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So read your Rutherford, folks...

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Overview of Oral Exam

1. History
2. Physical exam
3. Medical management (Heparin, ASA, statins, etc)
4. Explain rationale for differential diagnosis
5. Nonoperative management
6. Noninvasive studies
7. Anatomic imaging – memorize CT and angiogram findings of rarer things like carotid body tumor, fresh thrombus in ICA, inflammatory aneurysms etc
8. Endovascular options versus operative options
9. Explain rationale for operative plan
10. Treatment of complications
11. Follow up

Carotid Aneurysm:

1. History – TIA's, amaurosis, neck pain, dysphagia, CN compression. Mass effect and thromboembolic issues.
2. Physical – bruit, pulsatile mass
3. Work-up
 1. carotid duplex
 2. head CT/MRI
 3. angiography with back pressure measurement (end-hole balloon occlusion catheter)
4. Natural History
 1. dependent on etiology (atherosclerotic vs. post-traumatic vs. mycotic)
 2. most common complication is stroke (up to 50% in some series)
5. Treatment
 1. Resection with interposition bypass – Vein map patient preoperatively
 1. routine shunting (Pruitt shunt, can serve as distal control also)
 2. High exposure techniques
 1. nasotracheal intubation
 2. division of posterior belly of digastric muscle

1. When you divide this, glossopharyngeal nerve might be injured, CN IX involved in sour taste on back of the tongue, as well as gag reflex.
 3. subluxation of mandible
 4. ENT consultation
 5. Mobilize parotid gland
 6. Divide occipital branches around the hypoglossal
 7. Divide stylopharyngeus, styloglossus, stylohyoid.
 8. Use a balloon for distal control
2. Ligation
1. measure back pressure (if greater than 60-70 mmhg, usually ok, or at least 50% of systemic pressure). This can be done in IR with a balloon with end hole for 30 minutes or in the operating room with an arterial line set up.
 1. EC-IC bypass if back pressure not sufficient
 2. results in thrombosis from point of ligation to the origin of the first major intracranial collateral artery (usually ophthalmic)
 3. 7-10 days of postop heparinization to prevent propagation of thrombus

Notes: Cranial nerve injury rate is higher. Check both sides with an ultrasound. 21-50% mortality secondary to stroke if untreated. The presence of a CAA is an indication for repair. Bilateral 20% of the time. Symptoms occur secondary to thromboemboli. Angiography: although most involve the carotid bulb and extend up into the ICA, some extend into the carotid siphon. If asymptomatic fix, particularly if greater than 2 cm. Stroke/mortality 10% with operation. OR – can use balloon occlusion for distal control if too high. Ligate external carotid. Saphenous vein graft, end-to-end. 25% cranial nerve injury rate.

N.B. High exposures required – Carotid aneurysm, carotid fibromuscular dysplasia, carotid body tumor

Carotid FMD:

1. History – TIA's in young women. Cerebrovascular symptoms in young women.
2. Physical – bruit
3. Work-up
 1. Carotid duplex – 2-6 cm segment of medial fibrodysplasia adjacent to 2nd/3rd vertebra. Usually, the duplex will show no irregularities, but the contour of the vessel is irregular
 2. Check bilateral sides. Maybe check renal arteries as well?
 3. Angiography – Note FMD occurs in branchless medium size arteries
 4. CT scan potentially – 25% of patients have intracranial aneurysms.
4. Natural History
 1. if asymptomatic, <10% risk of stroke
 2. symptomatic lesions usually treated, no good studies to examine untreated symptomatic lesions.
5. Treatment
 1. Open graduated dilation
 1. high exposure
 2. clamp common carotid artery, traction on proximal internal carotid artery
 3. small longitudinal arteriotomy, dilators 1.5 – 3.5mm. Do not use anything larger than 5mm in diameter as that size might cause dissections.
 4. Do not clamp ICA, but only clamp CCA and ECA.
 5. allow generous backbleeding

6. consider 500ml of Dextran 40 at 25 ml/hr perioperatively
7. Do not shunt as you will embolize the webs.
8. Worry about glossopharyngeal nerve injury and hypoglossal injury.
2. open PTA vs standard PTA, just as you would with carotid intervention, but make sure you use embolic protection device.

Carotid Stenosis:

1. History – TIA, stroke, amaurosis
2. Physical – bruit
3. Work-up
 1. Carotid Duplex
 1. 1-15 % stenosis: duplex findings no flow reversal
 2. 16-49% stenosis: duplex findings spectral broadening
 3. 50-79% stenosis: PSV > 125 cm/sec EDV<140 cm/sec
 4. 80-99% stenosis: EDV > 140 cm/sec
 5. PSV Ratios: ICA/CCA 2:1 = 50% stenosis, 4:1 = 70% stenosis
 2. angiography if equivocal
 3. head MRI/CT if acute stroke suspected
 4. Make sure there is an EKG to rule out atrial fibrillation as a cause of stroke.
4. Natural History
 1. Risk of stroke in 2 years (NASCET) = 26%, reduced to 9% after CEA
 2. Risk of stroke in 5 years (ACAS) = 11%, 5% after surgery. 3.9% if you exclude the risks of carotid angiograms during the ACAS trial. 60% by arteriography = 80% by Doppler.
 3. 20% postoperative stroke for stroke-in-evolution
 4. OK to do CEA in patient with stable neurologic symptoms
 5. Best medical therapy at the time included aspirin alone. Now it should include a statin, aspirin, and if neurologic event happens while on these two medications, add clopidogrel
5. Treatment
 1. CEA
 1. routine shunt vs EEG vs stump pressures vs awake
 2. Use a shunt for all symptomatic patients.
 3. routine patch vs eversion technique
 4. doppler signals post procedure
 2. CAS for high risk (difficult local anatomy, contraindication to general anesthesia)
 1. Indications for carotid artery stenting
 1. Radiation to neck
 2. Previous CEA with symptomatic restenosis
 3. High medical risks
 2. Predictors of Stroke
 1. Tandem lesions, older age patients, more plaque at ostium of great vessels
 2. Lesion length greater than 1.5 cm
 3. Type of arch (Type I vs Type II vs Type III arch)
 3. Consider dextran 40 – 20 cc bolus, then 10cc/hr, preop aspirin
 4. Make sure you keep the blood pressure less than 140 mmHg post op with labetalol

Notes: 1. DDx Hypotension post-CEA: Hypovolemia, MI, anaphylaxis, intracranial bleed, stroke, autonomic dysfunction (usually resolves in 24 hours). Do not treat with medications unless they are symptomatic from the hypotension. 2. DDx of hypertension post CEA: pain, missed dose of antihypertensives, intracranial bleed. 3. DDx of headache post CEA: thrombosis of carotid,

hypertension, embolic issues.

DDx for stroke under the age of 40.

1. Non-atherosclerotic large artery disease
 1. Aortic dissection
 2. FMD
 3. Moyamoya (Tx is EC-IC bypass)
 4. Vasculitis
2. Cardioembolic
 1. Endocarditis
 2. Rheumatic heart disease
 3. Atrial fibrillation
3. Hypercoagulable states
 1. Antiphospholipid antibody
 2. Sickle cell disease
 3. DIC/TTP
 4. Protein C/S deficiency
 5. Antithrombin III
 6. Factor V Leyden
4. Substance abuse – Cocaine, methamphetamine, etc
5. Migraine
6. Cryptogenic
7. Dissection
 1. Trauma
 2. Spontaneous
 3. Hypertension
 4. Connective tissue disorders
 1. Marfan's Syndrome
 2. Fibromuscular dysplasia
 3. Ehlers Danlos Type IV

Hyperperfusion post CEA:

1. History – HTN, BP lability, headache
2. Physical – noncontributory
3. Work-up
 1. head CT (noncontrast)
 2. head MRI (FLAIR) – look for cerebral edema. T2.
4. Natural History
 1. 2-3% incidence
 2. 25-50% cerebral hemorrhage
5. Treatment
 1. aggressive blood pressure management
 2. EEG – check for subclinical seizure activity
 1. Possible anti-seizure medication
 3. Consider stopping antiplatelet/antithrombotic medication

Acute Stroke post CEA:

1. History – immediate vs. delayed
2. Differential diagnosis for stroke after CEA: intracranial hemorrhage, watershed infarction, hypoperfusion intraoperatively or post operatively, hyperperfusion, embolization during or after CEA with or without technical problem. HIT, low flow secondary to kink, embolization from cardiac source.
3. Physical – essentially MCA problems lead to upper extremity motor and sensory problems, ACA problems lead to lower extremity problems. Aphasia relates to the dominant hemisphere. Dysarthria is related to the nondominant hemisphere. Neglect of a side relates to the non-dominant hemisphere.
4. Work-up
 1. Carotid duplex (on way to OR if within 1 hour)
 2. CT scan (if >2 hours)
 3. Angiography (if > 2 hours)
5. Natural History
 1. reversible if treated immediately
6. Treatment
 1. re-exploration (for immediate, 1 hr delayed)
 2. cerebral angiography with intra-arterial thrombolysis (intra-cranial emboli, patent endarterectomy site)
 3. Essentially this should be the algorithm: if there is a deficit upon waking up of within 1 hour or so, go open the wound in the OR under local anesthesia. Why local: no hypotension on induction. Then perform intraoperative duplex. If the duplex shows a technical defect or clot, heparinize and open the carotid. If it doesn't, then get an angiogram and have neurointerventionalist help. If angiogram is negative, get a head CT to look for bleed.
 4. Timing of stroke after CEA
 1. Awakens with neurologic deficit: Do duplex as most likely the artery has no thrombus. Head CT and neurorescue are the best options. So just open up the wound, as you are already in the OR.
 2. Immediate post op period stroke after normal period. This is secondary to a technical error that results in postoperative thromboembolism – intimal flap, suture line problems, kink, clamp defect. Go to the OR and fix if problem occurs within 1 -2 of initial procedure, but realize the risk of hemorrhagic conversion is high. As an aside, if you see a clamp defect on completion duplex and the patient is asymptomatic, leave it alone.
 3. Deficit 1-2 days after CEA is usually secondary to cerebral hemorrhage and/or edema secondary to hyperperfusion. The syndrome is HTN/Headache/seizures. Tx = BP control and anti-seizure medications, in addition to minimizing anticoagulation.

Recurrent stenosis post CEA:

1. History
 1. early (<2 years) vs. late
 2. symptomatic vs. asymptomatic
2. Physical – bruit
3. Work-up
 1. carotid duplex
 2. angiography
4. Natural History
 1. Early – may regress, rarely progresses to occlusion
 2. Late – recurrent atherosclerosis, higher stroke risk
5. Treatment

1. redo-CEA
 1. patch vs. interposition
 2. CAS
6. Generally speaking, only treat symptomatic recurrent disease, as the majority of recurrent stenoses are intimal hyperplasia. So asymptomatic recurrence: leave it alone.
7. If immediately post op, restenosis is due to incomplete CEA or technical problem with repair. Within 2 years, you get myointimal hyperplasia, and after 2 years it is due to recurrent atherosclerosis.

Stump Syndrome:

1. History – TIA, stroke, amaurosis. If you have a symptomatic carotid from an occluded side, look at the duplex yourself. Also you might want to get a confirmatory study (CTA, MRA, or angiogram) to look for a string sign. Then if that is negative, then it is reasonable to assume you have stump syndrome.
2. Physical – noncontributory
3. Work-up
 1. carotid duplex
 2. carotid angiography
 3. head CT
4. Natural History
 1. limited data
5. Treatment
 1. ECEA + ligation of ICA
 2. In the operating room, look for atretic, small, ICA, without any backbleeding. If that's what you find, then ligate the ICA. Then heparinize for 7-10 days afterwards.

External carotid to internal carotid (EC-IC) bypass

1. Indications – essentially it is performed for symptomatic complete carotid occlusion and failed medical management.
2. Before you call neurosurgery, ligate cervical ICA and do external carotid endarterectomy.
3. Bypass is actually performed from the superficial temporal artery to a branch of the middle cerebral artery.

Fresh thrombus in ICA.

1. Remember on angiogram fresh clot looks like white filling defects
2. Heparinize, immediately go to the operating room
3. Standard carotid exposure, clamp the ECA and CCA, but don't clamp the ICA.
4. You want to let the internal carotid backbleed while you get the clot out
5. You also need to do a gentle #2 Fogarty catheter directed ICA thrombectomy. If you go too high, you can get a cavernous sinus-carotid fistula. The symptoms of this will be severe headache and ocular issues. Even if it is a redo carotid, do an open exposure.
6. If there is amaurosis fugax in setting of stenotic ECA and occluded ICA, then perform ECA endarterectomy with patch angioplasty and ICA ligation.

Subclavian Steal:

1. History – vertigo, nausea, imbalance, diplopia, perioral numbness. Generally, the symptoms are provoked by orthostatic maneuvers or BP meds, not by arm activity. You might see vertebrobasilar and hemispheric ischemia, if concomitant carotid disease. As a note – even if you have vertebrobasilar insufficiency, if there is concomitant carotid disease, you treat the CEA first.
2. Three presentations: (a) Arm ischemia/fatigue with activity (b) coronary subclavian steal syndrome (c) vertebrobasilar insufficiency.
3. Physical – palpation of pulses, check both arm BP 25 mm Hg difference is significant
4. Work-up
 1. carotid duplex – reversal of flow in vertebral artery
 2. arch angiography
5. Natural History
 1. persistent symptoms
 2. posterior/brain stem infarct
 3. coronary steal
6. Treatment
 1. subclavian transposition/carotid-subclavian bypass
 2. For the bypass: tunnel behind SCM, but anterior to IJ vein per the book. I do it posterior to the IJ, but anterior to the phrenic nerve. Do not do subclavian endarterectomy secondary to the fragility of the vessel – you will destroy it.
 3. subclavian stent. Avoid stenting if close to the vertebral artery or the LIMA.
 4. Other options: ax-ax bypass, sc-sc bypass, fem-sc bypass

Arm pain DDX: TIA, DJD, cardiac event, supra-aortic occlusive disease, TOS, embolic issues from aneurysms, carpal tunnel, causalgia.

Syncope DDX: Stroke, vertebrobasilar insufficiency, cardiogenic shock, hypotension, BP meds, arrhythmias.

Note: If true symptomatic vertebral artery disease, still treat anterior circulation first (i.e. CEA) instead of doing a vertebral bypass.

Carotid Body Tumor:

1. History – neck mass, pain, hoarseness, tinnitus
2. Physical – laterally mobile but vertically fixed neck mass. Also called Fontaine’s sign. Bruit over the mass
3. DDX of neck mass: lymphoma, met or head/neck cancer, parotid tumor, reactive lymph node
4. Exam: Laryngoscopy preop to assess for chord paralysis. Also might do a laryngoscopy prior to carotid aneurysm repair and prior to a CEA in the setting of contralateral CEA, redo CEA.
5. Good cranial nerve exam: Marginal mandibular, glossopharyngeal, vagus, hypoglossal, superior laryngeal.
6. Work-up
 1. Carotid duplex
 2. CT/MRI neck – Memorize the diagnostic angiogram findings: splayed out bifurcation with vascularity of the area between the ICA and CCA.
 1. Shamblin classification (displaced, compressed, encased)
 1. I – small, minimally attached to carotid arteries
 2. II – larger tumors with moderate attachments
 3. III – very large tumors encasing arteries

3. Angiography
4. Other: get a vein map, urine catecholamines, plasma metanephrines. Vein map in case you have to resect the entire thing and do a bypass, catecholamines, because 5% are actively secreting them and you might need to treat just as you would a pheochromocytoma (alpha blockade, special anesthesia, etc).
7. Natural History
 1. unpredictable malignant potential (2-50%)
 1. metastatic spread typically to lymph nodes, occasionally thyroid, kidney, pancreas, cerebellum, lungs, bone, brachial plexus, abdomen, breast (5% metastatic rate)
 2. local complications of dysphagia, CN injury, asphyxia
 3. 5-10% recurrence even after surgery
8. Treatment: nasotracheal intubation, vein mapping, plan to excise, but back up is excision and bypass
 1. resection
 1. consideration for preoperative embolization, particularly if $x > 5$ cm.
 2. Blood supply is the external carotid artery
 3. standard vs. modified "T" incision
 4. prep for saphenous vein harvest and high cervical exposure
 2. Radiation – high morbidity, palliative only
 3. Treatment Part II: Nasotracheal intubation, EEG, bipolar cautery in the periadventitial or subadventitial plane. I.D. CCA at the level of the omohyoid. Separate the hypoglossal, vagus, spinal accessory nerve, and glossopharyngeal nerve from the ICA side. If it can't be removed, then resect with interposition grafting.
 4. 25% temporary nerve problems 5% permanent. They may have labile BP afterwards. Screen family members.

Carotid Trauma:

1. History
 1. penetrating vs. blunt
 2. symptomatic (neurologic deficit) vs. asymptomatic
2. Physical
 1. Injury zone (penetrating)
 1. I – sternal notch to 1cm above clavicular head
 2. II – 1cm above clavicular head to angle of mandible, of course, deep to platysma
 3. III – angle of mandible to base of skull
 2. active bleeding/expanding hematoma
 3. upper extremity pressures
 4. neurologic exam
3. Work-up
 1. carotid duplex
 2. angiography. Cut off sign means either thrombosis or transection. Don't assume you need extravasation for a transection.
4. Natural history
 1. pseudoaneurysm formation
5. Treatment
 1. exploration
 1. penetrating zone II injuries
 2. blunt injuries with hard signs/pseudoaneurysm
 2. carotid stent (must be a covered stent)

1. pseudoaneurysm
3. anticoagulation (3-6 months)
 1. blunt injury carotid dissection
 2. blunt injury carotid thrombosis

Carotid dissections can be treated with anticoagulation alone (99%). For testing purposes, if they are still symptomatic, then potential operative approach would be to try to tack the right layers together with open procedure vs stenting procedure vs ligation \pm EC-IC bypass. Over 50% of patients with carotid dissections, get an ipsilateral Horner's syndrome: ptosis, miosis, anhidrosis. Imaging might find an intramural hematoma or "crescent sign". Tx wth anticoagulation for 3-6 months. If you see a Horner's sign, get a stat head CT before anticoagulation.

Aberrant Right Subclavian Artery:

1. History – dysphagia
2. Physical – arm BP's
3. Differential diagnosis: malignant and benign tumors of the esophagus, esophageal strictures, webs, diverticula, peristaltic disorders of the esophagus, and extrinsic compression by vascular structures.
4. Symptoms: Cough, shortness of breath, right arm claudication, right arm embolic, GI hemorrhage from esophageal erosions. Dysphagia lusoria from esophageal compression.
5. Work-up
 1. EGD. Endoscopy vs barium swallow shows extrinsic compression of the esophagus.
 2. CT scan
 3. angiography
6. Natural History
 1. aneurysmal degeneration
7. Treatment
 1. Ligation with re-implantation
 1. right supraclavicular/axillary approach with re-implant to right common carotid artery
 2. Right SC to right CC transposition, with transection of the right SC to the left of the esophagus. Mobilize the right SC from behind the esophagus and oversew the proximal stump. The broad based stump/origin of the right SC, does not necessarily need to be oversewn.
 3. ligation of proximal subclavian as far proximal as possible (mobilize retro-esophageal portion). If you need to ligate, one options is a left posterolateral 5th intercostal space thoracotomy and resection of the diverticulum. The other option- which essentially treats the diverticulum like a small penetrating ulcer, is to perform a TEVAR and simply cover the aneurysm. It will end up thrombosing.
 2. Surgical risks include recurrent laryngeal nerve injury, right brachial plexus injury, esophageal and tracheal injury. Also screen for AAA in these patients.

Innominate Artery Stenosis:

1. History – TIA, stroke, amaurosis, digital ischemia, vertigo, nausea, imbalance
2. The history will be concomitant right carotid and right subclavian symptoms. For example, right arm emboli and left arm weakness, etc.

3. Physical – arm BP difference
4. Work-up
 1. carotid duplex
 2. arch angiography
5. Natural history
 1. symptomatic vs. asymptomatic
6. Treatment
 1. anatomic open repair
 1. endarterectomy
 2. aorto-innominate bypass: Side biter on the ascending arch, end-to-end to innominate and oversew the stump. Tunnel behind the innominate vein. clamp arch near the origin of the innominate artery, while avoiding the LCCA. At the aortic wall, you need tacking sutures. Stay lateral on the wall of the ascending aorta, make sure the arch is not calcified before you clamp. Also, if debranching you can use a bifurcated graft and sew each limb to the carotid or the subclavian. You can also debranch with a bifurcated graft to the innominate and to the left subclavian. If the bifurcated graft is too bulky in the chest, other option is to sew a graft off the innominate artery bypass as a “Y” configuration.
 2. cervical reconstruction
 1. carotid-carotid bypass
 2. Ax-ax bypass
 3. Left SC to right carotid bypass
 3. stenting
 1. balloon expandable
 2. open retrograde approach
 3. femoral percutaneous approach

Buerger's Syndrome:

1. History – younger patients, smoker, claudication, digital ulcerations
2. Physical – ulcerations, +Allen's test, Raynaud's phenomenon
3. Work-up
 1. Labs:
 1. CBC, LFT's, creatinine, FBG, UA
 2. Cryoglobulin, anticentromere Antibody
 3. ESR, C-reactive protein, ANA, RF, complement
 4. serologic markers for CREST syndrome
 5. hypercoagulability screen – Antiphospholipid, Lupus anticoagulant, ATIII deficiency, Protein C and S, Factor V Leyden, prothrombin 2010 gene mutation
 2. TTE/TEE (r/o proximal emboli)
 3. CTA/MRA (r/o proximal emboli)
 4. angiography
4. Diagnosis: age less than 45 years of age, smoker, distal ischemia findings, lack of hypercoagulable state, diabetes, autoimmune, or embolic sources, and angiogram showing collateralization. “Corkscrew collaterals.” No large vessel disease.
5. Natural History
 1. persistent ischemia from smoking --> amputation
 2. Raynauds, pedal claudication, tissue loss, and migratory thrombophlebitis
6. Treatment
 1. smoking cessation

2. iloprost (during early critical ischemia phase)
3. Ca channel blocker (nifedipine/amlodipine) if significant vasospasm present
4. cilostazol
5. sympathectomy does not appear to effect amputation rates, may occasionally help healing of superficial ulcers
6. reports of spinal cord stimulators and ulcer healing

Thoracic Outlet Syndrome:

1. History
 1. Arterial – arm pain, digital ulcerations
 2. Venous – strenuous arm activities
 3. Neurogenic – pain and difficulty with daily activities
2. Physical
 1. pulses, blood pressures
 2. Elevated Arm Stress Test: EAST. Arms up and rapidly clench fist for 30 seconds to reproduce the TOS symptoms.
 3. Adson maneuver- Arm out at a right angle to the body and turn your head to that side – the pulse goes away.
 4. Exam: spasm over the left scalene triangle
3. DDx: cervical spine arthritis, degenerative disk disease, spinal stenosis, post-traumatic c-spine straine, fibromyalgia, shoulder tendinitis, cubital tunnel entrapment of ulnar nerve, carpal tunnel (median nerve) entrapment, peripheral neuropathy.
4. Workup
 1. CXR, cervical XR
 2. upper extremity plethysmography and segmental pressures with positional maneuvers
 3. nerve conduction and electromyography
 4. angiography (venous, arterial)
5. Natural History
 1. progressively disabling
6. Treatment
 1. Neurogenic
 1. physical therapy
 2. TOS decompression. Although cure unlikely, 70% get improvement after decompression. Continue physical therapy after decompression.
 2. Arterial
 1. TOS decompression
 2. resection of subclavian aneurysm if present
 3. Subclavian aneurysm can be from TOS or can be a primary atherosclerotic finding
 4. The symptoms produced will be either brachial plexus compression or thromboembolism
 5. Although you can treat with interposition grafting after resection, aneurysmorrhaphy will decrease the risks of brachial plexus injury when compared with excision.
 6. As an aside – axillary artery aneurysms are associated with crutch use and same thing applies, in reference to resection vs aneurysmorrhaphy with interposition grafting
 3. Venous
 1. thrombolysis
 2. TOS decompression during same hospitalization
 3. possible angioplasty and stenting
 4. possible venous bypass with superficial femoral vein

Hypothenar Hammer Syndrome:

1. History – workers using their hand as hammers, numbness, paresthesias, stiffness, coldness, blanching of one or more digits of the dominant hand
2. Physical – prominent callus over hypothenar eminence, positive Allen's test, pulsatile hypothenar mass
3. Mechanisms – Hook of hamate bone strike the unprotected palmar superficial branch of the ulnar artery.
4. Workup
 1. ultrasound to scan for ulnar aneurysm
 2. angiography
5. Natural History
 1. thrombosis of the distal ulnar artery
6. Treatment
 1. supportive care
 2. catheter directed thrombolysis if symptoms within 2 weeks
 3. if aneurysm discovered, resection of aneurysm with end-end, or saphenous vein graft from the ankle. Divide the palmaris brevis on the hypothenar eminence to expose this.
 4. Ligation alone is ok as well

Vasculitis Issues

1. Digital ulcerations are most commonly caused by vasculitis, Buerger's disease, and atherosclerotic issues (aneurysms, emboli). More than 1/2 from vasculitis are caused by primary Sjogren's syndrome – calcinosis, raynaud's esophageal involvement, calcinosis, and sclerodactyly, telangiectasias (CREST)
2. Tissue loss rules out primary raynaud's disease.
3. Finger pressures should be greater than 80 mmHg, less than 40 mmHg is low.

Complex Regional Pain Syndrome:

1. Diagnostic Criteria
 1. CRPS I
 1. initiating noxious event or a cause of immobilization
 2. continuous pain disproportionate to an inciting event with allodynia or hyperalgesia
 3. edema change in skin blood flow, or sudomotor activity in the region of pain
 4. exclusion of other conditions that otherwise would account for the degree of pain
 2. CRPS II
 1. evidence of peripheral nerve injury as the initiating factor
 2. continuous pain that is disproportionate to an inciting event, w/allodynia or hyperalgesia
 3. exclusion of other conditions that otherwise would account for the degree of pain
2. Stages
 1. I – hyperalgesia, allodynia, signs of vasomotor dysfunction, and edema
 2. II – dystrophic stage, often 3-6 months after onset, increase pain and sensory dysfunction and development of motor or trophic changes or both
 3. III – atrophic stage, characterized by decreased pain and sensory disturbances
3. Treatment
 1. drug therapy
 1. phenytoin, amitriptyline, carbamazepine, baclofen

2. nifedipine
3. phenoxybenzamine
4. NSAID
5. opiate analgesics
6. corticosteroids
2. physical therapy
3. TENS therapy
4. intermittent sympathetic blocks
 1. Cervicothoracic block
 1. Stellate ganglion nerve block
 2. C6 level
 3. Horner's syndrome will develop, this is a sign you are in the right place
 2. Lumbar Sympathetic block
 1. Bottom of L2
 2. At L4 the genitofemoral nerve so don't do a chemical ablative block here
 3. Lumbar sympathetic ganglia
 1. Post-sympathetic neuralgia risks high
5. For ablation (chemical, alcohol) or surgical sympathectomy
 1. Cervical
 1. Remove all below T1 (so T2 and T3)
 2. Leave stellate ganglion intact (otherwise Horner's syndrome)
 3. Surgery: collapse lung – visualize 1st 4 ribs/vertebrae
 4. The subclavian is the superior extent of the dissection
 5. Nerve of Kuntz- directly from the sympathetic chain to the plexus?
 2. Thoracic
 1. Remove only L2-L3
 2. L1 removal will lead to retrograde ejaculation
 3. Retroperitoneal dissection, ID psoas muscle, sympathetic chain is medial to the psoas muscle along the transverse processes. You have seen them when doing spine exposures for neurosurgery.
 4. On the right, under the edge of the IVC, on the left lateral to the aorta.
6. sympathectomy (if in stage II, and still responsive to blocks)
 1. Transthoracic has replaced
 1. Transaxillary approach
 2. Paravertebral approach
 3. Supraclavicular approach
 1. Horner's syndrome (permanent)
 2. Brachial plexus injuries

Abdominal Aortic Aneurysm:

1. History – abdominal/back pain
2. Physical – pulsatile mass, femoral pulses
3. Risks. FEV1 < 1L, smoking, DBP > 110 mmHg.
4. Workup
 1. aortic US
 2. CT scan
5. Natural History
 1. UK Small Aneurysm Trial

1. no survival difference between early surgery (4-5.5) and delayed (>5.5) at 5 years
2. 60% of patients randomized to observation eventually received surgery at 2.9 years
2. Aneurysm Detection and Management study
 1. no survival difference between early surgery (4-5.4) and surveillance at 4.9 yrs
 2. >60% in surveillance arm underwent repair
 1. 4-4.4 = 27%
 2. 4.5-4.9 = 53%
 3. 5-5.4 = 81%
 3. 5%/year rupture risk for 5cm aneurysms.
 4. Fix if 5.5 cm or if more than 5 mm growth in 6 months. If symptomatic repair.
 5. 10%/yr 6-6.9%
 6. Rupture risks overall:
 1. 4-5 cm 0.5-5% yearly
 2. 5-6 cm 3-15% yearly
 3. 6-7 cm 10-20% yearly
 4. 8 cm 50% yearly
6. Treatment
 1. EVAR (good anatomy, 1.5cm proximal seal zone, 1cm distal seal zone, aneurysm <7cm)
 1. EVAR can't cannulate gate: longer sheath, different catheter, snare from opposite side vs arm, converter/aorto-unibody, open conversion.
 2. open repair
 1. IMA – when ligated, collaterals from SMA and superior hemorrhoidal arteries are usually sufficient. At the preoperative CT, look at the IMA. If it is patent and doesn't backbleed, you need to re-implant it. If it is a huge IMA and there is mesenteric disease and/or internal iliac disease, you can't do an EVAR.
 2. Clamp internal and external first, then proximal clamp. Let it backbleed after a brief release of proximal clamp.
 3. A pararenal AAA includes one of both the renals, but does not include the SMA or celiac. Thus, clamp below the SMA, but above the renals, or in between the renals.
 4. Carrel patch for horseshoe kidney with a bunch of accessory renals. Never transect a horseshoe kidney. RP exposure. If you run into it from the front, then tunnel it behind the horseshoe kidney.
 5. A mass at the aortic bifurcation is a pheochromocytoma or a horseshoe kidney
7. Assess for complications before closing
 1. Urine output: ureter injury, hypovolemic, foley kinked, Doppler renal arteries
 2. Femoral pulses (push down on femoral pulses prior to releasing clamps in open repair) and LE: embolic issues requiring thrombectomy
 3. Colon/sigmoid ischemia
 4. Paraplegia
 5. MI

EVAR with endoleak:

1. workup
 1. CT scan
 2. angiography
2. Classification & treatment
 1. Type I – proximal or distal sealing zone
 1. proximal or distal cuff

1. Ia - Proximal
2. Ib- Distal
3. Ic- Through Iliac occluder
2. Type II – lumbar/IMA/accessory renal backbleeding
 1. embolization
 2. observation
3. Type III – structural or between pieces
 1. new limb/graft
 1. IIIa – junctional separation of endograft
 2. IIIb- fractures or holes involving endograft
4. Type IV – endotension/porosity
 1. re-line with new graft

Ruptured AAA:

1. diagnosis
 1. abdominal/back pain
 2. hypotension
 3. abdominal distension
 4. US or CT scan (if stable)
2. Treatment – Either straight to open repair if history/exam suggest ruptured AAA or straight to EVAR if you have a CT scan that shows appropriate anatomy. The other option is to go to a hybrid room and do an angiogram. If the anatomy is appropriate, do an EVAR. If not, then get a 12F sheath and CODA balloon up as proximal control. Then proceed with open repair.
 1. emergent open surgery
 1. Prep and drape from nipples to toes prior to induction. Cell saver set up and 8 units PRBCs.
 2. supraceliac clamp
 3. move clamp to infrarenal if neck available, otherwise clamp graft after proximal anastomosis completed
 4. Keep IMA ok to reimplant if colon looks ischemic.
 5. Check the feet prior to closing the abdomen
 2. EVAR
 1. better if contained rupture
 2. reliant or CODA occlusion balloon
 3. deploy graft
 4. Get a CT scan 24 hours after EVAR repair of ruptured AAA.

If you have a rupture after an EVAR, then you still have two endovascular options. First is to get an occlusion balloon through the left brachial or left axillary approach. The other option is to do an aorto-uni-iliac. The open approach is a bit different, although you can also use an occlusion balloon. When the proximal and distal fixation sites are intact, just transect the stent graft and sew the conventional graft to the fixed portion of the stent graft.

Inflammatory AAA:

1. diagnosis
 1. thickened aortic wall (thickened anterior and lateral rind) on CT scan
 2. urinary tract symptoms secondary to ureteral compression.
 3. Inflammatory aneurysms (vs infected aneurysms which are saccular) are fusiform, risks of rupture same as standard AAA.

2. Treatment
 1. retroperitoneal approach open repair
 1. may have difficulty with retroperitoneal fibrosis
 2. consider preop ureteral stents – never perform ureterolysis.
 3. RP approach: start midway between pubis and umbilicus and follow the course of the 11th/12th rib interspace. You can go as high as you want to get higher up on the aorta, however. Divide the lumbar branch of the left renal vein. The downfall of this exposure is the right renal artery and the right iliac artery.
 2. EVAR
 3. The inflammation resolves after you treat the aneurysm. Both EVAR and open AAA are options.

Type B Dissection:

1. History – severe chest/back pain, hypertension
2. Tear: intima -> media. Stanford A vs B. Acute < 14 days, Chronic > 14 days.
3. For Type A dissections, operative repair restores 95% of malperfusion syndromes.
4. Physical – check pulses
5. Workup
 1. CT scan
 2. TEE is also reasonable
6. Treatment
 1. Medical
 1. monitoring
 1. central line
 2. arterial line (right radial)
 2. BP control
 1. esmolol
 2. nicardipine/nipride
 3. pain control
 2. Intervention – patients with persistent symptoms despite maximal medical treatment, or patients demonstrating end-organ failure
 1. stent-graft of proximal flap
 1. IVUS
 2. endovascular fenestration +/- stenting
 3. open proximal graft replacement with distal fenestration

Inflammatory aneurysm

1. Know what the CT scan looks like: thick inflammatory rind anteriorly. These are usually fusiform, whereas infected aneurysms are saccular. Rupture rate of IAAA is no greater than a standard AAA.
2. Anterior and lateral surface has dense fibrotic layer. Renal vein and duodenum is stuck to the aneurysm wall. Can produce a dense retroperitoneal fibrosis.
3. Retroperitoneal approach allows for supraceliac clamp and avoids dissection around duodenum and renal vein.
4. Place preoperative ureteral stents if hydronephrosis. Do not do ureterolysis, no matter how easy it appears.
5. Unclear if steroids helps or not. It might not.
6. You can also just do an EVAR if the anatomy is appropriate.
7. After you repair these aneurysms, the dense retroperitoneal fibrosis.

Infected AAA:

1. Primary AAA infection rare. 15% of biopsied thrombus will be positive for organism, but this doesn't mean it is a primary infection. Generally, an infected primary AAA will be saccular, not fusiform. Look for fluid and air. Look for lumbar vertebral erosions.
2. They are prone to disruption with operative manipulation so clamp supraceliac if possible
3. If it is a primary infection arterial aneurysm, look for endocarditis, or other bacterial source for seeding the aorta that leads to aneurysm formation. Post-trauma is another reason it can happen.
4. superficial femoral vein reconstruction (NAIS): Neoaortoiliac system.
 1. Need preop DVT scan.
 2. Incision lateral to sartorius
 3. Identify sartorius and retract medially
 4. Then identify superficial femoral artery
 5. Posteromedially is the SFV
 6. Continue down as far as possible
 7. Proximally divide the SFV flush with the profunda femoris vein
 8. Valvulotome
 9. Wait until you are ready to use the vein before excising from the bed
 10. If you need to, go down to the level of the popliteal.
 11. Options
 1. Plication of aorta with subsequent size mismatch improvement for SFV
 1. Can do this as an aortounifemoral bypass
 2. Can also do a single SFV to the aorta, then take another SFV off the primary limb as an end-to-side SFV to SFV anastomosis.
 2. Other option is to pre-construct and join the two SFV's and then sew that to the aorta
5. extra-anatomic bypass with triple ligation of stump
6. rifampin-soaked dacron (15 minutes in 1mg/ml rifampin) + 6 weeks antibiotics
7. 1200 mg in 250 mL is another option. For 15-30 minutes

AAA/IVC Fistula:

1. 50% mortality. Get 2D echo as soon as possible.
2. New onset CHF with CT scan showing contrast in IVC during arterial phase
3. Specific risks are air embolism, pulmonary embolism, secondary to debris in the aneurysm that goes through the fistula. After the aneurysm exclusion and opening, backbleeding from the fistula is significant. Sponge stick above and below the IVC hole. If not suspected, a huge amount of backbleeding should make you suspect an aortocaval fistula. After repair of the AVF, and AAA repair, low cardiac output should respond to fluids and is related to low filling pressures. You might also be able to treat with EVAR \pm stent in IVC.
4. abdominal bruit, CHF
5. AV fistula is a volume overload state. Compression increases afterload and this leads to a reflex bradycardia. This is the Branham-Nicolladani sign.
6. Open repair
 1. control IVC with sponge-stick external compression, repair fistula from inside aorta

Aortoenteric Fistula:

1. EGD, CT scan
 1. CT scan: loss of plan between the aorta and duodenum with air in the aortic wall or the retroperitoneum. Use aortic homograft or rifampin soaked Dacron if there is a decision to

not do an extranatomic bypass. If you do this an intraoperative gram stain shows gram negative or fungal, replace Dacron with SFV-NAIS procedure, or ligate and do extranatomic bypass.

2. EGD should be done with a pediatric colonoscope to the level of D3/D4.
2. Treatment strategies
 1. Stable: staged extra-anatomic bypass first, followed by excision and ligation
 2. Unstable: repair duodenum first, then ligate aorta, then extra-anatomic bypass
3. General principles
 1. Supraceliac clamp prior to mobilization of duodenum, followed by two layered closure
 2. Rifampin soaked Dacron vs NAIS vs Aortic cadaveric homograft vs extranatomic.
 3. Omentum closure between duodenum and graft. Other options are anterior spinal ligament or jejunal serosal patch
 4. 6 weeks of antibiotics

Limb Ischemia post AAA:

1. check pulses after closing
 1. Kinking of graft
 2. Technical defect/intimal flap etc
 3. HIT
 4. Embolization
 5. Poor anticoagulation
 1. If platelet count is normal, then HIT is not a diagnostic possibility
 2. ATIII, Pn C and S, Fibrinogen
 3. Given FFP 2 units or 1 unit every 6-8 hours.
 1. This is a shotgun method for a variety of plasma deficiencies while on heparin
 2. ATIII deficiency the classic one that you give FFP while on heparin
2. thrombectomy
3. angiography

Mesenteric Ischemia post AAA:

1. usually due to embolization
2. patient's with prior colon resection should have IMA reimpaneled
3. early flex sig/colonoscopy
4. bowel resection. check for graft exposure. omentum to cover retroperitoneum

TAAA:

1. Risks
 1. paraplegia – dependent on extent, clamp time, and adjuncts like spinal drainage
 1. Type 2 – 25%
 2. Type 4 – 2%
 3. The rest are about 10%
 2. renal failure – 10-15% risk for all
 3. mesenteric ischemia
 4. Growth rate is 2 mm/year
2. Types
 1. Extent I – L SC to above the renals
 2. Extent II – L SC to aortic bifurcation
 3. Extent III – 6th ICS to aortic bifurcation
 4. Extent IV – Supraceliac to aortic bifurcation

5. Extent V – 6th intercostal space to just above the renals
3. surgical approach
 1. Indications: 6 cm in normal patients, less if the patient has connective tissue disorders
 2. Preop spinal drain. Leave in for 72 hours. Clamp drain for 24 hours prior to removal. Reimplant T9-T12 if large vessels or poor backbleeding. MAP > 80 mmHg. CSF drain 10 cc/hr max, keep CSF pressure less than 12 mmHg, by setting transducer at the level of the external auditory canal (EAC).
 3. Clamp/sew versus left atrial (via inferior pulmonary vein) to femoral bypass
 4. 6th or 7th interspace thoracoabdominal incision with retroperitoneal exposure
 1. incision from tip of scapula to umbilicus
 2. count ribs after opening auscultory triangle
 3. notch rib posteriorly, divide medial cartilaginous attachment of rib, ligate IMA
 4. divide external oblique, internal oblique and rectus muscles
 5. partial radial division of diaphragm
 5. endoaneurysmorrhaphy technique
 1. patch inclusion of intercostals 8-12, although most are T9-T12
 2. branch bypass vs. patch inclusion of celiac/SMA/renals
 3. When you are done, make sure the left renal artery bypass isn't kinked.
 4. For connective tissue disorders, can't do a Carrel patch – you have to reimplant each visceral vessel separately.

Spinal Cord Aside:

1. Anterior Cord Syndrome (1 artery)
 1. aka anterior spinal artery syndrome; due to dislocated bone fragment, or by herniated disc.
 2. motor- paraplegia bilateral
 3. sensory- loss of pain and temperature sensation (loss of spinal thalamic tract) with intact proprioception (posterior column-intact)
2. Posterior Cord Syndrome (2 arteries)
 1. posterior columns (fasciculus gracilis and cuneatus)
 2. loss of proprioceptive and vibration sense below the level of injury but with retention of touch, pressure, temperature and pain sense.

Iliac artery aneurysm

3. Common iliac artery
 1. EVAR vs open repair
 2. If there is 1cm of normal common iliac proximally, possible coil embolization of hypogastric followed by stent graft
4. Internal iliac artery
 1. Do sac-o-gram
 2. Coil embolize feeding branches to the internal iliac artery
 3. Then covered stent across origin of the hypogastric

Infected femoral aneurysm:

1. true vs. pseudoaneurysm (infected vs bland, secondary to puncture).
2. Any systemic illness, fevers, blood cultures
3. AFB limb or other prosthetic limb in groin
4. CT scan to assess proximal extent of infection
 1. Incorporated graft vs non-incorporated graft
 2. Presence of air/fluid

5. angiography to determine runoff
6. Revascularization options
 1. autologous vein (superficial femoral vein) – Bilateral DVT scan, harvest BL SFV
 2. obturator
 3. ax-popliteal
7. Intraoperative issues
 1. Cultures – staph epidermidis is hard to detect so culture explanted graft using sonication and Trypicase soy agar method (as opposed to blood agar method)
 2. Graft Preservation
 1. Cannot have pseudoaneurysm
 2. Cannot be thrombosed
 3. Anastomoses are intact
 4. No systemic signs of sepsis
8. Notes: If you have a prominent femoral pulse with a known anastomosis, assume the patient has a pseudoaneurysm. Then confirm with ultrasound, and assume it is a graft infection. CT to delineate the extent of the infection. Ax profunda from lateral to sartorius approach to avoid contamination. IV antibiotics for 2 weeks, then 3 months PO. Most common (80%) bugs are S. aureus, S. epidermidis, and E.Coli.
9. All prosthetic infections are the same principle. Bypass through clean fields and clean/new incisions, then wall off the clean incisions with dressings and occlusive tape etc, then take out infected graft. Other options is to use vein in the infected field.

Popliteal Artery Aneurysm:

1. Uncomplicated
 1. US/CT scan for diagnosis.
 2. Check other popliteal artery and check for AAA
 3. angiography for runoff
 4. endoaneurysmorrhaphy
 1. exposure
 1. divide medial gastrocnemius tendon if necessary
 2. open aneurysm and ligate side-branches
 2. graft vs. vein
2. Thrombosed
 1. Catheter Directed Thrombolysis
 1. t-PA 5mg bolus, 0.1mg/kg/hr infusion x12 hours, recheck angiogram
 2. endoaneurysmorrhaphy
3. Complications occur in these patients
 1. 2 cm PAAA
 2. Presence of intraluminal thrombus
 3. Poor runoff

Aortoiliac Stenting:

1. 10 mmHg pressure differential is significant
2. 100 mcg nitroglycerin or 10 mg papaverine should cause a 10 mmHg change for 2 minutes if significant
3. Predictors of success
 1. Patent profunda femoris
 2. Identifiable inflow lesion
 3. Rest pain (no tissue loss)

4. balloon expandable
 1. Ipsilateral retrograde
 2. If close to CFA, up and over (not involving the CFA)
 3. Success is a residual stenosis $x < 10\%$, $x < 5$ mmHg pressure drop
 4. If within 1 cm of the aortic bifurcation, then perform kissing stents
5. results
 1. infrarenal aorta
 1. 70-80% 3 year patency
 2. common iliac artery
 1. 77% 4 year patency (claudication)
 2. 67% 4 year patency (limb threat)
 3. 55% 10 year primary patency
 4. 64% 5 year primary patency (TASC B/C lesions)

Acute Aortic Occlusion:

1. History
 1. acute lower extremity pain (usually bilateral)
2. Physical
 1. pulses
 2. capillary refill
 3. sensation, limb viability
 4. discussion regarding impotence preop for all elective cases
 1. hypogastric blood flow restriction: penile brachial index of 0.6
 2. autonomic dysfunction so bladder sphincter does not tighten during ejaculation
 3. Nervi erigentes
 1. Sympathetic T12-L4 –ejaculation
 2. Parasympathetic S2-S4- vasodilation
3. Etiology
 1. embolic
 1. cardiac
 1. a fib
 2. prior MI
 3. dilated cardiomyopathy
 2. Thrombotic occlusion
 3. aortic dissection
4. Workup
 1. hypercoagulable w/u
 2. angiography in OR
5. Treatment
 1. if embolization identified, can do retrograde embolectomy
 1. Need to perform bilateral common femoral cutdowns
 2. The iliac side without thrombus should be controlled by clamping the femoral or by an occlusion balloon retrograde up the iliac, particularly, if only one leg is ischemic.
 3. Otherwise, the aortic thrombus will embolize to the opposite side
 4. This is true especially when you have a saddle embolus – sudden onset of rest pain in both legs, left barcahial angiogram, embolectomy, possible fasciotomy
 2. if thrombosis, need aorta-bifemoral bypass
 1. When you do an AFB, you need to control and clamp both renal arteries so that you can flush the plug out while protecting the renal arteries from embolic issues.

2. The other thing is that clot may squeeze up into the renals after you perform your clamp
3. Other option might be lysis with mechanical thrombectomy
6. Bilateral lower extremity pain. DDx is spinal cord injury, dissection, and acute aortic occlusion. Cause is generally cardiac embolism or thrombosis of severe AOI (dehydration, low cardiac output, septic shock). Generally, this is limited to the infrarenal aortic segment. Treatment is (1) aortobifemoral bypass vs ax-bifemoral bypass ± fasciotomy

Graft Infection (Aortic):

1. Presentation – fever, nausea, pulsatile groin mass
2. Workup
 1. CBC
 2. CT scan
 3. WBC scan
 4. angiography
3. Treatment
 1. Staged extra-anatomic bypass and excision
 1. extra-anatomic bypass (b/l ax-fem, or ax-bifem) – bilateral ax-fems allows lower incision
 2. excision of graft with ligation of aortic stump
 1. Debride aorta back to healthy tissue, which might end up compromising renal artery origins. So you have some options.
 1. This might require hepatorenal bypass on right
 2. Splenorenal vs renorenal vs ileorenal on the left
 2. The aorta needs to be ligated using a horizontal mattress suture, followed by a running locking suture with omental coverage.
 2. NAIS
4. Basic approach to all graft infections:
 1. stable graft infection – determine extent and then revascularize in a clean plane. Then disconnect graft through a clean plane that has no infection and close that wound. Last place to go is the dirty area and then remove the graft from there.
 2. unstable graft infection – remove graft and control bleeding. Then revascularize. If immediate, use saphenous vein, cadaveric aorta, NAIS. You can also heparinize and delay revascularization for 24 hours. If patient has no rest pain, then you can leave alone, but that is unusual.

Graft infections: If the graft is surrounded by a virulent organisms, standard gram stain and culture methods will suffice. If the organisms is not of high virulence, then a mechanical tissue grinding or ultrasound disruption of the biofilm of the explanted graft must be performed. This is followed by placement of the graft trypticase soy broth medium. This is particularly true when discussing staph epidermidis. Wait at least 5-7 days for final results

Graft enteric fistula:

1. Workup: GI bleeding in setting of previous AAA repair
 1. CBC
 2. EGD is the initial test and must be to the 4th portion of the duodenum
 3. CT scan w/ IV contrast only if EGD non-diagnostic
 4. Culture
 1. If extremely mild symptoms, might try Indium 111 leukocyte scan

2. Culture will show mixed flora – Gram negatives
2. Treatment
 1. Unstable
 1. laparotomy, supraceliac crossclamp. close enteric fistula
 2. excise graft, ligate aorta, cover with omentum.
 1. Ligation of aorta with horizontal mattress, then running locked 3-0 prolene closure
 2. If need more purchase for closure of healthy aorta, hepatorenal and/or splenorenal bypass (or some other way to revascularize the left renal artery).
 3. close wound
 4. bilateral ax-fem/ax-bifem.
 1. Or ax-profunda, ax-SFA, or ax-popliteal.
 2. Get into some sort of distal space that is not involved.
 2. Stable
 1. bilateral ax-fem/ax-bifem
 2. laparotomy, excise graft, close bowel fistula, ligate aorta

Other options: NAIS, cadaveric homograft.

Claudication:

1. H&P – smoking, HTN, DM, cholesterol
2. DDx: pseudoclaudication secondary to spinal stenosis, venous claudication secondary to iliofemoral DVT, muscle spasms etc
3. segmental arterial pressures with waveforms
4. If iliac occlusion only, ABI will be normal. Consider exercise ABIs (1.5 mph for 5 minutes)
5. Medical treatment
 1. quit smoking
 2. aspirin
 3. statin
 4. graded exercise 1 hour daily to the point of maximal pain, not the initial pain.
 5. +/- cilostazol (100mg tid), but get echo first, as cilostazol is contraindicated in CHF.
 6. f/u 3 months
6. decision for surgery
 1. disabling claudication (affecting work, activities of daily living) despite maximal medical therapy for 3 months
7. Claudication in a young patient
 1. aortic coarctation
 2. persistent sciatic artery
 3. popliteal entrapment – aberrant course of popliteal artery in relation to gastrocnemius muscle. On exam plantar flexion causes gastrocnemius compression of the popliteal artery. On angiogram, medial deviation seen on angiogram, mid popliteal occlusion, and post-stenotic dilation seen on angiogram. Always operate via posterior approach. Natural history is fairly aggressive so always operate. Check both sides. Active plantar flexion, passive dorsiflexion.
 1. Type I – Pop is medial to medial head of gastrocnemius. Divide medial head of gastrocnemius but reconstruct it \pm bypass
 2. Type II – More direct than type I, but same treatment.
 3. Type III – Normal pop course, but accessory band from medial head of gastrocnemius that attaches to the femur. Tx: divide band.

4. Type IV- Artery is deep to popliteus muscle or deep to a fibrous band. Tx: Divide popliteus or the band.
5. Type V – Also involves the popliteal vein and tibial nerve.
4. popliteal adventitial cystic disease
 1. Mid 40's, Sudden onset of claudication
 2. Subadventitial enlargement with encroachment on the lumen
 3. Knee flexion leads to loss of pedal pulse
 4. Tx: resection of cyst alone vs interposition grafting
 5. Scimitar's signs. Tx – excision and bypass.
 6. No medial displacement, unlike popliteal entrapment
5. Fibromuscular dysplasia of EIA

Rest Pain:

1. H&P – smoking, HTN, DM, cholesterol
 1. Medications
 1. Aspirin
 2. Statin
 3. B-blocker
 2. Smoking cessation
2. segmental pressures: 20 mmHg difference between levels is significant
3. ABI < 0.7 claudication, ABI < 0.4 rest pain.
4. angiography
5. bypass vs. endovascular
6. Bypass options if conduit limited:
 1. Spliced vein from two legs
 2. Lesser saphenous
 3. Arm vein: take basilic and cephalic out in continuity. If you use a valvulotome for the basilic segment only then the basilic is used for the proximal anastomosis and the cephalic for the distal anastomosis

Persistent Sciatic Artery:

1. Findings
 1. Asymptomatic or claudication
 2. Thrombosis of aneurysm leading to embolic phenomenon
 3. Nerve compression by sciatic aneurysm
2. History – younger age claudication/limb ischemia
3. Physical – absent femoral pulse, stronger popliteal pulse, pulsatile buttock mass
4. Variants
 1. Complete (continuity with popliteal, variable presence of common femoral/SFA)
 2. Incomplete (variable continuity with popliteal, intact CFA/SFA)
5. Treatment – depends on symptoms and extent of aneurysmal changes and must be followed.
 1. aneurysm
 1. coil embolization, bypass
 1. Interposition grafting from normal internal iliacs, EIA, or CFA to popliteal
 2. Ligation or endoaneurysmorrhaphy
 1. Do not excise secondary to sciatic nerve damage.
 2. High incidence of foot drop
 2. stent graft

2. limb ischemia
 1. bypass with exclusion/coiling

Failing Bypass Graft:

1. Graft Surveillance
 1. 3, 6, 12, yearly – ABI's, graft Duplex
2. angiography
3. balloon angioplasty (cutting for vein graft, rotate balloon 90 degrees and re-angioplasty) vs. open vein patch angioplasty

Graft risks:

	<u>High PSV</u>	<u>Vr</u>	<u>Low velocity</u>	<u>Change in ABI</u>
Highest risk	PSV>300	Vr>3.5	x<45 cm/sec	x>0.15 drop
High risk	PSV>300	ratio>3.5	x>45	x<0.15 drop
Moderate	180-300 cm/sec	>2	x>45	x<0.15 drop
Low risk	x<180 cm/sec	<2	x>45	x<0.15 drop

Thrombosed graft:

1. heparinization
2. classification
 1. I – limb viable, ok to delay revascularization
 2. IIa – marginal limb threat
 3. IIb – immediate limb threat
 4. III – irreversible limb changes
3. treatment
 1. if class I or IIa, CDT
 1. wire crossing lesion
 2. 5mg tPA bolus, 0.5mg/hr in catheter, 0.5mg/hr in sheath, 300 units/hr heparin in sheath
 3. monitor fibrinogen (>250)
 4. Contraindications to anticoagulation
 1. Absolute – Active internal bleeding, recent cerebrovascular accident (within 2 months), intracranial pathology
 2. Relative Major- Recent (within 10 days) major surgery, obstretic delivery or organ biopsy. Active peptic ulcer or other GI pathology. Recent major trauma. Uncontrolled hypertension.
 3. Relative minor contraindications – minor surgery or trauma, recent cpr, high likelihood of left heart thrombus, bacterial endocarditis, hemostatic defects
 5. Thrombolysis vs Mechanical thrombectomy. Risks with continuous tPA: embolic issues may occur before improvement. Access site problems. Spontaneous head or RP bleed.
 6. STILE, TOPAS, Rochester.
 2. if class IIb or III, immediate revascularization
 3. balloon angioplasty or open revision
 4. Thrombosis while on heparin? Check heparin levels and if normal, suspect AT III deficiency and give 1-2 units FFP q6 hours or give AT III concentrates
 5. chronic postoperative warfarin otherwise
 6. HIT: diagnosis of HIT is made by serologic assay or functional assay for heparin-dependant antibodies (serotonin release assay). Platelet factor 4 test. Get a baseline PTT. The

functional assay (ELISA) is only in those with low probability. Argatroban 2mcg/kg/min adjusted to a PTT 1.5 – 3 times normal – metabolized by the liver. Essentially, the first test is a serologic test which looks at the presence of heparin antibody. The second test, or functional assays, uses radiolabeled serotonin c-14 to assess the tendency of platelets to clot. Wait until platelets > 150 K before starting warfarin.

Bleeding and Clotting

1. **Bleeding disorders**: PT assesses II, V, VII, IX, X extrinsic, PTT assesses all other except VII. TT measures conversion of fibrinogen to fibrin.
 - a. Von Willebrand's disease: vWF Type I is decreased amounts, Type II is qualitative problems, and Type III is absent. Avoid NSAIDs, treat with desmopressin (ddAVP 0.3 mcg/kg over 30 minutes) if mild or Type I. More severe bleeding should be treated with replacement therapy (vWF). Maintain factor VIII and vWF to 50% normal during bleeding episodes or major surgery. Other treatment includes cryoprecipitate and also commercial vWF/FVIII replacements.
 - b. Both Bernard Soulier and Glanzmann's thrombasthenia is treated with platelet transfusions
 - c. Hemophilia A (Factor 8) and Hemophilia B (Factor 9). These are treated with recombinant factor replacement. Less than 1% normal is major, 1-5% is moderate disease. 5-40% is mild disease. If they have antibodies to F8 or 9, then rVIIa might be given. Hemophilia A – replace to 100% of activity and B replace to 50%.
 - d. Platelet issues are related to primary hemostasis. Secondary issues are related to factor deficiencies. Platelet problems present as bleeding in the gums, heavy menstrual problem. Hemophilias on the other hand, present as bleeding into joints and muscles after minor injury
2. **Clotting disorders**: Divided into two separate categories. The first group relates to decreases in natural anticoagulants in the body (ATIII, Pn C, and Pn S). The second group results in a gain in procoagulant function.
 1. Factor V Leyden (also known as activated protein C resistance). Aggressive treatment with homozygotes vs heterozygotes.
 2. Prothrombin 20210 gene
 3. ATIII deficiency – thromboembolism associated with the standard risk factors. Treat with heparin, although higher doses may be required. Give FFP or AT III concentrates. Need to get AT III levels to 80% of normal.
 4. Protein C deficiency
 5. Protein S deficiency
 6. During high risk time periods, protect the patients with heparin. Otherwise, wait until thrombotic episode to begin longer duration anticoagulation, but it is unclear to me.
 7. High risk states: smoking, pregnancy, OCP, cancer.

AV fistula post cardiac cath:

1. Duplex
2. US guided compression of fistula tract
3. open repair vs. coil embolization vs. stent graft
 1. Successful repair requires all 4 limbs of the fistula are controlled

Acute Limb Ischemia:

1. DDx: atheroembolism vs cardiac source vs thrombosis.
2. Life over limb – don't revascularize if the arrhythmia is life threatening.
3. Get EKG, maybe Echo.
4. The general question is this thrombosis of chronic disease (stenotic SFA) or an acute embolic issue. If palpable contralateral pulse or atrial fibrillation, then embolectomy. If not, then CTA.
5. Heparinization
6. classification
 1. I – limb viable, ok to delay revascularization
 2. IIa – marginal limb threat – some neurologic issues
 3. IIb – immediate limb threat – motor changes
 4. III – irreversible limb changes – mottling dead foot
2. Class IIa can get CDT, Class IIb or III go to OR immediately
3. Thrombolysis can theoretically be performed up to 14 days from initial symptoms. Consider 2D echo prior to thrombolysis to r/o cardiac thrombus.
4. to OR, embolectomy +/- angiography

Diabetic Foot Ulcer:

1. admit, infection control
 1. broad-spectrum antibiotics
 2. operative debridement/drainage
 3. Peripheral neuropathy – wounds over the weight bearing pressure points
 1. Usually the plantar surface of the metatarsal heads
 2. The lateral surface of the foot
 3. If it goes down to the bone, it is a malperforans ulcer.
2. segmental pressures with toe pressures
3. angiography
4. endo vs. open revascularization

Paradoxical Embolus:

1. presentation – any arterial bed ischemia (renal, LE, carotid) in setting of DVT (leg swelling)
 1. stroke with unilateral leg swelling
 2. murmur
2. carotid duplex - embolus
3. venous duplex – DVT
4. 2D echo with bubble study – PFO
5. Treatment
 1. IVC filter
 2. carotid embolectomy
 3. closure of PFO

Lymphedema:

1. Diagnosis – non-pitting edema where swelling starts in dorsum of the foot. In venous disease, it starts at the ankle and is pitting

1. venous duplex (r/o CVI)
2. CT/MRI (r/o malignancy, MRI can differentiate between lipedema and lymphedema)
3. lymphoscintigraphy
2. Classification
 1. Primary
 1. Congenital – present at birth or by age 2
 2. Praecox – puberty to 3rd decade
 3. Tarda – after age 35
 4. Familial
 1. Milroy's disease – present at birth, soon after
 2. Meige's disease – manifest during puberty
 2. Secondary
 1. Surgery
 2. Radiation
2. Treatment
 1. physical therapy
 2. decongestive therapy

Chronic Venous Insufficiency:

1. Always check for DVT and then check for reflux
2. Sickle cell should always be in the differential diagnosis of perimalleolar ulcers.
3. Then think about uncommon things like Klippel-Trenaunay Syndrome
 1. Port wine stain, limb hypertrophy, venous malformations, and capillary dermal hemangioma
 2. Never do venous disease surgery in these patients without a venogram or MRV as they often have absent deep systems, etc
4. CEAP classification
 1. Chronic Venous Disease
 2. Clinical: C2 varicose, C4 skin changes
 1. Class 0: no visible or palpable signs
 2. Class 1: telangiectasias or reticular veins
 3. Class 2: varicose veins
 4. Class 3: edema
 5. Class 4: skin changes (pigmentation, venous eczema, lipodermatosclerosis)
 6. Class 5: skin changes with healed ulceration
 7. Class 6: skin changes with active ulcer
 3. Etiologic: congenital, primary, secondary
 4. Anatomic: superficial, deep, perforating
 5. Pathophysiologic: reflux, obstruction, or both
5. Baker's cyst can cause compression of the popliteal vein (by this synovial cyst)
6. Basic physiology
 1. The GSV connects to the femoral vein via the saphenofemoral junctions, which has five (or 6) tributaries. Greater saphenous vein has both an anterior and posterior accessory vein. It connects to the lesser saphenous vein via the vein of Giacomini. The GSV connect to the deep system via femoral canal perforators (above knee), paratibial perforators (below knee), and posterior tibial perforators (at the ankle)
 2. With calf muscle contraction, the venous pressure should drop significantly. If it doesn't then, there is likely some venous dysfunction. Ambulatory venous pressures used to be obtained with a needle in the dorsal vein of the foot. Now ambulatory venous pressures can be performed with air plethysmography. This uses changes in leg volume as a surrogate for

pressure. Put a sleeve around the calf. The baseline is when a patient has legs up and supine. Then have the patient stand up.

1. AVP<40 ...low risk of ulceration
2. AVP>80 ...80 % chance of ulceration
3. Next, have the patient tip toe to get calf contractions. The rate of refilling of the limb veins is the venous filling index or VFI (mL/sec). This is a ratio of :
 1. $0.9 \times$ volume increase from supine to standing divided by the amount of time needed to reach 90% of the venous volume.
 2. Normal: VFI: less than 1.7 mL/sec
 3. Superficial incompetence: 2-30 mL/sec
 4. Deep incompetence: 7-28 mL/sec
7. Valve closure times >0.5 seconds indicates incompetent valves. Just look at it with ultrasound. So 500 msec for superficial veins, 350 msec for perforating vein, and 1000msec for deep veins.
8. Reflux time greater than 4 seconds with flow reversal
9. Venous claudication: bursting pain secondary to iliofemoral thrombosis. No arterial component.
10. Varicose veins: CEAP 2-3
11. Stasis dermatitis and dermal fibrosis: CEAP 4-6
12. Diagnostic
 1. Venous duplex with maneuvers to assess for reflux – in standing position
 1. Deep veins
 2. Saphenofemoral junction evaluation
 3. GSV – including anterior and lateral thigh branches.
 4. SSV
 5. Perforators – High thigh, above knee, paratibial (below the knee) and posterior tibial (at ankle)
 6. Valve closure time should be less than $\frac{1}{2}$ second. When a valve is incompetent, then there is greater than 4 seconds of reflux following compression of the leg or Valsalva, with a long segment of flow reversal.
 7. Also look at augmentation from femoral to look for proximal obstruction/iliac venous stenosis
 2. Air Plethysmography – APG
 1. Normal ambulatory venous pressure is 80-90 mmHg when standing and 40 mmHg when ambulating. Recovery should be less than 20 seconds to normal
 2. Refill time less than 20 seconds from APG means insufficiency. Put a tourniquet on and then if refill time is still less than 20 seconds there is deep insufficiency as well. If you put the tourniquet on and the refill time normalizes (greater than 20 seconds to fill the venous compartment of the leg), then there is superficial reflux only.
 3. Venous filling index less than 2 mL/second
 4. EF >60
13. Treatment
 1. Compression stocking, conservative. Jobst stocking, profore, Unnaboot
 2. endovenous ablation with RFA – remove the refluxing superficial veins from the venous circulation
 3. stab avulsion phlebectomy
 4. Open stripping of saphenous above knee with ligation of the saphenofemoral junction and associated 5 branches.
14. Historical but testable
 1. Vein Surgery: (predominant obstructive component)
 1. Palma procedure: femoral cross-over venous bypass for iliac vein occlusion

1. Obstruction of iliofemoral venous system: Bypass by saphenous vein cross over graft. Isolate the normal contralateral saphenous vein and divide it distally. Vein is tunneled suprapubically and anastomosed to contralateral femoral vein, distal to its obstruction.
2. Few candidates as natural history of iliac vein occlusion is recanalization
3. Majority of iliofemoral thromboses occur on left because the right iliac artery compresses the left iliac vein as it crosses the fifth lumbar vertebra
- 4.
2. May-Husni procedure: saphenopopliteal venous bypass for SFV occlusion
3. Transposition of deep femoral or saphenous vein valve
4. Transplantation of valve bearing segment of axillary vein
5. Direct repair
 1. Valvuloplasty: (for primary valvular dysfunction)(after dvt-too scarred and thickened wall-doesn't lend to this type of repair)
 2. Highest valve in superficial femoral vein: longitudinal venotomy to expose the valve cusps: suture plication with interrupted 8-0 monofilament

Ileo-femoral DVT/Phlegmasia:

1. hypercoag w/u
2. IVC filter
3. mechanically assisted thrombolysis
 1. Possis angiojet +/- tPA
4. Traditional CDT
5. open thrombectomy
 1. AVF using branch of saphenous vein to SFA
 2. Fasciotomy
6. Anticoagulation w/coumadin for 6 months
7. May-Thurner: Venous angioplasty and stenting. Iliac venous stent: Wallstent oversized by 15-20%, 16-18mm Wallstent vs aortic cuff. Warfarin post op.
8. Venous stents: Wallstents: 6-24 mm stents (go up by 2 mm) and lengths are 20-60 mm. Access sheath size ranges from 6-10F. Can also use aortic cuffs. Oversize both by 15-20%. Generally stent after May-Thurner.

Post-phlebotic syndrome:

1. DUS with reflux examination
2. compression
3. treat saphenous vein reflux if present.

DVT in pregnancy:

1. heparinization. No warfarin in pregnancy!
2. suprarenal filter if thrombectomy needed

Acute mesenteric embolic disease:

1. Presentation – acute abdominal pain
2. workup – CBC, lytes, amylase, EKG
 1. AXR
 2. CT scan w/IV contrast vs. angiography
 1. For embolus, generally it is lodged just beyond the middle colic takeoff

2. For thrombosis, the origin is involved
3. Treatment
 1. SMA thrombosis – bypass, iliac/supraceliac aorta – SMA, retrograde stenting of SMA, Hepatic to SMA bypass if celiac is ok.
 2. SMA embolus – thrombectomy
 3. warm up bowel, assess viability
 4. 2nd look laparotomy

Nonocclusive Mesenteric Ischemia:

1. History: Hypotension, poor cardiac output
2. CBC, lytes, amylase LDH
3. AXR
4. +/- CT scan
5. angiogram
 1. narrowing of multiple branch origins
 2. “string of sausages” sign – alternate dilation and narrowing
 3. spasm of mesenteric arcades
 4. impaired filling of intramural branches
6. 30-60mg/hr intraarterial papaverine
7. Swann Ganz catheter- make sure adequate volume, broad spectrum antibiotics

Chronic Mesenteric Ischemia:

1. GI workup
 1. EGD
 2. colonoscopy
 3. US, including mesenteric duplex
 1. Fasting
 2. Celiac >70% stenosis. PSV >200 cm/sec, EDV>55 cm/sec with retrograde hepatic artery flow
 3. SMA>70% stenosis. PSV>300, EDV> 45 with loss of the diastolic flow reversal
 4. CT scan
2. Angiography
3. revascularization
 1. endovascular
 1. predilation PTA
 2. balloon expandable stenting, 2mm of stent extruding into aorta
 2. open
 1. retrograde SMA bypass from iliac
 2. antegrade SMA/cealic +SMA bypass from supraceliac aorta
 3. bifurcated graft with two limbs: one to SMA and one to celiac.
4. Compare with median arcuate ligament syndrome: celiac compression on expiration, secondary to compression. Do not angioplasty or stent, as you need to lyse the compressive band.

Mesenteric Vein Thrombosis:

1. CT scan if no peritonitis
 1. visualization of clot
 2. bowel wall thickening, ascites, stranding of mesentery
 3. portal venous air, pneumatosis
2. hypercoag w/u

1. Protein C and S deficiency
2. Antithrombin III deficiency
3. Factor V Leyden
4. Prothrombin 2010
5. Other hypercoagulable states
 1. Malignancy, myeloproliferative disorders
 2. Polycythemia vera, oral contraceptives
 3. Cirrhosis, dehydration
3. treatment
 1. if peritonitis
 1. laparotomy + anticoagulation
 2. 2nd look operation
 1. Clinical assessment
 2. Doppler examination
 3. 1 gram of sodium fluorescein over 30 seconds, followed by Woods lamp UV examinations. Areas that are nonfluorescent and are bigger than 5 mm are a problem.
 3. Heparinize, antibiotics, coumadin for life
 2. no peritonitis
 1. bowel rest
 2. anticoagulation

Splenic Artery Aneurysm:

1. Risks: medial fibroplasia, grand multiparity (4-6 pregnancies), portal hypertension with splenomegaly
2. Other risks: chronic pancreatitis with pseudocyst formation, vasculitis, polyarteritis nodosa
3. Dx: CT/ultrasound, selective splenic artery angiogram via celiac artery
4. >2cm, fertile female – treat
5. If pregnant, you must operate and fix
 1. 80% are located in the distal 1/3 of the splenic artery
 2. Do not miss this in a pregnant patient, as they will rupture and 100% fetal death
6. Double rupture phenomenon
7. Treatment
 1. endovascular stent graft ± coiling
 2. coiling alone, proximal and distal to aneurysm
 3. ligation +/- splenectomy.
 1. Get into the lesser sac, ligate aneurysm proximal and distal with all the feeding collaterals
 2. Distal pancreatectomy and splenectomy
8. Vaccines
 1. N. Meningitides
 2. H. Influenza
 3. Pneumococcus

Renovascular hypertension:

1. DDx of surgical hypertension: coarctation, Conn's syndrome, Cushing's syndrome, renal artery stenosis.
2. RAS: Flank bruit, hx of flash pulmonary edema
 1. Most common cause of death is cardiac

2. ASTRAL trial puts all interventions into question
3. History
 1. deterioration of renal function after ACE-inhibitor
 2. SBP > 200 mmHg and DBP > 100 mmHg
 3. HTN with unilateral RAS
 4. Renal insufficiency and RAS is bilateral
 1. Clinical improvement predictors for CRI
 1. Bilateral RAS or unilateral RAS with solitary kidney and kidney size > 8 cm
 2. Rapid deterioration (within 6 months) of kidney
 3. Severe hypertension
 4. Low parenchymal arteriolar resistance (RI < 0.8)
 1. RI >0.8 = intrinsic parenchymal disease
 2. RI <0.8 = normal kidney
 3. RI = (PSV – EDV) /PSV
4. Renal artery duplex
 1. PSV>180 - 200cm/s = >60% stenosis
 2. RAR >3.5 = >60% stenosis, but aorta cannot be aneurysmal and kidney should be > 8cm.
 3. Acceleration time greater than 100 msec is also indicative of main RAS disease
5. Renal vein renin assay
 1. ↓ blood flow to involved kidney stimulates release of Renin
 2. Renin increases Angiotensin II and Aldosterone, resulting in vasoconstriction and sodium and water retention
 3. Normal kidney responds to increased blood pressure with natriuresis
 4. Compensatory natriuresis limits negative feedback on Renin, perpetuating renin-induced vasoconstriction hypertension
 5. Simply stated: unilateral ischemic kidney will hypersecrete renin and the normal kidney will suppress renin (ratio of > 1.5 : 1)
 6. Bilateral lesions limits this evaluation
 7. Need to sample from both renal veins and IVC simultaneously
 8. Need sodium restriction and discontinuation of beta blockers for this study
6. if unilateral disease and with normal renal function– functional study
 1. captopril renogram
 1. baseline scan after hydration and discontinuation of ACE-I
 2. 25-50mg PO captopril or Enalaprilat 50mcg/kg
 3. repeat scan in 60 minutes
 4. normal kidney responds with increased perfusion
 5. affected kidney responds with decreased GFR
7. angiography
 1. AP for left renal artery
 2. 15-30° LAO for right renal artery
8. revascularization
 1. renal artery angioplasty and stenting
 1. proximal/mid lesions
 2. FMD
 2. Aortorenal bypass - 12.5gm mannitol given early in operation, repeat dose before and after clamping
 1. distal lesions
 2. branch point lesions
 3. ex vivo repair

1. cold perfusion preservation – KHPO₄, KCl, NaHCO₃, Dextrose solution
3. Hepatorenal bypass vs splenorenal bypass (right vs left medial visceral rotation for exposure)
 1. In adult patients dacron is fine
 2. For pediatric patients, aortorenal bypass should be done with hypogastric artery
 3. Aortorenal bypass
 1. Right side: hepatorenal to distal right renal
 1. Ileorenal, route lateral to cava
 2. Kocker maneuver, mobilize right renal vein and lateral cava
 2. Left side: aortorenal, from iliac or from supraceliac aorta. Splenorenal is hard to do.
 1. Left RA bypass:
 2. Retroperitoneal approach with kidney up makes left renal bypass straight forward
 3. Distal left renal a exposure via midline approach requires mobilization of left colon!
 4. Splenorenal is technically challenging
 3. Both: bifurcated graft off of supraceliac aorta
 4. Nephrectomy in nonfunctional kidney with no reconstruction options
9. In all patients with renal insufficiency, don't forget CO₂ angiogram and IVUS
10. Bottom Line
 1. Reasonable indications for Renal Artery Revascularization
 1. Anatomic findings in setting of clinical findings
 1. Documented RAS (>70% diameter reduction)
 2. Fibromuscular dysplasia lesion
 3. Gradient > 20 mm Hg
 4. Kidney size > 8 cms
 5. Affected/unaffected kidney renin ratio of > 1:5/1
 2. Clinical
 1. Refractory or rapidly progressive hypertension
 2. Hypertension associated with flash pulmonary edema without CAD
 3. Rapidly progressive deterioration in renal function in setting of bilateral disease and RI <0.8
 4. Intolerance to medications

Aorta-bifemoral bypass with renal artery stenosis:

1. If concomitant AAA and would otherwise meet criteria for renal artery revascularization, then aortorenal bypass with AAA repair. Transverse incision.
2. angiography
 1. orificial vs. mid artery lesions
 2. Renal US to determine kidney size (<8cm, no improvement in revascularization)
 3. simultaneous ABF with renal artery endarterectomy/bypass
 1. transperitoneal approach
 2. divide base of mesentery, ligament of treitz, right colon attachments
 3. eviscerate bowel to RUQ, divide crus

Renal Artery Aneurysm

1. Risk rupture (kidney loss) vs embolization (hypertension)

2. 2 cm or greater need repair
3. Premenopausal women repair
4. Pregnant women, most rupture in the 3rd trimester
5. If concomitant hypertension, get lateralizing renins
6. Treatment options
 1. Nephrectomy if extensive intraparenchymal disease
 2. Embolization if isolated intraparenchymal lesion
 3. Ligation with bypass
 4. Call transplant for ex vivo repair if needed

Hepatic Artery Aneurysms

1. Pain, jaundice, hemobilia
2. Hepatic artery can be ligated so long as it is proximal to the GDA – this applies to trauma as well.
3. Anatomy
 1. Common hepatic
 2. Proper hepatic •
 3. Right hepatic: ~16% replaced off SMA. Supplies middle lobe liver •
 4. Left hepatic: ~25% replaced off left gastric. Supplies lateral and medial segments, +/- medial lobe •
 5. Right gastric: lesser curve stomach
4. Treat all
 1. If proximal to GDA then ligate proximal and distal to the aneurysm
 2. If distal to GDA then resect with RSVG bypass
 3. If intrahepatic then coil embolization

SMA aneurysms

1. These are almost always infected
2. Treatment
 1. Resection of affected SMA area with RSVG
 2. Antibiotics for a few months
 3. Generally these are from a cardiac infectious source
3. The other cause is polyarteritis nodosa
 1. Regress with steroid, cyclophosphamide, methotrexate

Polyarteritis Nodosa

2. Hallmark of PAN formation of multiple aneurysms
3. Kidney, heart, liver, and GI tract most commonly affected (aneurysm)
4. Branches of SMA, CA, SMA distal to middle colic artery. Associated with inflammatory destruction of the media. Rupture of intraabdominal PAN aneurysms has been well described.
5. Regress with vigorous steroid and cyclophosphamide therapy
6. should be recommended for all asymptomatic visceral aneurysm
7. Diagnosis P-Anca

Vascular Trauma Points

1. Abdomen
 1. Supramesocolic – aortic compressor vs supraceliac clamp, left anterior thoracotomy
 1. Ligate celiac
 2. Retrograde bypass to SMA
 3. Nephrectomy for renal artery bleeding
 4. If renal artery thrombosis for blunt injury, then end-to-end after resection of injured segment
 2. Inframesocolic – same exposure as infrarenal AAA
 1. Ok to ligate infrarenal aorta
 2. Ok to ligate left renal vein
 3. Lateral venorrhaphy for right renal vein
 3. IVC and branches – Right colon mobilization and Kocher maneuver
 4. Retrohepatic Cava
 1. Divide triangular ligament and anterior/posterior coronary ligament
 2. Watch out for the hepatic veins
 3. Clamp portal triad, infrahepatic cava
 4. If you need to – sternotomy and atriocaval shunt
 5. Lateral Pelvic
 1. Right iliac vessels – mobilize cecum and right colon
 2. Left iliac vessels – mobilize sigmoid
 3. Iliac venous confluence – Transect right common iliac artery

Injury of the Abdominal Vessels: Classification of hemorrhage by zone to obtain vascular control

1. Zone I Begins at the aortic hiatus and ends at the sacral promontory: Is generally approached via a left sided medial visceral rotation. Can also be accessed by using an extended Kocher maneuver . Will often require supraceliac control of the aorta. Contains the suprarenal aorta, the celiac axis, the SMA
 2. Zone II The right and left paracolic gutters Depends on expanding perirenal hematoma, Suggested vessel control before entering the hematoma, Management of renal vascular injuries. Get around Gerota's fascia. Lift the kidney up. Get a pedicle clamp around the entire thing. Nephrectomy.
 3. Zone III Begins at the sacral promontory and encompasses the pelvis - Difficult to manage due to colonic/GU injuries
2. Lower Extremity
 1. Ok to use PTFE, otherwise in LE use contralateral saphenous
 2. LE vascular injury: explore the vein. Concomitant venous and arterial injury warrants early fasciotomy
 3. Proximal and distal control
 4. Blunt vascular injury with associated thrombosis.
 1. #3 Fogarty catheter
 2. Resection with interposition grafting
 3. Shunt
 5. Shunt for orthopedic trauma.
 1. External Pruitt-Inahara or External Sundt shunt
 2. Stick around for the orthopedic portion of the case
 6. Dislocation of knee: Reduced ABI < 0.9. Then angiogram, duplex, or serial exams.
 7. Think about fasciotomy in trauma. CPK, urine myoglobin, fluids, fluids

1. 6 hour ischemia time
2. Compartment pressure 25-30 mmHg, or within 30 mmHg of diastolic pressure.
3. Urinalysis with +blood on dipstick but no red cells
4. CPK, urine myoglobin
5. Fluids fluids fluids
6. Technique
 1. Lateral incision is anterior to the fibula and lateral to the anterior tibial crest. Then get through the fascia for the following compartments.
 1. Anterior compartment – deep peroneal nerve (foot drop) and sensation between 1st and 2nd toes of the foot.
 2. Lateral compartment – superficial peroneal nerve
 2. Medial Incision is 2 cm posterior to the posterior crest of the tibia
 1. Then vertical incision identifies the septum between the superficial and deep posterior compartments
 2. If you take the soleus attachments off the tibia to open the deep posterior compartment fully
3. Upper Extremity
 1. Forearm compartment syndrome
 1. Most common cause is elbow dislocation
 2. The three compartments of the forearm
 1. Dorsal (Back of forearm) – Straight incision
 2. Volar compartments – Median/ulnar nerves. Radial and ulnar artery. Lazy “S” incision.
 3. Mobile Wad – does not need a separate incision
4. Injection Drugs
 1. Femoral vein injection can lead to DVT
 2. Arterial
 1. Mycotic aneurysms/pseudoaneurysms
 1. Septic emboli can result
 2. Thrombosis
 3. Rupture
 4. Bacteriology – staphylococcus aureus
 5. If above femoral or brachial bifurcation
 1. Ok to ligate after excision and debridement
 2. Saphenous vein graft vs contralateral SFV, but only in severe ischemia
 2. Embolic particles
 1. In the upper extremity, can get claw like hand
 3. Vasospasm (methamphetamine, cocaine)
 1. papaverine
 2. nitroglycerin
 3. Tx heparin bolus then drip
5. Thoracic vascular trauma
 1. Injury to the aortic arch is most often from penetrating trauma
 2. In blunt trauma the innominate artery is most commonly affected
 3. Subclavian Injury
 1. The subclavian artery is divided into three anatomic portions

1. First Portion- Origin of the aorta to medial border of the anterior scalene
2. Second Portion- The medial border of the anterior scalene muscle to where the artery crosses the clavicle posteriorly
3. Third Portion - From the clavicle to the medial portion of the pectoralis minor muscle
2. Resect clavicle if you need to for subclavian
3. Concomitant infraclavicular and supraclavicular approach
4. Left subclavian: trap door for origin injury, but only if you've done a sternotomy first
5. Otherwise, left posterolateral thoracotomy through 5th interspace
4. Importance of neck zones
 1. Zone I - sternal notch to 1 cm above the clavicular head - Initial studies should be directed at the vascular and aero digestive system. Needs thoracotomy.
 2. Zone II - above the clavicular head to the angle of the mandible- debate remains. Selective exploration versus routine exploration
 3. Zone III - from the angle of the mandible to the base of the Skull- Difficult to manage. Arteriography essential

Takayasu's Arteritis

4. Diagnosis- Elevated ESR and angiogram showing smooth tapering stenosis. Young females. Decreased peripheral pulses. Clinical.
5. Beds which can become ischemic: cerebrovascular, renovascular, peripheral, aortic.
6. Three phases
 1. Prodromal
 1. Malaise, fever, lethargy
 2. Constitutional vague symptoms
 3. Myalgias, arthralgias, anorexia, weight loss
 2. Inflammatory
 3. Burned Out
 1. Symptoms develop due to stenosis, embolization, occlusion, or aneurysm.
 2. Mortality due to stroke (uncontrolled hypertension), CHF (secondary to hypertension), MI or renal failure (both from hypertension)
7. Alternate view: Two phases
 1. Prepulseless phase (early)
 2. Pulseless phase (late)
8. Classification system
 1. Type I- Arch and great vessels
 2. Type II- Middle aortic syndrome. Distal thoracic and visceral abdominal aorta
 3. Type III- Aortic arch and abdominal aorta
 4. Type IV – Pulmonary arteries
9. Treatment: Corticosteroids, cyclophosphamide, methotrexate
10. Elevated ESR is predictive of the response

11. Operated when ESR has normalized, arterial bypass only in patients with persistent symptoms despite steroid treatment
12. Brachiocephalic disease: do an ascending arch to innominate bypass. Operate from healthy area, to healthy area. You might need to continue perioperative steroids as well.

Dissection Issues

1. Methods of dealing with Type B dissections
 1. TEVAR (right sided arterial line)
 2. Fem-fem for LE disease
 3. Stent graft from true lumen into visceral vessel
 4. Open fenestration
 5. Endovascular fenestration- bilateral groin access vs right arm access. Wire goes up both. IVUS – wire in both true and false lumens. TIPs needle vs Luck to get across the true to the false lumen or vice-versa. Snare wire vs predilation with balloon. Then standard balloon across the bridging area with potential dragging of the balloon down.

AV fistulas

1. Order of operations, non-dominant arm first
2. Vein map, $x > 3$ mm ideal, check pulses to assess inflow
3. Radiocephalic AV graft at wrist, work your way up
4. Brachiocephalic
5. Radiobasilic transposition in forearm
6. Radiocephalic straight PTFE
7. Brachiocephalic loop graft
8. Brachio basilic graft (one or two stage)
9. Brachial to axillary (in armpit) graft
10. Ax – ax loop graft (Armpit basilic to armpit brachial)
11. For prosthetic grafts, use a 4 – 7 mm tapered graft
12. For steal, get finger pressures, with and without AVF compression
 1. Options for steal: using a brachiocephalic as an example
 1. RUDI – Revision using distal inflow
 1. Ligate venous side of anastomosis
 2. Then take a vein graft from some distal artery and plug it into the upper arm cephalic
 2. DRIL- Distal revascularization, interval ligation
 1. Ligate the brachial artery distal to the fistula.
 2. Then bypass from upper arm brachial to beyond ligated area with saphenous vein
 3. PAI- Proximalization of arterial inflow
 1. Ligate the venous side of the fistula anastomosis
 2. Then take a graft from the axillary artery or armpit brachial and bypass to the venous side of the fistula