well in the preceding 24 h</p>

## MANAGEMENT OF CARDIAC ARREST

**GOAL** : to achieve Return of Spontaneous Circulation (ROSC)

### 1) Initial Evaluation

• Take <10s to assess if pulse is present

• Look for breathing → Gasping respirations } Common during seizure activity } SCA

↓  
can be mistaken for breathing & responsiveness

## 2) Initiation of CPR

Initiate chest compressions WITHOUT DELAY

- Generate cardiac output

• sequential filling & emptying of cardiac chambers

Rate = 100-120/min

Depress sternum by 2 inches  $\approx$  5 cm

Allow full chest recoil between compressions

Ventilation may be administered @ 2 breaths / 30 CCs  
if a second trained rescuer present

## 3) Rhythm based management

AED (Automatic External defibrillator)

Apply immediately

Non shockable rhythms

PEA / Asystole

CPR, Intubate, IV access

Epinephrine 1mg IV  $\text{Q } 3-5\text{min}$

ID & treat reversible causes

- Hypoxia
- Drug overdose
- Tamponade
- Pneumothorax

Bradycardia  $\curvearrowright$

- Atropine 1mg IV
- Pacing

shockable rhythm - VF/VT

150-200J biphasic shock

Resume CCs  $\times 2\text{min}$

Rhythm check

Shock again

- IV / IO access
- Advanced airway
- Epinephrine 1mg IV  $\text{Q } 3-5\text{min}$

IV amiodarone 300mg

Repeat 150mg

CPR, Repeat shock

## CARDIAC ARREST AFTER CARDIAC SURGERY

Non shockable rhythms → Tamponade  
Tension pneumothorax  
Severe hypovolemia



Shockable rhythms

Rule out / treat

3 failed cycles of CPR

Resternotomy

Failure

Atropine  
Pacing  
CPR

Internal Cardiac Massage



2 hand technique

Ⓜ hand over apex advanced posteriorly  
around the apex & palm up  
& hand flat

Ⓛ hand over anterior surface over heart

Flat palms, straight fingers

Compressions @ 100/min

- before initiating compressions,

- remove clots

- safeguard grafts

## AFTER ROSC....

- R/O and treat acute MI
- Optimize ventilation
- Optimize hemodynamics → SBP > 90 mmHg  
MABP > 65 mmHg
- Treat - Hypo & Hyperkalemia  
Drug toxicity  
Hypothermia

MAIN CONCERN: Cerebral hypoperfusion → Brain ischemia

Successful neurological recovery chances → < 30% @ 5min in absence of CPR

# HEART TRANSPLANT

Dr. Christian Barnard - 1967 - first human heart transplantation

## INDICATIONS

- m/c - cardiomyopathy  
 Ischemic heart disease } m/c  
 Valvular heart disease  
 Myocarditis  
 Congenital Heart Disease

in patients i end stage heart disease that has failed to respond to conventional therapy

- i predicted survival < 6-12m iout transplant
- iout irreversible damage to other organs

PRINCIPLE	COMMENT
<b>Advanced Disease Severity</b>	Refractory heart failure with a $VO_2$ of <14 mL/kg/min (<12, if on beta blockers) or percent predicted $VO_2$ <50%; combination of intolerance to disease modifying therapy, cardiorenal syndrome, use of inotropic therapy to maintain stability or need for a left ventricular assist system.
<b>Co-Morbidity</b>	Age is not an absolute contraindication, but frailty should be considered a relative contraindication; a BMI > 35 kg/m <sup>2</sup> should require weight loss; cancer should be dealt with on an individual basis (e.g., low-grade prostate cancer may not be a contraindication); poorly controlled diabetes mellitus or end-organ damage may be a contraindication; eGFR <30 mL/min/1.73 m <sup>2</sup> is a relative contraindication; severe cerebrovascular disease or peripheral vascular disease (which will limit rehabilitation or function) is also a relative contraindication.
<b>Donor-Recipient Matching</b>	Sensitized individuals with circulating antibodies should have a prospective or virtual cross match; pulmonary vascular resistance with a transpulmonary gradient >15, PVR >3 Wood Units and absolute PA systolic pressure >50 mmHg provided the systolic BP is >85 mmHg is a relative contraindication unless reactive.
<b>Psychosocial Issues</b>	Tobacco use in any form limits posttransplant survival and should be stopped for at least 6-months; substance abuse, including marijuana, should be a contraindication if the individual cannot demonstrate control and cessation; patients with severe cognitive-behavioral disabilities or dementia (inability to ever understand and cooperate with medical care) have the potential for self-harm and should not receive a transplant.

Definition of 'Advanced Cardiac Failure'

Generally ≤ 65y

health of other organ systems

Treat dental infections to prevent infective endocarditis

- Substance abuse

- Psychological conditions

Abbreviations: BMI, body mass index; eGFR, estimated glomerular filtration rate; PA, pulmonary artery; PVR, pulmonary vascular resistance;  $VO_2$ , peak oxygen consumption.

## DONOR CRITERIA

Age  $\leq 55$

No CAD

LVIH

Severe injury to allograft

Donor - recipient size match

Because cold ischemia time is  $\sim 4$ h -

ABO matching - decisive  
HLA matching difficult

Generally DBD  $>$  DCD

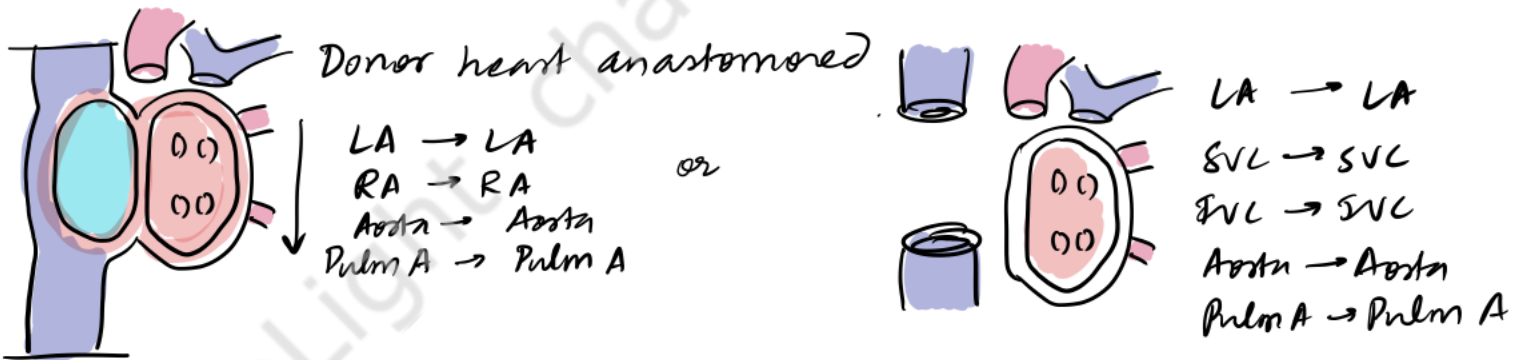
## PROCEDURE

Heart transplants - ORTHOTOPIC, BICAVAL (Piggy back heterotopic very rare)

- Median sternotomy
- Cardiopulmonary bypass

Recipient's native heart is removed leaving behind

- SVC
- IVC
- Aorta
- Pulmonary Artery
- Atrial cuffs & pulmonary veins



After anastomosis  $\rightarrow$  remove bypass  $\rightarrow$  Inotropes

Combined heart lung transplants for Pulmonary vascular disease & Cardiac dysfunction

Eisenmenger Syndrome

Heart disease & Pulmonary HTN

Idiopathic pulmonary fibrosis

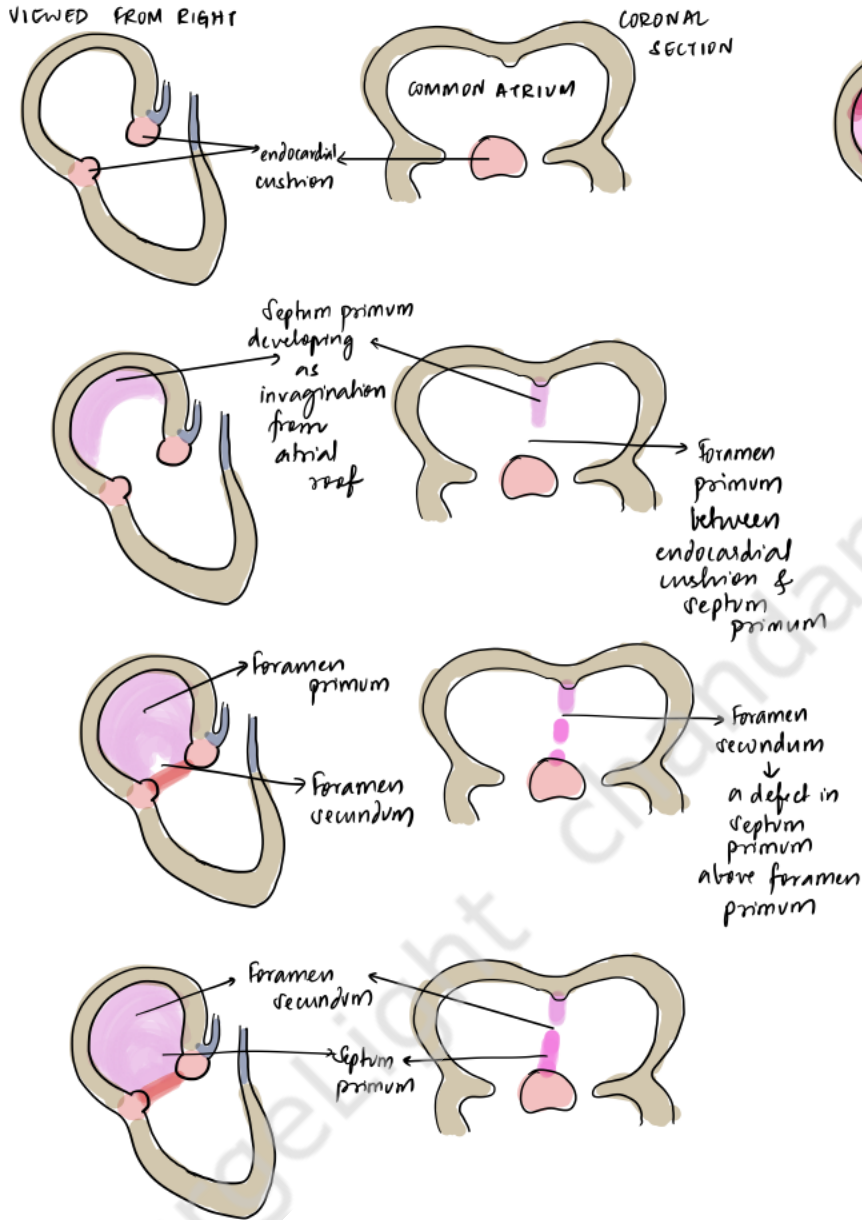
Primary Pulmonary HTN

# ATRIAL SEPTAL DEFECT

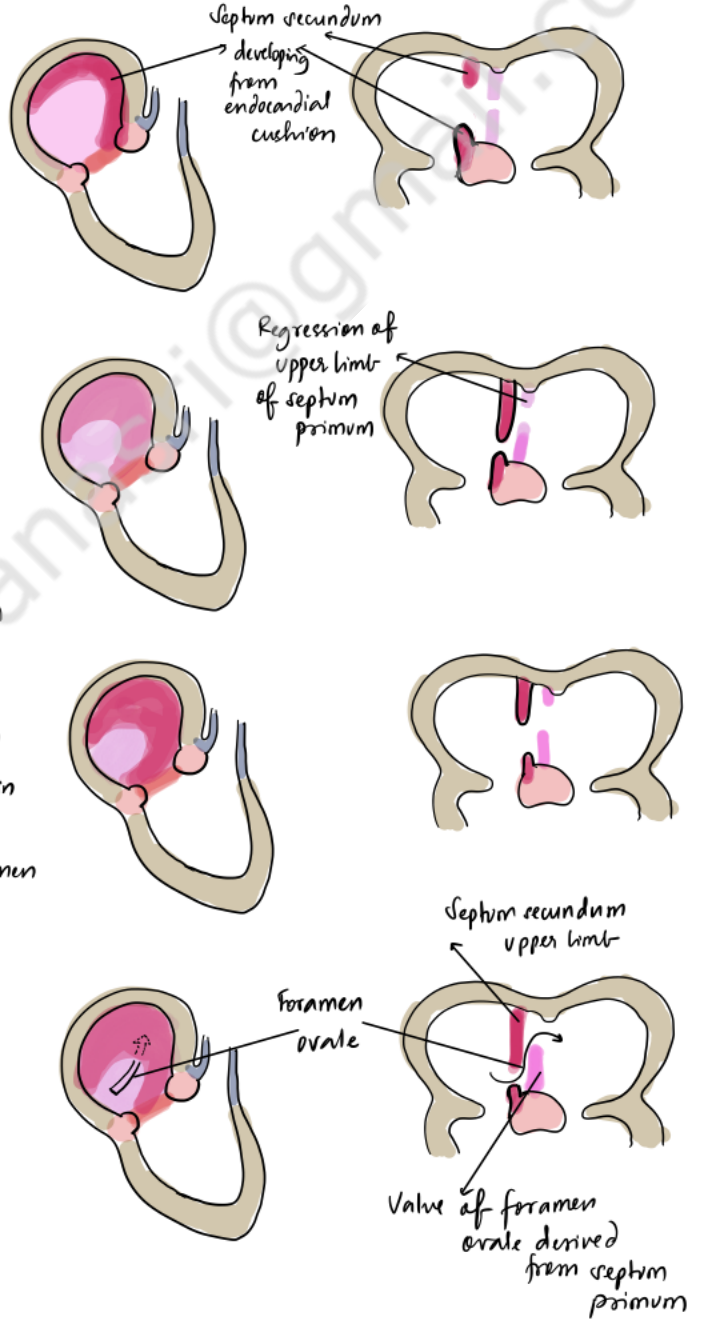
Defect in the interatrial septum

## EMBRYOLOGY

### DEVELOPMENT OF SEPTUM PRIMUM



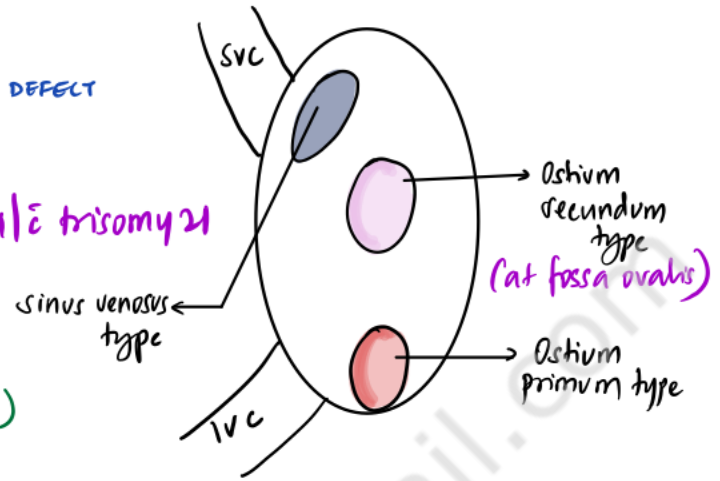
### DEVELOPMENT OF SEPTUM SECUNDUM



'Patent foramen ovale is not an ASD'

## TYPES OF ASD

- 1) **Ostium Secundum Type** - m/c - 80% - FOSSA OVALIS DEFECT  
- due to ↑ Resorption of septum primum or ↓ growth of septum primum
- 2) **Ostium Primum** - ATRIOVENTRICULAR DEFECT → a/c trisomy 21  
- due to failure of fusion of septum primum & endocardial cushion
- 3) **Sinus Venosus Type** - at SVC entry  
a/c anomalous pulmonary veins
- 4) **Rare types** - IVC defect (low sinus venosus defect)  
Coronary sinus septal defect



Primum defect presents earlier than secundum defect

DYSPNEA, RECURRENT CHEST INFECTIONS, PULMONARY HYPERTENSION

## PATHOPHYSIOLOGY

- $\text{L} \rightarrow \text{R}$  shunt is transient  $\text{R} \rightarrow \text{L}$  shunting in the times of ↑ Intrathoracic pressure  
↓  
• heart overload → ↑ Pulmonary blood flow → PHN → shunt reversal
- PARADOXICAL EMBOLIZATION → CVA risk

## MANAGEMENT

Simple secundum type ASD - transcatheter closure is atrial closure device

Larger defects - open surgical approach - CPB - primary/patch closure is pericardial/synthetic patch

Primary AV defect repair → + Mitral valve repair

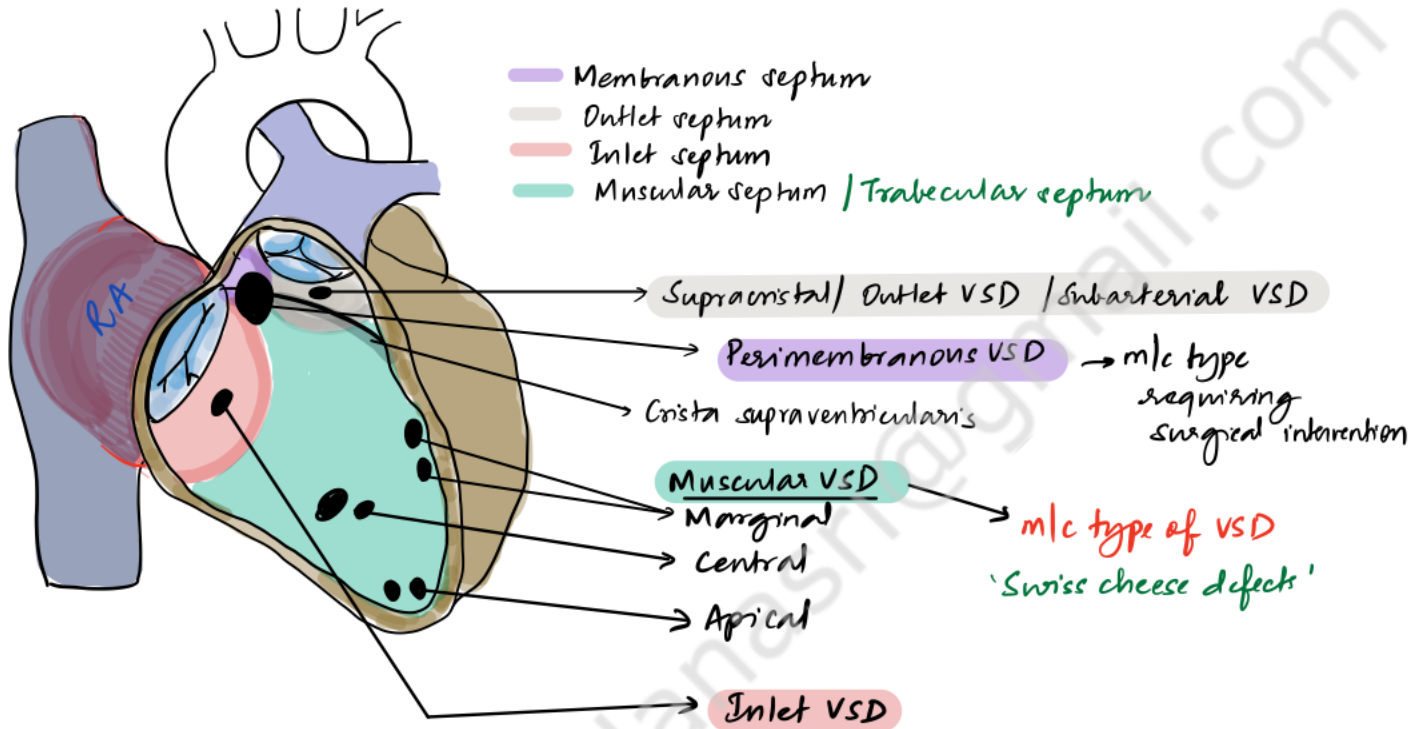
CLOSURE IS TAKEN UP IN 1<sup>ST</sup> DECADE EVEN IN THE ABSENCE OF SYMPTOMS TO PREVENT RISK OF

- RV Failure
- Endocarditis
- Paradoxical emboli

# VENTRICULAR SEPTAL DEFECT

VSD - pathological communication between the ventricles & a defect in the interventricular septum

TYPES OF VSD - based on location



Right ventricular view of interventricular septum

## PATHOPHYSIOLOGY

Depends on size of VSD

Large / Non restrictive VSDs

are atleast as large as aortic annulus

Allow free flow of blood from LV to RV

↑ RV pressure → Pulmonary hypertension  
↓ Congestive heart failure

Shunt reversal  
→ Eisenmenger's

Small / Restrictive VSDs

offer significant resistance to passage of blood through defect  
(LOUDER MURMUR)

↓ physiologic consequences

**BVT**  
- endocardial damage & high pressure jet through the defect  
↓  
Endocarditis



## Evaluation

CXR - Cardiomegaly  
Pulmonary plethora

Echo - Biventricular hypertrophy, defect size, flow - degree of shunting  
endocarditis

## TREATMENT

Percutaneous closure - Device closure (AMPLATZER)

↳ utility limited if/so paramembranous defects - 2/3 proximity of conducting system  
↳ Risk of embolization  
↳ RISK OF HEART BLOCK

Surgical closure → Primary mode

CARDIOPULMONARY BYPASS

Patch - autologous pericardial graft  
Dacron  
PTFE

Multiple / Swiss-cheese defects - cannot be repaired in infancy

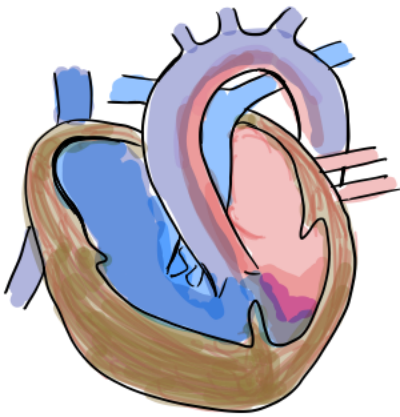
↳ R - temporary placement of pulmonary arterial band to control pulmonary flow

# TETRALOGY OF FALLOT

- Cyanotic congenital heart disease
- m/c cyanotic congenital heart disease found in children surviving to 1 year

- ① VSD - large perimembranous VSD adjacent to the tricuspid valve
- ② Over-riding aorta - aorta overrides the interventricular septum
- ③ RV outflow tract obstruction - hypoplasia/dysplasia of pulmonary valve  
- obstruction at the subvalvular/pulmonary artery level
- ④ RV Hypertrophy

Primary defect → Malposition of interventricular septum



## Pathophysiology

Presentation depends on severity of RVOTO

Severe pulmonary annular hypoplasia  
→ Cyanosis at birth

• m/c - significant cyanosis - 6-12m of birth  
due to worsening RVOTO & progressive RVH

## Presentation

- 'Tet spells' - triggered by stimuli causing ↓ SVR - fever/agitation/vigorous activity  
↳ worsening of R → L shunt → systemic hypoxemia  
SQUATTING → ↑ SVR → relief - Rx: Propranolol (↓ inotropy) & adrenergic agonists (↑ SVR)
- Clubbing
- Polycythemia
- Infective endocarditis
- Brain abscesses
- RVF → Congestive cardiac failure
- Dysrhythmias

## Evaluation

- CXR - Boot-shaped heart - Coarctation
- ECG - fls/0 RVH
- Echo
- ? Aortography to delineate coronary anatomy

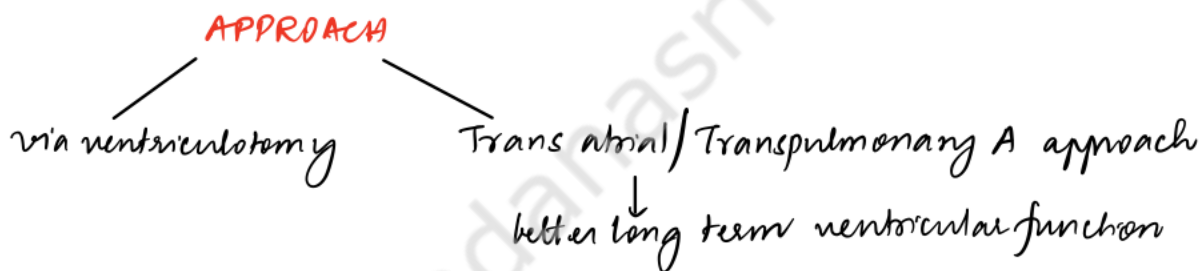
## SURGERY

### Correction

- Patch closure of VSD
- Relief of all levels of RVOTO + Pulmonary stenosis

Post repair pulmonary regurgitation possible

- Pulmonary valve repair
- Pulmonary valve implantation



### Palliative procedures

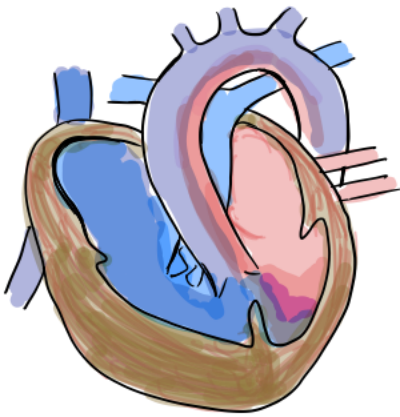
- Blalock - Taussig shunt - **SYSTEMIC → PULMONARY ARTERY SHUNT**  
↓  
Subclavian to pulmonary artery anastomosis

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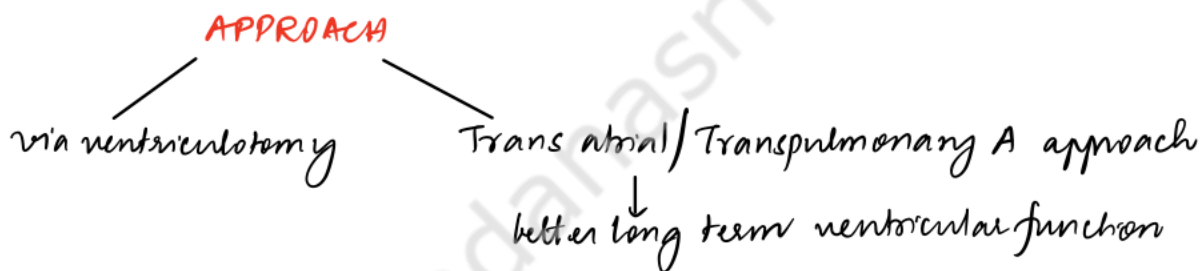
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↓  
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# COARCTATION OF AORTA

6-7% of congenital heart disease  
Boys  $\uparrow\uparrow$ ; Girls  $\in$  TURNER'S SYNDROME

Luminal narrowing in the aorta causing an obstruction to blood flow  
 $\rightarrow$  m/c located distal to the  $\textcircled{L}$  subclavian artery

EMBRYOLOGIC BASIS ————  $\textcircled{2}$  THEORIES

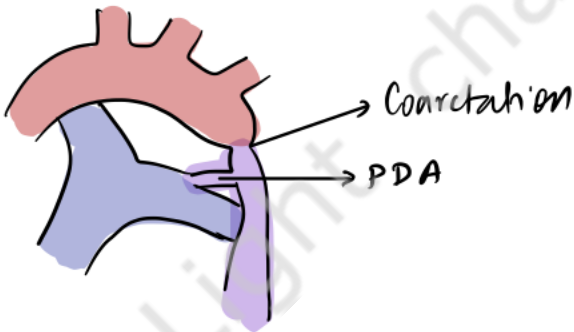
$\textcircled{1}$  d/t an obstructing shelf composed predominantly of tissue from the ductus, forms as the ductus involutes

$\textcircled{2}$  diminished aortic isthmus developing secondary to  $\downarrow$  aortic flow in infants  $\in$  enhanced ductal flow

## TYPES

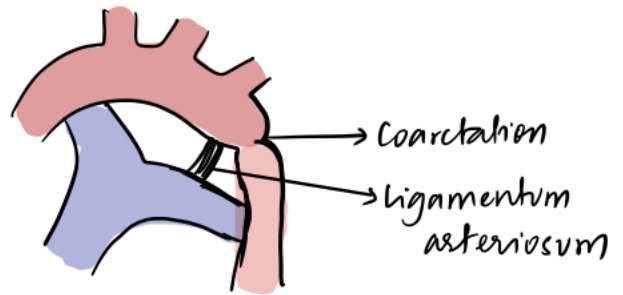
INFANTILE / PREDUCTAL

a/c LARGE PDA - patient depends on patent duct for systemic circulation until the coarctation is corrected



POSTDUCTAL / ADULT COARCTATION  
JUXTA / POST DUCTAL

the coarctation is present at/distal to the ligamentum arteriosum



## PATHOPHYSIOLOGY

• Development of extensive collateral circulation involving

- Intercostal arteries
- Internal mammary arteries

$\rightarrow$  Rib notching

$\rightarrow$  Prominent pulsation underneath ribs

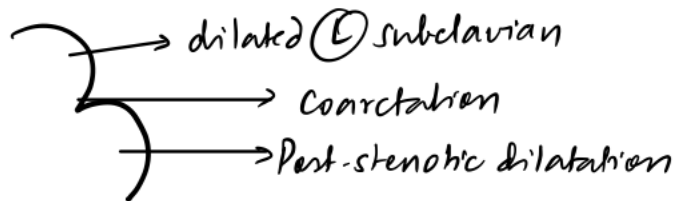
• Good v/l & cerebral perfusion; BUT  $\downarrow$  perfusion of lower body

$\downarrow$  Renal hypoperfusion  $\rightarrow$  RAAS  $\rightarrow$  hypertension

## Clinical features

- V/L / UL pressure gradient; Radial/radial / radial/femoral delay
- Continuous murmur - posterior thorax - flow murmurs
- Hypertension in older children

CXR - 3 sign replacing aortic knuckle



ECHO, DOPPLER - LV, collaterals, aortic anatomy

## Complications - Hypertension

- heart failure
- infective endocarditis
- Aortic rupture
- Hemorrhagic stroke (H+IV)

## MANAGEMENT

- Balloon dilatation - NOT PREFERRED - ↑↑ rate of re-coarctation
- Primary stent implantation - transcatheter

## OPERATIVE REPAIR - GOLD STANDARD

Sternotomy / (L) thoracotomy - patch aortoplasty (Dacron or subclavian artery flap)

- or -

Resection of coarctation segment  
+ end to end anastomosis

## AORTIC DISSECTION

- progressive separation of the aortic wall layers, following a tear in the tunica intima and media - permitting blood to create a false channel in the aortic wall typically between tunica media and adventitia

True lumen (lined by tunica intima) and the false lumen (between media & adventitia or layers of media) are separated by dissecting membrane

RE-ENTRY SITES: additional tears in the dissecting membrane which allow communication between true and false lumens

DISSECTION CAN PROCEED  $\left\{ \begin{array}{l} \text{DISTALLY - mlc} \\ \text{PROXIMALLY - retrograde dissection} \end{array} \right.$

### CONDITIONS ASSOCIATED TO DEVELOPMENT OF AORTIC DISSECTION

- 1) Connective tissue disorders - Marfan Syndrome
- 2) Hypertension - Atherosclerosis, hypercholesterolemia, smoking
- 3) Pregnancy
- 4) Cocaine abuse, Amphetamine abuse
- 5) Power weight-lifting
- 6) Aortic injury during endovascular procedures

### CONSEQUENCES OF AORTIC DISSECTION

- 1) Extension  $\left\{ \begin{array}{l} \text{:} \text{ Outer wall of the false lumen - extremely thin, inflamed, fragile} \\ \text{2) Rupture} \end{array} \right. \rightarrow \text{in the setting of ongoing hemodynamic stress}$
- into pericardial sac - cardiac tamponade
- into thoracic cavity -  $\ominus$  hemothorax

3) MALPERFUSION SYNDROMES -  $\Delta$ lt compression and compromise of the true lumen by the expanding false lumen

- Renal artery - Renal pain, Renal failure
- Mesenteric arteries - abdominal pain, bowel ischemia
- Spinal arteries - paraplegia
- Iliac arteries - limb ischemia - distal pulse deficits / Radial-radial discrepancy
- Head & neck vessels - CVA
- Coronary vessels - MI

4) Acute aortic valvular regurgitation -  $\Delta$ lt separation of layers within the aortic root  $\rightarrow$  aortic valve commissures become unbringed  $\rightarrow$  Dyspnea, murmur, crept, shock

Ddx: 1) INTRAMURAL HEMATOMA - blood collects within media  $\Delta$ lt hemorrhage from vasa vasorum; INTIMA INTACT

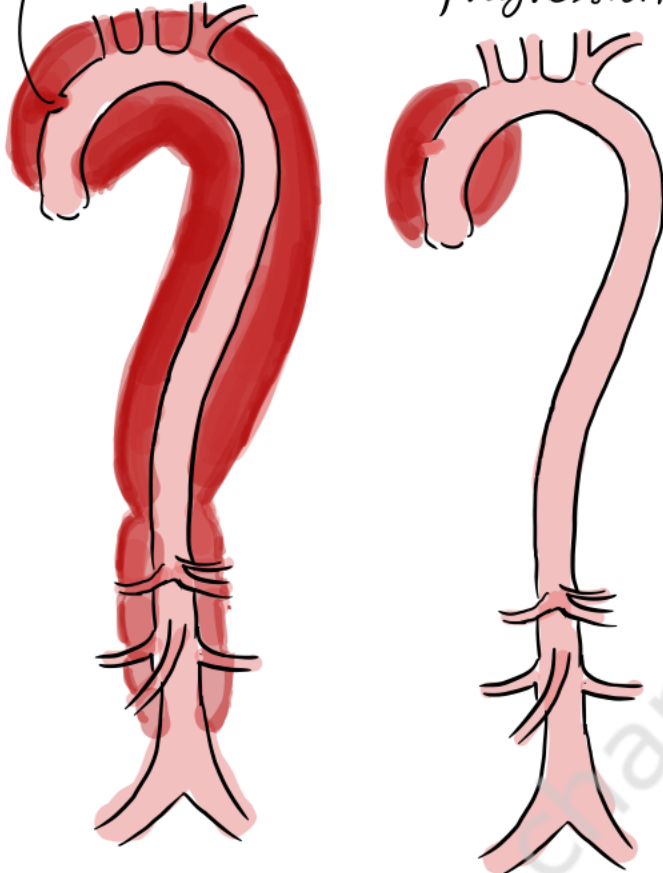
2) PENETRATING AORTIC ULCER - deep atherosclerotic plaques burrowing into the media - allowing blood to enter



# CLASSIFICATION

- Based on extent  $\left\{ \begin{array}{l} \text{DeBakey} - \text{I, II, IIIa, IIIb} \\ \text{Stanford} - \text{A, B} \end{array} \right.$
- Based on presentation  $\left\{ \begin{array}{l} \text{Acute} \\ \text{Chronic} \end{array} \right.$

Ascending aorta dissection also tend to have anterograde progression



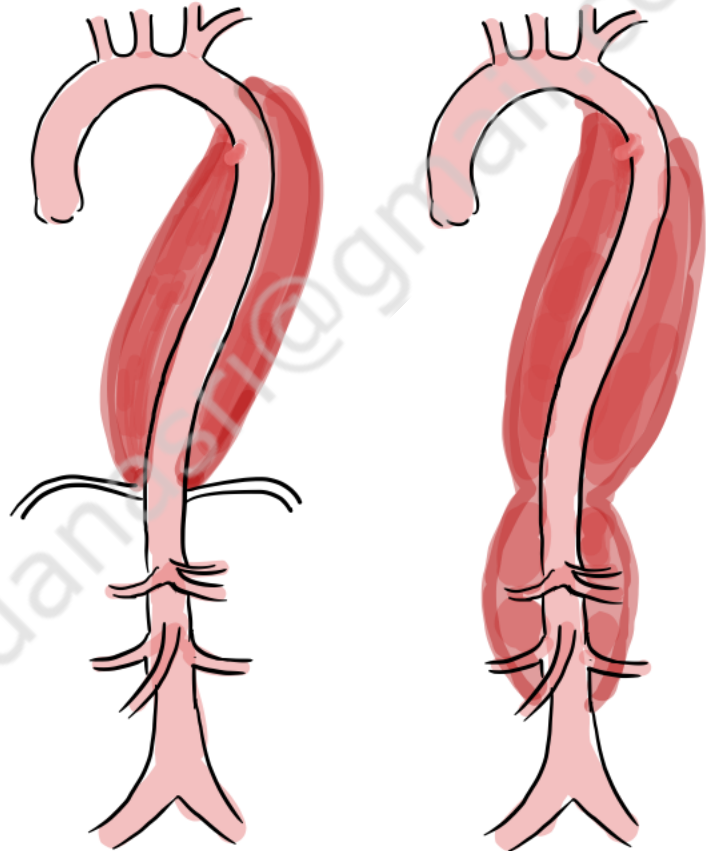
DEBAKEY-I  
- ASCENDING + DESCENDING AORTA

DEBAKEY-II  
ASCENDING AORTA

## STANFORD 'A' - ASCENDING AORTA

- acute presentation - chest/back pain
- profound hypotension esp: in cardiac tamponade acute aortic regurgitation
- SURGICAL EMERGENCY

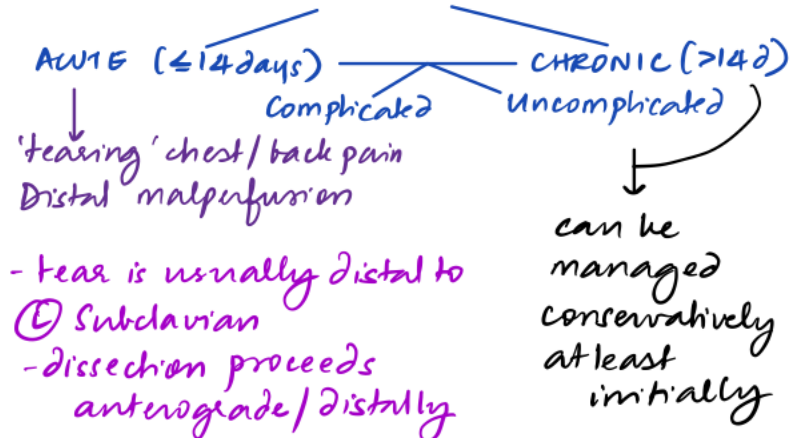
Descending aorta dissection - tear - usually distal to  $\odot$  subclavian A



DEBAKEY-IIIa  
DESCENDING THORACIC AORTA

DEBAKEY-IIIb  
DESCENDING THORACIC + ABDOMINAL AORTA

## STANFORD 'B' - DESCENDING AORTA



## EVALUATION

High index of clinical suspicion required

Chest pain - ECG - may show MI changes due to malperfusion syndrome

CXR - Widened mediastinum / abnormal aortic contour

Echocardiogram - Dissection vs aneurysm vs intramural hematoma

- aortic valve status

- cardiac tamponade

- if ascending dissection suspected → TEE → surgery

## CT/MRA

'DOUBLE LUMEN AORTA'

→ segments of aorta involved

aortic dilatation

threatening sequelae - impending rupture

branch vessel compromise

if CAD is suspected - Coronary angio pre-op

Malperfusion s<sup>o</sup> - R<sub>x</sub> in a hybrid operating room / angio suite

## TREATMENT

• Initial management :-

- Resuscitate - IV access, fluids, blood & blood products

- INITIAL MANAGEMENT STRATEGY - ANTI-IMPULSE THERAPY / BLOOD PRESSURE CONTROL

Goals - heart rate - 60-80 bpm

SBP - 100-110 mmHg

MAP - 60-75 mmHg

ESMOL  
LABETALOL β blockers ✓ - CIs - severe CHF, Bradycardia, ↑ grade conduction block, asthma

Direct Vasodilators - Nitroprusside

CCBs

ACE Is - Enalapril

- Pain relief - opioids

## ◆ ACUTE ASCENDING AORTIC DISSECTION

- Emergency surgical repair : Median sternotomy  
Cardiopulmonary bypass  
Hypothermic circulatory arrest  
Exploration, obliteration of false lumen  
Recon i graft  
± antegrade stenting of distal aorta

CHRONIC ASCENDING - SIMILAR - except that false lumen is not obliterated  
- dissecting membrane fenestrated

## DESCENDING AORTIC DISSECTION

Non-operative management: initial management strategy

- better outcomes
- monitor for expansion, rupture, malperfusion s/s
- if stable
  - ↳ shift to oral meds after CT confirmation
  - aggressive imaging followup
    - 6 weeks
    - 3-6 m
    - 1 y

### INDICATIONS FOR OPEN SURGERY

- Complicated acute distal aortic dissections
  - Rupture
  - Periaortic/Pleural collection
  - rapidly expanding aortic diameter
  - uncontrolled H/N
- Chronic cases
  - affected segment  $\geq 5.5$  cm diameter
  - connective tissue disorders

### ENDOVASCULAR MANAGEMENT

1) Rx of Malperfusion syndromes:

- Endovascular fenestration

↓  
Balloon used to create tear in dissection flap  
- allows blood flow into both lumens

- Aortic stenting - to overcome dynamic malperfusion
- Branch vessel stenting - to overcome static malperfusion

2) Endovascular stent grafts

- identify true lumen & intimal tear (using IVUS)
- use stent graft to seal entry site of dissection

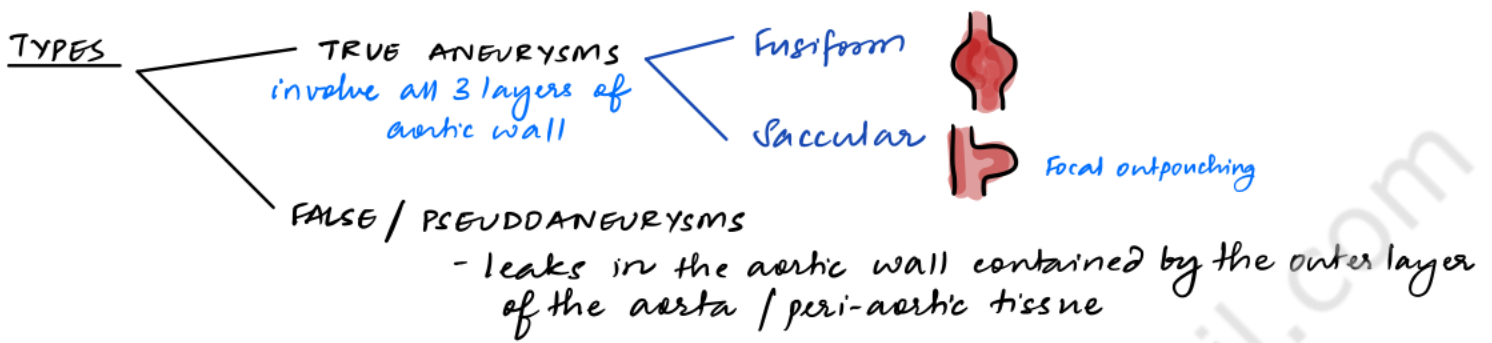
↓  
thrombosis of false lumen  
↓  
Aortic remodelling

### SURGICAL MANAGEMENT OF MALPERFUSION SYNDROMES

- Lower limb - femoral bypass
- Visceral - graft replacement / visceral/renal artery bypass
- open aortic fenestration

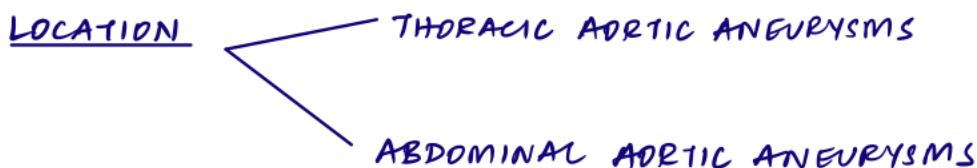
# AORTIC ANEURYSMS

An aortic aneurysm is a permanent, localized dilatation of the aorta to a diameter that is  $\geq 50\%$  than is normal at that anatomic level



## CAUSES

- **ATHEROSCLEROSIS (AAA)**
  - Non-specific medial degeneration (TAA)
  - ↑ Age
  - Male sex
  - smoking history
  - Family history
  - Hypertension
- **CONNECTIVE TISSUE DISORDERS**
  - Marfan's Syndrome
  - Loeys-Dietz syndrome
  - Ehlers-Danlos syndrome
  - Turner's syndrome
  - Familial thoracic aortic aneurysm (TAA)
  - Congenital bicuspid aortic valve (TAA)
- **Aortitis**
  - Takayasu arteritis
  - Giant Cell Aortitis
- **Infection - mycotic aneurysms**
  - bacterial endocarditis
    - Staph, Strep, salmonella
  - Previously - SYPHILIS - obliterative endarteritis of vasovasorum
- **Trauma - Pseudoaneurysms**



Laplace law = Tension = Pressure x radius

# ABDOMINAL AORTIC ANEURYSMS

• m/c location - infrarenal aorta - 90% ; fusiform

• Pathological focal dilatation of the aorta  $> 3\text{cm}$

or  $> 1.5$  times the diameter of adjacent normal aorta

(N) Aorta	Diameter	Annual risk of rupture	5-year rupture risk
Small AAA	2 - 3 cm	1%	5 - 10%
Moderate AAA	3 - 5 cm	2 - 5%	30 - 40%
Large AAA	5 - 6 cm	3 - 10%	$> 50\%$
Very large AAA	$> 7\text{cm}$	$> 10\%$	close to 100%

## RISK FACTORS

### FOR AAA DEVELOPMENT

- Tobacco use
- Hypercholesterolemia
- Hypertension
- Male gender
- Family history

### FOR AAA EXPANSION

- Advanced age
- Severe cardiac disease
- Previous stroke
- Tobacco use
- Cardiac / Renal transplant

### FOR AAA RUPTURE

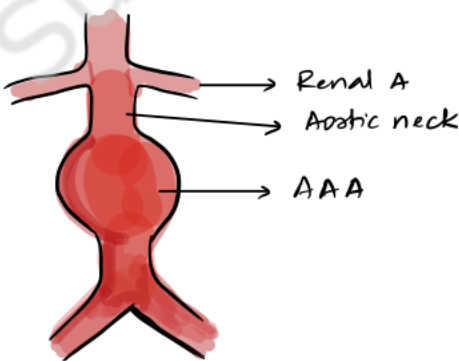
- Female gender!
- $\downarrow$  FEV<sub>1</sub>
- Large diameter
- $\uparrow$  MABP
- Current tobacco use

## CLASSIFICATION OF AAAs

Society of vascular surgery classification - based on extent & involvement of renal and visceral arteries

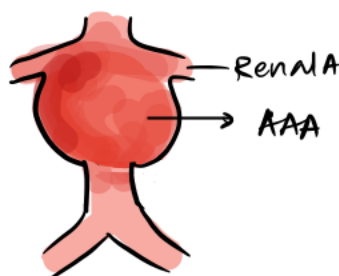
### INFRARENAL $\sim 80-90\%$

Proximal level of AAA below Renal artery level  $\bar{=}$  ADEQUATE AORTIC NECK for placement of clamp below renal arteries for surgical repair



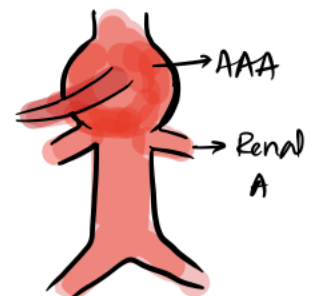
### JUXTARENAL/PARARENAL $\sim 8\%$

Proximal extent is at the level of the renal arteries with no normal aorta between AAA upper extent and renal arteries - clamp should be placed ABOVE one/both Renal As



### SUPRARENAL $\sim 5\%$

AAA originates above the level of renal As - may have renal artery / splanchnic involvement - REQUIRES RENAL / SPLANCHNIC REVAS



m/c in women

## CLINICAL FEATURES

- Majority - asymptomatic - detected incidentally while working up back pain
- Rarely - back pain / abdominal pain ± pulsatile mass
  - ↳ m/c - SUPRAUMBILICAL MIDLINE
  - ↳ tortuosity - INFRAUMBILICAL / LATERAL
- Ruptured AAA - back pain / abdominal pain ± pulsatile mass ± SHOCK
- Concomitant PAD - claudication / Renal artery disease

## EVALUATION

- USG - safe, non invasive
  - limited by bowel gas & body habitus
- CT/CTA - site, extent
  - vessel calcification
  - thrombus
  - concurrent occlusive disease

## ANATOMICAL INFORMATION RELEVANT FOR SURGICAL PLANNING

Length & diameter of aortic 'neck'  
Neck mural calcification  
Neck luminal thrombus  
Length & diameter of common iliac A

- MRA - cannot detect calcification
- Pre-op - coronary & renal evaluation

## INDICATIONS FOR INTERVENTION

- Size > 5.5cm
  - Growth > 5mm/6m  
> 1cm/year
  - Saccular rather than fusiform
  - Distal embolisation
  - tenderness
- Ruptured AAA - surgical emergency

# OPEN SURGICAL REPAIR

## APPROACHES

### Transperitoneal

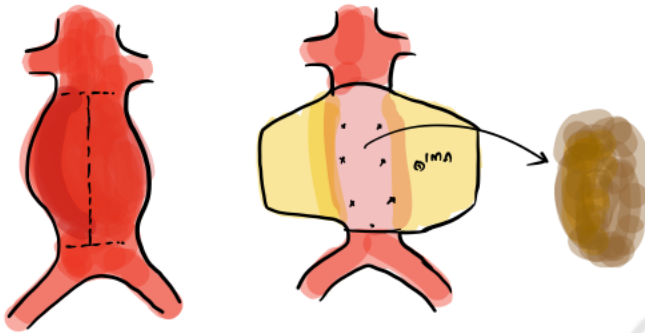
- Midline incision
- Left medial visceral rotation (Mattox) - exposes entire infradiaphragmatic aorta
- useful in emergency

### Retroperitoneal

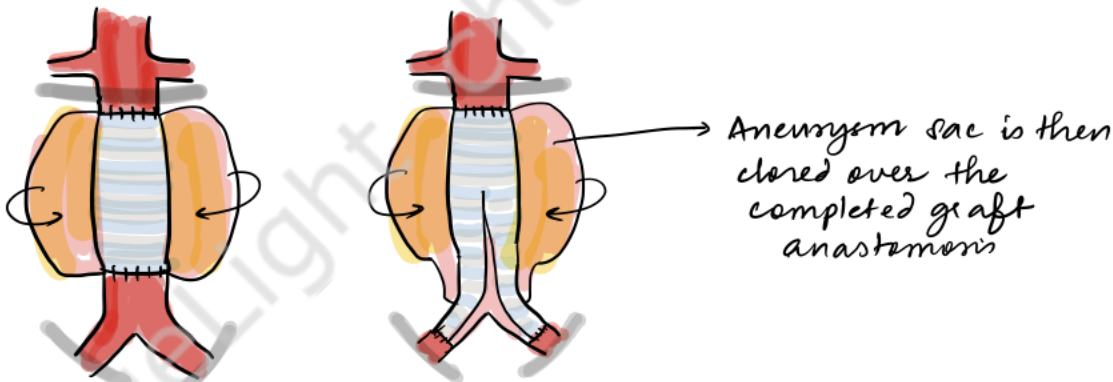
- ideal for juxta/supra-renal AAAs
- 'hostile' abdomen (adhesions)
- Needs familiarity

## PROCEDURE

- the aortic neck, B/L iliacs are dissected & clamped after heparinization
- Aneurysm is opened. Aneurysmal content is removed (Thrombus)



- Bleeding lumbar arteries & IMA - oversewn from within
  - Aneurysmorrhaphy is done by anastomosing a tube graft / bifurcated graft proximally & distally
- Based on extent ↓

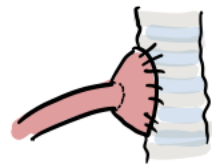


Restorative aneurysmorrhaphy  
= TUBE GRAFT

= Bifurcated GRAFT

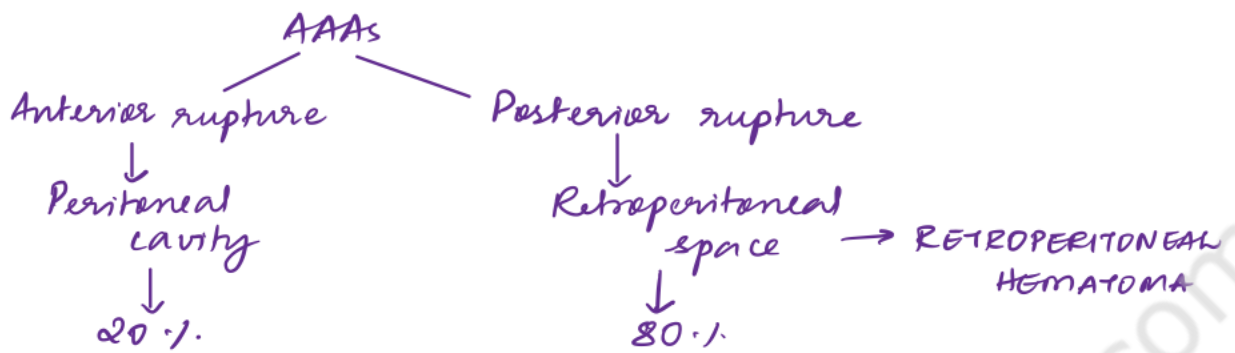
## IN CASE OF VISCERAL / RENAL / BRANCH VESSEL INVOLVEMENT

Reimplantation as **carrel patch** →  
or  
short bypass  
or



incorporated together by careful beveling

## MANAGEMENT OF RUPTURED ANEURYSM



- only 50% reach the hospital

'Permissive hypotension' - SBP < 100 mmHg

Surgical emergency

### COMPLICATIONS OF ANEURYSM SURGERY

- 1) Wound complications
- 2) Cardiorespiratory complications
- 3) Ischemic colitis - usually resolves spontaneously
- 4) Renal failure
- 5) Neurological complications - cord ischemia, sexual dysfunction
- 6) Antoduodenal / Antocolic fistula
- 7) Graft thrombosis / occlusion
- 8) Prosthetic graft infection



# ENDOVASCULAR APPROACH (EVAR) - Endovascular aneurysm repair

- Can be attempted in cases - long proximal aortic neck (>1.5cm)  
neck diameter 1.8-3.2cm
- ↓ mural calcification } <50%
  - ↓ luminal thrombus } circumference
  - sufficiently long & wide ilia

Seldinger technique - femoral approach → L<sub>1</sub> L<sub>2</sub> level

VASCULAR CLOSURE DEVICE deployed immediately below the aortogram lowest renal artery

↓  
Verified - completion angiogram

## COMPLICATIONS

- Endoleak (in EVAR)
- i - failure of the vascular closure device to achieve a satisfactory seal proximally / distally  
(failure to exclude aneurysm sac)
  - ii - filling of aneurysm sac by branch vessels
  - iii - breakdown of components of closure device
  - iv - seepage through graft

Graft migration

Graft fracture

Graft limb occlusion

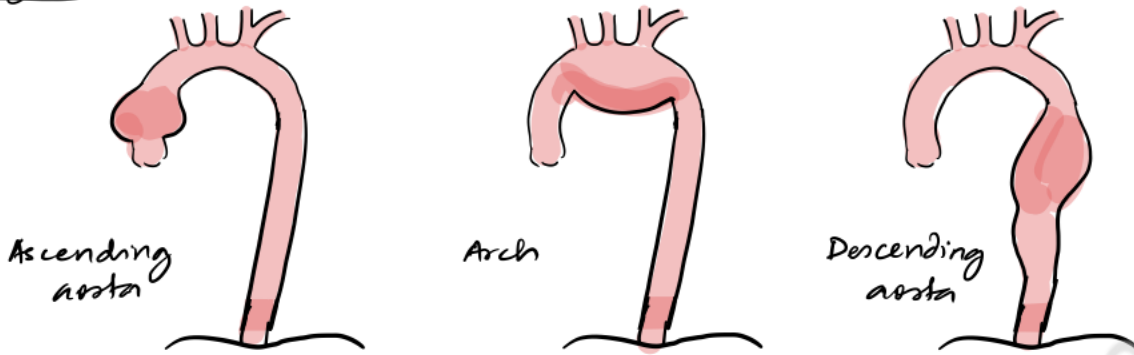
Needs lifelong surveillance

# THORACIC AORTIC ANEURYSMS

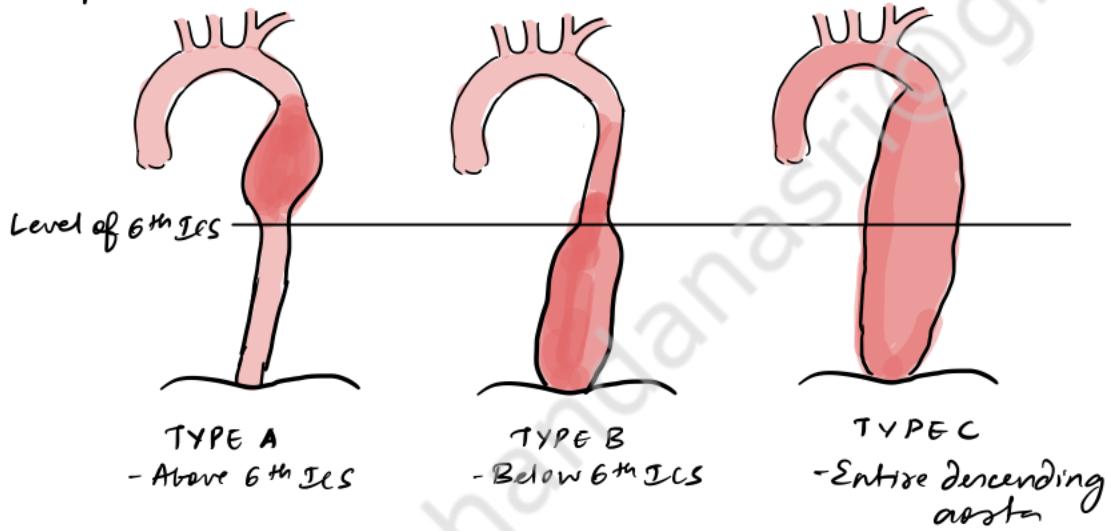
1<sup>st</sup> cause - degeneration

2<sup>nd</sup> 1<sup>st</sup> cause - late complication of chronic aortic dissection

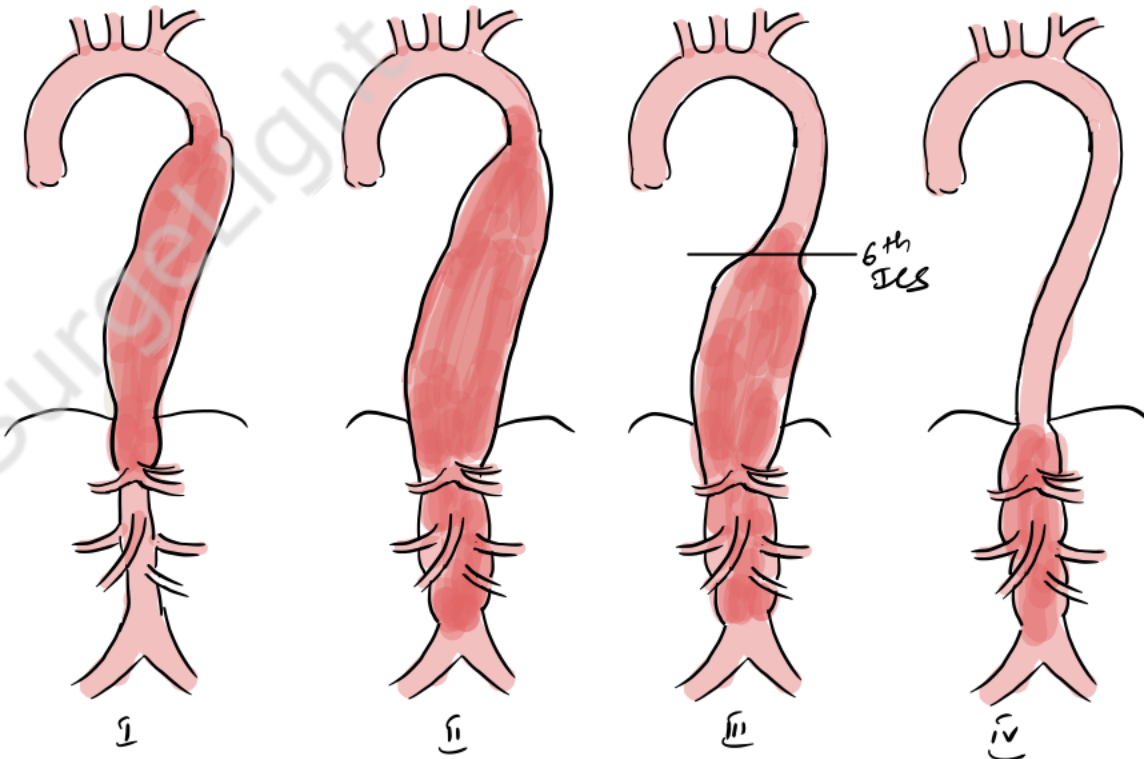
types



## DESCENDING AORTA ANEURYSMS



## CRAWFORD CLASSIFICATION - THORACOABDOMINAL ANEURYSMS



## Clinical features

- m/c - discovered incidentally
- Local compression & erosion
  - chest pain / back pain → desc. aorta
  - SVC compression (Asc. aorta & arch aneurysms)
  - erosion into SVC / RA - high output HF
  - stretching of ⊕ RLN - hoarseness
  - Desc. thoracic / Thoraco-abd - thoracic / lumbar vertebral body erosion
  - Airway obstruction
  - Esophageal compression - dysphagia
- Asc. aneurysms - aortic regurgitation
- Distal embolisation - esp. desc & thoracoabdominal - visceral / LL embolism
- Rupture - chest pain / shock / cardiac tamponade

## EVALUATION

- CXR - esp. lateral view - aortic silhouette - calcification
- Echo / USG
- CT / CTA
- MRA
- Aortography / Cardiac cath

## Indications for Intervention

Asc. aorta aneurysm  $> 5.5\text{cm}$  }  $> 0.5\text{cm}$  dilatation / year  
Desc. Aorta aneurysm  $> 6\text{cm}$  } <sup>or</sup>

Lower threshold for connective tissue disorders

## INTERVENTION

- Open Repair - AORTOPLASTY usually  $\bar{c}$  CPB
- Endovascular Repair - TEVAR - Thoracic Endovascular Aneurysm repair
- Hybrid repair

# ACUTE LIMB ISCHEMIA

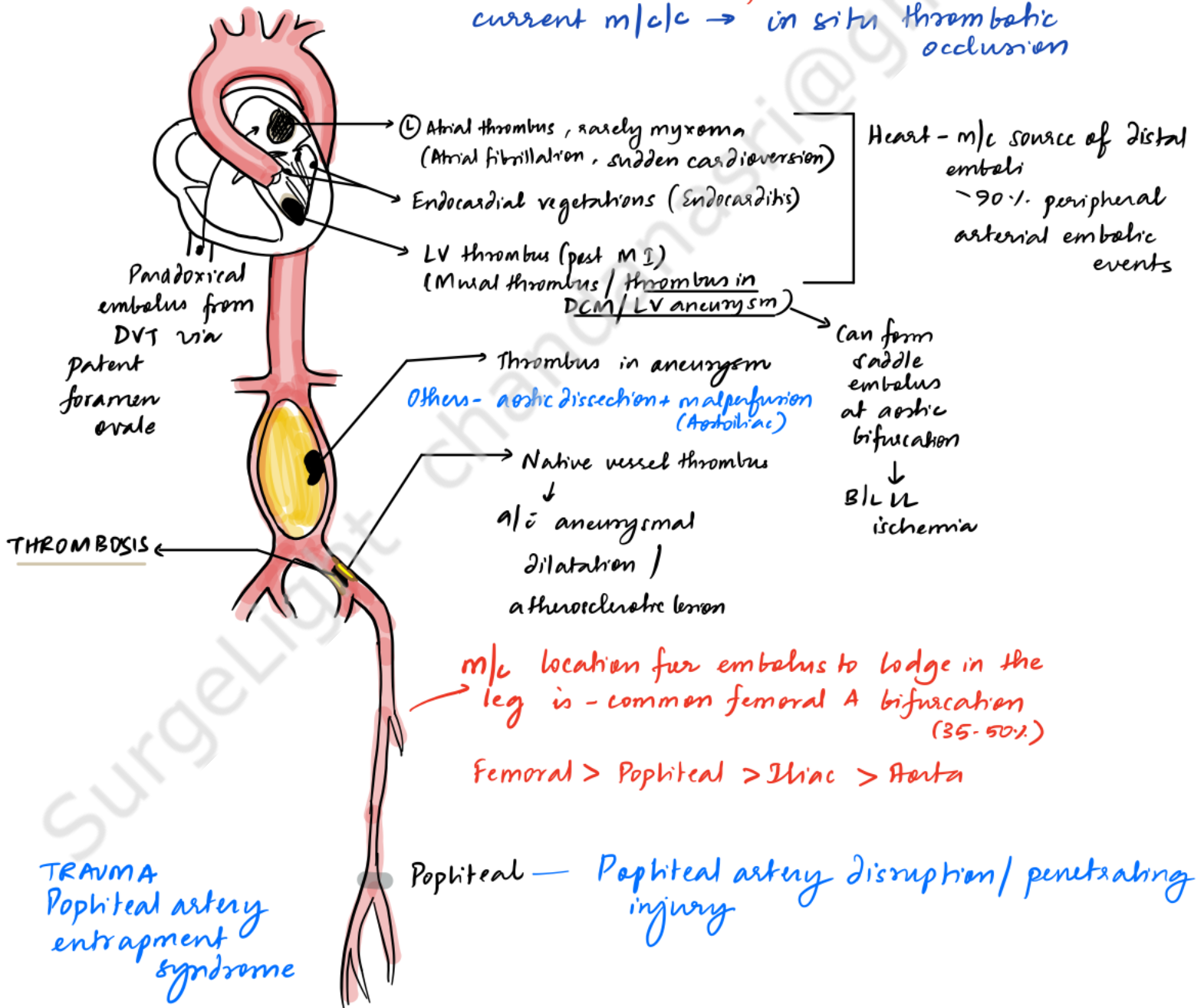
ALI - sudden loss of limb perfusion

↳ term is applicable up to 2 WEEKS after an initiating event.

## ETIOLOGY

- Embolism
  - Native vessel thrombosis
  - Reconstruction thrombosis
  - Trauma
  - Complications of a peripheral aneurysm
- } Thrombosis

EMBOLI - used to be m/c cause of acute limb ischemia in RHD era  
 alt ↑ cardioembolic phenomena  
 current m/c → in situ thrombotic occlusion



## PATHOPHYSIOLOGY

2/3+ ABRUPT + COMPLETE BLOCKAGE of the main arterial supply to an extremity

unlike CLI, collaterals are not well developed

Distal tissue bed in the limb becomes ischemic

aerobic  $\xrightarrow{\quad}$  anaerobic

Progressive ischemia - cell dysfunction & death

Nerve cells > Muscle cells

A patient WITHOUT UNDERLYING VASCULAR DISEASE who develops an acute arterial blockage has ~5-7h (6h) for revascularisation before irreversible functional tissue damage occurs (begins ~3h; complete by ~6h)

↓  
Reperfusion injury

## CLINICAL FEATURES

### EMBOLISM

- Rapid onset
- Prior cardiac event
- Generally no prior PAD history

Affected limb - Cold, mottled, Paralyzed  
- clear demarcation

Contralateral

limb - usually ⊖

(SADDLE EMBOLUS AT AORTIC BIFURCATION - B/L ALI)

Result of cardioembolic phenomena

Typically located at ARTERIAL BIFURCATIONS

### THROMBOSIS

- Vague onset
- No h/o recent cardiac event
- Usually h/o PAD +

Affected limb - Cool, bluish, paraesthesia  
No distinct demarcation

Contralateral limb

- usually - abnormal pulses  
(↓ pulses)

2/3+ Plaque rupture / hypercoagulable state

Typically - LONG SEGMENT OCCLUSIONS (diffuse)

Classical Presentation of ALI - Pain

(5+1) 'P's

Pallor

Paraesthesia

Paralysis → sign of very severe ischemia

Pulselessness

- Perishing cold / Poikilothermia

## LEVEL OF OCCLUSION

- AORTIC BIFURCATION - B/L Femoral pulse ⊖ & B/L LL ALI (m/c 2/3+ saddle embolus)
- DISTAL CFA (AT BIFURCATION) - Palpable femoral (proximal to occlusion) & absent distal pulses
- SFA EMBOLUS/THROMBUS
- POPLITEAL EMBOLUS/THROMBUS
- POPLITEAL TRIFURCATION EMBOLUS - Calf ischemia, absent pedal pulses + usually ⊕ Popliteal pulse

## GRADES OF ACUTE LIMB ISCHEMIA

I - viable; not immediately threatened

Capillary return ⊕

Muscle weakness ⊖

Sensory loss ⊖

Doppler signal ⊕ - arterial + venous

II a - marginally threatened, but salvageable if treated

Sensory loss - minimal (toes) or none

No muscle weakness

Doppler signal - arterial ⊖  
venous ⊕

II b - immediately threatened - salvageable if treated immediately

Sensory loss - moderate (more than toes)

at rest pain

Mild to moderate muscle weakness

Doppler signal - arterial ⊖  
venous ⊕

III - IRREVERSIBLE - Major permanent tissue damage

Absent capillary return - marbling / mottling

Profound sensory loss

Profound paralysis

Doppler signal - arterial ⊖  
venous ⊖

## EVALUATION

- Duplex scan

- Emergency CTA

## Management

### - PRELIMINARY

• Anticoagulation - for clot stabilization  
- to ↓ risk of 2° thrombosis of underperfused distal vessels

- INR 2-3

- UFH / LMWH

ease of reversal: ↙  
protamine  
sulfate

↘ ease of use, safety

• Correct dehydration, oxygenate

# MODES OF INTERVENTION

## ENDOVASCULAR PROCEDURES

### THROMBOLYSIS

#### CATHETER-DIRECTED

- Class I, II a AUI
- Catheter-directed approach → regional thrombus dissolution & minimal systemic fibrinolysis  
↳ however, it does occur
- agent - t-PA (alteplase, reteplase)

#### CONTRAINDICATIONS

- Active bleeding disorder
- GI bleeding < 10d
- CVA < 6m
- Head injury < 3m
- CNS surgery < 3m

#### Relative CE

- Major Sx/Trauma < 10d
- HTN (> 180/110)
- CPR < 10d
- Intracranial tumor
- Pregnancy

#### PHARMACOMECHANICAL

- Class I, II a AUI
- In addition to thrombolytic agent, mechanical clot removal devices are used to accelerate thrombolysis
- ↓ thrombolytic dose
- minimise bleeding risk

#### Strategies used

- Mechanical mixing
- Ultrasound energy
- Power pulse injection
- Microbubble technology

### PERCUTANEOUS MECHANICAL THROMBECTOMY

#### PMT devices

- hydrodynamic catheters
- rotational catheters
- Aspiration thrombectomy catheters

#### HYDRODYNAMIC

Use hydrodynamic jet to dislodge thrombus - which is then extracted

#### ROTATIONAL

Brushes / rotating wires - fragmentation of thrombus  
↓  
Recanalisation → Risk of distal embolisation

#### THROMBUS ASPIRATION

for fresh thrombus

Guidewire to reach thrombus  
↓  
Catheter threaded over it  
↓  
Thrombus aspirated

## SURGICAL REVASCLARISATION - Generally for IIb

↳ may have to be coupled & fasciotomies to address compartment S°

### BALLOON CATHETER THROMBECTOMY/EMBOLECTOMY

- for emboli / graft thrombosis
- Arteriotomy → passage of appropriate size Fogarty's Balloon embolectomy catheters to evacuate thrombus  
↓  
Check angio  
↓  
Closure of arteriotomy

### ENDARTERECTOMY

- for thrombotic in situ occlusion
- ± Patch angioplasty

### BYPASS PROCEDURES

done in case of failed Balloon thrombectomy after evaluating proximal & distal target vessels & on-table angiography

## Complications of ALI treatment

- Compartment Syndrome - when prolonged ischemia is flb reperfusion
- Ischemic neuropathy
- Rhabdomyolysis → Myoglobinuria → Acute tubular necrosis
  - ↳ Alkalinize urine
  - ↳ forced saline diuresis
- Reperfusion syndrome - Hypertension  
Hyperkalemia  
Myoglobinuria  
Renal failure

---

## AMPUTATION

- for failed revascularisation
  - for irreversible ALI
-



# CHRONIC LIMB ISCHEMIA

→ objectively proven arterial occlusive disease & symptoms > 2 weeks

## PATHOPHYSIOLOGY

Gradual progressive reduction in blood flow

↓  
Development of collateral circulation

↓  
Alternative route for blood flow — reduced symptom severity

↓  
CRITICAL STENOSIS / OCCLUSION → CRITICAL LIMB ISCHEMIA

## ETIOLOGY

- Atherosclerosis - smoking, HTN, T<sub>2</sub>DM, Dyslipidemia, Obesity, Age etc.
- Thromboangitis Obliterans / Buerger's Disease - smoking, male, < 45y
- Others
  - Popliteal aneurysm / Entrapment
  - Aortic coarctation
  - Fibromuscular dysplasia
  - Takayasu
  - Arteritis

## GRADES OF CHRONIC LIMB ISCHEMIA

FONTAINE	Characteristics	RUTHERFORD
I	ASYMPTOMATIC - (N) treadmill / Reactive hyperemia test	0
IIa	MILD CLAUDICATION - can complete TMT; Post TMT AP > 50mmHg but > 20mmHg ↓ than rest AP	1
IIb	MODERATE CLAUDICATION → in between SEVERE CLAUDICATION - cannot complete TMT + Post TMT ASBP < 50mmHg	2 3
III	Ischemic Rest Pain - Resting AP < 40mmHg, TP < 80mmHg flat / barely pulsatile ankle/metatarsal pulse wave	4
IV	Ulceration or gangrene Minor tissue loss - non healing ulcer, focal gangrene Major tissue loss - extending above transmetatarsal level; can't salvage functional foot	5 6

# INTERMITTENT CLAUDICATION

Cramp-like pain felt in a group of muscles

Due to anaerobic metabolism in muscles  $\Delta$  + unmet  $\uparrow$  in  $O_2$  demand brought about by exercise

- reliably and reproducibly brought on by activity (walking)
- not present on initiating the activity (first step)
- reliably relieved by rest (usually within 5 min)
- distal to level of occlusion  
(muscle group affected by claudication is classically one anatomical level below the level of arterial disease)

local intramuscular acidosis, substance P  
(Eutherford pg 1660)

## BOYD'S GRADING OF INTERMITTENT CLAUDICATION

- I - Pain appears on walking some distance  $\rightarrow$  Pt continues to walk  $\rightarrow$  pain RELIEVED
- II - Pain appears on walking some distance  $\rightarrow$  Pt continues to walk  $\rightarrow$  pain PERSISTS  
Pt continues to walk  $\bar{e}$  effort  $\leftarrow$
- III - Pain compels the patient to take rest

### LEVEL OF OCCLUSION

Aortoiliac obstruction

Iliac obstruction

Femoropopliteal (m/c)  
 $\hookrightarrow$  70%

Distal / Infrapopliteal

### SITE OF CLAUDICATION

Buttocks, thighs & calves (often B/L)

Unilateral thigh & calf; sometimes buttock

Unilateral calf

Calf and FOOT

Claudication distance: the distance that a patient is able to walk without stopping due to pain

Also affected by

- Uphill walking
- Carrying weights
- Anemia
- Heart failure

Progressively shortens as the disease / occlusion worsens

**REST PAIN** → 'CRY OF DYING NERVES' - ischemic neuropathy AS, C

→ Burning sensation

Pain at rest represents a significant decrease in circulation and involves the MOST DISTAL ASPECT OF THE LOWER EXTREMITY that is FARTHEST from the central source of circulation / blood flow

THE FOREFOOT AND DIGITS are commonly involved

In the absence of acute arterial occlusion, patients do not have any pain in the thigh and calf AT rest

Symptoms are classically relieved & dependency because gravity tends to facilitate circulation

Symptoms are aggravated if the patient lies down & elevates the extremity

Worse at night as BP ↓ at night → worsens hypoperfusion

Clutching the foot - gated theory of pain - ↓ pain

**CLINICAL DIAGNOSIS OF REST PAIN** - objectively confirmed by

SYSTOLIC ANKLE PRESSURE - < 50 mmHg  
TOE PRESSURE - < 30 mmHg  
ABPI - < 0.5

## ISCHEMIC ULCERS

It the effect of repetitive soft tissue trauma of very mild degree & erosion of overlying skin

↓  
Skin repair is hampered by inadequate tissue perfusion & oxygenation

- usually in the distribution of rest pain - between toes, dorsum of foot, malleoli - shallow, non-healing, pale erosions

→ (no usage of such a term in Rutherford, Schwartz, Sabiston, Bailey)

**PREGANGRENE** - changes in tissues

which indicate that blood supply is inadequate to sustain the viability of the tissue - rest pain

color changes

edema

hyperesthesia

± ischemic ulcer

## CRITICAL LIMB ISCHEMIA

**Definition:** a clinical state of advanced arterial occlusive disease  
(TASC) which places an extremity at risk for gangrene/limb loss  
Ischemic rest pain - of at least 2 weeks - requiring analgesics

ε Ankle systolic pressure  $< 50 \text{ mmHg}$   
Toe systolic pressure  $< 30 \text{ mmHg}$

In pts  $\bar{c}$  Ischemic ulcers / toe gangrene

Ankle systolic pressure  $< 70 \text{ mmHg}$   
Toe systolic pressure  $< 50 \text{ mmHg}$

ε ABPI  $< 0.3$  [  $0.5$  acc. to some ]

Tc  $\text{PO}_2 < 30 \text{ mmHg}$

Critical limb ischemia is a/c higher risk of limb loss  
in the absence of revascularization

- represents a reduction in distal tissue perfusion below  
resting metabolic requirements

### Clinical features of chronic limb ischemia

- 1) Intermittent claudication
- 2) Rest pain
- 3) Ischemic ulcers, gangrene
- 4) Other features of limb ischemia - dry skin, hair loss, sic tissue wasting, brittle nails, dark skin, guttering of veins, foot pallor on elevation  $\bar{c}$  dependent rubor (sunset sign),  $\uparrow$  capillary refill, diminished/alent pulses
- 5) Signs of occlusion/narrowing - bruit
- 6) Aortoiliac occlusion - Leriche s $\bar{c}$  - B/c alent femorals  $\bar{c}$  Bruit over iliac region  
Buttock + thigh claudication  
Impotence

## EVALUATION

- HANDHELD DOPPLER - as an extension of clinical examination of pulses  
↳ + sphygmomanometer  
↓  
ABPI, ASP, TSP, Segmental Brachial index

### ANKLE BRACHIAL PRESSURE INDEX

Brachial systolic pressure (used as a surrogate for central aortic pressure)

↳ generally accurate unless there is UL PAD  
∴ B/L pressure measured - higher value taken

Ankle systolic pressure - Highest of DPA/ATA, PTA/Peroneal for either ankle

Ankle Brachial → calculated for both LL

- ↳ without accounting for brachial pressure it is impossible to judge if
- low Ankle pressure is d/t systemic hypotension
  - high/normal pressure (despite significant vascular occlusion) is d/t systemic hypertension

ABPI is based on the principle of the changes in the pressure waveform as it moves through the vasculature

Peak systolic pressure is accentuated by the additive effect of the reflected pressure waves from the periphery

∴ Although, the mean pressure decreases as the pressure wave travels distally, the peak systolic pressure increases

↓  
∴ (N) Ankle pressure is ~10% higher than brachial pressure  
∴ (N) ABPI is 1.1  
(Range 0.9-1.29)

>1.3 → Arteriosclerosis - diabetes → not reliable  
0.9-1.29 → (N)  
0.5-0.9 → some degree of arterial occlusion - claudication  
<0.5 → Rest pain  
<0.3 → imminent necrosis - CLE

Drop of ABPI >20% following exercise } → flow limiting arterial disease  
failure to return to baseline in ≤3min }

•  $TcPO_2$ : transcutaneous oxygen pressure - non invasive method to quantify skin oxygenation

< 30 mmHg  $\rightarrow$  ischemia

< 10 mmHg  $\rightarrow$  poor prognosis

• DUPLEX - B mode + pulsed doppler spectral wave forms

B-mode - provides anatomical image of the vessel wall

vs

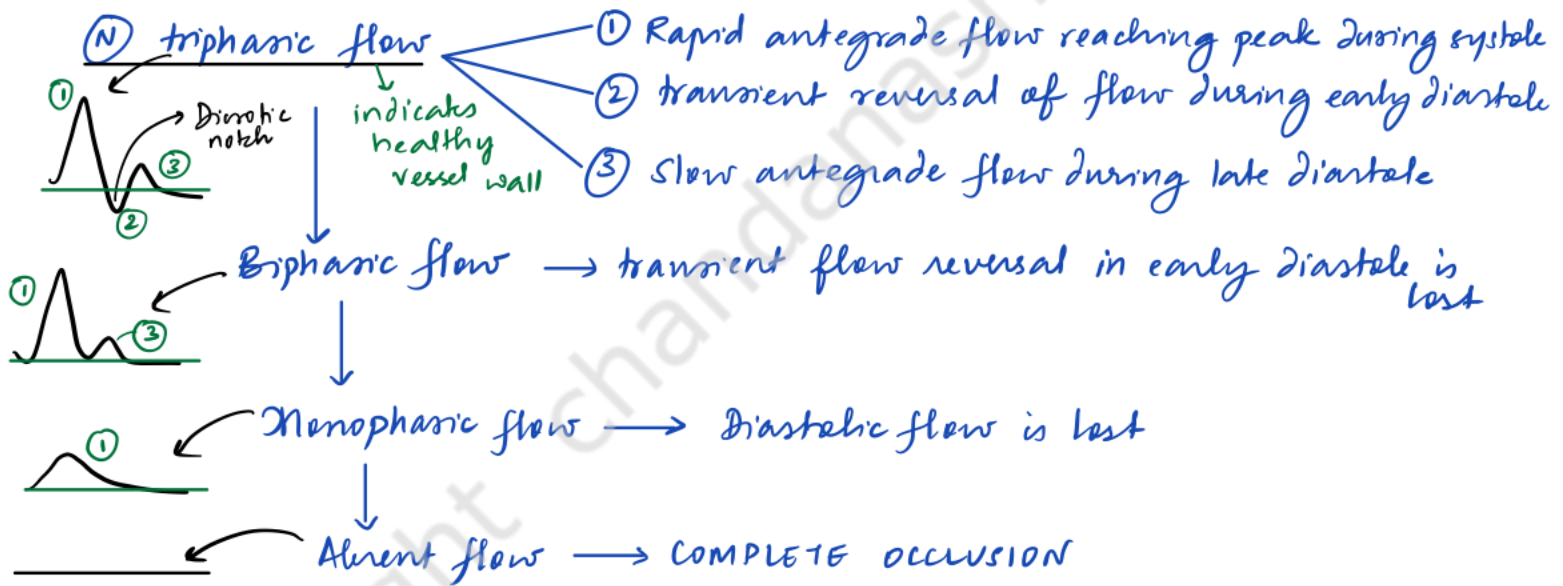
$\rightarrow$  Plaque / thrombus can also be visualised

$\rightarrow$  Stenotic segment can be imaged

Doppler - allows study of flow direction and velocity

Peak systolic velocities; end diastolic velocities

$\rightarrow$  waveforms



High flow velocity  $\Rightarrow$  Stenosis

$\frac{\text{Peak systolic velocity in affected segment}}{\text{Peak systolic velocity in (N) proximal segment}} \geq 2.0 \Rightarrow \geq 50\% \text{ stenosis}$

- Non-invasive
- safe
- best effective

- ARTERIAL SEGMENT visualisation  $\rightarrow$  limited by obesity bowel gas

# COMPLEX IMAGING

**ANGIOGRAPHY (ARTERIOGRAPHY)** - **invasive** - vascular access via seldinger technique

↳ **GOLD STANDARD** - Dx  $\xrightarrow{Flb}$  Rx

Needs - a C-arm fluoroscopy unit & image intensifier & digital image processing software

an arterial catheter  $\left\{ \begin{array}{l} \text{end hole} \\ \text{multiple side hole} \end{array} \right.$

injection  $\left\{ \begin{array}{l} \text{power injection} \\ \text{Hand injection} \end{array} \right.$

Contrast agents - Iodinated contrast  
CO<sub>2</sub>  
Gadolinium

Complete assessment of aortic and iliac inflow + Bilateral lower extremities  
needs 75-100ml contrast

Target vessel approached via vascular puncture by seldinger technique and dye injected  
(VASCULAR ACCESS  $\rightarrow$  Catheter placement in vascular bed that needs examination)  $\rightarrow$  femoral/axillary/brachial/radial

THE IMAGE OBTAINED IS A 'LUMENOGRAM'  
- misleading when plaques are eccentric

## INFORMATION

- Site of occlusion
- Extent of occlusion - length of lesion
- Nature of occlusion - % luminal compromise
- Run in - patency of vessel proximal to occlusion
- Distal run off - patency of vessel distal to occlusion
- State of COLLATERAL CIRCULATION

DSA - conventional angio  $\rightarrow$  digitally removing the background image to enhance picture of vascular anatomy  
-  $\downarrow$  contrast required for high-res image  
- better definition

Roadmapping - where a previously obtained arteriogram is superimposed on live image to guide the interventional procedure

Complications - Groin hematoma  
Thrombosis  
Pseudoaneurysm  
AV fistula  
Arterial dissection

Contrast nephropathy  
Allergic reaction

Radiation exposure

## CT angiography

- NON-INVASIVE (in the sense that the target vessel is not catheterised)
- IV infusion of contrast

↓  
Patient is advanced through a rotating gantry

↓  
serial transverse images

↓  
Images extracted from slices and rendered in recon format  
(POST-ACQUISITION IMAGE PROCESSING)

- issues - contrast adverse effects  
radiation exposure  
ARTIFACTS - calcification  
stents

## MR Angiography

Advantages - does not require iodinated (nephrotoxic) contrast  
Metallic stent artifacts can be dealt with using  
alterations in image acquisition & processing

Disadvantages - slow, expensive, claustrophobia issues  
Gadolinium - NSF  
CI in pacemakers, defibrillators, intracerebral shunts,  
cochlear implants etc.

IVUS : Intravascular USG  
small catheter-based transducers

- Wall anatomy
- endovasc R of aortic dissection

## OTHER CURRENTLY OUTDATED TECHNIQUES

Plethysmography - measures the changes in the volume of an organ/limb  
↓  
between systole & diastole  
↓  
reflects pulsatile blood flow

Photoplethysmography - uses Infrared rays to assess circulation



# MANAGEMENT OF CHRONIC LIMB ISCHEMIA

## Medical treatment

### RISK FACTOR MODIFICATION

- Smoking cessation
  - Physician advice
  - Nicotine replacement
  - Bupropion R<sub>x</sub>
- Antiplatelets
  - Aspirin 75-325 mg
  - ±/or
  - Clopidogrel 75mg
 } to ↓ risk of MI, stroke, vascular death
- Treat Dyslipidemia
  - Statins to target LDL < 100mg/dL
- R<sub>x</sub> HTN & T<sub>2</sub>DM

↳ offered to all Chronic limb ischemia/PAOD

### Intermittent Claudication

Risk factor modification +

A trial of Cilostazol + 3 months supervised exercise

Annual limb loss rate in  
 ABP < 0.5 } -8.5%  
 ABP < 40mmHg

∴ Trial of Medical management & risk factor modification

↓ Failure / Disease progression  
 Consider for intervention

## Revascularisation

### Critical limb ischemia

- those who progress from claudication to rest pain / present initially with rest pain

↓  
Imaging

↓  
Intervention

↙  
Surgical

↘  
Endovascular

#### • BYPASS

- AORTOBIFEMORAL
- AXILLOBIFEMORAL
- FEMORO-FEMORAL
- ILIOfEMORAL
- FEMOROPUfTEAL
- INFRAPOUfTEAL

#### • ENDARTERECTOMY

- ↓
- PTA ± stenting
  - Stenting
  - Subintimal angioplasty
  - Stent graft
  - Atherectomy
    - Mechanical
    - Laser

Choice of approach and procedure is made based on the location and extent of the lesion

- Aortoiliac disease
- Femoropopliteal disease
- Infrapopliteal disease

## Amputation

Patients presenting with non healing limb wound / dry gangrene / necrotising infection

↓  
OPERATIVE DEBRIDEMENT OR AMPUTATION

→ In case of severe sepsis, hemodynamic instability

↓  
AMPUTATION / DEBRIDEMENT before REVASCULARISATION

→ Stable patient with wound responding to Abx

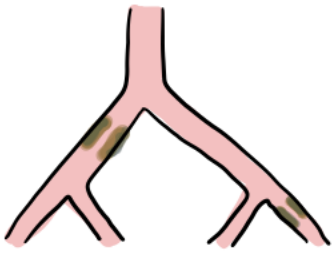
↓  
REVASCULARISATION BEFORE AMPUTATION / DEBRIDEMENT may minimise tissue loss

↓  
In pts who are not candidates for revascularisation, amputation done for

- spreading limb infections
- Gangrene
- intolerable pain

# AORTO-ILIAC OCCLUSIVE DISEASE

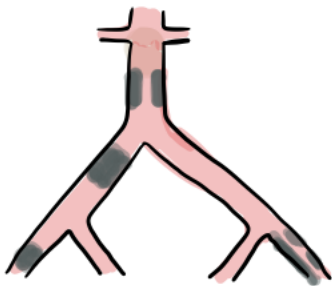
## TRANS ATLANTIC INTER SOCIETY CONSENSUS - TASC CLASSIFICATION



### TYPE - A

- U/L or B/L CIA stenosis
- U/L / B/L single short segment EIA stenosis ( $\leq 3\text{cm}$ )

Endovascular therapy is R<sub>x</sub> of choice



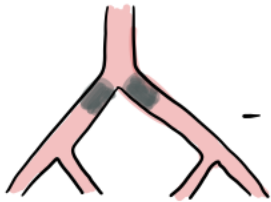
### TYPE - B

- short segment stenosis ( $\leq 3\text{cm}$ ) of infrarenal aorta
- single / multiple stenosis of EIA 3-10 cm (total) not involving CFA
- Unilateral CIA occlusion
- Unilateral EIA occlusion (not involving CFA / IIA)

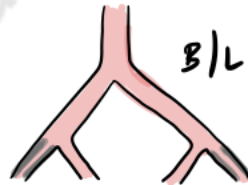
Endovascular > Surgery

### TYPE - C

Surgery > Endovascular

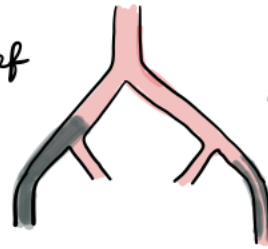


- B/L CIA occlusion



B/L EIA stenosis 3-10 cm not extending into CFA

U/L EIA occlusion involving origin of IIA / CFA



unilateral EIA stenosis extending into CFA

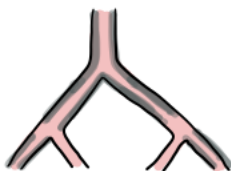
Heavily calcified U/L EIA occlusion  $\pm$  IIA / CFA involvement

### TYPE - D

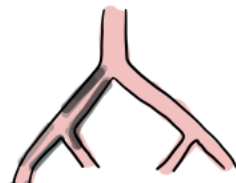
SURGERY is procedure of choice



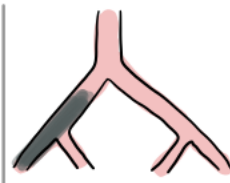
Infrarenal complete aortoiliac occlusion



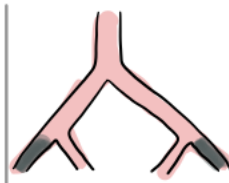
Diffuse disease involving aorta & both iliacs



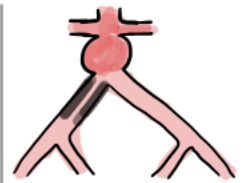
Diffuse disease involving U/L CIA, EIA, CFA



Unilateral CIA + EIA occlusion



B/L EIA occlusion

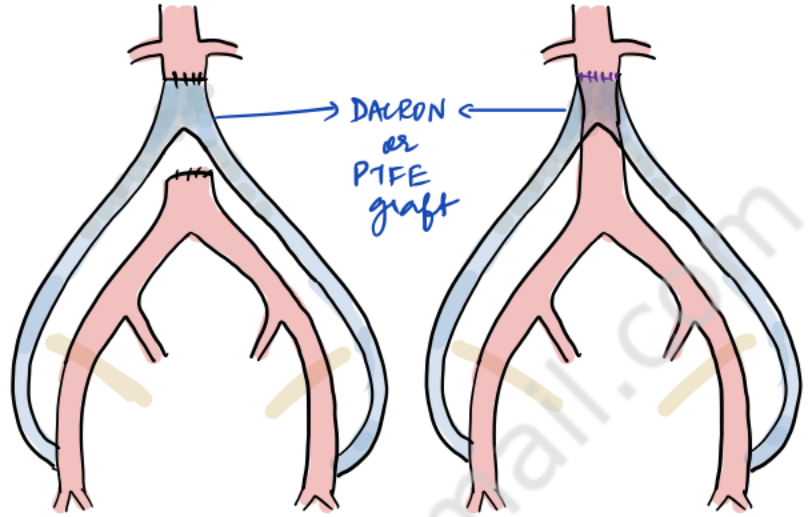


AAA requiring intervention + Iliac stenosis

# SURGICAL PROCEDURES FOR AORTO-ILIAc OCCLUSIVE DISEASE

## ① Aortobifemoral bypass

- Bilateral Femoral artery exposed via groin incision
- Aorta approached transabdominally / retroperitoneally
- Aorta cross clamped below renals
- Proximal aortic-graft anastomosis
  - end to end or side to end
- Distal anastomosis - just proximal to CFA bifurcation - end to side anastomosis



## ② Axillofemoral bypass

- extra-anatomic reconstruction that derives arterial inflow from the axillary artery to the femoral A
- preferred in pts with comorbidities that prohibit an abdominal vascular reconstruction

Axillary A exposed below clavicle - proximal anastomosis

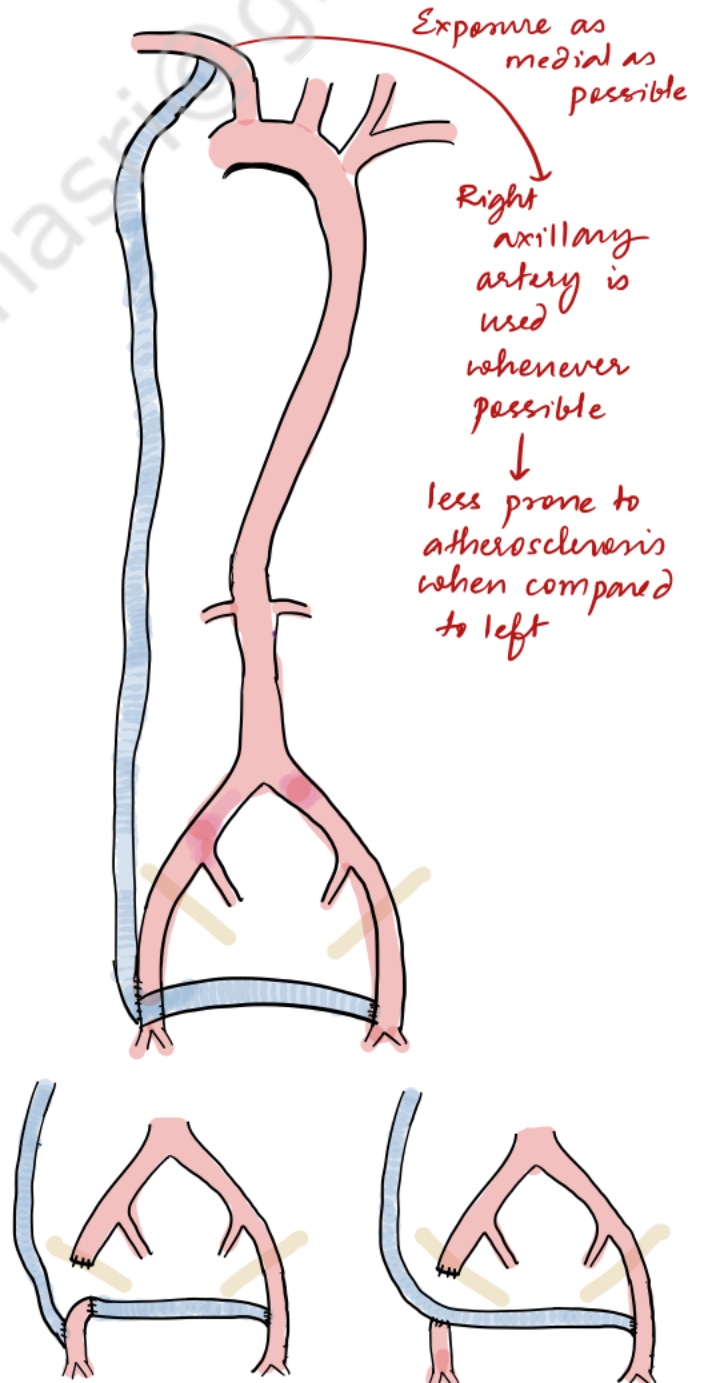
Graft is passed through a subcutaneous tunnel down the lateral chest wall, lateral abdominal wall to groin

Distal anastomosis

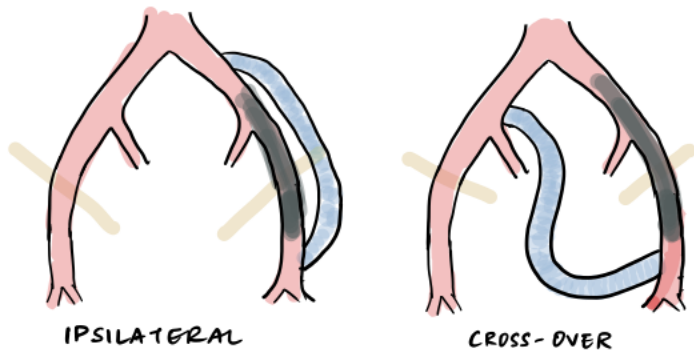
- ipsilateral CFA just proximal to bifurcation

A femero-femoral cross-over graft can be used to vascularise opposite LL if required

- Higher mortality
- Lower patency rates - 50% at 5y



### ③ Iliofemoral Bypass



### ④ Femero-femoral bypass



### ⑤ Aortobifemoral endarterectomy

- Circumferentially expose aorta and bilateral iliaes
- Clamp aorta, iliaes and lumbar (to prevent backbleeding during endarterectomy)
- Vertical aortotomy - remove plaque + intima upto level of iliaes
- close the incision
- done when there is risk of graft complications like infection  
long term patency comparable to aortobifemoral bypass

#### Disadvantages

- ↑ blood loss, sexual disturbances
- cannot be done if aorta is aneurysmal
- difficult if extensive external iliac disease - 2H problems in raising proper endarterectomy plane due to inherently adherent & muscular media in that location

#### Complications

- Cardiorespiratory complications
- Stroke
- Anticoag associated complications - bleeding
- Renal failure
- Graft thrombosis
- Hematoma
- Bowel ischemia
- Peripheral embolisation
- Erectile dysfunction
- Chylous leak
- Graft infection
- Anastomotic pseudoaneurysm

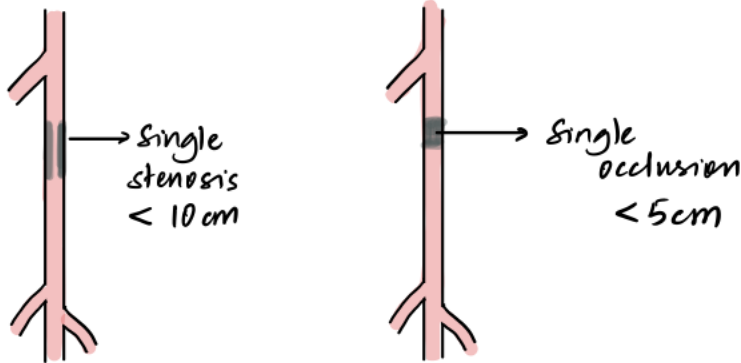
AORTOENTERIC FISTULA

AORTOURINARY FISTULA

# FEMOROPOPLITEAL DISEASE

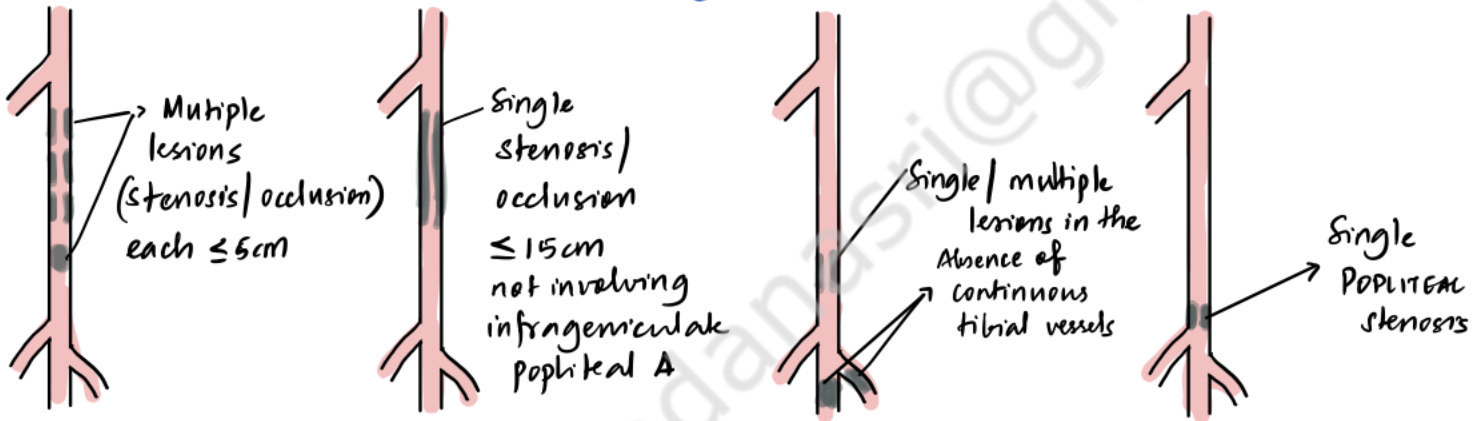
## TRANS ATLANTIC INTER SOCIETY CONSENSUS - TASC CLASSIFICATION

### TYPE A



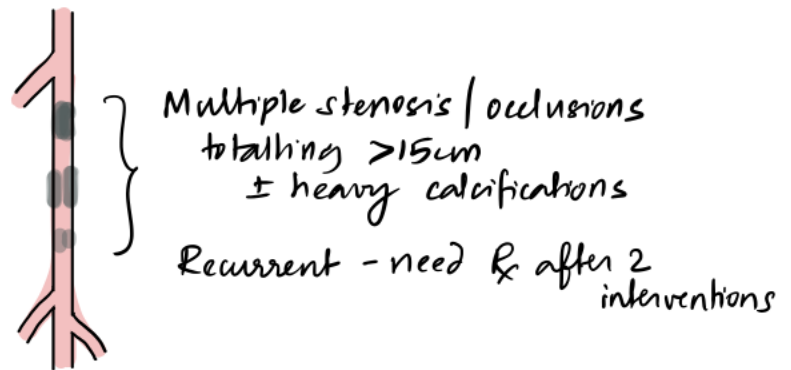
Endovascular procedure  
↓  
Treatment of choice

### TYPE - B - Endovascular > Surgery

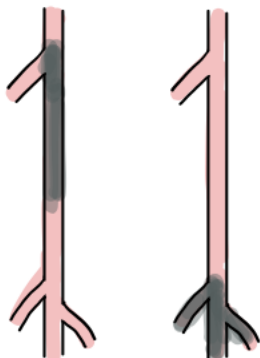


### TYPE - C -

Surgery > Endovascular



### TYPE - D → Surgery is procedure of choice



Chronic total occlusions of CFA, SFA  $> 20\text{ cm}$ , involving popliteal

Chronic total occlusion of popliteal A & its branches



# THROMBOANGIITIS OBLITERANS

SYN: BUERGER'S DISEASE

TAO is a chronic, inflammatory, thrombotic, non-atherosclerotic, segmental obliterative, tobacco-associated vasculopathy primarily involving the infrapopliteal (and infraulnar) medium-small arteries, predominantly in young, male smokers

## SHONOYA CRITERIA (5/5)

- Age at onset < 50y [ Papa & Adar: <30-40y ; <sup>Ohn,</sup> Mills & Peeter: <45y ]  
[ Jap MHW, European TAO - <50y ]
- Smoking
- Distal extremity ischemia (infrapopliteal / infraulnar)
- Upper limb involvement - OR -  
Phlebitis migrans
- Absence of atherosclerotic risk factors other than smoking (T<sub>2</sub>DM, HTN, Hyperlipidemia, proximal source of emboli, collagen vascular disease)

## EPIDEMIOLOGY

- Age < 45y - age of onset → Median age of diagnosis → 34y
- M >>> F (90% : 10%)  
(98% : 2%)
- Tobacco use → most consistent association  
- higher in those who smoke home-made tobacco products  
- role of ? Arsenic

## PATHOPHYSIOLOGY

### Uncertain

- Immunologically mediated injury (? autoimmune mechanisms) in response to smoking
- Endothelial dysfunction
- Hypercoagulable states
- Genetic predisposition
- Oral infection - inflammatory pathway?

THROMBOTIC DISORDER complicated by transmural neutrophilic infiltration

# PATHOLOGIC STAGES OF BUEGGER'S DISEASE

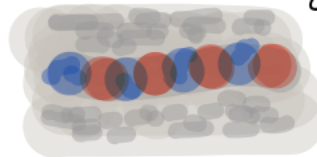
ACUTE PHASE	INTERMEDIATE/SUBACUTE PHASE	CHRONIC PHASE
<p>Panvasculitis</p> <ul style="list-style-type: none"><li>- small &amp; medium sized arteries</li><li>- superficial veins</li></ul> <p>Occlusive, highly cellular arterial thrombus &amp; MICROABSCESSSES</p>	<p>Progressive organisation of occlusive thrombus</p> <p>Partial recanalisation &amp; disappearance of microabscesses</p> <p>Deposition of immunoglobulins &amp; complement factors along the elastic lamina</p>	<p>Organisation of the thrombus &amp; extensive recanalisation</p> <p>Perivascular fibrosis</p> <p>Well preserved vessel wall architecture (even all stages)</p>

## CLINICAL FEATURES

- FOREFOOT CLAUDICATION / LOWER CALF CLAUDICATION ⇒ INFRAPOPLITEAL DISEASE  
(Isolated calf claudication - unusual)
  - Coldness
  - Burning sensation in hands & feet
  - Dependent rubor
  - Cyanosis - Raynaud phenomenon
  - Migratory superficial thrombophlebitis
  - Trophic nail changes, subungual splinter hemorrhages
  - ischemic ulcerations
  - Digital gangrene & Superadded infection
  - Multiple limb involvement \*\*
    - Isolated upper limb inv - 5%
    - Isolated lower limb inv - 75%
    - Upper + Lower limb → 20%
- ∴ TAO - Duplex of all 4 limbs

## DIAGNOSTIC EVALUATION

- 4 limb duplex - segmental inv, 'snake sign' / 'dot' sign - coarctation collaterals
- ABPI
- TcPO<sub>2</sub>
- Cardiac evaluation
- Blood work for glycaemic status, lipids etc.



Color Doppler

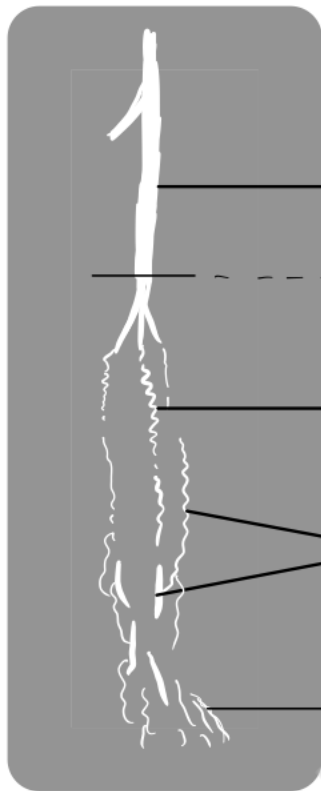


# ANGIOGRAPHY

- Conventional / DSA - invasive
- CTA - non invasive
- MRA

## Features

- Segmental involvement - diseased segments interspersed i normal segments
- more severe disease distally
- involvement of digital arteries
- (N) proximal arteries w/o evidence of atherosclerosis
- COLLATERALS - Corkscrew collaterals - Marderell sign  
Tree root / Spider leg



also seen in CRCT so

SLE

RA

APLA

Cocaine, amphetamine, cannabis abuse

INFRAPOPUITEAL DISEASE

Corkscrew collaterals

Segmental involvement

Distal involvement

R/O

- Thromboembolism

- other vasculitides

Scleroderma

PAN

RA

APLA

## BROWN VASOMOTOR INDEX

To determine the usefulness of sympathectomy in treating the vasospastic component of limb ischemia

Brown's vasomotor index =  $\frac{\text{Rise of skin temperature} - \text{Rise of mouth temp}}{\text{Rise of mouth temperature}}$  → after blockade

Sympathectomy is advisable when index is  $\geq 3.5$

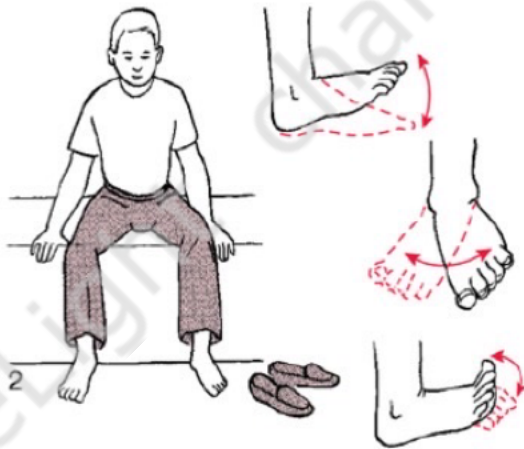
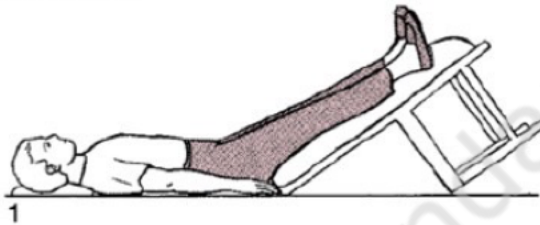
# MANAGEMENT OF TAD

## A. LIFESTYLE CHANGES

- 1) **SMOKING CESSATION** - Advice, counselling, Nicotine replacement, Bupropion
- 2) **LIMB CARE** - Daily foot hygiene, moisturization, lambis wool between toes, avoidance of trauma, barefoot walking
- 3) **EXERCISES** - Buerger & Allen exercises → supposed to improve claudication distance by inducing development of collaterals

### **Buerger-Allen exercises [ber'ger al'en]**

Specific exercises intended to improve circulation to the feet and legs. The lower extremities are elevated to a 45 to 90 degree angle and supported in this position until the skin blanches (appears dead white). The feet and legs are then lowered below the level of the rest of the body until redness appears (care should be taken that there is no pressure against the back of the knees); finally, the legs are placed flat on the bed for a few minutes. The length of time for each position varies with the patient's tolerance and the speed with which color change occurs. Usually the exercises are prescribed so that the legs are elevated for 2 to 3 minutes, down 5 to 10 minutes, and then flat on the bed for 10 minutes.



Buerger-Allen exercises. 1. Elevate feet on padded chair or board for 1/2 to 3 minutes. 2. Sit in relaxed position while each foot is flexed and extended then pronated and supinated for 3 minutes. The feet should become entirely pink. If the feet are blue or painful, elevate them and relax as necessary. 3. Lie quietly for 5 minutes, keeping legs warm with a blanket. From Black and Matassarini-Jacobs, 1997.

## MEDICAL TREATMENT

- Antibiotics } for ulcers & infection; rest pain, phlebitis
- Analgesics }
- CCBs - help & vasospasm
- IV sympathetic blocks & guanethidine
- Statins - pleiotropic effects
- PG analogs - Iloprost, Beraprost
- Cilostazol

## ENDOVASCULAR TREATMENT

Selective intra-arterial infusion of streptokinase / urokinase } Poor results  
Percutaneous subintimal angioplasty for limb salvage }

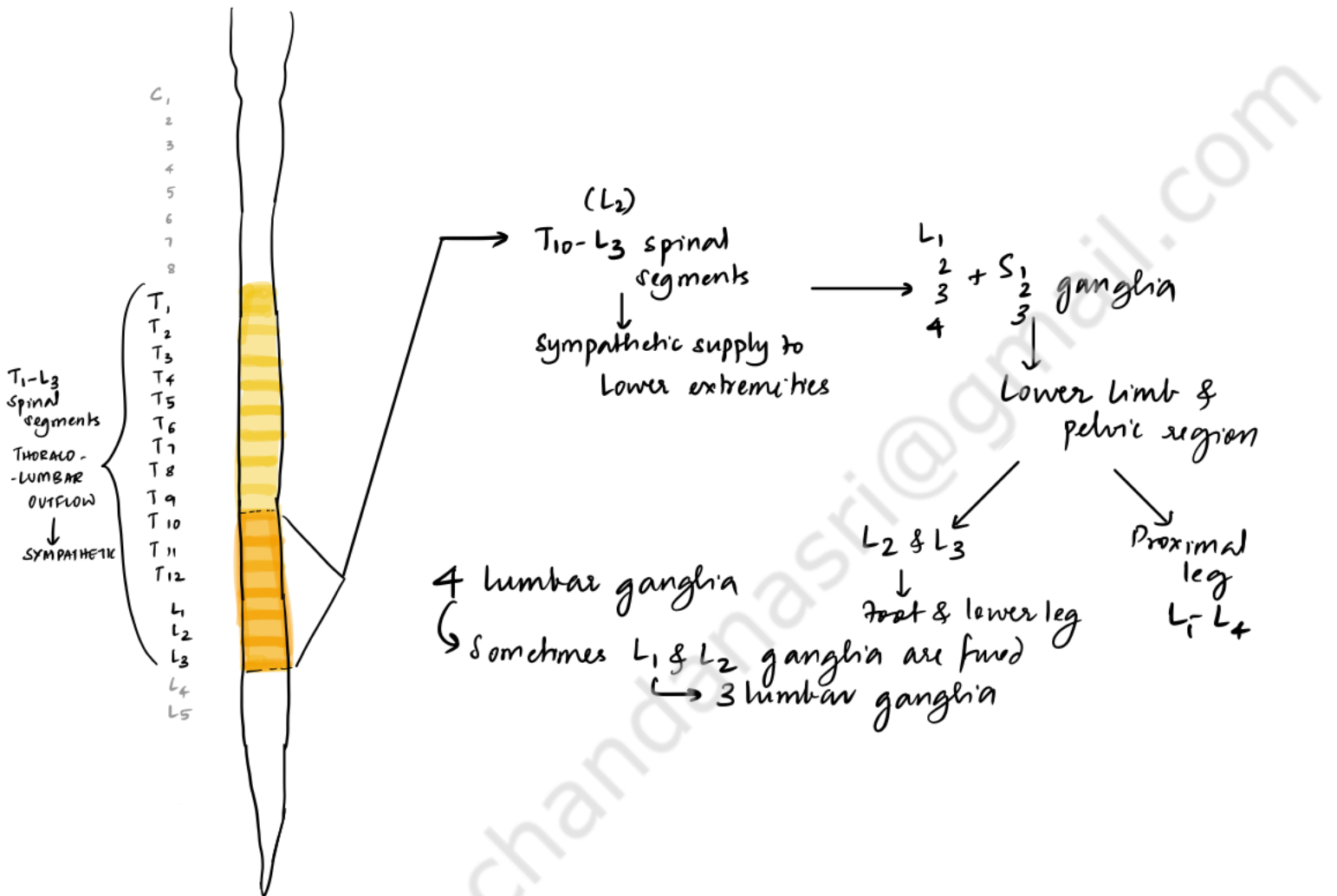
## SURGERY

- Revascularisation - generally not feasible because of diffuse, segmental arterial involvement and distal nature of the disease  
Suboptimal distal bypass patency rates
- Microvascular flap / Omental transfer
- Distal venous arterialisation
- Lumbar sympathectomy - doubtful, temporary
- Distraction vasculogenesis - unproven
- Debridement / Amputation

# LUMBAR SYMPATHECTOMY

## ANATOMY

Sympathetic outflow to the lower extremities originates in spinal segments  $T_{10}-L_3$



Crossover fibres in 15% pts - most at 4<sup>th</sup> ganglia

For most clinical indications, L<sub>2</sub> & L<sub>3</sub> gangliectomy is sufficient, but removal of L<sub>4</sub> ganglion is advised to reduce possibility of collateral innervation

- Sympathetic denervation causes vasodilation of arterioles in cutaneous beds - helps in healing cutaneous ulcers, reduces ischemic rest pain
- No role of lumbar sympathectomy in claudicants

## CHEMICAL SYMPATHECTOMY

Under fluoroscopic guidance - alcohol / phenol injected

absolute alcohol

6.5-7% phenol in sterile water

Landmarks: L<sub>1</sub> - junction of 12<sup>th</sup> rib & erecta spine

L<sub>4-5</sub> - level of transverse line joining posterior iliac crests

tips of needles placed against the bodies of L<sub>2</sub>, L<sub>3</sub>, L<sub>4</sub> bodies under fluoroscopic guidance

3ml injected through each needle

## SURGICAL SYMPATHECTOMY

OPEN

RETROPERITONEOSCOPIC

### Retroperitoneal approach

#### 'ANTEROLATERAL APPROACH OF FLOWTHOW'

- incision beginning at the lateral edge of the rectus muscle upto the anterior axillary line - midway between the costal margin & iliac crest
- muscles split along the line of their fibres
- Plane between transversalis fascia & the peritoneum easily developed by finger dissection to enter the retroperitoneum
- The ureter and genadal muscles are lifted up away from the psoas muscle towards the peritoneum as the dissection proceeds medially

- The lumbar sympathetic chain is located medial to the psoas muscle and lies over the transverse processes of the lumbar spine

(R) just beneath the edge of IVC

(L) - adjacent & lateral to abdominal aorta

- Genitofemoral nerve lies more laterally, on medial 1/3<sup>rd</sup> of psoas muscle

Transperitoneal approach

↓  
only applicable for sympathectomy combined with abdominal aortic procedure

CATTELL  
BRAASCH

MATTOX

tactile identification

↓  
characteristic 'snap' on plucking

↓  
Ganglion ID by counting up from sacral promontory

↓  
L<sub>3</sub> ganglion level  
L<sub>2</sub>, L<sub>3</sub> removed

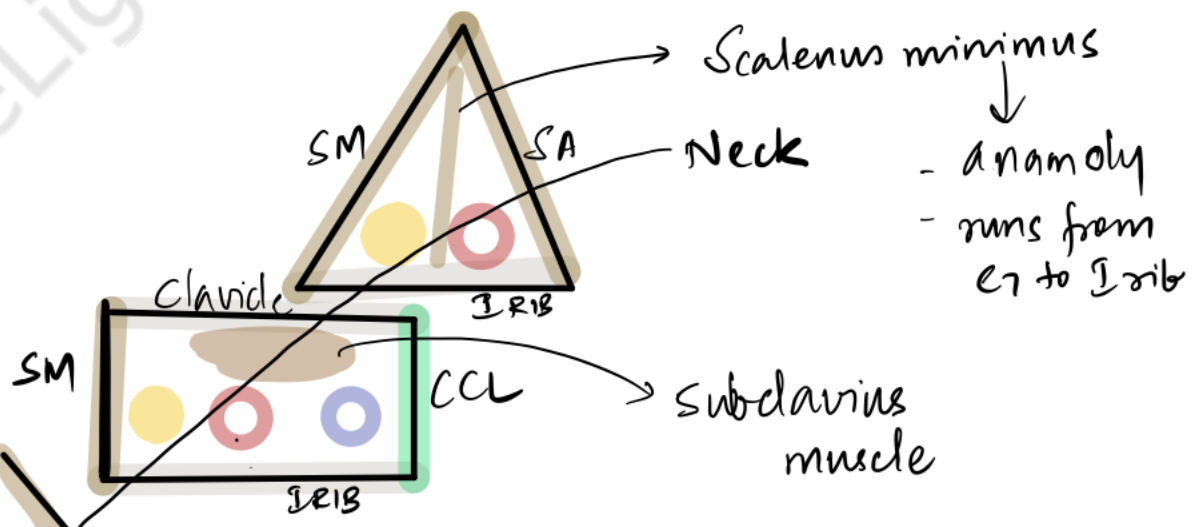
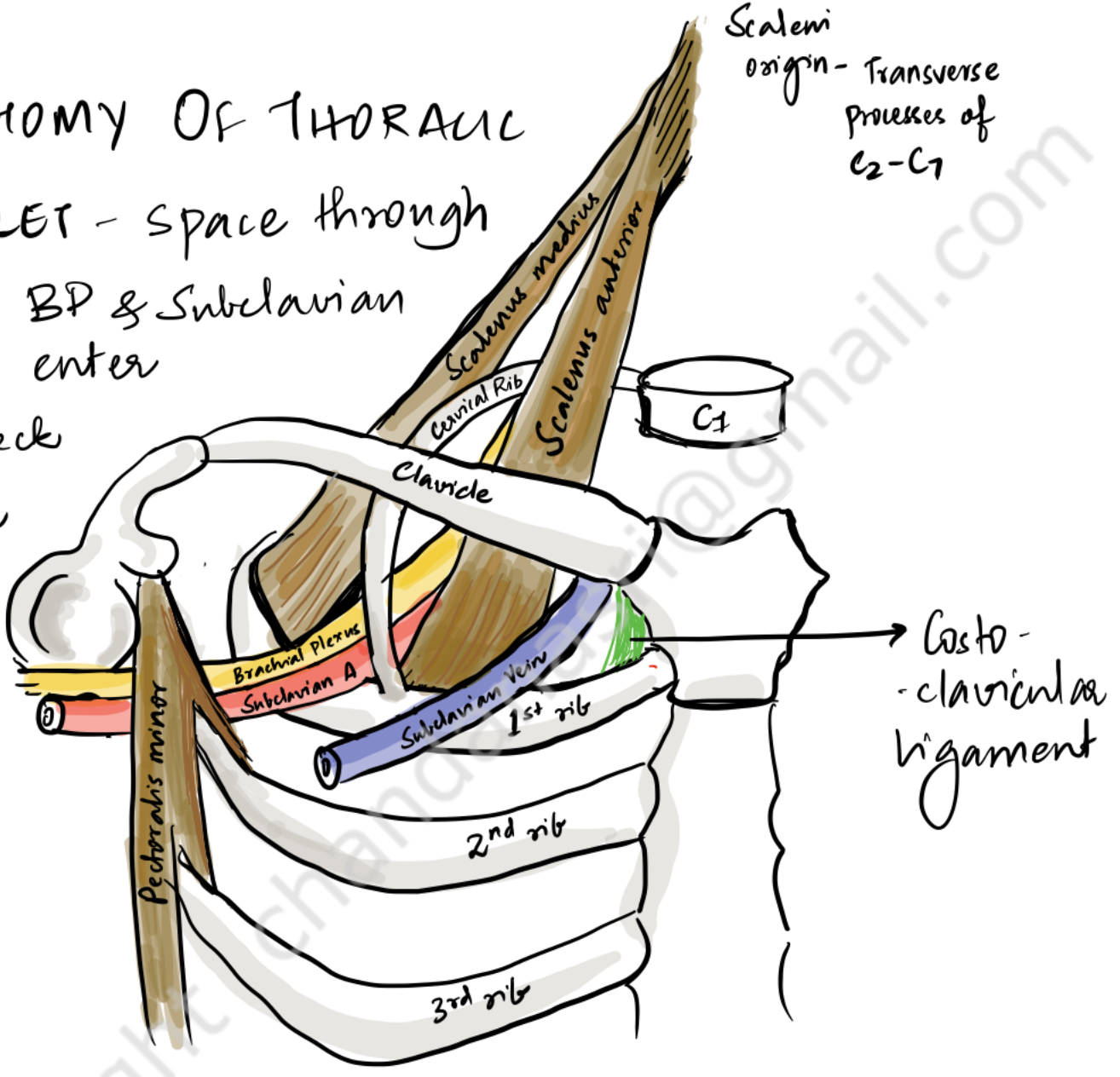
## COMPLICATIONS

- Neuralgia - along anterolateral thigh
- Retrograde ejaculation - seen in 25-50% cases post B/L L<sub>1</sub> sympathectomy
- ? Systemic arterial steal syndromes
- Injuries to genitofemoral n  
ureter  
lumbar veins  
aorta  
IVC

SurgeLight chandanasri@gmail.com

# THORACIC OUTLET SYNDROME

ANATOMY OF THORACIC  
OUTLET - space through  
which BP & Subclavian  
vessels enter  
from neck  
to axilla



Thorax ← Pectoralis minor

## Definition:

A constellation of symptoms occurring due to compression of the brachial plexus (Neurogenic TOS) and/or the subclavian vessels (Vascular TOS) at the thoracic inlet

Compression may occur at

- Interscalene triangle (Cervical outlet)
- Costoclavicular space (so called true thoracic outlet)
- Subcoracoid area

## ► Interscalene triangle:

Compression of BP & SA

- Causes: • Cervical rib
- Scalenus minimus

→ Clinical features: Neurogenic TOS  
Arterial Vascular TOS

- Rx
- Cervical rib excision
- Scalenectomy
- I rib excision

Vertebrobasilar insufficiency

(∵ Vertebral artery originates close to Sc. anterior)  
+ve ADSON'S, ROOS



## ▶ COSTOCLAVICULAR COMPRESSION

Compression of BP, SA, SV

→ Causes: Anomalous clavicle

Anomalous I rib

Cervical rib

Elongated anomalous

C7 transverse process

Fibrous & cartilagenous bands

→ Clinical features:

Neurogenic TOS

Vascular (Arterial + Venous) TOS

**ve HAUSTED'S TEST**

→ Rx - Excision of I rib

Excision of cervical rib

## ▶ SUBCORACOID COMPRESSION: Neuro + Vasc TOS

**ve Wright Hyperabduction**

[http://www.slideshare.net/povilas1/thoracic-outlet-syndrome-anatomy-symptoms-diagnostic-evaluation-and-surgical-treatment?from\\_m\\_app=android](http://www.slideshare.net/povilas1/thoracic-outlet-syndrome-anatomy-symptoms-diagnostic-evaluation-and-surgical-treatment?from_m_app=android)

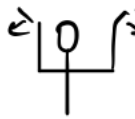
Nice  
reference

# CLINICAL TESTS - EVOCATIVE TESTS

contracts scaleni

holding breath raises 1st rib

- **Adson (scalene) test.** The patient inspires maximally and holds his or her breath while the neck is fully extended and the head is turned toward the affected side. This maneuver narrows the space between the scalenus anticus and medius, resulting in compression of the subclavian artery and the brachial plexus. Decrease or loss of ipsilateral radial pulse suggests compression.
- **Halsted (costoclavicular) test.** The patient is instructed to place his or her shoulders in a military position (drawn backward and downward) to narrow the costoclavicular space between the first rib and the clavicle, causing neurovascular compression. Reproduction of neurologic symptoms or decrease or loss of ipsilateral radial pulse suggests compression.
- **Wright (hyperabduction) test.** The patient's arm is hyperabducted 180 degrees, which causes the neurovascular structures to be compressed in the subcoracoid region by the pectoralis tendon, the head of the humerus, or the coracoid process. Decrease or loss of ipsilateral radial pulse suggests compression.
- **Roos test.** The patient abducts the involved arm 90 degrees with external rotation of the shoulder. Maintaining this body position, the modified Roos test is performed by opening and closing the hand rapidly for 3 minutes in an attempt to reproduce symptoms. Additionally, neurogenic compromise may be detected using provocative tests, such as percussion of the nerve (Tinel sign) or flexion of the elbow or wrist (Phalen sign).

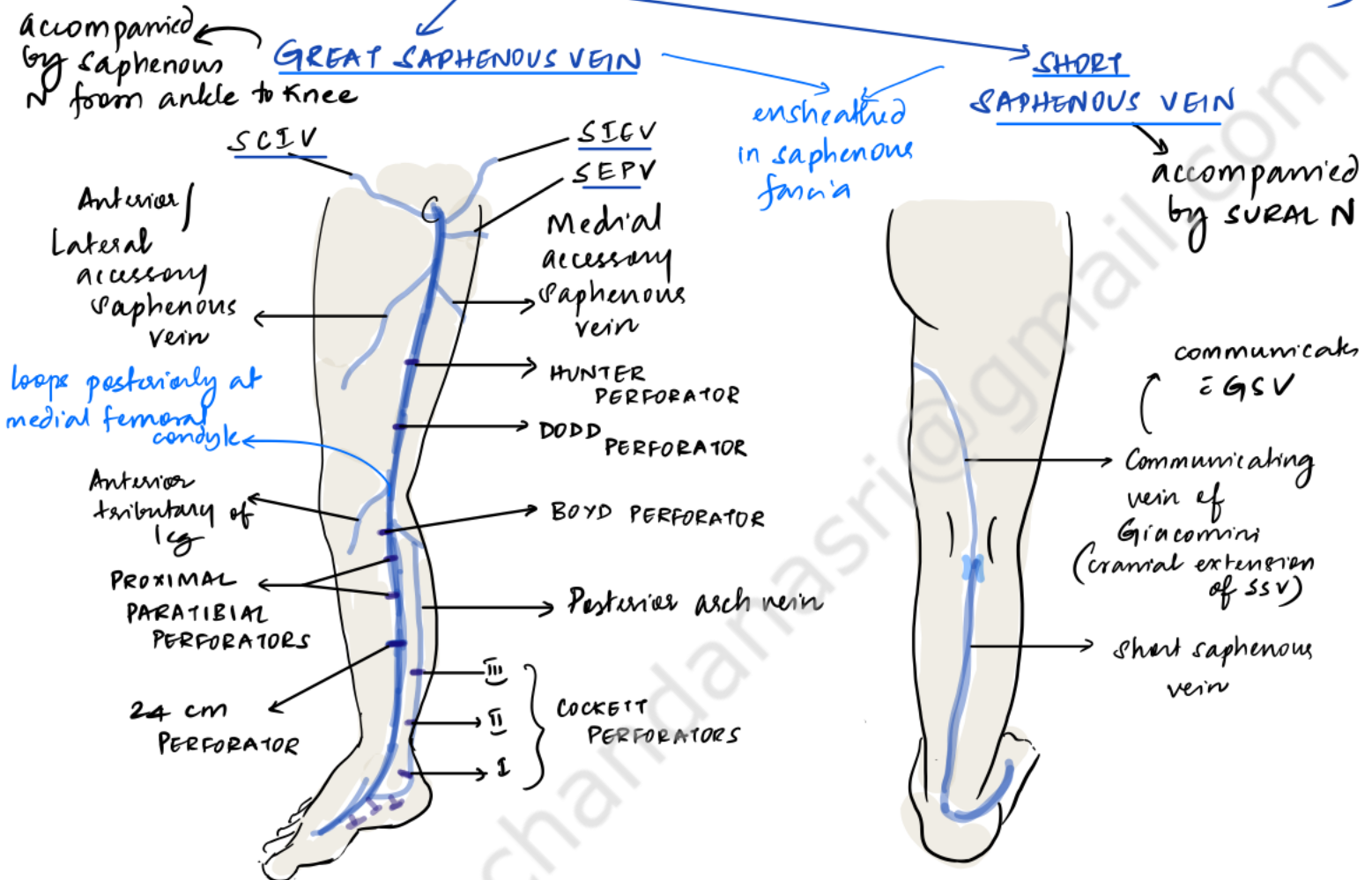


- **Allen's test** → to test integrity of palmar arch & its contributors

# CHRONIC VENOUS INSUFFICIENCY

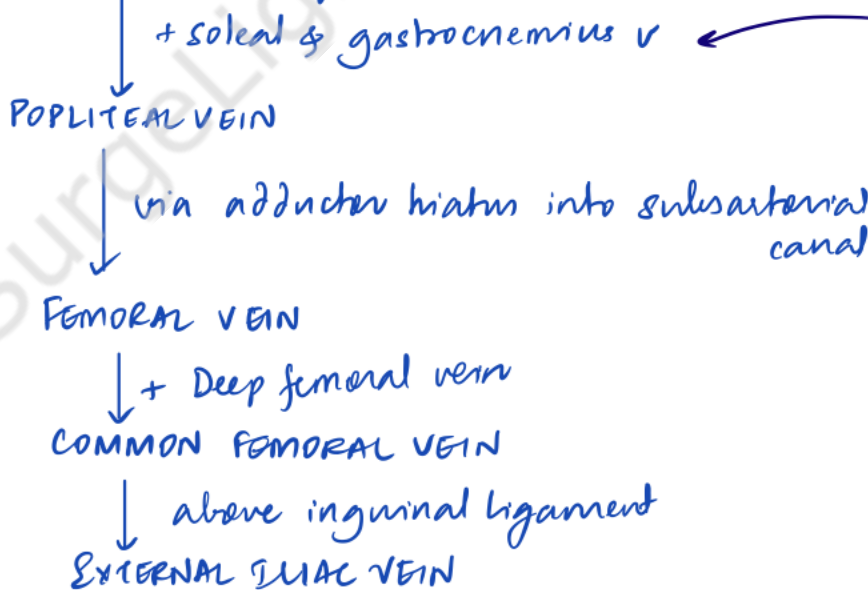
## LOWER LIMB VENOUS ANATOMY

① Superficial venous system - located superficial to the deep fascia (fascia lata)



② Deep venous system

venae comitantes of 3 central arteries (ATA, PTA, Peroneal A)



### Venous sinuses

- thin-walled large veins located within the substance of soleus & gastrocnemius muscles
- valveless
- linked by valved channels that prevent reflux

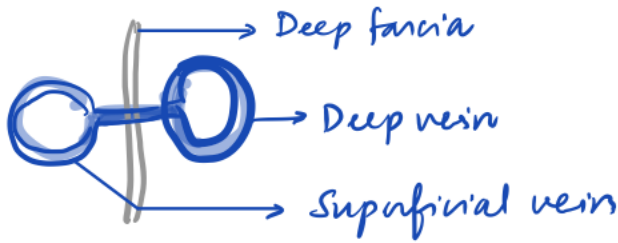
### ③ PERFORATORS

- Connect the direct and indirect systems

#### TYPES

##### DIRECT

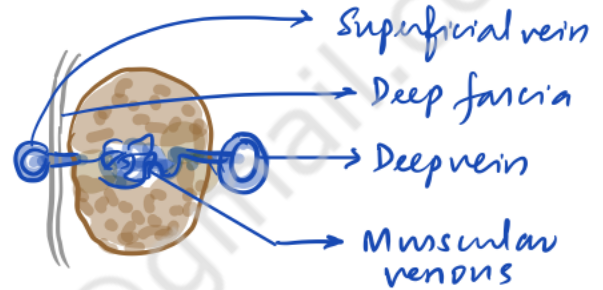
permit the superficial venous system to communicate directly with the main deep veins



more constant in location

##### INDIRECT

connect the superficial system to the deep system via a muscular vein



irregularly distributed sinuses

- perforators also communicate with each other via communicating vein over & beneath the deep fascia
- most perforators have valves
- Incompetent perforator veins play a key role in the genesis of venous ulcers.

### VENOUS PHYSIOLOGY

VEINS are capacitance vessels - contain ~60% of total blood volume

#### Venous hemodynamics

- Cardiac pumping generates dynamic pressure gradients, with arterial pressure being higher than venous pressure

#### IN SUPINE POSITION

Capillary pressure at the arteriolar end - ~32 mmHg

Capillary pressure at the venular end - ~12 mmHg

Right atrial pressure - ~4-7 mmHg

↓ Pressure gradient aids venous return

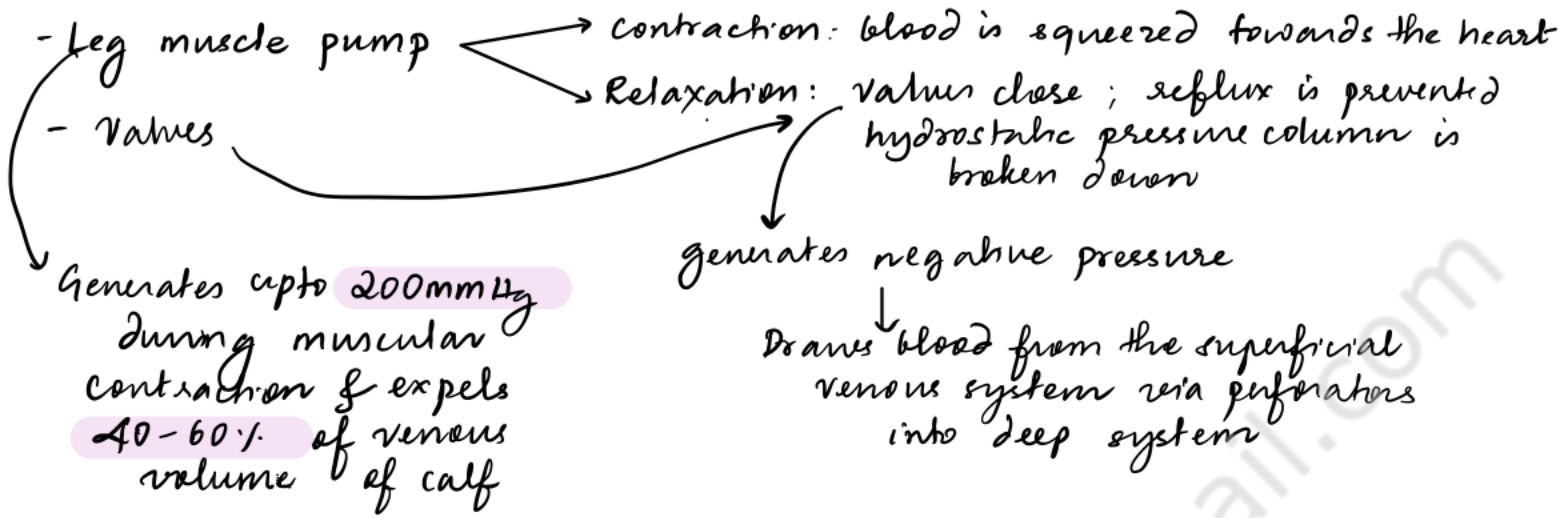
#### IN UPRIGHT POSITION

Venous flow in lower extremities is dominated by the effects of **HYDROSTATIC PRESSURE** (height of the column of blood below the atrium)

$\approx 0.77 \text{ mmHg/cm}$  of vertical distance from RA  
 $= 100 \text{ mmHg}$  in the foot vein

∴ For venous return, this hydrostatic pressure has to be overcome

## FACTORS AIDING VENOUS RETURN



AMBULATORY VENOUS PRESSURE - measured in dorsal foot vein (superficial system) based on the observations that variations of venous pressure in the superficial system reflect those in the deep system.

In normal subjects

- standing venous pressure is  $\sim 100\text{mmHg}$  (baseline)
- During exercise  $\rightarrow$  venous pressure falls to  $\sim 30\text{mmHg}$  (by calf muscle pump action & competent valvular mechanism)
- After exercise  $\rightarrow$  pressure slowly rises to baseline standing pressure

$\sim 90\%$  of baseline pressure is attained in  $\sim 1/2$  min

### ABNORMALITIES

- Failure of pressure to fall during exercise  $\rightarrow$  INEFFECTIVE MUSCLE PUMP
- Pressure rising during exercise  $\rightarrow$  OCCLUSION OF DEEP VEINS
- Rapid rise of pressure post exercise  $\rightarrow$  VALVULAR INCOMPETENCE

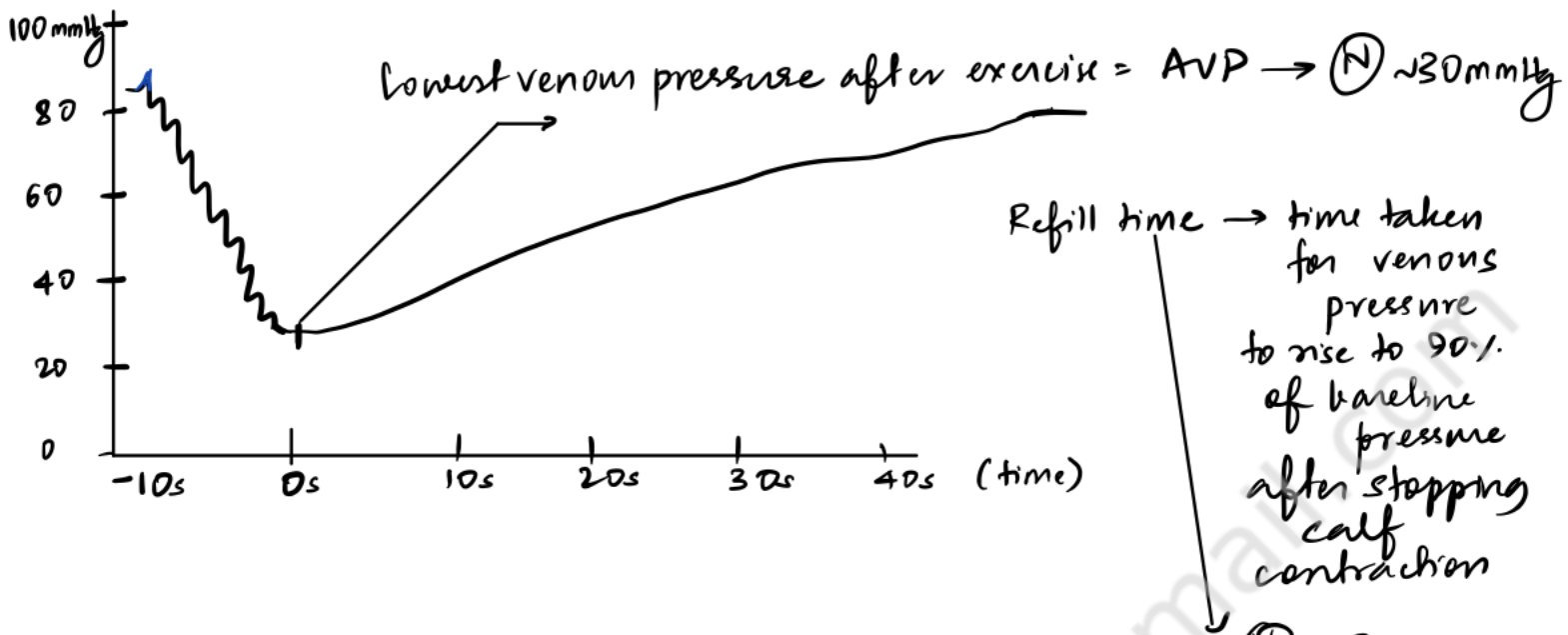
AVP definition: pressure measured in the dorsal foot vein after 10 tip-toe maneuvers (at 1/second)

(N)  $\rightarrow \sim 30\text{mmHg}$

Refill time: time taken to return to 90% of baseline pressure

$\uparrow$  AVP  $\Rightarrow$  more severe CVI;  $\uparrow$  AVP also perforator incompetence, obstruction correlates  $\bar{c}$  incidence of venous ulcer ( $80\%$  at AVP  $\geq 80\text{mmHg}$ )

Deep venous insufficiency  $\rightarrow$  higher AVP than superficial insufficiency  
HIGHEST AVP values - seen in those with deep valvular incompetence & proximal obstruction



shorter the Recovery time,  
greater the degree of  
reflux

## CAUSES OF VENOUS HYPERTENSION

### • Pressure gradient dysfunction

↑ Abdominal / thoracic pressure

- COPD
- Pregnancy
- Obesity
- Tumor
- Constipation

Decreased Calf muscle pump action

- Immobility
- Ankle joint fusion
- Paralysis

### • VENOUS SYSTEM ABNORMALITIES

#### STRUCTURAL DEFECTS

- Valvular agenesis / incompetence
- AV fistula
- Venous wall defects
  - Dilatation
  - Tortuosity
  - ↓ Compliance
  - ↓ Tone

#### OCCCLUSION

- Agenesis
- Thrombosis
- Pelvic / abdominal RT

#### COMPRESSION

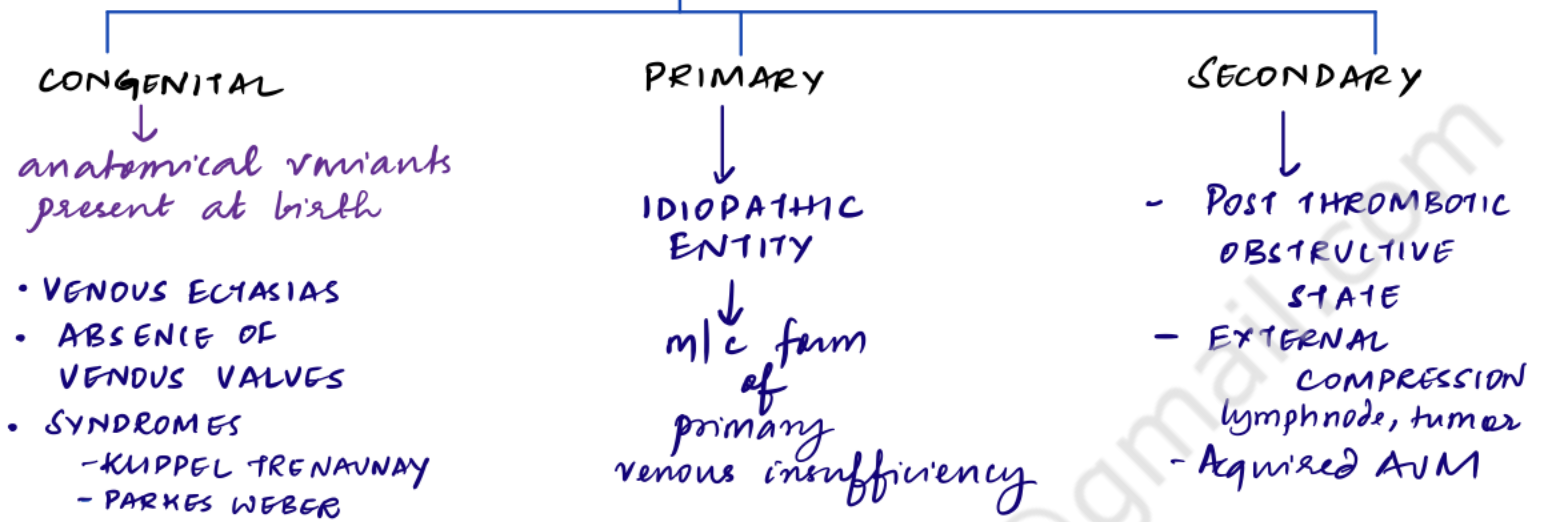
- May-Thurner Syndrome
- Pelvic / abdominal tumor

MAJORITY OF PATIENTS WITH VENOUS DISEASE have a PRIMARY problem with vein wall structure (leading secondarily to valvular dysfunction, rather than the other way round) and this is mostly confined to superficial veins

# CHRONIC VENOUS INSUFFICIENCY

- spectrum of pathological manifestations of venous hypertension

## CATEGORIES



## Primary venous insufficiency

- m/c cause of CVI
- mainly involves the superficial system
- can involve the perforator system as well

## SPECTRUM

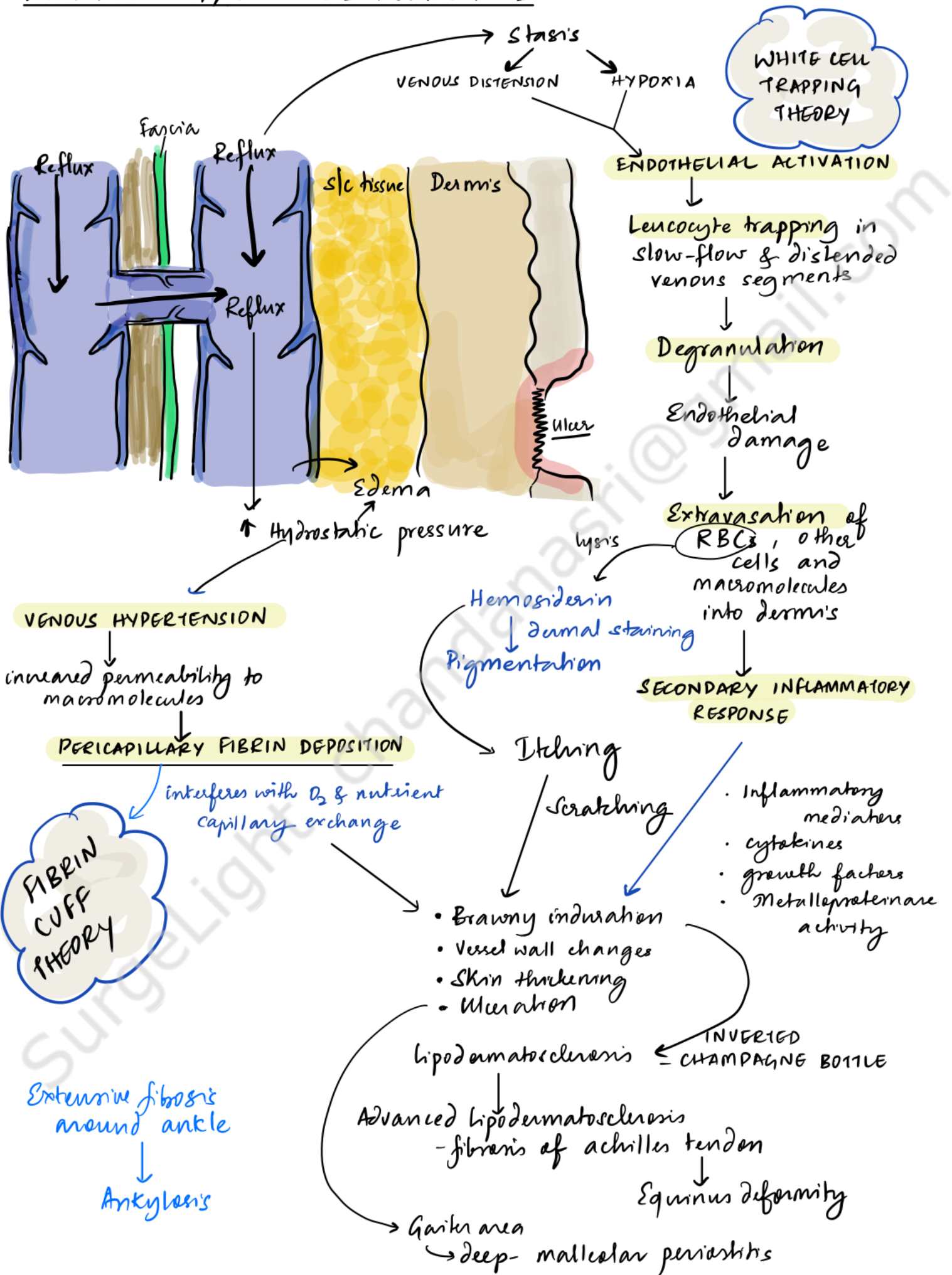
### VEINS

- TELANGECTASIA (thread veins/spider veins/hyphen-wels) - intradermal venules < 1mm in diameter
- CORONA PHLEBECTATICA / MALGOLAR FLARE: fan-shaped pattern of telangectasia on the ankle/feet
- RETICULAR VEINS - small dilated bluish SUBDERMAL veins 1-3mm
- VARICOSE VEINS: elongated, tortuous, dilated subcutaneous veins ≥ 3mm & demonstrable reflux
- SAPHENA VARIX: groin varicosity & expansile cough impulse

### STASIS

- Edema - Starling's forces disturbed d/t venous hypertension <sup>net filtration overwhelms lymphatics</sup>
- Eczema - itching
- Pigmentation - Hemosiderin, ankle & perforator area
- Lipodermatosclerosis - chronic inflammation & fibrosis of skin & subcutaneous tissue
- Atrophie blanche - localised areas of atrophic white skin surrounded by flare
- Venous ulcer

# PATHOPHYSIOLOGY OF STASIS DERMATITIS





# EPIDEMIOLOGY

- F > M
- Age > 40y
  - ↑ BMI
  - ↑ Height
- Pregnancy - impact of progesterone on venous tone, mechanical effects of gravid uterus
- Family history  
FOXC2 gene - Primary venous insufficiency
- Occupation - Prolonged standing, ↓ ambulation
- Lifestyle - sedentary

## CEAP Classification (Devised by American venous forum)

CLINICAL	ETIOLOGICAL	ANATOMICAL	PATHOLOGICAL
C <sub>0</sub> - No visible/palpable venous disease	E <sub>c</sub> - Congenital	A <sub>s</sub> - superficial system	P <sub>R</sub> - Reflux
C <sub>1</sub> - Telangiectasia, reticular veins, malleolar flare	E <sub>p</sub> - Primary	A <sub>d</sub> - deep system	P <sub>O</sub> - obstruction
C <sub>2</sub> - Varicose veins	E <sub>s</sub> - Secondary	A <sub>P</sub> - perforators	P <sub>R/O</sub> - reflux, obstruction
C <sub>3</sub> - Edema WITHOUT skin changes		Segmental subclassification (Sabiston 20e - pg 1824)	
C <sub>4</sub> - 4a - pigmentation, eczema 4b - lipodermatosclerosis			
C <sub>5</sub> - healed venous ulcer			
C <sub>6</sub> - active venous ulcer			

## CLINICAL TESTS

TRENDELENBERG  $\left\{ \begin{array}{l} \text{i} - \text{SFI incompetence} \\ \text{ii} - \text{Perforator incompetence} \end{array} \right.$

MULTIPLE TOURNIQUET TEST - to localise the incompetent perforators

PERTHE'S TEST

MODIFIED PERTHE'S TEST

SCHWARTZ TEST

FEGAN'S TEST

PRATT'S TEST

MORRISSEY COUGH IMPULSE TEST

Tourniquet, hand held  
Doppler

## Complications

- Superficial thrombophlebitis
- Acute bleeding originating in one of the thin walled varices
- Eczema
- Skin changes - Ulceration
- Contractures
- Periostitis

Venous ulcer

Allergy - dermatitis

Rupture - bleeding

Inflammation - thrombophlebitis

Calcification

Osteitis (periosteitis)

Sclerosis - lipodermatosclerosis

Equinus deformity

## EVALUATION OF VARICOSE VEINS

### ① VENOUS DUPLEX:

- High frequency linear transducer - 7.5-13 MHz - B-mode settings

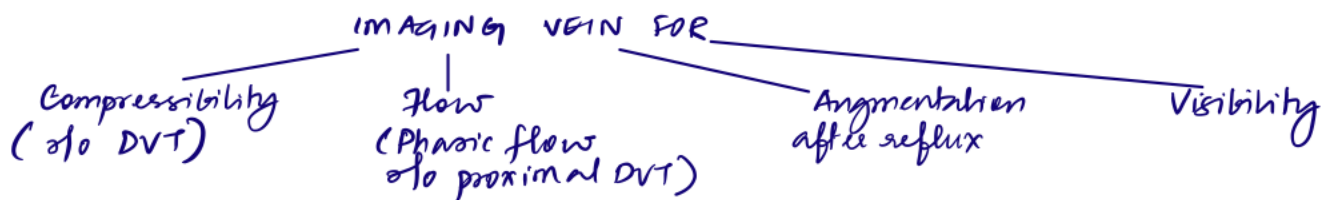
Pulsed wave - 3 MHz

- Color doppler - optimised for low-flow velocities within veins

Blue - towards

Red - away

Calf squeeze augments venous flow



- Presence of reflux in superficial / deep system
- exact distribution and extent of reflux in the superficial venous system, including affected junctions & perforators
- Presence of obstruction in deep system → DVT  
superficial system → thrombophlebitis
- Suitability of incompetent superficial veins for various modalities
  - DIAMETER
  - EXTENT
  - TORTUOSITY
  - SAPHENA VARIX
- Histo pelvic source of reflux / obstruction

SUPERFICIAL REFLUX - Retrograde flow lasting for  $\geq 0.5s$

DEEP REFLUX - Retrograde flow lasting for  $\geq 1s$

REFLUX → elicited by release of calf / foot squeeze / Pneumatic cuff deflation  
manual compression over varicosity clusters  
active foot dorsiflexion  
Valsalva maneuvers

MICKEY MOUSE SIGN - CFA, CFV, GSU - transverse views groin

• Groin - check for junctional incompetence

loss of phasic flow & expiration in CFV ⇒ proximal obstruction  
do pelvic imaging

## ② Venography

- In secondary venous insufficiency - like PTS - to image the extent if intervention is contemplated

Ascending venography

- Descending venography - done as a pre-operative adjunct when deep venous reconstruction is planned

- Varicography - recurrent varicose veins; anomalous veins

- MR venography - non-invasive

for evaluation of abdominal & pelvic venous vasculature  
(Iliac veins up to IVC)

Evaluation of pelvic congestion syndrome

# MANAGEMENT OF CHRONIC VENOUS INSUFFICIENCY

## CONSERVATIVE MEASURES

### LIFESTYLE MANAGEMENT

- Avoid prolonged standing
- Venous ulcer - avoid vigorous exercise

### EXERCISE

- ↑ MOBILITY & MODERATE PHYSICAL ACTIVITY → promote ulcer healing
- supervised exercise program to improve ankle mobility to prevent stiffness, contractures

### LIMB ELEVATION

- 30cm above heart level (Tees over nose) several times a day
- ↓ edema
- improves cutaneous microcirculation

### COMPRESSION THERAPY

- counters venous hypertension
- improves venous pump function
- reduces risk of VTE events

### Gradient elastic stockings

- Class 1: Prophylaxis; pregnancy, (10-20mmHg) symptom control in C<sub>1</sub>
- Class 2: C<sub>2</sub>-C<sub>3</sub>, DVT (20-30mmHg)
- Class 3 - Venous ulcer (30-40mmHg)

### CIRCAID

- UNNA BOOT - inner gauze: Calamine, Zinc oxide, glycerin, Sorbital, gelatin, Mg, Al + outer elastocrepe → stiff on drying → 50-60mmHg

- Layered elastic & non elastic compression bandages

- Intermittent pneumatic compression

### PHARMACOTHERAPY

- Diuretics ± in edema & CVI & concurrent CHF

- Zinc ±

- Pentoxifylline

- Phlebotropic agents

- Diosmin
- Flavonoids
- Ca Dobesilate

- Prostaglandins

- Aspirin

- Ifetrobane

## 4 LAYER BANDAGE

### ① ORTHOPEDIC WOOL

- Absorbs excessive exudates
- Distributes pressure
- Reduces pressure on areas unduly sensitive to pressure damage

### ② COTTON CREPE

- smooths the wool
- holds it in place

### ③ ELASTIC BANDAGE

- 1<sup>st</sup> compressive layer
- provides 1/3<sup>rd</sup> of the interface pressure

### ④ COHESIVE BANDAGE

- 2<sup>nd</sup> compressive layer
- ↑ stiffness
- 2/3<sup>rd</sup> of interface pressure

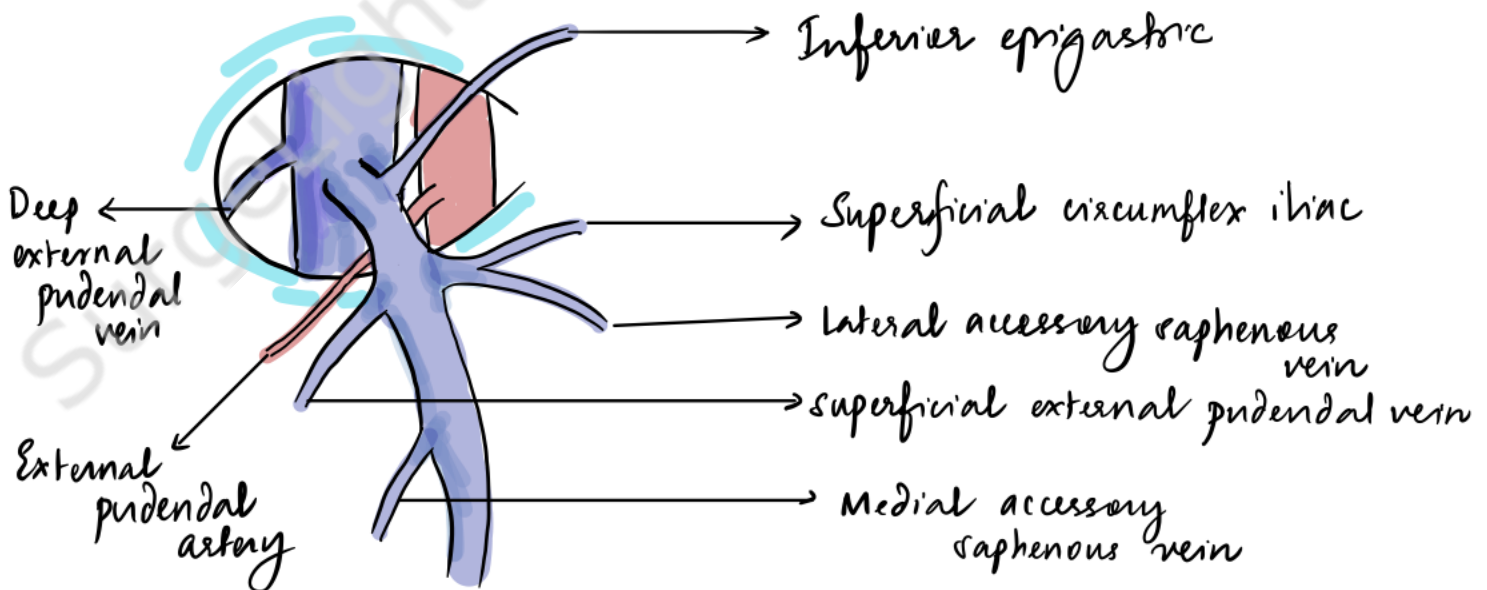
# INTERVENTIONAL PROCEDURES

## Removal / Ablation / Ligation of refluxing segment



### Definitive indications in the era of endovenous R

- 1) Very superficially placed involved venous segment
  - if the varicose segments are so closely applied to the skin that it is not possible to create atleast 1cm buffer zone between the vein & skin using tumescent anaesthesia (thermal ablation cannot be done safely)
  - will need stab phlebectomy
- 2) Grossly dilated segments / Aneurysmal segments  $\geq 2.5-3\text{cm}$ 
  - failure of EVA despite external compression & tumescent anaesthesia
- 3) Chronic thrombophlebitis, excessive tortuosity
  - prevent the advancement of catheter in endovenous techniques
- 4) Acute superficial thrombosis
- 5) Economic considerations & Physician choice



# SURGERIES

## FOR AXIAL VEINS

### TRENDELENBERG PROCEDURE

- Ligation of named tributaries
- Juxtafemoral flush ligation of the GSV
- ↓
- taking care not to
- narrow the Femoral vein
- leave a long stump

### LIGATION & STRIPPING OF GSV

- Reduces recurrence rates
- targeted stripping
- ↓
- strip only above knee to avoid saphenous (N) injury

### MYER / CODMAN STRIPPER

### Adjunctive procedures

- USG Guidance
- Tumescant anaesthesia
- ↓ Blood accumulation in stripping tunnel by tying a gauze to stripper
- leg elevation & compression while stripping

### ADDRESSING THE SSV

- Generally avoided ∴
- SSV incompetence is rare
  - does not contribute significantly to CVL of the limb
  - technical difficulties

### should be done if/when

- Posterior calf varicosities
- Recurrence
- isolated lateral malleolar ulcers
- ulcer recurrence after SEPS

### AMBULATORY PHLEBECTOMY

using multiple small stab incisions along the course of the varicosity

### 'STAB AVULSIONS'

- can be closed w/ steristrip
- can be done under LA

## FOR PERFORATORS

LINTON METHOD (1938 - Historical value): longitudinal incision to expose & ligate perforators - ↑ wound complications

COCKETT MODIFICATION - Extrafascial ligation

DODD, ROB - postromedial subfascial approach

PALMA - multiple small skin incisions along langer's lines for subfascial / subcutaneous ligation of perforators

→ Requires pre-op localisation

## NEWER ADVANCES

- TRIVEX - Powered phlebectomy - used to treat extensive branch varicosities (non axial / recurrent)
  - Pre-op marking of the entire course of the varicosity
  - 2mm incisions placed at the extremes of the varicosity
  - through the incisions, the transilluminator & resection devices are placed in the subcutaneous plane just deep to the varicosities
- SEPS - Subfascial Endoscopic Perforator Surgery

# ENDOVENOUS TECHNIQUES FOR THE MANAGEMENT OF CVI

## THERMAL METHODS

- ① USG guided catheter access into GSV  
i tip 2cm distal to SFJ
- ② Perivenous tumescent anaesthesia injected to create atleast 1mm cuff around vein - under USG guidance  
+ 50ml of 1% lidocaine + Adr  
+ 450ml NS  
+ 5-10ml 8% NaHCO<sub>3</sub>
- ③ Heat generation to destroy endothelial lining → non infective inflammatory reaction → thrombosis → luminal occlusion → Fibrosis
- ④ Compression - to aid apposition of vein walls & occlude lumen

### RFA

(VNUS, ClosureFAST)

Electromagnetic current → heat (upto 120°C)

Ablation cycle - 20s  
Coil length - 3cm, 7cm

Graded pullback - 7cm every 20s

↓  
Standardised treatment protocol

### Complications

- Vessel perforation
- Thrombosis/Phlebitis
- Skin injury/burn/pigment
- Recurrence
- Hematoma
- Infection
- Paresthesia

### EVLA

Small flexible optical fibre

↓  
LASER

↓  
targetted delivery of heat

810 - 1470 nm

Continuous pullback at 1cm every 3-5s  
i.e., 12-20cm/min

↓  
Requires experience

## NON THERMAL METHODS

### ① SCLEROTHERAPY

Injection of sclerosing agents into superficial veins → inflammation, thrombosis → fibrosis → obliteration

#### SCLEROSANTS:

OSMOTIC	DETERGENT	ALCOHOL
• Hypertonic saline (23.4%)	• STDS - 3%	Chromated Glycerin
• 10% NaCl + 25% D	• Ethanolamine oleate	
	• Polidocanol	

Liquid sclerotherapy - diluted sclerosant injected - dose depends on caliber & length of segment

Foam sclerotherapy - foam generated by Tessari technique - mixing sclerosant & air @ 1:5 ratio  
To-fro movement ~ 20 times  
Particle size ≤ 100µm

#### Catheter directed sclerotherapy

catheter based delivery of sclerosant under USG guidance

#### POST INJECTION COMPRESSION

### ② MECHANOCHEMICAL ABLATION 'ClariVein'

Mechanical injury to endothelium → vein spasm  
+  
Sclerosant → thrombosis - obliteration

### ③ ADHESIVE CLOSURE

'Venaseal'

Cyanoacrylate glue - polymerizes when it comes in contact i ionic substances (eg: blood components)

# RECURRENT VARICOSE VEINS

## Causes for recurrence

- Neovascularisation - development of new veins
- Reflux in residual vein
- Inadequate initial surgery
- Junctional reflux

Incidence - 10-20%.



## PROCEDURES FOR DEEP VENOUS INSUFFICIENCY

- Internal valvuloplasty
- External valvuloplasty
- External banding
- Value transplantation
- Value transposition

## PROCEDURES FOR SECONDARY VENOUS INSUFFICIENCY

### • POST THROMBOTIC LIMB

For chronic occlusion of distal femoral / proximal popliteal vein

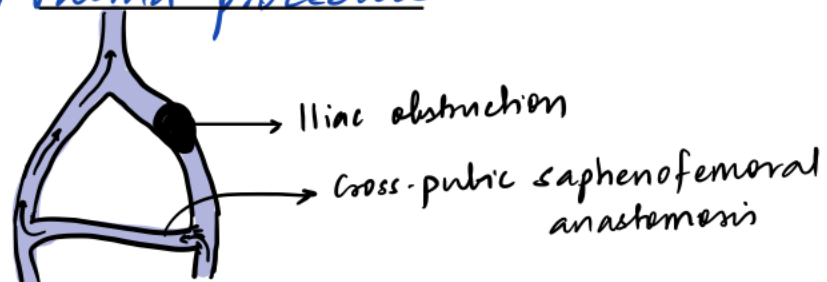
- Saphenopopliteal bypass - May-Husni procedure

(± temporary AV fistula to improve anastomotic patency)

For chronic unilateral iliofemoral obstruction, May-Thurner's<sup>o</sup>

- cross-pubic venous bypass / Palma procedure

- Hybrid procedures





# DEEP VEIN THROMBOSIS

Formation of intraluminal coagulum within the deep venous system

## PATHOPHYSIOLOGY - Virchow's triad

- 1) Stasis of blood flow
  - 2) Endothelial damage
  - 3) Hypercoagulability
- generally in secondary/provoked DVT
- generally in spontaneous/unprovoked DVT

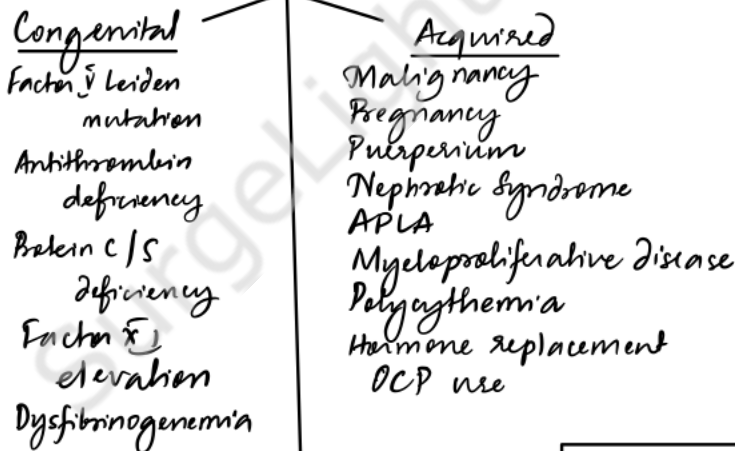
"Convergence of several risk factors on the background of an imbalance between COAGULATION & FIBRINOLYSIS"

The balance between recanalisation of venous lumen and recurrent thrombotic events → important determinant of long term outcome after an episode of DVT

## RISK FACTORS FOR VTE - VIRCHOW'S TRIAD

1. Stasis - stasis may contribute to the endothelial cellular layer coming in contact i activated platelets & procoagulant factors  
 ↳ contributing observation: satal sinuses are the m/c site for initiation of DVT
2. Hypercoagulable state
3. Venous injury

### Hypercoagulable states



### Mixed

- Homocysteinemia
- Hyperfibrinogenemia
- Factor VII, VIII, IX, XI ↑

### Stasis

- Major surgery
- Prolonged hospitalization
- Immobilisation
- Long-haul travel (>6h)
- Spinal cord injury

### Endothelial injury

- Trauma
- central venous catheter
- Surgery

### ANATOMIC FACTORS

May-Thurner Syndrome

↓  
 Compression of (L) iliac vein where it is crossed by the (R) iliac artery



### SCORING SYSTEMS TO PREDICT VTE RISK

- Rogers
- Caprini

## PATHOPHYSIOLOGY

Occlusive thrombus (Platelet aggregate  $\xrightarrow[\text{RBC}]{\text{Fibrin}}$  Thrombus)

Prepropagation (Proximal & distal)

Obstruction of venous outflow

24-36 hr (after sufficient fraction of outflow obstruction)

- Edema
- Pain

Recanalization

Collateral venous outflow

**PHLEGMASIA CERULEA DOLENS**: Extensive DVT of the major axial deep venous channels with relative sparing of collateral veins

- Pain
- Pitting edema
- Cyanotic congestion

**PHLEGMASIA ALBA DOLENS** - thrombosis extends to the collateral veins

- ↓  
massive fluid accumulation & significant edema
- extremely painful and edematous
  - pale d/t arterial insufficiency d/t dramatically elevated compartment pressures

Can progress to venous gangrene → need for amputation

## COMPLICATIONS

- 1) Pulmonary embolism - most dreaded complication  
In patients presenting w/ symptomatic DVT, 50-80% have asymptomatic PE  
In patients presenting w/ symptomatic PE, 80% have demonstrable asymptomatic DVT  
↓  
Pulmonary hypertension
- 2) POST THROMBOTIC SYNDROME → late complication in 20-50% pts w/ DVT  
↓  
consequence of VENOUS HYPERTENSION resulting from luminal obstruction & valvular reflux  
severity ↑ when iliofemoral segment is involved  
chronic edema, pain, venous claudication, varicose veins, venous ulcers

## CLINICAL FEATURES

- Requires high index of clinical suspicion - only 40% of DVT cases are clinically obvious
- Fever
- Painful edema
  - Phlegmasia cerulea dolens
  - Phlegmasia alba dolens
- Homan's sign - Calf pain on plantar dorsiflexion
- Moses sign - Calf tenderness

## EVALUATION

### ① DUPLEX ULTRASONOGRAPHY - best initial test - accurate, non invasive

- Anatomic distribution of thrombosed venous segments
- Degree of occlusion
- Collaterals
- Recanalization
- Echogenicity
- Compressibility
- Proximal extension
- used for follow-up

#### Drawbacks

- Observer dependent
- Poor info regarding iliac veins & IVC

### ② VENOGRAPHY → most accurate

- not routinely used for diagnosis
- done when intervention is planned

#### Ascending Venography

- Peripheral vein of the limb cannulated
- Tourniquet applied to prevent entry of dye into superficial system
- Contrast injection & bolus tracking
- Fluoroscopy
  - Luminal filling defect & surrounding rim of contrast - TRAM TRACK SIGN
  - Abrupt termination of intravascular contrast; meniscus formation

ASCENDING VENOGRAPHY - FOR DEEP VEIN THROMBOSIS, PERFORATOR INCOMPETENCE  
VENOUS ANEURYSMS & MALFORMATIONS

DESCENDING VENOGRAPHY - FOR EVALUATION OF VALVE REFLUX IN SUP & DEEP VEINS

MR Venography - less invasive, can directly image the thrombus

### ③ IMPEDENCE PLETHYSMOGRAPHY

- measures change in venous capacitance & rate of emptying of venous volume on temporary occlusion

- ④ **D-dimer** - marker for DVT  
 - product of fibrin proteolysis by plasmin - ↑ ilelo fibrinolysis of complexed fibrin (cross-linked degradation product)  
 In combination = clinical evaluation & assessment  
 NPV - 97-99% (negative test effectively xlo DVT)  
 sensitivity > 90-95% (but elevated foll. surgery ∴ ↑ levels not always = DVT)

- ⑤ **Iodine-125 fibrinogen uptake** - ≥ 20% ↑ in one area of a limb ⇒ DVT

MANAGEMENT - to mitigate mortality & morbidity a/c PE & PTS

ANTITHROMBOTIC THERAPY

Goal - INR 2-3

- Heparin + Vit K antagonist combo

- |              |               |
|--------------|---------------|
| UFH          | - Warfarin    |
| LMWH         | - Dicoumarol  |
| Fondaparinux | - Nicoumalone |

Min 5d

UFH - binds to antithrombin

- c- IIa, Xa
- c- Tissue factor

Antithrombin independent inhibition of thrombin via heparin cofactor II

80 IU/kg bolus IV  
 ffb 18 IU/kg/hr infusion IV

LMWH → c- Factor Xa

- Direct thrombin inhibitors

PARENTERAL

- Recombinant Hirsudin
- Argatroban
- Bivalirudin

ORAL

DABIGATRAN (antidote: idarucizumab)

- Direct Xa inhibitors

- Apixaban
- Rivaroxaban
- Edoxaban

Duration - min 3m  
 may be extended if 2nd episode, unprovoked/ cancer related

THROMBOLYSIS

Catheter directed >> Systemic

Maybe useful in EXTENSIVE PROXIMAL ILIOfEMORAL DVT

- Streptokinase
- Urokinase
- Alteplase
- Retepase
- Tenecteplase

Maybe used in the form of pharmacomechanical thrombolysis

Plasminogen

↓ thrombolytic

Plasmin

↓ Fibrin degradation

↓ Clot lysis

IVC FILTERS

Indicated in patients w/ lower extremity DVT

- absolute contraindications to anticoagulation

- severe bleeding as a complication of anticoag &

- Recurrent DVT/PE despite adequate anticoagulation

- Pt w/ severe pulmonary HTN

Complications

- bleeding at insertion site
- thrombosis
- breaking
- migration
- erosion through IVC

Removal recommended once rate is complete (~30-60d later)

VENOUS THROMBECTOMY

Reserved for acute iliofemoral DVT in

- pts who worsen w/ anticoagulation therapy

- phlegmasia cerulea dolens w/ impending venous gangrene despite fasciotomy

Operative venous thrombectomy

± ↓ intravenous angioplasty ± stenting

± AV fistula between GSV & SFA to maintain iliofemoral patency

± Pulmonary emblectomy for Pulmonary thromboembolism

# DIALYSIS ACCESS

- A good dialysis access → delivers a flow rate sufficient for effective dialysis
- is easy to cannulate
  - is durable
  - has low complication rate

## TYPES OF ACCESS COMMONLY USED FOR HEMODIALYSIS

### ① AUTOGENOUS FISTULA (AF)

ACCESS OF CHOICE - Better patency  
- lower complications

Arteriovenous fistula

↓ 6-12 weeks (maturation)

Arterialisation of vein

- Dilatation
- Wall thickening

↓  
Vein is cannulated for dialysis access

- Requires artery diameter > 2mm  
vein > 3mm
- Allen's test to determine dominant blood supply to the hand

### EXAMPLES

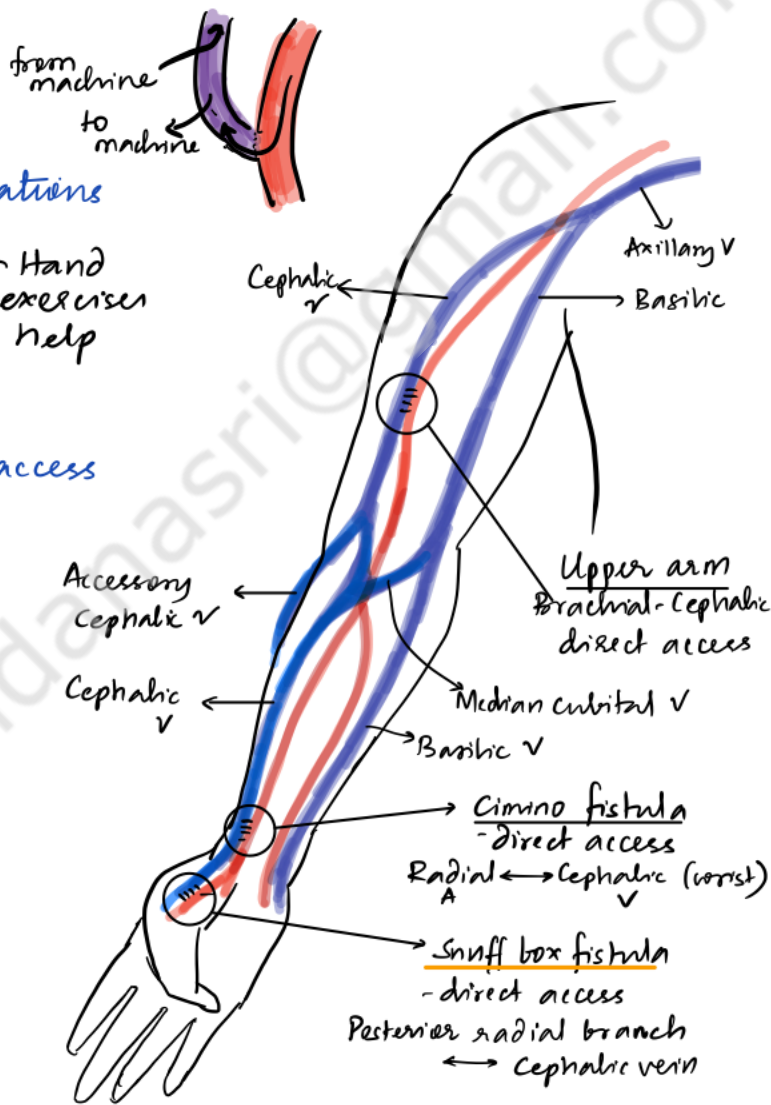
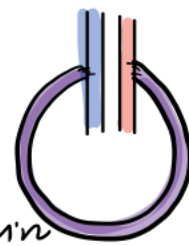
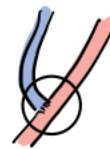
#### DIRECT- ACCESS

- 'Snuffbox' fistula - Posterior radial - Cephalic
- Cimino (Brescia Cimino Appel) fistula - Radial - Cephalic at wrist

#### TRANSPOSITION

The peripheral end of the vein is detached and anastomosed to the artery

- Eg:
- RADIAL ↔ CEPHALIC } forearm transposition
  - RADIAL ↔ BASILIC } forearm transposition
  - ULNAR ↔ BASILIC } forearm transposition
  - BRACHIAL ↔ CEPHALIC - upper arm transposition



TRANSLOCATION - access constructed from a segment of vein that has been completely mobilised, disconnected proximally & distally and placed in a location remote from its origin

## ② BRIDGING GRAFT PROSTHESIS

A PTFE/Dacron graft is used to bridge the artery and vein

- Larger surface area and ease of cannulation
- Shorter period of maturation (3-6 weeks)

- Loop / straight grafts

## ③ CENTRAL VENOUS CATHETERS

Placed in all patients requiring immediate dialysis after AF formation so that adequate maturation time can be allowed before cannulating the AF

- Cuffed CVC

- Contralateral IJV > Ipsilateral IJV > Contralateral subclavian > Ipsilateral subclavian  
Contralateral preferred over ipsilateral to limit the ipsilateral venous obstruction

## COMPLEX ACCESS

when all upper extremity access sites have been exhausted and when extensive central venous obliteration is not responsive to endovascular treatment

- JUGULAR VEIN - divided at angle of mandible  
↓  
turned down & transposed to axillary A
- AXILLARY A ↔ AXILLARY VEIN BRIDGING PROSTHETIC GRAFT
- Lower extremity - GSV → SFA/CFA transposition  
loop prosthetic graft - CFA ↔ Contralateral CFV

## Complications

- Infection → sepsis  
    - S. aureus
- Thrombosis / Occlusion  
    m/c/c - intimal hyperplasia at venous anastomosis  
    R open surgical thrombectomy ± angioplasty  
    endovascular thrombolysis
- Aneurysmal degeneration of fistula
- Arterial insufficiency / steal syndrome → compromised limb perfusion
- Rupture, hematoma, pseudoaneurysm

# VASCULAR ANOMALIES (ISSA classification)

## VASCULAR TUMORS

### 1) BENIGN

- Infantile hemangioma
- Congenital hemangioma
- Tufted hemangioma
- Spindle cell hemangioma
- Epithelioid hemangioma
- Pyogenic granuloma
- Others

### 2) LOCALLY AGGRESSIVE

- Kaposiform hemangioendothelioma
- Kaposi sarcoma

### 3) MALIGNANT

- Angiosarcoma
- Epithelioid hemangioendothelioma
- Others

## VASCULAR MALFORMATIONS

### SLOW FLOW

- Capillary malformations (port-wine stain) → flat pink patches  
Sturge Weber Syndrome
- Venous malformations (misnomer: Cavernous hemangioma) - skin/mucosal SC tissue
- Lymphatic malformations
  - Macrocystic → Cystic hygroma
  - Microcystic → Lymphangioma
- visceral lymphatic malformations = lymphangiomatosis
- Combined malformations
  - Klippel-Trenaunay Syndrome
    - ↓
    - Capillary-Lymphatico-venous malformations
  - Maffucci Syndrome

### FAST FLOW

- Arterial malformations
  - Aneurysms
  - Ectasias
- Combined malformations
  - AVM
  - AVF
  - Parkes Weber
  - CAVM
  - CLAVM

Hemangiomas - endothelial tumors, superficial - red; dermal (deep) - blue

### INFANTILE HEMANGIOMA

(CAPILLARY HEMANGIOMA)

'strawberry' hemangioma

Post natal proliferative phase (~9m)

↓  
Involution

50% → Residual lesions → Surgery

Multiple hemangiomas

Rx - Excision, sclerotherapy, Pulse dye laser

### CONGENITAL HEMANGIOMA

- Red-violaceous - coarse telangiectasias

Fully grown at birth; no post natal growth phase

Rapidly involuting (RICH)      Non involuting (NICH)



PYOGENIC GRANULOMA = Lobular capillary hemangioma

Solitary red papule - stalk

90% → Skin   
 60% Head & neck   
 20% - trunk   
 10% → Mucous membranes   
 20 - Extremities

Immature capillaries interspersed fibroblastic tissue  
Resembles granulation tissue in an edematous matrix

Bleeding → Crusting → Shrinking → Regrowth  
↑ in Pregnancy - ? Progesterone effect

Rx - excision, laser, electrocauterisation

### CIRROID ANEURYSM

- Rare AV fistula/malformation seen in the scalp (rarely, extremities)
- Congenital anomalies; can be traumatic
- 90% in relation to superficial temporal artery  
rarely - occipital artery in addition to superficial temporal A
- Scalp/facial lesion is markedly tortuous subcutaneous vessels  
- PULSATILE LESION
- rarely - bone thinning  
extending into cranial cavity

Complications - Ulceration  
Hemorrhage

Evaluation: Doppler USG, CT, CTA, X Ray

Rx - Ligation of feeding vessel → Excision  
Endovascular & percutaneous modalities

ANEURYSMAL VARIX = AV FISTULA

## ARTERIOVENOUS MALFORMATIONS

congenital abnormal vascular connections  
between arteries and veins  
without intervening capillary beds

↓

multiple large feeding vessels

- numerous arteriovenous  
communications

↓

Regrow if not completely  
excised

Locations - limbs  
Visceral  
Lung  
Liver  
Bowel  
Brain  
Spine

### PATHOPHYSIOLOGY

Structural changes -

↑ limb length & girth  
erosion of subjacent bone

thrill / murmur

Physiological changes

Hyperdynamic circulation  
CHF

Complications

Hemorrhage, thrombosis,  
embolism,  
CHF

Rx - Surgical ligation of feeding  
vessels  
Excision  
Embolisation + Excision

## ARTERIOVENOUS FISTULAS

acquired communications  
between artery &  
vein

↓

usually single communication

- secondary to trauma

↓

obliteration of the  
communication will  
suffice

Locations:

Any site of trauma  
truncal / extremities/  
visceral

### PATHOPHYSIOLOGY

Hyperdynamic circulation

1) At fistula level → dilatation  
i formation of aneurysmal  
fibrous sac - warm, pulsatile,  
soft, compressible swelling  
i continuous machinery  
murmur

2) Distal to fistula  
↓ diversion of arterial  
blood - distal part becomes  
ISCHEMIC

High pressure - arterialisiation  
of veins, development of  
varicose veins

3) PROXIMAL - BRANTHAM/NICOLADONI  
SIGN

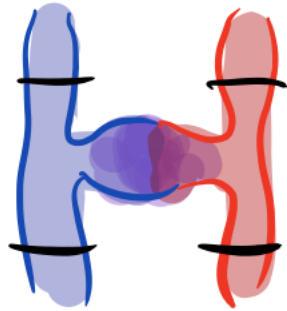
Pressure over artery proximal to  
fistula → ↓ swelling, ↓  
PR, ↓ PP

Investigations for AVM/AVF - Doppler / CT / CTA / Ech / echo

## SURGERY FOR AV FISTULA

Excision of fistula + Reconstruction of artery and vein + grafts

Emergencies - Quadruple ligation



## HUNTER'S LIGATION / LIGATION OF FEEDING ARTERY

↳ should NOT be done

- Collaterals
- Ischemia

# LYMPHATIC SYSTEM

## Embryology

- ~ 6-7 wk gestation - 6 embryological lymph sacs appear - outgrowths from venous channels (embryonic veins in the jugular & perimeronephric areas)

② Paired

② Unpaired

2 jugular sacs - at the junction of subclavian V and IJV

1 Retroperitoneal sac - at root of mesentery

2 iliac sacs - near femoral veins

1 Cisterna chyli

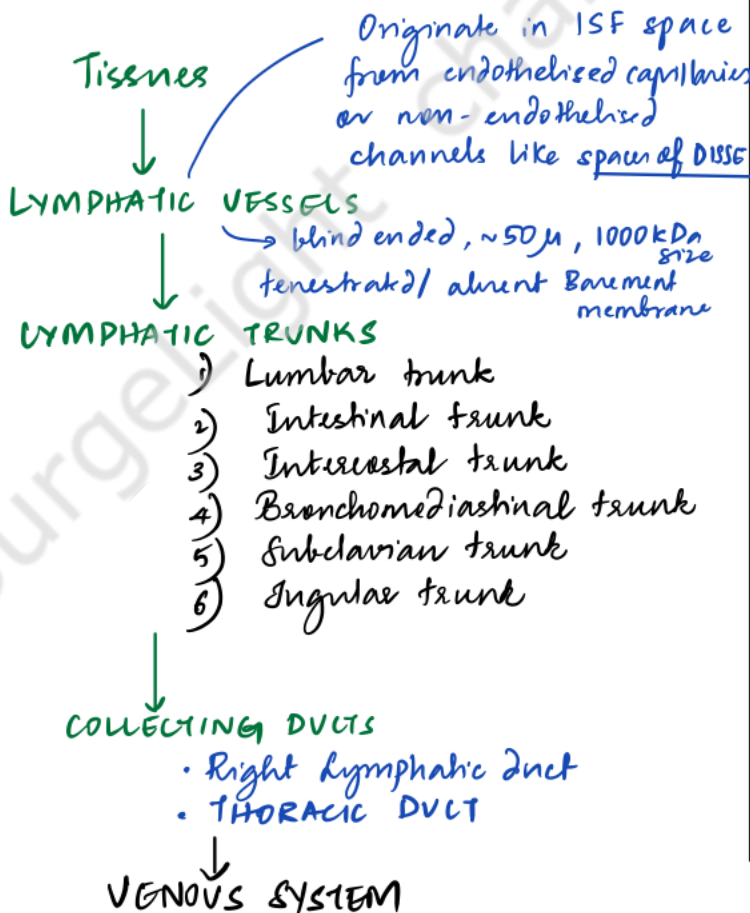
- ~ 9 weeks of gestation - multiple endothelial channels connect these sacs to form a complicated network (of lymphatic vessels)

- During early fetal development, these sacs (except cisterna chyli) are invaded by mesenchymal cells to form LYMPHNODES

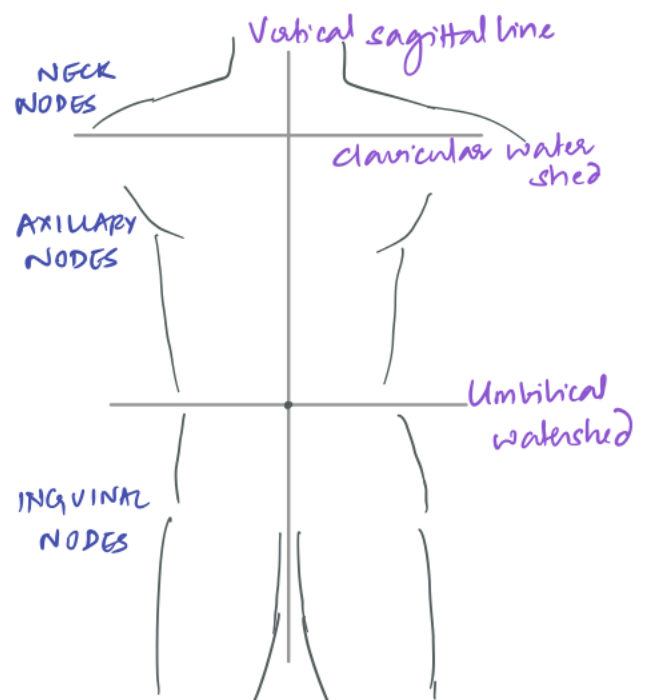
↳ Lymphocytes are derived from primitive stem cells of yolk sac mesenchyme

DEVELOPMENT REQUIRES: Prox-1 (Transcription factor)  
VEGF-C

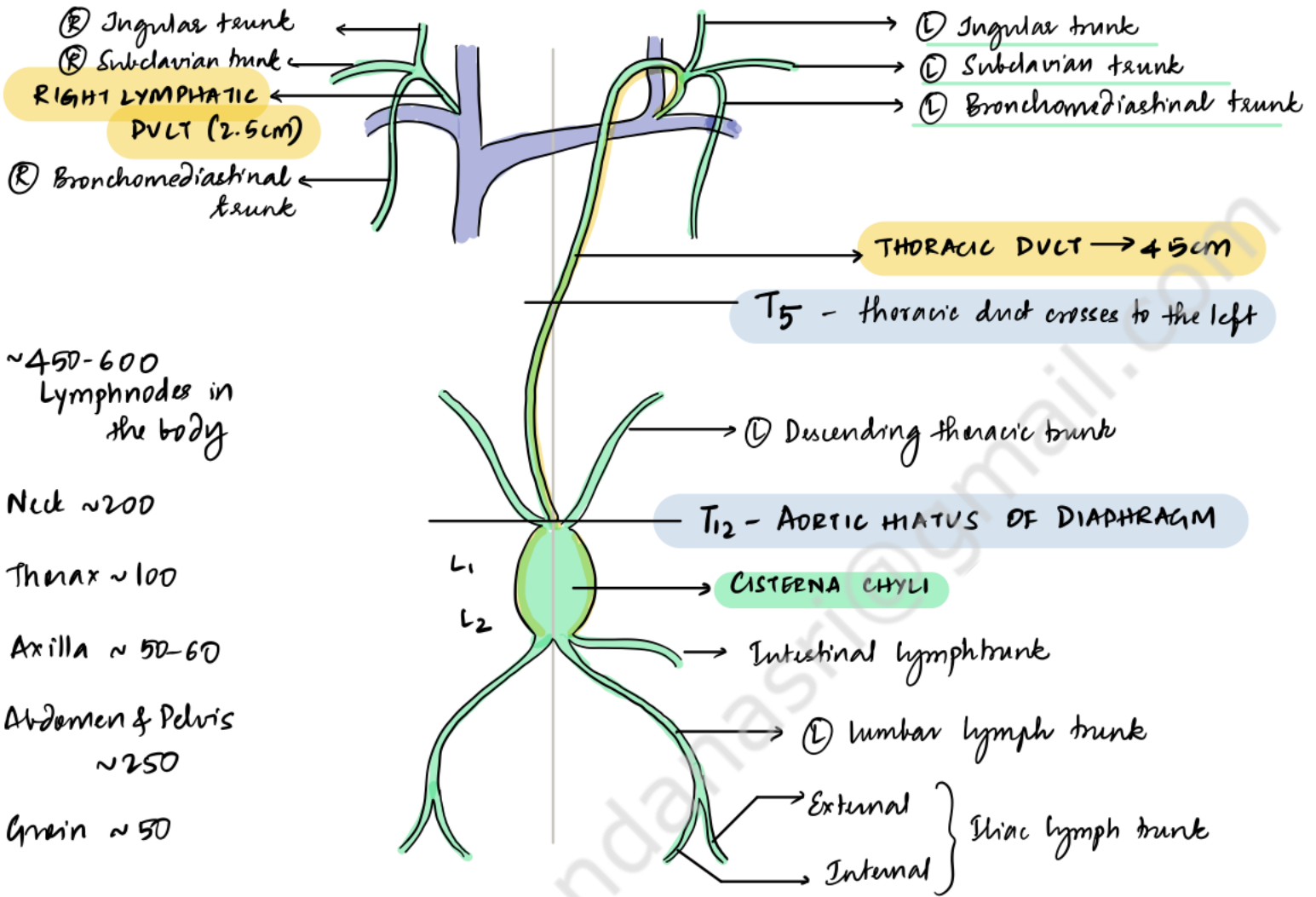
## Flow of lymph



## WATERSHED AREAS OF CUTANEOUS LYMPHATIC DRAINAGE



# ANATOMY



~450-600  
Lymphnodes in  
the body

Neck ~200

Thorax ~100

Axilla ~50-60

Abdomen & Pelvis  
~250

Genia ~50

**LYMPHOID ORGANS:** Lymphnodes  
Spleen  
Peyer's patches  
Tonsils  
Thymus

## LYMPHATICS

Lymphatic capillaries  
(Initial lymphatics)

→ Terminal capillaries → Lymph trunks

- valves  
- contractile

90%  
EPIFASCIAL ← DCEP

- majority is conducted AGAINST  
VENOUS FLOW FROM DEEP TO  
SUPERFICIAL in epifascial  
lymph trunks

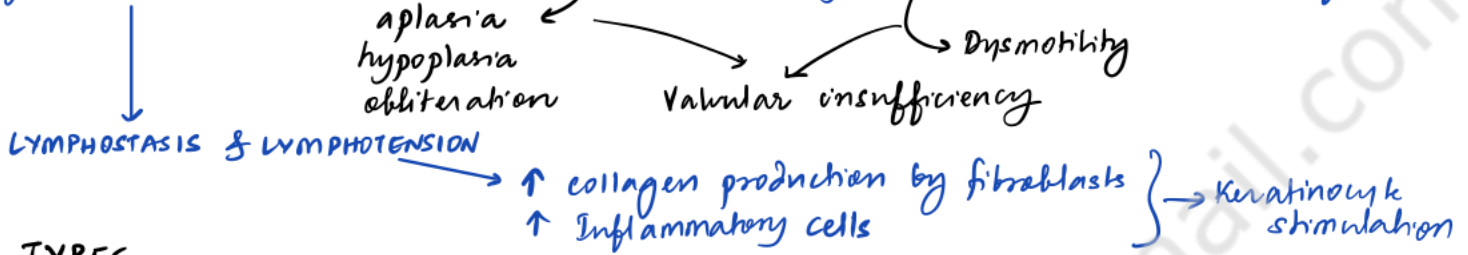
**PHYSIOLOGY** - ISF volume is determined by Starling's forces  
in health, they is NET CAPILLARY FILTRATION - removed by lymphatics

# LYMPHEDEMA

Definition: Abnormal limb swelling due to accumulation of ↑ protein IS & 2° to defective lymphatic drainage in the presence of (near) normal net capillary filtration

generally confined to **EPIDERMAL SPACE**

Lymphedema occurs due to structural &/or functional abnormalities of lymphatics



## TYPES

### PRIMARY LYMPHEDEMA

Structural defect of the lymphatics arising congenitally - uncertain/improven

TYPES - based on age of onset, family history & lymphangiographic features

< 2y

**Congenital**  
10%

NONNE-MILROY DISEASE

- M > F
- Whole leg
- U/L = B/L
- Family history +
- Progressive

AD

Hyperplasia - ↑ number of lymphatics & LNs but defective function ± Chylous ascites Chylothorax Protein losing enteropathy

Variable response to compression Rx

2-35y

**Praecox**  
(80%)

LETESSEUR MEIGIE DISEASE

- F > M
- Ankle, calf
- B/L
- Family history +
- Slow progress

Absent / ↓ distal superficial lymphatics  
↓  
APLASIA/HYPOPLASIA

**DISTAL OBLITERATION**

Good response to compression therapy

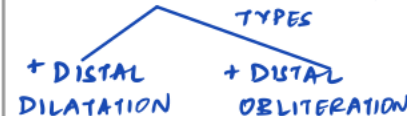
> 35y

**Tarda**  
10%

PROXIMAL OBLITERATION

- M = F
- Whole leg, thigh only
- U/L
- No family history
- Rapid progress

Obstruction at the level of INGUINAL/ARTEILIAC NODES (PROXIMAL OBLITERATION)



Benefits from lymphatic bypass &

POOR response to compression therapy

### SECONDARY LYMPHEDEMA

defective lymphatic drainage due to distinct acquired cause - **mlc**

- Progresses faster than primary lymphedema

## CAUSES

- 1) Trauma & tissue damage
  - lymphnode excision
  - Radiotherapy
  - Burns
  - Large wounds, scarring
  - Varicose veins & vein harvest
- 2) Malignant disease
  - LYMPHOMA
  - LYMPH NODE METS
  - PRESSURE/INFILTRATION
- 3) Venous disease
  - Chronic venous insufficiency
  - Post thrombotic syndrome
  - Venous ulcer / IV drug use
- 4) Infection - Cellulitis/Erysipelas, Filariasis, Lymphadenitis
- 5) Inflammation - RA, Psoriasis
- 6) Endocrine - Prethibial myxedema

# Clinical Features

Tree-trunk pattern, loss of perimalleolar shape

'Buffalo hump' feet, square toes (molded by footwear)

→ EDEMA

GRADES

Subclinical / latent

Excess interstitial fluid  
Histological abnormalities in lymphatics & lymph nodes  
BVT  
no clinically apparent lymphoedema

Reversible component

Grade I : PITS ON PRESSURE  
↓ i limb elevation & rest

Grade II : EDEMA DOES NOT PIT  
not much ↓ i limb elevation / rest  
**+ve STEMMER SIGN**

↓  
skin over the area cannot be pinched due (toes)  
to subcutaneous fibrosis

Grade III : EDEMA is associated : irreversible skin changes like : FIBROSIS  
PAPILLAE

changes

- Dermal thickening
- Hyperkeratosis

↓  
Chronic eczema  
Fungal infection  
Dermatophytosis  
Onychomycosis  
Fissuring, verrucae, papillae

Dilated dermal lymphatics (LYMPHANGIOMA)

↓  
Blister on the skin surface  
↓  
'weeping' lesions  
↓  
Fibrosis  
Hard nodules

< 5cm

lymphangioma circumscriptum

> 5cm, widespread

lymphangioma diffusum

Complications

- Chronic lymphoedema  
↓  
lymphangiosarcoma

## SEVERITY OF UNILATERAL LIMB EDEMA

- MILD : < 20% excess limb volume
- MODERATE : 20-40% excess
- SEVERE : > 40% excess

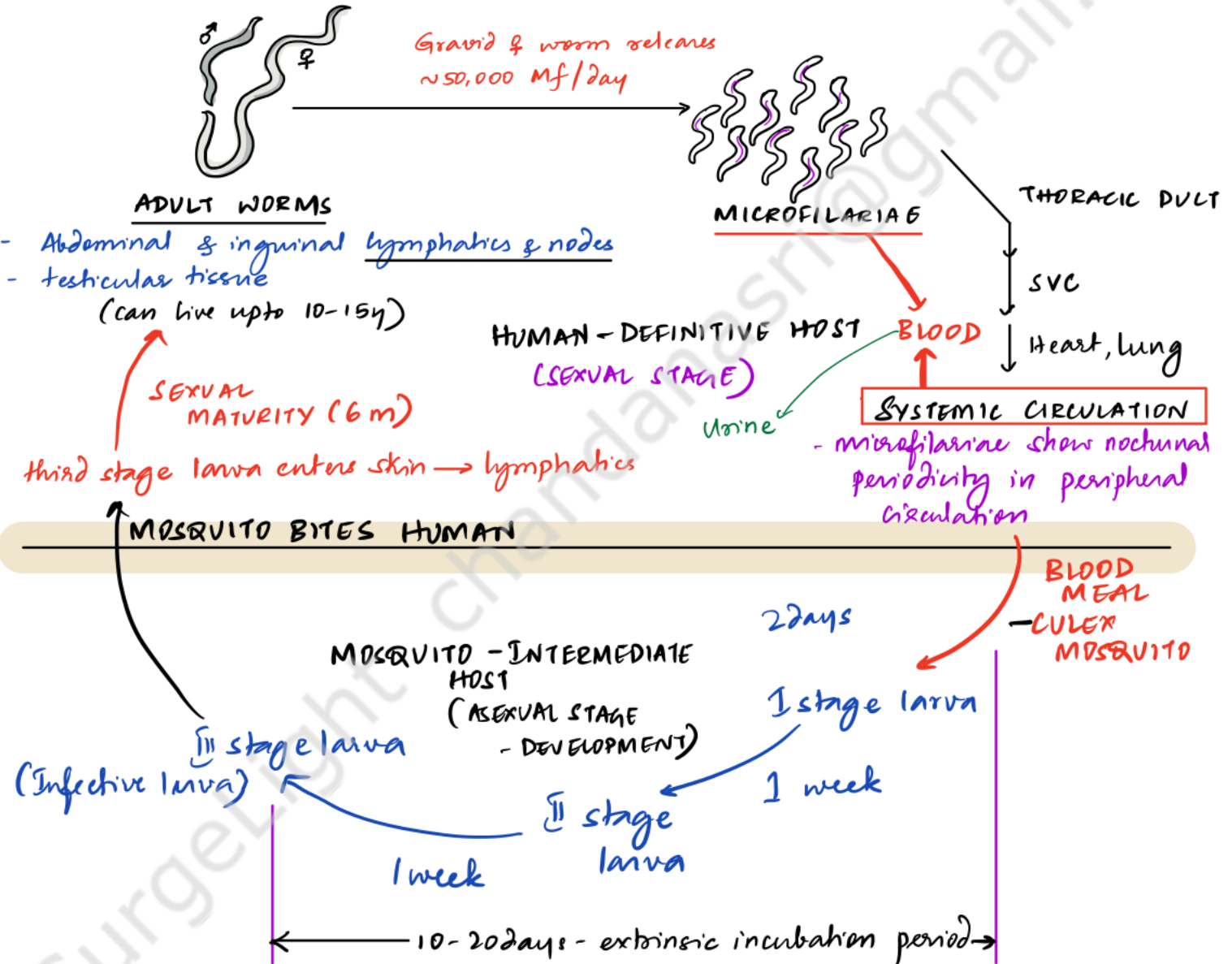
# LYMPHATIC FILARIASIS

- m/c/c of lymphedema worldwide
- Africa, India, South America

Wuchereria bancrofti - nematodic parasite - ovoviviparous

VECTOR: Culex quinquefasciatus (female): INTERMEDIATE HOST

## LIFE CYCLE



## Pathogenesis

Adult worms in lymphatics & lymphnodes → obstruction & fibrosis

due to physical load → 2<sup>nd</sup> inflammation

Progressive lymphedema



## CLINICAL FEATURES

### ACUTE

- Filarial fever
- Lymphadenitis
- Lymphangitis
- Epididymo-orchitis

### CHRONIC

LYMPHEDEMA (as above)

### HYDROCELE

Lymphatic varices

Abdominal - Chylous ascites  
Thoracic - Chylothorax  
Chyluria, Protein losing enteropathy

Massive lymphedema

'ELEPHANTIASIS'

Carrier stage: Microfilaria in blood - infective but not symptomatic

### LYMPHEDEMA

Chronic manifestations appear after repeated acute attacks over several years

LYMPHATIC OBSTRUCTION - limb edema

→ cutaneous lymphatics - skin thickening

Recurrent lymphangitis → FIBROSIS OF LYMPH CHANNELS

→ grossly swollen limb

→ thickened skin

} Elephantiasis

Secondary streptococcal infection

PULMONARY EOSINOPHILIA - dry cough, allergic manifestations

### Evaluation

- CBC - Eosinophilia
- Blood smear - microfilaria  
(sedimentation concentration)  
DEC provocation

Occult filaria - lungs - <sup>micro</sup>filaria  
blood - no microfilaria

### TREATMENT

- DEC - Diethyl Carbamazine - microfilaricidal

6mg/kg → 12 days (6 days a week × 2 weeks)

once infection resolves → treat like primary lymphedema

- Antivector measures

Other parasites causing filarial lymphedema - Brugia malayi  
Brugia timori

# EVALUATION OF LYMPHEDEMA

- Mainly a clinical diagnosis - Classical features especially in later stages

ROLE OF INVESTIGATIONS: in atypical presentations

evaluation of multifactorial edema - dx, ex, prognosis

ROUTINE : - Complete hemogram  
- RFT  
- LFT - total protein, albumin  
- TFT

FBS  
CRP  
Peripheral smear for microfilariae  
CXR, USG

(Duplex US - to diff CUE from early LE)

LIMB VOLUME MEASUREMENT - severity assessment  
expressed as % of (N) limb; Water plethysmography (Gold std), Perometry (infrared rays)

## IMAGING MODALITIES

### LYMPHANGIOGRAPHY

Patent / Isosulfan blue is injected into interdigital web space

↓  
Lymphatic absorption

↓  
Lymphatics become visible

↓  
One of the lymphatic vessels is cannulated  
= 30G cannula

↓  
CONTRAST (LIPIODAL) INJECTION  
(~7ml @ 1ml over 8min)

↓  
X-Ray after 24 hrs

Cumbersome; Gold standard

Indications - Lymphangiectasia or lymphatic fistulas & candidates for microvascular surgery

### ISOTOPE

### LYMPHOSCINTIGRAPHY

Tc- labelled sulphur colloid  
or  
protein (human albumin)

↓  
injected into interdigital web space

↓  
Taken up by lymphatics

↓  
γ-camera

3hrs

30-60min

↓  
INGUINAL NODES

↓  
Para-aortic nodes

↓  
Thoracic duct

Imaging done = whole body γ camera

Patterns - ↑ Cutaneous collaterals = obstruction of primary axial channels

Dynamic scintigraphy  
static + post-exercise imaging of lymphatic transport

### LYMPHO FLUOROSCOPY

Indocyanin green is injected intradermally

↓  
Image is obtained using an infrared camera

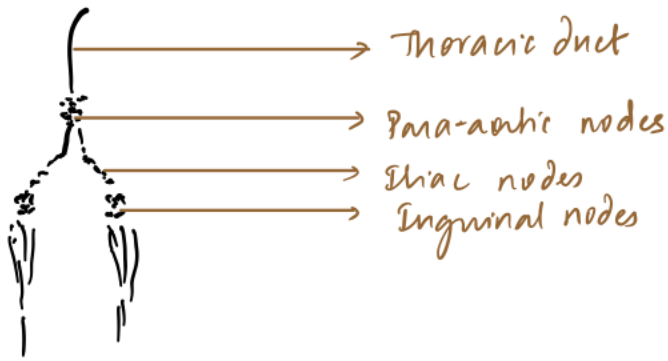
↓  
Exercise maneuvers

↓  
Mapping = infrared camera

- Can be used to screen for lymphedema in early cases

- Can be used to aid manual lymphatic drainage therapy

# (N) LYMPHANGIOGRAM



## PRIMARY LYMPHEDEMA PATTERNS

### MILROY DISEASE

Hyperplasia



### LETESIER MEIG DISEASE

Distal hypoplasia



### LYMPHEDEMA TARDA

Proximal Hypoplasia

± Distal hyperplasia

± Distal hypoplasia



## CT SCAN

- Single axial cut through mid-calf

LYMPHEDEMA

↓  
coarse, non-enhancing reticular honeycomb pattern in an enlarged subcutaneous compartment  
**EPIFASCIAL**

LIPEDEMA

↓  
↑ subcutaneous fat

VENOUS EDEMA

↓  
↑ volume of muscular compartment  
**SUBFASCIAL**

- To rule out abdominopelvic malignancies & image regional nodes

**MRI** → classical circumferential reticular pattern (honeycomb) within epifascial compartment

**FNAC of local nodes** → if malignancy is suspected in 2° lymphedema

# MANAGEMENT OF LYMPHEDEMA

GOALS: PAIN RELIEF, MINIMIZE SWELLING, PREVENT RECURRENT INFECTIONS  
PREVENTING PROGRESSION TO LATE STAGE DISEASE

NON SURGICAL - Bed rest, limb elevation, analgesics, Drugs - flavinoids, oxerubins

## SKIN CARE

R of bacterial & fungal infections

Wash regularly & moisturising product

↓  
Dry

↓  
Emollient

prevent maceration

Lymphonhea - R & i  
elevation, compression,  
cautery

## MANAGEMENT OF EDEMA

### MANUAL LYMPHATIC DRAINAGE (MLD)

- evacuation of fluid & protein from ISF by massage techniques

↓  
decongestion of lymphatic channels

↓  
promotion of collateral channels

→ Intensive, done by therapist  
SLD - Simple lymphatic drainage  
- maintenance, done by pt.

### MULTILAYER LYMPHEDEMA BANDAGING

### COMPRESSION GARMENTS

### INTERMITTENT PNEUMATIC COMPRESSION

30-60 mmHg; 4-6 hr/day using  
single/multi-chamber pumps

## EXERCISE

Rhythmic isometric movements  
(like swimming)  
coupled with massage

↓  
improves lymphatic drainage by augmentation of muscle pumps

## COMPRESSION GARMENTS IN LYMPHEDEMA

Control of lymphedema requires higher pressures

arm 30-40 mmHg      leg 40-60 mmHg

### Classes of Compression Hosiery

#### BRITISH

Class I - 14-17 mmHg  
Class II - 18-24 mmHg  
Class III - 25-35 mmHg

#### AMERICAN / INTERNATIONAL

Class I - 20-30 mmHg  
Class II - 30-40 mmHg  
Class III - 40-50 mmHg  
Class IV - 50-60 mmHg

Compression is contraindicated if ABPI is  $< 0.5$

ABPI - 0.5-0.8 - 14-21 mmHg compression may be given

ABPI  $> 0.8$ : Compression based on severity of lymphedema

**SURGICAL MANAGEMENT OF LYMPHEDEMA** - Stage II, III & severe functional impairment  
 (reserved for a small fraction of patients)

**BYPASS PROCEDURES**  
 (RECONSTRUCTIVE)

Indications

Patients with **PROXIMAL** (Primary / secondary) **OBSTRUCTION** & **PRESERVED DILATED DISTAL LYMPHATICS**

Eg: UL edema 2° to axillary lymphadenectomy

LL edema 2° to inguinal or pelvic lymphadenectomy

1° Lymphedema with proximal obstruction & distal hyperplasia

**PROCEDURE:**

Residual dilated lymphatics are anastomosed to

- nearby veins
- or
- transposed healthy lymphatic channels

- **NOODOVENOUS BYPASS** (Neulubowicz)

- **LYMPHATICOVENOUS ANASTOMOSIS**

- **LYMPHATICO-LYMPHATIC ANASTOMOSIS**

- **Omental Pedicle**

- **Skin bridge** (Gillies)

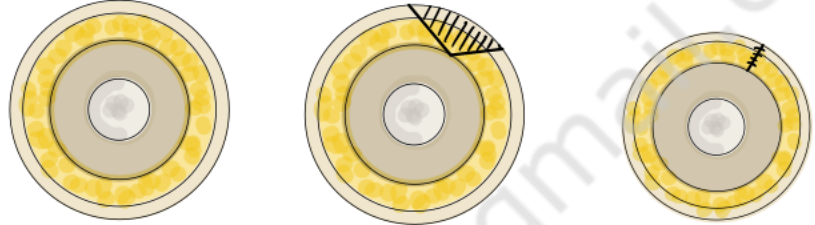
- **Ileal mucosal patch**

**LIMB REDUCTION PROCEDURES**  
 (EXCISIONAL)

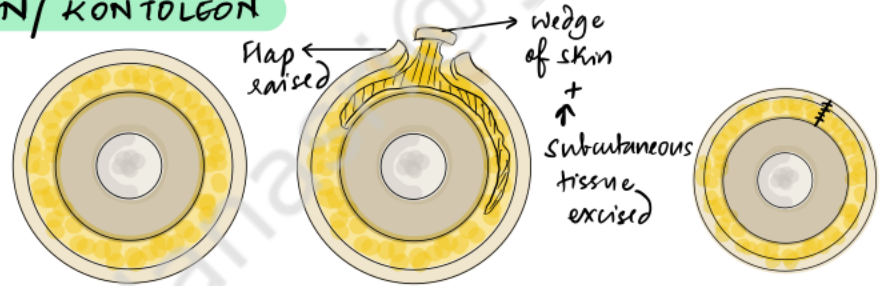
Done when swollen limb interferes with mobility & livelihood

• **Liposuction**

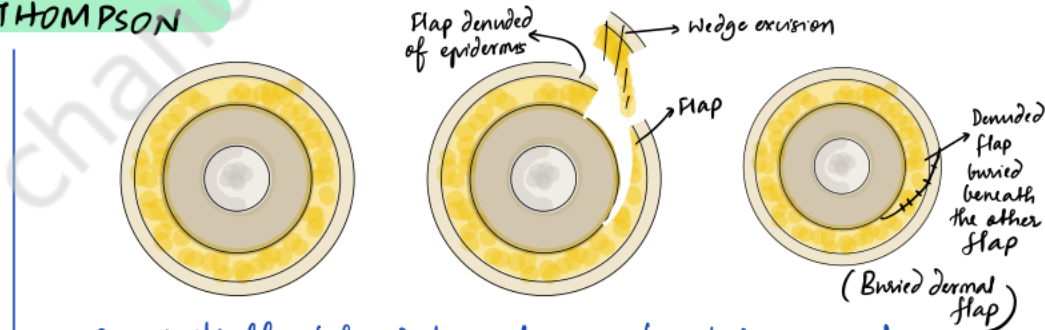
• **SISTRUNK** - excision of a wedge of skin & subcutaneous tissue & primary closure



• **HOMAN / KONTOLGON**

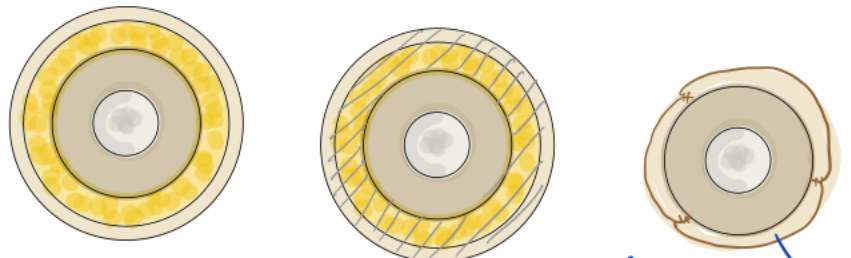


• **THOMPSON**



↳ was initially believed to create new lymphatic connections between superficial and deep systems  
 • risk of pyodermal sinus

• **CHARLES**



Circumferential excision of skin & subcutaneous tissue (down to & including deep fascia)  
 coverage with thick SSGs or FTSGs

# LYMPHANGIOMAS

CYSTIC HYGROMA syn: cavernous lymphangioma

It is a congenital condition resulting from the failure of lymphatic sacs to develop proper connections and drainage with the lymphatic system, resulting in the formation of focal lymph cysts (sequestration)

## CLINICAL FEATURES

m/c site - posterior  $\Delta$  of neck  
axilla  
mediastinum  
groin

Partially compressible - brilliantly transilluminant swelling

## PRESENTATION

Antenatal period - Polyhydramnios (fetus cannot swallow amniotic fluid)

Mass may be apparent at birth / may appear & enlarge rapidly in early weeks / months of life

→ can cause obstructed labour

Must present by 2 years of age

Neck lesion may extend into axilla / mediastinum (~10%)

CYSTS are lined by ENDOTHELIUM & filled with LYMPH

↳ unilocular < Multiple cysts - infiltrate surrounding structures distort local anatomy

Adjacent connective tissue } extensive lymphocytic infiltration  
Esp in tongue, floor of mouth, deep neck

- May contain nests of vascular tissue → poorly supported vessels  
→ bleeding → sudden enlargement discoloration

- Infection by staph / Strep

- Respiratory distress d/t airway compromise

↓  
Perinatal asphyxia → 'EXIT' procedure - Ex-utero intrapartum technique

↓  
Orotracheal intubation / Emergency tracheostomy while infant remains attached to placenta

## Management

- Image guided sclerotherapy
- Cystic excision may be done when aspiration / injection sclerotherapy is inadequate
  - CONSERVATIVE EXCISION & UNROOFING OF REMAINING CYSTS & REPEATED PARTIAL EXCISION OF RESIDUAL CYSTS & SCLEROTHERAPY

Total removal of all gross disease

- not possible due to extensive nature & intimate relationship w nerves and blood vessels
- not necessary - because they are always benign

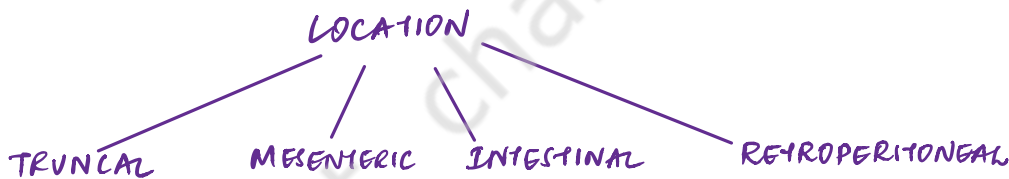
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## CAPILLARY LYMPHANGIOMAS

Failure of embryological remnants to connect to efferent channels

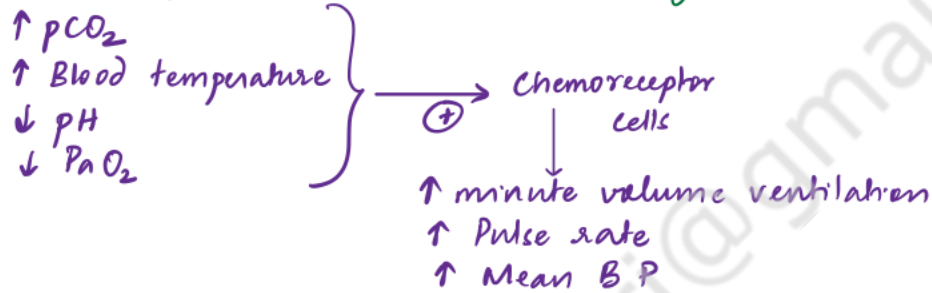
↓  
Cystic lymphatic malformations

↓  
SIMPLE CAPILLARY HEMANGIOMAS



## CAROTID BODY TUMOR

- CAROTID BODY** - originates from
- neuroectodermal-derived neural crest lineage of the third branchial arch
  - Mesoderm → stroma
- located in the adventitia/periadventitial tissue at the ECA bifurcation along the **POSTERIOR SURFACE**
- innervated by glossopharyngeal nerve (**exclusively sensory**)
- Blood supply - predominantly ECA  
- also, vertebral artery
- Largest mass of chemoreceptive tissue anywhere in the body**



**CAROTID BODY TUMORS** - belong to the paraganglioma family

**PARAGANGLIOMAS** - family of neoplastic tumors that can occur anywhere along the autonomic ganglia

∴ Neural crest ectoderm differentiates into chemoreceptor cells that migrate in close association with the autonomic ganglion cells.

### PARAGANGLIOMAS

SYMPATHETIC	PARAGANGLIOMAS	PARASYMPATHETIC
• Paravertebral thoracic sympathetic chain -	Posterior mediastinal paraganglioma	Paraganglia in middle ear along Jacobson's N / Arnold's N } Glomus tympanicum
• Retroperitoneal sympathetic nerve fibres	Retroperitoneal paraganglioma	Paraganglia in adventitia of jugular bulb } Glomus jugulare
• Organ of Zuckerkandl -	Paraganglioma of Organ of Zuckerkandl	Vagus-m/c nodose ganglion within carotid sheath } Glomus vagale
• Sympathetic nerve fibres in pelvic organs	Urinary bladder paraganglioma	Carotid body at CCA bifurcation } Carotid Body tumor
		Aortic-pulmonary paraganglia } Anterior mediastinal paraganglioma



## CAROTID BODY TUMORS

- Rare tumors
- Sporadic > Hereditary
  - ↳ 10-35% - autosomal dominant
- Tumors vs Hyperplasia
  - ↓  
a/c hypoxia - COPD, congenital heart disease, high altitude (prolonged hypoxemia)
- 5th-7th decade

### Highly vascular tumors

Synthesize neuropeptides & enzymes → detected by ITC

5-7% → malignant → risk max in young pts & family history

### SPREAD - locally invasive

- adventitia of adjacent carotid vessels - along posterior aspect of bifurcation
- widen the angle between ECA & ICA - 'Splaying' - 'Lyre' sign
- encase the main trunk & proximal tributaries of ECA (rarely involve ICA) - ? because ECA is the vascular supply to Carotid body.
- May spread to local lymph nodes → malignant

### Clinical features

- asymptomatic neck mass - near angle of mandible
- fixed mass (no longitudinal movement; mild side to side) - firm, smooth, lobulated
- transmitted carotid pulsations
- 30-40% → audible bruit
- Cranial nerve involvement - Hypoglossal N > Vagus > Laryngeal N > Symp chain
  - ↓  
Horner's Syndrome
- Large tumors - extend to base of skull
  - bulge in lateral wall of oropharynx & deviation of soft palate
- TIA - rare (usually ⊕ if there is associated carotid plaque)

# INVESTIGATIONS

- Carotid **DUPLEX** - non invasive,
- Selective Carotid angiography - Gold standard; but **INVASIVE**
  - demonstration of Carotid bifurcation
  - overall size, extent & **VASCULARITY** of tumor
  - major arterial supply of tumor
  - presence of **aberrant ascending cervical/vertebral artery branch**
  - demonstration of plaques in carotid
- MR/CT - recon gives very good picture
  - non invasive

can show presence of other concomitant cervical paragangliomas  
 → pre-op embolization if necessary

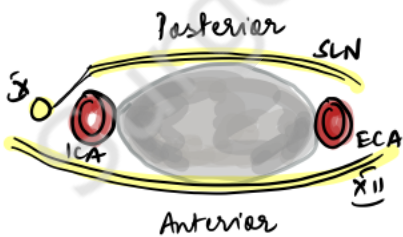
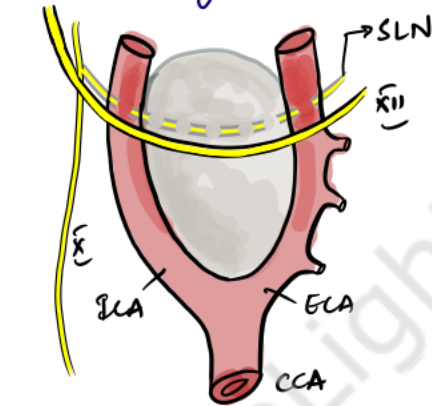
# CLASSIFICATION

## SHAMBUN CLASSIFICATION

### GROUP I

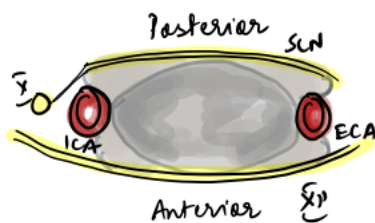
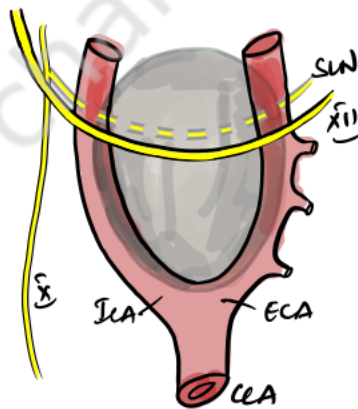
Tumors that can be freely dissected from the wall of the Carotid artery

generally < 5cm



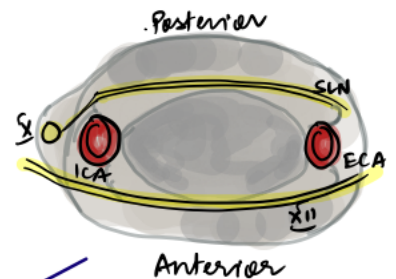
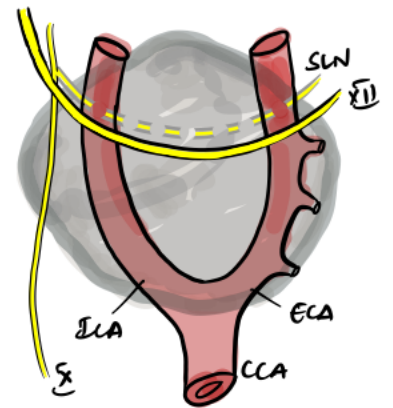
### GROUP II

Tumors that partially surround the circumference of the carotid artery



### GROUP III

Tumors intimately adherent to the entire circumference of carotid bifurcation



Generally require pre-operative embolization

## MANAGEMENT

- Surgical excision ± pre-operative embolisation of feeding vessels

Complications - Bleeding  
Cerebrovascular complications  
Cranial nerve deficits

- Radiation therapy

Adjunct

Pre-op radio  
- Bulky  
- Inoperable

Primary RT

- Recurrent tumors
- Poor surgical candidates

SurgeLight chandanasri@gmail.com