Canadian Vascular Surgery Minimum

Unofficial 2008 Review Notes

University of Ottawa

Anton Sharapov, MD

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What follows, is a collection of notes gleaned from multitude of scraps, scribbles, summaries of texts (primarily Rutherford and Moore), and in-training written exams in Ottawa. Originally I started writing things down in preparation for my Canadian and American Vascular Boards. I did not find a unified editable collection of review notes anywhere, hence I decided to do this project.

These notes are FAR from being comprehensive. Also, being a huge fan of "Made Ridiculously Simple" series and "Whatever for Idiot's" frachise (more by necessity, rather than by choice), I may have oversimplified things a bit to make it easier to understand and memorize. So consider information critically.

This is a composite body of work spanning several years of study notes written by the Canadian Vascular surgery fellows and which were passed on to the next generation. I edited these and added a few of impressions & biases of mine own. My study partners **Wesam Abuznadah, MD**, a fellow at U of Calgary and **Hao Ming Wu,** fellow at U of British Columbia, were instrumental with several revisions of the draft. Our thanks go to the generations of scribblers and note takers, to the "Big R", as well as to our staff surgeons. We formed a google study group, and conduted almost daily conference meetings over Skype which was helpful.

This is not a substitute for reading Rutherford or doing actual oral exams. I know of several successful vascular surgeons who never did read Rutherford from cover to cover but they are just plain brilliant and full of unhibited genius. So unless you are all that, read your "big R". I did.

This is a work in progress. You can't quote what's written here as the statements contained herein may all be pronounced blasphemous 5 years down the road☺ However, this is loosely based on what's expected to know on the written part of the Canadian Vascular Board. Feel free to email me at antonsharapov@yahoo.com with constructive ctiticism etc.

To navigate, press CTRL and click on the item in the Content.

Contents

PREOP:

1. Clinical risk indexes:

- Goldman Cardiac risk index
- Modified Lee
- Detsky CRI
- Eagle CRI
- American Heart Association guidelines:

- Variables considered:
  - age, recent MI, CHF, ECG abn, aortic stenosis, emergency OR, poor general health, intra-abdo/thoraci, aortic surgery.

- Objective measures:

  - ECG – if abnormal, 3 fold increase in periop complications, if normal – not predictive….

  - Exercise ECG - less applicable in vascular surgery: suboptimal/submaximal effort due to disability, high false-negative.

  - Holter – good predictor if abnormal. 10% can't interpret meaningfully, hence false positives: due to ECG changes not due to CAD.

  - Stress-Thallium: under conditions of near maximal coronary flow (dipyridamole/adenosine induced), heterogenous perfusion areas are identified.

    - Reasonable test to order if pt is deemed intermediate risk by clinical assessment. Good alternative – dobutamine ECHO.

NUCLEAR MEDICINE TESTS:

- Perfusion studies:
  - Myoview – test of myocardial perfusion only,
  - PET – more sophisticated form of myoview:
    - tells where perfusion is happening
    - distinguishes scar vs viable vs necrotic myocardium

- Blood pool study:
  - MUGA – myocardial perfusion AND ventricular function.

- Provides the most accurate assessment of LVF

- Stress:
  - Add persantine, dobutamine, adenosine.

2. *How does Persantine scan work?*

- Baseline thallium nucleotide scan is done
- Dipyridamole is administered
- Dipyridamole dilates coronary circulation
- As a result, flow to NON-stenotic vessels is increased
- Stenotic vessel distribution shows delayed early uptake
- On repeat imaging, thallium gets into the remaining myocardium (interpreted as redistribution or delayed uptake) OR does not (interpreted as scar)
- Aside: dipyridamole (or persantine) is a phosphodiesterase inhibitor – will increase cAMP, decrease Ca++, and decrease platelet aggregation…

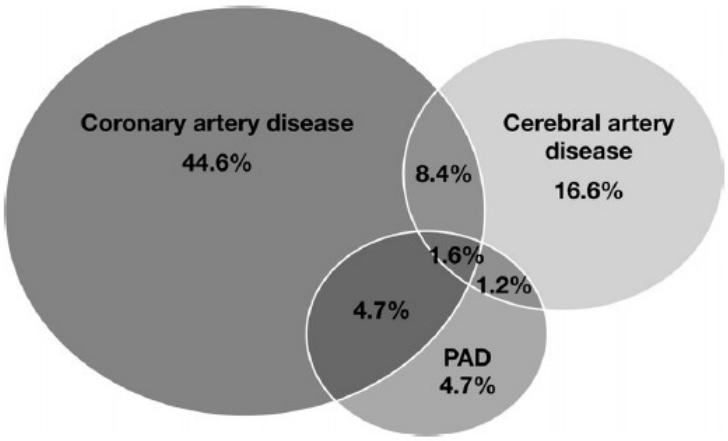


Fig. A7. Typical overlap in vascular disease affecting different territories.[26] Based on REACH data. PAD – peripheral arterial disease.

TASC II

50% of pts with PVD have CAD
25% of pts with PVD have Carotid disease

10% of pts with Carotid disease have PVD
30% with Carotid disease have CAD

20% of pts with CAD have Carotid disease
10% of pts with CAD have PVD

3. *Role of CAD screening preop?*

- role undefined…
  - CAD is prevalent, but rates of MI are fairly low
    - Aorta 2%, CEA 1%, infrainguinal 4% (Rutherford)
  - Cardiac screening detects primarly HD sig stenosis
    - Acute coronary syndrome does not occur with most HD significant stenosis Most authors state risk stratification is imprecise at best
  - Long term benefits of CAD revascularization (if it is performed) may not apply for pts with PVD…
- There are no validated invasive or non-invasive methods to ID plaques that are vulnerable to disruption
- Hence preop optimization should aim at plaque stabilization
- It is agreed there is role for BB and statins preop
- Well done negative provocative test have high NPV
  - Positive test, however, does not have high PPV

4. *CARP study:*

- Coronary Artery Revascularization Prophylaxis (CARP) trial
- Hypothesis:
  - among stable patients with CAD that is amenable to CABG or PCI, coronary artery revascularization prior to elective surgery improves long-term survival.
- multicenter, randomized, controlled, cooperative trial

- 18 Veterans Affairs Medical Centers.
- Patients scheduled for aortic and infrainguinal vascular operation eligible
  - Screen-> angio->randomized to revasc vs no revasc
  - Screened 5800, randomized 500
  - Result:
    - revascularization procedure delays/prevents the vascular operation
    - does not improve either short- or long-term survival.

5. *Courage study:*

- Predictive value of POSITIVE preoperative pharmacologic nuclear perfusion scanning is POOR (5-20%)
  - NPV is excellent (98-100%)
- 2006, Study setting:
- Patients with
  - chronic angina pectoris,
  - stable post-myocardial infarction (MI) patients
  - asymptomatic patients with objective evidence of myocardial ischemia

        - i.e. positive non-invasive tesing/scanning
    - 2287 pts randomized to either aggressive med therapy vs PCI plus therapy
    - At 5 years, same number of pts is angina free
    - No difference noted.
    - Role of PCI preop should be questioned…

6. *POISE study:*

- Multiple SMALL studies showed beneficial effect of Beta blockers on CV mortality in perioperative setting (e.g.DECREASE trial)
- This was tested in a 7000 pt RCT in pts undergoing non-cardiac surgery
- Metoprolol vs placebo
- Overall there was reduction in MI but there was increase in stroke and overall mortality in Metoprolol group
- for every 1000 patients treated, metoprolol would prevent 15 MIs but there would be an excess of eight deaths and five severe disabling strokes
- effects attributed to high dose BB that compound perioperative shock
- recommendation is to use lower dose, start BB early to allow accommodation of dose, and avoid extended release preparation.
- http://cme.medscape.com/viewarticle/574526

7. *Respiratory assessment and fitness for thoracotomy:*

- PFT: if FEV1 > 60% or DLCO > 60% - will tolerate up to pneumonectomy
- FEV1 and DLCO < 60%
    - Quantitative lung scan
        - If predicted post op FEV1 and DLCO > 40%
            - May procede to surgery
        - If PPV FEV1 and DLCO < 40%
            - Perform xercise testing:
                - If VO2 max > 20 ml/kg/min
                    - Procede to surgery
                        - 15-20 ml – 66% risk of complicatios
                        - > 20 ml – 10% risk of complications
                - If VO2 max < 15 ml/kg/min
                    - Beware of 100% risk of complications

8. *Eagle criteria:*

- Age > 70
- Diabetes
- Angina > class 3
- CHF
- Prev MI

- ECG changes
- Aortic stenosis
- Ventricular arrhythmia

9. Modified Lee criteria

Predicts risk of major cardiac complications:

- DM (insulin dependent)
- CVA/TIA
- Angina/MI/ previous CABG/PTA
- CHF
- Renal insufficiency > 180 (> 2)
- High risk procedure
  - Abdo, thoracic, aortic, visceral
- cardiac risk index has been validated in number of studies
  - risk of major vasc complications is equal to the number of RF squared

Number of risk factor	Riks of major cardiac complications
0	0.5%
1	1.3%
2	4%
3	9%

10. What are METS?

1MET

 Can you take care of yourself?
 Eat, dress, or use the toilet?
 Walk indoors around the house?
 Walk a block or two on level ground at 2 to 3 mph or 3.2 to 4.8 km per h?

4METs

 Do light work around the house like dusting or washing dishes?
 Climb a flight of stairs or walk up a hill?
 Walk on level ground at 4 mph or 6.4 km per h?
 Run a short distance?
 Do heavy work around the house like scrubbing floors or lifting or moving heavy furniture?
 Participate in moderate recreational activities like golf, bowling, dancing, doubles tennis, or throwing a baseball or football?

>10METs

 Participate in strenuous sports like swimming, singles tennis, football, basketball, or skiing?

11. Arterial MRI studies:

- 2 D TOF
- 3 D TOF
- 3 D phase contrast
- 3D TOF gadolinium phase contrast – BEST
- "spin echo technique" – black blood technique, for large a. visualization
- "gradient echo sequence" – bright blood, for smaller a. (renal, peripheral)

HEMODYNAMICS AND DOPPLER:

12. Hemodynamic principles:

- Stenosis causes local disturbances in laminar flow
  - In fluid, in the stenotic segment velocity GOES UP
  - Upon leaving stenotic segement velocity GOES DOWN
- This interaction sets off a series of multidirectinal velocity vector forces
- These vectors are perpetuated along the course of the tube due to inertia (fluid has mass)
  - The more mass fluid has - the more inertia it has
- This leads to turbulence and energy losses
  - Commonly picked up as pressure drop/velocity drop off
- With time, these tubrulant vectors (driven by inertia) are redirected by the viscosity forces into a laminar flow.
- It is an interaction of the disrupted FLOW in the stenotic area (interplay between inertia and viscocity of the fluid) and a given RESISTANCE of a vessel (defined by radius and length) that will determine how much energy (pressure) will be lost during this interaction.
- Reflected in Omh's law:
  - Change in the direction and absolute velocity will result in energy losses
  - Sparks (energy) fly when you scrape(resistance) the sidewalk at 100 km/h (flow)
  - Omh: energy (Pressure change)=Resistance*flow
- Resistance **$R = 8L\eta/\pi r^4$**
  - this represents minimum resistance in the circuit,
  - determined by the length, radius of the vessel and the viscocity (**η)** of the fluid.
- The energy drop for a given flow will increase if kinetic contribution is added – i.e. if acceleration and/or deceleration is seen in the flow.

A hemodynamic version of the Omhs' law:
Poiseuille's P_i-P_{ii}= Q * R= Q* 8Lη/πr^4

- **P_i-P_{ii}** – pressure or energy change
- Q - flow
- η – coefficient of viscocity
- Pressure gradient across the stenosis is increased:
  - the longer the lesion,
  - the higher the density & viscosity,
    - density or mass determines kinetic component as well, **1/2ρv_i ^{2}**
  - the greater the diameter reduction
    - **Most important**

- Where can the energy in the flow be lost?
  - Viscocity loss – see Poiseulle equation – the longer, the narrower the stenosis - the greater the loss
  - Inertial loss – kinetic energy **1/2ρv_i ^{2}**
    - **This is the most significant way to lose energy in the circulation**
    - In stenotic area, flow increases!
      - See high velocity jet on doppler
    - **velocity change is seen at the entrance (goes up) and the exit of the stenotic segment (goes down).**
    - **In both circumstaces, energy is dissipated…**
    - **So if you have 2 stenosis 2 cm each, energy loss will be MORE than a single 4 cm stenosis.**

Total vascular resistance:

- Collaterals:
  - Compensate for the occlusion of main conduit (iliac/fem/tib)
  - Pre-existing vessels, some are formed in response to hypoxia
  - Zones:
    - Stem
    - Midzone
    - Re-entry vessels
- Segmental resistance (iliac/femoral OR collaterals) + outflow resistance
- Segmental resistance increase with iliac/SFA disease
  - Even multiple collaterals can never bring down total resistance to normal once iliac/femorals are occluded…
- Outflow: combined resistance of arterioles, capillaries, venules, and veins
- With arterial disease, segmental resistance is increased (occlusive disease), and outflow resistance is reduced as a compensation (dilation at rest)
  - Hence:
    - Diseased limbs always have higher segmental resistance

- Even with exercise relative drop in outflow resistance is SMALLER in diseased limbs compared to normal
  - Because in these patient outflow is maximally dilated at rest

Bernoulli's principle:

- **Hemodynamic version of the law of conservation of energy.**
- **The fluid's energy is determined by**
  - **the existing bank account (Pi – initial pressure),**
  - **its' density & speed (i.e. kinetic energy)**
  - **and elevation above ground (gravity).**
- **Without friction and with continous flow, the energy content remains constant.**

$\mathbf{P_i + \rho g h_i + 1/2 \rho v_i^2 = P_{ii} + \rho g h_{ii} + 1/2\ \rho v_{ii}^2}$ ρ – density of blood

- Relationship between kinetic, gravitational energy and pressure
  - in a frictionless system
- Under steady flow, frictionless systemn - energy remains the same.

- **Reynolds number: very important number**
  - Dimensionless quantity.
  - Represents an interplay between inertial and viscous forces.
  - When number is < 2000, local disturbances are dampened by viscous forces and flow is laminar.

  - **$R_e = \rho v d / \eta$**
    - i.e. Laminar flow (<2000) is favoured in
      - viscous (high η)/low density (low ρ) fluids
      - slower flow (low velocity V)
      - smaller diameter of the conduit.

So, if the velocity is increased (as in stenotic iliac lesion during exercise), the Re can rise and eventually cross 2000 mark – turning flow into TURBULENT. This translates into energy losses and pressure drop past stenosis. Outflow resistance reduction (due to maximum vasodilation) is designed to counteract this but can't fully compensate for the energy loss at the stenotic segment.

- Minimum length of tube needed to turn laminar flow into turbulent OR to re-establish laminar flow is defined as an entrance length:

  - **Entrance length: $L_x = K * r * R_e$ K=0.16**
- the length of the tube needed to change a turbulent flow into laminar is smaller if:
  - the radius of the tube is small

- the fluid is more viscous
- the flow is slow

- **Laplace: Wall tension = P*r**
- Larger tubes have larger wall tension.
- Larger aneurysms are more prone to rupture

- Doppler: **frequency shift $\Delta f = 2 F_o * \cos \theta / c$**
  - F_o carrier frequency
  - C – speed of sound 1540 m/sec
  - Θ – angle of incidence
  - Why factor 2 is in equation?
    - Some say it accounts for two directions of sound travel – to the moving RBC and back to the receiver
    - I think it may be simplier than that:
    - Cos is not linear, error inreases with greater angle:
      - Cos 0 - 1
      - Cos 30 – 0.86
      - Cos 45 – 0.70
      - Cos 60 – 0.5
      - Cos 90 – 0
  - So if you keep angle at 60, cos of 0 is 0.5, hence multiplying it by 2 will yield error factor of 1 i.e. no error…

- 2 degree angle error
  - If confuse 58 degrees with 60 - PSV assessment is 6% off
  - If confuse 78 degrees with 80 – PSV assessment is 17% off

13. Non-invasive testing overview:

- Establish clinical indication first
- Use to plan angiography or survey grafts/known stenosis
- Start with resting ABI and PVR.
  - If these are abnormal may order targeted arterial duplex to see waveforms and velocities
  - If ABI are normal but clinically suspect claudication
    - Order stress testing/hyperemia

14. Doppler wave forms:

- Pulse contour –

- normal – tri or bi phasic
- Frist wave –
  - prograde energy from the cardiac output,
  - 2nd wave – reflection from the peripheral resistance,
  - 3rd wave – reflection is overcome by the last kick of the prograde effect of systolic flow.
- Velocity increases rapidly in early systole, reaches peak, then drops off, reversing in early diastole.
  - In late diastole, velocity tracing becomes positive before returning to the zero-flow baseline.
  - With increasing vasoconstriction, the reverse-flow component becomes exaggerated.
    - Same seen with microemboli…
  - With decreased resistance, reversed phase may disappear – wave becomes biphasic
    - seen normally in renal, celiac, fed state SMA and cerebral circulation

- Abnormal pattern– with proximal stenosis
  - Blunt slow upstroke 1st phase
  - drops 2rd phase
  - then 3nd stage disappears
  - Peak pulsatility index goes down…

- Monophasic waveform:
  - blunted with slow upstroke and prolonged hump
  - tracing more continuous and less pulsatile.

- Spectral analysis – summarizes and describes available range of vector velocities in the vessel.
  - The more the turbulence, - the more direction and velocity change and the broader the spectrum.
  - Look for the disappearance of the clear window under the tracing

- Presence of reverse flow component is highly predictive of intact inflow
- Absence of reverse flow may be related to other factors:
  - Low resistance, hyperemia, vasodilators
  - monophasic wave due to severe inflow disease.

- Exaggerated 2nd component – increased outflow resistance (microembolism)

- Hence the following means are available to analyze waveforms:

- **Peak to Peak pulsatility index**

- PTP frequency difference of the Doppler waveform divided by the mean frequency
  - i.e. peak of systolic first wave + peak of the reflected (2nd) wave in early diastoli of the same cycle
  - NOT peak to peak of two different cycles
- In normal limbs, PTP increases from proximal to distal
  - Because MEAN pressure drops in periphery
- Provides good description of proximal inflow lesion:
  - i.e. first wave is blunted/decelerated and reversed wave is reduced
- With proximal stenosis, this increase from proximal to distal is not seen
- E.g.
  - Normal: CFA 13 -> distal 18
  - Aortoiliac occlusion 2->4
  - SFA occlusion 6->4
  - both 3->3

- Pressure Pulsatility index – if > 4 in CFA – likely rules out aortoiliac disease
  - Abnormal value – interpret with caution

- Laplace wafeform transformation
- Power frequence spectrum analysis
- Pulse transit time

These evaluate lesion indirectly… Preferred method is direct examination of the lesion with B-mode/wave form analysis.

15. *Critical stenosis – residual radius and surface area of the lumen:*

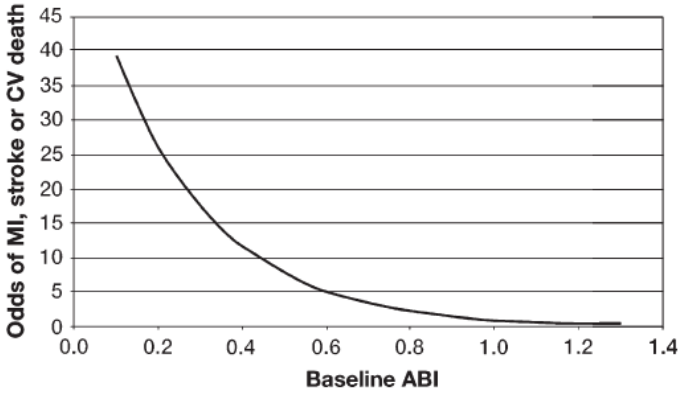
- The change in energy content of the flow is a function of radius of the vessel raised to the 4th power AND velocity of the fluid.
  - Poiseuelle's equation
- Initially, with small reduction of radius, the energy (pressure) loss is small, however pressure drops exponentially when critical radius reduction is reached
  - 50% radius or 75% surface area
  - Both due to velocity change AND radius change
- At this critical point, Pressure drops precipitously – i.e. critical stenosis is reached.

- In simple terms, narrowing at which pressure and flow begin to be affected.
- Does not happen until 50% diameter reduction is reached
  - equivalent of cross-sectional area of 75%

16. Resting ABI – advantages and disadavantages:

- Advantages of resting ABI:
  - Quick, easy, cheap
  - Excellent Prognostic survival info (Framingham study)
  - Reproducible
  - Can be used to follow and assess effect of tx
    - (min 0.15 change is clinically sig)

- Limitations of resting ABI:
  - Does not localize occlusive disease precisely
  - Doesn't measure internal iliac and profunda (i.e. non-axial arteries)
  - Doesn't detect multilevel disease
  - Doesn't reliably predict probability of thrombotic episode in graft/PTA site
  - Doesn't detect occlusion distal to ankle
    - Unless measure Toe Brachial Index
  - artificial elevation in calcified vessels
    - Renal Failure and DM – i.e. false negative is possible
  - can't use in large wounds
  - Sources of error:
    - Cuff size (need to be at least 50% larger than limb diameter)
    - Large collaterals will elevate ABI
    - HTN and CO variation
- ABI is an excellent predictor of CV mortality:



Additional notes on ABI:

- Pressure difference between DP and PT should be less than 10 mm Hg. > 15 – suspect stenosis

- Ankle pressure > brachial pressure by 12-24 mm

  - augmented systolic pressure but diastolic is less

  - mean pressure is same
- how to ID ABI in calcified diabetic foot:
  - elevate foot
  - note disappearance of distal Doppler signal
  - multiply distance in cm by 0.735
- ABIs
  - Don't measure non-axial pressure
  - Do not distinguish vasospasm vs stenosis
  - Do not pick up non-flow limiting lesions in trauma
    - In trauma, > 0.9 – no need to angio

Additional notes on PVR:

- Reflect total volume of the limb
- Ideally measure upper, lower thing an calf
  - Normal - Calf amplitude exceeds thigh amplitude by 25%
    - If not – suspect SFA occlusion
  - Inaccurate for aortoiliac disease
    - Unless use 2 thigh cuffs
  - Accurate for femoral lesions EVEN with aorto-iliac disease
- upper thigh pressure
  - by cuff- always exceed brachial by 30-40 cm

        - Thigh Brachial index 1.3-1.4
            - However, direct (invasive intraarterial) pressure is same as brachial
        - If cuff pressure is same or less – suspect significant aortoiliac stenosis

- Pressure gradient between levels > 30 mm Hg – suggest obstruction
    - Upper thigh pressure (profunda AND SFA) vs lower thigh pressure
        - Should be same
        - If different by 15 mm – SFA disease
        - Horizontal difference > 20 mm is significant
- Multilevel disease is difficult to distinguish on PVR

17. Stress testing and ABI:

Exercise increases cardiac output and flow through the aorta/iliac system. If the velocity is increased (as seen in stenotic iliac lesion during exercise), the Raynold's number can rise and eventually cross 2000 mark – turning flow into TURBULENT. This translates into energy losses and drop in pressures past stenosis. Outflow resistance reduction (due to maximum vasodilation) is designed to counteract this. However it can't fully compensate for all the energy loss at the stenotic segment.

Advantages:

- Uncovers lesions that are asymptomatic at rest
    - particularly in the iliac system
- Allows to establish functional significance of the lesion
- Allows combined assessment of walking ability and restriction imposed by orthopedic, neurologic and cardiolpulmonary disease

Principle:

- Normal individuals do not drop ABI after 5 min.
- Magnitude of drop reflects degree of obstruction

Technique:

- Supine for 20 min, baseline ABI
- 2 mph at 10% incline walk for 5 min OR until claudication/restriction
- Supine position – remeasure ABI q 2 min until pre-exercise value achieved OR 20 min elapsed.

- Other facts on Exercise testing:

- Do not use in CLI, cardiac cripples
- Post exercise – re-measure brachial pressure – it usually rises, so need to establish new baseline.
- The more proximal the obstruction, the greater the effect of exercise on ankle pressure
- Ankle pressure < 60 mm post exercise – test is positive
- Reactive hyperemia:
    - Substitute for exercise
    - 3-7 min suprasystolic pressure on thigh
    - Monitor ankle pressure at 15 sec then 30 sec interval
        - Normal response – initially drops to 80% but comes back to 90% within 30-60 sec.
        - Toe pressures
            - Assessment of functional severity
            - Shows degree of maximal dilation of peripheral bed
            - reappear almost immediately, but with 2 fold increase in amplitude.
            - Abnormal – toe pressure does not come back for > 120 sec
- Direct pressure and Papaverin – 30 mg intraarterial – > 20 mm pressure drop significant

- Surgical sympathectomy and hyperemia responce:
    - If posthyperemia response – twice prehyperemia – may benefit from sympathectomy
        - Measures an ability to dilate in response to a release in vascular tone
        - Not a test of integrity of sympathetic system

- To check function:
    - PVR will decrease with deep breath if sympathetic is intact

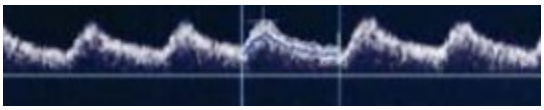
18. Extremity arterial duplex and stenosis:

- No stenosis: psv <150, ratio , 1.5
- 30-50% : psv 150-200, ratio 1.5-2
- 50-75%: psv 200-400, ratio 2-4
- >75%: psv >400, ratio >4
- Monophasic non-continuous staccato/thump proximal to stenosis (no diastolic)
- Monphasic continuous flow distal to stenosis

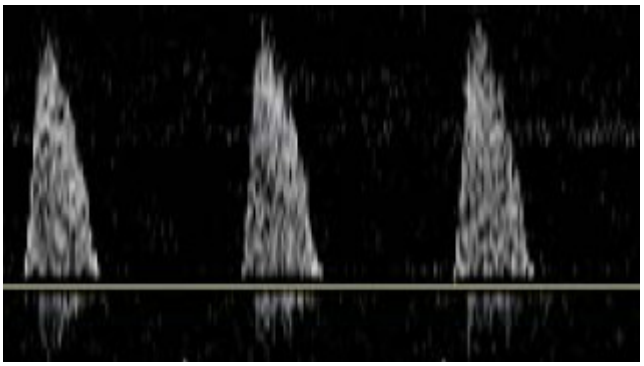
Artery	Normal Diameter in B mode	Normal PSV
EIA	8 mm	120 cm/sec
CFA	8 mm	115 cm/sec
Proximal SFA	6 mm	90 cm/sec
Distal SFA	5.5 mm	92 cm/sec
popliteal	5 mm	70 cm/sec

Aortoiliac and peripheral waveform in stenosis

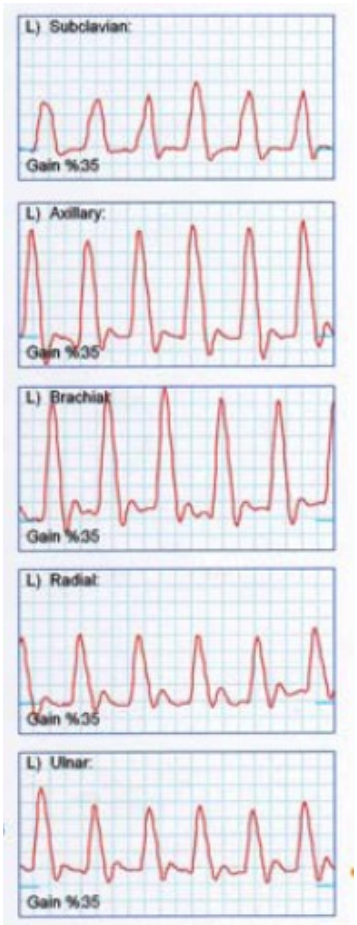
- 100% PSV step up (velocity ratio 2) compared to normal segment proximally is significant
- Best accuracy – ratio 2.5-3
- Color doppler without velocity waveform analysis is poor at quantifying stenosis



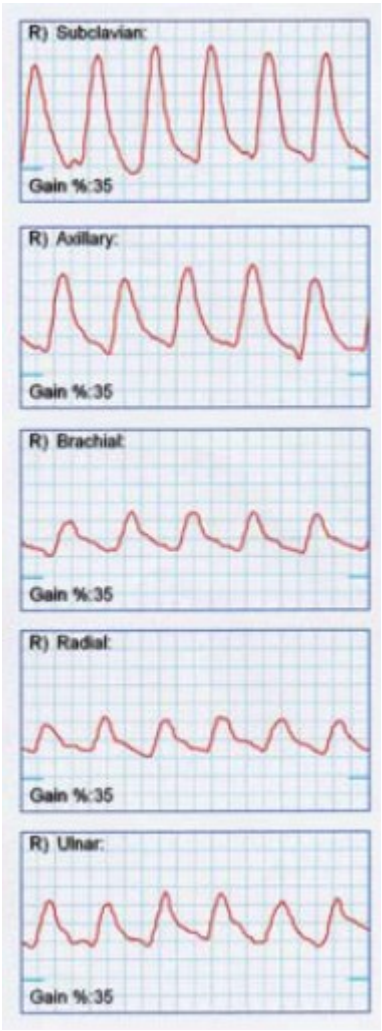
Inflow problem (low amplitude, reduced upstroke)



Outflow stenosis (no distal flow, no diastolic flow)



Normal upper extremity waveform



R subclavian stenosis

19. Venous graft follow up:

- <45 cm/sec, no diastolic flow – predictors of early failure
    - <45 cm/sec but normal flow may be seen in large diameter vein
- Surveylance of grafts improves primary assisted patency by 20%
- Intervene if PSV>300, ratio > 3.4

20. Why does steal after fem-fem or ax-bifem occur rarely?

- Because usually the inflow is NOT limited…
    - With ample inflow, both receiving beds receive adequate blood supply
    - There is increased (double) flow in donor artery if outflow is increased
    - The flow is distributed to each extremity
        - according to the resistance in each
        - which is more or less equal under normal resting circumstances
- Vascular steal arises when 2 run off beds with different resistance are supplied by a **limited source of inflow**
    - Competition arrises
    - If inflow is limited, the run off bed with less resistance will take the flow preferentially over the other bed rendering it clinically ischemic
    - With presence of PVD, the overall resistance of diseased limbs is high
        - high segmental resistance, maximally dilated outflow bed resistance that can't drop its resistace more with excercise
    - Hence initially ischemic limb may be rendered even more ischemic

21. Carotid ultrasound – Washington, NASCET and ECST criteria

- Washington criteria – based on ECST, NOT NASCET angiographic correlation
    - ECST – outlines hypothetical normal carotid bulb and measures stenosis wrt this
    - NASCET – compare distal ICA to stenosis
        - May get negative stenosis figures
        - Compared to ECST, predicts less severe stenosis

- Angiogram – underestimates stenosis
- MRA probably equivalent to duplex US
- High sensitivity study
    - Able to recognize an abnormality
    - needed for symptomatic pts
- High specificity study
    - abile to recognize normal artery
    - needed for asymptomatic pts

Washington Criteria

- Remember that it OVERESTIMATES the stenosis (ECST, not NASCET criteria)
- Remember, that it gives ranges that do NOT apply for ANY study – i.e. NASCET (70% stenosis) and ACAS (60% stenosis)

- Normal: no plaque, smooth walls, boundary layer separation in bulb
- < 15 % mild SB
- 16-49% marked SB, no systolic window
- 50-79% PSV >125, PDV <140 cm/sec, marked SB, ICA/CCA > 1.8
- 80-99% PSV >125, PDV > 140 cm/sec, poststenotic turbulence, ICA/CCA > 3.7
- Occlusion: no flow
    - May be wrong in 3% of cases – hence ALWAYS confirm this with angio or MRI.

In measuring carotid velocity:

- keep gain low – may artificially cause Spectral Broadening
- keep sample volume low (1.5 mm)
- notice post-stenotic turbulence and color flow mosaic

22. Normal Carotid and Vertebral flow velocities:

- CCA
    - No defined abnormal PSV

- Normal flow most of the time <100 cm/sec
    - If Decreased. First compare R and L
        - If bilateral – aortic stenosis or myocardial failure
        - If unilateral
            - CCA origin stenosis – all velocities past CCA are dampened
    - If elevated
        - Hyperdynamic state if bilateral
        - r/o contralateral CCA/ICA occlusion
            - compensatory flow
- flows increase from arch to CCA bifurcation
    - 9cm/sec per cm
    - Measure CCA PSV for ration at a DEFINED location
        - Usually 4 cm below bifurcation
        - Width of a trasducer

- ICA – normal velocity 54-88 cm/sec
- ECA – normal velocity 77-115 cm/sec
    - No criteria for stenosis
    - Suspect stenosis if >125 cm/sec and post-stenotic turbulence
    - See notch with temporal tap
- ICA and ECA should LOOK and SOUND different
    - If not, suspect ICA occlusion and confusion of branch of ECA for ICA…
- Normal vertebral flow
    - <60 cm/sec

- Increased flow seen in
  - Dominant vertebral (MC on the L)
  - Near occlusion of ICA

23. Consensus panel on US criteria on stenosis:

- These are more practical then Washington – based on Nascet technique and range of measurements of stenosis:
- <50%
  - PSV<125
  - EDV<40
  - Ratio <2
- 50-69
  - PSV 125-230
  - Visualized plaque
  - EDV 40-100
  - Ratio 2-4
- >70
  - PSV > 230
  - Visible plaque
  - EDV > 100
  - Ratio >4

Aside notes on Abnormal velocities:

False elevation in ICA PSV :

- Contralateral occlusion
- Hyperdynamic state
- Bad angle
- Post stent or endarterectomy

PSV in carotid stent:

- >150 cm/sec is NOT >50% stenosis
- Psv>300 and EDV > 140 – predicts high grade stenosis but need angio to confirm

Carotid occlusion

- Waterhammer waveform (sharp up and below zero line downstroke, no diastolic flow
- Acute thrombus
- No flow in ICA
- In imaging:
  - Use power Doppler
  - If pulsed:
    - Keep PFR low to detect low flows
    - Increase Doppler gain to ID slow velocity

Innominate stenosis:

- Decreased CCA wave
- Reversal of flow in ICA and CCA
  - Crouching bunny waveform
- Carotid steal can only happen on R side
  - See reduced PSV in ICA, reversal of flow in diastoli

Long severe stenosis in ICA will have reduced PSVs, not eleavated...

- Hence always rely on ratio ICA/CCA in these cases.
- Make sure CCA is not elevated (i.e. >100 – due to c/l occlusion

24. Other useful velocities measurement for carotids

- >60% stenosis – external Oregon validation with angio
  - PSV >260, EDV >70, ratio > 3.2.
  - Accuracy 90%
- NASCET > 70% stenosis
  - PSV >280, EDV > 80, Ratio >4
  - PPV 95%
- >80% stenosis
  - PSV >250, ratio >4
  - 90% accuracy for 70-99% range
- Intraop duplex assessment of CEA
  - Repair if PSV>200
- Subclavian artery stenosis
  - Retrograde (notched) vertebral flow
- No graded PSVs values vs occlusion for vertebral artery flows
- Vertebral steal:
  - See either reversal of flow or stalled flow
    - Pre-steal - Back (systoly) and forth (diastoly)
    - Don't confuse with phasic flow in vertebral vein

25. Frequency of Surveylence of known stenosis

- Asymptomatic > 60 stenosis%
  - If PSV<175 cm/sec
    - Progression is 4% over 21 months
    - Image annually
  - If PSV > 175%
    - Progression 26% over 14 months
    - Hence image q 6 month

26. Transcranial Doppler

- Color flow, B-mode, pulsed Doppler
- Indications
  - Evaluate cerebral vasospasm (post SAH)
  - Bubble study (for patent foramen ovale)
  - Screening children with sickle cell disease (I don't know what that means)
  - ? inraop monitoring and assessement for shunt need
  - ?early diagnosis of hyperperfusion syndrome
    - Need to know MCA flow preop
    - If see 2 fold increase in mean velocity – have your diagnosis
    - Not cost effective to justify routine use
  - There is NO indication for TCD in routine carotid US
- How to do:
  - 2 MHz

- Transtemporal window and suboccipital window
- ID MCA, ACA, P1, P2
- Get mean (not angled corrected) velocity – assume 0 degrees correction
  - N=30-80
  - Vasospasm > 120

- For RPVI exam, you **have to know** what arteries are sampled in which window and the direction of flow in these areteries – away or toward the transducer
  - Like, you are going to need to use this in real life…

27. Renal artery US

- Do clinical profiling first
  - Athero - >50yoa, RF, HTN
  - FMD – young female with HTN
- Overnight fast
- Longitudinal view of supraceliac artery first
  - Convert to transvers
  - ID L renal vein
  - ID origin of both renal aa.
  - Image:
    - Orifice
    - Proximal
    - Mid
    - Distal
    - Interlobar and arcuate flows:

        - Upper pole
        - Mid pole
        - Lower pole
    - Measure kidney size
        - Compare size, > 1.5 cm difference significant
        - < 9 cm pole to pole suggest atrophy
- Indirect measurements of flow: i.e. measure random artery in parenchyma
    - NOT prospectively validated
    - Resistive index: (1-EDV/PSV)
    - If >5% b/l difference, diagnose 50% stenosis
        - Not work if bilateral disease
    - RI <0.8
        - 96% sensitive
        - 53% specific
        - To predict improvement in renal failure post stenosis repair
    - RI > 0.75 AND EDV <90
        - May get reduction of 5+/-5 mm Hg pressure
        - Likely NO clinical benefit
    - TRANSPLANTED kidney
        - RI > 0.8
            - PPV 88%, NPV 93% to predict death, need for dialysis and Cr clearance deterioration
- Normal flow – low resistance, PSV <180 cm/sec
- Stenosis

- RAR > 3.5 - 60% stenosis
  - 84% sensitivity
  - 97% specificity
  - PPV 94%
  - Prospectively validated
- PSV >200 cm/sec - 60%
  - But with RAR is MORE predictive – i.e. can diagnose 60 % stenosis if RAR>3.5 regardless of PSV value

- Dupplex of renal artery – CAN'T see accessory arteries
- Renal artery aneurysm:
  - Associtated with
    - Vasculitis
    - FMD
    - Dissection
  - macroaneurysm

28. Mesenteric duplex

- Screen for CMI
- 6 h fast
  - Fasting: Low flow, high resistance
  - Fed: high flow low resistance
- 2-4 MHz, subxiphoid and R lateral (liver window)
- SMA
  - PSV 275 cm/sec – 70-100% stenosis
  - EDV 55 cm/sec - > 50% stenosis
- Celiac

- High flow, low resistance all the time
- PSV >200 cm/sec
- EDV >55 cm/sec
- Retrograde flow in CHA if celica is stenosed/occluded
- Difficult to assess flow in celic if SMA disease is present
- With median arcuate syndrome, PSV goes up with EXPIration, normalizes with inspiration.

- CHA
  - Normal PSV 60-125
  - Low resistance
- Portal vein
  - In fasting state – PSV 8-18
- Varies 70-100% in size with respiration
- Gradient with IVC 5-7 mm
- Portal hypertension
  - Size >13 cm
  - Slow flow
  - Flow away from liver (hepatofugal)
    - In N, should see same direction of flow in CHA

29. US and EVAR

- Most of the time useless – difficult to get good pictures
- Endoleak type 2
  - If <80cm/sec- likely to thrombose
  - If >100 cm/sec unlikely to thrombose

30. Ultrasound of Transplant:

- Liver
  - Hepatic artery stenosis
    - Difficult to get true angle PSV
    - Parvus tardus most common finding in stenosis
      - AT >0.03, RI is less than one
    - Thrombosis – emergent exploration (bile ducts depend on it)
  - Hepatic artery spasm:
    - High resistive flow
    - Reduced PSV, no flow in diastoly, may be reversed flow
    - RI is 1.0
- Kidney:
  - Renal a. stenosis –
    - PSV >200, renal – iliac ratio > 1.8
    - On intrarenal spot testing – tardus parvus – AT >0.1
  - Elevated RI
    - Renal VEIN thrombosis
    - Fluid collection
    - Obstruction
    - Rejection
    - ATN
    - Drug nephrotoxicity
  - Intrarenal AVF
    - Post biopsy

- Rare cause of ischemia, may cause htn

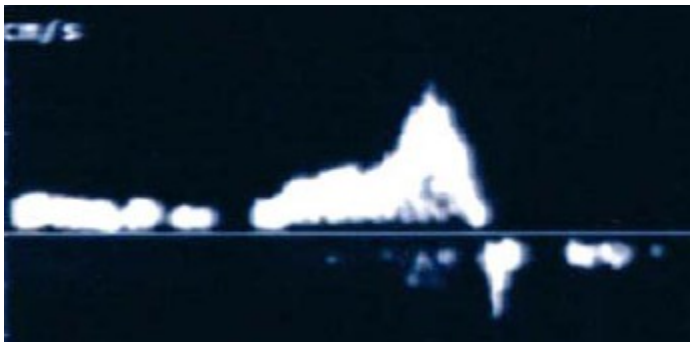
31. Ultrasound and chronic venous insufficiency

- Duplex assessment
  - r/o DVT
  - assess outflow
    - augmentation studies
  - deep and superficial flows, perforators
  - reflux: lying and standing plus cuff
  - r/o AVM

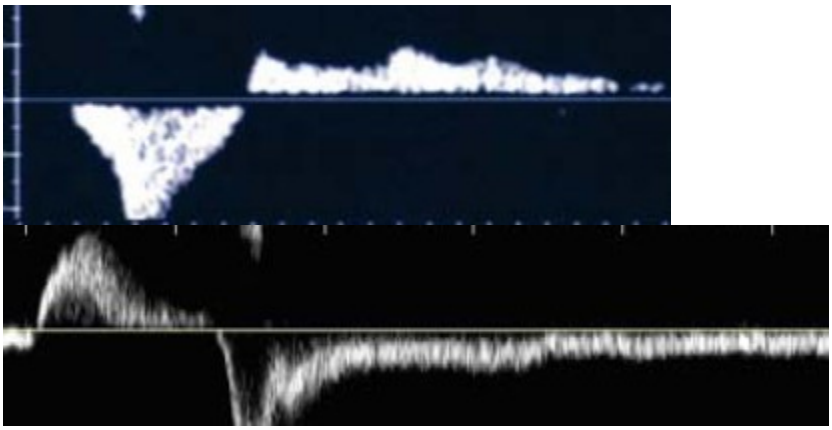
- AVP
  - Ambulatory venous pressure
  - Venous pressure measured directly in dorsal vein after 10 dip-toes (1/sec)
  - Calf contractions increase outflow
- Strain gauge Plethysmography
  - Used for DVT
  - Not anymore
    - Assess baseline value
    - Measure increase in volume after calf contractions
    - N 2-3% change above
    - If outflow pathology – see < 2% volume reduction
- Impedance plethysmography
  - For DVT
  - Not used anymore

32. Characteristics of venous flow:

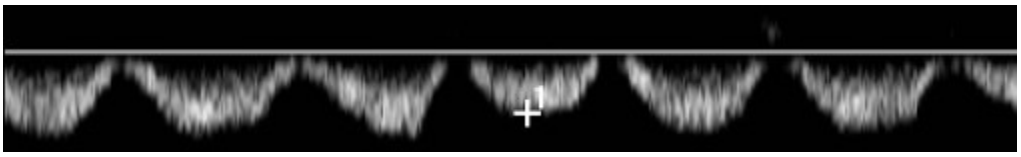
- Phasic
    - If continuous – suspect proximal obstruction OR collaterals from prior DVT
    - If pulsatile – suspect RHF, fluid overload, AVF
        - In upper extremity, it is NORMAL to expect to see some pulsatile flow because of proximity to the heart. Pulsatility is ABnormal in leg veins
- Unidirectional
- Responds to
    - Respiration
    - Valsalva



Competent valves



Incompetent vein



AVF in leg vein: pulsatile flow

Continuous flow – outflow obstruction

33. Ultrasound and Dialysis access

- Normal graft velocities
  - Psv 150-300
  - Edv 60-200
  - Marked spectral broadenings

- critical velocity in dialysis graft PSV <150 cm/sec
- normal flow:
  - >800 cc/min
  - early stenosis 500-800 cc/mn
  - severe stenosis <500 cc/min

ATHEROSCLEROSIS & RISK FACTORS' TX:

34. Physiologic role of endothelium :

- Normal endothelium function:
  - Modulator of coagulation system
    - Heparin, tPA, thrombomodulin
  - Permeability barrier for nutrients&fluids
    - Lipids, glucose, water
  - Permeability barrier to cells
    - Regulates inflammatory response
    - Normally Nonadherent surface for ppt/neutrophils
      - Regulated by ICAM, VCAM receptor expression
    - Inhibit SMC proliferation within intima
      - TGFb – transforming GF
  - angiogenesis modulation
    - VGEF to grow new vessels
  - Vascular tone modulation
    - NO & prostacyclin vs endothelin & angiotensin

- Endothelial products:
  - EDRF (NO) & Prostocyclin - dilating
  - Angiotensin and endothelein – constricting
  - Tpa, heparin, thrombomodulin

- What damages endothelium
  - Mechanical & ***low*** Sheer stress
  - Metabolic stress – excess of LDL, glucose
  - Immunologic stress - infection
  - Vasoconstrictor stress
  - smoking

35. Factors important for atherosclerotic plaque development:

- Shear stress
- Flow separation and stasis
- Turbulence and Oscillation of shear stress vectors
- Hypertension and Heart rate
  - These contribute to turbulence and sheer
    - low HR - less atherosclerosis in carotids
    - high HR – less atheroscleosis in infrarenal aorta
- structural disorders – pseudoxanthoma elasticum

- vasa-vasorum obliteration & fibrosis – due to radiation

36. Role of macrophages in atherosclerosis, list macrophage secreted GF:

- Injured/altered endothelium exposes adhesion molecules that attracts monocytes
- Monocytes migrate into subendothelial space and turn into macrophages
- MPH pick up oxidized LDL – turn into foam cells
- This uptake causes MF to synthesize
    - Monocyte colony stimulating factor (M-CSF)
    - Granulocyte colony stimulating factor (G-CSF)
    - epidermal GF (EGF)
    - platelet derived GF (PDGF)
    - transforming GF alpha/beta
    - Vascular endothelial GF (VEGF)
    - monocyte chemoattractant protein 1 (MCP 1)
- more monocytes are attracted, plaque starts to remodel

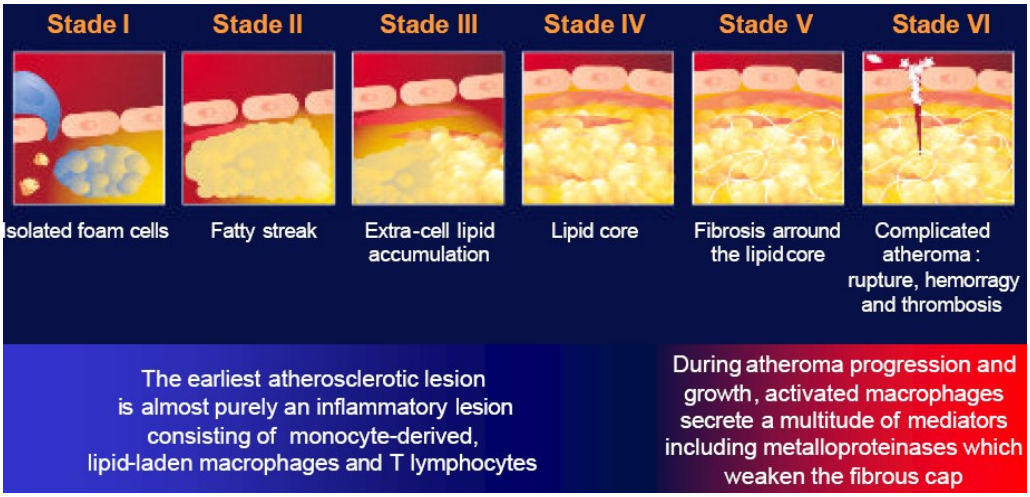
37. What happens in atherosclerosis:

- Primarily an ENDOTHELIAL disease that spills over to the media
- Endothelial injury OR alterered permeability to lipoproteins first
- more adhesive molecules formed
    - ICAM, PECAM – ppt and WBC movement into vessel wall
- cytokine production by endothelium: PDGF, FGF< TGFb, IL 1
    - local cells (SMC) and new arrivals (monocytes) are transformed and caused to proliferate/move to intima
    - monocytes form mast cells ->foam cells
- lipid accumulation in foam cells, which later spills outside to the media & around SMC
- lipid core is formed under endothelium
- collagen and connective tissue is formed ->fibrous plaque
- plaque can rupture - >
    - leading to luminal thrombosis and atheroembolism

38. Stages in atherosclerosis and types of plaques:

1. Isolated Foam cells
    a. transformed monocytes->macrophages with lipid
2. fatty streak (collection of foam cells)
3. fat accumulation outside of foam cells
    a. lipid droplets BETWEEN SMC distorting their arrangement in media

4. formed lipid core in INTIMA
5. tough fibrous cap
6. ruptured cap/complex atheroma



39. Name different mediators secreted by endothelium:

- Procoagulant – plasmingen activator inhibitor (PAI), von-Willebrant Factor
- Anticoagulant – heparin, thrombomodulin, tPA
- Vasodilator – prostacyclin, NO
  - Prostocyclin also increases cAMP (reduces plt aggregation)
- Vasospasm mediator – angiotensin 2, endothelin

40. Endothelial progenitor cells:

- Found in circulation
- Released by BM in response to ischemia and trauma
- Capable of endothelial repair, serete tPA
- Decreased in pt with cardiovascular disease and smokers
- Present in both young and old

41. Mechanism of action of Nitric oxide, or Endothelial Derived Relaxing Factor:

- Smooth muscle relaxant
- Inhibits ppt and WBC aggregation

42. Effects of smoking:

- Histologically, smoking damages endothelial cell:
  - swelling,

- bleb formation,
- subendothelial edema,
- thickening of basement membrane,
- widening of endotethelial junctions.

- increases viscocity and decreases oxygen transport:
  - carboxyHG due to CO which leads to increase in hematocrit
  - aggregation of WBC and ptt
  - fibrinogen content in blood increases

These events lead to:

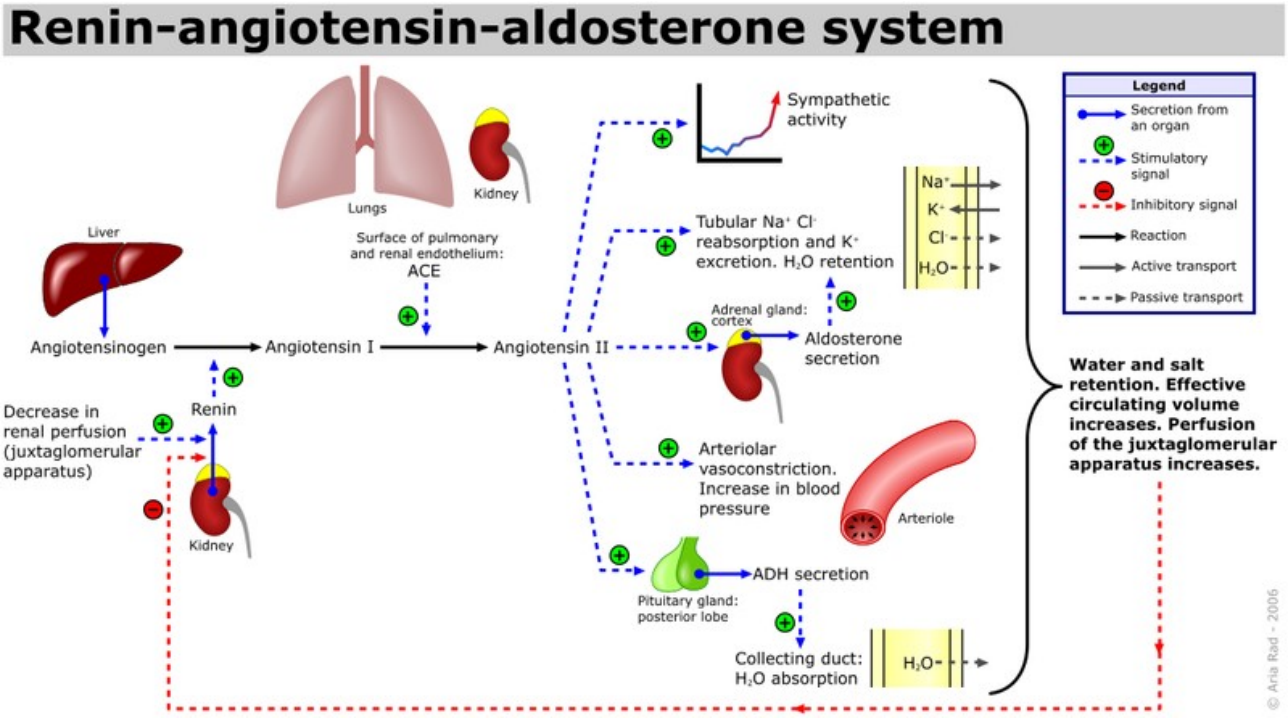
- Increased thrombogenicity
  - Due to increased viscosity due to increased hematocrit
  - Due to decreased fibrinolytic capability/increased fibrinogen
  - Due to direct endothelial injury and vasospasm
    - due to decreased NO production
    - due to increased platelet aggregation
- Altered Lipid metabolism – decreases HDL, uptake of LDL increases
- Reduced oxygen delivery:
  - Due to increased carbon monoxide

Summary:

- Affects ALL functions of endothelium
- Increases thrombogenicity
  - Increases viscocity of blood
  - Increases fibrinogen
- Decreases oxygen delivery (carbon monoxide effect on Hg)
- Affects lipid metabolism
  - LDL up, HDL down

43. Mechanism of action of Angiotensin II:

- Vasoconstrictor
- Induces inflammatory cytokine IL 6
- Releases aldosterone
- Releases ADH
- Increases sympathetic tone
- Vessel and myocardial wall hypertrophy



44. ACEI effects:

- Mechanism:
    - inhibits conversion of AT I to AT II
    - upregulates bradykinin –
        - causes cough and vasodilation
    - inhibits aldosterone
    - inhibits ADH
    - increases LV contractility
    - remodels myocardium and vessels
    - LDL oxygenation reduction
    - numerous pleotrophic effects on endothelium (SMC inhibition, NO induction, ppt adhesion inhibition)

OVERALL EFFECTS: TRIPPED OUT antihypertensive

- Reduces risk of MI, CVA, death due to CV cause (HOPE)
- In acute MI: decreases death, progression to CHF and need for hospitalization due to CHF (AIRE)
- Compared to other anti hypertensive meds, in diabetic pts have BETTER
    - prevention of proteinuria,
    - nephropathy progression,
    - preservation of renal function,
    - control of HTN.
- Contraindications:

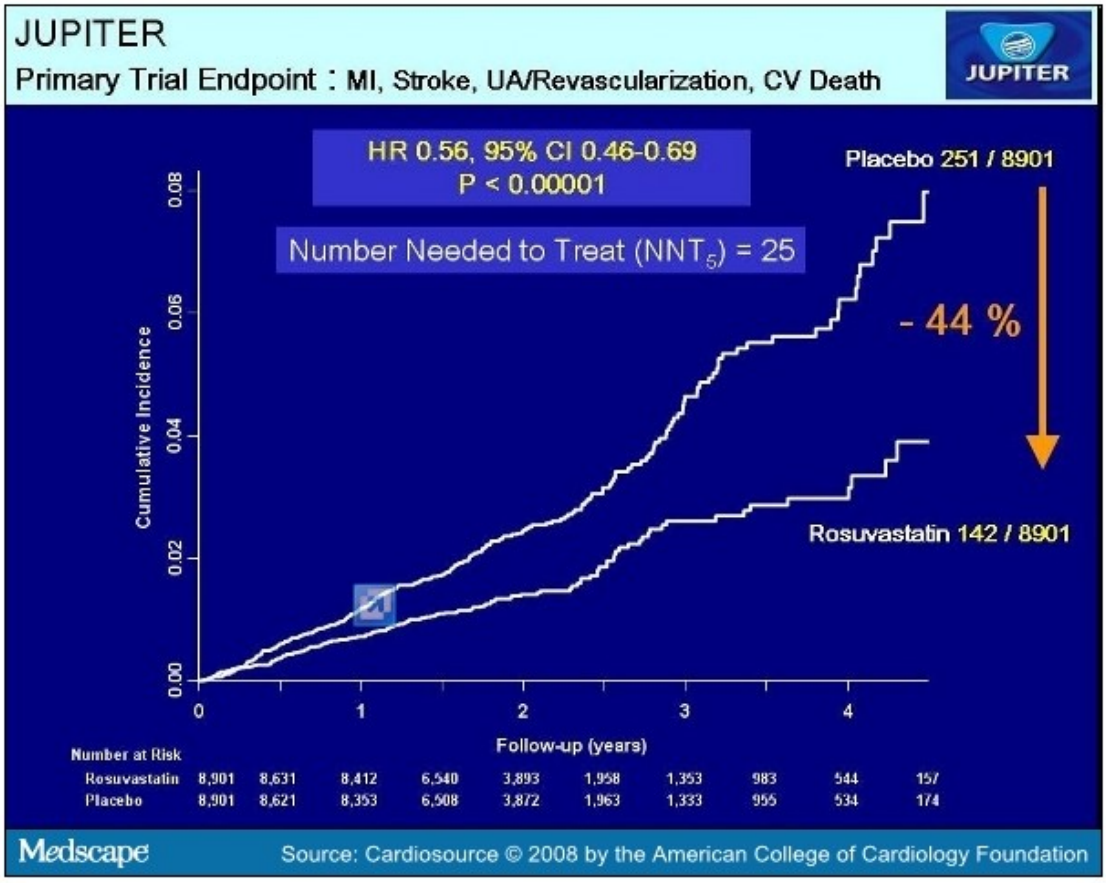
- known hypersensitivity
- life threatening angioedema
- **pregnancy (teratogenic)**
- **bilateral RAS** or sole kidney RAS (AT II is needed to maintain GFR – ACEI will cause acute RF)
- aortic stenosis
- Hypertrophic Cardiomyopathy – risk of hypotension from fixed outlet obstruction

45. What are the effects of statins?

- Cholesterol lowering - reduce LDL and improve HDL
- Pleotrophic effects – EXTREMELY important:
  - Positive effects seen in people with NORMAL cholesterol
  - Stabilize plaque
  - Reduce macrophage activity in the plaque
  - Cause plaque fibrosis
    - Rupture prone (echolucent, lipid laden) transform into tough stable plaque (ehogenic, fibrous)
  - Nutritive effect on endothelium that changes procagulant/anticoagulant properties of endothelium
    - Reduce venout thromboembolism
    - Stabilize AAA and reduce their growth (yes, sir)
- Overall, reduce stroke, reduce CV mortality.

46. Jupiter trial, 2009:

- Pts with normal lipid profile but high CRP
- Males > 50 yoa, females > 60 yoa, NO history of DM, CVA, MI
- 17,000 pts randomized to placebo vs 20 mg of rosuvastatin
- At 2 years trial stopped
- Impressive reduction in:
  - in the various combined endpoints, which included stroke, heart attack, angina, and bypass surgery (44%).
  - combined endpoints of stroke, myocardial infarction, and cardiovascular death (47%) as well as a
  - reduction in total mortality (20%).



47. What mechanical factors can injure endothelium?

- Embolectomy cath
- PTA/wire
- Endarterectomy
- Valvulotomy
- Overdistension of vein graft
- Anastomosis construction

48. Steps in intimal hyperplasia development:

- Endothelial injury
- Coverage of denuded area by carpet of platelets
- Ppts release GF
  - PDGF, EGF, FGF
- Growth factors stimulate endothelial AND smooth muscle cell proliferation
- Platelets are displaced by neo-endothelium
- Medial SMC proliferation caused by platelet GF
- **SMC migration across internal elastic membrane into intima**
- **Generation of exracellular matrix by SMC in initima**

In the end, luminal diameter is decreased…

49. How can intimal hyperplasia be prevented/treated?

- Antiplatelet agents – inhibit platelets - mediators of hyperplasia
    - ASA, GP IIBIIIA, plavix
- Seeding of grafts with endothelium – experimental, only prox and distal ends of the synthetic graft are seeded.
- Inhibit SMC:
    - Drugs – sirolimus, tacrolimus
    - Local irradiation
    - Nitric oxide
    - Gene therapy
- Further surgical reconstruction: animal studies suggest that IH is self-limiting - **once complete, renewal or continued hyperplasia is unlikely – hence surgical reconstruction of stenosed graft is feasible.**

50. How can RF for atherosclerosis be modified:

- DM control – diet, exercise, medication, weight loss, foot care
    - Reduces MI/death due to vascular causes
    - UNCLEAR if it prevents progression of ulcers,
    - No eveidence it prevents amputations or infections
- HTN – diet, exercise, medication
    - decreases
        - all cause mortality by 12%,
        - stroke mortality by 36%,
        - coronary mortality by 25%
- Dislipidemia – diet, exercise, medication, weight loss
    - stabilize plaque and progression of PAD
        - 40% reduction in progression of IC
    - for every 10% reduction of total cholesterol, 15% reduction in mortality
    - improved patency of infrainguinal bypass
- Smoking – cessation program, drugs, support groups
    - halt claudication -> rest pain progression
    - improves patency of revascularization procedures
    - improves survival
    - no evidence it improves symptoms of IC
- hyperhomocysteinemia – by diet and folate
    - impact unknown yet

51. Target for lipids:

- Ideally LDL < 3 mmol
- HDL > 1 mmol
- However, if risk for CAD
  - low – LDL < 5 mmol, TC/HDL < 6
  - moderate – LDL <3.5 mmol, TC/HDL < 5
  - high – LDL <2 mmol, TC/HDL <4
- If have DM – LDL < 2 mmol

52. PAD and risk reduction:

- Life style modification:
  - Weight loss
  - Exercise
  - Cease smoking
  - Control HTN
  - Control Lipids
  - Control DM
- Specific Drugs:
  - Platelet inhibitors:
    - ASA, COX inhibitor,
      - limits production of thromboxane A2
      - inflammation, platelets' aggregation and vasoconstriction is limited
      - Platelets are affected irreversibly
      - endothelium quickly regenerates
        - prostacyclin secretion is restored.
        - This happens with low dose ASA –
        - Explains better small dose effectiveness compared to a large dose.
      - given periop, see reduction of CABG failure by 10%

    - Clopidogrel/plavix/ticlid:
      - ADP receptor inhibitor
      - Prevents activation of GPIIb/III a complex
        - An ADP dependent process
      - ADP/collagen/platelet activating factor (PAF)& adenosine induced ppt aggregation is limited

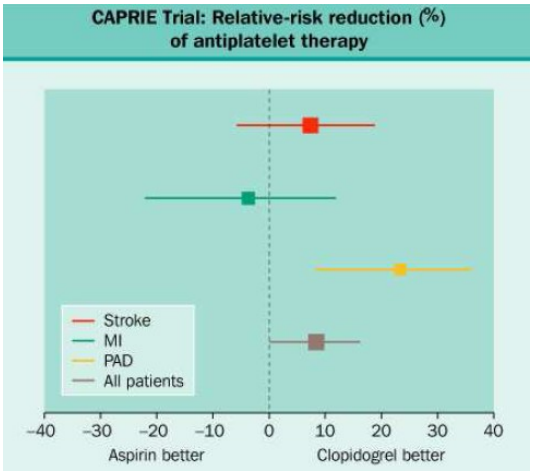
    - GP IIb/IIIa receptor inhibitor –
      - used post cardiac stents,
      - inhibit binding of fibrinogen/vWF to the above named receptor
      - possible anti SMC effect

- vasodilators:
  - cilostazole, 100 mg bid,
  - phosphodiesterase inhibitor,
    - increases cAMP in ppts
    - reduces ppt aggregation
    - increases SMC relaxation.
  - CI in CHF.
  - S/e: n/v, diarrhea, rash, dizziness, palpitations
- rheologic:
  - pentoxifylline –
    - 1200 mg od,
    - increases RBC deformability and decrease for viscosity.
    - 20% improvement in claud distance in some studies over 6/12.
  - s/e: n/v/dizziness

- ACEI
  - Vessel:
    - Dilates (through NO)
    - Remodels (SMC effect)
  - Platelets
    - Reduces aggregation
  - Lipid
    - Decreases LDL oxygenation
  - Renal protection
  - Controls HTN (anti aldosterone and ADH effect)

- Statins:
  - Vessel:
    - Remodels via effect on SMC
      - proliferation and migration inhibition
    - Improves endothelium function
  - Plateletes
    - Reduces aggregation
  - Lipid
    - Reduces cholesterol
    - Reduces LDL oxidation and uptake
  - Reduces insulin
  - Increases fibrinolysis\

ASIDE:



Osler notes…
Role of antiplatelets, statins and ACE inhibitors in the management of patients with peripheral arterial disease.

http://ves.sagepub.com/cgi/reprint/40/4/312 - v. useful article plus TASC

Antiplatelet therapy: inactivate platelets thus lowering thrombotic complications of PVD associated with ulcerated plaque rupture. May have benefit in maintaining patency of prosthetic grafts.

Evidence: In pts with PAD particularly as it was shown to decrease overall reduction in cardiovascular events. This was initially demonstrated in pts with PAD and coexisting CAD and Cerebral arteries Disease (Antithrombotic Trialist's collaboration (25% reduction in CV events); and later confirmed for all subgroups of pts with PAD (23% odds reduction). Compared to ASA, Clopidogrel offers 24% better risk reduction in CV events in symptomatic pts with PAD (absolute RR is only 1.2%). Combination of ASA/clopidogrel is required post SFA stent insertion to reduce risk postoperative instent thrombosis. Antiplatelet medications have beneficial effect on patency of prosthetic lower extremity bypass grafts: RR of occlusion while on the ASA therapy is 0.78

The CAPRIE trial examined the relative safety and efficacy of daily doses of 75 mg clopidogrel vs 325 mg ASA in nearly 20,000 patients with ischemic stroke, MI, or PAD.The results of the trial showed that clopidogrel was more effective than ASA in preventing the primary study end point, a composite of ischemic stroke, MI, or vascular death. The trialists found a significant 8.7% relative-risk reduction ($P = .043$) for clopidogrel over ASA.

Post hoc analyses of the CAPRIE trial have shown that certain subgroups of patients with high stroke risk, including those with diabetes mellitus,those with prior cardiac surgery, those receiving concomitant lipid-lowering therapy, and those with a history of more than 1 ischemic event, received significant advantage from clopidogrel over ASA. For example, in the subset of CAPRIE patients with diabetes, annual event rates for the

composite of vascular death, MI, stroke, or rehospitalization for ischemia or bleeding were elevated compared with the rate in nondiabetic patients, corresponding to an amplified benefit of clopidogrel over ASA in these high-risk patients (21 vs 9 events prevented per 1000 patient-years for this end point).

Statins: they lower LDL, TG, LP(a) – factors involved in pathogenesis of atherosclerosis. Also, there is evidence they modulate arterial wall inflammation, plaque stabilization, endothelial dysfunction, and thrombosis, reduce fasting insulin concentration.

Evidence: Heart protective study demonstrated that 40 mg of simvastatin at 5 year f/u in pts with PAD resulted in 12% reduction of total mortality, 17% reduction in vascular mortality, and 24% reduction in CAD. These findings led to recommendation to lower LDL to <2.59 mmol/L. 1 mmol/l reduction in LDL is associated with 20% RR in major CV events – regardless of the baseline lipid level (i.e. including normal range) and only depended on the baseline assessment of CV risk, with PAD pts being at high end of the spectrum.

ACEI and ATRB: useful adjunct to BP management, particularly in pts with diabetes and PVD. Moreover, it ACEI and ATRB were shown to effect remodeling of the myocardium and vessel wall: they share similar to statins pleotrophic effect on arterial wall.

Evidence: HOPE study demonstrated 22% reduction in CV events in patients on ramipril, independent of the blood pressure lowering effects. They have numerous pleotrophic effects on the arterial wall inhibing SMC migration and proliferation, oxidation of LDL, platelet inhibition, stimulation of NO secretion.

53. Conservative measures of treatment of claudication:

- Most effective – walking
    - Endothelial function optimization
    - Alters muscle metabolism (anaerobic training)
    - Re-trains to use more proximal muscles
    - Collateral development – unlikely….
    - Improves metabolism of lipids and glucose
- Drugs – cilastazole, pentoxyphylline (placebo?)
- Smoking, RF – no clear evidence that it will reverse claudication, but will control progression of atherosclerosis and possible conversion to claudication.
    - 11% of smokers with IC will undergo amputation, compared to 0% in non-smokers
    - 3 fold higher risk of needing intervention if pt has 40 pack year of Smoking
    - Cessation will improve patency of bypass 3 fold…

- Statins – will halt progression to CLI but won't help symptoms of IC

- 40% reduction in progression to CLI

54. DD of claudication:

- Atherosclerosis
- Non-atherosclerotic disease
  - Coarctation
  - Entrapment
  - Adventitial cystic disease
  - Persistent sciatic a
  - FMD of EIA
  - Pseudoxanthoma elasticum
  - Endothelial fibrosis of cyclists
- mimickers
  - Venous claudication
  - Chronic compartement syndrome
  - Peripheral nerve pain
  - Spinal cord compression (OA)
  - OA hip

55. Risk factors and marker of increased risk for PVD:

RF:

- HTN
- HL
- DM
- Smoking
- Homocyseinemia

Markers

- CAD
- Previous PVD events
- Sedentary lifestyle
- Fibrionogen
- Obesity
- Fam hx
- Inflammatory mediators

p. 1897

56. What enzymatic deficiency is found in hyperhomocysteinemia?

- Homocystein, product of methionine
- 3 enzymes
  - cystathione B- synthetase (CBS)

- homocysteine methyl transferase (HMT)
- methylene tetrahydrofolate reductase (MTHFR)

- homocystein is not fully metabolized => partial metabolites accumulate
- homocysteine thiolactone (HTL) accumulates
  - toxic to endothelium
  - changes luminal charge
  - causes cellular aggregation
  - accelerates atherosclerosis (LDL metabolism)
- treatment
  - folic acid
  - vit B6, 12, Choline, Betain
    - these help to metabolise HTL
  - folic acid treatment, however, did not demonstate any effect on CV mortality…

SYMPATHECTOMY

57. How does sympathectomy work?

- Increase in blood flow
  - Drop in resting vasomotor tone
  - Most of increase is non-nutritive, via AV shunting
  - Diminishes after 5 days (5^{th} day phenomenon)
    - resting vasomotor tone returns to normal in 6 month
- Collateral flow increase:
  - Average 11% increase in flow in animal models
- Alteration in pain perception
  - Effective for rest pain
  - Central and peripheral signal conduction attenuation

- Overall - between healing superficial ulcer and relief of rest pain, sympathectomy is more likely to help RP:
  - needs less increase in blood flow to relieve rest pain compaired requirements for ulcer healing
  - effective pain impulse conduction interruption & attenuation

58. Indications for Lower extremity sympathectomy:

- hyperhydrosis
- Complex regional pain syndrome
- LE vasospasm - Raynaude's DISEASE
  - Rare indication but responds very well
- PVD:

- Ischemic rest pain: criteria….
  - ABI > 0.3
  - Absent neuropathy
  - Limited tissue loss
- Ischemic ulceration: criteria…
  - ABI >0.3
  - Shallow ulcers, non-infected
  - Will heal in 33%, but won't prevent amputation
- As an adjunct to Arterial reconstruction
  - Particulary if small vessel anastomosis is involved
    - Dextran 40 infusion may be just as effective for perioop prevention of thrombosis of difficult distal anastomosis.
  - May not improve long term patency of bypass

Technique:

- Retroperitoneal dissection
- ID psoas muscle
- Chain lies over transverse process medial to psoas m.
  - On the R – under IVC edge, on the L – lateral to aorta
  - Remove ONLY L2 -3
    - If remove L1 – retrograde ejaculation
    - L4 does nothing

- Complications:
  - Post sympathectomy neuralgia
    - Seen in 50%!
    - Anterolateral thigh ache
    - Worse at night, unaffected by activity
    - Goes away in 12 months
  - Gen-fem n. injury
  - Male sexual dysfunction:
    - Retrograde ejaculation
    - If bilateral L1 sympathectomy – 25-50%
  - Failure to achieve adequate levels of pain control

59. Lumbar sympathectomy: outcome …

- Excellent outcome expected in:
  - Complex regional pain syndrome
  - hyperhydrosis
- good/fair outcome expected in:
  - Raynodes
    - However, response is transient …
  - Buerger's disease

        - Non-bypassable atherosclerotic occlusion with limited tissue loss
    - Poor outcome seen in:
        - Claudicants
        - DM neuropathy

60. Upper extremity sympathectomy:

- Indications:
    - Hyperhydrosis
    - CRPS
    - Raynauds disease (high recurrence)

How:

1. Open (transaxillary - painful, paravertebral – extensive dissection, supraclavicular – high incidence of Horner's)
2. Thoracoscopic:

- Collapse lung
- Visualize 1st 4 rib/vertebra
- ID subclavian a. – superior extent of dissection
- Sympathetic chain – dorsal, phrenic/vagus nerve – ventral
- Remove all sympathetic ganglia below T1 – i.e. t2 and t3.
- Leave stellate ganglion intact – otherwise horner's:
    - Upper ptosis (Muller's muscle denervation)
    - Lower upside ptosis
    - Myosis
    - +/- enhydrosis and loss of ciliospinal reflex (neck pain – skin prick - causes ipsilateral pupil dilation)
    - No Facial sweating

Most common complications:

- Compensatory hyperhydrosis (100%)
- Horner (up to 40%)
- Intercostals neuralgia (30%)
- Phrenic nerve is NOT damaged in thoracoscopic approach.

VASCULITIS

61. Raynaud's:

- Syndrome –
    - due to occlusive pathology,
    - may be unilateral, may lead to ulcers
    - Associated with
        - CTD, atherosclerosis, hyperviscocity, vibrational trauma

        - 90% - CTD – scleroderma, sjogren, SLE, RA, myositis
    - On hx:
        - r/o joint pain, rash, muscle pain, systemic sxs, hypothyroidism, repetitive trauma, frostbite, PAD/sxs of TOS
    - Labwork:
        - SLE – homogenious ANA
        - Sclreroderma – speckled ANA
        - CREST – anticentromere AB
            - Calcinosis, raynauds, esophageal dismotility, sclerodactyly, telagectasia
- Disease –
    - vasospastic, bilateral, no ulcers
    - Etiology is uncertain
        - Primary problem is on ENDOTHELIAL level
            - More vasospastic than relaxing
        - Exaggerated response of SMC to sympathetic stimulation
        - Sympathetic pathway is overstimulated
    - Color change – white, blue, red –
        - 98% precipitated by cold, 2% by emotions

- Differentiate spastic DISEASE - primary VS obstructive SYNDROME -secondary
    - Spastic: PVR and waveforms normal at room temp, worse with cold
    - Obstructive: **at room temperature – SEE abnormal PVR**

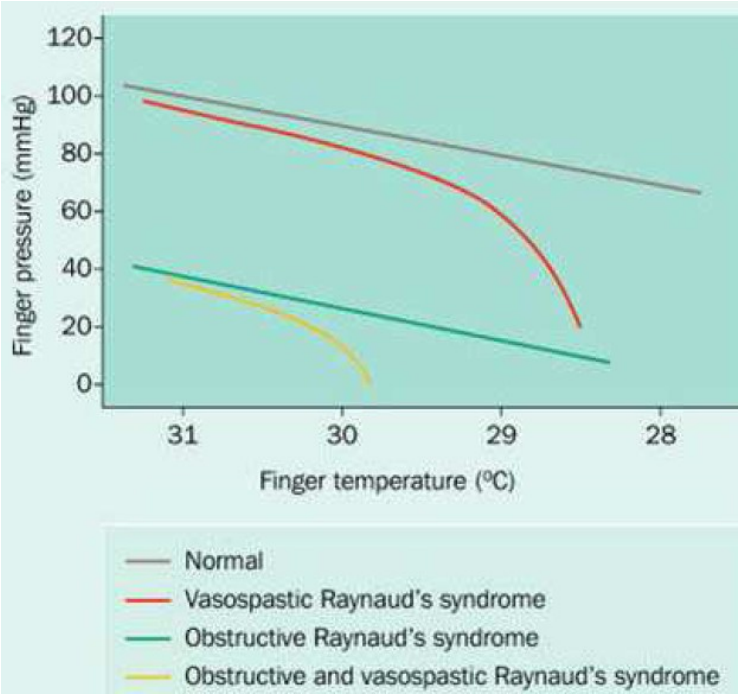
Diagnosis of DISEASE: thermal test done only when obstructive component is excluded.

- Early vasosaspm with temperature drop (two cuffs: proximal digit cold, distal digit warm)
- Recover takes > 20 min
- Have reactive hyperemia on rewarming
- See Peaked pulse (suggestive of vasospasm)

20% of Raynaud's have scleroderma,
80% of scleroderma have Raynaude's
MCTD – 5%
Atherosclerosis – 8%
Buerger – 4%

Table 93-1. Clinical Features to Distinguish Primary from Secondary Raynaud's Syndrome

TYPE	GENDER	OTHER FEATURES
Primary	Usually female	Age < 45 years
		Vasospasm of multiple or all digits
		Normal vascular examination
		No skin abnormalities
		Normal laboratory studies
Secondary	Male or female	Any age
		Single digit involved
		Abnormal pulse examination
		Vascular laboratory abnormalities
		Positive autoantibodies



Treatment:

- Avoid cold, betablockers, smoking
- CCB, alpha blockers, yohimbin helpful
- In rare cases – consider sympathectomy
- Prognosticate –
    - Evaluate presence of symptoms of CTD
    - Perform CTD antibody screen
- if CTD screen positive, patient may have progession to obstructive pattern
        - i.e. in this case, dealing with early Raynaud's syndrome NOT just Raynaud's disease
    - If there are no CTD symptoms at presentation

        - risk of CTD is 6% at 3.3. years
    - If symptoms of CTD are present:
        - risk of CTD is high (up to 50%)

62. Connective tissue disorders:

- Systemic sclerosis (aka scleroderma)
    - Strictly speaking – small vessel VASCULOPATHY, NOT VASCULITIS
    - **NOT an inflammatory vasculitis**
        - Likely due to SMC proliferation
    - Luminal narrowing
    - Most commonly CTD associated with Rayaud's
    - Unknown etiology
    - Fibrosis of skin and internal organs

\

- Subtypes of scleroderma:
    - Diffuse:
        - **10 year survival 40-60%**
        - Pulm hypertension
        - Renal failure
    - Limited scleroderma
        - CREST
            - **10 year survival 70%**
            - Calcinosis, raynaudes, esophageal dismotility, sclredodactyliy, telangectasia
            - More benign
            - Less heart/lung/kidney problems
        - Diagnosis:
            - Clinical
            - AB:
                - Positive ANA
                    - speckled pattern
                    - seen in 95% of pt
                    - NONSPECIFIC
                        - see also in SLE - homogenious
                - Scl-70 more specific
                - **ESR NORMAL**
- SLE
    - Young females, but all age groups are susceptible
    - Arthlagia, skin rash, pericarditis, pleuritis, Glomerulonephritis
    - Positive ANA – homogenious pattern
    - Raynaud phenomenon is seen in 70%
- Rheumatoid arthritis and Sjogren:

- Small vessel vasculitis with obliterative fibrosis

- Mixed CTD:
  - Overlap of two CTD
    - Usually SLE and Scleroderma
    - Polyarterirtis
    - Mixed bag

63. Diffirential diagnosis of positive ANA:

- SLE – homogenious ANA
- Sclreroderma – speckled ANA
- CREST – anticentromere AB

64. Vasculitis:

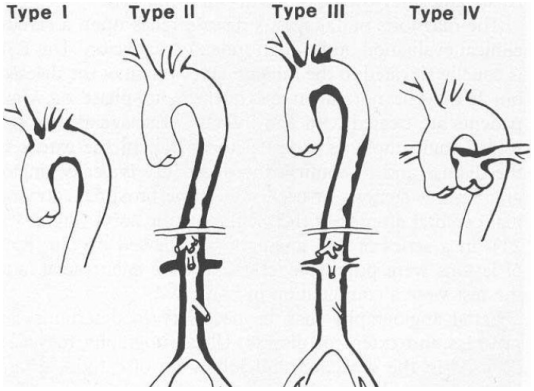
Table 28-5. Classification of Vasculitis*

Vessel size	ANCA NEGATIVE	ANCA POSITIVE
Large	Takayasu's arteritis	-
	Giant cell arteritis	-
Medium	Kawasaki's disease	Churg-Strauss angiitis (P-ANCA)
	Polyarteritis nodosa	-
	Behçet's disease	-
	Drug abuse vasculitis	-
Small	Henoch-Schönlein purpura	Wegener's granulomatosis (C-ANKA)
	Essential cryoglobulinemia	Microscopic polyangiitis (P-ANKA)
	Arteritis of connective tissue (Scl, SLE, RA, MCTD)	

65. Types of Takayasu Arteritis:

modified Ueno classification

- aortic arch only: 40%
- descending thoracic and abdo aorta – middle aortic syndrome : 11%
- involves both 1. and 2. : 65%
- pulmonary artery involvement +/- 1-3: 15%
- all to a happy total of 131% ☺



Rutherford:

- most common large vessel arteritis
- disease of the young < 30 year olds
- both stenotic and aneurismal disease
- in NA, most common presentation is upper limb ischemia, closely followed by CVA.
- HTN in 33-88%
    - renal a. stenosis, may be missed unless pressure in both arms is measured
- Isolated AAA
- Aortic regurgitation (ascending a. dilation) – 20%
- Pulmonary HTN (unexplained dyspnea)

- **10% of active disease have normal CRP and ESR**
- Most often disease is diagnosed at reconstruction
    - i.e. not in an active stage

- Unknown etiology, Pan-arteritis, Patchy involvement
- Granulomatous lesions, but no caseation and cavitation
- Disease is transmural
    - i.e. there is NO role endarterectomy or patch angioplasty – prefer bypass…
- Responds to systemic steroids and cytotoxic drugs
    - if in active stage

Stages:

- Prodromal part
- Inflammatory
- Burned out

Clinical presentations:

- Stroke
- CHF
- HTN

- CRF
- Aneurysm
- Pulm HTN
- Aortic regurgitation

  - Mortality due to uncontrolled hypertension: stroke, CHF
  - Can't follow stages using labs, need to have serial imaging.
  - Main pathology is stenosis/obstruction, occasionally aneurysm:
  - Surgery is contemplated in burnt out stage only

  - Long stenosis –
    - Sparing ascending aorta (in 95% of cases)
    - carotid
      - bypass to the level of the carotid bulb
    - Renal artery
      - may consider PTA 1st for renal a. stenosis
      - poor long term results for other locations
    - Infrarenal aorta – up to (but not at) the level of bifurcation
      - May consider thoracic aorta -> single iliac bypass – will reperfuse the other extremity via preserved bifurcation…
    - Innominate artery/subclavian:
      - Bypass to subclavian/axillary artery –
        - Not for ischemic symptoms
        - Primaly purpose: to be able to diagnose HTN (by arm BP measurement)
        - Complications of HTN is the most important cause of mortality in Tak…

Important: make sure disease is in burned out stage before bypassing.

66. Giant cell arteritis:

- Elderly affected
- Predominantly middle sized vessels and aortic involvement
- Frequent involvement of ECA branches, including ophthalmic artery
  - Painful temporal a.
  - Retinal ischemia leads to blindness
  - Jaw claudication in 50%
- Can have aortic arch involvement only
- Polymyalgia rheumatica common
- Criteria of Am Col Rheumatology:
  - Age > 50 yoa
  - Localized headache

- Temp artery tenderness on exam
- ESR > 50
- TA biopsy positive for GCA
  - Up to 20% of pts have normal bx
    - 40% negative with aortic arch syndrome only

67. Behcet disease diagnosis:

- Systemic vasculitis, unknown origin
- Hallmark: Oral/genital ulcers and recurrent uveitis
- NO lab markers

Diagnostic criteria:

- Major – oral ulcers
- Minor: need 2
  - Genital lesions:
    - recurrent ulcer
  - Eye lesions:
    - Ant/post uveitis/retinal vasculopathy – seen in 80%
      - May lead to blindness
  - Skin lesions:
    - erythema nodosum, pseudofolliculitis, acne,
    - Pathergy:
      - clear pustule 48 h post skin puncture
  - Other:
    - venous thrombosis (UE, LE, SVC, IVC)
      - MOST COMMON VESSEL PATHOLOGY
        - 50% of pts
      - Due to:
        - Prothrombic state
        - Endothelial injury
        - Defective fibrinolysis

    - Arterial pathology:
      - SECOND MOST COMMON VESSEL PATH
        - Up to 34% of pts
      - aneurysms -
        - more common than occlusive
        - AAA >pulm >fem >pop>brachial >iliac
        - **Leading cause of death in BD – RUPTURE**
        - Intracranial aneurysms described
      - occlusive arterial disease LE and UE
        - better prognosis

        - increased risk of bypass thrombosis
    - pericardis 3-6%
    - Arthritis
    - GI ulcers
    - CNS (seizures/meningitis/palsy),
    - Glomerulonephritis - 7.5%

Summary:
- sore mouth AND sore eye/penis/skin/other
- pathergy
- die from AAA rupture
- no lab test, clinical diagnosis
- more venous rather than arterial problems

Treatment:
- Immunosuppressive for ophthalmic/neurologic/vascular complications
- No serologic markers to follow
- Large dose steroids for vascular, may need second agent
    - Cyclosporine
- For oral/genital ulcers – THALIDOMIDE
    - if this drug is given for females, consider doing hysterectomy or TL first

68. OTHER Mid & small vessel vasculitis:

- PAN – polyarteritis nodosa
    - Systemic necrotizing vasculitis
    - Aneurysms/ruptures/thrombosis in any organ
    - Common presentation:
        - abdo pain in young adults
            - Mesenteric aneurismal involvement
    - ESR up, but ANCA is negative
    - Angio – multiple visceral aneurysms
    - Hep B antigen positive in 30%, associated with HIV

- Kawasaki:
    - Affects infants/children < 5yoa
    - 20-30% - coronary aneurysm
    - Other: pericardial effusion, MR, CHF
    - Multiple other (aorta, viscera) aneurysms with age

- Churg-Strauss – three stages

i) Allergic phase – sinusitis, rhinitis, Asthma
ii) Eosinophilia with eosinophilic infiltrates
    - pneumonia, gastroenteritis, neuropathy

iii) Vasculitis
- Mc site –coronary, but any vessel can be involved
- **P**-ANCA positive
- More **P**roximal vessels involved

69. Small vessel arteritis:

- Wegener's
  - Necrotizing granulomatous vasc
  - Classic lesions: Kidney and Upper resp tract
  - Presentation: digital ischemia and nail fold infarct
  - **C-ANCA positive in 90% in active disease**

- Microscopic polyangiitis
  - Just vasculitis of small vessesl, P-ANCA positive

70. Small vessel pathology leading to digital ishchemia: DD

- ANCA positive:
  - Microscopic polyangiitis
  - Wegener's
- ANCA negative:
  - CTD
  - Cryoglobulinemia
- Trauma:
  - Vibration
  - Frost
- embolic

71. Arteritis associated with aneurysm formation:

- Takayasu
- PAN
- Kawasaki
- Behcet
- Drug induced

Other W&W conditions that can have AAA: Ehlers-Danlos, Marfan, Turner, PCKD

WEIRD & WONDERFUL

72. Buerger's disease diagnostic criteria:

- Predominantly male, but may see in female

        - In North America, up to 15-30% of pts are WOMEN
    - Onset < 45 yoa
    - Smoking hx
    - Infrapopliteal/infrabrachial arterial involvement leading to ischemia
        - documented clinically (RP, ulcer) and objectively
    - No other RF for atherosclerosis (DM, htn, lipids)
    - Echo and angio exclusion of:
        - proximal embolism (cardiac, TOS, aneu, arch, athero)
        - trauma
        - local lesions (adv cyst d, pop entrapment)
    - Lab test exclusion of autoimmune, CTD, DM, myeloproliferative DO
    - Other features:
        - Migratory phlebitis, Raynaud's, instep claudication

Etiology: no one knows, smoking, genetics, hypercoag and endothelial dysfunction, immunologic mechanism

Histologically:
- thrombus is *inflammatory*
- inner elastic *lamina is spared*
- no acute phase reactants (unless acute infarction of limb)
- markers of immune-activation are absent
- discontinuous lesions

Summary:
- distal arterial disease
- WITH smoking
- WITHOUT DM, lipid, HTN, embolus, CTD, myelo

Treatment:
- Stop smoking, no nicotin patches/gum, pain management, ASA, CaCB, iloprost, debride, bypass or amputate if necessary
- Pain control – spinal cord stimulator

Amputation rate:
- in smokers – 43%,
- ex-smokers – 6%

73. Angiographic features of Buerger's disease:

- Normal proximal a.
    - i.e. no atheroscelrosis
- Involvement of vessels distal to brachial and pop artery
    - Segmental involvement (normal intermingles with abnormal)
    - Severity increases distally

- Collateralization in the vasa-vasorum – cork-screw collaterals
- No source of embolism

74. Uncommon causes of aneurysms

- Arteritis (see above)
- Marfan:
    - Must have clinical findings, not genetically documented classic FBN1 mutations alone
        - 25% of the patients have new mutation
    - Defective elastic tissue
    - Disease continuum
    - Tall thin, long arm/legs, Arm/height > 1.05
    - Pectus carinatum/excavatum
    - Aortic dilation
        - Ascending involved in 80%
    - BB prophylaxis essential
    - Replace ascending aorta/sinus when >5 cm
        - In pregnancy – use BB,
        - dilation to > 4cm is high risk for rupture
- Ehlers-Danlos:
    - Collagen synthesis problem
    - Skin hyperelasticity, fragility, joint hypermobility
    - 11 types – diverse clinical presentation
    - Type 6 has vascular relevance
        - Only 4% of all types
        - Reduced/abnormal type 4 collagen
            - Thin skin, easy bruises,
            - "Alien from the flying saucer" face (thin lips, prominent eyes, narrow nose, no sc fat)
        - Most relevant presentation is Perforation:
            - Vessels
            - Uterus – 11-25% of pregnancies in these pts
            - Colon (sigmoid)
        - MC cause of death – arterial rupture
        - Multiple aneurysms, only 16% aware of diagnosis prior to rupture
            - Unusual compartement syndroms (buttock with gluteal a. rupture)
        - In emergency, ligation preferable to bypass
        - Endo may have role
        - Avoid sports
- Turner, polycystic kidney associtated with TAA

75. Types of collagen:

- 1 –MC, 90%, tendons, ligs, bones
- 2 – hyaline cartilage
- 3 – vascular structures and colon
- 4 & 5 – basement membranes and CT matrix

76. Features of pseudoxanthoma elasticum?

May present as young male with retinal hemorrhage, coronary artery disease, and bilateral leg claudications.

Pseudoxanthoma elasticum:

- CTD that causes elastin degeneration -> calcification
- Predisposes to Early AGRESSIVE diffuse atherosclerosis
- 70% of patients are < 35 yoa
- Skin, eye, cardiovascular system
- Xanthomas along neck/groin flexion lines (chicken skin)
- Spontaneous retinal hemorrhages -> blindness

- CAD
  - Don't use LIMA/RIMA, only GSV…
- Stroke
- Soft tissue calcification (elbow, hip)
  - Dd trauma, scleroderma, hyper PTH

Aggressive atherosclerosis RF modification.

77. Pathology of radiation vasculitis:

- Causes accelerated atherosclerosis
- Injury to vasa-vasorum
- Ischemic necrosis of vessel wall
- Fibrosis of internal elastic lamina
- Thickening of adventitia
- Long smooth tapering stenosis

78. Clinical syndromes associated with cystic medial necrosis:

- Marfan
- Ehlers-Danlos
- Some types of Neurofibromatosis
- All mucopolysaccharidoses

Hyaline degeneration of media, replacement with mucoid basophilc substance.

Presents as:
1. Aortic/carotid dissection
2. Disseminated arterial rupture
3. Spontaneous rupture

79. Visceral Splanchnic Artery aneursyms:

- Splenic (60%),
- hepatic (20%),
- SMA (6%),
- Celiac (4 %)
- Gastric/gasroepiploic (4%),
- intestinal/pancreatic (2%).

i.e. After celiac, go clockwise: gastric, gastroepiploic, intestinal, pancreatic

80. Classification of splenic a. aneurysms:

Usually saccular, at bifurcations, multiple in 20%.

- True
  - Associated with arterial fibrodysplasia (FMD)
  - Associate with portal HTN/splenomegaly
  - Pregnancy induced (multiparity)
  - r/o PAN, Kawasaki, Ehlers-Danlos
- False
  - Pancreatitis induced
  - Trauma
    - Penetrating
    - Blunt
  - infected

81. Indications and treatment for splenic artery aneurysm repair:

- >2 cm in good risk pts, some say > 3 cm
- Pregnant or Potentially pregnant pt
- Symptomatic

- Rupture - < 2%
  - If pregnant – 95% of aneurysm diagnosed during pregnancy are ruptured
  - Double rupture – first in lesser sack, then periotoneal cavity

Repair:

- ligate or splenectomy
  - For proximal: simple ligation/exclusion no reconstruction
  - Mid: usually false aneurysms associated with pancreatitis
    - ligation,
    - then open aneurysm,
    - ligate branches/artery form within
  - Distal – splenectomy

82. Hepatic a. aneurysm:

- 2 times more common in males
- 25% medial degeneration – MC cause according to Rutherford Companio n
  - In 35 % atherosclerosis is seen – but this is co-incidental
- 22% pseudoaneurysm
  - trauma
- 15% mycotic due to IV drug abuse
- Other causes:
  - PAN
  - Trauma,
  - amphetamines
- 80% extrahepatic, 20% hepatic
- 20% rupture rate
- May ligate if in CHA,
- hepatic artery branches involvement may require reconstruction.

- Hepatic localization:
  - Embolectomy vs lobectomy

83. SMA, celiac, gastroepiploic aneurysm:

- SMA
  - 60% mycotic
    - Current papers says less
    - Non-hemolytic strep
    - More common in younger
  - 20% atherosclerosis
  - Drugs – cocain and ergot
  - Rupture can be seen in up to 40%
  - Do not stent these…

- Celiac aneurysm –
  - mostly degenerative (i.e. secondary to atherosclerosis)
  - rupture 13%
- Gastroepiploic

- If see multiple onces, particularly in GDA distribution – look for high flow situation
- 90% present with rupture
  - 70% into GI
  - 30% intraperitoneal
  - Ligate/embolise all

84. Renal a. aneurysm:

- Degenerative
  - FMD
  - Vasculitits
  - PAN
  - Behcet
- 90% extrarenal
- 3% rupture
- Repair in:
  - Impregnatable females
  - Symptomatic
    - Htn
    - Hematuria
    - Pain
    - Hydronephrosis
  - >3-4 cm in asymptomatic

85. Complex regional pain syndrome :

Aka causalgia…

- This syndrome is **complex**
  - involves autonomic, vascular, motor, cutaneous, inflammatory changes
- it is **regional**
  - sxs and findings are beyond the original region of injury
- it is very **pain**ful
  - severity usually out of proportion to the initiating event

- Type 1 – due to inciting noxious event
  - Trauma (MC)
  - Non-traumatic
    - Post prolonged bed rest
    - Post MI/CVA/neoplasms
      - Shoulder-hand syndrome
  - Idiopathic
- Type 2 – due to nerve injury

flare->dystrophy->atrophy

- Stage I acute:
  - Hot&sweaty, swollen, burning hyper-pain
  - Tx: physical therapy main stay. If can't do this because of pain, try SYMATHETIC BLOCK – may be long lasting
  - steroids, local nerve block, TENS (transcutaneous electric nerve stimulator)

- Stage II dystrophic:
  - cold, mottled, osteoporotic/brittle nails, continuous pain
  - Tx: physio but may try sympathectomy
    - UNLESS there is dramatic response to sympathetic BLOCK first, do not go for sympathectomy
  - TENs, steroids

- Stage III atrophic:
  - atrophic, contracted, pain elsewhere
  - Tx: sympathectomy less successful, physio, TENs, antidepressant

Most helpful confirming diagnostic feature of CRPS is RESPONSE to sympathetic block. Other tests – abnormal sweating, thermography.

86. Types of FMD:

Cause:

- Estrogen effect
- Mural ischemia (lack of branches)
- Repetitive trauma (bending and stretching)

Think of Jabba the Hutt –

- tough fibrous core,
- tough fibrous/hyperplentiful middle,
- malignant/dysplastic exterior…

  - Inside – intimal FIBRO plasia – 5%
    - M=f
  - Middle –
    - medial FIBRO plasia – 85%
      - most common
    - medial HYPER plasia – 1%
      - rare
  - perimedial DYSPLASIA – 10%

- Most common is medial fibroplasia

- String of beads aneurismal dilatation
- **Middle to distal renal a. affected**
- b/l in 55%
- R sided more common than L
- Histology –
    - fibrous connective tissue replaces Smooth Muscle Cells
- 10% have berry aneurysms in head
    - 80% solitary
    - 20% multiple

87. Most common arteries affected with FMD;

- Renal artery
- Carotid arteries
    - 0.4% of all carotid angios show this, 65% bilateral
- External Iliac arteries

Treatment of RENAL FMD: PTA…
Treatment of CAROTID FMD:

- Standard – open gradual intraluminal dilation. 2 mm to 6 mm to the base of the skull, back flush debree from ICA.

- Other option: PTA, but in a review of 170 cases 5% had neuro deficit. Clinical Scenarios in Vasc Surgery do not recommend this as a

88. Portal hypertension and bleed:

- Treat definitively after first bleeding episode
    - 70% will rebleed with 75% mortality rate
- Banding and observation is contraindicated if pt leaves far away
    - Does not work well for gastric varix
- Beta blockers reduce rebleed by 50%
- TIPS – 90% success, but 25% portosystemic encephalopathy

89. Indication to treat vasc malfomations:

- absolute
    - local effects:
        - distal steal/ischemia
        - non-healing ulcer
        - bleeding
        - Leg length discrepancy
        - Aneurismal degeneration
    - systemic effects:

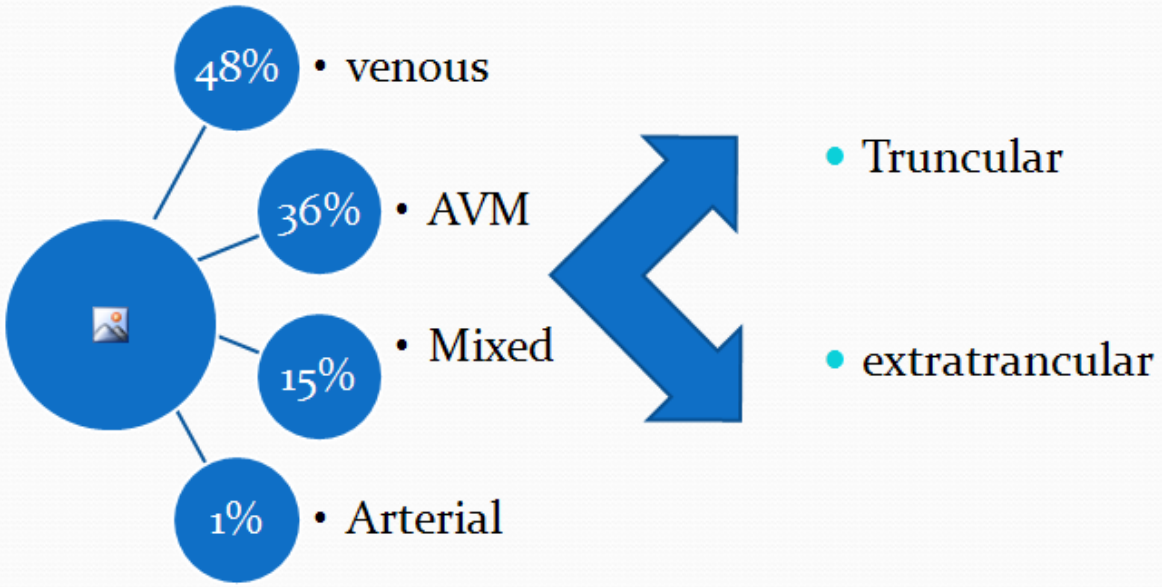
- CHF
- DIC

- Relative:
  - Cosmesis
  - Disabling Pain
  - Limiting claudication
  - Compression of structures

90. Hamburg classification of vascular malformations:

- Tumors
  - Abnormal endothelial turnover
  - MC infantile hemangioma
    - Starts at birth but usually seen at 2/52
    - Grows with child, regresses with age
      - 90% gone by 9 yoa

- Malformations
  - Normal endothelial turnover



91. Klippel-Trenaunay:

- Slow flow AVM
- soft tissue/skeletal hypertrophy
- Capillary malformation

- Lymphatic hyperplasia
- Anomalous lateral leg vein of Servelle
- Other: hematuria, hematochezia, constipation, bladder outlet obstruction

Parkes webber – same plus Clinically FAST flow AVM. KT has them too but it is micro and not clinically significant…

Facial V1-V2 capillary malformation – suspect intracranial/leptomeningeal/oribtital vasc malformation. V3 carries no such prospect.

92. Sclerotherapy:

For varicosities – ethanolamine, polidocanol, NOT ethanol
VM – STD, ethanol.

93. Vascular tumors:

- Vascular leyomyosarcoma:
  - Most commonly found in IVC, not arteries. Dismal survival – discovered with mets.
- MC tumor to grow into IVC – RCC
- MC tumore to obliterate IVC in RP - sarcoma

94. Congenital defects and symptoms:

- Double aortic arch –
  - vasc ring around trach/esophagus, -> dysphagia/dyspnea
- Ductus arteriosus –
  - **Most common congenital abnormality**
  - shunt from aorta into pulm artery -> pulm HTN
- R abberant subclavian a. –
  - Takes off distal to Left subclavian artery, behind Trach/Eso or between trachea and esophagus, causing to dysphagia lusoria & dyspnea
  - May have aneurismal dilatation at origin (known as Kommeroll diverticulum)
- Persistent sciatic artery -
  - prone to aneurismal dilation
- Bovine arch – Innominate and L CCA are joined:
  - True common trunk seen in 8%, common origin only is 16%
- L vertebral off aortic arch: 8%

95. Persistent sciatic artery:

- Persistence of fetal circulation
- Off internal iliac, down to popliteal along sciatic n,

        - diminutive SFA present, no femoral pulse
    - Prone to degeneration
    - Presents with buttock mass, emoblizaiton, sciatic nerve pressure, embolization
    - 70% unilateral
    - Pop pulse but no femoral pulse
    - Aneurysm in 40%
        - at greater trochanter
    - Repair aneurysm vs ligation plus fem-pop bypass

96. Abberant Rt. Subclavian artery:

- 0.5-1% of people
- Caused by involution of R 4th aortic arch.
- Instead, R subclavian artery forms from 7th intersegmental artery **distal** to L subclavian artery
- Passes usually posterior to esophagus,
    - but can pass btw esophagus 80%, trach 15%, anterior to trach 5%
- Can result in dysphagia lusoria
- Note:
    - dysphagia lusoria can be caused by persistent abberant L SCA originating from R sided arch pressing on esophagus. RARE
- Kommerell's diverticulum – remnant of the 4th aortic arch at the aorta
- According to Rutherford text, repair only for
    - symptoms or
    - >4 cm in asymptomatic pt
- Repair:
    - endo exclusion plus extrathoracic reconstruction (hybrid)
    - branched endo graft
- If Kommerell is seen – which is most of the cases
    - need either
        - branched endo,
        - aortic arch repair

- Pts will also have bovine arch, L vertebral taking off aortic arch, R sided thoracic duct and anomalous R recurrent laryngeal nerve – direct course off vagus.

97. What is required to have a normal erection?

- Nerves:
    - Nervi erigentes – sympathetic T12-L4 and parasympathetic S2-S4
        - Parasympathetic –

        - cause vasodilation, leads to tumescence
      - Sympathetic –
        - Regulates ejaculation
    - nerves form plexus at the root of IMA and aortic bifurcation.
    - Nerves course anterior to L common iliac artery
      - Careful with dissection
  - Arteries:
    - Internal pudendal a. – need this as a requirement for successful erection

Mechanism of erection:

- Increased arterial inflow into corporeal bodies
- Normally blood in cavernous body is desaturated.
- With blood flow, oxygenation of cavernosal nerves and endothelium produces more NO
  - i.e. Hypoxia decreases NO
- NO stimulates cGMP production
  - promotes SMC relaxation

- Infilling of the cavernous body and SMC relaxation:
  - distends penis and occludes venous outflow.
  - Pressure goes from 15 mm to 90 mm
- Eventually, at maximum upward posture, cavernous artery (center) flow ceases.
  - Maximum pressure of 120 is reached.
  - With reduced flow, desaturation takes place -> the thing goes flaccid…

98. Erectile disorder:

- Persistent/repeated inability to achieve erection to perform an intercourse
- At least 3 months
- No ejaculatory disorder

Causes of dysfunction:

Most common underlying mechanism – failure of cavernosal smooth muscle relaxation.

- Vascular (macro AND micro circulation)
- Endocrine – 3-4%
- Metabolic
- Neurogenic
- Psychologic
- Drug induced –
  - Antihypertensive
    - Note ACEI are sparing

To test for vascular insufficiency, try PGE1 injection – if adequate errection is achieved, then vascular supply is ok.

Vascular causes:

- Poor arterial inflow
    - AIOD
    - Steal to external iliac artery
    - Occlusive disease of penile arteries
    - Atheroembolic occlusions
    - Blood pressure effect on arteries (Beta blockers)
- Venous leaks at the cavernosum bodies
    - Trauma to tunica albugenea
    - Congenital leakage

- cavernosum is messed up
    - Fibrosis (post priapism)
    - Peyronie's – deformity invading into SMC
    - Refractory smooth muscle – does not respond to stimulation
        - Hormonal (prolactin, low testosterone,blood pressure med)
        - Metabolic (DM, uremia)

Work up:

- Prolactin, testosterone, glucose, PSA
- PGE1 injection

- **Try oral drugs first,** then intracavernous injection, then vacuume constrictors.
    - Sildenafil is NOT recommended for women
- IF these fail, then invasive testing – angio/venogram. If these fail with additional trial of Drugs/ICI/VC – then try prosthesis.

EMBOLISM, THROMBOSIS & LIMB ISCHEMIA IN GENERAL

99. Causes of arterial occlusion in general:

- embolism
- thrombosis
    - pre-existing occlusive disease
        - ruptured plaque
    - poor inflow:
        - low flow state
        - sluggish blood flow leading to thrombosis
    - bypass/conduit occlusion

        - disease
        - mechanical problem
    - outflow occlusion
        - blood flow backed up leading to thrombosis
    - hypercoagulable state
        - thrombosis in normal vessels
        - congenital, malignancy, post-op, trauma
- trauma & dissection
    - disruption of normal vessel

100. Most common sources of embolism:

- heart
    - ASC heart disease:
        - MI,
        - Arrhythmias
    - Atrial myxoma
    - Valvular heart disease:
        - RF,
        - Degenerative,
        - Congenital,
        - Bacterial,
        - Prosthetic
- artery to artery:
        - aneurysm,
        - atherosclerotic plaque
- Idiopathic
- Paradoxical
    - Patent foramen ovale – up to 25% of population have it

101. Most common sites of embolisation:

- femoral – MC – 35-50%
- popliteal – 2nd MC – 20-30%
- cerebral – 20%
- upper exteremity – 15%
- visceral – 10%

During embolectomy, longitudinal arteriotomy is recommended – if embolectomy fails, may do bypass. Use AIR for #1 and 2 Fogarty – air is more responsive to change in diameter than saline – less chance of endothelial injury.

Livido Reticularis – most common cutaneous sign of microatheroemboism (trash foot and trash can). Symptom wise, can see fatigue and weight loss if atheroembolism is dissiminated.

102. Atheroembolic Renal falure: i.e. parenchimal causes…

- DD
  - ATN due to contrast nephropathy
  - ATN due to ischemia
  - Emtolism
    - Arterio-arterial
    - Cardiac embolism
      - Clot
      - Atrial myxoma
      - SBE, ABE
  - Weird and wonderful:
    - Necrotizing vasculitis
    - Thrombotic thrombocytopenic purpura
    - Antiphospholipid antibody
    - Multiple myeloma

- Lab – unhelpful in general
  - Eosinophilia in atheroembolism
  - ESR, CRP up
  - UA – see urine sediment in ATN (dirty brown cast)
- Important to distinguish contrast vs ischemia:
  - Consider time-frame:
    - Contrast nephropathy/ATN –
      - renal failure within 72h
      - renal failure usually recovers
      - **normal blood pressure. i.e no HTN**

    - Atheroembolism –
      - rise in creatinine may be delayed by a week
      - **refractory hypertension**
      - renal failure mostly non-refersible
      - poor outcome:
      - 1 year mortality 64-81%, due to cardiac, CVA, GI ischemia

Treatment and prevention:

1. Stabilize plaque:
   - Statins
   - Antiplatelets
   - ACEI
   - Platelet infusions?
   - Iloprost? (see Rutherford, does work in some studies)
2. Surgical control of source
   a. Only if medical therapy failed

b. Open surgery vs endo

Arch/thoracic aorta plaque –

- if >5mm thick:
  - 33% annual risk of vascular events vs 7% in control.
- Overall, non-calcified & at least 4 mm plaque are a risk factor for atheroembolism
  - It is suggested to start warfarin on these pts (ACCP, 2001)
    - Better then ASA alone
    - Combined with statin
- Surgery
  - Option for minority only
  - only on highly selected pts, low OR risk, have multiple documented embolic events despite medical therapy

see aorta part of oral in training exams….

103. Causes of arterial thrombosis:

- Atherosclerosis – exposed ruptured plaque
- Low flow:
  - CHF
  - cardiogenic shock
  - hypotension
- vascular graft
  - thrombogenic
  - disease progression in graft (Atherosclerosis)
  - initimal hyperplasia
  - mechanical: kink, valve
- trauma:
  - intimal flap vs spasm vs compression
    - penetrating
    - blunt
    - drug abuse
- hypercoagulable state (see below)
- outflow obstruction:
    - arterial – disease progression, dissection
    - venous – compartment, phlegmasia

104. Etiology of post op acute Leg ischemia post AAA repair:

Most common RECOGNIZABLE cause:

- Raised intimal flap 25%
- Kinking 8%
- Post op hypotension 8%

Causes expanded:

- Thrombosis:
  - limb of Dacron graft
  - diseased iliac, CFA, SFA, profunda
  - pop a. aneurysm

- emboli:
  - thrombus
    - from the heart
    - from proximal vessel with inadequate heparinization
  - dislodged plque (atheroemboli)

To prevent:

1. Heparinize pt prior to clamping
2. Back bleed iliacs prior to proximal clamp removal
3. Flush graft prior to distal clamp removal
4. Liberal use of Fogarty
5. Do not leave OR without checking feet first
6. Do not clamp CIA
   a. Clamp IAA and EIA instead, less chance will trash clot in CIA downstream

Management:

1. Prep both groins
2. Explore index groin:
   a. Check inflow
   b. Check anastomosis
   c. Check SFA/profunda
3. Consider thrombectomy vs fem-fem vs axfem
4. Consider profundoplasty
5. Monitor Hg and consider fasciotomies if > 6 h ischemia

105. Infrainguinal graft thrombosis:

- Early failure rate 5-10% within 30 days
- Bad prognosis even in successfully thrombectemized grafts:
  - At 1 year – 50% amp, 25% RP, 15% died
- Approach:
  - Confirm thrombosis

- Minimize clot propagation (AC)
- Assess neurologic and motor status
- Review indication for bypass (IC vs RP/ulcer)
  - Decide if need to intervene at all
- Decide if graft can be salvaged
- See what conduits are available

Cause:

- Graft thrombogenicity
- hypercoag state
- poor run off
- poor inflow
  - undetected asc disease
  - poor cardiac output
    - dehydration
    - cardiac decompensation

- **in 60% of cases failure is due to the conduit problems**
  - **80% of these is correctable**
- **Technical errors responsible for 4-25% graft failures**
- **At exploration, 50% of grafts have no apparent problems.**

- If decided to salvage, decide surgery vs thrombolysis.
  - Results of both are poor.
    - lysis won't help in pts with DM & recent graft.
  - Prefer surgery for most
- Outcome is better if technical problem (cusp, twist or stensis) is identified.
- See notes on Thrombolysis

106.Ischemia & reperfusion effects on organs:

- Ischemia depletes intracellular energy source:
  - switch to anaerobic metabolism
  - generation of toxic radicals.
  - Adhesion molecules generated – inflammatory cells come in

  - In the end:
    - Organel and cell membranes are disrupted as ionic pumps stop
    - Influx of Ca++ causes cell death and fluid extravasation
    - Transudation of fluid cauase edema and capillary occlusion

  - Reperfusion effects –
    - washes out cell death debrie into circulation –

- high K, urea, acid, myoglobin
  - arrhythmias and renal failure
- brings in oxygen to damaged tissues
  - oxygen free radicals form.
  - radicals overwhelm damaged antioxidant system –
  - cause further damage to healthy, surviving membranes – further cell damage.
    - SIRS and organ dysfunction

- No reflow phenomenon with reperfusion:
  - Mini-compartment syndrome
    - No obvious obstruction
    - No flow through microcapillary bed
  - Unclear etiology.
  - Described in coronary literature ("broken heart syndrome")
  - Several causes seen:
    - spasm of the microcirculation,
    - local platelet activation,
    - microvessel embolization
    - tissue edema

COAGULATION & ANTICOAGULATION

107. Summarize coagulation cascade:

Extrinsic –

- tissue factor activates VII
- VIIa activates small amount of factor X **AND factor IX.**
  - This represents an important feature – CROSS talk to Intrinsic pathway
  - VIIa is quickly inactivated by Tissue Factor Pathway Inhibitor

- Xa (initially activated by VIIa) activates small amount of VIII and V

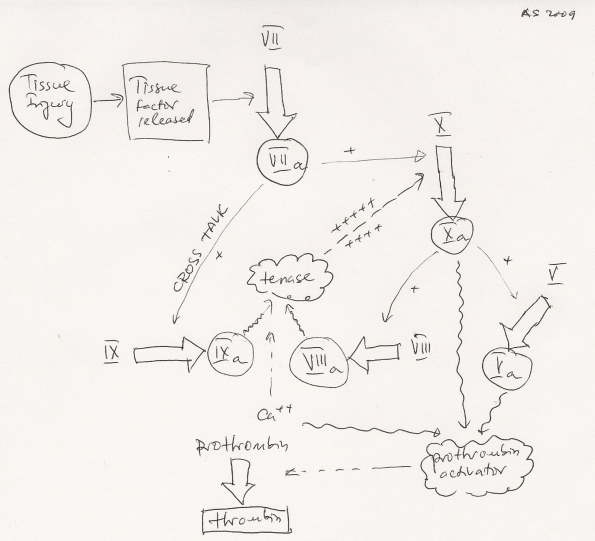
- activated factors IX (from VIIa), VIII (from Xa), and plus Ca^{2+} form tenase complex on the surface of ppt
  - 50 times more active than VIIa.
  - Massive amounts of Xa are formed
- Xa, Va, Ca^{2+} and ppt activate thrombin that converts fibrinogen to fibrin.

Intrinsic: XII->XI->IX…

- Common pathway – X activates II, II creates fibrin

- But the most important source of activated factor X is from tenase action.

Fibin is stabilized by factor XIII



Plasmin inhibitors: procoagulants…

- Plasminogen Activator Inhibitor – 1 (PAI-1)
  - inactivates tPA…
  - excess causes arterial AND venous hypercoagulable state
- Alpha 2 antiplasmin
  - Inhibited by dextran 40

Anticoagulation arm:

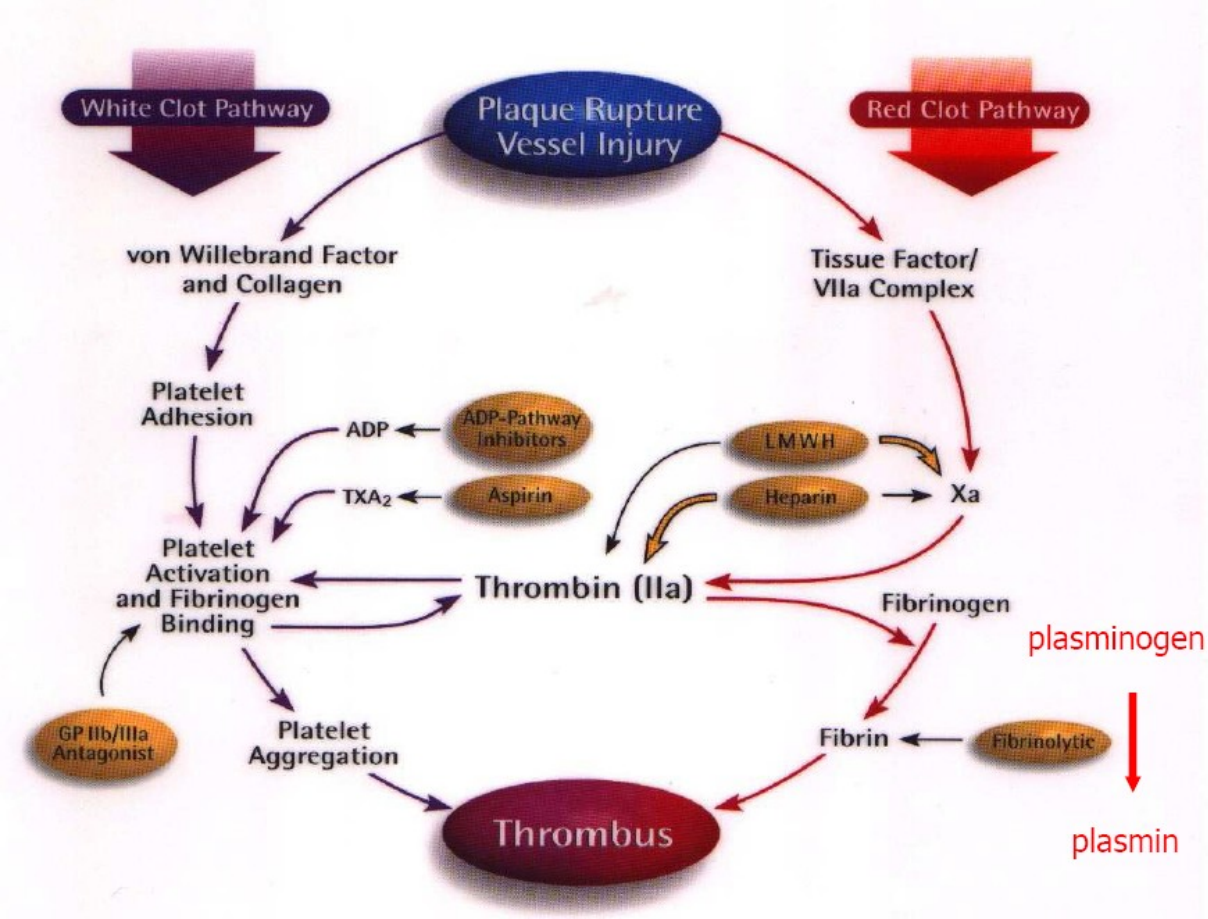
- Plasmin – breaks down fibrin
  - Inhibited by PAI-1 and alpha2-antiplasmin
- Antithrombin III –
  - Always active
  - Action is amplified by the presence of Heparin
  - Most sensitive enzymes to hep-ATIII is factor II
    - Also works vs IX, X, XI,

- Prorein C - > binds thrombin and then thrombomodulin.
  - This activates PnC and, together with pn S, blocks factor V and VIII.
    - Factor VIII is needed for activation of factor X
    - Factor V is needed for activation of factor II
    - **major anticoagulant**
    - **resistance of factor V to APC is MC cause of Hypercoagulable state**
- Thrombomodulin –

- binds thrombin inactivating it.
- it accelerates activation of protein C thousand fold.

- tPA – activator of plasminogen, secreated by endothelium

- Tissue pathway pathway inhibitor (TPPI) - anticoagulant
  - Protein that inactivates VIIa and Xa
  - Most of it is bound by endothelium and can be released by heparin
  - Has anti-inflammatory properties
- Prostocyclin –
  - generates cAMP – reduces platelet aggregation



108. How does Dextran 40 work?

- Polysaccharide
- Increases electronegativity of RBC, plt, wbc, reduces aggregation
- Inhibits alpha-2 antiplasmin
- Reduces factor VIII-VW activity
- Decreases viscocity

- 70% eliminated within 24 h (urine)
- Complications:
  - May aggravate renal failure (diuretic)
  - Anaphylaxis
  - Pulm edema
  - Cerebral edema

109. Warfarin: mechanism of action and complications:

- Interfers with utilization of vit K by the liver during synthesis of X, IX, VII, II (1972).
- Can't synthesize carboxyglutamyl residues for Ca++ binding
- Produced factors are antigenically similar but have abnormal Ca++ binding
- Warfarin-induced skin necrosis
  - Dermal gangrene of the breast, thigh or buttocks
  - Rare
  - Due to transient hypercoag state (pn C&S synthesis suppression)
  - Need to bridge warfarin with heparin
- Bleeding (5% per year)

110. Contraindications to warfarin therapy:

Contraindication	Percent affected
Uncontrolled hypertension (>180/100 mmHg)	14.0
Frequent falls or blackouts	13.2
Inability to comply with treatment	9.8
Daily use of NSAIDs	9.2
GI or urinary bleeding in last six months	1.0

Exclusion criteria used in the major intervention trials of anticoagulation for patients with atrial fibrillation

1. Bleeding disorder or abnormal coagulation at baseline
2. Uncontrolled hypertension (> 180/100 mmHg)
3. Active bleeding
4. Haemorrhagic retinopathy
5. History of intracranial haemorrhage
6. Use of non-steroidal anti-inflammatory drugs
7. Chronic alcohol abuse
8. Risk of gastrointestinal bleeding (active peptic ulcer disease, positive faecal occult blood testing, known oesophageal varices)
9. Planned surgery or invasive procedure
10. Pregnancy or breastfeeding

11. Psychiatric disorder or dementia
12. Expected poor compliance
13. Limited life expectancy
14. Significant renal dysfunction (creatinine > 0.25 mmol/L)
15. Platelet count < 100 x 109/L
16. Other: Recent stroke or transient ischaemic attack (previous two years)

Patients were also excluded if they refused to participate or if their doctor considered the risk of anticoagulation was too great.

Harm:benefit analysis in prescribing warfarin:

- **The risk of major bleeding in patient with AF treated with warfarin:**
  - **1-4% per year,**
  - **with an intracranial bleeding rate of 0.2-0.5% per year**.
  - The fatality rate mirrored the intracranial bleeding rate.
- In observational studies of ambulatory patients the risk of major bleeding is 4-9% per annum.
- Major determinants of warfarin-induced bleeding:
  - intensity of anticoagulation,
  - patient characteristics,
  - the concomitant use of drugs that interfere with haemostasis
  - the length of therapy

Intensity of anticoagulation and duration of therapy

- The risk of bleeding increases when INR exceeds 4.0.
- INR > 4.0 the most important risk factor for intracranial haemorrhage,
- independent of the indication for warfarin.
- **The risk of major bleeding:**
  - **greatest in the first month of therapy (3%)**
  - **decreases with time to 0.8% per month for the remainder of the first year and to 0.3% per month thereafter.**

AF and age:

- **Framingham study: the incidence of stroke due to AF increased with age:**
  - **1.5% for 50-59 years**
  - **23.5% for those aged 80-89 years.**
- The prevalence of AF in pts 80 years old is10%.
- Advanced age is not itself a contraindication to warfarin.
- Studies AF support the ongoing benefit of anticoagulation with increasing age.

- **Warfarin therapy reduces the risk of ischaemic stroke in patients with non-rheumatic AF from 7.4% to 2.3% per year.**
- Age is, however, a RF for more unstable prothrombin time results.
    - For every 10-year increase in age there is a 15% increase in the risk of anticoagulation having to be suspended because of a raised INR.

http://www.australianprescriber.com/magazine/27/4/88/92/#t1

111. What drugs affect warfarin:

- **Potentiate/augment**

- Allopurinol
- Aminoglycoside
- Amiodarone
- Oral hypoglycemics
- Acetomenophen
- Cipro
- Cimetidine
- Erythromycin
- Fluconazole
- Isoniazide
- Metronidazole
- Omeprasole
- Phenytoine
- Propranalol
- Tetracycline
- Alcohol in liver disease

Remember these: hypoglycemic, Aminoglyoside, cipro, metronidazole, omerazole AUGMENT

- **Inhibit:**

- Anti-histamines
- Azathioprine
- Barbiturates
- Carbamazepine
- haldol
- Cyclosporine
- spironolactone
- Rifampin
- Sucralfate
- Vit K

Remember these: Haldol, spironolactone, cyclosporin, barbiturates INHIBIT

80. Heparin vs LMWH:

Heparin: 4000-40,000 Da,

- polysaccharide from pork/beef lung,
- how it works:
    - increases affinity of antithrombin to thrombin & binds thrombin directly;
    - inactivates platelets.
    - Releases Tissue Factor Pathway inhibitor
    - Releases endothelial TPA
- Activity vs X and II – 1:1 ratio,

- half life 60 min, some say 90 min
- cleared by endothelium and macrophage binding,
  - renal if high dose of heparin is given,
- more chance of platelet inhibition compared with LMWH,
- reversal with protamine,
- can be given iv and sc,
- monitor anti Xa, PTT, ACT,
- more bleeding and HIT complications – HIT seen in about 5% risk

Low Molecular Weight Heparin – 4000-8000 Da,

- enzymatic depolymerization of UFH,
- activity vs X and II in 2-4:1 ratio,
- like heparin, releases Tissue Factor Pathway Inhibitor,
- half life 6 h, renal clearance,
- less ptt inhibition, only sc administration,
- no need to monitor,
- less bleeding and HIT complications- HIT seen in about 0.5%

112.Protamine mechanism of action:

- Cation that binds heparin 1:1 ratio
- Restores ATIII to its' inactive state

113.Direct Thrombin inhibitors:

Lipirudin , Argatroban, hirudin. Inhibit Thrombin directly. Can't be reversed.

Argatroban:

- Ask for hematology consult
- Half life – 45 min, onset in 30 min, max effect in 1-2 hours
- Give infusion of 2 mcg/kg/min
- Keep aPTT at 1.5.-2.5
- Can't reverse

DISORDERS OF COAGULATION

114. HIT:

- > 50% ppt drop OR ppt <100,000 OR resistance to heparin with thrombotic complications while on heparin
- Can be seen as a laboratory finding (just HIT – heparin induced thrombocytopenia) or a clinical finding with thrombosis (HITT – heparin induced thrombotic thrombocytopenia)
- Platelet drop in 5 % of pts on heparin for > 5 dyas
- Majority with ongoing heparin use

- More chance with UFH then LMWH
- Type 1 – non immune related, inconsequential
- Type 2 – Ig G vs PF4
- Ds:
  - platelet aggregation,
  - serotonin release assay,
  - ELISA (for Ig G and PF4-heparin complex)
- Alternative to heparin: lepirudin, argotroban, danaparoid.

115. Hypercoagulable state:

- High risk of thrombosis:
  - Antithrombin deficiency
  - Pn C& S def
  - HIT
  - Antiphospholipid

- Lower risk of thrombosis:
  - Factor V Leiden
  - Hyperhomocystenemia
  - Prothrombin 20210 polymorphysm

Another way to classify hypercoagulable state:

Arterial:

- Platelet abnormalities
- hyperfibrinogenemia
- lipoprotein (a)
- atherosclerosis

BOTH venous and arterial:

- hyperhomocysteinemia
- HIT
- elevated PAI-1
- antiphospholipid AB:
  - cardiolipin/lupus anticoagulant

Venous:

- Factor V leiden
- pn 20210 A polymophysm
- pn C & S
- antithrombin
- dysfibrinogenemia

116. Antiphospholipid antibodies: -LAC, ACL and SLE:

- IG directed vs phospholipids
- Family of pns, one of them is LE (lupus anticoagulant)
- They have both pro coagulant and anticoagulant activity -
    - In vitro, they prevent coagulation factors from interacting, slowing coagulation (PTT prolongation)
    - In vivo – they do not inhibit any coagulation activity, but encourage it through poorly understood mechanisms –
        - endothelial damage,
        - inhibition of prostocyclin secretion,
        - interference with fibrinolysis.

- SLE can be associate with
    - Systemic arterial/venous thrombosis
    - Recurrent abortion
    - Neurologic disease

- Lupus anticoagulant & anticardiolipin antibody seen in association with SLE:
    - **LAC – 34% (vs 2% in general population)**
        - **6 fold risk of DVT**
    - **ACL – 44% (vs up to 7% in general population)**
        - **2 fold risk of DVT**
        - Risk of arterial thrombosis is 25%...

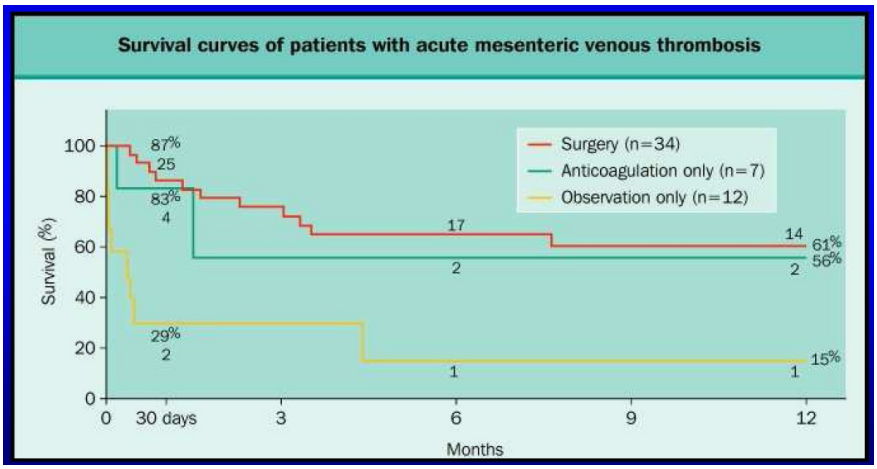
117. Resistance to activated factor V:

- Most common abnormality associated with VTE
- Resistance to inactivation of factor V by activated protein C
- Hence, activation of II is not inhibited
- Clinical effects depend on wether two (homo) or one (heterozygous) copies of the mutated gene are present
    - Heterogenous – 7 fold increase of thrombosis,
    - homo – 80 fold

118. Causes of mesenteric thrombosis:

- Idiopathic – most common
- Hypercoag state (dehydration aggravates this)
- Venous congenstion
    - CHF
    - portal hypertension
- injury:

- trauma
- post surgery
- inflammation (IBD, pancreatic)
- infection (sepsis, abscess, peritonitis)



86. Common acquired causes of bleeding and their treatment:

- Heparin use –
  - anti II via AT III, TFPI, tpa, platelets
  - elevated PT/PTT/ACT,
  - use protamine
- Argatroban/hirudin –
  - direct anti II,
  - high PT/PTT,
  - use FFP
- warfarin, liver failure, malnutrition, biliary obstruction:
  - lack of X, IX, VII, II (1972)
  - elevated PT only
  - use FFP/vit K, octaplex
- dilution
  - fewer molecules or cell membranes around
  - replace missing substances
- bone marrow failure
  - thrombocytopenia
  - ID by bone marrow bx
  - platelet transfusion
- acidosis/hypothermia
  - diminished enzyme/platelet function
  - correct cause/warm up

- DIC
    - Global activation of entire clotting
    - Consumptive coagulopathy
    - Correct cause, replace factors
    - Do NOT give antifibrionlytics
- Thrombolytic therapy
    - Reduced fibrinogen, clot lysis
    - Elevated FDP, ECLT
    - Give cryoprecipitate
- Primary fibrinolysis
    - Reduced fibrinogen, clost lysis
    - Elevated FDP, ECLT
    - Give antifibrinolytics (gamma amino caproic acid)
- Uremia
    - Impaired platelet/endothelium function
    - Lengthened bleeding time
    - dDAVP
- asprin/GP IIb/IIIa use
    - permanent platelet dysfunction
    - lengthened Bleeding time
    - platelet transfusion
- specific inhibitor
    - antifactor, usually VIII
    - seen as resistance to factor replacement

give higher dose, immunosuppression

THROMBOLYSIS

119. Types of thrombolytics:

Streptokinase – rarely used, does not directly activates plasminogen, need to form activator complex first.

Urokinase: directly activates plasminogen – both circulating AND bound to fibrin

- CDT with infusion within the thrombus
- Dose:
    - 250,000 IU lacing bolus sprayed into clot
    - then infusion 4000 IU/min fo 4 h, then 2000 IU/min for up to 36 h
- Re-evaluated pts at 6-12 hours

tPA – activates fibrin bound plasminogen ONLY thus limiting systemic effects

- CDT
- loading dose is 2-5 mg
  - May give up to 3 times at 5-10 min interval,
  - then infusion at 0.05 mg/kg/h – i.e. 3.5 mg/h for 70 kg pt.
- For all thrombolytics monitor fibrinogen
- Complication of thrombolysis:
  - puncture site hemorrhage
  - distal embolization
  - pericatheter thrombosis
  - intracranial hemorrhage (<1 %)

alteplase – like tPA, but higher affinity for fibrin and longer half-life

120. Contraindications to thrombolysis:

- Absolute
  - CV event (including TIA) in past 2 months
  - Intracranial trauma (e.g. neurosurgery) in past 3 months
  - active bleeding
  - GI bleed in past 10 days
- Relative major
  - Trauma & surgery:
    - major non-vascular trauma or surgery in past 10 days
    - CPR in past 10 days
    - recent eye surgery
    - puncture of uncompressible vessel
  - uncontrolled HTN (>180/110)
  - IC tumor
- Relative minor
  - hepatic failure with coagulopathy
  - bacterial endocarditis
  - pregnancy
  - diabetic hemorrhagic retinopathy

121.Thrombolysis studies summary:

Rochester trial: surgery vs thrombolysis

- 114 pt randomized
- Same limb salvage at 1 month
- Same survival at 30 days
  - but worse survival for surgery at 1 year

STILE trial:

- surgery vs thrombolysis for ischemic LE.
- Initially pts were not randomized according to duration of ischemia
- all analysis was done after the trial was completed on subsets of pt population
    - hence, source of bias…

    - Acute (<14 days since onset) at 30 days
        - Overall, no difference,
            - **except need for amputation after failure of surgery**
        - Surgery had more amputation but it is non-significant…

	Lysis	Surgery	
Amputation	6%	17%	P=0.06, NS
Amp after failure	**30%**	**68%**	P=0.01
Bleeding	6%	0%	
Mortality	5%	5%	

    - Chronic (>14 days since onset) at 30 days
        - Same mortality (4%) and amptation (4%)
        - **For very high risk pts, survival at 1 year was better in lysis compared to surgery (7% vs 32%)**

    - Native vessels: 237 pts
        - Less amputation and ischemia at 1 year with surgery

	Lysis	Surgery
Ischemia @ 1 year	64%	35%
Amputation @ 1 year	10%	0%

    - Prosthetic:
        - Acute: lysis better than surgery

	Lysis	Surgery
Amputation 1year	20%	48%

        - Chronic:
            - Same rates of amputation for surgery and lysis

    - Topaz:
        - Doze phase – 4000 u/min followed by …
        - RCT: same limb salvage, same survival, less surgery in thrombolytic group

Thrombolysis trials conclusion:

    - 1 year mortality is 10-20%

- Lysis has intracranial bleeding rate of up to 2%,
  - All bleeding complications - 5%
  - With tPA less…

- **In acute setting vs chronic setting, lysis is equivalent to surgery wrt mortality and limb salvage with several caveats:**

  - **Lysis has advantage:**

    - In acute setting,
      - less amputations if procedure fails
      - better for prosthetic occlusion
    - In chronic setting:
      - May have long term advantage for very high risk pts

  - **Native artery thrombosis is better addressed through surgery**

For vein grarts, best results with thrombolytics are achieved for pts without DM and in late failures as opposed to early.

Because thrombolysis trials did not demonstrate SUPERIORITY of thrombolysis, it is not clear which methods should preferentially be used in ALI. Overall, in practice surgery is preferred.

122.Intraoperative thrombolysis: how to…

- Important part of thrombectomy
- Camerota RCT of urokinase showed that intraop route is safe, no plasminogen depletion, and lower mortality compared to placebo
- After thrombectomy is done – do angio.
  - If clot is gone – occlude artery and bolus IA lytic in distal bed
    - 2-8 mg tPA or 100,000-250,000 urokinase
  - Incomplete thrombus – either repeat bolus or restore perfusion and start IA drip with catheter
  - Extensive residual thrombus –
    - Isolated limp perfusion
      - Exsanguinate limb
      - 250 mm blood pressure cuff to thigh
      - Canulate pop vein with red rubber cath
      - Canulate AT and TPT
      - Infuse 500,000 uro or 50 mg tpa into each artery in 500 cc of NS over 20 min
      - Drain vein, flush out with another 1 L NS/heparin
      - Restore circulation

- Can keep cuff for up to 1 h
- Most of plasminogen activator escapes via vein. Some will go into bone marrow ->systemic

Angiojet :

- Reduced incidence of distal embolisation
- No prospective study surgery vs mechanical device is available
- Retrospective study:
  - better survival, better patency, same amp free survival

	angiojet	Surgery
4/12 patency	78%	67% p=0.017
6/12 survival	88%	75% p=0.02
Amp free survival 12/12	77%	61% p=0.07

DVT

123.Diagnostic criteria for DVT: CDF…

Acute – less than 2 weeks
Subacute – between 2 weeks and 6 months
Chronic – more than 6 months

- C =Venous incompressibility
  - Most important criterion, the rest are supportive
- D= distended large vein in ACUTE setting
  - Thrombus visualization – echolucent
  - Vein is contracted and clot is heterogenious in CHRONIC DVt
- F=Absent or diminished spontaneous flow
  - Absence of respiratory phasicity
    - inspiration augments UE flow, decreases LE
  - Absent or incomplete color filling of lumen
- Additional:
  - Compare with contralateral side
  - <50% diameter increase with Valsalva (i.e. no response to proximal obstruction)
  - immobile venous valves

124. RF and outcomes for DVT:

- 80% of PE are clinically silent

- VQ has high false positive rate

Rutherford:

DVT – seen in 20% of pts undergoing Gen surgical procedure
Fatal PE:

- Gen surgery – 0.7% without prophylaxis
  - Down to 0.1% with heparin
- Elective hip – 2-3%
- Hip fracture – 4-7%

Rutherford p. 2136

- After DVT, veins WILL recanalize
- Rate and recurrence of DVT will determine viability of valves
  - And incidence of CVI and PTS
- In 2/3, valves will be damaged and will lead to post-thrombotic sequellae
- Histological evidence:
  - Thrombus organization rarely involves valve cusp
  - Clear zone around valve due to endothelial fibrinolytic activity
- 25 year study of Mohr: 1500 pts

Time	CVI incidence
1	7%
5	14%
10	20%
20	25%
Venous Ulcers at 20 years	Surpising 3%

- However, proximal obstruction will increase risk of CVI
- 95% of pts with iliofemoral thrombosis treated by AC alone have ambulatory venous hypertension at 5 years
  - 90% have symptoms of CVI
  - 15% have ulcers
  - 15% debilitating venous claudication
- Argument to clear iliofemoral clot burden
  - Either with CDT or open thrombectomy
  - See iliofemoral thrombosis notes

RF for DVT:

- Age : 40 yoa
- Immobility
  - hospitalization

        - neurologic/paralysis
        - travel
    - Trauma
        - surgery – depending on type
            - neuro/ortho> general>ENT
        - trauma
    - Venous htn
        - CHF
        - venous insufficiency
    - Hypercoagulable state
        - OCP
        - fam hx
        - see HCS
        - malignancy
        - pregnancy
    - Systemic inflammatory state
        - SLE
        - IBD

Low risk

1) **< 40** yrs age
2) **None of the risk factors listed**
3) General anesthesia for **< 30 minutes**
4) Minor elective, abdominal, or thoracic surgery.

Without prophylaxis proximal **DVT risk < 1.0 %**

Fatal PE < 0.01 %

Prophylaxis - **early ambulation**

Moderate risk

1) **> 40** yrs
2) General anesthesia **> 30 minutes**
3) 1 or more risk factors

- **DVT 2 - 10 %**
- Fatal PE 0.1 - 0.7 %
- Prophylaxis – LDUH 5000u Bid or Tid, **OR** intermittent compression until ambulation

High risk

1) **> 40** years
2) Surgery for **malignancy** or an **orthopedic procedure**
3) General anesthesia > 30 minutes
4) Have an **inhibitor deficiency state** or **other risk factors**

- Proximal DVT **10 - 20 %**
- Fatal PE 1.0 - 5.0 %
- Prophylaxis – **LMWH od <u>AND</u>** IC until ambulation

- Dr. Wells (University of Ottawa), et al: Lancet 1997; 350: 1795-1798

- active cancer (ongoing treatment/diagnosed within 6 months or palliative care)- score 1
- paresis, paralysis or recent plaster cast immobilisation of lower extremity- score 1
- recently bedridden for more than 3 days and/or major surgery within 4 weeks- score 1
- localised tenderness over distribution of deep veins- score1
- entire leg swollen- score1
- calf swelling more than 3 cm compared with asymptomatic side, measured at 10 cm below tibial tubercle- score 1
- pitting oedema (greater in symptomatic leg)- score 1
- collateral superficial veins (non-varicose)- score 1
- alternative diagnosis as likely or greater than that of DVT- score SUBTRACT 2

In patients with symptoms in both legs, the most symptomatic leg is used
score 0 or less- low risk (3% probability DVT)
score 1 or 2- moderate risk (17% probability DVT)
score 3 or more- high risk (75% probability DVT)

Categories of Risk for Venous Thromboembolism in Surgical Patients†

Low risk:
Minor surgery in patients <40 years of age with no additional risk factors present*
- Risk of calf DVT: 2 percent
- Risk of proximal DVT: 0.4 percent
- Risk of clinical PE: 0.2 percent

Moderate risk:
Minor surgery in patients with additional risk factor present*, **or**
Nonmajor surgery in patients aged 40–60 with no additional risk factor, **or**
Major surgery in patients <40 with no additional risk factors
- Risk of calf DVT: 10–20 percent
- Risk of proximal DVT: 2–4 percent
- Risk of clinical PE: 1–2 percent

High risk:
Nonmajor surgery in patients >60 or with additional risk factor present*, **or**
Major surgery in patients >40 or with additional risk factor
- Risk of calf DVT: 20–40 percent
- Risk of proximal DVT: 4–8 percent
- Risk of clinical PE: 2–4 percent

Highest risk:
Major surgery in patients >40 with additional risk factor present*, **or**
Hip or knee arthroplasty, hip fracture surgery, **or**
Major trauma, spinal cord injury
- Risk of calf DVT: 40–80 percent
- Risk of proximal DVT: 10–20 percent
- Risk of clinical PE: 4–10 percent

DVT: deep vein thrombosis; PE: pulmonary embolism

* **Additional risk factors** include one or more of the following: advanced age, prior venous thromboembolism, obesity, heart failure, paralysis, or presence of a molecular hypercoagulable state (eg, protein C deficiency, factor V Leiden).

† Revised from Geerts, WH, et al. Chest 2001; 119:132S.

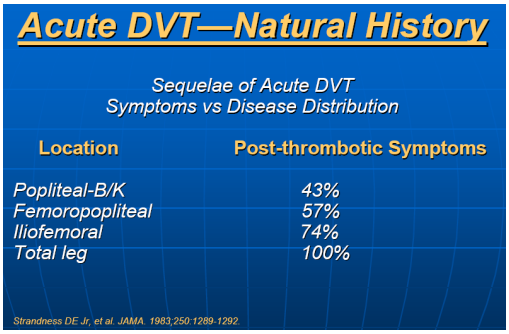
125.D-dimer in diagosis of DVT:

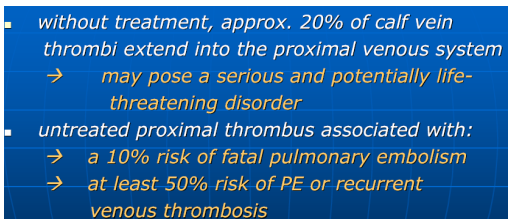
- Measure D-dimer in outpatients with low pretest clinical probability
- Product of fibrin degradation
  - Low pretest probability risk pts – NPV 99%,
  - high pretest probability risk patients – NPV 35%
- If pretest probability is low, order D-dimer.
  - If it's low, you've ruled out DVT
- If Pretest Prob or D-Dimer is high – need to order duplex
  - One negative duplex rules out DVT
  - Cost of first follow up duplex is 390,000$ per life saved
  - Cost of second follow up duplex is 3.5 mln $ per life saved

126.Ways to treat DVT:

- Elevation/bed rest +/- AC
- AC alone

- AC + IVC interruption (filter)
- CDT
  - Will decrease post thrombotic sequellae of iliofemoral dvt, NOT fem-pop (large venous registry)
  - 2 small RCT did not show difference with AC for fem-pop
  - Great for acute DVT (<10 days)
  - Large RTC is needed – pending TOLEDO
  - If fails – PC mechanical thrombectomy
- Surgical thrombectomy - for ILIOFEMORAL DVT
  - For active ambulatory pts OR **to prevent** venous gangrene in palliative pt
  - Swedish RCT – 10 year patency 80% vs 30 % (AC alone)
  - Much better patency and valve function compared to AC alone
- Surgical bypass if IVC/Iliac occlusion AND sxs





Treatment:

- Heparin->warfarin
- Enoxoparin (1mg/kg bid or 1.5mg/kg od)->warfarin

How long:

- Above knee DVT, no PE –
  - 3 months treatment
  - Elevate leg, compression hose, may return to work in 2 weeks
  - Duplex at 72h, 1 month, 6 months
  - Multiple follow up duplex is NOT cost effective…
- Above knee DVT with PE or recurrent:
  - 6 month treatment
  - 3rd episode – life time AC

Below knee DVT – some say follow with Duplex, some say treat… I'd treat for fear of 20% progression. Alternatively, may follow with serial Doppler US in 1 week. The only DVT that is safe not to treat is that of soleal sinuses and observe

Most common vein to thrombose – peroneal, least common – anterior tibial.

127.Indications for IVC filter:

- Absolute:
  - Need to anticoagulate but pt has contraindications
  - Need to anticoagulate but developed complications with AC
  - Post pulmonary embolectomy
  - prev caval interruption (ligation or filter) failed
- Relative:
  - Floating Iliofem thrombus
  - Propagating iliofemoral thrombus while on AC
  - Septic PE
  - Chronic PE in pt with Pulmonary hypertension and core pulmonale
  - Pt with > 50% pulm vessel occlusion who can't afford to lose more pulm function with recurrent pulmonary emb
  - high risk of falls – as in severe ataxia (can't anticoagulate)

aside:

- PREPIC
- 400 pt with proximal DVT
- All are AC'd, half have IVC filter put in
- At 12 days more PE in non IVC group 4% vs 1%
- However, there was NO difference in Morbidity or mortality related to PE at 2 years
- There is increased risk of subsequent DVT if have IVC filter in place
  - 10% vs 20%
  - Still, no role for prophylactic anticoagulation if have filter in place
- Are IVC filters allTHAT necessary?
- Hence argument for retrievable filter…
- Temporary filter – have wire/sheath attachment
- Retrievable filter – like regular filter but can be removed at a later date
- Cancer pts have very high rate of DVT and PE

128.Complications of IVC filter:

- @ placement:
  - Access bleeding/arterial injury/pseudoaneurysm
  - IVC/SVC injury
  - Incomplete deployment
  - Misplacement
  - Renal contrast load
- While in place:
  - Migration
  - Obstruction with clot
  - Infection
  - Recurrent PE

- @ removal:
    - Access bleeding/arterial injury/pseudoaneurysm
    - Failure to retrieve
    - Renal contrast load
    - injury to SVC/IVC

129.Migratory phlebitis:

- Seen in pancreatic ca, buerger disease, behcet's disease, PAN.
- Superficial phlebitis has 20% risk of PE – NOT a benign entity

130.Effort thrombosis: classify

Frequency:

- Primary – effort – 25%
- Secondary –
    - Central lines 40%,
    - infection, prev DVT, UE AVF, hypercoag state, trauma – 35%

path:

- venous HTN, related to obstruction, rather than reflux
- AC makes no difference on degree of disability
- PE incidence – 7%

- Primary –
    - Sometimes see anatomic defect in TO
    - young males
    - dominant extremity in 75%
    - 75% report strenuous repeated activity prior to onset
- Secondary –
    - CV catheter MC, malignancy, infection, trauma, thrombocytosis
    - to treat, just remove catheter, consider AC only…

131.Treatment options for primary axillary vein thrombosis:

Observe/elevate/AC vs **intervene**

Treatment options for primary axillary vein thrombosis:

1. AC, and wait for the clot to go away, vein to recanalize
2. Catheter Directed Thrombolysis, to actively open up vein
3. Thoracic outlet decompression if external compression demonstrated,
4. Open Angioplasty vs stent for intrinsic residual stenosis in the vein,
5. surgical bypass for failed thrombolysis AND disabling symptoms.

Confusion and variation begin when you factor in timing and order of these interventions… Here is the above list with timing considerations:

A. CONSERVATIVE approach:

- Arm Elevation/Rest alone
    - not practical for most active patients
    - for high risk only with minimal function and AC Contraindications
- **Anticoagulation/arm elevation/rest alone**
    - **Preferred approach of most of conservative hematologists**
    - **Poor functional results according to Rutherford…**

- Please note, that AC alone makes no difference on degree of future disability – i.e. it does not improve function much.
- PE incidence – 7%

B. INTERVENTIONAL approach:

- open up vein with CDT (most commonly) vs open surgical (not common as a stand alone – unless combine with immediate TOD and open venoplasty)

Questions arise as to WHEN to do TOD if such is diagnosed:

- open up vein with CDT and/OR anticoagulate for 3 months, then venogram
    - **if external compression alone, then TOD. Most common scenario.**
    - If internal defect alone, open venoplasty vs balloon/stent
    - if normal venogram – 3 months of AC/AE/R.
        - Least common scenario.
        - Then decide on stopping AC vs continuing…

Adjuncts for vein defects:

- for short stenotic segment
    a. open venoplasty
    b. endovascular plasty +/- stenting
- for long stenotic segment
    a. trial of AC/AE/R. If fail, then consider venous bypass or IJ turndown

TOD:

- On exam, **the classic answer would be to lyse (if no contraindications), keep AC for 3 months, then bring for delayed TOD if indicated by venogram.**
- MAKE sure you let the pt choose and review complications of thrombolysis (IC hemorrhage) as well as contraindications to thrombolysis.

132.Iliofemoral Venous thrombosis:

- Aggressive approach –
    - high rate of sig post thrombotic sequellae that are difficult to manage conservatively
    - early relief of obstruction may prevent such sequellae.
    - Phlegmasia cerulean dollens – obstruction of ALL venous outflow.
    - Phlegmasia alba dollens – obstruction of only MAJOR venous outflow, with patent superficial outflow.
        - Alba – because of concomitant arterial vasospasm.
- Iliofemoral thrombectomy:
    - anticoagulate
    - CT first to assess caval involvement
    - +/- IVC filter
    - GA, 10 mm PEEP to decrease PE chance
    - Fascitomy first if phlegmasia cerulean dollens
    - GSV junction dissection, CFV to inguinal lig
    - Longitudinal venotomy of CFV
    - Fogarty proximal, esmark distal extremity
    - GSV to SFA anastomosis, close in 6 weeks
        - Diameter fistula/artery ratio 1/3
        - No more then 300 ml/min flow
    - Completion venogram – A MUST
        - If iliac v. stenosis – plasty/stent
    - Keep anticoagulated

CHRONIC VENOUS INSUFFICIENCY

133.Venous flow characteristics:

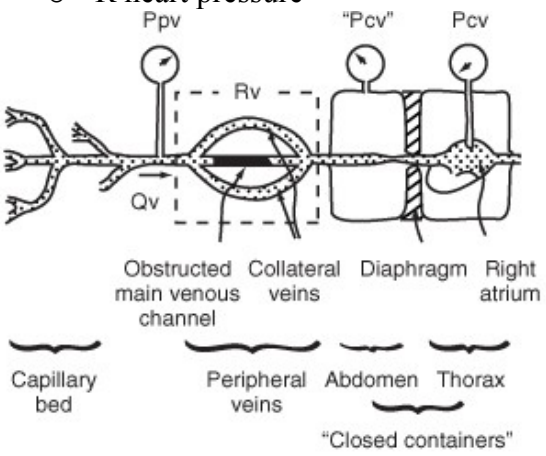
- phasic
    - if continuous
        - suspect proximal obstruction
    - if pulsatile
        - suspect AVF or Fluid overload (R CHF, venous HTN)
- unidirectional
- spontaneous
- responds to inspiration and expiration/Valsalva

- Upper extremity: UP with inspiration and DOWN with expiration
  - i.e. augmented by negative pressure of the chest
- Lower extremity: DOWN with inspiration and UP with expiration
  - i.e. augmented by negative pressure in the abdomen

134. Determinants of venous flow:

From distal to proximal…

- Arterial inflow
- Body position
- Calf muscle activity
- valves
- Peripheral resistance
- Abundance of collaterals
- Intraabodminal and intrathoracic pressure
  - Phase of respiratory cycle
- R heart pressure



135. Cause of Chronic venous insufficiency:

- Venous stasis
  - Due to valve/endothelial damage, stasis leads to nutritional deprivation of skin/tissue.
  - However, no changes in O2 sat shown…
- AV fistula theory
  - Hypothesized arteriovenous connections
  - Increased shunted flow
  - Not confirmed objectively

- Fibrin cuff: diffusion block
    - Cuff on precapillary vessels, acting as diffusion barrier.
    - Not true

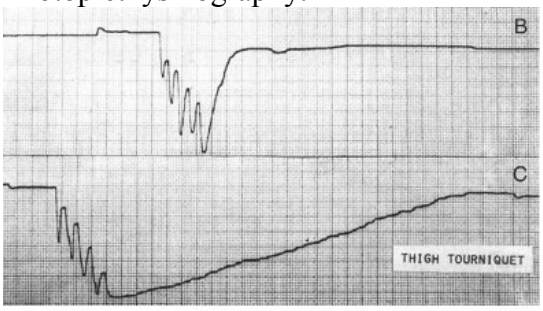
- Leukocyte trapping:
    - Accepted theory.
    - Activation of neutrophils in venous microcirculation.
        - leads to degranulation, endothelial damage.
    - affects diffusion of nutrient and oxygen,
    - results in ulcers and skin damage.

136. Wave forms of venous plethysmography in severe SFJ reflux:

Ambulatory venous pressure:

- AVP – pressure in the dorsal pedal vein after 10 calf contractions
    - <40 – low risk of ulcer
    - >80 – 80% chance of ulceration
    - Return to baseline in Normal should be > 30 sec
        - i.e. DELAYED refilling is NORMAL
- PPG and APG are non-invasive substitutes of AVP

Photoplethysmography:



B – abnormal, VRT (venous refilling time) is short
C – normal

Baseline –

- pt standing,
- followed by 10 tip-toe calf contractions –
    - empties the leg vein
- return of venous blood is documented
    - either by strain-gauge
    - photopletysmography…

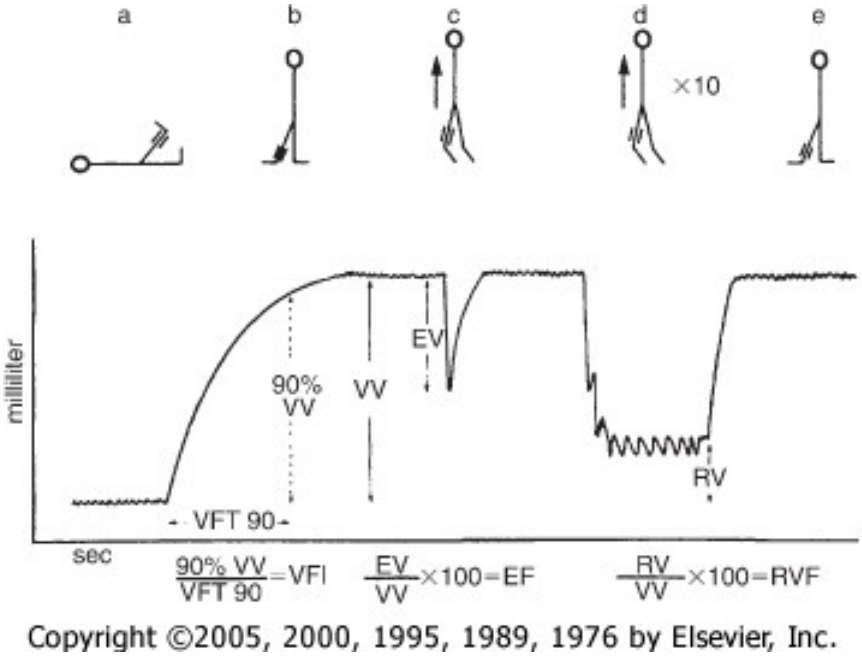
Photo Plethysmography –

- PPG uses a transducer that emits infrared light from a light-emitting diode into the dermis.
- The backscattered light is measured by an adjacent photodetector
  - displayed as a line tracing.
- As blood fills in, it absorbs the light
  - This returns curve to baseline…
  - The faster the pooling, the faster the return to baseline

- Rapid return to baseline is characteristic of venous insufficiency.

Air plethysmography:

- Unlike PPG, it samples large calf volume.



Air plethysmograph data obtained from tracings. VV, venous volume; VFI, venous filling index; EF, ejection fraction; RVF, residual volume fraction; EV, ejected volume; RV, residual volume; VFT, venous filling time.

Lie down, then stand up, then empty veins with single contraction (EF), then empty veins with multiple contractions.

- Normal VV is 80-150 ml
- Residual volume is <35%

- **VFI - < 2 ml/sec,**
    - **if reflux it is > 30 ml/sec**
        - **Ziebel says 7 ml/sec**
    - i.e. curve is steeper with CVI

- EF – ejection fraction – after single tip-toe – **normal > 60%**
- Some use TIME to refill alone – **should be less than 25 sec to diagnose insufficiency**

Note , do not confuse VFI (normal < 30 ml/sec, some say 20…) and time to return to baseline (normal > 30 sec)

- Can't do PG on pts who can't stand unassisted or can't do tiptoe.

Duplex of SFJ reflux:

- Do supine exam for DVT
    - May do Valsalva –
    - less than 2 sec reversal of flow at SFJ should be seen when pt supine
- Stand up, cuff to lower thigh, Doppler at SFJ
- Inflate cuff, noting disappearance of prograde flow
- Rapidly deflate cuff – noting reversal of flow at SFJ.
    - N <0.5 sec of reflux, if more – reflux.

137. Venous disease assessment: CEAP classification

- Venous hypertension causes extravastion of albumin and RBC.
    - RBC can't re-enter circulation – break down and products of their decomposion stimulate vicious inflammatory reaction.
    - WBC migrate in
    - through TGF-β-1, fibroblast creat intense fibrotic reaction – UNIQUE to CVI. In the end, fibrosed inverted bottle neck legs are formed.
- Varicous veins – with venous HTN, SMC transform form contractile to secrotory subtype.
- Traditional thinking –
    - primary CVI – thickened vein, normal valve
    - Secondary CVI – thickened valve, normal vein
    - In reality, see elements of both - i.e. both vein and valve may be abnormal…

    - C – clinical signs, add A (asymptomatic) or S (symptomatic)
        - 1. Telagectasia, reticular veins, malelar flair. Diameter < 3 mm
            - These do not need duplex study
        - 2. Varicose veins – i.e. diameter > 3 mm
            - These need duplex study
        - 3. Edema, no other changes
        - 4. **Skin changes – pigmentation, lipodermatosclerosis**

        - 5. Skin changes + healed ulcers
        - 6. Skin changes + active ulcers
    - E – etiology
        - C – congenital, since birth
        - P – primary, idiopathic
        - S – secondary
    - A – anatomy
        - S – superficial
        - D – deep
        - P – perforators
    - P – pathophysiology
        - R – reflux
        - O- obstruction
        - R,O – both

Venous clinical severity score: validated 30 point tool to assess disease.

138.Approach to venous ulcers:

- Confirm venous etiology
    - History/exam/duplex – GSV/deep/perf incompetence
    - On hx:
        - Varicosity (since birth vs EVENT)
        - DVT/pregnancy/vein tx/harvest
        - Occupation
        - OCP/fam hx
        - Race
            - African American – perimalelar ulcer – think sickle cell

- R/o arterial/neuropathic/infection contribution
- Heal ulcer conservatively
    - Elevation, Compression, infection treatment
- If does not heal after 3 months, r/o malignancy and consider an intervention
    - Indication
        - bleeding
        - Pain
            - Aching pain
            - Leg heaviness
            - Easy leg fatigue
        - Superficial thrombophlebitis
        - cosmesis
        - CEAP 4 - 6
            - Ulcer
            - External bleeding
            - Ankle hyperpigmentation

- Lipodermatosclerosis
- Skin atrophy

Expanded Aside:

Classify varicosities –
- congenital,
- primary (underlying structural problem),
- secondary

Distinguish primary from secondary varicosities:

Primary:
- early onset, fam hx of varicosities.
- Precipitatitng factors: after pregnancy, external compression (may thurner, pelvic mass).

Secondary:
- will have hx of normal limb, then DISTINCT event (DVT/trauma/iatrogenic surgery),
- hypercoagulable state/fam hx of such,
- contraceptive use,
- AV fistula.

History should always include:
- profession/long upright standing.
- Review previous ulcer/vein surgery/treatment
  - compliance and work up.
- history of vein harvest,
- DVT
- pregnancy,
- hypercoagulability,
- trauma
- avf

- hx of IC/rest pain
- RF for Atheroscelrosis as the ulcers will turn out to be mixed in origin
  - smoking, htn, hl, homo, dm, cva/mi
- hospital admission

Aside:

Classify varicosities –
- congenital,
- primary (underlying structural problem),
- secondary

Distinguish primary from secondary varicosities:

Primary:
- early onset, fam hx of varicosities.
- Precipitatitng factors: after pregnancy, external compression (may thurner, pelvic mass).

Secondary:
- will have hx of normal limb, then DISTINCT event (DVT/trauma/iatrogenic procedure/surgery),
- hypercoagulable state/fam hx of such,
- contraceptive use,
- AV fistula.

On exam:

- With primary, likely see SFJ involvement only, i.e. predominantly superficial reflux
    - i.e. no perf/deep vein problems
    - less remarkable CVI stigmata.
- Secondary will have more advanced disease
    - Deep refulux, perforators, significant CVI stigmata

- inspect –
    - swelling, varix, ulcer - vein: pink, clean bottom, flush with skin level, moderately tender to painless, better with elevation, bleeds on probing, gaitor area,
    - normal pulses,
    - signs of CVI
        - telangecatsia/reticular (1),
        - varicose (2),
        - swelling (3),
        - skin changes - atrophy blanche, lipodermatoscleoris (4),
        - healed ulcer (5),
        - active ulcer (6).

- Aside:
    - Arterial ulcer - pale fibrinous necrotic bottom, distal foot, painful, punched out, poor pulses, better with dependency, signs of PAD and RF on history plus IC/rest pain.

Exam cont'd
- auscultate: bruit for PAD
- palpate: Trendelenberg, pulses
- Asses reflux in superficial and deep system and perforators.

Trendelenberg test –

- supine, elevate leg, compress GSV junction or LSV junction.
- Stand up, maintain compression:
    - watch fast refilling of vein from below – check perforators
- Release compression –
    - see fast refilling from above – check in line valve insufficiency

Finally, investigations:

- Duplex
    - First check for DVT when supine, and > 2 sec reflux at SFJ with valsalva
    - Upright – check for >0.5 sec reflux with cuff deflation distal to SFJ

- Venography – rarely 1st
    - Ascending for obstruction
    - Descending for reflux
    - Pressure gradient studies if suspect obstruction
- ABI +/- angio if necessary
- CT/MRI if suspect central obstruction

Ambulatory venous pressure:

- Pressure in the dorsal pedal vein after 10 consequitive calf contractions.
- Should dip from 80 mm Hg standing to 20-30 mm.
    - If going up – suspect proximal obstruction
- Should take 30 sec or more to return to 80 mm with standing quietly:
    - If faster – suspect valve incompetence

PPG and APG are non-invasive substitute for invasive AVP measurement.

Conservative treatment:

- Compression
- Dressing change – DRY gause
- Elevation
- ASA/iodosorb/pentoxifylline/Prostoglandin
    - p. 2245-47
    - NO occlusive hydrocolloid dressings (duoderm)
        - same healing rates as dry, may be more infection

Compression: Rutherford: 30-40 mm Hg below knee
Sigvaries recommends the following CI:

Absolute contraindications:

- Advanced peripheral obstructive arterial disease
- Severe neuropathy (diabetic)
- Congestive heart failure (active)
- Septic phlebitis
- Plegmasia coerulea dolens

- Unaboot – glycerin, zn oxide, chamomile, sorbitol, mg, al silicilate

If mixed ulcer in old gentleman and ABI 0.6 - may try light profore dressing for 3-4 weeks

If not improving - review diagnosis:

- r/o non-compliance
- mimickers:
    - CTD
    - HTN
    - Malignancy
    - Pyoderma gangrenosum
    - Calcifilaxis in dialysis patients
- local and general factors preventing healing:
    - infection
        - r/o osteomyeltis and w&w infections
    - ongoing trauma
    - stalled wound
        - need for debridement,
        - needs growth factors
    - malnutrition
    - immunocompromised state
        - malnourished
        - cancer
        - chemo

Consider adjuncts -

- intermittent compression
- promogran/regrenex/platelet spin-off to kick start stalled wound
- abx iv/po if cellulitis
- antimicrobial in the wound
- consider hyperbaric oxygen
- consider apligraf/pentoxyfilin, iloprost

If surgery necessary –

- consider location and see if stripping GSV/ligation would help.

Indication for surgical intervention: after 3 months of intensive compliant conservative management…

1. ulcer
2. pain
3. bleeding
4. superficial thrombophlebitis and thrombosis of GSV
5. cosmetic

Healed ulcer is not likely to open up again. However, with maintenance program, 30% recurrence is expected. If no maintenance is adhered to, 100% recurrence is guaranteed. Overall, 6% of pts with CVI will progress to ulcer over 5 years (Minessota study).

139.Name veins ligated during saphenous vein stripping:

- superficial epigastric
- superficial circumflex iliac
- superficial external pudental
  - these may be left intact if you believe in neovascularization
  - Rutherford does…
- greater saphenous
- anterior accessory
- posterior accessory

140.Why does vein stripping surgery fail:

- Surgical error: failure to remove the GSV from the circulation
  - Ligated SFJ only, not stripped
  - Missed duplicated saphenous vein
  - mistook accessory saphenous vein for the GSV
- Non-saphenouse source of venous HTN:
  - perforating veins with incompetent valves
- genetic tendency to form varicosities
- neovascularization

aside: contrary to previous dogma, Rutherford points out that leaving some branches intact actually protects from neovascularization… So it pays to be "sloppy"

141.Venous claudication: cause

- Bursting deep leg pain with exercise
- Proximal venous obstruction, distal veins are usually normal

- Due to exercise induced hyperemia AND increased venous outflow resistance.

142. Chronic IVC/iliac/deep vein obstruction:

- Intrinsic venous defect:
  - Acute DVT
  - Endothelial scarring:
    - Post chronic DVT
    - Radiation
    - Abnormal venous webs, hypoplasia, aplasia

- External compression
  - Retroperitoneal fibrosis
  - Benign/malignant tumor
  - Cyst
  - Aneurysm (arterial)
  - Bands/slips
  - May Thurner syndrome (L common iliac vein compressed by R CIA)

Testing:

- Duplex
- Plethysmography
- CT/MRI abdomen
- Resting arm/foot venous pressure difference > 4 mm Hg
- Resting supine central/femoral pressure difference > 5 mm Hg
- 2 fold increase in femoral pressure after exercise
  - 10 dorsiflexions at ankles
  - 20 isometric calf contractions
- Venography – ascending !
  - for OR planning
  - B/l femoral vein puncture

Venous stent: Gianturco Z stent: self-expanding, 8-40 mm, good loop strength, no forshortening, can use across tributaries (renal vein) – i.e. large cells

143. SVC obstruction:

Blindness and cerebral hemorrhage is an uncommon complication.
Cause:

- Cancer (lung, mediastinal LN, tumors) – 85%
- Benign –
  - MOST Common – mediastinal fibrosis,
  - Yatrogenic – due to catheters
- Treatment:
  - If Cancer – radiation therapy of primary

- Image from basilic vein bilaterally, then attempt to stent with Gianturco Z stent
- If fails, consider central bypass vs IJ-femoral extra-anatomic with GSV

144. Why bypasses for venous repair are prone to failure?

- Low flow due to
    - collateral circulation
    - distal venous obstruction
    - incompetent valves
    - **remedy:**
        - **+/-bypass, AVF, periooperative intermittent compression**
- easily compressible grafts
    - due to low pressure in the circulatin
    - dependence on intraabodminal/thoracic pressure
    - location behind inguinal lig/liver
    - **remedy:**
        - **external support, large diameter graft**
- increased thrombogenicity
    - most pt have lack of pn C, S, AT III
    - synthetic grafts more thrombogenic
    - **remedy:**
        - **use autologous graft**
        - **use anticoagulation**
        - **surveillance**

145. List LE perforators:

- Connect GSV and deep system
- Normally, direct flow form superficial to deep
    - Except in foot – no valves there
    - Allows for measurement of AVP in superficial dorsal vein
- Cockett I, II, III
    - go from posterior arch vein (NOT GSV) to post tibial vein
        - aka vein of Leonardo
    - 4 cm posterior to medial edge of tibia
    - along Linton's line:
        - 6, 12, 18 cm from floor,
    - cockett 1 at med malleolus
        - can't get this on with SEPS
- Paratibial perforators
    - Proximal location
    - 2 cm medial to the medial edge of tibia, go from GSV to posterior tib and pop vein
- Thigh:

- Hunterian (H for high) – in the mid high thigh
- Dodd's (D for distal thigh)
- Boyd's (B for below) – below knee

- Vein of Giacommi – connects GSV and LSV
- 10% of veins are completely duplicated
- 25% - partially duplicated
- No valves in CFV or iliac veins
- To study perforators, use descending phlebography
  - Low false positive
  - High false negative
  - Duplex is better

146.Indications for perforator ligation:

- have to have CEAP class 4 and above….
- Failure of conservative tx of severe stasis symptoms
- Recurrent cellulitis/recurrent DVT during conservative treatment
- Relative – non-compliance

Contraindications to perforator ligation:

- Chronic PAD
- Infected ulcer
- Morbid obesity
- Non-ambulatory and high risk pt
- Relative: CRF, DM, Rheumatoid arthritis

Results:

1. Difficult to distinguish between contribution of GSV L/S and perforator surgery
2. ulcer healing at 10 y – 60%
3. recurrence 20% (40% in post thrombotic limbs)
4. improved hemodynamics (Ambulatory Venous Pressure) in some but not all studies
5. possible that sclerotherapy may be better than SEPS or surgery…

Surgery:

1. Subfacial Endoscopic Perforator Ligation (SEP)
2. Open – modified (for cocket I)
3. Open for the rest of them – modified Linton (not done much)

Results:	SEPS	Open
Ulcer Healing	94%	88%
Ulcer recurrence	11%	22%
Wournd complications	5%	24%

DIALYSIS ACCESS

147.Principles of AVF creation:

DOQI wants at least 50% AVF as autologous access.

- If short term dialysis needed < 3 weeks – temp line (quinton)
- Check GFR if <30 ml/min
- Create access if anticipate dialysis within a year
- Autologous if possible
- Non-dominant limb
- Distal portion of upper limb first
- Venous imaging if:
  - Edema in arm
  - Collateral veins on chest on exam
  - Previous lines/pacemakers (several)
  - Previous arm/neck surgery
- Subcutaneous placement

- Order of placement in upper extremity:
  - Radiocephalic
  - Basilic transposition in forearm
  - brachiocephalic
  - basilic vein transposition above elbow
  - loop forearm AV PTFE
  - upper arm PTFE
- order in LE:
  - try to use all arms first
  - SFA/SV loop
  - PTFE loop

What is normal AVF flow:

- total AVF flows above 600 ml/min are enough for dialysis.
  - ideally, aim for a flow of ~1000 ml/min.
  - minimum flow required is 350 cc/min noted on 6 occasions/month
- If an AVF flow falls by 25% or more in any given 4 months,
  - AVF exam with ultrasound or a fistulogram.
- 2000 ml/min is too high.
- US Sign of mature vein
  - 4 mm, >500 cc/min – 95% maturation certainty
  - <4 mm, <500cc/min – 33% maturation certainty

Vein mapping:

- Vein - Need 2.5 mm below elbow, 3 mm above

- Artery – at least 2 mm diameter
- Use tourniquet for vein mapping
- If vein > 5 mm deep, may need to superficialize it at the later date
- High radial a. take off may lead to steal in radiocephalic AVF

148.Access options in Central Vein Occlusion:

- Recanalize vein endovascularly
- go to lower extremity,
    - Superficial femoral vein transposition
    - may use GSV if sure no need for lower extremity revascularistion in the imminent future.
        - GVS vein (does NOT dilate much even if transposed to arm)
- reconstruction (jug turn down, bypass)
- IVC canulation
    - percutaneous tunneled catheter
    - HeRO device http://www.veithsymposium.org/pdf/vei/2090.pdf
- Weird and Wonderful:
        - Ax-fem-vein ptfe bypass as venoud outlflow
        - Axillary-axillary artery loop
            - Bunger, CM JVS 2005, 42(2)-290-295

149.Complications of AVF creation:

- Failure to mature
- Stenosis (particularly at the venous end)
        - Leads to thrombosis
        - Surgery and percutaneous thrombolysis equally effective
- Aneurismal dilatation
- Bleeding that can lead to…
    - Pseudoaneurysm
- Seroma that can lead to …
    - Infection
- Steal that can lead to:
    - Heart failure
    - Swelling and Venous hypertension
    - Neurophathy – monomelic neuropathy

150. Treatment of AVF steal:

Some coolness, stiffness and swelling are common after normal AVF creation. So may want to wait awhile to see if sxs settle.

Reminder: steal happens with reduced inflow AND increased outflow resistance in the arm compared to AVF

- Distalization of arterial inflow –
  - for brachioceph AVF, move inflow distally to radial a.
  - in a way, this is like controlled banding or creating a smaller inflow
- Proximalization of arterial access –
  - for brachioceph AVF,
  - move inflow to the axillary artery.
  - This moves pressure sink area proximally and
  - This improves inflow and decreases outflow resistantce to the arm compared to AVF.
- DRIL:
  - Proximilazation of inflow and increased resistnace to reversed flow
  - ligate distal to AVF
  - bypass from 2-3 cm above AVF to the point beyond ligation.
- Distal radial a. ligation if rad-ceph AVF:
  - in a way, a form of DRIL
  - must have continuous palmar arch
- Ligate fistula
- Banding fistula

http://www.fistulafirst.org/pdfs/Surgical_Salvage.pdf

LYMPHEDEMA

151.Lymph physiology:

Lymph is propelled by intrinsic contraction of lymph vessels with directing valves. External pressure has little effect on transport. With dilation, when chronic, ability to contract is lost. This results in settlement of protein rich fluid in the tissues.

152.Classify lymphedema:

Differential diagnosis first:

- Acute:
  - Cellulites
  - DVT
  - Trauma
  - Fracture
  - Torn ligament/muscle
  - Ruptured Baker's cyst
  - Acute limb ischemia

- Chronic
  - AV malformation
  - Lymphedema
  - CVI
  - Systemic disease
    - CHF
    - Low protein
      - Liver failure
      - Nephritic syndrome
    - Reflex sympathetic dystrophy (acute stage)
  - fat

Etiologic classification:

- Primary:
  - Congenital < 1 yoa
    - Non-familial
    - Familal (Milroy) – Roy/king by birth
  - Praecox 1-35 yoa
    - Non-familial
    - Familial (Meig) – got my first Mig before 35
  - Tarda > 35 yoa
    - Ta-DA! I am old now…
- Secondary:
  - Filariasis
  - Lymph node/vessel injury:
    - surgery
    - infection
    - tumor
    - rads
    - trauma

Anatomic classification:

	Distal hypoplastic 80%	Proximal hypoplastic 10%	Hyperplastic 10%
Gender distribution	Females	Any gender	Any gender
Laterality	Bilateral	unilateral	Bilateral
onset	At puberty	Any age	Congenital
Fam hx	Positive	No	Positive
progression	Benign, slow course	Fast progression	Progressive
Response to TX	Well to conservative tx	Poor to conservative tx **	Responds to conservative tx ***

- ** proximal hyperplastic – May be candidates for microvascular reconstruction

- *** hyperplastic
    - mesenteric lymphatics is incompetent –
        - reflux of chyle and pn losing enteropathy.
        - Chylous drainage via vagina, scrotum, lower extremities.
    - May be treated with retroperitoneal excision of incompetent lymphatics.

153. Long term complications of Lymphedema:

- Local:
    - infection
    - Fibrosis
    - Neoplasia (lymphagiosarocoma, Stewart-Thrives)
- Systemic:
    - Malnutrition
    - Immunodeficiency

154. Treatment of lymphedema:

Indications:

- Improvement of limb function: MOST EFFECTIVE indication
    - severe impairment of mobility
    - Reduction of pain
- Cosmetic improvement
- Reduction of complications
    - cellulitis, lymphangitis, lymphangiosarcoma

Treatment:

- Conservative
    - Two phases – active decongentstion then maintenance
        - elevation/compression/manual decongestion
        - excerise
        - prevention/treatment of cellulitis
            - penicillin 500 mg TID at first sign of infection
        - benzopyrones
            - may be effective in reabsorbtion and mobilization of tissue pns
        - compressive garment is used for maintaining limb size
- surgery: < 10 % of all pts need this
        - excisional operations RARE
            - charles
            - Linton - staged subcutaneous excision beneath flaps
        - microsurgical lymphatic reconstration: V.RARE
            - lymphovenous anastomosis
            - lymphatic grafting

    - free lymphatic flap
- liposuction

UPPER LIMB

155.Genearl causes of upper limb ischemia:

- In general, upper limb ischemia is due to spasm, obstruction, or emboli.
- When performing an angio, always use vasodilators to assess distal spasm – more ommonly seen in upper than in lower limbs.
- If see hand weakness post brachial puncuture – alsway explore median nerve – small hematoma in the sheath will cause permament damage.

    - spasm - ergotamine, IV drug abuse, raynaude disease
    - obstruction:

        - large vessel –
            - Atherosclerotic ulcer – more proximal sublcavian
            - TOS – more distal subclavian
            - Arteritis
                - Tak, GSA
            - Radiation
            - FMD,
            - DISSECTION
        - small –
            - CTD
                - Scl, RA, SLE, Sjo
            - metabolic
                - DM, CRF
            - myelo
                - thrombocytopenia, PCV, Cold cryglob, hypercoag, leukemia
            - trauma
                - vibration, cold injury, AVF
            - other
                - Burger, intraarterial injection, cytotoxic drugs - chemo

    - Emboli:
        - aneu - ventricular, inno, subcl, axil, brach, ulnar
        - plaque - arch, inno,subclavi
        - heart - AF, post MI, valve

156.Symptoms & signs of TOS:

- Pain & paresthesia,
- Worse with arm elevation
- Occipital headache
- Weakness/muscle atrophy (uncommon)
- Local tenderness in scalene triangle
- Elevation Arm Test (EAST) –
  - with arm exernally rotated/abducted, elevated
  - can't repetitively open/close fist for > 30-60 sec
- Adson test:
  - disappearance of radial pulse when head rotated the other way, breath in
- Positive provocative tests seen in normal limbs as well.

157.Approach to pt with TOS

TOS:

- Neuro – 95%
- Vein – 4%
- Artery – 1%

Overview:

- Review occupation, neurological, arterial, venous, Raynaud symptoms.
- Assess location, laterality, degree of disability.
- Review pmx of compressive syndrome, neck/shoulder trauma, vibrational/occupational injury, CTD, hypercoag state, AF.

Specific History:

- occupation, provocative maneuvers, hand dominance

- numbness/weakness (neurogenic)
- ulcers/hand fatigue/claudication/nail splinter hemorrages (arterial)
- swelling/catheters/recent strenuous activity (venous)
- vasomotor symptoms (Raynoudes)

- bilaterality
- duration,
- what was tried for relief, course of sxs over time,
- disability - at work and life style limiting

- prev hx of
  - AF/anticoagulation
  - DVT/HCS
  - CTD/arteritis

    - Central lines
    - Trauma:
        - Neck
        - Rotator cuff tear
        - Hand
        - Vibration
        - Frost bite
    - DDD in neck
    - Entrapment
        - Ulnar
        - median
- meds: OCP if female
- fam Hx: hypercoag state
- ROS: functional status, degree of disability.

Exam:

- blood pressure both arms, pulse quality
- pulse distribution/bruits
- provocative testing –
    - Adson - radial goes when head turned the other way, breath in.
    - EAST - external rotation, abduction - can't sustain fist clinching for more than 30 sec. High negative predictive value test.
- wasting, weakness, sensation, reflexes, Tinnel, Phalen test.
- Check for Rotator cuff tears/weakness, bicipital tendonitis
- splinter hemorrages/ulcers/Allen test.
- the rest of exam - i.e. other extremity, HS, abdo etc.

work up:

- no test alone is indicative or fully diagnostic
- go by presentation hx and physical
- CXR to check for 1st rib
    - See in 1% of population, more in women, 95% of arterial TOS have it.
- Dupplex to assess vein and artery
- CTA if suspect aneurysm
- MRI – but may not show much…
    - Will rule out cervical disk disease, syrinx, spinal cord problems
- Nerve conductions studies are likely to be normal
    - If abnormal – see median motor and ulnar sensory problems

- Rutherford recommends test injection of lidocain in to ASM
    - If better, then may benefit from surgery discussion
- Surgery is better viewed as DIAGNOSTIC rather than therapeutic

158. Complications of 1st rib resection:

- Wound hematoma/infection
- Injury to
    - intercostobrachial n.
    - brachial plexus
    - long thoracic n.
    - thoracodorsal n.
    - phrenic n. – temporary palsy seen in 10% of cases
    - sympathetic chain
    - vein
    - artery
- pneumothorax
- lymph leak
- persistent/recurrent TOS

159. Indications for surgery of a subclavian a. aneurysm:

- Symptomatic:
    - rupture,
    - thromboembolism,
    - pressure: pain, brachial plexus
- Size > 2 cm
- Note that 30-50% pts with atherosclerotic (most common type) aneurysm will have aneurysms elsewhere

160. Vibration white finger:

- Raynaud's phenomenon due to prolonged use of vibratory tools
- Numbness, tingling -> tips blanching ppt'd by cold
- With time affected area progresses

- Only 1% progress to ulcer/gangrene
- Mechanism unknown.
- Cold provocation testing diagnostic
- Angio: digital a. occlusion findings

- Tx: avoid cold, may try CCB for advanced cases.
- Sympathectomy is RARELY needed
- If digital gangrene – consider prostoglandin E2/iloprost iv

161. Hypothenar Hammer Syndrome:

- Anatomy:

- Ulnar aretery over hypothenar imminence is vulnerable
- Terminal branches or ulnar a. may be involved (deep & superficial palmar)
- Arise in the canal of Guyon. Boundaries:
  - Medial – pisiform/hook of hamate
  - Dorsally – transverse carpal lig
  - Ulnar a. is superficial (skin, Palmaris brevis)
- Intimal damage – occlusion/aneurysm
- Histo: FMD in media, intimal disruption
- Raynaud's phenoemenon, digital ischemia
  - But always spares thumb due to radial artery supply
- Rarely ulnar a. occlusion
  - Tx: conservative, CDT if within 2 weeks
  - Resect/repair ulnar a. aneurysms to prevent embolism
    - For graft – harvest distal GSV (size match)
    - Ligate only if palmar arch is patent

162. Occupational acro-osteolysis:

- First seen in workers exposed to polyvinyl chloride
- Resorption of the distal phalange tuft, similar to scleroderma
- Raynaud's phenomenon
- Angio
  - digital occlusion AND hypervascularity adjacent to bony resorption

163. Athletic injuries:

- Digital artery occlusion
  - Trauma in catchers
  - Cleland ligament: from phalanx to sc tissues, on palmar surface over PIP
- Embolization
  - Quadrilateral space
    - teres minor sup, humerus lat, teres major inf, long head of triceps med
    - Posterior circumflex a., axillary nerve
    - artery prone to aneurysm in pitchers, volleyball
  - thoracic outlet (leading to subclavian aneurysm/thrombosis)

Lower LIMB

164. Differential diagnosis of bilateral lower limb swelling:

- Systemic: MC
  - Pitting edema

  - CHF – better in am after supine, but may get exacerbation of CHF
  - CRF – not better in am after supine
- Drug induced – Adalat – mild
- Iliofemoral thrombosis
- Aorto-caval fistula
- Lymphedema
  - Toes – > groin, squaring of toes
  - Unilateral most commonly
  - Hyperkeratosis of skin, fibrosis at LAST stages
- CVI – MOST COMMON CAUSE in NA
  - Stigmata of CVI
  - Almost never involves feet
  - The only condition to cause scarring of perimaleolar skin (lipodermatosclerosis)
- Fat legs

165.Approach to distal peroneal a.

  - medial
  - posterior
  - lateral with fibula resection

exposure:

- peroneal a. is continuation of tibioperoneal trunk
- Medial:
  - in the upper calf, TPT is exposed thus:
    - skin, sc fat, preserve GSV, fascia, divide soleus muscle over angled dissector, divide anterior tibial vein
    - beware of dense venous plexus
  - in the middle calf:
    - muscles of the deep compartment
      - from medial to lateral –
        - FDL – flex dig longus,
        - FHL – flex hal longus,
        - PT – post tib
    - remove soleus from the edge of tibia,
    - reflect soleus and GC posteriorly:
    - PT artery is found between posterior surface of the FDL and soleus – leave PT artery in the fascia/areolar tissue attached to soleus
    - Keep going deeper
    - Peroneal a. is found on the posterior surface of FHL, close to the fibula.
  - Distally:
    - Find peroneal between FDL and FHL.
- Posterior approach:
  - Direct, fewer wound complications, for short bypass with LSV

- Supine
- Incision in distal third over ***lateral*** edge of the calcaneal tendon
- Retract CT medially and FHL laterally

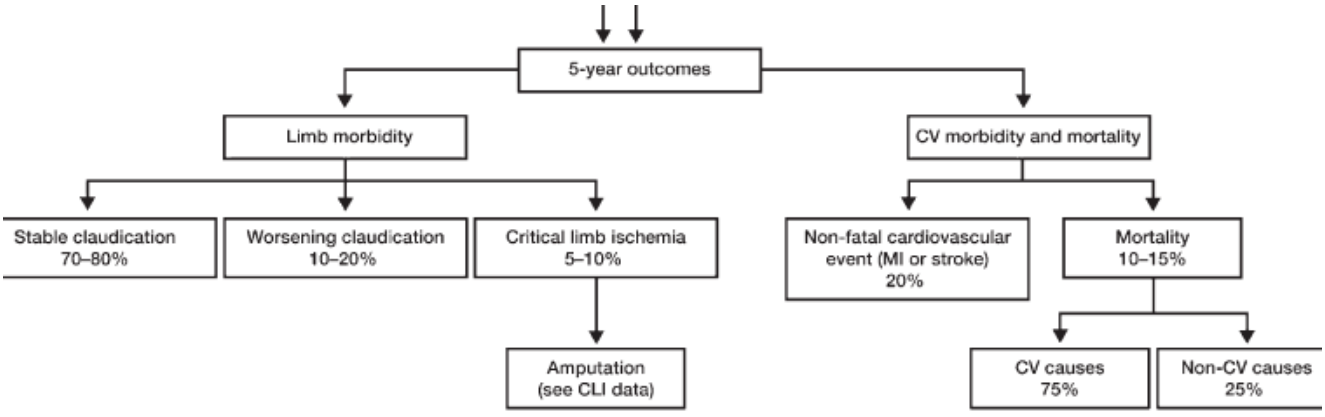
- Lateral approach:
  - 10-15 cm incision
  - ID Common peroneal n., protect
  - Separate muscle of lateral compartment from fibula
  - Excise fibula, may do so subperiosteally, dril in places of resection for clean cut
  - Peroneal a. is Sitting right there in the fibular bed.

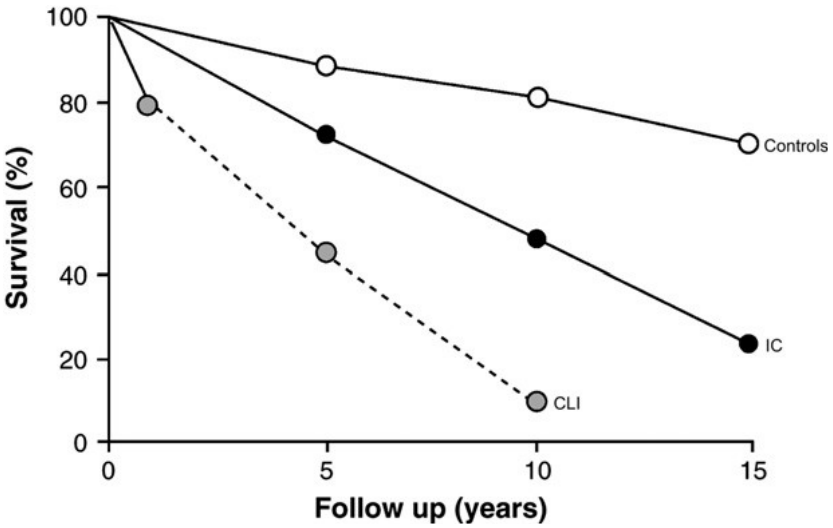
166. Fate of a claudicant:

- IN GENERAL, 20% deteriorate, 10% CLI, 2% lose limbs (more if diabetic).
- Decrease in ABI < 0.5 is best predictor of deterioration in IC and mortality
- At 5 years:

  - CV MORBIDITY (non-fatal MI or stroke) 20%
  - CV MORTALITY 10-15%.

- **Risk of limb loss in IC can be stratified:** p. 16, Rutherford
  - It is too simplistic to say for all IC risk of limb loss is 1% per year
    - As is suggested by EPIDEMIOLOGIC data from Framingham
  - **If clinically confirmed, risk of LIMB loss is closer to 5% per year**
    - 3.7% if diagnosis is established clinically
    - 5.8% if diagnosis confirmed with non-invasive studies
    - 8.5 % if ABI between 0.4-0.6
      - Hence term subcritical ischemia





From TASC II:

	Intermittent claudication	No intermittent claudication
5 year survival	85%	95%
10 year survival	50%	85%

ABI <0.5 is an important predictor of overall survival.

Claudication management options:

Grade it first – with treadmill. Maximum Walking Distance is MORE predictive than Pain Free Walking Distance.

ABI is the best predictor of abbrevitated survival.

Goal:

- Increase quality of life
- increase Pain Free Walking Distance, and Maximum Walking distance
    - cilastazole
    - walking exercise
- delay progression to CLI
    - modification of RF
        - Thorough discussion re: natural hx of claudication.
            1. Benign – 20% worse, 10% CLI, 2% limb loss
                1. See above – may be as high as 8% per year..
                2. Overall felt NOT to be a limb threatening issue
            2. Problem is global: need to address all vascular beds
                1. i.e. pain in the leg is Life style issue

Management of risk factors:

- interestingly enough, this <u>will not affect claudication distance</u>…
    - success in slowing the progreesion of PAD and improving IC *<u>has not been proved</u>* by statistically significant valid clinical trials. Rutherford P. 602

- but it **will address the global progression of atheroscleoris**…
    - has been shown to reduce the risk of CAD and progression to CLI

Specifically, what needs to be done:

- Smoking cessation
    - Smoking improves IC distance in some but not other studies, hence no consensus
    - Cessation will improve patency of bypass 3 fold…
    - 11% of smokers with IC will undergo amputation, compared to 0% in non-smokers
    - 3 fold higher risk of needing intervention if pt has 40 pack year of Smoking

- HTN control
- HL control
    - Reduces progression to CLI
- DM if present
    - Treatment of DM was NOT shown to reduce amputations…
- Antiplatelet medications
        - **Walking therapy: the only non-operative treatment shown to improve IC.**
            - Supervised better than non-supervised
                - 150 m difference, Cochrane, 2006
            - 30 min/3 times a week for 3 months,
                - increase to 60 min a session
                - increase speed from 2 mi/h to 3 mi/h
                - initial goal 10 min pain free walking then start pushing
            - improvements in walking efficiency, endothelial function and metabolic adaptations in skeletal muscle
            - Cochrane meta-analysis:
                - 150% increase in MWD
    - DRUGS:
        - Cilostazole:
            - decreased platelet aggregation & TG, increased SMC relaxation & HDL metabolism
            - About 140 m (450 f)
                - Rutherford), Taks II – 50-70 m...
                - 30-100% MWD
            - Mc side effect: headache, diarrhea
        - Pentoxyfylline: rheologic drug

        - Some studies show benefit, others don't
            - Placebo vs drug: 24% vs 40% in PFWD
            - 32% vs 20% in MWD
        - Overall likely small improvement: 50m (160 f)
    - Operative intervention: bypass vs PTA.

RCT: walking vs endovascular, 2009

- 76 pts ENDO vs 75 pts walking therapy
    - Hospital based
    - Proven mild, moderate and severe Claudicants
- Measure Functional capacity, MWD, PFWD, ABI improvement over 12 months
- Improvement in walking:

	1 week	6 months	12 months
Endo 1st	88%	75%	68%
Walking	16%	77%	65%

Equivalent objective and subjective improvement between two therapies…

Endovascular therapy **does not create new indications** for an intervention.

It defines a population with an accepted indication that allows acceptable improved outcome with lesser intervention.

- Ideal technical outcome of an open bypass:
    - patency, limb salvage and survival
    - **seen in at least 90% of bypasses**
- **Ideal functional outcome after an open bypass:**
    - Hospital free life, reintervention, wound healing, significant change in AMBULATORY potential
    - **seen in only 15% of all bypasses…**
- CANDIDATES FOR ENDOVASCULAR INFRAINGUINAL THERAPY:
    - Economic claudicants
        - exhausted conservative trial of RFM, walking, and cilastazole and who understand that:
            - intervention is done for Life style issues, not limb salvage
            - compared to open intervention, PTA offers an uncertain but reasonable durability
            - compared to open intervention, PTA carries SMALLER chance of conversion to CLI and limb salvage situation

- patients with CLI with non-prohibitive tissue loss AND limited preop ambulatory capacity
  - despite an excellent anticipated TECHNICAL outcomes of the open bypass in the end are predicted to have suboptimal FUNCTIONAL outcome
    - based on their feable health status, nursing home situation, poor premorbid ambulation.
  - In these subselected cases, minimal intervention that converts CLI (rest pain or ulcer) to claudication status is worthwhile.
- SIA as a Technical adjunct to open approach allowing for performance of hybrid cases when purely open approach is either impossible or impractical:
  - TASC II, B type SFA lesion
    - short conduit - SFA/pop inflow source management
  - CFEA open and SFA angio

If claudicants represent 15% of the operated upon poplution, then i'd hazard 5% would to be task A and B, and hence candidate for endo first option. As far as the CLI it is hard to predict the numbers... 30%?

167.CLI criteria:

- Severe RP requiring opiate analgesics for >4 weeks and either
  - Ulcer
  - Ankle pressure < 40 mm Hg
  - Toe pressure <30 mm Hg

Fontaine		Rutherford		
Stage	Clinical	Grade	Category	Clinical
I	Asymptomatic	0	0	Asymptomatic
IIa	Mild claudication	I	1	Mild claudication
IIb	Moderate-severe claudication	I	2	Moderate claudication
		I	3	Severe claudication
III	Ischemic rest pain	II	4	Ischemic rest pain
IV	Ulceration or gangrene	III	5	Minor tissue loss
		IV	6	Ulceration or gangrene

168.Natural hx of pts with CLI:

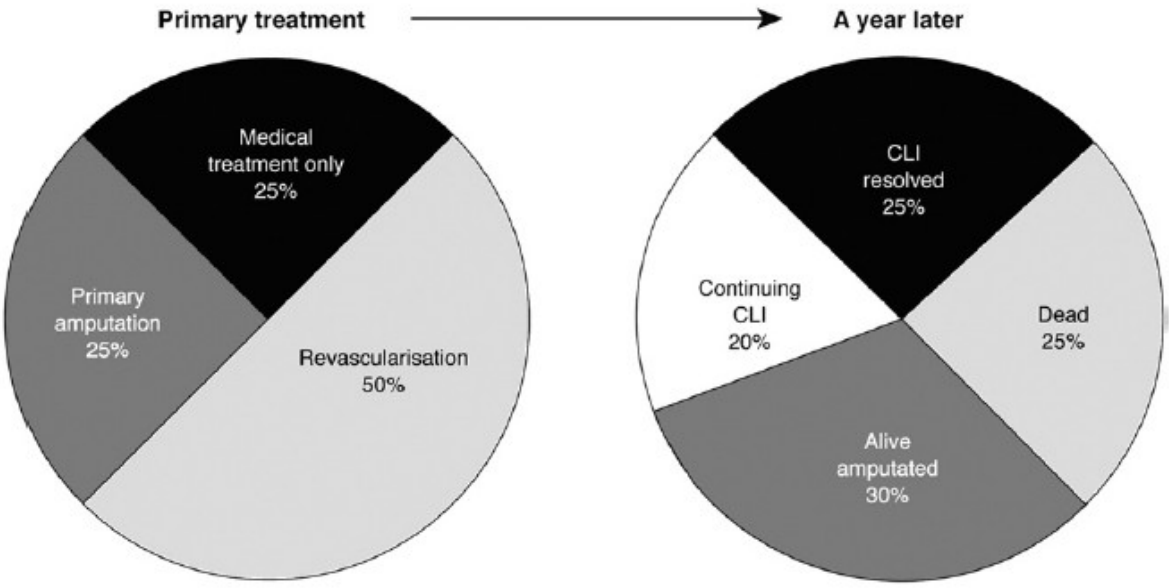


Fig. A5. Fate of the patients presenting with chronic critical leg ischemia. CLI – critical limb ischemia.

- CLI at diagnosis:
  - 50% receive revascularisation
  - 25% are given conservative tx
  - 25% receive primary amputations
- At 1 year:
  - 25% CLI resolved
  - 20% ongoing CLI
  - 30% are ALIVE but amputated
  - 25% dead

Note, that prospect of limb loss is not universal. With conservative management, 50% will keep their limb and 40% of ulcers will heal.

169.TASC classification for iliac and SFA lesions:

- **Iliac:** need to know only A and B – as these are the for endo, the rest is open…
  - A
    - CIA stenosis: uni or bilateral
    - EIA stenosis <3 cm: uni or bilateral
  - B
    - < 3 cm aortic stenosis
    - Unilateral CIA occlusion
    - Unilateral EIA occlusion
      - Not involving IIA or CFA
  - C

- b/l CIA occlusion
- b/l EIA stenosis not extending into CFA 3-10 cm
- uni EIA stenosis extending into CFA

▪ D

- Infrarenal occlusion
- Aortic Aneurysm
- Uni stenosis of both CIA/EIA
- b/l occlusion of EIA

o **SFA**

▪ A

- Single stenosis <10 cm
- Single occlusion <5 cm

▪ B

- Multiple stenosis/occlusion < 5cm each
- < 5 cm calcified
- Single stenosis < 15 cm, not involving pop
- **Single of multiple lesions in the absence of continuous tibial vessel involvement**
  - **To improve inflow for distal bypass**

▪ C

- Multiple stenosis/occlusion >15 cm
- Recurrent stenosis/occlusion after 2 endo treatments

▪ D

- Entire SFA occluded
- Pop or trifurcation occlusion

170.BASIL study:

- Randomized prospective trial
- 27 UK hospitals
- 450 pts with CLI were randomized
- Bypass vs PTA as 1st treatment with CLI due to infra-inguinal disease
- Crossover of patients was allowed after randomization
- Two strategies "broadly similar" wrt
  - amputation free survival,
  - all-cause mortality
  - quality of life in the SHORT term.
  - Surgery 1/3 more expensive
- Kaplan-Meier plots for long term:
  - amputation free survival and all cause mortality favour PTA 1st for the first 2 years,
  - bypass was better past 2 years.
- Overall, at the end of 3 years,
  - **Surgery – 57% amputation free survival**

- **PTA – 52% amputation free survival**
- **Overall longevity - 50%**

- **Hence, if life expectancy is less than 2 years, offer PTA.**
- **If life expectancy more than 2 years, offer surgery .**

171. Vein advantages over prosthetic:

Combines many of the ideal qualities of the graft…

- Biocompatible
  - Resists thrombosis
  - Excellent ingrowth
  - Less infectable
  - Heals if infected
  - durable
- similar to native biophysical match:
  - impermiable
  - Better size match
  - Compliance similar
  - Grows with arterial system
- Logistical
  - Cheap
  - Available
  - Easy to handle

As a result, vein has better patency

172. Options for bypass graft material in descending order of preference:

- GSV
- LSV
- Arm vein
- Composite graft (with adjuncts – venous cuff/patch)
  - Do not do straight ptfe-vein anastomosis
    - Rather, ptfe goes to blind arterial segment/distal SFA
    - Vein takes of distal to the ptfe, NOT off the hood of the prostetic bypass
- SVF
- Prosthetic (heparin bound>PTFE, Dacron)
- Cadaver GSV
  - Cryopreserved
  - 1 year patency 28-80%
  - 2 year patency 19-40%

        - Early failure 17%
        - Late aneurysm 5-30%
        - Limb salvage 60-100%
            - P.720-21
    - Allogenic vein/artery

Note:

- in thigh and calf, 65% of GSV is single, 35% - double system
- most common location of abberantly placed GSV is ANTERO-LATERAL to usual location.
- Cryopreserved veins are NOT better than abx bonded graft in infected field wrt patency\
- According to Rutherford's Companion, HUV is BETTER than multispliced vein for distal bypass. It also has same rate of infection as autologous vein. HUV has outer supporting mesh and needs special handling.
- Antiocoagulation, cuff patch will improve patency of BK prosthetic bypass

173.Synthetic grafts – characteristics:

- woven
    - long (warp) and circumferencial (weft) thread pattern
    - low porosity
    - strong
    - stiff
    - doesn't dilate
    - poor handling, fray at edges
    - recommended for ruptures and thoracic replacement
- knitted
    - predominantly long (warp – long way from home, StarTrek)
    - leaks – need preclotting
    - flexible, comfortable
    - dilates with time
- veloure
    - micro loops on the outside
    - designed to improve incorporation into tissues and protein adhesion – to reduce porocity.
- Krimping
    - adds flexibility and elasticity
    - may interfere with laminar flow
    - smaller diameter may cause increase thrombogenicity
- biologic coating

- with albumine
- to improve porocity issue
- more expensive…
- saves time – no need to preclot

Endothelial seeding:

- still experimental
- do not decrease anastomotic hyperplasia
- however, show better patency at 3 years
- only 40% of attached cells remain on graft at 24 h

174.Adverse effects of Vein harvest:

- Dissection technique – trauma to vessel wall, tying branches too close
- Forceful dilation – keep under 200 mm Hg, otherwise will slough endothelium;
  - It leads to injured vessel wall that will have:
    - Leukocyte infiltration
    - intimal thicking via intimal hyperplasia
      - neointima, SMC ingrowth in intima, intimal matrix deposition
    - subintimal fibrosis

Endothelial disruption leads to the following effects:

- loss of fibirinolityc ability
- increased LDL uptake
- loss of NO/prostocyclin secretion

- Temperature – conflicting
  - 37 degrees C vs 2-4,
  - less prostocyclin & NO synthesis suppression with room storage…
  - Rutherfords companion recommends COLD
- Solution:
  - Warm whole blood better than cold crystalloid.
  - Papaverin prevents vasospasm

Hence: gentle dissection, under 200 mm, heparinized cold saline with papaverine

175.Expected patency of vascular grafts & procedures:

Iliac angioplasty:
2222 limbs
76% claudicannt
1 year – 85%
5 year – 70%

SFA – pooled results: TASC II

	1 year	3 year	5 year
PTA: stenosis	75%	60%	55%
PTA + stent: stenosis	75%	65%	-
PTA: occlusion	65%	50%	40%
PTA + stent: occlusion	70%	65%	-

i.e. stent offered about 5% improvement

5 year fem pop:

	Claudicant	CLI
Vein	80%	65%
AK PTFE	75%	50%
BK PTFE	65%	65%

I am puzzled as to why TASC II offers no explanation for patency for BK PTFE being equal for claudicants and CLI…

AK PTFE vs vein: several RTC analysis, at 5 years – TASC II results

Vein	75%
PTFE	50% (optimistic)

There is NO doubt that ABSOLUTE patency of AK vein graft is superior to PTFE…

Classic Veith RCT study:

4 year results	vein	PTFE	Stat significance
Below knee	68%	47%	0.025
Above knee	61%	38%	**0.25 (NS)**

Comment – low numbers, but trend was toward vein superiorty in above knee…

VA boston study, 2000
AK location, no DM, no women
5 year results
In this study, HUS was used as well and did better than PTFE…
(J Vasc Surg 2000;32:268-77.)

	claudication	CLI
Vein	80%	68%
PTFE	33%	37%

Klinert, 2004 Metaanalysis
At 4 years, there is 20% difference between vein and PTFE
Selected 4 quality RCT

69% vs 49% in favor of vein.

NOTE: 2009 Cochrane analysis on AK vs BK vein/PTFE is USELESS – no recommendation given, studies are poor, etc. Done by the RN, not MD. Heavy criticism of this study at 2009 Harvard Review course.

Limb based	5 year	10 year
Aortobifemoral: claudication	90%	85%
Aorto bifemoral: critical limb ischemia	90%	80%
Axillo-uni	50%	
Axillo-bi-fem	70%	
Fem-fem	75%	

1999 metaanalysis:
Improvement in patency of prosthetic grafts (but not autogenous) with antiplatelet tx.

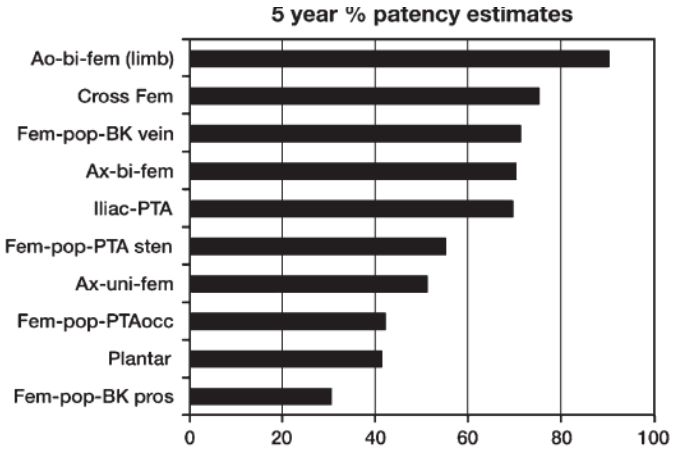


Fig. F8. Results summary: Average results for surgical treatment. Ao-bi-fem – Aortobifemoral bypass; Fem-pop – femoropopliteal; BK – below knee; Ax-bi-fem – Axillobifemoral; PTA – Percutaneous Transluminal Angioplasty; Ax-uni-fem – Axillounifemoral bypass; pros – prosthetic.

5 year patency according to TASC II:

- 90% group:
  - Aorto-bi-fem
- 70% group:
  - Fem-fem
  - Fem-pop BK vein
  - Iliac-PTA
  - Ax-bifem
- 50-55% group:
  - Ax-unifem
  - SFA angioplasty for stenosis

- 40% group:
  - SFA angioplasty for occlusion
  - Fem-distal bypass
- 30% group:
  - Fem-pop-BK prosthet

Other info re patency: OPEN SFA BYPASS RESULTS according to TASC II

	Claudicant	CLI
Vein	80%	65%
AK PTFE	75%	50%
BK PTFE	65%	65%

Results of SFA endovascular patency according to TASC II, cumulative

	1 year	3 year	5 year
PTA: stenosis	75%	60%	55%
PTA + stent: stenosis	75%	65%	-
PTA: occlusion	65%	50%	40%
PTA + stent: occlusion	70%	65%	-

-

176.Complications after infrainguinal revascularization:

Table F8. Cumulative observed morbidity outcomes for bypass in critical limb ischemia

Parameter	*Short term (first year)*	*Long term (3–5 years)*
Mean time to pedal wound healing	15–20 weeks	–
Incisional wound complications*	15%–25%	–
Persistent severe ipsilateral lymphedema§	10%–20%	Unknown
Graft stenosis**	20%	20%–30%
Graft occlusion	10%–20%	20%–40%
Graft surveillance studies	100%	100%
Major amputation	5%–10%	10%–20%
Ischemic neuropathy	Unknown	Unknown
Graft infection†	1%–3%	–
Perioperative death (primarily cardiovascular)	1%–2%	–
All death (primarily cardiovascular)	10%	30%–50%

*Not all requiring reoperation.
§Not well-studied.
**Greater in series of composite and alternate vein conduit.
†Greatest in prosthetic grafts.

ESRD is special case: for infrainguinal bypass….

	ESRD with ESRD	CLI with NO ESRD
Perioparative mortality	5-10%	2-3%
Survival	50% at 3 years	50% at 5 years
Wound & incision healing	Poor	Better

Those who persue aggressive indiscriminate revascularization of these pts are not practicing evidenced based medicine. (Rutherford)

ASIDE:
Cryopreserved vein for infrainguinal bypass:
1 year patency – 28-80%
2 year patency – 19-42%
Early failure – 17%
Late aneurysm – 5-30%
Limb salvage – 60-100%

So patency rates are NOT sterling, so make your own conclusions.

177.Graft surveilance:

- 15-20% of graft fail within 5 years
    - 10 % - see failure in first 30 days:
        - Poor quality vein
        - Poor anastomosis
        - Poor outflow/inflow
- **In up to 60% of ealy failure are due to graft lesions**
    - Poor vein vs valve vs twist vs anastomotic narrowing
        - **Technical errors responsible for 4-25% graft failures**
        - **At exploration, 50% of grafts have no apparent problems.**

- Failure in first 2 years:
    - Mostly due to intimal hyperplasia

- IT is critical to detect failing graft:
- Most veins don't stay open after mechanical thrombectomy or thrombolysis
    - Patency after thrombolysis is 20% at 1 year
    - At 1 year,
        - 50% are amputated,
        - 25% have rest pain,
        - 15% dead…
        - But limb salvage may be better
    - Surgery is a beter option if:
        - Taken to OR immediately
        - Mechanical problem is detected (twist, valve cusp)
- Traditional ways to detect failing graft lack sensitivity
    - Recurrent symptoms
    - Loss/reduction of pules
    - ABI change of > 0.2
        - PPV 12-34%, i.e. LOW
- 8% of lesions develop in first year
    - Lesions developing in first 3 months are more threatening than late ones

- There is 2-4%/year late graft stenosis IN ADDITION to 10% life time risk of progression of atherosclerosis.

- First surveylance duplex study: at discharge or 1 month,
    - then biannually for 2 years,
    - then annually for life
- Check GVF, PSV in the lesion. Grade risk level.
- Revise grafts at high risk

RISK LEVEL	HIGH-VELOCITY CRITERIA		LOW-VELOCITY CRITERIA		ABI REDUCTION
Highest	PSV > 300 cm/sec or Vr > 3.5 *Or* EDV > 100 cm/sec	*and*	GFV < 45 cm/sec	*or*	>0.15
High	PSV > 300 cm/sec or Vr > 3.5	*and*	GFV > 45 cm/sec	*and*	<0.15
Intermediate	PSV 180-300 cm/sec or Vr > 2.0	*and*	GFV > 45 cm/sec	*and*	<0.15
Low	PSV < 180 cm/sec	*and*	GFV > 45 cm/sec	*and*	>0.15

ABI, ankle-brachial index; PSV, peak systolic velocity; Vr, ratio of the PSV within the lesion to the PSV in a proximal normal segment of the graft; GFV, graft flow velocity; EDV, end-diastolic velocity.

- Grafts at risk: these need angio and likely revision….
    - low-flow velocities in the graft
        - peak systolic velocity < 45 cm/sec throughout the graft
    - PSV > 300, EDV > 100

- These need to undergo angiography:
    - **drop in ABI exceeding 0.15 in the absence of detectable graft lesions**
        - need arteriography to search for inflow, outflow, or missed graft lesions

- Grafts with low and intermediate risk – follow: q 3 months
    - 50% of these will progress.

Vein graft surveillance improves long-term vein graft patency by approximately 15%

- RCT by Lundel, multiple observational studies
- Cost effective (multiple studies)

In general, open repair with patch or interposition gives best results.

178.Iliac PTA – risks of procedure:

- Dissection
- Rupture
- Distal embolisation
- Thrombosis
- Restenosis
- False aneurysm
- Infection (if use stent)

179.Primary and secondary patency:

Primary –
- % of grafts that remain patent, without failure, over a given time
- Includes grafts that were rescued PRIOR to occlusion (assisted 1^{o} patency)

Secondary -
- % of grafts that remain patent AFTER it was restored following failure
- Does NOT include procedures when new graft is inserted

Life table method:
- Can be used for data sets MORE than 30 pts only
- Initially used to measure survival of cancer pts and effect of therapeutic intervention
- Events (graft failures) are grouped into intervals
- Survival is then calculated for each interval
- Major assumption:
  - all withdrawals occur in the middle of the interval
  - failure rate is stable over the interval duration

Failure rate= number of failures/(number at risk – ½ number of withdrawals)

- Curves are straight lines, not steps

Information needed for life table:
- Interval period
- Number of pts at risk for each interval
- Number of pts withdrawn per interval
- Number of failures per interval

Cumulative patency
- Cumulative patency for 1st interval = Success rate = 1 – failure rate
- Cumulative patency of the 2nd interval = success rate for 1st interval X success rate for 2nd interval

Kaplan-Meier Method:
- Product-limit method
- Date is not grouped in intervals
  - Or interval are very small and contain only one observation point
- Event occur at individual failure points
- No corrections are needed for withdrawals
- Graphically represented as stair –cases
  - Between events, nothing is known about the failure rate – it is assumed to be 0…
- Appropritate for ANY data size

Comparison between patency: use log-rank test of significance

180. Tests for evaluation of the donor iliac artery prior to fem-fem:

- Palpation of fem pulse
- Thigh-brachial index
- Thigh plethysmography – brisk upstroke and good excursion
- Duplex of iliac –
    - Triphasic
    - No PSV elevation
    - Spectral window preservation
- BIPLANAR angiography – evidence of anatomic stenosis
    - If questionable lesion – walking ABI or angio with vasodilation
- Direct Fem artery pressure measurement
    - Normal – less than 5 mm Hg gradient with CL side
    - Normal – less than 15% drop with vasodilation

181. Popliteal aneurysm:

- Male, elderly pts – 6-7th decade
- 50-70% are bilateral
- 30% have AAA
- 30% have fem Aneu
- 40% involve tib/per trunk

- Classification:
    - Fusiform vs saccular
    - Prox, mid, distal
- Asymptomatic in 37%
- If symptomatic:
    - Ischemic (progressive embolization, thrombosis) – 55%
    - Local effect 6%
        - Pop mass
        - Pain
        - Venous compression/edema/phlebitis
    - Rupture 1.4%
- When to fix:
    - All symptomatic
    - Asymptomatic > 2 cm
- OR:
    - Endo vs open
        - Single small RCT showed equivalence of results
        - Antonello, 2007 update

		Open 27 pts	**Endo 21 pts**	**p**

12 months	1°	100%	86.7%	NS
	2°	100%	100%	NS
72 months	1°	71.4%	88.1%	NS
	2°	88.15%	85.9%	NS

- Open:
  - Medial vs posterior approach
  - Huang, 2007, 4 year
    - Primary patency 85% (vein), 50% (ptfe)
    - Secondary patency 94% (vein) vs 63% (ptfe)
- Consider intraoperative thrombolysis/isolated limb perfusion

If elective, <1% mortality and limb loss
If emergent 5% mortality and up to 20% limb loss
IF see both AAA and popliteal – fix popliteal aneurysm first – otherwise risk thrombosis in periop period. (Rutherford)

182.Approaches to pop aneurysm repair: advantages and disadvantages

Medial:
ADVANTAGES:

- easy GSV harvest,
- supine/not prone position – easier for GA, no risk for retinal ischemia
- familiar approach,
- can get far remote and distal access to SFA/trifurcation
- avoid tib/peroneal nerves

DISADVANTAGES:

- Saphenous n. injury
- Difficult to get into aneurysm
- Long scar

Posterior:

ADVANTAGES

- Easy access to aneurysm – ligate all the feeding branches
- Easy way to decompress aneurysm
- Can do interposition repair
- Easy access to LSV
- Can go further distally – just split the gastrocnemius

- Smaller cosmetic incision

DISADVANTAGES;

- Unfamiliar territory
- Pop vein and tib/per nerves in the way – injury potential
- Can't go far proximal
- Can't easily get GSV
- Prone position – more difficult to GA (risk to eyes and airway loss)
- Wound healing problem

183. Goals of peripheral aneurysm treatment:

- Eliminate source of atheroembolism/thrombosis/rupture
- Eliminate mass effect
- Maintain distal perfusion
- Prevent recurrence

184. Nerves encountered in popliteal a. exposure:

- Tibial
- Medial sural – off tibial
- Common peroneal
- Lateral sural - off common peroneal
- Medial receives a branch off lateral branch, and from median (lesser) sapheous nerve

Aside:

- Saphenous n. – medial calf/foot
- Sural n. – lateral foot
- Superficial peroneal – foot dorsum
- Deep peroneal – 1st web
- Tibial – plantar surface

185. Cystic adventitial disease:

- Mucinous cyst/gangion in the arterial wall
- Mc location popliteal,
  - Second most common site - EIA
  - Also seen in CFA, radial/ulnar, GSV at SFJ
- Male more common than female
- Theories:
  - Origion form Ganglion –
    - Since lesions are seen near the joint
  - repetitive trauma

- systemic disorder
- developmental – the most plausible theory of all
  - inclusion of mesenchymal mucin secreting cells in adventitia during aberrant embryonic development

- Note: apart from developmental, there is no support to the theories presented here

- Treatment:
  - cystotomy - cyst evacuation (NOT resection)
    - open - BEST compared to percutaneous
  - replacement if thrombus/fibrosis/aneu

186. Angio findings in adventitial cystic disease

- Smooth tapering stenosis
  - concentric cyst – hour glass appearance
  - eccentric cyst – scimitar sign
- arterial occlusion (seen later)
- Proximal artery free from atherosclerosis

187.Causes of popliteal a. occlusion in order of frequency.

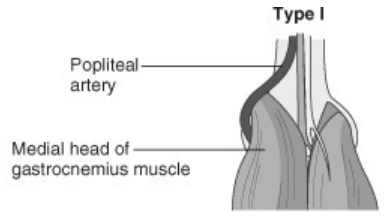
- Proximal source:
  - Embolus
  - Aortic dissection
- Local source:
  - Thrombosis of atheroscleoritic lesion
  - Aneurysm
    - In old – degenerative AAA
    - In young – think infected aneurysm (IV drug use)
  - Pop entrapment
  - Cystic adventitial disease
  - Trauma (knee surgery, disarticulation)
  - FMD
  - Buerger's
  - Pseudoxanthoma elasticum
  - Persistent sciatic artery

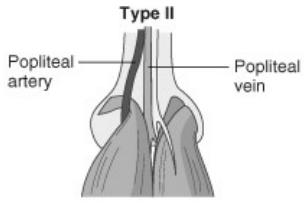
188.Types of popliteal entrapment:

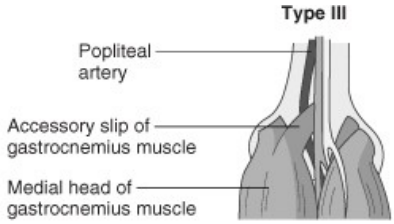
- normally pop a. goes between heads of gastroc (HOG).

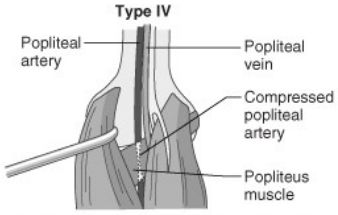
- Type 1 – pop. a. lies abnormally: medial to normally placed medial HOG
  - NOT the most frequent type
- Type 2 – pop. a. is compressed by Abnormally (more laterally) placed HOG

- Type 3 – pop. in normal position, compressed by abnormal lateral slip of muscle/tendon
- Type 4 - pop a. is compressed as it runs deep to popliteus muscle
- Type 5 – any type PLUS pop vein +/- tibial n. involvement
  - Vein is involved in 1/3 of cases
- Type 6 – functional – i.e. no abnormality is identified but pop artery is compressed in certain positions









When examining, make sure:

- Ascertain absence of atherosclerosis RF and vasculitis:
  - HTN, DM, HL, smoking, FH of premature asc, homocysteinmeia
- Examine contralateral extremity

- Evaluate arterial, venous and neurologic status

- Check out pulses during stress test: tensing of the gastroc
  - Passive dorsiflexion
  - Active plantarflexion
    - Note: stress test is positive in 1/3 of normal individuals
- Angio findings:
  - Medial deviation of pop artery
  - Mid pop segmental occlusion (with stress)
  - Post-stenotic dilatation

- All pts with type 1-5 should be fixed – progress to occlusion/aneurysm
- For type 6 – indications is less clear…
  - may be entrapped by slightly malpostioned soleus/medial HOG
  - may consider division of the muscle head if no other explanations of sxs.
- Resect artery if: fibrosis, aneurysm, thrombus on intimal surface

189. Causes of early limb edema after a femoro-tibial GSV bypass

- At one year, 10-20%
- Disruption of lymphatics (most important)
  - Elevate, compression dressing
- Surgical trauma – inflam response, increased IF accumulation
- Venous interruption
  - Not a contributing factor unless there is
    - DV incompetence
    - DVT

190. Causes of compartment syndrome:

- Definition of Compartment syndrome:
  - Pressure increase in the constrained space,
  - Most commonly due to interstitial fluid swelling

Causes:

- Restricted compartment volume:
  - Casts
  - Constrictive dressing
  - MAST
  - Circumferencial eschar
- Increased volume of compartment:
  - Space occupying lesions
    - Hematoma
    - Abscess
    - Synovial fluid (ruptured Baker's cyst
    - Inadvertent infusion of crystalloid

- Swelling of soft tissue, primarily muscle
  - Ischemia-reperfusion injury
  - Venous outflow obstruction
  - Trauma

- Risk factors for CS:
  - >6 h ischemia
  - Combined vein and artery trauma
    - Particularly if accompanied by vein ligation
  - Popliteal artery injury
  - Massive soft tissue injury

Sxs:

1. pain out of proportion to the physical findings, getting worse
2. distal motor/sensory dysfunction according to compartment
   a. weakness of dorsiflexion and numbness of 1st dorsal web space in LE
   b. weakness in wrist extension and numbness in 1st web space in UE
3. muscle pain, worse with passive flexion/extension

Arterial perfusion pressue – gradient between MAP and interstitial pressure.

- Intervention is recommended when compartment pressure is within 20 mm Hg of the diastolic or 30 mm Hg of the MAP for > 4 hours.
- Absolute numbers are generally misleading…

Indirect test to RULE OUT CS:

- do DUS of the veins in the affected compartment – preservation of normal respiratory phasicity will r/o CS… p.1061
  - legs – flow augmented with expiration and inhibited with inspiration
  - arms – the opposite

Tx:

- Early recognition:
- 4 compartment fasciotomy in lower extremity
- Upper extremity Curvilinear volar incision
  - antecubital fossa->palm, open up Carpal tunnel
- Other places – longitudinal over involved compartement
  - E.g. over dorsum of metacarpal/metatarsal bones

Aside:

- **chronic compartment syndrome** – repeatative lower extremity anterior compartment (most common) pain with minimal exertion.
- Normal circulation.

- Ds – tenderness over compartment and loss respiratory phasicity of veins in the compartment.
- Tx: cosmetic incision and fasciotomy curative.

191.Calf compartments:

- Anterior: TA, EHL, ED, Peroneus tertius, deep perineal n.
- Lateral: Peroneus Brevis/Longus
- Post Deep: TP, FHL, FD, popliteal, tibial n.
- Post super: GC, Soleus, plantaris

192.Acute limb ischemia classification:

- Category 1:
  - viable, no motor/sensory loss, arterial Doppler present
  - may do angio
- Category 2a:
  - marginally threatened, mild sensory loss, no motor loss, no arterial Doppler signal, venous Doppler present
  - may do angio, but consider surgery soon
- Category 2b:
  - immediately threatened, mild motor/sensory loss
  - have to go to OR, angio on table if needed
- Category 3:
  - irreversible, anesthetic, paralytic, no arterial AND venous Doppler.
  - Too late, amputation

Signs suggestive of irreversible loss:

1. Fixed cyanosis
2. Marbled mottling
3. Myositis: dough consistency, calf pain, muscle rigidity
4. Anesthetic sensory loss
5. Complete paralysis
6. No spontaneous venous doppler

193.Amputation level and bones:

- BKA – tib/fib
- Syme – distal tib/fib, 0.5 cm prox to ankle joint
- Lisfranc – forefoot disarticulation at tarsal-metatarsal joint
- Chopart – midtrasal disarticulation – at talo-navicular joint

194.Define different types of amputations:

Failed infrainguinal revascularization will NOT affect level of amputation, nor will it increase perioperative amputation mortality. However, it may prolong wound healing and long term survival of the pt.

- Toe –
    - OM, neuropathy confined to distal/mid phalanx,
    - fishmouth incision, preserve small button of prox phalange over MT head,
    - no prosthesis needed
- Ray –
    - localized gangrene/infection to MTP crease or involving head,
    - racquet incision, divide neck at 45 degree plantar bevel,
    - orthotic may improve balance/minimize skin trauma
- TMA –
    - total plantar flap,
    - gangrene/ischemia of multiple toes sparing plantar skin
    - may need to modify shoe with steel shank to allow normal "toe" push off and prevent excess dorsiflexion
- BKA –
    - INDICATION: Gangrene/infection/unreconstructable RP/non-healing ulceration and CAN'T do more distal amputation.
    - MC posteriorly based myocutaneous flap on GC and Soleus muscles.
    - Need prosthesis. 40% energy increase, 30-70% ambulate again
- Knee disarticulation –
    - occasional use,
        - if good blood supply, but has contracture or can't do BKA.
    - Difficult prosthesis.
    - Types of flaps:
        - Long anterior,
        - equal ant & post,
        - equal med & lat flaps.
    - Condyles – must be transected to allow prosthesis
- AKA -
    - in bed ridden,
    - Gangrene/infection/unreconstructable RP/non-healing ulceration and CAN'T do more distal amputation
    - fish moth, ant & post flap
    - the longer the shaft, the better the ambulation potential
    - up to 50-70% energy increase
    - 10-30% ambulate
- Hip disarticulation (rarely performed)
- Aside:

- Finger amputations are better dealt with by plastic surgeon
- Replant may not be as functional as amputation
  - One study, showed 90% excellent functional outcome with finger amputation vs 44% with replant
- Having said this, do not rush to ray amputation – it will decrease grip strength and ability to supinate.

195. Tests to use to select level of amputation:

- In general ratio of AKA vs BKA is 1to 1
  - Clinical:
    - >60 mm ankle – ok for BKA
      - Accuaracy 50-90% I.e. could be a coin toss!
    - > 40 mm toe – ok for BKA
  - Angiography (poor)
  - Skin temp > 90F
  - Segmental and toe pressure
  - Transcutaneous oxygen saturation (most accurate)
    - < 30 mm Hg, probably ok
  - Intradermal isotope blood flow
  - Other:
    - Skin fluorescence & Laser Doppler
    - Skin perfusion pressure

196. Complication of below knee amputation:

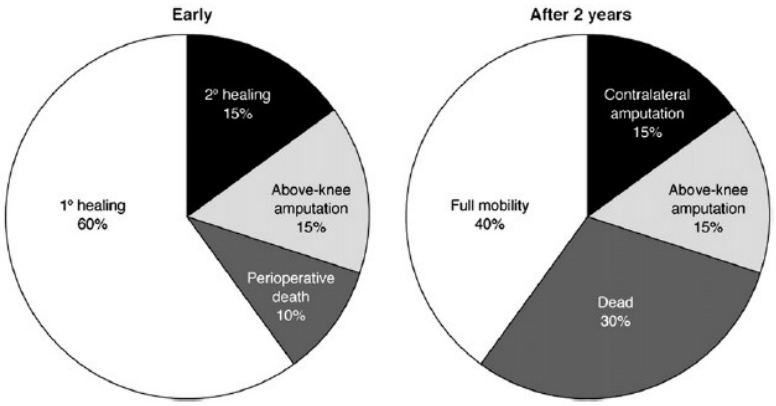


Fig. A6. Fate of the patient with below-knee amputation.

TASC II

- Systemic:
  - Death
    - Early 10% for BKA,15% for AKA
      - ESRD, sepsis, acute ischemia DOUBLE mortality

        - Hence, physiologic (dry ice) and two stage amputation
    - At two years 30%
- MI
- PE/DVT 40%
- Pulmonary 10%
- Renal insufficiency
- sepsis
- stroke
- depression – seen in 35%

- Local:
    - Early
        - bleeding/hematoma
        - pain – phantom vs stump
            - 70% have it post op
            - 25% pain severe
            - Eventually settles
        - stump non-healing 15%
        - stump infection – 30%
        - revision to AKA – 15%

    - Late:
        - contralateral amputation 15%
        - above knee amputation 15%

        - MSK problems:
            - Flexion contracture
            - bone spurs and osteoporosis
            - adherent scar
            - stump edema/congestion
            - excessive residual soft tissue
            - callous and cyst
        - neuromas
        - failure to rehabilitate
            - only 40% walk at 2 years
            - rutheford is more optimistic
                - 50% have SOME ability to ambulate at 1 year

197.Compare arterial, venous and neuropathic ulcers.

- Arterial – dorsum foot/distal, no bleeding with manipulation, irregular edge/poor granulation, no surrounding inflammation, atrophic changes/no pulse, pain worse at night/better with dependency/worse with elevation.

- Venous – gaiter area, venous ooze on manipulation, shallow round edge/good granulation, surrounding inflammation, lipodermatofibrosis/pigment, mild pain, relieved by elevation
- Neuropathic – under calluses/pressure point/1&5 MPJ, brisk bleeding with manipulation, punched out, deep sinus, surrounding inflammation, has neuropathy, normal circulation, no pain.

So:

- Consider location, response to manipulation, blood supply, pain response to position, stigmata of associated disease

198. Pathophysiologic mechanisms in diabetic foot:

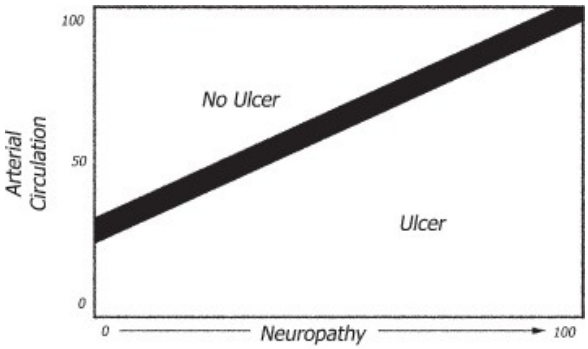
- Arterial ischemia in tib/fib vessels
  - There is NO microcirculation problems contrary to initial animal experimentations
  - Profunda and crural vessesl are affected
- Neuropathy:
  - loss of noci/proprioception leading to trauma
  - Lumbical muscle denervation
    - Foot architecture messed up:
      - cavus & claw toe
      - flexture contracture
  - Loss of neuroinflammatory response:
    - response to infection is blunted
    - Normally, Pain/infection causes release of neuropeptides (substance P) that release histamine, increase permeability and attract cells to site of injury.
    - Neuropeptides ARE released but there is no humeral response…
- Sympathetic dysregulation:
    - AV shunting (less perfusion to the tissue, more to the skin
    - no sweat -> dry skin
- Glycosylation of basement membrane and tissue proteins ->
  - Limited joint mobility
  - Extravasated albumin cause tissue edema
    - difficult to heal incisions
    - note: there is NO effect on oxygen or nutrient diffusion

All leads to propensity to repeat trauma, loss of protective reflexes and weight bearing redistribution. Decreased arterial circulation complicates matters further.

If no palpable pulses – need angio. If diminished pulses – i.e. less than triphasic – non-invasive study first to see if DSA is required.

Direct quote from Rutherford:

"The presence of neuropathy generally requires a revascularization under condition of perfusion that would not require revascularization in the absence of neuropathy. "



Non-invasive tests of arterial inflow, including TC O2 sat, can be poorly predictive of healing under these circumstances.

Osteomyelitis vs Charcot foot :

- Charcot foot: progressive degenerative osteoporosis of foot bones secondary to neuropathy. Leads to deformities, foot swelling and erythema.
- Charcot foot presents with swelling and erythema.
- 24h bed rest without antibiotics should settle Charcot foot, but not osteomyelitis.

Evaluating pt wth Dibetic ulcer/foot:

- Drainage sxs
- Usual PVD questions
- Sepsis review
- RF and RF Tx

- On Exam:
- Pulses – dp and pt gotta be triphasic
- ID ulcers/sinus, dd arterial/venous/neurophatic
- Probe/debride necrosis
- Look for exposed bone
- Do plain x-ray looking for gas…

Overall principles in treating diabetic foot:

- Debride/drain obvious infection
- Control systemic sepsis/hyperglycemia

- Assess for Asc occlusive disease, as well as neuropathy
- Define status of FOOT arteries
- Restore maximum perfusion possible
- Look for, drain, debride residual infection/necrosis
- Manage open wounds with dressing

CAROTIDS

199.Branches of External Carotid artery:

- Anterior
    - Superior thyroid
    - Lingual
    - Transverse Facial
    - Facial
- Posterior
    - Occipital
    - auricular
- Ascending
    - Ascending pharyngeal
- Terminal
    - Superfical temporal
    - Internal maxillary

200.Internal carotid artery anatomy:

- Cervical segment –no branches
- Petrous segment – small pterygopataline to internal maxillary
- Cavernous segment – aka carotid siphon
    - Gives off ophthalmic artery
- Cerebral segment
    - MCA
    - ACA

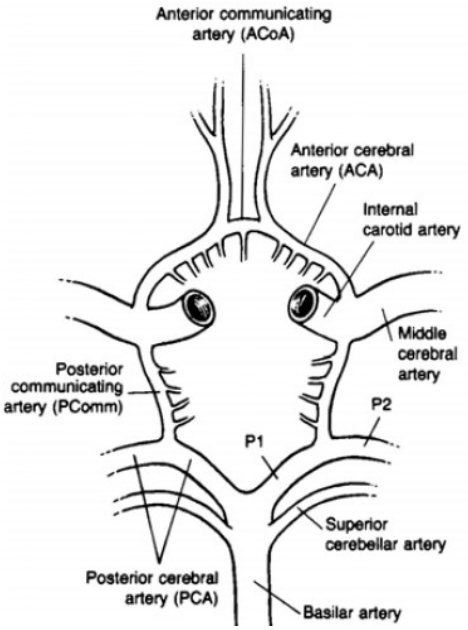
Circle of Willis is present in complete form in only 20% of pts.

201. External carotid to internal carotid a. collaterals:

- Intracranial-extracranial
    - Pre-willisian anastomosis
        - Orbital-ophthalmic

- Meningo-hypophysial
- Occipito (ECA) to atlantic branch of verteb ral
- Deep /ascending cervical (ECA) to lower vertebral
- ECA to ECA across midline
- Rete mirabile
    - Dural arteries to brain surface

- Small inter-territorial communications
    - Leptomeningeal collaterals
        - Between terminal cortical branches for main cerebral arteries across the border of vascular territories



202. Clinical presentations of cerebral syndromes:

TIA - <24 h duration
Crescendo TIA – same sxs but increased frequency, ok between episodes.
Stroke in evolution – stars as TIA but extent of deficit and frequency is increasing with overall worsening neuro status

Gaze toward side of lesion…

- ICA – monocular ipsilateral visual symptoms plus MCA
- MCA –
    - CL hemiparesis & hemianesthesia
    - **Aphasia – can't find words**
        - **L sided territory**
    - **Dysphagia – can't pronounce words, slurrs**
        - **R SIDED TERRITORY**
    - CL neglect
    - Apraxia (can't carry out purposeful movement)
- PCA –
    - homonymous visual field loss
    - denial of blindness - Anton's syndrome☺
        - have to have bilateral vertebral occipital lobe phenomenon
        - can't see with unilateral stroke…
    - visual hallucinatinos
    - loss of reading (alexia)
    - confusional state
    - amnesia
    - discoordination
    - **blind, hallucinate, alexic, amnestic**

- ACA –
    - CL sensory changes
    - inability to walk (gait appraxia)
    - apathy, mutism
    - **can't walk, not spontaneous, urinary incontinent**
- VB
    - crossed findings (ipsi CN palsy and CL extremity sensory/motor deficit) – in brain stem
    - vertigo, disorientaton, disequilibrium
    - speech problems
    - hiccups
    - **hiccups and disequilibrium**

Table 136-2. Signs and Symptoms of ICA-Related Ischemic Events

VASCULAR TERRITORY	SYMPTOMS	SIGNS
ICA	Transient monocular visual changes lasting about 3-5 minutes and presenting as	Amaurosis fugax
		Rarely Horner's syndrome
	Blindness	

	Blurry or foggy vision Blind spots, colors, shapes Tunnel vision In only 10-15%, curtain-like blindness ascending or descending throughout visual field Rarely headaches	
MCA	Difficulties in comprehension or language production, difficulties performing motor tasks or calculations, incoordination, numbness/tingling on one side of body, weakness in arm and leg	Aphasia, **head and eye deviation toward lesion, apraxia, neglect, anosognosia,** contralateral sensory deficit, contralateral paresis, confusional states
ACA	Numbness/tingling on one side of body, weakness of leg more than arm, difficulties walking	Contralateral sensory deficit, contralateral paresis, **apathy, mutism, reduced spontaneity, gait apraxia, urinary incontinence**

203. Visual syndromes:

- Basics:
  - occipital cortex, then to midbrain, then to chiasma, then optic nerves.
  - Pre-chiasmal fibers – outside travels to ipsilateral temporal retina,
  - fibers supplying nasal retina are on the inside and cross over.
- Optic nerve severed – complete blindness
- Chiasm injury:
  - longitudinal cut – cut inside cross over fibers that supply nasal retina –
    - bilateral heteronymous hemianopsia – bi temporal blindness
  - transverse partial cut into fibers that travel on the outside that supply temporal retina –
    - Contralateral nasal blindness
- Prechiasm fibers – contralateral homonymous hemianopsia –
  - R brain is watching L side – L nasal, and R temporal fields are affected

204. Indication for carotid duplex:

- See Doppler notes in 2nd section…
- Indications:
  - Bruit in asymptomatic pt
    - Seen in 5% of pts above 50 yoa

- Present in only 20% of pts with HD significant stenosis
- Amaurosis fugax
- Focal TIA
- Follow up of known >20% stenosis in asymptomatic
- Drop attacks (rare)
- CEA setting
    - CVA in candidate for CEA for OR planning
    - Intraoperative assessment of CEA
    - CEA without arteriography
    - Follow up after CEA (single study at 12/12 after surgery)

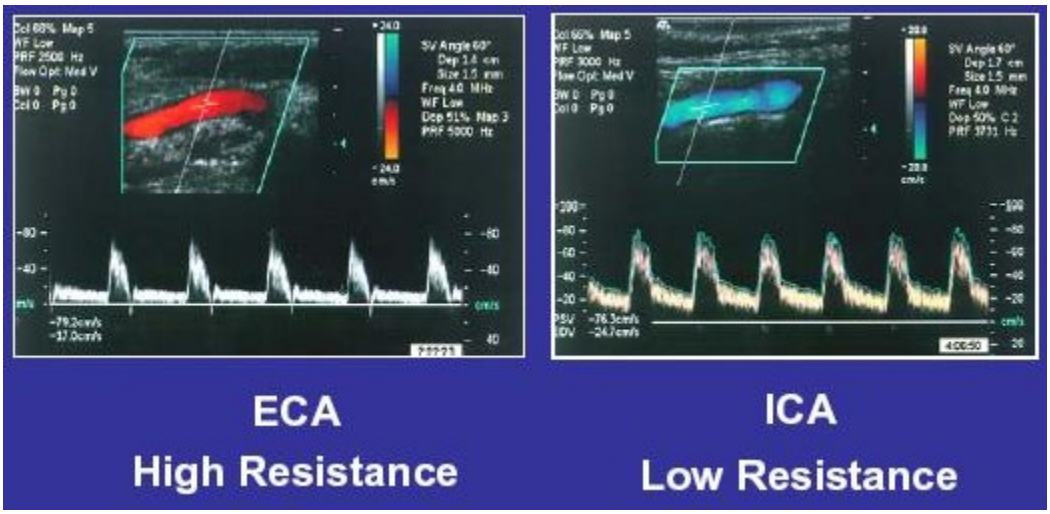
- Can trust US if:
    - No proximal CCA irregularities
    - No kinks/loops
    - No aneurysms
    - Good quality study

Indications for carotid angio:

- Can't trust duplex (kink, aneurysm, proximal CCA irregularity)
- In equivocal duplex findings
- Duplex can't show extent of disease
- Suspicion of tandem or arch lesion
- Uncommon carotid pathology
- Trauma
- W/u for stent

205.ICA and CCA Doppler profile

- CFA – three components –
    - forward flow, reverse flow, forward flow.



- ICA profile – biphasic because of elevation of baseline –
  - i.e. no reverse flow
    - NOT because of disappearance of the 3rd component of forward systolic flow
    - Happens because of low resistance in ICA.
      - 2nd – reversed – component is gone
    - Note – spectrum window is clear under the curve – no broadening…

  - Washington criteria – based on ECST, NOT NASCET angiographic correlation
    - ECST – outlines hypothetical normal carotid bulb and measures stenosis wrt this
    - NASCET – compare distal ICA to stenosis
      - May get negative stenosis figures
      - Compared to ECST, predicts less severe stenosis
  - Angiogram – underestimates stenosis
  - MRA probably equivalent to duplex US
  - High sensitivity study
    - Able to recognize an abnormality
    - needed for symptomatic pts
  - High specificity study

- abile to recognize normal artery
- needed for asymptomatic pts

206. Carotid duplex artery stenosis criteria: Washington Criteria

- Size of carotid:
  - At bulb 0.94 cm M, 0.92 F
  - ICA 0.55 cm M, 0.49 F

- Remember that it OVERESTIMATES the stenosis (ECST, not NASCET criteria)
- Remember, that it gives ranges that do NOT apply for NASCET (70% stenosis) .
- May be applicable to ACAS (BY ANGIO 60% stenosis)

- Normal: no plaque, smooth walls, boundary layer separation in bulb
- < 15 % mild SB
- 16-49% marked SB, no systolic window
- 50-79% PSV >125, PDV <140 cm/sec, marked SB, ICA/CCA > 1.8
- 80-99% PSV >125, PDV > 140 cm/sec, poststenotic turbulence, ICA/CCA > 3.7
- Occlusion: no flow
  - May be wrong in 3% of cases – hence ALWAYS confirm this with angio or MRI.

207. Consensus panel on US criteria on carotid stenosis:

- These are more practical then Washington – based on Nascet technique and range of measurements of stenosis:

- <50%
  - PSV<125
  - EDV<40
  - Ratio <2
- 50-69
  - PSV 125-230
  - Visualized plaque
  - EDV 40-100

        - Ratio 2-4
    - >70
        - PSV > 230
        - Visible plaque
        - EDV > 100
        - Ratio >4

208. Other useful velocities measurement for carotids

- >60% stenosis – external Oregon validation with angio
    - PSV >260, EDV >70, ratio > 3.2.
    - Accuracy 90%
- NASCET > 70% stenosis
    - PSV >280, EDV > 80, Ratio >4
    - PPV 95%
- >80% stenosis
    - PSV >250, ratio >4
    - 90% accuracy for 70-99% range
- Intraop duplex assessment of CEA
    - Repair if PSV>200
- Subclavian artery stenosis
    - Retrograde (notched) vertebral flow
- No graded PSVs values vs occlusion for vertebral artery flows
- Vertebral steal:
    - See either reversal of flow or stalled flow
        - Pre-steal - Back (systoly) and forth (diastoly)

- Don't confuse with phasic flow in vertebral vein

209.Frequency of Surveillance of known asymptomatic stenosis

- Asymptomatic > 60 stenosis%
  - If PSV<175 cm/sec
    - Progression is 4% over 21 months
    - Image annually
  - If PSV > 175%
    - Progression 26% over 14 months
    - Hence image q 6 month

210.Mechanisms of stroke:

Rutherford:

- Ischemic (80%) – non painful
- hemorrhagic (20%) – painful
  - dd of ICH
    - trauma
    - tumor – primary vs met
    - HTN
    - Amyloid angiopathy
    - AVM
    - aneurysm

Ischemic:

- 20-30% are due to major Exracranial and intracranial cerebral vessels
- 30% are due to embolism (close to 50% in pts younger 40 yoa)
  - Unlike carotid origin, these are NOT territorial
- 40% of stroke – no known cause

Srokes happen because of:

- Embolism
- Thrombosis
  - Extracranial
  - Intracranial
    - lacunar
- Hypoperfusion

- Flow related phenomenon are due to decreased cardiac output and occlusion

Pathology leading to stroke:

- Atherosclerosis 90%
  - Artery-artery atheroembolism (MC)
  - thrombosis
- Embolism
  - Cardiac (AF, post MI, valve)
  - > 50% in pts <50 yoa
- Other:
  - FMD
  - Kinks & loops
  - Traumatic occlusion
    - Intimal dissection
  - Inflammatory angiopathy
    - takayasu, GCA
  - Aneurysm
  - Intracranial vessel disease:
    - Vasculalitis
      - PAN
    - Weird and wonderful:
      - moyamoya, fibrinoid necrosis, amyloidosis,
      - arteritis
        - CTD
        - allergic, granulomaout, infectious
        - P. 1883

Summary of Non-ASC causes: FMD/kinks/dissections/arteritis/lacunar

211.Lab and investigations for TIA:

NEW definition: **a transient episode of neurological dysfunction caused by focal brain, spinal-cord, or retinal ischemia, without acute infarction**. NOTE – no more 24 h time line is given

DD:

- Partial complex seizure
- Complicated migraine
- Demylienating process
- Metabolic (glucose, liver, lyte abnormality)
- Psychiatric - conversion disorder

Sroke in young: 40% are cryptogenic

- embolism

- Dissection
- FMD
- Moyamoya
- CTC
- TTP
- hypercoag

Preliminary BW:

- cbc (plt, RBC),
- Cr
- gluc, HgA1C,
- lipid profile,
- ESR
- fibrinogen, homocystein,
- PT/PTT/INR
- drug screen,
- rheum (RF, ANA, APL, lupus),
- hypercoag - if young
    - FVL, pn 20210, AT def, fibrinogen, homo, ACL/LAC, pn C&S, PAI-1, lipid profile and lipoprotein a.
- r/o mimickers: check lytes, gluc, LFTs, cardiac screen/holter,

IF ECG is abnormal, order ECHO.

- Ideally **diffusion weighted MRI** of the head, if not available CT head
    - According to the latest recommendations of the AHA/ASA 2009

Why CT? to r/o

- SOL
- hemorrhage,
- old strokes,
- lacunar strokes,
- check laterality (and likelihood of embolic disease).

however, DW MRI is best for TIAs...

- Doppler/imaging – within 24 h by guidelines, accredited lab

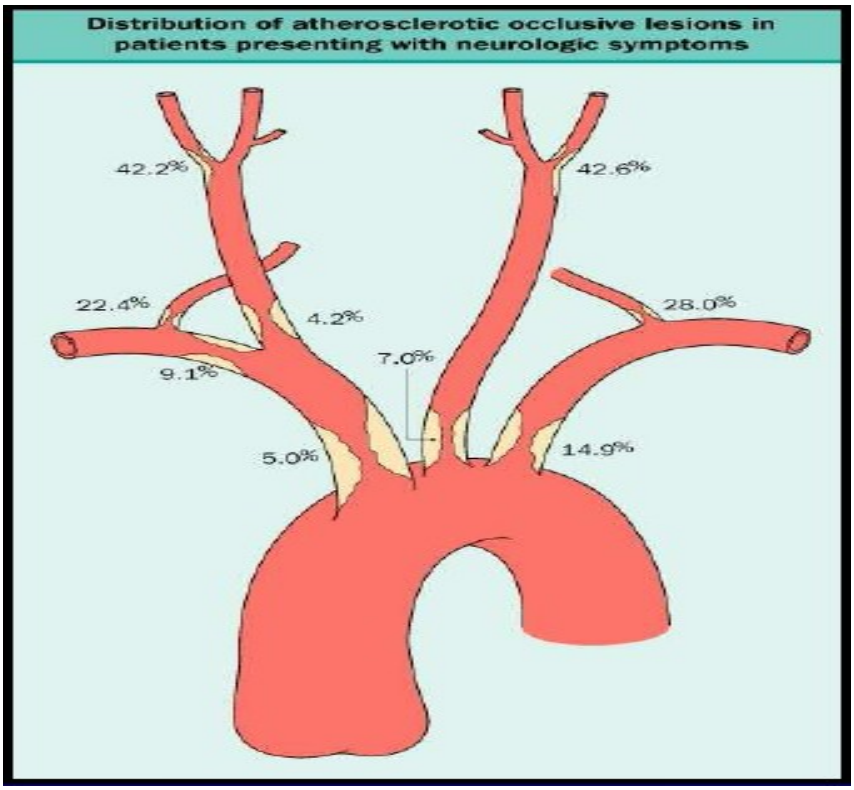
If pt had stroke:
tPA – indicated within 3 hours of stroke, not later. Seizures is contraindication to tPA.
Maintain sligh hypertension, no hypotension
Antiplatelets and statins, no heparin.
Urgent studies (as for TIA)

Aside:

- Auditory evoked potentials – only good for diagnosis of demyelinating disease (e.g. MS), not for anatomic localization.
- Only 30-50% of CVAs are preceded by TIA
- Mortality of initial stroke 30%
    - Second stroke 35%
    - Subsequent strokes 65%
- 6 months post stroke –
    - 50% have residual deficit,
    - 25% non-ambulatory,
    - up to 20% aphasic,
    - 25% require care,
    - 30% depressed

- Stroke in evolution:
    - Mortality 20-80%
    - Complete Recovery with medical therapy 4%
    - Recovery with surgery – up to 70%

- Asymptomatic stenosis:
    - Seen in 30% >50yoa
    - 80% of stenosis progress but prognosis is generally benign
        - If >80% with risk factors up to 35% risk of TIA/stroke/occlusion at 6 moths, 46% at 12 months
- Bruits
    - Seen in 5% of >50yoa
    - Only 20% of bruits are associated with >50% stenosis
    - Only half of HD significant stenosis have bruit
    - Stronger predictor of CAD



212.Recurrent CVA after TIA or CVA:

- Stroke after TIA
  - Highest in the first 2 weeks
  - Overall 6% per year
    - 10% 1 year, 16% at 2 years, 26% at 3 years,
    - then declines…
  - Stratifiation of TIA risk:
    - if risk factors present:
      - A=>60 yoa,
      - B=BP >140/90.
      - Clinical=hemispheric vs monocular,
      - D=duration > 1 hour,
      - D=DM
  - risk of recurrence may be as high as 8% in 2 days according to $ABCD^2$ criteria
    - Total scores ranged from 0 (lowest risk) to 7 (highest risk).
    - Stroke risk at 2 days, 7 days, and 90 days:
      - Scores 0-3: low risk
      - Scores 4-5: moderate risk
      - Scores 6-7: high risk

Guide to ABCD2 Total Score		Stroke risk after TIA at:		
Risk Group	(Score)	2-day	7-day	90-day
Low	0 - 3	1%	1.2%	3.1%
Moderate	4 - 5	4.1%	5.9%	9.8%
HIGH	6 - 7	8.1%	12%	18%

- Recurrent CVA after CVA:
  - 9% per year, steady… p. 1880

	1st year CVA recurrence	Annual recurrence	Recurrence declines?	Post CEA annual recurrence?
CVA	10%	9%	No	2%
TIA	10%	6%	Yes	<1%
asymptomatic	-	2-5%	-	0.3%

Moores data: If ulcer is > 40 mm^2 and cavernous – then annual stroke may be as high as 7.5%, even when no significant stenosis is present… Controversial.

213. Timing of CEA with respect to CVA

After TIA – calculate ABCD2 rates and may offer surgery same admission
After CVA – if neurologically recovered, within 2 to 4 weeks. 2009 Harvard review course recommends within 2 weeks but there are no RCT to support this…

Old adage: "if even happened withing a day, fix in a day; if within week, fix in a week; if within month – fix in a month" – may have some value…

214. Contraindications to CEA

- Carotid aneurysm
- Major previous stroke with significant neurologic dysfunction
- Acute major stroke
  - Premature blood flow increase may exacerbate deficit:
    - Area lost its' autoregulatory abilities
    - Blood brain barrier is disrupted
      - Edema
      - Intracranial bleed

Hence, may have to wait up 6 weeks then r/a neuro status.

- If complete recovery is seen earlier, Rutherford suggests may operate before 6/52

215.Complications of CEA:

M&M about 6% for symptomatic and 3% for asymptomatic:

IMMEDIATE:

- neuro - periop stroke/hyperperfusion,
    - see hyperperfusion on 3-7 day
- local - bleed/infection/Cranial n (hypoglossal, marginal) – most reversible ,
- systemic - HD instability/MI.

DISTANT –

- restenosis - 10% at 2 y, 17% at 10 y - int hyperplasia vs ASC... patch reduces incidence.
- Patch aneurysm

216.Conduct of CEA:

Goals – remove plaque, repair artery, avoid complications

- Consent: indications, risk and benefit discussion
- Beach chair position, neck extended, head turned
- Regional anesthetic, monitor CL arm activity and frequent neuro checks
- Ear-lobe – to nipple prep
- Cut along ant border of SCM
    - Skin
    - Platysma
    - Reflect SCM laterally
- Go in front of jugular vein, ID facial vein
    - Key to bifurcation
- Enter sheath, preserve ansa and vagus
- Dissect “patient away from artery”
- Loop CCA, ECA, sup thyroid, ICA
    - Preserve hypoglossal
- Inject bifurcation with lidocain, don’t dissect there
- Ask neuro to maintain BP, neuro check, heparin, circulation time
- Test clamp ICA with baker clamp, neuro check
- Clamp CCA, ECA, clip sup thyroid
- CCA-bulb-prox ICA longitudinal arteriotomy
- Penfield or Dallar tool plus Lower
- Transect plaque in CCA, work it up around ECA orifice, transect at the base of the ICA
- Eversion ECA endarterectomy
- Continue EA plane to ICA, feather out or tack with 7:0

- Clean surface
- Patch
- Unclamp ICA – then reclamp
- Unclamp ECA and CCA
- Finally unclamp ICA, neuro check
- Protamine, hemostasis, consider drain
    - Less hematoma
- Close platysma
- Staples
    - Remove these POD 1 and replace with stereostrips.

217.Indication for ECA endarterectomy:

- Occluded ipsilateral ICA with ongoing symptoms refererrable to the side where ECA stenosis is found
    - Amputate ICA, flush close after ECA endarterectomy

218.Difficult access to ICA: high ICA, difficult ICA

- Standard access – upper third of C2
    - Cut sternocleidomastoid branch of occipital artery
        - will allow to lift hypoglossal up
    - Extend incision to mastoid
- Divide posterior digastrics m – middle third of C1
    - Mobilize and elevate lower pole of parotid gland
    - Transect SCM at mastoid process
- Sublaxate TMJ (call for head and neck surgeon to help)
    - Don't dislocate
    - Need nasotracheal intubation
- Resect styloid process – upper half of C1 in 50% of cases
- Cut of posterior portion of the mandible ramus – gets you above C1 in 100% of cases
    - Lateral mandibulectomy
    - preserve inf. Alveolar nerve

- Finally, going retrojugular on initial approach exposing the carotid can get you surprisingly high… (Dr. Hajjar/Lewis)

219.Nerves encountered during CEA –

Injury rate – overall 10%... Most are transient.

- Hypoglossal 4% - tongue deviation, swallowing

        - MC in Nascet/rutherford
    - Vagus 3% - hoarse voice, airway problems
        - Osler course make it most common injury
        - Very often overlooked, hence making it likely the MC CN injury
    - Marginal mandibular 2% - drooping corner of mouth, drooling
    - Greater auricular – numb ear
    - Superior laryngeal – sustain high pitch
    - Spinal accessory – shoulder drop
    - Sympathetic chain – horner's

220.Nascet findings:

- 2 year results
- Asa vs ASA/CEA
    - i.e. NO statins. Medical management is outdated.
- Ratio of stenosis diameter to normal proximal ICA

- 26% vs 9% for 70-99%,
    - NNT 6 for < 75 yoa
    - NNT 3 for pts > 75 yoa
    - major/fatal stroke 13% vs 3% ARR 10%
- 22% vs 16% for 50-69%, NNT 17
    - No difference in major/fatal stroke
- Need complication rate < 5%

Aside:
ECT - > 70% stenosis
High 30 days surgical stroke of 7.5%
3 year 6% vs 11% fatal/disabling stroke in favor of surgery

Final conclusion:

1. CEA is offered in all symptomatic pts with severe stenosis (> 70%)
    a. Pts > 75 yoa have most benefit
2. In high-moderate stenosis (50-70%):
    a. for male – if good operative risk for OR
    b. for women – only for pts with persisting symptoms unresponsive to medical therapy, hemispheric TIA AND risk PVD factors
    c. There is no reduction in fatal/disabling strokes in Nascet study

Aside: if see clamp defect on completion angio – leave it alone, don't fix it unless it is HD significant, it will heal. (Osler)

Dr. H.J.M. Barnett: The appropriate use of carotid endarterectomy. JAMC, 2002

Symptomatic > 70% stenosis.
All pts are better off with surgery, but…
Most benefit seen in:

- Healthy elderly pts > 75 yoa
- Hemispheric TIA (NOT monocular)
- Pts with tandem extracranial and intracranial lesions
- Pts without angiographic evidence of collateral pathways

Perioperative risk is higher in the following pts, BUT surgery is still beneficial:

- Widespread leukoaraiosis
    - Significant risk factor for stroke
    - Poorly defined hypodense white matter lesions
        - Unlike sharply defined infracts
    - Overall benefit is smaller
- Occlusion of CL carotid artery
- Intraluminal thrombus

Symptomatic < 70% stenosis:
For most benefit smaller. The following pts may be HARMED, particularly if they have few Risk factors :

- Pts with monocular TIA
- Women

The presence of the following RF increase benefit:

- >75 yoa
- Male
- IC

Caveats:
CEA carries 2% incidence of Disabling stroke. So:

- Precise measurement of stenosis is essential
- Follow exclusion criteria:
    - Impending organ failiure
    - Serious cardiac dysfunction
    - Late stage cancer
        - These pts not likely to benefit…

221.Asymptomatic Carotid artery stenosis and ACAS findings:

- 39 centers, elite surgeons
- Good risk pts, mostly men
- 5 year results
- Most events occur after 3 years
    - Stat significance only after 5th year

- Stenosis > 60 %
  - But NO dependence between extent of stenosis vs benefit
  - 11% vs 5%, ARR 6%, NNT 17
    - men 12.1% vs 4.1% - ARR 8%
    - **women 8.7% vs 7.3% ARR 1.5% not stat significant**
- Fatal stroke and disabling stroke – no stat difference
- Need complication rate < 3%
- Females or > 80 yoa likely no benefit
- Recommended for good risk male pts with 3 year life expectancy, at least 60% stenosis. Results for women less certain.

ACST findings:
http://stroke.ahajournals.org/cgi/content/full/strokeaha;35/10/2425

- Larger study
- community surgeons, no elite requirements
- the only study to show stat sig difference in fatal/disabling stroke of 2.5%..
- females have TINY stat significant benefit, uncertain clinical benefit

So would I offer surgery on asymptomatic side?

- risk of stroke for asymtomatic < 80% is generally low
  - 1-2%/year,
    - 425 pt, stable 50-79% stenosis
    - 5 year cumulative risk of event is 5.4%
  - Recommendation to consider OR above 80% stenosis only
    - NNT is 83 according to Barnett paper (1 % yearly, i suppose)
    - 17 according to NEJM review (for 5% ARR over 5 years...).

- Risk of an event with stenosis above 80% - may be as high as 11% per year.....
- the greater the stenosis, the more RF there is, the greater the incidence of stroke..
  - some report incidence of stroke in >80% as high as 35% at 6/12
- also, only 30-50% of pts with CVA have an antedecent TIA
  - so can't rely on waiting for reversible symptoms of TIA as a warning system for incoming stroke…

- **Still, the most common event in ACTS/ACAS – MI, NOT stroke..**

- **ACAS acquired statistical significance for strokes only after a small burst of strokes after 5th year of follow up**
  - **i.e. no significance would have been reached if only followed for 4 years**
  - **only 1/3-1/2 of ischemic strokes were referable to ipsilateral carotid stenosis.**

Unfortunately, the very same factors that make stroke more likely without an operation, also increase perioperative risk of stroke…

- In ACAS, the following factors increased **perioperative risk of stroke, TIA, or death**
    - Anatomical factors:
        - Contralateral carotid stenosis >60%
        - Contralateral siphon stenosis
    - Clinical factors:
        - Prev CVA
        - Hypertension
        - DM
        - Female gender
        - Age >75 yoa
        - CHF
    - Procedural factors:
        - Combined carotid-cardiac surgery

i.e**. CL disease, PVD factors, female, age >75**

- At the same time, the following factors increase risk of having a stroke in asymptomatic stenosis if followed non-operatively:
    - Anatomical factors:
        - Soft, echolucent plaques
        - CL ICA occlusion
        - Silent ipsilateral infarction on CT
    - Clinical factors:
        - Htn
        - Dm
        - Smoking
        - Hyperlipidimia
        - Hyperhomocystein

I.e. PVD risk factors…

- so the decision to do surgery in asymptomatics is not an easy one…
    - on the one hand, multiple RF make pts more prone to stroke (from baseline 2% to as high as 11%)
    - on the other, the very same RF make pt more prone to perioperative CVA/death.

- Also, the benefits of CEA do not get realized until 3rd year post op and it does not appear to be cost effective in 80 year olds, or females
- Medical therapy is getting better. Jupiter trial showed 50% reduction in strokes and MIs with statin therapy at 2 years in asymptomatic pts.

- **Conclusion:**
    - If pt has
        - less than 3 year life expectancy,
        - CL occlusion/stenosis,

- has FEW vascular RF,
- is a female
- under 80% stenosis,

… medical therapy is beneficial. Particularly statins (Jupiter trial).

- Who is an ideal asymptomatic candidate for CEA?
  - male
  - < 75 yoa,
  - >80% stenosis
  - RF for PVD
  - No contralateral occlusion

- If decision to operate is made, it is imperative for the operator to have less than 3% combined M&M stroke risk to ensure gradual realization of 6% (at most) of absolute risk reduction over the next several years...

- So for the exam intent, unless you can prove to the examiner your record of asymptomatic CEA is low and you understand the risk variation with different RF thrown in the mix, I'd be careful suggesting CEA for an asymptomatic pt….

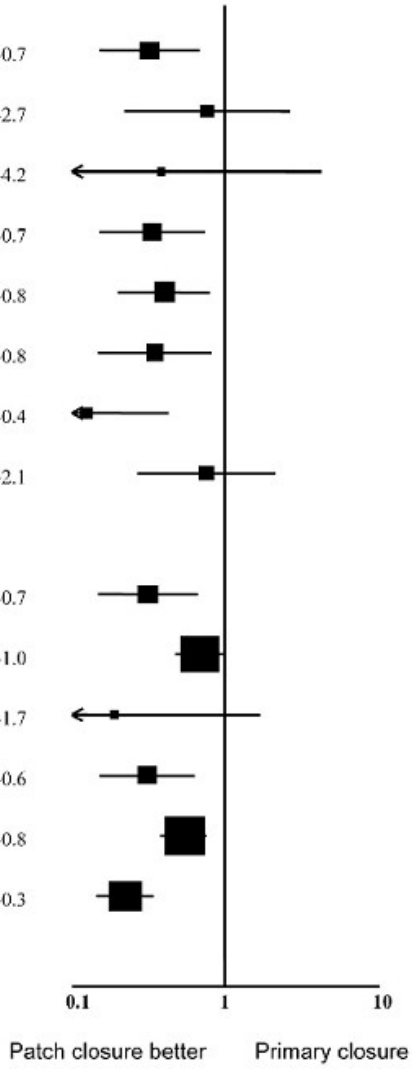
- Carotid Stent - still investigational. Crest –for symptomatic pts is not out yet… Reserved for high risk Symptomatic pts (technically or physiologically). For asymptomatic – use stent in off lable investigational setting only

222. Carotid patching – advantages and disadvantages:

- Disadvantages:
  - Longer clamp time
  - Potential blow out if use vein
  - Potential infection if use prosthetic

- Advantages
  - Lower restenosis rate
  - Reduced acute carotid thrombosis
  - Reduced periop neurologic rate
    - All these were suggested by Cochrane 2006 metaanalysis, 40 perioperative strokes are prevented per 1000 pts… No RCT done.

Outcome	Patch closure Events / Patients	Primary closure Events / Patients	Odds Ratio	95% CI	Significance
Perioperative 30 day results					
Ipsilateral stroke	10 / 625 (1.6)	23 / 480 (4.8)	0.32	0.2-0.7	p=0.001
All death	5 / 577 (0.9)	5 / 442 (1.1)	0.76	0.2-2.7	p=0.6
Fatal stroke	1 / 577 (0.2)	2 / 442 (0.5)	0.38	0.0-4.2	p=0.5
Any stroke	9 / 577 (1.6)	20 / 442 (4.5)	0.33	0.2-0.7	p=0.004
Stroke or death	13 / 515 (2.5)	23 / 378 (6.1)	0.40	0.2-0.8	p=0.007
Return to theatre	8 / 731 (1.1)	17 / 550 (3.1)	0.35	0.1-0.8	p=0.01
Arterial occlusion	3 / 641 (0.5)	17 / 466 (3.6)	0.12	0.0-0.4	p=0.0001
Cranial nerve injury	8 / 375 (2.1)	7 / 250 (2.8)	0.76	0.3-2.1	p=0.7
Long term Follow up					
Ipsilateral stroke	10 / 641 (1.6)	24 / 500 (4.8)	0.31	0.1-0.7	p=0.001
All death	65 / 577 (11.3)	69 / 442 (15.6)	0.69	0.5-1.0	p=0.1
Fatal stroke	1 / 577 (0.2)	4 / 442 (0.9)	0.19	0.0-1.7	p=0.2
Any stroke	11 / 577 (1.9)	26 / 442 (5.9)	0.31	0.2-0.6	p=0.0009
Stroke or death	75 / 515 (14.6)	91 / 378 (24.1)	0.54	0.4-0.8	p=0.004
Restenosis	31 / 641 (4.8)	93 / 500 (18.6)	0.22	0.1-0.3	p<0.0001



- Complications of CEA:
- Immediate:
    - Neuro
        - death
        - Hyperperfusion
        - stroke
            - Embolism/thrombosis
            - Disabling 2%
            - If pt is Symptomatic risk is up to 6%, if asymptomatic 3%
    - Local:
        - CN injury

        - Bleeding
        - Wound dehiscence
        - infection
    - Systemic:
        - HD instability
        - MI
- Long-term:
    - Recurrent stroke AFTER repair
        - Annual risk
            - 0.3% if asymptomatic
            - 2% if symptotic post CVA
            - <1% if symptomatic post TIA
    - Recurrent stenosis
    - Patch blow out/aneurysm

223.Recurrent stenosis after CEA:

Defined as recurrent stenosis >70%.

- IH in first 2 years, then atherosclerosis
- Metaanalysis:
    - 10% at 2 years, 17% at 15 years.
    - Highest in first year, then low rate
        - Generally 1% per year
- Only one third to one half are symptomatic
- Incidence Significantly reduced by patching:
    - down to 2% at 2 years p.2103
    - acas – 70-80% reduction with patching

- If symptomatic, consider longevity AND nature of lesion:
    - At 5 years – 74%, at 10 years 42%
    - Most lesions are IH, not athero
        - Low risk for atheroembolism

- Management options include:

- Aggressive medical therapy
    - Dipiridomole and ASA is better than Plavix and ASA
    - Statins (Jupiter trial)
- CEA+patch
- Resection+autologous interposition grafting
- CAS

- If asymptomatic, options include:

- Dupplex follow-up – 1/3 of lesions will regress with remodeling
- OR – very rarely as natural hx is benign and repeat OR is morbid affair

224. Carotid shunt complications?

Shunt types – outlying (Pruitt-Inohara) vs inline (Javid)

Need 50 ml/100 mg/min of brain tissue perfusion during clamping

- Dissection
- Embolization
- Migration
- Poor distal endpoint managment
- Blockage by debris

- May forgo shunt if >50 mmHg back pressure.
- If pt had previous CVA use shunt routinely (unless doing it awake) – p. 1981.

Javid, Argyle, Pruitt-Innohara.

225.CEA and CABG – decision making in vasc surgery: p. 86

- CABG by itself causes cerebral atheroembolism
- Risk of stroke for CABG alone is 6.7% in Hertzer RCT
  - Seems high, but that's what "Decision making in Vascular Surgery" state

- Sorting out what is responsible for the event post combined repair is difficult

- First, define high vs low neuro risk
  - High – these need to be addressed either before or during CABG
    - bilateral severe (>80%) asymptomatic stenosis
    - severe symptomatic
    - unilateral severe asymptomatic with CL occlusion

  - Low risk – unilateral severe asyptomatic: this can wait and followed on duplex

- Secondly, define High vs low Cardiac risk
  - Unstable angina HIGH risk
  - Stable angina LOW risk

Combinations to be considered:

- Pts which is High neuro and High Cardiac risk:
    - Controversial, now that CAS and PCI are available
    - Traditionally Combined open:
        - Accept high risk of M&M combined 15- 17%
    - Or…Local CEA then 48h CABG
        - That's what I'd suggest on my exam
    - Or… PCI and CEA/CAS combined/staged
    - No good data to support one or the other

- Pts with High neuro and Low cardiac:
    - CEA first, then CABG in 4-6 weeks
    - Recent review (aug 2008 by Claggett)
        - CAS was worse than CEA for symptomatic,
        - CAS better than CEA for asymptomatic combinations with CABG

- Low neuro and High cardiac:
    - Cardiac first, then monitor carotid
    - Combined risk of complications should be less than 8%
        - If can assure this, may do combined, otherwise stage

- Low neuro and Low cardiac risk:
    - May do either, depending on the institution results – combined vs staged

226. CAS trials:

Initial early death and mortality were 10%, but current series (17,000 pts) – 5%.
Recurrent stenosis: 10% at 24 month. Predisposing factors:

- Female
- CRP elevation within 48h
- Redidual post procedural stenosis
- Incomplete appostion
- Age > 75

High risk pts: p. 2010
Physiologic criteria:

- Severe CAD requiring PCI or CABG
- Hx of CHF
- Severe COPD requiring home O2 and FEV_1<20% predicted
- Severe renal failure (Cr > 3 or 240)

Anatomic criteria:

- Prior CEA with restenosis
- CL vocal cord paralysis
- Surgically inaccessible lesion – at/above C2, inferior to clavicle
- Radiation induced stenosis
- Prior ipsilateral radical neck dissection

SAPPHIRE trial of CAS in HIGH RISK patients

- randomized to CAS (*n* = 167) or CEA (*n* = 167)
- combined endpoint of stroke, death, and MI.
- hypothesis that CAS *was not inferior* to CEA
- Most of the randomized patients were asymptomatic – 70% in each group

- At 30 day – more MI in surgery group 4.4 vs 8 %, the rest equal.
    - 30 day the risk of stroke
        - CAS 3.1%, CEA 3.3% NO DIFFERENCE
    - 30 day Mortality
        - CAS - 0.6% vs CEA 2.0% $P = 0.36$ NO DIFFERENCE
    - 30 day MI
        - CEA - 6.6% vs CAS group (4.4%), $P < 0.05$).
            - most of the MIs were non-Q,
            - identified on routine postprocedure laboratory studies,
    - 30 day combined endpoint death/stroke/MI same:
        - CAS - 4.4% vs CEA- 9.9% $P = 0.08$

- 1 year data:
    - Major ipsilateral stoke – CAS 0% vs CEA 3.5%. $P = 0.02$
    - MI - CAS 2.5% vs CEA 8.1%. $P = 0.03$
- **1 year combined endpoint at 1 year favored the CAS group**
    - **12.0% vs. 20.1% $P = 0.05$**
- **Conclusion – CAS is non-inferior….**

The ARCHeR

- study of a stent and protection devicea
- registry of high-risk patients
- the composite endpoint (stroke/death/MI) was 7.7%,
    - included a 5.3% stroke risk;
    - the risk of stroke or death was 6.6% at 30 days.
    - Patients having CAS for restenosis following CEA had an extremely low risk of stroke (0.7%);
    - those patients with end-stage renal disease had an extraordinarily high risk (28%).
    -

The EVA-3S study

- Endarterectomy versus Angioplasty in Patients with Severe Symptomatic Carotid Stenosis
- Randomized,
- NOT HIGH RISK symptomatic patients **with >60% carotid** stenosis to CAS or CEA
- Requirements:
  - vascular surgeon
    - min 25 CEAs (with no upper limit of MI/CVA)
  - interventional physician
    - 12 CAS procedures OR
    - 35 interventions in the supra-aortic trunks,
    - at least 5 of which were on carotid artery
    - at least two procedures with a new device

- primary end point: composite of stroke and death at 30 days.

- 261 patients underwent CAS and 259 had CEA and were analyzed for primary outcome measures.
- trial was designed to show noninferiority
- stenting was found to carry a greater risk than endarterectomy.
- The 30-day
  - incidence of any stroke or death:
    - 3.9% after CEA and 9.6% after CAS
  - disabling stroke or death:
    - 1.5% after CEA and 3.4% after CAS
    - **i.e. ARR 2%**
- No stat difference in Systemic complications or local complications
- Cranial nerve injury was more common after CEA (7.7%).
- no difference in results from high vs low enrolling centers
- no difference btw experienced vs less experienced operators

- The trial was stopped prematurely after enrollment of 527 patients because of "both safety and futility," as CAS carried significantly higher risk than CEA.
- Criticism:
  - 20 pts in stent group were done without cerebral protection
  - 5 different stents, 7 different embolic protection devices used
  - Low experience of interventionalist was required

Summary:

- So EVA3 –
  - no high risk,
  - CEA 4% vs CAS 9.6%
    - fatal/disabling -1.5 vs 3.5 in favor of surgery
- Sapphire –
  - high risk
  - CEA 20% vs CAS 12% in favor of CAS

SPACE trial
- Stent-protected angioplasty versus carotid endarterectomy in symptomatic patients
- 1200 symptomatic patients
  - TIA or moderate stroke within 180 days
- randomized to CAS or CEA
- Embolic protection used only in 27% of pts
- The primary endpoints
  - ipsilateral ischemic stroke or death within 30 days of the procedure
- death or stroke was 6.84% in the CAS group and 6.34% in the CEA group.
- The authors concluded that the study failed to prove the non-inferiority of CAS compared with CEA (p-value of 0.09)
- Important age related outcomes of stenting:
  - No difference in stroke rate in less than 75 yo
  - 11% vs 7% in stroke in > 75 year olds (in favor of CEA)

Crest trial:
- Final results are still pending
- Lead in phase resuls:
  - morality and stroke in CAS
    - 3.4% for asymptomatic and 5.6% for symptomatic
  - Outcome heavily dependent on age:

Age (N)	Stroke/death %
<60 (120)	2 (1.7%)
60-69 (229)	3(1.3%)
70-79 (301)	16(5.3%)
>80 (99)	12 (12.1%)

Pending studies –
International Carotid Stending study (ICSS) – only symptomatic pts
Asymptomatic Carotid Surgery Trial #2 (ACT2)
Asymptomatic Carotic Trial (ACT1)

Metaanalysis:

Luebke's – 2007 – 30 day stroke or death 1.39%
Bahmanandam's – 2008 – 30 day risk of stroke 1.38%
These results, when compared to CEA are marginally higher.

228.L ICA occlusion. What do do?

- See if symptomatic. If yes:
- Confirm flow distribution with Xenon CT/PET
- If definite hypoperfusion, may be one of the rare cases of extracranial to intracranial bypass
    - Another indication for EC/IC bypass- moyamoya and high carotid aneurysm that can't be ligated and reconstructed at neck
- Recognized that sxs are hemodynamic, NOT embolic
    - Otherwise, all that need to do, ligate origin of ICA and open up ECA
- COSS study pending in 2009, December
- Previous study of ECICB study in 1986 – bypass = medical therapy…

229. Vertebral insufficiency:

DD of syncopy:

- Stroke
- VBI
- Cardiogenic shock
- Hypotensive state
- Epileptic seizure
- Metablic state

Rull out

- Local:
    - Labyrinthine
    - Subclavian steal
- Systemic:
    - Orthostatic drop
    - Meds
    - Extrinsic compression
    - Anemia
    - CHF
    - Arrhythmia
    - Malfunctioning pacemaker
    - Venomotor paralysis of diabetics
    - Brain tumor

Usually see immediate sxs with rapid head shaking or turning if choclearvestibular problems are the source of symptoms. If problems with compression of vertebral artery and low flow – see delay of several seconds, position dependent.

- Studies: MRI (brainstem infarct), angio (positional and cranio-caudal loading), Holter

VBI: includes sxs and strokes
Sxs:

- Syncopy
- Diplopia
- Vertigo
- Ataxia

- 30% microembolization – predominant cause of strokes
  - From innominate, subclavian, vertebral
- 60% Low flow – predominant cause of sxs but not strokes
  - Plaque
  - Osteophyte

Concomitant vertebral and carotid a. repair:

- Has to be on the same side
- VA is dominant, stenosed > 75% OR responsible for emboli
- Pt understands that risk of complications exceeds aggreagated M&M for individual repair of carotid and vertebral lesion (Rutherford companion).

V1 – most common stenosis at orifice due to atherosclerosis
V2 – most common pathology – compression
V3 – MC pathology – trauma, FMD, dissection. Stays open due to collaterals from occipital a.
V4-surgically incaccessible

230.Revascularization of vertebral artery:

- Subclavian bypass (rare)
- Carotid transposition – most common, for V1 lesssions
- Carotid bypass – for V2 lesions, at the base of the skull.
  - Off common
  - Off external
  - Off occipital
  - Off cervical ICA

231.Describe steal, outline treatment:

For steal, one needs to have:

- Dominant vertebral a. affected (**50% Left**, 25% both or R)
- Proximal subclavian (or innominate on the R) stenosis
  - Reduced inflow

- Decreased peripheral resistance in upper extremity (due to exercise) causes reversal of flow in vertebral artery. If artery is dominant, significant vertebro-basilar insufficiency is seen

To diagnose steal:

- Duplex
  - will have same direction in CCA and subclavian (red) , but reversed in vertebral (blue).

To treat suclvian steal is to treat proximal subclavian stenosis:

- Endo
  - PTA, dodgy as may occlude/dissect vertebral/LIMA/RIMA origin
- Open:
  - Transthoracic
    - Thrombo-endareterectomy
    - Ascending Aorto innominate/sublavian bypass
  - Extrathoracic:
    - Carotid subclavian transposition
    - Carotid subclavian bypass
    - Axillo-axillary BP (rare, despised by purists)
    - Femor->axillary BP (very desperate and rare)

232.Branches of subclavian artery:

- Vertebral
- Thyrocervical trunk
- Costocervical trunk
- Internal mammary
- Descending or Dorsal scapular (50% of time)

233.Branches of axillary artery:

- 1st part
  - supreme thoracic
- 2nd part
  - thoracoacromial
  - lateral thoracic
- 3rd part
  - subscapular
  - posterior humeral circumflex
  - anterior humeral circumflex

234.Features of spontaneous carotid dissection:

- Cause: FMD, marfan, Ehlers-Danlos, mucopolysacharidosis
    - Blunt trauma: important, but clearly this is not SPONTANEOUS category
- Most diagnosed post factum after neuro deficit set in
- Younger pts
- Clinically see:
    - Headache +/- neck pain
    - oculosympathetic syndrome
    - Neuro deficit:
        - Stroke/TIA/SAH
        - Palsies of lower CN (VII, IX, X, XII)
    - Combinations for the above three
        - Unilateral headache +/-
        - Ipsilateral oculosympathetic syndrome
        - delayed focal neuro deficit
- Angiographically:
        - Appears as "string sign"
        - luminal smooth stenosis
        - tapered occlusion, distal branch occlusion
        - low flow in MCA
        - abrupt reconstitution of lumen
        - pseudoaneurysm of extracranial arterial segment
- Treatment:
        - USUALLY medical –
            - Anticoagulation
                - hep->warf 3 months
                - reimage at 3 months
                    - if resolved, ASA
                    - if not – 3 months of anticoag, repeat dril
        - surgical – not common
            - thrombectomy,
            - progressive intraluminal dilation,
            - endarterectomy,
            - intimectomy,
            - graft interposition vs ligate vs EC/IC bypass

Prognosis:

- If no symptoms or mild – 90% recovery to good function.
- If symptoms – 40%...

See treatment of blunt carotid injury in Trauma

235. Carotid FMD:

- Most common distal ICA
  - Doppler may miss it
- 5% risk of stroke over 5 years – i.e. don't treat asymptomatics
- Have 10-50% incidence of intracranial aneurysm – always check for these and fix prophylactically
- Have 8-40% incidicence of renal a. involvement

236. Extracranial Carotic artery aneuryms:

DD – dilated tortuous subclavian and proximal CCA

- Etiology
  - Atherosclosis 70%
  - Trauma – penetrating and blunt
  - Dissection
  - FMD
  - Post CEA with vein patch angioplasty
  - Infection – used to be most common cause, from tonislitis
  - Media problem:
    - Marfan syndrome
    - Cystic medial necrosis
    - Idiopathic medial arteriopathy
- Mc location – CCA at bifurcation, 2nd mc – subclavian.
- Sxs and signs
  - Pulsatile mass in neck or ***tonsilar fossa***
  - Compression:
    - Auricular pain
    - Dysphagia
    - Horner's syndrome – compression of stellate ganglion
  - TIA or strokes
  - Hemorrhage: RARE

DD:

- Kinked /coiled artery
  - Carotic kinks – 4 times more common in females
  - Coils – more common in kids
- Carotid body tumor
- Non-vascular neck tumor

Investigations:

- Duplex, angio, CT
- If distal ICA involved – balloon occlusion test –

- 30 min occlusion and assessment of neurologic status as preparation for ligation
- Back pressure >50 mm Hg– safe to ligate…

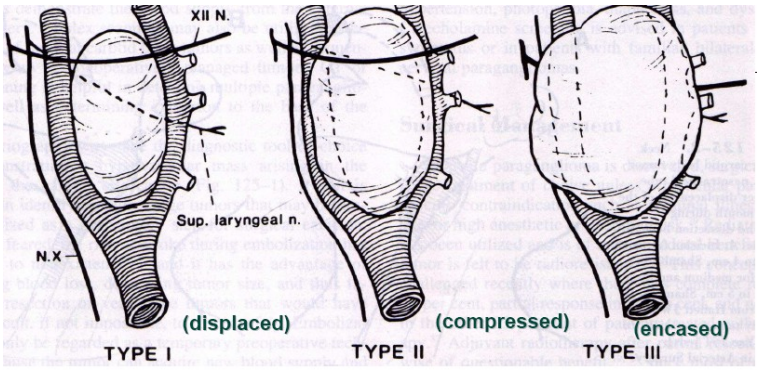
Tx:

- Resect and restore arterial continuity for CCA and proximal ICA lesions
- Distal ICA
  - Endo (stent, embolization)
  - Open
    - ligation – only if back pressure is > 50 mm Hg and ok occlusion test
      - Keep pt anticoagualted for 10/7 to minimize thrombus propagation
    - Bypass – consider extra-intracrainial bypass if failed balloon occlusion test.
    - Note, that hemodynamic results of EICB are inferior to carotid repair

237.Carotid body tumor:

- Arises from afferent ganglion of Glossopharyngeal n.
- Paraganglioma, chemoductoma
- Chemoreceptor responsive to hypoxia, hypercarpnia, acidosis
  - if stimulated, will increase RR, tidal volume, HR, BP, vasoconstriction, catecholamine release
- Embryology:
  - Neural crest ectoderm and mesoderm that migrated along the afferent nerves
- Autosomal dominant inheritance, but most sporadic
- 5% guideline:
  - Metastatic
  - Biochemically active – pheo…
  - Bilateral (30% of these are familial)
    - May be part of MEN 1 and 2
- Differentiate from hyperplasia in high altitude dwellers
- On exam –
  - pulsatile, not expansile,
  - can move it sideways but not up and down (Fontain sign)
- Sensitive to rads but the only definitive control is surgery

Shamblin classification:



- Displaced
- Compressed
- Encased

Clear off ICA, then resect with ECA. See Oral exam file.

238. Innervation of carotid body:

- afferent input to the reticular formation in the medulla via glossopharengyal n.
- Connects CB to brain stem so that it can respond to hypoxia (primarily), and (lesser degree) hypercarbia/acidosis
- Stimulation produces increased RR, BP, vasoconstriction

MESENTERIC ISCHEMIA:

239. What are the non-atherosclerotic causes of chronic mesenteric ischemia?

- Aortic dissection
- FMD
- Radiation injury
- Buerger's
- Drugs – Cocaine, ergot
- Embolism
  - From AF
  - From aneurysm
- vasculitis
  - Takayasu
  - Neurofibromatosis

        - These form middle aortic syndrome
    - Rheumatoid arthritis
    - SLE
    - PAN

240.Causes of intestinal ischemia:

- Embolus (most common)
    - Sudden
    - Known AF/MI/source of embolism
    - No preceding GI hx
    - Diarrhea and abdo pain
- Thrombosis
    - Gradual
    - Preceding hx of chronic problems
    - Diarrhea (malabsorbtion, xyloze test)
    - Gasless abdomen on X-ray
    - Collaterals on angio, orificial obstruction
- Non-occlusive
    - Due to cardical failure, septic shock, dialysis, digitalis like drugs
        - **String of sausages on angio AND pruning of arterial branches**
        - **Decreased venous flow**
        - Papaverin IA is helpful
- Venous Thrombosis
- Acute IMA occlusion
    - MC due to ruptured AAA
        - Loss of collaterals in IMA and IIA distribution

241.Doppler findings in mesenteric ischemia:

- SMA – >275 PSV, >45 EDV. For >70% stenosis
- Celiac - > 200 PSV, >55 EDV. For 70% stenosis
    - Normal flow in Celiac is 100cm/sec
    - Normal flow in IMA 93-187
- Note:
    - Normal flow in celiac is biphasic – low resitance system
        - It does not change with fasting/fed state
    - SMA will change phasicity with fasting
        - Fasting – triphasic
        - Post prandial – bi (drop in peripheral resistance)
        - Also, replaced RHA may make flow biphasic
        - Hence, all measurements in SMA are done in FASTING state

242. Common variations of Common Hepatic Artery:

Normal anatomy seen in 80%
10% LHA is off L gastric artery
11% RHA and 4% CHA can take off SMA

243. Treatment of mesenteric ischemia:

- Indications:
  - Classic hx
    - Post prandial pain
    - Food fear
    - Weight loss
  - 2 out of 3 vessels occluded/stenosed
  - Mimickers ruled out
    - Pancreatitis
    - Cancer
    - PUD
    - psychiatric

- Celiac and SMA orificial stenosis tx:
  - Pta/stent – poor patency rates, poor durability, less symptomatic improvement
  - Bypass:
    - Supraceliac:
      - Supraceliac aorta less diseased
        - More diff to expose then iliac
        - May need to enter chest…
      - Supraceliac to CHA/SMA
      - Bifurcated Dacron
        - Vs Seatle slug
          - 8 mm single Dacron to Longitudinal opening of base of celiac onto aorta
          - Long patch angioplasty of the celiac origin with the hood of the graft to the SMA.
          - Hood starts on aorta and ends on the Celiac.
      - Retropancreatic tunneling to SMA
    - Iliac/infrarenal aorta:
      - Bifurcated
      - Easier to expose
      - Risk of kinking
      - Inflow may be more diseased

- Transaortic endarterectomy:
  - Most difficult
  - If have simulataneous renal a. revascularization
    - Medial visceral rotation
    - Control supraceliac aorta

244.Differences between acute and chronic mesenteric ischemia:

- Acute
  - previous embolism elsewhere
  - no previous GI symptoms
  - clear source of embolism
  - patent SMA origin, meniscus, no collaterals
  - sparing proximal jejunum
- chronic
  - vasculopath
  - previous postprandial angina/weight loss
  - low flow or intra plaque hemorrhage
  - no embolism
  - clot at SMA origin, see collaterals
  - entire SMA distribution knocked out

245.How to determine intraoperative bowel viability?

- Clinical:
  - visible palpable pulsations in the mesenteric arcade
  - normal colour/appearance
  - peristalsis
  - bleeding from cut of surface
- Laboratory:
  - Doppler signal on antimesenteric side
  - Woods lamp and fluorescin injection
  - Surface oxymetry
  - Intracolonic pH monitoring and IMA stump pressure
    - for large bowel

RENOVASCULAR DISEASE

246.Differentiate Renovascular Hypertension from other causes of HTN?

- More common in Caucasians
- Younger age

- Thin pts
- Less fam hx
- Recent onset
- Accelerated course
- More severe (Diastolic > 105)

Other important clues:

- Onset of azotemia on ACEI
- Hypokalemia while off diuretics
- Hypertension resistant to 3 drug
- Unilateral small kidney
- Abdo bruit

247.Mechanism of renal HTN:

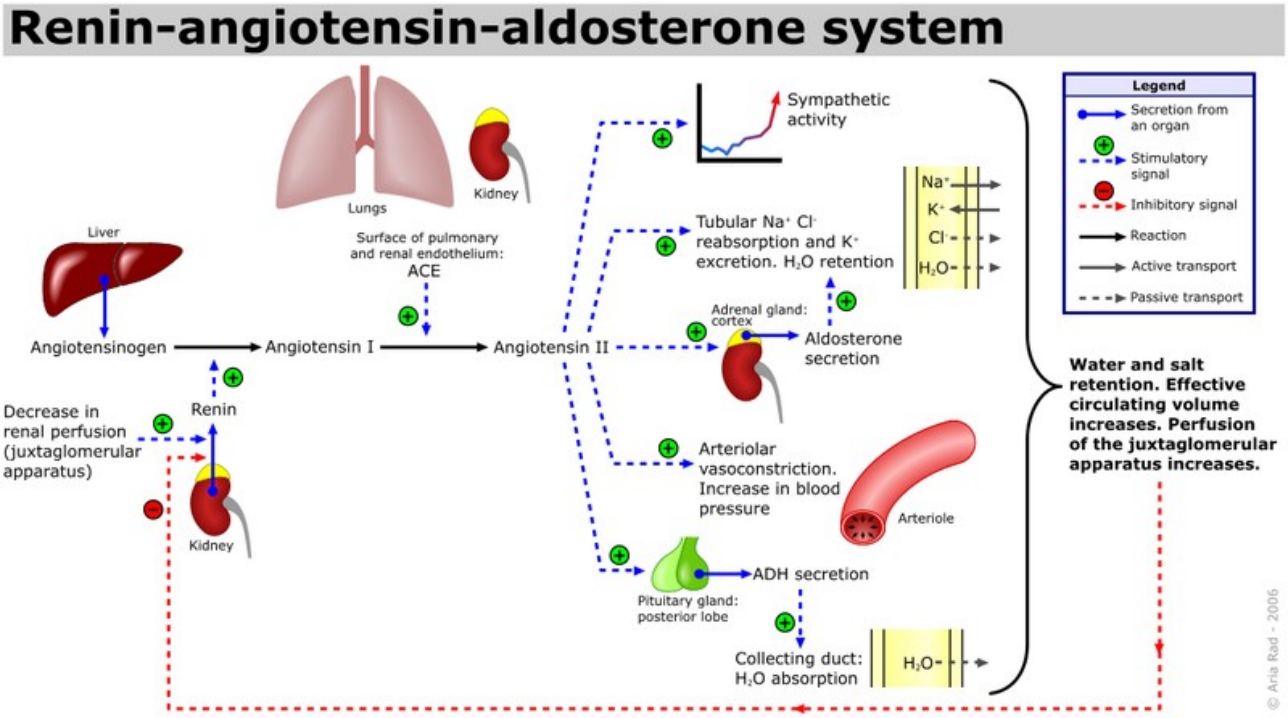
- Unilateral:
  - One clip model
  - Renin driven
    - On stenotic side:
      - one sided renin secretion -> aldosterone,
    - the other (Normal) side
      - compensates by natriuresis
      - keeps the volume down
    - hypovolvemia and hypoperfusion drives up renin

Unilateral stenosis leads to overproduction of renin which – through Angiotensin II, drives pressure up, remodels CVS, retains Na, and may have direct toxic effect on tubular elements of kidney. This effect is counteracted by contralateral normal kidney with natreuresis.

- Bilateral:
  - two clip model
  - volume expansion,
    - initially, driven by b/l secretion of renin-> aldosteron
    - Eventually aldosterone is suppressed
    - New set point for expanded effective circulating volume is established
    - CVS undergoes compensatory hypertrophy

Bilateral RAS leads to hyperaldosteronism, hypervolemia, and then sustained adaptaion of the CVS to higher pressures. Renin will be suppressed but HTN persists due to hypervolemia and adaptive CVS changes persist

It is difficult to distinguish early ischemic nephropathy (reversible) from chronic parenchimal disease (not reversible). In the end, hypervolemia, vascular hypertrophy and vascular reactivity sets in.



248. Captopril nephrogram:

- Can't use for bilateral RAS and if Cr is elevated (above 1.2 or 100)
- Angiotensin II –
    - constricts efferent arterioles, maintaining GFR when blood flow is reduced to glomerulus.
    - With chronic RAS, GFR become tightly dependent on increased parenchimal parachrine ATII.
    - Increase in ACEI acitivity drops AGII production
        - GFR drops as well.

- Nuclear perfusion scan done at baseline, then 1 h after 25-50mg captoril.
- Will see decreased excretion of trace with RAS side compared to normal contralateral renal a.

Diagnosis is made if

- Peak uptake is delayed > 11 min
- peak of GFR is delayed

- asymmetry of uptake between kidneys
- cortical retention of radionuclide

249.Renal a. duplex:

- Accessory a. seen in 10%
- Aberrant 20% (enter kidney outside of hilum)

- Ideally, would like to interrogate entire renal artery with 60 degree angle corrected Doppler.
  - PSV > 180, RA/Aortic PSV ratio > 3.5 - > 60% stenosis
  - EDV > 150 => 80% stenosis

- Parenchimal angle-independent spot readings. Allow to measure RI, wave shape, AT

- Resistive index
  - Peak systolic frequency shift – PDFS/PSFS
- > 5% difference btw kidneys indicative of > 50% stenosis
- RI > 80% - highly unlikely to benefit from surgery or revascularization
  - Seen in intrarenal vessel disease
  - Seen in subcapsular collection
  - Seen in low CO
- Tarda and parva waveforms
    - acceleration time > 0.07 sec
    - acceleration time index >3 m/sec^2

250.Surgical causes of hypertension:

- RAS
- Aortic coarctation
- Pheochromocytoma
- Conn's
- Cushing's

251.Causes of RV HTN, RV hypertension:

- RAS
  - Atherosclerosis
  - Congenital – bands/webs
  - FMD
- Embolism, leading to parenchimal disease:
  - Heart
  - Aortic atherosclerosis
  - Aortic aneurysm
  - Renal artery

    - Renal aneurysm
  - Dissection
    - Aortic
    - renal
  - AVM
  - Trauma
  - Weird & Wonderful
    - Vasculitis:
      - Takayasu
      - PAN
      - Neurofibromatosis
      - Necrotizing Angiitis
    - Post surgery:
      - Post bypass stenosis
      - Post transplant stenosis

252.RV HTN treatment and results:

In treating RV HTN your options include:

- Medical therapy
- Interventional:

  - Indications for intervention:
    - RV HTN
    - Ischemic nephropathy
    - Acute traumatic occlusion of renal artery (see trauma)
    - Concomitant supre-renal aortic clamping requiring reconstruction of renal a.

Before considering revascularzation, look at Renal perfusion scan…

- Measure cortex width (cortical atrophy – > poor response to revasc)
- Renal perfusion scan (DMSA better than DTPA)

- **It looks at time to the Peak activity – measures perfusion of the kidney**
  - Good study to order to work up failing graft due to anastomotic failure
- **Excretion – measures cortical function**
  - Cortical atrophy – bad prognostic sign,
    - as is pole-pole kidney size < 8 cm

Surgical reconstruction:

- Aortorenal bypass
- Thromboendarterectomy

  - Transrenal
  - Transaortic
- Splanchnorenal bypass: have to ensure celiac artery patency
  - Hepatorenal – easier, kocherize duo, ID portral triad, short bypass to R renal
  - Splenorenal – BIG deal, need to rotate viscera, discect behind pancreas
- Ex-vivo reconstruction

Endo: PTA/stent

- Ideal for FMD (medial fibroplasia type)
- Primarily for HTN treatment,
- not effective for CRF long term

Nephrectomy – only if kidney is non-functional AND disease is non-reconstructable

- Why?
  - Equivalent blood pressure response with revascularization and nephrectomy
  - Improved renal function after revascularization confers a survival advantage.

Offer surgery to pts with severe HTN that is difficult to control medically on multidrug regimen.

Role of PTA/stent in management of Renal Artery Stenosis:

- There is no indication to intervene as BP is optimally controlled
- PTA is not durable for renal failure.
- PTA has no effect on survival.
- Restenosis could be up to 37%
  - less with stenting.
  - ASTRAL study is designed to compaire PTA vs medical therapy. Preliminary results - Equivalent?

- PTA works best for non-osteal lesions due to to FMD.

- May be offered for pts who are at high risk of open surgery.

Results of renal PTA vs surgery: cumulative data from Rutherford

	HTN	HTN	HTN	CRF	CRF	CRF
	Cure	Stabilized	Failed	Better	No change	Worse
Surgery, p.1813-14	12%	73%	15%	40%	50%	10%
ENDO, p. 1840	10%	50%	40%	20%	60%	20%

- Same cure for HTN btw surgery and ENDO but 3 time more failure with endo…

- Initial 20% vs 40% improvement in CRF but that does not last for ENDO
    - At 5 years only 25% sustain their renal function
        - 75% get worse OR go on dialysis
        - Compare that to 55% dialyais free stay for 5 year for open surgery
- Open surgery for Renal failure:
    - The worse the failure the better the response to revascularization
        - Creatinine level → Improvement in RF:
            - < 1.8 → 30%
            - >3.0 → 60%
        - For prognostic info to see if there is going to be an improvement in RF post surgery, the most important is the RATE of renal function decline before surgery
    - Diabetics may not respond to revascularization

253.Indications of concomitant aortic and renal reconstruction:

p. 1817, 1807

Asymptomatic pts :

- ***NO role for prophylactic reconstruction.***

Assuming that due to progression of atherosclerosis RV HTN occurs first followed by renal failure

- If we were to take 100 asymptomatic pts
    - expect RV HTN to develop in 44.
- With medical management:
    - out of 44, 16 (36%) will progress to loss of the renal function.
- If these 16 pts are operated upon, 11 (67%) will regain RF,
    - the rest (5 pts) won't.
- **So 5% of pts will be lost if no surgery is offered.**

100-> 44->16->5….
If Surgery was offered at the outset on all 100 pt:

- then expect to have
    - OR mortatlity of 5.5%,
    - early technical failure of 0.5%
    - late failure in 4% –
- **i.e. a total of 10 pt (%) will be harmed if surgery is done prophylactically.**
- Risk benefit analysis does not support prophylactic repair in asymptomatic pts

Symptomatic patients:

- Unilateral disease:
    - If HTN – if mild, do captopril, if positive – OR
    - If HTN severe – empiric OR

- Bilateral disease:
  - If RAS >80% - empiric OR
  - IF RAS 60-80% - check HTN
    - severe – OR
    - If mild – check CRF –
      - If azotemic - empiric OR,
      - if not – medical therapy

Empiric repair:
- for pts with Hypertension or Hypertension AND CRF
- a causal relationship between RAS and these sequella has not been established

example:

60 yom needs open AAA repair, tight bilateral > 80% stenosis, Cr 200. Would you offer surgery?

Would like to know if he is hypertensive.
- If he is, then offer surgery
  - Additionally, Pts has azotemia which strengthens indication for intervention
- If pt has unilateral RAS and high CR but NO HTN, then there is no role for reconstruction.

254.Renal artery aneurysm repair indications: RA aneu

90% extra, 10% intrarenal

Ethiology:
- Atherosclerotic
- Most common – medial degenerative process
- FMD
- Dissection
- Vasculitis (PAN, Behcet)
- trauma

- Symptomatic
  - Rupture (calcification not protective)
  - pain
  - Embolization leading to HTN, CRF
  - Hematuria
  - Collecting duct obstruction
  - acute dissection threatening kidney viability
- any size in women of Child bearing age & pregnant

        - OSLER suggests watchful waiting for last trimester, repair in the first.
    - > 3-4 cm in asymptomatic

Management options:

- Repair with interposition graft
- Ex-vivo repair with autogenous vein reconstruction
- Prox/distal ligation with aortorenal/hepatorenal/splenorenal bypass
- Nephrectomy along with aneurysm
- Transcatheter embolization of saccular aneurysm or stent

255.Approach to renal arteries:

- Midline or Tansverse incision
    - Advantage of transverse incision –
        - handle instruments perpendicular to longitudinal axis of the body
            - Supraumbelical
            - Mid axil->mid clavicular
- R renal:
    - R medial rotation of colon and Kocherization of duo/panc head
    - Dissect middle of R renal a. first
        - If start distally - troublesome bleeding
        - Retract R renal vein cephalad
            - May need to ligate adrenal/small branches
        - Then dissect osteum
            - Ligate lumbar veins
            - Push IVC laterally
- L renal:
    - L medial rotation: better than transmesenteric
    - Ligate L gonadal and adrenal vein
    - Retract L renal vein cephalad
- Aorta is dissected for 5 cm infrarenally
- Fluid load and give 12.5 g of manitol before clamping
- GSV graft –
    - spatulate branched portion and anastamose it to aorta first
    - Tunnel R graft retrocaval, L graft behind R renal vein

256.Ex-vivo reconstruction: indications

- When reconstruction > 45 min
- All lesions involving branches (RAA, stenosis, AVM, dissection)

- Failure of prior reconstruction

257.Indication for repair of renal artery in trauma:

- Stable pt
- In the setting of laparotomy for other reasons
- Time to injury more than 6 h (if one kidney is damaged)
- Time to injury more than 20 h (if SOLE or BOTH kidneys are damaged)
  Why conservative approach?
- Success of revasc is only 30%
- If successful – 12-50% chance of hypertension,
- if decided to treat concervatively – have 40% chance of HTN

AAA

258.AAA epidemiology and cause:

Definitions:

- Aneurysm if more than 1.5 normal diameter
- Arteriomegaly - > 1.5 diameter in long multiple segments of arterial system with no discernable aneurysm.

- 2-6 times more common in males
- 2-3/1000 person/years
- Above 65 yoa – 5% males, 1% females,
  - In pts with FH of first degree relative:
    - 25% males, 5% females (i.e. 5 fold)
- In men, AAA begins at 50 peaks at 80
  - 7% of these is familial
- In women, begins at 60
  - 12% of these is familial
- If pt has AAA, there is…
  - 25% chance of having iliac aneu
    - Common iliac aneurysm
      - 10 times more common than internal iliac
      - Expect growth rate of 4 mm/year
      - No ruptures seen less than 3.8 cm
      - R twice more common than L
      - Repair if above 3 cm?
      - EVAR preferred
        - JVS 2008, 47:1203-11
  - 12% chance of having thoracic aneu

- <3% of AAA will have fem Aneu

- If see – need to fix if
  - symptomatic (mass/Atheroembolism)
  - if >2.5 cm if asymptomatic
- Conversely, 50% of fem aneu will have AAA
  - according to Rutherford (osler course say 90%)
- 30% of fem aneu will have pop aneu

- 3-7% AAA will have pop Aneu
  - 30% of pop aneu will have AAA (50% according to osler)

P. 1535

Cause:

- Proteolytic enzymes (MMP-2, 9, tissue inhibitors of MMP - TIMP)

- Decreased elastin in infrarenal aorta
- Decreased vasa-vasorum in infrarenal aorta
- Reflected pulse waves from aortic bifurcation
- Inflammation/infection
- Genetics

259.Principal matrix fibers in aorta, what changes are seen in AAA?

- Elastin and collagen
- MMP – matrix metalloproteases responsible for degradation
- Degradation of elastin is responsible for growth
- Loss of collagen is responsible for rupture

260.Risk factors for AAA DETECTION:

INCREASED RISK in…

- Smoking
- FH
- Older age
- Male gender
- High chol
- CAD
- COPD
- Tall statue

DECREASED RISK in…

- N abdominal imaging within 5 years
- DVT
- DM
- Black race

- Female gender

261.Natural history AAA

- Rate of expansion for aneurysm between 4-6 cm is 10% per year
- Rupture risk is related to size
- <4 cm 0
- 4-5cm 0.5-5%
- 5-6 cm 3-15%
- 6-7cm 10-20%
- 7-8 cm 20-40%
- >8 cm 30-50%

Selection of patients for surgery:

- Risk of rupture
- Risk of surgery
- Overall pt fitness
- Life expectancy

- **Most pts do not benefit from repair until 5.5 cm, unless it is woman (5 cm)**
- <5 cm – follow with serial US at 6 months intervals
- 5-5.5 cm – elective repair in young, low risk , good life expectancy such that eventual repair is almost certain if 5.5 cm threshold is reached
- For higher risk pts, consider raising optimal threshold size, e.g. 6 cm.
  - See modified Lee criteria

- Screening reduces mortality of AAA by 50%.
- 50% of aneurysm between 4.0 and 5.5 cm require fixing within 3 years (ADAM, UK)
- 75% of aneurysms between 5.0 and 5.5 cm required repair…

Note mall aneurysms:
24,000 consequitive autopsies over 23 years
473 AAA found
118 of these – ruptured
13% of these are under 5 cm (difficult to predict size on autopsy)
Risk of rupture for AAA 4.6-5.4 is about 0.5-1% annually.

262.Relevant CT findings in pts with AAA

- Venous anomaly
  - Retroarotic L renal vein
  - Duplicated IVC
  - Left sided IVC

- Renal anomalies
    - Horseshoe
    - Pelvic kidney
- Neck of aneurysm
    - Proximity to renal a
    - Accessory renal a.
- Other items to consider:
    - Inflammatory?
    - Iliac aneurysm?
    - Presence of Aortoiliac occlusive disease?
    - Other significant non-vascular pathology:
        - GB cancer, bowel Ca etc
    - SMA/Celiac/IMA/IIA/renal stenosis, arcs of Reolan

263.RF for AAA rupture

Normal rate of growth – 0.4 cm/year

PROVEN
- Large size
- HTN
- COPD
- Female gender
- smoking

SUSPECTED
- Familial inheritance, particularly in females
- Eccentric shape
- High expansion rate
- Absent/minimal thrombus

264.Ruptured AAA management:

- Abdo pain/back pain in pt with known AAA
- Fainting/hypotension/abdo pulsation
- Tender pulsatile mass
    - r/o CHF, swollen legs, abdo bruit, IVC dye in arterial phase
- Establish IV but keep BP at the minimal level to allow normal mentation
- If stable, may consider CT for planning EN ROUTE to OR
- In OR – prep first, induce when ready to cut
- Supraceliac clamp if large hematoma at neck
- No heparin if large hematoma
- Try to get away with shortest procedure (i.e. tube graft)
- Back bleed iliacs prior to completion

- Consider need to leave abdo ipen

What determines poor outcome in rupture?

1. Admission and intraop SBP <90
2. Preop Cr > 200 mmol (>2.5)
3. Preop Hg <100
4. OR blood loss >7 L
5. More then 10 u pRBC transfusion
6. OR u/o < 200cc total
7. Temp <91F (33 C)

265.Inflammatory AAA:

- Same rate of rupture as AAA
- Unclear if this is distinct entity vs part of the spectrum
- No role for primary management of other organ involvement
  - i.e. DO not decompress/release obstructed ureters
  - may stent them
  - most will settle conservatively after AAA repair
- Role of EVAR is being defined
- Use Teflon pedgets if repair open – do retroperitoneal

266.Indications for retroperitoneal RP repair:

- Hostile abdomen
- Inflammatory AAA
- Horseshoe kidney
- Ascitis
- Peritoneal dialysis
- obesity

267.Anatomic criteria for EVAR:

- Neck >15 mm long, <30 mm wide, < 60 degrees angulated
- Neck is non-divergent, no calcium, no thrombus
- Bifurcation minimum
  - 24 mm Cook, 28 mm Talent if bifurcated
  - Non-issue for AUI
- < 90 degrees aortoiliac angle
- Iliacs at least 7 mm

268.Types of endoleaks:

- a. proximal, b distal, c through occluded CL iliac in AUI

- sac flow via –
  - a. single vessel (not possible for long),
  - b more than 2
- structural failure – A – junction, B – tear or #
- porosity
- endotension – sack expansion with no endoleak

269.Specific complications of EVAR -endovascular AAA repair

Overall, risk of Major Adverse Events after EVAR is up to 30%. Most of these can be managed endovascularly. Freedom from Rupture at 9 years is about 94% (800 cases from MGH, Boston).

EARLY

- Radiation exposure to pt and personnel
- contrast allergy
- renal failure
- Access trauma:
  - perf,
  - dissect,
  - thrombosis
  - Microembolization of plaque or AAA thrombus
- Graft displacement or misplacement
  - Occlusion of RA, IIA, SMA
- Endoleak
- Postimplantation syndrome
  - fever
  - backache
  - malaise
- graft limb compression – thrombosis, stenosis, occlusion

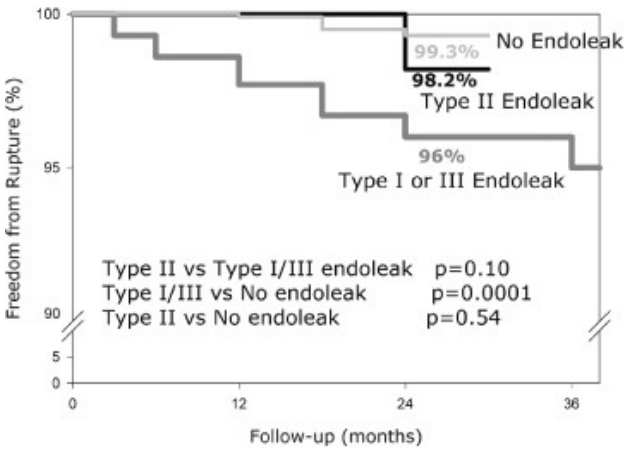
LATE

- Graft migration
- Endoleak
- Limb stenosis, kink, thrombosis
- AAA rupture

270.Follow up after EVAR. Endoleak treatment:

- CT scan at 1 month, then annually
- The purpose of EVAR is to prevent rupture of the aneurysm
- early identification of endoleaks is intended to help achieve this goal.
- treatment of endoleaks - the most common reason for readmission of patients after EVAR.

- 20% of pts experience endoleak
  - 7% at first CT
    - Most early endoleaks (70%) disappear
  - 13% later
- Risk of rupture of type IA/combined with III is LOW
  - in EUROSTAR registry (2800 pts) is 1% per year…
  - 4% over 3 years
- Still, all type 1 should be fixed:
  - extension vs palmaz vs external banding vs conversion to open



Freedom from aneurismal rupture after EVAR, categorized by endoleaks presence.

- Type II: seen in 40% before 30 days, the rest later
- No risk of rupture with type II
- No general agreement about the need for graft related interventions in pts with endoleaks
- Controversy re: most appropriate type of intervention for type II.
  - Coil, glue, laparascopic/open clipping vs observation

Type 2 with sac shrinkage
- No intervention, continue follow-up

Type 2 with sac expansion
- Seen in 10% of cases
- Most will recommend intervention
- Sac expansion→ migration & distortion of fixation sites →type I/III leak→increased risk of rupture

Type 2 with stable sac size
- Controversial
- Safe to observe according to Silverberg et al…

Sack enlargement is seen in:

- in 20% of patients with type I/III endoleak
- In 10% of patients with type II endoleak
- In 5% with no endoleak

271.Dream, EVAR 1 and EVAR 2 findings

- DREAM
  - 400 pt, RCT, the first to show 3% ARR in favor of EVAR
- EVAR 1 –
  - **540 pt in each group**
  - Fit pts, evar vs open
  - 1.7% vs 4.7% mortality at 30 days
  - 3% vs 7% mortality at 4 years
  - Overall mortality same at 4 years
    - 26% vs 29%, NS
  - Disease specific mortality at 4 years – 3% less for EVAR
  - Quality of life improved for EVAR for 3 months only
  - Conclusion:
    - Continue to explore the issue
    - Not enough evidence to change practice
- EVAR 2
  - Unfit pts, evar vs observation
  - 9% mortality at 30 days in EVAR group
  - At 4 years, Overall mortality is 67% vs 65%, same…
  - No improvement in quality of life

272. Harvard Medicare Registry study

- Registry review, NOT RCT
- 23,000 pts who had EVAR matched to 23,000 of pts who underwent open
- 20% women, 10% had MI previously
- Periop mortality 1.2% vs 4.8%
  - **ARR for 67-69 – 2%**
  - **ARR for >85 yoa – 8.5%**

Complications:

	EVAR	open
MI	7%	9.5%
pneumonia	9%	17%
Renal failure	5.5%	11%
Dialysis	0.4%	0.5%
Acute mesenteric isch	1%	2%

All have significant p

- Mortality benefit persisted for about 3 years
  - 1 year for 67-74 yoa
  - 4 years for >85 yoa

4 year observation	EVAR	open
rupture	2%	0.5%
reintervention	9%	2%
Laparotomy/hernia complications	4%	10%

Mortality comparison between EVAR vs open, Harvard registry

Age group	endovascular	open	Absolute difference
ALL	1.2%	4.8%	3.6%
67-69	0.4%	2.5%	2.1%
70-74	0.8%	3.3%	2.5%
75-79	1.3%	4.8%	3.5%
80-84	1.6%	7.2%	5.6%
>85	2.7%	11.2%	8.5%

Who goes home? Direct home vs rehab outcomes: EVAR vs open:

age	EVAR	OPEN	Absolute difference
ALL AGES	94.5%	81.6%	12.6%
67-69	97.8%	92.6%	5.2%
70-74	96.8%	88.7%	8.1%
75-79	94.4%	80.4%	14%
80-84	90.6%	67.7%	22.9%

>85	84.6%	57.1%	27.5%

Conclusion:

- Largest observational study of open vs EVAR
- EVAR Survival benefit depends on age
  - the older - the more and longer
  - Survival benefit of EVAR disappears with time
- Functional outcomes of EVAR are better
  - the older, the better – after 80 yoa astounding 25% ARR!
- EVAR reinterventions were balanced by laparotomy complications in open group
- More ruptures with EVAR

273. Indications for angio in pt with AAA: angio for AAA

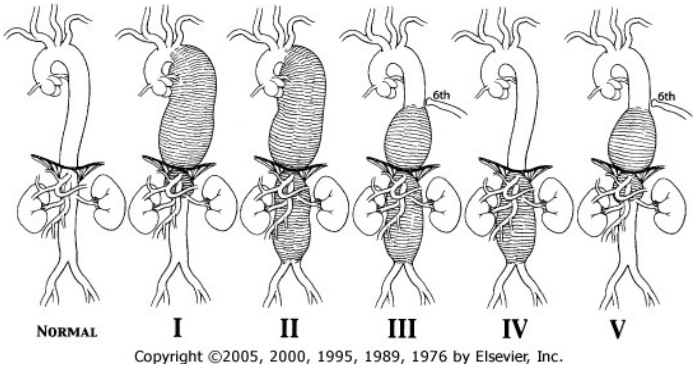
- TA/suprarenal aortic aneurysm
- Chronic aortic dissection
- Horseshoe kidney
- Suspected renovascular or visceral arterial disease
- Ilio-fem occlusive disease
- Associated peripheral aneurysm

THORACOABDOMINAL ANEURYSM

274.Thoracoabdominal aneurysm (TA)

Notes on anatomy:

- Think of a giant slug gradually sliding down then crawling back up
- High high, high low, low low, very low, middle
- No renal involvement in type 1 and 5
- Type 2 is the most extensive
- Type 4 is a bad version of suprarenal aneu (prox suture line above celiac)
- Most are degenerative
  - Thinning of media, destruction of SMC & elastin
- 20% are familial



- Syndromes associated TAA:
  - Marfan (MC)
    - Familial – 75%
    - New onset – new mutation 25%
  - Turner
  - Ehlers-Danlos
  - Polycystic Kidney
- Dissection (20% of TAA are due to AD)
  - Conversely, up to 40% of AD end up in aneurysm at 7 years
- Infection/trauma (minority)
  - Salmonella, H.Flu, Staph, TB, Treponema)
- Predictors of rupture: normal rate of growth – 2 mm/year
  - Size > 7 cm
  - 1cm/year expansion
  - HTN (diastolic)
  - Smoking
  - COPD
  - Gender (F>>M)
  - Age – up 2.5 folds for every decade
    - For >70 yoa, 50% risk of rupture within 1.5 years
- Risk of repair:
  - COPD
  - **Renal Failure – Cr > 200 (2.5) – poor prognosis for repair**
  - **Longevity assessment**
  - **Mortality of repair:**
    - **10% in center of excellence**
    - **20% country wide**

- Presentation:
  - Pressure effect
    - Dysphagia
    - Horseness

        - SOB
        - Visceral pressure
        - Ureteric compression
    - Rupture
        - Intraperitoneal
        - Into pleural cavity
        - Into RP
        - Into GI (duo)
        - Into IVC
        - Into ureter
    - Atheroembolism:
        - Visceral vessels
        - Lower exremity

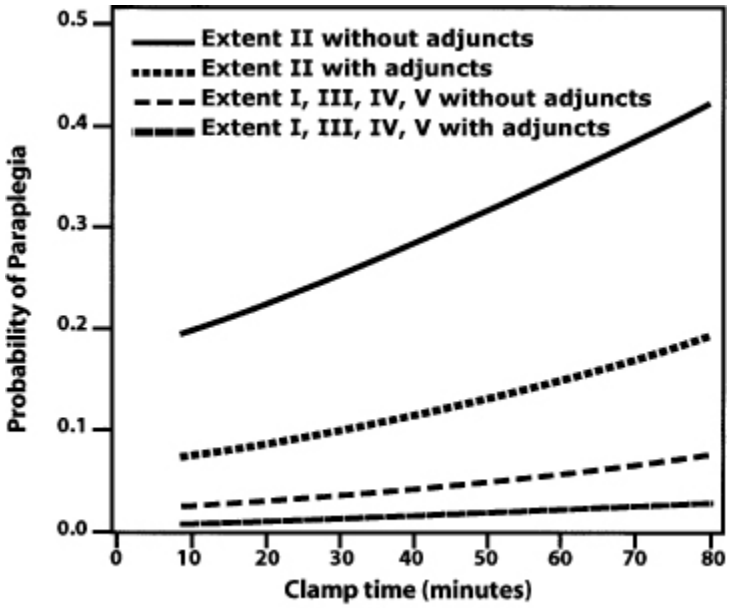
275.Decision making in assessing pt with TAA:

- Riks of OR
    - Surgical detail
        - Extent
            - Prox & distal
            - Visceral involvement
        - Aortic quality
            - Calcified
            - Thrombus in T9-L2
        - Comorbidities
            - Modified Lee, especially Renal failure
        - Life expectancy

    - Risk of Rupture:
        - HTN
        - Smoking
        - COPD
        - Size

In general, do not operate on TTA < 6 cm (except in type 4 – 5 to 5.5 as in infrarenal and Marfan pts)

- Overall, 30 days mortality is close to 10%...
- Type II had 29% risk of spinal cord ischemia before institution of adjuncts
    - Less with adjuncts..



276.Strategy for renal protection in TAA:

Goals: reduce renal oxygen use, reduce direct renal tubular injury, maintain perfusion

- Hold nephrotoxins (ACEI, aminoglycosides)
- Distal aortic perfusion
  - Only protective when renal a. do not require reconstruction
  - i.e. for type 1 and 5…
- Selective visceral perfusion
  - It DOES protect the liver
- Retrograde hypotermic renal venous perfusion to 15 degrees
  - But keep core body temp at 32-33 degrees
  - Holds a lot of promise according to Safi…

So far, none of these techniques have been shown to definitively reduce incidence of RF

277.Why is the spinal cord at risk during repair of TAA?

Spinal cord blood supply

- one anterior – principal
  - varies in size
  - discontinuous in some people
  - receives radicular branches from intercostals or the upper lumbar arteries.

- largest of these aortic branches is called the great radicular artery of Adamckiewicz or arteria radicularis magna (ARM)
  - arises between T9 and T12
- two posterior
  - arise cephalad from branches of the vertebral a.
  - run through the total length of the spine
  - end in a conus plexus of lumbosacral branches.

The most likely cause of paraplegia after thoracoabdominal aortic aneurysm surgical treatment, either temporary of permanently is the interference with the Adamckiewicz artery.

Aside:

- anterior spinal syndrome – b/l paralysis and loss of pain/temperature. Intact proprioception

- Posterior: loss of proprioception and vibration. Preservation of touch, pain and temperature

278.Spinal cord protection methods during TA repair:

- Distal aortic perfusion
  - Passive (Ax-fem, Gott shunt)
  - Active
    - L atrial-fem bypass
    - Complete cardiopulmonary bypass

- Perioperative CSF drain
  - Spinal cord pressure= MAP – CSF,
  - keep CSF pressure at <10 mm Hg
  - keep MAP up
  - drain 10-15 cc/h
  - d/c drain on POD 3
- intercostal aa. Reimplantation (T9-L2)
- expeditious operation
- other
  - Hg >100
  - CI >2
  - MAP 90-100
- Hypothermia
  - Spinal (4 degrees C)
- Pharmacology
  - Nalaxone

- Steroids
- Magnesium
- Calcium channel blockers
- Oxygen free radical scavengers
- barbiturates

aside:

Different techniques were developed since the first TAAA repair in 1954:

- 1980's Crawford's inclusion technique:
  - preserve posterior wall of the aneurysm
    - don't do this for Marfan
  - reimplantation of the celiac, superior mesenteric, and renal artery islands of aortic wall into the prosthetic graft.
    - L renal is sewn in as a bypass
  - called the Clamp-and-sew technique.
    - "the less clamping time, the less the incidence of paraplegia"
      - goal was to complete all the anastomosis in about 30 minutes
    - Sequential advancing cross-clamping also minimized ischemic time to spinal cord and abdominal organs.

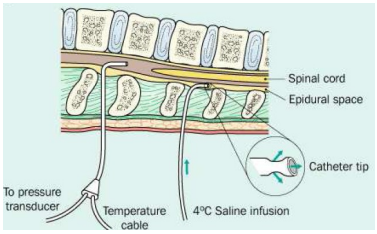
- Temporary Aortic Bypass:
  - axillo-femoral bypass
  - left atriofemoral bypass using a centrifugal pump without heparin.decompression of the proximal aorta
    - perfuse cord in a retrograde fashion distally
    - allows more time to perform the procedure

- Reimplantation of Intercostal arteries:
  - The vast majority of surgeons re-implant intercostals when patent
    - particularly in the T9-T12 area.
  - Some localize the Adamckiewicz artery preoperatively with CT- Scan or MRA,
  - some use somatosensory evoked potentials (SEPs) or motor-evoked potentials (MEPs) monitoring to ascertain which islands of intercostals or individual arteries to reattach.
    - Intercostal arteries can be reattached
      - directly to the graft, through an individual bypasses or
      - with the use of an oblique distal aortic transection with preservation of the posterior wall of the aneurysm

- Cerebrospinal fluid drainage:

- In intraoperative period and extending for several days after the operation
- supported by a Systematic Review of the literature.
- Safi et al technique, a constant pressure of 10mmHg is maintained.

- Hypothermia:
  - Selective cooling of the spinal cord:
  - Infusion of 50 ml of iced saline into the epidural space for 30 min before aortic clamping.
  - technique was not consistently proven as effective.



-
  - the goal is to decrease the temperature and decrease metabolism, THUS extending the period of ischemic tolerance.
  - hypothermia reduces the loss of ATP stores with earlier resolution of lactic acidosis.
    - Protection of tissue damage from ischemia may also reduce reperfusion injury.
    - With the use of the heat exchanger, various degrees of hypothermia can be achieved (e.g. 28° C, 33° C).

- Pharmacologic agents:

  - Naloxone during spinal cord ischemia, steroids and papaverine. These therapies were not proven effective.

279.Bleeding during TAA, cause:

- Hypothermia
- Coag factor exhaustion
  - Liver hypoperfusion
  - Post bleeding
  - Long OR time

Hence, warm up, be quick, perfuse liver, replace coag factors (FFP, cryo), give volume.

280.When should we NOT cover SCA in thoracic endografting?

- Prominent/dominant Vertebral on the L
- LIMA graft is present

- Extensive coverage of the decending thorasic aorta is planned– i.e. T9-L1
- Prev AAA repair (lumbar and IMA collaterals are gone)

AORTIC DISSECTION:

281.Aortic Dissection:

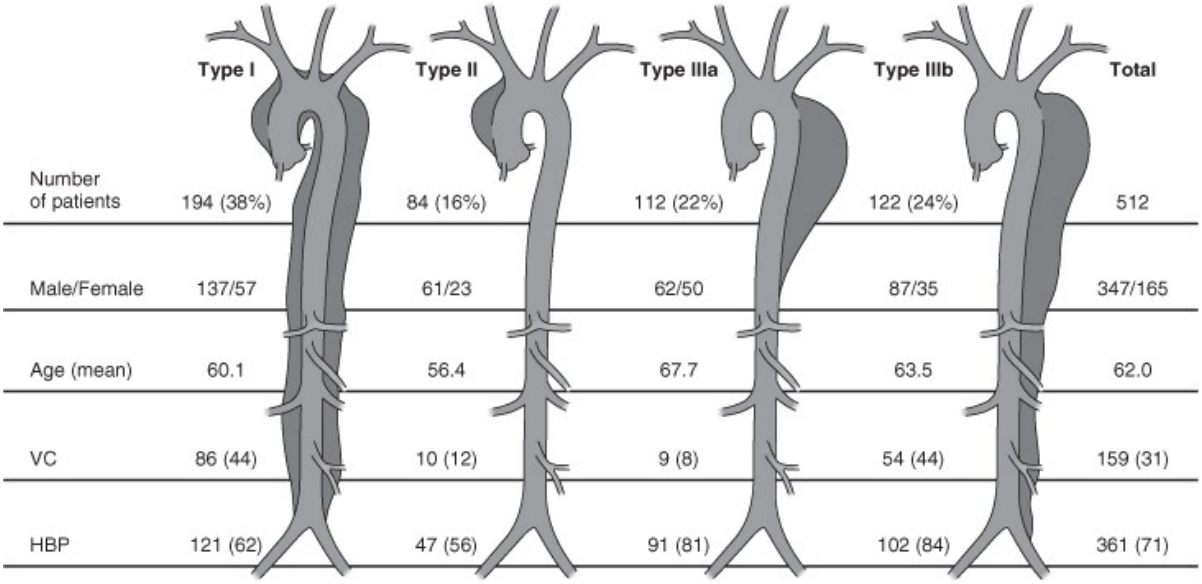
Acute dissection:

- Type A: all need repair by cardiac surgeon.
  - If there is concomitant mesenteric ischemia, FIX MESENTERIC ISCHEMIA 1st…
- Type B:
  - aggressive pressure control/impulse control therapy
  - surgery for:
    - rupture
    - expanding aneurysm (i.e. near rupture)
    - branch occlusion
    - high risk of rupture
      - Marfan
      - Diam > 5 cm
      - Long term steroid
    - failure of medical therapy
      - (i.e. rupture/expansion/branch occlusion) manifested by ongoing HTN, PAIN

Chronic dissection with aneurismal dilatation – same indications as for AAA

Aside:

Classification and management of aortic dissections.



Classification:

Sandford (S for simple) –

- A - ascending,
- B – descending

DeBakey classification (D for difficult)

- 1 origin in ascending, goes at least as far as the arch or further
- 2 origin in ascending, limited to ascending
- 3 a – origin in descending, limited to descending
- 3 b – origin in descending, goes to abdominal
- 3 c – origin in descending, goes proximally to arch

Treatment of type B:

Medical –

- 3:2 = A:B
- More in men
- less than 50% present with HTN
- impulse control with BB/dilators is mainstay
- better survival rate with medical therapy (istead trial, IRAD registry)
- goals of treatment:
    - Stabilize extent of dissection
    - Reduce intimal flap mobility
    - Relieve dynamic aortic branch obstuction

    - Decrease risk of rupture
- indications for surgery - only for complications
- goals of surgical treatment:
    - Induce aortic remodeling through FL thrombosis
- Indications
    - Recurrent pain
    - Rapid aortic expansion
    - Rupture
        - <4% of acute presentations,
        - 20% during the course of disease
    - Branch Vessel occlusion
- Malperfusion seen in 25-40% of acute dissection
        - Most important source of M&M
        - Dynamic obstruction 80%
        - Static 20%
        - False lumen: L renal a
        - True lumen: R renal, visceral
    - Diagnosis and management are usually delayed
        - 50-80% mortality if renal ischemia
        - 87% if mesenteric ischemia
- mortality for open surgery for malperfusion > 20%
    - Hence endovascular option is attractive
        - reduced upfront M&M
        - but INSTEAD trial showed same results for stent vs medical in acute AD
            - 3% medical 10% stent mortality at 1 year, p NON-significant
- Surgical Options:
    - Open
        - Central aortic replacement – for rupture (used very rarely)
        - Open fenestration for branch occlusion (used rarely)
            - Principle:
                - Wide resection of the dissected septum
                    - Equalize flow through both lumena
                - 9-10th IC space thoracoabdomianal exposure
                - Septectomy may be extended into visceral vessels
                    - If small aorta/poor flow/suspected osteal obstruction
                    - Resect septum
                - Inspect/tack peri-osteal intima
                - Fenestrate to infrarenal level with Teflon pleget
                - Replace infrarenal aorta with distal double-Teflon pleget anastomosis
    - Endo

        - Entry site sealing
        - Endo fenestration
        - Problem with endo:
            - Endovascular approach only seals endoluminal source of bleeding
            - Large vasa vasorum and intercostals may still contribute to late rupture/growth
                - Surveillance necessary
            - May not provide long-term survival benefit
                - Bridge therapy through rupture/emergency situation?
- FDA Gore TAG trial: applies to thoracic aortic repair

complication	open	stent
paraplegia	14%	3%
stroke	10%	5%
rupture	0%	0%
reintervention	10%	4%

- Note, Safi results cannot be reproduced by most centers (i.e. 6% spinal cord ischemia without adjuncts and 2% with). Results in real world practice are WORSE.
- Conclusion:
    - For uncomplicated type B – medical therapy
    - For complicated type B – consider expeditious diagnosis and treatment
        - Rupture or complications will likely require open repair
            - Stent if local expertise/logistics available
            - IF significant comorbidities – consider stent
            - Stent entry point and enlarge true lumen if dynamic branch obstruction. Consider fenestration if no outflow for FL
            - Stent individual branches/or individual vessel orifice if static obstruction
            - Consider open if failed
    - Carefully follow chronic dissections for future TAA
        - 14% at 4 years
        - 40% at 7 years

AORTOILIAC OCCLUSIVE DISEASE (AIOD)

282.Approach to pt with AIOD:

In AIOD setting, always ask re

- Buttock pain/impotence
- Blue/painful toes
- IC/RP/ulcer
    - Claud distance
- Management of RF

If pt has AAA in addition to AIOD, then add…

- AAA stuff…
    - Epi (race, gender, age)
    - Symptoms (abdo, back pain, distal atheroembolism)
    - High probability risk factors – smoking, lipids, CAD, FH
    - Low probability risk factors – DM, DVT, N abdo imaging
    - Risk Factor for repair: MI, CVA, CRF, COPD, HTN, level of activity, longevity assessment

283.Indication for End to End vs End to Side for ABF:

- End-to-End ABF bypass configuration
    - Do this when EIA open and you can reperfuse internal iliacs
    - Better HD configuration (theoretical)
    - Better tissue coverage
    - Indicated for aneurismal aortic/iliac disease
    - Easier clamp placement
- End-to-Side ABF bypass configuration:
    - When preservation flow is required in the following systems:
        - IMA flow
            - colon is preserved
        - iliac system flow
            - occluded EIA
            - if bypass occludes, pt is back to original state, with residual iliac system function, allowing at least AKA to be done
        - accessory renal a. flow and horseshoe kidney
        - median sacral and lumbar a. flow
            - spinal cord preservation

- End to side theoretically has more risk of atherembolic complications and less chances of impotence.

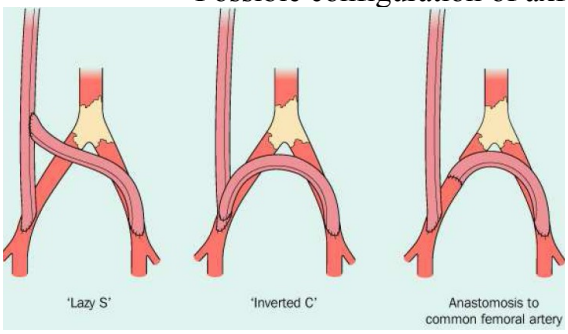
Ouflow management for ABF:

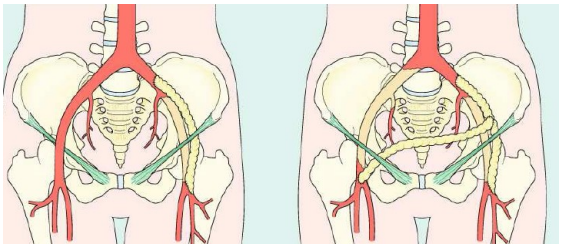
- If profunda is small/diseased AND pt has distal ulcer, add distal bypass
- Profunda-popliteal collateral index (PPCI)
    - If > 0.5 – profundoplasty alone will not improve sxs

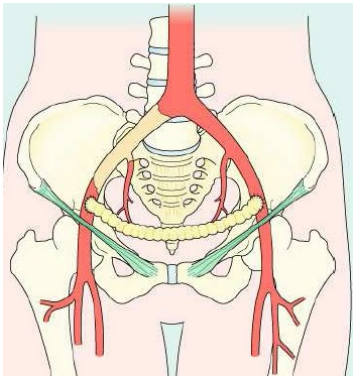
- If <0.2 – profundoplasty alone is adequate

- Best indications for profundoplasty:
  - Rest pain and minimal tissue loss
  - No DM
  - Patent distal pop and outflow with PPCI < 0.2

Possible configuration of axfem:







- Patency depends on indication.
- Patency of ABF: 90% at 5 years, 70% at 10 years, 3% periop mortality
- Axillo-bi-fem patency: 70% at 5 years
- Axillo-uni-fem: 50% at 5 years

284.Indication for Axillo-bi-femoral Graft:

- AIOD in pts with CRITIAL limb ischemia not suitable for ABF:
  - Poor surgical/medical risk

        - Hostile abdomen
    - To revascularize lower extremities following removal of an infected graft
        - Aortic graft
        - One limb of ABF
        - Fem-fem graft
    - Treating LE critical limb ischemia following aortic type B dissection

Complications:

1. Upper limb ischemia
    a. Steal
    b. Thrombosis with Upper Extremity embolization
    c. Anastomotic pseudoaneurysm
    d. Arterial distortion causing kinking/thrombosis
2. Brachial plexus injury

- Note, if anastomosis is made to 1st portion of axil artery, anastomotic disruption with sudden arm abduction is less likely.

- Hemodynamic results of ax-fem MAY NOT significantly improve claudication distance.

- Traditionally axillo-Bi-fem are considered to have better patency than uni.

    - However, best axfem patencies were obtained for uni configuration
    - Rutherford's companion states that may be patencies are equivalent.

285. Types of endarterectomy. Discuss aortic endarterectomy

- Controlled arterial injury that heals by intimal re-growth

- Why EA is possible?
    - Cleavage plane and endpoint is smooth:
        - Disease is limited to initima and inner media
        - Disease is segmental
    - Residual adventitia/outer media resists dilation

Contraindications to endarterectomy:

- Aneursmal disease
- Takayasu arteritis
- Radiation arteritis
- Medial calcification/Transmural calcification
    - Multiple adventitial defects post procedure

- Two types of atherosclerotic involvement:

  - Type 1:
    - m:f=2:1,
    - younger,
    - aorta, CIA
    - spare EIA, CFA, outflow
  - type 2:
    - m:f=5:1
    - older
    - type 1 plus outflow involvement,
- Atherosclerosis occurs at:
  - Origin of aortic branches
  - Sites of arterial fixation
  - Sites of turbulence

- Types of EA:
  - **Open**
  - **Semi-closed (remote)**
    - Two longitudinal arteriotomies at the end and beginning of the vessel, develop distal point first, then go proximal.
  - **Extraction EA**
    - With hemostat, develop plane then pull out plaque
    - Antegrade –
      - for aortic/branch vessels
      - distal extent of plaque is ID'd externally by palpation
      - Atherosclerosis is orificial and smoothly tapers in the distal vessel
    - Retrograde –
      - for CFA/EIA
      - proximal endpoint is separated by crushing the artery
      - distal endpoint is controlled with talking suture
  - **Eversion** –
      - Transect artery
      - Evert
  - **Selective**
    - I don't understand this one☺

- General guidelines for aortic EA:
  - BILATERAL EA to iliac bifurcation for type 1, and to CFA for type 2
  - Entire media removal -> leads to 2-5 mm dilatation of the artery
    - May be responsible for durability of repair
  - Unobstructed outflow to profunda

286. Causes of AV communications involving aorta and its' branches:

- Congenital – Vascular malformations
- Acquired
    - **Rupture of degenerative AAA (MOST COMMON)**
    - trauma (low velocity penetration)
    - iatrogenic
        - renal Bx,
        - spine OR – aorta/IVC/iliac damage
        - mass ligation of splenic/renal pedicle
    - erosion
        - due to sepsis/aortitis
        - tumor (renal Ca, mesenchimal tumor btw Ao/IVC)

287. Potential physiologic and anatomic consequences of a large AVF:

- Trauma to the endothelium
- Hemodynamic consequences:
    - Drop in total peripheral resistance
    - Increased central venous pressure
        - Drop in mean arterial pressure
        - Increased heart rate and stroke volume – increased cardiac output
        - Increased blood volume
    - Gradually increased LVEDP and volume
    - Cardiac failure

288. Aortocaval fistula:

- Commonest cause – ruptured AAA
- Also see arto-renal vein
    - Won't see infrarenal IVC contrast
    - No leg edema
    - Hematuria – 72%, flank pain
- Clinical features
    - Acute:
        - Bruit/thrill > 2/3
        - AAA/mass
        - CHF > 1/4
        - Decreased distal pulses
        - Swollen lower extremity, venous HTN > 1/3
        - Hematuria >1/4

- Chronic: RARE
  - LE swelling
  - venous thrombosis
  - perineal/hemorrhoidal varices
  - hematuria
  - abdo bruit
  - high output CHF
  - Peripheral circulation steal

Role of EVAR?
In clearly decompensated CHF pt, high risk for open, as a temporizing strategy may consider EVAR…

COMPLICATIONS

289.Cardiac and Respiratory Complications of vascular surgery:

	MI	Fatal MI
Aortic surgery	2.2%	1.4%
Infrainguinal bypass	4%	1.8%
Carotid endarterectomy	1%	0.4%

i.e. MI for aorta 2%, bypass 4%, CEA 1%... Half of these is fatal…Myocardial ischemia may be as common as 20-40%...

- Role of screening: undefined…
  - CAD is prevalent, but rates of MI are fairly low (see table above)
  - Atherosclerosis does not occur with most HD significant stenosis
    - Cardiac screening detect primarly HD sig stenosis
  - Most authors state risk stratification is imprecise at best
  - Long term benefits of CAD revascularization (if it is performed) may not apply for pts with PVD
  - Revascularization EVEN for hemodynamically significant lesions may not be better than aggressive medical management
    - CAPRI (CABG) and COURAGE (PCI) trials
- There are no validated invasive or non-invasive methods to ID palues that are vulnerable to disruption
- Hence preop optimization should aim at plaque stabilization
- It is agreed there is role for STATINS in reduction of CV mortality
  - Jupiter trial

- Beta Blockers are useful as well
  - Caution, as they may be harmful in some pts (POISE study)
- Well done negative provocative test have high NPV
  - Positive test, however, does not have high PPV

Respriratory:

- Nosocomial pneumonia – leading cause of nosocomial infectious death
  - Pt may develop inanition pneumonitis
    - Debilitated, nutritionally depleted, can't clear secretions
    - Atelectasis -> pneumonia
- Risk Factors – smoking, COPD, obesity, URTI, poor health
- Quit smoking at least 1 month ahead
- Routine CXR preop useless –
  - 14,000 pts reviewed -> in 140 CXR abnormal,
  - In only 14 pt abnormal findings caused change in management
- Acute Respriratory failure: hypoxic, hypercapnic, both
- Prevention of post op problems:
  - Prevent atelectasis
  - Minimize artificial ventilation
  - Strict glucose control - QUESTIONED in recent reviews…

290.Ischemic neuropathy:

- Large nerve – due to thrombosis, embolism, injury
- Small nerve – PAN, Rheumatoid vasculitis, Churg-Strauss, Wegener, DM
- Some say muscle is more susceptible to ischemia than nerve

- Acute ischemia –
  - if more than 24h denervation – axonal degeneration **of both myelinated and non**-myeliated nerves
- Chronic ischemia –
  - **mostly myelinated n.** affected – de and re-myelination, edema.

- Temporal and functional effect on nerves in humans in not well defined.
- Sensory deficits, no DTR seen in 50% of pts with PVD
- Extent proportional to severity
- Stocking and glove distribution, distal muscle wasting/weakness (foot ->proximal)
- DD – uremia, DM, drug, alcoholism. BUT in these – distribution is SYMMETRICAL, in ischemia – it is limited to most PVD affected limb

Diagnosis:

- If ankle pressure >50mm, toe pressure >30 – diagnosis is unlikely
- Electrophysiologic studies

- Treatment –
  - reperfuse,
  - may expect slow regeneration of nerve fibers and relief of symptoms

- **Ischemic mononeuropathy: due to large vessel occlusion** – thrombosis/embolus/PAD
  - Peroneal
  - Tibial
  - Femoral
  - Lumbosacral plexus:
    - Motor and sensory
    - More than one dermatome, several nerves
    - Areflexic flaccid limb
    - Dd – poor recovery in these…
      - spinal
        - Spastic, hyperreflexive, extensor plantar response, dissociated sensory loss
      - Cauda equine
- **Ischemic monomelic neuropathy with vascular access surgery**:
  - Majority are diabetics
  - after antecubital fossa access
    - major watershed area for vasa nervorum for all three nerves
  - symptoms within minutes/hours after access
  - bounding radial pulse
  - tx: ligate fistula

291. Complications of aortic surgery:

- Hemorrhage
- Death
- Ureter injury
- Ischemia
  - Myocardial
  - Colon
  - Renal
  - Limb
  - cerebral
  - spinal
- Infection:
  - Pneumonia
  - wound
  - graft

- Wound related
  - Nerve injury
  - Non-healing
  - Seroma/lymphocele

- Impotence/retrograde edjaculation
- Recurrence:
  - Graft dilatation (knitted)
  - Pseudoaneurysm

292.Strategy to minimize renal damage during aortic clamping?

- Manitol – 12.5 – 25 g before clamping
  - Increase urine flow volume
  - Decrease effects of cortical perfusion reduction
  - Free radical scavenger
- Fluid load before clamping
- Keep warm ischemia under 40 min
- Distal aortic perfusion
  - May reduce spinal ischemia
  - Effect on renal failure is less clear cut with octopus devices
    - Works for type 1 and type 5 TAAA, when you don't need to reconstruct renals
- Cold perfusion with 500 cc 4 degrees NS
  - When anticipating > 45 min clamp time
  - Effects seen with 10 degrees temp lowering
- Avoid atheroemoblism

Unproven – dopamine, fenoldapam

293.Clinical characteristics, risk and diagnosis of ischemic colitis:

- 0.5-10%, overall 2 %
  - If looked for aggressively, will find out in 6% of elective cases
- Mechanism:
  - Thrombosis of intestinal arcaded due to hypotension
    - Unrecognized hypovolemia periop
  - Embolisation of aneurismal contents
  - Traction injury
  - Inapporptiate IMA ligation
    - Not from within of aneu
    - Unrecognized significance of IMA with celiac/SMA stenosis
  - Use of pressors

- i.e. just reimplanting IMA is NOT going to solve all the problems…

- Early post op (24-48 h) – bloody/brow diarrhea in 1/3 of pts
- Abdo pain/distention/fever/oliguria/thrombocytopenia/leukocytosis
    - Ds: clinical presentation plus flex sig
        - High index of suspicion
        - If suspect – scope, if more than mucosa – Hartmann.
    - RF predisposing to colon ischemia
        - Technique:
            - Improper IMA ligation – too distal
                - Particularly if meandering a. is preserved
            - Loss of internal iliacs
                - Embolization during manipulation
            - Retractor injury
                - To collaterals
                - To colon
        - Operative procedure:
            - Ruptured aneurysm
            - Increased X-clamp time
            - Periop hypotension/hypoperfusion
        - Patient:
            - Old age
            - Comorbidities:
                - Previous colectomy/loss of collaterals
                - Prev rads
                - SMA/celiac artery disease

Post op Renal failure increases risk of death 3 fold (to 15%)
Lung and heart problems increase risk of death 2 fold (to 10%)

294. Colon ischemia avoidance after AAA repair:

- Assess IMA backbleeding –
    - re-implant if none or < 40 mm Hg backpressure
    - Ligate IMA at orifice -> preserve arc of Rhiolan
    - check with tonometric colonic mucosa pH: should be > 6.86
- Preserve internal iliacs:
    - Avoid embolus,
    - don't ligate
- Preserver profunda collaterals
- Avoid mechanical mesentery injury – e.g.with retraction
- Better anesthesia:
    - no anemia/hypotension/hypothermia
- Better technique:
    - minimal cross clamp time

295.How to prevent sexual disfuntion with aortic surgery:

- DOCUMENT PREOP
- R sided dissection of aortoiliac segment
- Minimal division of longitudinal periartic tissues to the left of the infrarenal aorta
- Don't dissect at the base of IMA
- Don't cut tissues over L CIA
- Maintain internal iliacs
- Ligate IMA from within
- Use of retroperitoneal approach may be beneficial

Aside, if done with precautions, aortoiliac reconstruction (for AIOD) may restore potency in 25% of pts. However, if no precautions are taken, incidence of impotence is close to 100% even among those that were potent.

296.How to improve pelvic circulation:

- Correct inflow – aortic/iliac disease
- Improve internal iliac
    - Angioplasty
    - Endartherectomy
    - Bypass
- support collaterals:
    - IMA reimplant
    - Profundoplasty
- Check for patency of arch or Riolan
    - IMA reimplantaion if poor collaterals

297.Incidence of complications with different type of access:

- Axillary – 5-15%
- Translumbar – 5-15%
- Femoral – 1-10%

298.Pseudoaneurysm formation: causes

- no previous surgery
    - infection due to IV drug use
    - trauma
    - percutaneous intervention – BY FAR the most common cause of PA overall…
- prev surgery, i.e. anastomotic - 2nd most common
    - 70% degenerative,
    - 25% infection,
    - 5 % suture break

In general, causes are….

- disease related
  - increased outflow resistance due to AsC progression
- arterial wall weakness related
  - progression of AsC
  - infection
  - aggressive Endarterectomy
  - disruption of vasa-vasorum
- prosthetic graft related
  - compliance mismatch
  - graft dilatation and deterioration
- anastomosis related
  - tension due to position (groin/flex)
  - tension due to graft shortness
  - uneven tension at anastomosis
    - short graft
    - native artery elongation
  - suture tear

- so = relating to the artery, graft, suture line, infection, physical stress, tech errors..
- when to fix?

  - Aortic – at 4 cm
  - Iliac – at 3.5 cm
  - CFA – at 2.5 cm
  - when symptomatic
  - During repair, look for the sign of infection… if infected, see infected graft notes.

299.Post Angio pseudoaneurysm: why?

- Interventional rather then diagnostic procedure
- Multiple catheter exchange
- Technical errors/inexperience
- Peri-interventional Multiple (e.g. lovenox, plavix, warfarin) anticoagulation
- Female gender
- > 60 yoa
- Lack of closure device
- Poor selection of access

300.Intraartearial drug injection – mechanism of injury & tx

Mechanisms:

- Vessel obstruction with particles
- Direct endothelial damage with thrombosis – venous and arterial

- Hypersensitivity vasculitis
- Vasospasm

Clinical manifestations:

1. Traumatic complications:
    a. AVF
    b. pseudoaneurysm
2. Infectious complications:
    a. Infected pseudoaneurysm or arteritis
    b. Mycotic pulmonary aneuryms: dyspnea, cyanosis, hemoptysis
    c. endocarditis
3. Thrombotic complications leading to ischemia:
    a. In situ thrombosis at injection site
    b. distal embolization -> ischemia
4. Pharmacologic effects of the drugs:
    a. NOMI/MI with Cocaine

Therapy:

Angiogram to ID local arterial injury

1. intimal flap,
2. AVF,
3. pseudoaneurysm,
4. thrombosis,
5. distal vasospasm

Pharmacologic therapy:

1. Vasodilators (intra-arterial papaverin, tolazoline (25-50 mg) iv severe vasospasm
2. Anticoagulants – heparin, dextran 40

If infected pseudoaneurysm – treat as infected graft.

301. What increases contrast nephropathy?

- Advanced age
- Pre-existing CRF
- Hypovolemia
- Hyperosmolar agents
- DM
- Large volume of contrast
- Repeat doses of contrast
- Co-ingested nephrotoxin
    - ACEI
    - ASA

        - aminoglycosides
    - Multiple myeloma
    - Heavy proteinuria
    - High osmolarity agents

Note, non-inonic agents have similar incidence of adverse side effects as ionic..

302.Advantages of low osmolarity contrast to compared to high?

- Reduces risk of severe allergic reactions by 80%
- Reduces post-venography phlebitis (less endo injury)
- Less nephrotoxic

303.Gadopentate Dimeglumine aka $CO_{2...}$

Low risk of renal disfunction, but poor resolution. Can cause mesenteric ischemia with gas embolism.

304.Complications of blood transfusion:

- fatal ABO incompatibility 1: million
- non-fatal ABO incompatibility 1:250,000
- febrile reaction 1:100
- transfusion related lung injury (rare)
- GVHD in immuno suppressed
- Infections:
    - viral Hep B, C, HIV, HTLV
    - bacterial contamination (platelets)
    - Chagas' disease

305.Difference between seroma and lymphocele?

- Lymphocele has feeding lymph channel. When excising, need to ligate it. Surgery is indicated for enlarging LC or ones close to the prosthetic graft (risk of infection form adjacent LN). Prior to this, legs up, compression, prevention of infection
- Complications of chronic lymph leak:
    - Malnutrtition
    - Lymphocytopenia
    - Anemia
    - Infection of underlying graft
- If chose to investigate – 5 ml of isosulfan blue between toes,
- for mesenteric lymphleaks – 4 h preop 24 oz of whipping cream via NG.
- Percutaneous treatment: talc, alcohol, bleomycin, fibrin – effective, coming into foreground

INFECTED GRAFT

306.Native vascular vessel infection:

	MYCOTIC ANEURYSM	MICROBIAL ARTERITIS	INFECTION OF EXISTING ANEURYSM	POST-TRAUMATIC INFECTED FALSE ANEURYSM
Etiology	Endocarditis	Bacteremia	Bacteremia	Narcotic addiction, Trauma
Age	30-50	>50	>50	<30
Incidence	Rare	Common	Unusual	Very common
Location	Aorta	Atherosclerotic	Infrarenal	Femoral
	Visceral	Aortoiliac	Aorta	Carotid
	Intracranial	Intimal defects		
	Peripheral			
Microbiology	Gram-positive cocci	*Salmonella*	*Staphylococcus*	*Staphylococcus aureus*
		Others	Others	Polymicrobial
Mortality	25%	75%	90%	5%

- Post traumatic infected false aneurysm – MC
- Microbial arteritis with aneurysm - common
- Infected pre-existing aneurysm - unusual
- Mycotic aneu (preantibiotic >90%, now <10%) RARE

Does not include:

- **infection from contiguous source**
- **aorto-enteric fistula**
- **vascular synthetic graft infection**

1. **Mycotic: mortality 25%**

- Due to endocarditis
- 30-50 yoa
- Location:
    - Femoral 89%
    - Upper ext 13%
    - aorta 12%
    - Cranial 4%
    - SMA
    - Where – site of bifurcations, AVF, coarctations.

Bugs:

- if no IVDU – Strep Viridance 22%, SA 20%
- IVDU – SA 36%, Ps. Au 16%

2. **Microbial arteritis with aneu: mortality 75%**
- 50 yoa
- Due to bacteremia
- More common than mycotic
- At the site of Atherosclerosis
- Most commonly aorta (3:1 compared to peripheral sites)
- **77% of all infected aortoiliac aneurysm…**
- MC E.Coli, **Salmonella – predisposition to Aorta**, SA
- Also, AIDS, CRF/hemodyalysis pts are susceptible

3. Infected pre-existing aneu: 90% mortality
- 15% of AAA grow stuff – unknown significance
    - 38% ruptured
    - 13% symptomatic
    - 9% elective
- Bugs:
    - Staph – 41% - St. Epi is most common

4. Post traumatic infected: 5 % mortality
- Most common, lowest mortality
- IV drug use
- Post percutaneous procedure – Risk Factors:
    - Long procedure
    - Repeat cath
    - Difficult access
    - Arterial sheath in >24 h
    - CHF
    - Use of angioseal
- Most Common microorganism – Staph Aureus.
    - Fungal RARE – in DM and immunosuppressed.
- Rutherford's companion states that unless purulence and gross uncontrolled infection, always try to revascularize with autologous in situ repair AND muscle flap (Sartorius)

Presentation: difficult to detect.
- Fever of unknown origin
- Positive blood culture
- Erosion of lumbar vertebrae
- Female sex
- Presence of uncalcified aneurysms
- First presentation of an aneurysm after bacterial sepsis

If see aortic infection – likely microbial arteritis leading to aneurysm
If see femoral infection – likely infected pseudoaneurysm, second possibility - mycotic aneurysm

Lab:

- Negative blood cultures, intraoperative Gram stain
  - ARE NOT sensitive enough to exclude ds
  - In ruptured AAA:
    - Blood culture is positive in 69% , Gram stain – in 50%
  - only 11% Gram stain is positive in non-ruptured AAA
- Even in aneurysm wall culture was found to be positive in only 92%…
  - p.1588

- DSA appearance:
  - saccualr aneu in normal vessel,
  - multilobulated aneu,
  - eccentric aneu with narrow neck
- Lumbar osteomyelitis
- Indium -111 labeled WBC helpful for prosthetic graft infection, NOT infected aneu…

Incontrovertible principles of treatment:

1. **Control hemorrhage**
2. **Confirm ds: gram, culture for bacteria/fungi/TB**
3. **Operative control of sepsis: resect, debride, abx irrigation, drain**
4. **Post op wound care: dressing change, repeat debridements**
5. **Long term abx**
6. **Consider reconstruction through non-infected field: THE ONLY TENENT OPEN TO** CONTROVERSY…

307.CT findings for infected prosthetic graft:

- Fluid around graft
- Gas around graft
- Pseudoaneurysm
- Soft tissue stranding
- Adjacent vertebral osteomyelitis
- Hydronephrosis
- Retroperitoneal abscess

308.Risk Factors predisposing to graft infection:

- Bacterial contamination of the graft
  - Perioperative contamination
  - Hematogenous spread from remote source

        - Erosion of graft into GU/GI tract
        - Contiguous process
    - Risk factors for contamination:
        - Procedure related:
            - Emergency surgery
            - Remote infection
            - Prolonged preop/post op stay
            - Operative particulars
                - Reoperative procedure
                - Simultaneous GI procedure
                - Crush/Rough tissue handling
                - hematomas
                - Contact between skin and graft
            - Post op wound infection
        - Altered host defense:
            - Advanced age
            - Female gender
            - Aspirin use (hematoma)
            - Malnutrition
            - Leucopenia
            - Malignancy
            - Steroids
            - Chemo
            - DM
            - CRF
            - Autoimmune disease

309.Prevention of graft infection:

- Minimize preop stay (to reduce colonization by resistant flora)
- Treat remote infection prior to surgery
- Antiseptic preop shower (Cochrane, 2008)
- Immediate preop shaving (Cochrane, 2008)
- Prophylactic abx preop
- Meticulous sterile surgical technique
    - Autogenous tissues for bypass or endarterectomy
    - Gentle tissue handling, no crush
    - Iodine impregnated drapes to limit contact btw skin & graft
    - Meticulous hemostasis – no hematomas/lymphleaks
    - Meticulous skin closure
    - Rifampin bonded graft (Cochrane, 2008)
    - Close suction drain (Cochrane, 2008)
    - Avoid simultaneous GI procedures
- Early recognition/aggressive treatment of wound infections

- Support nutrition

310.Investigation of pt with draining R groin wound post ABF

- CBC, ESR, CRP
  - sepsis, inflmmatory state
- BUN/CR, lytes,
  - ok for contrast, need for fluid resusc
- Blood culture, wound culture
  - to ID microorganism
- Groin US/duplex
  - to see if graft patent, pseudoaneurysm
- CTA abdo/DSA and runoff
  - fluid/gas around graft
  - vascular reconstructive options
- Indium labeled WBC scan
  - indirect evidence of infection

311.Infected ABF graft: Draining sinus in groin post ABF: approach –

- Concerned with graft infection
- Review old OR notes and indications
- Review current status of PVD and need for revasc
- Culture blood and site
- Confirm patency of graft (dysfunctional graft)
- Image – US/CT/angio – i.e. establish the presence of…
  - Involvement of anastomosis
  - Pseudoaneurysm
  - extent of involvement
  - undrained fluid collection/abscess
  - reconstructive potential
- Treatment:

  - Eradicate infection
    - Antibiotics:
      - Broad spectrum antibiotics to start with
      - Narrow to culture specific abx when pathogen is known
      - Continue abx long term
    - Control source of infection:
      - Remove infected graft
      - Debride to healthy tissue
      - Flap tissue coverage/drain/leave open

- Reconstruction of distal circulation
  - Limited to groin only?
  - Can profunda/SFA bifurcation be reconstructed?
  - Can graft be preserved?
  - Can we consider insitu reconstruction?

If infection is limited to groin, main body may be preserved.
Circulation may be reconstructed:

- via obturator bypass to SFA/pop OR fem-fem (medial tunneling)
  - if only one limb is involved and main body is ok…
- Via Ax-SFA/profunda/pop tunneling laterally in the area away from the site of infection
- Thoraco-SFA/profunda/pop
- Weird&wonderful – carotid popliteal bypass

If infection reaches main body bifurcation, an entire graft must come out. First, revascularize with Ax-distal fem, then remove abdominal portion of the graft, debride aorta, get anterior spinal ligament and omental pedicle to bolster aortic stump. Finally, remove groin limbs and oversew native vessels.

312.Classification of graft infection:

Table 59-2. Clinical Classifications of Prosthetic Graft Infections

Appearance Time after Implantation
Early: <4 mo
Late: >4 mo
Szilagyi's Classification (Applicable to Postoperative Wound Infections)
Grade I: cellulitis involving wound
Grade II: infection involving subcutaneous tissue
Grade III: infection involving the vascular prosthesis
Bunt's Classification (Modified)
Peripheral graft infection
P0 graft infection: infection of a cavitary graft (e.g., aortic arch; abdominal and thoracic aortic interposition; aortoiliac, aortofemoral, iliofemoral graft infections)
P1 graft infection: infection of a graft whose entire anatomic course is noncavitary (e.g., carotid-subclavian, axilloaxillary, axillofemoral, femorofemoral, femorodistal; dialysis access bridge graft infections)
P2 graft infection: infection of the extracavitary portion of a graft whose origin is cavitary (e.g., infected groin segment of an aortofemoral or thoracofemoral graft, cervical infection of an aortocarotid graft)
P3 graft infection: infection involving a prosthetic patch angioplasty (e.g., carotid and femoral endarterectomies with prosthetic patch closure)
Graft-enteric erosion
Graft-enteric fistula
Aortic stump sepsis following excision of an infected aortic graft

i.e. Bunt:

- P0 – cavitary graft infection, AAA and ABF
- P1 – extra-anatomic
- P2 – infection of fem portion of ABF or cervical of aorto/carotid

- P3 – patch angioplasty infection
- GE erosion
- GE fistula
- Aortic stump

313.CT findings of aortoenteric fistula:

- Periaortic gas or fluid
- Proximal pseudoaneurysm formation
- Bowel wall thickening
- Retroperitoneal stranding
- Plane between duodenum and aorta obliterated
- IV contrast seen in bowel

Note, that barium enema or barium GI contrast is contraindicated in AEF – will obscure picture and may cause retroperitoneal spillage/infection/sepsis.

314.Selection of pts for infected graft preservation:

- Not Dacron
- No anastomotic involvement
- No sepsis
- No pseudamonas

    - Note: Dacron may still be preserved but less chance of success compared to PTFE

How:

- General: optimize heart, lungs, kidney, nutrition, work out revascularization potential, map veins
- Pretreat with 3 days of broad spectrum iv abx
- Debride, irrigate in OR
- Sterilize wound:
    - Vanc/gent beads changed every 7 days
    - 1% iodine dressing TID
- Continue culture specific abx
- Sartorius muscle flap coverage
- Long term po antibiotics

On exam, be careful with presenting preservation of graft as a first choice, list it as one of the options only.

315.Selection of infected graft for insitu replacement:

- No sepsis
    - No positive blood and tissue cultures

        - Biofilm culture positive for Staph Epi
    - No graft-enteric- fistula

How:

- Iv abx broad range
- Sterilize field
    - Debride
    - Irrigate
    - Vanc/gent beads q 7 days
    - 1% iodine saline dressing
- Confirm sterilization on culture
- In situ PTFE- rifampin soaked graft vs fem-fem
    - Gentamicin impregnated thrombin glue on anastomosis
- Sartorius muscle flap for groin
- 6 months iv abx then 3 months po antibiotics

On exam, be careful with presenting insitu replacement of infected graft as a first choice, list it as one of the options only.

316. Results of aortic graft infection treatment:

- Staged (ax-fem first, then in 2 days excision of graft) is best
- Don't use pledgets on aortic stump – infection nidus
- If ax-fem got infected, then consider need for revasc…
    - if ischemic, then may do thoracic-fem bypass.
    - If see monphasic signal or 40 mm Hg at ankle, may consider to forgo revascularization

	Mortality	Amputation	Re-infection	Survival > year
Ex-situ bypass & excision	20%	15%	10%	80%
In-situ with vein	10%	5%	1%	80%
Rifampin PTFE	10%	5%	15%	85%

Advantages of in-inisitu vs ex-situ:
Less periop mortality (10% vs 20%)
Less amputations (5% vs 15%)
Similar survival overall

317. Aorto-enteric fistula:

- Primary
    - Less common overall
    - Degenerative sterile AAA expansion most common in this category

    - Duodenal Ulcer (second MC)
    - Cancer, FB
- Secondary
    - Infection at suture line –
        - leads to pseudoaneurysm, expansion, pressure on the duodenum
    - Infection likely latent –
        - skin flora that gets activated with dips in immunocopmetence (p. 904)
    - Pulsatile pressure
    - Duodenum – injury during Transabdominal mobilization
        - Initial fistulae were at body of the grafts when homografts were used
- Manifestation:
    - GI bleed, sepsis, abdo pain – rare
        - GI bleed – seen in 21% of all AAA repairs,
        - only 0.4% of all these bleeds will have AEF
    - Fever/malaise
    - Septic emboli
        - Common – 27%, lead to:
            - Multifocal ostemyelitis and cellulitis
            - Hypertrophic osteoarthropathy
    - Abdo pain due to pseudoaneurysm pressure
- Evaluation
    - Hx and physical
        - UGI bleed, lower GI bleed (aotoappendicial fistula/to limb), AAA repair, systemic signs, abdo mass, LE multifocal cellulitis
    - EGD – to r/o other source of bleed, to 4th portion
    - CT
    - Indium 111 scan
    - Angio to define run off and renal a. location
    - In half the cases need to explore in OR
- Treatment is surgical:
    - Id presence of active hemorrhage
    - Classify AEF – primary vs secondary
    - Indication for repair: AIOD vs aneurysm
        - If EtS AIOD – then take down and patching of the aortotomy is an attractive option…
    - Extent of sepsis

    - Goals:
        - Save life, then preserve limb. How:
            - Control hemorrhage
            - Repair GI tract
            - Control infection

- Maintain adequate distal perfusion

Surgery: DETAILED consent first: honest, realistic, detailed…

- **Scenario 1: pt is bleeding:**
  - lines/abx/emergent surgery
  - TP vs retroperitoneal
    - RP if difficult neck is anticipated,
      - can't see R CIA, R renal and can't do Right ax-fem
  - Supraceliac control of bleeding first – place clamp but don't close it until needed
    - Medial visceral rotation vs through the crus
  - Distal control
  - Peel of duodenum, put a stitch to control spillage if needed
  - Resect/debride infected aorta
  - Decide on in-situ vs extra-anatomic
  - Repair duodenum:
    - Primary, roux-en-y, +/- gastrostomy, jejunostomy
  - Assess extremities
    - If monophasic signal/>40mm Hg at ankles may forego revasc

- **Scenario 2: No bleeding:**
  - Confirm diagnosis AND rule out GI bleed (other sourcesss)
    - CT, EGD, tagged RBC, WBC scan
  - Consider extra-anatomic revascularization first prior to excision.

- If primary AEF – only 30% are infected
  - May consider in-situ repair with life long surveylance
    - Possible if minimal retroperitoneal soiling and no sepsis
    - Allows for simple durable revascularization
    - Uncertain long term potential for infection
  - Safer option is an extra-anatomic repair

- Bacterial seeding in AEF can occur in about 25% of all the EAB…
  - For typical synthtic graft, the risk is about 10%....
- Composite extra-anatomic bypass –
  - SFV to infected groins, prosthetic to axilla… any merit? P.910
- Role of EVAR?
  - Limited… may be considered for primary
  - AEF after EVAR has been described as well…

- Results of Aorto-Eneric fistulae repair:
  - Natural history – bleeding, sepsis, death

- Operative repair -30-40% mortality
- Amputation 10%
- 3 year survival 50%

TRAUMA:

318. Carotid a. injury and neurologic deficit. When to fix?

- **Neurologically asymptomatic, no occlusion** - fix
- **Neuro asymptomtic, occlusion**
  - Some may say "thank you lucky stars"
  - Rutherford states repair to prevent delayed complications
    - AVF
    - Thrombus propagation
    - pseudoaneurysm
  - Anticoagulate and don't fix it…

- Comatose, no occlusion – be careful to r/o other causes of coma
- **Comatose, occlusion** - fix
  - Initial anectodal reports of converting ischemic stroke to hemorrhagic.
  - Concerns about distal embolizaiont during carotid repair are unfounded according to Rutherford
  - Controversion area but Rutherford suggests to explore
  - Try to restablish back flow with up to the level of the skull fogart
    - re-perfuse if backbleeding
    - If can't backbleed or too extensive – ligate, +/- anticoagulate.
  - All available evidence suggests **optimal neurologic outcomes are obtained with operative repair because most deficits remain unchanged or improve.**
    - Can't always discern the etiology of coma: alcohol, metabolic, durgs, shock, vs vascular injury

Comatous pts	mortality	Optimal normal outcome
28 pts ligated	60%	15%
42 pts reperfused	25%	50%

p.1009

- Minor carotid injuries can be followed with angio/DUS

319. Treatment of blunt carotid injury:

- Carotid-cavernous sinus fistulae – balloon occlusion technique

- Dissection – AC alone, rigorous follow up
    - 90% recovery to normal function if AC started before neuro deficit, 40% after the onset.
    - Survey for dilatation:
        - 62% reverted to normal
        - 29% progressed to pseudoaneurysm
    - Stents – controversial
- Pseudoaneurysms
    - Surgical repair if easy to access or simple
    - Otherwise AC, ligate,
    - Rarely EC-IC/cervical petrous ICA bypass
- Vertebral artery injury:
    - If penetrating and exsanguinating:
        - Treat it with ligation: very little downside…
        - 3% chance of brainstem stroke if L is ligated, 2% if right
        - May try endo to occlude
    - If blunt/occluded/AVF/pseudoaneurysm:
        - Observe with AC, follow with angio
        - Consider endo occlusion (thrombosed artery in fact may be completely transected).
        - This will address concerns re: rebleeding
    - If dissection – usually see in V3 segment, 1-2/52 post trauma: 80-90% present as posterior circulation infract…
        - P.1012

320.Chest vascular trauma:

- Ascending aorta and arch – require full cardiopulmonary bypass, hypothermia and cardioplegic arrest
    - Aside: arch vessels may be reconstructed with side biting clamp to assending aorta and a bypass
- Distal aorta:
    - Clamp and sew
    - Off load heart:
        - Atrio-femoral bypass (Most common)
        - Axillo-femoral (time-consuming)
        - Full cardiopulmonary bypss (used rarely)
- Aortic trauma repair general points:
    - Pre-warm room and fluids to 40 degrees
    - Prox control btw L CCA and L SCA
        - Watch out for vagus and thoracic duct
        - L vertebral artery takes off arch in 8% of cases

- Do not debride aorta
- Do not sacrifice intercostals
- Move clamp closer to injury
- Fine suture and knitted graft
- For grafts taking off ascending aorta, use single limb, multiple if necessary
    - Premanifactured bifurcated are too bulky, may not fit in anterior mediastinum…
    - Use bovine pericardium to cover this

Midline sternotomy: helpful points

- Skin from Sternal notch to xyphoid
- Develop retrasternal plane above and below, no need to connect these
- ASK anesthesia to deflate lungs to minimize chance of pnemo
- Oscilating saw
- Divide thymus
- L brachiocephalic vein:
    - Preserve
    - ligate its' thyroid tributaries
        - will allow mobilization of this vessel.

Anterior thoractomy: for subclavian exposure

- Avoid R sided central lines – may cause pneumo –
    - won't be able to do single lung ventilation
- Double lumen ET tube

- Supine, roll under L shoulder and hip to bring chest up 20 degrees
- L infrapectoral incision
- ID 5th rib, go above it – 4th intercostal space
- Rib spreader – may need to divide internal mammary a/v
- Deflate lung, push it down
- ID arch under mediastinal pleura
- Incise it, preserving L vagus nerve coursing anterolaterally over origin or LSA
- Thoracic duct is posteromedial - preserve

L Postero-lateral thoracotomy: for descending aorta exposure

- Bean bag, strap hips,
- True lateral postion, roll under R axilla, L arm over Mayo stand
- Incision from below L nipple to 1 inch below tip of scapular
- Divide serratus ant, lats, trapezius – slide shoulder girdle up
- 4th ICS for middle descending aorta, 6th ICS for distal
- Verify ICS from above by counting
- Protect phrenic and vagus nerves coursing over arch

321.Radiographic clues to potential blunt aortic injury:

- Loss of the shadow of the aortopulmonary knob
  - more specific than wide mediastinum
- Loss of parevertebral stripe
- Loss of aortopulmonary window

- Depression of the LMSB > 140 degrees
- Deviation of nasogastric tube
- Lateral displacement of trachea

- Apical hematoma
- Wide midiastinum > 8 cm
- Massive left hemathorax

- Fracture of the sternum, scapula, multiple left ribs, clavicle, pelvis
- Blunt injury to the diaphragm

322.Most common blunt vascular thoracic injuries:

- Descending aorta distal to LSA
- Innominate artery

323.Conservative treatment of thoracic aortic injuries:

- Three categories in multiply injured pts
  - Massive injuries, exsanguination on site
  - Unstable during transport, transient responders:
    - high mortality rate due to multisystem trauma
  - HD stable, confined mediastinal hematoma:
    - these can be observed.
    - Death is due to head injury

- If chosen to observe, use
  - Impulse reduction therapy
  - MAP at minimum (<90)
  - Ensure stability of mediastinal hematoma on serial imaging
  - Pt is fully informed re: risk and benefits
  - Management during delay is supervised by vascular surgeon
- Can delay up to 72 h
  - fibrinous organization of mediastinal clot takes place

324. Abdominal vascular trauma:

Korean/veietnam war – 3%
Civilian:
- Penetrating:
  - gun shot – 14%, stab 10%
- blunt 3%
  - MC renal, then SMA

Retroperitoneal hematoma: when to explore

1. Explore all penetrating
   a. **Exception – stable perinephric NOT involving the hilum**
2. Blunt: explore only if
   - Leaking
   - Expanding (some say only RAPIDLY expanding)
   - Pulsatile
   - Paraduodenal
   - **Root of mesentery (i.e. SMA) PLUS ischemic bowel**
   - Zone 3:
     - Intraperitoneal Bladder injury
     - male urethral injury
     - pulseless leg

aside:
- ***blunt injury to the iliac artery – leads to thrombosis due to stretch.***
- ***EXPLORE only if:***
- ***Intraperitoneal leak***
- ***Expanding***
- ***Absent or diminished femoral pulse***
  p. 1039

On exam it is safe to simply do fem-fem, if there is no need to explore the abdomen (and L hematoma…). However, in stable individual, Rutherford suggests exploration and, possibly, iliofemoral bypass…Hemodynamic and patency result of fem-fem in a young pt is FAR inferior to iliofem. For Iliofem – use SFV if contamination… But for exam purpose, do not do iliofem – will be too contaminated for the prosthetic, and will take too long to get SFV.

- Unexplored hematomas are to be followed by Doppler and Doppler.
- Enteric spillage is not contraindication to prosthetics:
  - so consider extrananatomic bypass only in case of purulent peritonitis and infected graft (Rutherford)
  - Multiple trauma experience suggests safety of prosthetics even in the face of entric contamination. Cover this with grafts
- Supra mesocolic a.control –

    - L visceral rotation, diaphragmatic control, L chest
- Inframesocolic -
    - Transverse colon reflected cephalad, small bowel to the R.

SMA – retropancreatic portion (may transect neck, or supraceliac proximal control), infrapancreatic portion (retract pancreas up).

When to ligate aortic branches:

- SMA – only in the presence of necrotic bowel AND only at the origin – above gastroduodenal and inferior pancreatico-duodenal arcade.
    - Elsewhere – high incidence of bowel ischemia.
- Celiac – ok if SMA is ok
- CHA – proximal to take off of GDA.

- L renal vein ligation at IVC is tolerated well,
- R renal vein ligation is always followed by nephrectomy.

- NEVER ligate CIA or EIA without reconstruction, even in pts in critical condition.
    - Place temporary shunt if necessary.

- May ligate iliac veins –
    - If repair will result in stenosis > 50%
    - Prophylactic fasciotomies are controversial
    - No role for complex spiral graft reconstructions

- IVC – may ligate if infrarenal
    - Wrap and elevate legs…
- Renal a. injury –
    - revascularize if
        - within 6 h, if single or
        - within 20 h if SOLE kidney injury or bilateral damage
            - authorities vary on exact timing
    - Success of revascularization is poor at about 30%...
        - in 12-50% of revascularized pts hypertension develops
        - If non-revascularized, HTN develops in 30-40% within 1 month to a year, mean 3 month
    - Some recommend surgery for Renal Artery repair only in:
        - Intraoperative identification of renal artery injury
        - Stable pt
        - Solitary kidney present or bilateral injury
        - The rest – monitor for hypertension…

325. Extremity trauma notes:

- Most common type of vascular trauma (80% of all types)
  - Penetrating 90%, blunt 10%
- Hard signs – go to OR, haparinize if possible

- Angiogram indications in extremity trauma:
  - Significant blunt injury with
    - #/dislocation AND signs of ischemia
    - ABI <1
  - Multiple penetrating wounds

  - P.1003
- Angio is 92-98% accurate, most errors involve false positive
- Always have global approach to trauma
  - r/o OTHER vascular injury
  - r/o other NON-vascular injury
  - assess neurologic and anticipated FUNCTIONAL status of extremity
    - i.e. mangled extremity check.

Hard signs:

- Any of 6 "P"
  - Pain
  - Pallor
  - Pulseless
  - Paresthesia
  - Poikilothermia
  - paralysis

- Ongoing bleed
  - External
  - Internal:
    - Pulsatile hematoma
- Bruits and thrill
  - AVF

Soft signs:

- Distal pulse deficit
- ABI < 1
- Presence of soft signs of arterial injury
  - proximity to major vessels

- small non-pulsatile non-expanding hematoma
- h/o prehospital bleed/shock
- peripheral nerve deficit
- fracture dislocation

Minimal in line intimal angio injury –

- intimal flap,segmental narrowing, HD insig AVF/pseudoaneurysms – heal without surgery
- can be serially observed with duplex/angio.
- Non essential vessels/aneurysms can be embolized.

note: for proximity to major vessel injury, most would do serial ABI only, no angio.

Occult injury:
If chose to observe, must be able to follow with physical exam and duplex. Missed injury seen in only 2-3% of cases (Osler)

Posterior knee dislocation:

- Reduce first
- Check ischemia/pulses
- Explore if no pulse after reduction or ischemia
- If ABI is reduced but no ischemia – angiogram -> fix only major problems.

326. Traumatic AVF:

- Iatrogenic (post procedure)
- Non-iatrogenic (true trauma)

If central in origin, unlikely to close, so will require repair.
If peripheral and is NOT associated with true trauma (i.e. iatrogenic origin post needle stick), 90% will close in 4 months (average length of closure is 28 days).

Indication for repair:

- Fluid overload
  - Venous HTN
  - CHF
- Distal ischemia
- Non-compliant patient
- Central location/neck
- Post trauma (as opposed to procedure)

General recommendation is to wait 4 months unless there are indications (above). Repair failures. Proximal and distal control. Expect major blood loss.

If in mid SFA – consider short covered stent… May try US guided occlusion.