VENOUS PHYSIOLOGY

VENOUS RETURN: muscle pump, gravity, venous tone (sympathetic control), breathing
UNIDIRECTIONAL FLOW maintained by VALVES + CALF PUMP
*Retrograde flow= flow away from heart ie reflux/venous incompetence
*Calf contraction + patent veins + free outflow + valvular competence (reduce pressure in superficial veins from 90 to 30mmHg)
VENOUS HYPERTENSION Increased: reflux/prox resistance/obstruction/obesity/standing
Decreased: muscle pump/elevation/compression/losing weight

CHRONIC VENOUS INSUFFICIENCY

9% of adult population; M>F; ulcers affect 1% adult pop and 72% recurrent

Aetiology

MACROCIRCULATION: impairment of calf contraction//patent veins//freedom of outflow//valvular competence
(i)**Superficial reflux** (LSV/SSV)
(ii)**Deep reflux**: 1^0 (congenital valve absence) or 2^0 (obstruction and prox resistance/valvular incompetence)
(iii)**Perforator reflux** (usually with superficial/deep venous insufficiency; correlates with severity of CVI)
(iv)**Combination of all three**
Obstruction: (i)congenital: K-T (ii)acquired: thrombosis, post-thrombotic stenosis
Proximal resistance in raised venous pressure: compression/deep venous insufficiency/ AV fistula/tric incompetence
Valvular incompetence: (i)congenital (ii)acquired: eg post-thrombotic, post-injury

MICROCIRCULATION: venous hypertension → microcirculatory changes → ulceration
(i)**White cell trapping hypothesis**: WCs plug capillaries, adhere to VEC → release proteases → ulceration
(ii)**Fibrin cuff hypothesis**: wide pores between VECs→fibrinogen extravasates→fibrin→ischaemia→ulceration

Investigations

Hand-held Doppler//Duplex US: anatomy + patency + reflux + VVs + plan treatment//Venogram: IVC+iliacs//CT//MR
Functional assessments: ambulatory venous pressure (overall venous function, inc. calf pump)//plethysmography

Management

1 .GENERAL: Elevation/less standing/exercise/lose weight/compression
*bed rest reduces venous pressure to 12-15 mmHg → ulcers WILL heal

2. GRADUATED ELASTIC COMPRESSION:
Class I:14-17mmHg Class II:18-24mmHg Class III:25-35mmHg (pressure @ ankle, decreasing prox)
First-line for CVI: symptom relief/ulcer healing in 93%/prevent recurrence
Healing rate increases with degree of compression; trade-off is worsening compliance

3. OTHER:
Dressings
Emollients
Pentoxifylline: several RCTs comparing with + without compression demonstrate effectiveness in ulcer healing
Nutrition: Vit A/Vit C/zinc etc

4. INTERVENTION
1. Superficial venous surgery: RCT compared SVS + compression vs compression alone → lower 12mth recurrence
2. Perforators: subfascial endoscopic ligation/endovenous methods = weak evidence so benefit remains unclear
3. Deep vein reconstruction: for severe or treatment resistant cases = weak evidence so benefit remains unclear
4. Venous bypass: proven obstruction/occlusion (DVT) + severe symptoms; leave 4 yrs, post-thromb limb may resolve
**ILIACS: fem-fem crossover w/ LSV || DEEP THIGH VEINS: saphenopopliteal bypass w/ LSV*
5. Stenting: iliacs in May-Thurner syndrome → 90% 1yr patency
**M-T=pulsatile compression of L.CIVby R.CIA → luminal web/membrane → left leg DVT/venous HTN (20% adult pop)*

VARICOSE VEINS

Abnormally dilated, tortuous superficial veins

Epidemiology: (Edinburgh and Bonn population studies)
F>M but ESI overall/most adults have reticular veins (C1)/25-40% C2-6/developed countries
Deep reflux in men, superficial reflux in women
Risk factors: age/female/FHx/DVT/obesity/pregnancy/occupation

Aetiology:
Primary: valve defect
Secondary:
(i)Obstruction (thrombosis/post-thrombotic web/compression)
(ii)Raised venous pressure resists outflow (compression/deep venous insufficiency/ AV fistula/tric incompetence)
(iii)Valvular reflux: (i)congenital (ii)acquired: post-thrombotic etc
*pregnancy: enlarged uterus compresses iliacs/ P4 relaxes vascular smooth muscle

Pathophysiology
Retrograde flow= flow away from heart ie reflux/venous incompetence
No valves in IVC, first is in EIV so if deficient, column of blood from RA to SFJ
If deep veins blocked, superficial veins may be sole drainage of limb
2 theories: **Ascending** (Trendelenburg)= incompetence starts at SFJ/SPJ
Descending = progressively proximal venous incompetence

VENOUS HYPERTENSION
Increased by reflux/obstruction/obesity/standing
Decreased by: muscle pump/elevation/less standing/losing weight/compression

Clinical features
VARICOSITIES & RETICULAR VEINS
ANKLE SWELLING: usually ankle, worse at day's end; impaired lymphatic drainage due to venous HTN
SKIN CHANGES: gaiter area
PAIN: dull; worse on standing for long periods; night cramps

CEAP classification
C1: RETICULAR VEINS: thread veins (<1mm)//reticular veins (1-3mm)
C2: VARICOSITIES (dilated, tortuous superficial veins >3mm)
C3: OEDEMA (ankle, worse at day's end; impaired lymphatic drainage due to venous HTN)
C4: SKIN CHANGES (gaiter area)
C4a: PIGMENTATION (haemosiderin deposition from RBC extravasation)
C4b LIPODERMATOSCLEROSIS (i)skin/subcut fibrosis (ii)inflamm/induration (fibrin)→gutters +fixed flexion deformity
OTHER: eczema, atrophie blanche, corona phlebectatica (malleolar flare)
ULCERATION: Full thickness skin defect >4wks; prolonged healing time (consider arterial disease)
C5: HEALED ULCER
C6: ACTIVE ULCER

E: P=primary S=secondary C=congenital N=no cuase
A: s=superficial d=deep p=perforators n=no venous incompetence
P: r=reflux o=obstruction r,o=both n=no venous patholophysiology

Complications

Haemorrhage: dilated vein under pressure → trauma → profuse bleeding (elevate, compress, patient recumbent)
Phlebitis: vein becomes hard and tender w/ overlying skin inflamed (elevate, compress, antibiotics)
Venous ulcers
Oedema
Atrophie blanche
Oedema

History

Symptoms, causes of DVI, effect on life
Correlation between size of veins and symptom severity is poor
History of DVT/thrombophilia or clotting risk factors/COCP use/prior venous intervention

Examination

Inspection:
(a)varicosities (saphena varix)/ankle swelling/skin changes (PLEU) esp gaiter area/ complications (haemorrhage/phlebitis)/cross-groin collaterals in DVI/stars, flares, blow-outs
(b)elevation/dependency tests (Trendenelburg/tourniquet tests)
Palpation:
(a)texture of skin and subcutaneous tissues for fibrosis; tenderness in LPS
(b)palpate along course of veins for fascial defects
(c)palpate SFJ and SPJ + cough test
(d)palpate varicosities: pulsatile in AVf/TReg and painful in phlebitis
Percussion:
(a)Tap test: block SFJ and tap varicosities and if thrill, they belong to this system (column of blood)
(b)Do in reverse to assess valvular incompetence
Auscultation: machinery murmur in AV fistula

Investigations

NICE: duplex US= confirm the diagnosis of VVs+ extent of truncal reflux+ plan treatment(1^0/recurrent)
Doppler: uniphasic signal w/ no sound on release= competent; biphasic w/retrograde flow on release: incompetent
Duplex US: (i)reflux/occlusion in deep and superficial systems (ii) anatomy(iii) suitability for ET ablation (3 factors)
Venogram (CT/MRI; traditional method obsolete): incomp perforators/incomp pelvic veins/visualise iliacs and IVC

Management

A. CONSERVATIVE

NICE: DO NOT OFFER COMPRESSION UNLESS UNSUITABLE FOR INTERVENTION

Pregnancy/elderly/co-morbid/patient choice/symptoms mild or unconvincingly of venous source

1. Reduce venous hypertension: elevation/less standing/exercise/lose weight/compression
2. Compression stockings (Class II):

C2-4: improve symptoms/retard progression/conceal varicosities/delineates if venous disease causing symptoms

C5: reduces re-ulceration C6: Ulcer healing 93%

*issues: compliance/exclude arterial disease first

INTERVENTION: SURGERY/ENDOVENOUS THERAPY

Indications: symptoms, skin changes/ulceration, complications

NICE: offer endothermal ablation first; if unsuitable, UFG; if unsuitable to both then offer surgery

Aim: remove/obliterate incompetent venous channel

*deep venous occlusion: don't treat as superficial veins may be sole drainage channel

*mixed superficial and deep reflux: treat superficial disease, deep veins may become competent

*counsel patient that repeated treatments may be required

*occurrence after recurrence is higher than after primary treatment

SURGERY: SFJ/SPJ LIGATION + STRIPPING + phlebectomies

Day case/GA/prophylactic Augmentin reduces wound infection

GSV: Trendelenburg (reduces bleeding)/no vein divided until SFJ identified/ligate GSV close to CFV; divide DEPV

Stripping: no consensus on best stripping method; reintervention 60% at 11yr if stripped

SSV: Prone or lateral/SFJ marking mandatory/flush ligation avoided (nerve)

Stripping not accoc'd with sural n. injury, has better outcome

Complications: Bruise $_{\text{(stripped trunk)}}$/bleeding$_{\text{(groin)}}$/DVTPE/nerve injury ($_{\text{CPN/saph <10\%/sural>10\%)}}$

Outcomes: recurrence 20-80% at 5 years; 60% re-intervention at 11 years if stripped

Recurrence: fail to ligate all perforators/DVI/AV fistula (if wrong diagnosis)/de novo varicosities

**Endovenous ablation first-line for recurrence*

EVIDENCE:

REACTIV (surgery vs compression): better QoL and symptom relief with surgery but recurrence common

ESCHAR: superficial venous surgery reduces risk of recurrent ulceration

ENDOVENOUS THERMAL ABLATION

Mechanism: thermal damage to intima → obliteration of incompetent venous channel

EVLA: laser fibre causes diffuse thermal damage to intima ||RFA: RFITT (pullback) and VNUS (segmental)

Tumescent anaesthesia: (dilute LA w/ adrenaline) less pain, less bleeding, heat buffer

Suitability: (i) straight veins (ii) >1cm deep after tumescent anaesthesia (iii) >3mm diameter ie C2

Pros: avoid GA/office-based/less morbidity/less recurrence|| Cons: expensive equipment/learning curve

Complications: burns/DVTPE (bruising/bleeding/nerve injury less common)

EVIDENCE: less morbidity/better closure rates than surgery and UGFS(>90%)

ULTRASOUND-GUIDED FOAM SCLEROTHERAPY

Aim: induce chemical ablation and fibrosis in empty veins

Indication: C1/2 disease

Sclerosant: 3 types (detergent/osmolar/chemical irritant); Tessari method (foam) increases potency and volume

Cannulate w/ US/elevate limb/inject/eccentric bandages

Complications (vol. injected): thrombophlebitis/DVTPE/pigmentation/headache $_{\text{esp migraine pts}}$/CVA/

EVIDENCE: low morbidity BUT lowest closure rate + least effective long-term

INCOMPETENT PERFORATORS

Can become competent after GSV/SSV stripping; **treat if persistent/recurrent disease** (ligate/EVLA/UGFS)

VARICOSITIES

Treat at primary session/treat after trunk if necessary/don't treat at all

(i) stab phlebectomy (ii) UGFS

ATYPICAL VARICOSE VEINS

<u>VENOUS ULCERS</u>

90% of all leg ulcers

Aetiology: usually deep venous incompetence

Features: Ragged or sloping edge with pink/blue rim of advancing epithelium if healing
Scarring with white rim around ulcers (atrophie blanche)

Investigations:

US venous duplex + US arterial duplex

Other: AutoAb screen, RhF, CBG, biopsy

Management:

1. Elevate/less standing/exercise/lose weight
2. 4 layer compression bandaging (NA/wool/gradiated compression/tubular bandage)
3. Ligate incompetent perforators
4. Treat ischaemia

Other leg ulcers (10%):

Arterial

Diabetic/neuropathic

Malignant (SCC/BCC ie Marjolin's)

Traumatic

Rheumatoid

<u>POST-THROMBOTIC LIMB</u>

30% of DVTs//10% at 10 yrs → CVI w/ ulceration//50% recanalise by 90 days

Functional outflow obstruction due to - residual obstruction (fail to recanalise+ obliterated segments + synaechae)
-valvular incompetence

Cannot dilate to increase flow → venous HTN → CVI

Investigations: US Venous Duplex +/- venogram

Treatment: (a)prevent clot propagation (b)prevent PE (c)lifelong compression (d)restore venous function recon/bypass