

# Vascular Surgery Exam Prep

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# **Chapter 1**

## **About**

This content was developed by the Audible Bleeding Team to accompany our board review podcast series. Each chapter covers a key domain of vascular surgery, essential for board review preparation, and is associated with an Audible Bleeding episode which you can access through the link embeded into the text.

### **1.1 Usage**

This e-book is not intended to be a comprehensive board review guide, but instead serves as an easily accessible resource, paired with our podcast content to help streamline exam preparation.

Please consider this a “living document.” We plan to post regular updates and respond to your feedback. We are utilizing an open source annotations software called [hypothes.is](#) to allow you to take note along side our e-book. Feel free to post your notes publically if you think other readers would benefit from your additions, or create a small private group for your local trainees or faculty members. We will work to incorporate relevant notes and additions into future editions.

### **1.2 Comments, Questions or Contributions**

Please visit our [github](#) page or send us an email.

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# Chapter 2

## Cerebrovascular

**UNDER CONSTRUCTION - This Chapter is Currently in DRAFT mode**

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### 2.1 Available Guidelines

Society for Vascular Surgery clinical practice guidelines for management of extracranial cerebrovascular disease [AbuRahma et al., 2022]

### 2.2 Presentation and Diagnosis

#### 1. What is the definition of crescendo TIAs?

The presenting symptoms of extracranial carotid disease is of the utmost importance to understand. Focal neurologic symptoms that occur suddenly and then resolve within 24 hours are called a transient ischemic attach (TIA). If the deficit persists beyond 24 hours, then it is considered a stroke. Strokes are then graded according to the National Institute of Health Stroke Scale (NIHSS).[Fischer et al., 2010]

A particularly confusing distinction is between a crescendo TIA and a stroke in evolution. A crescendo TIA is frequent repetitive neurological attacks without complete resolution of the deficit between the episodes, usually over a 24 hour period. The result of which is the same neurological deficit. If the patient exhibits progressive deterioration in neurological function between TIA episodes this is classified as a stroke in evolution not a crescendo TIA.[Ricotta] These are both particularly concerning findings and may be an indication for an emergent intervention.

## 2. Who needs to be screened for carotid disease?

Not everyone should be screened for carotid disease, however, as only 15% of patients who have a stroke experience a warning TIA prior to their event, there is merit in screening high risk patients.[Rockman, 2019] The purpose of carotid bifurcation imaging is to detect “stroke-prone” carotid bifurcation plaque and identify a high-risk patient likely to benefit from therapy designed to reduce stroke risk.

Traditionally, the clinical sign associated with asymptomatic carotid stenosis is the presence of a neck bruit on auscultation. Focal ipsilateral carotid bruits in symptomatic patients has a sensitivity of 63% and a specificity of 61% for high-grade carotid stenosis (range, 70%-99%). As these figures suggest the absence of carotid bruit does not absolutely exclude the presence of a significant carotid bifurcation lesion, therefore additional criteria are used to determine which patients would benefit from screening:

- Evidence of clinically significant peripheral vascular disease regardless of age
- Patients aged >65 years with a history of one or more of the following atherosclerotic risk factors:
  - CAD
  - Smoking
  - Hypercholesterolemia

In general, the more risk factors present, the higher the yield of screening for the patient. Presence of a carotid bruit, AAA or family history of disease alone is not sufficient to warrant imaging.[AbuRahma and Perler, 2022]

## 3. Ultrasound Imaging

- US is considered the best, least invasive, and cost effective method for detecting carotid stenosis on screening.[Shaalan et al., 2008] For more see 12.2.
- 50-69% stenosis of ICA - US has a low sensitivity for 50-69% stenosis. A negative ultrasound in symptomatic patients necessitates additional imaging. Typically on scanning if 50-69% ICA stenosis is present:
  - PSV 125-229 cm/sec
  - EDV 40-100
  - Internal/Common Carotid PSV Ratio 2-4
- 70-99% stenosis of ICA- Typically on scanning:
  - PSV >/= 230 cm/sec

- EDV >100 (EDV > 140 cm/sec most sensitive for stenosis >80%)
- Internal/Common Carotid PSV Ratio > 4
- Velocity-based estimation of carotid artery stenosis may need to be adjusted in certain circumstances:
  - Higher velocities in women than in men.
  - Higher velocities in the presence of contralateral carotid artery occlusion.
- High carotid bifurcation, severe arterial tortuosity, extensive vascular calcification, and obesity may also reduce the accuracy of US imaging.

#### 4. Other Imaging Modalities

- CTA
  - Pro - fast, sub-millimeter spatial resolution, visualize surrounding structures.
  - Con - cost, contrast exposure.
- MRA
  - Pro - no contrast administered; can analyze plaque morphology.
  - Con - Does not visualize calcium in plaque; overestimates the degree of stenosis (False positive for 50-69% to be read as >70%).
- Catheter-based digital subtraction imaging (DSA)
  - Still considered by many the gold-standard imaging modality.
  - Reserved for individuals with conflicting less-invasive imaging or those considered for CAS.
  - Con - cost, contrast, and risk of stroke.

## 2.3 Management

### 2.3.1 Medical Management - Asymptomatic Patients

First line management for asymptomatic patients with carotid stenosis is medical optimization of risk factors. The benefit of prophylactic intervention (CEA/CAS) of even high grade stenosis is estimated at a 1-2% stroke reduction risk per year. [Naylor, 2015] In addition, keep in mind that intervention (CEA/CAS) has only demonstrated a benefit in asymptomatic patient with life expectancy greater than 3 years. [Bulbulia and Halliday, 2017, Halliday et al., 2010, Rosenfield et al., 2016]

#### Hypertension

- Blood pressure reduction, with a target of <140/90 mmHg through lifestyle interventions and anti-hypertensive treatment is recommended in individuals who have hypertension with asymptomatic carotid atherosclerosis or those with TIA or stroke after the hyper-acute period.
- Each 10-mm Hg reduction in blood pressure among hypertensive patients decreases the risk of stroke by 33% in patients aged 60-79.[Aiyagari and Gorelick, 2009]

### **Diabetes**

- In diabetic patients glycaemic control to nearly normoglycemic levels (target hemoglobin A1C <7%) is recommended to reduce microvascular complications and, with a smaller evidence base, macrovascular complications other than stroke.[Ricotta]

### **Hypercholesterolaemia**

- Statin agents are recommended targeting LDL of 100 mg/dL, for those with coronary heart disease or symptomatic atherosclerotic disease, and LDL of 70 mg/dL for very high-risk persons with multiple risk factors.
- High dose statin therapy in patients with TIA/stroke reduce future rates of stroke or cardiovascular events but not overall mortality at 5 years. [Karam et al., 2008]

**Smoking Cessation** - Encouraging smoking cessation is paramount for these patients. Smoking and carotid disease have been found to have a dose dependent relationship based on pack-years. Risk of stroke has been shown to reduce to baseline within 2-4 years of smoking cessation.[Hicks et al., 2015, Kawachi et al., 1993, Petrik et al., 1995, Wilson et al., 1997]

**Antiplatelet therapy** - Single agent antiplatelet therapy, most often aspirin, is often all that is needed, but this should be weighted with other cardiovascular risk factors. A number of trials have showed limited benefit of adding an additional antiplatelet or routinely testing for clopidogrel resistance.[Ricotta]

### **2.3.2 Medical Management - Symptomatic Patients**

Patients who present with a TIA should be placed on dual antiplatelet therapy, often aspirin and clopidogrel, which are continued through the workup and treatment of carotid disease. Patients often also have their blood pressure medications reduced to allow permissive hypertension and maximize perfusion to at risk cerebral tissue.

While we are not often directly involved in the management of acute stroke, vascular surgeons should be aware of best practices in this area. Management of acute stroke currently follow these timelines[Powers et al., 2018]

- < 4.5 hours from onset of symptoms - medical thrombolysis with tPA unless contraindicated.

- Patient age >80 and diabetes are contraindications to tPA administration after 3hrs.
- Other contraindications include - hypertension, intracranial hemorrhage, recent stroke or cranial trauma, spine/brain surgery within 3 months, GI bleed within 21 days.
- <6 hours from onset of symptoms - catheter directed therapy
- After 6 hours, there is limited benefit to thrombolytic therapy.

### 2.3.3 Surgery - Carotid Endarterectomy

#### Indications for Surgical Intervention

- Recommendations on when to operate after a TIA/stroke.
  - Acute stroke with a fixed neurologic deficit of >6h duration - When the patient is medically stable, intervention in 14 days or less after stroke is preferable. [Rothwell et al., 2004, Meershoek and de Borst, 2018]
  - Consider urgent intervention in a medically stable patient with mild-moderate neurologic deficit, if there is a significant area of ischemic penumbra at risk for progression on imaging that is contributing to the patient's presentation.
  - Stroke in evolution (fluctuating / evolving neurological deficit) or crescendo TIA (repetitive transient ischemia with improvement between events) - If the patient's neurological status is not stabilized by medical intervention then an urgent CEA should be considered.
  - In this clinical context CEA is preferred to CAS based on an increased embolic potential of carotid lesions. [Rantner et al., 2017]
- What is the only emergent indication for CEA?
  - Crescendo TIAs or a stroke in evolution with a surgically correctable lesion identified on imaging.

#### CEA Intraoperative Techniques

- General concepts
  - Patch angioplasty or eversion endarterectomy are recommended over primary arterial closure due to decreased ipsilateral stroke, perioperative carotid occlusion, return to the operating room, restenosis and 1-year stroke rates.[Goodney et al., 2010, Rerkasem and Rothwell, 2009] Patch repair is considered the standard of care surgical management for most extracranial carotid lesions.[Arnold and Perler, 2019]

- Neuromonitoring/Shunting options during a carotid endarterectomy[Chongruksut et al., 2014, Wiske et al., 2018]
  - Local/regional anesthesia with direct neurological monitoring - the benefit being that the patient is awake and moving to command throughout the case. Indications for shunting include lateralizing deficits, seizure, lack of consciousness and severe anxiety. Improved neuromonitoring, however, has not been shown to reduce myocardial infarction rate with CEA.
  - Stump pressure- Clamp the inflow and place a butterfly attached to a line tubing into the internal carotid artery. If the stump pressure is > 50 mmHg the surgeon can chose to proceed without shunt placement, if < 50mmHg then a shunt should be placed prior to proceeding.
  - EEG Neuromonitoring - EEG tech places neuromonitoring, monitored by the technician and neurologist remotely intraoperatively. Generally the surgeon should clamp the ICA for 3 minutes before proceeding, if the patient develops neurological deficit/EEG abnormalities with slowing and decreased alpha and beta waves, then unclamp, await normalization of EEG, then proceed.
  - Cerebral Oximetry - 15% decrease in either hemisphere should be shunted.
  - Transcranial Doppler (TCD) - severe slowing in the middle cerebral artery. An RCT demonstrated that TCD and cerebral oximetry changes most accurately predicted cerebral ischemia. Detection of a greater than 50% drop in middle cerebral artery velocity using transcranial Doppler is 100% sensitive for detecting cerebral ischemia. [Moritz et al., 2007a] For more see 12.3.
  - Non-selective shunting - the surgeon uses a shunt for all CEAs as standard practice, this is often done for all CEAs performed under general anesthesia
- Techniques to reach high internal carotid lesions?[Beretta et al., 2006]
  - A good knowledge of the available imaging while preparing for the procedure is essential when considering the level of the carotid bifurcation.
  - Nasotracheal intubation will allow for jaw closure and neck extension to facilitate exposure of higher lesions.
  - Division of the posterior belly of the digastric muscle, stylopharyngeal muscle, styloglossus muscle, stylomandibular ligament or the styloid process. Care should be taken to identify and preserve the glossopharyngeal nerve.
  - Division of the occipital artery.

- Dissection along the posterior parotid gland.
  - ENT surgeon assisted mandible subluxation with assistance if previous techniques fail.
- What is the best technique for a patient with an anatomically kinked internal carotid artery?
  - Eversion carotid endarterectomy (only indication for selection for this technique over patch endarterectomy), transection allows for reduction of redundancy at re-anastomosis.
  - Otherwise, no advantage has been shown between eversion or patch, both can be shunted.
- Nerve Injuries – where you would encounter these and, if injured, what deficit would be seen?
  - Hypoglossal: Most commonly injured. Usually passes above the bifurcation of the carotid artery. If injured the tongue will deviate toward the side of injury on protrusion.
  - Glossopharyngeal: Commonly injured during high dissections. Found under the posterior digastric muscle. Injury can be devastating for patients resulting in difficulty swallowing and high aspiration risk.
  - Vagus: Usually found adjacent and lateral to the common carotid. Injury can occur with carotid clamping. Patients develop hoarseness due to innervation of the laryngeal muscles from the recurrent laryngeal nerve which is a branch of the vagus.
  - Marginal Mandibular: Branch of the facial nerve. Runs inferiorly to the angle of the mandible. Commonly a retraction injury during high dissections leading to the corner of the ipsilateral lip drooping, can be confused with a significant neurological deficit following the case.
- Completion duplex for CEA
  - Can identify technical issues with carotid endarterectomy such as clamp site injury, thrombosis, intimal flap or dissection, or missed disease.[Lipski et al., 1996]
    - \* Acute thrombus may be hypoechoic and difficult to see on B-mode, color flow necessary to determine luminal narrowing or occlusion. For more see 12.2.
  - Revision required if residual plaque identified with severe velocity elevation.[Weinstein et al., 2015]
    - \* Elevated velocities can be seen for many reasons that do not require revision, including incorrect technique (compression with

probe), tortuosity, size discrepancy, ICA spasm.[Ricco et al., 2013, Weinstein et al., 2015]

- One study demonstrated that 2.3% of carotids required revision for concerning findings on completion duplex ultrasound.[Ascher et al., 2004]

### **Postoperative Complications**

- What to do if the patient develops neuro deficits following carotid endarterectomy: Two main concerns- an intimal flap causing thrombosis or an embolic event resulting in a stroke. Perform a thorough neurological exam to confirm in operated carotid territory.
  - If in OR – perform duplex, very low threshold to re-explore and confirm patency of ICA with duplex or doppler. If ICA open and concerned for distal embolization, then perform a cerebral angiogram and proceed with thrombolysis or thrombectomy.[Fletcher et al., 2016]
  - If in Recovery or on the floor – many would consider CTA first line vs duplex to look for thrombosis.
- Risk factors and how to manage hyperperfusion syndrome?
  - Defined as an ipsilateral headache, hypertension, neurological deficits likely in distribution of the carotid which can progress to seizures and intracranial hemorrhage (75-100% mortality). Can present 2-3 days post surgery.[Kaku et al., 2004]
  - Patients with uncontrolled hypertension are at risk for hyperperfusion syndrome, clinical practice guidelines from SVS recommend strict BP control following CEA, with target pressure being less than 140/80mmHg or 20mmHg from preoperative baseline.[Abou-Chebl et al., 2004]
- High risk groups
  - ESRD patients have higher rates of perioperative stroke, but also have higher rates of stroke if not revascularized. [Klarin et al., 2016]

### **Long term complications and follow up**

- Recommend f/u US duplex of bilateral carotids at </=30 days. >/= 50% stenosis further imaging is indicated.
- Ipsilateral stenosis
  - 0-3mo likely due to inadequate endarterectomy or clamp injury.[Kang et al., 2014]
  - 1-3y likely neointimal hyperplasia, smooth and regular. If <80% stenosis and asymptomatic, then duplex surveillance and antiplatelet.

If progresses, then often endo treatment first.[Garzon-Muvdi et al., 2016]

- Contralateral stenosis:
  - The risk of progression for moderate stenosis at the initial surveillance to severe stenosis in the contralateral artery can be as high as 5 times.
  - Requires post-operative surveillance.
- Carotid artery patch infections
  - Rare complication (<1%) presenting as phlegmon, pseudoaneurysm, sinus tract or carotid cutaneous fistula.[Stone et al., 2011]
  - Treatment is excision and replacement with autologous tissue.[Fatima et al., 2019]

#### 2.3.4 Endovascular - Carotid Artery Stenting

- In patients aged >70 the risk of intra- or post procedure stroke was the highest, if undergoing CAS, presumably due to calcified disease in the arch.
  - Lesion-specific characteristics are thought to increase the risk of cerebral vascular events after CAS and include a “soft” lipid-rich plaque identified on noninvasive imaging, extensive (15 mm or more) disease, a pre-occlusive lesion, and circumferential heavy calcification.
  - This risk can be reduced, but not eliminated, by using flow-reversal embolic protection rather than distal filter protection.
- Limited data is available on CAS in asymptomatic patients - currently is not supported by guidelines or considered reimbursable.
- Consider CAS in symptomatic patients with >50% stenosis who are poor candidates for CEA due to severe uncorrectable medical comorbidities and/or anatomic considerations:
  - Ipsilateral neck dissection or XRT - equivalent periprocedural stroke rate to CEA, but increased later stroke rate. CEA higher rates of cranial nerve damage (9%). [Giannopoulos et al., 2018]
  - Contralateral vocal cord paralysis.
  - Lesions that extend proximally to the clavicle or distal to C2.
- Transfemoral Approach vs Transcarotid approach
  - ROADSTER Trial - single arm study with flow reversal for cerebral protection. Suggested lower rates of post-op stroke in patients undergoing TCAR.

- TCAR contraindications include previous CCA intervention, CCA disease at entry site, <5cm CCA (clavicle to bifurcation) for sheath, CCA < 6mm, contralateral recurrent laryngeal or vagus nerve injury.[Kwolek et al., 2015]
- Patient needs to be preloaded with dual antiplatelet medications for at least 5-7d, should delay procedure if has not been appropriately loaded with antiplatelets.[Brott et al., 2010, Kwolek et al., 2015, Valls et al., 2017]
- Studies with TCD have shown that there may be reduced embolization rates with TCAR over transfemoral stenting.[Plessers et al., 2016]
- Complications
  - Post stenting ICA thrombosis should first be treated with abciximab and catheter directed thrombolysis, followed by endo-salvage with suction thrombectomy, repeat angioplasty or stenting. May require eventual conversion to CEA.[Coelho et al., 2019, Naylor et al., 2018]
- Post-CAS follow up - Dual-platelet therapy should be continued for 1 month after the procedure, and aspirin should be continued indefinitely.
  - In stent restenosis (>50%) - 4-times higher rate of stroke than no recurrence of disease, but more benign than de novo disease.[Clavel et al., 2019] Stenosis should be confirmed with CTA. No clear difference between treatments in literature - PBA, Cutting, DBA, stenting, open conversion.[Arhuidese et al., 2017] Repeat angioplasty or stent have low incidence of periprocedural stroke but failed to improve long term stroke/death/MI or patency rates.[Chung et al., 2016]

### 2.3.5 Summary of Prospective Trials

1. Asymptomatic Carotid Atherosclerosis Study (ACAS)
  - Compared medical management with CEA in asymptomatic patients with > 60% stenosis.
  - 5-year stroke and death rate was 5.1% vs 11%.
  - In women, the benefit of CEA was not as certain as 5y stroke and death rates were 7.3% vs. 8.7%.
  - This trial was conducted pre-statin and clopidogrel era.
2. North American Symptomatic Carotid Endarterectomy Trial (NASCET) [North American Symptomatic Carotid Endarterectomy Trial Collaborators, 1991]
  - Compared medical management vs CEA for symptomatic patients with moderate (50-69%) and severe stenosis (>70%)

- Only moderate impact for patients with moderate stenosis (50-69%)
  - Symptomatic patients with >70 % stenosis benefited from CEA, at 18 months 7% major stroke in surgical arm, and a 24% stroke rate in medical arm. 29% reduction in 5-year risk of stroke or death.
    - Patients with severe >70% stenosis had such a dramatic effect the trial was stopped early for this subset and all referred for endarterectomy.
  - No benefit is shown in symptomatic patients with < 50% stenosis
  - European studies have shown similar results
    - ACST = ACAS
    - ECST = NASCET.
  - **Take a listen!** Click hear to listen to Dr. William Jordan discuss this landmark paper!
3. Carotid Revascularization Endarterectomy versus Stenting Trial (CREST).[Silver et al., 2011]
- Compared CEA vs. CAS in both symptomatic and asymptomatic patients.
  - Composite endpoint of 30-day stroke, MI, death equivalent between CEA and CAS.
  - CAS had a significantly higher incidence of stroke and death than CEA and CEA higher incidence of MI.
    - Follow up at 10 years demonstrated no difference in composite stroke/MI/death but increased rate of stroke/death in stented patients likely attributable to increased periprocedural stroke. [Brott et al., 2016]
    - CAS patients with stroke had a higher impact on QOL than CEA patients with MI.[Brott et al., 2010, Redfern et al., 2011, Mantese et al., 2010]
  - Subanalyses identified that older patients (>70y) had better outcomes after CEA than CAS, the QOL impact of stroke was more significant than that of MI, and anatomic characteristics of carotid lesions (longer, sequential, remote) were predictive of increased stroke and death after CAS.
  - Unfortunately, this study provides a benchmark to strive for, but no other large trials have achieved these results.
4. ROADSTER[Kwolek et al., 2015]
- Single arm feasibility trial of transcarotid carotid stenting.

- The results of the ROADSTER trial demonstrate that the use of the ENROUTE Transcarotid NPS is safe and effective at preventing stroke during CAS. The overall stroke rate of 1.4% is the lowest reported to date for any prospective, multicenter clinical trial of CAS (i.e. CREST). No direct comparison.
- 5. Trials to look out for in the next few years:
  - CREST-2 - a multicenter, randomized controlled trial underway evaluating revascularization against modern intensive medical management.
  - ACT-1 and ACST-2- the role of intervention in asymptomatic patients, designed to compare the early and long-term results of CEA vs CAS and best medical management.
  - ROADSTER-2 - TCAR.

## 2.4 Uncommon Carotid Disease

### 2.4.1 Complicated Extracranial Occlusive Disease

- Occluded Carotid: What is the management of an occluded carotid?
  - Leave it alone.
- What if an occluded carotid on imaging is still causing TIAs?
  - Ongoing symptoms in the setting of carotid occlusion is known as carotid artery stump syndrome. Symptoms are likely due to emboli from friable intima of the internal carotid or from the external carotid through collaterals. Treatment is external carotid endarterectomy and ligation of the internal carotid.[Hrbáč et al., 2012]
  - The addition of oral anticoagulation is likely to reduce the rate of recurrent stroke.
- Simultaneous coronary and carotid disease
  - Patients with symptomatic carotid stenosis will benefit from CEA before or concomitant with CABG. The timing of the intervention depends on the clinical presentation and institutional experience (GRADE 1, Level of Evidence B).
  - Patients with severe bilateral asymptomatic carotid stenosis, including stenosis and contralateral occlusion, should be considered for CEA before or concomitant with CABG (GRADE 2, Level of Evidence B).
  - Patients undergoing simultaneous CEA/CABG demonstrate highest mortality. [Naylor et al., 2003]

- What about tandem lesions in the carotid in a symptomatic patient, carotid bulb and carotid siphon lesion (high ICA)? How should you treat this?
  - Treat carotid bulb and extracranial disease first, likely to be the embolic source.
  - Treatment of intracranial disease has been shown to increase rates of stroke and death.[Chimowitz et al., 2011]. Extracranial to intracranial bypass has increased risk of stroke over BMT.[Group, 1985]
- What if the patient has severe vertebrobasilar insufficiency and carotid artery disease?
  - Should undergo carotid revascularization first to improve flow. Then reassess for vertebrobasilar insufficiency.
  - Vertebrobasilar insufficiency characterized by dizziness, ataxia, nausea, vertigo and bilateral weakness. [Lima Neto et al., 2017b, Nouh et al., 2014]

#### 2.4.2 Vertebrobasilar Disease

- How do you treat isolated vertebral disease?
  - Most common etiology is atherosclerosis, but can also be caused by embolism, dissection or migraines.[Lima Neto et al., 2017a]
  - Symptomatic primary vertebral disease with >60% stenosis should be treated. Vertebral divided into segments. V1 preforaminal (lower than C6), V2 foraminal (C6-C2), V3 (C2-dura), V4 (intracranial).[Morasch, 2019, Berguer et al., 1998]
  - Disease of V1 should be treated with vertebral transposition of bypass with vein.[Rangel-Castilla et al., 2015]
    - \* Most common complication is Horner's Syndrome due to disruption of the sympathetic chain (20%), next is TIA (1-3%).[Coleman et al., 2013, Ramirez et al., 2012]
  - Disease of V2 is most difficult to access and should be treated with bypass from ICA to V3.[Berguer, 1985]
  - RCT of BMT vs endo treatment did not show superiority of endo treatment, recurrent symptoms most common, periprocedural stroke was rare.[Antoniou et al., 2012, Coward et al., 2007]

#### 2.4.3 Carotid artery dissection

- Patients with carotid dissection should be initially treated with antithrombotic therapy (antiplatelet agents or anticoagulation) (GRADE 1, Level of Evidence C).
- Indications for endovascular treatment of carotid artery dissection [Cohen et al., 2012, Markus et al., 2019, Pham et al., 2011]
  - Ongoing symptoms on best medical therapy

- Contraindication to antithrombotics
- Pseudoaneurysm

#### 2.4.4 Internal Carotid Artery Aneurysms

- Defined as dilation of the bulb 200% of the internal carotid artery or 150% of the common carotid artery. Like popliteal aneurysms the major risk is thromboembolization.[Bush et al., 2019, Fankhauser et al., 2015, Pourier et al., 2018]
- Treatment is resection with primary repair or interposition. Distorted anatomy increased the risk for cranial nerve injury (12%).[Welleweerd et al., 2015]
- Redundant ICA - should be repaired if patient symptomatic due to kinking. Treated with resection and primary repair with shortening and straightening.[Ballotta et al., 2005]
- Fibromuscular Dysplasia (FMD)[Olin and Sealove, 2011] - for more, see 7.4
  - Asymptomatic disease can often be treated with antiplatelets alone.
  - Duplex ultrasound demonstrates classic “chain of lakes” appearance. For more see 12.2
  - Associated with intracranial aneurysms (13%), therefore may be reasonable to also pursue brain imaging if identified. However, no formal guidelines exist.[Lather et al., 2017]

#### 2.4.5 Carotid Body Tumors (CBT)

Shamblin criteria utilized to determine difficulty of resection and is associated with rates of carotid reconstruction or cranial nerve injury.

Blood supply primarily from the external carotid artery.[Robertson et al., 2019, Davila et al., 2016] Embolization helps reduce risk of perioperative blood loss not associated with reduced cranial nerve injury. Resection 24-36hrs after embolization.[Power et al., 2012]

One quarter (25%) are associated with a germline mutation or familial history. Genetic testing recommended in patients with multifocal tumors, associated paraganglioma, pheochromocytoma or family history. SDHD gene mutation is most common associated with familial paraganglioma.[Davila et al., 2016, Kruger et al., 2010]

Evaluating for carotid body ligation - When involved with a CBT or other skull base tumors, cerebral perfusion should be thoroughly evaluated prior to surgical resection to determine whether a patient will tolerate carotid ligation. Evaluation includes balloon occlusion testing with xenon-enhanced SPECT imaging.

Low risk for ligation if no neurologic deficits (after 30min and hypotensive challenge) and normal diffusion of SPECT (ratio of >0.9). Angiography with contralateral occlusion evaluates collaterals.[Sugawara et al., Tansavatdi et al., 2015]



# Chapter 3

## Upper Extremity and Thoracic Outlet

**UNDER CONSTRUCTION - This Chapter is Currently in DRAFT mode**

**21 Jan 2021:** *Kush Sharma, MD and Ashraf Mansour, MD*

### 3.1 Overview of Upper Extremity Disease

#### 3.1.1 Anatomy

**What are the zones of the upper extremity?**[Illig, 2019]

Division of the upper extremity into three zones:

1. Intrathoracic Zone: Inclusive of the aortic arch and its branches, innominate artery (dividing into right subclavian and right common carotid), left subclavian artery and left common carotid artery, as well as the innominate veins, and SVC.
  - Arch classification helps determine difficulty of accessing the right innominate from a femoral approach. Type 1 innominate does not break the plane of the outer curve, Type 2 it is between the outer curve and inner curve and Type 3 it falls below the inner curve of the aortic arch.
2. Thoracic Outlet: Extends from the base of neck to the axilla. Includes the subclavian, proximal vertebral, and proximal axillary arteries, as well as the corresponding veins).
3. Axilla to Fingers: The arm.

**What are some common exposures for major upper extremity arteries?**[Illig, 2019, Gary G Wind and R. James Valentine, 2013]

1. **Proximal Right Subclavian Artery:** Access through a median sternotomy.
2. **Proximal Left Subclavian Artery:** An anterolateral thoracotomy is required in emergent settings for proximal left subclavian artery control. For proximal, mid and distal exposure is required a “trap door” exposure is used: anterolateral thoracotomy, third space sternotomy, and left supraclavicular incision.
3. **Mid and Distal Subclavian Artery:** Access through a supraclavicular Incision. After division of the platysma and clavicular head of the SCM, a fat pad of varying thickness is encountered which contains the omohyoid muscle. This should be divided and placed supero-laterally. At this point, the anterior scalene muscle is exposed medially with the phrenic nerve running in a lateral to medial direction, which should be identified and preserved. In a carotid/subclavian bypass division of the anterior scalene should be performed as close to the first rib as possible. Once divided the subclavian artery is exposed.
4. **Axillary Artery:** Exposure is gained through an infraclavicular incision, below the middle third of the clavicle. Pectoralis major is divided and pectoralis minor is freed at the lateral margin of the wound. The axillary vein should be identified and followed supero-deeply to identify and expose the artery. Anatomically bound by the first rib proximally and the lateral edge of the teres major muscle distally. For exposure of the first part of the axillary artery, the ipsilateral arm is abducted approximately 90 degrees and a horizontal skin incision 2 cm below the middle third of the clavicle should be made. The underlying pectoralis major is split by bluntly separating the fibers, exposing the tough clavipectoral fascia. At the lateral wound margin, the pectoralis minor can be freed and laterally retracted. The axillary vein is the first structure to be encountered in the fascia and the artery lies just superior and deep to the vein. Make sure to protect the nerves of the brachial plexus which lie deep to the first part of axillary artery and are at risk of injury during blind placement of occluding arterial clamps.
5. **Brachial Artery, Upper Arm:** The brachial artery's superficial location makes it vulnerable to injury and accounts for most vascular injuries of the upper extremities. Brachial artery exposure involves a 5-8 cm longitudinal incision in the groove between the biceps/triceps muscles on the medial aspect of the arm. In the lower half of the arm, take care to avoid basilic vein damage as it runs in the subcutaneous tissue. The neurovascular bundle is exposed by incising the deep fascia at the medial border of the biceps muscle, which then should be retracted anteriorly. The basilic vein should be identified and retracted into the posterior wound. On open-

ing the brachial sheath the median nerve is the most superficial structure and should be identified, protected and retracted. The brachial artery lies deep to the median nerve and is flanked by two brachial veins. Be mindful that the ulnar nerve lies posteriorly.

6. **Brachial Artery, Antecubital Fossa:** Exposure of the brachial artery in the antecubital fossa requires a transverse skin incision 1 cm distal to the midpoint of the antecubital crease. After deepening, mobilize the basilic vein medially to avoid injury. The medial antebrachial cutaneous nerve should be identified and protected. The bicipital aponeurosis should be divided to exposure the brachial artery, which is flanked by two deep veins crossing tributaries. To isolate the brachial artery ligation and division of these tributaries is required. The brachial artery bifurcates at the radial tuberosity into its radial and ulnar branches. Immediately after the ulnar artery arises from the bifurcation origin, it gives off a short common interosseous branch, which bifurcates at the hiatus in the proximal interosseous membrane.
7. **Radial Artery at the Wrist:** This requires a 2-3 cm longitudinal incision generally between the palpable radial artery and cephalic vein. Incision of the antebrachial fascia medially to the radius should expose the radial artery. Two veins accompany the artery and should be dissected away to isolate the artery. The superficial radial nerve and its medial/lateral branches course between the cephalic vein and radial artery in this area.
8. **Ulnar Artery:** The ulnar artery courses beneath the superficial flexor muscles of the proximal forearm, emerging near the ulnar border at the midpoint between the elbow and the wrist. In the distal forearm, the ulnar artery runs just deep to the antebrachial fascia and is easily exposed through a longitudinal incision placed radially to the flexor carpi ulnaris. The palmar branch of the ulnar nerve courses superficially to the antebrachial fascia and should be preserved during arterial exposure to avoid injury.

#### **What common aberrant upper extremity/arch anatomy is important to be aware of?**

Thoracic aortic disease is associated with aberrant anatomy, with high rates of bovine arch (25%), isolated L vertebral (6-8%), and aberrant R subclavian (1-2%).[Dumfarth et al., 2015]

1. **Bovine Arch:** Where the innominate and left common carotid arteries share a common origin.[Layton et al., 2006]
2. **Aberrant Vertebral Artery:** Where the vertebral artery arises directly from the aortic arch.
3. **Aberrant Right Subclavian Artery:** Where there is no innominate artery and instead the right common carotid arises directly from the aorta. Confusingly the right subclavian artery, then originates from the aortic

arch distal to the left subclavian artery. To supply the right arm it therefore, needs to pass behind the esophagus. (Dysphagia Lusoria)

### 3.1.2 Epidemiology, Etiology, and Diagnostic Evaluation

**How does evaluation of upper extremity ischemia differentiate from lower extremity ischemia?**

- Upper extremity ischemia <5% of patients with limb ischemia and in contrast to lower extremity, atherosclerosis is not a major contributor to upper extremity ischemia[Shuja]
- Vast majority of cases caused by autoimmune/connective tissue disorders

**How can upper extremity disease be classified?**

Anatomic Location:

- Large vs. Small Vessel

Disease Process:

- Vaso-occlusive disease often requires endovascular/surgical management.
- Vasospastic disease is more responsive to pharmacological management.

### 3.1.3 Evaluation

**How should patients be evaluated who have concern for upper extremity disease?**

1. Detailed H+P evaluation (pulse palpation, auscultation at supraclavicular/infraclavicular fossa may reveal a bruit concerning for subclavian artery stenosis, upper extremity neurovascular/skin exam)
2. Brachial/forearm blood pressures and if suspected claudication, measured at rest and 2-5 minutes after exercise. Look for a gradient of >20 mmHg is considered significant
3. Some or all of 6 P's of acute limb ischemia with symptoms occurring within 14 days are deemed acute
4. Doppler insonation of radial, ulnar, palmar, and digital arteries
5. Vascular Lab Evaluation
  1. Segmental Pressure Measurements
  2. Duplex Ultrasound (look for large vessel occlusive disease)
6. Other Imaging
  1. CTA/MRA
7. Clinical Lab tests

1. Inflammatory disorders-CBC, ESR, ANA, RF
2. Hypercoagulable screening

## 3.2 Vaso-occlusive Disease

### 3.2.1 Acute Limb Ischemia

**What is the procedure of choice in acute limb ischemia of the upper extremity?**

With acute presentation of upper limb ischemia and a localizing examination, may be reasonable to proceed directly to OR for embolectomy to minimize ischemia time.[Wahlberg et al., 2006, Henke, 2009]

**What are some postoperative complications of an embolectomy for acute limb ischemia?**

Brachial sheath hematoma - parasthesias and weakness in the median nerve distribution require emergent re-exploration and decompression. Even small hematomas that are not readily identifiable on clinical exam, can cause compression on the median nerve and are a surgical emergency.[Tran and Andersen, 2011]

Compartment Syndrome - If prolonged ischemia or no hematoma found, and forearm compartments appear tense, then should proceed with fasciotomies to treat forearm compartment syndrome. Rarely performed prophylactically due to significant morbidity.[Gelberman et al., 1981, Kistler et al., 2018, Leversedge et al., 2011]

- Three compartments - forearm volar (flexor, superficial, and deep), mobile wad (lateral), and dorsal (extensor, superficial, and deep). Volar compartments most susceptible to ischemia and compartment syndrome, most vulnerable muscle are flexor digitorum profundus and flexor pollicis longus.[Ronel et al., 2004]
- Volar/Henry approach decompresses lateral and volar compartments with a single incision, includes carpal tunnel release.

### 3.2.2 Chronic Limb Threatening Ischemia

#### 3.2.2.1 Demographics and Presentation - by anatomic level

**What are causes and symptoms associated with subclavian/axillary occlusive disease? [Jack L Cronenwett et al., 2020]**

- Etiology: Atherosclerosis is the most common cause of subclavian/axillary occlusive disease. Left SCA > Right involvement. Less common causes include Takayasu disease, giant cell arteritis, or arterial TOS

- Symptoms: Upper extremity arm/hand ischemia or neurologic symptoms due to subclavian-vertebral steal. Because significant collaterals, minimal pain on exertion even with subclavian occlusion
- Innominate disease can present with complex steal syndrome from the vertebral artery.[Rodriguez, 2016]

**What are causes and symptoms associated with brachial/forearm occlusive disease?**

- Etiology: MCC of brachial artery occlusion is cardiac origin embolus. Atherosclerosis RARELY affects the brachial artery. Distal axillary/proximal brachial stenosis can be from repetitive trauma from crutch use.
  - Radiation arteritis - may be seen with radiation after breast cancer therapy. Lesions characteristically tapered with smooth border. May occur alongside a brachial plexopathy.[Goldstein et al., 2010, Modrall and Sadjadi, 2003]
- Forearm occlusive disease can be seen in advanced ESRD/DM where calcific atherosclerosis of radial/ulnar arteries is present. Less common causes include Buerger's disease or Raynaud Phenomenon

### 3.2.2.2 Management

**How/when is upper extremity occlusive disease treated?**

Indications for treatment

- 70% stenosis and associated symptoms or deficit
- Lesion with ipsilateral ICA requiring treatment
- Pre-occlusive lesion in a good surgical candidate (>5y life expectancy)

Innominate or SCA Occlusive Disease

- Endovascular with balloon expandable stent via femoral or ipsilateral brachial artery. [Chatterjee et al., 2013, Bradaric et al., 2015, Saha et al., 2017, Palchik et al., 2008, Mordasini et al., 2011] Preferred in:
  - Short segment or ostial disease with adequate distance to the vertebral artery origin.
  - History of neck surgery or radiation.
- Surgery:
  - Bypass from aortic arch through median sternotomy - Most durable option, particularly for dense R innominate disease, so preferred in patients who can tolerate. Endarterectomy not appropriate if disease involves the origin.[Aiello and Morrissey, 2011, Mansukhani et al., 2018,

Berguer et al., 1998, Daniel et al., 2014, Reul et al., 1991, Berguer et al., 1999, Byrne et al., 2007]

- \* Complications include compression of the graft on sternal closure, so proximal anastomosis should be placed to the side of the aorta.
- Ipsilateral CCA to subclavian artery (bypass or transposition) - Recent NSQIP study showed no difference in stroke and death for CS bypass vs transposition, therefore procedure should be determined by anatomic constraints.[Cinà et al., 2002, van der Vliet et al., 1995] Small studies and systematic reviews have shown that patency of carotid subclavian bypass is better with synthetic graft than with autologous vein.[AbuRahma et al., 2000, Illuminati et al., 2018, Ziomek et al., 1986]
  - \* An additional indication for carotid subclavian bypass is as a staged revascularization prior to TEVAR for aneurysmal disease requiring coverage of the LSA
  - \* **How does the exposure differentiate in transposition vs bypass?**
    - Arterial transposition via a short, transverse cervical incision above the clavicle between two heads of SCM (bypass is lateral to entire SCM)
    - Sub-platysmal flaps created and avoid EJ vein damage
    - Omohypoid divided between heads of SCM and IJ mobilized laterally (bypass IJ is mobilized medially to expose CCA and care must be taken to avoid phrenic nerve in more lateral approach)
    - CCA is reflected medially with vagus nerve
    - On the left side, the thoracic duct is identifiable and divided followed by dividing the vertebral vein
    - Subclavian artery and proximal branches identified (anterior scalene is in lateral dissection)
  - \* **What are some common complications after carotid subclavian bypass in order of highest to lowest incidence?**
    1. Phrenic nerve palsy (most common) - most often managed conservatively.
    2. Recurrent laryngeal palsy
    3. Lymphatic leak
    4. Neck hematoma[Voigt et al., 2019]

\* When carotid-subclavian bypass compared to transposition?

1. Vertebral artery takes origin from the subclavian artery in a very proximal position or is dominant over the contralateral side, then bypass preferred. [Morasch, 2009b]
2. For coronary-subclavian steal with patent internal mammary artery to coronary artery bypass graft, then Bypass (a carotid-subclavian transposition requires a more proximal clamp with occlusion of inline antegrade flow to the coronary bypass during the procedure) [Cua et al., 2017]
  - Contralateral CCA (anterior or retropharyngeal)

Brachial/forearm Occlusive disease

- Endovascular: PTA evidence is anecdotal with stents for lesions unresponsive to PTA or dissection following angioplasty.[Cheun et al., 2019, Nasser et al., 2014, Dineen et al., 2007]
- Surgery: GSV vein bypass remains standard for revascularization with bypasses to superficial or deep palmar arch have good patency rates. Tuneling is subcutaneous if to distal ulnar or superficial palmar arch whereas anatomical to distal radial artery over the anatomic snuffbox.[Chang et al., 2003, Masden et al., 2012, Spinelli et al., 2010]

### 3.3 Vasospastic Disorders

#### 3.3.1 Iatrogenic or Vasopressor Induced Ischemia

You are called to the intensive care unit for a septic patient with acute bilateral upper limb ischemia, what is the management strategy?

Critically ill patients on vasopressors with bilateral upper extremity ischemia are often managed best with supportive measures and attempts to wean vasopressors (especially norepinephrine).[Landry et al., 2018]

Limited role for radial artery embolectomy, only in clear ischemia isolated to the distribution of an occluded radial artery, often secondary to line placement.[Valentine et al., 2005]

#### 3.3.2 Raynaud's Phenomenon

What is Raynaud's and what causes it? [Shuja, Landry, 2019]

Exaggeration of normal physiologic response with episodic pallor or cyanosis of the fingers caused by small digital artery vasoconstriction occurring in response to cold or emotional stress. There is an abnormality with sympathetic nervous

system, resulting in a multifactorial problem involving a combination of vascular, neural, and humoral factors.

**What are the subtypes of Raynaud's phenomenon and what is the underlying pathology?**

1. Primary: Raynaud's disease-idiopathic form that is a benign process not associated with structural vascular change. Triggers include (cold, emotional stress, caffeine) resulting in digital smooth muscle contraction and temporary digital hypoperfusion.
2. Secondary: Fixed vascular obstruction to blood flow decreasing threshold for cold induced vasospasm or progress to tissue loss. Diseases associated include mixed connective tissue disease, SLE, and rheumatoid arthritis, and scleroderma (accounts for 80-90% of cases). In setting of lower digital blood pressure, symptomatic digital ischemia or tissue loss under low stress conditions. With cold/emotional stress, vasoconstrictive response of digital artery smooth muscle further causes arterial closure and resultant symptoms

**What are diagnostic criteria for Raynaud's?**

- Clinical (Progression of ischemia with white -> blue -> red finger discoloration. Episodes can be self-limited and may last from less than a minute, but generally not longer than 10-20 minutes)
- Qualitative testing for severity of cold sensitivity in Raynaud's syndrome can be useful. Most basic test is cold sensitivity and recovery after ice water immersion. >10 minutes return to baseline pressure concerning for Raynaud's
- Segmental pressures with finger systolic blood pressure can differentiate purely vasospastic vs occlusive disease. Difference of more than 15 mm Hg between fingers or absolute finger pressure <70 mm Hg may indicate occlusive disease
- Serologic evaluation (ANA/RF)

**What are appropriate treatments for Raynaud's phenomenon?**

1. Medical-cold/tobacco avoidance lead to improvement in around half of patients. Calcium channel blocker (nifedipine ER 30mg qday) and losartan (50mg BID) have been shown to be most effective.[Landry, 2013, Wigley and Flavahan, 2016] Other drugs include alpha blocker, sildenafil, fluoxetine (SSRI), reserpine, cilostazol, captopril.
  - OUTCOMES ARE POOR IN PATIENTS WITH ARTERIAL OBSTRUCTION. If there is an asymmetric vascular examination, then further non-invasive vascular imaging is needed.
2. Surgical-thoracic sympathectomy (used for treatment of digital artery vasospasm/digital ischemic ulceration). For vasospasm, thoracic sympathect-

tomy is initially successful, but symptoms return generally within 3-6 months.

3. Immunosuppression/immunomodulation for connective tissue disorders associated with secondary Raynaud phenomenon

See Landry's Review in JVS for a good treatment algorithm.[Landry, 2013]

### 3.3.3 Ergotism

**What is Ergotism?** [Stanley et al., 2014]

- Etiology: Ergot is a parasitic fungal disease that has a particular prevalence for infecting rye plants and ergot alkaloids have been linked to epidemic poisonings that manifested as ergotism from consumption of rye
- Modern day is rare

**What causes Ergotism and how do patients present?**

- Ergotamine is chemically like endogenous catecholamines/indolamines and when applied clinically, it behaves as an agonist to alpha-adrenergic, serotonergic, and dopaminergic receptors. Despite limited bioavailability, vasoconstrictive effects have been reported to last for 24 hours or longer
- Gangrenous-mild limb pain followed by burning pain/shooting and
- Convulsive-heaviness in limbs and head associated with diarrhea. Could result in tonic-clonic spasms

**How can you diagnose Ergotism and what is the process for treating this disease?**

Upper extremity ischemia (i.e. digital ulceration) in the setting of ergot alkaloid use (typically for migraines). The treatment includes:

- Volume expansion and IV heparin as anticoagulation
- IV infusion of nitroprusside, nitroglycerin, iloprost or combination
- Infusion of Ca 2+ channel blockers
- Surgical: for thrombosis, consider thrombolysis

### 3.3.4 Buerger's Disease

**How is Buerger's disease categorized?**

Non-atherosclerotic, segmental, inflammatory disease of small/medium sized arteries in distal extremities of tobacco users distinct from either atherosclerosis or immune arteritis[Jack L Cronenwett et al., 2020, Le Joncour et al., 2018]

**What clinical criteria can help diagnose Buerger's?**

Smoking history, onset before 50 years, infrapopliteal arterial occlusions, upper limb involvement, absence of atherosclerotic risk factors besides smoking

**What is important about diagnosing Buerger's**

- Typically a diagnosis of exclusion
- Must rule out proximal embolic source, trauma, local lesions (e.g. pop entrapment or cystic adventitial disease), autoimmune disease, hypercoagulable status, atherosclerosis

**What physical exam and non-invasive/invasive imaging findings of Buerger's?**

- Distal, but not proximal arterial disease (palpable brachial/popliteal but absent/reduced at ankle or wrist)
- DBI<0.6 and flat/reduced digital waveforms
- CTA/MRA/DSA or duplex - characteristic findings of serpiginous/corkscrew collaterals, occlusion of distal calf/pedal arteries, and normal proximal arteries.[Busch, 2011, Fujii et al., 2011]

**What is the mainstay treatment in Buerger's disease?**

1. Smoking cessation! Only treatment to improve symptoms and reduce amputation risk if achieved before onset of gangrene or tissue loss. Important to remember following treatments will likely fail without smoking cessation.
2. If smoking cessation does not improve, medical management with antiplatelet agents, immunomodulators, vasodilators, anticoagulants
3. Endovascular-distal small vessel intervention
4. Surgical-upper extremity autogenous vein bypass-limited success due to poor outflow
5. Sometimes can consider upper extremity sympathectomy, but unproven benefit
6. Amputation-reported in 30-40% who are followed longer than 5 years[Olin, 2018]

**3.3.5 Large Artery Vasculitis**

**What are common characteristics for patients who are suspected to have a large vessel vasculitis?** [Shanmugam, 2019a, Weyand and Goronzy, 2003]

- Affect aorta and major branches

- Present with non-specific heterogenous symptoms making the diagnosis challenging. Most commonly, they present with systemic or constitutional symptoms (fatigue, fever, weight loss, arthralgias)
- Frequently, diagnosis made with presence of constitutional symptoms, elevated inflammatory markers, and dedicated imaging (MRA, CTA, DUS, or PET)

**How can you differentiate Takayasu arteritis vs giant cell arteritis?**

#### 3.3.5.1 Takayasu arteritis

1. Aorta and primary[Ehlert and Abularage, 2019b]
2. Young patients <20 years and female in 80-90% of cases, Asian populations
3. Criteria (ACR)
  1. Onset <40 years
  2. Claudication of an extremity
  3. Decreased brachial pulse
  4. >10 mmHg SBP between arms
  5. Bruit over subclavian arteries or aorta
  6. Arteriographic evidence of narrowing/occlusion in aorta/primary branches/or large upper/lower extremity arteries
4. Initial therapy for acute vasculitis should include steroids and close observation. If persistent severe symptoms after acute phase, then consider surgical repair. Mid aortic syndrome likely needs open repair from uninvolvled aorta-usually thoracic aorta-to bifurcation bypass with jump graft to involved visceral vessels.[Ehlert and Abularage, 2019b]

#### 3.3.5.2 Giant cell arteritis

1. Aorta and main branches, but predilection for carotid artery branches[Bongartz and Matteson, 2006]
2. Diagnosis:
  1. Age at disease onset > 50 years
  2. New headache
  3. Temporal artery abnormality
  4. Elevated ESR (>50) - can be normal in up to a quarter of cases.
  5. Abnormal artery biopsy (gold standard test)
3. Other symptoms include jaw pain with mastication or visual changes

4. Associated with Polymyalgia rheumatica, characterized by morning stiffness in shoulders/hips occurring in 40-50% of patients
5. Arteriography/MRA/CTA/PET may be used to assess large vessel involvement - classic finding is smooth, tapering stenosis.

**How should patients be monitored with active large artery vasculitis?**

- Lab data tracked at least monthly for 6 months with close follow-up to ensure appropriate response to medical treatment and enable physicians to assess for adverse effects of medical treatment
- Repeat tests after remission reached and imaging choice to evaluate large vessels (DUS/CTA/MRA)

**What is the medical treatment for GCA and when do you consider surgical treatment?**

- Medical-steroid therapy. In as many as 50% of patients who have a large vessel vasculitis refractory to glucocorticoid therapy alone, patients will trial immunomodulators or cytotoxic drugs (i.e. methotrexate, azathioprine, mycophenolate, tocilizumab, or leflunomide)
- Intervention-once remission, treatment of symptomatic arterial lesions or those at aneurysm size threshold should be considered and as many as 50-70% with large vessel vasculitis will require intervention.
  - Endovascular-angioplasty/stent/stent graft for large vessel vasculitis have all been described, however higher restenosis in endovascular compared to open treatment
  - Open Surgery (gold standard)-lesions are long, fibrotic and therefore less amenable to endovascular treatment. Bypass grafts from aorta-CCA are the most common (CEA should be avoided due to pathology involved)
    - \* Upper extremity bypass with autogenous vein to the brachial artery
    - \* Aortic aneurysms should be managed with open surgery[Janssen et al., 2008]

**3.3.6 Other Vasculitis syndromes**

Polyarteritis Nodosum - Focal necrotizing lesions primarily affecting medium-sized muscular arteries, peak incidence in 40s. Multiple saccular aneurysms secondary to inflammatory destruction of vessel media.[Shanmugam, 2019b, Weyand and Goronzy, 2003]

Behçet Disease - recurrent oral pathos ulcers, genital ulcers and uveitis.[Shanmugam, 2019b, Weyand and Goronzy, 2003]

## 3.4 Aneurysmal Disease

### 3.4.1 Subclavian Artery Aneurysms

**How are subclavian aneurysms caused and how can they present?**  
[shadman Baig and Timaran, 2019]

#### 3.4.1.1 Etiology

- Degenerative - atherosclerotic or due to aberrant right subclavian with degenerative changes in proximal subclavian known as “Kommerell diverticulum”
- Traumatic - blunt, penetrating, iatrogenic with attempted catheter placement
- Radiation induced, sometimes seen after radiation for breast cancer.[Mohan et al., 2016]
- Thoracic outlet obstruction - no need to investigate for aTOS if there is another plausible cause.

#### 3.4.1.2 Presentation

- Rare, comprises 1% of peripheral aneurysms.
- Exam-pulsatile supraclavicular mass or bruit, absent/diminished pulses, signs of microembolization (“blue finger”)
- Most discovered incidentally, however referred chest, neck, shoulder pain, upper extremity ischemia due to thrombosis or distal embolism, brachial plexus compression, hoarseness from right recurrent laryngeal nerve compression
- Dysphagia from esophageal compression in aberrant right subclavian artery
- Rarely rupture

#### 3.4.1.3 Evaluation

**What are diagnostic studies and treatment modalities for subclavian aneurysms?**

- CXR-mediastinal mass may suggest neoplasm
- MRA/CTA important to delineate extent of aneurysm and proximity to ipsilateral vertebral artery

#### 3.4.1.4 Management

- Open Repair-resection/endoaneurysorrhaphy with end to end (small aneurysms) or interposition prosthetic graft[Vierhout et al., 2010]

- Proximal-median sternotomy with supraclavicular fossa extension for adequate proximal control for right side, however supraclavicular with left anterolateral thoracotomy for left subclavian aneurysm
- Mid-Distal-supraclavicular/infraclavicular generally adequate for control where again resection of the clavicle may be needed
- Endovascular Repair-transbrachial/transfemoral approach with covered stent[Maskanakis et al., 2018]
  - Must consider vertebral artery origin. Can cover vertebral artery if contralateral vertebral artery is patent and of adequate size, however posterior circulation stroke may occur when the contralateral vertebral artery is highly stenotic, hypoplastic or occluded.
- Hybrid Repair-embolization/coils of proximal subclavian artery combined with subclavian transposition or carotid-subclavian bypass
- For aberrant subclavian artery aneurysm, resection or exclusion of the aneurysmal artery with vascular reconstruction of the subclavian artery is recommended. Especially in the setting of dysphagia lusoria, subclavian artery reconstructed by interposition graft where proximal anastomosis is on ascending aorta. Alternatively, left posterolateral thoracotomy for proximal aneurysm resection and right supraclavicular incision for reconstruction of subclavian artery by end to side to the right CCA has been reported.

### 3.4.2 Axillary Artery Aneurysms

How are axillary aneurysms caused and how can they present?

#### 3.4.2.1 Etiology

- Blunt/penetrating trauma
- Congenital (infrequently reported)
- Post-traumatic axillary aneurysms (repeated abduction/external rotation downward toward humeral head in baseball pitchers)

#### 3.4.2.2 Presentation

- Exam-pulsatile supraclavicular mass or bruit, absent/diminished pulses, signs of microembolization (“blue finger”)

What are diagnostic studies and treatment modalities for axillary aneurysms?

#### 3.4.2.3 Evaluation

- Ultrasound

- CTA/MRA of upper extremity

#### **3.4.2.4 Management**

- Open Repair-resection with interposition vein grafting or prosthetic if inadequate vein is present.
- Endovascular repair-covered stent graft can be placed with occasional embolization with micro coils to isolate sac and prevent retrograde endoleaks

### **3.4.3 Brachial Artery Aneurysms**

**How are brachial aneurysms caused and how can they present?**

#### **3.4.3.1 Etiology**

- False aneurysms secondary to repetitive trauma
- Iatrogenic complications - Seen in 1-3% of brachial artery access - increased risk in older age, female patients, and larger sheaths.[Treitl et al., 2015]  
More on appropriate access technique can be found in 9.2.4.3
- IV drug abuse (infected pseudoaneurysms in antecubital fossa)
- Connective tissue disorders (ex. type IV Ehlers danlos)

#### **3.4.3.2 Presentation:**

- Exam: pulsatile mass
- Local pain or symptoms of median nerve compressions
- Hand/digital ischemia from thrombosis/distal embolization

**What are diagnostic studies and treatment modalities for brachial aneurysms?**

#### **3.4.3.3 Evaluation**

- Duplex Ultrasound
- CTA/MRA of upper extremity may be needed to delineate extent of aneurysm

#### **3.4.3.4 Management**

- Open Repair (preferred)-resection with patch or interposition vein grafting
- Endovascular repair-rare and generally in a traumatic setting

- Iatrogenic injuries-due to access and nonoperative treatment for small/asymptomatic pseudoaneurysms that are likely to thrombose spontaneously. Direct suture repair with evacuation of hematoma is possible. Thrombin injection is less favorable due to location and short neck.
- Neurologic symptoms often from median nerve compression and require urgent repair, open surgery best for decompression.

### 3.4.4 Radial Artery Pseudoaneurysm

**How do you manage a patient who presents with a radial artery pseudoaneurysm after a coronary angiogram one year ago?**

Small pseudoaneurysms <3cm have a high rate of spontaneous thrombosis. However, larger pseudoaneurysms or those with symptoms require treatment. Often best managed with open excision and primary repair or interposition graft.[Tosti et al., 2017]

Can also be seen as a result of trauma.[Bagir et al., 2017]

## 3.5 Occupational Vascular Disease

**There are some occupational vascular disorders than contribute to vascular disease in the upper extremity. Hand arm vibration syndrome and hypothenar hammer are of particular importance. Can you talk to us about the key information from these syndromes? [Eskandari and Morgan, 2020]**

### 3.5.1 Hand-Arm Vibration Syndrome

#### 3.5.1.1 Etiology

- Vibrating handheld machines (eg pneumatic hammers and drills, grinders, and chain saws)
- Linear relationship between exposure over years and onset of this syndrome
- Exact mechanism unknown, but thought that endothelial damage with sympathetic hyperactivity -> finger blanching attack

#### 3.5.1.2 Presentation

- Various stages seen where early results in slight tingling/numbness and lateral, the tips of one or more fingers experience attacks of blanching that is usually precipitated by cold

- Blanching typically lasts 1 hour and terminates with reactive hyperemia, but prolonged exposure can cause bluish black cyanosis of fingers

#### **3.5.1.3 Evaluation**

- Detailed history with use of vibrating tools/symptoms of Raynaud phenomenon
- Objectively: cold induced ischemia with recording time until digital temperature recovers
- Digital occlusion with noninvasive digit pressures or duplex scanning

#### **3.5.1.4 Management**

- Avoidance of vibratory tools
- Nifedipine (Ca<sup>2+</sup> channel blocker) in advanced cases
- IV prostacyclin (ie prostacyclin) for digital gangrene
- Surgery-cervical sympathectomy or digital sympathectomy rarely needed

### **3.5.2 Hypothenar hammer syndrome**

#### **3.5.2.1 Etiology**

- Repetitive use of palm of hand in activities that involve pushing, pounding, twisting - particularly to the ulnar artery as it exits guyots canal and crosses the hook of the hamate.[Ferris et al., 2000]
- Name comes from reports of mechanics, factory workers, carpenters or laborers who habitually use their hands as a hammer are at risk for disease
- Repetitive trauma leads to thrombotic occlusion, aneurysm formation or both

#### **3.5.2.2 Presentation**

- Asymmetrical distribution involving dominant upper extremity where cyanosis and pallor can occur and digits affected are ulnar distribution in nature
- Cool/mottled digits or severe cases with ischemic ulcers

#### **3.5.2.3 Evaluation**

- Duplex ultrasound
- CTA or MRA

- Arteriography (gold standard) with corkscrew pattern typically in affected vessels

#### 3.5.2.4 Management

- Conservative-smoking cessation/hand protection/cold avoidance.[Carr et al., 2019]
- Medical-calcium channel blockers/antiplatelet
- Surgical (severe digital ischemia/aneurysm)-ligation if adequate collateral or interposition vein graft has good long term patency.

#### 3.5.3 Environmental Exposures

**Exposure to what environmental agents can result in upper extremity ischemia?**

Acroosteolysis

- Exposure to polyvinyl chloride can result in ischemic hand symptoms similar to those of Raynaud syndrome
- Angiography-damage to digital arteries with multiple stenosis/occlusions or hyper vascularity adjacent to areas of bone resorption
- Treatment-supportive

Electrical burns

- <1000 V cause injuries limited to immediate skin/soft tissue, however >1000 V cause damage from entry to exit point
- Results in arterial necrosis with thrombus or bleeding and gangrene of digits develop
- Initially can be occlusion/thrombosis or spasm, however later damage can cause aneurysmal degeneration
- Treatment-dependent on soft tissue/bone injuries as well. Can have reconstruction with free flap due to local vascular damage or occlusion of major artery requiring bypass grafting

Extreme thermal injuries

- Workers at risk with chronic exposure to cold (slaughterhouse, canning factory, and fisheries)
- Raynaud syndrome symptoms due to vasomotor disturbances in the hands when exposed to extreme chronic thermal trauma
- Treatment-Supportive

### 3.5.4 Sports Medicine

**How can athletes specifically be affected by upper extremity ischemia?**

Athletes who engage in strenuous or exaggerated hand/shoulder activity may be susceptible to upper extremity ischemia from arterial injury manifested by Raynaud syndrome, symptoms of sudden arterial occlusion or digital embolization

## 3.6 Vascular Trauma-Upper Extremity

This is discussed in detail here: 10.1, so we will go over some important specifics for upper extremity vascular injury. [Kauvar and Kraiss, 2020]

### 3.6.1 Subclavian Artery Trauma

**What is the management strategy for an iatrogenic placement of a subclavian artery catheter?**

Critically ill patients can be treated effectively with the use of a closure device or covered stent placement.[Yoon et al., 2015, Tran et al., 2009, Cohen et al., 2014] Cut down in the mid subclavian artery is very difficult to perform and results in increased blood loss and worse outcomes.

Trauma to the proximal subclavian should be accessed on R through a median sternotomy or trapdoor incision. On the left, the proximal subclavian is accessed best through an anterolateral thoracotomy. For more on exposures, see 12.4.

### 3.6.2 Axillary Artery Trauma

**What is the mechanism and management of upper extremity axillary artery trauma?**

#### 3.6.2.1 Etiology

- Predominantly in penetrating trauma with equal incidence in proximal/middle/distal divisions and brachial plexus injury in >1/3rd of arterial injury

#### 3.6.2.2 Evaluation

- Physical exam with deficiencies in upper extremity pulses/ischemic changes, but may not be present given collateral flow from axillary artery to upper extremity
- High index of suspicion with location of injury proximity to course of axillary artery

- Upper extremity Doppler or CTA if patient is stable for diagnosis

### 3.6.2.3 Management

- Primary repair or treated with interposition graft
- If hemodynamically stable, can consider covered stent based on location to thoracic outlet via femoral/brachial approach

## 3.6.3 Brachial Artery Trauma

**What is the mechanism and management of upper extremity brachial artery trauma?**

### 3.6.3.1 Etiology

- Frequently associated with humerus fractures/elbow dislocation
- Penetrating trauma

### 3.6.3.2 Evaluation

- Pulse deficit in majority (>75% of cases)
- Upper extremity Doppler or CTA

### 3.6.3.3 Management

- Given course, can be extensively mobilized and repaired in end-to-end fashion in 50% of cases. Otherwise, treatment with an interposition graft

## 3.6.4 Radial/ulnar artery trauma

**What is the mechanism and management of upper extremity radial/ulnar artery trauma?**

### 3.6.4.1 Etiology

- Associated with significant soft tissue pattern

### 3.6.4.2 Evaluation

- Pulse deficit in >80% of patients
- Doppler based Allen test-confirmed radial/ulnar contribution to palmar arch

### 3.6.4.3 Management

- If Allen test reveals a patent palmar arch, the injured artery can be ligated
- If palmar arch is not patent in the absence of contribution of the injured artery, it should be repaired
- If both are damaged, preference to ulnar artery as dominant contribution to hand
- Generally, repair can be done in an end to end fashion given mobility of the vessel

## 3.7 Compression Syndromes

**The main syndromes are quadrilateral space syndrome and humeral compression of the axillary artery. What important information here do our listeners need to know?**

### 3.7.1 Quadrilateral space syndrome

#### Anatomy

- Bordered by teres minor superiorly, humeral shaft laterally, and teres major inferiorly, and long head of triceps muscle medially
- Posterior humeral circumflex artery and axillary nerve in space

#### Etiology

- Compression of posterior humeral circumflex occurs with abduction/external rotation
- Typically seen with chronic overhand motion athletes (pitchers/volleyball players)
- Vascular-repetitive mechanical trauma to posterior circumflex humeral artery
- Neurogenic-fixed structural impaction of quadrilateral space by fibrous bands or space-occupying lesions

#### Presentation

- Muscle atrophy, paresthesias, poorly localized shoulder pain and pain in quadrilateral space

#### Management

- Medical: Oral anti-inflammatory medications, PT, limitation of activities
- Surgery: decompression with neurolysis/excision of fibrous bands or other space occupying lesions

### 3.7.2 Humeral head compression of axillary artery

Anatomy

- 3rd portion of axillary artery compressed by head of humerus

Etiology

- Arm is abducted and externally rotated with downward compression of humeral head to axillary artery

Presentation

- Arm fatigue, loss of pitch velocity, finger numbness, Raynaud, cutaneous embolization

Evaluation

- Provocative maneuvers with impedance of flow through axillary artery on ultrasonography
- Arteriography with rest and provocative position

TManagement

- Supportive with avoidance of throwing motion
- Surgical-saphenous vein patch for no improvement or structural injury may require resection with saphenous vein bypass anatomically or extra-anatomic tunneling above pec minor

## 3.8 Thoracic Outlet Syndrome

**27 Nov 2019:** Nedal Katib, Prince of Wales, Sydney Australia

Thoracic Outlet Syndrome = A constellation of signs and symptoms relating to the compression of the neurovascular structures that occurs as these structures travel between the thoracic aperture and the upper limb.

Types: Neurogenic, Venous and Arterial

- vTOS – 2-3%
- aTOS – 1%
- nTOS – >95% [Humphries and Freischlag, 2019b]

### 3.8.1 Anatomy

Understanding the anatomy of what is collectively referred to as the thoracic outlet is the best way to thoroughly appreciate this topic.

Anatomy from anterior to posterior

- Subclavian vein

- Phrenic nerve
- Anterior scalene muscle attachment to the first rib
- The subclavian artery
- The brachial plexus
- The middle scalene muscle.

Three spaces where the neurovascular structures are at risk of compression:

1. Interscalene Triangle
2. Costoclavicular Passage[Gary G Wind and R. James Valentine, 2013]
3. Subcoracoid Space[Gary G Wind and R. James Valentine, 2013]

#### **Interscalene Triangle:**

Appreciating the attachments of the Anterior and Middle Scalene Muscles on the first rib becomes important in the diagnosis of the various types and also the ultimate surgical management of the compression.

#### **Anterior Scalene:**

Attachments: Anterior Tubercles of the four ‘typical’ cervical vertebrae (3-6) AND the scalene tubercle on the upper surface of the first rib.

- Phrenic nerve runs along anterior scalene muscle and injury can cause ipsilateral diaphragm paralysis.

#### **Middle Scalene:**

Attachments: The posterior tubercles and intertubercular lamellae of all the cervical vertebrae AND the Quadrangular area between the neck and subclavian groove of the first rib. [McMinn, 2019]

- Long thoracic nerve runs along middle scalene muscle and injury can cause winged scapula.

#### **The First Rib:**

- The broadest and flattest of the ribs and is an ‘Atypical Rib’.
- The upper surface of the first rib has the scalene and quadrangular tubercles for attachments of the anterior and middle scalene muscles respectively. There are also three grooves for the Subclavian Vein, artery and the Lower Trunk of the Brachial Plexus.
- The Inferior Surface is smooth and inferior and medially has an attachment for the suprapleural membrane, Sibson’s fascia AKA scalenus minimus, which is tethered to the C7 vertebrae.
- This is the passage of the subclavian vein largely as it emerges through the tight space created by the clavicle, the subclavius muscle and the

costoclavicular ligament and also more posteriorly this can also compress the artery and nerves as the space can also be narrow in relation with the scapula and subscapularis. [Gary G Wind and R. James Valentine, 2013]

**Subclavius Muscle:**

- Attached to the costochondral junction of the first rib and is inserted into the subclavian groove on the inferior surface of the clavicle. [McMinn, 2019]
- This space is best appreciated by intimate knowledge of three things:
  - The Coracoid Process and its attachments
  - The Pectoralis Minor Muscle
  - The Clavipectoral Fascia

**The Coracoid Process:**

- Arising from the Scapula as a ‘process’, this broad-based bony landmark offers attachment to muscles and ligaments.
- The relevant attachments being the pectoralis minor muscle occupying the medial border for about 2cm behind its tip. The tip itself having a medial and lateral facet for the short head of biceps and the coracobrachialis muscles respectively.

**Pectoralis Minor Muscle:**

Attached to the bone of the third, fourth and fifth ribs AND the medial border of the coracoid process.

**Clavipectoral Fascia:**

A sheet of fascial membrane that fills the space between the clavicle and pectoralis minor splitting and encompassing the subclavius muscle. Its superior portion is what can be thickened and become a tight band referred to as the costocoracoid ligament.

**Phrenic Nerve Anomaly:**

The Phrenic Nerve normally runs anterior to the Subclavian Vein. A rare anomaly is the nerve compressing the vein anteriorly and in very rare circumstances due to the timing of development can run through the vein itself.

Anomalous anatomy can also cause TOS especially when patients have a Cervical Rib and anomalous first ribs or a congenital band attaching to the first rib.

- Incidence of anomalous first ribs and cervical ribs is 0.76% and 0.75% respectively.
- Incidence of bands are as high as 63% in the general population. [Humphries and Freischlag, 2019b]

**nTOS**

- Scalene Triangle compression – most common cause of brachial plexus and neurogenic TOS
- Cervical Rib and Anomalous First Rib

**aTOS**

- Cervical Rib and Anomalous First Rib
- Scalene Triangle compression

**vTOS**

- Costoclavicular Passage
- Subcoracoid Space

**3.8.2 Presentation**

- Identify symptoms and thoroughly interrogate timing
- Exclude history of trauma
- Associated symptoms like headache, visual disturbance, neurology in the upper limb
- Exclude Carpal Tunnel and Antecubital Tunnel Syndromes if symptoms are isolated to the arm or forearm or hand
- Patients with vTOS may present acutely and have acute or sub acute Upper Limb DVT
- Patients with aTOS need to be investigated and assessed urgently given risk of ischemia.

**3.8.3 Evaluation****3.8.3.1 Clinical Examination**

Provocative maneuvers are largely used for nTOS. While these are described and mentioned in most texts their utility largely is beyond the scope of a vascular surgeon's assessment and diagnosis of nTOS.

**Adson Test**

- Extended abducted and externally rotated arm – palpate radial pulse
- Rotate and laterally flex the neck to the ipsilateral side while inhaling deeply.
- A positive test results in reduction or complete obliteration of radial pulse

**Roos Test / EAST test**

- Patient seated and both arms abducted 90 degrees and externally rotated and elbows flexed at 90 degrees.
- Open and close hands for 3 minutes or until pain or paraesthesia sets in.

#### Elvey's Test

- Abduct both arms to 90 degrees with elbows extended and dorsiflex both wrists.
- If pain is elicited as wrists dorsiflexed then test is positive.
- A further manoeuvre is then performed, laterally flex the head on each side, if pain is elicited on the contralateral side to which the head is flexed then test is positive. [Humphries and Freischlag, 2019b]

#### 3.8.3.2 Non-invasive imaging or vascular lab studies

- DBI
- Arterial Duplex
- Venous Duplex
- CT – CTV commonly performed in acute upper limb DVT and suspicion of vTOS
- CTA for the evaluation of aTOS and excluding other causes of embolisation
- MRI – for further evaluation of the anatomy and related neurovascular compression
- Electromyography and Nerve Conduction Studies for nTOS

#### 3.8.4 Management

After the appropriate evaluation, if decompression of thoracic outlet is deemed appropriate, there are multiple approaches to the first rib resections.

|                            | Advantages  | Disadvantages  |
|----------------------------|---|--|
| <b>Transa<br/>xillary</b>  | <p>Cosmetically more appealing as it has a limited hidden scar</p> <ul style="list-style-type: none"> <li>Good for scalene triangle *Supraclavicular access and debulking and vicular** cervical rib resection</li> <li>Required for aTOS if arterial reconstruction necessary</li> <li>Good access for venous *Infraclavicular decompression</li> <li>Allows for excision of subclavius muscle and costoclavicular ligament</li> </ul> | <p>Difficult to visualize the anatomy, dependent on good assistance</p> <p>Risk of injury to T1 nerve root, phrenic nerve, long thoracic, brachial plexus , subclavian vein and arterial with limited exposure to repair</p> <p>Not able to approach cervical ribs, scalene triangle or patch vein.</p> <p>Unable to decompress venous compression or visualize vein adequately</p> <p>Cosmetically less appealing</p> |
| <b>Paracla<br/>vicular</b> | Useful if mixed etiology TOS to adequately decompression all neurovascular structures   | <p>Unable to expose subclavian artery or decompress brachial plexus.</p> <p>Difficult to access most posterior aspect of rib</p> <p>Cosmetically less appealing</p> <p>Requires two incisions one above and below the clavicle</p>   |

Siracuse et al. [2015]

### 3.8.4.1 Post operative complications

- Post operative patients with hemodynamic instability and ipsilateral effusion on xray should go back to OR for exploration and hemorrhage control. Rinehardt et al. [2017]
- Chyle leak often managed with adequate drainage and medium chain fatty acid diet. Persistent leak should undergo wound exploration and ligation of thoracic duct (most often encountered on the left) or VATS thoracic duct ligation.[Delaney et al., 2017] IR embolization has also been shown to be effective.[Cope et al., 1999]

### 3.8.5 vTOS

### 3.8.5.1 Demographics

- Incidence: 2/100,000 persons
- Age: 18 years to 30 years [Illig and Doyle, 2010]
- M>F
- **Paget Schroetter Syndrome**
  - First defined by Hughes in 1949 in reference to Sir James Paget who in a hundred years earlier defined acute arm swelling and pain as possibly related to vasospasm and then von Schroetter who in 1884 attributed to the presentation to subclavian and axillary vein thrombosis. [Humphries and Freischlag, 2019a]
  - Now vTOS and Paget Schroetter Syndrome are used synonymously.
  - Paget Schroetter Syndrome accounts for 10-20% of all upper extremity deep vein thrombosis. [Sekhar, 2018]

### 3.8.5.2 Presentation

- Upper Limb edema, pain and cyanosis. Edema affects the shoulder, arm and hand and characteristically non pitting.
- Collateral vein dilatation over the shoulder, neck and anterior chest wall to accommodate for the increased venous hypertension. [Humphries and Freischlag, 2019a]
- Pain on exertion of the upper limb described as stabbing, aching or tightness.
- The reported incidence of PE following Upper Limb DVT is <12%. [Humphries and Freischlag, 2019a]

### 3.8.5.3 Evaluation

- A differential diagnosis for Upper Limb DVT
  - vTOS
  - Congenital Phrenic Nerve anomaly
  - History of Fracture, Clavicular Fracture and malunion
  - Repetitive arm provocative manoeuvres, check occupation and history of body-building
    - \* Pectoralis Minor Hypertrophy.
- Exclude Pulmonary Embolism
- Exclude Venous Gangrene and Phlegmasia of the upper limb

### 3.8.5.4 Management

Limited evidence due to lack of RCT's. Majority of evidence based on retrospective studies.

- Prevent immediate risk
- Return patient to unrestricted use of the affected extremity
- Prevent recurrence of thrombosis without the need of long-term anticoagulation
- Prevent long term Post Phlebitic Limb Syndrome

#### 3.8.5.4.1 Medical Management

- As per ACCP Guidelines: Initial management is anticoagulation regardless of etiology. [Kearon et al., 2016]
  - The limitations of anticoagulation alone are that the slow recanalization of the thrombus may lead to eventual valvular damage and intravenous scarring. [Sekhar, 2018]
  - Thrombolysis has been considered superior to anticoagulation alone in minimizing valvular damage due to residual clot. [Urschel and Patel, 2008]
  - Systemic Lysis – non favored due to risk of intracranial hemorrhage. [Grunwald and Hofmann, 2004]
  - Catheter Directed Lysis (CDT) – carries a lower risk of intracranial hemorrhage.
  - Patient should be maintained in a compression sleeve until definitive decompression can be performed.
- Optimal timing of CDT
  - Within 14 days of onset of thrombosis. Excellent results have been reported following CDT if initiated before 14 days. [Wilson et al., 1990]

#### 3.8.5.4.2 Surgery

- After initial management patients are generally divided into two groups, unsuccessful or successful thrombolysis.
- Persistent stenosis or signs of extrinsic compression, on venography, has generally been perceived as a significant risk of recurrent thrombosis.
- Surgery for vTOS remains to be mainly Rib Resection and decompression of the subclavian vein with or without venolysis and patch plasty either surgical or endovenous.

- Surgical treatment of severe resistant subclavian vein stenosis in the setting of vTOS is rib resection by paraclavicular approach and vein patch plasty. [Melby et al., 2008]
- Venous occlusion in vTOS may be treated with jugular turn down or venous bypass to IJ of SVC if patients remain symptomatic. [Vemuri et al., 2016]

### 3.8.5.5 Summary

#### 3.8.5.5.1 Controversy

- There is a lack of consensus around the necessity of surgical rib resection, the timing and the requirement for vein patch plasty.
- Options post recanalization:
  - Deferring surgical decompression for 1-3 months after thrombolysis to allow for healing of the venous endothelium and resolution of the acute inflammatory process. [Humphries and Freischlag, 2019a]
  - Decompression during the same admission, as the thrombolysis, with the main benefit being to reduce the risk of re-occlusion. [Humphries and Freischlag, 2019a, Molina et al., 2007]
  - Post decompression venography and treatment 2 weeks post rib resection may help to prevent recurrence and long term vein patency. [Chang et al., 2012]

#### 3.8.5.5.2 Landmark papers

1. Lugo J et al – Acute Paget Schroetter syndrome: does the first rib routinely need to be removed after thrombolysis? Annals of Vascular Surgery 2015 [Lugo et al., 2015]
  1. Systematic literature review analysis. Patients divided into three groups
    1. First Rib resection (FRR) – n=448
    2. First Rib resection and endovenous venoplasty (FRR and PLASTY) n=68
    3. No further intervention after Thrombolysis n=168
  2. Symptom relief after initial follow up more likely in FRR (95%) and FRR and PLASTY (93%) compared to no rib removed (54%)
    - p<0.0001
  3. Results showed superior patency with FRR and PLASTY and FRR compared to anticoagulation alone.

4. Conclusion was that patients are more likely to experience greater long-term results with FRR compared to no FRR.
2. Sajid MS et al – Upper limb vein thrombosis: a literature review to streamline the protocol for management. *Acta Haematology* 2007 [Sajid et al., 2007]
  - Comprehensive review identifying the key papers on this topic and allows for a clear view of the best management strategy.
3. Vemuri, C., Salehi, P., Benarroch-Gampel, J., McLaughlin, L. N., & Thompson, R. W. (2016). Diagnosis and treatment of effort-induced thrombosis of the axillary subclavian vein due to venous thoracic outlet syndrome. *Journal of Vascular Surgery: Venous and Lymphatic Disorders*, 4(4), 485–500. [Vemuri et al., 2016]
  - Comprehensive summary of management strategy for effort induced thrombosis.

### 3.8.6 aTOS

#### 3.8.6.1 Presentation

- Most common: Hand ischemia due to arterial compression or microembolization with subclavian artery aneurysm and pulsatile supraclavicular mass [Boll and Valentine, 2019]
- Less common: Exertional pain, unilateral Raynaud's Phenomena, retrograde embolisation and neurological symptoms
- Differential Diagnosis
  - Trauma
  - Primary and Secondary Raynaud's Phenomena
  - Small Vessel Vasculitis
  - Connective Tissue Disorders
  - Thromboangiitis Obliterans
  - Arterial Embolisation – Aortic or Central Source
  - Radiation Arteritis
  - Atherosclerotic / Dissection causes
- The different anatomical abnormalities causing aTOS [Boll and Valentine, 2019]
  - Cervical Rib (60%)
  - Anomalous First Rib (18%)

- Fibrocartilaginous band (15%)
- Clavicular Fracture (6%)
- Enlarged C7 transverse process (1%)

### 3.8.6.2 Evaluation

#### Clinical Examination

- Audible Bruit / Palpable thrill over the supraclavicular fossa
- Pulsatile mass
- Distal ischemic lesions in the distal hand – Splinter hemorrhages
- Positive Adson Test

Most useful studies are pulse volume recordings (PVR) and duplex to identify aneurysm or sites of embolization. Stress test is not reliable for diagnosis. [Vemuri et al., 2017, Criado et al., 2010]

### 3.8.6.3 Management

- Symptomatic patients are generally indicated for treatment. Unlike asymptomatic patients in whom it may be appropriate to manage conservatively. [Boll and Valentine, 2019]
  - Scher Staging of aTOS
    - \* Stage 0: Asymptomatic
    - \* Stage 1: Stenosis of Subclavian Artery with minor post stenotic dilatation with no intimal disruption
    - \* Stage 2: Subclavian artery aneurysm with intimal damage and mural thrombus
    - \* Stage 3: Distal embolisation from subclavian artery disease
- Supraclavicular rib resection is the most suitable for adequate arterial reconstruction. Transaxillary has been argued to offer more complete rib resection however arterial repair is not possible in this approach.
- Subclavian artery repair is necessary in Scher Stages 2 and 3 and in some cases 1. Arterial repair with conduit either GSV, Femoral Vein or prosthetic have been described. Ringed PTFE offers good patency and resistance to kinking in this functional anatomical location.

### 3.8.7 nTOS

### 3.8.7.1 Demographics

Neurogenic TOS is largely a clinical diagnosis with symptoms and signs pertaining to nerve compression most commonly the lower trunk of the brachial plexus.

- F>M – 70% Female
- Ages 20-40
- Occupational Exposure
- Trauma history - often associated with recent history of neck trauma.

### 3.8.7.2 Presentation

- Symptoms [Sadeghi-Azandaryani et al., 2009, Sanders et al., 2007]
  - Paraesthesia (98%)
  - Trapezius pain (92%)
  - Neck, shoulder or arm pain (88%) - usually involves nerve roots so isolated hand symptoms is rare.
  - Supraclavicular pain with or without occipital headache (76%)
  - Chest pain (72%)
  - Weakness
  - Swelling
- Positional Effects
  - Reproducible exacerbation of symptoms
  - Lying supine with arms overhead
  - Overhead activities -occupational or recreational
- Weakness and Muscle Atrophy
  - Hypothenar atrophy
  - Drop-off in athletic performance
  - Inability to carry out activities of daily living

### 3.8.7.3 Management

As the patient has already seen multiple specialists and physiotherapists, the role of the vascular surgeon with nTOS is often limited but should focus on:

- Exclude other causes
- Confirm diagnosis – Neurophysiologic Tests (EMG and NCS)

- Seek alternate opinion
- Trial of Physiotherapy and non-operative management - patients should be evaluated and undergo a 6 week course of physical therapy. This physical therapy focuses on scalene and pectoralis stretching improving mobility of the shoulder and strengthening the arm. Many improve with physical therapy. [Balderman et al., 2019]
- Anterior scalene lidocaine block may provide temporary symptom relief (~7 days) and may help identify those patients most likely to benefit from surgical decompression. [Salhan et al., 2016, Lum et al., 2012b]
- Botulinum injection may give an average of 6 weeks of relief. [Salhan et al., 2016]
- Be selective in patients who may require surgery

Surgery with Rib resection often is accomplished with transaxillary or suprACLAVICULAR approach, particularly if scalenectomy or cervical rib resection is necessary.

## 3.9 Upper Extremity Lymphedema

### 3.9.1 Etiology

Often due to breast cancer therapy and axillary lymph node dissection.[Morrell et al., 2005]

### 3.9.2 Management

Initial treatment includes decongestive therapy, such as daily massage for 2-4 weeks. Once the girth and symptoms stabilize, then can transition to long term management with compression sleeve.[Mondry et al., 2004]

Severe cases may require lymph node transfer.[Warren et al., 2007]

Lymphangiosarcoma in the setting of lymphedema (Stewart-Treves syndrome) without distant disease is best managed with wide local excision and adjuvant chemoradiation. Prognosis is poor.[Sharma and Schwartz, 2012]



# Chapter 4

## Abdominal Aneurysms

**UNDER CONSTRUCTION - This Chapter is Currently in DRAFT mode**

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### 4.1 Pathogenesis, presentation and risk factors

**What is an abdominal aortic aneurysm (AAA)?** [Moore et al., 2019]

- Defined as a localized dilation of an artery to a diameter greater than 50% (1.5x) of its normal diameter. It is generally accepted that >3cm in adults is considered aneurysmal for the abdominal aorta.
- AAAs can be described as:
  - Infrarenal – distal to the renal arteries with normal aorta between the renal arteries and the aneurysm origin.
  - Juxtarenal – aneurysm extends to the renal arteries but does not involve them
  - Pararenal – aneurysm involving the origin of at least one of the renal arteries
- Estimated 1.1 million Americans have AAAs, which equates to a prevalence of 1.4% in 50-84 year old general population.
  - Men 65-75y with a history of smoking have a prevalence of 6-7% - hence the need for screening.[LeFevre and Force, 2014, Chaikof et al., 2018a]
  - Men without a history of smoking have a prevalence of 2%.

- Women who are current smokers 2%, and women with a history of smoking 0.8%. [Wanhainen et al., 2006]
- AAAs are 3-7x more prevalent than thoracic aortic aneurysms and can co-exist with other aneurysms throughout the arterial vascular system like popliteal artery aneurysms.
  - In a 10-year review originating from Ireland, 50% of patients that presented with unilateral popliteal artery aneurysms had associated AAA. In patients with bilateral popliteal aneurysms, 63% of those had associated AAA. [Duffy et al., 1998]
  - Patients with femoral aneurysms have an 85% chance of an associated AAA. [Diwan et al., 2000b]
  - Conversely, if a patient is first found to have a AAA, there is an 11% chance of having associated popliteal artery aneurysms >15mm. [Tuveson et al., 2016]
  - Another study showed a rate of femoral-popliteal aneurysms in AAA patients is approximately 14%. [Diwan et al., 2000b]
  - This association stresses the importance of a good physical exam when evaluating a patient with a AAA and is commonly tested on exams. [Chaikof et al., 2018a, Diwan et al., 2000a]

**What is the pathogenesis of an abdominal aortic aneurysm?** [Moore et al., 2019]

- More than 90% of AAAs are associated with atherosclerosis.
- Other causes include cystic medial necrosis, dissection, Marfan's syndrome, Ehler's-Danlos syndrome, HIV and syphilis.
- Elastin and collagen are the major structural proteins responsible for the integrity of the aortic wall and defects in these cause degeneration and further aneurysmal change.
- For example, a mutation in fibrillin in Marfan's syndrome causes elastin fragmentation and pathological remodeling of the wall of the artery to form cystic medial degeneration.
- Several investigations have also shown that upregulations of metalloproteinase activity, specifically MMP-2 and MMP-9, have an essential role in aneurysm formation. Imbalances between aortic wall proteases and antiproteases cause degradation of the extracellular matrix and loss of structural integrity of the aortic wall.
- Increased thrombus burden is associated with wall thinning, medial loss of smooth muscle cells, elastin degradation, adventitial inflammation and aortic wall hypoxia which all increase the rate of AAA growth.

- Of note, intraluminal thrombus rarely embolizes and is not an indication for anticoagulation.[Singh et al., 2019, Cameron et al., 2018]
- Infective aortitis is a rare but highly morbid cause for aortic aneurysm formation. Treatment in this setting should be more aggressive with the addition of antibiotic therapy and open surgical repair with rifampin soaked graft whenever feasible.[Gornik and Creager, 2008, Paravastu et al., 2009, Rasmussen and Hallett, 1997]
- For more about aortitis, see 3.3.5.

**What are the risk factors for AAA occurrence and growth?** [Moore et al., 2019]

- Risk factors for AAAs are similar to the risk factors for occlusive atherosclerosis and include age, tobacco use, hypertension, male gender and hypercholesterolemia.
- It has been found that diabetes is protective for AAA progression and rupture.
- Cigarette smoking is the single most important modifiable risk factor to prevent occurrence and growth of AAAs. Smoking increases the rate of growth by 35% for abdominal aortic aneurysms.
- Medical therapy has been studied with disappointing results. Beta-blockers and ACE/ARB inhibitors have been studied but have not shown any effect on growth of AAAs.
- Men more likely to have AAA, but women develop at a later age with more aggressive growth and risk for rupture. [Lo and Schermerhorn, 2016b]
  - Stronger association with smoking in women.
  - Women have same outcomes from endo repair, higher complications from open repair due to poorly managed CV risk factors.
- Fluoroquinolones[Lee et al., 2015, 2018, LeMaire et al., 2018, Pasternak et al., 2018]
  - In a recent study just published in JAMA Surgery this January, the group at UNC showed an increased short-term risk of developing an aortic aneurysm with fluoroquinolone use. [Newton et al., 2021]
  - They reviewed all prescription fills for fluoroquinolones or comparative antibiotics from 2005-2017.
  - This included >27 million US Adults aged 18-64 years old with no history of aneurysms.
  - 18% of the prescriptions were fluoroquinolones.
  - Fluoroquinolones were associated with increased incidence of aortic aneurysms. Compared to the other antibiotics, fluoroquinolones were

associated with a higher 90-day incidence of AAA and iliac aneurysms as well as more likely to undergo aneurysm repair.

- They recommended that fluoroquinolone use should be pursued with caution in all adults, not just high risk individuals, and they recommended broadening of the warnings from the FDA.
- Fluoroquinolones playing a role in dissections and aneurysm formation is often a highly tested question

#### **What is the dreaded complication of AAA?**

- Aneurysm rupture is the fear with a diagnosis of AAA. The risk of rupture increases yearly as the aneurysm expands. Once an aneurysm develops, it tends to enlarge gradually yet progressively. This is an important concept to grasp for testing.[Lindsay, 2019a, Tracci et al.]
- Growth rate
  - For smaller aneurysms (3-5cm in size), the growth rate is approximately 2-3mm/year
  - For larger aneurysms (>5cm), the growth rate is higher at 3-5mm/year.
- Rupture risk, historically, has been described as:

Table 4.1: Historical reporting of AAA rupture risk[Tracci et al.]

| AAA Size | Cumulative Annual Rupture Risk |
|----------|--------------------------------|
| 3-3.9 cm | 0.3%                           |
| 4-4.9 cm | 0.5-1.5%                       |
| 5-5.9 cm | 1-11%                          |
| 6-6.9 cm | 11-22%                         |
| >7 cm    | >30%                           |

- Newer data suggests the true rupture risk per year is decreasing with time. Rupture risk has recently been reported to be much lower.
  - A systematic review from the UK published in JVS in 2015, the rupture risks were far lower than previously reported and what is documented in most textbooks. Data included 11 studies, reviewing 1514 patients, published between 1995 and 2014.[Parkinson et al., 2015]

Table 4.2: AAA rupture risk based on most recent publications

| AAA Size   | Cumulative Annual Rupture Risk       |
|------------|--------------------------------------|
| <5.5 cm    | <1%[Oliver-Williams et al., 2019]    |
| 5.5-6.0 cm | 3.5% [0-9%][Parkinson et al., 2015]  |
| 6.1-7.0 cm | 4.1% [0-9%][Parkinson et al., 2015]  |
| >7.0 cm    | 6.3% [0-14%][Parkinson et al., 2015] |

- Factors that increase the risk of rupture other than the size of the aneurysm are smoking, COPD, hypertension, transplant recipient, and rapid enlargement (defined as 1 cm/year or more).

## 4.2 Evaluation and Diagnosis

**What is the work up for a AAA?** [Moore et al., 2019]

- 75% of all infrarenal AAAs are asymptomatic when first detected and often incidentally discovered on unrelated imaging.
- Symptoms - Some patients may report symptoms such as abdominal, flank or back pain from pressure on adjacent somatic sensory nerves or overlying peritoneum. Tenderness by itself is not a reliable indicator of impending rupture. Other symptoms include thrombosis and distal embolization.

### 4.2.1 Imaging

- Ultrasound, when feasible, is the preferred imaging modality for aneurysm screening and surveillance.
  - The Society for Vascular Surgery (SVS) recommends a one-time ultrasound screening in men and women ages 65 to 75 years with either a history of smoking or a family history of AAA, as well as men and women over the age of 75 with a smoking history in otherwise good health who have not previously undergone screening. [Chaikof et al., 2018b]
  - For more technical details on duplex screening see 12.6.1.

Table 4.3: Recommended Intervals for AAA Surveillance[Thompson et al., 2013, Chaikof et al., 2018b]

| Size on Imaging | Surveillance Interval |
|-----------------|-----------------------|
| 2.5 – 2.9 cm    | 10 years              |
| 3 - 3.9 cm      | 3 years               |
| 4 - 4.9 cm      | 1 year                |
| 5 - 5.4cm       | 6 months              |

- It is important to note that these screening guidelines are Level 2, Grade C evidence from the SVS.
- Traditionally, once duplex reveals an aneurysm 5cm in size, an initial CTA is performed and patients are followed with additional CT scans to assist with operative planning.
- CT Angiograms are helpful in operative planning and determining candidacy for EVAR. You can assess the relationship of the aneurysm to the renal arteries, assess the access vessels, and measure seal zones
  - The maximum aneurysm diameter derived from the CTA should be based on outer wall to outer wall measurement perpendicular to the path of the aorta (the centerline of the aneurysm).
- MRA is recommended for patients with renal insufficiency who cannot tolerate iodinated contrast.

### 4.3 Management

**What are the indications for repair?** [Moore et al., 2019]

- The current recommendation to repair a fusiform aneurysm is 5.5cm for men (Level 1, Grade A evidence), 5.0cm for women as they have a higher risk for rupture, and rapid growth (>5mm over 6 months). [Chaikof et al., 2018b]
- For saccular aneurysms, the SVS practice guidelines recommend elective repair (Level 2, Grade C evidence). [Chaikof et al., 2018b]
  - Studies show equivalent wall stress in saccular aneurysms at much smaller sizes when compared to fusiform aneurysms. This has led to the notion that they have a higher rupture risk at smaller sizes.
  - A study published in Annals of Vascular Surgery in 2016 showed a significant portion of ruptures <55mm in size were saccular in nature. [Kristmundsson et al., 2016]
    - \* Specific size guidelines for repair are currently lacking because of their infrequent presentation.

**What are the options for repair, and how do you choose?** [Moore et al., 2019, Fairman, 2019]

- Two options: open repair and endovascular aortic aneurysm repair (EVAR).
  - When attempting to decide between the two, one must consider the patient's perioperative risk as well as the patient's anatomy, which will be reviewed further here.

- When reviewing the patient's risk for surgery, there are many tools to assist, which are outlined in the Society for Vascular Surgery's practice guidelines.
- The VSGNE or Vascular Study Group of New England developed a risk prediction model for mortality which can assist in your decision making. This is endorsed by both SVS and the Vascular Quality Initiative.[Chaikof et al., 2018a, Eslami et al., 2015]
  - This risk model looks at open vs endovascular repair and further delineates infrarenal vs suprarenal clamps
  - It includes aneurysm sizes with 6.5cm as the cut off.
  - It includes age above or below 75yo.
  - Gender and comorbidities are included like heart disease, cerebrovascular disease and COPD.
  - An important risk factor is also renal function which is delineated by creatinine at 1.5-2 or >2.
  - Each of these risk factors is assigned a point grading.
  - These points are added together and they place the patient on a spectrum of mortality risk. Depending on the amount of points accumulated, the risk is divided into low, medium, high or prohibitively high-risk groups
  - This is something that can help both the patient and physician in deciding on surgery and how to proceed.
- Recent studies have shown that decreased aerobic fitness and high frailty score predicted increased morbidity and mortality after open aneurysm repair.
- High-risk patients are defined by the following in the SVS guidelines:
  - Unstable angina or angina at rest
  - Congestive heart failure with EF < 25-30%
  - Serum creatinine level > 3 mg/dL
  - Pulmonary disease manifested by room air PaO<sub>2</sub> < 50 mmHg, elevated PCO<sub>2</sub>, or both.
- To help delineate a patient's risk, a preoperative workup is necessary. The SVS practice guidelines recommend the following: [Chaikof et al., 2018b]
  - Determine if the patient has an active cardiovascular condition. Coronary artery disease is responsible for at least 50% to 60% of perioperative and late deaths after operations on the abdominal aorta,

therefore, it is important for patients to undergo cardiac evaluation prior to surgery.

- \* Unstable angina, decompensated heart failure, severe valvular disease, significant arrhythmia -> Cardiology consultation (Level 1, Grade B)
- \* Significant clinical risk factors such as coronary artery disease, congestive heart failure, stroke, diabetes mellitus, and chronic kidney disease -> Stress test (Level 2, Grade B)
- \* Worsening dyspnea -> Echocardiogram (Level 1, Grade A)
- \* All patients undergoing EVAR or open repair require EKG
- \* In patients capable of moderate physical activities, such as climbing two flights of stairs or running a short distance (MET  $\geq 4$ ), there is no benefit in further testing.
- \* If coronary intervention is required, this takes precedence over aneurysm repair.
- History of COPD
  - \* Pulmonary function test with ABG (Level 2, Grade C)
  - \* Smoking cessation for at least 2 weeks prior (Level 1, Grade C)
  - \* Pulmonary bronchodilators at least 2 weeks before aneurysm repair (Level 2, Grade C)
- In patients who are deemed high risk, EVAR is the most attractive option in anatomically suitable patients
- Morbidity and mortality rates are lower for EVAR than open repair in the short term. This is illustrated in multiple studies.
  - The EVAR-1 trial, a randomized prospective UK study including 1082 patients, compared EVAR with open AAA repair in patients who were fit enough to undergo open surgical repair from 1999-2003. The 30-day mortality rate was reduced in the EVAR group (1.7% vs 4.7%), although secondary interventions were more common in the EVAR group (9.8% vs. 5.8%). [Greenhalgh, 2004]
  - The DREAM trial, a multicenter randomized trial from 2000-2003, compared open repair with EVAR in 345 patients with a reduction in operative mortality (4.7% vs 9.8%) with the majority of complications accounted for by pulmonary issues. [Prinssen et al., 2004]
- This early survival benefit with EVAR over open repair disappears by the third postoperative year.

- The Open vs Endovascular Repair (OVER) trial included 881 patients from 42 VA centers randomized to either EVAR or open repair. This demonstrated that perioperative mortality was improved in the EVAR group (0.5% vs 3.0%), yet no statistically significant difference was seen in mortality at 2 years (7.0% vs 9.8%). [Lederle et al., 2019]
- Late mortality seems to be higher in EVAR due to ruptures from endoleaks that do not occur in open repair. [Rajendran and May, 2017]
- Reviewing the anatomic criteria for traditional EVAR may rule out EVAR as an option in some patients. These criteria vary slightly depending on the particular device being used.
  - Neck
    - \* A neck length of at least 10-15mm from the renal arteries to the aneurysm start with a diameter of 18-32mm.
    - \* It is important that the neck is relatively free of thrombus or calcification to decrease the risk of endoleaks.
    - \* Infrarenal necks 4-15mm would require fenestrated endovascular repair. The only FDA approved device for fenestrated repairs is the Cook Z-Fen device.[Oderich et al., 2014b]
      - Z-fen requires diameter 19-31mm, angle <45deg angle to long axis of aneurysm, ipsilateral iliac fixation >30mm and 9-21mm in diam, contralateral iliac >30mm and 7-21mm diam. Multiple or early branching renal arteries can limit this repair as well. [Oderich et al., 2014a]
      - Parallel grafts for short necks (i.e. snorkel or chimney) require 20mm seal for good outcomes. [Donas et al., 2015]
  - Angulation
    - \* Neck angulation should be < 60 degrees for current devices
  - Access vessels
    - \* Access vessels must be adequate for delivery of the device depending on the sheath size required (6-8mm)
  - Aortic bifurcation
    - \* The aortic bifurcation must be >20mm in size to accommodate the graft opening to full caliber
  - Iliac landing zone
    - \* Adequate seal zone in the distal common iliac arteries of 10-15mm in length and diameter of 7.5-25mm.

- \* If covering the hypogastric arteries is necessary unilaterally to obtain a seal, you can embolize the hypogastric artery (to prevent retrograde flow) and extend the graft into the external iliac artery.
- \* If this is an issue bilaterally, an iliac branch device can assist in maintaining perfusion into the hypogastric arteries.

#### Special considerations

- Aortocaval fistula - sometimes aneurysms can be complicated by fistula with the IVC. This can occur with primary fistula, rupture and after repair. This is rare, 1% in elective AAA and 6% in rupture.[Brightwell et al., 2013, Schmidt et al., 1994] High risk of persistent endoleak due to ongoing flow into IVC. Open repair often best.[Orion et al., 2016]

#### 4.3.1 EVAR

**Can you briefly go over the steps of an EVAR?** [Moore et al., 2019]

- EVAR now accounts for approximately 70-80% of elective abdominal aortic aneurysm repairs and 65% of iliac aneurysm repairs in the United States and many other countries.
- Performed in the operating room or IR suite with a fixed or portable C-arm
- Anesthesia
  - Regional block, local anesthesia or general anesthesia depending on surgeon preference and patient risk
- Groin access and short sheath placement
  - Percutaneous - Closure devices are introduced prior to inserting the large sheaths containing the stent-grafts
  - Cut-down
- Pigtail catheter is used to perform an aortogram of the abdominal aorta and iliac arteries
- The renal artery orifices are marked. If there is any concern about good visualization, IVUS (intravascular ultrasound) can be used to assist.
- Systemic heparin is given
- Bilateral femoral sheaths are exchanged over a stiff wire for the necessary sheaths required for the device size chosen.
  - Main trunk and ipsilateral limb sheath on one side
  - Contralateral limb sheath on the other side

- The main body is positioned in the proximal neck and a repeat angiogram is commonly performed to confirm the positioning of the device at the desired level just below the lowest renal artery. It is best to position the main body so that the gate is directed at the simplest angle to cannulate.
- The main body is deployed to the point where the gate is opened
- Contralateral limb gate cannulation is performed using a wire and directional catheter.
- Once in the gate, a pigtail catheter is formed within the main body and must be able to spin freely 360 degrees to confirm placement within the endograft
- The contralateral limb is introduced and deployed taking care to preserve flow to the internal iliac artery.
- The remainder of the main body is deployed and iliac extensions deployed if required.
- The stent graft is ballooned at the neck, within the gate, at the bifurcation, and distal iliac seal zones.
- An aortogram, usually multiple in different views, is performed to exclude any endoleaks.
- The sheaths are removed, and the groin sites are closed using Perclose devices if performed percutaneously, or primary repair if open cutdown performed.
- Check pedal pulses at the end of the case to ensure no thromboembolic events or femoral artery access injuries have occurred. If there is concern, an ultrasound duplex can be performed intraoperatively.

**You mentioned endoleaks, can you discuss the complications specific to EVAR and the management? [Moore et al., 2019]**

- 15 year follow up from EVAR-1 shows inferior late survival and higher re-intervention in EVAR patients when compared with open repair. [Patel et al., 2016]
- Many of the cardiopulmonary complications inherent with open repair do not occur with EVAR as there is no aortic cross clamping.
  - In a study from Mayo clinic evaluating elective infrarenal AAA repairs from 1999 to 2001, Elkouri et al found that cardiac and pulmonary morbidity after EVAR was drastically reduced compared to open repair (11% vs 22% and 3% vs 16%, respectively). [Elkouri et al., 2004]
- Risk of ischemic colitis remains as the IMA is covered with EVAR. It is lower than with open repair but remains 1-2%.

- Renal insufficiency may occur secondary to contrast administration in a patient with underlying chronic kidney disease. Thromboembolic events may occur from thrombus-laden aortic necks with wire and device manipulation to the renal arteries as well.
- Iliac limb occlusion - can occur within hours of EVAR (3-5%). Risk factors included iliac artery tortuosity, calcified plaque burden or occlusive disease, external iliac artery dissection[Vacirca et al., 2019], inadequate outflow due to access site complication. Post operative CFA duplex will show damped waveforms.[Greenhalgh et al., 2010, Taudorf et al., 2014]
- Emergent conversion from EVAR to open are associated with significant increase in morbidity and mortality. Risk factors include aneurysm diameter, young age, female gender and nonwhite race.[Ultee et al., 2016a]

#### 4.3.1.1 Endoleaks

Defined as persistent blood flow within the aneurysm sac following EVAR.

##### 1. Type I

- A leak at the graft ends secondary to inadequate seal proximally (1a) or distally (1b)
- If identified intraoperatively, Type I endoleaks require attention with further balloon angioplasty, proximal or distal extension, or endoanchors.
  - Use of endoanchors in thrombus and calcium-laden aorta is not recommended.[Jordan et al., 2014] Outcomes are similar with or without suprarenal fixation.
- If seen in follow up surveillance, intervention is necessary.

##### 2. Type II

- Sac filling secondary to retrograde filling via a branch vessel off of the aneurysm sac such as a lumbar artery or the IMA
- If identified intraoperatively, this typically does not need to be addressed in the OR. Typically, type II endoleaks spontaneously thrombose and therefore can be observed.
- It is common to continue monitoring even if there is persistent flow as long as there is no aneurysm sac growth. Type II endoleaks persist in 5-25% of patients. Risk factors for persistent endoleak include anticoagulation[Bobadilla et al., 2010], patency/number/diameter of IMA/lumbar arteries[Ward et al., 2014], hypogastric coil embolization, distal graft extension, absence of COPD, age >80yrs, and graft type. Risk of rupture is 1.5-3%. [Lo and Schermerhorn, 2016a, Abu-

luggage et al., 2010, Timaran et al., 2004, Shalaby et al., 2016, Sarac et al., 2012, Jones et al., 2007]

- If the leak persists for > 6 months with sac enlargement >5mm, intervention is recommended. Several techniques exist to eliminate type II endoleaks, most frequently embolization. After multiple attempts at repair and if continued sac growth with impending Type I leak, then consider open conversion.[Kelso et al., 2009, Mohapatra et al., 2019]

### 3. Type III

- Separation of graft components
- Usually identified in follow-up surveillance and necessitates intervention.

### 4. Type IV

- Secondary to a porous graft which typically does not occur any longer as endograft material and devices have improved. If seen, no intervention is needed at the time, and they usually thrombose on their own.

### 5. Type V

- Increasing aneurysm sac size with no identifiable endoleak. Commonly referred to as endotension.
- Usually necessitates graft explantation and open repair or re-lining of the graft.

#### 4.3.2 Open Repair

Now we can move onto open repair. Describe an open infrarenal aneurysm repair. [Moore et al., 2019, Gary G Wind and R. James Valentine, 2013]

- After thorough preoperative evaluation and clearance, the patient is taken back to the operating room. An epidural may be placed preoperatively depending on institutional preference. The patient is intubated, and arterial and central venous catheters are placed. The abdomen is prepped from chest to bilateral thighs.
- A cell-saver should be available to optimize resuscitation during the procedure due to expected large amounts of blood loss. Balanced resuscitation to prevent coagulopathy is important with significant blood loss.
- Exposure
  - Trans-peritoneal or retro-peritoneal. First we will describe the most common approach: trans-peritoneal.

- Surgical steps
  1. Mid-line laparotomy, transverse or chevron-style incision
  2. A retractor system such as an Omni, Bookwalter or Balfour retractor is used to assist in exposure depending on physician preference.
  3. The transverse colon is retracted cephalad, and the small bowel is retracted to the patient's right to expose the aorta. The duodenum is mobilized and the ligament of Treitz is divided. The posterior peritoneum is opened along the anterior wall of the aorta.
  4. The aneurysm sac is now in view and careful dissection proximally for clamp site is achieved. Identification of the left renal vein crossing the aorta is key and can be divided if necessary.
  5. Identification of the renal arteries proximally is required if there is a plan for suprarenal clamping.
  6. Isolate bilateral common iliac arteries for distal clamp site. Use caution when dissecting the fibro-areolar tissue overlying the left common iliac artery as it contains nerves that control sexual function. Damage can result in retrograde ejaculation.
    - You can avoid nerve injury with mobilization of the sigmoid colon medially and identifying the iliac bifurcation distally, thus avoiding transecting the tissue overlying the left common iliac artery.
    - If the iliac arteries are severely calcified and pose risk for injury with clamping, intraluminal balloon catheters can be inserted for distal control instead.
    - Also, you must be cognizant of the location of the ureters crossing over the iliac bifurcation to prevent injury.
  7. After proximal and distal clamp sites have been identified, systemic heparin is administered by anesthesia.
  8. Clamp the distal vessels first to prevent distal embolization.
  9. Open the aneurysm sac in a longitudinal fashion toward the patient's right to avoid the IMA and clear the sac of thrombus. Extend proximally to normal aorta and then t off the incision on the aortic wall.
    - Some physicians prefer to transect the aortic wall as opposed to leaving the posterior wall intact for the anastomosis.
  10. Lumbar arteries on the posterior wall are ligated using figure-of-eight sutures.
    - Back-bleeding lumbar vessels can be the source of significant blood loss.
  11. Graft

- A tube graft or bifurcated graft depending on the patient's anatomy and aortic diameter is chosen. Dacron or PTFE grafts are most common, and the choice depends on physician preference. This is anastomosed proximally in a continuous fashion.
  - Once complete, the graft is flushed forward to flush out any thrombus. The graft is then clamped and the aortic clamp removed to test the anastomosis. Repair if needed.
  - The distal anastomosis is completed to the aorta or bilateral iliac arteries depending on extent of the aneurysm.
  - The graft is flushed forward prior to completion to remove any thrombus within the graft. The anastomosis is completed and clamps removed.
12. Hypotension may occur at this point from re-perfusion of the lower extremities and pelvis. Anesthesia should be notified that unclamping will occur soon prior to completion of the distal anastomosis to allow for fluid resuscitation in preparation.
- The graft can be slowly unclamped or partially clamped to assist with blood pressure management during this time. You can also place manual pressure on the iliac arteries or femoral arteries to slowly release flow and avoid significant hypotension.
13. Next, the IMA must be addressed. The IMA orifice is identified within the aneurysm sac.
- Chronically occluded or pulsatile back bleeding -> ligate.
  - Anything between occlusion and strong pulsatile back bleeding requires further evaluation. This should be performed at the end of the case after the internal iliacs have been reperfused. Methods to measure perfusion:
    - \* Place vessel loops or micro bulldog on IMA and assess the sigmoid colon. If there is a poor doppler signal on the antimesenteric border of the sigmoid colon, the IMA should be reimplanted.
    - \* Insert blunt-tip needle through the IMA orifice and pull vessel loop around needle to secure and connect to a transducer. Pressure less than 35 mmHg requires reimplantation. [Hoballah, 2021]
  - The Carrel patch technique involves excising a circular button of the aortic wall around the IMA and anastomosing it to the graft wall.

- Newer studies have shown that IMA reimplantation does not eliminate the risk of ischemic colitis after open AAA repair. In a study out of George Washington University in DC published in JVS in 2019, there was still significant risk of ischemic colitis rates with IMA reimplantation. [Lee et al., 2019]
    - \* Using NSQIP data collected prospectively and studied retrospectively
    - \* Out of 2397 patients undergoing AAA from 2012-2015, 135 patients (5.6%) had ischemic colitis.
    - \* 672 patients were evaluated further after exclusion criteria applied (suprarenal clamp, emergent or ruptured, occluded mesenteric vessels)
    - \* Of these, 637 patients had IMA ligation, 35 had IMA reimplantation
    - \* Reimplantation was associated with - More frequent return to the OR (20% vs 7.2%), Higher rates of wound complications (17.1% vs 3%), Higher rates of ischemic colitis (8.6% vs 2.4%)
  - Difficult to interpret impact of revascularization of IMA on ischemic colitis rates, due to selection bias, but should be noted that patients who require revascularization still may experience colon ischemia.
14. To finish, the aneurysm sac is then closed over the graft to protect the viscera, and the retro-peritoneum is reapproximated. Occasionally, a vascularized omental pedicle flap may be used to separate the graft from the duodenum to prevent an aorto-enteric fistula if the peritoneum cannot be closed securely.

- Steps for the retro-peritoneal approach:
  - Positioned semi-lateral with the left side up with bilateral groins exposed for femoral artery access. This is done in a lazy lateral position where the patient's upper body is near complete lateral but the hips are rotated to the patient's left in attempt to keep both groins in the field in case they need to be accessed.
  - An oblique incision extends from the left 11th intercostal space or tip of the 12th rib to the edge of the rectus abdominus muscle, through the external and internal oblique muscles, transversalis fascia until you are just superficial to the peritoneum. Using blunt finger dissection, the peritoneum is dissected from the abdominal wall posteriorly over the psoas muscle until the aorta is reached.

- \* Benefits include less postoperative ileus, less intraoperative hypothermia, lower IV fluid requirements, and less post-op respiratory compromise.
- \* A disadvantage is the difficulty addressing the right iliac artery from this approach.

#### 4.3.2.1 Complications

**What are some of the complications with open aortic aneurysm repair?** [Moore et al., 2019]

- Recent studies have demonstrated equivalent outcomes in younger healthy patients undergoing endovascular and open repair. [Liang et al., 2018]
- Myocardial dysfunction which is usually secondary to cardiac ischemia or hemorrhage.
- Abdominal compartment syndrome secondary to coagulopathic bleeding postoperatively or third spacing of fluids can cause abdominal compartment syndrome requiring emergent laparotomy.[Cheatham et al., 2007, Mehta et al., 2005, Rasmussen and Hallett, 1997]
  - Signs include unexplained oliguria, difficulty maintaining adequate ventilation–elevated peak inspiratory pressure, and hypotension with significant abdominal distension is concerning for abdominal compartment syndrome. A sustained bladder pressure > 25 mmHg with associated organ dysfunction (elevated peak airway pressures, new onset acute renal failure) is indicative of abdominal compartment syndrome.
  - Abdominal compartment syndrome can still occur after EVAR during an aortic rupture in up to 10-20% of patients, therefore, one must keep a heightened suspicion for this in the post-operative period.[Veith et al., 2009]
  - It is important to note that a patient with a soft abdominal exam can still have abdominal compartment syndrome particularly with an enlarged body habitus.
  - Laparotomy may initially worsen hypotension and hemodynamic instability due to reperfusion injury.
- Renal failure can occur due to suprarenal aortic clamping, atheromatous embolization or hypotension causing acute tubular necrosis (ATN).
- Postoperative ileus is common. Duodenal obstruction from dissection of the ligament of Treitz can mimic a gastric outlet obstruction.
- Ischemic colitis of the left colon and rectum is the most serious gastrointestinal complication.[Moghadamyeghaneh et al., 2016]

- Incidence ranges from 0.2 - 10%, 3-4x more common after operations for occlusive disease than aneurysmal disease.
- It is important to study the collateral pathways on the preoperative CT scan and the patient's history to assist in surgical decisions regarding IMA reimplantation including:
  - \* Stenosis/occlusion of the SMA
  - \* Previous colectomy
  - \* IMA or Hypogastric artery occlusion
- Earliest manifestation is postoperative diarrhea, especially bloody diarrhea.
- Sigmoidoscopy is needed for diagnosis.
  - \* Mild colon ischemia with patchy mucosal involvement should be treated with bowel rest, fluid resuscitation and antibiotics. Transmural necrosis requires emergent operation with colon resection. Patients can be left in discontinuity or an end colostomy performed depending on stability.
  - \* The mortality rate with colon ischemia after aneurysm surgery is about 25% but reaches over 50% if bowel resection is required.[Brewster et al., 1991] This is a very heavily tested topic for both general surgery and vascular surgery boards.
- Distal ischemia from embolization downstream can lodge in larger vessels or cause microembolization, colloquially known as “trash foot”.
- Infection is rare but can be associated with graft-enteric fistula which is another highly tested topic.[Fatima et al., 2013, Oderich et al., 2011, Smeds et al., 2016]

#### 4.3.2.2 Postoperative Surveillance

**What is the post-operative surveillance required for open and endovascular approach, and how do they differ? [Moore et al., 2019]**

- That is a great question because it highlights why open repair has continued to be so important, especially for young, healthy patients.
- Post-operative surveillance is necessary in the immediate post-operative period for open repair to evaluate incisions. Follow-up is only needed every 5-10 years, unless the patient becomes symptomatic.
  - Peri-anastomotic degeneration occurs in 0.5-10% of cases. Can be diagnosed with CT scan or ultrasound.

- In contrast, EVAR patients require a strict postoperative surveillance regimen to allow for detection of endoleaks, aneurysm sac expansion, stent fracture, limb kinking and material fatigue.
  - CT scans at 1-, 6- and 12-month intervals initially then annually are recommended which raises concerns related to cost, cumulative radiation exposure, and contrast administration.
  - Some physicians may elect to use ultrasound for surveillance with CTA prompted if an endoleak is identified or the sac is enlarged, particularly in patients with stable aneurysms.
  - The long-term follow-up is often inconsistent and a study of 19,962 Medicare beneficiaries undergoing EVAR from 2001 to 2008 showed that 50% of patients were lost to annual imaging follow-up at 5 years after surgery.[Schanzer et al., 2015]
- Some patients will elect for open repair to avoid frequent surveillance if they are a candidate for both, while other patients will select endovascular management to avoid the short-term effects like longer hospitalizations, post-operative pain, and longer recovery time to baseline functioning in open surgery.

#### 4.3.3 Ruptured Aneurysms

**Although elective repair is important, can you touch on the management of a ruptured AAA (RAAA) as our last topic of the session?**  
[Moore et al., 2019, Lindsay, 2019b]

- Ruptured AAAs have declined secondary to improved medical management, decreased rates of smoking and superior diagnostic imaging and surveillance.
- Traditionally, it has been taught that 50% of ruptured AAAs die in the field and of those remaining, 50% will die in the hospital. With time, the in-hospital mortality rate has decreased.
  - In one study out of Finland, of 712 patients with ruptured AAAs from 2003-2013, 52% died prior to arrival to the hospital. Of those that were offered surgery, 67% of patients were alive at 30 days indicating a mortality rate of 33%. [Laine et al., 2016]
- Diagnostic triad on presentation:
  - Pain, syncope and known or palpable AAA.
- When a ruptured AAA is suspected or diagnosed, permissive hypotension is key in the initial management before surgery.
  - Allowing systolic arterial pressures of 50-70 mmHg as long as the patient is mentating appropriately.

- Limits internal bleeding which further limits loss of platelets and clotting factors.
- Initial management involves many considerations like patient stability, patient's anatomy and the surgeon's experience with either open or endovascular repair.
- Due to the developments of endovascular techniques, it is ideal to have a CTA prior to the operating room to determine if the patient is a candidate for an EVAR.
- There are two options for expedient aortic control in an unstable patient with a ruptured aneurysm.
  - Open supraceliac aortic clamping
    - \* Achieved by retracting the stomach caudally, entering and dividing a portion of the gastrohepatic ligament, reaching under and medial to the caudate lobe, dividing the pars flaccida, and identifying the spine. The aorta lies to the patient's left of the spine and is bluntly dissected anteriorly and laterally for aortic clamp placement.
    - \* Another method of supraceliac exposure and control is to mobilize and reflect the left lobe of the liver, sweep the esophagus to the patient's left, divide the right crus of the diaphragm and bluntly dissect both sides of the aorta then apply the clamp.
    - \* A nasogastric tube can help identify the esophagus when placing this clamp to ensure the esophagus has been swept to the patient's left and protected.
    - \* The clamp should be moved down to the desired position for repair (supra or infrarenal depending on anatomy of the aneurysm neck) to decrease ischemia time to visceral vessels as soon as possible.
  - Percutaneous occlusive aortic balloon
    - \* Gain percutaneous access and place an occlusive aortic balloon for stabilization in the distal thoracic aorta. This will require a long support sheath, usually 12fr in size, to prevent distal migration of the occlusive balloon.
- EVAR has been used increasingly to treat ruptured AAAs and offers many theoretical advantages over open repair.
- Less invasive, eliminates risk of damage to periaortic and abdominal structures, decreases bleeding from surgical dissection, minimizes hypothermia and third space losses, and lessens the requirement for deep anesthesia.

- EVAR has been deemed superior to open repair for the treatment of RAAA in many studies.
  - In a study out of UVA published in JVS in August 2020, they looked at ruptures in the VQI database from 2003-2018. This resulted in 724 pairs of open and endovascular pairs after propensity matching. [Wang et al., 2020]
    - \* There was a clear advantage of endovascular compared to open repair in patient's with suitable anatomy.
    - \* Length of stay was decreased with 5 vs 10 days in open. 30 day mortality was much lower at 18% vs 32%. Major adverse events like MI, Renal failure, leg ischemia, mesenteric ischemia, respiratory complications were much lower in the EVAR group at 35% vs 68% in the open group.
    - \* All cause 1 year survival was much higher with EVAR at 73% vs 59% in the open group.
- Despite improved RAAA results with EVAR, conversion from EVAR to open AAA repair appears to have the most unfavorable outcomes in terms of mortality.
  - Conversions can be early or late and are due to access-related problems, errors in endograft deployment, graft migration, persistent endoleak, graft thrombosis, or infection.
  - In a study evaluating 32,164 patients from NSQIP with 300 conversions (7,188 standard open repairs and 24,676 EVARs), conversion to open repair was associated with a significantly higher 30-day mortality than standard open repair (10% vs 4.2%) and EVAR (10% vs 1.7%). In addition, conversion patients compared to standard open patients were more likely to undergo new dialysis (6.0% vs. 3.5%), cardiopulmonary resuscitation (5.3% vs. 1.9%), postoperative blood transfusion (42.3% vs. 31.6%), and have a myocardial infarction (5.0% vs. 2.2%). [Ultee et al., 2016b]
  - A more recent systematic review and meta-analysis found the elective late open conversion has comparable outcomes to primary open aneurysm repair. 36% are able to complete with infrarenal clamp alone. Highest complication rate in urgent setting (10x higher mortality). [Goudketing et al., 2019]

## 4.4 Iliac/Peripheral Aneurysms

### 4.4.1 Demographics

CIAA aneurysm defined as >1.5cm.

#### 4.4.2 Presentation

Often found incidentally, but carries risk of rupture and distal embolization.

- Distal embolization is rare, but when it occurs patient should undergo embolectomy and treatment of aneurysm thought to be the embolic source.[Bacharach and Slovut, 2008, Ferreira et al., 2010, Nachbur et al., 1991]

#### 4.4.3 Management

Treatment threshold for CIAA is often accepted at 3.5cm.[Dix et al., 2005, Laine et al., 2017]

Treatment options include

- Open repair
  - If open repair involves aneurysm or occlusive disease of internal iliac arteries, should attempt to revascularize at least one IIA via a jump graft.[Krupski et al., 1998, Huang et al., 2008]
- Iliac branched device to preserve hypogastric. Minimum size requirements include:
  - CIA 17mm in diameter
  - EIA 6.5-25mm in diameter, 10mm seal zone
  - IIA 6.5-13.5mm in diameter, 10mm seal zone[Schneider et al., 2017]
- Extension of EVAR into external iliac and coil of internal iliac artery
  - If internal iliac arteries also aneurysmal, ensure that outflow vessels are all coiled. Posterior branch gives rise to the superior gluteal artery, which is high risk for retrograde filling.[Ryer et al., 2012]
  - Coil embolization can result in ipsilateral buttock claudication in 12-22% of patients. Treatment is with medical therapy and walking regimen, resolution usually occurs at 6 months.[Papazoglou et al., 2012, Stokmans et al., 2013]
  - If the IMA is chronically occluded, then coverage of the IIA may compromise colonic blood flow and increase risk of ischemia.[Angilettta et al., 2011, Karch et al., 2000]

# **Chapter 5**

## **Lower Extremity**

**UNDER CONSTRUCTION - This Chapter is Currently in DRAFT mode**

This chapter is lengthy and covers all vascular disease of the lower extremity, progressing in sub-chapters through peripheral arterial occlusive disease, critical limb ischaemia, acute limb ischaemia, compartment syndrome, lower limb amputation, and some of the more rare non-atheromatous conditions.

### **5.1 Pathophysiology of Peripheral Arterial Occlusive Disease**

Accompanying Episode: Peripheral Arterial Occlusive Disease (Part 1): Nedal Katib and Danielle Bajakian

#### **What is Peripheral Arterial Disease (PAD)?**

Peripheral Arterial Disease encompasses extremity arterial disease but generally is used to describe lower limb arterial occlusive disease. Peripheral Artery Occlusive Disease (PAOD) is a more specific term and this encompasses atherosclerotic disease of the lower limb arteries.

The disease has a stepwise spectrum of presentations from asymptomatic disease to intermittent claudication and finally chronic limb threatening ischemia (CLTI) formerly known as Critical Limb Ischemia (CLI) [Aboyans et al., 2018]

#### **What is the underlying pathophysiology of PAOD?**

The understanding of the underlying pathophysiology which results in occlusive arterial disease of the lower limb has evolved over the last 50 years. Atherosclerosis remains the main pathological process resulting in occlusive disease with smoking being the primary modifiable risk factor.

Although the rising incidence of diabetes has created a second population of patients with a significant PAOD burden.

Atherosclerosis, in summary, begins with an injury to the intimal lining of the arterial wall, which can result from smoking, hypertension or advanced age and ultimately a chronic inflammatory reaction resulting in plaque build up and calcification that may result in progressive stenosis and occlusion or plaque rupture with acute occlusion.

### **What are the risk factors for atherosclerosis?**

Modifiable:

#### **1. *Smoking***

- The **most significant modifiable risk factor** for developing peripheral arterial disease
- Causes Endothelial dysfunction by reducing nitric oxide and triggering reactive-oxygen species [United States Surgeon General, 2014]
- Causes a prothrombotic environment by causing an increase in thromboxane A2 and decreasing Prostacyclin thus overall resulting in an increased prothrombotic environment for platelets.
- Smoking has a stronger association with Intermittent claudication than with Coronary Artery Disease! [Gordon and Kannel, 1972]

#### **2. *Diabetes , Metabolic Syndrome and Insulin Resistance***

- Diabetes Mellitus, after smoking, is the most significant modifiable risk factor for developing peripheral arterial disease. Both insulin resistance and hyperinsulinemia are independent risk factors for developing peripheral arterial disease.
- The Odds Risk for developing PAD in patients with DM ranges from 1.89 to 4.05.
- An increase in HbA1C by 1% correlates with a 28% increase risk of developing PAD. [Adler et al., 2002]

#### **3. *Hypertension***

- The **most common** cardiovascular risk factor worldwide.
- The Incidence of PAD increases to 2.5-fold in patients with Hypertension. [Kannel and McGee, 1985]

#### **4. *Dyslipidemia***

- A strong association has long been identified as a risk factor for cardiovascular disease.
- 25% cardiovascular event reduction for each 39 mg/dL (1mmol/L) reduction in LDL. [Group, 2007]

## *5.1. PATHOPHYSIOLOGY OF PERIPHERAL ARTERIAL OCCLUSIVE DISEASE*87

Non Modifiable:

### **1. Age**

- Age is identified as a risk factor for PAD regardless of gender.
- Prevalence of PAD increases with age: 15% > 70 years of age.

### **2. Gender**

- The Framingham Study has found that the risk of developing PAD is doubled in men.

### **3. Ethnicity**

- The MESA study showed a higher prevalence of PAD (ABPI <0.9) in African Americans compared to Whites. 7.2% versus 3.6%. [Bild, 2002]
- Cross Sectional analysis 6653 subjects all with ABPI assessment revealed a prevalence of PAD (<0.9) of 4%. Non-Hispanic Whites: 3.6%, Asian: 2%, African American: 7.2% and Hispanic: 2.4%. (p<0.01) [Allison et al., 2006]

**What are the some of the major population-based trials looking at the natural history?**

1. The Framingham Heart Study: The original Cohort from the town of Framingham, n=5183 patients followed over time for over 30 years. There have been multiple subsequent recruited populations since. The majority of information we have about risk factors related to cardiovascular health comes from this study. [Mahmood et al., 2014]
2. The Rotterdam Study: 1990, Longitudinal Study, >7000 participants.
3. CVHS – 1989-1999 Longitudinal Study : n>5000 Multicentre Study.
4. MESA – Cross Sectional analysis 6653 subjects all with ABPI assessment revealed a prevalence of PAD (<0.9) of 4%. Non-Hispanic Whites: 3.6%, Asian: 2%, African American: 7.2% and Hispanic: 2.4%. (p<0.01) [Allison et al., 2006]
5. The Edinburgh Study: The EAS began as a cross sectional study of 1592 men and women in Edinburgh with the goal of examining the frequency of risk factors for peripheral arterial disease. The subjects were followed over 20 years. [Fowkes et al., 1991]

**How does Diabetes confound the clinical picture of PAOD?**

- Increasing Incidence of Diabetes world-wide. [Boulton et al., 2005]
  - 2.8% in 2000, 4.4% in 2030
  - 25% of patients with diabetes develop a DFU at some stage in their lives

- Limb Loss every 20 seconds world-wide to Diabetes

**What's the pathophysiology of Diabetes and PAOD** [Armstrong et al., 2012]

- Sensory Neuropathy, Motor Neuropathy and Autonomic Neuropathy
- Structural and Gait abnormalities
- Arterial disease
  - Large Vessel
  - Small Vessel
  - Both
- Foot infection[Bandyk, 2018, KAYSSI et al., 2019, Lepäntalo et al., 2011]

Given this diverse and confounding pathology the normal progressive history of PAD is somewhat different. What's most concerning is the neuropathy resulting in initial presentation being ulceration. This results in a lack of a 'safety net' where presenting with progressive claudication allows for a period of detection, management and risk factor modification before they develop tissue loss and are at risk of amputation.

Since 2014 and the publication of WIfI a lot has changed in the way we view PAD leading up to last years new Global Vascular Guidelines on CLTI, which as a term has replaced CLI. [Mills et al., 2014b, Conte et al., 2019]

## 5.2 Intermittent Claudication

**What is Intermittent Claudication and the classic patient presentation?**

The original population studies we mentioned determined the epidemiology and natural history of Intermittent Claudication based on historically validated and widely accepted questionnaires, namely the Rose [Rose, 1962] (which later was adopted by the WHO) and subsequently the Edinburgh questionnaire [Lend and Fowkes, 1992].

All questionnaires are based on a number of key diagnostic clinical factors that define claudication, they are:

- Onset
- Calf involvement
- Reproducibility
- Relief with Rest
- Not occurring at Rest

The progression historically graded by Fontaine (1954) [Fontaine et al., 1954] followed by the Rutherford Grading System (1986, Revised 1997) [Rutherford et al., 1997]

Rutherford et al. Ad Hoc Committee on Reporting Standards, SVS/North American Chapter ISCVS:

| Grade | Category | Clinical Description  |
|-------|----------|---|
| 0     | 0        | Asymptomatic -no haemodynamic significant occlusive disease |
| I     | 1        | Mild Claudication   |
| I     | 2        | Moderate Claudication                                       |
| I     | 3        | Severe Claudication   |
| II    | 4        | Ischaemic Rest Pain   |
| III   | 5        | Minor Tissue Loss   |
| III   | 6        | Major Tissue Loss   |

Leriche syndrome - Triad of claudication (buttock, thigh, and calf), impotence and decreased pulses signifies aortoiliac occlusive disease. [Frederick et al., 2010, Leriche and Morel, 1948, Setacci et al., 2012]

Differentiating neurogenic claudication from vasculogenic claudication can be difficult. Vasculogenic claudication is relieved by cessation of ambulation and mostly affects a unilateral calf. Neurogenic improves with postural changes, more often bilateral and affects the thighs. [Nadeau et al., 2013]

#### **What is involved in the work up of patients with PAOD/Intermittent Claudication?**

##### **5.2.1 History / Clinical Examination**

SVS Guidelines:

“We recommend using ABI as the first-line non-invasive test to establish a diagnosis of PAD in individuals with symptoms or signs suggestive of disease. When the ABI is borderline or normal ( $>0.9$ ) and symptoms of claudication are suggestive, we recommend an exercise ABI.”

*Grade 1 Level of Evidence A*

- ABPI
- Exercise ABPI
- Ultrasound

**What is an ABPI and how is it measured?**

The AHA came out with guidelines on how to perform an ABI and to standardise the method to allow for more comparable results from studies.

Divide the higher of the PT or DP pressure by the higher of the right or left Brachial SBP (*Class 1 Level of Evidence A*) [Aboyans et al., 2012]

Sensitivity and Specificity both >95% (when ABPI cut off  $</=0.9$  – in detecting  $>/= 50\%$  stenosis) [Yao et al., 2005, Ouriel et al., 1982]

| ABPI      | Interpretation   |
|-----------|------------------|
| >1.4      | Non-compressible |
| >0.9-1.39 | Normal           |
| 0.5-0.9   | Mild to Mod PAD  |
| 0.0-0.5   | Severe PAD       |

### What is Exercise ABPI studies?

Constant Load Testing – (unlike the Graded Test – Bruce Protocol).

The mechanism for changes in ABPI are related to the fact that exercise increases SBP and decreases SVR and therefore will affect flow across a stenosis making it more physiologically relevant during exercise. [Nicolaï et al., 2009, Alqahtani et al., 2018]

Walking distance has been shown to correlate with level and severity of POAD. [Strandness and Sumner, 1975]

Patients with single level iliac disease may present with buttock/thigh claudication with preserved pulses. Exercise treadmill ABI particularly useful in determining the severity of disease in these patients. [Aboyans et al., 2010]

For more on exercise testing see 5.

### What is the ultrasound duplex criteria for defining PAOD?

| Stenosis Category | Peak Systolic Velocity | Velocity Ratio | Distal Artery Spectral Waveform       |
|-------------------|------------------------|----------------|---------------------------------------|
| Normal            | <150                   | <1.5           | Triphasic,<br>Normal PSV              |
| 30-49%            | 150-200                | 1.5-2          | Triphasic,<br>Normal PSV              |
| 50-75%            | 200-400                | 2-4            | Monophasic,<br>reduced PSV            |
| >75%              | >400                   | >4             | Damped,<br>monophasic,<br>reduced PSV |

| Stenosis Category | Peak Systolic Velocity                     | Velocity Ratio | Distal Artery Spectral Waveform |
|-------------------|--|----------------|---------------------------------|
| Occlusion         | No Flow – B<br>-mode,<br>Terminal<br>Thump | NA             | NA                              |

Adapted from Stone and Hass. Vascular Laboratory: Arterial Duplex Scanning. Rutherford's Vascular Surgery and Endovascular Therapy. 2019.[Stone and Hass, 2019]

#### What Guidelines are there pertaining to PAD Management.

SVS Guidelines (2015)

- The SVS published the SVS practice guidelines for atherosclerotic occlusive disease of the lower extremities:**Management of asymptomatic disease and claudication.** *Conte and Pomposelli et al. JVS 2015* [Conte et al., 2015b]

Other Guidelines:

- TASC 1 -2
- European Guidelines (2017)
- AHA Guidelines (last update 2016)

#### What is the initial management of Asymptomatic Patients with PAD?

- Smoking Cessation – Multidisciplinary comprehensive smoking cessation interventions – repeatedly until tobacco use has stopped (Grade A – 1)
- Intervention is not only not recommended, but invasive treatment is recommended against, in the absence of symptoms (Grade A -1)

#### How can we medically (non-invasively) manage Asymptomatic PAOD based on the SVS Guidelines?

- Anti-platelet Therapy
  - The Aspirin for Asymptomatic Atherosclerosis Trial – n=3350, aspirin versus placebo. 8 years follow up no difference in events [Fowkes, 2010] – therefore benefit unknown
- Statin Therapy
  - The Heart Protection Study [Group, 2007] - this study looked at Statins in patients with PAD but not completely asymptomatic, they had other risk factors such as diabetes, IHD, cerebral disease or hypertension. Without these risk factors Statin therapy benefit unsure.

- However, the AHA from the Framingham Study does recommend using Statins if 10-year risk based on risk calculators >7.5% (which would be positive if PAD present).
- Exercise and Limb Function
  - No clear evidence that physical therapy improves QoL
- Surveillance
  - No benefit from US surveillance, unclear benefit of ABPI surveillance.

### 5.2.2 Management

**How can we medically manage Intermittent Claudication based on the SVS Guidelines?**

- Smoking Cessation – Multidisciplinary comprehensive smoking cessation interventions – repeatedly until tobacco use has stopped (Grade A – 1)
- Dyslipidaemia: Statin Therapy Recommended – most recent evidence on lipid therapy has suggested focusing on reducing 10-year cardiovascular event risk rather than specifically reducing lipid levels. (Grade 1-A)
- Statin Therapy – Aspirin therapy (75-325mg daily) is recommended to reduce cardiovascular events in patients with PAD (Grade 1-Level A) [Collaboration, 2002]
  - There is evidence that Clopidogrel 75mg compared to Aspirin is better in event reduction (CAPRIE)[Committee, 1996b]– replacing Aspirin with Clopidogrel Grade 1-Level B)
- Diabetes Mellitus – Optimization of HbA1C < 7% (Grade 1-Level B)
- Hypertension – Indicated B-Blockers for hypertension (Grade 1-Level B) (there's no evidence that Beta Blockers worsens IC)
- Homocysteine – Recommendation against Folic Acid and Vit B12 ( Grade 2 – C)

**To improve Limb Function in patients with IC:**

- Cilostozol use – IC without CHF – 3-month Trial (Grade 2 - A)
  - If unable to tolerate Cilostozol – Pentoxifylline (400mg TDS) (Grade 2 – B)
  - Based on Meta-analysis 26 trials [Stevens et al., 2012]
- Exercise Therapy
  - First Line Therapy recommended SEP: minimum three times / week (30-60 min/session) for at least 12 weeks (Grade 1 Level A)

- Meta-analysis of 32 RCT's: Placebo versus exercise: Walking Time, Walking Ability, Pain Free Walking and maximum walking distance improves. BUT no difference in ABPI, Mortality or amputation. [Lane et al., 2017]
- Meta- Analysis of 14RCT's: SEP better than Non-Supervised Programs. [Hageman et al., 2018]

**What is the management for patients with Intermittent Claudication?**

- With appropriate medical management and risk modification, 25% of patients will improve, 50% will remain stable and 25% will progress and requiring intervention. [Conte et al., 2015a, Norgren et al., 2007]
- **Patient Selection for Intervention:**
  - 20-30% of patients with IC who adhere to risk factor modification will have progressive symptoms that will eventually be treated with intervention.
  - Patient selection should be based on QoL and functional impairment in an active person (loss of ability to perform occupation or that limits basic activities of daily living) rather than haemodynamic (ABPI or US) or anatomical disease progression/severity.
- **Always remember multifactorial causes of immobility – particularly in the elderly.**
  - SVS recommends that invasive therapy for IC have a >50% likelihood of sustained clinical improvement for at least 2 years.
- **Anatomical Selection:**
  - Aortoiliac Disease:
    - \* Previous TASC Classification has attempted to categorise anatomy of disease and subsequent recommendation of Endovascular versus open surgery. But as the authors of the SVS guidelines highlight, “improvements in technology and endovascular techniques have resulted in EVT replacing open surgical bypass as a primary treatment for both focal and advanced AIOD in many cases.”
    - \* The majority of evidence is non randomized and meta analyses of non-randomized series.
    - \* Endovascular procedures over open surgery for focal AIOD causing IC. (Grade 1 Evidence B)
    - \* Endovascular interventions as first line for CIA or EIZ occlusive disease-causing IC. (Grade 1 Level B)

- \* Hybrid recommended for Iliac disease involving CFA. (Grade 1 Level B)
- \* Direct Surgical reconstruction (bypass, endarterectomy) in patients with reasonable surgical risk and diffuse AIOD not amenable to endovascular approach, after one or more failed attempts at EVT, or combined occlusive and aneurysmal disease. (Grade 1 Evidence B)
- Infrainguinal Disease:
  - \* When you look at the historical data comparing all EVT together they are less durable than surgical bypass, especially when there's diffuse or long segments of occlusion/multilevel infra inguinal disease.
  - \* Most recommendations are based on low level evidence when comparing EVT versus Open Surgery
  - \* Focal + Not involving SFA origin = EVT (Grade 1 Level C)
  - \* SFA 5-15cm, self-expanding stent (with or without paclitaxel) (Grade 1 Level B) – NB: (This was in 2015 pre Katsanos Paper)
  - \* Recommend against infrapopliteal treatment for IC (Grade 1 Level C)
  - \* Initial Surgical Bypass (with vein: Grade 1 Level a): If
    - Diffuse FP disease
    - Small Calibre <5mm
    - Extensive calcification in SFA
    - Average or low operative risk (Grade 1 Level B)

## 5.3 Chronic Limb Threatening Ischemia

**03 Jan 2021: Neda Katib and Danielle Bajakian**

### 5.3.1 Etiology and Presentation

**Since Our previous discussion of PAD how does this pathology then progress clinically into the more advanced stages?**

The latter stages of both the Rutherford and Fontaine Classification systems highlight this progression, with the Rutherford classification of Stage 5 being specifically minor tissue loss with focal gangrene, and stage 6 as major tissue loss identified by speeding of gangrene beyond the Trans metatarsal level.

Rutherford et al. Ad Hoc Committee on Reporting Standards, SVS/North American Chapter ISCVS:

| Grade | Category | Clinical Description  |
|-------|----------|---|
| 0     | 0        | Asymptomatic -no haemodynamic significant occlusive disease |
| I     | 1        | Mild Claudication   |
| I     | 2        | Moderate Claudication                                       |
| I     | 3        | Severe Claudication   |
| II    | 4        | Ischaemic Rest Pain   |
| III   | 5        | Minor Tissue Loss   |
| III   | 6        | Major Tissue Loss   |

**What is Chronic Limb Threatening Ischemia (CLTI), sometimes previously call Critical Limb Ischemia (CLI)?**

In the last decade leading up to the recent Global Vascular Guidelines (GVG) published last year, the term (Chronic Limb threatening Ischaemia) CLTI has been gradually replacing CLI. The GVG mentions that their “promotion” of the term CLTI is partly due to terms such as “critical or severe limb ischemia” failing to “recognize the full spectrum and inter-relatedness of components beyond ischemia that contribute to major limb amputation...”

**What was the original definition and threshold for CLI and how can we make sure we elicit the right symptoms from the patient?**

John Cranley back in his publication in 1969 defined Ischemic Rest Pain as,

“...pain that occurs in the toes or in the area of the metatarsal heads. Occasionally...in the foot proximal to the metatarsal heads. Elevation of the limb above or at the horizontal position aggravates the pain and pendency...brings relief...” [Cranley, 1969]

Nocturnal Rest Pain: Worse due to horizontal positioning and systolic BP drop during sleep.

### 5.3.2 Evaluation

**What aspects of the clinical assessment is important?**

Clinical Assessment involves a full history (the differential mentioned above) and examination.

- Clinical Examination:
  - Berger's Test (Beuriger 1908) / AKA Ratschows Test (Max-Ratschow-Klinic) identifies when there is critical ischemia without necrosis yet or gangrene, and is characterised by pallor when the leg is elevated

above the level of the heart, which then turns red when hanging down over the edge of the bed. This redness is referred to as “Sunset appearance” and its due to abnormal autoregulation. Its been described that normally only a third of the capillary bed is open at any time but in a state of critical ischaemia because of the autoregulation being paralyzed a significantly higher portion of the capillaries open up.

- The ischaemic Angle: A refinement to Berger’s Test: The angle of elevation from the horizontal at which the Doppler Signal of the PT or DP disappears. This is also referred to as the ‘pole test’, whereby the foot is raised alongside a calibrated pole marked in mmHg.

- Tissue Loss:

- Gangrene Dry or Wet (infection)
- Level of tissue Loss
- Probing To Bone/ Exposed structures: Tendons, Soft Tissue, bone, Joint Capsule.
- Examination of an Ulcer (may have many aetiologies) – important not only to identify extent of disease but also to exclude other aetiologies:
  - \* Such as Venous, Mixed, infective, autoimmune, inflammatory, malignancy or trauma.

- Foot Infection - Signs of infection, erythema, rubor, cellulitis, tenderness or unexplained hyperglycemia in diabetic patients should prompt urgent referral.[Kalish and Hamdan, 2010]

**What is the natural history of CLTI and what do we know about its prognosis?**

Fortunately, only a small portion of patients with Intermittent Claudication will go on to develop rest pain or tissue loss. Its estimated that anywhere between 5% -29% of patients with PAD or IC go on to develop CLTI over 5 years.

However, those that do develop CLTI, have a high risk of limb loss (greater than 20% annual risk) and a high mortality (10-15% annual risk), the majority of terminal events being related to cardiovascular events. Limb loss or mortality may reach as high as 50% in 1 year. [Adam et al., 2005, Norgren et al., 2007]

CLTI measurements most predictive of non-healing are ankle pressure <50mmHg, ABI <0.4, TcPO<sub>2</sub> <20mmHg, and TP <20mmHg.[Gerhard-Herman et al., 2017, Wickström et al., 2017]

**What has changed in the last few decades?**

Prevalence of smokers-Ex and Current (decrease) and patients with diabetes(increase).

Estimated 4.4% of the world will have diabetes by 2030. 25% of patients with Diabetes will develop a foot ulcer.

**Briefly outline the underlying pathophysiology associated with peripheral neuropathy in Diabetes?**

The loss of the basic nociceptive mechanisms in the foot amongst diabetics, presents as a loss of protective sensation (LOPS).

Neuropathy can be divided into three types:

1. Sensory: “stocking-glove” distribution
2. Motor: Intrinsic muscle wasting – resulting in deformities
3. Autonomic: Sympathetic nervous system pathology

Along with Neuropathy, Diabetic Patients also have Structural deformities and Gait disturbances in addition to Angiopathy or small vessel disease.

There is often an overlap in the pathological processes in patients with CLTI or CLI.

Diabetic foot ulcers have a very high rate of recurrence with 40% of patients developing a new ulcer within 12 months of healing a previous ulcer. [Armstrong et al., 2017]

**What is the WIFI Classification? [Mills et al., 2014b]**

Interestingly in the original article by Bob Rutherford regarding Diabetes and PAD:

- “It was generally agreed that diabetic patients who have a varied clinical picture of neuropathy, ischemia and sepsis make the definition even more difficult and it is desirable that these patients be excluded...diabetic patients should be clearly defined as a separate category or should be clearly defined as a separate category.”

Since then, the SVS, while acknowledging that we can no longer exclude these patients and treat them separately given the overlap, have decided that a new classification system is necessary, as one of the key authors (Joseph Mills) states:

- “We classify things into groups to differentiate, remember and compare, observe and predict their behaviour over time.” –Joseph Mills

WIFI stands for: Wound, Ischemia and foot Infection. Most of the existing Vascular and non-Vascular classification systems don’t include all three components or fail to stratify the degree of ischemia and presence of gangrene.

Principles of WIFI:

1. Grades, Classes and Stages – Each of the three categories (WIFI) have Grades 0,1,2,3: Resulting in 64 Classes.

2. Delphi Consensus – Clinical Stages 1 (Very Low), 2 (Low), 3 (Moderate), 4 (High Risk/Benefit).
  1. What is the one-year risk of amputation with medical therapy alone?
  2. What is the potential benefit from successful revascularization?
  3. Analogous to TNM Staging

“It is intended to be an iterative process with the goal of more precisely stratifying patients according to their initial disease burden, analogous to TNM cancer staging, but not to dictate therapy.”

**What about a differential diagnosis or other causes of similar pain as rest pain?**

Acute Lower Limb ischemia is a different clinical picture, but there may be some overlap with Acute or Chronic disease such as in the case of in situ thrombosis in the lower limb arterial system.

Other causes of ischemic pain include:

- Buerger's Disease, or Thromboangiitis obliterans - for more see 3.3.4
- Scleroderma
- Fibromuscular dysplasia
- Popliteal Artery Entrapment
- Cystic Adventitial Disease
- Persistent Sciatic Artery Disease

**What is the Rutherford Acute Ischemia Grading System?**

Although Acute Ischemia is very different from chronic ischemia, patients with progressive chronic PAD can develop an acute picture whether from embolism or in-situ thrombosis secondary to plaque rupture. See 3.2.1 for more.

**What are the recent CLTI Guidelines?**

In 2019 the SVS, the EVS and the World federation of Vascular Societies (WFVS) joined forces to put together the structure and funding of the Global Vascular Guidelines Initiative (GVI). Importantly all sponsorship was directly from the societies and any direct industry sponsorship or external sources were excluded. They put together a steering committee responsible for recruiting a large and diverse writing group and outlined the scope and developed the section briefs of the guideline.

They determined that:

“The term “critical limb ischemia” (CLI) is outdated and fails to encompass the full spectrum of patients who are evaluated and treated for limb-threatening ischemia in modern practice.”

CLTI was promoted as the term of choice and was defined by the target population

The target population were:

1. Ischemic Rest Pain with confirmatory hemodynamic studies
2. Diabetic Foot Ulcer or any lower limb ulceration present for at least 2 weeks
3. Gangrene involving any portion of the lower limb or foot

Exclusion from the population:

1. Purely venous ulcers
2. Acute Limb Ischemia/acute trash foot/ischemia due to emboli
3. Acute Trauma or mangled extremity
4. Wounds secondary to non-atherosclerotic conditions

Methodology of the guidelines utilised the structure of GRADE.

They Highlighted particular important sections in the evaluation and management of patients with CLTI: namely Patient Risk stratification, Limb Assessment and Severity of Limb Threat and the development of a specific evidence-based revascularisation guideline in CLTI.

One think to notice (which the authors also highlight in the text body) is compared to most guidelines, unfortunately in this area, particularly when it comes to revascularisation, the level of evidence is generally LOW. Again, highlighting the importance of these guidelines in developing a standard approach and appropriately stratifying patients in not only management but ongoing research.

#### **What clinical evaluation is necessary for the patient with CLTI?**

In addition to the History and Examination, and the WiFi assessment mentioned above. For patients with diabetes and an ulcer a full assessment of neuropathy and a “probe to bone” test for any open ulcers is recommended as part of good practice.

We mentioned in our previous discussion the non-invasive methods of assessment for these patients. One additional point to make is the importance of TP and TBI in these patients.

It's been shown that, healing of an ulcer or tissue loss is unlikely if a patient's toe pressures are less than 55mmHg. And Toe Pressures have been validated in multiple studies to correlate with Amputation free survival and wound healing: Amputation Free Survival TP <30mmHg 2.13 HR (1.52-2.98). [Wickström et al., 2017, Hicks et al., 2018]

Patients with ESRD or DM develop medial calcification and often have elevated ABI (>1.3) - which is associated with an elevated risk of cardiovascular mortality.

Toe pressures are particularly important in this scenario. [Resnick et al., 2004, Vitti et al., 1994]

Non-invasive assessment for wound healing, tcPO<sub>2</sub> greater than 40mmHg has the greatest correlation with amputation stump healing.[Malone et al., 1987]

### 5.3.3 Management

**What are the recommendations for patients with CLTI when it comes to Medical Therapy and Risk Factor Modification?**

- Treat all patients with CLTI with an antiplatelet agent (**Grade 1 Level A**)
- Consider Clopidogrel as the single agent (**Grade 2 Level B**) – CAPRIE[Committee, 1996a]
- High-intensity statin therapy to reduce all-cause and cardiovascular mortality - Atorvastatin 80mg or Rosuvastatin 40mg (**Grade 1 Level A**) [Arya et al., 2018, 201]
- Control Hypertension to BP target <140mm Hg systolic and <90mm Hg diastolic in patients with CLTI (**Grade 1 Level B**)
- Offer Smoking Cessation interventions and ask all smokers or former smokers about status of tobacco use every visit (**Grade 1 Level A**)
- Diabetic foot wounds with signs of infection, erythema, swelling, pain and foul smelling drainage should be treated with IV abx, plain radiography, ESR, CRP and cultures. Signs of systemic sepsis, such as fevers, tachycardia or shock, such as hypotension, should warrant urgent debridement and drainage, regardless of vascular status.

**What imaging assessment is required?**

The CLTI Guidelines outlines an algorithm of attaining Arterial Anatomy Imaging. Starting with US and then depending on the information required, CTA, MRA or eventually digital subtraction angiography. They emphasize the importance of obtaining good quality imaging to appropriately stage and be able to compare the level and degree of disease.

For wounds with concern for underlying osteomyelitis - initial workup is with plain radiography, which can identify soft tissue emphysema, evidence of osteomyelitis or presence of a foreign body. High suspicion of early osteomyelitis with negative x-ray may warrant an MRI to detect.[Giurato et al., 2017]

**What is the Global Limb Anatomic Staging System (GLASS)?** [Conte et al., 2019]

Because the existing arterial anatomical staging of disease is very vague, and are “lesion focussed” and not all encompassing (beyond the concept of ‘in-line pulsatile flow to the foot’), GLASS attempts to incorporate all aspects in its

staging to improve vascular care and evidence-based revascularisation (EBR) outcomes.

GLASS incorporates two novel and important concepts: The Target Arterial Path (TAP) and the estimated Limb-Based Patency (LBP) and it's a grading system based on anatomical and subjective assessment of calcification.

GLASS focusses on Infrainguinal disease, with the Aorto Iliac (AI) segment considered the inflow disease which includes the Common Femoral Artery and the Profunda Artery. Therefore, the GLASS grades assume the inflow vessels are treated and adequately ‘dealt with’.

Infrainguinal disease assessment for Femoropoliteal (FP) and Infrapopliteal (IP) is based on length of disease and the extent of CTO’s. The FP and IP GLASS Grades are then combined into Stages 1-3.

The calcification scale is a dichotomous subjective assessment of the degree of calcification and if there is >50% circumference of calcification, diffuse or bulky calcification or “coral reef” plaques, then there is an increase in the within-segment grade by one numerical value.

There is also mention of the Inframaleolar (IM) degree of disease (PO, P1-absent arch, P2-no target artery crossing into foot) which is not included in the GLASS staging given little evidence on the outcomes this difference makes on overall patency and limb salvage.

Once the GRADES (0-4) of FP and IP disease are determined then staging (1-3) can be performed based on the matrix or grid that is provided. Staging then allows for estimated Peripheral endo-Vascular Intervention outcomes (PVI) to be predicted, Immediate Technical Failure (ITF - <10% or < 20% or > 20%) and 1-year Limb Based Patency (LBP - >70%, 50-75% or <50%).

#### **What is the Target Arterial Path (TAP)?**

“The selected continuous route of in-line flow from groin to ankle. The TAP typically involves the least diseased IP artery but may be angiosome based.”

#### **What revascularisation management strategies exist for CLTI?**

The mainstay of management for patients with CLI or CLTI has always been based on the fundamental principle of limb salvage and given the high risk of limb loss in these patients there’s been a low threshold to revascularize these patients if they have occlusive disease that is treatable. But strategy has varied significantly.

The CLTI Guidelines provide an approach to dealing with this complex condition on planning three aspects to each case:

1. Patient Risk Estimation
2. Limb Staging
3. Anatomic Pattern of Disease

### **What is involved with the Patient Risk Estimation?**

Good Practice Statements (Recommendations section 6)

“Refer all patients with suspected CLTI to a vascular specialist for consideration of limb salvage, unless major amputation is considered medically urgent.”

“Offer primary amputation or palliation to patients with limited life expectancy, poor functional status (e.g. non ambulatory), or an unsalvageable limb after shared decision-making.”

Recommendation 6.3:

- Estimate periprocedural risk and life expectancy in patients with CLTI who are candidates for revascularization. **Grade 1 (Strong) Level of Evidence C (Low)**
- Average Surgical Risk: \<5% operative mortality and 2-year survival >50%
- Severe Surgical Risk: \>/= 5% operative mortality and 2-year survival </=50%

Understanding disparities is important when interpreting risk for amputation. Observational studies have found certain populations to be at higher risk of amputation regardless of disease severity, these include African Americans, lowest median income, medicaid insurance, uninsured, or those from regions with less access to vascular surgeons.[Hughes et al., 2019, Ho et al., 2005, Eslami et al., 2007]

### **What is involved and recommended with the Limb Staging and recommendation for Management?**

Use an integrated threatened limb classification system (such as WiFi) to stage all CLTI patients who are candidates for limb salvage. **Grade 1 (Strong) Level of Evidence C (Low)**

Perform urgent surgical drainage and debridement (including minor amputation if needed) and commence antibiotic treatment in all patients with suspected CLTI who present with deep space foot infection or wet gangrene. (Good Practice Statement)

Offer Revascularisation to all “average surgical risk patients” (\<5% operative mortality and 2-year survival >50%) with advanced limb-threatening conditions (e.g. WiFi stage 4) and significant perfusion deficits (e.g. ischemia grades 2 and 3). Particularly if they have good saphenous vein. **Grade 1 (Strong) Level of Evidence C (Low)** [Adam et al., 2005, Norgren et al., 2007]

### **What is involved in the Planning of the Anatomic pattern of disease and its effects of revascularisation strategy?**

The overall pattern of arterial occlusive disease is a dominant factor in guiding type of revascularisation and timing of such.

**Do all patients require direct in-line flow to the foot as a primary technical outcome with revascularisation?**

One important patient population to identify that do not necessarily require direct in line flow are those with rest pain “for which correction of inflow disease alone or treatment of FP disease even without continuous tibial runoff to the foot may provide relief of symptoms. This may also be the case in patients presenting with minor degrees of tissue loss.”

**What are some essential Key Factors to consider before deciding Open versus Endovascular according to the CLTI guidelines?**

1. The “availability of and quality of autogenous vein conduit”<sup>7</sup>
2. Patient overall risk (as mentioned above) and Limb Staging
3. The Target EndoVascular Intervention (TVI) outcomes

**What evidence do we have for deciding between Endo and Open? What is the BASIL trial?**

The evidence is largely retrospective or non-controlled, industry sponsored and in overall quality poor. BASIL (Bypass versus Angioplasty in Severe Ischemia of the Leg) remains the only multicentre RCT (BASIL -2 and 3 underway) directly comparing an endo versus open strategy in CLTI and Infra-inguinal occlusive disease.

BASIL compared POBA and Bypass across multiple centres (27 centres, n=452, 1999-2004) in the UK. Primary endpoint was amputation-free survival. [Bradbury et al., 2005]

**Major Findings:**

1. At 6-months follow up: no difference in AFS.
2. Intention-To-Treat Analysis of overall follow up showed no significant difference in AFS and overall survival.
3. Among patients who survived >2 years, overall survival was better for those treated with Bypass as a first approach
4. Analysis to treat:
  1. Prosthetic Bypass Patients did very poorly (even compared to POBA)
  2. Patients who had bypass after failed POBA had significantly worse AFS compared to those treated with a bypass as initial treatment

**Criticism:**

1. Majority had POBA alone (not currently best endovascular option)
2. 25 % of Open Bypass were Prosthetic

3. The Technology and Technical Skill with growing operator experience in Endovascular has improved.

For isolated tibial disease, first line endovascular treatment of choice is trans-luminal or subintimal angioplasty. Atherectomy, stenting, and drug coated balloons are often used, but should not be considered first line at this time, but this data is rapidly evolving. Patency is often poor but limb salvage is reasonable.[Popplewell and Bradbury, 2019, Kayssi et al., 2016, Mustapha et al., 2016]

Optimal followup for endovascular interventions of the lower extremity have not been established, but should at least include a pulse exam, ABI and duplex to establish a new baseline after intervention.[Zierler et al., 2018, Mohler et al., 2012]

**What is new with the future management guidance for CTLI? What is BASIL 2 and 3 and BEST-CLI?**

BASIL 2: Infrapopliteal Disease: Vein Bypass First vs. Best Endovascular Treatment first

BASIL 3: PBA +/- BMS vs. DCB +/- BMS vs. DES

(Both Follow up 24-60 months, Primary Endpoint AFS)

BEST-CLI: Open Bypass versus Endovascular Intervention, Primary Endpoint: MALE-Free Survival. Major Above-the-Ankle Amputation, Major Bypass or Jump/interposition graft revision or the need for thrombectomy or thrombolysis (MALE).

**Some important take away lessons for unique scenarios in operative planning for CLTI based on anatomy and conduit.**

- Single segment GSV best conduit for infrageniculate bypass. [Arvela et al., 2010, Avgerinos et al., 2015, Moreira et al., 2016]
- Fem-AK pop bypass with prosthetic may be preferred to contralateral GSV. [Moreira et al., 2016]
- Bilateral external iliac occlusion may be best treated with end to side aorto-bifemoral bypass to allow for continued perfusion of the pelvis. [Jquinandi et al., 2008, van den Akker et al., 1992, Brewster and Darling, 1978]
- Profunda-popliteal collateral index (Segmental pressures AK-BK/AK) of less than 0.25 may suggest that there is sufficient collateral network between profunda and popliteal that SFA treatment may not be necessary. [Boren et al., 1980, Mawatari et al., 2000]
- Mal perforans ulcer over the R first metatarsel head with long occlusion of BK pop, PT and AT with reconstitution of the DP and PT should have

an AK pop to DP bypass to perfuse the appropriate angiosome with the shortest bypass.[Hingorani et al., 2016, Jongsma et al., 2017]

### 5.3.3.1 Complications

**What are some complications of lower extremity revascularization procedures?**

For a comprehensive list of access complications after endovascular therapy, see 13.2.

Femoral exploration carries high risk for infection or lymphatic leak. Lymphatic leaks often resolve spontaneously if they are small and can avoid super-infection. Infected lymphatic leaks, particularly in the setting of prosthetic bypass require exploration. Often times definitive therapy requires alcohol ablation or muscle flap coverage. The most important aspect of lymph leak is prevention with careful dissection and tissue management during femoral exploration.[Obara et al., 2014, Weaver et al., 2014]

**Some important things to keep in mind when considering amputations**

- Transmetatarsal amputation is performed with transection of the metatarsal bones just distal to the heads. Best to transect in a graduated parabola with the 5th metatarsal most proximal. Patients may benefit from concomitant achilles tendon lengthening to prevent equinovus deformity that can result in ulceration of the medial aspect of the TMA.[Eidt and Kalapatapu, 2019]

## 5.4 Acute Limb Ischemia

**17 Oct 2020: Alex Forsyth and Sarah Carlson; Boston University**

**What is acute limb ischemia and what does it encompass?**

Acute limb ischemia is defined as any process that leads to an abrupt cessation of blood flow to a limb resulting in ischemia. There are a few causes, but the most common two are embolic and thrombotic.

- Embolic
  - Cardiac
    - \* Typically due to a fib
    - \* Arm ischemia is most commonly due to cardiac embolism
    - \* Endocarditis – as seen in IV drug users or patients with bacteremia from other causes
    - \* Cardiac tumors – such as atrial myxoma

- Atherosclerotic (e.g. iliac disease embolizing downstream to the lower leg)
- Paradoxical embolism (Thromboembolic venous system with PFO)
- Aneurysm (e.g. thrombus from within an aortic aneurysm embolizing downstream to the leg)
- Thrombosis
  - Aneurysm
  - Bypass graft
  - Acute on chronic progression of atherosclerosis
- Dissection
- Thoracic outlet syndrome (in the upper extremity)
- Vasospasm (severe)

For clarification: (1) Acute on chronic progression of atherosclerosis: – e.g. once a chronic stenosis becomes critically tight, platelet thrombus can develop leading to an acute occlusion; or unstable plaque can “rupture” leading to an acute occlusion of a chronic lesion. And (2) Regarding aneurysms – especially small aneurysms (such as popliteal) – these are less likely to rupture, but more likely to thrombose and cause an acute limb ischemic event.

#### 5.4.1 Presentation and Diagnosis

**What is the patient presentation of ALI? Are there any differences for upper vs. lower extremity presentations?**

Classically remembered by the 5 or 6 Ps depending on who you ask

- Pain: usually located distal to the occlusion and gradually increases in severity as the duration of ischemia continues. The pain may also decrease after a time due to ischemic sensory loss
- Pallor: the limb appears pale compared to the non ischemic limb. There is delayed capillary refill as well
- Poikilothermia: (just a way to make “cold” into a “P” – really means cold limb) means literally the inability to regulate one’s body temperature, or dependent on ambient temperature as cold blooded animals are. If there is no perfusion of warm blood to the limb, it acclimates to the ambient temperature.
- Pulseless: self explanatory, but a good thing to think about is if the contralateral limb has normal pulses, it suggests the absence of chronic limb ischemia and that an embolus or other cause of ALI.

- Paresthesia and Paralysis are the last two Ps. Paresthesias are an earlier sign of ischemic nerve dysfunction and paralysis is a later sign. In the lower extremity, ischemic changes often affect the anterior compartment first, and sensory loss over the dorsum of the foot is one of the earlier neurologic deficits in ALI

This is why a thorough physical exam is key; comparison of both limbs and a good pulse exam including handheld doppler exam. It can be difficult for a junior resident to tell whether a limb is acutely threatened, especially in patients with chronic disease where the presentation of an acute change can be more subtle. This is why the attending surgeon will always ask the consult resident “how is the motor and sensory function” in addition to the pulse exam...this helps us gauge the chronicity and therefore the urgency of intervention.

Acute paralysis, mottling of bilateral lower extremities and absent femoral pulses should raise concern for aortic occlusion.[Wang et al., 2016]

#### How is ALI classified?

From Rutherford RB, Baker JD, Ernst C, et al. Recommended standards for reports dealing with lower extremity ischemia: revised version. J Vasc Surg. 1997;26:517–538. [Rutherford et al., 1997]

| Classification     | Prognosis   | Muscle                 |                              |                  |               |
|--------------------|---|------------------------|------------------------------|------------------|---------------|
|                    |   | Sensory Loss           | Weakness                     | A rterial Signal | Venous Signal |
| I . Viable         | Not immediately threatened  | None                   | None                         | Present          | Present       |
| II.                |   |                        |                              |                  |               |
| Threatened         |   |                        |                              |                  |               |
| a . Marginally     | Salvageable if promptly treated (<24hr)   | Minimal (toes) or none | None                         | Weak/absent      | Present       |
| b . Immediately    | Salvageable with immediate revasc (<6hr)  | More than toes         | Mild, moderate               | Absent           | Absent        |
| III. I rreversible | Major tissue loss or permanent nerve damage inevitable; Minimal benefit from revasc | P rofound anesthetic   | P rofound pa ralysis (rigor) | Absent           | Absent        |

**What does the work up for an acute limb entail? How is the diagnosis made?**

- The diagnosis can often be made on history, physical exam, and bilateral ABIs. Imaging can be done in patients in who the diagnosis is uncertain
- As with most urgent cases, the type of imaging done depends on the availability of at your institution, but generally imaging, such as a CT angiography or arteriography should be done on viable and marginally threatened limbs. Arteriography often can distinguish between embolic vs arterial thrombosis which may help to direct therapy.

The situation varies depending on how severe the presentation is and how quickly you can obtain imaging. Also depends on renal function and whether you want accept two contrast loads (CT followed by endovascular intervention). As a rule of thumb, if I can feel femoral pulses I would typically be more inclined to proceed with on-table angiogram without a CT scan. If femoral pulses are absent I would be more concerned about aortoiliac disease and I would prefer to have a CT scan so I know what I'm getting into in the operating room and can have a better plan.

In patients with severe renal insufficiency, MRA or MR time-of-flight can be helpful, but these studies usually take a little longer to obtain and may not be quickly available in an acute threatened limb situation. Consider going straight to angiography, as to get the most immediate and best imaging of the tibial vessels. [Creager et al., 2012, Earnshaw, 2019]

Bedside ultrasound can also be very helpful even if you're not a certified ultrasonographer yourself, if you have access to color flow doppler US it can be very helpful.

#### 5.4.2 Management

**What is normally done in the initial management of ALI?**

- Anticoagulation - IV unfractionated heparin is immediately administered to prevent proximal and distal progression of secondary thrombus as long as heparin is not contraindicated. The dose should be titrated to maintain activated partial thromboplastin time between 50 and 80 seconds (2-3 times normal values)
- Supportive care (IV fluids)
- A full set of labs including serum chemistry panel with BUN and Cr, CBC, baseline coagulation studies should be obtained. Baseline plasma CPK can be helpful to follow for evidence of rhabdomyolysis after reperfusion

A good rule of thumb for IV heparin is to start with a bolus of 80-100 units/kg, and then drop at 18units/kg/hr – titrating to PTT at 2-3x normal

**What are some of the options for treatment of ALI?**

- Medical primarily with anticoagulation using heparin or a direct Xa inhibitor
- Acute or chronic limb ischemia may be more technically challenging and require more complex reconstruction with a combination of endovascular and open techniques. [de Donato et al., 2018 Jun - Dec, Creager et al., 2012]
- Open
  - Thrombectomy – balloon catheter based (Fogarty embolectomy balloon – Dr. Fogarty invented this while he was a medical student)
    - \* Patients with acute limb ischemia, neuro deficit and distal pop embolism may be best treated with popliteal exposure and open thrombectomy. [Darwood et al., 2018, Kempe et al., 2014]
  - Bypass
    - \* Patients with aortic occlusion and significant comorbidities, and significant aortoiliac baseline disease may likely benefit from primary axillo-bifemoral bypass. [Mohapatra et al., 2018]
    - Endarterectomy – not usually the go to but might be used for the common femoral
- Endovascular
  - Pharmacologic catheter directed thrombolysis
    - \* Absolute contraindications include active bleeding disorder, CVA (<6mo), CNS injury or head injury (<3mo), or GI bleed (<10d).
    - \* Relative contraindications include recent major surgery, uncontrolled hypertension, intracranial tumor, pregnancy, recent eye surgery, hepatic failure, CPR (<10d), or bacterial endocarditis.
    - \* 1-2% risk of hemorrhagic stroke. If neurologic deficit develops during thrombolysis, stop lysis and perform immediate head CT. [Ouriel et al., 1998]
  - Percutaneous thrombus aspiration – useful for small fresh thrombi such as after angioplasty, as distal diameter of the catheter tip limits the size of the thrombus that can be removed.
  - Mechanical thrombolysis and aspiration are also useful for patients with contraindications for thrombolytic therapy, and also may allow for a lower dose of a thrombolytic agent, but risk damage to the arterial wall

**Who gets which kinds of treatment? Who needs emergent treatment?**

- Class I might just need medical therapy like anticoagulation and revascularization can be elective.

- Class IIb patients do not need immediate revascularization
  - If symptoms have been present for less than 2 weeks endovascular therapy is preferred
  - If more than 2 weeks or lytic therapy has failed then surgical intervention is preferred
- Class IIb need immediate revascularization. Historically surgical revascularization has been preferred because of its immediacy, but catheter directed thrombolysis and percutaneous mechanical thrombectomy have shortened time to revascularization.
- Studies:
  - STILE trial – one of the first large RCTs comparing catheter thrombolysis with open surgery; overall the study showed some short term benefit to open surgery however this can probably be attributed to a couple things: (1) in 28% of patients randomized to CDT they weren't able to get a catheter in place so these patients were considered treatment failures and crossed over to the surgery arm, and (2) patients with very long durations of ischemia – up to 6 months) were randomized, and when they looked at patients who had been symptomatic for less than 2 weeks, the thrombolysis patients actually did better. [Investigators, 1994]
  - TOPAS trial – larger RCT which enrolled patients who had an acute arterial occlusion of less than 14 days; this showed no difference in mortality or amputation-free survival but higher major bleeding in the CDT group. [Ouriel et al., 1998]
  - Meta analysis released originally published in 2002 but updated in 2013 and 2018 demonstrated no difference in mortality or limb salvage between surgical and thrombolytic therapy, but endovascular demonstrated higher rates of complications including ongoing limb ischemia and bleeding within 30 days of treatment. Previously they had reported higher rates of stroke in the thrombolysis category but the most recent update is unable to support this finding [Darwood et al., 2018]
    - \* All this to say, it is very reasonable to think about a catheter-directed therapy especially if the presentation is acute, less than 2 weeks or so. That said, there are certain anatomical locations that most surgeons would favor a simple open procedure – e.g. embolism to the common femoral or brachial arteries – these are typically pretty simple to treat with a cut down and balloon thrombectomy.
    - \* One thing it is important to consider when doing a therapeutic infusion is that you might place a tPA infusion catheter at time

zero and then bring the patient back 24 hours later; patient needs to be advised that they'll need to lie flat for a day or even two days. Setting expectations with patients is important.

- Class III is usually treated with primary amputation because revascularization is unlikely to restore function to the limb and restoring bloodflow can cause the patient serious harm.

**What are the risks of revascularization for a class III or prolonged ischemia?**

- Myonephropathic metabolic syndrome: muscle cells undergo liquefactive necrosis due to ischemia. Potassium, myoglobin, lactic acid, and superoxide accumulate and can perfuse through the body or can have a sudden increase in the event of revascularization which leads to hyperkalemia, arrhythmias, pulmonary edema, metabolic acidosis, myoglobinuria, and can even cause sudden death from heart and/or renal failure. [Obara et al., 2018] Treatment of this is largely supportive with fluids

## 5.5 Compartment Syndrome

**What is the pathophysiology and manifestations of compartment syndrome?**

- Increased intramuscular compartment pressure results from increases in capillary permeability due to ischemic reperfusion. The increase in pressure leads to neuromuscular dysfunction and interferes with circulation. Irreversible damage occurs when pressures exceed 30mmHg in each compartment
- Ischemia reperfusion causes increased capillary permeability due to free oxygen radicals, neutrophils and endothelial factors that collect during ischemia. Severity depends on time to reperfusion, muscle mass, and flow pattern (i.e. direct or collateral). Symptoms often develop within 6hr of reperfusion. [von Keudell et al., 2015]
- The pathophysiology underlying acute extremity compartment syndrome is related to the arteriovenous pressure gradient theory. Increased compartment pressure reduces the gradient – reduces arterial pressure and increases venous pressure.[Frink et al., 2010, McQueen and Court-Brown, 1996, Elliott and Johnstone, 2003, Papalambros et al., 1989]
- Clinically they can have neurological dysfunction with sensory motor deficits, but the most common presentation is a tense extremity with pain on passive movement of the muscles in the compartment, which is often dorsiflexion/plantar flexion of the ankle. A sensitive indicator is loss of two point discrimination

**How do you diagnose a compartment syndrome?**

- Physical exam (tenderness, especially over anterior compartment), paresthesias, especially between first and second toes
  - Anterior compartment: deep peroneal nerve (this is a VSITE favorite)
  - Deep posterior compartment: tibial nerve [Velmahos and Toutouzas, 2002]
- Compartment pressures: how do you do this?
  - Need a needle to access the compartment and a pressure monitoring system (can be handheld Stryker kit, or just a hollow bore needle connected to an arterial pressure bag).
  - Normal compartment pressure is <10-20mmHg; greater than 30 is highly concerning. Probably even more accurate than an absolute number is comparing the compartment pressure to the mean arterial pressure or diastolic pressure. If the compartment pressure is within 40mmHg of the MAP (for example, MAP is 60 and compartment pressure is 25 – this is concerning) – OR – if the difference between compartment pressure and diastolic pressure is less than 10 (for example, diastolic pressure is low at 30, and compartment pressure is 22).

If clinical suspicion of compartment syndrome is high, I tend not to be reassured by “normal” compartment pressures. It’s relatively low risk to do fasciotomies, but the risk of limb loss is so high for a missed compartment syndrome...I would much rather err on the side of caution if there’s any question.

**Treatment is a fasciotomy – can you tell us a little about the types of fasciotomies?**

- Forearm and upper arm fasciotomies are often performed by orthopedic or hand surgeons. The forearm fasciotomy includes dorsal and volar incisions to release the dorsal and volar compartment, and mobile wad while avoiding numerous superficial cutaneous nerves. The arm fasciotomy releases the medial, lateral, and deltoid compartments through medial lateral incisions
- Fasciotomies can be done in the thigh as well with a medial and lateral incision to release the lateral, medial and posterior compartment
- The most common type is a lower leg 4 compartment fasciotomy

**How is a lower leg 4 compartment fasciotomy for the lower extremity performed?**

1. A longitudinal incision is created between the fibular shaft and the crest of the tibia over the intermuscular septum and the anterior and lateral compartments are opened. If tissues are swollen occluding the view of the

intermuscular septum, the perforating vessels can be followed down to it. Nerves including the peroneal nerve are most at risk near the fibular head

2. A second incision is created on the medial surface of the lower leg approx. 1cm posterior to the edge of the tibia to avoid the greater saphenous vein. The superficial posterior compartment is incised. The gastrocnemius-soleus complex is taken down from its attachments to the tibia in order to access the deep posterior compartment.
3. The incisions are made generously – sometimes the skin incision can be a little short of the fascial incision, but they should be nice and long in order to fully release the compartments
4. After hemostasis be sure to apply loose dressings, and the leg should be elevated to reduce edema that can complicate closure. Closure can be done in 48-72 hours but may be delayed and dressed with wound vacs to attempt primary closure. If primary closure is not possible, a split thickness skin graft can be used for closure

**Who should a prophylactic fasciotomy be performed on?**

- Patients with high occlusion and extensive ischemia,
- Acute ischemia of greater than 6 hours with few collaterals
- Patients with combined arterial and venous injury
- Patients who are obtunded making serial examination difficult

**What is the prognosis for a patient with ALI? What are some patient factors that lead to a poor prognosis?**

Amputation rates after acute limb ischemia are typically described in the 10-20% range, and mortality is also in the 10-25% range whether you're talking about surgery or catheter-directed procedures (that's excluding the patients who present with Rutherford class III and by definition have an unsalvageable limb). Many factors determine likelihood of amputation; typically, patients with more medical comorbid conditions tend to do worse as you might expect: baseline CAD, kidney disease and smoking are predictive of worse outcomes. There is a trend toward improved limb salvage rates (decreased amputation rate) over time, and I think this speaks to wider availability of different limb salvage techniques among vascular surgeons across the globe.

## 5.6 Blue Toe Syndrome

### 5.6.1 Etiology and Presentation

Blue toe syndrome is characterized as a painful discolored toe in the setting of a normal vascular examination or palpable pulses.

Acute onset of blue toe after MI - consider cholesterol embolization from wire manipulation in the aorta. [Saric and Kronzon, 2012]

### 5.6.2 Evaluation

Patients should have a work up for an embolic source. Patients without a clear recent source of embolization on history or physical, should undergo CTA of chest/abdomen/pelvis to look for an arterial lesion, echocardiogram to identify a cardiac source, and a Holter monitor to evaluate for an underlying arrhythmias.

If no obvious source of embolism can be identified, further evaluation for underlying cancer may lead to a prothrombotic state.

### 5.6.3 Management

Treatment of blue toe from cholesterol embolization best with single antiplatelet and statin. Anticoagulation may precipitate further embolization. [Ghahramani et al., 2016, Quinones and Saric, 2013]

Treatment of blue toe from a specific thrombogenic arterial lesion may be best with anticoagulation. Recurrent symptoms on anticoagulation may require stent coverage or, in a young patient, may require thrombectomy. [Reyes Valdivia et al., 2017, Jeyabalan et al., 2014, Verma et al., 2014]

## 5.7 Non-atheromatous Popliteal Artery Disease

### 5.7.1 Popliteal Artery Entrapment Syndrome (PAES)

#### 5.7.1.1 Etiology and Presentation

Most often seen in young healthy patients (often athletes) who present with claudication.

Etiology from embryologically abnormal lateral attachment of medial head of the gastrocnemius muscle.[Gokkus et al., 2014, Lejay et al., 2014] Type IV PAES caused by compression with popliteus muscle.

#### 5.7.1.2 Evaluation

Physical examination finding will be loss of pedal pulse on active plantarflexion or passive dorsiflexion of the foot.[Gokkus et al., 2014, Lejay et al., 2014]

Best diagnosed with axial cross-sectional imaging from the MRA to identify the abnormal muscle course and insertion. [Sinha et al., 2012, Lejay et al., 2014]

#### 5.7.1.3 Management

Symptomatic PAES should be treated, even if presenting with mild or moderate claudication, due to risk of progression and thrombosis from scarring.[Forbes and

Kayssi, 2019]

### 5.7.2 Cystic Adventitial Disease

#### 5.7.2.1 Etiology and Presentation

Controversial etiology, often considered related to repeated popliteal trauma due to knee flexion, causing fluid to collect between the adventia and media layers of the artery wall. Presentation overlaps with atherosclerotic lesions of the popliteal segment, but in patients without traditional risk factors.

#### 5.7.2.2 Evaluation

Often diagnosed on duplex ultrasound or CTA. For a representative image, see 12.5.1.4.

#### 5.7.2.3 Management

Compression can sometimes be relieved by percutaneous drainage of large cystic portion that is causing luminal compression. PTA and stenting should be avoided as often poor durability. Operative resection and reconstruction with interposition can be effective but is a final option.[Li et al., 2017]



# Chapter 6

## Mesenteric Disease

**UNDER CONSTRUCTION - This Chapter is Currently in DRAFT mode**

**09 Apr 2020:** *Matt Chia, MD and Nick Mouawad, MD*

Mesenteric vascular disease can be broken down into three disease states that we'll cover today. There's the arterial disease, which is clearly separated into acute mesenteric ischemia and chronic mesenteric ischemia. Then there is venous disease, which we'll touch on briefly. There are also a handful of somewhat related diseases that we'll also sprinkle into these discussions, like median arcuate ligament syndrome and SMA syndrome, but that's overall where we're headed.

Mesenteric collaterals are important for protecting against mesenteric ischemia. The gastroduodenal and pancreaticoduodenal arteries connect the celiac and SMA. The meandering artery (arc of Riolan) and marginal artery of Drummond connect IMA and SMA. Hemorrhoidal branches connect internal iliacs and IMA. [Chandra and Quinones-Baldrich, 2010, van Gulik and Schoots, 2005]

### 6.1 Acute Mesenteric Ischemia

#### 6.1.1 Presentation and Evaluation

**Can you tell me about the classic presentation and approach to patients presenting with acute mesenteric ischemia?**

These patients present with the sudden onset of abdominal pain. Nausea, vomiting, distention, and diarrhea (possibly bloody, described as "sudden and forceful evacuation") are the common symptoms. Pain out of proportion is the classic buzzword, and can be hidden on multiple choice tests with a pain score of 10/10 with only mild abdominal tenderness on physical exam.

Etiology most often from acute embolism (40-50%), which is often secondary to cardiac source (atrial fibrillation or recent MI).[Wyers, Mark C and Martin, 2019]

Vitals typically normal, possible tachycardia.

Lab evaluation typically unremarkable. Leukocytosis, hemoconcentration, and acidosis (high anion gap) all are frequently found, but the absence of these definitely does **not** rule out acute mesenteric ischemia.

D-dimer has been proposed as a reasonable rule-out test for acute mesenteric ischemia.

### How about radiology studies?

#### Plain film:

Frequently normal, may show ileus in the early stages. In late stage acute mesenteric ischemia, findings on plain film can include bowel wall edema (thumbprinting) or pneumatososis.

#### CT angio:

Probably represents the most common diagnostic modality to diagnose acute mesenteric ischemia. CTA has the advantages of speed, availability, and non-invasiveness when compared to conventional angiography, and also allows for some assessment of the degree of bowel involvement.

Preferentially, a “negative” oral contrast agent would help prevent oral contrast from causing artifact or obscuring evaluation of the vessels, although the availability of these agents may limit their use.

### How about mesenteric duplex (vascular lab studies)?

Mesenteric duplex has the advantage of being able to see the velocity of flow across a stenosis, giving you a good method of quantifying the significance of a stenosis. However, bowel gas often limits the acoustic windows for visualizing the mesenteric arteries, and so we usually will have patients fast for several hours before a study. Also, duplex is more sensitive for proximal disease rather than distal mesenteric involvement. For these reasons, mesenteric duplex is considered the gold standard for evaluating chronic mesenteric ischemia, but has no real role in the evaluation of acute mesenteric ischemia. Can you imagine, having a tech mash a transducer into a patient with acute abdominal pain?!

## 6.1.2 Management

### What's your approach to the initial management of the patient?

1. Resuscitation. Fluids, fluids, fluids. These patients are really volume down, and are headed towards a profound distributive shock that will

be worsened by your eventual plan for revascularization (think ischemia-reperfusion injury). Also look for electrolyte imbalances and correct those early.

2. **Antibiotics.** These patients are not usually septic on initial presentation, but are at high-risk, so broad-spectrum antibiotics with gut coverage (Gram negative and anaerobes) are standard of care.
3. **Heparin.** In the absence of a clinical contraindication, these patients should be systemically heparinized, with a bolus, as soon as the diagnosis is made.

**Which blood vessel does acute mesenteric ischemia typically involve?**

The SMA. This makes sense if you think of each mesenteric distribution. The celiac distribution has organs that have redundant blood supply (like liver and stomach). The IMA is frequently occluded in patients with AAA, but the patients are rarely ever symptomatic due to collateral flow. The hypogastrics (which ARE mesenteric vessels, especially in the situation of an occluded IMA) principally supply the rectum from a mesenteric standpoint.

For embolic pathology, the acute angle of the SMA seems to predispose it to capture emboli from above, but this is more theoretical than proven. The embolism is often lodged distal to middle colic vessel.[Wyers, Mark C and Martin, 2019, Kazmers, 1998]

**Tell me about the two main pathologies, and how they would differ in terms of the anatomy and operative findings.**

So the two most common etiologies of acute mesenteric ischemia are embolism and thrombosis.

Embolism is the more common, where preexisting thrombus (think atrial fibrillation, mural thrombus from thoracic aneurysm, etc.) or plaques from atherosclerotic disease break off and lodge in the SMA. The classic operative finding is that an embolism lodges just distal to the middle colic artery, where there is a significant caliber change in the SMA. This is distal to the first few jejunal branches off of the SMA, leading to the classic sparing of the proximal jejunum and transverse colon. In other words, the mid to distal jejunum and all of the ileum will be ischemic, but other areas of the SMA territory are spared. Atherosclerotic debris is typically smaller, and results in smaller, more patchy areas of ischemia.

Thrombosis occurs primarily as a plaque rupture of preexisting atherosclerotic disease, resulting in acute thrombosis at the site of the disease. Thus the patients will often present with an acute-on-chronic symptomatology, having classic symptoms of postprandial abdominal pain, food fear, and weight loss, but with a sudden onset of severe symptoms. This frequently allows for the development of mesenteric collaterals, which may make the onset of acute symptoms more insidious than for embolic pathology. In the majority of situations, the

atherosclerotic disease is most severe right at the origin (consistent with what we know about shear stress and branch points in blood vessels). This means that when the plaque ruptures, the entirety of the SMA occludes, leading to ischemia of the entire territory, as opposed to the jejunal-sparing distribution seen in embolic disease.

**Describe the operative steps to getting exposure of the supraceliac aorta (or the celiac artery).**

1. Divide triangular ligament to mobilize left lobe of liver
2. Divide gastrohepatic ligament to enter the lesser sac
3. Retract liver to right with a self-retaining retractor
4. Push esophagus left (use NGT to assist with identification)
5. Divide peritoneum overlying crura to identify celiac vessels
6. Typically trace common hepatic artery backwards to identify celiac artery
  - Watch out for the left gastric vein as it crosses the celiac artery as it drains the lesser curve of the stomach into the portal vein.
  - About half of the time, the phrenic artery takes an origin from the celiac artery and must be controlled during exposure.
7. To expose the supraceliac aorta, divide the median arcuate ligament and separate the left and right crura from each other.

**So through this kind of exposure, what mesenteric vessels do you get access to?**

You can trace most of the proximal celiac distribution right at the origin, and through this exposure you get access to the origin of the SMA if you mobilize the superior border of the pancreas. The neck of the pancreas and the splenic vein cross the anterior of the SMA, obscuring the rest of the mid and distal SMA from the superior approach.

**How about getting to the rest of the SMA?**

There are a couple of places you can get exposure to the SMA.

Most commonly you'll hear it described at "root of the mesentery." Specifically, lifting up the transverse colon will stretch out its mesentery (i.e. transverse mesocolon). At the bottom, or "root" of the transverse mesocolon, a transverse incision is made. If the middle colic artery is palpable in the mesocolon, the incision can be made around it, and you can trace the middle colic backwards to the SMA. Usually you'll find the SMV first, and the SMA will be just to the left of it. Be sure to identify and preserve small jejunal branches during the dissection. If needed, careful dissection superiorly, going behind the inferior border of the pancreas can get a little more proximal exposure. Embolectomy

through a transverse arteriotomy in the SMA is the best approach.[Wyers and Martin, 2019, Kazmers, 1998]

Alternatively, you can get to the SMA from a lateral approach, specifically from the left side. Begin by dividing the ligament of Treitz and mobilizing the 4th portion of the duodenum. The SMA is found in the tissues just cephalad to the duodenum. You can also improve your proximal exposure if needed by retracting the inferior border of the pancreas cephalad to the level of the left renal vein.

Other options include a retroperitoneal exposure, like you were preparing to treat a thoracoabdominal aneurysm. Additionally, the more distal SMA can just be identified in the small bowel mesentery.

**So for the operative strategy for acute mesenteric ischemia, tell me about the general approach to the patient.**

1. Resect frankly necrotic bowel and contain gross spillage. Once you revascularize the bowel, compromised-appearing bowel may improve and not need immediate resection. Thus the first step is only damage control, to remove anything completely unsalvageable that is making the patient sick, or anything causing gross contamination of the operative field. The key is you're not doing anything definitive with the bowel as your first step.
2. Revascularization. SMA embolectomy is the initial management of choice for embolic disease. Thrombotic disease, on the other hand, may be more challenging to treat by embolectomy alone, and frequently are treated with a bypass. (More to come on these procedures).
3. Re-assess bowel viability. Clinical status permitting, 20-30 minutes should be taken to fully assess the results of the revascularization before proceeding with resection. Perfusion can be assessed by many methods, including clinically, by Doppler, pulse oximetry (a.k.a. photoplethysmography), fluorescein fluorescence, etc. The take home is to give the bowel enough time to be perfused before going ahead with resection.
4. Proceed with temporizing or definitive bowel repair. Resection, leaving in discontinuity, primary anastomosis, diversion, etc. All of these are options on the table, but the key here is that all of the previous steps occur before addressing the bowel.
5. Consider second-look laparotomy. Many times, bowel may look questionable even after revascularization and thorough re-assessment. To preserve the most bowel length, it may be reasonable to leave borderline bowel alone at the index operation and do a “second look” to fully reassess the bowel, especially after the patient has benefitted from aggressive resuscitation in the ICU.

Endovascular approaches to acute mesenteric ischemia have been described but data is limited. Exploratory laparotomy and embolectomy should be preferred

management.[Wyers and Martin, 2019]

**Let's talk a little about the steps for an SMA embolectomy.**

Typically, you'll expose at the root of the transverse mesocolon. After obtaining proximal and distal control, an arteriotomy is made, and embolectomy can be performed by passing Fogarty catheters in both a retrograde and antegrade fashion. The arteriotomy can be made transversely for an embolectomy, and thus could be closed primarily. A longitudinal arteriotomy may be advisable if you have a high suspicion that you'll need to do a bypass, and if not, may be closed with vein patch angioplasty (remember that the field is contaminated or dirty in many situations).

**So if I'm gonna do a bypass, what are my options for conduit?**

Yeah, so again, because the field is frequently contaminated or dirty, a good conduit is saphenous, followed by femoral vein. Thus every patient undergoing surgery for acute mesenteric ischemia should have both legs prepped out in the field. Prosthetic conduit has the advantages of being more resistant to kinking (externally reinforced), likely better patency than vein (although data are a little mixed), but in the situation of gross contamination may be less preferred than vein. Other less common options include cryo-preserved cadaveric homograft, or rifampin-soaked prosthetic.

**And what are some of my options for constructing a bypass?**

Short retrograde aorto-SMA bypass:

This bypass takes its origin off of the aorta just below the SMA, anastomosing typically end-side onto the SMA just below its origin in order to bypass ostial or very proximal disease. This is a relatively quick bypass, with only one field of dissection directly from the aorta below the SMA onto the proximal-mid SMA. The length of the bypass is very short, limiting concerns with kinking or twisting of the bypass. However, this may not always be feasible, as SMA disease often coexists with significant aortic disease. Additionally, the other bypasses described have better reported patency.

Long retrograde R iliac-SMA bypass ("C-loop"):

This bypass originates from the right common iliac artery, which presents a number of distinct advantages over an aorto-mesenteric bypass. First, using the iliacs avoids the hemodynamic consequences of an aortic cross-clamp, which may be contraindicated depending on your patient's medical condition. Second, you can avoid showering, causing dissection, or otherwise injuring your clamp sites if you have significant disease in the mesenteric segment of the aorta, which is common in patients with chronic mesenteric ischemia. The graft should be tunneled in a gentle C-loop towards the SMA to avoid kinking or twisting. The proximal anastomosis is performed end-side on the iliac artery, and the distal can be performed either end-end or end-side depending on the anatomy of the

disease. Especially when using prosthetic in a contaminated field, you can consider taking an omental flap to wrap or cover the prosthetic.

Antegrade supraceliac aorta-SMA bypass:

This bypass originates from the supraceliac aorta. If revascularization of both celiac and SMA is planned, a bifurcated graft can be selected. A side-biting aortic clamp can be used to mitigate the hemodynamic effect of an aortic cross-clamp. The tunnel to the SMA is created with gentle finger dissection in a retropancreatic plane, taking care to avoid injury to the SMV.

**What other options have been described for treatment of acute mesenteric ischemia?**

Retrograde open mesenteric stenting (ROMS)

So ROMS is a hybrid procedure involving an upper midline laparotomy that is used to evaluate the bowel. Through this incision, SMA exposure is obtained just as in a traditional open fashion at the root of the mesentery. The mid-SMA is then punctured under direct vision, and the area of disease is attempted to be treated from a retrograde approach back into the aorta. If bowel ischemia is found, the upper midline is easily lengthened into a traditional vertical laparotomy incision.

Endovascular treatment (percutaneous thrombectomy / thrombolysis / pharmacomechanical thrombectomy)

Some authors have described completely endovascular approaches to treatment of acute mesenteric ischemia. However, the major limitation is the inability to assess the bowel. These patients are frequently those who are deemed to be lower-risk for frank bowel ischemia or perforation, but the rates of laparotomy and bowel resection after these treatments have been described at over 20%. Probably not your first answer for oral boards.

### 6.1.3 Other Etiologies

**What are some other, more rare etiologies of acute mesenteric ischemia?**

Embolism is the most common etiology of acute mesenteric ischemia (40-50%), and thrombotic etiology composes another quarter to a third of these populations. The other two etiologies to consider are non-occlusive mesenteric ischemia and mesenteric venous thrombosis.

**Perfect, so how does a non-occlusive mesenteric ischemia (or NOMI) patient differ from what we've been talking about?**

So NOMI patients typically do not have a focal lesion like you see with embolism or thrombosis. What happens to these patients is that classically they're pretty sick patients with some predisposing factors, most commonly ESRD. On top of that, there was some clear inciting hemodynamic event causing sustained

hypotension, such as recently getting a session of hemodialysis or undergoing cardiopulmonary bypass. The presentation is more indolent and less obvious than embolic or thrombotic acute mesenteric ischemia, and the imaging findings are more consistent with a diffuse vasospasm and hypovolemia picture.

Treatment is primarily conservative, with the emphasis on resuscitation and addressing whatever the underlying etiology is. Adjuncts to this include placement of infusion catheters into the affected vessel with infusions of vasodilators (most commonly papaverine at 30-60mg/hr) or prostaglandin [Wyers, Mark C and Martin, 2019, Trompeter et al., 2002] NOMI with portal venous gas, pneumatisis and free air often indicate need for laparotomy.

#### **How about mesenteric venous thrombosis?**

This is the most rare and most difficult to diagnose. They have a very slow course, frequently with a lot of other workup already done. There's a wide variety of causes that have been reported, and any of the things that contribute to Virchow's triad have been reported (thrombophilia from coagulopathy or malignancy, venous stasis from abdominal hypertension or obesity, direct injury from trauma, surgery, or inflammation). The diagnostic test of choice is a CT with portal vein contrast, which most commonly identifies thrombosis in the superior mesenteric vein (but can also involve the IMV, portal vein, or splenic vein). The treatment of choice is therapeutic anticoagulation.[Acosta and Björck, 2019]

## **6.2 Chronic Mesenteric Ischemia**

### **6.2.1 Presentation and Evaluation**

#### **How does chronic mesenteric ischemia differ in its presentation?**

Chronic mesenteric ischemia is characterized by post-prandial abdominal pain, typically 30-60 minutes after eating (think after gastric emptying time). This pain is usually severe, crampy, and resolves after minutes to hours of time. The pain leads to food fear and eventual unintentional weight loss. The next step in evaluation in these patients is a mesenteric duplex to evaluate for elevated velocities.

However, the clinical presentation here is key. Chronic mesenteric ischemia is pretty unlikely in patients who do not have this constellation of clinical symptoms, and it's really common for patients to be referred with imaging findings of elevated mesenteric velocities on duplex who have none of the clinical findings, and thus do not benefit from any intervention.

#### **What are the duplex criteria for chronic mesenteric ischemia?**

Yeah, so really the two vessels we're concerned with here are the celiac artery and the SMA. It's pretty rare that stenosis of the IMA results in clinically significant ischemia, and also pretty rare that isolated disease of either the celiac or the

SMA would be enough to cause significant symptoms. Typically you see the classic symptomatology of chronic mesenteric ischemia in patients with occlusive disease in both the celiac and the SMA.

The key numbers to remember here are peak systolic velocities (that's PSV) of 200 cm/s for the celiac artery, and 275 cm/s for the SMA. These correspond to a stenosis of at least 70%. Remember, the **superior** mesenteric artery has **higher** velocity criteria, in case you forget. These classic numbers come from a study done in 1993, and many other studies have demonstrated other thresholds. Make sure to check with your friendly neighborhood vascular technician to see what the thresholds in your local lab are!

In addition to the PSV, you can also use the end diastolic velocity (EDV) to predict stenosis. For the EDV values in the celiac and the SMA, think approximately 50 cm/s predicts a stenosis of 50% (easy to remember, **50-50**). This is known as Bowersox criteria, and is actually >45, but 50 is easier to remember.[Bowersox et al., 1991]

You can also predict a higher degree of stenosis with EDV values. This is easy for the SMA, where an EDV of 70 cm/s predicts a stenosis of 70% (**70-70**). For the celiac, it's a bit higher, where an EDV of 100 cm/s predicts that same 70% stenosis.

So to review the numbers: [Moneta et al., 1991, 1993, Zwolak et al., 1998, AbuRahma et al., 2012]

- PSVs: 200 in the celiac or 275 in the SMA gives you 70% stenosis
- EDVs: 50s in the celiac or 50s in the SMA gives you 50% stenosis
- EDV #2: 100 in the celiac, or 70 in the SMA gives you 70% stenosis.

Final thought. When the celiac artery is severely narrowed or occluded, often you'll have retrograde flow coming from the SMA via collaterals (these are your pancreaticoduodenal arcades via the GDA). You can see this as backwards flow in the common hepatic artery, and this has 100% specificity for disease in the celiac artery.[Warncke et al., 2019]

### 6.2.2 Management

#### What's next in the approach to these patients?

Frequently they're going to get some kind of axial imaging, either CTA or MRA, to help plan for their revascularization, although this isn't strictly necessary. Often endovascular approaches are the mainstay of treatment. Remember that the angle of the SMA frequently favors a brachial approach, although you can also engage the SMA from a femoral approach with preformed catheters (think Sos, Cobra, etc) or steerable sheaths. Remember to watch out for median nerve compression from brachial sheath hematomas if you've opted for a percutaneous brachial approach, with a low threshold to evacuate the hematoma and

perform median nerve decompression if the patient develops symptoms in the median nerve distribution postoperatively. Also remember that a brachial sheath hematoma will not be easily palpable or visible at the skin level, so the neurologic exam postoperatively is key.

Single vessel SMA revascularization is widely accepted for chronic mesenteric ischemia without necessarily needing to revascularize the celiac axis. [Oderich and Ribeiro, 2019a, Foley et al., 2000]

#### **Any other technical tips for endo intervention on the mesenterics?**

The best view for diagnostic angiography in mesenteric ischemia is the lateral view.

Most often balloon-expandable stents are selected for the mesenteric circulation. The precision of deployment and the increased hoop strength of a balloon expandable stent make them more favorable here over the radial force of a self-expanding stent. You can also flare the proximal end of a balloon-expandable stent into the aorta, making sure that you've really treated any ostial disease. Some authors advocate for use of embolic protection devices to prevent distal embolism, and other authors also advocate for the use of covered stents for the mesenteric circulation due to better patency.[Oderich et al., 2013, Oderich and Ribeiro, 2019a]

#### **What is the surveillance protocol for an SMA stent?**

At most institutions it is 1mo, 6mo and annually thereafter.[Mohler et al., 2012, Zierler et al., 2018]

#### **How about open revascularization?**

In most centers, up to 80% of CMI is treated endovascularly. Open is often reserved for those that fail endovascular therapy.[Oderich et al., 2010, Oderich and Ribeiro, 2019a]

So we talked a lot about open revascularization earlier in the acute mesenteric ischemia segment. The techniques are broadly similar, with the exception that often for chronic mesenteric ischemia, frequently you'll be planning to revascularize both the celiac and the SMA. The approach for this is with a bifurcated graft from the supraceliac aorta, taking one limb down to the celiac artery, and the other limb tunneled in a retropancreatic fashion to the mid-SMA. Remember that this approach requires a supraceliac aortic cross clamp, which your patient may not be able to tolerate. But single revascularization of SMA is often acceptable with appropriate collaterals. Taking a retrograde bypass off of the common iliac artery as we previously described may be a better option for patients with significant comorbidities. A further option that completely avoids a bypass is to go right for a trapdoor endarterectomy of the celiac artery and the SMA, allowing you to address coral reef or proximal/ostial disease (such as flush occlusions) that are difficult to treat endovascularly.

## 6.3 Mesenteric Aneurysms

### 6.3.1 SMA Aneurysm

If symptomatic, most SMA aneurysms are mycotic in nature and require treatment. Treat with resection and saphenous vein interposition and 6 weeks antibiotic therapy based on intraoperative cultures.[Lee et al., 2008 Nov-Dec]

Asymptomatic aneurysms can be observed up to 2.5cm.[Kwong et al., Stone et al., 2002]

### 6.3.2 Splenic Aneurysm

Post splenic aneurysm embolization, L flank pain in stable patients likely represents splenic infarction. Treat with hydration and analgesia. Rates are lower in stented patients.[Kwong et al., Piffaretti et al., 2007, Zhu et al., 2019]

Splenic artery embolization also an effective way to treat thrombocytopenia, particularly chemo induced, when splenectomy is too high risk.[Bhatia et al., 2015]

## 6.4 Median Arcuate Ligament Syndrome (MALS)

**Can you tell me about median arcuate ligament syndrome and how that differs from chronic mesenteric ischemia?**

Sure, so median arcuate ligament syndrome (MALS), which has many names (Dunbar syndrome, celiac axis compression syndrome, etc.) is a somewhat controversial entity that occurs when repeated compression of the celiac artery occurs against the median arcuate ligament during respiratory variation. The thing you want to visualize here is that during full exhalation, the lungs are completely emptied, and the diaphragm moves up at a sharper angle. This angulation kinks off the celiac artery more severely, so velocities in **exhalation** are **higher** in MALS. However, these findings are common in asymptomatic patients, and so just like chronic mesenteric ischemia, the clinical presentation is key. They'll have a similar presentation as chronic mesenteric ischemia patients, with post-prandial pain, food fear, and weight loss, but often the symptomatology is a little more indolent in these patients. Because of this, these patients have frequently gotten the million dollar workup for nonspecific GI pain. Duplex ultrasound shows celiac PSV >200 and EDV >50, normalizes during deep inspiration, and post-stenotic spectral broadening.[Zwolak et al., 1998]

The treatment of choice for suspected MALS is a laparoscopic median arcuate ligament release, frequently performed by a MIS/foregut surgeon. A key point here is that you don't want to be fooled into putting a stent in these patients before they've gotten their median arcuate ligament release, because the dynamic

motion of the diaphragm is likely to crimp or bend the stent if that hasn't been treated yet. It may be that some patients benefit from endovascular treatment after release, though, as some think that the chronic damage from MALS can result in intimal damage/scarring that persists even after the extrinsic compression is treated by the median arcuate ligament release.

## 6.5 SMA Syndrome

### What is SMA syndrome?

So SMA syndrome, also called Wilkie's Syndrome is a rare entity where the 3rd portion of the duodenum gets compressed between the SMA and the aorta, causing a functional gastric outlet obstruction. Patients are typically emaciated, having lost a significant amount of weight before their symptom onset. What's happened is they've lost the retroperitoneal fat pad that normally surrounds the SMA, and so the angle between the SMA and the aorta becomes more acute, pinching off the duodenum.

Classic findings in fluoroscopic swallow in SMA syndrome are delayed gastric emptying, a dilated duodenum, to and fro peristalsis in the proximal duodenum and cutoff in the third portion. EGD can show pulsation in the 3rd portion of the duodenum.[Warncke et al., 2019]

The treatment of choice here is enteral feeding with a nasojejunal tube or other surgically placed tube and TPN, because what really will help them here is weight gain. GJ bypass or duodenal mobilization is sometimes needed for treatment, but is controversial. It's not really a mesenteric vascular disease, but it sometimes shows up on exams as a related entity.[Welsch et al., 2007, Merrett et al., 2009]

## 6.6 Uterine Fibroids

Symptomatic uterine fibroids are now often treated with uterine artery embolization. Many patients require bilateral uterine artery embolization (UAE), but may only need unilateral if blood supply is unilateral. Infarcted tissue can be expelled or become infected even years after initial embolization. [Stępniaak, 2018]

# Chapter 7

## Renal

**UNDER CONSTRUCTION - This Chapter is Currently in DRAFT mode**

**29 Jun 2020:** Dr. Cullen McCarthy and Dr. Matthew Edwards; Wake Forest

### 7.1 Pathophysiology

#### What is renovascular hypertension?

Hypertension as a result of progressive renal artery stenosis

- While renal artery stenosis is a relatively common finding in older patients with hypertension, it's relatively uncommon as the primary cause of hypertension.

#### What is ischemic nephropathy?

Decreased renal function and/or chronic kidney disease that results from atherosclerotic renal artery stenosis due to a reduction in glomerular filtration rate (GFR) and rise in creatinine produced by any cause of diminished renal blood flow. Primary mechanism is chronic hypoperfusion. [Rickey and Geary, 2019]

#### Who is at risk for ischemic nephropathy?

Renal injury can develop in anyone with a kidney or kidney region beyond a critically stenotic artery

Now, this is usually in patients with atherosclerotic disease, but any flow limiting lesion—such as coarctation of the aorta, mid-aortic syndrome, or fibromuscular dysplasia—can cause ischemic neuropathy OR renovascular hypertension.

In terms of clinical practice, the prevalence of renovascular hypertension is probably less than 1 % in patients with mild hypertension but may be as high as 10 to 40 % in patients with acute (even if superimposed on a preexisting elevation in blood pressure), severe, or refractory hypertension.

**That's the best you can do, 10-40%? That's a big window.**

Yes, and it illustrates the biggest issue we have with renovascular disease. We know 5-22 % of patients 50 years or older who have advanced CKD have some degree of renal artery stenosis and 23 to 54% of these patients have bilateral renal artery disease.

- Bilateral renal arterial stenosis is associated with more widespread atherosclerotic disease, higher serum creatinine levels, and higher mortality than unilateral disease
- Renal artery revascularization among patients in these studies infrequently produced a meaningful recovery of kidney function, which would have supported the diagnosis

**So not everyone with flow limitations to their renal vascular will get renovascular hypertension or ischemic nephropathy?**

Not at all, actually. Flow limiting lesions may be an “incidental” finding in patients who have CKD or hypertension that is caused by a separate disorder (eg, diabetic nephropathy and essential/primary hypertension).

- Because of this, it can be very difficult to distinguish between patient whose disease is induced by renal artery stenosis and those who have alternative causes of CKD or renovascular hypertension.

**So you're telling me that we have no idea who has clinically significant disease and who doesn't?**

No. Fortunately there are clinical findings that suggest that renovascular disease is an underlying cause:

- Recent or rapid development of severe hypertension.
  - Relatively specific for renovascular hypertension and is the strongest predictor of antihypertensive benefit from revascularization.
- Severe hypertension that may be treatment resistant.
  - Some patients with ischemic nephropathy are normotensive, which may be due in part to a reduced cardiac output
- Acute rise in serum creatinine following the administration of angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARBs).
  - Rise in serum creatinine is more common with agents that block the renin-angiotensin system than with other antihypertensive drugs

because glomerular filtration rate (GFR) often depends upon the efferent arteriolar actions of angiotensin II in this setting.

- More common with bilateral as compared with unilateral disease because there is hemodynamic compromise to the entire renal functional mass
- This usually resolves after withdrawal of the drug.
- Restoring the renal blood supply in such cases can recover the ability to use these drugs for blood pressure control.
- Significant variability of serum creatinine concentration that may be due to changes in volume status
- A rapid rise in arterial pressure associated with sudden development of left ventricular failure (“flash pulmonary edema”).
  - This finding is more common with renal artery stenosis because bilateral disease is also associated with diuretic resistance and sympathetic adrenergic activation
- Deterioration of kidney function after placement of an endovascular aortic stent graft– IATROGENIC
  - EVAR with isolated L renal coverage -> splenal renal bypass, ligate splenic artery distally and then perform end-to-end anastomosis to renal artery. [Benjamin and Hansen, 2019]

#### **So it matters which kidney is affected or if it's one or both?**

Yes, effect of the stenosis may not be clinically apparent due to compensatory function of the unaffected contralateral kidney

Most cases of renal artery stenosis affect one side much more than the other; one kidney is affected with the second kidney being essentially normal, hence the designation “unilateral” disease.

- In patients with unilateral renal artery stenosis, contralateral kidney is damaged by uncontrolled hypertension and circulating angiotensin II and aldosterone. [Rickey and Geary, 2019]

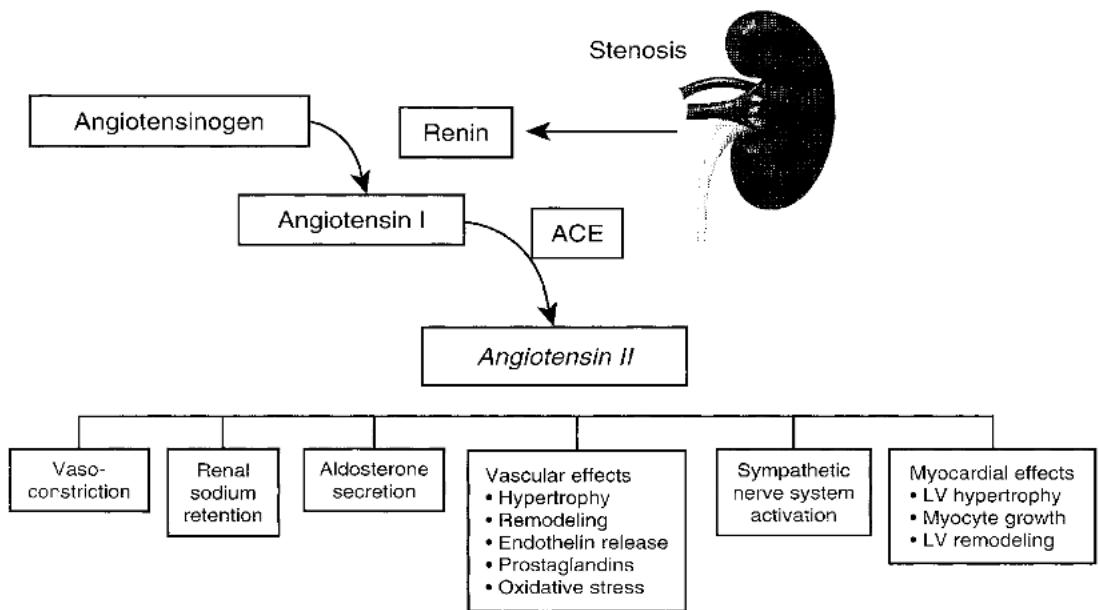
Patients who are diagnosed with ischemic nephropathy usually have high-grade stenosis of both renal arteries or stenosis to a solitary functioning kidney.

- It is this subpopulation of the disease that merits specific consideration because of its additional contribution to fluid retention, loss of kidney function, and congestive heart failure.

#### **What are the pathophysiological mechanisms at play here?**

Like we said, first you need a flow limitation. We mentioned several, but by in large There are two major causes of renal artery stenosis:

1. Atherosclerosis – you'll generally see this in patients over 45 years old, likely with known PVD/CAD—though it can occur as an isolated renal lesion—usually involving the aortic orifice or the proximal main renal artery.
2. Fibromuscular dysplasia – These patients are most often women under the age of 50 years and typically involves the mid- or distal main renal artery or the intrarenal branches.
  - Rarely iatrogenic from malposition or migration of endovascular aortic stent grafts over the renal orifices.
3. Takayasu's arteritis - although a rare cause of renovascular hypertension, this is the most common presentation of Takayasu's arteritis (60%). First line therapy is open revascularization. [Ehlert and Abularage, 2019a, Weaver et al., 2004, Zhu et al., 2012]



I think the flow limitation part has been well established. What next?

Renal (especially bilateral) hypoperfusion induces activation of the renin-angiotensin-aldosterone system which increases vascular tone and impairs

sodium excretion resulting in expansion of the extracellular fluid volume.

## 7.2 Medical Management and Evaluation

**Can't we just treat their hypertension and give these patients an ACE inhibitor at this point?**

Sure. And oftentimes we do. In fact, many of these patients can be treated with medical therapy without loss of function or irreversible fibrosis, sometimes for many years

- Studies in human subjects demonstrate that, despite a moderate reduction in renal perfusion pressure (up to 40 %) and in renal blood flow (mean 30 %), glomerular filtration is reduced but tissue oxygenation within the kidney cortex and medulla can adapt without the development of severe hypoxia.
- But this only works to an extent.

**Explain that...**

As the hypertension is treated, we're lowering the pressure gradient across the stenosis and can actually increase the degree of renal malperfusion and worsen the renal function.

- Oftentimes this loss of kidney function is a reversible consequence of antihypertensive therapy but it to some degree limits our ability to control the hypertension medically without causing further damage to the kidneys.... And it can also reflect progressive narrowing of the renal arteries and/or progressive intrinsic kidney disease as more advanced vascular occlusion, corresponding to a 70 to 80% narrowing of the renal artery, leads to demonstrable cortical hypoxia.

**Can we tell who has cortical hypoxia through diagnostic tests?**

A: To some degree. Cortical perfusion can be measured by blood oxygen level dependent magnetic resonance (BOLD-MR). Additionally, inflammatory markers sampled from renal veins of stenotic kidneys correlated strongly with the degree of hypoxia (as measured by BOLD-MR), particularly after correction of the stenosis with angioplasty

**So we have a patient with evidence of malperfused kidneys, either through worsening renal function or uncontrolled hypertension, with known discrete stenoses, and we even got a BOLD-MRI which confirms it. Let's just revascularize them and be done with it?**

Not so fast. Although vascular stenosis or occlusion can initiate these processes, long-standing ischemia causes parenchymal injury characterized by inflammation and fibrosis which eventually becomes an irreversible process. At some

point, restoring renal blood flow provides no recovery of kidney function or clinical benefit.

**So how can we determine who has CKD or hypertension due to renovascular stenosis that we can actually help?**

This is probably the most important question since in this whole disease process.

- To start, if a patient has the clinical manifestations of ischemic nephropathy or renovascular hypertension as we discussed above, a presumptive diagnosis of ischemic nephropathy can be made if there is radiologic documentation of significant stenosis (usually more than 70 % luminal occlusion) of both renal arteries or of one renal artery to a solitary functioning kidney.

**But how do we know the vascular occlusive disease posing critical hemodynamic limitation to kidney function?**

- Generally, luminal occlusion of at least 60 to 75 % is required to limit blood flow and reduce perfusion pressure
- This degree of stenosis is usually associated with a measurable translesional “pull-back” pressure gradient of 10 to 15 mmHg.
- Doppler ultrasound criteria [Hoffmann et al., 1991, Zierler and Dawson, 2016]
  - Peak systolic velocities above >170 cm/sec with post stenotic turbulence to identify less than 60 % luminal stenosis.
  - Renal aortic ratio >3.5 required to diagnose greater than 60% stenosis.
  - Elevated velocities can be seen with tortuosity, but this should be able to be confirmed with B-mode.
- Identifiable levels of cortical hypoxia (measured by blood oxygen level dependent magnetic resonance BOLD-MR) are usually associated with translesional velocities above 385 cm/sec or reduction of single kidney glomerular filtration rate (GFR) in the range of 20 to 25 mL/min.
- MRA can be used for evaluation
  - Pro - no radiation, good imaging of distal renal arteries, no degradation from ostial calcium.
  - Cons - requires GAD -> interstitial fibrosis in CKD [@galanNephrogenicSystemicFibrosis] and degrades with motion and respiration [Nelson et al., 1999]

**Most importantly: is the condition of the kidneys such that restoring renal blood flow is likely to benefit function?**

Short answer, we still can't be certain.

Long answer, we can at least have some idea by considering the renal resistive index, the six-month trajectory of kidney function, and the size of the kidneys or by performing a kidney biopsy (which is not usually done).

- None of these factors predict the outcome of revascularization with certainty.
- Improved and validated methods to evaluate the salvageability of kidney function in this disorder are greatly needed and are the holy grail of this disease process.

**Let's go through some of these:**

Renal Resistive index:

Some studies indicate that elevated resistive indices in segmental vessels (above 0.80) measured by duplex ultrasound denote poor prognosis for renal recovery while a low resistive index is a favorable sign.

Trajectory of kidney function

The most consistent predictor of good recovery of kidney function after revascularization has been a recent deterioration of kidney function (ie, in the prior six to twelve months). Similarly recent progression of hypertension is a sign of likely good recovery.

Kidney size

Very small kidneys (less than 8 cm in longest diameter) are usually considered unlikely to recover after revascularization.

Kidney biopsy

Previous studies suggest that biopsy demonstrating preexisting atheroembolic changes and interstitial fibrosis indicate a limited potential for recovery.

- Biopsies are not usually performed.

Comparison of kidney morphology with kidney function

Some investigators have recommended assessing morphologic parameters, such as renal parenchymal volume and cortical thickness with MRI, and comparing these parameters with kidney function measured by radionuclide scanning

- In a stenotic kidney, apparently normal morphology combined with reduced function may indicate a “hibernating kidney” that could be salvaged with revascularization.

**So how do we get a definitive diagnosis?**

A definitive diagnosis is not usually made before revascularization. In practice, confirmation of the diagnosis is based upon stabilization or improvement of the GFR after successful revascularization.

### 7.3 Operative Management

Now we think our patient's renal artery stenosis maybe is causing hypertension or decline in renal function and we can possibly reverse it... how do we treat it?

For starters, all of these patients should receive medical therapy to control their hypertension in addition to routine CKD care and surveillance. They need to be aggressively treated for secondary prevention of cardiovascular morbidity with aspirin, statins, cessation of smoking, and, in patients with diabetes, glycemic control.

Second, once diagnosis has been made we have 2 therapeutic alternatives... Which are?

First, medical therapy alone- this generally involves ACE-I or ARB and as we discussed.

RAS can worsen ACE-I and ARB induced renal dysfunction due to systemic hypotension, efferent arterial vasodilation, and reduced glomerular hydrostatic pressure, in turn lowering GFR. [[Rickey and Geary, 2019, Schoolwerth et al., 2001]]

Okay, in other words we can have chronic normalization of the systemic pressure that might eventually lead to ischemic atrophy due to the reduced renal perfusion pressure distal to the stenosis? Any other concerns with medical management alone?

- We're addressing or prevention progression of stenosis in those with atherosclerotic disease.

Since this isn't a vascular medicine podcast, what's our other option?

Procedural intervention (open or endovascular) along with medical therapy.

Now you're talking. Who should we fix operatively?

Some but not all patients should undergo revascularization, Patient selection single most important factor.

Depends upon the hemodynamic severity and likely recoverability of kidney function

You mentioned recoverability before, can you once again touch on some recoverability indicators?

- A short duration of blood pressure elevation prior to the diagnosis of renovascular disease, since this is the strongest clinical predictor of a fall in blood pressure after renal revascularization
- Failure of optimal medical therapy to control the blood pressure

- Intolerance to optimal medical therapy (eg, deterioration of renal function during antihypertensive drug therapy)
- Recurrent flash pulmonary edema and/or refractory heart failure
- Otherwise unexplained progressive renal insufficiency, particularly if proteinuria is absent
- CKD stage 3a and 3b most likely to benefit from revascularization. [Singer et al., 2009]
  - Lower GFR likely to progress to ESRD
- Degree of stenosis, age, pre-procedure BP control and meds are not associated with improvement. [Textor et al., 2013]

**But do we have any good data proving our interventions help?**

This is where things can get muddy.

Early on, observational studies demonstrated a high rate of procedural success with percutaneous transluminal renal angioplasty (PTRA) and stent placement (~85%) in patients with ostial atherosclerotic disease, as well as a high rate of clinical success measured by improvements in blood pressure and kidney function in 50 to 75 % of subjects.

**Anything better than observational studies?**

Unfortunately, randomized trials showed no additional benefit from stenting when added to medical therapy with respect to blood pressure control, renal function, cardiovascular events, and mortality. But these studies have their own limitations.

**The one we keep hearing about is the CORAL trial. Tell me about that.**

- CORAL trial [Cooper et al., 2014]
  - Cardiovascular Outcomes in Renal Atherosclerotic Lesions (CORAL) trial
  - 947 patients (80 % had unilateral disease) who met the following two criteria:
    - \* Unilateral or bilateral atherosclerotic renal artery stenosis >60 % if diagnosed with conventional angiography, peak systolic velocity >300 cm/second if diagnosed by duplex Doppler ultrasonography, Luminal narrowing >80 % if diagnosed with magnetic resonance angiography or computerized tomography angiography (or >70 % with additional evidence of renal ischemia)
    - \* Systolic hypertension despite two or more antihypertensive medications and/or an estimated glomerular filtration rate (eGFR) <60 mL/min/1.73 m<sup>2</sup> that was presumably due to the stenosis.

- \* All patients received antiplatelet therapy plus best medical therapy including ARB
- Revascularization had no additional effect on the primary outcome (a composite of cardiovascular or renal death, stroke, myocardial infarction, hospitalization for heart failure, a reduction in eGFR by more than 30 %, or end-stage renal disease) as compared with medical therapy alone (35.1 versus 35.8 %).
- No effect on any of the individual components of the primary outcome.
- Low procedural complication rate ~2%
- Similar findings in the ASTRAL trial [Investigators, 2009]

**Well, that sounds pretty convincing.**

- Limitations on existing treatment data:
  - Considerable selection bias – For the most part, the patients enrolled in these trials did **not** meet the criteria for selecting patients likely to benefit from intervention (eg, short duration of blood pressure elevation, hypertension resistant to medical therapy, recurrent flash pulmonary edema):
    - \* CORAL [Cooper et al., 2014]
      - Patients hospitalized for heart failure within 30 days of screening for the trial were excluded, thereby limiting the number of trial participants with recurrent flash pulmonary edema.
      - Mean number of antihypertensive medications used by CORAL participants at baseline was 2.1- many had not failed optimal medical therapy
      - More than 25 % had controlled blood pressure upon entry into the trial.
      - Mortality and event rates lower than in most previous registries, suggesting that many high-risk patients were not enrolled.
    - \* ASTRAL [Investigators, 2009]
      - Large number of patients had stenoses that were probably not clinically significant (50 to 70 %), and patients were excluded if their primary doctors felt that they “definitely” needed revascularization.
    - Results of the trials differ substantially from observational reports of “high-risk” subsets

- \* For the most part, patients selected by their treating clinicians to undergo revascularization have derived greater benefit from revascularization than did patients enrolled in the trials who were randomly assigned to revascularization

### 7.3.1 Endovascular Therapy

We've determined our patient is an appropriate candidate for intervention, and we don't fully buy into CORAL, what can we do?

- Percutaneous renal angioplasty/stenting in addition to medical therapy
  - Most commonly employed if technically feasible.
  - Most amenable lesions to angioplasty are those producing incomplete occlusion in the main renal artery.
    - \* Total occlusions and ostial lesions extending into aorta generally do not respond well to angioplasty alone due to elastic recoil.
  - Quick results: maximum antihypertensive response is generally observed at 48 hours after the procedure
    - \* But BP levels and antihypertensive drug requirements often change over subsequent weeks
  - In general, the effects of revascularization on blood pressure were greater in bilateral disease, but effects on renal function and mortality did not differ in those with bilateral as compared with unilateral stenosis .
  - Most atherosclerotic lesions are now treated with primary stenting to avoid rapid development of restenosis.
    - \* A higher initial primary success rate, defined as less than 50 % stenosis (88 versus 57 %).
    - \* At six months, a higher patency rate (75 versus 29 %) and a lower restenosis rate (14 versus 48 %).
    - \* Twelve patients assigned to PTCA alone underwent stenting because of treatment failure within six months. These patients had a similar blood pressure response as those initially treated with stenting.
  - Performing a renal angiogram [Edwards and Cooper, 2019]
    1. Supine with arms overhead or straight out to the sides
    2. LAO 15-20deg
    3. Flush catheter placed just above the renals

4. Breath hold, high frame rate and non-DSA due unavoidable patient movement

### What about complications?

- Complication rate with percutaneous transluminal renal angioplasty with or without stenting is between 5 and 15 %
  - Mostly minor: puncture site hematoma and renal artery dissection.
  - Serious complications more rare: renal artery thrombosis or perforation, AKI 2/2 atheroembolic disease (~1%) or radiocontrast agent injury.
  - Mortality exceedingly rare

### Outcomes data

- In the correct patient population:
  - Unilateral disease
    - \* PTRA alone results in normalization of blood pressure (removal of antihypertensive drug therapy) ~8-20%
    - \* Some improvement 50-60%
    - \* Failure rate ~20-30%
    - \* Restenosis rate of 8 to 30 % at two years (without stent)
    - \* Better results with unilateral fibromuscular disease.
    - \* Less consistent for patients with chronic hypertension compared with patients who have an acute elevation in blood pressure
  - Bilateral disease
    - \* 25-30% will recover kidney function to a meaningful degree, sometimes avoiding progression to end-stage kidney disease (ESKD) and/or the need for renal replacement therapy.
    - \* ~50% will have little immediate change in kidney function but will “stabilize”
    - \* ~20% will have a progressive deterioration of kidney function, sometimes related to the procedure

### Guidelines

- 2005 ACC/AHA guidelines on peripheral artery disease recommends that a stent be placed in patients undergoing PTRA for treatment of atherosclerotic renal artery stenosis [Hirsch et al., 2006]
  - PTRA without stent placement is rarely performed unless the anatomy precludes stenting.

- POBA without stenting is generally less successful and associated with more complications (eg atheroemboli)

**How durable is PTA/stenting?**

- Restenosis
  - ~11-17%
    - \* 11-39% during the first one to two years
  - Detected as a rise in blood pressure requiring more intensive therapy
  - Angioplasty/stenting injures the vascular endothelium, which may result in restenosis.
  - Symptomatic stenosis leading to a rise in blood pressure or a fall in GFR are less common and are reported in 10 to 20 % of patients

**How do you follow these patients after stenting?**

- Follow-up of patients who have had a renal artery stent should include serial measurements of blood pressure and estimation of GFR.
  - Post-stent duplex ultrasound @2-4weeks with
  - Repeated examinations on a quarterly basis (not much data)
  - Patients who develop an increase in pressure or reduced GFR after stenting should undergo duplex ultrasonography to identify restenosis
  - Retreatment with angioplasty with or without repeat stenting can be attempted, but the restenosis rate after repeat angioplasty is increased.
    - \* Surgical reconstruction may be pursued in patients with recurrent episodes of restenosis and loss of kidney function.

### 7.3.2 Open Surgery

**What about an open operation?**

- Surgical revascularization used in addition to medical therapy
  - Less common since the widespread application of effective antihypertensive drug therapy and endovascular stents (mid 90s)

**So who still gets open repair? [Benjamin and Hansen, 2019]**

- Younger patients
- Unfavorable anatomy (i.e. occlusion or branch disease)
- Failures of endovascular therapy (i.e. in stent restenosis)
- Need for concomittent aortic revascularization.

**How do we do it?**

- Involves bypassing the stenotic segment or of removing a small atrophic kidney with nearly complete arterial occlusion.
  - From the aorta or hepatorenal or splenorenal bypass to avoid diseased aorta.
  - Bilateral: either bilateral repair or unilateral repair with contralateral nephrectomy of a nonfunctioning, atrophic kidney.
- Bilateral ostial disease in a young patient can be treated with transverse arteriotomy and bilateral renal endarterectomy. Close primarily or with PTFE/polyester patch. Second line is bilateral bypass with GVS. [Benjamin and Hansen, 2019]

#### **How do outcomes compare to PTA/stenting?**

- Equally or more effective than PTCA in the treatment of atherosclerotic disease, with cure of or improvement in the hypertension occurring in 80 to 95 % of patients.
  - Cure of hypertension after surgery is most likely in patients who have been hypertensive for less than five years
- Lack of complete response was usually associated with one of two factors:
  - Presence of underlying primary/essential hypertension
  - Development of intrarenal vascular disease due to exposure of the contralateral kidney to the elevated blood pressure.

#### **Guidelines recommendations?**

- 2005 American College of Cardiology/American Heart Association (ACC/AHA) guidelines [Hirsch et al., 2006]
  - Open surgery in patients with atherosclerotic renal artery stenosis largely restricted to those who have multiple small renal arteries, have early primary branching of the main renal artery, require aortic reconstruction near the renal arteries for other indications (eg, aneurysm repair or severe aortoiliac occlusive disease), or to avoid manipulation of a highly diseased aorta or failed endovascular stents (using specific surgical techniques, including splenorenal, ileorenal, or hepatorenal bypass procedures).

#### **So why not do it instead of stent?**

- In-hospital mortality: ~3-10 % in high volume centers
  - Risk factors diffuse atherosclerosis, advanced age, chronic kidney disease, heart failure, or chronic lung disease.
  - No deaths in 105 procedures for fibromuscular dysplasia (FMD).

## 7.4 Fibromuscular Dysplasia (FMD)

You mentioned FMD as a cause of renovascular hypertension, tell me more about that...

Fibromuscular dysplasia (FMD) is a noninflammatory, nonatherosclerotic disorder that leads to arterial stenosis, occlusion, aneurysm, dissection, and arterial tortuosity.[Olin and Sealove, 2011]

- Virtually always diagnosed radiographically – formerly pathologically, but rarely sent for specimen in modern diagnosis or treatment

How do we classify it?

- Most commonly classified by angiographic appearance:
  - Multifocal FMD (more common)
    - \* angiographic appearance of a “string of beads.”
    - \* corresponds pathologically to medial fibroplasia, the most common histologic type, and to perimedial fibroplasia, which is less common.
  - Focal FMD (less common)
    - \* angiographic appearance of a “circumferential or tubular stenosis”
    - \* corresponds pathologically to intimal fibroplasia but medial hyperplasia and periarterial hyperplasia may also have a focal appearance.
  - These two different angiographic subtypes of FMD (multifocal and focal) have different phenotypic presentations and natural history
    - \* Is FMD is, in fact, a single disease?

Where does it occur?

- Has been observed in nearly every arterial bed
- Involvement of the renal arteries ~75-80%
- Involvement of the extracranial cerebrovascular arteries (eg, carotid and vertebral arteries) ~75%
  - 2/3 of patients have multiple arteries involved.

Who has FMD?

- ~90% of cases in adults are in women.
  - No female predominance among children with FMD.
- Mean age at diagnosis was 52 years, with a range of 5 to 86 years

- In the past, it was believed that FMD was a disease of young women. However, older now know to make up a large proportion of affected
- 35-50% of cases in children and 5-10% of cases in adults under the age of 60 years with renovascular hypertension
- Often an incidental finding:
  - 4.4% of potential kidney donors had evidence of FMD.
  - CORAL trial:
    - \* FMD was discovered in 5.7% of the total study population (8.8% of enrolled females)

### **What causes FMD?**

The exact etiology of FMD remains unknown, but some mechanisms have been proposed

- Most often results from medial fibroplasia (60-90% of cases). Collagen deposits in the media result in elastic fibrils and fibromuscular ridges. [Olin et al., 2014]
- Genetics may play an important role in development
  - Some studies report autosomal mode of inheritance with variable penetrance
  - Potential association with a single nucleotide variant in the phosphatase and actin regulator 1 gene (PHACTR1)
  - Variant rs9349379 is also a risk locus for coronary artery disease, migraine headache, and cervical artery dissection.
- Predominance of young/childbearing age women hormonal influences are thought to play a role
  - Remains unproven.
- Mechanical factors such as stretch and trauma unproven.

### **Does FMD present differently than the atherosclerotic renovascular disease we talked about?**

- Varies widely depending on artery affected and as it results from:
- Ischemia related to stenosis
- Dissection and occlusion of major arteries (renal infarction, stroke, myocardial infarction)
- Rupture of aneurysms
- Embolization of intravascular thrombi from dissection or aneurysms

### **What are the common presenting symptoms and signs**

- Manifestations of renal FMD (eg, hypertension, flank pain) are more likely to occur in men, as are arterial dissections and aneurysms.
  - Most common presenting signs:
  - Hypertension – 67% (66% of women and 74 % of men)
- But overall hypertension is the most common manifestation of renal artery FMD in both genders
  - Flank pain and abdominal pain can result from ischemia, aneurysm rupture, or dissection of renal and mesenteric arteries, respectively.

**Are these dissections common?**

- High prevalence of aneurysm and/or dissection
  - Aneurysm (22%) and dissection (26%).
    - \* 34% of aneurysms were renal
    - \* 11% of dissections were renal
  - 42% had an aneurysm and/or dissection.

**So should we screen for these dissections in a patient with known FMD?**

- Every patient diagnosed with FMD should have one-time, head-to-pelvic CTA (or MRA) is an alternative.
  - CTA of the neck and head on one day followed one week later by CTA of the chest, abdomen, and pelvis

**When should we suspect FMD?**

- Hypertension (particularly in a woman under the age of 60 years) with findings that would prompt an evaluation for secondary hypertension:
  - Severe or resistant hypertension.
  - Onset of hypertension before the age of 35 years.
  - A sudden rise in blood pressure over a previously stable baseline.
  - A significant increase in the serum creatinine concentration after the institution of therapy with an angiotensin-converting enzyme (ACE) inhibitor or angiotensin receptor blocker (ARB) in the absence of an excessive reduction in blood pressure.
  - An epigastric/abdominal bruit.
- Renal artery dissection (or carotid, vertebral, coronary)
- Aneurysm in a visceral, carotid, vertebral, or intracranial vessel.
- Renal infarction.

**How do we diagnose this and/or distinguish it from renovascular atherosclerotic disease?**

- Confirmed by diagnostic imaging that reveals consistent findings
- Noninvasive imaging test is usually performed first. This includes CTA, MRA, and Duplex ultrasound.

**Let talk about CTA...**

- CTA is preferable due to higher spatial resolution than MRA, less dependence upon technical expertise, and a shorter scan time
- Excellent diagnostic accuracy for FMD of the main renal arteries, although the sensitivity decreases when FMD is only present in the smaller branch renal arteries.
- Multirow detector CT scanners, which offer more rapid image acquisition, variable section thickness, three-dimensional rendering, diminished helical artifacts, and smaller contrast requirements, may gain an increased role in the diagnosis and follow-up of renal artery FMD

**What about MRA?**

- Inconsistent detection of FMD and is performed if CTA is contraindicated
- The spatial resolution in the branch vessels is not adequate, and artifact may occur, suggesting “beading” when none is present.
- May miss mild FMD.
- Can be useful for detecting aneurysms and dissections - can be difficult to diagnose on duplex ultrasonography.[Renaud et al., 2012]

**And finally, Duplex ultrasonography**

- Detects elevated blood flow velocities in the mid and distal portions of the renal artery, most common locations for FMD.
- Increased peak systolic velocity, turbulent blood flow, and tortuosity of the mid and distal artery.
  - % diameter stenosis reports less helpful and usually inaccurate
- lowest spatial resolution of all of the cross-sectional imaging modalities
- most operator dependence
- first choice only in high-volume centers with extensive expertise in this technique

**What about non invasive testing?**

- DSA is performed in patients if there is a high clinical suspicion of FMD, and treatment with revascularization is planned if a stenosis is found.

- Can improve visualization of the arteries by eliminating background soft tissue and bone and has higher spatial resolution than any of the other imaging modalities
- Can measure the pressure gradient across the stenosis
  - \* Pressure decrease threshold of 10 % or more of the mean pressure should be used to decide whether a lesion is hemodynamically significant
  - \* IVUS and optical coherence tomography (OCT) can be helpful in determining if a dissection or intramural hematoma is present as well as help to determine if angioplasty has improved the stenosis.
- Negative DSA excludes a diagnosis of FMD in the vascular bed that was imaged.

**Any place for pathologic diagnosis in modern therapy?**

- Histopathology (and histologic classification) is **no longer** part of the diagnosis.
  - Only in the rare patient who requires surgical revascularization or resection of an aneurysm.

**So how do we treat this, what warrants intervention and are those interventions different than what we offer atherosclerotic disease of the renal arteries?**

- All patients with FMD should be placed on antiplatelet therapy (ASA) unless otherwise contraindicated
- Antihypertensive therapy
  - Most patients will require antihypertensive therapy, even if they undergo revascularization.
  - Majority of patients with **focal** FMD have their blood pressure cured with angioplasty

**But what about revascularization?**

- Revascularization Goal: control of hypertension
  - BP can be controlled in most adults with multifocal FMD with a mean of two antihypertensive medications
  - Weigh risks and benefits in well controlled hypertension.
- **No** randomized trials comparing revascularization with medical therapy

**Well then who do we treat?**

- Recent-onset hypertension, with goal to cure hypertension.

- Resistant hypertension despite compliance with an appropriate three-drug regimen.
- Patients unable to tolerate antihypertensive medications or who are non-compliant with their medication regimen.
- Adults with bilateral renal FMD, or unilateral renal FMD to a single functioning kidney, and unexplained progressive renal insufficiency thought to result from renal artery stenosis
- Hypertensive children.
- may be at higher risk than adults for progressive renal parenchymal loss, and therefore could benefit from revascularization even if their hypertension can be well-controlled with one or two antihypertensive medications.

**And what kind of results do we get with revascularization?**

- Hypertension is cured or improved following revascularization in a large proportion of patients with FMD.
  - Much better than 2/2 atherosclerosis
  - Varies considerably from study to study, although hypertension control improves in most patients and depends in large part upon the definition of cure.
  - Not good data on stabilization of either GFR or renal size in patients with FMD.

What options do we have in terms of revascularization?

**Angioplasty and open surgery**

Do we have good results treating FMD with these?

- Patients most often treated with angioplasty alone with good success. [Davies et al., 2008, Jenkins et al., 2015]
- Improvement in blood pressure (including those with and without cure) was similar with PTA as compared with surgery (86 versus 88 %).
- Older age and longer duration of hypertension prior to revascularization were significantly associated with a lower cure rate.

**How do these compare?**

- PTA achieves similar technical success and is associated with a lower risk of adverse events in observational studies
- Most patients with FMD who are selected for renal revascularization have PTA rather than surgery
- Major adverse events were more frequent with surgery (15 versus 6 %).

**So why choose open surgery?**

- Cure rates were higher with surgery (54 versus 36 %).
- Surgery rather than PTA if PTA fails or if the arterial anatomy is not amenable to PTA
  - Patients with small renal arteries (<4 mm), with branch renal artery disease, or with extensive intimal fibroplasia.

**So how do we perform Percutaneous transluminal angioplasty for FMD?**

- Without stent placement... unlike PTA for atherosclerotic RAS

**Why not place a stent?**

- Patients do very well with angioplasty alone, no reason to place a stent.
  - Lesion is so fibrotic that the pressure gradient cannot be obliterated with an angioplasty, a stent will not correct this problem
    - \* Such patients should be referred for surgery.
- Usually have stenoses in the mid and distal portions of the artery rather than at the ostium or proximal portion (as occurs with atherosclerosis).
  - Should surgical revascularization become necessary due, for example, to in-stent restenosis, patients may require more complex branch repair to bypass the occluded stent since the stent often covers the renal artery up to the point of the first intrarenal branch.

**Do we ever place stents?**

- Stents placed when a dissection results from the performance of PTA or in the rare instance in which a perforation of the renal artery occurs during angioplasty.

**And we're getting good outcomes with PTA alone?**

- Technical (angiographic) success rates for PTA 83-100
- Rate of restenosis 12-34% over follow-up intervals of six months to two years
  - Difficult to determine if patients with FMD develop restenosis, or if the lesion was not completely treated correctly the first time.
  - Not necessarily associated with recurrent hypertension.

**But generally we can achieve significant and sustained reductions in systolic blood pressure, diastolic blood pressure, serum creatinine, and number of antihypertensive agents.**

- Systolic blood pressure response was better in patients with FMD affecting the main renal artery than in patients with branch vessel involvement.

**Any specific technical tips?**

- Cutting balloon angioplasty should be avoided
  - Increased risk of rupture
- Post angioplasty visual inspection alone is **not** accurate.
  - Measure pressure differential using a pressure guidewire, with a mean gradient of <5 mmHg across the treated segment suggesting a satisfactory result
    - \* Measure before and after angioplasty
  - Post-procedure renal duplex scanning
    - \* Degree of turbulence is less prominent, and velocity elevation in the mid-distal renal artery returns to normal.
  - Intravascular ultrasound or optical coherence tomography (OCT) is occasionally used to evaluate the elimination or reduction of various endoluminal defects.

**What should we do if it doesn't work?**

- If either has no improvement in blood pressure or an initial improvement followed by recurrence, repeat angiogram and PTA.
  - Restenosis may actually represent inadequate angioplasty during the first procedure
- Persistent HTN despite technically successful PTA suggests that the cause of hypertension is unrelated to fibromuscular disease or is related to small vessel disease within the kidney (nephrosclerosis) due to longstanding hypertension.

**What kind of complications do we see after this?**

- Mostly related to vascular access
- Rarely: renal artery perforation, dissection, or segmental renal infarction may occur.
- Decreasing over time- 16 % in 1998 to 3 % in 2001

**Ok, lets switch gears to open revascularization?**

- Aortorenal bypass with a saphenous vein graft is the most common technique
  - Artificial graft material used occasionally

**For everyone? What about for pediatric patients?**

- Pediatric patients: hypogastric artery grafts are used or else aortic reimplantation of the renal artery is performed because vein grafts become aneurysmal

**How does this compare again to PTA?**

- Similar success rates compared to PTA (82-89% patency) but with higher morbidity.
  - Perioperative mortality appears to be very low (~1.2%)
  - Usually limited to complex cases so success and complication would probably be higher if simpler cases were included.

**What's Monitoring and follow-up look like for these patients?**

- Medical management only:
  - Renal artery stenosis and kidney dysfunction may progress despite good blood pressure control
    - \* Mostly in patients with focal FMD and intimal fibroplasia
  - Every patient with FMD should have measurement of serum creatinine and renal artery duplex ultrasound every 12 months.

**And After revascularization?**

- Duplex ultrasonography and serum creatinine measurements performed on the first office visit post procedure, then every six months for two years, and then yearly, if stable.
- With worsening or new hypertension, or unexplained increase in the serum creatinine, he or she should be imaged at that time with duplex ultrasound (or CTA if the ultrasound is equivocal or poor quality).

## 7.5 Renal Artery Aneurysms

Ok, that's a pretty good review, but let's switch gears and talk about renal artery aneurysms

### 7.5.1 Demographics

- Renal artery aneurysms are rare - Autopsy studies have revealed an incidence of 0.01% to 0.09%.
- Females > males, although females = males with FMD excluded
- Although arteriosclerotic changes have been identified in most aneurysms in patients with multiple lesions, this is not a uniform finding, suggesting that arteriosclerosis may not be the most important factor in the genesis of renal artery aneurysms.
- More likely due to a congenital medial degenerative process with weakness of the elastic lamina.

- Fibromuscular dysplasia (FMD) is often a direct contributor to the development of an aneurysm.
  - Medial fibroplasia is typically associated with multiple stenoses and post-stenotic dilatation of the distal two thirds of the renal artery.
  - Renal artery aneurysms in association with FMD are generally only a few millimeters in diameter.
  - The typical angiographic appearance of a renal artery involved with medial fibroplasia is a “string of beads.”
- A rare cause of renal artery aneurysms is Ehlers-Danlos’ syndrome.
  - This disorder is associated with extreme arterial fragility and spontaneous rupture.

### 7.5.2 Anatomy

- Most frequent site of involvement is primary bifurcation, intraparenchymal (<10%)
- Most are saccular
- Right slightly more common than left, bilateral 10%
- 90% are extraparenchymal

### 7.5.3 Presentation

- Majority are associated with hypertension (70%) [Coleman and Stanley, 2015]
- 10% mortality
- 90% risk of kidney loss
- Less than 3% rupture.

### 7.5.4 Management

#### Size criteria currently controversial\*

- Uncontrolled hypertension is an indication for repair when smaller than 2.5cm. [Coleman and Stanley, 2015]
- VLFDC recently proposed a 3cm threshold for asymptomatic renal artery aneurysm. [Klausner et al., 2015]
- Many aneurysms with circumferential calcification which could offer protection against rupture

In an elderly patient, observation of this aneurysm with Duplex surveillance is the appropriate treatment.

For larger aneurysms in younger patients, aneurysmorrhaphy with primary repair or patching, interposition grafting, or bypass can be performed with low mortality. [Coleman and Stanley, 2015]

- Comparison of ex vivo or insitu renal artery reconstruction have shown no difference in mortality, morbidity, LOS or reoperation.

Endovascular techniques such as coiling have been reported to be successful in treating these saccular aneurysms; however, most aneurysms occur at branch points making covered stent placement difficult. [Coleman and Stanley, 2015]

- Renal artery dissection caused by guide wires or catheters can occur, but is rare.

## 7.6 Renal Artery Dissection

Renal ischemia if patient has new hypertension, flank pain, hematuria and proteinuria. [Müller et al., 2003]

Endo avoided if branch involvement.

Bypass, in situ repair, auto-transplant if renal branch involvement and possibility of renal salvage.

Nephrectomy required in uncontrolled hypertension, extensive dissection and irreversible ischemia.

## 7.7 Renal Vein Thrombosis

### 7.7.1 Diagnosis

CT scan is best. Difficult to visualize native renal vein on duplex imaging. [Asghar et al., 2007, Velazquez-ramirez and Corriere, 2019]

### 7.7.2 Management

- Renal vein thrombosis initially managed with heparin, then warfarin for 6mo. [[Asghar et al., 2007, Velazquez-ramirez and Corriere, 2019]]
- Thrombectomy or thrombolysis reserved for acutely threatened kidney in young patient, complication of AC or thrombosis of solitary kidney with renal failure.
- Nephrectomy for post-infarct hemorrhage.
- Thrombolysis requires arterial and venous access - venous access to debulk and arterial access to drip and clear small intra-paranchymal veins.

## 7.8 Renal Arteriovenous Fistula

Relatively common complication of renal biopsy (9-18%). [Schwarz et al., 2008]

Presentation - bruit over kidney, renal impairment, varicocele, hematuria and abdominal pain. [Hunter et al., 2019]

Diagnose with duplex, CTA or MRA.

- Duplex will show marked turbulence, elevated PSV and high end diastolic flow, low resistive index. [Ozbek et al., 1995]

Management

- Indications for treatment [Merkus et al., 2005, Morimoto et al., 1995]
  - Gross hematuria requiring blood transfusion
  - High output cardiac failure
  - Worsening hypertension or renal failure
  - Persistence at >1yr
- Treated most often with angiogram, covered stent or highly selective micro-coil embolization. [Ginat et al., 2009, Saliou et al., 1998]

# Chapter 8

## Thoracic Aorta

UNDER CONSTRUCTION - This Chapter is Currently in DRAFT mode

### 8.1 Aortic Dissection

01 Nov 2021: Matt Spreadbury, MD; Adham Elmously, MD; Einar Brevik, MD and Joseph Lombardi, MD

#### 8.1.1 Demographics

**What is an aortic dissection?**

It's when a tear occurs in the intima that results in separation of layers of the intima and media and allows blood to flow through the false lumen.

**How common are they and how serious are they?**

Acute dissections occur around 3/100000 - 2-3x more common than ruptured aortic aneurysm. For Type A dissections, early mortality 1-2% per hour - if untreated, 20% die within 6 hours, 50% within 24 hours, 70% first week.

Main cause of death in type A is aortic rupture into the pericardium, acute aortic regurgitation, and coronary ostia compromise. While patients with descending thoracic aortic dissections are more likely to die from end organ compromise due to obstruction of visceral or extremity vessels in the acute phase of the disease.

**The time frame is also important.**

- Hyperacute <24 hours
- Acute < 2 weeks
- Subacute 2 weeks – 3 months -> TEVAR

- Chronic >3 months -> Chronic aneurysmal degeneration/ partial false lumen thrombosis (highest risk) = operative treatment

### 8.1.1.1 Classification and Terminology

**When we think about aortic dissections there are a few classifications, how can we break it down?**

Historically, there are the Stanford and Debakey Criteria.

Anatomical Stanford

- Type A - involves the ascending aorta, 2/3 (most common)
- Type B - arises from distal to L subclavian, 1/3

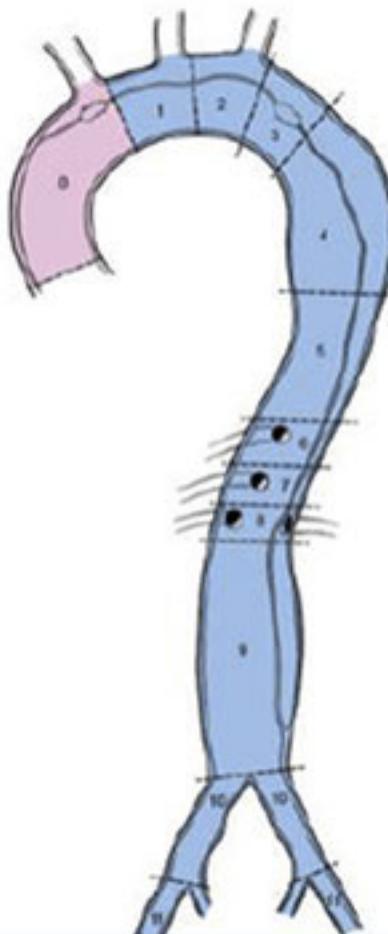
Debakey

- A
  - 1 - ascending + descending
  - 2 - ascending only
- B - distal or at the LSCA.
  - 3a - Descending aorta above diaphragm
  - 3b - Descending aorta above and below diaphragm

**How about the new system proposed by Dr Lombardi, the SVS-STS classification system?**

The new system published in 2020 keeps A and B and adds a number system which divides the aorta into zones from 0 proximally to 12 distally in the mid SFA. [Lombardi et al., 2020b]

## Society for Vascular Surgery and S Reporting Standards for Typ



| Type   | Proximal Extent | Distal Extent |
|--|-----------------|---------------|
| <b>A<sub>D</sub></b>                                     | 0               | 0             |
|  | 1               | 1             |
| Entry tear:<br><b>Zone 0</b>                             | 2               | 2             |
|  | 3               | 3             |
|  | 4               | 4             |
| <b>B<sub>PD</sub></b>                                    | 5               | 5             |
|  | 6               | 6             |
| Entry tear:<br><b>≥Zone 1</b>                            | 7               | 7             |
|  | 8               | 8             |
| <b>I<sub>D</sub></b>                                     | 9               | 9             |
| Unidentified<br>entry tear<br>involving<br><b>Zone 0</b> | 10              | 10            |
|  | 11              | 11            |
|  | 12              | 12            |

- Type A is now JUST the ascending aorta to the innominate, also called Zone 0.

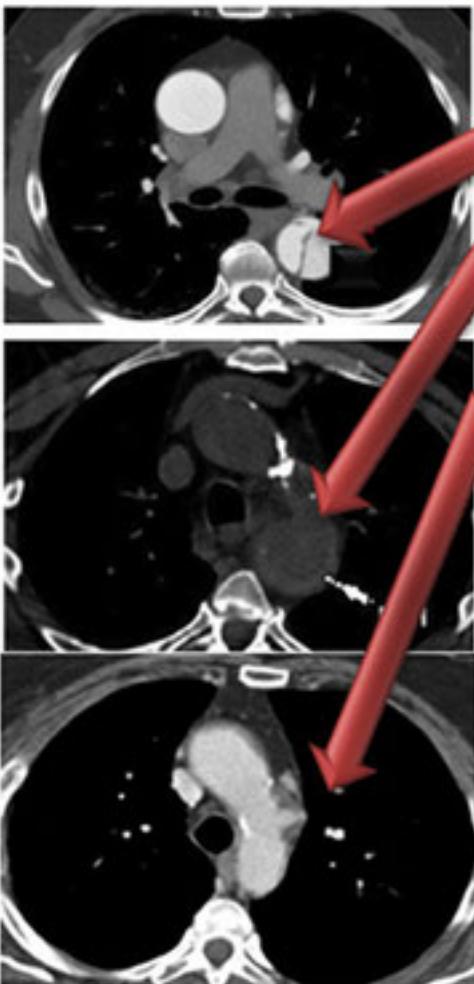
- Type B is now an entry tear in Zone 1 or greater and distally to whichever zone the dissection lands in.

#### 8.1.1.1.1 Penetrating Aortic Ulcer and Intramural Hematoma This anatomical classification is based on reading the CT angio. What else could we see on a CT angio that we have to know about?

So aside from the aortic dissection its self, you could see a bleb of contrast sticking out. That could be an **penetrating aortic ulcer**. That is an atherosclerotic plaque that penetrates the internal elastic lamina of the aortic wall.[Ciccone et al., 2016]

Another key finding can be an **intramural hematoma** which is a hyper-dense crescent shaped hemorrhage within the aortic wall. There is no identifiable direct communication between the true and false lumen. IMH are classified in the same way but with the abbreviation IMH p-d zones.

## Society for Vascular Surgery and Reporting Standards for Type I Aortic Dissection



|                                 |  |
|---------------------------------|--|
| <b>Aortic Dissection</b>        | Tear in the intima that results in the false lumen   |
| <b>Intramural Hematoma</b>      | There is no identifiable discrete tear; instead, there is a dense, crescent-shaped hematoma          |
| <b>Penetrating Aortic Ulcer</b> | Atherosclerotic lesion that penetrates the aortic wall. Often diagnosed in presence of a false lumen |

### Aortic Dissection Acuity

|                    |  |
|--------------------|--|
| <b>High Risk</b>   | Refractory pain or HTN<br>Bloody pleural effusion<br>Aortic diameter > 40 mm<br>Radiographic or angiographic malperfusion<br>Readmission<br>Entry tear: Lesser curve location<br>False lumen diameter > 22mm |
| <b>Complicated</b> | Rupture<br>Malperfusion  |



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What's the significance of these two in combination?

There is a higher chance of aortic rupture if a penetrating aortic ulcer is seen

with intramural hematoma.

**When a patient presents with an aortic dissection how can we classify them clinically?**

- Uncomplicated
  - Stable hemodynamics
  - No evidence of malperfusion
  - Pain controlled
- Complicated
  - End organ ischemia / malperfusjon
  - Rupture or impending rupture
- High risk for early complication or continued growth[van Bogerijen et al., 2014, Reutersberg et al., 2018]
  - Uncontrollable pain / hypertension
  - Bloody pleural effusion
  - Aortic diameter >40mm / False lumen diameter > 22mm
  - Readmission
  - Radiographic only malperfusion
  - Entry tear on the lesser curve
  - Number vessels originating from false lumen and length of dissection.[Brunkwall et al., 2014, Kamman et al., 2017, Nienaber et al., 2009]

**What is the danger of a false lumen? How does it lead to symptoms and malperfusion? Likewise which arteries commonly branch off the true lumen?**

The false lumen can lead to end organ ischemia as the intimal flap can cover the ostia of branching vessels. This can be a static or a dynamic obstruction.

Likewise it also leads to weakening in the wall of the aorta which can become a threatened rupture or rupture if the diameter of the false lumen is larger than 22mm.

The celiac trunk, SMA, right renal typically come off the true lumen. Left renal comes off the false.

Also the dissection most commonly goes down into the left common iliac rather than the right. You might be able to detect downstream effects of this on clinical exam with reduced left sided groin pulse.

The presence of multiple false lumens is associated with increased risk of aortic dissection related death.[Sueyoshi et al., 2013]

### **What kind of patients get aortic dissections?**

Hypertension (older patients) / cocaine or Meth (younger patients)

Marfans, Loeys-Dietz, Ehlers Danlos Type 4, Turners, arteritis, Bicuspid aortic valve.

We also have a traumatic cause of aortic dissections. That being called blunt thoracic aortic injury:

- Grade 1: intima tear
- Grade 2: IMH
- Grade 3: Pseudo aneurysm
- Grade 4: Aortic rupture.

#### **8.1.1.2 Presentation**

##### **How do these patients present?**

Signs and symptoms – Chest pain 90% tearing pain radiating between the shoulder blades.

Chest pain extending to the abdomen abdomen? Think mesenteric ischemia or aortic tear

Type A - Stroke 5-10%, Syncope 15%, tamponade, carotid dissection, paralysis.

Others: MI – Hypovolemic shock – leg ischemia

#### **8.1.2 Evaluation**

##### **What is the workup?**

Physical Exam – Asymmetric pulses / blood pressure differences / Diastolic murmur,

Investigations - CXR, EKG, D-dimer + Troponin, CTA, ECHO for type A.

The big distinction is to find out if this is a type A or type B because the treatment strategy is completely different.

- Type A need an emergent operation
- Type B starts with medical management, follow up CT angiogram +/- Trans esophageal echo in the OR. Reevaluate at 24 hours.

### 8.1.3 Management

#### **What are the details of Type A treatment?**

Operative treatment. 30% op mortality. Cardiothoracics take the lead on this one. However vascular surgeons should be involved in the management of type A as after the repair, a type A can become a functional type B.

Type B is in the realm of vascular surgery. What is the first management step after we have diagnosed a type B dissection?

Invasive impulse therapy. That means reducing the force of transmitted impulse down the aorta. Blood pressure goals of 100-120mmHg. Hr < 60bpm.

#### **How would you achieve that?**

Start with a beta-blocker (esmolol or labetalol) first then a vasodilator (nitroprusside). This is to stop the sympathetic surge after vasodilation that could increase pressure and thus tearing forces inside the aorta worsening the dissection.

Initial CT, 72 hours, 3 months x 4, q6 months x2, q12 month. (Descending thoracic aorta that dilates first.)

#### **Why isn't open surgery indicated for type B dissections?**

Open surgery is not recommended due to the high mortality 30% if < 48 hours. 18% if > 49 hours.

In the acute setting mortality can be up to 50% with a 20% paraplegia risk. Its been described as sowing tissue paper.

#### **What is the management plan for a complicated Type B aortic dissection?**

Start with invasive medical management and plan for TEVAR. The goal with TEVAR being to direct the blood flow into the true lumen and seal the entry tear. If there was a dynamic obstruction (flap occludes branching vessels.) Then TEVAR would reestablish the true lumen hence removing the dynamic obstruction. Endovascular fenestration can also equalise the pressure in the true and false lumen. [Lombardi et al., 2020a]

For a static occlusion there could be a thrombus or stenosis in the branched vessel so a stent might be indicated.

#### **What are the major risks of TEVAR in the management of Type B aortic dissections?**

- Retrograde type A (reported 2% in literature however it can be around 20% in some experiences). Carries a high mortality (ranging from 7-50%). Factors associated with retrograde dissection include:

- Over-sized endograft - every 1% over 9% results in an increase in retrograde dissection of 14%. [Canaud et al., 2014]
- Proximal landing zone more than 40mm (2% -> 18%)
- Periprocedural hypertension
- Underlying aortopathy.[Tjaden et al., 2018]
- Paraplegia (reported around 5%)
- Stent induced new entry.

#### Is there a role for TEVAR in uncomplicated type B dissections?

The INSTEAD and INSTEAD XL trials looked at uncomplicated Type B dissections. There was NO statistical difference at 2 years comparing OMT vs TEVAR but at 5 years there was good aortic remodeling and better long term survival in patients treated in the sub-acute stage.

Timing for TEVAR is a difficult choice. In chronic dissections the septum thickens leading to a potentially difficult TEVAR. Anecdotally, TEVAR is best at 2 weeks to 3 months.

It should be noted, that both the thoracic and abdominal aorta often continue to dilate even after repair so follow up surveillance is paramount.[Famularo et al., 2017]

If repair must extend into Zone 2, then pre-TEVAR carotid subclavian bypass should seriously be considered in elective/urgent inpatient setting to reduce the risk of periprocedural stroke (1.9% vs 14.3%). [Bradshaw et al., 2017, Teixeira et al., 2017] Revascularization of the L subclavian with a L subclavian bypass is particularly important with a LIMA CAMB or a dominant L vertebral and a transposition is not appropriate.[Morasch, 2009a, Matsumura et al., 2009]

## 8.2 Aneurysms - Thoracic and Thoracoabdominal

02 Dec 2021: Rachael Forsythe, Mr. Mohamed Barkat, Mr. Nick Greaves, and Mr. Michael Jenkins

### 8.2.1 Anatomy and Demographics

#### Can you take us through the Crawford classification to start off?

The Crawford classification is relatively recent, to be honest it was 1986.[Crawford et al., 1986] And I think it was really important because it's very practical classification, depending on body cavity and how to get to an aneurysm. It's a bit old in terms of anatomy because they don't really follow a pattern one

would expect going one to four would be getting either more extensive or less extensive. And it's not quite like that.

So type one is from the left subclavian down to just below the diaphragm. And that's crucial because that distinguishes it from a thoracic aneurysm, which you can get to just from the chest and from a practical surgical approach. That's a very important differentiating marker, but you have to go into a second cavity.

Type two is the biggie. So this is from the left subclavian all the way down to your bifurcation. So both abdominal and thoracic exposure, all the visceral, renal arteries and a lot of intercostal and lumbars. So big impact for cord supply, et cetera.

So three is for mid chest down to and involving the viscera, renals, and bifurcation. And that's the one, which really is I suppose, the differentiator between whether you go for the full support that's needed in a type two, or whether you can risk a clamp and go approach.

And type four is characterized by being in most patients accessible from the abdomen. I say in most patients, because there are some anatomical situations with body habitus, which means that going into the left chest is useful even for type four aneurysm.

Then type five. It was an additional classification that came in later, which is a bit like a type three at the top of the type one at the bottom effectively, but not everyone uses that.

**There appears to have been an increase in the incidence of thoracoabdominal aneurysms. Do you think it's a true increase or does this relate to having scans for other reasons picked up by accident and then going on from that? What sort of many referrals do you get in a year and what's your turn down? How many numbers are you actually doing in your practice year on year?**

So that's interesting because the overall incidence of infrarenal atherosclerotic aneurysms is going down every year in the national UK vascular registry. And I suppose that is a legacy of the end of smoking for a big group of patients. I think thoracoabdominal aneurysms are going up partly because we are now imaging more and more people. and therefore we are imaging the chest and seeing them, which perhaps wasn't the case, previously. So that's an artificial increase in incidence, but also the rate of aortic dissection is on the rise, so chronic post dissection aneurysms are increasing. I think it's a small group, but there is more knowledge about connective tissue disease and genetic studies and family screening, which perhaps is also a small part of annual increase. So over the next, 10, 20 years, certainly aneurysms are not going to go away. And that group of patients that have been affected are still around and there is a huge number now within the national UK screening program under surveillance for small aneurysms, so it will come to fruition.

### 8.2.1.1 Etiology

You've just touched on a bit of difference in the etiology between thoracoabdominal aneurysms and abdominal aortic aneurysm. So is there a huge difference in the etiology between these two type of aneurysm from your experience?

On presentation, the majority of aneurysms are asymptomatic. So they're found incidentally. It is rare that you get a symptomatic aneurysm. Some can get tender as they approach a time when the wall is going to breach pre rupture. I think thoracoabdominal aneurysms tend to be a bit more symptomatic than infrarenal. These are ones that go into the arch and present with hoarseness from recurrent laryngeal tension or bronchial compression.

There is a group that cause dilatation of the crus--I've seen, the crus of the diaphragm acts like an extrinsic wrap and it can be extremely tight at that point. These patients present with excruciating pain, radiating around their cost al margin. This is tension on the crus and it's often not thought of as a sign of an aneurysm, but I think in that group, it is. The larger the extent of aneurysm, things like weight loss and general poor health begins to become increasingly seen in that group with chronic aneurysms. So there are some subtle differences.

The other group that tends to occur, not in infrarenal aneurysms, is the post dissection aneurysms and the majority of those are known about. But there was a time when type A dissections, as in Stanford type one, which went all the way down were only monitored really with echo and the ascending. The rest was forgotten about, they got lost from cardiothoracic follow up. That group is a different group because we already know they've got an aneurysm. So there are subtle differences.

And then the connective tissue group, I suppose, is a bit different because they tend to have more extensive aneurysms rather than just confined to the infrarenal segment, which is by far and way, the most common for abdominal aneurysms.

So you've mentioned a few different etiologies. Moving on to specifically thoracic aneurysms, you said a quarter of these are associated with dissection in the past, and a small portion will have underlying connective tissue disease. Do you think these patients will have a different threshold for intervention compared to the non-connective tissue disorder patients?

I think we need to be aware that the connective tissue disorders we talk about—Marfan's, Loeys Dietz, vascular Ehlers Danlos—are probably the tip of the iceberg of a number of other cases that we don't have genetic sequencing for, but behave differently. The data from a dimension point of view is not quite as robust as one would hope, but the threshold that most people would agree for non connective tissue disorders would be about six centimeters. That takes into

account the increased risks of operating both in the chest and the abdominal segment.

I think most people would agree that five centimeters is a better cutoff for connective tissue patients. Some cardiac surgeons would repair a young Marfan ascending aneurysm at four and a half centimeters. It does vary a little bit geographically, a bit like the threshold for infrarenal aneurysms, which varies between Europe, the States and the UK. So you've got to look at the patient in front of you and make a decision. I would certainly increase the threshold for someone who was not so fit and perhaps lower it a bit for a younger patient.

Also, be aware if it's something odd anatomically—a saccular bulge, something where you think there could be a mycotic element—those are all very different you can't be reassured by a dimension in an axial plane that is safe. These findings up the ante, in terms of whether you would pair at an earlier threshold. I'd include there a blowout for an eccentric penetrating aortic ulcer (PAU) or anything like that, which is not a conventional fusiform aneurysm.

**Can you talk us through the Ishimaru zones of the aorta and why do we need to know about these?**

Ishimaru was one of those useful classifications, there's lots of classifications in medicine, but it's nice to find one that's actually got a use. This classification made sure that everyone was on the same page when reporting seals zones for thoracic devices. That is important for two reasons. 1) the extent of coverage and 2) the complications increased, the more proximally you go. The main complication for that is stroke. So historically, Ishimaru decided to classify these zones.

- **Zone 0**, the first most proximal zone, is the ascending aorta up to and including the brachiocephalic trunk
- **Zone 1** is between the brachiocephalic trunk and including the left common carotid artery
- **Zone 2** is between the left common carotid artery and including the left subclavian artery
- **Zone 3** is the aortic segment just distal to the left subclavian artery.
- **Zone 4**, added by some people, is distal to the T4 level, which is much lower down the thoracic aorta

Most thoracic stenting will go really to zone three or perhaps into zone two. The more proximal you go, the more work needs to be done in terms of either extra anatomical debranching or using some form of fenestrated or branched arch device with an increase in stroke risk. What this allowed people to do is compare different series. You're not just saying these were a group of TIVA patients, but you could define exactly how proximally they go.

And the same applies for the Crawford classification. It allows comparison

within thoracoabdominal groups, and that's important with both survival and complication rates. Because if your series is mainly extent 2 thoracoabdominal aneurysms, you're going to have a very different outcomes from someone who's got mainly type four aneurysms. Both classifications allow you to look at and compare data between groups.

### 8.2.2 Management

#### 8.2.2.1 Ascending aorta and arch

So if a patient has a thoracic aneurysm that affects their ascending aorta, we obviously involve the cardiac surgeons. The patient may require an elephant trunk procedure prior to intervention on the descending aorta. Can you briefly summarize the difference between a conventional elephant trunk and a frozen elephant trunk, and when to use one over the other?

This is particularly pertinent to aortic dissection. The main purpose of repair in Type A dissection is to protect the heart. People die of either rupture into pericardium causing tamponade or stripping the coronary ostia off and getting myocardial ischemia. So the priority of repair in acute ascending aortic dissection is primarily to protect the heart and to some extent to ensure a true lumen flow distally. Therefore, it was very popular because it was the least invasive to do a short interposition ascending repair, but that really left problems for later.

Therefore, as people became more adept with cardiac and cerebral protection, it became more popular to do a more extensive repair in the first sitting. This involves an arch repair and an acceptance that eventually the descending thoracic aorta will still need to be repaired, but at a later stage. Now you have to be aware that the ascending and arch is done from a median sternotomy, and it's really difficult to get beyond the left subclavian from that position.

So when the arch was done, it was felt that the elephant trunk came from leaving an extra piece of dacron within the descending thoracic aorta in the true lumen of a dissection or in the main lumen of an aneurysm by a double sewing technique on the distal anastomosis and an inverting it. It would be left free and perfused in the descending, thoracic aorta. The benefit of that was when one came back to then do the descending thoracic segment via left thoracotomy, you could very quickly open the aorta and clamp that Dacron for a ready, made proximal anastomosis, which was much easier. It meant you didn't have to grow up and dissect to the left subclavian with scar tissue and a previous anastomosis.

Now, what industry realized is that they could help with this procedure by actually facilitating a device which had three or four branches on a piece of Dacron, which was sized to be an arch replacement. There were ready-sewn 10 millimeter branches with an extra pipe for rewarming. These could then be

sewn to be a innominate, carotid and subclavian and attached to that piece of Dacron was then a stent graft, which could then be placed distally in lieu of what was previously a floppy piece of Dacron.

And two manufacturers, JOTEC and Terumo Aortic, have made these devices, which facilitate and make things much easier. Therefore, so-called FET or frozen elephant trunk, has now become quite popular. I don't quite know why it's called frozen, but I think it perhaps means that the thoracic segment is stiff with a supported stent graft rather than just a floppy Dacron segment.

### 8.2.2.2 Isolated Thoracic Aneurysms

**So if we look at it at the isolated thoracic aneurysm, can you tell us briefly about the principle of TEVAR and what's the optimal landing zone. Finally, in your opinion, is rapid ventricular pacing required when deploying these stents?**

So I think the isolated, thoracic aneurysm is a perfect application for TEVAR because it fulfills all those minimally invasive credentials—an endoluminal approach and if you can get a good seal zone, proximally and distally, it saves a thoracotomy and it's a massive difference for the patients. Unfortunately I suppose those cartoons you see on the industry advertisements are vanishingly rare, when you've got a perfect proximal and distal landing zone and have really straight thoracic aorta.

I think for the majority of cases, you need a 2cm seal zone proximally and distally. However, if you have a tortuous aorta, you likely need a longer seal zone. You want to land in an area where there is a good parallel walled segment. You have to be careful of so-called "bird-beaking" when a stiff, less conformable device lands perfectly on the outer curve, but holds off on the inner curve. This results in a lip, which protrudes and allows blood to get under that and cause stent graft crushing. I think modern devices have become much more conformable. We've learned the lessons of oversizing and the problems associated with oversizing. We've learned how to taper stent grafts when there's mismatch between proximal and distal landing zones.

These have all significantly improved things, but there's still no getting away from the fact that some anatomy is not well adjusted to the currently available stent grafts and Gothic arches are a particular problem. I've already mentioned tortuous and large sacks, which allow a stent graft to move away from the center line to the outer curve.[Iwakoshi et al., 2019] If that sac doesn't shrink, you can imagine that draws on both the proximal and distal seals zone, and reduces them.

I think you can sometimes get away with a shorter landing zone. If, for example, you're doing a post traumatic aneurysm where it's only on one segment of the outer wall where there's a problem. But for the vast majority of true aneurysms, you need that seal zone and you do need to oversize to achieve that.

You can go more proximally, and I think the majority of aneurysms at the left subclavian area, you can just drop the blood pressure to a systolic of 70 or 80 pharmacologically. Once you go more proximal to that, especially if you've got a custom device, with a scallop or branched device where you need an absolutely critical landing zone, you then really need to achieve a short transient period of circulatory arrest, either with adenosine, which is perhaps sometimes a bit unreliable, or rapid pacing.

Rapid Pacing can be difficult and does come with complications though, such as ventricular puncture. There are some newer techniques in terms of caval occlusion balloons, which basically stop venous blood returning to the heart with a reduction in output. Those are gaining popularity certainly in Germany and they can be quite effective for that. I think having tip capture on devices and better, more accurate deployment and better imaging does allow you to be more accurate in deployment, but there are still problems.

These devices have been inserted to very torturous iliac systems sometimes and they retain some energy. When you deliver them by removing that sheath, sometimes that energy is still there and they can jump forward as well as back. This can cause problems because you end up covering a vessel that you didn't intend to.

**So you've just explained in detail that TEVAR is a great option for the majority of cases, but I presume you still believe there is a place for open repair in isolated, thoracic aneurysms, and why?**

I'm not so sure in isolated, thoracic aneurysms, unless for some reason they're not suitable for TEVAR. I think if you've got problems with a mycotic aneurysm or a fistula into the bronchus or esophagus, these are our big problems with high mortality and your duty bound to go for open repair.

Patients with hemoptysis and a previous aortic repair should carry a high suspicion for an aortobronchial fistula. TEVAR is considered preferred repair, due to high morbidity and mortality with open repair. Bronchoscopy should be avoided due to high risk of rebleeding.[Bailey et al., 2011, Léobon et al., 2002, Quintana et al., 2006]

Certainly with connective tissue patients, open has advantages. In some unique situations it is possible to bridge between repairs. I think in some of the younger connective tissue patients, they will end up having certain segments repaired at different stages of their life. Although it's probably now accepted that "all endovascular" is not a good option for these patients, if you've got Dacron proximally and distally already, bridging those segments with an endovascular device might work well. Although the Dacron will slowly dilate, it won't dilate as much as aortic tissue in these patients. So there are options, but I think in reality, isolated, thoracic aneurysms on reasonable rare and TEVAR can be used for the majority of them.

### 8.2.2.3 Thoracoabdominal Aneurysms

**Moving on to thoracoabdominal aneurysms.** Earlier we used the Crawford classification to describe the anatomical extent of the thoracoabdominal aneurysm. So let's talk firstly, about type 1, 2, 3, and 5, which involved the thoracic or aorta to a greater or lesser extent as well as the abdominal aorta. We'll come back to type four later on in this podcast.

**So type 2 open thoracoabdominal aneurysm is arguably the most invasive operation a patient can undergo and carries 30 day mortality in excess of 10 to 15%. What is the size threshold for type two that you currently use in your practice and what is the evidence for this?**

So, as I suggested earlier, the evidence is that it's a bit historical, and most people would actually say about six centimeters. I think that can be brought down a little bit for connective tissue patients and can go up a little bit for atherosomatous patients. But in general, you're talking about a younger segment of patients. This is not an operation for people in their eighties. Outcomes stratified by age greater than 50 found a higher rate of death, paraplegia, renal failure, cardiac complications and length of stay.[Coselli et al., 2017] These patients need to be in very good shape because it is a big onslaught physiologically on them. It's not just getting them off the table, it's getting them out of intensive care and out of hospital and to recover back to their baseline.

I think that's one of the things that can be really difficult about this disease. You are taking patients who are effectively functioning quite well, and they're often relatively asymptomatic and you're putting them through a prophylactic operation to try and prevent rupture, which no one quite knows if or when it'll happen.

**So to that end, can you talk us through the decision making process when you're assessing a patient with a thoracoabdominal aneurysm types 1, 2, 3, or 5?**

I think it is really dependent on anatomy, physiology, and the patient in front of you. Unfortunately, it is a bit of a bespoke assessment for an individual patient. So firstly, I think you have got to look at what are the options for that patient, which goes from conservative to endovascular, to open surgery.

The endovascular domain has changed hugely now with the increasing availability of branched devices. When I started, there weren't custom devices. There were some FEVAR devices just coming out, and we did use long covered stents going through devices with TIVA above. However, there wasn't the option for what we've now got from an endovascular perspective.

So I think you've got to choose the patients that are either unsuitable for endovascular, or are we really going to benefit from a durability of an open approach. Those are the two big differences, and they are differences. One, you're

going for gold standard approach because you think they're life expectancy warrants that. The other one is much higher risk, because they haven't got another endovascular option. These range from people with very tortuous anatomy, to difficult renal arteries, such as early divisions, that make them unsuitable as target vessels. Another reason is complex dissections, which are not suitable for an endovascular approach. Therefore, it is quite variable, but the first thing you must consider is whether this patient is going to get through this and get out of hospital. And that really is looking at them in the eye and considering what's their quality of life or what's their family support, and are they up for this?

The second one then is anatomical difficulties—Is there a shaggy aorta, atherosomatous disease, or calcification of a target vessel, etc. Severe aortic wall thrombus is associated with solid organ infarction (24%), acute kidney injury, without dialysis (21%) and delayed oral intake by 3.4d, however rarely resulted in mortality (0.5%).[Ribeiro et al., 2017] Rarely is this a hundred percent or 0% decision, but it's about building up relative contraindications. Some of them are pretty simple, like access problems for endovascular, and it's a combination of those which then will sway you in one way or the other. And if anything, that decision-making is probably more pertinent for type three and type four, where there really is a different option and these patients are in their sixties and seventies with classic atherosomatous disease. The really, young fit non-connective tissue patient with a type 2 TAAA is a rare beast.

**For connective tissue disease, many textbooks prefer an open approach over an endovascular approach do you still agree with this?**

Yes, I do. I think certainly, what we know about patients who've had total endovascular approaches for connective tissue diseases is that they don't last. They may last five years or so and then you get progressive aortic dilatation and loss of those seal zones. Whether that is the natural history of the native disease or whether it is regional force hooks, barbs or whatever is related to the device is unclear.

But when you sew these pensions and just touching the back end of a needle on the anterior wall can create a massive problem or just being a little bit clumsy and you get a radial tear. It's very clear that a stiff device sitting in those aortas for many years with 60-70 beats per minute going through it and often hypertension, isn't going last.

However there are some caveats, as I mentioned earlier, bridging between previous prosthetic open repairs is a possibility. And sometimes in a lifesaving situation, such as when someone has presented with a rupture, you may have to use an endovascular approach. However, be prepared then to treat that as a bridge and go back to do a definitive repair when things have calmed down and in the cold light of day.

**So we don't really have the scope to dive into complex endovascular repair in this episode, but can you describe the broad principles of**

**your operative approach to open thoracoabdominal aneurysm repair, type two, because it's the most extensive?**

In terms of the patient, for anything juxtarenal or type four, I tend to have a patient supine on their back, but with a break in the table at the level between the costal margin and the anterior superior iliac spine. Breaking the table at that level, you can massively increase the exposure to that segment.

However, there are some patients even with a type four repair that demand a left thoracoabdominal incision. For a type four, if you're doing a left thoracoabdominal approach, it's usually for someone with a very narrow acute angle of the costal margin, such as a more petite frame female patient or a crus that is very high on the aorta. And if you look at CT scans, which is something I've realized over many years, that not everyone is the same. So where the celiac and SMA are can be in a different proportion, depending on where the costal margin and skeleton is, which can make them easier or hard to get to. And certainly in a situation, if there is a rupture within the left side of the abdomen, you wouldn't really want to go into that without getting proximal control. Because as you do the visceral rotation, you could have catastrophic bleeding.

So under those circumstances, I'd also go into the left chest with a reasonably low segment so that you can place a clamp ready before doing the visceral rotation. So for type four the one above then, the higher you go, the higher your thoracotomy and the more tipped over you need the patient. So for a type two, you're going probably fifth intercostal space and the shoulders are at 90 degrees and the pelvis is 60 degrees. For type three, you're slightly further over, so you're down to about 60 degrees and you're perhaps in the seventh intercostal space. So the lower you go, then right up to being pretty much supine.

The left arm is put over top. You mark the scapular and you go into the chest and abdomen. I tend to go intraperitoneally into the abdomen, so I can see the bowel, although there are people who do a retroperitoneal approach. I tend to divide the diaphragm down to the central ligament and mark that. Then effectively you are looking at your clamp zone. You dissect around your clamp zone, both proximally and distally. We don't tend to expose the celiac and SMA in particular, or the right renal. It's important to find the left renal artery. For the visceral rotation, you effectively extend the left colon up, find the white line and get under that and get right onto psoas. The key is to get right down on the psoas early on, and then it is a relatively bloodless field. Everything has taken me to leave the ureters out of the way. I always take for left kidney up. You will see in the textbooks and some advocate to keep the left kidney down. I think that's utter madness to be honest and I can't see any point in it.

So for me, everything rotates up then out of the way. And it looks a bit strange, like a postmortem. It looks very odd to see the whole left side abdominal cavity when all you've got is diaphragm muscle, psoas muscle and nothing else left. It does give you a very safe exposure then to the aorta. I don't tend to go round the aorta with slings, I think that can cause problems. I go around with my

fingers and be very aware of the right sided lumbar or intercostals because you really don't want to cause a problem there because you easily get into the right chest from that exposure. So you have to be a bit careful so you can see exactly where you're getting round.

**So in specifically with a type 2 TAAA, many people establish a left heart bypass. Do you always operate in that way and do you use a cardiac surgeon and the setting?**

I think type 2 you are mandated to have some sort of adjunct to keep the rest of the body perfused while you're doing the proximal anastomosis. I think in an elective setting left heart bypass is the right way forward, because it does give you more flexibility and it allows you then to sequentially clamp and keep the legs, the viscera or the kidneys perfused. There are some negative aspects to left heart bypass, though, in terms of needing a much higher ACT and bleeding is the enemy here. The more you anti-coagulate, the more problems you've got. It's also not pulsatile flow and you're putting all your cells through a pump and you've got to be aware of increased problems around stroke and everything else that goes with that. I think it is very convenient for that set up, because you can get to the inferior pulmonary vein quite easily and femoral cannulation is quite easy for you to return and then you've got the option then of individually calculating celiac and SMA for perfusion, whereas usually renals are cooled down.

If you've got a perfusionist there as well, you can get cold perfusate for the renal arteries. So it gives you more flexibility, and if things go wrong, you've then got an option to switch to conventional bypass if needed or even drain out and go to circulatory arrest and cool-down.

So it does give you more options, however, in a more emergency setting, an approach that tends to work quite well for us is to do an ax-fem approach. You use an ax-fem graft onto the right subclavian artery, not tunneled but on the outside of a body onto the right femoral and use the other limb as a single cannula, usually for the SMA, because the SMA is the king vessel with all of this. Most of the other things can go, but if you lose the SMA, you've got a dead patient. And that allows you to do an emergency procedure, a mid type 3 approach, without some of the problems associated with left heart bypass. It allows you to have a lower ACT; it gives you pulsatile flow; you're not smashing your cells up all the time; and it's relatively quick and straightforward to do. You don't have to mobilize a perfusionist and everything else that goes with that. It's worked well for us in emergency settings. It's better than a clamp and go approach because with a clamp and go approach, for the type 3 repair, you need to complete in 15 minutes, your inlay anastomosis at the top, the clock is already on. If you get down to the visceral segment and you've got to do some removal of thrombus to get your Corolla patch on or you've got to reimplant the left kidney separately, it's a lot of time pressure and there's no opportunity for something to go wrong there. That's where this gives you a bit more breathing space that you've got the lower body perfused while you're doing your proximal

anastomosis.

**8.2.2.3.1 Complications** As you've mentioned, open repair is a high tariff, high risk operation, but apart from death, are there any specific complications to the open approach?

The big problems are bleeding and clamp times. Those are the things that cause problems for those patients who survive. So problems with large transfusion which cause problems with ventilation afterwards. The most common complications after a TAAA are pulmonary with 8.5% of one cohort requiring a tracheostomy.[Coselli et al., 2016] Renal ischemia is an issue of people will quote a 40 to 50 minute renal warm ischemic time, you are then getting increasing problems with likely renal failure. They may need to go on the hemofilter for temporary support, but these patients have already lost a lot of nephrons, so they haven't got a huge amount of capacity to lose more. That's why eGFR or high creatinine is a really important prognostic marker of outcome in this patients. I think it's like a barometer on their micro-circulation.

The big one and the Achilles heel of these approaches, whether it's endovascular or open is spinal cord ischemia and that is because of the segmental of blood supply to the cord comes from all the intercostals and lumbars. If you've got that aneurysm extending both proximally and distally involving the internal iliacs the left subclavian, then you're taking out your crucial collateral supply. And that is why trying to maintain blood supply, for example getting the lengths back in circuit early, whether that's removing the large sheaths, during a long branched approach or getting the leg supply earlier in an open approach, and keeping the left subclavian in circuit. These are all other important things, paying attention to the mean arterial pressure in theater, have a spinal drain working, all of that is crucial in terms of trying to protect the cord as much as possible.

Post TEVAR leg weakness needs to undergo urgent evaluation. Differential diagnosis includes spinal ischemia due to subclavian or intercostal artery coverage, distal embolization or stenosis of the TEVAR graft. Stenosis should be treated with angioplasty and extension.[Buth et al., 2007]

**So the major issue is the spinal cord ischemia, and we mentioned briefly the blood supply of the spinal cord for the sake of the exam. Can you recap for us the blood supply of the spinal cord?**

So it's complicated actually. There are anterior and posterior spinal arteries, but I think depending on what level you are in the cord, they get a blood supply from a more dominant approach. So higher up in the cord—cervical and higher thoracic—vertebral arteries are important, so hence the importance of a left subclavian bypass.

The lower you go, then they become a bit less important and the actual segmental arteries, intercostals, become dominant. Much is talked about the artery of

Adamkiewicz. I think, yes, there may be a dominant vessel at about that level, but it's rarely one, absolutely single one that if you preserve that one, then the rest don't matter. I think they all matter.

And then the more distally you go, collateral supply from internal iliacs, median sacral, all those contribute to a collateral circulation that should be seen in some ways all connected. And that is why, more recently in endovascular approaches, staging repair is really useful because you can allow the surviving arteries then to remodel and increase flow. It's why, during a type 2 repair, when you get the whole aorta open, it's very important to block the intercostals early—either by sewing them off or putting a little Pruitt balloon in and to stop losing blood from that collateral supply. It keeps the pressure in the spinal cord. Also that's why, the differential position of that blood supply is why patients then get a particular form of problem with motor problems and sometimes preservation of sensation.

**So purely for exam purposes and a bit of physiology. Can you remind us how to calculate the spinal cord perfusion?**

All that is really is map minus the CSF pressure. So it's your mean arterial pressure, take out the CSF pressure and that's what you've got perfusing the spinal cord. It is a useful equation to remember because it allows you to decide what's the way to enhance spinal perfusion as much as possible, and that is to increase MAP and decrease your CSF pressure. We do that by enhancing the mean arterial pressure as much as possible, and draining off CSF with a spinal drain to reduce your CSF pressure and allow more blood into the cord.

I think people perhaps get a bit hung up on spinal drainage and it's importance. It is just as important to maintain oxygenation and hemoglobin over a hundred, reduce blood loss and all those other things are as crucial. Spinal drainage is an adjunct, but it shouldn't be seen in isolation. It's one of many adjuncts to try and help prevent spinal ischemia.

**So in regards to the mechanism for spinal cord ischemia during open thoracoabdominal aneurysm repair, do you think it's related the blood loss, clamp time or or implantation of the intercostals?**

I think it's multifactorial. So I think the majority is due to ischemia scheme in terms of a watershed situation. Segments of a cord just not getting enough blood. And the very reason the more typical area around T7, 8, 9, to 10, is because it is in the middle between the vertebral and internal iliac artery supply. This is probably a kin to a splenic flexure of the colon, it's between two territories.

However, there is also potentially problems with microemboli or trashing of a spinal arteries, which can occur in some patients. It's a slightly confusing situation, but I think in terms of intraoperatively, blood loss, dumps in blood pressure, with hypotensive episodes really don't help. When do you remiplant? Again, that is a bit confusing. Some people would argue if there's massive back bleeding, you don't need to be reimplant because they're getting collateral

supply from somewhere.

I think it will depend on how the case is going. We do use motor evoked potentials (MEPs) but that's quite a specialist situation and you're very reliant on the readings you get. They can be confused by leg ischemia, cooling down or edema. But if you have already got the legs back in circuits and got the MAP up and things weren't improving and there was a sizable intercostals that could be reimplanted, well, then that's worth doing. And that's why the technique of using Pruitt balloon occlusion methods in pairs rather than ligating is good because you can then just reimplant those. If you don't need to implant them, you can just ligate and remove them. Whereas if you've already ligated them, you've got a problem because you've already destroyed the ostia. We tend to leave a branch already attached in the mid thoracic aorta to save an anastomosis. You can, if you've got really good intercostals, fillet the branch to open it up and sew it on like a long patch longitudinally along the paired intercostals and then plumb it back on the other end so it's a circuit in series with your main aortic graft. That probably decreases the resistance to flow and that might remain patent for a bit longer, rather than having a big 10mm graft going into a single pair of intercostals with what are likely to have quite a high resistance, and they probably don't last as long.

Severe spinal protocols have been described to reduce risk of spinal ischemia or mitigate the impact of spinal ischemia in patients who develop weakness.[Estrera et al., 2009, Yanase et al., 2012] An easy way to remember the important components are COPS:

- C - Cerebral spine drain status - keep for 7 days, pressure less than 5
- O - Oxygen delivery - supplemental oxygen, increase hgb, increase cardiac index
- PS - Patient Status - Keep BP greater than a MAP of 90mmHg

**So do you think we're good at predicting preoperatively who may get spinal cord ischemia? You mentioned a few risk factors already that can predict who will or won't post-operatively.**

I think we're utterly hopeless at it actually. I really don't think that there's a good method. You can see people having an infrarenal repair getting paralysis and you can completely replace the whole aorta from the arch down to the iliacs with prosthetic and have people walking around without a problem at all. So I don't really understand it and I think that comes into the business about whether it is about emboli, flow, or collaterals. I think it's a combination of things. When people started embolizing intercostals preoperatively prior to endo repairs, people thought this is a man thing—you want to keep them perfused surely. But I think it does show the importance of maximizing the collateral supply, and I think some people have a better collateral supply than others. And of course, what we don't know is when you get down to the more microscopic level of actual arterial supply at cord level, what that is like in an

individual patient. You can't see on imaging. If that is already compromised, I suspect those patients are more at risk than whether they have a macro vascular problem when things go wrong terms of hypotension or blood loss.

Many reports state that the highest risk factor for spinal cord ischemia includes length of aortic coverage or repair, followed by whether they have had a prior repair, preop hemoglobin, and intraoperative hypotension.[Bisdas et al., 2015]

**We haven't really touched on complex endovascular repairs yet, but can you explain when you might choose a hybrid approach to this thoracoabdominal aneurysm repair?**

So visceral hybrid operation came in really as a bridge between open surgery and endovascular. This is a bit dated now, because it is before custom devices and we only had thoracic stents at this time, the era of the Talent stent, which was a very early thoracic device made by Medtronic. Perhaps not the perfect name because it wasn't a particularly talented, it was a difficult thing to deploy, but it actually got us into the thoracic aorta. What we realized was that if you could operate in one cavity, such as the abdomen, but still excluding any aneurysm in the chest without cross clamping and without rendering that patient ischemic over a large part of their body—they could withstand that hit much better than open cavity surgery with cross clamp and massive reperfusion.

So the concept of a visceral hybrid was to do an extra-anatomical bypass of the celiac, SMA and both renals from either the distal aorta or the iliac vessels. So patients would only get sequential ischemia of one organ at a time, say the right kidney or the celiac territory, and during that 10 minute anastomosis, the remainder of the body and organs were perfused. And finally, at the end that, you could then put in a thoracic device as a freebie, because you'd already perfused the organs from distally. You'd ligated those target vessels and then you would just exclude the aneurysm so that you were transferring blood from above the repair to below the repair and retrogradely perfusing the vital organs.

So it was very attractive and we started doing it quite early at St. Mary's and initially we had some very good results in the first 30 or so patients. And as always, then it allowed you to consider older, sicker patients but it wasn't easy surgery. And actually I think because it was doable in terms of just an abdominal approach and relatively familiar territory for anyone who's done occlusive disease for the viscerales or renals, I think a number of centers started doing small numbers and not getting so good results. And then when better, custom made devices came in, I think its purpose became less and less because why would you do that if you had an opportunity to do something which was even less invasive.

So what we've been left with now to consider a hybrid type approach effectively are those patients who are physiologically not fit enough for an open approach and those patients who are anatomically not suitable for a total endovascular approach. In some ways, they are the worst of both worlds in that outcome group. And so I don't think you'll ever be able to compare outcomes legitimately

with the fitter group having a fully open approach and the anatomically suitable patients having a total endovascular.

But I must say we still see quite a lot in our multidisciplinary reviews coming back. What surprises me is the durability of those grafts. The visceral grafts remain patent for many years, some of them are 15 years now. Occasionally there will be an accessory or small diseased renal artery, and the renal graft will go down, but the others have been extremely durable. And even though we've had to re-line the stenting portion or extend it, the grafts have maintained really well.

A unique situation that uses a similar technique is with mycotic aneurysms of the visceral segment of the aorta. The mainstay of treatment is a debranching of the aorta and debridement of the infected aorta with in situ revascularization or ax fem-fem bypass. There is an evolving role for FEVAR, such as with most aneurysmal disease.[Sörelius et al., 2016, Sule and Dharmaraj, 2016]

**So moving on to Type IV thoracoabdominal aneurysm repair. These can be repaired using endovascular or open techniques. Can you talk us through your approach to open Type IV repair in terms of exposure and any tips such as how to minimize the visceral ischemia?**

So I think an open Type IV from a subcostal approach is a doable and durable procedure. And what I mean by that is that the hit to the patient is not enormous, and a reasonably fit patient can get through that reasonably well. You have to be a little bit selective, but not super selective. For example, in my first hundred, I had one 30 day death and one in hospital death, which was someone at about three months who just didn't get better, so that puts it into perspective that actually it's an operation with a very good outcome, if you get it right at the beginning.

The approach I mentioned earlier, nothing too special. This is a supine patient. Getting the break on the table in the right position to allow you to extend the abdomen. You lower both ends of a table to open up that segment of the abdomen and get better exposure. I tend to do the vast majority via a subcostal approach, some people would call it rooftop, then extending on the left side down below the costal margin a bit. And it gives you really good exposure even if you have to go down to the iliacs. It does cause a bit more of a problem getting to the right iliac bifurcation. The left is easy, because it's right there next to you. So that's an added complication.

The visceral rotation needs to be done carefully because what you do not want is to create blood loss right at beginning. And as I mentioned earlier, it's getting under the left colon along the white line onto psoas and then getting up under psoas and I always tell the registrars when they're doing it, you've got to go as far as possible on psoas from below. And when you think you've gone as far as possible, you've got to go another five centimeters. So you get right up to the diaphragm from below. And then you can get into the supra-colic compartment around the splenic flexure and get your fingers either side of the

lienorenal ligament and take that off the lateral and the posterior abdominal wall. The more and more you do, the more you are freeing and the whole of the viscera comes up towards the right side of a patient and eventually they will rotate up and you've exposed the whole left wall of the aorta.

The first thing I then do is ensure that I've got a clamp zone, and I do that by dividing the crus which sometimes can be really tense at that level. The crus can act as an extrinsic wrap around the dilated aorta, to an extent that the distal end of the crus is almost ligamentous, causing a tight band on the aorta. I tend to get my finger underneath that before it with a diathermy and opening it, and then very carefully choosing the clamp zone and getting my fingers right round that area, so I know I've got a healthy clamp zone.

The next thing I then do is look for the left renal artery. I've already tended to make a decision about what I'm going to do with the left renal artery, which depends on the number of things. So it depends on, to some extent, the age of a patient, where the left renal is in terms of a clock face. So if you're looking at an axial cut to the CT, the more the left renal is around before three o'clock, up to two o'clock, the easier it is to incorporate because you've got less aortic tissue there. Once you're beyond 3:30, round to four o'clock, it's quite a long way away and it's quite posterior on the sidewall of the aorta, or if the left renal is more distal compared to the right renal. What that means is then it's a long way away from the right renal artery, which means you're going to be leaving a larger patch of aorta. This is usually bad news, unless you have a more elderly patient.

You're making this decision about whether you're going to try and incorporate the left renal artery into a big big patch or whether you're going to reimplant it separately or perform a jump graft. So you've already thought about that, but it's crucial to find the left renal because when you've clamped and you've done your left aortotomy, you've got to get that aortotomy underneath and posterior to the left renal artery so that the orifice goes up with that segment of the aorta.

Then I tend to get the iliacs out. The left is easy, it's there. I have no qualms on the right by going back over through the peritoneum to find the right iliac artery to expose that. I don't go around any of them. I just find a very discrete clamp zone. You don't need much, just literally enough to put a clamp on.

And then I tend to take the fat and lymphatic tissue off the left side of aorta. So when you open the aorta, you've not got a lot of immediate bleeding. If you've got a retro-aortic left renal vein, you've got a line gate that causes otherwise you'd be going through it. And surprisingly, on CT, the main left renal vein is anterior, but there is often a vein there which has stretched across, either a lumbar or phrenic vein, which you should deal with prior to your aortotomy.

It's about getting everything set up before you cross clamp, because once you cross clamp, the clock is on. So it's getting the whole team ready for that moment and making your anatomy as perfect as possible before you do that. Obviously it goes without saying that you have cell salvage and we use a Belmont

with the big bucket on the top of it, so that you can rapidly give blood back when you need it.

**Can you talk a little bit more about a Carrel patch and when would you choose to do a jump graft to the left renal rather than encompass all the vessel vessels onto that patch?**

I tend to think of a Carrel patch more in a way with a type 3 repair, because with a type 3 repair, you've done your proximal anastomosis and then what you're doing is you're putting on a patch of aorta to include the celiac, SMA, and right renal, which tend to be together, whereas sometimes the left renal is a bit away. The more tissue you leave there, the more chance that aortic tissue will dilate to over time, hence the patch aneurysm. If your patient has got some form of connective tissue disease, we tend to reimplant directly.[De Rango et al., 2011, Afifi et al., 2017] There is a very nice graft available with four side bunches already put on and you can go straight onto the ostia. So you're basically leaving no aortic tissue at all.

For type four, you are doing an oblique anastomosis and you have to be aware that the right side of the aorta is really left there. It's a compromise, these patients with atherosclerotic type aneurysms, they may be in their sixties, seventies or even into their eighties. Even the good candidates, they're not going to live 25-30 years, and therefore over time that segment may begin to dilate a bit. I think that is acceptable. Half of that segment is Dacron and the other bit may dilate.

I think if you've got a Carrel patch, which really balloons out, that's something which is different. We don't have good dates as to when those will give away and rupture. I think what is clear is that if you've got dehiscence between your suture line and your graft, that is a dangerous situation, akin to a false aneurysm. And although it may look similar on CT, that's a different beast and they're at risk at any stage. True aneurysmal dilation of a segment that is left behind is a bit different and I think that's a bit safer. In essence, what you're trying to do is a compromise between leaving as little aortic tissue as possible, but also getting the patient alive through their first repair safely.

If I know I'm not going to incorporate a left renal, then I will sew on a six or eight millimeter side branch onto the tube graft before I cross clamp, because that saves you one anastomosis. And then now my favorite technique is just to amputate the left renal artery and sew that directly onto your branch and leave that in a bit of a lazy C configuration in the paracolic gutter, so as the rotation comes back, it's not under tension. The other technique is to have very short graft, right from your aortic prosthetic, but you've got to really judge how that will sit when the kidney is back in its anatomic position. So it can't come off the anterior aspect of the tube graft. It needs to be further down and that's awkward to judge and I think we're probably not good at that so the lazy C is a better approach.

I much prefer that to trying to either reimplant the native artery directly onto

the Dacron, as they tend to restenose or try to bring the aortic sidewall down on to the Dacron. I think that's fraught with difficulty. You've often got calcification at the ostium, and it's also very difficult because you've got the rest of your anastomosis really close and sometimes not enough room to bring your clamp down, which is exactly what you want to do. The left kidney will take a bit of an extra ischemic hit, but you want to be perfuse the right renal and viscera. You've got to have to bring your camp down below your proximal anastomosis.

When it comes to late complications from TAAA repair, 8% are from progression of aortic disease and 3% are related to the graft repair. Risk factors associated with late events include female sex, partial aneurysm resection, expansion of remaining native aorta or initial aneurysm rupture.[Clouse et al., 2003]

**Have you ever had to treat the Carrel patch aneurysm in your career?**

We have a number from previous Type 3 repairs and I think these are very suitable for fenestrated or branched devices. You've got a beautiful lending zone, proximally and distally. Why not? These tend to be well-suited to that and it's a much easier approach mixing endo and open, changing from one to the other, I think that's fine. It's just at a different stage of a patient's life and repair

**Any final summary or closing words about aortic surgery?**

We have a bit of a tipping point with open aortic surgery at the moment, certainly in the UK. Looking at registry data, many are tipping back to open now. Whether that was as a result of NICE guidelines, I suspect not, perhaps that just put it more into focus. I think many people are seeing more problems with patients surviving after what was very good endovascular surgery at the time, but they've outlived the repair. Now we've got problems with patients where you got to wonder whether that wasn't perfect anatomy and should they have had open surgery?

Now the difficulty we've got at the moment is the patients that perhaps weren't that suited for endovascular repair, did well initially from it and they are paying the price later on. But those very patients are the ones that are also more difficult from an open approach. They are patients with shorter, more tortuous necks or more calcification. There is a concern that there is an era of surgeons now that haven't done as much open aortic surgery, who may not be as confident with adverse anatomy.

That lack of confidence influences your decision-making, so patients don't get a full appraisal of what options are available. The default then may be to go with a less than perfect and endovascular option. I agree, this may be less easy to kill a patient upfront with an endo option but you perhaps pay for that later on. This is a very difficult thing to get around and we've got to get to a compromise of finding the patients with the likely better life expectancy and sending them to groups with a larger open practice and get them through a big operation safely and capitalize on those benefits.

The really lucky ones are the ones that are anatomically really suited for endovascular repair and their sack really shrinks and their seal zones remain good. They are the best of both worlds as they tend to also be fitter with less extensive disease and get the benefits from a less invasive approach. Not all patients are the same and we've got to now accept that we've gone down the line of working with industry, which they didn't necessarily focus on durability. What they wanted to do is to extend the applicability, to treat more patients and get better, lower profile devices to go for these percutaneous cardiology market in the United States. And we're now paying the price of some of those decisions, so we've got to reboot and take a stance on which patients are going to actually benefit from which approach and have a better evaluation of that going forward.

### 8.2.3 Additional Resources

- Yale Vascular Review Podcast Episode 1: Thoracoabdominal Aneurysms

## 8.3 Aortopathies

**18 May 2020:** *Dr. Anna Ohlsson and Dr Sherene Shalhub; University of Washington*

### What are the common genetic aortopathies?

There are several well-known genetic disorders which account for genetic aortopathies. The most well-known are Marfan syndrome, Loeys-Dietz Syndrome, and Vascular Ehlers-Danlos Syndrome,

There are less commonly known ones such as Familial Thoracic Aortic Aneurysms and Dissections due to pathogenic variants smooth muscle cells genes such as *ACTA2*. There are others in which the causative gene is not known.

### Why are they such a big deal?

These are cases in which the building blocks of the aortic wall are defective. What I mean by this, is that these patients have pathogenic variants in the genes that affect cell signaling or smooth muscle cell structure that lead to suboptimal composition of the aortic wall. These alterations ultimately lead to cystic medial necrosis in the aortic wall.

As such they are at more risk for aortic aneurysms and dissections that can lead to the premature death of the patient.

To put the frequency in perspective, Marfan syndrome occurs in 1:5000 of the population while Vascular Ehlers-Danlos syndrome (also known as VEDS) occur in 1:50000 of the population.

**Let's dive into them then – what are the defining features of each and the high yield information?**

The high yield information is being able to pair the genetic syndrome and phenotype with its associated genetic mutation. A useful exercise following this broadcast is to list the disorders in a table and write out their associated gene mutation, what protein defect or deficit occurs, the typical phenotype, and the common vascular pathology associated.

But before we dive in, I want you to keep in mind some of shared features. One is that the associated aneurysms and dissections tend to occur at younger ages and dissect at lower blood pressures than what we see with sporadic dissections (these are the dissections that are not familial or associated with a syndrome)

One is that these are inherited in an autosomal dominant manner but there can be variation in how the pathogenic variants are expressed among affected people and even within families. The other thing to remember, is that in roughly half of these cases, the affected patient is the first in their family to have a given pathogenic mutation. The flip side of this, is in half the cases, there is a family history of aortic aneurysms, dissections, and sudden death.

**We will start with Marfan syndrome.**

Marfan syndrome is caused by pathogenic variants in the *FBN1* gene (also known as fibrillin-1 gene). These variants lead to improper formation of the microfibrils that maintain elastin, a key component of the arterial wall.

These patients are prone to aneurysmal degeneration and dissections of the aortic root but can also dissect the descending thoracic aorta. They commonly have lens dislocations (ectopia lentis). They have common skeletal features such as

being tall, thin, with long arms and legs, scoliosis, pectus deformities (carnitatum or exicavatum), and club feet. They can also have a history of spontaneous pneumothoraces and mitral valve prolapse.

**How is Marfan syndrome similar or different from the other genetically triggered aortopathies that you mentioned?**

Loeys Dietz Syndrome is similar to Marfan syndrome in all the features including the aortic root aneurysms. They don't seem to have lens dislocation and they have other unique features such as bifid uvula or cleft palate, and hypertelorism (which is an abnormally increased distance between the eyes). What is different about Loeys Dietz Syndrome from Marfan syndrome is that they can have arterial aneurysms of other arteries instead of the aorta, such as the SMA, axillary, or other peripheral arteries.

Vascular Ehlers-Danlos syndrome has some shared features to Marfan Syndrome with both such as spontaneous pneumothoraces, but these patients tend to be short and can have easy bruising. They also have similar features to Loeys Dietz syndrome in terms of arterial aneurysms. Common features of VEDS would be thin translucent skin where you can easily see their veins, thin lips, thin bridge

of the nose, large eyes, easy bruising, acrogeria – or an aged appearance of the hands

However, unlike Marfan and Loeys Dietz, the majority of VEDS patients tend to not have aortic root aneurysms. One thing to remember about VEDS is that it is a *subtype* of Ehlers Danlos syndromes. It's very important to distinguish it from the other subtypes because most of the other 12 Ehlers-Danlos syndromes are not associated with arterial pathology. So people with vascular EDS are prone to arterial, uterine, and intestinal rupture and their average lifespan is 48 due to these highly morbid pathologies. 25% of patients with vEDS will have experienced some clinical manifestation by age 20, and that number is close to 90% by age 40.

**I remember learning about classic Ehler's Danlos presenting with hypermobile skin and joints. Is this something you see with Vascular EDS as well?**

Patient's with vEDS don't have the same hypermobile skin or joint laxity as we classically think of with classic Ehler's Danlos. In fact, some vEDS patients report losing confidence in their physicians who ask them about joint and skin hypermobility because it suggests to them that their doctor doesn't know about their disease process. These patients often know more than most of the doctors they meet about their condition, and it's a source of constant frustration for them. It can also be a problem if the severity of the disease is underestimated, as we discussed they can present much younger than most patients with highly morbid issues – like arterial rupture.

**You mentioned arterial pathology in Loeys Dietz and VEDS. Can you tell me more about that?**

In both types you can see subclavian, carotid, SMA, and iliac artery aneurysms and dissections, as well as less frequently vertebral, SFA, and popliteal aneurysms and dissections.

**How do you diagnose these genetically triggered aortopathies?**

There are clinical diagnostic criteria for each, but ultimately genetic and laboratory testing is very important for the final diagnosis.

Ghent's criteria is used to clinically diagnose Marfan's syndrome. The big ones are aortic root dilation, known family history of Marfan's or not, the diagnosis of ectopia lentis which clinically is manifested as iridonesis (lens shimmering). Additionally, genetic testing for pathogenic FBN1 variants is also diagnostic.

To date, there are 5 types of Loeys-Dietz as of last check. These are due to pathogenic variants in the TGF-B signaling pathway, such as TGF-beta receptors and SMAD3 genes.

Vascular EDS is caused by a mutation in the COL3A1 gene which encodes a defective type of III procollagen. The defect in the procollagen makes it unable to properly fold into a triple helix that forms the normal collagen structure. This

causes the defective procollagen to be degraded intracellularly and as a result there is an overall deficit in type III collagen which is an important component of arterial walls and other structures. The confirmatory test for VEDS is collagen testing which can confirm the collagen III defect.

#### **How would you manage these patients?**

Medical optimization and surveillance is key to try to extend the time as much as possible before they get a dissection and avoid it if at all possible.

We start with lifestyle modification. Avoid “burst” exertions such as sprinting and weight-lifting. Anything that very strenuous. That’s not to say that they shouldn’t exercise. Light exercise is encouraged, but this would be activities like light jogging, swimming laps, or biking.

In order to minimize aortic shear stress, a resting heart rate of under 70 beats per minute and an exercising heart rate under 100 should be the goal. This can be accomplished with beta blockers. Propranolol has been shown to significantly decrease the rate of aortic growth in Marfan’s patients with a baseline aortic root diameter under 4cm. There is research into the use of Losartan in murine models that suggests it inhibits TGF-beta in the aortic wall, which is an important pathway that contributes to the breakdown of the wall. However, randomized controlled studies have failed to show an increased benefit of Losartan over beta blockers in Marfan’s patients. ACE inhibitors are also being tested and are shown to decrease the risk of type b aortic dissection over 6 years.

In vascular EDS instead of propranolol, celiprolol has been studied by the French and shown to reduce vascular rupture from 50 down to 20% in vEDS, although the mechanism of this is not yet clear and does not appear to be necessarily the same as decreasing shear stress as in Marfan’s syndrome. In general taking care of these patients involves trying to minimize complications from procedures and interventions. For instance, use ultrasound for any line that is necessary and avoid arterial lines, intramuscular injections, or other invasive lines if possible to minimize the chance of a complication. Patients are advised to wear medical bracelets notifying that they have vEDS.

We also discuss the importance of forming a care team based on their needs. This usually includes a cardiologist, a cardiac surgeon, a vascular surgeon, and a primary care physician.

#### **What about surgical treatment for those who need it?**

For patients with Marfan’s, prophylactic surgery is recommended for aortic root dilation  $>5\text{cm}$  or thoracic aorta  $>5.5\text{cm}$ . Often times the thoracic and abdominal aorta are involved. Open and endovascular surgery are options for these patients. Open procedures often include open thoracoabdominal aortic aneurysm repairs, open cardiac surgery for arch replacement, or cervical debranching procedures. Endovascular procedures can include regular TEVARs or branched TEVARs which require extensive aortic coverage. Open surgery can be well tolerated and is ideal in the sense that you can replace the entire aorta which

avoids the future complications from continued aneurysmal degeneration, loss of proximal or distal seal zones, or device issues that can plague endovascular methods. However open surgery, of course carries higher complication risk and morbidity up front and does share some complications with endovascular treatments as well. Sometimes these patients will have hybrid procedures and often their care will require multiple surgery teams including cardiac and vascular surgery. An important thing to be up front with all of these patients about is that this is a long term relationship with their surgeon, as they often require multiple staged procedures, things aren't fixed in one procedure, and even after they have been surgically addressed there is a lifetime of maintenance and surveillance. Ultimately, the decision for open vs. endovascular approaches will vary between patients based on their specific anatomy and arterial issues, what their body can tolerate, and ultimately what their goals of care are.[Lum et al., 2012a] Some may require having their entire aorta replaced, while others may only need ongoing medical therapy and surveillance and it's important to set expectations early.

**What about VEDS, when surgery cannot be avoided? How do you mitigate the risk of complications?**

The tissue is very fragile. So using instruments that are the least traumatic is key – like Fogarty clamps for vessels. Sutures often must be pledgedged to reinforce them. Leave no tension on anastomoses or suture lines. Always keep a backup plan in mind – when arteries cannot be repaired, can they safely be ligated or embolized? Generally any large bore access for endovascular treatment is avoided because access site complications are high and can lead to devastating consequences. In situations of extremis, like a rupture, these patients' tissues have been known to completely breakdown. Try to avoid the worst case scenario, but of course sometimes it's the only option left to get out of the OR. Be upfront with the patient about how complications may arise, set expectations, and think about goals of care early.

**We discussed earlier that these aortopathies can have shared phenotypic characteristics, some of which can be used in a clinical diagnosis, but are all of these genetic aortopathies syndromic?**

Let's start by saying that all patients with Marfan syndrome and VEDS can have the syndromic features we just talked about. However, it's not always the case and the absence of these features does not exclude the diagnosis. In fact, we recently treated a middle-aged woman with an aortic dissection who had Marfan Syndrome confirmed with genetic testing. She had been diagnosed prior to her dissection because her daughter had undergone genetic testing. However, on meeting her, I would not have guessed she had Marfan Syndrome, had I not known. She was average height, obese, and had no other relevant physical findings on exam or history.

This ties into another genetic aortopathy that we have not discussed yet which are the familial thoracic aortic aneurysms and dissections. They do not have

any syndromic features. For example, patients with ACTA2 pathogenic variants that cause alpha actin mutations which again contribute to degeneration of the arterial wall. These patients tend to present 10 years younger than sporadic thoracic aortic aneurysms, generally in their late 50s compared to late 60s, and women seem to be less often effected than men.

**Dr. Shalhub, I know vascular genetics is one of your passions. Is there anything else you want people to remember from this broadcast?**

Don't forget the family. Once you've made the diagnosis in one of them, remember it is autosomal dominant, so it's important to make sure the family understands and that they are set up with the appropriate care team and monitoring. They may not all develop the same medical issues, however as we discussed, ongoing medical management and lifestyle changes are the key.

### 8.3.1 LINKS:

VEDS Research Collaborative study:

<https://www.vedscollaborative.org/get-involved>

<https://depts.washington.edu/vedscoll/>



# Chapter 9

## Venous Disease

**UNDER CONSTRUCTION - This Chapter is Currently in DRAFT mode**

**28 Nov 2021:** *Mr. Andrew Nickinson, Mr. Aminder Singh and Mr Manj Gohel*

### 9.1 Chronic Venous Insufficiency

#### 9.1.1 Terminology and Presentation

**What is chronic venous insufficiency and how common is it?**

So I thought the first question was going to be a nice, easy uncontroversial one and you've landed with CVI. What you've hit upon is one of the main problems with venous disease, which is terminology. So if you type in chronic venous insufficiency into Google, you end up with, this enormous range of descriptions and how its used in lots of different ways, by lots of different people.

One of the problems is that this led to a lot of confusion among people within the space let alone everywhere else. So really important documents to guide people to is the VEIN-TERM consensus document published in 2009.[Eklof et al., 2009] The lead author was Dr. Bo Eklof and this was a document where a whole group of venous experts around the world got together and said, okay, we need to be clear and define what all these different terms mean.

And what are the terms that he found was chronic venous insufficiency. And the official definition is it is a venous disease between C3 and C6 on the CAEP classification. We'll talk about that a bit later, I'm sure. But between C3 and C6. In practice, it is used to describe the entire spectrum of venous disease ranging from thread veins C1 all the way up to venous ulceration. But technically it is C3 to C6. And I would urge anybody who is learning about the terminology to look at the vein term documents.

And silly little things like great saphenous vein and small saphenous vein—I think UK is the only place where long saphenous is ever used and the real problem was LSV in lots of other countries will be the lesser saphenous vein, which is the small saphenous vein. So again, I'd urge anyone to look at that document.

**How common is chronic venous insufficiency?**

Chronic venous disease is very common, with the weather getting a bit nicer, people wearing shorts, you're spotting, venous disease all over the place. The studies that have been done indicate that at least a quarter, probably up to a third of people have C2 to C6 disease. So very common problem, lots of people that need treatment.

**Now we would like to discuss the classification and scoring systems, you mentioned CAEP, could you expand on that?**

So CEAP--clinical, etiological (with an E because it's American), anatomical, pathophysiological-classification system was introduced, I think in the mid 1990s, by the American Venous Forum and American colleagues. And it's a classification system.

So if you have a patient that comes into your clinic with a venous problem, it's a system to allow you to describe that patient's current situation in an objective, clear way. What it does not do is give you any information about prognostication and it isn't useful for measuring responses to treatment.

So it is purely a descriptive classification system. And the most commonly used bit is the clinical bit, which is C1 one to C6. And again, CAEP was just recently revised last year, a 25 year revision, which again, I would urge trainees to have a look at, Dr. Fedor Lurie was the lead author, and just some useful revisions of CEAP.[Lurie et al., 2020]

Now in response to the limitations of CAEP, the Venous Clinical Severity Score (VCSS) was created. So this is a 10 item scoring system, each item scores between zero and three. So you get a maximum score of 30. Now this is designed to be responsive to treatment and complimentary to CAEP.[Vasquez et al., 2010]

There are lots of other things that have been described, quality of life tools or VDS, but I would say that those two are the most relevant and important ones to know about. But many of the QOL tools are too cumbersome to use in day to day practice. We are in dire need of a easy to use clinical tool that is validated for assessment of patient reported outcomes. And again, the number of papers I've reviewed where people have reported CAEP scores as one of the outcome measures in response to treatments. That is not what it's designed for, for example, you can never get lower than C5. You can never get better than a healed ulcer. So again, that's the important distinction.

### 9.1.2 Evaluation

**When we do see patients in clinic with chronic venous insufficiency, which type of imaging modalities should we use to investigate the function and the anatomy of the lower limbs?**

If a patient comes to clinic, we can't underestimate the importance of the clinical assessment. And when you assess these patients, you really looking for two things, you are looking for the impact of their venous disease on their quality of life and the impact of the venous disease has on their normal function.

But also you're looking for complications of venous disease. This is a clinical assessment. But then when ordering investigations, you really want to identify underlying treatable causes of their venous disease. And that's the goal of any investigation.

Venous duplex imaging is obviously the first line, gold standard that pretty much anybody will go to around the world. And what that does is essentially gives you information about the flow, both in the normal antegrade, but also retrograde or refluxing flow in superficial and deep veins.

It'll give you information about the anatomy of the veins , which will help you to plan treatment, but it also gives you lots of other information as well. It gives you information in the common femoral vein, for example, if there's good phasicity of flow or if there's no scarring. It helps you to evaluate whether there might be some outflow obstruction, so duplex imaging is the absolute first line, gold standard. For more on venous duplex scanning, see 12.7.

Now, once you've done the duplex imaging, if there's clinical suspicion, then of course we can move on to other investigations. So proximal CTV, MRV, venography, IVUS, all sorts of other weird and wonderful things. But clinical assessments and duplex assessment are the bedrock of every assessment of every venous patient. So that's the foundation.

### 9.1.3 Management

**Moving on to the treatment of superficial disease. When we talk about treatment of superficial venous incompetence, I don't think we can really get away from talking about the two major studies, the ESCHAR[@gohel2007] and the EVRA[Gohel et al., 2018] studies, which I know you've been involved with. Can you review what these studies showed and their importance in the care of our patients.**

Before I go into the trials, I'm just going to go back a step and just talk about the general mindset that's important for these patients. So what we're treating with these patients is not superficial venous reflux. What we're treating is chronic venous hypertension. So we're treating a global pathophysiological entity of which the superficial reflux is one correctable factor. And the reason that's

important is that there may be other correctable factors, such as venous outflow obstruction or other sort of mobility related things can be adjusted.

But they're also going to be lots of uncorrectable factors, such as poor mobility, heart failure, dependency, ankle stiffness. If your ankle is stiff, you can't use your calf muscle to pump effectively. You don't have that mechanism for clearing the pressure out of your legs. So the success of these treatments, particularly for the ulcer population, has to be taken in the context of treating venous hypertension as well.

Having said that the aim of these trials was really very simple. Most patients with venous ulceration have got superficial reflux. So what is the role of treating the superficial reflux? That's the simple question that was asked by these trials.

So the ESCHAR trial recruited between 1999 and 2002, 500 patients with healed and open leg ulcers. They either randomized to compression or compression and traditional, superficial venous surgery, stripping or ligation. And that trial was very clear. It showed that with venous stripping there wasn't a benefit for healing, but there was a significant and sizable reduction in the risk of ulcer recurrence.[Gohel et al., 2007]

So one of the criticisms of ESCHAR was lots of people didn't have the same surgery. Lots of people had just ligation alone, et cetera. We don't really do stripping anymore, it's all endovenous. So EVRA really aimed to bring the ESCHAR trial up to modern practice. We randomized 450 patients with open leg ulcers to early endovenous treatments, delivered within two weeks versus a more delayed approach. What EVRA showed, again is common sense, if you do with the underlying problem, the venous ulceration healed significantly quicker.[Gohel et al., 2018]

So put them together clear, unequivocal argument for treating the superficial reflux in these patients as quickly as possible.

#### 9.1.3.1 Endovenous

**Can you broadly outline the different treatment options that can be offered to patients with superficial venous reflux?**

I have counted about 42 different ways of murdering a saphenous vein. Long and short of it, there are lots of different treatment options. Do something that you're trained in and proficient with and something that's appropriate. Thermal ablation is in the NICE guidance and in lots of other international guideline documents has been combined, whether it's radiofrequency or laser. Although foam sclerotherapy therapy, when studied in randomized trials, has lower technical success rates and lower vein closure rates, the clinical success rates in terms of healing are just as good. This sort of five, six, even 10% reduction in closing the vein, doesn't really seem to translate into a significant drop in clinical effectiveness.

So I think the most important thing is not necessarily what you use, it's doing it quickly and delivering it to the people that need it.

**When we are consenting patients for endovenous intervention, what risks should we discuss with the patient and that they need to be aware of?**

You can spend an hour doing this, if you're being very diligent. But what I say to patients are that there are generic risks of venous thromboembolism is the risk of any intervention. And I think that's worthy of specific discussion after endovenous procedures, but I quote a risk of around one in 200 for most endovenous interventions.

There's always a risk of bruising. And if you do phlebectomies, what I often tell people is that bruising sometimes looks worse than it feels. And with the thermal interventions in particular, I think the risk of nerve related complications, so some numbness or some nerve pain, are worthy of mentioned, and of course the risk of recurrence.

Foam sclerotherapy has a different mechanism of action. It's a detergent that's injected into the vein that kills the endothelium. And there are some people that have associated foam sclerotherapy with some neurological events. So a very large registry that was organized by the manufacturers of STS recently looked at 10,000 patients in the UK and found very few events-around one in 4-5,000. Some people say that if there is a history of migraine, then the risk of these neurological events might be a little bit higher.

So, a little bit of caution, although in practice, I've treated many people with migraines. But if you believe the literature, one in four or five, people have got a little ASD and obviously we treated lots of those people, so the risks are very low but it's just important to have that discussion beforehand.

**Are there any techniques that we can use to mitigate some of these risks?**

So I think VTE is something that is really worth specifically discussing. I t's a very rare event after superficial venous interventions, but it's potentially catastrophic. It's newspaper worthy, if young people end up having a very bad outcome. What we've got to realize is that the rate of VTE events is very low, there are almost certainly some people that have got a higher risk. And so what we've got to get slightly smarter about is identifying the people that have a higher risk of VTE and maybe giving them prolonged course of thromboprophylaxis, as well as appropriate counseling and mitigation as well.

So it's about getting smarter about risk assessment. The current risk assessment tools are not really fit for purpose when it comes to superficial venous intervention. So the department of health or the Caprini tools don't really take into account some of the really important factors that I think do impact on VTE risk after superficial venous interventions.

There's nothing validated, there's nothing widely available, but we have a local, specific risk assessment for patients having superficial venous interventions. There are major risk factors we look out for, such as previous ipsilateral VTE, active malignancy, but also significant chronic inflammatory conditions (i.e. inflammatory bowel disease), these patients have a much higher risk of VTE than we previously recognized. There are also minor factors, superficial vein thrombosis, obesity, comorbidities. All of these things are put together and we come out with a score. For patients above a certain threshold, they get offered extended rivaroxaban or low molecular weight heparin. So then we can get smarter.

In addition, it is important to implement mitigation. So traditionally things like a contraceptive pill, HRT and Tamoxifen, we wouldn't usually stop them for a local anesthetic procedure, but being on an estrogen containing pill doubles or triples your risk of VTE. It's an easily reversible thing in the short term that can be done. So for my patients, I would normally recommend that we stop the pill a full cycle before, and similarly a month before and after the procedure.

**You mentioned it an extended course of anticoagulation for those patients with risk factors. Is there a specific timescale locally that you use for this?**

The venous forum produced some guidance the year before last in exactly this area. We often only do about a week or 10 days for a lot of these patients. However, if there's an ongoing, persistent risk factor, then actually the VTE risk persists for four to six weeks, and thus there may be an argument for up to six weeks of therapy in particularly high risk patients.

However, if they are really that high risk, you've really got to question whether a superficial venous intervention is needed, balancing the risks and the benefits. But since for some patients the high risk period persists for at least another six weeks afterwards, be careful not to stop prophylaxis too soon.

**Moving on to a more specific question about treatment, what is the role for treating and competent perforator veins in these patients?**

In most sensible practices, the more advanced the venous disease, the more likely you are for the disease to be recurrent, the more likely there is to be deep venous disease or posts thrombotic disease, and the more likely you are to finding competent perforators. If you imagine these patients with venous ulcer, it is very common to find perforators, and if you take a sort of super aggressive role saying you've got to obliterate every single bit of reflux you can find, then you'd be doing a couple of perforators in every single patient, and that is the approach of a number of centers around the world.

The pragmatic reality is, number one, if you ablate the superficial reflux, we know that a lot of these perforators actually become competent because we've got rid of the outflow or you've changed the dynamics in some other way, and they've become competent. Number two, even if they've stayed incompetent, the clinical benefit is still there and is usually still pretty durable.

Now, of course, there may be some people that develop recurrent problems and then there can be a more targeted approach to the perforator. The ESCHAR and EVRA studies did not target perforators at all and the outcomes in the EVRA study was the best healing rate of any published prospective leg ulcer study. So, putting all that together, it's difficult to make a case for aggressive treatment of perforators first up. Having said that for some people with recurrent disease, it's not uncommon to have a big mid thigh **Huntarian perforator** or **Cocher's** perforator lower down on the medial side. So, I do treat perforators, but almost always it's in recurrent disease with recurrence or deteriorating symptoms.

#### 9.1.3.2 Open Surgery

So we've discussed quite a bit about endovenous intervention. Is there a rule for open surgery in some patients?

Open surgery remains the most commonly used superficial venous intervention around the world, and there are still lots of centers in the UK that primarily offer open surgery. Being balanced, it's a very effective treatments if you do it well and if you use modern approaches tumescent anesthesia ultrasound guidance, then actually some of the traditional issues with open surgery, which are often technical and complication related, don't really apply.

Having said that all the randomized trials have shown the same thing while the effectiveness may be as good in open surgery done well, the recovery is much better after endovenous ablation. So it's difficult to make the case for open surgery when you've got something that is so well established and the complications just so low, and it's cheaper when you look at their theater capacity, et cetera.

So, I think there has to be a steady drift towards endovenous, but there are lots of things stopping this. In Germany, for example, the reimbursement is greatest for open surgery. So surprise, surprise, there are still lots and lots of open varicose vein operations. And again, for you guys, if you're going to change practice, the single biggest driver for change in practice is reimbursement. If you can, you can change where the money goes. You can change whatever you like. So, the final thing is I've treated maybe three patients with open surgery in the last five years. One was a GP, who I had treated the other leg with open surgery a few years earlier, and she said, "I want the same operation, please." So I did struggle to argue with that. The other two were big three, four, five centimeters saphenovarices in the groin. So specific indications, but not really for most patients,

#### 9.1.3.3 Medical Therapy

So we've talked about endovenous and surgical treatment, but are there any pharmacological therapies that can be used in patients with chronic venous insufficiency?

So we're pretty skeptical in the UK, when it comes to venoactive or pharmacological treatments. If you go to Europe, they love them. They sell like hot cakes. They're over the counter in a lot of pharmacies. The drug with probably the most evidence is something called Daflon, micronized purified flavonoid fraction.

So flavonoids are naturally occurring, venoactive compounds. There are a whole variety of effects, only some of which we understand. But a lot of studies have shown improvements in edema and heaviness. Daflon is marketed by a French company and it's got good evidence, but they do not see places like the UK as a big enough market because of our inherent skepticism over these sort of things, to make it worth their while to go through the marketing and the regulatory stuff. So it's not available. But patients that have gone to France and picked it up over the counter and have said it works well. So there may be a rolling and pentoxyphyllin and these other things—there's some good evidence that it accelerates healing of leg ulcers.

But I don't think we're very receptive in the UK in general, not in the vascular surgery community to prescribe pharmacological or herbal type remedies.

**What are the healthcare costs associated with the lower extremity venous disease?**

The educated guesstimates in the UK, NHS, is around 2 billion pounds a year. The UK NHS budget is about a 120 billion. So, up to about 2% of the budget, and that's just C6 disease. We're not even going into all of the other diseases, the associated cellulitis, but also not only the healthcare costs, but the societal costs, the time off work, the other things. And then of course the superficial vein thrombosis, VTE and other associated diseases, it's a massive underestimate

#### **9.1.4 Other Complications of Superficial Venous Disease**

##### **9.1.4.1 Superficial Vein Thrombosis**

**You mentioned superficial vein thrombosis, also known as superficial thrombophlebitis. What is it and how do you manage it?**

I'm very pleased you used both terms because I'm going to take the opportunity to encourage robustly the use of superficial vein thrombosis rather than thrombophlebitis. The problem with thrombophlebitis is it is too closely associated with an infectious etiology. And I can only imagine how many useless courses of antibiotics have been prescribed for the treatment of what has been presumed to be an infection. Of course, there's no infection, it's a thrombus in a superficial vein. I think what's really important is that traditionally it's been seen as just a bit of a nuisance and let's not worry about it, treat with analgesia and anti-inflammatories, and you'll be fine.

It's a really sinister pathology and an important cause of VTE. And again, if you think about it, think of how many thousands, millions of people that have varicose veins. They don't all get clots in their varicose veins. They've all got

stasis to a certain extent but they don't get clots. So it's more than just the flow dynamics is going on here. These people are thrombogenic. They have a thrombogenic innate quality that leads to this. That's shown out in the study, some really good work in some French studies. They identified patients who were scanned with superficial vein thrombosis, and a quarter of them on their first duplex scan had a DVT. A lot of these DVTs were remotes from the superficial vein thrombosis. So they're not all extending into the deep vein—they were remote—confirming this idea that actually a thrombogenic problem is going on here.

So in the latest ESVS guidelines, there is quite a lot of stuff about SVT treatment algorithms. I would urge people to have a look at that, and it's important to risk stratify people. The closer you are to the junction, the higher VTE risk, and actually for anyone other than just a bit of clot in a varicosity, anticoagulation is the treatment of choice to reduce progression to VTE.

So look at the guidelines, it's very clear what we should be doing with these people, evidence-based. Once the anticoagulation is done, they need to be re-scanned and that residual incompetent, scarred thrombogenic, saphenous vein needs to be ablated, otherwise they'll recur. So a real change in mindset and level of aggression.

#### 9.1.4.2 Bleeding

##### **What's your approach to managing patients with bleeding varicose veins?**

Really important question. It is a vascular emergency. There are several depressing reports of patients having died from bleeding from a varicosity. These are often elderly patients who are frail, who have arthritis in their hips, and therefore can't bend down to press on the bleeding varicosity down by the ankle ulcer. There's some very grim photos in various case reports and it's really sad. There's two or three photos that I've seen where an elderly patient has not wanted to wake their partner and so has gone and laid down in the bath with this bleeding vein that they couldn't control and basically exsanguinated.

So, it's a vascular emergency and these patients should be seen and assessed and their superficial venous disease should be dealt with as soon as possible. We had an emergency clinic running and anyone with bleeding veins have the same urgency as the CLI patients and they were treated there and then.

In terms of the treatment, there's two aspects: you want to decompress the venous hypertension by ablating the saphenous reflux, but the specific bleeding area, I think there's a real case for some local foam sclerotherapy. You want to block off that vulnerable bleeding vein to ensure that this doesn't happen again. But again, if anyone receives a referral, these are people to see straight away.

## 9.2 Acute Thrombosis - Deep venous thrombosis (DVT)

So let's move on to discuss deep venous thrombosis. You're recently involved in the European society of vascular surgery, venous thrombosis guidelines. In the most part, calf DVTs are managed by medical teams or dedicated DVT services within the hospital. So in this section, we'll be focusing mostly on proximal DVT.

### 9.2.1 Physiology

In medical school, we've learned a lot about Virchow's triad and the etiology of venous thrombosis. Does that simple concept still hold true today?

I think it does. There's a few caveats and a few nuances, but I think the principle that the flow, the vessel wall, and the blood constituents are the main factors that affect whether or not you get thrombosis in a vein is still pretty solid. However, there is also a real change in mindset about the whole idea of provoked and unprovoked DVT. Again, I would urge people to look at some of the new areas that we've covered in these guidelines.

### 9.2.2 Etiology

You mentioned there about potential risk factors for DVT, and that can be classified into these provoked, which can be transient or persistent, and then unprovoked risk factors. Can you talk about some of the specific factors and causes?

In general, what's been happening for several years is a recognition that there are some DVTs that happened with a clear provoking factor—the communist major, transient, provoking factor is something like surgery, for example. So a lot of DVT are caused with that clear provoking factor, but there's an enormous population of people that don't have a clear provoking factor at all, or have a minor factor that may or may not have contributed.

So traditionally there's been a dichotomy between provoked and unprovoked. If it's provoked, they get a limited period of anticoagulation if unprovoked it's long-term. And certainly the last big thrombosis guidelines was the ACCP guidelines, the American CHEST guidelines, and that's what they suggested, but it's much more nuanced than that.

What we've got to remember is that there are some provoked DVTs where there's much higher risk, and those are there's some unprovoked DVTs, which actually there may be other factors that you need to take into account. So I think for those people that are interested in this area, it's really important to get to the nitty-gritty. So, long-winded way of answering your question. But here are some risk factors:

- Major provokes
  - Surgery
  - Major trauma
- Minor provoked
  - Shorter periods of immobility - a few days sick in bed with the flu
  - Contraceptive pill - depending on whether it was just started or the patient has been taking for two to three years

In the guideline document is a big, long list of other things where for people to think about it and consider.

**In those patients where, after you've taken a thorough history, there's no obvious provoking factors. Is there a role for screening for occult malignancies and thrombophilias?**

For thrombophilia testing, the history of it is really interesting. When they were first identified and our ability to screen them first became available, there's a lot of excitement because people thought they were going to be able to identify a clear cause for all of these unprovoked DVTs. The reality is that at least 50% of them, there's no identifiable thrombophilia. So what it almost certainly means is there's lots of thrombophilias that we haven't yet identified, we can't test for. So it hasn't really been the panacea in that regard. The other pragmatic reality for the thrombophilias is that if we're going to be putting people on long-term anticoagulation anyway, the additional value of testing for these things is really very minimal.

And the ones that are really important—the thrombosis history is so stark that they normally smack you in the face. So I think it's generally gone out of fashion. And again, there's only very specific circumstances where we would suggest testing them:

1. If you're going to be trying to stop anticoagulation, for some reason, the patients don't want to be on it, or if there's bleeding risks
2. There's a clear family history. Then it might be worth excluding some of the more sinister thrombophilias.

Otherwise, certainly not a role for routine testing and similarly for malignancy—for unprovoked DVT, the prevalence of an underlying malignancy is somewhere between 6-10%. The studies that have looked at an aggressive, thorough assessment process rather than a more selective process, have picked up a few more cancers, but the additional cost in terms of anxiety, additional investigations, et cetera, and no demonstrable improvement in outcome has meant that the guidelines have not recommended routine cancer screening beyond the appropriate age and sex specific screening—PSA, breast assessments, colonoscopy, etc.

### 9.2.3 Evaluation

#### What is the algorithm for investigation?

The most important components is the first component of the algorithm, which is an assessment of the pre-test probability of DVT. So anyone who comes with a DVT, you can go through a validated tool. The most common is the Wells probability score, which is a series of questions and give you an indication as to whether a DVT is likely or unlikely.

If a DVT is likely, then the algorithm is to do a scan, very straightforward. If a DVT is unlikely, then the algorithm is to do a D-dimer. Because if that is negative, DVT is excluded. The guidelines are very clear on the assessment process, but a lot of this won't reach our radars as vascular specialists at all until the DVT has been diagnosed, so we don't appreciate the importance of the early stages. So again, a quick plug for the guidelines, there's a very nice algorithm, beautiful colors—I definitely recommend having a look.

### 9.2.4 Management

#### 9.2.4.1 Medical

**So the exact choice and duration of anticoagulation in the management of a DVT is probably beyond the scope of this podcast, but on the whole are DOACs preferred over vitamin K antagonists, such as Warfarin and why?**

In a word, yes, lots and lots of studies, funded by big companies that make DOACs, have been done and they've all shown fairly consistent things. They've shown that the DOACs are as effective as the vitamin K antagonists at preventing DVT. They have almost always a significantly lower bleeding risk, but they've got this enormous added convenience of not needing regular blood tests, not having this dietary effect , and the erratic control, et cetera. So the general direction of travel is inexorably towards more and more use of DOACs.

A couple of other areas where DOACs have really shined: The tendency is to recommend longer courses of anticoagulation and often indefinite anticoagulation. A number of studies, both rivaroxaban and Apixaban have looked at the effect of a lower dose of DOAC in these patients. So if you need to extend anticoagulation, can you use a lower dose, a prophylactic dose, so 10mg of rivaroxaban or 2.5mg BID of Apixaban. The results have been impressive because the prevention of recurrent VTE is very good, the same as the therapeutic dose, but the bleeding risk is much lower. So you've got this additional benefit of being able to give an even load those with even lower bleeding risk.

And the real icing on the cake for the DOAC has been a few recent studies that have been looking at cancer patients. Traditionally cancer patients where low-molecular weight heparin only, not for DOACs. But the Hokusai study[Raskob et al., 2018] and the Caravaggio study[Agnelli et al., 2020] more recently have

both looked at Edoxaban and Apixaban in the context of cancer and have shown that actually they're very effective.

In almost all of the areas, DOACs are demonstrating their superiority. The one very notable exception is antiphospholipid syndrome, particularly triple positive antiphospholipid syndrome. This is a pretty nasty acquired thrombophilia where Warfarin is still the gold standard.[Pengo et al., 2018]

**Patients with a proximal DVT, in the iliofemoral or the femoropopliteal segment, is there a role for elastic compression in the acute phase?**

Traditionally people have been a bit nervous about putting compression on patients with acute DVT, concerns about causing propagation and causing PEs and all this sort of stuff. A very important study was the ideal DVT study[Ten Cate-Hoek et al., 2018], run by a series of Dutch colleagues, and a number of other additional studies more recently that have built on the original IDEAL DVT study.[Schreurs et al., 2022]

The short answer to your question is that early compression is really important. It's a really positive thing to do within 24 hours and it's proper compression. It's not just the Ted stocking and see what happens. It's 30 to 40 millimeters of mercury, so that in practice is a class two stocking with a bandage on top. The benefits are reduces the early pain and swelling of the DVT, but also interestingly, it reduces the risk of post-thrombotic syndrome. So if you put compression on early, it reduces the risk of them developing post-thrombotic syndrome. So a lot of focus has been on clearing the vein and early thrombus removal and thrombolysis and thrombectomy and all this sort of aggressive stuff. But just by doing the compression, we can have at least part of the same effect and we've just not been doing it. Very rarely do these patients get proper compression.

So there is a big section in the ESVS guidelines about the role of compression and algorithm as to when, how to use it, and how long do you use it for. What we want to see now is the DVT pathways around the country, around the world, updated and amended with this latest guidance.[Kakkos et al., 2021]

**9.2.4.1.1 Complications - Post Thrombotic Syndrome One of the concerning complications of a DVT, particularly a proximal DVT is post-thrombotic syndrome (PTS). Can you recap the symptoms of PTS and how it's diagnosed?**

PTS is a fairly amorphous entity that includes a series of patient symptoms and clinical signs that develop after deep vein thrombosis. That's deliberately very vague, because the condition is so variable. In terms of diagnosing PTS, the official diagnostic tool is a score of five or more on the Villalta scale. The Villalta score is another one of these tools that have been developed for both making a diagnosis, but also assessing progression of PTS.[Kahn et al., 2014, Villalta et al., 1994]

But in terms of sort of symptoms—people get swelling, heaviness, pain. There is a very unique symptom, which is venous claudication, which is almost pathognomonic of post-thrombotic syndrome with venous outflow obstruction. It's this pain which has a bursting tense quality in the calf on exertion, which does settle on stopping, but it takes a lot longer than arterial claudication. So sometimes people will take half an hour to get better and the leg has to be elevated. So that's venous claudication and that's often the most disabling symptom. But of course, people can develop the same clinical complications than you can with any venous disease—skin changes, lipodermatosclerosis, venous leg ulceration, et cetera. Those are the summary of symptoms for PTS, seen in up to 50% but probably closer to 25% of proximal DVT.

#### **9.2.4.1.2 Complications - IVC Filters A quick question about IVC filters. As vascular surgeons, we sometimes get asked about the appropriateness of inserting an IVC filter. When would you consider one and how long can one stay in situ for?**

So short answer is I virtually never consider one, and if already in situ, remove as soon as possible. Let's expand on this a little bit. So the history of IVC filters is a painful story, particularly in the US, super aggressive use of IVC filters, very few of them retrieved, lots of IVC occlusions, and major lawsuits against big companies. That's led to a worldwide, reluctance to use IVC filter, so now we may not be using them in some cases when we should be using them. The traditional indications and reasons for using IVC filters included weird and wonderful things like floating thrombus and other factors.

The reality is the only real indication that we could come up with was when there's a proximal DVT, and there's a clear contraindication to anticoagulation. So if there's no anticoagulation, then the risk of propagation and PE is reasonably high, so there is a strong case for a temporary IVC filter. If a patient is anticoagulated, very rarely can the case be made for an IVC filter. I don't want to go into nuanced details—such as where somebody already had a PE there's right heart strain, there may be some anatomical features that you're worried about, their anticoagulant control is borderline—there might be specific cases, but this is really very rare. Most people anticoagulated do not need an IVC filter.

#### **9.2.4.2 Surgery**

##### **So clot burden reduction techniques are a hot topic at the moment. Before we talk about endovenous techniques, is there a role for surgical venous thrombectomy? And when would you consider this?**

Open thrombectomy is part of the spectrum of early thrombus removal techniques. It used to be very popular, a very satisfying technique—I think I've done it three times. They've all been for the same sort of indication, which is phlegmasia, severe limb threatening post DVT change, and in a patient where they

couldn't tolerate anticoagulation and therefore weren't candidates for thrombolysis. So you're with a patient where the leg is threatened, you can't anticoagulate them, you can't thrombolize them. Then in that case this is a sort of last ditch approach. But if that's not the case, then almost always a catheter based approach is less invasive and more acceptable.

In addition, most people have got more training. Trainees should read some of the descriptions of open thrombectomy— it's a very satisfying technique, but it's quite involved. It involves finding and exposing the common femoral, deep femoral and femoral veins, applying tight compression around the lower leg to milk the clots out distally to proximally. So the original descriptions are really interesting, but really very rarely would we need to do that.[Comerota and Aziz, 2019]

#### 9.2.4.3 Endovascular

**So you've mentioned catheter directed techniques for clot burden reduction. Some recent trials have challenged our thinking on the appropriateness of these techniques, such as the ATTRACT and the CaVent trial. What's your take on the current evidence?**

This is very controversial. The fundamental issue here is that the trials that have been done almost certainly would not have included a large number of people who would benefit from interventions in the opinion of the investigators. So if somebody has got severe DVT, it is unlikely, particularly in the US, where the ATTRACT trial was done, but these people would have been randomized into the trial.

So the immediate criticism of the trials is that this is not reflective of the population. You've excluded the people that benefit, so it's an underestimate of the benefit of the technique. But if we look at trial results ourselves, there is a modest benefit. In the CaVent trial, PTS rate was reduced. The quality of life was less impressive, but the PTS rate was reduced moderately.[Haig et al., 2016] And in the ATTRACT trial, when they subgroup analyzed the iliofemoral DVT group, then there was a reduction in the number of people who develop moderate and severe PTS. So I think the numbers are something along the lines of, you'd have to treat 10 people with thrombus removal to prevent one person developing moderate to severe PTS.[Vedantham et al., 2017]

So then the discussion is are those numbers needed to treat appropriate for intervention? Particularly when you then start to consider the additional need for stenting surveillance, et cetera. So I think the jury is very much out and it's turning into a Brexit or a Donald Trump like discussion. You've got really stark, strong, firmly held unshiftable views on both sides of the argument and what we need is a bit of nuance. What we need is everyone accepts that some people benefit let's all work together, try and work out, which people benefit. Let's work on case selection.

**So we'll put you on the spot then, when would you offer thrombolysis? Which patients would you offer it in and which circumstances would you also stent?**

There's no role for thrombolysis in anything other than the iliofemoral segment. So if it's infrainguinal, proximal DVT, even if it's severely symptomatic, the studies that actually cause harm by attempting early thrombus removal. That's the first important point.

Even if they have got severe symptoms, there's always a role for conservative management, compression elevation and a lot of these people have a dramatic improvement in their symptoms. If they don't, and there's an honest discussion, and the bleeding risk is low then potentially there is a role. But when there's a treatment with a borderline benefit or the benefit is unclear, then I think what we've gotta do pick low risk patients, so low bleeding risk. We've got to choose teams and techniques with the highest risk of technical success. And we've got to pick people where we think the benefit is greatest, i.e. those that have the highest risk of PTS.

Whether or not you stent is very difficult. We had a case not so long ago where we had a very nice result from early thrombus removal, we stented, and the patient thrombosed again early and subsequently was identified to have triple positive antiphospholipid syndrome. Obviously there wasn't the opportunity in the time to make that diagnosis prior to the decisions around the thrombus removal, so you end up stung and now this patient has a stent and can be very difficult to open up again. The advocates and the aggressive stenters would say, you can't leave a significant stenosis after thrombus removal, otherwise they will rethromboses. Whereas others will say, if we go back to the early studies, like CaVent, very few, if any, people actually had stents, but actually their results were still reasonable.

I think once you get on the roller coaster of thrombus removal, it's very difficult not to then proceed also to stenting, if you see a significant lesion, for example, at the May-Thurner point. Final comment, I'll make is that the use of recanalization and stenting for chronic PTS is a very effective technique. So another approach, if you're not sure, is to manage them conservatively, but always have that up your sleeve, if they get significant PTS at six months, no earlier, then we could potentially consider recanalizing and stenting at that point.

**Following thrombolysis, what duration of anticoagulation is required?**

So the whole point of anticoagulation after DVT is to reduce the risk of propagation and pulmonary embolus, firstly, but also then to reduce the risk of recurrent VTE. The aims don't really change if you thrombolyzed or if you put a stent in. Some people used to say, I put a stent in, we've treated the May-Thurner and that was the main cause of DVT, so we can stop the anticoagulation. That's not the case. So it shouldn't change and it shouldn't be any shorter if you've thrombolized and put a stent in.

Now, whether it should be longer because you put a stent in that's debatable. Some people have very aggressive anticoagulation protocols after stenting, but that's to do with the stent rather than the DVT. So remember the aims, which are to reduce the risk of recurrent DVT and PE and to stop propagation of the current event.

### 9.3 Phlegmasia

**Some patients may present with very profound, lower limb swelling. Pain and cyanosis with a threat to viability of the limb. Can you explain for us what phlegmasia is and an approach to treatment?**

There's two sorts phlegmasia that are described, but to be honest, I don't think anybody has a detailed and robust understanding of what these actually mean. Phlegmasia alba dolens is the white leg, cerulea dolens is the blue leg and the severe, form a phlegmasia. Theoretically, cerulea dolens has a limb loss rate of 30-40%--severe limb threat, compartment syndrome, skin blistering, swelling, etc. The reality is that it's often over-diagnosed, so you'll see a lot of people saying I did venous thrombolysis for phlegmasia and it's just a big swollen engorged leg.

The reason it happens is almost certainly a profound obstruction of venous outflow. So the venous blood in the leg cannot escape. You get this cycle of worsening pressure, leaking of fluid from the capillaries, increasing pressure on the soft tissues. Then that starts to threaten tissue perfusion and potentially if it's bad enough this will threaten the actual arterial profusion of the leg as well.

So that's the sort of most severe and extreme form. I don't think anybody has confidently linked the anatomical findings and features of a DVT to what the leg looks like. There was a patient I saw who literally had every deep vein acutely thrombosed from the IVC down to both lower legs and was almost asymptomatic. So we've got this very uncomfortable lack of understanding between the symptoms and the clinical and anatomical pattern of DVT.

### 9.4 Deep Venous Obstruction

#### 9.4.1 Etiology

**So finally, let's move on to talk about the treatment of proximal deep venous insufficiency, both obstruction and incompetence. What are the common causes for deep venous obstruction? Thinking about post-thrombotic and non-thrombotic obstructions.**

So again, the terminology is important. So it's venous outflow obstruction. Obstruction implies that there's a physiological barrier causing the consequences. As opposed to occlusion, which is an anatomical term. So venous outflow obstruc-

tion, is essentially anything that reduces the ability of venous blood to escape the leg via deep venous channels will cause an obstruction.

And again, going back to a bit of physiology. Poiseuille's Law talks about the flow rate being the fourth power of the radius. So it's really interesting. A question to ask is that if you've got a two centimeter vein, how many, one centimeter veins would it take to carry the same potential flow as a single two centimeter vein?

The long story short is that it takes 16 one centimeter veins to carry the same amounts of potential flow as a single two centimeter vein. So it doesn't take much of a narrowing to have a great potential impact on flow. Of course, most of the time, you don't need that flow potential, it's only an absolute extremes of circulatory stress that you'll need that sort of potential, but it is interesting. And again, it also challenges when people say there's great collaterals and that will be enough. Well unless they're enormous, you need 16 of them to have the same potential blood carrying capacity.

So in terms of the causes, the most common by far is post-thrombotic disease. So when you've had a DVT, one of three things happens. If you're lucky, the vein will recanalize completely. If you're unlucky, it will obstruct completely. And if you're somewhere in between, you'll end up with partial stenosis and obstruction. What's important is even if the vein is open, what you almost always lose in the post-thrombotic vein is a lack of compliance. It's not the same compliant vein that can respond to demands and physiological need that you have, in a primary non-scared vein.

Other potential causes are a non-thrombotic iliac vein lesion, or May-Thurner lesion. So the original paper, 1957, is very interesting reading.[May and Thurner, 1957] There were a couple of Austrian pathologists who were looking at kind of various specimens and they identified that a very high proportion of people, particularly those had left-sided DVT had, not only the overriding iliac artery causing compression, but they had a secondary tight fibrosis in the iliac vein. So it's not just the compression because any one of us in a certain position we'll have a squashed vein. It's the fact that there's a secondary inflammatory fibrotic change, which is what's causing the obstruction. So that's a true May-Thurner or non-thrombotic lesion.

The other big group of patients of course, is malignant lymph node or retroperitoneal fibrosis related venous outflow obstruction. But post-thrombotic by far was the most commonly diagnosed pathology.

#### 9.4.2 Evaluation

We may see patients who come in with severe venous disease with skin changes, for example. When would you consider imaging the proximal deep veins in addition to infrainguinal superficial and deep veins of the leg and what are the best modalities to do this?

So we are really getting down to the importance of a detailed assessment of the entire superficial and deep venous system. Every patient with venous disease should have a whole leg infrainguinal duplex to start with—that's deep and superficial veins. There's no real role for just looking at superficial veins. So you need to understand the context. In terms of if their obstructed or if they're refluxing. Again, remember that, reflux on a duplex, yes, all we see is arrows going in certain directions. But all reflux means is that after a calf augmentation maneuver, there is some retrograde flow—in a superficial vein, more than half a second and in a deep vein, more than one second. That's all it means. So for example, you can have retrograde flow for 0.6 seconds in a saphenous vein, and that will be labeled as reflux for all the rest of the time that superficial vein is doing the right job, it's returning the blood to the heart. So just be wary of interpreting what these arrows mean. For more, see 12.7.

In terms of how that affects your decision-making—one approach is to consider the overall burden of venous disease, such as those with venous hypertension. And what I try and do is draw a pie chart and say, you got venous hypertension, here are the different causes of your venous disease—that might include superficial reflux, deep venous reflux, deep venous obstruction, and other factors. You try and apply some clinical judgment as to what the different factors are.

Now, even if they've got significant, deep venous reflux and superficial venous reflux, there is still a role for treating superficial reflux in most of these cases. I still come across colleagues and other people who say, look, you shouldn't treat the superficial veins if there's deep reflux. But all the studies—EVRA, ESCHAR—have shown significant benefits, even if there's deep flux.[Gohel et al., 2007, 2018] So again, the key message is treat the superficial reflux if it's there.

Now, deep venous obstruction and looking for it is a different matter. There are again, lots of approaches, some people are very aggressive about evaluating the iliac veins—CTVs and IVUS on everybody. The problem is if you get super aggressive, then you start to over-diagnose and if you start to over-diagnose, you start to over treat and this all gets very messy.

I think the sort of people that have got clear signs and symptoms of major venous outflow obstruction, particularly things like venous claudication, history of previous DVT, really disproportionate venous changes to the superficial and the infrainguinal imaging. Those are the people that I would image.

And on the duplex imaging of the leg, if there are changes in the common femoral vein that make you think there is a proximal problem, particularly a loss of phasicity of flow then that's pretty concerning for a significant venous outflow problem. In an unobstructed venous system, the common femoral vein should really reflect the right heart, which has some phasicity. There should be transmission of that respiratory phasicity. So if that isn't there, then you're thinking, hang on, there's something interrupting that transmission, which is almost always an obstruction somewhere. So those are the people I'd image, but I wouldn't over image—because then you end up with a headache and a

problem—Should I treat or should I not treat?

**So you mentioned IVUS, what is intravascular ultrasound and what are the particular benefits of using this technique over venography alone?**

IVUS an approximately nine French probe that is inserted into the vein and it gives you a 360 degree view in B-mode ultrasound—traditionally without color flow, just B mode—of the anatomical structures. What it's very useful for is identifying venous anatomy, confluences, identifying the size of veins, so it allows you to plan sizes of stents. It really is an important adjunct to venography and other imaging modalities. We did our first 30 or so cases of deep venous stenting without IVUS and thought we were brilliant and IVUS was a waste of money. And then we started using IVUS and realized I can't believe we were doing this without. Once you start using it, you suddenly realize that it's a really useful adjunct.

In terms of when you use it, in some parts of the world it's commonly used for diagnostic because the risk of over-diagnosis, but more commonly, almost routinely, for therapeutic, particularly when we're stenting deep veins to plan our landing zones.

**You talked about over-diagnosis. Can you talk us through some of the decision-making processes that you go through when you're assessing a patient who has a deep venous obstruction?**

There are lots of patients that have this anatomical change, the number of referrals I get from people who have done a CT and they've picked up a problem. The most important thing is the clinical status and the clinical picture. So have they got symptoms—go about right at the beginning of the podcast—have they got symptoms? Have they got complications? If they haven't got significant symptoms and they haven't got significant complications then there's not really an indication for treatments.

So leg swelling was initially, frequently pushed as, oh you're swelling will get better, if we get rid of your obstruction. That's only the case in probably less than 50% of patients. The reason for that is that, yes the original etiology for the swelling may well have been venous, but you almost always get a secondary lymphatic dysfunction when you've had swelling, for whatever reason—whether it's heart failure, whether it's venous, the lymphatic system gets damaged. And so even if you get rid of the original cause, the lymphatic damage persists, of course, and the swelling persists.

The symptoms are very important and in terms of complications, if somebody has intractable ulceration, et cetera, these are all important indications. But a bit of skin change, a bit of mild swelling, careful case selection is very important for these procedures.

### 9.4.3 Management

#### 9.4.3.1 Endovascular

**So following a deep venous recanalization and stenting, what surveillance do you offer for these patients?**

Surveillance has been evolving. The first availability CE (UK version of FDA) marked deep venous stents in the UK was around 2012. A few services across the country really got started at a few centers in 2012, 2013. Initially there was an early scan and then a scan maybe at six weeks and three or six months down the line.

I think we realized that in general, the people that developed problems, often develop them very early. So we are scanning these people the day after the intervention—obviously there's intraoperative quality control and venograms and scanning—but the day after the intervention to ensure that there's no early thrombus problems. Then we scan a couple of weeks after to make sure that there's nothing that's happened early, because there's an opportunity to re-intervene, to balloon, to correct any technical issues, at that point. After that point, it's usually six weeks, three months, six months, and then annually thereafter.

So that's a sort of protocol that we have and in general, what we've seen is that people that develop problems tend to develop them relatively early. So once you get to a year, then in general, the patency rates are very good. In our center, the chronic PTS stenting primary patency rates at around three years are over 80%-85%. Of course, there are some that have problems, but it's important to remember that, even those that have thrombosed, they generally go back to the level of symptoms they have prior to intervention. Which is very reassuring. The last thing you want to do is to make people worse with a novel intervention.

**And so what about your anticoagulation and antiplatelet regime for these patients?**

There was a systematic review done a few years ago where all of the published literature for deep venous stenting was reviewed and I think the authors identified 28 different regimens for anticoagulation of the stenting.[Notten et al., 2021] The honest answer is nobody knows, our approach is to have low molecular weight heparin as the primary anticoagulant for the initial two week period. And then at the two week point, they undergo a stent. If there's no issues, they get transitioned to a DOAC.

In addition, the patients get six weeks of clopidogrel as an anti-platelet. And again, the reason for that is that it's a foreign material into the vein. Yes, the role of anti-platelets of preventing venous thrombosis is very limited but there has to be some platelet related activity and platelet activation involved there. So it makes logical sense to have some anti-platelet activity. Of course the downside

is that there's an increasing bleeding risk. These are the constant tightropes that you're walking with these patients.

#### 9.4.3.2 Surgery

**We've talked about endovascular and endovenous treatments, but is there a role for open surgery in these patients?**

So in the iliac segment, very rarely. In some patients, you can do PTFE reconstructions and bypasses. And of course the traditional operations of Palma-Dale[Palma and Esperon, 1960] bypass and May-Husni[Shaydakov et al., 2015] type procedures. They all sounded very elegant in the books, but the reality is a great saphenous vein going across the lower abdomen to the other side is not really going to sort the problem out in a durable way for all sorts of obvious reasons—the size of the vein, the pressure and all that sort of stuff. So there's really very limited role for those sorts of procedures.

Now, the one area where there may be a role for open reconstruction is in the common femoral vein. The consistent observation is that good inflow is absolutely imperative to get good outcomes in these patients. And sometimes if the common femoral vein has got a lot of scarring, then it's very difficult to reconstruct that inflow endovascularly. So a number of authors have advocated the use of endophlebectomy, opening up the common femoral vein, and cutting out the scarring and the tribulations, putting a patch on it. Sometimes even putting an AV fistula—a little six millimeter graft from the common femoral artery onto the vein to really drive the flow through the stent. It's a pretty complicated operation and you can imagine you're dissecting down and there's lots of little veins everywhere and lots of lymphatics. The wound complication rate is very high.

So really very limited role only—the endovascular approach is really the way forward for these patients.

## 9.5 Pelvic Congestive Syndrome

### 9.5.1 Presentation

**So finally, let's talk about pelvic congestion syndrome. What is pelvic congestion syndrome and how does it present?**

So it often doesn't present anywhere near a vascular surgeon. There are a number of women who present with these chronic heaviness and dragging symptoms in the lower abdomen. There may be some association with menses, there's often dyspareunia and a whole variety of other symptoms.

There may be associated vulvar varices or posterior buttock and thigh varices all as a result of pelvic venous incompetence. But it's a complicated field—there are some people that have pelvic symptoms, there are some people that have

the varicose veins or some will have a combination of the two. Almost always they will be seen and initially assessed by the gynecology teams rather than coming to the vascular surgeons.

### 9.5.2 Evaluation and Management

**When we do see these patients, are there any treatment options available for them? And when would you consider treating?**

In a highly selective way—this is venous disease in a nutshell—the association between anatomical changes and clinical symptoms is ropey at best. In terms of postpartum women, an enormous proportion of these women have got venous reflux in the pelvis—if you look hard enough. so the problem is that if you start looking for May-Thurner syndrome, if you start looking hard enough, you start identifying these symptoms and then you lose the ability to really discern who's going to benefit most.

So again, I would go back to clinical symptoms as the single most important thing. If they've got severe clinical symptoms majorly affecting their quality of life and associated significant venous changes, then those people to try to intervene on. It's not necessarily a benign procedure. Pelvic vein embolization involves implantation of coils. There's concerns about non-targeted embolization coils have ended up in lungs. There are a number of people with unknown nickel allergies that have problems with these coils. So again, it's not an area to be over aggressive. So careful case selection is important. So again, a quick plug for the ESVS guidelines. There is a very good section on pelvic venous disease assessment.[De Maeseneer et al., 2022]

What's very important is again, to differentiate between pelvic congestion syndrome and pelvic origin varicosities. One thing that is certainly being advocated is that if the varicose veins are coming from the pelvis, but there's no pelvic congestion syndrome, it's perfectly reasonable just to deal with the visible varicosities from below—with some foam sclerotherapy or other treatment from below, rather than being super aggressive embolizing from above. But I think anybody who's involved in managing this really needs to work closely with the gynecologists. There are gynecological diagnoses that need excluding before we start to address the venous disease. And there are a number of validated questionnaires and other assessment tools that are probably quite important. So not for the faint-hearted.

## 9.6 Venous Trauma

Iliac vein injury often able to be accessed by ligating the internal iliac artery and mobilizing the common and external iliac artery. Transection of iliacs should only be used as a last resort if this fails. [Lee and Bongard, 2002]

For more on venous trauma, see 10.3.



# Chapter 10

## Vascular Trauma

### 10.1 Peripheral

**22 Sep 2019:** *Kevin Kniery, MD, MPH; Todd Rasmussen, MD*

#### Hard and Soft Signs of Vascular Injury

Hard signs of a peripheral vascular injury:

- Obvious bleeding (arterial or venous)
  - can be pulsatile or even history of bleeding in the field
- Expanding hematoma
  - underneath a closed wound/injury
- Profound ischemia of the extremity
  - determined by the absence of a palpable pulse or dopplerable signal beyond the area of injury
- Presence of an audible bruit or palpable thrill near site of injury
  - concern for an arterial-venous fistula, often high flow

Soft signs of a peripheral vascular injury:

- Injury pattern often associated with a vascular injury (i.e. posterior knee dislocation)
- Diminished blood flow to extremity
  - weak, but audible arterial doppler signal
- Penetrating wound in proximity to a major axial vessel
- Peripheral nerve injuries

- Femoral nerve injury
  - \* paresthesia to anterior thigh
  - \* decreased hip flexion and knee extension
- Sciatic nerve injury
  - \* paresthesia to lateral leg and dorsal/lateral/plantar foot
  - \* weakness of foot plantar flexion
- Tibial nerve injury
  - \* paresthesia over heel
  - \* weakness of plantar flexion
- Deep peroneal nerve injury
  - \* paresthesia of first digital interspace
  - \* foot drop[Bulger et al., 2014]

### **Initial principles of managing a bleeding extremity**

- DO NOT GET DISTRACTED
  - These patients are often poly-trauma patients with likely other injuries
  - Follow ABCDEs of the primary survey
    - \* Identify bleed and hold pressure to stop the bleeding
    - \* Pressure can be manual pressure, such as placing a finger in the wound, or applying a tourniquet
    - \* Once bleeding is effectively controlled, RESUSCITATE and continue the primary survey to address other life threatening injuries

**In Rich's Vascular Trauma Book, they described the injury extremity index. Could you explain this concept?**

If I had to take one tool with me into resuscitation room or an austere location to evaluate for vascular injury, it would be the continuous wave Doppler. It's only more powerful if you can combine it with a manual blood pressure cuff and together allow one to obtain an objective measure of perfusion. One can slowly inflate the blood pressure cuff, just proximal to the arterial signal, kind of like taking a blood pressure, and measure the pressure at which the arterial signal goes away. That can then be compared to a normal or non-injured extremity. If there is no flow limiting arterial injury, the ratio should be one or slightly greater than one.

Conversely, if the ratio for the injured extremity is less than 0.9, then that's indicative of flow limiting injury in the artery to that extremity. If the patient happens to be in shock or cold and clamped down, the injured extremity index can be repeated 10 or 15 minutes later as the patient is resuscitated and warmed.[Rasmussen and Tai, 2022]

**So what do you think the role of CTA is in the workup of extremity with concern for vascular trauma?**

If there are hard signs of arterial injury, oftentimes the patient will require an intervention without obtaining formal imaging. The question about imaging

often comes into play when there's a soft sign of vascular injury, such as a reduced injury extremity index, audible bruit, or injury pattern. We spent decades saying that the traditional arteriography was the gold standard, but I think we can say now that CTA has become the gold standard for the evaluation of extremity vascular injury. CTA has improved in quality and it can also get imaging of the head, torso, or other other extremities.

**And so whether the patient has a hard sign or a soft sign with a vascular injury, can you talk us through some of the principles of operating on peripheral vascular trauma?**

Foremost important is to resuscitate the patient and to be mindful of his or her physiology and hemodynamics. As we said, an extremity vascular injury can be somewhat distracting. Make sure the patient is being resuscitated and warmed, ventilator settings are being corrected, and monitoring is in place – which takes a lot of communication with the anesthesia team, the circulating nurse, the technicians, and the blood bank.

I think once the patient is set and you have the team resuscitating him or her, then you can turn your attention to the injured extremity. I will prep that patient widely and be mindful that the incision is likely to take more than what you think it will. For example, for an above the knee popliteal artery, I will still prep that patient from their umbilicus down to both legs; because you need to anticipate getting proximal control, or doing an angiogram at the femoral artery level and anticipate the potential harvesting of saphenous vein for conduit.

I also leave the tourniquet on and just prep it into the case. Taking that tourniquet down prematurely can be problematic for a couple of reasons. It may result in arterial bleeding, which in itself can be catastrophic, but it can also result in hypotension which poses risk to multiple organ systems. So, I just prep them into the field and then communicate with anesthesia to tell them when I am going to let this tourniquet down. Prior to doing so, I ensure they are ready, they have blood going, and have a secondary measure to control bleeding as you let that tourniquet down.

For the incisions, they just need to be probably twice as long as you think they do. You can't fix what you can't see and if you can't see it, you can't control it, and you certainly can't repair it.

**Do you routinely heparinize all these patients?**

I hate to equivocate, but it depends. It depends on whether or not they have other injuries. So if it's an isolated extremity injury and the patient does not have any torso bleeding, head trauma, or very extensive soft tissue injury, then I will then be more inclined to use systemic heparin during revascularization. If they have any of the above injuries, then I will use regional heparin flushed up and down the injured vessel.[Liang et al., 2016, Fox et al., 2012]

**One question that I've heard come up a handful of times is can you use saphenous vein from the injured extremity?**

Our teaching, as you referenced, has always been to use a saphenous vein from the contralateral extremity, but I am quite practical about it. I certainly have used ipsilateral saphenous vein. If there's no deep vein injury and it's part of the incision and exposure, then I will use ipsilateral reversed greater saphenous vein. If there is concern for deep venous injury or if I have another person to harvest and who can prep the other leg, then often I'll defer to that and use contralateral. I'll be more deferential to using the contralateral saphenous vein in the reverse configuration.[Liang et al., 2016, Fox et al., 2012]

**So you have a vascular injury and you have debride it back to healthy tissue. How do you make the decision between, primary repair, patch repair, or interposition graft with autologous vein or prosthetic?**

You know, you can picture it now, you have vascular control with clamp proximal and distal. Now you're setting up, you are really shifting gears into a different phase of this operation. It's important to just take a deep breath, I think at this point, and check with anesthesia and make sure the patient's doing all right from a global standpoint.

I think the other thing is that at this point, I have sometimes overlooked the importance of performing a proximal and distal thrombectomy using the Fogarty catheter. I'll make sure that I've passed a Fogarty catheter distally to completely clear the outflow of thrombus. Then I'll use regional heparin, 40-60cc of heparin saline down the outflow before I apply the clamp. I'll do the same in the inflow and that buys you a little time as you're sort of thinking about and sizing up what's in front of you.[Liang et al., 2016, Fox et al., 2012]

I think in my experience, the majority of patients need an interposition graft. Certainly, if it's a grazing wound or a stab wound from a knife, there may be a role for primary repair or patch. But if one thinks there's going to be any compromise of the arterial lumen or tension on the repair, then you're better off with an interposition. [Liang et al., 2016, Fox et al., 2012]

There are some special cases where primary repair might be appropriate. Veins are much more compliant and you can often get away with a lateral venorrhaphy. Then sometimes the upper extremities, such as the brachial artery, has more redundancy. If it's a very focal injury and you can get a little stretch, then you will often be able to get the brachial artery back together primarily.

I suspect in the majority of my experience, vascular injuries require an interposition graft. Then in regards to conduit, because most of the wartime injuries were contaminated and quite dirty, we deferred to autologous saphenous vein the vast majority of the time. However, if you are in a pinch, can't find vein or don't have time to harvest vein, the wound isn't too contaminated, and the injury is proximal, axillary or subclavian, proximal femoral, or iliac, then I think using a prosthetic, either Dacron or expanded PTFE Gore-Tex is also acceptable. Some recent civilian literature says PTFE may be better than vein, but that has not been our experience in the deployed setting.

Now that we have covered some basic principles, lets dive in a little deeper on each kind of peripheral vascular injury. First we're going to talk about junctional hemorrhage, which is an injury in the junctional area between the torso and extremity, such as the distal external iliac/common femoral or subclavian/axillary region. These are some our biggest fears and most difficult to deal with. Can you take us through a hematoma in the groin or pelvis just above inguinal ligament—what are the best ways to get control of this?

For the iliac artery or even just the proximal common femoral, I think a retroperitoneal exposure to the lower quadrant of the abdomen is preferred. We sometimes refer to that as a transplant incision, which is quite suitable depending upon the injury pattern.

As I alluded to before, don't underestimate the exposure you're going to need. So, if it truly is a groin hematoma, think that this could be common femoral or external iliac and in those cases, either a transplant incision in a retroperitoneal fashion to get that controlled up to the common iliac, or just doing a laparotomy and finding it through the abdomen would suffice.

**What if you encountered an internal iliac injury deep in the pelvis that you weren't able to control, what are the consequences of ligating that?**

That's real tiger country, in those cases it's really damaged control. One of the things we haven't mentioned is the important tenant – that maybe arguably is one of the most important tenants in vascular damage control – is ligation. We've talked a lot about setting up for a vascular repair, but one of the reasons to check with anesthesia and to assess the whole of the patient is to recognize that in some scenarios the best thing is ligation and then dealing with whatever consequences may result from that.

Keeping in mind, the idea of life over limb and not being too aggressive about trying to do some sort of exposure and repair of a vessel that takes hours and many, many units of blood. So in that context, I think, if you find bleeding from the internal iliac or hypogastric vessels, I think ligating those is quite acceptable. There can be instances of pelvic ischemia, but if the contralateral iliac artery is open, then that's quite rare.

Also, often one is dealing with artery and vein injury because they're right there together. And as I mentioned, that's real tiger country. If that's happening in those situations, keep in mind the tenants of exposure, light, two suctions, and don't use too small of a needle. That's where the SH blunt tip needle on a 3-0 or 4-0 prolene needle works well. Don't try to be too finesse about this because, in the pelvis, you won't be able to see your needle.

**Let's take the same injury and move it now to the external iliac—what are the consequences of ligating the external iliac artery and what are some of your options?**

Because it's the axial vessel to that extremity, I would not ligate the external iliac artery with such impunity. I think in those situations, controlling it and making that assessment of whether or not to repair it is paramount. I think that the consequences of ligating an external iliac artery are going to be significant and include proximal extremity ischemia, including thigh, and probable need for above the knee amputation and even disarticulation of the hip.

So I think the external iliac needs to be preserved at all costs. And whether you reconstruct immediately or use a temporary vascular shunt as a damage control is dependent upon how the patient's doing or if you are in the field and there are other triage concerns.

**Some can find shunts a little tricky, can you give some advice to listeners on how to best use temporary vascular shunts for damage control?**

I think it's important to know what shunts you have available before you are in this situation. So stroll through the operating room or your stock room, wherever you are, when it's not busy and check out the stock of shunts. There are four or five different types of shunts, anything from what's referred to as the Javid shunt, Sundt shunt, or there's the Argyle shunt. Then there can be makeshift shunts, such as small caliber chest tubes, which are temporary plastic tubes that can be placed into the injured vessel proximally, and then controlled with either a ligature around the vessel to lock that shunt place or a rubber vessel loop.

And then you want to make sure that you did not blow out any clot with that shunt. So you let some heartbeats go with red blood running through the shunt, then occlude it with the clamp, and then insert it to the vessel that is distal to the injury.

Likewise, you have to make sure that the thrombus has been removed, and I mentioned this earlier from that distal vessel. I think it is very important before you place the shunt in that you try to clear as much of the thrombus as you can, proximal and distal, and then place that shunt in and secure it either with a heavy silk tie or plastic vessel loops.

Most of those shunts you can listen with the Doppler and you will see arterial flow in it. So you can listen on the surface of the plastic shunt with a little water or acoustic gel and hear the arterial flow in those shunts.

**For our military docs out there, if they're deploying to a role and their supply sergeant calls them and asks what kind of shunts do you want us to have — what sizes and what types? What recommendations would you give to make sure that you have them on hand?**

Yeah, it's a great question for now and it's a challenge for us in the future. I think here and now, I would try to really make sure that the surgeon put his or her hands on Argyle shunts. The Argyle shunts come four in a container, there's a 12 French, 14 French, 10 French, and an 8 French all sort of in the

same container. And it's important to make sure he has those. As the larger 14 French Argyle can work on an iliac. So that would be one, and another would be to look for a 14 French chest tube. And I would say, "so these are my shunt options and I would need to have these available."

The other shunts, the Sundt and Javid shunts, are the other ones you could ask for, but then you only have one size for those. And so those and the thrombectomy catheters is what I would have. Because I think it's very difficult to do an effective arterial repair without the ability to do the thrombectomy, proximal and distal, because you will be repairing that segment into thrombosed vessel with thrombosed inflow and thrombosed outflow.

I think you have to get that vascular repair kit, if you will, assembled and communicated with your scrub team ahead of time, so as to include the specifics of each item: specific heparin saline dose, shunts to be used, SH needle and some of the smaller ones, and thrombectomy catheters. It is important to go through that series of tools that you may find yourself needing. Also, keep in mind, we don't use pulmonary artery catheters as much anymore, but they can be used as a thrombectomy catheter in a pinch. And then those thrombectomy catheters can be used as good proximal inflow control. You can insert a large thrombectomy catheter proximal and inflate the balloon with a three-way stopcock and that will afford you inflow control in a pinch as well.

**Say you're having a patient transferred in from the Role II to the higher level of care. Do you recommend these patients be heparinized, if able, when they have shunts whether arterial or venous**

If they can be, then yes. It is dependent on whether there are other injuries to the head, torso, extensive soft tissue, etc. If there is massive bleeding, then the recommendation is to not shunt or put these patients on heparin.

The best anticoagulant is flow. If you have high flow in those larger proximal vessels (above the knee or elbow), the shunts will stay patent without heparin (typically for 4-6 hours). The distal shunts, if they do clot off, what we have found is that they do not cause harm when they clot off. You basically are just back to square one with the occluded vessel and you have to do the thrombectomy, assessment, and repairs.

So even if the shunts do thrombose, our experience has been – at least in the short term of the temporary shunts – that in two, four, five, six hours they do not cause harm when they thrombose. You just have to do the thrombectomy and assessment at that time.

**To close out iliac artery injuries, if you did need to ligate either the common or external iliac artery due to severe injury, what kind of options do you have there?**

Well, that all really depends on the patient and their overall condition. If you have found yourself not being able to put a shunt in and in that scenario you had to ligate the external iliac, that patient is presumably in a really bad situation.

You can ligate it temporarily. So thinking that I am going to ligate it and then get some help. But in 2-3 hours, I am going to have to go back and try to re-establish inline flow with another set of hands, with more blood, a different surgeon, different lighting, different tools, because leaving it ligated will incur a tremendous amount of ischemia into that extremity.

There are the options of a cross femoral graft. I don't think I have ever done that in the setting of trauma, bringing a PTFE graft from the other groin, the other femoral artery over to the injured femoral artery. That would be extraordinary or unusual. But when the options are just life over limb, and you have to just say: "I can't repair it." The scenario, whatever the reason, does not allow me to establish inline flow and the patient's going to get an amputation and we are going to work on saving his or her life, and move on.

**As we move our way down the leg, I want to stop and focus on the common femoral and just discuss the principles of controlling the vessel — the options and if ligation is an option at this level for any of the branches? Also, in residency, we would talk about blast injuries and accessing the common femoral to get hemorrhage control. Is this a common maneuver performed in the combat trauma?**

The common femoral is not much less consequential than the iliac. In fact, it's probably every bit as consequential, as far as ligating it. So it can be ligated as a damage control maneuver, but the consequences will be significant if ligating the common femoral – just as significant as the external iliac.

There's three femoral arteries: the common, the deep femoral, and the superficial femoral. I think similar to what I mentioned before, all efforts should be maintained to try to maintain or establish flow through those three, if at all possible, and balancing the situation at hand, which sometimes means I have got to ligate it – because of the flow of casualties, because of the capability I have at a given level. Again, ligation if its life over limb is still an important damage control maneuver. You just have to be ready for the consequences of that and move on.

So controlling it, I think the Fogarty catheters are useful to control the deep femoral. Sometimes from the open common, you can insert a small Fogarty down into the orifice of the deep femoral and inflate that balloon and control back bleeding from the profunda. Often there are two profunda femoris arteries. They are difficult to control as they are right adjacent to the deep femoral vein. So, oftentimes controlling those deep femoral vessels from the inside using the small Fogarties and a three-way stopcock is quite handy. Then, the same tenets apply with either placing a shunt and trying to do a primary repair or some sort of interposition repair.

**The popliteal artery is an area that we frequently see injuries and they can be quite difficult to manage. Can you discuss just a little bit about how you best expose and set up the patient when you are trying to access the popliteal artery, both above and below the knee.**

Yeah, I will. And I skipped a question there. You asked about accessing the common for control. I think that's acceptable, I mean, it just depends on where the injury is and who is managing the case. I think if it's an individual who has done, 15 or 20 or 30 of these sorts of cases, and they feel comfortable about accessing the common femoral to control it – I don't think there's anything at all wrong with that. On the flip side, if it's a distal SFA (superficial femoral artery) injury or a popliteal artery injury, in those situations, most of the time it should not require just a virgin cut down on an uninjured common femoral.

Now for popliteal, I think it is really important to get in-flow control to the popliteal. One has to expose the above knee popliteal artery, which is really the distal segment of the superficial femoral artery as it comes through the adductor magnus or Hunter's canal.

You would start with a wide, long incision from above the knee to the mid thigh. It is important to make sure to have a small sort of bump with rolled towels that are placed underneath the calf, so that you prop the leg up in a "frog leg position" and let gravity pull a lot of the musculature of the leg down. Then, find the the distal superficial femoral which is proximal to the above knee popliteal, just at or beyond Hunter's canal.

And now move the bump above the knee. Then, make a separate medial incision below the knee, again using sort of gravity in a way to pull the gastrocs and soleus down, so that you can open up that below knee popliteal space – you are taking the muscles right down on the medial, inferior edge of the tibia to open up that below the knee popliteal space.

In this situation, you are going to need Weitlaner retractors. Oftentimes, we have something called a popliteal retractor, which has different depths of blades on the retractor. That's also referred to as a Henly popliteal retractor or Pilling retractor (must be the company that makes it). Those are awfully handy because you may need varying depths in blades, deeper than the standard Weitlaner retractor.

And then being able to really put a narrow handheld retractor, like an appendiceal or a Wylie renal vein retractor, that allows you to really retract. If you are above the knee and you are not that well exposed, you can put that narrow handheld on the inferior aspect of the wound and sort of toe in and really get down into the popliteal space itself.

If you are below the knee, you put that narrow handheld in the proximal extent and again, toe in and you really can try to get that popliteal space itself exposed. If you need to, you can join the above and below knee incisions – sometimes with the trauma, the injuries are joined anyway. Those are sort of some tenets.

Lastly, lighting. It can not be overstated how important lighting is, sometimes that's a luxury for those of you who are in a deployed setting. But these are the top line things to think about with popliteal artery exposure.

\*\*From one of my own clinical experiences, especially in young patients, with

repairing or bypassing the popliteal artery, many times these patients have very severe vasospasm at the end of the case. So that, even though you may do a beautiful bypass, you have minimal Doppler signals in the foot at the conclusion of the case. Have you experienced this and do you have any thoughts on this?

Yeah, for sure. If the bypass has sort of an above knee-to-below knee and you have excluded or ligated the injured popliteal artery, then absolutely, a lot of times the patient will be relatively cold and in a vasoconstrictive condition. As long as you feel like there's flow in the reverse vein bypass – ie: you can hear audible flow and it's not a water hammer signal, meaning that there's some diastolic flow in the arterial signal, and then there's some sort of weak signal at the ankle – most of the time, I will just be done, warm the patient up, and resuscitate them. If any of those things are not true, you don't have any arterial signal in the graft, or if you do and it's only a water hammer signal, or where there's no signal at all at the foot or beyond the graft – then, you have to open it up. Pass thrombectomy catheters, proximal and distal, and get that regional heparinized saline, and really make sure that there's not a real problem. But, that's really a judgment call and it is anxiety-provoking for sure.

But you have to have faith. I do think that if you feel like technically you saw all of your stitches, you felt it was good, you had some good outflow back bleeding before you did the distal, you didn't forget to the thrombectomy proximally, and you have signals – even if they are in spasm and you feel it's not as good as it should be – in some of those cases, you warm the patient up and resuscitate them and the signal will improve.

You can also do an on-the-table arteriogram, but that puts you in an austere location and can be a little bit of going down a rabbit hole. You will see the spasm and then say: "well, geez, now i need to intervene on the spasm." An on-table arteriogram is certainly an option and it should not be discounted, but I also would encourage folks that as long as those things are true – you saw your anastomosis, you thrombectomized proximally and distally, you feel good about your inflow/outflow, you have an arterial signal that has got some diastolic flow (not water hammer in the graft), and you have some signal beyond it – warm up the patient and see how they do.

**As we continue down the leg, we encounter our tibial vessels. How do you decide on repairing tibial vessels versus just ligating them?**

So there's a great paper I would refer you and your listeners to that was published in the Journal of Vascular Surgery in 2010 with Gabriel Burkhardt as the lead author.[Burkhardt et al., 2010] It was a review of the U.S. military experience with the practice of selective tibial artery repair. When you think about what that means, selective repair, it means we repair some but not all tibial arteries. So how do you determine which ones to repair? That was a good review article in the Journal of Vascular Surgery that I think looked at maybe a hundred tibial artery injuries. What we found from that experience was some principles of selective repair which led us fix those patients in which

all three tibial arteries were injured. This means that it was not just the posterior tibial artery, but because of the injury pattern, the peroneal and anterior tibial had also been severed and there was just no flow in the foot. So in those situations, we did repair them with a reverse saphenous vein graft. That's the exception. Majority of times, as you are alluding to and in that case series, we did not repair them because we could ligate the single tibial artery injury since there was redundant arterial flow to the foot. So you can ligate it, listen on the table, and if the other tibial vessels are patent then it can remain ligated and should remain ligated because tibial artery repairs are even more technically challenging and time-consuming.

**We are going to shift our focus to cover venous injuries. What are your thoughts? We have been talking a lot about the possibilities of ligating arterial injuries. How do you approach venous injuries and ligation?**

This would also fall under the descriptor of selective repair, which means you repair some but not all. Without going into too much detail, it depends. In almost all of the upper extremity veins, can and just should be ligated. There is not enough muscle mass and flow in the upper extremity veins to bother with repair. The exceptions might be a proximal axillary vein or subclavian vein. But, by and large, the distal axillary, brachial, basilic, cephalic veins can be ligated. In the lower extremity, similarly, the small tibial veins can and should be ligated – there should be no attempt to repair those. Coming proximally in the lower extremity, popliteal and then the femoral vein, if the patient is physiologically stable and performing a repair is possible or placing a venous shunt (we have had good luck venous shunts staying patent), then we have deferred to fixing the veins in those cases. If the patient is not in profound shock and if the expertise is there and present to do it with operating time, I think there is benefit to fixing those veins. There have been studies that confirm that this approach affords better arterial flow in the arterial repair because the venous outflow is not impeded.[Quan et al., 2008, Clouse et al., 2007, Rasmussen and Tai, 2022] Even if those veins thrombose over time, they do it slowly and do not lead to pulmonary emboli. So, when we can, we will fix those veins in the popliteal and femoral segments. Conversely, if the capability does not exist, the surgeon is not accustomed to repair, the patient is not doing well physiologically, or there's the need for the operating table – then you just have to ligate those. I think ligating those veins in the popliteal or femoral segment are also acceptable as a damage control maneuver.

**We could not finish a peripheral vascular trauma talk without discussing fasciotomies. What are your advice for listeners regarding fasciotomies?**

In the military setting, we have been liberal about the performance of two incision-four compartment-lower extremity fasciotomy. I think they should be done more liberally in our setting in the military. We advocate for them and have shown that they can be done without substantial additional morbidity and

mortality. In fact, waiting to do a fasciotomy or missing a fasciotomy has been shown to be associated with mortality from early data from the wars. So part of that is in our system, we often are going to lose track of these patients. We will see them at a Role II, and then we would like to think, they don't need the fasciotomy at the Role II because they will do it at the Role III. And then we are not going to do it the Role III, because they can do it in the Role IV. But again, we are going to be evacuating these patients at altitude and they are going to go out of our care. So I err on the side of prophylactic, two incision-four compartment fasciotomy in the setting of lower extremity vascular injury certainly with wartime vascular injuries.

## 10.2 Abdominal Arterial

**22 Dec 2019:** *Kevin Kniery, MD, MPH; Adham Elmously, MD; Todd Rasmussen, MD*

We are going to skip the preoperative workup and description of anatomy and zones of abdominal vascular trauma, so please review this on your own. As a side note, we are also going to skip the management of iliac artery injuries as we covered that as part of our peripheral vascular trauma session.

**Scenario:** “You have a patient with a gunshot wound to the abdomen. General surgery has already prepped them from the neck to the knees, and performed a laparotomy from the xiphoid to the pubis. They packed the abdomen and temporarily have control. There was note of pulsatile bleeding from Zone I. Can you take us through how to get supraceliac control?

A case like this should not proceed without close communication of the entire resuscitation team. I would start by making sure that I was on good terms with my anesthesia colleagues. So, they know what we were facing in this situation and that they were pursuing all the key tenets of damage-control resuscitation — leading with blood product or whole blood resuscitation, keeping the patient warm, etc. As far as from a technical standpoint, you can't sew what you can't see. So, I immediately assess my exposure — which is the laparotomy. The laparotomy in this case was from the xiphoid to pubis. That's a great start, but for the approach you have referenced, we will need to come up higher and go between the costal margin and almost begin the first 2 to 3 centimeters of a sternotomy. It is not a sternotomy, but, it comes along the side of the xiphoid to release the inferior costal margins of the thoracic area under the diaphragm to pull the costal margins laterally and up. You want to suspend your costal margins so that you are almost suspending the patient's rib cage up and away from the aorta and the abdominal contents. So making a good assessment of your exposure initially is really important. In a case that I am prepping and operating on, I try my best to use a retractor. Such as the Omni, that will allow

for retractors to be placed underneath the costal margin so one can spread those costal margins and try to lift the thoracic area away from the abdominal contents. If you do not have an Omni retractor, you can still make sure that you are suspending the superior aspect of your exposure.

Once that is done, the other part of communicating with anesthesia is to see if they have a nasogastric tube down. In this case, supraceliac control is almost always facilitated by having a nasogastric tube in the esophagus to allow one to open the esophagus here and be able to get around that esophagus circumferentially — with either umbilical tape or penrose drain. This will allow for appropriate left-sided retraction of the esophagus, away from the crus that is surrounding the aorta.

The next maneuver is mobilizing the left lateral segment of the liver, so that it can either be brought cephalad or tucked inferiorly and brought to the right side (or the whole portion of the liver). So now, you have the left lateral segment out of the way. At this point, one is left with the crus that is overlying the aorta.

The other maneuver here, dependent upon the situation, is to put the patient in reverse Trendelenburg to let the stomach down and pull it inferiorly so that you create as big a window as you can — with the esophagus retracted to the patient's left the stomach pulled down. Sometimes, you can have a resident or a an assistant put their hand on the stomach and retract it towards the pelvis.

Now, you have a window in which you can work. Next is to divide the skeletal muscle, which is the crus overlying the aorta. This can be facilitated with a large right angle and a bovie extender, and hopefully you can feel the aorta with a pulse (even a weak pulse) and divide the crus over the aorta with blunt dissection on either side. Typically, you do not try to spend time to get circumferential control of the aorta. It is not like an elective aneurysm repair where you want to get around the entire aorta. In these cases, if you can get on either side of the aorta down fine, that is what is needed and is most efficient in these emergencies.

**Scenario:** You enter a patient's abdomen and there is a large rush of blood. You think it may be coming from somewhere in the upper aorta. Is it worth taking the time to get supraceliac control before doing a medial-visceral rotation and exposing the aorta in that way? Or are there any scenarios in which you do a medial-visceral rotation first before you get supraceliac control? Can you take us through a medial-visceral rotation and pitfalls/advantages of doing each of those approaches, ie when to leave the kidney down and when to take it up?

It is worth getting super celiac control if it is a Zone I hematoma, especially if you have some time with someone holding pressure. Now, if you initially believe that the bleeding is from Zone I, but when you are actually able to enter the abdomen it looks like the bleeding is coming from the spleen or a different area, then you can readjust. The left medial-visceral rotation is designed to give

optimal exposure of the para-visceral segment of the aorta. The para-visceral segment of the aorta includes the supraceliac segment all the way down to the aortic bifurcation, primarily the left common iliac artery. In this case with the left medial-visceral rotation, it is performed through dividing the white line of Toldt along the sigmoid colon and the left colon. You want to establish this plane in the retroperitoneum, which a good portion is achieved through steady blunt dissection facilitated with Bovie electrocautery. In the setting of trauma, this dissection plane is taken from the sigmoid/left colon up to divide the attachments ingrained in the spleen and the left kidney, then to the midline and then ultimately to the patient's right. In the setting of trauma, the left kidney is almost always brought up. It is generally easier and faster, because you do not have to take the time to find the plane above the left kidney.

Check out this video for an example of a left medial visceral rotation

#### **Management of bleeding from the spleen**

One of the key tenets of splenic injury is exposure — good retraction with incision up to xyphoid and alongside the left costal margin. Compromised exposure is a contributor to splenic injury.

**With regard to exposure and eviscerating the bowel, there are some nuances. A method includes finding a moist lap pad that is large enough to cover the bowel, then place a clamp around it with someone holding it outside the abdomen. However, this method may not be as secure and also result in the bowel still being in the way. Do you have any tips on how to eviscerate and control the small bowel?**

This is difficult to describe, because sometimes you just have to feel it. However, this is the main purview and job of the surgeon that is on the patient's right. It takes a strong and big right hand, extending as wide as possible, to get the bowel over to the patient's right side — pulling it from the left to the right. It is not a subtle maneuver. Also, I do not use lap pads. I use an operative towel and ask to moisten the blue towel. You take the blue towel in your left hand and put it underneath your right hand (which has the viscera) to try to get it down to the base and laid out — the blue towel and your right fingers are essentially coming across the aorta passed midline. Then, my preference is to use the Omni retractor to take the place of your right hand, which will come on top of that blue towel and hold all the viscera to the right of patient's midline.

**With the left medial visceral rotation (Mattox maneuver), we perform the maneuver because of a suspected aortic injury or a close branch off the aorta. However, sometimes we need to do a right medial visceral rotation (Cattell-Braasch maneuver), especially when concerned about an IVC injury. What are some of the nuances of a right medial visceral rotation?**

As you are on the other side, it still requires dividing the white line of Toldt starting at the cecum and coming along the right colon. You bring the cecum

and right colon cephalad and then to the patient's left. It is actually not so much of a rotation to the midline and then the left, but bringing everything up cephalad and then left to the patient's left costal margin — almost bringing the cecum and right colon up onto the patient's left chest. This is a plane that is also similar to the medial visceral rotation on the left side as it is largely developed with blunt dissection. However, it is a combination with opening the retroperitoneum with some Bovie electrocautery. You will also almost immediately see the left iliac vein and then the vena cava. Then, you continue to bring the left colon up until you see the right renal vein and kidney. At the superior extent, this will expose the head of the pancreas and duodenum. And similarly, you use a blue operative towel to cover and bring up the viscera to be held with the large retractors so your hands are free to operate. Spine exposures with orthopedic and neurosurgical colleagues and transplant procurements are also a great opportunity to learn the tenets of rotating the visera either right or left and exposing the retroperitoneum.

Check out this video for an example of a right medial visceral rotation.

#### **The technique for exposure of the infrarenal aorta**

The exposure of the infrarenal aorta is also called the transperitoneal inframesocolic exposure and is considered to be a straightforward maneuver. Inframesocolic means that the transverse colon is reflected cephalad. One of the first aspects of this maneuver is to hold up the transverse colon and drape it up cephalad to patient's costal margin (can be held up with a blue towel and retractor). Next, you divide the ligament of Treitz (by the surgeon on the patient's right) and get down to the fourth portion of the duodenum which should be to the left of patient's midline. Once divided, the left colon remains on the patient's left and everything else goes to patient's right. However, this is not a medial visceral rotation because only the fourth portion of the duodenum and all of the small bowel is partitioned to patient's right. At this point, one should see the infrarenal aorta. The left renal vein is the most cephalad extent and the aortic bifurcation is the most caudal extent (located at root of colon mesentery). The left renal vein can be ligated if necessary to facilitate aortic control. However, it is best to try to preserve the branches of the left renal vein that drain the left kidney (gonadal and lumbar) so that dividing the left renal vein is not as much of a detriment to the kidney.

#### **Tips for getting umbilical tape or vessel loop around aorta, especially in supraceliac region.**

You can achieve this with a combination of Metzenbaum scissors and controlled pressure. You are trying to free the aorta from the connective tissue around it to get back to the spine. You can use a metallic pediatric yankaeur (small profile and blunt tip) as a dissection tool and push with the Metzenbaum scissors along with direct feel to get the aorta freed. Then, you can use a right angle to get underneath the aorta. You also want to make sure to identify the lumbar vessels — "find the outside before you find the inside" — so you can clip them with

medium/large Weck clips.

**Techniques of exposing and repairing branches off of aorta: Celiac Artery**

The origin of the celiac artery can be seen with left medial visceral rotation as it will show the entire paravisceral segment of the aorta and also with an anterior approach through the lesser sac (very similar to exposure needed for the supraceliac aortic exposure). The stomach is retracted caudally towards the pelvis, lesser sac is opened, and then the stomach is pulled down further. The pancreas is pulled down as well and the origin of the first centimeter of the celiac artery is now exposed. For injuries to the proximal portions of the celiac, if able to be repaired primarily — use 4-0 or 5-0 prolene sutures. If injury is large and there is destruction of vessel wall, then primary repair is not an option and this becomes a damage-control situation to control the bleeding. With damage control, ligation is even considered as an initial step to save a patient's life.

**Techniques of exposing and repairing branches off of aorta: Common Hepatic Artery**

The common hepatic artery is best visualized through that similar transperitoneal anterior approach for exposure of the celiac artery through the lesser sac. Prior to identifying the common hepatic, you have to find the celiac and follow the branches. Patient should be in reverse Trendelenberg, the stomach inferior, and costal margins flared up to have the appropriate space. This will expose the second and third portions of the celiac artery, and its major branches — including the common hepatic. The common hepatic artery can not be visualized using left medial visceral rotation. Primary repair is ideal, however, in damage control surgery ligation may be the only choice. If possible, ligation of the common hepatic proximal to the GDA is preferable as there will be retrograde flow from the GDA into the proper hepatic to maintain the viability of the duodenum, pancreas, and liver.

**Techniques of exposing and repairing branches off of aorta: Gastro-duodenal Artery (GDA)**

The gastroduodenal artery is the first branch off of the common hepatic artery and defines the junction between the common and proper hepatic arteries. Primary repair should be sought if possible. If it is a damage control situation, then the GDA must be ligated to control hemorrhage. If ligated, assess patient's condition, resuscitate, utilize the doppler, and assess the area distal to ligation to evaluate the consequences. For example, if ligation showed appropriate collaterals, then the vessel can remain ligated. However, if there is clear ischemia, then there is a difficult decision between repair vs. shunt vs. permanent ligation along with its metabolic consequences. The decision is dependent on each situation. The principles of damage control resuscitation and damage control surgery should always be maintained.

**Techniques of exposing and repairing branches off of aorta: Superior**

**Mesenteric Artery (SMA)**

The SMA is divided into segments based on its course, from aorta to pancreas to root of mesentery to the small bowel. Approach to SMA visualization is dependent on the segment that needs to be exposed. The first segments of the SMA can be seen through left medial visceral rotation. The distal segments of the SMA can be seen through the anterior transperitoneal approach. Since the SMA (second segment) is near the head of the pancreas, if the proximal segment of the SMA is injured, then the head of the pancreas may need to be divided to get appropriate exposure. If the segment of the SMA that is injured is distal to the pancreas, then it can be visualized through the inframesocolic approach. With regards to ligation, distal SMA segments will be more tolerant of ligation. All efforts should be made to maintain flow through the SMA. Primary repair can be performed on small injuries with 4-0 or 5-0 prolene suturing. Larger injuries can be repaired with patch angioplasty or interposition graft (PTFE, Dacron, or autologous tissue such as proximal greater saphenous vein or deep femoral vein). Autologous vein is preferred in contaminated field, but can also use rifampin-soaked Dacron. Tunneling is dependent on location, proximal segments would need 6-8 mm of graft while distal segments would need 3-4 mm. It is important to remember that a majority of these axial injuries are typically unsurvivable and proximal SMA repair is uncommon in the trauma setting. Even if the SMA is repaired and the bowel appears viable, it is preferred to delay abdominal closure and perform a second look 24-36 hours later or sooner depending on the patient's condition.

**Techniques of exposing and repairing branches off of aorta: Inferior Mesenteric Artery (IMA)**

The majority of times, the IMA can be ligated. Prior to ligation, assess the patency of the iliac vessels — specifically the internal iliac vessels. Also, if both celiac and SMA are patent, then the IMA ligation can be tolerated. If there is concern for atherosclerotic disease or occlusion in the internal iliacs, then ligation may not be the preferred option and re-implantation may be considered.

**Techniques of exposing and repairing branches off of aorta: Renal Arteries**

It is important to consider the broad principles and approach: 1. Urgency of situation 2. Optimal Exposure 3. Resuscitation Efforts 4. Communication with Team 5. Appropriate Equipment

Decision to maintain patency of a vessel vs. ligation is dependent upon the situation. During damage-control procedures where bleeding is not controlled and location of hemorrhage not known, the inclination is to ligate. The risk is organ ischemia, but the benefit would be patient survival.

The renal artery is similar to the SMA, unlikely to be reconstructed in a trauma setting. In renal trauma, the renal artery is either not injured (with only injury to renal parenchyma) or renal artery is injured and thrombosed. If renal artery

is injured and thrombosed, salvageability is unlikely due to prolonged warm ischemic time of the kidney. Repair of the renal artery would truly occur if there is an expanding hematoma in Zone II that warranted exploration and it was found that the source of the hemorrhage was from an injured renal artery that still has flow. In this case, again, would do primary repair with smaller injuries with a 4-0, 5-0, or even 6-0 monofilament prolene suture. If a primary repair would compromise the lumen, then consider patch angioplasty. However, most commonly, if renal artery is injured it is ligated for damage-control. With regard to incidental CT findings, an intimal flap, fistula, and/or possible pseudoaneurysm of the renal artery, the injury can be observed if there is continued renal perfusion and no flow limitation. If it is a flow limiting injury with more than 50% stenosis caused by the intimal flap, it may be amenable to endovascular placement of bare metal stent to tack down the flap. Distal branch artery injuries can also be managed with endovascular coil embolization. Again, the decision to take the time to coil embolize is dependent on the patient's current hemodynamics. Most commonly, if there is an expanding hematoma in Zone II and the renal artery/parenchyma is damaged and confirmed as source of hemorrhage, a nephrectomy is warranted and there is no indication for repair.

#### **General Guidelines of Penetrating vs. Blunt Retroperitoneal Exploration**

Zone I hematomas: explore whether penetrating or blunt mechanism.

Zone II hematomas: always explore if penetrating, do not explore during blunt unless expanding. Kidneys are in Zone II and are in the lateral gutters.

Zone III hematomas: always explore if penetrating, do not explore during blunt unless expanding.

### **10.3 Abdominal Venous**

**18 Jan 2020:** *Kevin Kniery, MD, MPH; Adham Elmously, MD; Todd Rasmussen, MD*

**Kevin:** so for the sake of time we're going to skip the preoperative workup and description of anatomy and zones of abdominal vascular trauma So please review this kind of on your own Um and just as a side note we're also going to skip the management of iliac artery injuries We covered that as part of our peripheral vascular trauma talk It can fit into abdominal vascular trauma but for the sake of time we're going to just jump into a true abdominal vascular trauma So we're gonna start out with a little scenario just to kind of get our feet wet hereso doctor asks me it's in a you have a patient with a gunshot wound to the abdomen A general surgery has already prepped them from the neck to the knees and performed a laparotomy from the xiphoid to the pubis They pack the abdomen and temporarily have control on what they said was pulsatile bleeding from zone one can you take us through how to get a super celiac control And

in a case like this and in a trauma

**Rasmussen:** Sureso I think you know I think for cases like this [00:02:00] just a couple of starter pointsyou know no case like this Should proceed without you know close communication with the entirety of the resuscitation team So I think if I was coming into a case like this or participated in a case like this with you know with our team I would start by making surethat I was on good terms with my anesthesia colleagues that they knew what we were um facing in this in this situation that they were pursuing all the key tenants of damage control resuscitation and and and all that That means meaning you know leading with a blood product or whole blood resuscitation keeping the patient warm and and all of those tenants So I would start with thatand I think as far as technicallythe from a technical standpointin these types of cases I will almost always you can't so what you can't see and I'll I'll often really immediately assess my exposure I mean meaning the laparotomy [00:03:00] So you mentioned thatLaparotomy in this case was from the to the pubis And and and that's a great start Butbut for the approach that you have referenced is likely to be needed in this case um I'll often reset and actually come up a higher go between the and the costal margin and almost begin you know two to three centimeters of Of a of a sternotomy It's not a sternotomy but it is at least coming along the SciFest side of the xiphoid and really release thoracic the inferior costal margins of the thoracic under the diaphragm so that she can really pull the diaphragm I mean sorry the cost of margins laterally and up so that you want to suspend your costal margins so that you're almost suspending the patient's rib cage up in a way from the aorta and the abdominal contents So I think making a good assessment of your exposure initially really important Hopefullyyou know [00:04:00] in in the case that I am prepping and in an operate on these cases I'll try as best I can to use a retractor Such as thethat will allow for retractors to really be placed up underneath the costal margin So again you can spread spread those costs origins and try to lift the thoracic almost lift the cost of margins away from the abdominal contents If if you don't have an Omni you can still make sure that you're trying to OSE and suspend thatum superior most aspect or cuddle Or cranium aspect of yourof your exposure Once that's done And the other part of communicating with anesthesia is to see if they have a nasal gastric tube downsuper celiaccontrol of or in this case is almost always facilitated byhaving a nasal gastric tube in the esophagus to allow one to two You know to pop esophagus um in this area and and then be able to get around that esophagus circumferentially and sometimes place some sort of [00:05:00] umbilical tape or sometimes a Penrose drain to allow for apt left sided Retraction of the esophagus away from the the a cruise that's surrounding the aorta The other areas is really mobilizing the left lateral segment of the liverso that that can either be brought cephalad or tucked you know once it's mobilized can be Tucked in in area early and brought to the right side or the whole portion of the liver so that you've got the left lateral segment out of the way And now at this point you know one is left with the cruise that's overlying the aorta The other maneuver here sometimes dependent upon the situation is is put into patient a little bit of reverse Trendelenburg to

let the stomach and pull that stomach the inferiorally so that you create as big a window as you can Oh with the esophagus retracted to the patient's left the stomach kind of pulled down Sometimes you can have a resident or a an assistant put their hand on the [00:06:00] stomach and retract it towards the pelvis if you will Sometimes a little bit of reverse T-Bird helps with that and you can kind of hopefully picture this So now you've got the separated you've really The cost of margin suspended and now you've got a window in which you can work And and at this point now it's maneuvers dividing the skeletal muscle which is the cruise overline the aorta This can often facilitated with a large right ankleyou know in a Bovie extenderand you know in this case you know you can almost always see that you're dividing skeletal muscle over the aorta if there's a pulse hopefully there's even a weak pulse you know one can feel the aorta and just divide the cruise over the aorta and and try to do some blood dissection on either side to allow your patient of the spineOn either side you know in these cases I typically don't try to spend time getting circumferential control of the aorta It's not like an elective aneurysm control So or elect bang chrism [00:07:00] repair where we'll sometimes get round the entire order and these cases if you can get on their side of the aorta down fineyou know that that is is what is needed and and is most efficient in these emergent sort of cases So up there if you have other questions I mean it's a it's a unexposed which requires you know a comprehensive set of of things to think about But I'll I'll

stop there for now

Do you have any

questions or comments

**Adham:** Somy question is sometimes you're faced with a situation of entering a patient sad them and obviously there's a large rush of blood and you think it may be coming from somewhere in the upper aorta Do you think it's worth taking the time to get super Celia and control in the in the way that you described before doing a video this rotation and exposing the order that way Or are there any scenarios in which you should do a medial visceral rotation first before you do that So take us through can you take us through a [00:08:00] medial visceral rotation When did she to want to leave the kidney down when to take it off sort of the falls and advantages of doing each of those approaches

**Rasmussen:** Yeah I think so That's a great question I think It is worth getting super celiac control If in the scenario that you described where where you you know the description is a zone one a hematoma that's bleedingandyou know you you've got some a little bit of time you know somebody may be holding pressure or but I do think it's worth getting super celiac control if you think it's from the zone one Now if it's you know the initial description was on one and then you get in there and you look and it's actually it looks to be from the spleen or or from a different area then then you can readjust But if it's truly a a a zone one hematoma that's got active bleeding then I to answer your question I do think it's worth taking the time to get super celiac controlthe medial visceral rotation

of courses [00:09:00] is designed to to to to give upyou know optimal exposure to the left medial visceral rotation is is designed to give optimal exposure of of the para visceral segment of the aorta Sothat is you know the the super celiac segment all the way down to You know to the aortic bifurcation and and and really the left primarily the left iliac common iliac artery initially although it can be taken across the ORIC bifurcation to see the right iliac as well in this case that that is performed through dividing the white line of told along the sigmoid sigmoid colon and the left colonand you know establishing this plane and the retroperitoneumand and a good portion of this is is steadyyou know blunt dissection it's facilitated with Bovie electrocautery But it it is also um you know there's a plane in that in this area that is developed with with blood [00:10:00] to section as well And in the setting of traumaalmost always this this detection plane is taken from the sigmoid the left colon up to divide the the attachments and grained the spleen and and the left kidney all the way to the to the midline and then to the patient's right ultimatelyand I think in in the setting of trauma the kidney is Almost always brought up into this and and just bring the kidney up I think it's generally easier It's a little bit faster because you don't have to take the time to to find that plane you know above the left kidney and to leave it down in the setting of trauma Um I would almost always bring that left kidney up

**Kevin:** Okay

**Rasmussen:** if that's yeah

**Kevin:** Yeah I think that's perfect one question I have is it seems like the spleen is always getting in the way and always bleeding at the end of one of these rotations Do you have any tips for managing bleeding from the spleen

**Rasmussen:** Well I think thisgoes back to I think my initial comments and and probably one of the key tenants of of this type of a procedure is exposure and making sure that if you're going to undertake this that you've really Gotcha Retractor you've got your incision up along the alongside not just to the XY FOI but along side the XY Floyd and the cost of margin so that you can really bring that the left cost of margin both of them and suspend it up Because I think and in many cases what you described is a result of just not being able to see welland soyou know I think the the best I would the best tip I could give is is to really try to make sure that that your exposure is as best you can and if you're compromising on exposure um you knowwhich which is easier said than done I I understand that But if you feel like you're compromising on exposure or retraction that is that that's I think a contributor

to to

to splenic

injury

**Adham:** I have a question Speaking of exposure specifically about eviscerating the battle I find myself as the as the senior residents sometimes appreciating

actually having difficult and nuances to do this The way that I've learned to do it is kind of taking all the bowel and finding a lap pad large [00:11:00] enough that's moist I'm placing some sort of clamp or something around it and then having someone hold it outside the abdomen but I've always found that it sort of ends up being clunky or the bowel falls out of the time Do you have any tips on how to eviscerate and control the small balance situation like this

**Rasmussen:** That's a great question And it is it's it is very difficult to describe this using in a discussion It's one of those things where you just got to feel it But in my experience this is the and I hope those who are listening and you can kind of picture yourself in the situation This is really the the main Purview and job of the surgeon that's on the patients right So you can picture in a you're on the patient's rights and and it takes a it takes ait takes a strong and and I think big I don't necessarily mean physical but it takes a strong and a big right hand to get that bowelthatyou know Often and over [00:12:00] and to the patient's right side you're you know you're pulling it from the left to the right And and I you know I'm sitting here to describing it and my hand is I'm trying to extend my hand as wide as I can you know and and it's it's not a subtle move It's notit it shouldn't be a rugged but it needs to be very it can't be done with a gentle it's Until a type of thing you've gotta be strong with it And I don't use in this situation I'll get operative towels I'll ask them to moisten blue towel or wet blue towel And you know basically what you do is with you know with your left hand as you take that blue towel and you put it underneath your right hand which has the viscera in it and you know you sort of You tried to get it down at the base so that you really that blue towel here and your right fingers are coming across the aorta you knowcause you're taking it from the you know from the patient's left to the patients to the midline and then [00:13:00] try to get it over to the right and then put this big blue towel this wide blue towelyou know underneath your fingers and then you know lay it out and and then hopefully I mean this is where I I actually like in my situation or in my preferences to use the Omni but you're going to need big a wide you know retractor that's going to take the place of your right hand That's going to now come in on the top of that blue towel And and hold all of that and maybe a couple that hold that visceral you know to the patient to the right of

the patient's Midland

**Adham:** That's one of those maneuvers that I feel like is very much easier said than them and I've been

humbled by it

**Rasmussen:** it's very difficult to describe

**Adham:** I want to just ask a follow up question about the middle of the medial visceral rotation that I asked her My question just to clarify question is is there ever a situation in which you're faced with a bleeding error where because of the time it takes to get super sealing I control that you would rather do a visceral rotation and be controlled being on it that way Or is your approach

always Basically without fail too if you have a high aortic injury to get your con control and then immediately rotate the viscera and expose the order that way Secondly

**Rasmussen:** Well I think there's great question I mean I think that if you there there are scenarios where if you if you If you feel like you know you see the bleeding let's say you know it's zone one and all of a sudden you you know you're getting ready to do this and you see that it is a spurter from the SMA you know a superior mesenteric artery and it doesn't look like it's the aorta for example And because there's some structures of course in zone one that aren't I mean obviously they're not some are not the aorta So I think that the scenario that you would forego super celiac control would be a scenario in which you you made an observation that you felt like you saw the bleeding It wasn't necessarily from the aorta and you felt like you could kind of get it controlled Locally you know without having to occlude a inflow from the aorta So but that if it's unknown you know and you have the you had just you don't know where it's coming from in zone one I think short of that I would advocate for trying to get a super celiac control

**Kevin:** Great so when we talked to the left medial this rotation for our kind of med students out there we're we're thinking that there's likely an aortic injury or a a branch close off the aorta Um sometimes you'll need to do a right medial visceral rotation though and that's generally when you're kind of worried about either generally a [00:14:00] IVC injury so Dr said can you just take us through a few of the nuances of a right medial Chris rotation

**Rasmussen:** Right So this is you know this from obviously from the other side and this involves dividing the the White line of toll starting often at the and the coming along the right colon and bringing they you know the Sikh cephalad and and and the entire right colon you know to the patient's left now and then cephalad it's I'm surprised most of the time in these cases it's actually Not so much a rotation to the mid line and then the left as it is bringing everything up cephalad to almost to the patient's left costal margin for example They really are almost bringing the CQM and the and the right colon You know to the up onto the patient's left chest in a way and and this is a plane that also similar to the medial visceral rotation on the left [00:15:00] side This is a plane that is largely developed with blunt dissection It's a combination Once the The parent Niamh retroperitoneum has been opened with some Bovie electrocautery than the plane should develop And in this case you'll see almost immediately the the left iliac vein and then the vena cava And and you continue to bring the the left colon up until you see the the left renal vein or I'm sorry the right renal vein and and the kidney And And and you know it did it's superior extent This will of course expose the head of the pancreas the duodenum and the head of the pancreas And similarly um You know this the visitor in this situation I get a a wet blue towel or operating towel I guess it can be white towel but I don't really like to use a lap sponge cause it's too small and it doesn't quite have enough substance And you will one will you know place that

that [00:16:00] Blue operative towel down along your fingers that are bringing this this all of the vis are up so that you really get it all partitioned away with with a substantive operative towland that that has to be then you know cold in with a large retractors so that your hands are free to actually operate

**Kevin:** Perfect one thing I just want to remind some of our residents in medicines out there is I I would really go onto YouTube after this and just look up left and right middle vis rotation that'll really help solidify these points It's one thing hearing it but I think if you watch some videos on it I think it'll really help bring it all togetherand if you also want to  
hear

**Rasmussen:** Yeah I think I was going to say the same or to obviously have an Atlas but are you know something because it's it is you got to kind of combine I think the word that we're talking about here with with some sort of visual aid the end the thing that I would recommend and make a point is for those who have the opportunity to assist with spine exposures So in these maneuvers that [00:17:00] we often perform as assisting our orthopedic or neuro surgical colleagues to expose do anterior exposure to the spine many of these maneuvers we're talking about are performed in the spine exposure which which sometimes can be overlooked by trainees And and even They sometimes is well that's not a very good operation You don't really get to so and and it's not really a basket operation Boy I'll tell ya Um high exposures are a great way to begin to nerve or learn the tenants of of rotating the visceral either to the right to the left and exposing the retroperitoneum

**Adham:** I think alsoprocurements have been extremely helpful for liver and kidneythis right sided rotation Is it usually the first step of any procurement And then you're kind of seeing the entire intro part IOM So it's also a very good opportunity to learn some anatomy

**Kevin:** Absolutely And

**Rasmussen:** Great point

**Adham:**we haven't discussed the infer renal aorta yet [00:18:00] I think probably a slightly more straightforward approach but can you discuss your technique for inferiority exposure and then talk about I'm kind of coming across the renal vein when you can like gate it how to get it out of the way things like that

**Rasmussen:** Right So the the most You know the most common and maybe most straightforward exposure is what we refer to as a you know transperitoneal and from he's a colleagueexposure of thethe infer renal aorta you know this here again people who are listening and looking either at a videos or oror an Atlas kind of recommend or recognize that the infer music colleaguemeans that the transverse colon isreflected cephalad andit's one of the first moves is to really hold up the transverse colon um in the patient's abdomen and and drape it up You know cephalad almost on the patient's costal margin And often you [00:19:00] know I'll set initially set a retractor Now that also goes into a big to

me is is partitioned with a operative towel a moistened or wet operative towel to get the transverse colon um and retracted cephalad and then the next move is to divide the ligament of Treitz and this to me is always also a maneuver that's led by the surgeon on the patient's right Um whether that's the primary surgeon and the the attending or resident however you're configured this this maneuver dividing the ligament of Treitz and and getting the you know fourth portion of the denim which is to the left of the midline in a dividing that ligament of Treitz that peritoneum and and and bringing that over to the patient's right is is the next move Um and and that begins this Process of you know getting really the the bowel Nowin this case the the left colon is left remains to the patient's left [00:20:00] but you're bringing all all bowel to the patient's right this this is not a medial visceral rotation of of the entirety of the viscera This is only bringing the duodenum the fourth portion of the Dudina and all of the small bowel Partitioning it to the patients Right And and this also again if one can visualize this be done by the patient the surgeon to the patient's right and the right side of the patient bringing that small bowel all the way across This also takes a wide and broad and strong hand This can't be done with I don't know how else to say it It's just a broad and strong sort of maneuver with with the hand to bring this across and then and then partition get another wet blue towel I keep saying blue I guess it could be white but operative towel and Yeah Put that towel over your fingers And you know as you bring the small bowel over that's the infer renal aortayou know this [00:21:00] positioning Then retractors one will you know at the most cephalad extent of that exposure begin to see the left renal vein at the root of the of the colon mesenteryand you know at the at the caudal most portion of this you'll see the aortic bifurcation Um andyou know that's that's sort of the extent of this Thethe the left renal vein Mmm Hmm Can be like if if it if it facilitates aorta control Um obviously if one is going to locate the left renal vein it's it's optimal to leave the branches of the left renal vein So the gonadal eh the the lumbar vein which also often will drain the left kidney and then the the gonadal So if those are left intact dividing that left renal vein can usually be accomplished without causing a significant detriment to the left kidneyI will say that dividing the left renal vein is easier said than [00:22:00] done I mean it's it is you know it's a thin wall structure which of course can bleed significantly itself So you know these are all Sort of decisions that have to be made depending upon what what Andrew you're dealing with and and the extent of your exposure

**Adham:**I think one question I forgot to ask you earlier was I think Infor renally they are it is a little bit easier to get around completely input you know an umbilical tamper a vessel loop around But like you mentioned earlier Super celiac area That's a lot harder to do when sometimes the goal is not to always do that but you have tips on how to do that Because again I'd been humbled by that I'm in a procurement one time when the attending allowed me to try and get around it and I tried to bluntly with my fingers to get around the posterior Huerta and had a lot of trouble So do you have any tips about that

**Rasmussen:** Well I think this isI mean this is just takes um you know experi-

ence and and and And and and time I mean I in my own mind as I see this I I you know [00:23:00] this is a combination of sort of a little bit of Metzenbaum scissors a little bit of kind of pushing you know cause what you're trying to do is to to to free the aorta from from the connective tissue around it to get back to the spine You know sometimes actually almost always I'll use a metallic Pediatric yang cower That is sort of one of my the suction or dissection tools of choice is is pediatric Yankauer which is small and profile It's got a blunt tip It will you know it is a suction device And so that's usefuland you know kind of this combination of pushing a little bit of maths here and there Metzenbaum scissorsand and a little bit of feeling with your finger so you can get that connective tissue aorta freed and then you know you can begin one can begin to you know use a right angle a big right angle and and try to to you know to get underneath it Obviously what you're looking for here in [00:24:00] part is a Is lumbar you know you're looking for the lumbar vessels which you know those are the things you need to leave behind for at least by as we tell our residents you know find the outside before you find the inside you can find them outside before you find the inside of a lumbar Then you can clip ityou know another I think very useful tool in this situation is is having large Weck cliffs you know because you're not it's very difficult to pass you know a silk to lie gate a lumbar And in these situations a lot of times you know you need those large WEC medium to large Weck clips to help you know get on either side of a of a lumbar artery or vein

**Kevin:** Great one quick point back to their renal vein I kind of picked this up just as fast past week is you can really get a lot of mobilization on the renal vein just by ligating some of the smaller branchesout distantly on it not even the gonadal but sometimes the adrenal vein and a few of those veins And you can really get you know five centimeters a [00:25:00] mobilization of that renal vein which should give you enough distance to clamp Um But now we're going to go intoeach vessel off the aorta I know this isn't exactly what you're going to see in a trauma You don't have isolated celiac artery injury or isolated SMA injury but just for the sake of learning we're going to talk through each vessel and talk about the options of exposing it and then repairing it So starting approximately a Dr. Rasmussencan you discuss what the best ways to see the celiac artery are andany options for repairing it

**Rasmussen:** Yeah So the you know the celiac arteryI guess the origin of the celiac arterycan be seen either through the lesser SAC this is again through you know an an sort of a I guess an anterior approach through the stackand this is would likely be very similar to the exposure we talked about with a super celiac aortic exposure where the cause retracted you know caudally down towards the pelvis[00:26:00] and um You know that opening lesser sack and pulling the stomach down and and sort of getting the pancreas to come down That will expose then the the origin of the first centimeter to the celiac artery If the proximal segment of the celiac artery is injured and bleeding then then the repair optionsyou know I I think lend into the category of any vascular know major vascular three If it can be prepared repaired primarily then

you know using

four Oh five Oh proline future If it does a grazing wound and repairing it primarily can be done then then that's that's one option if it's a largeyou know celiac artery injury that that cannot be repaired primarily and there's destruction of the wall Oftentimes you're an individuals in a dam Really a damage control situation there And repair is not so much Your first a lot of thought You're [00:27:00] really situation trying to just control bleeding and save patient's life And and so in those cases you know controlling the celiac artery and even consider you know ligating it may be necessaryyou know as a damage control maneuver at least initiallythe other way to see the origin of the celiac artery is still left medial visceral rotation So the maneuver we described before will show the entire we'll expose the the para visceral segment of the aorta

um to include the first centimeter to the celiac artery

**Adham:** so any keys to identifying the common hepatic artery and againthoughts on ligation strategies and repairs

**Rasmussen:** Right So I think the you know I think I mean the common a paddock you'll need to that will not be able to be visualized through the left medial visceral rotation that the common hepatic and getting out into the [00:28:00] segments there of of the the major branches of the celiac one we'll need then in that case to be coming from a you know a transparent to Neal you know anterior approach with a lesser SAC And you know di identifying the common a paddockyou know is isMmm You know requires sort of a recognition of where the celiac artery is You're in the lesser SAC The stomach really has to be Ah inferiorly This is again if you find yourself operating in this space you know the the costal margins as I said to begin really have to be flared And almost suspended upso that you've really opened this Sometimes reverse Trendelenburg is useful getting a hand a resident or a med student's hand to pull the stomach downand open up that that spaceum it you know that that will then should expose then the you know the the second and third segments of the celiac and its major branches to include the common [00:29:00] hepatic artery

**Kevin:** Great And so as far as like getting the common hepatic artery how does the GDA play into this And by that I mean the gastroduodenal artery

**Rasmussen:** I mean you know the the GDA is as I guess is most commonly the case is you know and and listeners know is Really one of the first or the first branch of the common hepatic artery and and really defines the junction between the common hepatic and proper paddock arteriesand I thinkI mean ideally um The common hepatic should should not ideally you don't want to legging any of these arteries right So I think that we start with that premise to say geez if we can get a repair done that's going to be our that's going to beoption awhether that's the common hepatic proper paddock or GDA just like any artery in the body like 18 it may be required as it is a damage control maneuver just to control hemorrhage [00:30:00] let the patient recover a little bit talk to anesthesia and sort of see how things are going Make an assessment

of of the region distal to where you've liked it So so this is just a basic tenant of like aiding any artery in a damage control situation we recognize no matter which artery rely gating the consequences of that ligation or we need to assess those consequences and then and then address them if we're able if patients you know able to um to move to the next step So obviously liking the comment of paddock distal to the GDA likely to Render you know a significant portion of the of the duodenum the pancreas and and then the liver is schematic and so you know if the light station can occur proximal if the injury is proximal to the GDA um then you know that would and G and perfusion can be maintained into the GDA then that's that's preferable[00:31:00]but if I was in one of these situations or when I am And we find ourselves having to like ate it or we like hated it We didn't quite know cause we didn't but we had to you know I was bleeding and there was a clip when we had a damage control over and now Jeannie or the common paddock or the proper paddocks lie dated Now what I usually will talk to anesthesia We'll get the patient as resuscitate as we can We'll get out the Doppler we'll make an assessment because you know dependent upon patients can tolerate but depending on the resuscitation status collateral where your legation isyou know they may be able to tolerate having any one of those vessels legged If on the other hand without your Doppler and you get the patient resuscitate as possible and there's clear aschemia then you're going to be faced with the decision whether or not we need a reconstruct the this leg vessel shunted or you know it may be situation where you have to deal with the metabolic consequences and leave the [00:32:00] leave at like eight There's not one answer You know that I think there's a there's not one answer that applies to all situations It's a I think it's a learning and adhering to the principles of damage control resuscitation and damage control surgery

**Kevin:** Absolutely so off the record dr real quick I think you miss said the distal and proximal cause you can lie gate the you can like it The common a paddock Proximal to the GDA because you'll have retrograde flow from the GDA into the proper hepatic Right could you just say that sentence and one more time and then

**Rasmussen:** Sure what I guess what I'm what I'm meant to say And I don't you know actually looking at a diagram so I can try to see the so what I meant to say is that you can often lie gait the common hepatic proximal GDA keeping a retrograde filling into the GDA and maintaining a viability of the Dudina and the pancreatic head

**Kevin:** Perfect Thank you Okay And now let's dive into the Um and this is you know very close in proximity to the celiac artery So many of the options to visualize it will be the same but there's some unique differences A is with its relationship with the pancreas Um so briefly doctor asked me Jason what are our options to see the SMA and and and do we kind of think of SMA kind of in portions a little differently than some

other vessels

**Rasmussen:** Definitely So the the SMA you know the the pathway really to see the first centimeter to the SMA superior mesenteric artery is through the left medial visceral rotation which will show the entire pair visceral segment of the order including [00:33:00] for centimeter two of the superior mesenteric artery If more exposure needs to be um you know if one is trying to expose more distal segments of the SFA then one probably have to go back to the anterior approach not the left mijo visceral rotation and go through the approach And in this case then the the superior mesenteric artery is of course closely related to the head of the pancreas And and and and so in this situation that the head of the pancreas Well it needs to be you know divided or you know the the SMA is closely adherent to the head of the pancreas in this case distal of the segment of the superior mesenteric artery distal to the pancreas is often now best seen in an infra And these a call like approach And this this is the superior mesenteric artery exits the second segment and its relation with the [00:34:00] pancreas and comes out into the the root of the mesentery and into the into the small bowel This is where for example Mmm Mmm One with lift the transverse colon cephalad as we described before and and retract the small bowel to the patient's right and see the the root of the SMA below and and inferior to the to the transverse colon So yes there there are these stink segments of the superior mesenteric artery expose Each of those segments um has a little bit of a different anatomic approach And and then and each of those segments has you know I think considerations that are important that relate to surrounding structures you know whether that's the obviously the pancreas the root of the transverse colon

**Kevin:** thank you for taking us through those complicated anatomy of the SMA as it kind of traverses the abdomen you know in [00:35:00] all of these vessels obviously we you know primary repair is kind of our our first option if possible And then many times you may need to interposition graft Just you know I know this is a question that is very case dependent is there a portion of the SMA that you can ligate and or is a is SMA just a no

ligation zone throughout

**Rasmussen:** Well that's a great question And I think similar to other other large axial vessels or arteries you know the more distal segments is just going to be better tolerated than like eating the you know the proximal proximal segments So um You know I would not say it's a no ligation zone in its throughout its entirety but I do think you know that if possible efforts should be maintained or efforts should be undertaken to maintain flow through the SMA through you know through through its entirety and you know repairing the SMA [00:36:00] just it depends upon the injury if it is a small grazing wound Then then you know perhaps a primary repair can be undertaken with a FORT or a five O proline suture if it's a more substantial wound um and and one can get it controlled and isolate it you know than than repair can be Um Undertaken with a patchy angioplasty or even an interposition graft That's pretty rare And because those wounds are just so mortal um it's very different You know patients

who have those sorts of axial injuries at the proximal portions of these message check vessels typically don't surviveum And so you know it's very difficult I I can't think of a time when I've necessarily been repairing for trauma the proximal segment or two of of the SMA it's a little bit different for an elective tumor resection atherosclerotic occlusive disease and such But for trauma[00:37:00] Um you know it's it's it's pretty uncommon to be doing extensive repair of of these SMA segments I don't I don't know what your experience is or our thoughts are on that

**Adham:** I certainly have had zero experience preparing SMA injuries Um but

**Rasmussen:** Yeah I think it's just pretty uncommon

**Adham:**I know that like you said in her position grafting is probably more theoretical than realistic in a situation like this But can you talk about the technique for it Maybe it may be useful for other types of situations but what what material are you using where you're where you're bringing the graph from and how you tunnel it

**Kevin:** I was just going to say I think you guys are exactly right And and Adam and I both experienced this is yeah many times we're not seeing traumas with these injuries but many times we're getting called by the surgical oncologist with a kind of can't cancer injury to these vessels And so I think when we're discussing SMA we can kind of keep it in the perspective of how do we replace it if we need to Um and in the situation where they need to do some larger sections So with that in mind a doctor asked [00:38:00] can W can you tell us how you would bypass it

**Rasmussen:** Yeah So I think that theI think that in these cases you know and as I said in a rare situation in the setting of trauma if you've if you've or able to establish proximal and distal control and side-branch control of this segment and then you know your options for reconstructing it um with an interposition graftyou know would include using a prosthetic like PTFE or Dacron or an autologous tissue like vein And here it it just it depends it depends on how prepared you know you are with the with patient for example as a patient prepped where the one could harvest either the very proximal segment of greater saffron is Fein or or a segment of deep femoral vein which which mayactuallyYou know have a better size match to the proximal portions of the SMA Um and and if that's the [00:39:00] case you know if there's Interra contamination either from a tumor resection or from an injury than it is preferable of course to to use autologous conduit such as deep vein deep femoral vein a small segment of deep femoral vein or if the if the saphenous at its proximal extent is It's large enough you can one can use thatif that's not possible for example the leg isn't prepped or or the expertise that is not available to to harvest the deep vein then I think using a small segment of rifampin soaked Dacron graft would be probably what I would useand you know you get some rifampin from the pharmacy you get a probably a six or eight millimeter Diameter a segment of Dacron and that's your inner position graph as far as tunneling it that and and

and how to route it That sort of depends upon the injury obviously the proximal most segments of the SMA are you know right underneath the pancreas if this involves for example a Whipple procedure where the head of the pancreas has got to come out and with that approximal portion of the SMA then um you know then that's probably gonna require a six or eight millimeter diameter interposition graft you know to replace that And if it's more distal and the SMA and now it's you know in the infra means a colic The transverse colon is off and you're and they end up in the injury or the segment is for example below the transverse colon and that's going to be you know three or four millimeters and it may be there that vein you know sadness pain would work or in some of those cases a patch angioplasty using vein is actually

easier and better than trying to do an interposition graft to sort of debris the the injury and then and then do a patch angioplasty using a segment of a saphenous vein

**Adham:** All right One follow up questionin these situations with SMA [00:41:00] injuries so you were a parent and the bowel doesn't look infarcted or threatened at the time Do you think it's mandatory to leave these patients open and do a second look even if everything is fine on the first timedo you think you can kind of take it on a case by case basis

**Rasmussen:** Yeah I think and I think you're getting at a an important point here which is one we've emphasized throughout our discussion is that none of these procedures these techniques that we're describing take place in isolation right They have to be in conjunction with anesthesia and an assessment of the patient's physiology And the whole team including the intensive care unit team et cetera So it's a really good point My own preference in these situations is to leave the abdomen open with a temporary abdominal closure of some sort and plan a look Um you know 24 to 36 hours later I I really That I think it I think we know that the morbidity of [00:42:00] of that approach is very low I suspect you know um I think it probably provides not just reassurance but probably some real advantages in case the the perfusion actually is not adequate and the patient becomes acidemic and the bowel is compromised you know subsequent to To a pure initial departure from the or

**Kevin:** Great And just to breeze through the inferior mesenteric artery would you agree that you can generally like gate this with impunity if it's damaged in a trauma

situation

**Rasmussen:** I think never say like eight with impunity but I because there's always situations where You know I think it has to be thoughtful a ligation and in this case yes I think the majority of times the the inferior mesenteric artery can be ligated I think the thoughtful approach here is to make an assessment as of to the patency of the iliac vessels and specifically the internal iliac vessels I think

if What we're [00:43:00] describing which is ligation of the inferior mesenteric artery one can be more assured that that's going to be tolerated if both internal iliac arteries are patent for example if the SMA is paid and the celiac is paid and then then the inferior mesenteric artery is almost always tolerated Um if for some If it's an aged person who's got occluded internal iliacs or they've got atherosclerosis lucid disease and their mesenteries are not good then I think you have to be a little worried about ligating the Ima in some cases

**Kevin:** Right And just for residents that'll be rounding I think we generally discuss when talking about the Ima and you know open aorta situations if it's occluded Or it's pulsatile then we feel comfortable like eating it It's when it has that kind of a slight dribble back that we may consider re implanting it just to kind of prepare you for your rounds dr SPC and we're going to jump into the last vessel of branches [00:44:00] of the aorta Not an easy one to discuss either we're going to get the renal arteries and You know I I know it differs kind of whether it's proximal or distal it very much as far as your exposure but how do you think of the renal arteries in your management of them in a traumatic situations

**Rasmussen:** think that it's I think that what I'm thinking about in that question is Is similar these other vessels that that and hopefully it's helpful as a broad principle and an approach you know we started this discussion talking about exposure and making sure communicate with anesthesia and you know having the right suction devices and retracting devices and headlights in these broad concepts to make sure that you you've thought about um going into these cases you know I think um when it comes to the renal arteries like like all these festivals um it's useful to think well I'm either in a in a fairly elective situation you know meaning I've got things under control for the moment Patient's physiology is a [00:45:00] solid um you know there's there's whatever bleeding there is is controlled and I I've got a reasonable exposure in contrast to I am in a uncontrolled situation and there's the patients in a in a really bad situation you know the bleeding is not controlled I don't know where the bleeding is coming from et cetera And I think because it helps one to Have a mindset about whether I'm pursuing damage control surgery you know am I going to have to how how much am I going to work to maintain a patency of this vessel or am I going to be more tolerant of ligating it and just as a damage control man over and having to deal with the consequences but recognizing I need to do it to say the patient's life So the renal arteries are like that I think because it is similar to the SMA it is pretty unlikely in the setting of trauma to be reconstructing a renal artery I think that's pretty rare in trauma that the kidney the renal artery is either [00:46:00] Not injured you know not injured And you know there's there's hematoma that is from the parenchyma of the kidney or surrounding vessels or the renal artery is injured and Broncos you know and and And if it's certainly if it's injured in thrombosis by the time you know an individual gets to it it's probably too late The scheming time or mosquito time to the kidney's been too great and there's not going to be value of of trying to reconstruct it in the setting of trauma And so if it happens to be an in between and I say well let's say that

a actually it is one of these cases where you know explore the The the zone two hematoma and a is coming from a main renal arterythen you know and there's flow in it but it's bleeding Then I think the principles that we have described such as either primary repair four or five Oh potentially probably in this case maybe a six O monofilament sutures such as proline It would be useful if that appears to [00:47:00] compromise the lumen andeither a patch angioplasty of that artery or you know a bypass But again that is extremely rare in the setting of traumayou know if it's going to require more than than a primary repaireyou know it's I think in my experience equally likely that you know you're going to beligating that vessel for damage control

Um but but it's pretty rare to be doing a complex renal artery repair in the setting of of trauma similar to the proximal SMA injuries for example

It just pretty rare

**Adham:** right Can you talk to us about your emotions You're dealing with your renal hematomas whether they're Penetrating or blind I know the old adage of every penetrating renal hematoma should be explored and blood one should not be explored unless they're expanding May not be true but can you kind of walk us through how you approached this

**Rasmussen:** Right So you know zone two this would be a you know zone one or the central dominal hematomas and you know the rules that [00:48:00] you're alluding to or that you know almost all or all zone one hematomas should be exploredZone two hematomas which are the kidneys in the lateral gutters basicallyyou know the the general rule is that if it's a expanding hematoma regardless if the mechanism is blunt or penetrating then those hematomas Should be explored in zone two Um the general rule goes as most of your listeners know if it's a blunt injury and the zone two hematoma is not expanding then that does not need to be exploredIn contrast if it's a zone to hematoma from a penetrating mechanism then most of the time though than need to be explored even if the hematomas is stable Those are the general rules that we we use to guide our approach to zone two a retroperitoneal or zone to retroparotoneil hematoma So I use those I think that's those are pretty goodYou know I I think there are times [00:49:00] when a blunt you know one finds themselves in in in the belly because there was a hemoperitoneum from a another injury And and and there is a blunt mechanism and there's a zone two hematoma I have explored those in the past And I think that if I don't think those guidelines are meant to be You know a dictums that can never beadjusted for an individual case I'm not sure if that

**Kevin:** So one question I know a lot of times many of these cases we don't already have the laparotomy performed Um you know they're in the trauma Bay They get a CT scan and they they call and say there's an intimal flap There's a fistula maybe a pseudo aneurysm and the renal artery any kind of general principles for

some of these sort of

incidental findings

**Rasmussen:** and I think that's a that's probably a so what you described there is is the most obviously I guess the most common scenario in which which it's a The CT finding you know right in or just after the resuscitation room of a of a main [00:50:00] renal artery injury branch renal artery injury or maybe you know parenchymal injury to the kidneyand in those casesI think if the let's say it's the intimal flap that you described If the intimal flap is Either not Mateen and there's perfusion in the kid on that CT scan for exampleand it does not appear to be flow limiting then that can be observed some of those like other blond arterial injury some of those will healif it is amenable to an endovascular placement of a bare metal stent that Can be used in some institutions to tack downor stabilize that detection Um and that will be if if you really feel like it is a flow limiting more than 50% stenosis caused by that intimal flap the more distal branch artery injuries can often also be managed with endovascular techniques such as [00:51:00] coil embolization Replacement of an occlusive you know hemostat coil or other deviceand I think coil embolizing or or or treating those depends uponyou know how the patient is doing how much blood they're requiring how large the hematoma isand then what their hemodynamics arebut I think for those reasons what we described five or 10 minutes ago um having to fix these main these renal artery injuries in the ORs pretty uncommon because they've either been diagnosed and managedbased off CT with or without endo Endovascular technologies and oftentimes the management of a renal artery injury If you're in the or exploring and expanding you know hematoma and one of the zone twos is is going to be nephrectomy you know because it's it's that major injury in which there's just not there's not the [00:52:00] indication for repair

**Adham:** all the ranches of the aorta from the diaphragm Down to the renals and Ima And I want to thank you very very much for taking a complex topic and it's sort of convoluted in breaking it down from technical steps to algorithms for exposure to how to repair I think this was overall an excellent episode Well we haven't touched on the endovascular management of trauma which we'll save for another episodebut thank you so much for for a great discussion I for recipes

**Rasmussen:** well I applaud you for tackling this topic is unruly you know it both in the the anatomy the anatomic exposure operative exposure and then the seemingly endless number of scenariosThat can be considered for either observation repair or damage control ligation I mean it's a it's a complex topic I would commend you for tackling it and I hope that our discussion is is useful for for you and

and your listeners

**Kevin:** [00:53:00] Definitely I think this gives people the the groundwork to really make some safe decisionsone thing I want to mention to our listeners if you click on our show notes or you go to audible bleeding.com we're going to have links to different YouTube videos that will have breakdowns of many of

these exposures so you can listen to this and then go watch it later to really reinforce it so thank you dr recipe set again for an extremely complex episode that you're breaking down onto a podcast

Thank you for listening to audible bleeding. This is part two of six of our vascular trauma series with dr mucin. Be sure to tune in to hear the rest of the series. Yeah.

Check out Dr. Rasmussen's book

<https://www.elsevier.com/books/richs-vascular-trauma/9781455712618>

<https://www.amazon.com/Richs-Vascular-Trauma-Todd-Rasmussen/dp/1455712612>

## 10.4 Cerebrovascular Trauma

**25 May 2020:** *Kevin Kniery, MD, MPH; Adam Johnson, MD, MPH; Nicole Rich, MD, MPH; Todd Rasmussen, MD*

### Tenets of Cerebrovascular Injury

1. Control of catastrophic bleeding — with manual compression and resuscitation
2. Ensure that injury is not causing airway compromise
3. Evaluate the neurological status of patient — have a thorough baseline neuro exam and look for signs of ischemia, hemispheric stroke, arousability/mental status
4. Prevent secondary injury — maintain blood pressure to maintain cerebral perfusion pressure and prevent hypoxia

### Blunt Carotid Injury Patterns

- Mechanism of Injury: history of a motor vehicle collision, blunt force to neck, severe hyperextension, etc.
- External signs of injury: marks on neck or around the thoracic inlet (ie “seatbelt sign”))
- Unilateral neurodeficits — could suggest hemispheric ischemia or stroke (less common, but possible)
- Accompanying Injuries: concomitant closed head injuries (TBIs), complex facial fractures, cervical vertebral body and transverse foramen fractures

### Blunt Carotid Injury Classification System and Management

Well, I think that as we think about this one thinks about, the anatomy of the vessel wall, the arterial wall. that helps us conceptualize the degree or the severity of, of these blood carotid injuries. They can be, as simple as a, as an intimal tear, , where, you know, it is, the intima has been raised off of the media and it creates a minor flow defect.

, which may be prone to platelet aggregation. but it's, it's not a, , a long dissection and it's, it's not a flow limiting defect, and that is the [00:08:00] most minor. , and then of course, the most severe on the other end of the spectrum is a blond carotid injury. That's resulted in. In, in complete thrombosis of the artery so that, , it's either the vessel has been transected from the blunt injury and is thrombosis or, , or the, the injury is this, such that the intimal is lifted up and it has created a longer this section that is, that is now thrombosed and occluded the vessel.

And then there's the degrees of injury between those two extremes where. There can be a longer does section, that, has thrombus associated with it and is visible on the cat scan or the imaging modality, and or, a dissection that can be flow limiting the defect that can be flow limiting.

They may not have thrombus and it, or B along the section, but it, but it is in itself flow limiting, meaning that it. It's, , compromises greater than 50%, for example, [00:09:00] of the, of the lumen of that pestle. So, you know, the, the varying degrees of severity, you know, with our imaging technologies today, mostly CTA, it is possible to pick up, you know, or is.

If you're able to pick up or proceed the different, , types of severity from a, again, from a real mild intimal defect that's not flow limiting, that has no thrombus all the way to the more obvious, , you know, , , injury where there's complete thrombosis.

: Is there any role for ultrasound, , in addition to CTA, if you do see some sort of intimal flap, , to maybe see the, if there's any hemodynamic impact of it, or is it. Does CTA give you the information that you need.

I think that's a really good point. I love, I really like ultrasound. I think you alluded to this, with your comment, you know, CT gives you, gives one a static image. , it's often quite detailed and, , boy, I really also think that CTA [00:10:00] is, I mean, in many ways it is the gold standard, but it is a static image.

Yeah. In contrast, duplex ultrasound, which combines B mode, you know, ultrasonography with POLST Doppler , gives one a real dynamic, assessment of the area of interest. Meaning is, is the, is there, I mentioned the word flow limiting is, is there a elevated velocity and a detriment in, in, in.

Or a change or a difference in the velocities around that area. So I think, I think you can get a lot of information with duplex though. Of course, the limitation with duplex, and we may talk about this, when we talk about the different zones of. Of of, of neck injury, you know, the, the limitation with duplex is it, it you cannot see above the angle of the mandible.

So you quickly kind of run out of real estate distally where you can use duplex. It's not good for distal internal carotid injuries and you cannot see, you know, proximal to the thoracic outlet. [00:11:00] So, you know, there is a limited

window in which duplex is useful. But, , but in that window, I think it gives really important information.

\*\*That's a great point. So going back to the grading of the injuries, , Dr. Rasmussen, you described to us a grade one and grade two injuries, which involve intimal injuries, , less than 25% in grade one or grade two with a greater than 25% luminal narrowing. can you talk about the management of grade one and two injuries.

Yeah, I think I'm grade one and grade two injuries. Um. Most of the time are managed non-operatively and can be managed expectantly with, , use of antiplatelet therapy, potentially anticoagulation. depending upon, the patient's global burden of trauma. Do they have an associated TBI? Do they have other, other injuries?

But. if, if there are, if the patient is [00:12:00] neurologically intact, and a grade one or grade two injury is seen on imaging, and the patient is able to have anti-platelet or anticoagulation therapy, then those, those types of great injuries, grade one and grade two are managed. Non-operatively, , with, , I guess you'd say medical management.

If the patient changes, meaning that he or she was initially neurologically normal, and medical management is initiated and then the patient changes has hemispheric symptoms indicating that your medical management is failing. Then that is a situation where, , an intervention is, is this likely to be needed or would be indicated, if that makes sense.

: That drives me is to have a question. have you ever been in a situation where you have a grade one or two injury, but a [00:13:00] patient that you're unable to get a neuro exam on? Are we obligated to, ask for things such as transcranial Doppler or EEG or, or, or something to prove that there's no injury from that or, you know, what are your thoughts on a situation like that?

Great question, and I think I'm. I'll probably use this phrase multiple times in this discussion. Is it? It depends, partly it depends on what modalities, one has available to them in their institution, but I think the answer to that, if I could, if I come down from on it, is, no, you're not obligated.

I think it's a luxury. , that some places may have that if you have access to EEG or transcranial Doppler, it might be able to be used or sort of a luxury in your diagnosis, but I don't think that's really necessary. I would mostly treat them, as I said, with medical management and then imaging, serial [00:14:00] imaging, both.

Of their neck and, of their brain. and I think in a way, and then you mentioned it earlier, Kevin, I thought that was really important. , supportive measures, like preventing, secondary injury, you know, which means optimize the profusion pressure to the brain. , try to avoid or lessen the likelihood of secondary insults such as hypotension, shock and, and such.

So I don't think you're obligated to get those things. I think that things like EEG, transcranial Doppler are more in the category of a luxury if you have them. but otherwise I would manage with a serial imaging.

\*\*So Dr. Rasmussen in a patient who remains neurologically intact and you plan to, that medically manage and do serial imaging, what's the best timing for repeat imaging?

Yeah, that's a great question. And I, I think, for, for patients that we manage and in my experience, we manage, we at least image him twice in the hospital. While they're an [00:15:00] inpatient. So, , to make sure that they're not changing or evolving, you know, in the acute phase of their injury. So, you know, if, , if, .

The typical patient is someone with a, you know, with a blunt, head injury and they have a finding at the time of, , of a grade one or grade two carotid injury. we get the CT at the time. They're admitted to diagnose it. We follow them, you know, over the course of three or four or five days as they recover from their injury.

They need another imaging study, I think within, you know, within the first week to make sure that that. Grade one or grade two injury has not evolved into an either a pseudo aneurysm, because remember, there's a, by definition, a disruption of the arterial wall or an extension, you know, from a grade one or grade two into something that's more, more significant.

So I think the initial scan on admission, of course, and then within the first seven days to make sure they've not, that injuries not evolving. [00:16:00] and then typically after that, I think, again, it depends on. sort of the severity and how the patient's doing, but probably at a month after discharge from the hospital and then most of them will heal.

, certainly grade ones will heal, hopefully grade two, but, but they do need followed, , to the point of resolution or healing as an outpatient. , usually with CT scans or CTA.

\*\*Okay, great. And series of these patients have shown that 60% of them will have their injuries devolve or change in greater severity in this period. Is the pattern that you see typically that they tend to improve over time?

Well, I think the, I think the grade, you know, the grade ones, , I think they do. they're, those are the ones where that's a minor or limited intimal disruption. A small intimal flap that. that is not flow limiting. It's not associated with thrombus or platelet aggregation or thrombus.

So I think most of the grade ones do, and I [00:17:00] think, I'm sure the grade twos, I think it certainly a greater percentage of those are prone to evolve into, you know, a longer deception formation of thrombus around them or into a pseudo aneurysm. And so that's the, the point of, of serial imaging. You know, within the first week of the injury.

, and then certainly, you know, , another one within the first month of the injury he had at best case, certainly if they begin to evolve or you see on that second CT. That the image that the injury's changing, let's say it's a grade two to your point that you think and you see signs that it's evolving, then by all means then that that needs then more intensive imaging and likely if it's evolving into something more significant, it needs an intervention.

\*\*Okay, so a pseudo aneurysm is what we consider grade three. what is your approach to managing a pseudo aneurysm of the carotid?

Yeah, so I think the, the suitor aneurysms [00:18:00] of the carotid, you know, that in a way it depends. It's influenced certainly on, the patient's neuro status or the location of the pseudo aneurysm. And then I think the expertise of, of the team that's managing the patient. but I think that the pseudo aneurysms, all of them, no matter what size they, they need to be treated, they, they require, you know, they require an intervention to fix them.

there certainly are some patients who, um. Who's you know, have a high mortality. They, they're, they're, they're quite ill or have a high, high injury severity score. They may not make it. There's some patients that one may pass and not fix a pseudo aneurysm or grade three entry, but by and large, most do because the pseudo aneurysms will.

, will, they will not go away. Then they're likely to get bigger and become associated with either expansion and mass effect or bleeding or thrombus formation, stroke. so, the, the ways to treat them, I think are [00:19:00] these days primarily with endovascular techniques that include covering, , the pseudo aneurysm with a small coverage stent.

Or what we'd call a stent graft, a small covered stent to seal the, the opening of the pseudo aneurysm. You know, there are some reports and experiences using what we call a bare metal stent, through which coils can be placed to thrombose the pseudo aneurysm to cause it to clot. And then the bare metal stent then, you know, maintains the integrity of the wall.

Um. Such that, flow is maintained through the, through the lumen, those both use of a stent graft or recovered stent or coiling of the pseudo aneurysm, or interventional techniques or endovascular techniques that are performed most commonly through a transfemoral approach, an RJ order gram, and then selection of the.

of the [00:20:00] effected carotid artery and then placement of the, of the endovascular treatment. I think those, these days are probably the most common. Certainly if, it is, in, you know, in zone two of the neck, and we can talk about that a little bit. , and it's amenable to an operation. there's another indication for an operation in that area than, than certainly open repair of the pseudo aneurysm can also be.

Undertaken, but I think more commonly these days for grade two injuries, they're managed with an endovascular technique.

: In your book, it's mentioned that, considering waiting seven days to decrease neuro events, , what's the thought behind this?

Yeah, definitely. I think that, if a patient, um. You know, Harrigan, it depends. I think that it depends on the patient. There are some patients who just are in no shape to undergo. you know, , , an intervention such as this in the acute, , in the acute setting, the first two or three days and, and certainly in those patients waiting is, is just, you don't have a choice.

You need to wait. I think Yeah. If the patients, [00:21:00] you know, are awake, let's say they're awake and otherwise normal, you know, in those patients, if they're, if it's a pseudo aneurysm, the patient's awake and, , able to be examined. I, I often don't wait seven days. I think the waiting period probably more pertains to patients with polytrauma, maybe an associated TBI and, and a, and a more complex, , complex course.

I think if it's more. A cut and dry, the patient's awake, , able to be examined. They're out of the unit. , then I don't wait necessarily seven days.

: Got it. So, , for the grade four injuries, , the occlusions, we just do medical management for these patients if they're already included. can you just talk to us briefly about, how you decide on medical management, whether you were using anti-platelets or dual anti-platelets or anticoagulation?

How do you go about this?

Yeah. So this is really, an area where it is, you know, we don't have a lot of good, we don't have really any good data. We have. [00:22:00] Really good clinical experiences by a lot of expert groups, but we don't have a lot of, prospective data or level one data that guides our management in these cases.

and so I think, patients with occluded, let's say a blunt carotid and secluded grade for injury, patients who do not have associated injuries or conditions. That preclude the use of anticoagulation, then I will use the anticoagulation. We'll, we'll give them, , intravenous heparin, and, monitor them with repeat imaging and obviously, monitor them to make sure they're not over anticoagulated in pain.

And that tends to be the exception because many of these patients have poly-trauma, so they have an associated TBI. Or other body regions that have been severely injured. So that anticoagulation is not an option. And in those cases, , we are really left with using, um. , [00:23:00] an antiplatelet like, clopidogrel or aspirin.

In those cases, I'll often use both, , dual antiplatelet therapy, clopidogrel and aspirin. And these are in patients who cannot receive full anticoagulation because of other injuries. one point there, I think if, if one's going to use anticoagulation, and I think this group, you know, Kevin, you and Nicole are, and those of us who get called frequently for bleeding complications are sensitive to this.

But in these, these patients, we're going to use anticoagulation on for a blunt grade four bland carotid injury. I really try to avoid, , protocol based, bolusing. Of anticoagulation. I think our internal medicine friends and often our intensivist get into these protocols where they'll use large boluses of heparin as if the patient's being treated for a PE.

And I think that, I try to avoid that because I, in my experience, that leads to [00:24:00] bleeding complications either in the brain. The neck or somewhere else in these injured patients. So I typically will, when I start anticoagulation on these patients start pretty slow with a goal PTT of 50 to 70 and in the first 24 hours, I often will say 50 is better than 70 because these patients with this type of trauma are just prone to bleed.

and then work up slowly to therapeutic anticoagulation without boluses. and again, these are patients that they use the anticoagulation and the don't have on the, they shouldn't have other major injuries. So then they get transitioned to an oral anticoagulant for, 30 to 90 days.

: That's a really good point.

\*\*Okay, great. So why don't we move on to discuss blend for tibial artery. Injuries, Dr. Rasmussen, when you're assessing a patient's imaging who is presenting with a trauma and a blunt vertebral artery injury, what are the kinds of things that you look for in terms of the dominance of the vertebral,[00:25:00] and how that affects your management?

Yeah, good point. And you've used. Kindly, , , tips, pointing me in the right direction. I think it, depends upon, you know, one of the main things is, , well, first of all, starting with a physical exam, and as we've mentioned, you know, how, how is the patient, when you first assess them, do they have.

No signs of bleeding in the neck, airway compromise, et cetera. But then, you know, quickly with these patients, and to your point, you get to the imaging and, and I think one of the key things is it, which vertebral, is it the dominant vertebral artery that is affected? Or is it the diminutive, for TBL? Or, or maybe you can't tell.

but I think that is important. and then I think, It's similar in some ways to the carotid injuries. It's the severity of that vertebral injury, both its location, anatomically which of the vertebral segments. And then, you know, what is the, what is the severity of the injury?

Is that a mild. grade one intimal flap [00:26:00] that's sort of seems non flow limiting, or is it a full full blown occlusion, , , of the vertebral? And then lastly, is it associated with a baseline artery defactor posterior circulation, stroke? Those are sort of the things that I try to go through in my mind.

Um. As I look at the imaging, you know, one thing that's important to mention, and we were just talking about this with one of our residents or trainees, , last

week we did a first rib resection and talked about, the severity of, of impact that it takes to make these create these sorts of sorts of injuries.

You know, I think. What I would say is yes, be focused on the vertebral artery, or in the case of the carotid, be focused on the carotid, but also be aware that in, oftentimes these cases can be associated with other torso, vascular injuries, blunt aortic injuries and such. So, you know, like, like we always try to do ourselves or teach.

You know, don't get tunnel [00:27:00] vision with these patients. Look at their imaging with regards to the vertebral, but also be aware that, , they often have other injuries that you need to open the aperture to see.

: That's great. from my experience, , most of these injuries of the routine rules are managed non-operatively or occasionally with embolization, maybe, by neuro, I R. unless there's maybe a pseudo aneurysm, and per your book, 90% of these stenotic lesions will resolve. Um. Is that the experience that you have to,

Yes. And I think the, you know, this, they fall into a similar management pattern, is the blunt carotid injuries. maybe not quite the same because their natural history is a little different, but, but I think the, you know, as far as serial imaging, management with, , either expectant medical management for burn for those that are, that are less severe, and then endovascular management.

with those that are more severe, I think, you know, in a way, they do fall into a [00:28:00] similar management pattern. And yes, , the majority of those that, , that are low grade do resolve for or become asymptomatic over the long period. Meaning they may, the receivable may, may, if it's thrombosis, I mean, oftentimes that does not result in.

Stroke. It just, depends, you know, but many of the smaller injuries certainly do resolve over time. , and, and that can be seen with the serial imaging approach that we've already talked about.

\*\*So Dr. Rasmussen, Kevin mentioned that a lot of these vertebral injuries, especially if they're kind of high up. In the neck or the skull base are managed by neuro IRR or conservatively, but if you need to expose the proximal vertebral artery in the neck, what's your approach for that surgically.

Right? So that's a, that is exposed through a supraclavicular incision. and for those who have, um. Looked at up or [00:29:00] performed, a carotid subclavian bypass, for example, or the, the exposure of the subclavian artery. Most mostly that exposure, or most commonly that supraclavicular incision is, is described for exposure of the, the, of the subclavian artery.

but through that same supraclavicular exposure, um. No, the vertebral artery can be found a little bit more proximal on the subclavian artery. So that's, that's sort of the, that's the, that's the incision or approach. and you know, it depends upon the, you know, one, you know, what one is operating for, if it's,

if it's for, for bleeding and the intent is to, to control bleeding or ligated, that's actually most commonly.

The case, I mean, is, is that, , you know, that exposure is then used because it's a hematoma and, and there's, , there's bleeding. you know, then the intent is to expose, , the, the vertebral is, it comes off of the, the subclavian and to [00:30:00] litigate it, it's rare that one would be repairing or trying to restore flow in the setting of trauma.

to the vertebral artery, you know, through that incision. We don't really typically do for Teebo reconstructions for trauma. typically if you're doing it through an open operation, it's really to control bleeding and, and, , probably gonna end up lying eight in it, just because a re implanting the vertebral, you know, on the common carotid or doing some sort of achievable reconstruction is just not well suited for for the trauma situation, if that makes sense.

: Yeah. And one quick thing before we move on, I just wanted to touch you on a paragraph that I took from your book. that I think is really important because we don't do. Artery exposure as much. I've only really done one in fellowship. I've been a lot of credit subclavians where I see it, but an actual exposure for TB and your book, it mentions you go in between the two heads of the SCM.

you open up the credit chief and you retract the carotid immediately, and this will you, then you'll be able to identify this is a critical [00:31:00] structure of the vertebral vein, which directly posterior, which is directly posterior to the carotid. And this vertebral vein is sort of a landmark that helps you identify the vertebral artery and the proximal subclavian artery.

And the one of these that I've done that was exactly the, anatomic, exposure

Yeah. Good point. We didn't tie, I didn't talk about the anatomy or the specific anatomical aspects. That's exactly right. I mean, I think one of the things, a couple of things that are useful in addition to to the vertebral vein, I think that, um. I think that identifying, the sternal head and the clavicular head of the sternocleidomastoid, you know, and trying to find it really in a lot of ways, you can go between those two heads of the sternocleidomastoid.

and it's in that sort of, that groove there where one can, and, and oftentimes that's just a, I mean, it is, there's an open plane there between the, and if you can look at your anatomy book, kind of get that out and see the. The sternal head and the clinic killer had, , really the window there, lead you then, you know, to that area where posteriorly, there [00:32:00] will be the, the vertebral artery will come off.

And so claiming, you know, you can feel the carotid, and it's just beyond, it's just beyond the takeoff, at least on the right is just beyond the takeoff of the carotid artery.

: Great. And now that we're kind of talking about operations and we're going to keep diving into penetrating injuries of the neck. So dr , we just want to

just kind of start with some of the basics, , when we're talking about the zones of the neck, , and, and what are the anatomic considerations of each of these zones.

can you just tell us what zone one is and kind of what you're thinking of if you hear of a vascular injury in this area?

Yeah, so, you know, so zone one, so the zones of the neck, you know, really, refer to the, the anterior, aspects of the neck. and so the, the sort of the anterior triangles of the neck, and, um. The zone one really is, is the most proximal. It's the inferior, , aspects of the, , of that, of that.

: So doctor asked me, he said, I think we're going to dive now into penetrating carotid trauma and start with sort of the basics, , diving into the zones of the neck and some of the things you take into consideration with each of these zones in the neck as far as getting control.

So the zones of the neck, that are, have been used to help us conceptualize, you know, penetrating injury mostly. . Oh, really? Apply to the anterior portion of the neck. , kind of the anterior triangle of the neck. zone one is, is the, is the, , is the sort of the first and the most, I guess.

proximal zone, and it's really defined, by the sternal notch to the cricoid cartilage. So it's a tight or narrow zone that really involves the thoracic outlet. zone two is the cricoid cartilage to the angle of the mandible, which is, sort of the largest of the, of the three zones, , in the [00:33:00] neck, the intern neck.

And then zone three is from the angle of the mandible to the base of the skull. Like, zone one's on three's pretty tight. there's not a lot of space there. and those zones, I think are useful and that have been described. , there was original paper led by, dr rune, R O, N, H, J rune, and, , in the journal of trauma in 1979 that, first described those, those zones, it was called evaluation and treatment of penetrating cervical, , injuries and in those zones I think are really useful just as a, to help us conceptualize into, to try to, , manage, different forms of penetrating injury or different areas of penetrating injury.

so those are the zones. , I think that. You know, teaching, , at least over the years has been the, penetrating injuries to zone one and zone three largely, require, you know, imaging at the outset. and maybe either non-operative repair or at least imaging first with, , because they're very difficult areas to expose penetrating injuries to [00:34:00] zone two.

traditionally have been in the teaching has been that those are all managed with, , an open operation and exploration of the neck.

: and then for zone three, sort of, I think, much like zone one, , we depend on sort of imaging and angiography, , for treatment of these injuries. kind of want to dive straight into this and, , discuss how do we manage credit injuries. So we're, we're going to just pitch some scenarios at you and we, can discuss some of the nuances of it.

Um. If you have a penetrating carotid injury, zone two, , and the patient is neuro intact. I mean, it's this kind of the most straightforward situation. But, , I guess, I guess a better question to you is, is , if they're neurologically intact, how do we determine whether or not you're going to repair the credit injury

Yeah. I well, I think probably the case-based scenarios are probably best because. You know, each management, as I mentioned, the zones are there to provide some conceptualization, help us to categorize our management. But, , the, management of any given cases is often, individualized.

So in this particular case, yes, I mean, whether or [00:35:00] not they're, neurologically intact certainly matters. I think with penetrating zone to injury, um. I would still operate on that. I think that, that, that, I think that paradigm is changing with modern imaging technologies, CTA mostly, and clinical experience.

But I think that teaching is in the case that you've presented a penetrating zone to neck injury with a carotid or penetrating zone to wound with a carotid injury, then that patient would be. Operated on with an open operation.

: the neurodeficit portion of it, you know, historically there was concerns over if you repair this carotid injury, you know, that you could create intracranial hemorrhage. So, say you have a patient that comes in with a zone, two injury with, , you know, on the right neck with. Left-sided neuro symptoms. w what are your thoughts on that? Does, does the neuro status play into it or do you repair, the carotid.

Yeah, I think that, and these days, you know, as I mentioned or alluded to, we have the benefit of CT imaging. So, you know, the, the [00:36:00] paradigm of that was. I mentioned the, AGA rune paper from 1979 and that really set up the, the zones, you know, that was in really, that was in an era where we did not have, and for many decades after that, we didn't really have the benefit of routine high quality contrast CT imaging.

Today we're operating in that, , in this new era with. Routine, high contrast or high quality contrast CT scan. So, I think in your case, you know it, if the injury has a CT scan, which is likely to have these days unless the, unless the injury was associated with bleeding and the patient was just taken immediately to the, or, you're going to have a CT image.

If the CT image shows. You know, a grade one type of injury, you almost extrapolating the, you know, the, the blunt characterization or the blunt categories. If it's zone one, then I suppose, you know, those may also, you know, may not choose to operate on those, but I think penetrating wounds, I would, [00:37:00] I would operate on them and I think, um.

No. Cause I think they're going to evolve. And they, the other thing about exploring zone to the neck is that you can explore for other Aerojet aerodigestive injuries. I mean that, that's the other dictum or part of this is to make sure

that there's not an injury to the, to the, , to the airway or to the esophagus, as, as, as you perform the zone to neck exploration.

So, um. You know, I think that they get, , an exploration. They get a, you see the carotid, , and , , you explore and you see the injury and then you manage it depending upon what the injury looks like at the time of open exploration. I mean, it's hard. I think it would be hard for me to sit on a penetrating zone to wound that has a.

Carotid injury, I, it's potentially described. , and I think, but I think, there's a couple of reasons that I would err on the side of, of operating on that.

: Yeah. And I think, , historically, they talked about, like I mentioned, the intracranial hemorrhage. So if you had, say, a, , a very, , beat [00:38:00] up looking carotid artery, or maybe. near thrombosis that they would leave it, because they're afraid of reproducing the brain, causing intracranial hemorrhage.

But it seems like the consensus now, at least from your book and the East guidelines is, to repair these deficits, , or to repair these credit injuries because the mortality and final nerdy neuro status, , has been shown to be better in these patients. even if they started with a neurodeficit.

And I think that we, you know, as vascular surgeons who do carotid endarterectomy, , routinely, I think we're more so, I don't, I do not. So, first of all, this is a complex, . Complex scenario. So I, I'm definitely won't throw shade on any previous experiences with this sort of heroin, injury pattern.

I do think that we, um. You know, for those of us who manage and perform carotid endarterectomy routinely, we sort of are, are familiar, I guess, when maybe not good with it, but we're at least familiar with trying to mitigate hyperperfusion syndrome or reprofusion syndrome. And I think that is, you know, that's just part of the, the management.

There's the technical management of the injury, and then there's the communication with anesthesia to try to lower the blood pressure and the overall profusion pressure. in these patients after you repair the carotid, it's not unlike communicating with anesthesia about resuscitation, right?

We need to, communicate with anesthesia in this case, to mitigate hyperperfusion or reprofusion and worsening of a brain injury. We, we, need to, , [00:39:00] communicate with anesthesia as well as do a good job of fixing the, the injury.

\*\*That is a great point. what about in a situation where you have a patient who has a large infarct. On the same side as a carotid injury. What is your approach in that situation?

Yeah. So I think, you know, and it depends upon the injury. you know, I think if this is a penetrating zone to wound. you know, in nearly all instances, you know, so you're going to explore it. because it's his own two neck injury. I mean, I, I think, um. You know? Okay. So there may be some that are, that are sort of there.

I'm trying to avoid the words. It depends, you know, I suppose there are some minor penetrating injuries where the, the image, , you know, it's a small stab wound or ice pick type of thing. , in, in, in, there's, there's, there's no hematoma. It's looks like there's a injury to the carotid. That's, , that's, that's, .

, resulted in, , in an infarct or a stroke, but there's no bleeding or pseudo aneurysm. there might be scenarios where you would observe it. I, I don't, that's not a practice that I, a spouse. I would explore all of these. I still sort of fall, fall back on exploring all penetrating zone two injuries. and in that case then you're going to be in the neck anyway.

You're going to see the injury to the carotid. And I guess your question. Nicole is in the setting of an infarct, would you then repair the carotid? I mean, your options are either to repair it, do nothing to it or, or lie gated and I guess as a vascular surgeon, you know, I would repair it. I think observing [00:40:00] it while you're there looking at it is then you risk it evolving into a pseudo aneurysm and you having to re operate on it, you know, days or weeks later.

As it is, the small injuries of all. and I think, you know, the, the question of, well, if he's already at the patient already has an infarct, would you like it? I think that that don't, that's just not something that is a vascular surgeon, that, that I would pursue. I think, I would try to, , I would try to repair the injury reperfused the hemisphere.

acknowledged that, yes, you could make things worse with reprofusion. but, , you also may take up a number that was ischemic and you may reproduce it and, and push the penumbra in a positive direction. So, you know, there's not a lot of data in this area. And I mentioned the word heroin. you know, these are the most challenging of, of injury scenarios for sure.

: Definitely. Yeah. there's no easy answers there,

I mean, what do you think, Nicole? What do you guys think and what's your experience? I mean, I, [00:41:00] I'm, I'm interested also in what you guys think about that case.

: I have one experience with this. It was in residency. we had a, a person that was sleeping and, , a, a pressure washer guy came up to him and asked him to move. He refused to move and they got in a fight and I got the pressure washer straight to the neck. , tore open his carotid. They came into the ER holding pressure on his neck and huge hemiparesis on the contralateral side.

in that case, we did repair the carotid, and he did have a dense hemiparesis after the case. you know, whether litigating would have helped or not, you know, we don't know, but that, that's my, that's my one experience.

\*\* I actually haven't seen a situation like this,

but it's definitely a tough, it's a tough situation. I think if you have an injury that's going to cause potential for bleeding or a pseudo aneurysm. And the vessel is payment, then my [00:42:00] inclination would be to repair it. if you're

doing, if you have like an occlusion of the artery, when you explore it, you know, say you try to do a thrombectomy and you're not getting any retrograde bleeding.

That might be a situation where I've considered ligating it, but these are tough calls.

I think you're, I think you're spot on. And I think as surgeons, you know, at you know, at some point, , you know, I won't speak for other of my colleagues, , and I'm not saying that we. Try to teach this mantra to our trainees. But you know, sometimes what comes to mind is, well, don't overthink it. you're a vascular surgeon.

You've explored a vascular injury. There's a hole in the artery or disrupted artery, do what we do, which is fix it. And, again, I don't mean to be, , overly simplistic. I'm not trying to be, , sarcastic, but I, I think we get to that point. Where you know you're going to have to fix it.

It would be very difficult to just not do anything. And I think, in the case that you described Kevin, I think that's important. You do need to know going into these, that the outcome's going to be likely to be stroke. You know, whether you make the stroke better or worse, you're not [00:43:00] really going to know by fixing the artery.

Um. And so the outcome's going to be pretty bad. , either way, I think that's important for family and the patients and, for the care team to recognize. these situations, but, to me fixing it as, unless Nicole, you bring up a good point. If the carotids occluded, then yeah, I think then that is a time when I would like eat it.

And I think this is where we fall back on our experience with carotid endarterectomy and managing, you know, a string sign that we thought was there from a carotid occlusive disease. And you explore the string sign. And by the way, the carotid was occluded. In those cases, in a setting of a carotid endarterectomy, we actually likely ligated.

, and so I think we fall back on some of the similar principles that we pursue and we're fixing age related carotid disease.

: Definitely, , and for completeness sake, there's another large. That's hole in the neck, , that we encounter injuries to, , what is your thought on, , internal jugular injuries? , can you like eight one, two, or

Yeah, I think, , I mean, you certainly can. [00:44:00] One can certainly, , like eight, these I think, and not have. consequential effects in most patients. internal carotid arteries that are, I'm sorry, the internal jugular veins that I have repaired have been in the situation of a significant TBI.

So a patient has, you know, , bleeding in the neck. You explore, explore the. the neck and the patient also has a penetrating wound to the head and intracranial hypertension and, you know, needed a decompressive craniectomy or something.

in those cases, I have repaired the internal jugular vein to maintain venous outflow of that side of the brain.

you know, I'm never sure whether or not that is necessary, but, you know, I think it, it makes sense to me. I think, . In one case, I remember before I repaired the internal jugular vein, you know, we transduced a venous pressure, , towards the cranial side, and the venous pressure was, you know, 40 or 50 millimeters of mercury, and there was a gradient, right?

Eight millimeters of [00:45:00] mercury on the, on the cardiac side, or the proximal or central side. So to me, if there's a gradient, and the patient's got a TBI, you know, I think fixing it is at least reasonable. but most of the time you can lie gate, the external jugular, , the internal juggler.

and there's enough collateral, venous drainage that, , the patients will do fine.

\*\*Okay. So going back to repair of the carotid artery and these, , trauma situations. So, you know, we fall back on the standard steps that we use in, you know, other types of vascular surgery. , the first thing being getting proximal and distal control. And then Dr. Rasmussen, can you speak to us about heparinization and how you manage that in the trauma setting?

Yeah. I think this is, I'm in this injury pattern more than, more than most or more than any other arterial injury pattern. You, you've, you have to use heparin in these cases. So, you know, it's a, , in some injury patterns, you kind of have to be ready for a wild ride. Because because you explore the neck for bleeding [00:46:00] in a, and if the patient has poly trauma, there's bleeding elsewhere.

and now you're going to fix the carotid artery, , in some manner. You're going to either put in a shunt or, or just repair it primarily, or you're going to have to, if you're going to clamp the carotid artery, , in my experience, you're going to need to use heparin. And so what I mean by a wild dried is, you know, you're.

Explore the patient for bleeding, they're already bleeding. And now what do you do? You give more heparin. the key there is to try to fix the carotid is, you know, as quickly as you can, , try to manage the, the other areas of bleeding while the patients have denies. and then, you know, bring them off of pepper and after the carotid is repaired.

I think unlike, for example, I think there's extremity arterial injuries that can sometimes be managed without heparin. But I think if you're going to go to the trouble of, , in the, in the technical, challenge of, fixing a traumatic carotid injury, I think in nearly [00:47:00] every case you got to give systemic, , heparin.

: so you know, you have proximal and distal control. Now the patients have pronounced, and how, how do you decide, whether to use a shunt or not and what type of shot do you use.

Yeah. So, you know, so the tenants are not, and so once you've done that, the tenants are similar to other Cherrelle injuries. you need to, identify the injury and then. Remove the burden of thrombus. Often that can be done with flushing, you know, back bleeding of the internal carotid or back blading and for bleeding.

And then, passage of a thrombectomy catheter, obviously passing a thrombectomy catheter up. The internal carotid needs to be done. you know, very carefully. It's usually a two or three Fogarty catheter and just a centimeter two. You really don't want to be putting a Fogarty catheter. Distill into the internal carotid more than a centimeter too.

Hopefully you can rely on back bleeding, you know, and back bleeding from that internal can quote unquote blow out any thrombus that's there. same with the proximal, but do [00:48:00] be aware that you need to get rid of the thrombus burden in those arteries before you fix them. whether or not to use a shunt, I mean, I think in most cases of trauma, um.

I, I have to say that I do not use a shunt, because it's, it's too, it adds just another level of complexity to a case that's already complex. and if I were to use a Shaunte, I would use either an Argyle, Sean's or a short son. Shant, S U N D T. , or the Argyle. Shawn's. I think it's reasonable to do that.

let's say you've got a long segment, the loss of the carotid artery, you're going to need to do an interposition graft, let's say. and, and in that case, you, you know, you're going to have to go harvest vein. you know, in those cases, I suppose, sure. It's, it's reasonable in those cases to put in a shunt to reproduce that hemisphere of the brain.

, while the conduit is, is harvested or prepared. so certainly there are times when I have used a shunt in the setting of trauma, but, I always also, I also try to keep in mind [00:49:00] too, in the case of carotid trauma, to try to keep it as simple as possible. Keep in mind, That the internal carotid can also be transposed over onto the external carotid artery as a transposition.

You know, in some cases, depending upon the precise location of the injury, so that, you know, you don't need an interposition graft. And in those cases, you know, you would not use a either. Um. So, you know, I think, yeah, be, be mindful to use the shot and be ready, sort of think about it, but also, if you find that fixing, it sets up pretty quickly, either with an interposition graft or with a carotid transmission transposing the internal onto the external.

If that sets up pretty quickly, , I do not think you should feel obligated to use a shunt in the setting of trauma.

\*\*Okay. Great. so. Options for repair dictated somewhat by the injury and the size of the injury. you mentioned harvesting vein. I think that's a good point, especially in cases where you're [00:50:00] exploring the neck and there's also an aerodigestive injury, in order to avoid putting prosthetic in the vein option is nice.

what if you have a small injury and you're just planning to do a patch?

So, For the patch, I would either use, , personally, I would use Dacron or, , bovine pericardium. You know, I think something, some prosthetic that's available, , you know, hemophilia Dacron patch, , or bovine pericardi as a patch. And I think, you know, you raise a really good point there that I think if it's, clearly a contaminated wound.

A penetrating wound where, you know, whether it's because there's an injury to the esophagus or the airway, or it's a shotgun wound, and so there's a large soft tissue injury or a fragment wound that's really contaminated. Then I think in those cases, yes, I think efforts to harvest vein sappiness Maine probably, are advised, um.

I will say that if it's the opposite of that, if it's sort of a simple penetrating wound, there's no evidence, at least overt evidence of aerodigestive injury. There's no really other contamination. Let's say it's a stab wound. , , you know, and it's not a big [00:51:00] soft tissue all quickly, , also use, , , Dacron or PTFE and, or position graft and not use the, as I mentioned before, I think, um.

You know, you try to be mindful of keeping these operations, you know, as, as, , expeditious as possible. You try to move them along. And, in the case where there's not a lot of contamination, or any contamination, I also will then not use me. You know, I think, , , using a Dacron interposition graft or PTFE in this larger vessel, these common carotid is, .

\*\*Okay, great. Then I think one important point also to mention is that you need to debris back any devitalized tissue. so you know, if there's a defect in the artery, but the tissue around it is pretty ratty. You know, you may have to actually debried it back and make the defect larger initially before you repair it.

Really good point. Yeah. This ma'am. And the same with us, Sean. That's why. You know, I said, be ready to use a shot. [00:52:00] Certainly in some situations you need to use a shun, but you know, using the shot, you can also, , either create a concrete, you know, an injury or cause you to need to debris the artery back a little further.

so really good point.

: , start, you're asking some, one point that I picked up from reading your chapter here is that, , we're talking about back bleeding from the internal carotid and it made the point of back bleeding is restored. , you should perform an interrupt angiogram. To document complete evacuation of distal thrombus.

can you just speak to this.

Yeah. I think, a completion study, um. is is definitely, , something to, consider or to perform. Now, I think we've used the, we've, we've used the phrase during our discussion here this afternoon, , a luxury. You know, there's some of these

things that you have the luxury to do. You know, in some cases of trauma, you, may not have the luxury to perform.

an on the table. Arteriogram so, in those cases, I would use duplex, , if you have duplex or even, use , the, [00:53:00] duplex function of, the ultrasound that you have in the, or to show flow. you know, and then the, the most simple thing is a continuous wave Doppler.

And so some form of assessment of your repair certainly is indicated. the complexity of that completion exam depends on what. Capabilities you have and how the patient's doing. so certainly in an ideal situation, you're fully outfitted. You've got, , experienced colleague or partner or fellow with you, and, you know, the patient is on the right imaging table and you can do a completion arteriogram then absolutely.

I would, , put a, 18 gauge butterfly in the common carotid artery. Make sure it's. The aired so that you don't inject air and shoot a completion arteriogram, , both the repair and the distal internal carotid to assure that there's no thrombus in situations where you're not that outfitted and you don't have that luxury then using a duplex ultrasound.

I think is, [00:54:00] is fine. And in those cases, you don't have duplex or the patients just, . , has enough injuries that are in his, in a complicated enough situation using a continuous wave Doppler and, and, and just, finishing up is also

\*\*I'm curious. Dr. Rasmussen, do you use a completion imaging study for elective carotid repairs?

At this point? yeah, we use, , ultrasound. We use duplex ultrasound, but not arteriography. I mean, I think that the arteriography point is, , is useful. I appreciate that. And, , I think, for those who have done that before and are used to doing it, it's reasonable. The, the counter to that would be, well, let's say you see a little bit of thrombus. in the distal internal, beyond your room repair, what are you going to do about it? so it, it's not to make light of or diminish the need for, confirming a technically adequate or perfect repair. but I think, [00:55:00] to some of my previous comments, you know, we as surgeons, I think, try to strike this balance between, I mean, um.

You know, being perfect and then spending too much time overthinking it and, , carotid angiography completion angiography in the setting of trauma may just not be feasible in a lot of cases. or people may not have the experience in doing it.

\*\*Right. Okay, so what if there's a concomitant tracheal or esophageal injury? Do you do anything

Well, I mean, I think we, we didn't mention the word, we didn't mention drains. So, you know, in all of these cases where we've explored his own to penetrate a

neck injury, those patients are all drained. , with, , you know, for me, , , a seven millimeter flat JP drain or, or maybe more, I guess that's a great question.

I don't know that there's a situation where I really buttressed , or done used any adjuncts [00:56:00] to cover my repair. the vascularity of the neck. um. is good and if not, great. And I think that helps. I think if you've drained the, , the, , the wound that is also sort of a mitigating step.

And I'm not sure that I have buttressed, , or done anything, more than that to protect the arterial repair. And maybe it's because I've not had any that have had. overt esophageal injury. They may be, you know, micro injury or something like that. Small. do you think, Nicole?

\*\*I've heard of using a muscle flap to separate the. Esophageal injury from the arterial injury, but I'm not sure if that's always necessary. And I do like the idea of the drain, because that would prevent any

collection from building up there that could compromise your arterial repair.

I think that those are both. Good points. I think the drain is in my mind, the drains of mosque. Even if you explore the zone too. Yeah. You still know, [00:57:00] because remember many of this opera deal injury, you just not going to see, and honestly. I don't know that any of us are familiar enough with exposing the esophagus, , through that wound, we, we may sort of see the esophagus, and do the best we can, but many of this off the GL injuries will be pretty small, or we may not see them.

, so I think that, , the drain is a must, , in these injuries, at least one and a muscle flap, I guess is something to consider if there's gross contamination. You know, either spillage from a softened geo wound or they're just gross contamination, the wound.

EAST Blunt Cerebrovascular Injury Guidelines

<https://www.east.org/education/practice-management-guidelines/blunt-cerebrovascular-injury>

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## 10.5 Endovascular Approaches

**09 Feb 2021:** *Kevin Kniery, MD, MPH; Marlin "Wayne" Causey MD; Todd Rasmussen, MD*

**Kevin Kniery:** [00:00:00] Welcome back to behind the knife and audible bleedings vascular trauma podcast series with Dr. Todd Rasmussen. Today are in our final episode. We're going to discuss the endovascular management of acute

vascular injury specifically. We're going to focus on. Thoracic , , blunt aortic injury and endovascular treatment options for EXITO subclavian injuries.

Um, today we're lucky enough to have Dr. Todd Rasmussen. He's a Colonel United States air force and his professor of surgery and an associate Dean of research at the uniform services. University of health sciences is an attending vascular surgeon at Walter Reed national military medical center. Welcome back Dr.

Rasmussen.

**Todd Rasmussen:** Thanks very much. , Kevin for the opportunity I look forward to. A good discussion on a, on a, I think a really important topic. Thank you. Great.

**Kevin Kniery:** [00:01:00] And , , we also have Dr. Wayne Kasi. He's the chief of vascular surgery at San Antonio military medical center at a level one trauma center in San Antonio, Texas.

And he's an associate professor of surgery at the uniformed services, university of health sciences. So a welcome,

**Wayne Causey:** thanks, Kevin.

**Kevin Kniery:** All right , well, let's just dive in. We're going to focus first on the blunt, thoracic aortic injury and the endovascular management of that. So I think it's important to understand.

how this occurs and what patients is occurs in. So blunt, thoracic Edric injury is rare, but it's very lethal. It's less than 1% of all blunt traumas, but it's the second leading cause of death in blunt trauma.

**Wayne Causey:** Yes, Kevin, that's why whenever I'm called about a patient who has a blunt aortic injury.

I really want to know what their other injuries are because these are usually very high velocity mechanism injuries, and there's a particular concern that they'll have an intracranial injury.

**Kevin Kniery:** Right. So, you know, when you're the trauma resident , , taking pages, you know, the things you want to think about, that'll clue you into , , you know, we need to make sure they don't have this as a high-speed motor [00:02:00] vehicle collisions, motorcycle crashes vehicle versus pedestrians and falls from Heights are some of the most common causes of blunt, thoracic aortic injury.

Yeah, I

think

**Wayne Causey:** it's most vascular surgeons have seen. It's pretty amazing. Now the types of injuries people can survive. I mean, I've seen people in cars who fallen over overpass, fallen off of overpasses being hit by trains, the

prehospital management and the vehicle safety features that exist now have really expanded what survivable.

**Kevin Kniery:** Yeah. And , um, yeah. Quickly, we're going to just kind of review some of the anatomy of where these injuries occur is. Um, so the aortic arch itself is relatively mobile. It doesn't have a lot of tethering, but the descending thoracic aorta itself is actually kind of tethered. And so , , it's it's at that junction just distal , , to the.

Subclavian artery , um, is where these injuries generally occur. Most of them curved within about a half centimeter to two centimeters of the left subclavian artery. And so it's at that junction that , , that the aorta tears due to the deceleration. Um, so Wayne, do you have any particular thoughts on the diagnostic methods for these?[00:03:00]

**Wayne Causey:** Well, I think most trauma centers have a pretty good protocol, such as the level one trauma center we work at. Typically the patients who come in with a really high velocity mechanism, injury, get a CT angiogram of the chest, abdomen and pelvis. And oftentimes if there's an extremity that's injured as well, that'll just be carried down through the , , lower extremities as well, or the upper extremities.

**Kevin Kniery:** Yeah. Great. So yeah, CTA , um, is critical in these patients , um, to help , , diagnose this. And so it's important to realize that , , patients with these injuries, the majority of them greater than 75% never make it to the hospital. Um, and those that do make it, the hospital are likely going to be severely injured and in multiple other ways.

And it's not gonna be an isolated injury. Um, so quickly, we're just gonna go over the SVS grading system. There's a couple of ways to grade these. The current one in the SVS is , , has four grades. The first one is a grade one, intimal tear. Grade two is an intramural hematoma. Grade three is a pseudo aneurysm.

So there's an actual contour of the aorta is [00:04:00] disrupted. And , , and then grade four is what they consider rupture. So those are the four SVS grading systems and , um, When do you want to talk a little bit about how Dr. Starnes kind of breaks it down? Yes,

**Wayne Causey:** Kevin that's the official grading system. I remember when behind the knife first started , , one of the first or second episodes was an interview with Dr.

Ben Starnes. And I remember listening to his blunt, thoracic aortic injury recommendations on the way to the hospital, as I would consult on one of these blunt, thoracic aortic injuries. And he really categorize these into three different categories based on his Harbor view data. , that was a minimal aortic injury , , which had a 10 millimeter or less intimal tear, a moderate, which had a pseudo aneurysm.

And then the maximal, which I think he summed up very well and saying that there was a unilateral, hemothorax active contrast. Extravasation a periodic hematoma greater than two centimeters in maximum width. And those were the patients that needed to go to the operating room, basically at the time of the consult.

**Kevin Kniery:** Yeah. Yeah, that's a great point. So the, [00:05:00] the Dr. Star and slash Harbor view categorization is really just three categories and they kind of depend on when you operate. So the minimal ones you don't operate on. Those are just the intimal tears, the moderate ones. They need an operation, but not emergently.

And the Maximo ones need a, an emergent operation. And so I think that was part of his kind of reasoning for it. One of the things

**Wayne Causey:** that. Struck me with that podcast on behind the knife was he mentioned that what's the point of having a grading system that. Doesn't have a treatment with each different grade.

And so as Kevin goes through these, you'll see that there's a little bit of overlap between the two grades. And the point of that previous podcast was if you're going to have three treatments, then you need to, which is imaging or surveillance , , delayed repair, and then urgent, emergent, or urgent repair.

Then there needs to be three different grading scores,

**Todd Rasmussen:** right? Yeah. Yeah. I think, I think Dr. Starnes in that , , the grading system, you mentioned, I always find it to be sort of [00:06:00] practical,, , it's very practical, , for, for us., the, the four category classification is, is a little bit more technical and maybe a little less practical, but , um, yeah, I agree.

I think it's , , That's true. You know, the other thing before we get too far into this, I do think it's useful for your listeners., , Kevin T there, there's a couple of classic, , um, you know, papers , , on. Blunt aortic injury and , and, and they really speak to, for those who are interested in vascular trauma, interested in this condition, I would ch I would encourage you to dig into just two or three papers.

One of them is the original double AST paper led by Dr. Dimitrios and the team at LA County in the late 1990s. Um, on blunt aortic injury, which not surprisingly in the late 1990s, that double AST, what we refer to as the WST one study showed that nearly all a blenny already injuries were fixed with an open thoracotomy and clamping and sewing into graft.

Um, the WST [00:07:00] repeated that study about 10 years later and in 2006 , , or so I think it was actually published in 2008 , um, where they , , double it's called the double AST two study. So in the journal, both of these were in the journal of trauma at the time. Now it's the journal of trauma and acute care surgery.

Again, led by Dr. Dimitrios says,, and, and in that 10 year period, the management of blood aortic injury had completely. , evolved from open repair to end a repair. And we won't go into the details of those studies, but , um, you know, and then you go three or four years later, and now there's the SBS that's writing these guidelines.

So which you've seen in those three papers, the WST one in 1998 or nine was T2. In 2008, both in the journal of trauma. And now the leading organization writing the guidelines is the SBS and not the WST. What you see there is this evolution of, of the management of this, , this, injury pattern from open, you know, to [00:08:00] nearly exclusively endo now.

**Kevin Kniery:** Yeah, that is a fascinating evolution. Um, and very interesting. I, I've heard of those papers, but I didn't realize the transition from the, the trauma journal to the vascular journal for the kind of guidelines on it. Um,

**Todd Rasmussen:** surprisingly again , um, at the risk of you guys not inviting me back, I don't mean to monopolize the conversation, but , um, you know, the thing that's happened with that evolution is the mortality, the survival of, of operating on these, these injuries has improved.

Rates of paraplegia have decreased blood utilization is decreased. You know , patients, patients have a better chance in 2020 than they did in 2000, you know, and it's largely because of the evolution to endo , , technologies that we've. We've mentioned, and I

**Kevin Kniery:** bet there's not a whole lot of other specific things in trauma that have had that big of a change or improvement in outcomes in that amount of time.

So it's pretty, it's actually pretty special kind of a pathology and improvement there.

**Todd Rasmussen:** Yes, sir. I [00:09:00] encourage one of your listeners to write that up. It'd be a great review. , you know, two decade review , , two and a half decade review of, of just the broad strokes, how that practice has changed and how paraplegia rates are down on mortality rates.

It's still a highly lethal injury, but if patients make it to the hospital, as you were referring to , um, modern grading, modern management techniques, give them a much better chance.

**Kevin Kniery:** Absolutely. And I, and I'll include all those papers in the show notes. So you can just go click the links in the show notes here.

Um, we're going to get back into the kind of management of this. Um, there's some things that all of these patients are going to need, whether they're going to get surgery or not. Um, you really want to focus on anti impulse therapy. And generally you can do it with a beta blockade and a short acting beta blocker is the best.

Um, so that's going to be as small as generally the, the BA the beta blocker of choice. And of course you have to talk with your neurosurgery colleagues. Um, and we're gonna discuss a little bit this more lately, but. Um, you know, generally want the blood pressure less than 120 systolic. Um, it's, there's no guidelines on that, but it's just sort of consensus of, of surgeons.

But, you know, [00:10:00] obviously it depends on their head injury and other what other issues they have going on. Um, and generally , , As far as, you know, managing this small tears, the less than 10 millimeter ones , , the guidelines recommend a CTA within 30 days. If you have a larger flap, greater than 10 millimeters, this is something you're going to image sooner.

, generally within seven days. And as Dr. Kazi pointed out the other day is that many of these patients are getting rescanned for lots of different things. Reasons, whether it's their blood organ , , solid organ injuries and other things, and really kind of try and work it into one of those or kind of fit the imaging , um, to help them out.

And then , um, if it's an in, we're gonna talk a little more about this in detail, but if there is an external aortic contour abnormality, generally, you're going to repair that within less than one week. And then of course, if the patient is hypotensive and has a hematoma , um, concerning for a rupture, this should be repaired urgently.

**Wayne Causey:** One of the things that comes up often I find is that. Patients who ha who need to have their blood pressure elevated for one reason or another such as a traumatic brain injury. And if you go back on the behind the knife conversation , um, a couple of years ago, this [00:11:00] was really brought to life as if they need to ramp up the blood pressure to treat intracranial pathology.

Oftentimes that's going to require a very urgent repair , um, so that, that blood pressure can be brought up because you will not be able to do that anti impulse therapy.

**Kevin Knieriy:** Dark there. Are you saying on the, if there is a grade one injury , um, per the say either, either guidelines, the Starnes or the SPS guidelines , , that you would this, if, and they needed a high blood pressure for their brain, you'd potentially , , repair it with a T VAR.

, even though there is no pseudo aneurysm.

**Wayne Causey:** That's a great question. I think that Dr. Starnes covered that a little bit and I'm not gonna speak too much on what he said, but he found that the intimal terrors at 30 days in that podcast, that they were not going to need to be re-imaged or repaired at all.

And so he put each of those patients on 325 milligrams of aspirin for 30 days and said in that podcast that he didn't Ramage them. So in that particular case, no, I would not repair an intimal. Tear. Um, but if it was a [00:12:00]

significant other type of aortic injury, I think I would lean towards repairing that.

What are your thoughts, Dr. Rasmussen?

**Todd Rasmussen:** Yeah , I, I agree. I think that , um, you know, the, sort of the grade ones that, , the, the , um, Seattle group has described,, I, I, I don't think that they , um, You know, parsed out the patients who might, for example, need a higher cerebral profusion pressure. And therefore they, they're not as amenable to , , you know, anti impulse therapy or hypotensive, resuscitation, whatever.

I don't think they really parse that out, but I don't. I also don't. I also don't think that they, you know, they th th that they recommend or others around the country recommend, you know , um, Fixing grade one injuries in, in those patients. I think that they're, if they're grade one , Um, they, you know , those, those generally don't need to be treated.

And , , now, I mean, I think there's always room as in all of these complex situations for,, for, for another CT, you know , for, for a followup CT to see, I mean, if , , if a provider's really nervous about it and , , you know, the. The neurosurgeons have had to drive up the pressure for the [00:13:00] TBI or, , to, to maintain, , , CPP then, then , um, you know, you can always image them again to confirm that.

But , , I agree. I think if, if they're a grade one injury , um, right now they, they, you know, non non-operative therapies is safe and indicated.

**Kevin Kniery:** Great. So let's talk a little bit more about the nitty gritty of actually repairing these things. It's important for , , residents out there to understand , , the T-bar devices that are currently in the market.

Um, these are generally designed for aneurysmal disease and not for the trauma population. , the Trump populations going to be smaller. They're going to be , um, Younger and healthier vessels and it's , um, you know, these devices weren't really made. So the devices that we currently have, they are, you know, generally require large diameter , , access.

Their compliance is not , , as ideal for these kinds of , , smaller aortic arches. And the actual size of the devices are relatively large compared to these aortas. Um, the average size of the aorta proximal to the injury in a study was shown to be about 19 millimeters. And our current smallest [00:14:00] device is about 22 millimeters, which is, you know, an adequate oversizing, but you wouldn't, you know, that's , , only, you know, wouldn't want much bigger than that.

So , , Dr. W what is your experience with the current generation of devices?

**Wayne Causey:** Yeah, I think the good news for a lot of these devices is that the technology seems to be advancing. To where arch confirmation is starting to have a lot of technological advances so that the young patient with the steep aortic arch will better be treated in the coming decade.

Dr. Rasmussen, this always brings up an interesting question in regards to, so the sizing of an Intergraph, so in a patient's injured and they're in the trauma Bay and they get their CT angiogram. Oftentimes they're under resuscitated. How do you go about assessing what you think is the proper size of the order and choosing your endograft.

**Todd Rasmussen:** So I think , um, it's a great question. And , um, one of the things just to add to what you've, what you've said , , Wayne is that , um, and it really, I think fits with our , um, the, you know, the line of our discussion about the evolution of, , of, of, not of this injury pattern. Now our ability to [00:15:00] have classifications that are pretty technical , , with degree of injury.

then the evolution of. Of management techniques is now the evolution of these stent graphs. And, you know , um, one of the, I think things to keep in mind is that. TEVAR for blunt threat , um, thoracic endovascular, aneurysm repair T VAR extent graft , , for blunt aortic injury, you know, a decade ago was done with really very clunky endograft technology and none of that technology.

Let's say for the, for example, the WST two study that I cited , , the , the, the endovascular repairs in that study were often , , you know, iliac limbs , , for the, you know, not really designed for, for blunt aortic injury. Now, fast forward to 2020. And we have, for example , , one of the devices that is made by Gore is actually has an indication , , for , , an aortic transduction.

You know, and so now , , you know , the, the technology , , is really evolved now to, to suit the, you know, the condition, which is sort of exciting. And I think gives us as vascular [00:16:00] surgeons and, and gives patients a lot better options. Um, maybe we can go over some of the specific devices , , next, your point, or your question about , um, you know, imaging is, I think is, is.

Is an important one for listeners to understand. And I think w just to be clear, what we're talking about is is that if , , if , , Patient's aorta is imaged in the cat scanner when his or her systolic blood pressure is 90 and they're relatively hypovolemic then, you know , is, is the , , how accurate is it has a diameter of 18 millimeters, for example.

And is that a vasoconstrictive or just a hypovolemic , , aorta. And I think it is, I think that it is, it is, it is not the greatest , , measure and that providers should To keep that in mind that when patients are resuscitated, that the diameter of the aorta is actually going to be larger. So usually what I would recommend is to , um, you know, still oversize, , the, the stent graft by, you know, 20% or so based on your original measurement , , again and here , , unlike aneurysm disease [00:17:00] where you know, you're trying to.

I think get a seal and it really diseased , um, and dilated aorta here. , you know, the seals zone in, in the, in the pathology is, is almost always much more focal. You're really just trying to cover and seal the proximal intimal tear. Um, and so I think that, you know, oversizing by roughly 20% , um, Using intravascular ultrasound or IVUS, I think is also useful.

Sometimes when you get them, the patient to the, or for the TVR, for the thoracic endovascular repair, using intravascular ultrasound real time to confirm or refute your original , um, you know, your original CT scan is. Is , , is also useful. Um, but I think keeping in mind the blood pressure and their volume status when the scan was taken oversizing by somewhere around 20 to 25% and then using IVIS.

So I think are useful tips. What do you think, or how do you guys do it in San Antonio? Oh, that's a

great

**Wayne Causey:** point. I mean , we, we set up IVUS for every single aortic case so that it can be there. It's mounted to the system and. [00:18:00] Even if we don't use it for the particular case, we know that everything's there and available for us to use it.

If we need to re image or resize and aorta or some other blood vessels so that it can be adequately sized. I remember one time we had a 17 year old with a blunt traumatic aortic injury and he needed an emergent repair and we didn't have the smallest endograft at the hospital, but fortunately there was a one from a local hospital that could be sent over, but.

That was a little bit of a troubleshooting. I was, what is, what was I going to do as far as putting a larger endograft into a relatively smaller we're to knowing exactly what we talked about that open repair has much worse outcomes than an endovascular repair. And I think my, the way I was going to approach that one was I was going to use the IVIS after I deployed the endograft to make sure I didn't have a retrograde dissection , , or something , , some major complication related to the procedure.

**Todd Rasmussen:** Yeah, I think, I think that also , , Wayne speaks to the,, um, the,, in, in some ways, and I, I don't want this [00:19:00] to be not, I don't want to overstate this, but in some ways this condition is , um, although the overall patient complexity can be a much greater, some ways, just the aortic. Pathology is simpler than, than a complex dilated aortic aneurysm and in an elderly patient.

And, and I think that makes it more amenable to,, , to , um, you know, sort of belts and suspenders repair, meaning that you just really need to seal the, the entry point. I mean, again, it's not, I'm not trying to oversimplify it because it is serious. And these certainly , , some of these, a grade three or four by imaging can certainly rupture and cause mortality.

But, but even in the WST two trial in 2000, Eight that was published in the journal of trauma, the success and the, in the unmistakable , , advantage of endo repair over open. That was all using very primitive, endo materials, you know, as I mentioned , so, you know, I think,, , it's really an exciting part of vascular , , practice muscular [00:20:00] trauma is, is the evolution of , , of these technologies.

But , , I appreciate, and I agree with, and it definitely used them. Belts and suspenders sorts of endograft that you got. It's not exactly the one you want. Um, but you use what you can and just try to seal the, the proximal portion of the intimal tear.

**Kevin Kniery:** Yeah. And , , you know, there, there's a lot of technical aspects of these and , , kind of , , you can miss , um, the zone you can deploy to proximal, to distal.

One of the issues that comes up and you see sort of in a case reports of , , you know, malpositioned , , devices is what they call bird beaking and where the , um, the lesser curve, the graft isn't. Approximated well , um, to the lesser curve of the aorta and a doctor asked me, said, I wonder if you had any tips for our listeners , um, on how to avoid a bird beak.

**Todd Rasmussen:** Yeah. Great question. A couple of things. Um, so I wish we could , , illustrate this , , for your listeners. Um, you did a nice job bird beaking to me and , , you know, you guys jump in or correct me if you've got a different way to articulate it. On just [00:21:00] a call. We don't have a whiteboard , um, bird beaking, as you said, is the, is sort of the lifting of the inferior aspect of the very proximal.

, endograft where the fabric is. That that, that ring, if you will, of stent covered stent lifts up off of the inner curve of the aortic arch. And if it lifts up, then it's prone to get an, a, you know, the jet of flow and pressure that then, you know, would cause an endo leak. And so to get that first covered stent ring to lay down , , so that it doesn't lift up and cause this, I guess the bird beat comes, cause it sort of has this appearance of a.

I'm a triangle , , on the, on the imaging, I guess that's where bird big comes from. Um, to get that, to lay down a couple of things, one , um, I think getting a stiff wire , um, , , around the outer edge of the art. So using an Amplatz , , or a lender, Quist wire, that's really , um, all the way into the aortic root, the ascending aorta and actually [00:22:00] pushing it.

Um, so that it's got sort of a constant. Tension that , um, allows you to get the endograft forward and, you know, making sure that you have a stiff wire and platform. Is one, you know , that's, that's a basic principle of endograft deployment anyway, independent of the bird beak. But I think that helps. And then I think another is to consider if there's, if it's close or if it's an angulated arch to consider taking the endograft over, across the origin of the left subclavian artery , um, to where the , um, You know, to where the there's more room, if you will, and a flatter inner arch or inferior aspect of that curvature to lay the graph down.

So covering the left subclavian, using a stiff wire with forward pressure on that wire during positioning and deployment. And then the third thing, you know, some of the, the Gore , , conformable , , Thoracic Intergraph now actually has a kind of a ratchet on it that, that allows one to have some control of that first

stent so that you can actually kind of conform or lay it down.

Um, I think that that's real. Um, [00:23:00] I'm not quite sure how effective, I don't know what, you know, Wayne, what you think about the conformable Gore graft, but I think that maybe another advantage that comes as graphs that you can actually. Manipulate them when they're in place and try to lay down that first , , covered stent component.

**Wayne Causey:** Right? I, I think that's ideal. I mean, we have recently. Switched all our inventory. So we let the old stuff, you know, use it or, or expire. And then everything that we purchased now for blunt, thoracic aortic injury is conformable. And I like to use the double curve Lundquist. And just like you said, I. I have the assistant , , whoever's holding the wire actually put forward pressure.

So that, that wire is really snugged against the greater curvature of the order aorta. And also that the double curve lender request is basically sitting at the , , at the heart so that it can really give me as much tension as I can. And I think that bringing it across less of cleaving, if you think it's going to be bird beak is, [00:24:00] is.

Absolutely essential that if you need to cover it so that you don't have it sitting oddly on the inner curvature, that that's ideal. And I agree with you a hundred percent.

**Kevin Kniery:** Yeah. So , , another kind of issue , , that we can sometimes run into in our trauma patients is getting the devices there. Um, some of the iliac arteries are , , relatively small.

Um, you know, granted the devices are getting smaller themselves, but Dr. Rasmussen, do you have a size , , that you would consider doing an Elliot conduit , , for these graphs? Is there kind of a rule of thumb with our current generation?

**Todd Rasmussen:** That's a great idea. A great question. A great idea. Sometimes to do it in Elliot conduit, if you need it.

I think , um, Boy, I hate to , , to wimp out and just say it depends. Um, so I would say that, you know, if , if, if the iliac , um, but it does, but it does depend , um, you know, it depends on the age of the patient. I mean, if they're calcium free , um, you know, it's a young patient, like many or most of our trauma patients , um, and the iliac is, you know , um, Seven millimeters or greater.

Um, and it's sort of a straight shot we'll give it a try. seven. [00:25:00] To eight millimeters is sort of on the small side of this. Um, but I will often, but if it's calcium free in, in a young patient, then, you know, w w we'll try it through the, through , , through the femoral without a conduit.

Um, I think that if gets smaller than that, if it's a female and they're hypotensive, when the images come through and you, you know, you're ultrasounding them in the, or using duplex to size up their common femoral and you, you know, whether it's shock or just they're native, they're just small. If it gets to be,

you know, less than six or seven millimeters, then I think , , You know , , exposing either the common femoral or the external iliac through retroperitoneal approach and putting on a Dacron conduit is it's easy enough , , and should be done.

Um, So it depends, but you know, the flip side, I guess , , Kevin is that if it's , , you know, if there's a lot of calcium and it's an elderly patient and it looks like there's atherosclerosis, then I have a lower threshold to put on a conduit because you know, you could cause problems. It's not as accommodating of a passage of the sheet.

If they're, if they're calcified or [00:26:00] highly

**Wayne Causey:** ambulant, Yeah, I think if it measures seven on the seat, the angiogram and the trauma Bay, that it's likely, like you said, if it's not calcified and not diseased and not injured, otherwise it's, it's a good chance that you're gonna be able to deliver that endograft without any difficulty.

Um, you may, I sometimes consider, I use do a most percutaneously, but cut down on the femoral artery, just like you said, just to make sure that it's delivered safely. And if there's a problem with removing it, that I'm ready to fix it.

**Todd Rasmussen:** I think that's a great point. If it's a small one and you feel like you're kind of right on that edge, then I have a low threshold to just cut down.

And that way, you know, you afford yourself, I think a little bit , um, more assured sort of placement sure. Placement with an open, which is also relatively easy or should be , to, to do it open the other. The other thing, I don't know if you've used, I sometimes will oil the sheets with mineral oil, you know, I'll put it on the.

On the , , I mean, you know, the other question here we haven't really talked about is heparin heparin eyes or not. We can get to that, but sometimes I'll get some mineral oil on the back, you know, and I'll just cover the sheath. If you think [00:27:00] it's going to be tight and sort of make that , um, make the sheath as you're passing it from the femoral up the, the small externally, like into the end of the AOR to make it slick, if you will.

**Kevin Kniery:** Yeah, you , , are reading our minds, sir. So I was actually going to bring this question to Dr. Kazi. Um, many of these patients are multi injured as we've discussed and have , , head injuries, maybe even head bleeds. Um, do you recognize in all of these cases, is there cases where you won't

**Wayne Causey:** have her nice? Well, it's interesting in our group, we have a variation of practice patterns.

, I guess number one is if the neurosurgeon or someone. Completely contraindicate you to using heparin. And then you're a little bit in a bind. I haven't really found that to be largely the case, but it does come up every now and then I

personally like to Hep denies, even if it's just a little bit of heparin , um, 3000 of heparin, just to , , allow me to have the optimal device, but I don't think it's necessary.

One of the things that you and I discussed recently because we repaired A symptomatic AAA three days ago on a COVID positive patient. Kevin was, we were concerned about the thrombosis [00:28:00] with the large sheets. Um, Dr. Rassman, what do you think with the COVID patients , um, and heparin in general , , do you, do you have denies all of your T VARs and what ha what if you had a COVID positive patient?

**Todd Rasmussen:** That's a great question. I mean, I think , um, You know , I, I remember , um, my first sort of TVRS for blunt aortic injury when I was in San Antonio and I was amazed at , um, how. Um, if the patient's condition allows it , , and I think most of them do , , operative planning. So imaging , , trying to get some resuscitation, maybe even, you know, a 12 to 24 hour delay and the repair.

If you get everything lined up and it's a straightforward , , repair , um, how you, you can really get these stent grafts in and. Sort of 30 minutes or less. Um, I don't, I don't want to exaggerate or tell a fish story here. So I , , you know, I know it's never as quick as we surgeons think it is, but the point is if you can get it done quickly and the patient has other , , contraindications, a head bleed, solid organ injury, you know, big hematoma and a fractured femur or [00:29:00] something, then I think they can be done safely without heparin.

Um, and I think , um, one just needs to be mindful of the time. Um, and then really flushing out the femoral and then assessing the access side or site , , when you're done. Um, so I think they can be done safely without heparin. Um, now the, the flip side is if the patient has no solid organ injury, TBI hematoma, and the thigh from a femur fracture , then, then proceeding with the use of heparin is it would be preferred.

You know, I think that's always preferred. Um, so I think, you know, I guess what I would say is I would prefer to use heparin. I do in all the cases that I can , , but I don't sweat it too much if I can't, because of other conditions, the patient has other injuries. I just am more attentive to the time my sheets are in.

And then to what's happened. What's the status of the Elio femoral segment. After I've removed the sheets. I think in COVID positive patients, I think they've shown [00:30:00] themselves to be relatively hypercoagulable and I would , , I would want to I'd, you know, I I'd have a more , um, you know , , th I think the imperative to anticoagulate coagulate, those patients would be higher because of their , , shown propensity to develop , , thrombus.

**Wayne Causey:** Yeah, that's exactly what Kevin and I did with that endo AAA patient is we pepper to them more than we normally would. And then followed act pretty closely to make sure they stayed over two 50,

**Todd Rasmussen:** a really good point. And we didn't really mention it, but the act, the activated clotting time, I think, you know, we talked about operative planning and, you know, for your listeners, I think.

You know, we tell our trainees the best, the best resident, the best surgeons are pessimists. You know, it sounds bad. I don't want to be a downer before the holidays, but you have to anticipate things going wrong and never be the optimist. Right., if I have a major injury or condition, I want my doctor to be worried that everything's going to go wrong.

, I'm exaggerating a little bit to make the point. The part of that is the act machine. Like how many times have we been in the employment? And we need an activated clotting time because we really, whether it's for carotid or endarterectomy or this type of an [00:31:00] injury, and you know, the, our staff, our anesthesia colleagues, not necessarily to a fault of theirs, they'll say, Oh, we don't basically team.

Machine's not working. Or we don't have it or something. Right. So part of the pre-op planning for, for house officers is, you know, among all the other things is where's the act machine. And could we draw a test act before the case starts because Dr. Kazi really needs the act on this one. And , um, the last time you, you know, the worst time to hear , well, the act machine is not available.

It doesn't work. Cause when you've got a, you know, an 18 French sheath up a small iliac and an undergrad in the order, So I agree with the act and , , I don't want to sound too much like a pessimist. Maybe I'm just a realist , um, that, you know, putting that on the pre-op timeout, for example, if you're going to do team steps or time out , , make sure that you're you're you got that act lined up.

**Kevin Kniery:** Yeah, absolutely. , so there's two kind of main points we want to cover here , , to wrap up , topic. And one of them is the subclavian artery. And as we discussed before, the average distance between the injury and [00:32:00] a subclavian is about 5.8 millimeters. And most of these devices recommend a two centimeter coverage and healthy vessel.

Um, and then two centimeters distal in order to adequate coverage. so. And , and, and using those numbers, the registry suggests about 40% of patients have their subclavians covered in these cases. So, Dr. Rasmussen, what are your thoughts on covering subclavian artery? Do you , um, do anything to revascularize it AF if you do , , cover it and any, any thoughts you have on this?

**Todd Rasmussen:** Yeah. Great question. this also has evolved , , and I think can be institution specific. Although I think most institutions now have gone to not. „ um, preemptively, revascularizing the subclavian. So maybe for, if we take a step back , , I don't want to take too much time, but I think for your listeners, what we're talking about here is , is, is covering with that covered stent.

So you've got to get that covered stent graft up over. In into the distal part

of the aortic arch to seal to [00:33:00] get a good two centimeter length of seal before that aortic tear that happened most commonly at the aortic isthmus. Right. And so sometimes that requires covering the left subclavian artery. And while that seems crazy, most patients do just fine with that.

Actually they may have a little bit of arm claudication, but do just fine. And what that allows us to do is to then get that two centimeters of seal. , where the stent graft is really well. Well, opposed to the inner curve of the aortic arch, if you will, , or, or the outer curve., the, the things that I think about, I think , , where's the vertebral artery.

So the, so the risks of covering the left side, Clarion, I mentioned arm , , arm claudication , um, or Ms. Schema, so real close attention to , , and then another complication could be stroke. Right. So that , um, if , if, if the patient has, , , the dominant vertebral artery is on the left and it comes off the left subclavian, and for some reason they don't have a right to vertebral, then you really need to revasc you've got to, you can't cover the left subclavian without [00:34:00] revascularizing.

Um, you know , um, that subclavian and the verge , um, so paying attention to where the w what the, the dominant vert is, where it's from, what is the right vertebral artery look like? Is there atherosclerotic disease in the arch and the great vessels to include the birds? And then the third main complication is spinal cord ischemia , um, and weirdly , , you know, for your listeners, you think , well, how can covering the left subclavian lead to spinal cord ischemia?

Um, and if you. Think back to how our spinal cord is perfused. Um, it is rare in a very it's uncommon , but, but there are collateral pathways through the, through the Ima, the internal mammarys and to, through other,, , collateral circulations that originate off the subclavian. Whereas subclavian profusion can be important to perfuse the spine.

So, you know, Making sure that you have those three things in mind, left arm, claudication, posterior circulation, stroke, spinal cord ischemia. And as long as you have those front and center , um, you know, most of the time you can still cover that left subclavian [00:35:00] without. Revascularizing it. And you will be cognizant of the situations where you do need to revascularize it either before you do the T bar or at the same time.

Um, or sometimes afterwards.

**Wayne Causey:** Yeah, this is always an interesting situation, particularly some more rare scenarios that often comes up as well as , , as you said, the right for T-ball already may be occluded. There's a dialysis access and the left arm or the one that concerns me the most is if there's a Lima to an LA , , I don't, I agree. I agree.

I don't think there's an easy answer on these. , we have , , laser finished rated a few endograft for these rare cases with pretty good result. And I do think that

there's some graphs on the horizon. That's going to allow us to preserve that left subclavian artery , um, in the

**Todd Rasmussen:** future. Really good point about the Lima. So for the listeners, that means that the patient's had a cabbage and they've got their eyes left. Ima going into the, for example, anterior , , or to the, into a coronary. So you can't, you know, you can't cover that without you might cause a M I, [00:36:00] And I think good point on the future of the graphs, I think we do have already branch graphs that , , may not yet be approved for trauma, but they're probably coming, which will help.

And did we talk about, or maybe I'll ask a question. I don't know if that's fair. , , Kevin and Wayne, but did we talk about spinal cord drainage? I mentioned, you know, spinal cord ischemia and a rare incidence in paraplegia, you know, do you guys , , in San Antonio preemptively , , do spinal cord drainage , , for blunt aortic injuries that you're going to put an undergrad in or not?

**Kevin Kniery:** , no. Um, in general, it's thought that , , likely due to the short distance of coverage with these graphs, that there's not a lot of , , intercostals that are kind of covered and putting the spinal cord at risk. I think the, the risk of spinal cord ischemia is extremely low in these cases, but it's something you certainly.

Have to have in mind and have your anesthesia team or neurosurgery colleagues , um, aware of and available for , , should the situation change. And so certainly a motor in [00:37:00] neuro exam at the conclusion of the case is critical. And I think we would respond , , with , , elevating the blood pressures and , um, you know, potentially placing a spinal drain if there was concern for , , spinal cord ischemia , , following the case, but , , routinely , , we do not.

um , And, and that's sort of our kind of algorithm for that. How about you, Dr. Mucin?

**Todd Rasmussen:** Yeah, I agree. I don't have a whole lot more to add. We do it selectively very uncommonly and that's also because of the risks associated with , , placing a spinal cord drain. You know, those aren't minimal both placing the drain and then monitoring it effectively.

Um, I think we've got away from that. I think it is important for your listeners to understand also that. And I think you referred to this, that the risk of spinal cord ischemia, when you cover, you know, a segment of the, of the, of the descending thoracic aorta, the risk is low because we're not paving the entire or such a long segment.

But the risk is , , you know, it's out there for what, 24 hours, 48 hours. So there's a rare, you know, again, thinking about being a [00:38:00] pessimist , , I guess I should get away from that term. That sounds bad around the holidays. I'm not trying to be , , but you know, the resident's house officers, you have to realize that, that the patients who, for example, come out of the, or with a normal neuro exam, there is an incidence it's low to 3%.

But patients who can develop spinal cord ischemia like 24 or 48 hours or even 72 hours later. So we really have to be mindful of, of the spinal cord ischemia complication, even though it's uncommon.

**Wayne Causey:** Yeah, I think the post-operative care can't be understated. The fact that you avoid hypotension, you make sure that the patient's properly resuscitated.

And if there is any concern for a neurologic , , event that's occurring, that you have a system in place to where a lumbar drain can be placed relatively expeditiously. A couple of things that come to mind. When I think about spinal drains, particularly for elective cases are someone who's had a priority repair , , Chronic [00:39:00] kidney disease to a significant degree, stage four or five, or even in stage renal disease.

And , um, and whether I'm going to need to cover a large segment. And luckily for most aortic injuries, you're talking 10 centimeters for a covered endograft. And so again, that's going to be the, the shortest device that's available. And so it's going to give you the lowest risk. As far as an aortic coverage standpoint occurs.

**Kevin Kniery:** those are great points and things to keep in mind when , , fixing these patients. , so as we wrap up a blunt, thoracic aortic injury, we're going to cover one of the more important points. There's a lot of important points here. , but this one , , is the timing of the repair. Um, You know, generally it was thought initially that, you know, you should repair these sooner rather than later.

Um, but Dr. Rasmussen, , what, what are your thoughts on the timing? And, and specifically, I think we're referring to the, kind of the, the SVS grade three, the pseudo aneurysms, , what, what are your thoughts on the timing of repair of these.

**Todd Rasmussen:** Um, so another great topic, great question in this, , , pertaining to this injury pattern.

I think that , um, you know, I think that I'll start with the premise that [00:40:00] delayed repair is, is certainly feasible. Um, in many cases and in. Many cases it's preferable. And so , um, that's sort of the premise of , of, of the answer and delayed repair, meaning 24 to 48 hours later is feasible and preferable in many cases.

Um, I want to be careful because there's certainly some cases , , where it's, you know , , a grade four or a grade three that's, that's evolving into a four , , where the patient, you know, the delayed repair is not. Feasible, , um, but I think that Delaine w what we've understood, I think with our experience broadly on this , um, in the U S is that once you get a patient under your.

Care in, into your ICU. If they have a grade three repair and they've survived that far and you now can get them on anti impulse therapy, um , and, and

appropriate resuscitation, that, that waiting 24 hours again is safe and, and preferable. , and I think the majority of cases,, and, and why is that?

Well, I think it's because it allows us to do good. [00:41:00] Operative planning. We've, we've mentioned sort of the team steps, if you will, the things that we want to troubleshoot and anticipate for the repair , , whether it's size of vessel access assembling the right endograft the right team to include spinal cord drainage or what, you know, the whole thing.

I think we do a better job. We're giving ourselves 24 to 36 hours, for example. And I think it also allows patients other injuries to be stabilized. So whether that's a serious TBI , um, you know, long bone fracture, solid organ injury, , , I think it, it helps us. So , um, so I think Delaine is, is , is, is feasible.

We've just published. I, we mentioned. Well mentioned a whole lot of papers here, but , , there is a paper in the journal of vascular surgery , , that is a large assessments and just came out in the journal of vascular surgery, Ramon. , is the senior author, Dr. most of you know, is that you, the university of Texas health science center, and it's a great paper for a lot of reasons.

It's, it's the timing of repair for blunt aortic injuries. So it's this [00:42:00] paper addresses exactly your question. Um, it's a great paper cause it's got a trauma and vascular surgeons on it. , Bryan Eastridge, Don Jenkins are on it. , but it's got some really , , awesome , , vascular surgery fellows from Cleveland clinic who did this when they were in Cleveland.

, and what that analysis of the national trauma DataBank shows is, is just that is that it's actually a survival benefit associated with delayed repair. Um, and I won't go into the paper. Your readers can look at JBS and, and Ramon, Dr. , , , is you can sort of pub met him, or we can send that paper out on your, on your , , to your listeners , but, but it does show that there's a survival benefit for those who have a greater than 24 hour.

, delay in their TVR. And that's, there's an analysis that we did in that paper that, that controls for severity of injury, you know, meaning that it's not just because those with less severe injuries , um, survive longer. there's undoubtedly some survivor ship. Bias and that , , definitely acknowledged that, but it, but that paper speaks to its at large NTDB [00:43:00] analysis of this injury and it speaks to the, value of , of, of trying to delay repair if possible.

**Kevin Kniery:** Yeah, I think it's a really important , , topic , um, to have published the data on, because I think many surgeons and ER docs , and, and when they hear of a aortic transection or pseudo aneurysm feel , um, and they may pressure , , vascular surgeons to, you know, get in there and fix it. And I think , , knowing this information is critical and supportive of our community.

Yeah.

**Wayne Causey:** And the, and the delay also allows you to. Really get an idea of what's the constellation of the Andrew patterns that are occurring. I mean

, if, if there's some infectious process that's concomitantly going on with the injured patient's care that can an infected endograft and the thoracic order can be a nearly.

If not insurmountable problem to try to tackle and , , repair. Um, we, I can remember one case where the endograft was infected and before we could even wrap our hands around it, the aorta had ruptured and the patient ended up succumbing to their injuries. But[00:44:00] , , th th delaying it by a little bit of time affords you the opportunity to really gather as much information as you can, to make the best decision on when and how to prepare the aorta.

Yeah.

**Todd Rasmussen:** Yeah. And some of the delays, I mentioned 24 hours, but I think Wayne is you're pointing out. It can also be seven days, right. It can be, you know, as long as no, the thing is that, you know, you can't ignore the patient, you got to still stick with anti impulse therapy.it's , and you know, very meticulous care cause you can.

You know, there are cases certainly where a delay, you know, can fail, you know, and those are cases where we lose track of their blood pressure and, and , , you know, something happens in the, or for a femur fracture or something, and we lose track and , , we're not communicating as a multidisciplinary team and you can turn a stable.

, you know, grade three injury that you're observing for four, three or four or five days into one that ruptures that's were very rare. But again , , we want to be mindful of, of , , just because we're delaying doesn't mean you get a free pass until the, or, and , , five [00:45:00] days

**Wayne Causey:** later. Right? I think if I may, personally, if I'm going to be watching.

, grade three aortic injury , um, up to seven days that patient's going to stay in the ICU with very close monitoring that entire time until the time of the repair.

**Kevin Kniery:** These are fantastic points. And I think we really covered blunt, thoracic aortic injury , , quite in depth. And I think our listeners will really enjoy this.

We have two more topics that are much briefer to cover here as we close out the endovascular management of trauma. Um, so we're gonna move down the order a little bit. We're going to do below the diaphragm, and this is pretty rare, but there are. Important to keep this in mind. Um, the blunt abdominal aortic injuries in the endovascular approach to this.

Um, so many times it's a similar sort of patient population as the , , thoracic aortic injuries , , where it's a multiple injured trauma. And when we described the blunt abdominal work injury, they in, in repairing it for endovascular, there's three zones that are thought of , um, and the numbers ones, and three are the ones that you can repair , um, endovascular.

So the zone one is from the diaphragm down to the SMA. Um, zone [00:46:00] two is the SMA and renal arteries are relatively small segment, but , , that's the zone two. And then zone three is from the inferior renal inferior to the renal arteries, to the aortic bifurcation. Um, so. Zone one and zone three, you know, above , , from above the SMA two.

And then also below the renal arteries are the two areas that are amenable to endovascular repair.

**Wayne Causey:** Yeah. Fortunately, Kevin, these things are extraordinarily uncommon. I mean, most vascular surgeons at level one trauma centers, I would wager can count on one or two hands, depending on how old they are. The number of these injuries they've had to manage from an endovascular standpoint.

I think Dr. Rasmussen, what do you think about coverage of the celiac artery? Um, and what are the factors you think about if you had to prepare something in that area?

**Todd Rasmussen:** Yeah, I think that , um, I think there's increasing , um, literature, including I think , , an article and I'm just, won't pull the journal here, but I think it's.

Yeah, it's this November , , JVs about the feasibility and safety of covering the celiac artery it's [00:47:00] what's in this month. JBS is not pertaining to trauma, but it's talking about , , covering the celiac for , , for T VAR EMR, for example, for aneurysm disease. I think we're learning that you can cover the celiac safely.

In many cases, I think it, it reminds me of what the left subclavian was 10 years ago. No, no, we were a little nervous. We knew there were some patients you couldn't cover it in. And now 10 years later, we pretty routinely covered that left subclavian. , and I think the celiac artery is similar. It reminds me of that.

I think in patients, obviously you've got to look at the make sure they've got and SMA , , probably an Ima and then internal iliac arteries, you know, looking at the blood supply to the whole of their mesentery. And if any of that's compromised, then that would give me. Um, you know, pause, certainly if they've had a, you know, a gastrectomy here or some unusual intra-abdominal operation, that's interrupted their bowel.

If you will, the gut circulation that would give me pause to cover the celiac. Um, you know, but otherwise I think if it really gets you out of a [00:48:00] jam , , which these injuries are like, so yeah, you're right. Wayne, they're pretty rare, but man , they're, they're really hard to. Try to repair open. So that gets a person out of a jam and you need to cover the celiac.

I think he can.

**Kevin Kniery:** Yeah. I think those are fantastic points. And, you know, just like with the , , thoracic injuries and other, you know, situation where you have to be prepared to respond , um, with a bypass, potentially if, if , um, you know,

you have a schema that you didn't expect. Um, and so it's just things to keep in mind.

And one thing I think the we like talked about here is the IVIS is also very helpful. It's kind of like a, with. Dissection cases. A lot of these cases are sort of , um, kind of variants of dissections of, of the traumatic. And so the IVIS can really help characterize this. You can help identify these large intimal flaps , um, in pseudo aneurysms or , , kind of, and see the branches and help plan your stint graft placement all with IVIS.

So, So, one thing to keep in mind when, especially if these patients might not tolerate a lot of contrasts and other kind of situation to keep in mind, this is an, a patient with significant Tesla. Intestinal contamination , , the endovascular approach to this [00:49:00] would help , , eliminate any or. You know, minimize any risk of contamination.

, so Dr. Kazi, as far as the types of graphs , um, that you're thinking of when you have a patient , , what are you using? Are you using Tavarez covered since Eric cuffs? What's your preferred method?

**Wayne Causey:** My preferred method is to use whatever. Means is going to cover the least amount of aorta, but still seal the injury.

Um, so the answer to that is, yes, I I'd use all of them. Um, some of the newer covered balloon expandable and post dilatable stints have changed our management, particularly in the smaller aorta is at our level one trauma center. We carry. Basically the entire catalog of balloon expandable coverage stents.

, one that Kevin recently showed me , um, was we did an iliac branch case last week and the , , covered stent. I didn't realize started up. It started eight millimeters and goes all the way up to 11. I'm sorry. 16 millimeters. Previously, it was a 11 millimeter stent that could be post dilated up to 16. So in the smaller aorta is , , I think these have a role , um, and being able to post dilate them.

The [00:50:00] question is how to keep it in place when you're post dilating it so that it doesn't move. And so if that's a concern, then oftentimes I will go to a, , thoracic graft or an aortic cuff or , , something that I know is going to deploy and stay stable the entire time.

**Kevin Kniery:** And I think , , now we're going to move into our final topic of the day is XLO sub cleaving injuries and endovascular management.

We chose these three topics is endovascular care has really kind of in some ways revolutionized , , the management of these. And so , um, as we know the injuries to the XL, subclavian is very rare. And it's likely due to the protective nature of the skeleton, but the fact of the skeleton there also makes it very difficult to obtain proximal and distal control.

So , , Dr. Kazi, if you have a patient with some kind of contained some Flavian injury , um, and you're going to approach this from an endovascular approach

, , H w what approach do you prefer to, to access this.

**Wayne Causey:** My preferred approach is from the arm, if at [00:51:00] all possible , um, that could either be brachial or radio.

Um, depending on the, again, if you have a covered stent that can be post dilated to a size that can be delivered through a seven French sheath, and you can use a, a radial access for those patients in that , , Can be done percutaneously with low risk. Otherwise I do like to cut down on the brachial artery, if I'm , um, going to the brachial artery for trauma just seems to be more reliable for me and sometimes even quicker.

But, , I, I like the brachial approach, but sometimes I'll do both the brachial and the femoral approach , , and combine that with a through wire. If I have a really tough or challenging aortic arch and I need to deliver something. Say to the proximal, right. Subclavian. Um, and you've got , , a type three arch or something to that effect.

And I liked the through wire.

**Kevin Kniery:** Great. And so as far as you know, like discussing our stint options here, and I I've heard , , debates even on Twitter about this, about whether to use a self-expanding scent, because it might give you more flexibility, especially if it's in the axillary artery , um, versus balloon expandable , um, more precise [00:52:00] deployment, especially if near kind of branch vessels.

Um, Dr. SBS. And do you have a, do you have a preferred stint or do you just base it on the situation?

**Todd Rasmussen:** I think that we , um, had , , you know, I think initially we aired. Um, and well, I don't know, we erred on the side of self-expanding initially, if you think back , um, you know, when we first started treating this injury pattern with endo techniques covered stents, all of the covered stents available were self-expanding.

We didn't really have , um, you know, 10, 15 years ago, we didn't have viable , um, Balloon expandable covered stents. And so I think, you know, therefore that's what we got used to, whether it was the via Bon or the old fluency or whatever the self-expanding. And so we got used to that and I think more recently in the last three to four, five years , , there's now , um, really nice balloon expandable covered stents, which are flexible, I think it's dealer's choice in a lot of ways. I do think there is truth to , um, balloon expandable for more precise landing. You know, I just think that [00:53:00] premise of stenting is holds true, meaning that if you gotta really nail something , , you know, close to a Lima an internal mammary , um, for you read in relation to the vertebral.

Um, you know, blue expandable is I think more accurate if it's , , more distally and needed in a longer stand in the axilla than using a self-expanding is just fine. Another thing that, you know, I think you may get to, but sometimes there's you need to come from both right.

The arm, retro grade. Um, but if he can't retrograde, you know, sometimes coming antegrade , um, for imaging and sometimes to , to, to really make that connection between the disruption but coming from the groin into the. Origin of the subclavian axillary , , antegrade is, is needed as well.

**Wayne Causey:** one of my techniques for that was to have a through wire. So I had snare wire and the descending thoracic aorta. especially for challenging arch. Then I would come across with a sheath from the femoral artery and then deploy my stint with a through wire. Um, and that I felt, gives me a pretty [00:54:00] precise deployment , , particularly if I'm really trying to nail something.

And I want to make sure that there's no curves or bends and that the stent delivery. Which is usually a little stiffer than the sheath is going to be able to make it around and be very precise. Particularly if you're trying, like you said, nail on a vertebral artery or , um, and Ima

**Todd Rasmussen:** yeah, we had one where we, our last one we did here, a couple of my partners are pretty crafty and skilled and, and we, we actually had to put us , , we had to, I think we used a snare, right.

from the arm. We couldn't get , , the wire to make the approach. So we put a aim up, you know, antegrade through the lifts of Clayton, got a snare sort of out there, and we're able to snare the free wire and bring it through. That's pretty rare, pretty uncommon. Um, but you know , , I think those are all techniques that we're learning , , for the, kind of the fringes of these injury patterns, which used to be too severe for us to treat with endo.

And now we're actually finding with technology and experience that actually you can, you can treat the vast majority of these. [00:55:00]

**Kevin Kniery:** Yeah. These are , , A lot, a lot of options and things are going to continue to evolve as our technology improves. It's kind of a theme of this episode. ,

**Wayne Causey:** I seem to find that when people have these injuries there, for some reason that maybe this is just my own experience, it seems to be that they're more contra-indicated for anticoagulation.

I don't know if it's because the , , nature of the injury to injure something in the, you know, in the. Bony thorax, such as scapulothoracic dissociation or a sub claiming artist, pseudo aneurysm. I remember I can remember there's cases where the central line goes in for a head injured patient because they need to monitor their CVPs.

Um, I prefer to have a brachial open approach to that. And when I can't give anticoagulation, just because I can, then I'm set up to do an embolectomy if need be for the brachial artery.

**Kevin Kniery:** Yeah, I think that, I think that's a great point. And just wrap this up, and as far as discussing, you know, options you have from the

endovascular approach, there really it's up to your imagination.

, , you know , , whether it's [00:56:00] approximate rights of Cleveland injury, approximal, left, subclavian and injury. Um, you know, you can use a hybrid approach. I've seen multiple examples of , , large hematomas where , , proximal, distal control are obtained , , whether it's over a three wire from the arm.

Um, and then you can approach it open. Um, it really helps in that situation. Um, I know , , recently we had a case where we did some parallel stenting of the brachiocephalic in order to, to treat injury., and, and you can even use , , the approach to embolize. You can embolize the subclavian arteries , um, and you know, and do things , , with.

As long as you're respecting the people already. So , , keep in mind, you know, there's not one kind of recipe for treating these exits of Cleveland injuries , um, and really kind of adjusting it to the patient. you know, kind of the last thing I'll mention is , , sometimes these patients, you know, would probably do better with a bypass, especially with some axillary artery injuries.

You know, it's going to be in a pretty mobile spot , , but they're too sick. And so , , I've seen it even done where they use a stint graph to temporize things, to keep inline flow. And then at a more stable time, maybe a week, a month later, take them back and perform the bypass

**Todd Rasmussen:** [00:57:00] before you end. I don't want to get you , , let you off the hook.

I think the question for you guys and your listeners and , and, and this may be the most important question of our discussion is to what degree does it, should we in the military? Try to take these endo technologies into our deployed hospitals. Um, we've just discussed a range of injuries in which patients do better.

Right? So they bleed less. They're often less , , less frequently paralyzed from T-bar for example , , they survive more, they , , with these endo technologies. So are we telling ourselves that that's okay for civilians off the street in the U S. , but not good enough. , but, but our, what our service members have to just kind of get by and the deployed range.

Do you know when they're down range with these injuries or is it our responsibility to develop systems of casualty care in which we can take these technologies down range? What do you think?

**Wayne Causey:** Yeah, I think that's a great point. I mean , um, Obviously the farther [00:58:00] out you get. So like when I was in a Ford surgical team in Afghanistan, we, it took us three months to get , um, the most primitive x-ray machine you could think of.

And just to get a chest x-ray to see if there was a pneumothorax. But when you get back to the rural three , um, level, which for everyone, for the listeners, that's a combat support hospital. So there were, for instance, there would be

one in Baghdad. So there's one that triages to the next level of care from all the forward surgical teams to a combat support hospital.

There's, there's two in Afghanistan, for instance, but , um, They don't are always equipped with endovascular technologies as Dr. Rasmussen's really al-luding to. And as a matter of fact, there, there are going to be no thoracic endo to repair there's. Um, the capabilities is digital subtraction angiography. Um, usually it best to say, you know, treat a bypass.

Yeah. But I do think that one of the things we brought up in this podcast and Dr. Brass means I'd be interested in your, your thoughts is that we have demonstrated that there is a delayed [00:59:00] timing of repair for a Arctic injuries. And I do think that deployment and placing an, a thoracic endograft or a covered stent.

In say Germany, Walter Reed or San Antonio is much better than trying to do the same thing in Baghdad or bog room or Kandahar Afghanistan, because , um, there are the, a septic techniques are not going to be the same there as they are here. And the bacteria and the microorganisms that exist in those environments are also significantly different than what we experienced in the United States.

**Todd Rasmussen:** Yeah, I think it's a great question. And, you know, I kinda, I get a little bit fired up about it. Um, I know you've got a lot of young, you know , you've, you've guys got the future , , who are your listeners? You know, and I think there there's a ton of , um, brain power, you know, intellectual capacity that listens to this , , and it's to your guys's credit.

I think it's, it's going to be up to, to the next generation, right. To figure out. How do we integrate , , endo [01:00:00] technologies for wartime scenarios and , um, you know, and I think to it's , it's, it's like it's too big of a topic to answer that with one question. But I think if you take it in , in, in sort of intervals and you say , well, in the near term, right?

So from now to 2022, 2023, Maybe all we can do is get like what you said, Wayne. Let's make sure we have vascular go teams on the West coast and the East coast. , who can go into the Pacific or the European theater, African theater , , with endo stuff. Right? So that if, if we can't get a casually back, we can , um, you know, figure out what we can do in the near term.

But, you know, in the, in the midterm, whose fault is it that there's no digital subtraction NGO to rule three? Is that, do we just going to blame it on the system and say, it's a damn shame. Where is that our fault? Right. Cause we're not articulating this. And I think, you know , we, we should think about that.

I think that, yes. , so you know, most of the time a covered stent for sure. Yeah, absolutely. Wayne. I mean, you'd definitely rather have a T-bar and, you know, Germany or , , [01:01:00] Walter Reed or bamse than, than in Ballade or Bodrum, Afghanistan, for example. But some patients can't wait. Right. And

so. You know, how do we establish an endo specific trauma inventory and get it into the cash?

Right? So the, for the select cases who can't wait, what if it's a type three or type four, , blunt, aortic injury, do we then commit that service member to, you know, an antiquated treatment, maybe, , if it's a one cause, and it's particularly relevant in this discussion, because you've mentioned injury patterns that can be highly lethal, but are particularly amenable.

To end of therapies, you know, and there's no answer to it. And I don't mean to get too fired up, but I, I'm excited to sort of pose that question to the young and innovative, you know, listeners that you guys have to help us think about. What does a trauma specific endo inventory, how can it be brought to, you know, resource limited circumstances or scenarios to, to provide the benefit that these technologies have to some of these patients.

It's great discussion. And [01:02:00] I really appreciate the opportunity to spend some time with, , with you guys. , and, and , it's been fun.

**Kevin Kniery:** Absolutely. And doctor asked me to sing. You have to thank you for this entire series for joining us and taking time out of your very busy schedule to get down and dirty and talk very specific and very technical.

Um, I think this will be a resource for years to come for both the listeners of behind the knife and audible bleeding. So thank you. Thank you again, and thank you, Dr. Kazi for joining us and helping us with this discussion. Yeah. Thanks

**Wayne Causey:** Kevin.

**Todd Rasmussen:** Stay safe and stay well.

Seminal Papers in Blunt Thoracic Aortic Injury

- AAST 1997 Paper: <https://pubmed.ncbi.nlm.nih.gov/9095103/>
- AAST 2008 Paper: <https://pubmed.ncbi.nlm.nih.gov/18545103/>
- JVS 2011 Paper: <https://pubmed.ncbi.nlm.nih.gov/20974523/>
- Timing of repair of BTAI JVS 2020: [https://www.jvascsurg.org/article/S0741-5214\(20\)31575-5/fulltext](https://www.jvascsurg.org/article/S0741-5214(20)31575-5/fulltext)

Dr. Ben Starnes' podcast on Behind The Knife on BTAI: <https://bit.ly/2LuycWq>

# Chapter 11

## Angioaccess

**UNDER CONSTRUCTION - This Chapter is Currently in DRAFT mode**

**28 Mar 2020:** *Young Lee, MD and Matthew Smeds, MD*

### 11.1 Planning access creation

Management of Dialysis access is an important topic of discussion, not only because it is a significant part of board examinations, but also because health-care costs continue to rise for ESRD patients, particularly during the transition from CKD to ESRD. This is attributed to use of dialysis catheters and frequent hospitalizations for arteriovenous access failures and related procedures.

The National Kidney Foundation-Dialysis Outcomes Quality Initiative (NKF-KDOQI) and SVS has provided guidelines in the follow areas:[Foundation, 2015, Sidawy et al., 2008]

- Timing of referral to access surgeons
- Operative strategies to maximize placement of autogenous AV accesses
- First choice for autogenous access
- Choice of AV access when a patient is not a suitable candidate for a forearm autogenous access
- The role of monitoring and surveillance of AV access management
- Conversion of a prosthetic AV access to a secondary autogenous AV access
- Management of nonfunctional or failed AV access

**This brings us to the question, who needs dialysis access?**

Patients should be referred to a vascular surgeon for access when their creatinine clearance is <25mL/min which is CKD stage 4. You want to provide adequate time for your autogenous access to mature, so the ideal time for access creation would be > 6 months for anticipated need of dialysis. This allows for time for any subsequent interventions if your access is not maturing.

**Should prosthetic access also be placed several months before anticipated dialysis?**

Prosthetic access patency is limited by duration of access placement, thus, if a patient requires prosthetic access, placement should be delayed until about 3-6 weeks before initiation of dialysis.[Foundation, 2015]

**For dialysis access creation, which site should be considered and used first?**

Due to the easier accessibility and lower infection rates, upper extremity access sites are used first. Furthermore, you want to place your access as far distally in the extremity as possible to preserve the proximal arm for future accesses.

**What are some important considerations in a patient's history when planning a dialysis access?**

It is important to find out recent history of:

- Previous access procedures
- Peripheral IV lines
- Sites of any existing or previous indwelling catheters including pacemakers and defibrillators
- Trauma or surgery to the upper extremities

Moreover, you also want to consider the patient's quality of life, thus, noting which extremity is dominant is important. If possible, you want to create your dialysis access in the nondominant arm so that when the patient is receiving dialysis multiple times a week, they are able to use their dominant arm during their dialysis sessions.

However, placing a fistula with lowest rate of failure is preferred to using non-dominant hand. Therefore, placement of access on the patients dominant side would be preferred over placement of access with high risk of failure (i.e. on the side of an implantable cardiac device).[Sgroi et al., 2019]

**As with any preoperative planning, physical examination is extremely important. Central venous stenosis can cause problems such as prolonged bleeding after dialysis sessions at the puncture site. What are some signs of central venous stenosis?**

Unilateral arm swelling or edema and prominent venous collaterals are signs of central venous stenosis. Central venous stenosis can lead to venous hypertension which affects access patency and function, and also causes disabling edema.

Beyond signs of central venous stenosis, when examining a patient, an Allen's test should always be performed to evaluate palmer arch patency.

**Preoperative planning should also include arterial and venous assessments. What are your size requirements for the artery and the vein to be used in your dialysis access creation?**

First, you want equal pressure gradients in bilateral upper extremities. If there is a pressure differential  $>20\text{mmHg}$  or pulses are non-palpable then patients should undergo a duplex ultrasound to evaluate for arterial insufficiency and the artery should be greater than or equal to 2mm. Segmental pressures have limited applicability. Patients with correctable lesions on duplex may consider undergoing angiography and intervention to optimize inflow.[Sidawy et al., 2008]

A venous duplex should also be done to evaluate for diameter, distensibility and continuity. A vein mapping is useful to determine the size of the patient's superficial veins at various points in the forearm and upper arm. The vein should ideally be at least 3mm, but 2.5mm will likely dilate after regional anesthesia and be sufficient for access.[Huber et al., 2002, Smith et al., 2012]

**Autogenous access should always be considered first due to higher patency rates, lower infection rates, and longer duration of access survival. What are the different configurations of autogenous accesses?**

The first and best option would be direct arteriovenous anastomosis. However, if that is not possible, then venous transposition should be considered next followed by venous translocation.

Venous transposition is for deeper veins such as the basilic vein, which is transposed so the vein lies just below the skin for easier access for puncture during dialysis sessions. This can be done in either a one-stage or two-stage procedure. In a 2-stage procedure, the direct arteriovenous anastomosis is created during the first stage and once the vein has arterialized 4-6 weeks later, the second stage of transposition is done when the vein is easier to mobilize. Translocation procedures include harvesting the femoral or saphenous vein and using it as a conduit for AV access creation in the upper extremity.

**When can a venous transposition be done in a one stage procedure?**

Many surgeons will do a brachiobasilic arteriovenous fistula creation with the transposition in one-stage. However, if the vein is  $<4\text{mm}$ , it is generally recommended to do a two-stage procedure so that if the fistula fails to mature, the patient does not have to undergo a second operation with extensive dissection of the vein. When a patient has not been initiated on dialysis and there is marginal vein that would require transposition, but carry a significant risk of graft failure then a first-stage BB fistula may be appropriate.

When comparing single- to two-stage brachiobasilic fistula, two-stage creation had high rates of primary patency at 2-years. However, there was no differ-

ence in infection rate, steal syndrome, hematoma, pseudoaneurysm, stenosis, failure rate, primary or secondary patency at 1-year or secondary patency at 2-years.[Jun Yan Wee et al., 2018]

**It was mentioned earlier that the dialysis access should be created as distally as possible on the extremity. What are some of the most distal locations?**

The snuffbox fistula, which is the posterior radial branch to cephalic direct access and Brescia-Cimino-Appel (often shortened to just Cimino) fistula which is the radial-cephalic wrist direct access are two of the most distal fistulas that can be created. Overall, these have comparable patency and ischemia risk. While snuff box fistulas carry a 10% failure rate at 1mo, the majority are able to be converted to RC fistulas.[Siracuse et al., 2019]

**What are your arterial and venous options in the upper extremity?**

In the forearm, you have your radial, ulnar, and brachial arteries and cephalic and basilic veins. In the upper arm, you have your brachial or proximal radial arteries and cephalic, basilic, brachial and axillary veins.

If adequate vein is available, most common access sites distal to proximal include:

1. Snuff box
2. Radiocephalic
3. Basilic transposition in forearm
4. Brachiocephalic fistula - these have a few advantages over more distal fistulas, including higher maturation rates, decreased time to maturity, higher patency and functional primary patency.[Nguyen et al., 2007]
5. Brachiobasilic fistula[Gilmore, 2006]
6. Vein transposition from a remote site - Femoral vein transposition can be used for upper extremity dialysis access in patients with previous upper extremity access complicated by infection, thus not a candidate for prosthetic, but no central stenosis. There is a high rate of hand ischemia due to larger conduit (up to 43%).[Huber et al., 2004]

**If you need to use a prosthetic graft, what would you use?**

PTFE is the most commonly used prosthetic graft. Either a 6mm graft or a tapered 4-7mm graft can be used. The tapered grafts help to ensure that the size of your arterial anastomosis isn't too large to minimize chances of steal. However, recent publications found that there is no difference between tapered and non-tapered grafts in terms of primary patency, steal syndrome, complication rates or need for reintervention.[Roberts et al., 2019, Han et al., 2017] Both the 6mm PTFE graft and the tapered 4-7mm PTFE grafts are standard sizes used for dialysis access creation.

Overall AVG have benefit of earlier cannulation, discussed later, however they have lower patency rates and higher infection rates. There is no difference between grafts and fistulas for perioperative morbidity and mortality.[Macsata and Sidawy, 2019]

Most prosthetic access will fail because of the development of venous outflow stenosis as a result of smooth muscle proliferation at vein graft interface. Thus, a major cause of graft failure is due to thrombosis. Heparin-bonded PTFE (HB-PTFE) grafts are an options, however, studies have shown no improvement in graft patency with HB-PTFE compared to standard PTFE grafts.[Shemesh et al., 2015, Davies et al., 2016]

There are also early cannulation prosthetic grafts, which is another option for patients who need more immediate dialysis access. These grafts are constructed in 3 layers with an elastomeric membrane in the middle between two layers of ePTFE material. This allows for cannulation as early as 24 hours from implantation because the graft configuration minimizes dialysis needle bleeding. This graft options allows for avoidance of a central venous catheter. Studies have shown similar patency rates compared to standard ePTFE, earlier cannulation and catheter removal, and decreased catheter related complications.[Al Shakarchi and Inston, 2019, Wagner et al., 2019]

**For patients with central venous stenosis or occlusion, what is another alternative upper extremity access creation?**

Two overall strategies for managing patients with severe central venous stenosis or occlusion are lower extremity AV access of hybrid catheter/grafts. Patency of femoral vein transposition is better than hybrid catheter/grafft placement.[Brownie, 2016, Glickman, 2011] We will cover specifics of these techniques later.

For lower extremity AV access, femoral vein transposition has been shown to have the best outcomes, followed by saphenous vein transposition and graft placement. Lower extremity access should be avoided in patients with peripheral vascular disease or lower extremity ischemic symptoms—when in doubt perform duplex and segmental pressures to fully evaluate. Veins should also be evaluated to ensure patency more proximally.[Parekh et al., 2016]

For patients where lower extremity access may not be appropriate, the hemodialysis reliable outflow (HeRO) device can come to the rescue. These devices may not have been encountered in your training, so we will go into detail here. This device is composed of 2 components: a graft which is made of 6mm PTFE with a titanium coupler at one end, and a venous outflow component of a 19 Fr silicone catheter reinforced with a nitinol braid to prevent kinking. The graft portion is anastomosed to an artery, usually brachial, and is tunneled subcutaneously and the venous component is percutaneously placed into the right atrium via the IJ or subclavian vein. The two components are connected with a titanium coupler at the deltopectoral groove. If you need more immediate dialysis, the super

HeRO comes to the rescue in which the graft portion is the early cannulation graft.

For patients with thoracic central venous occlusion, there is also the SURFACER inside-out access catheter system. This is a device used to cross central occlusions from the right femoral vein. It allows placement of a tunneled central venous catheter, but it can also be utilized as an adjunct for HeRO placement. In a multicenter study, the results from the SAVE (Surfacer System to Facilitate Access in Venous Obstructions) registry showed that in 29 or 30 patients with thoracic central venous occlusion, a central venous catheter was successfully placed. Moreover, there were no device-related adverse events, catheter malposition, or intra- or postprocedural complications. [Gallieni et al., 2020]

Hybrid catheter/grafts are good alternatives in patients with previous line infections, central stenosis, peripheral vascular disease and no suitable vein in the upper extremities. They have shown fewer bacteremia episodes than catheter, low primary patency rate and acceptable secondary patency rates.[Al Shakarchi et al., 2015] Absolute contraindications include donor artery <3mm, inability to dilate outflow vein to 19f, allergy to device materials (ePTFE, silicone, titanium, nitinol), and current active infection.[Medical]

## 11.2 Techniques for access creation

**The techniques of arteriovenous fistula creation are common across access sites. Can you go through the techniques?**

First the vein is identified and the distal end is transected and flushed with heparin. By flushing with the heparin, you are able to access the caliber and extent of the vein as well as identify any side branches

Then after distal and proximal control of your artery, a 4-6mm arteriotomy is made. The length is limited to decrease incidence of arterial steal. The artery is then flushed with heparin to avoid thrombosis during the anastomosis and an anastomosis is created between the side of the artery and the end of the vein. A 6-0 or 7-0 nonabsorbable continuous suture should be used to create the anastomosis to avoid future dilation of the anastomosis.

For prosthetic accesses, the length of the arteriotomy does not have to be limited to 4 to 6mm since the incidence of arterial steal is limited by the graft diameter. Both arterial inflow and venous outflow vessels need to be dissected prior to graft anastomoses. The graft should be tunneled close to the surface of the skin to allow for easier cannulation. Meticulous attention to sterile technique is important to avoid graft infections. And as with the fistula creations, a 6-0 or 7-0 nonabsorbable continuous suture should be used to create the anastomosis to avoid future dilation of the anastomosis.

**What are some other options if an access is not able to be created in the upper extremity?**

Autogenous accesses can also be created in the lower extremity. Femoral artery to femoral vein or saphenous vein anastomosis can be created. Both veins have to be transposed. Synthetic grafts have high infection rates (as high as 22% in some series) and only a 50% patency at 6 months.[Antoniou et al., 2009, Lazarides et al., 2018, Pike et al., 2019] Risk of lower extremity ischemia increased with burden of PVD, AV fistula vs graft, distal arterial inflow, and large femoral vein mismatch, but can be tempered by banding/narrowing of femoral vein at the time of surgery.[Antoniou et al., 2009, Bourquelot et al., 2012, Gradman et al., 2005]

Access creation in the chest wall or cervical region is also possible with axillary artery to ipsilateral axillary vein loop access, axillary artery to contralateral axillary or jugular vein straight access (ie necklace access) and brachial artery to jugular vein straight access. Keep in mind that for these fistulas, the central veins must be patent.

**Are there benefits to different anesthetic techniques used during access placement, such as regional anesthesia?**

Regional anesthesia for AVF creation has been associated with higher periooperative flow and lower rates of vasospasm resulting in higher rates of short term patency. Regional anesthesia has not been associated with perioperative rates of major morbidity or mortality.[Aitken et al., 2016, Siracuse et al., 2014]

**What is the role for endovascular creation of AV fistula?**

Endovascular approach to fistula creation without open surgery is another option for dialysis access creation. The results of the NEAT study (Novel Endovascular Access Trial), prospective multicenter study which showed that 98% of the 80 patients enrolled had EndoAVFs created. Of these, 87% were physiologically suited for dialysis, and functional usability (2 needle cannulation) was 64%. Primary patency was 69% and cumulative patency was 84%. Total complication rate was 8% and this is a promising alternative to surgical arteriovenous fistula creations. [Lok et al., 2017]

**When is the newly created dialysis access ready for use?**

A good way to remember this is the rule of 6's. Fistulas should be created about 6 months prior to start of hemodialysis. It is ready to use when the fistula is 6mm in diameter, has a flow of 600ml/min, is 6mm from the surface of the skin and usually takes 6 weeks to mature.

Prosthetic AV accesses can be used as early as 2 weeks postoperatively. If you use the 3-layer early cannulation grafts, the access can be used as early as 24 hours after access creation. This is great because it offers the potential for avoidance of dialysis catheters in patients who need dialysis immediately.[Glickman et al., 2015]

## 11.3 Maintenance and complications

### 11.3.1 Failure to mature

**What are some reasons why an access may fail to mature?**

Sometimes your access may have arterial inflow stenosis. This is difficult to detect clinically because there will be a palpable thrill, however, due to the stenosis, the flow is not sufficient enough for dialysis. Dialysis access duplex is a useful way to assess flow volumes and identify areas of stenosis that could be further assessed with fistulogram. In the absence of arterial inflow issues, collateral or large venous branches can divert blood away from the main access channel resulting in insufficient flow. This can be resolved by coiling or ligating the large venous branches that limit maturation of the fistula.

**If the newly created AV fistula is not maturing, what are some secondary procedures to help with maturation?**

Open procedures include vein patches, interposition vein grafts, vein transposition to proximal arteries, branch ligations, and vein superficialization. Endovascular procedures include arterial and venous angioplasties to improve inflow or outflow issues.[Sidawy et al., 2008] In particular, balloon assisted maturation (BAM) has been described to decrease maturation time. A prospective randomized controlled study by Elkassaby et al. showed that BAM significantly decreased maturation time and had higher successful functional maturation. However, BAM was also shown to have increased fistula complication rates.[Elkassaby et al., 2021]

### 11.3.2 Access Failure

**Once a dialysis access is created, maintenance of the access is extremely important. The flow disturbances and hemodynamic changes associated with AV access creation causes intimal hyperplasia leading to venous outflow stenosis. This can ultimately lead to access thrombosis and failure. What are some methods of detecting access failure?**

One way of detecting a well functioning access is a strong thrill at the arterial anastomosis which continues a few centimeters into the outflow vein. If you feel a pulsation near the venous outflow, then a stenosis or thrombosis is likely. If you feel a thrill distal to the area of pulsation, then you have likely localized your area of stenosis. It is important to note that you may feel a pulsation at a pseudoaneurysm independent of venous outflow issues.

Other signs of outflow stenosis are collateral veins or upper extremity edema.[Padberg et al., 2008] This is indicative of venous hypertension likely secondary to stenosis. You will typically see this in the shoulder area or anterior chest as a result of subclavian vein stenosis/thrombosis. Moreover, these high venous pressures as a result of the stenosis can result in excessive and prolonged

bleeding after removal of needles from the dialysis puncture sites. This is often the first sign of elevated venous pressures. These patients should undergo a fistulogram to evaluate for underlying outflow stenosis.[Caro Monroig et al., 2018] Sometimes, a hematoma from dialysis access cannulation can result in compression of the fistula. In these cases, the hematoma should be drained to relieve the compression.

The most common cause of graft failure in upper extremity fistulas is venous outflow stenosis and in grafts is venous anastomotic intimal hyperplasia.[Berman and Gentile, 2001, Padberg et al., 2008] Diagnostic fistulogram is a very useful tool to diagnose and treat potential access complications. Diagnostic fistulogram is often performed with proximal access of the fistula a few centimeters distal to the anastomosis. Compression of venous outflow is necessary to evaluate for inflow and anastomotic issues.[Bountouris et al., 2018]

Finally, even patients with normal physical exam can have issues while on the hemodialysis circuit, such as recirculation. When in doubt, if there are recurrent issues with hemodialysis, the patient should undergo a fistulogram to evaluate for occult venous outflow stenosis that may be missed on other diagnostic modalities.[Sidawy et al., 2008]

**What are some endovascular interventions for a failing access?**

First line therapy for outflow stenosis is a simple balloon angioplasty of the stenosed area.[Berman and Gentile, 2001] Insufflation times are generally up to 2-3 minutes. Treatment of stenosis 2/2 intimal hyperplasia often require high pressures of 20 ATM or more. However, this is a double edge sword because this can lead to trauma in the veins stimulating a further intimal hyperplasia process. Some advocate a cutting balloon before high pressure dilation. There are also studies showing improved primary patency with drug coated balloons with no difference in survival compared to plain angioplasty.[Han et al., 2017, Chen et al., 2020, Moreno-Sánchez et al., 2020, Yin et al., 2021] Stenting is also an option to treat residual stenosis or dissections after balloon angioplasty. Covered stents have shown good patency results.

Some unique situations include, recurrent cephalic arch stenosis which may be best treated with bare metal stenting.[Shemesh et al., 2008] Proximal occlusions (i.e. subclavian) may be best treated primarily with a covered stent.[Agarwal, 2015, Anaya-Ayala et al., 2011]

**If endovascular interventions fail, what are some open options for managing a failing access?**

Generally an interposition graft or patch angioplasty is performed and the results of the two techniques are largely equivalent.

**If an AV access has ultimately failed and thrombosed, what are your endovascular options at this point?**

Some endovascular options are catheter directed thrombolysis with about 2-4mg

of tPA injected into the clot, followed by balloon angioplasty (typically an 8mm by 8cm high pressure balloon). A mechanical thrombectomy device, such as angiojet, can also be used in combination to thrombolysis.

Alternatively, an open thrombectomy with a thromboembolectomy balloon and patch angioplasty of venous stenosis areas can also be used. Moreover, a hybrid approach of open thrombectomy with percutaneous interventions of venous stenosis areas has been described.

Immediate postoperative thrombosis of an AV graft is likely technical - inadequate inflow (i.e. small brachial artery) or outflow stenosis/occlusion. Open revision and thrombectomy is often the best option.[Paulson et al., 2002]

**What are some ways to manage failing AV access in the setting of ipsilateral vTOS?**

Performing a first rib resection in a hemodialysis patient is high risk and controversial. However, if there is a prominent external jugular vein, then there are reports of external to internal jugular vein transposition offering more in line drainage and access salvage.[DeGiovanni et al., 2020]

### 11.3.3 Steal Syndrome

**Earlier, you mentioned steal syndrome, can you explain to us what this is?**

Steal syndrome is also known as Access Related Hand Ischemia = ARHI. It is an uncommon but devastating complication of access creation. All patients with arteriovenous fistulas have some degree of physiologic steal or reversal of flow in part of the artery distal to the fistula. However, this is not sufficient enough to cause ischemia. Rather, ischemia results from inadequate collateral circulation and inability of peripheral arteries to meet the increased demand. Diseased vessels do not dilate and stenosis of arteries leads to decreased distal perfusion pressure. Furthermore, hypotension during dialysis further decreases perfusion causing symptoms. Steal can be limb threatening and is graded as follows:

- Grade 0 - asymptomatic, no flow augmentation or steal
- Grade 1 - asymptomatic, mild ischemia with signs of cool extremity and flow augmentation with access occlusion (May be seen in over half of AV access patients)[Leake et al., 2015]
- Grade 2 - moderate/intermittent ischemia that is experienced only during dialysis and patients feel claudication.
- Grade 3 - severe, ischemic pain at rest with tissue loss.

**What are some symptoms and signs of Steal syndrome?**

Symptoms include coolness, parasthesias, rest pain, and weakness. Signs of steal include cool to touch, pallor, cyanosis, delayed capillary refill, absent

pulses/signals, diminished sensation, weak grip, and in severe cases ulceration or gangrene. If the patient shows improvement with access compression, diagnosis is confirmed.

Patients with acute pain and loss of pulses after AVF creation should first have arterial injury/thrombosis ruled out, but then after diagnosis of steal syndrome should undergo fistula ligation.[Schanzer and Eisenberg, 2004, Yevzlin et al., 2016]

#### **When is an intervention necessary to treat steal syndrome?**

You do not need to intervene for grade 0 and 1.[Leake et al., 2015] For grade 3 an intervention is mandatory. The goal of treatment includes symptom resolution and access preservation, and this is achieved by reducing access flow and increasing distal arterial flow.

The most important finding on duplex ultrasound to evaluate for steal syndrome is fistula flow rate. High flow rates suggest a primary problem with the fistula, low or normal flow suggests underlying PVD and inflow insufficiency as etiology.[Al Shakarchi et al., 2016, Leake et al., 2015] Approximately 5% of steal is secondary to inflow stenosis.

#### **What are your intervention options for resolving steal syndrome?**

There are multiple ways to manage steal syndrome, detailed as follows:[Leake et al., 2015, Gupta et al., 2011]

- AV Fistula Banding is a simple option to reduce access flow. This is done by suture plication, placement of single narrowing tie or wrap by constrictive cuff to cause a stenosis in the AV access near the arterial anastomosis. A minimally invasive approach is used by the MILLER banding which uses an endoluminal 4 or 5mm balloon as a sizer and a suture is placed around the access with the balloon inflated. This procedure increases arterial inflow towards the hand. Flow should be maintained at 600ml/min.
- Revision using distal inflow (RUDI) involves ligation of the fistula at the arterial anastomosis and reestablishment of flow via a more distal artery by bypass or vein translocation. This allows for decreased flow through the access by reducing the fistula diameter and by taking inflow from a smaller vessel. However, ultimately, the fistula is placed at risk
- Proximalization of arterial inflow (PAI) involves ligation of AV anastomosis, and the inflow is moved to a more proximal level with a prosthetic interposition. Dialysis can be continued via the vein. The main advantage is the native artery's continuity.[Zanow et al., 2006]
- Distal revascularization-interval ligation (DRIL) is ultimately considered the best option by many vascular surgeons due to the excellent results shown. There is an arterial bypass created originating proximal to the access and ending distal to the access, with ligation of the artery distal to the anastomosis. This prevents retrograde flow from distal vessels and allows

for a low resistance pathway for arterial supply to the hand. DRIL may be particularly useful in patients with distal brachial, proximal ulnar/radial disease, as this can bypass the underlying stenosis as well.[Leake et al., 2015]

- Distal radial artery ligation (DRAL) can be performed for palmar arch steal syndrome from radio-cephalic av accesses, to prevent reversal of flow in the palmar arch. However, the ulnar artery patency needs to evaluated first.

#### 11.3.4 Neuropathy and Neuropraxia

**What complication often presents like steal syndrome, but with easily palpable distal pulses?**

Access creation can result in neuropathy. It is important to note that over 2/3s of the patients have preexisting peripheral neuropathy. Neuropathy is also graded:

- Grade 0 - asymptomatic
- Grade 1 - mild intermittent changes (pain, paresthesia, numbness with sensory deficit)
- Grade 2 - moderate persistent sensory changes
- Grade 3 - severe sensory changes with progressive motor loss (motion, strength, muscle wasting).

Ischemic monomelic neuropathy is rare but occurs acutely after AV access creation. Within hours of surgery, patients develop acute pain, weakness, or paralysis of hand and forearm muscles with prominent sensory loss. However, the hand is warm with palpable pulse or audible signal in distal radial and ulnar arteries. It is important to note that pain out of proportion is what differentiates IMN from ARHI. Treatment is access ligation or emergent augmentation of flow.

#### 11.3.5 Perioperative neuropraxia

**What are some common technical complications after placement of AV access?**

Post operative numbness of the thumb, index and middle finger and weakness of abduction and grip strength may suggest median nerve neuropraxia and will likely resolve. However, you need to rule out steal syndrome.[Talebi et al., 2011, Vahdatpour et al., 2012]

Postoperative hematoma can often just be observed. However, signs of median nerve compression mandate immediate operative brachial sheath evacuation or the neurologic deficits may become permanent.[Padberg et al., 2008]

Prosthetic grafts can result in seroma from ultrafiltration of the graft and most resolve without intervention.

### 11.3.6 Bleeding and Aneurysm

**What presentations require urgent revision?**

Fistulas that present with bleeding from a visible ulcer, wet scabbing, frank infection or acute pseudoaneurysm often require urgent open revision. Some access may be salvaged, but in acute presentations with significant risk of life threatening hemorrhage the safest option may be to ligate the access and place a temporary dialysis access line.[Galbusera et al., 2009]

**Many fistulas develop aneurysms over time. When do fistula aneurysms require revision?**

- Pseudoaneurysms often result from trauma due to repeated punctures or poor technique
- True aneurysms result from long standing hemodynamically significant outflow stenosis.

Both can lead to cannulation difficulties, increased risk of thrombosis, pain, bleeding and cosmetic deformities. AV aneurysms should be revised if they develop skin thinning, ulceration or bleeding. Early skin changes can be observed. Intervention often requires revision with open plication/excision or ligation. When isolated to a short segment, attempts should be made to salvage the fistula.[Al-Jaishi et al., 2017, Al-Thani et al., 2017, Pasklinsky et al., 2011]

## 11.4 Dialysis Catheters

**While ideally every patient would have surgically placed access, many patients still receive dialysis through catheters. What is the difference between an acute and chronic hemodialysis catheter?**

Chronic catheters have a subcutaneous cuff at the exit site and tunneled to the vein. This decreases infection rates and makes them less likely to become dislodged. Tunneled hemodialysis catheters can be used up to 12 months.

**If catheters cause so much problems such as infection and central venous stenosis, what would be an indication for them?**

The most common indication would be for urgent hemodialysis. But other indications include patient who are not operative candidates due to advanced comorbidities, or patients who are unable to have an AVF or AVG due to anatomic feasibility. Temporary dialysis access may also be needed in patients who have just had a peritoneal dialysis catheter placement or in chronic peritoneal dialysis catheter patients requiring abdominal or inguinal surgery.

**Which site is the most ideal site for a hemodialysis catheter?**

The right internal jugular vein is preferred because it has the best patency.

If all traditional access sites are inaccessible, translumbar IVC catheters may be used as a last resort as salvage. Overall complications are similar to other permanent dialysis access sites, however obesity is a relative contraindication due to potential migration into the soft tissues.

**Every procedure has potential complications. What are the immediate complications of catheter placement?**

When placed in the internal jugular veins, there is always a chance of a pneumothorax or hemothorax. Wire embolism can occur if control of the wire is lost during the procedure. If the guidewire is placed too far, then there is always a chance of arrhythmia. Thus, the best place for the wire is through the IVC. With a left internal jugular vein approach, there is always a risk of thoracic duct laceration. If a leak is apparent, then the catheter needs to be removed immediately and a pressure dressing applied.

**How do you manage infected hemodialysis lines?**

Infected hemodialysis lines should be removed. Ideally you should avoid replacing any lines until blood cultures have been negative for 48hrs, particularly subsequent permanent lines[Group, 2006]

## 11.5 Peritoneal Dialysis

**Although not often managed by the vascular surgeon, we should still be aware of another method of dialysis in renal failure patients. What are the indications for peritoneal dialysis?**

Peritoneal dialysis is better tolerated and less disruptive to daily life, so is often a better option for young, active patients with less comorbidities. PD can be considered first-line or in patients who are not candidates for HD, but require high patient engagement and education and so may not be appropriate for patients with other chronic diseases limiting independence, unstable housing, cognitive decline or poor management other medical issues.[Ansari, 2011]

From a technical standpoint, PD may be difficult in patients who have had multiple previous open abdominal surgeries, but can be tolerated in patients who have had minimally invasive or minor abdominal procedures.[Sinnakirouchenan and Holley, 2011] Long dwell times with glucose solution can sometimes lead to hyperglycemia. The major reason for conversion from PD to HD is PD catheter infection.[Li et al., 2017]

# Chapter 12

## Vascular Lab

**UNDER CONSTRUCTION - This Chapter is Currently in DRAFT mode**

**06 Jan 2022:** *Alaska Pendleton, MD and Anahita Dua, MD*

*Acknowledgements: A special thanks to Drena Root, Technical Director at the Massachusetts General Hospital Vascular Center, without whom this imaging would not have been possible.*

So let's start with an overview of ultrasound modalities, waveforms, and changes proximal and distal to flow-limiting stenosis.

### 12.1 Overview

**Brief overview of ultrasound:** Ultrasonography uses sound waves with frequencies higher than those audible to humans. Ultrasound images are created by sending pulses of ultrasound into tissue using a probe. Reflected pulses are recorded and displayed as an image. There are many types of ultrasound images, but two modes commonly seen in vascular ultrasonography are B-mode and doppler:

**B-mode (Brightness) imaging** is a 2-D, black and white display of tissue acoustic impedances.

**Doppler mode** uses the Doppler effect to measure and visualize blood flow.

**Spectral Doppler** converts frequency shifts from moving blood to velocities using the Doppler equation and displays a “spectrum” of these frequencies as Doppler waveforms.

**Color Doppler** presents velocity information as a color-coded overlay on top of a B-mode image. Duplex ultrasonography is a term commonly used for the

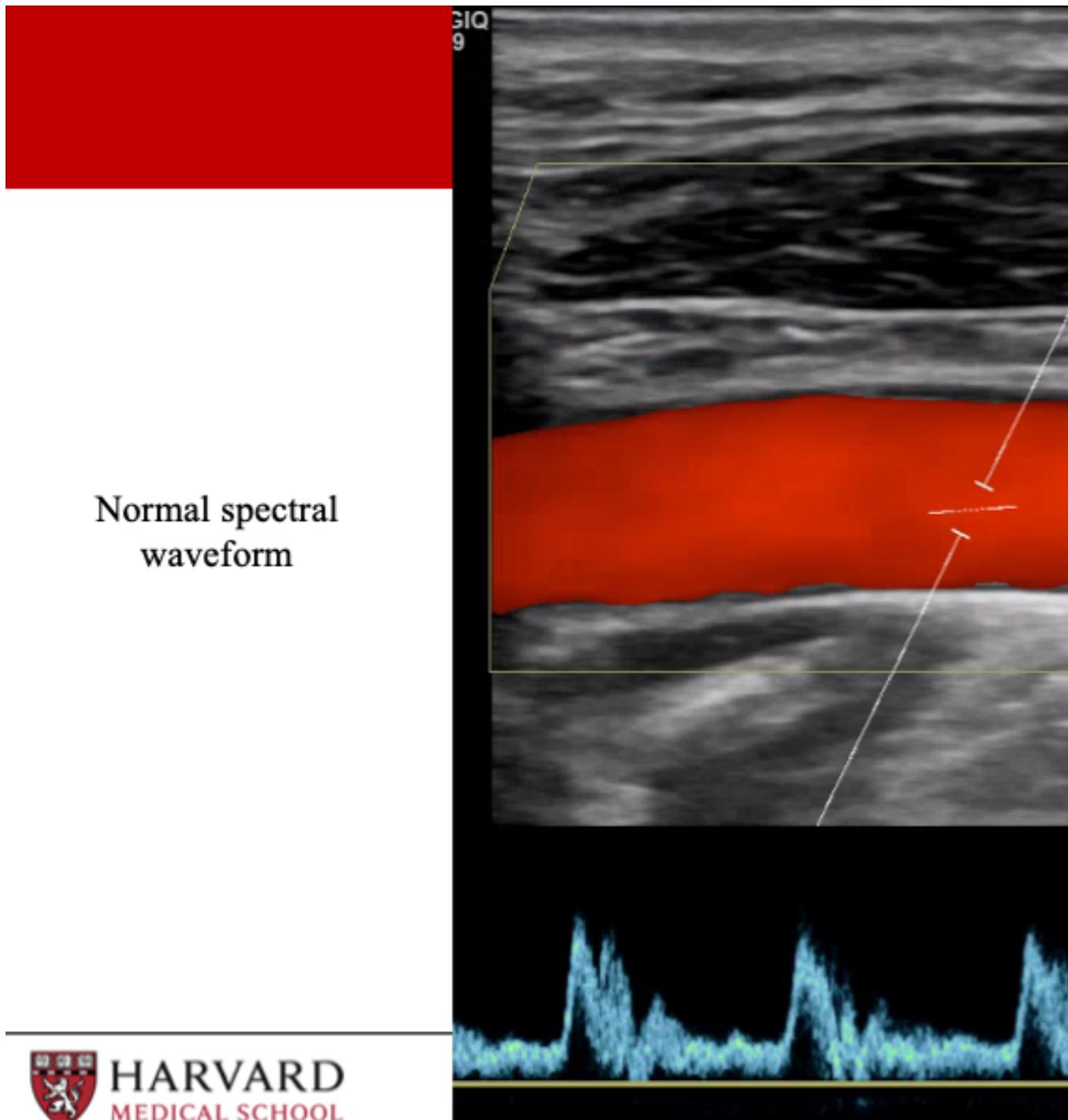
simultaneous presentation of B-mode and doppler data.

**Power Doppler** is less frequently used, but is based solely on amplitude of the doppler signal without giving information on direction. This is particularly useful to detect certain low or abnormal flow states, such as testicular/ovarian torsion, carotid string sign, slow intrarenal flow, supporting occlusion seen on B-mode or color.[Pellerito and Polak, 2019]

### 12.1.1 Waveforms

#### What do normal spectral waveforms look like?

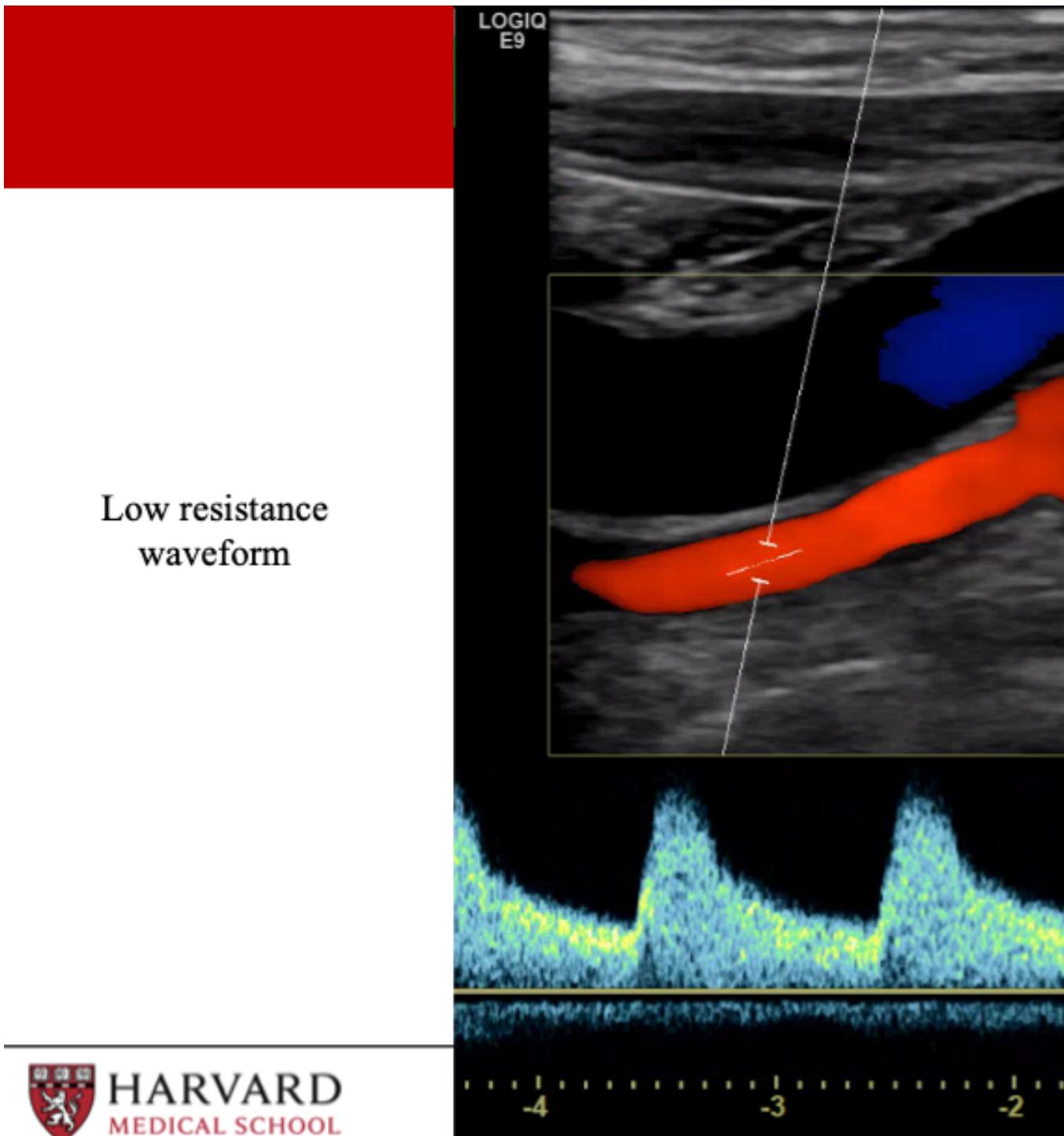
Normal spectral waveforms have a brisk upstroke, sharp peak, rapid downstroke. A “spectral window” under the waveform, that is the black space between the spectral waveform and the 0 velocity axis, represents the absence of lower velocities - indicative of laminar flow within the vessel (Image 1: normal spectrum waveform)



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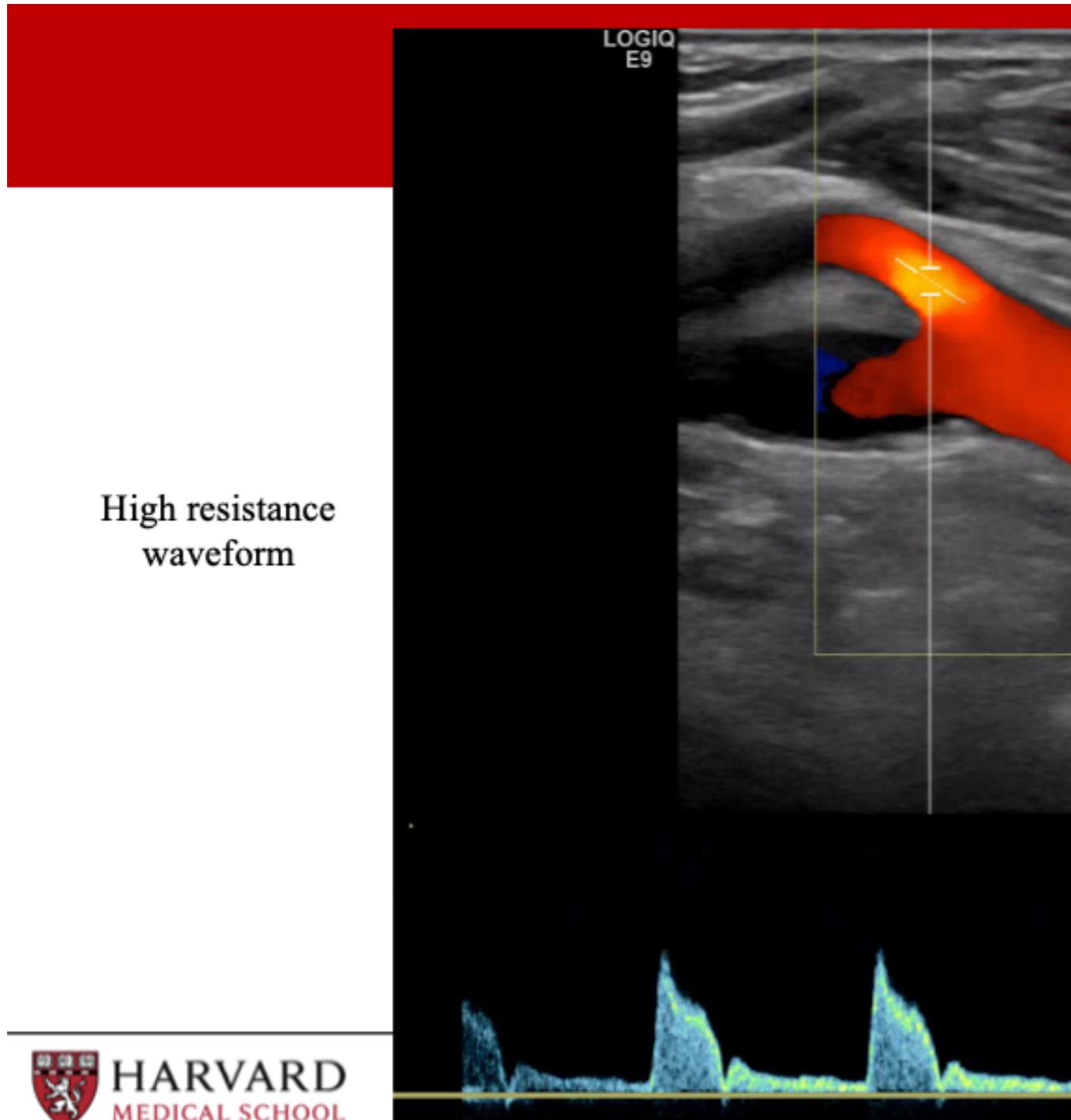
**How can we differentiate low vs high resistance waveforms?**

Waveform profiles change depending upon the nature of the distal vascular bed being supplied. Organs like the brain, kidneys, liver, spleen, peripheral muscle during exercise, and postprandial SMA have constant high metabolic demand, and are therefore low resistance vascular beds. Low resistance waveforms for arteries supplying these organs demonstrate constant forward flow throughout the cardiac cycle because the distal bed being supplied has low resistance leading to high end-diastolic flow.



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In contrast, high resistance waveforms are seen for arteries supplying resting peripheral muscles, fasted mesenteric beds (such as the fasting SMA), and the external carotid artery. High resistance waveforms are characterized by triphasic morphology, with a sharp peak, early diastolic flow reversal, brief forward flow (elastic recoil of the artery), and then no flow during the remainder of the diastolic phase.



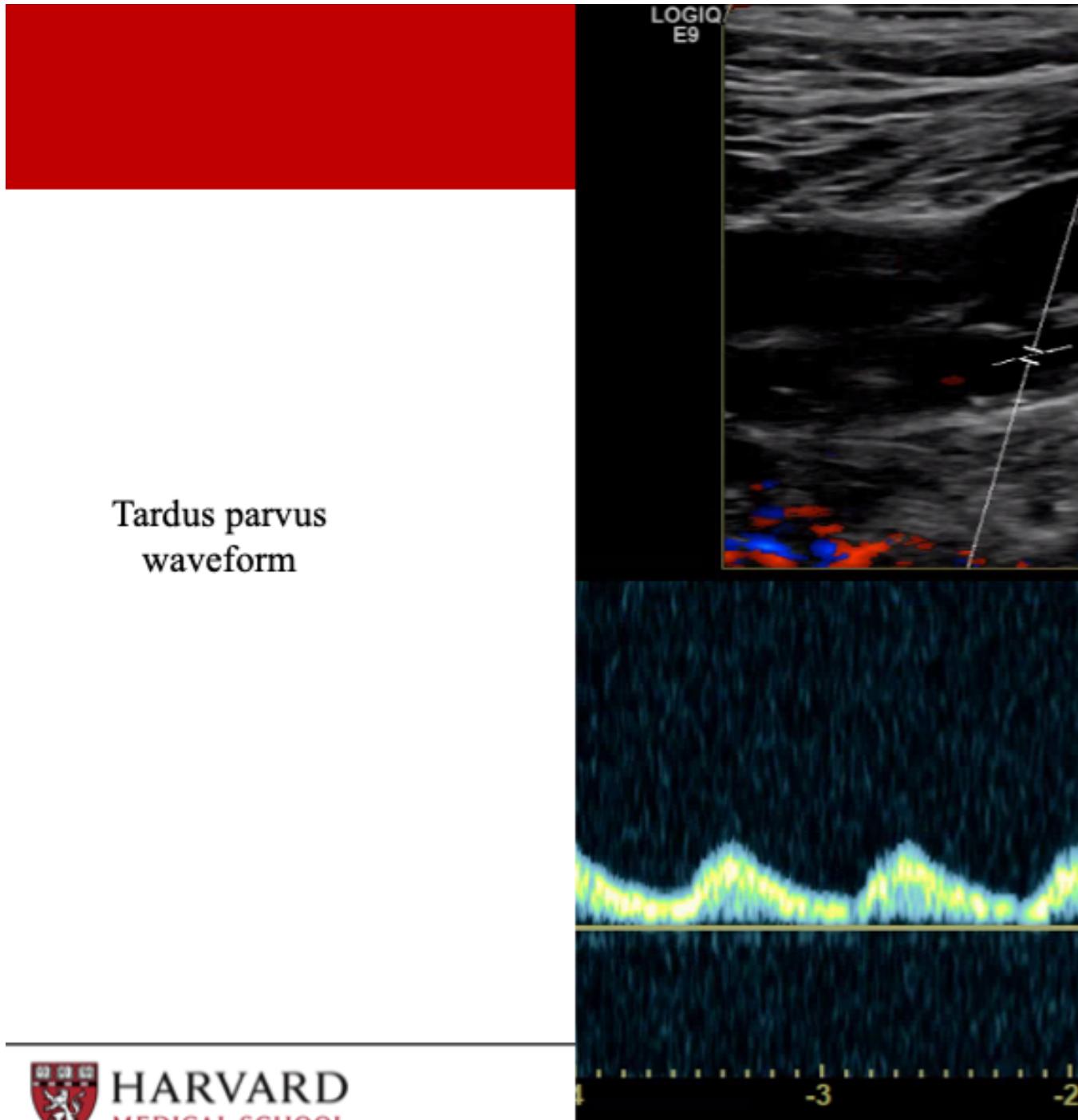
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**How does flow-limiting stenosis change the waveform?**

First let's define Stenosis: A hemodynamically significant stenosis (area reduction >50%) will result in a doubling of velocity from the inflow segment to the area of maximal stenosis (velocity ratio >2).

**Now what do waveforms look like AFTER a flow-limiting stenosis?**

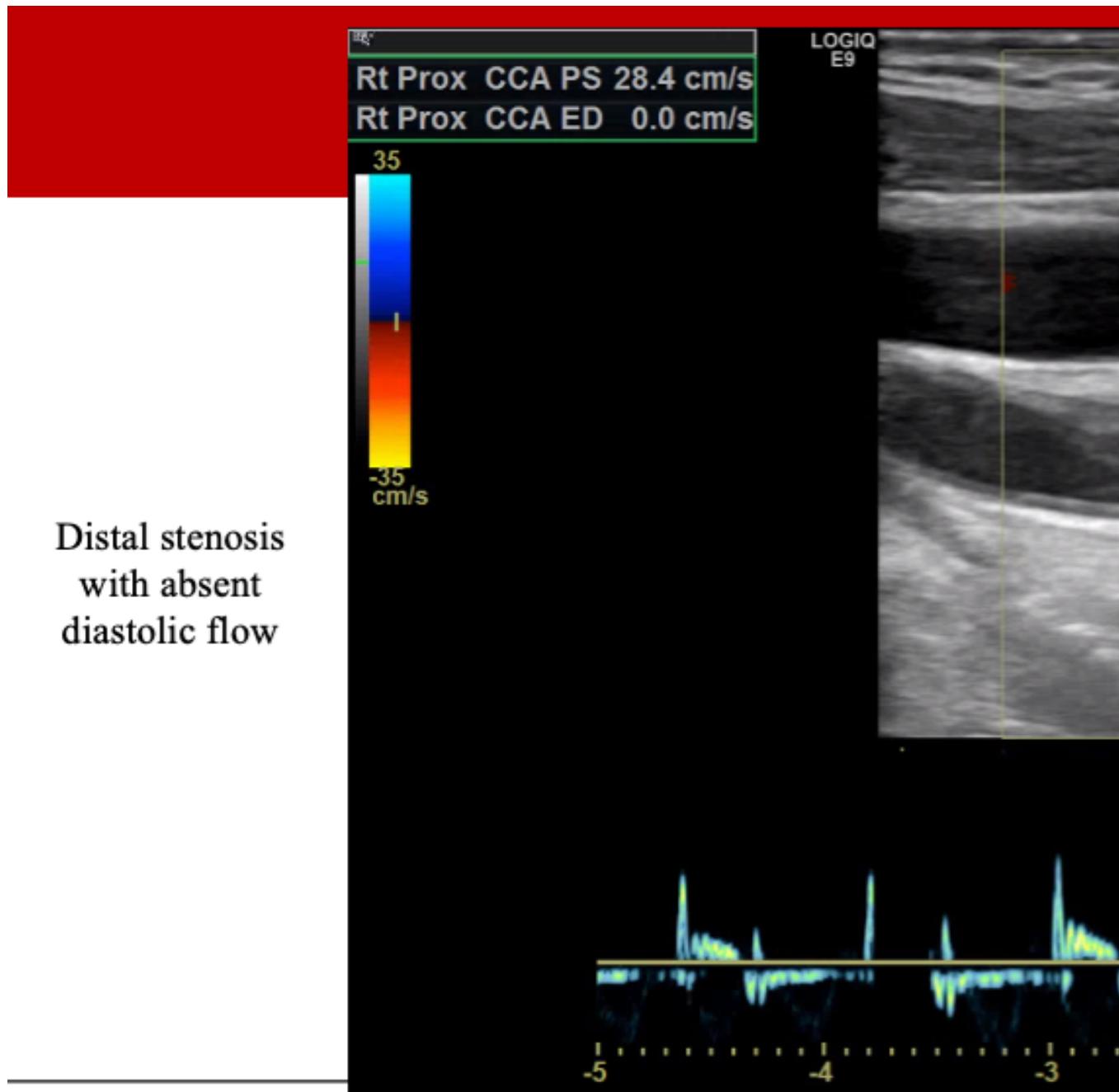
Tardus et parvus refers to a pattern of Doppler ultrasound spectral waveform resulting from arterial stenosis. The tardus et parvus waveform is delayed with prolonged systolic acceleration (tardus) and diminished with a small systolic amplitude and rounded systolic peak (parvus). This phenomenon is observed downstream from the site of stenosis. Tardus parvus in the CFA represents upstream (iliac) stenosis. Tardus parvus in the brachial artery likely represents upstream (subclavian or axillary) stenosis.[Hwang, 2017, Pellerito and Polak, 2019]



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**So what will the waveform look like BEFORE a flow-limiting stenosis?**

Distal stenosis: Distal occlusive disease will result in a high resistance waveform, with absent diastolic flow. (Image 5: Distal stenosis with absent diastolic flow)



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**Are there any considerations we need to understand when assessing a stented vessel with ultrasound?**

Stenting decreases vessel compliance and increases observed velocities. There is no defined consensus, but in-stent restenosis is classified by some as follows:

- >50%
  - SFA -  $V_r > 1.5$  or  $PSV > 200$  [Baril et al., 2009, Kawarada et al., 2013]
  - ICA -  $ICA/CCA > 2.7$  and  $PSV > 220$ .[Lal et al., 2008]
- >80%
  - SFA -  $V_r > 3.5$  or  $PSV > 275$  [Baril et al., 2009, Kawarada et al., 2013]
  - ICA -  $ICA/CCA > 4.15$  and  $PSV > 340$ .[Lal et al., 2008]

It should be noted that restenosis of stents vs vein grafts in the lower extremity will have higher velocity for the same percentage of stenosis. Tardus et parvus waveforms distal to a stent should raise concern that stenosis is hemodynamically significant.[Baril et al., 2009, Kawarada et al., 2013]

**Ultrasound compared to other diagnostic modalities**

Duplex is often adequate to visualize any peripheral arterial bed, even the aorta and iliacs.[Muela Méndez et al., 2018]

- CT - limitations include ionizing radiation, iodinated contrast and is not appropriate in patients with CKD.
- MRA - is performed with gadolinium-based contrast and is not appropriate in patients with ESRD.

Now that we've covered the basics, let's move through by organ-system high-yield vascular lab studies, findings, and pathologies. We'll start with the extracranial evaluation, a highly-tested area of vascular ultrasonography.

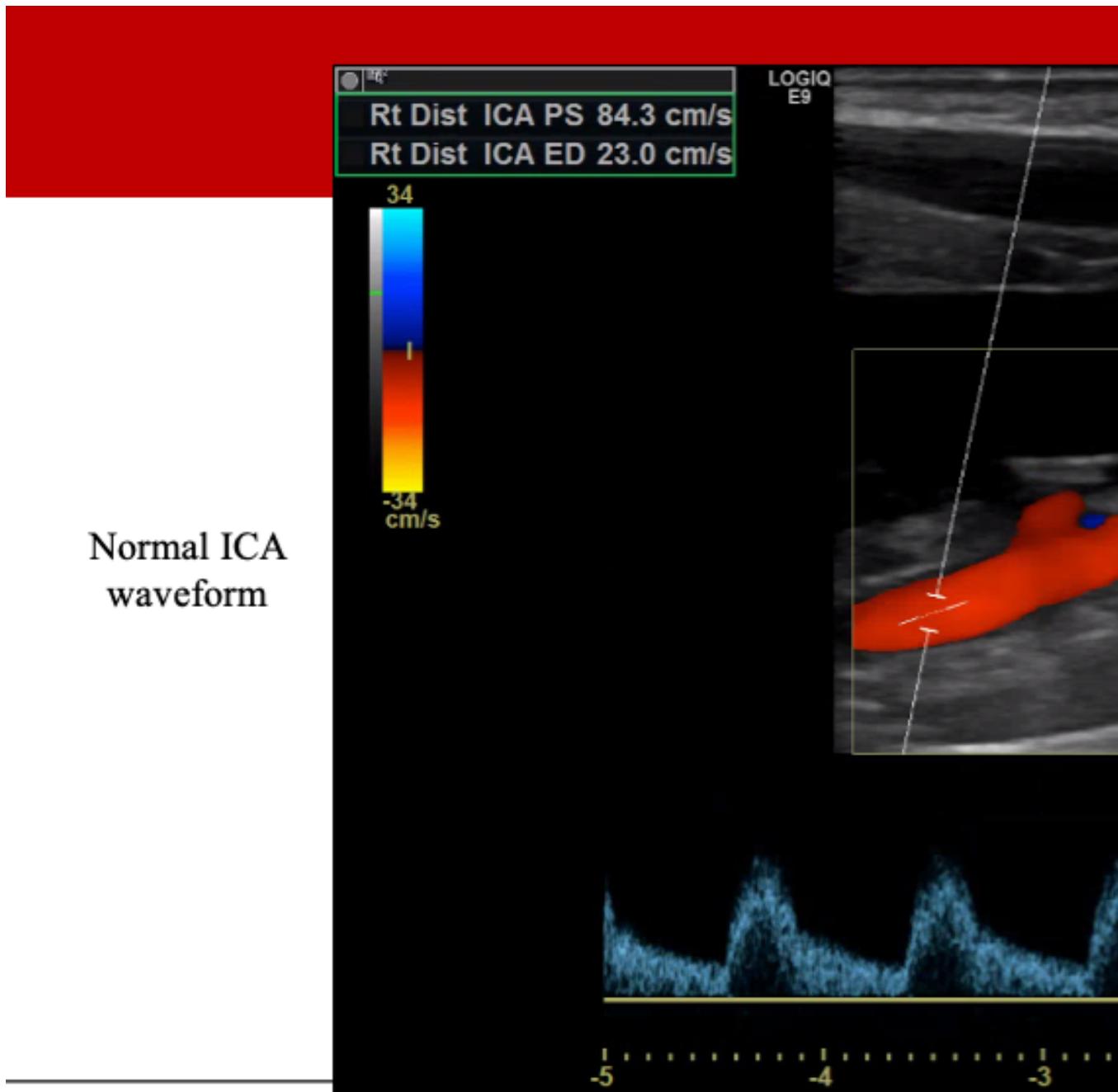
## 12.2 Extracranial

**What does a typical extracranial evaluation involve?**

Examine CCA (2 views), ICA (2 views), ECA, vertebral arteries

**What are normal ICA and ECA waveforms?**

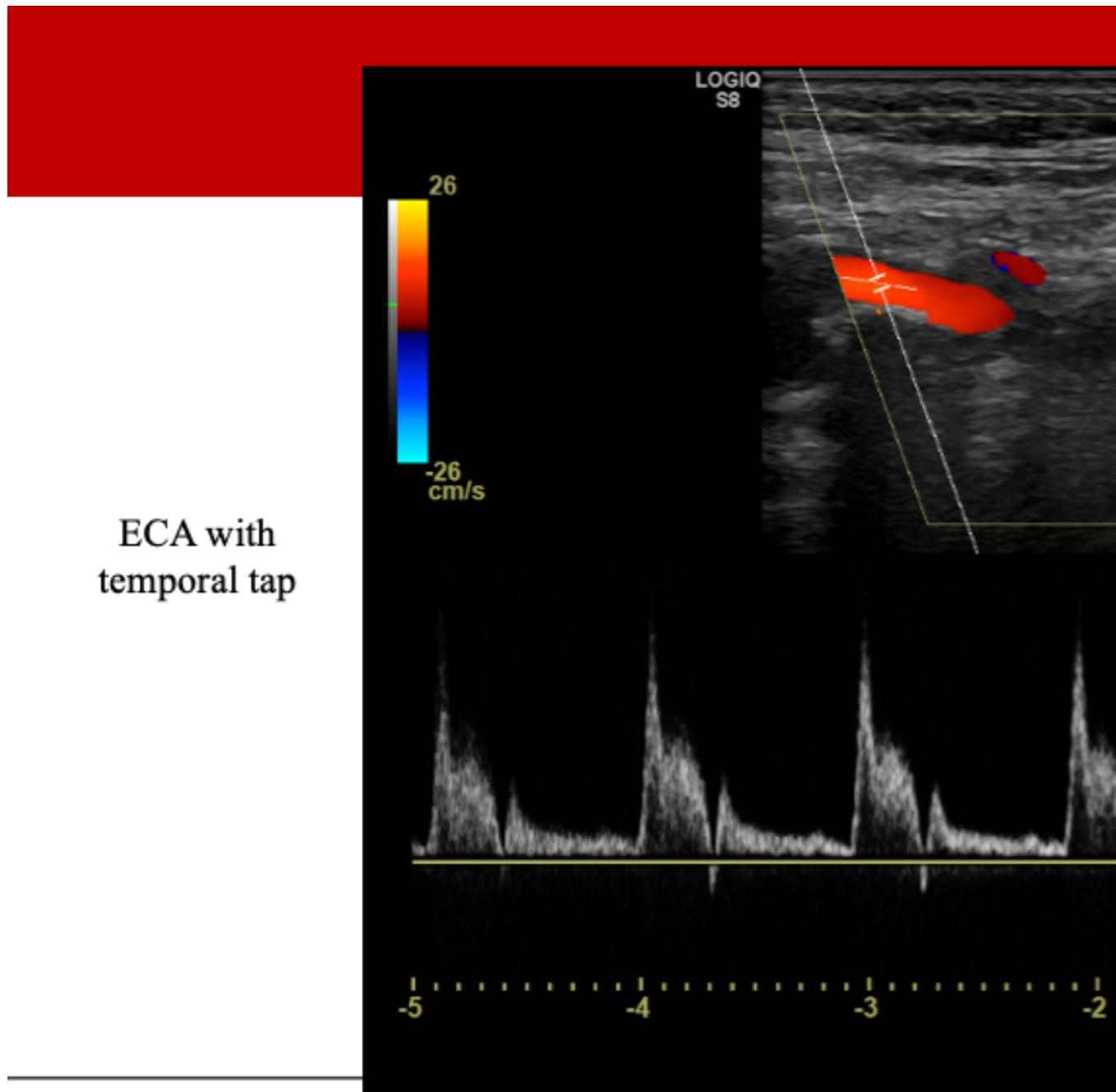
Normal ECA vs ICA waveform: The external carotid artery waveform reflects a high resistance vascular bed. This means minimal diastolic flow. Conversely, the ICA waveform reflects a low resistance vascular bed with antegrade diastolic flow.



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This makes sense, as the ICA is supplying the brain while the ECA is supplying the face. Intuitively, the common carotid artery is a mixture of the ICA and ECA waveform morphologies - like the ICA with forward flow throughout diastole, but less as compared to the ICA due to the high-resistance influence of the ECA.

Another way of differentiating the external and internal carotid arteries is the “temporal tap”. Tapping on the superficial temporal artery (a branch of the ECA) will be transmitted as small pulsations in the diastolic component of the external carotid artery.[Pellerito and Polak, 2019, Size et al., 2013]

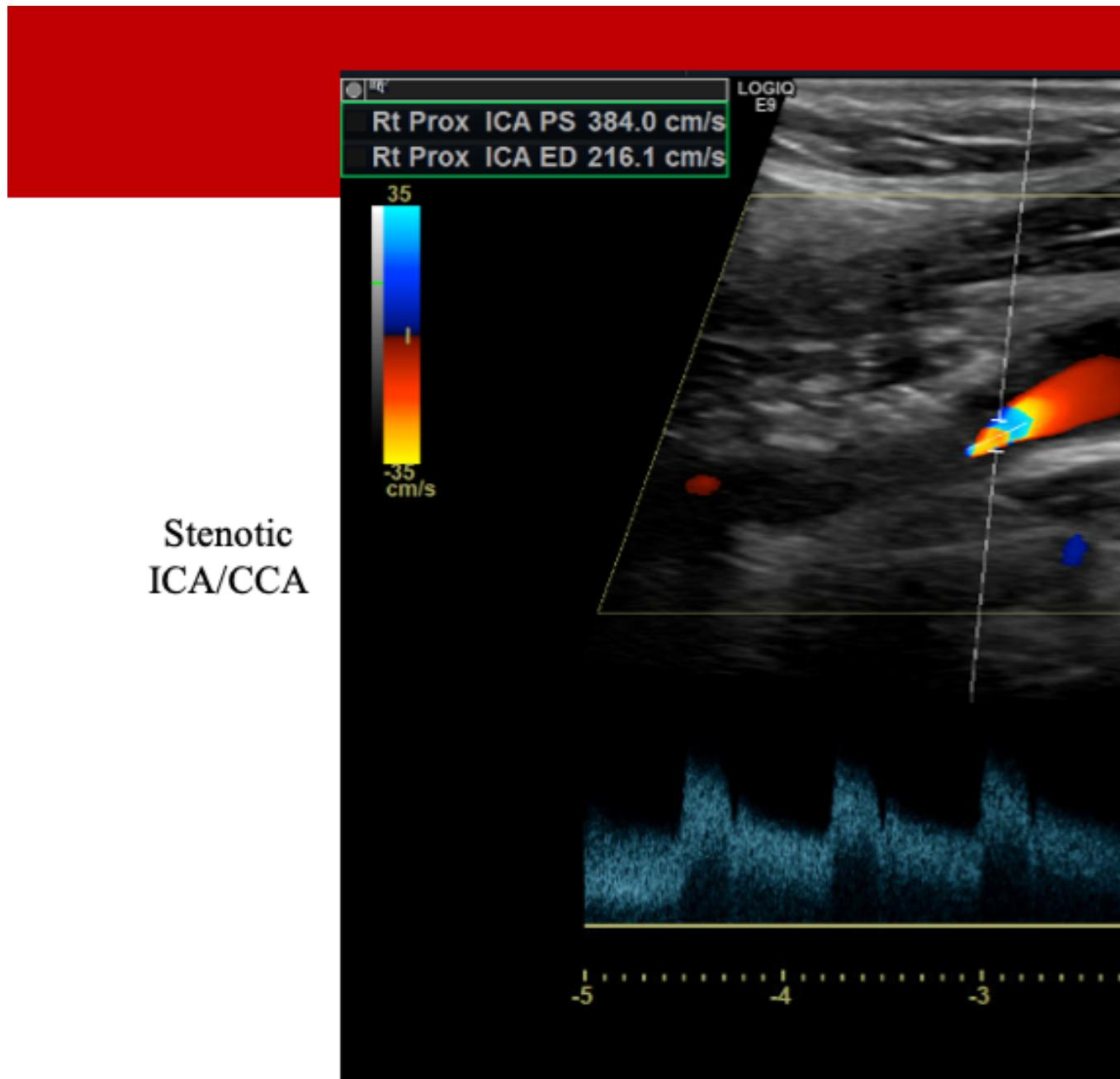


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**Can you talk about diagnostic criteria for ICA stenosis?**

Parameters for ICA stenosis: These are a few numbers that are (unfortunately) essential to memorize for the VSITE and RPVI.

Although criteria differ between guidelines, the Carotid Consensus Criteria, define ICA stenosis  $\geq 70\%$  as a peak systolic velocity  $\geq 230$  cm/sec, EDV  $> 100$  cm/sec, and ICA/CCA ratio  $> 4.0$ . Of note, post-stenting criteria vary from pre-stenting criteria. Stenosis criteria are not clearly defined for the CCA or ECA.



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**When is surgery indicated for ICA stenosis?**

Parameters for when surgery indicated:

Asymptomatic Carotid Atherosclerosis Study (ACAS) >60% stenosis asymptomatic, NASCET >50% stenosis symptomatic

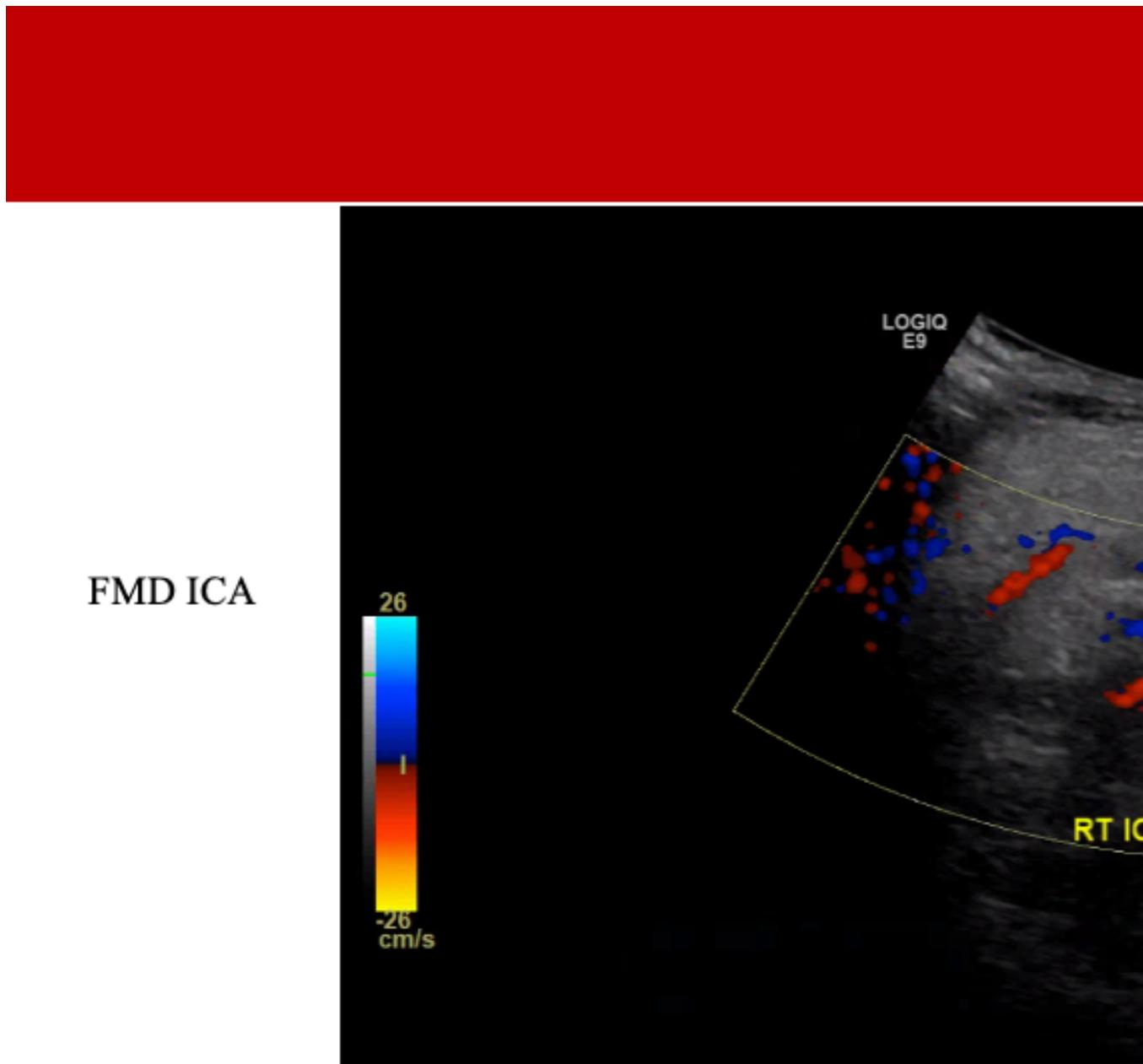
For more see 2.

**Let's talk about other pathologies that can be visualized on extracranial ultrasound:**

Pathologies: Stenosis (plaque), dissection (flap), aneurysms (rare), occlusion (no flow, do not operate), carotid body tumor (splaying of ECA/ICA, fed by ECA branches), FMD.

FMD is frequently encountered on the VSITE/RPVI. How would this appear on the exams?

Fibromuscular dysplasia of the internal carotid arteries affects women more commonly than men. Duplex findings show a “chain of lakes” appearance, demonstrative of multiple septa and small aneurysms. Velocity elevations and increased turbulence in the waveform patterns is typically found on Doppler interrogation.[Olin et al., 2012]



Aortic stenosis: Tardus parvus waveforms in both common carotid arteries can be a sign of more proximal disease of the aortic valve or global myocardial dysfunction.

### How to treat?

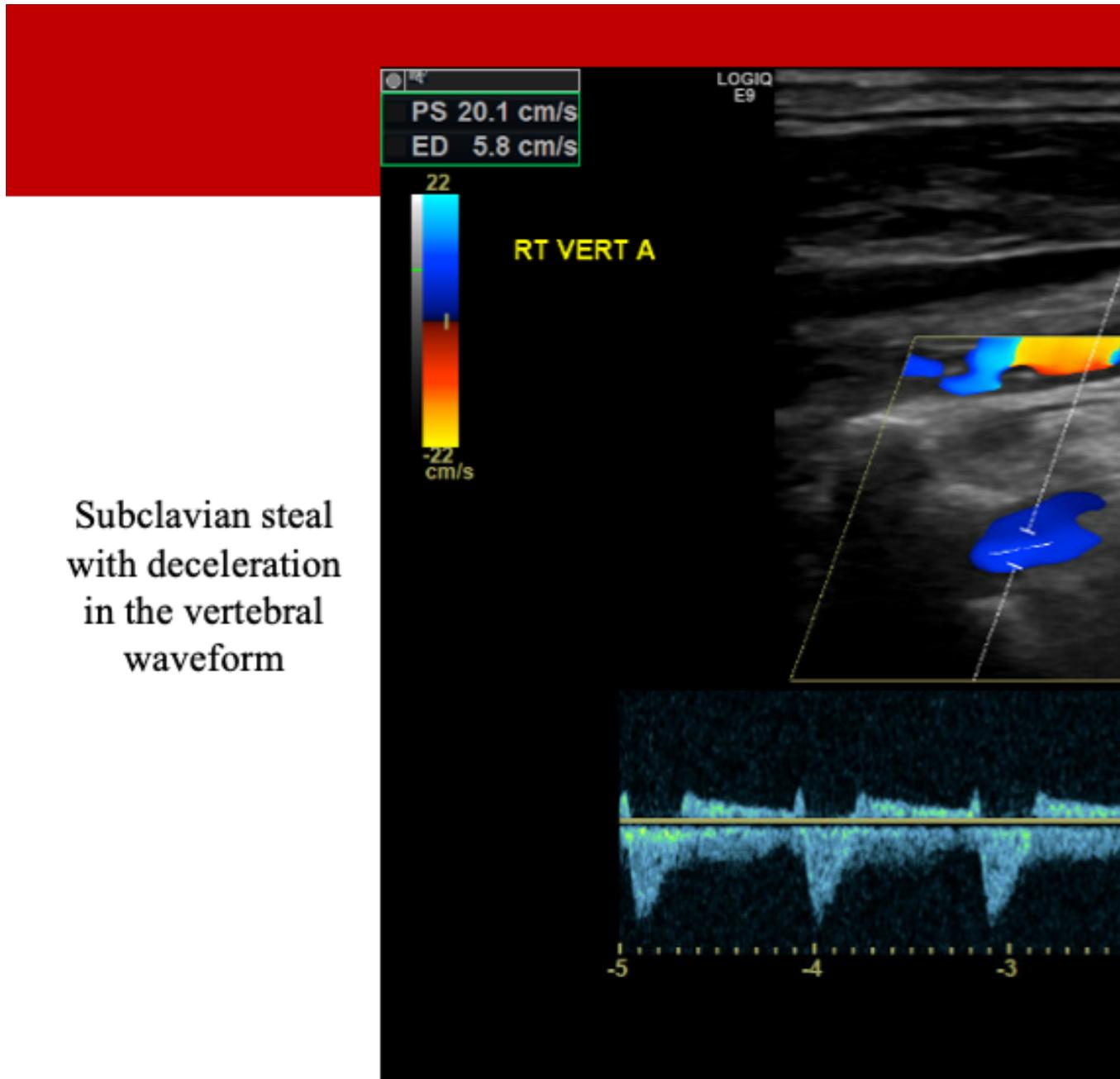
Aspirin if asymptomatic, POBA if symptomatic.

### Are there other frequently tested pathologies demonstrated on extracranial exam?

Subclavian steal: Subclavian steal occurs when a proximal subclavian stenosis or occlusion leads to reversal of vertebral artery flow. This causes “stealing” of blood from the posterior cerebral circulation, and presents as vertebrobasilar insufficiency.

### How does this look on duplex?

Normal vertebral flow looks very similar to ICA: antegrade low resistance waveforms with constant forward flow throughout the cardiac cycle. As subclavian stenosis progresses, one can see mid-systolic velocity deceleration ('bunny ears'), with mild stenosis bidirectional flow can signify “pre-steal” phenomena, and with severe steal, there is a complete reversal of flow in the vertebral artery towards the arm rather than towards the brain.[Kalaria et al., 2005, Mousa et al., 2017]



Subclavian steal  
with deceleration  
in the vertebral  
waveform



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Innominate stenosis: A phenomenon that is related to this, is innominate stenosis. Here again the patient will present with vertebrobasilar insufficiency, indicative of diminished vertebral antegrade flow, but additionally will experience right hemispheric insufficiency secondary to diminished R ICA antegrade flow. The right-side duplex will demonstrate flow reversal in the vertebral artery, abnormal waveforms in the subclavian, as well as steal pattern waveforms in the common and internal carotid arteries. The common denominator for all of these findings is significant disease in the innominate artery.

**What other extracranial arteries of the head and neck can be evaluated separately?**

Temporal arteritis - well visualized with high frequency transducer (10 MHz or greater). Pathognomonic halo effect of anechoic/hypoechoic edematous tissue surrounding the lumen due to concentric inflammation.[Ball et al., 2010, Schmidt, 2014]

### 12.3 Intracranial

So that covers extracranial vascular lab evaluation. What about intracranial?

This is less frequently tested, so we will discuss just a brief overview of views and some of the more commonly tested pathologies related to transcranial doppler (TCD).

**Intracranial:**

- Three primary views: temporal, foraminal (occipital), orbital views
  - **Temporal view:** Used to interrogate PCA, ACA, MCA, and ICA. The MCA, ICA and PCA flow direction is towards the probe, the ACA flow direction is away.
  - **Occipital view:** Basilar and vertebral arteries (both away).
  - **Orbital view:** Ophthalmic and ICA
  - Arteries differentiated by depth. MCA 3-6 cm, everything else deeper.

**What are some frequently tested pathologies that are identified on TCD?**

Indications often tested:

- MCA spasm (severe PSV>200): Can be seen in sickle cell disease with studies indicating a strong correlation between mean velocities of >200cm/s and the rate of stroke in children with sickle cell disease. With blood transfusions, stroke risk can be reduced from >10% to <1% per year. [Bulas et al., 2000]

- Lindegrad ratio is the MCA velocity divided by the distal ICA velocity and can also be used to assess MCA spasm.
  - \* Ratio of 3-6 is indicative of mild-mod vasospasm.
  - \*  $>6$  is severe vasospasm.
  - \*  $<3$  is a sign of hyperemia.[Kirsch et al., 2013]
- Cerebral ischemia during CEA: Comparing transcranial Doppler sonography, near-infrared spectroscopy, stump pressure measurement, and somatosensory evoked potentials, cerebral ischemia was most accurately predicted by the percent change in transcranial Doppler detected middle cerebral artery velocity. Detection of a greater than 50% drop in middle cerebral artery velocity using transcranial Doppler is 100% sensitive for detecting cerebral ischemia.[Moritz et al., 2007b]
  - TCD can also demonstrate microemboli (high spikes of white vertical lines on spectral doppler) during CEA.
- A shower of bubbles in both cerebral hemispheres upon injection with agitated saline is consistent with a R to L cardiac shunt and patent foramen ovale.[Zito et al., 2009]
- Elevated contralateral ICA velocities can demonstrate compensatory flow and collateralization and can be a sign of persistent MCA occlusion.
- Mechanical Compression of the vertebral arteries: TCD will show normal posterior circulation velocities at rest, disappearance of waveform on provocative maneuvers, and increase in velocity once back in a neutral position representing a reactive hyperemia.[Vilela et al., 2005]
- Vasomotor activity can be evaluated as well with TCD. Hypercapnia or breath-holding should result in vasodilation and increased flow. Hypocapnia will result in decreased flow. No change in response to hyper or hypocapnia can show poor cerebral autoregulation.[Müller et al., 1995]
- Reversal of flow in the ophthalmic artery can be a sign of ipsilateral ICA occlusion.[Guan et al., 2013]
- Brain death determination - In patients with cerebral arrest (brain death) TCD will demonstrate a low amplitude, high-resistance waveform with to and fro flow with zero net flow.

## 12.4 Upper Extremity

Having covered head and neck vasculature, let's move on to peripheral vasculature. This is a huge area both on the VSITE/RPVI and in practice. In this section we'll cover first the upper, then the lower extremity vasculature.

**So first, what are characteristics of waveforms in the peripheral vasculature?**

Peripheral: Normal waveforms are indicative of high resistance distal beds, so we would expect triphasic waveforms

**What are normal arterial parameters in the upper extremities?**

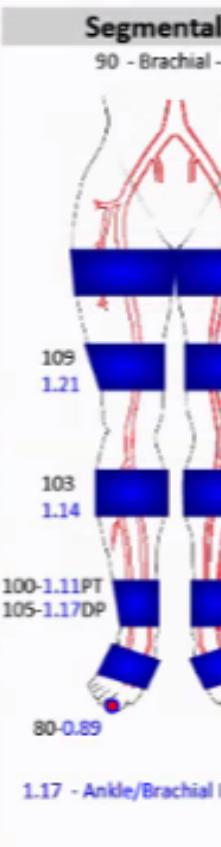
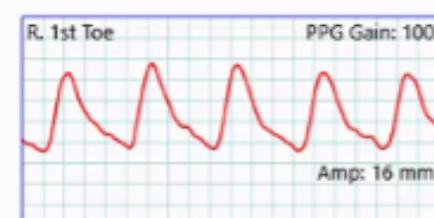
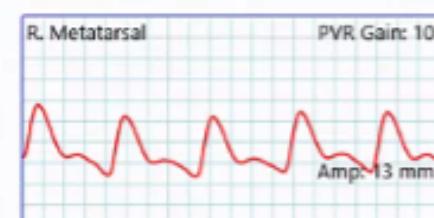
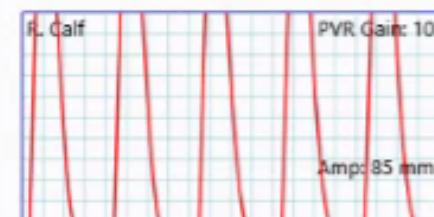
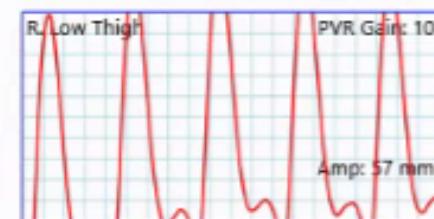
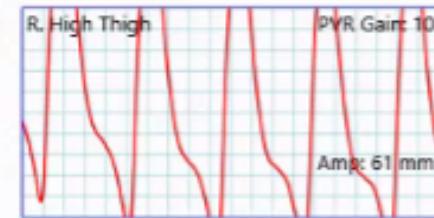
Normal pressure gradient between the right and left brachial pressures is <20 mmHg. Normal finger pressure is >80% of the ipsilateral brachial systolic pressure. Digital brachial index <0.8 is abnormal and <0.5 is diagnostic of ischemia. Absolute digital pressure <70mmHg is abnormal and <60mmHg is diagnostic for ischemia. A gradient between digits of >15 mmHg is considered abnormal. These criteria are used in occlusive disease and steal syndrome.[Chloros et al., 2008, Sen and Tripathi, 2016]

**Let's talk about some of the most frequently tested pathologies, starting with arterial TOS.**

Arterial TOS: Results from compression of the subclavian artery at the level of the first rib within the scalene triangle. Arterial TOS testing is done by placing a sensor, most often photoplethysmography (PPG), on one finger of each hand, recording the resting waveforms and then recording while during maneuvers to evoke arterial compression in the thoracic outlet.[Size et al., 2013]

**Can you tell us a little more about PPG testing?**

Photoplethysmography (PPG) uses an infrared light to illuminate superficial tissue. The reflection is received by a photosensor, and amplitude of the reflected light is proportional to the volume of red blood cells in the sample area. A normal digital arterial PPG has a brisk upstroke with a narrow systolic peak, and a dicrotic notch on the downslope during diastole.[Carter and Tate, 2001]

**Normal PPG**

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Digital PPGs change with progression of peripheral vascular disease. The first changes are a loss of amplitude and loss of the dicrotic notch. More advanced disease findings include a flattened systolic peak and a prolonged upstroke. Significant arterial TOS is suggested when there is a loss or persistent flattening of the digit waveforms during any of the positional changes that can compress the subclavian artery (either with the clavicle, first rib and scalene muscle). However, it should be noted that up to one-third of patients without arterial TOS may have some degree of subclavian artery compression with positional maneuvers.

#### **What are other diseases affecting the upper extremity?**

Raynaud's: Vasospastic disorder characterized by temporary vasospasm. Diagnosis may be assisted by decrease in digital waveforms with immersion of the hand in cold water.

Thromboangiitis obliterans: Is a segmental non-atherosclerotic inflammatory disorder characterized by microthrombosis that primarily involves the small- and medium-sized arteries. Ultrasonography may demonstrate the classical “corkscrew” collateral development at the level of occlusion. TA has a male predominance and first-line treatment is smoking cessation.

#### **12.4.1 Hemodialysis Access**

Before we segue to the lower extremities, this is a good time to discuss an entity frequently tested on the VSITE that constitutes for many vascular surgeons a notable portion of their practice: hemodialysis access, and specifically fistulas.

Fistulas: Ultrasound is one of the key modalities used in identifying suitable anatomy for fistula placement, suitability of a fistula for dialysis, and finally complications of fistulas.

##### **So first, assessment for fistula placement:**

The optimal configuration for an AVF is determined on the basis of vein mapping and noninvasive studies. Veins should measure >3 mm in diameter (>2.5 mm may be acceptable, as veins are likely to dilate under anesthesia), and there should be no arterial inflow stenosis or venous outflow stenosis. Duplex ultrasound arterial imaging can be performed at the same time as vein mapping and can provide important predictors of fistula maturation, such as arterial diameter and flow. The minimal arterial lumen diameter is 2 mm.

##### **How can we tell if a fistula is ready to be used for hemodialysis access?**

Rule of 6's: At six weeks post-creation the diameter of the fistula should be at least 6 mm and the depth no more than 0.6 cm. The flow rate should be at least 600mL/min, and the length of the fistula should be 6 cm to allow for a successful two-needle dialysis.

Brachial artery volume flow rate (VFR) is one of the best measurements to

identify poor fistula maturation.  $>800$  ml/min is ideal, but  $>600$  is often sufficient.[Ko et al., 2015]

#### **What do normal fistula spectral waveforms look like?**

Waveform: The arterial waveform should demonstrate very low resistance throughout diastole. End diastolic velocity should be one half to two thirds of peak systolic velocity in a well-functioning fistula. As a side note, this is also what one would see in an iatrogenic arteriovenous fistula, as between the femoral artery and vein.[Teodorescu et al., 2012]

#### **Let's discuss commonly encountered complications and pathologies identified in association with fistulas:**

Pseudoaneurysms: Pseudoaneurysms commonly occur when a puncture fails to seal and the blood is contained by the surrounding soft tissue. As in other locations, pseudoaneurysms are defined on imaging by a communicating neck between the arterial vessel and pseudoaneurysmal sac with "to-and-fro" waveform at duplex.[Mahmoud et al., 2015] While small pseudoaneurysms can be managed without intervention or surgery, larger pseudoaneurysms, pseudoaneurysms associated with infection or overlying skin changes or bleeding may require excision and repair.[Bandyk, 2013]

Steal syndrome: Hemodialysis-related steal, also known as access-related hand ischemia, which may occur in over half of all patients undergoing access creation. Steal is characterized by retrograde diastolic flow distal to the donor artery. Of note, reversal of flow in and of itself is not sufficient to cause distal ischemia with an intact palmar arch. This is commonly seen after access creation and represents physiologic steal phenomenon, rather than symptomatic steal syndrome. Digital pressures  $<60$  mm Hg are highly sensitive and specific for predicting steal. Patients who have no symptoms (Grade 1 access-related hand ischemia), may be closely monitored without any intervention.

#### **How do we treat more severe steal?**

Flow rate measurements of the fistula can help determine the optimal treatment (banding, revision using distal inflow, distal revascularization with interval ligation, proximalization of arterial inflow or ligation of the fistula). For more on steal, see 11.3.3

#### **So steal can occur in the context of high fistula output, can we talk a bit about low fistula flow, as from stenosis?**

Central venous stenosis: Venous outflow stenosis is the most common reason for arteriovenous graft failure. A low flow rate results in recirculation during the dialysis session. Venous obstruction manifests as arm swelling, and with central venous stenosis may present with collateral development over the upper extremity and chest wall.

Stenosis of fistula: Arterial and mid-graft stenosis can also cause complications, but are less common than venous stenosis. Stenosis on imaging will be rep-

resented by narrowing of luminal diameter of 50% (which correlates to a 75% reduction in cross-sectional area) on b-mode ultrasound, PSV>400, Vr >2.5, high-resistance waveform proximal to the stenosis, and tardus parvus waveform distal to the stenosis.[Bandyk, 2013]

## 12.5 Lower Extremity

**Can you please tell us about some of the diagnostic modalities that are used to examine perfusion in the lower extremities?**

ABIs: Ankle-brachial index measurement requires calculating the ratio of the highest ankle systolic pressure (posterior tibial artery or dorsalis pedis) over the highest brachial systolic pressure. Regardless of whether you're doing the R or L ABI, use the higher arm pressure for both ratios. Normal ABI >0.9, severe disease indicated by ABI<0.5, and CLI by ABI<0.3.

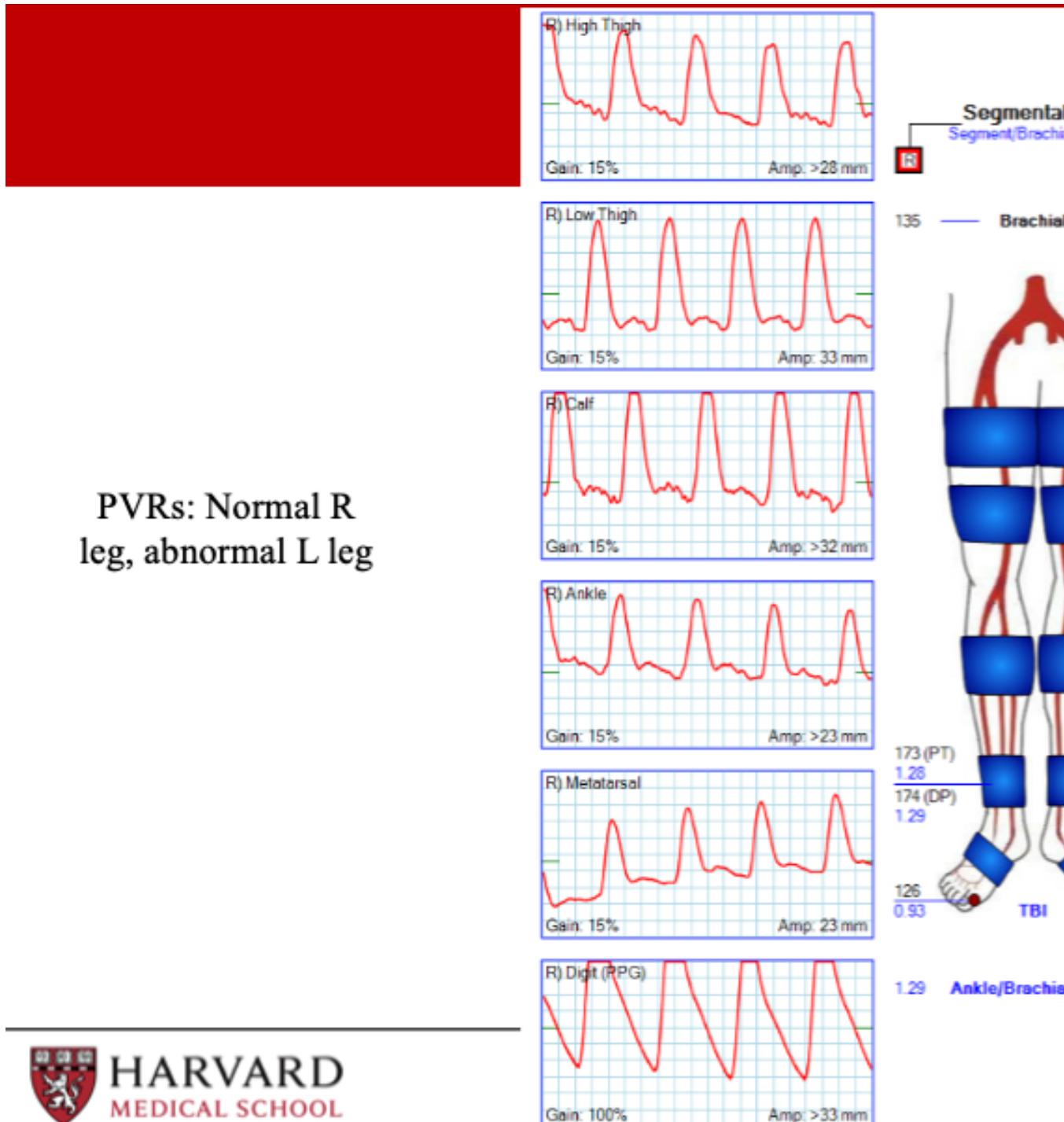
The ankle-brachial index in diabetic patients is frequently unreliable due to incompressibility of the tibial vessels at the level of the cuff secondary to calcification. Consequently, toe pressures are mandatory in all patients with diabetes mellitus. Normal TBI>0.7.

Of note, monophasic waveforms in the brachial artery likely represent proximal occlusive disease in the upper extremity and therefore make the ABI non-diagnostic because the upper extremity blood pressure is not likely not representative of central blood pressure.[Chen et al., 2014]

TcPO<sub>2</sub>: When there is significant tissue loss, preventing TBI measurement, another option is transcutaneous oximetry (TcPO<sub>2</sub>).[Mills et al., 2014a] Transcutaneous oximetry is a non-invasive method of measuring the tissue partial pressure of oxygen through a heated sensor on the skin. A TcPO<sub>2</sub> value of 40 mmHg is the critical value below which wound healing is impaired and ischemia develops.

**Are there other non-invasive ways of determining extremity perfusion?**

PVRs: Pulse volume recordings. Normal PVR waveforms have a rapid upstroke, sharp peak, prominent dicrotic notch and downslope. This typically uses 4 cuffs. High thigh cuff should be 30% greater than brachial pressure, hence a thigh-brachial index of 1.3 is normal. However, ABI/PVRs may not demonstrate significantly abnormal values/waveforms in individuals with single level disease.



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Exercise Test: During exercise testing, patients are placed on a treadmill after baseline resting ABIs are measured. The patient is then asked to walk for 5 minutes or until physical discomfort requires test cessation. The point when the patient reports pain starts is the initial claudication distance and the point where the patient stops is defined as the absolute claudication distance. Diagnostic criteria for a positive test include a drop-in ankle pressure of greater than 20 mmHg from baseline, drop in ABI greater than 0.2 from baseline, or inability of ankle pressures to return to baseline after 3 minutes.[Strandness and Bell, 1964]

**So just to briefly summarize, what are parameters associated with poor wound healing?**

Parameters associated with poor wound healing: Considering these various testing modalities, what are factors associated with poor likelihood of wound healing? An ankle pressure <50 mmHg, ABI < 0.40, TcPO<sub>2</sub> <20 mmHg, or a toe pressure <20 mmHg are considered predictive of non-healing.

**What is the utility of duplex in diagnosing lower extremity occlusive disease?**

Duplex ultrasound is utilized to further delineate the anatomy, level and severity of occlusive disease. Increased PSV can identify the site of a hemodynamically significant stenosis. Diminished waveforms often suggest more proximal disease.

Of note, diffuse narrowing of the SFA can result in tardus et parvus waveforms in the popliteal, without necessarily demonstrating any areas of high velocity.[AbuRahma and Perler, 2022, Pellerito and Polak, 2019]

While duplex ultrasound most easily visualizes infrainguinal arterial disease, it can also be used to evaluate for iliac artery stenosis. Patients with an iliac Vr>2.5 or monophasic/biphasic waveforms in the CFA, suggest that further imaging of the iliac system may be required.[Heinen et al., 2018]

**Ultrasound is frequently used for graft surveillance. Can you talk about graft surveillance parameters?**

Suggested bypass vein graft surveillance with ABI, clinical exam and duplex should begin immediately after surgery and then continues at 3, 6, and 12 months and then every 6 to 12 months thereafter.[Conte et al., 2006, Zierler et al., 2018] A velocity ratio (Vr) is often utilized in graft surveillance and is defined as the peak systolic velocity (PSV) at the site of a stenosis divided by the PSV in a normal vessel segment proximal to the stenosis.

- The highest risk for graft thrombosis, and highest cause for concern, is suggested by PSV >300 cm/s, Vr >3.5, a mid-graft flow velocity <45 cm/s or a drop in ABI >0.15. [Bandyk et al., 1988, Zierler et al., 2018]
- Increased risk grafts have a PSV 180-300 cm/s and a Vr 2.0-3.5.
- Spectral pulse wave doppler is more sensitive than color doppler to identify low flow states or thrombosed grafts.[Hedrick et al., 2005]

- Criteria for prosthetic grafts is higher than vein bypass. [Zierler et al., 2018]

Intervention for recurrent vein bypass graft stenosis early after angioplasty may be best treated with an open revision and patch plasty.[Jongsma et al., 2016]

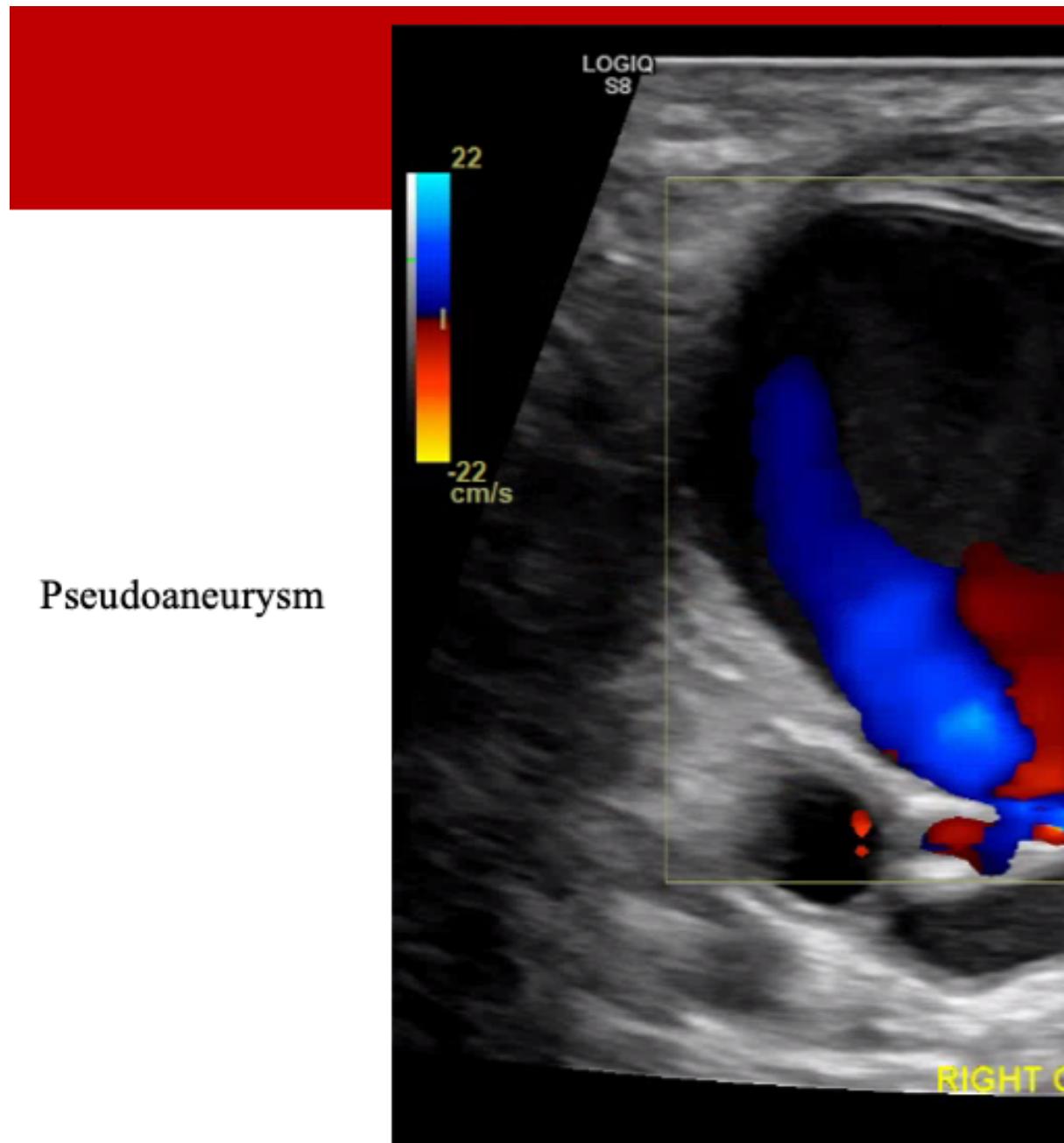
Presenting with new swelling, pain and fluid around a prosthetic graft on duplex ultrasound may be a sign of underlying graft infection. PET and WBC scans may be positive up to 4 months after implantation, so do not provide much utility in the early post-operative period.[Puges et al., 2019]

### 12.5.1 Pathologies

**Let's talk about pathologies frequently encountered in the lower extremities:**

#### 12.5.1.1 Pseudoaneurysms (particularly femoral)

Gray-scale ultrasonography demonstrates a hypoechoic cystic structure adjacent to an arterial supply. Color Doppler typically demonstrates a “yin-yang sign” within the pseudoaneurysm sac. The hallmark ultrasound sign is identification of a neck between the sac and the feeding artery with a “to-and-fro” spectral Doppler waveform measured at the neck. This represents the flow in and out of the PSA during systole and diastole.

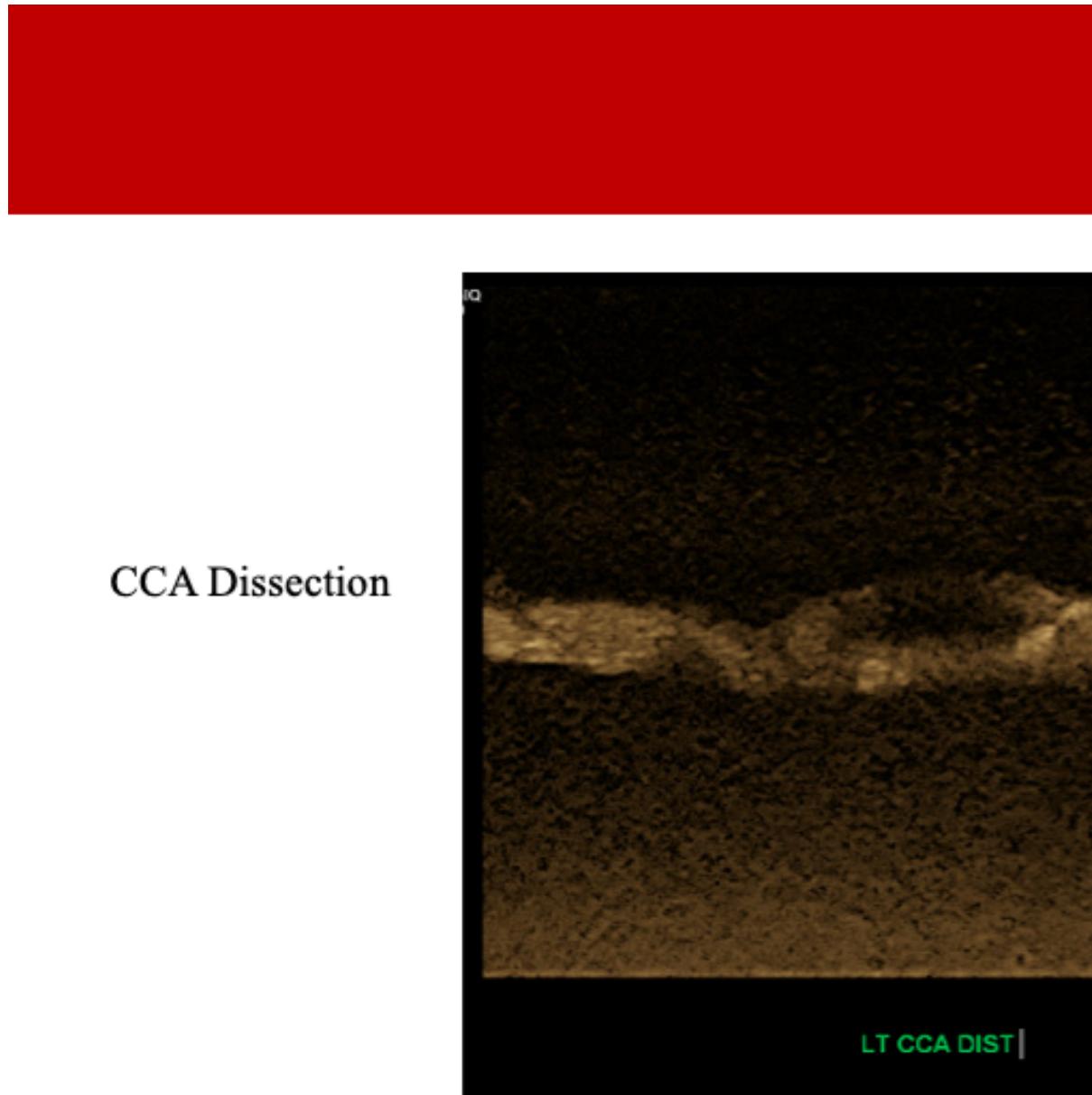


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Anastomotic pseudoaneurysms develop in around 3% of femoral anastomoses and can be associated with arterial wall weakness, endarterectomy, mismatch between artery/graft, dilation/deterioration of graft material, increased/uneven tension of the anastomosis, or underlying infection.[Pellerito and Polak, 2019]

#### 12.5.1.2 Dissections

Characteristic ultrasound findings on color Doppler include a parallel blood-flow channel that separates the true and false lumen, with diminished blood flow distally.[Ge et al., 2015, Kobayashi et al., 2018] Traumatic intimal flaps after knee dislocation can have a normal ABI and be asymptomatic, but they should be followed as they can progress.[Gaitini et al., 2008] Of note, if the false lumen is filled by a thrombus, it may not distinguishable from an intramural hematoma or noncalcified plaque.



CCA Dissection

**12.5.1.3 Buerger's Disease**

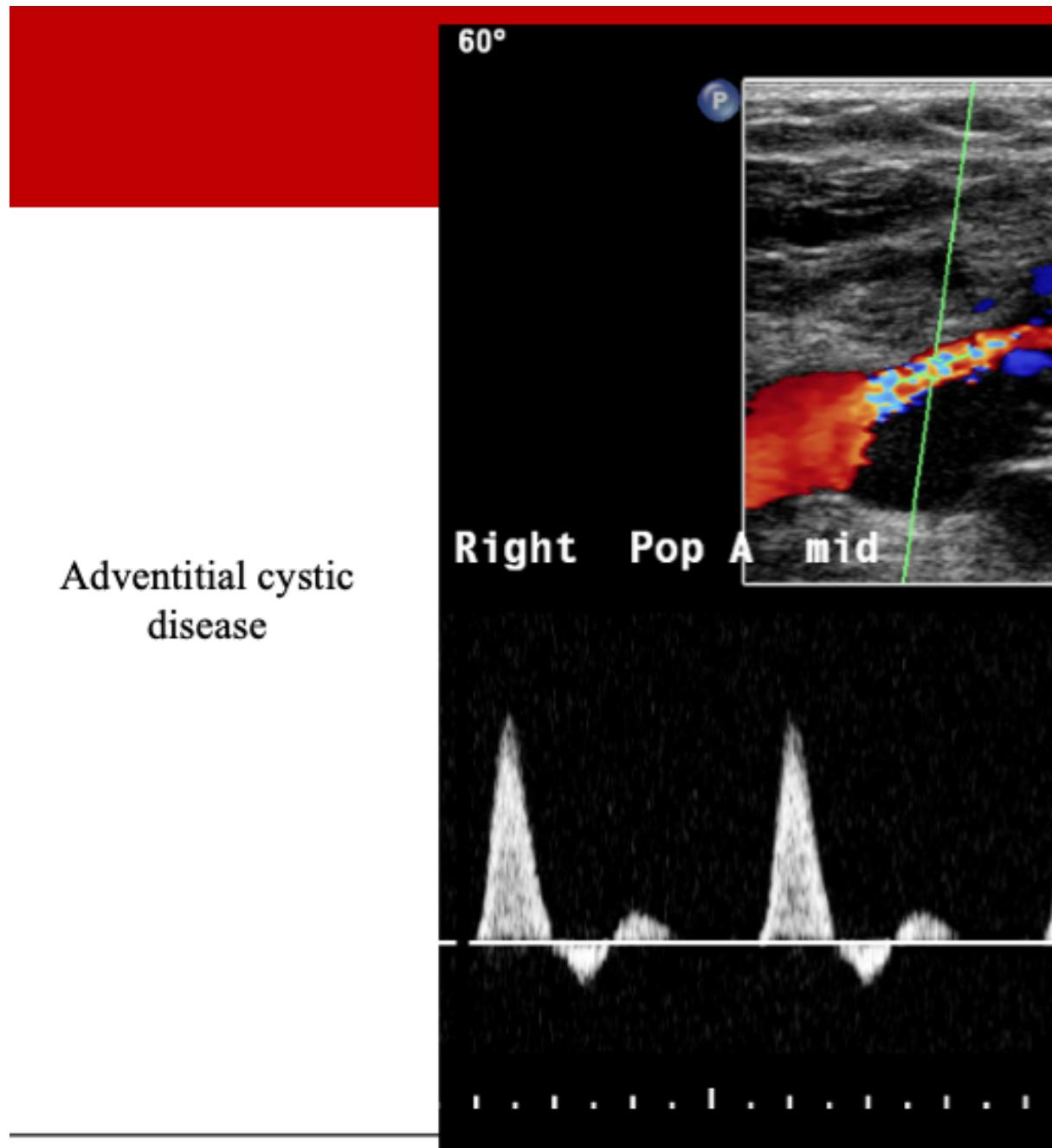
Also known as thromboangiitis obliterans, this is a rare disease, but with characteristic findings on ultrasound of serpiginous/corkscrew collaterals, occlusion of distal calf/pedal arteries, and normal proximal arteries.[Busch, 2011, Fujii et al., 2011]

For more see 3.3.4

There are several disease pathologies that are frequently tested relating to the popliteal fossa. Can you talk briefly about these?

**12.5.1.4 Cystic Adventitial Disease**

This is a rare (but often tested) pathology. Adventitial cystic disease is a non-atherosclerotic etiology of claudication, most often affecting the popliteal artery in the lower extremity and leading to stenosis or occlusion. Duplex imaging of the popliteal artery will demonstrate an anechoic or hypoechoic *intraluminal* region with a smooth contour and stenosis documented by velocity increase on spectral doppler. Angiography will often show a classic “scimitar” sign - a smooth well-defined crescent-shaped defect.[Shaw et al., 2007, Winn et al., 2015]

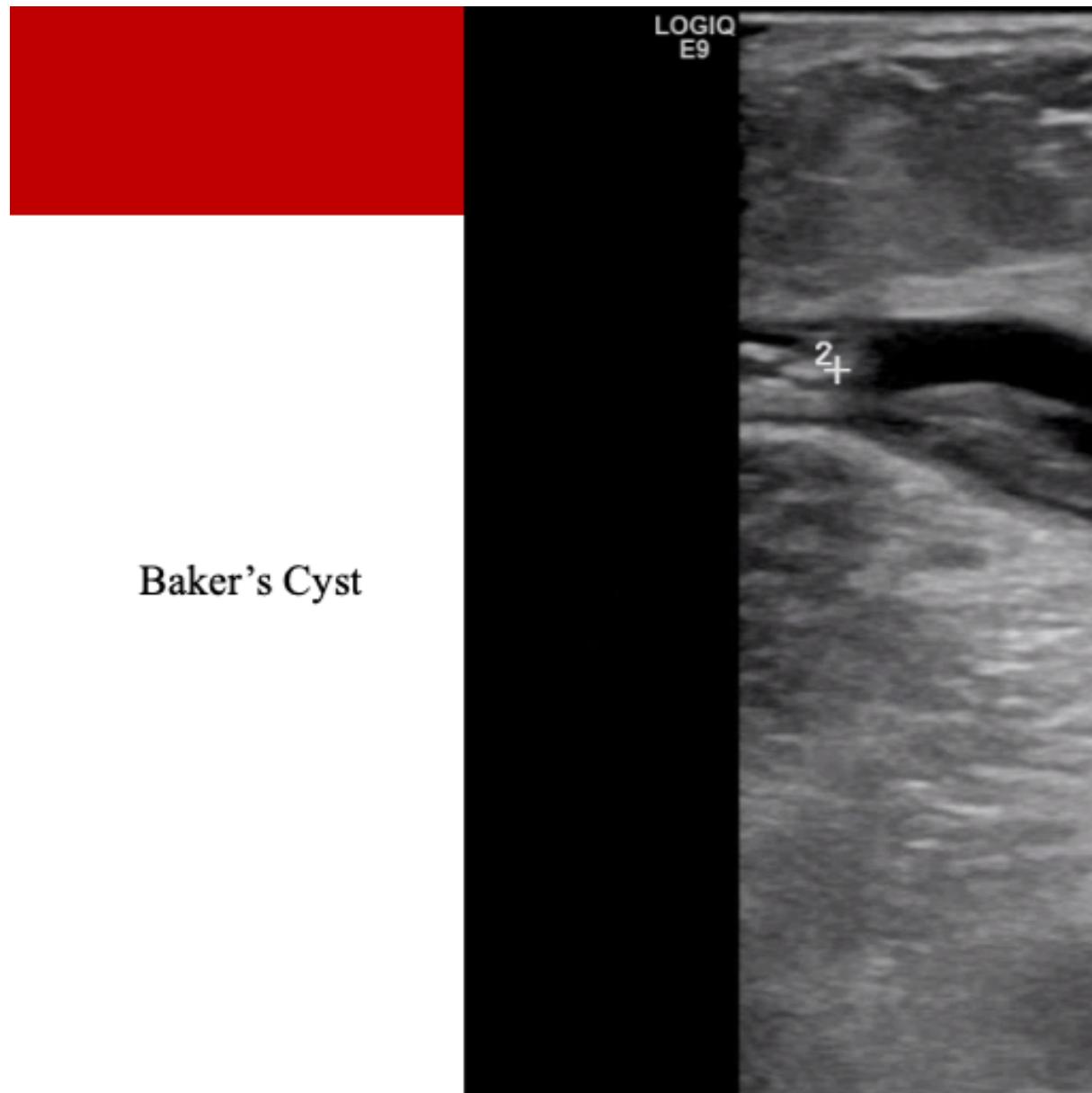


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For more on adventitial cystic disease, see 5.7.2. This is not to be confused with:

#### 12.5.1.5 Bakers Cyst

A benign, cystic structure found in the popliteal fossa and arising from the joint capsule. Flexion of the knee may result in compression of the popliteal artery by the cyst. On b-mode ultrasound, a well-defined, anechoic cystic structure with a ‘neck’ extending into the joint space between the semimembranosus tendon and the medial head of the gastrocnemius will be identified.



Baker's Cyst



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TRV LT

Popliteal entrapment syndrome: decrease in ABI or loss of distal pulses with passive dorsiflexion or active plantar flexion of the foot caused by compression of the popliteal artery by the gastrocnemius.

## 12.6 Abdominal

### 12.6.1 Aorta

**Abdominal Aortic Aneurysms (AAA):** Ultrasound screening of a AAA is non-invasive, accurate, and cost-effective.[Chaikof et al., 2018a]

- Sensitivity of 98% and specificity of 99% for AAA diagnosis.[Wilmink et al., 2002]
- Aneurysm is measured in transverse view, anterior to posterior diameter, outer wall to outer wall.[IAC, 2021] Size criteria for AAA repair is 5.5 cm men, 5.0-5.5 cm in women.
- Can be limited by bowel gas or obesity - so recommend fasting.
- For more details, see4.

#### Can ultrasound be used for graft surveillance s/p EVAR?

Surveillance color duplex ultrasound is safe if CT imaging at 1 year exhibits no sac growth, graft migration, or endoleak (or stable type II endoleak). Contrast enhanced duplex, in particular, has a high sensitivity and specificity to detect endoleaks.[Abraha et al., 2017, Kapetanios et al., 2019, Chaer et al., 2009]

Type II endoleak classification is subdivided on surveillance duplex. Type IIa has a single inflow vessel and a “to-and-fro” appearance, whereas Type IIb has multiple vessels and maintains a monophasic flow. Type IIa endoleaks with velocities greater than 100cm/sec are not likely to spontaneously resolve and may be resistant to therapy.[Arko et al., 2003, van Marrewijk et al., 2004]

Type II endoleaks with stable sac size can continue surveillance with color duplex.[Chaikof et al., 2018a, Karthikesalingam et al., 2012, Chaer et al., 2009] Any findings concerning for growth, endoleak or flow in the sac should have confirmatory CTA performed.[Maleux et al., 2017]

Surveillance duplex does not reliably detect graft migration given challenge of renal arteries visualization in a long axis view of the aorta.[Busch et al., 2009]

Velocities within stents are elevated from baseline due to stent rigidity, but a limb velocity >300cm/s with a Vr >3.5 is associated with a high risk of limb occlusion from stenosis or kinking and should undergo further investigation or intervention.[Chaikof et al., 2018a, Blom et al., 2012]

#### What other pathologies can be identified on ultrasound evaluation of the abdomen?

CIA aneurysms: SVS defines CIA aneurysms as any permanent, localized dilatation of the iliac artery  $>1.5$  cm in diameter (diameter 1.5x the normal diameter)

Para-anastomotic pseudoaneurysms: As previously discussed (reported in up to 0.5% to 10% of cases)

Penetrating aortic ulcers: Describes an ulcerating atherosclerotic lesion that penetrates the intima and progresses into the media. Associated with atherosclerotic plaque on ultrasound

Dissections: (as already discussed) a dissection flap is usually identified and color flow demonstrates dual channels (true and false lumens). Turbulent flow patterns are frequently encountered.

### 12.6.2 Mesenteric Vasculature

So far we have steered clear of the mesenteric vasculature. But this is an area frequently encountered on exams. Let's start with mesenteric vessel stenosis.

**Celiac and SMA stenosis:** May present as chronic mesenteric ischemia. PSV  $>275$  cm/s in the SMA or  $>200$  cm/s in the celiac artery indicates  $\geq 70\%$  stenosis. Normal SMA Doppler waveforms in the fasting patient show high resistance waveform, PSV  $<275$  cm/s and no spectral broadening. In the postprandial state, the waveform becomes low resistance, with a slightly increased PSV and little to no spectral broadening. Significant SMA stenosis may be differentiated by the presence of spectral broadening and elevated PSV ( $>275$  cm/s) and EDV ( $>55$  cm/s).[AbuRahma et al., 2012, Oderich and Ribeiro, 2019b] Distal to the stenosis, one would expect a tardus parvus waveform. EDV  $>45$  in SMA or  $>55$  in celiac are predictive of stenosis (would expect higher diastolic flow in celiac trunk given low resistance vascular bed of liver and spleen).

Reversal of flow in the hepatic artery and mildly elevated SMA velocities may indirectly signify severe stenosis or occlusion of the celiac artery.

Note that aberrant anatomy with the hepatic artery arising from the SMA, will result in the proximal SMA demonstrating a low resistance waveform, even in the fasted state.[Pellerito and Polak, 2019]

Mesenteric duplex is still valid even if the patient has not fasted. The images may be technically more difficult to acquire due to bowel gas, however if velocities can be obtained, you are likely to see elevated PSV and low resistance waveform in the SMA. The EDV will also be elevated and should be interpreted with caution.

**IMA Stenosis:** PSV  $>200$  cm/s or aortic ratio  $>2.5$  has a high sensitivity for hemodynamically significant stenosis. However, this is rarely clinically significant and does not require treatment.

**What other mesenteric vessel pathologies may be identified on vascular ultrasound?**

Dissections: Rare without concomitant aortic dissection, describe only in case reports.[Gouëffic et al., 2002, Oglesby and Sorrell, 2006]

Aneurysms: Rare. Repair >2 cm celiac, hepatic, SMA aneurysms and >3 cm splenic and renal.

Median arcuate ligament syndrome: MALS can cause significantly elevated velocities at the origin of the celiac artery. Testing is for reversible mechanical compression, as opposed to a fixed lesion from atherosclerotic disease. During deep inspiration or in the upright position the MAL is elevated and celiac velocities should normalize if stenosis is secondary to MALS.[Tembey et al., 2015]

Hepatic artery thrombosis: Mesenteric duplex is often performed in liver transplant patients. Hepatic artery thrombosis most often occurs within the first 30 days after liver transplantation, with an incidence of 1.5-9% and a mortality rate of 75%. [García-Criado et al., 2009, Sanyal et al., 2012]

### 12.6.3 Portal Vein

**So I recognize that we are jumping ahead here discussing venous circulation, but as this represents another component of a mesenteric vascular exam, let's discuss portal venous ultrasonography.**

Normal portal venous flow is hepatopetal (toward the liver), whereas abnormal portal venous flow is hepatofugal (away from the liver). (The root “fugua” means to flee, or flight). Flow should be in the same direction as the hepatic artery.

Other abnormalities that may be visualized are portal vein thrombosis - often associated with portal venous hypertension in patients with chronic cirrhosis, hepatitis, or hepatocellular carcinoma. Acute portal vein thrombosis on ultrasound demonstrates dilatation of the portal vein with hypoechoic intraluminal thrombus. Chronic portal vein thrombosis is characterized by a contracted vein with heterogeneous/hyperechoic echoes and may be associated with collateral formation.[Chawla and Bodh, 2015, Nouvini and Hapani, 2013]

Mesenteric duplex can also assess for functioning of TIPS (transjugular intrahepatic portosystemic shunt). A sign of a failing TIPS is portal vein peak systolic velocity less than 30cm/s.

### 12.6.4 Renal arteries

**Awesome, and so before jumping fully into venous circulation, let's complete our discussion of abdominal ultrasound evaluation with a discussion of the renal vasculature.**

Renal Pathologies: ostial (atherosclerotic) vs mid-artery (FMD)

Atherosclerotic stenosis: PSV > 285 cm/s and Renal-aortic ratio (RAR) >3.5, suggest >60% stenosis.[Schäberle et al., 2016] However aortic PSV needs to be

within normal range (40-100) to be appropriately interpreted.

The renal resistive index (RRI) is calculated as the RA (PSV-EDV)/PSV. The RRI ( $>0.8$ ) is an indicator of intrinsic parenchymal renal disease. RRI has been used for assessment of transplant renal allograft rejection, decision making for treatment of renal artery stenosis and to evaluate progression of CKD. Diabetic nephropathy will demonstrate elevated RRI, signifying parenchymal disease.[Viazzi et al., 2014] One study showed it was associated with poor renal and overall outcome in critically ill patients.[Le Dorze et al., 2012]

FMD: Fibromuscular dysplasia (FMD) will demonstrate tortuous renal arteries with turbulent flow and increased PSV of the mid-distal portions. Beading may be difficult to see on ultrasound (compared to the extracranial carotid). Atherosclerosis disease is primarily ostial in nature.

In-stent restenosis: Higher cut off values are required to determine in stent restenosis.[Chi et al., 2009, Schäberle et al., 2016]

Renal artery aneurysms: Can see a yin-yang sign. [Gutta et al., 2008, Ham and Weaver, 2014]

## 12.7 Venous

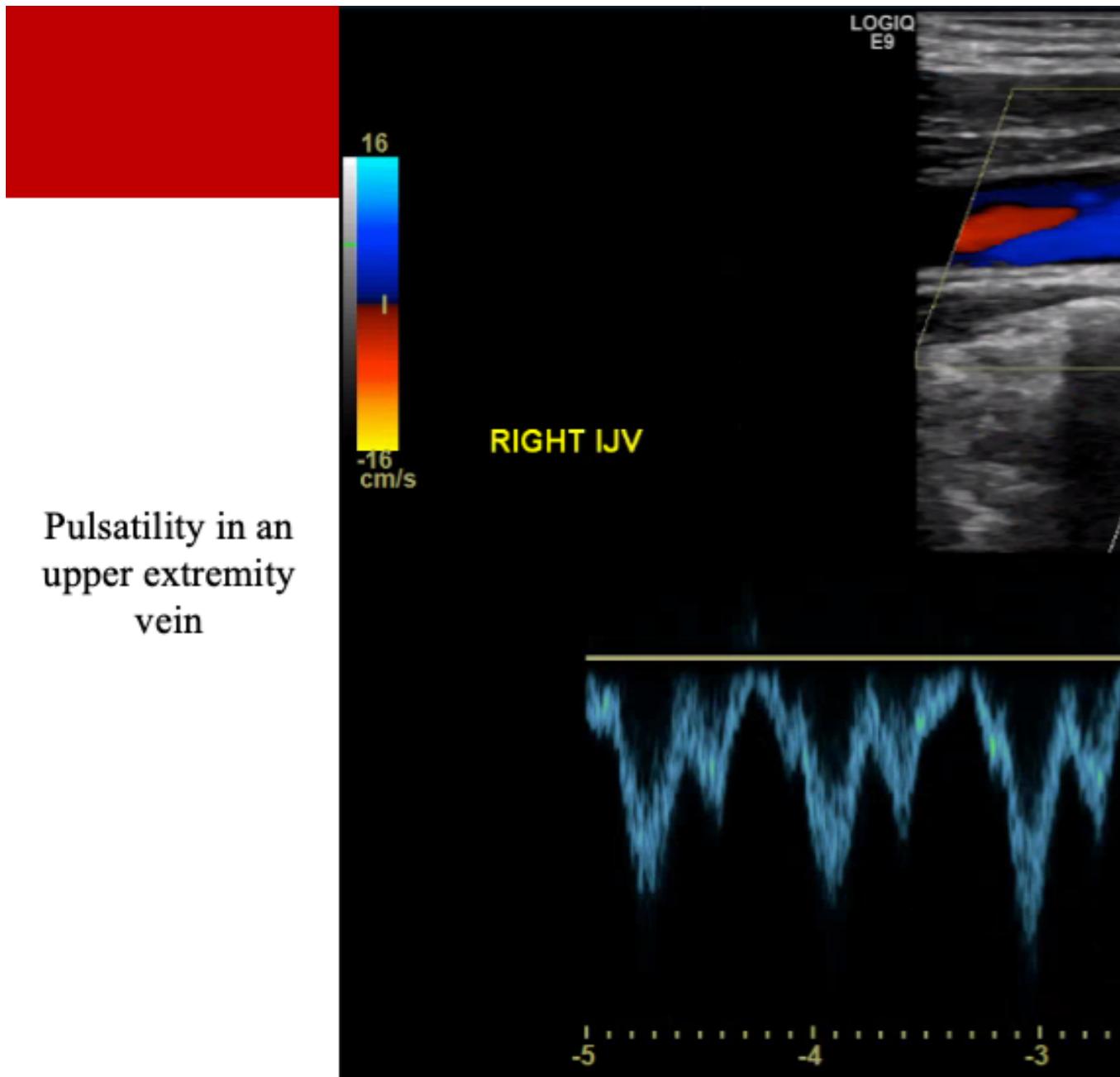
**Venous Exam:** Uses a linear transducer. Complete deep venous reflux duplex examination includes color and pulsed wave spectral doppler imaging. Spontaneous Doppler waveforms as well as provocative maneuvers are recorded in the common femoral, femoral, popliteal, and tibial deep veins. Superficial veins (GSV, SSV, and perforator veins) are evaluated with provocative maneuvers to test valve competency. Diameters are also included for superficial veins. Transverse B-mode images are used for vessel compression (as when looking for thrombus) to ensure that the vein is fully compressible under probe pressure as opposed to simply slipping out of view, as may occur in longitudinal views. Reflux exam should be performed with a patient standing, with assessment performed on the non weight-bearing leg.

**We've talked extensively about normal arterial waveforms; what do normal venous waveforms look like?**

Normal venous waveforms: Normal flow patterns of iliac and femoral veins demonstrate phasicity and should augment with distal compression. In the upper extremity central veins, doppler waveforms normally demonstrate pulsatility and phasicity.

**What does this mean? Pulsatility, phasicity, and augmentation?**

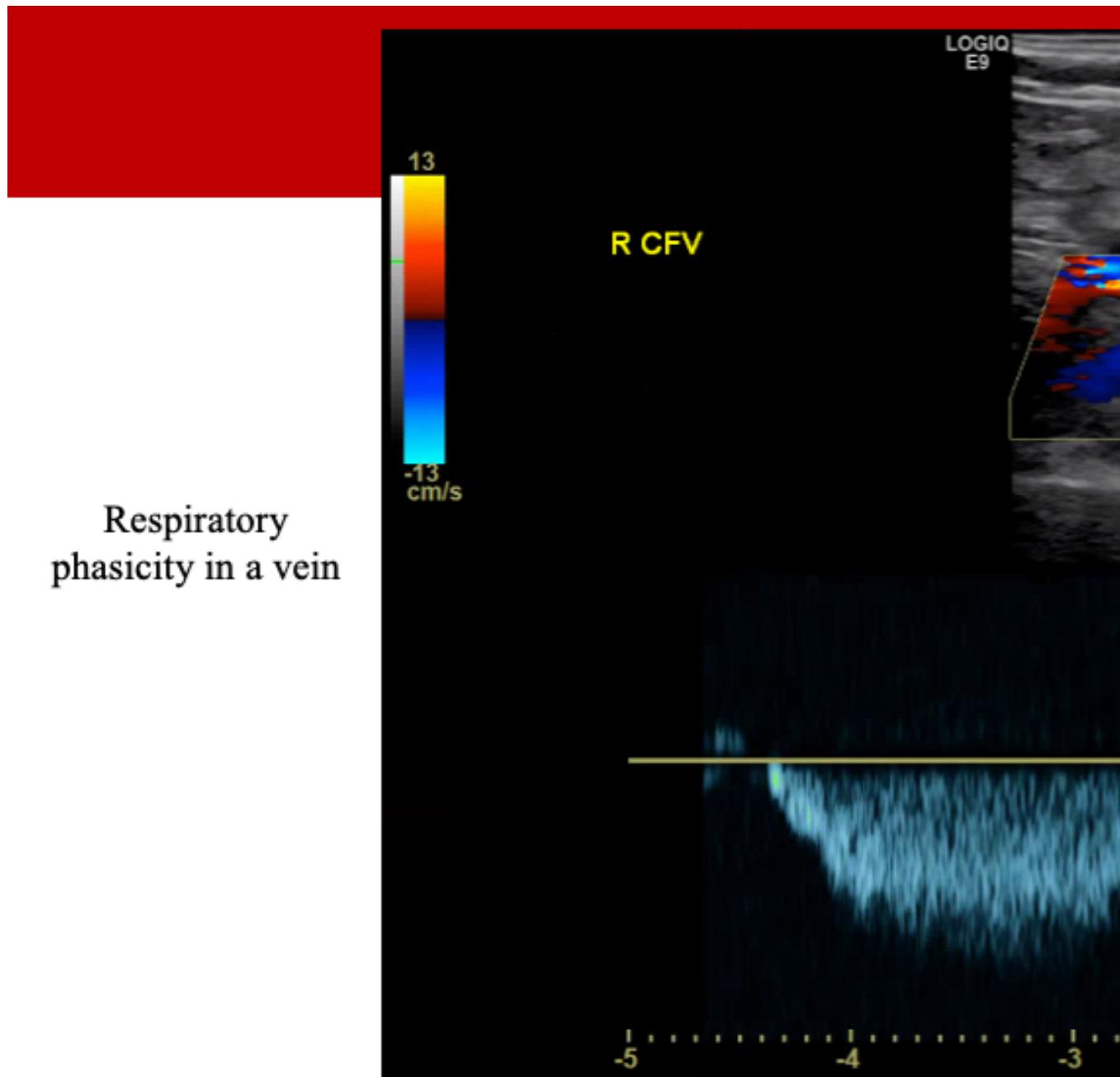
**Pulsatility:** Refers to changes in the venous waveform in accordance with the cardiac cycle. Pulsatility is normal in the upper extremity veins central veins, given their proximity to the heart.



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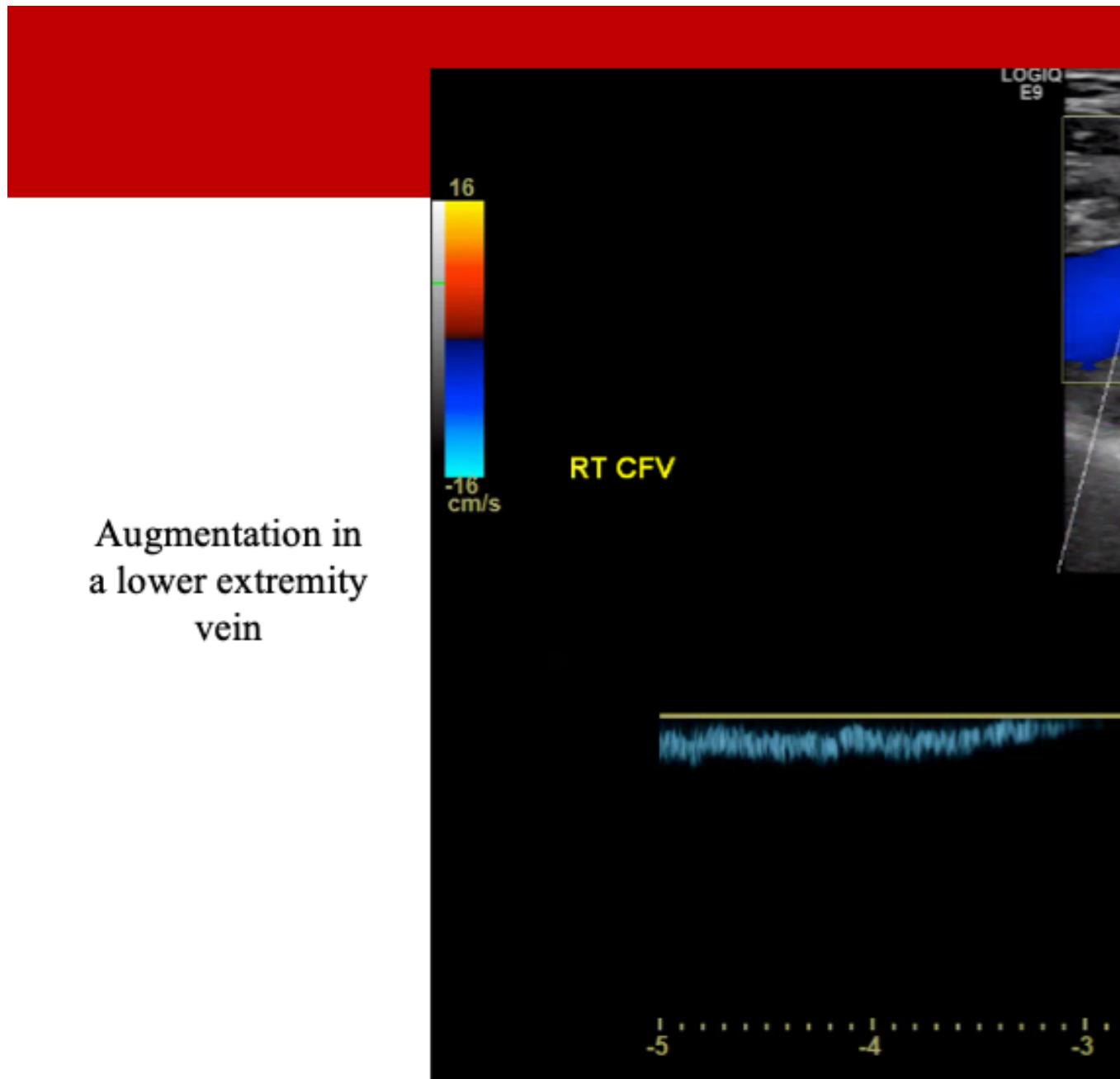
This is an abnormal finding in the lower extremity veins, and may be a sign of pulmonary hypertension, right heart failure, or tricuspid regurgitation.

**Phasicity:** (also called respiratory phasicity) is variation in the waveform with respiration. This results from increasing and decreasing intrathoracic pressures secondary to respiration. Phasicity is an indicator of a patency proximal to the point of measurement. So if we see lack of phasicity (continuous flow) in the left femoral vein but normal phasicity in the right, we would be concerned for left iliac vein occlusion or stenosis. If we saw absence of phasicity (continuous flow) in the bilateral femoral veins, we would be concerned for IVC obstruction or stenosis.



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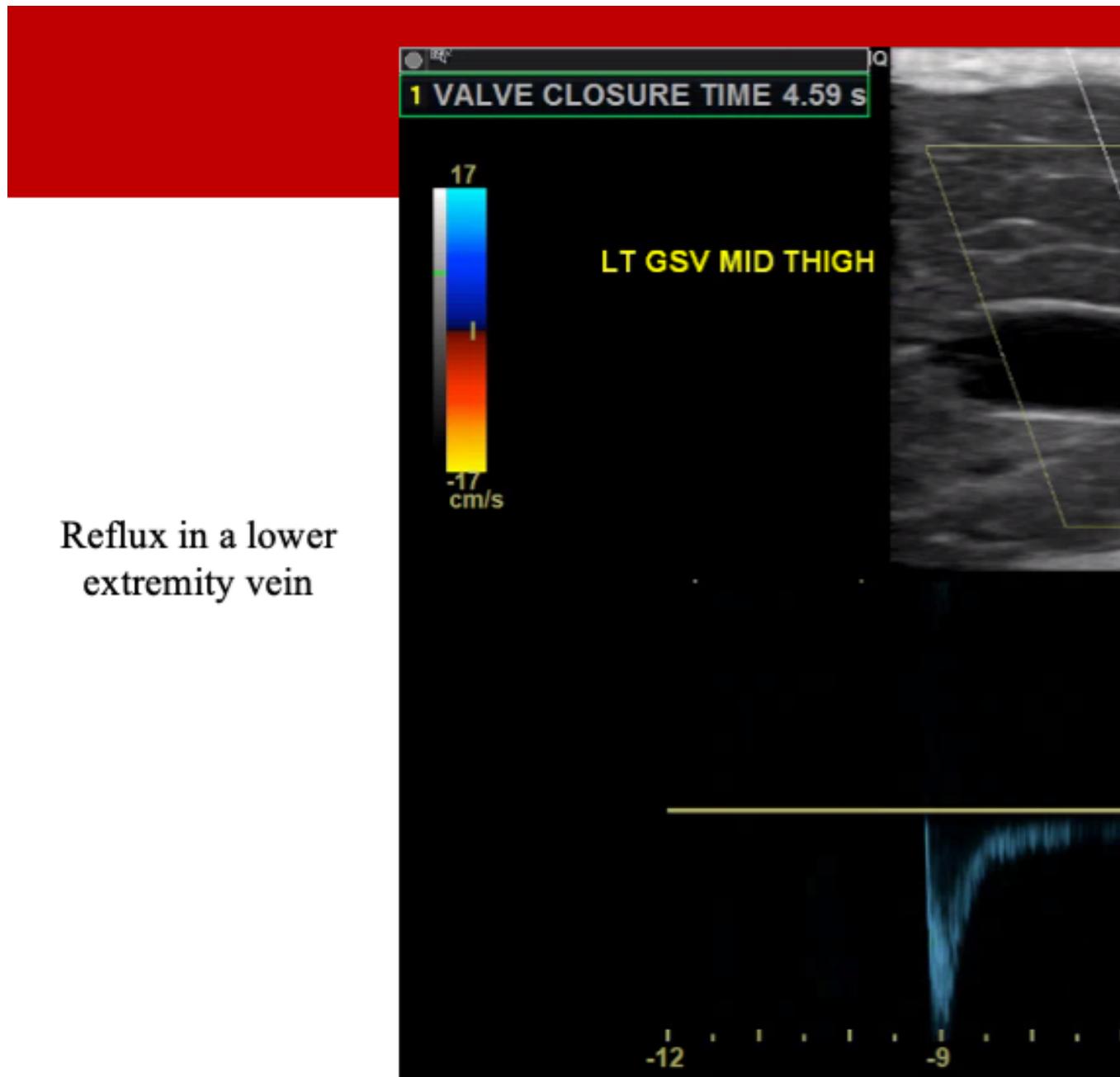
**Augmentation:** Distal compression that augments forward flow. For example, if we are measuring flow at the femoral vein and we squeeze the calf and we see augmentation in the waveform, this indicates lack of occlusion in the venous system from the knee to the probe.



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**What are venous pathologies that are commonly tested/encountered?**

Reflux: Venous reflux due to valvular incompetence is best assessed with duplex scanning in the upright position. Reflux in the common femoral vein and the saphenofemoral junction may be elicited with a Valsalva maneuver (which increases intra-abdominal pressure), but release of a pneumatic cuff compression is a more reproducible method. Reflux is identified as reverse flow - that is, away from the heart - following valsalva or release of the compression cuff.



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Consensus guidelines suggest a cutoff value of 1 second for abnormally reversed flow (reflux) in the femoral and popliteal veins and of 0.5 seconds for the great saphenous vein, small saphenous vein, tibial, and deep femoral veins.

Perforator veins: Perforator veins connect the deep and superficial venous systems, penetrating the deep fascia overlying the muscle. Size  $>3.5$  mm and reflux  $>350$  ms (deep to superficial) is associated with perforator reflux. Pathological perforators are found in association with a healed or non-healed ulcer.

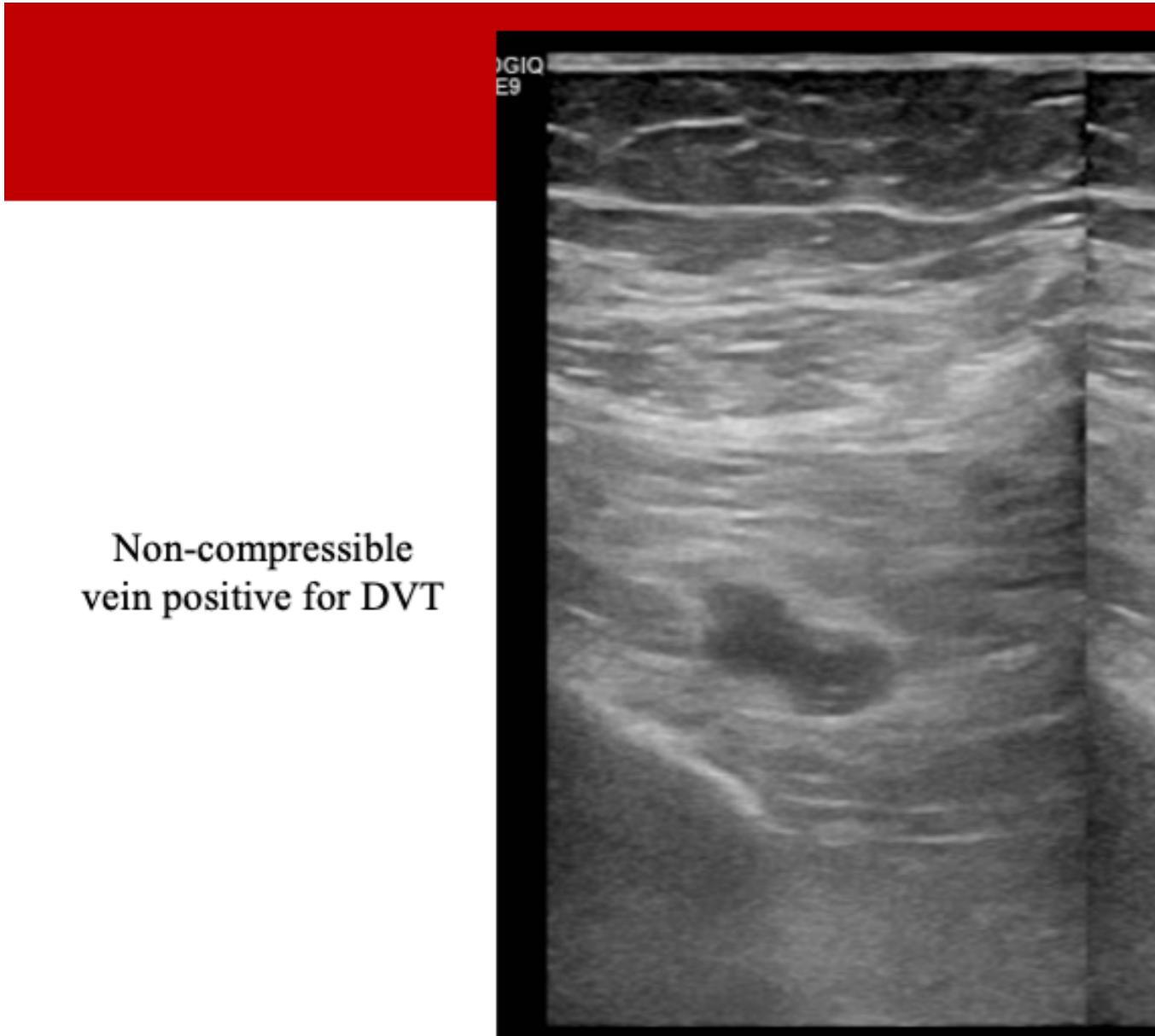
### What are some other examples of reflux?

Ovarian vein reflux: The ultrasound evaluation of pelvic congestion syndrome is performed in steep reverse Trendelenburg and standing positions with a low-frequency probe. Reflux is identified during the Valsalva maneuver. There are no validated criteria for the duration of reflux. Rather, an ovarian vein diameter  $>6$  mm is considered significant.

May Thurner: Also known as iliac vein compression syndrome, refers to a chronic compression of the left common iliac vein by the overlying right common iliac artery (CIA), with or without deep venous thrombosis. Notably, patients present with unilateral (left) lower extremity edema and pain, varicosities, DVT or venous ulcers. Intravascular ultrasound will demonstrate  $>50\%$  stenosis of the iliac vein from compression. Distal waveforms will demonstrate absence of phasicity if obstruction/stenosis is severe with continuous flow.

### Ok, let's transition from reflux to thrombosis:

Thrombosis: Characteristics of acute thrombus are an echolucent and incompressible thrombus in a thin-walled vein with significant distension.



Non-compressible  
vein positive for DVT

RT FV DIST



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Acute thrombus typically causes the vein to dilate with a diameter greater than the diameter of the adjacent artery. Venous wall thickening/scarring, a contracted vein, recanalization, and collateralization are found in chronic thrombosis.

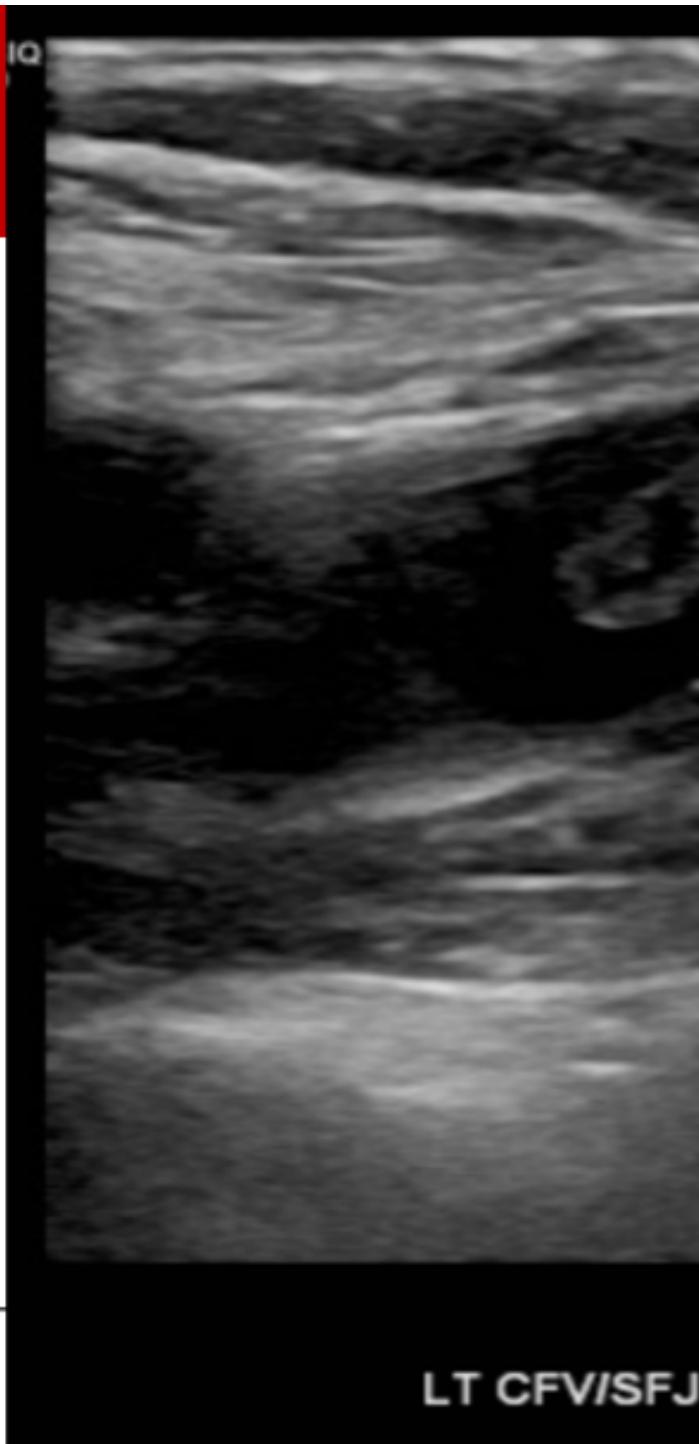
**Let's discuss 2 Specific examples of venous thrombosis: VTOS and EHIT**

Venous TOS: Venous thoracic outlet syndrome is thrombosis or severe stenosis of the subclavian or axillary veins secondary to chronic extrinsic mechanical compression. Repetitive injury to the subclavian vein at the level of the costoclavicular space results in chronic injury to the veins. Venous duplex may show a dilated, non-compressible vein consistent with an acute subclavian vein DVT, or lack of pulsatility/phasicity if obstruction/stenosis is more centrally located.

EHIT or Endovenous Heat Induced Thrombosis: S/p endovenous thermal ablations (RFA or laser ablation) of the GSV. 4 Grades: Grade 1 is thrombus in the GSV up to the level of the CFV. If < 50% of the CFV lumen is involved this is EHIT grade 2. EHIT grade 3 is extension into the CFV occupying >50% of the lumen and grade 4 is occlusion of the CFV.



EHIT Grade 2



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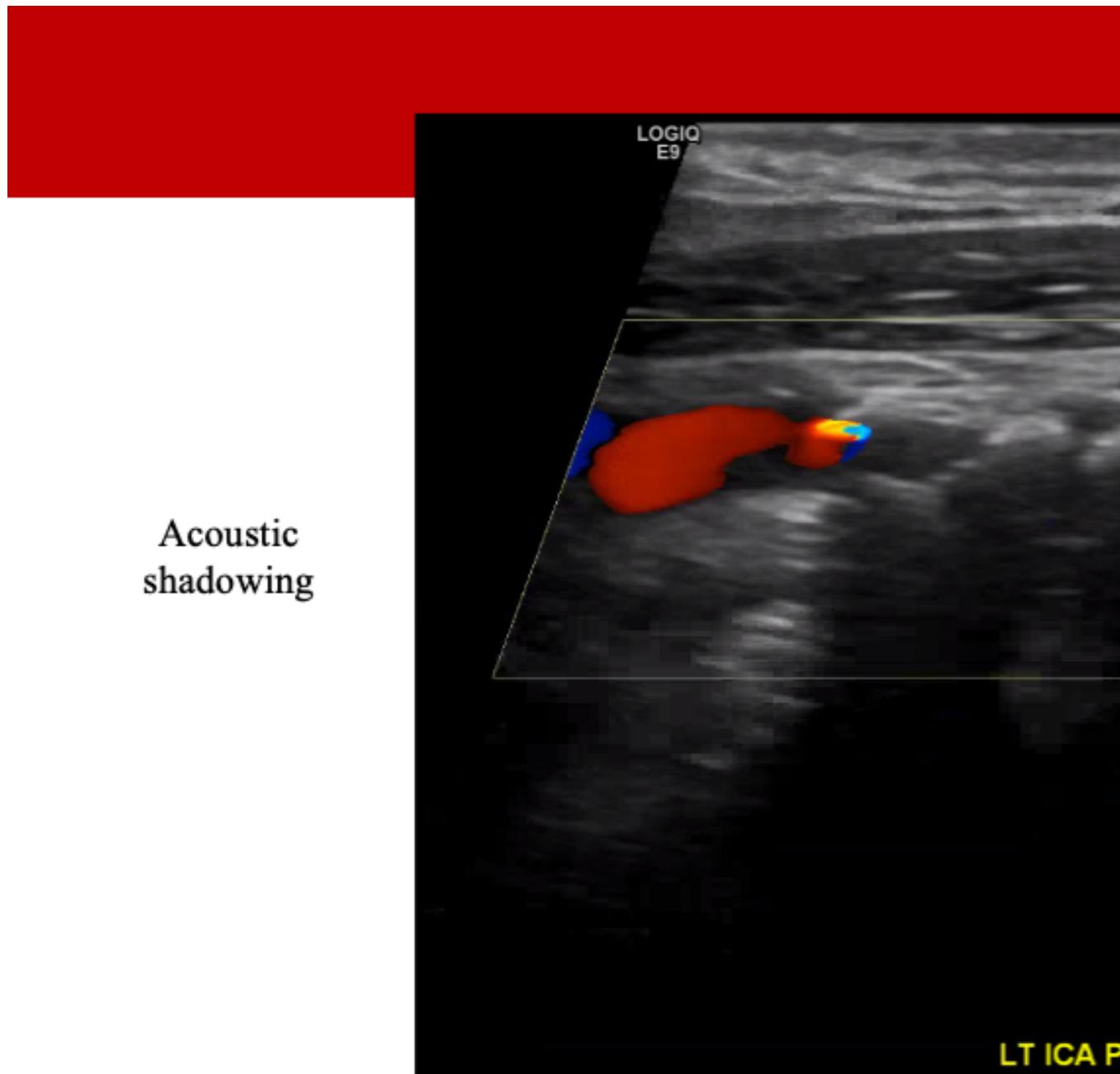
LT CFV/SFJ

EHIT grades 3-4 are typically treated with anticoagulation to reduce risk of PE. To minimize the risk of EHIT, the catheter should be positioned at least 2 cm from the saphenofemoral junction.

## 12.8 Artifacts

Finally, let's wrap up this episode with a discussion of imaging artifacts. These are frequently encountered and tested in vascular ultrasound, and it is important to recognize imaging artifacts in order to prevent incorrect interpretation.

**Acoustic shadowing:** Shadowing on an ultrasound image is characterized by a signal void behind structures that strongly absorb, reflect, or refract ultrasonic waves.



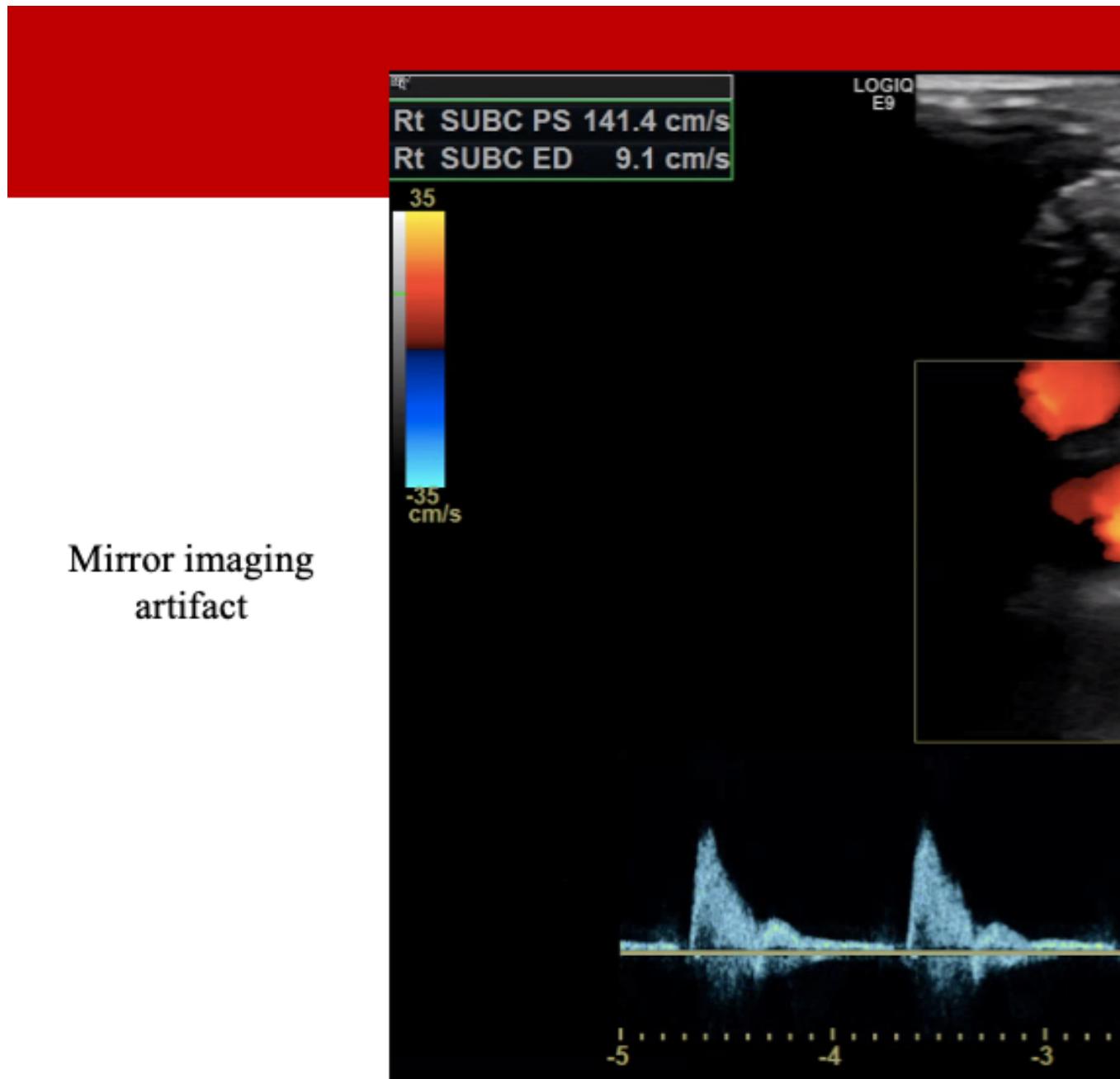
Acoustic  
shadowing



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Practically speaking, this most typically occurs deep to strongly reflective surfaces such as calcified plaques, and appears as a “dark area” beneath the plaque. **Acoustic enhancement** is essentially the inverse situation, and appears as a “bright area” deep to structures that transmit ultrasound waves exceptionally well. This can happen deep to fluid-filled structures such as cysts.

**Mirroring:** A mirror-image artifact is caused by reverberation of ultrasound and shows structures that exist on one side of a strong reflector as also being present on the other side of the reflector.



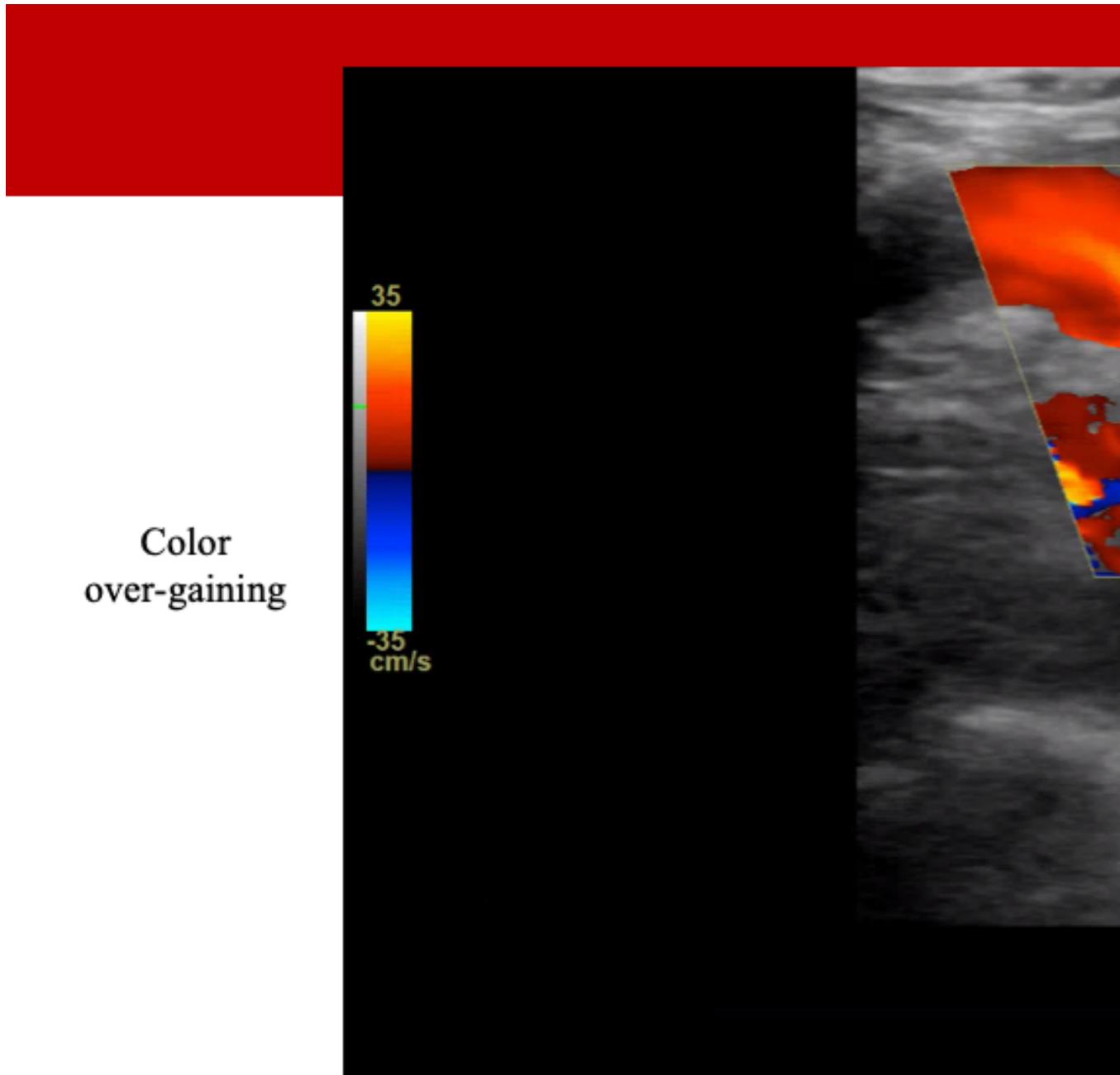
HARVARD  
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This is often seen around the pleura and the diaphragm, due to the strong reflection of ultrasound from the air-filled lung. These artifacts can occur in both B-mode imaging where you see the mirrored image and Doppler, in which you see the mirrored waveform.

**Refraction:** A refraction artifact is the result of ultrasound waves passing through tissues with different propagation velocities (such as air and water) and causes a structure to be improperly positioned laterally in the image. This is the phenomenon that results in a straw appearing bent when in a glass of water.

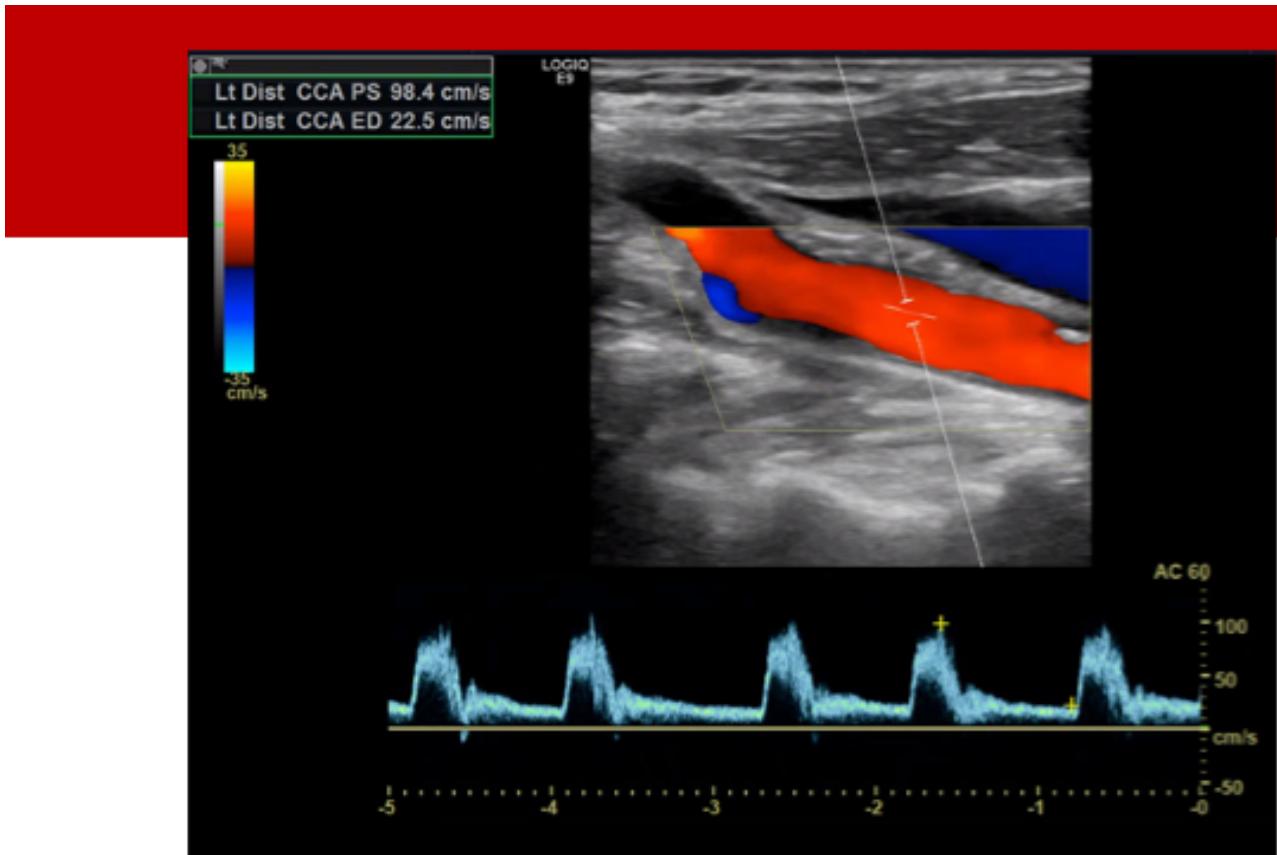
**Speed artifact:** Depth determination by an ultrasound machine is based on calculations using an average propagation velocity of sound in soft tissue of 1540 m/s. If the ultrasound wave passes through a medium at a different speed than predicted by the machine, an inaccurate image depth will be displayed. If the ultrasound passes less quickly through the material than soft tissue (as occurs in air or fluid), then the image will be displayed deeper than the true depth. In practical application, this is what causes a “bayonet” sign, or apparent bending of a needle when it passes from soft tissue into a cystic structure.

**Inappropriate color gain:** Overly gained images will show “speckling” in areas in which no flow is present (such as in soft tissue). Increasing the gain, increases both the signal and the noise.[Kremkau, 2021]



Under-gaining will result in reduced sensitivity to low velocity flow.

**Inappropriate angle correction:** Make sure the angle correction cursor is centered in the vessel and parallel to the walls, otherwise the Doppler velocity measurement will be incorrect.



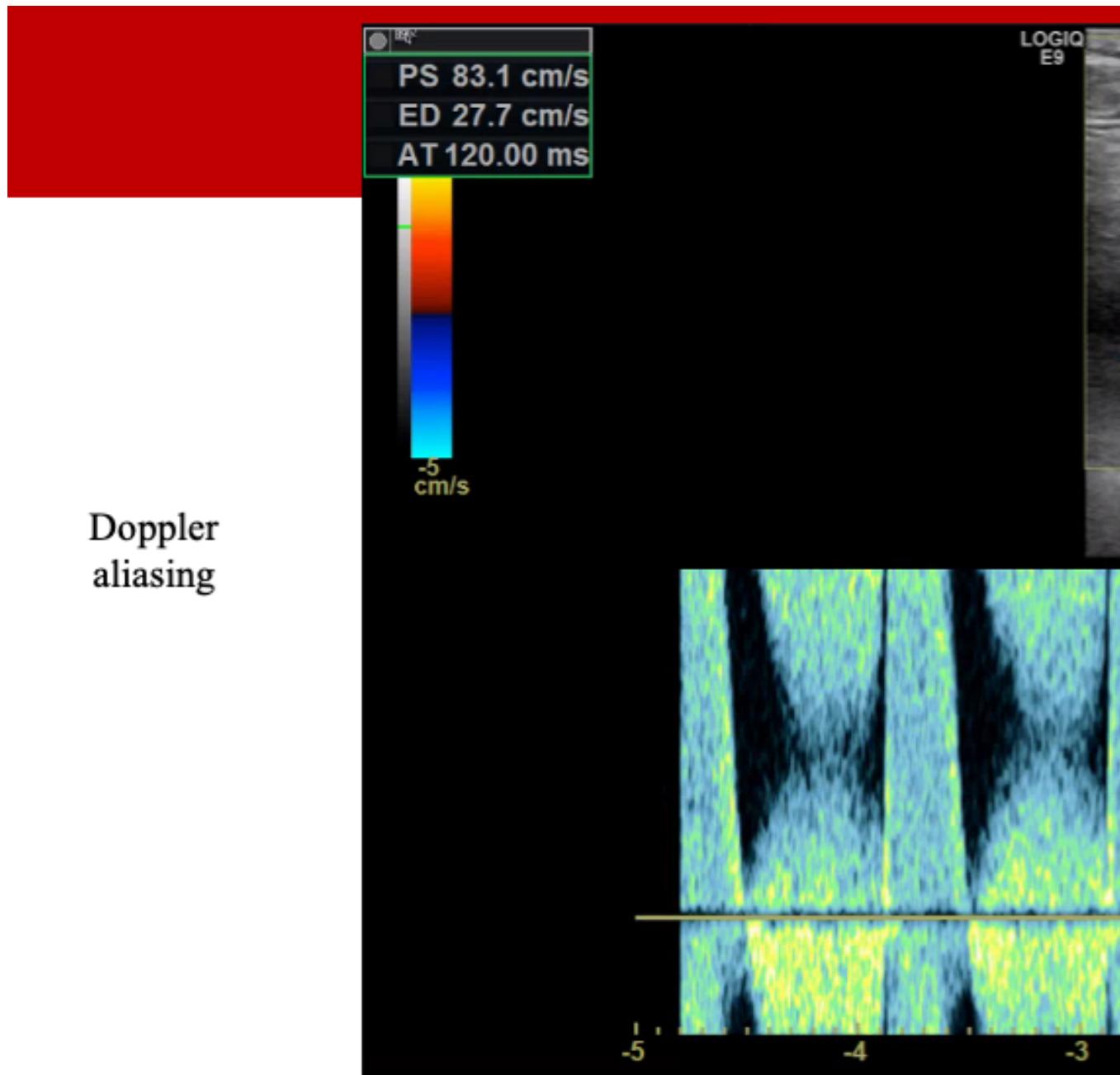
Inappropriate  
angle correction



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Finally, can we talk about aliasing, as I feel like this comes up a lot on exams:

**Aliasing:** Unlike continuous wave Doppler, pulsed wave and color flow Doppler are characterized by rapid pulses of ultrasound waves (at a rate called the pulse repetition frequency). The Nyquist limit defines the frequency at which aliasing will occur, as equal to the PRF/2. So what does this mean practically? In pulsed wave doppler, if the velocity of blood is greater than  $\frac{1}{2}$  the PRF, the peak velocity will be cut off, and wrapped around to the bottom of the scale.



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This results in inaccurate measurement of peak velocities, and may be remedied by increasing the PRF and hence the scale. In color flow doppler, aliasing appears as red to blue hues without separation of a black region indicating no flow. This occurs in areas of high velocity (such as immediately post-stenosis). This can be remedied with an increase in the color scale. Of note, if asked to determine the direction of blood flow in a vessel demonstrating aliasing, one should assess the flow in low-velocity areas of blood flow, as seen along vessel walls.

## 12.9 Accreditation and Credentialing

**Intersocietal Accreditation Commission (IAC) Accreditation** - voluntary, not linked to reimbursement but may be required by some payers. Medical director must have a certain training/experience, including RPVI as one way; technical directors must have credentials through ARDMS, CCI or ACR with recredentialing every three years.[IAC, 2021]

- Physician accreditation with RPVI requires recertification every 3 years with 30 hours of CME specifically in the area of vascular ultrasound.

Regulations for a completed vascular exam report[IAC, 2021]

- Patient identifier
- Date of exam
- Sign/symptom as indication - medical necessity should not just include “rule out”
- Reasons for any limitations to the examination
- Comparisons to any previous related studies
- Reported by medical staff within 2 working days and final report verified within 4 working days.

Troubleshooting examinations

- Any concerning acute findings, such as mobile intra-arterial material concerning for embolism, should be reported to the supervising/reading physician immediately before completing the examination.[of Diagnostic Medical Sonography, 2015]
- Some modifications to tests can be made if the patient is unable to tolerate, such as standing or tolerating a valsalva maneuver.[of Diagnostic Medical Sonography, 2015]

# Chapter 13

## Endovascular

### 13.1 Vascular Access

**Authors:** Sammy Siada, DO, RPVI; Rafael Demarchi Malgor, MD, MBA, FACS

Endovascular procedures are the cornerstone of any modern vascular surgery practice. Because most endovascular procedures are performed percutaneously using arterial or venous access, it is critical that vascular surgeons are facile with various techniques and devices used for endovascular access. Today we'll be discussing the various access sites, techniques for access, closure devices, and complications.

#### What factors play a role when choosing a site for access?

The factors to think about when thinking about which vessel to access are:

- The appropriateness of the access site the procedure performed
- Ability to obtain hemostasis at the conclusion of the procedure
- Ability to convert to open if necessary
- Effects of access on the tissues supplied by the accessed vessel and distal limb perfusion

#### What makes a vessel appropriate for access?

One of the most important factors when planning your access is the size of the vessel. The vessel needs to be able to accommodate the catheters and devices that will be used to perform the procedure. For instance, a brachial artery with less than 4mm diameter should not be accessed by a large bore sheaths, such as a 12Fr sheath.

The vessel also needs to be in a location that can allow access to the target vessel of interest. Additionally, the vessel needs to have an area that is relatively healthy to access the vessel safely and minimize complications. Heavily calcified vessels especially those with anterior wall calcification might not be appropriate for access.

**What about the ability obtaining hemostasis at the end of the procedure?**

The ability to obtain hemostasis is critical to be able to perform endovascular procedures safely which is one reason why the common femoral artery is the most commonly accessed vessel.

Hemostasis is most commonly achieved through manual compression by compressing the artery against the femoral head. The brachial artery can also be compressed against the humerus, but because it's a more mobile vessel, compression is less effective and can lead to hematoma or pseudoaneurysm formation which may necessitate an operation to prevent compression of the median nerve

Patients who will need to be uninterruptedly anticoagulated peri and postoperatively pose a challenge to hemostasis. The use of closure devices is very important in these situations to prevent access bleeding.

A variety of closure devices can also be used to assist in hemostasis, each with their own inherent advantages and disadvantages. In general, closure devices are contraindicated in small diameter and heavily calcified vessels.

**In any minimally invasive procedure, there is always a chance that you may need to convert to open. How does converting to open play a role in vascular access?**

Conversion to open is uncommon with vessel access accounting for <5% of the cases. Sometimes a large sheath is accidentally pulled out and a cutdown becomes necessary to repair the artery. Closure devices aren't 100% effective in hemostasis and may also require a cutdown for definitive control if they fail, especially when obtaining large bore access.

This makes choosing the right vessel critical. For example, if a large sheath is accidentally pulled out of the CFA during an EVAR, the repair can be done through a straightforward groin cutdown. In contrast, the subclavian artery is rarely accessed percutaneously because converting to open would require a more challenging peri-clavicular incision or even a thoracotomy for repair.

**Large diameter sheaths are often used, particularly in aortic procedures. These sheaths can be occlusive which can result in downstream tissue ischemia. What considerations should be taken when thinking about downstream tissue ischemia?**

When performing diagnostic procedures using small diameter sheaths and catheters, anticoagulation may or may not be necessary depending on how diseased the access vessel is.

However, when using large devices (e.g. in EVARs), the sheaths can be partially or completely occlusive which mandates full anticoagulation to prevent thrombosis. The other thing to consider is the length of time that the sheath remains in the vessel as the leg can only tolerate ischemia for 4-6 hours. This is usually pertinent when performing complex endovascular aortic procedures.

To minimize downstream tissue ischemia, a large bore sheath should be pulled back to decrease the length of vessel obstruction by its shaft in order to unblock proximal vessel collateral branch vessels. For instance, when performing an aortic procedure through a femoral access attempt to pull the sheath back into the external iliac artery to increase distal limb perfusion through the internal to femoral artery collateral branch vessels.

The long story short is to be liberal with anticoagulation when there is reduced flow in the vessel such as the iliofemoral system during EVAR or tibial access

**Do the principles that we've described also apply to veins?**

The same principles apply but there are some notable differences between arterial and venous access.

Veins are a low-pressure system, so hemostasis is easier to achieve and hemorrhagic complications are much less common. However, this poses a challenge during access as there is less radial force keeping the vein open making the vein more susceptible to compression by the ultrasound probe and the needle.

If a large bore sheath is necessary to perform a venous procedure, a suture-mediated closure device can be utilized to achieve hemostasis especially in patients that will be kept fully anticoagulated

Additionally, a syringe may be needed to confirm access and can also prevent air embolism

**Let's talk about accessing the common femoral artery. Why is the CFA the most common vessel used for access?**

It is large caliber and can accommodate large sheaths up to 26-28 Fr. It also allows for a wide set of procedures and is ergonomically easy to work with given its location. It is relatively easy to hold manual pressure and if a conversion to open is needed, a femoral cutdown is relatively straightforward.

**Where in the common femoral artery is the best spot to access?**

The ideal puncture site is in the CFA in the medial third of the femoral head in between the inguinal ligament and the femoral bifurcation in the middle of the femoral head.

Accessing the vessel above the inguinal ligament makes compressing the artery very difficult which can lead to life-threatening retroperitoneal bleed.

A puncture that is too distal and into the SFA increase the risk of thrombosis or dissection causing acute limb ischemia as well as AV fistula formation between

the superficial femoral and profunda femoris artery.

#### **What are the different ways to obtain CFA access?**

There are three different ways to access the CFA: manual palpation, fluoroscopic guided, and ultrasound guided.

With manual palpation, a finger is placed above and below the desired access point directly on the pulse and the needle is inserted in between the two fingers.

Fluoroscopic guidance uses bony landmarks relative to the position of the needle.

The standard of care in the modern era for obtaining CFA access is to use ultrasound guidance. Ultrasound allows visualization of the vessel and surrounding structures. PAD within the vessel can readily be identified with ultrasound, allowing safe access in a relatively disease-free part of the artery. Ultrasound also clearly shows the femoral bifurcation. Using ultrasound allows for subtle corrections in the angle of the needle and how it interacts with the surrounding tissues. It is rapid, real-time, inexpensive, and safe.

#### **What anatomic considerations should be taken when accessing the CFA?**

The CFA is the continuation of the external iliac artery as it courses under the inguinal ligament. It is about 5-8 cm in length and then bifurcates into the superficial femoral and profunda femoris arteries.

The inguinal ligament is a good external landmark to estimate where the CFA is. It is critical to emphasize that the inguinal ligament does not correspond to the groin crease and this is especially true in obese patients. An imaginary line is drawn from the ASIS to the pubic tubercle. The artery generally runs a third of the way from the pubic tubercle to the ASIS. A metallic instrument can be placed in this area to mark it externally and a fluoroscopic image can be obtained to identify the relation of the instrument to the medial third of the femoral head. This imaginary line also marks the superior-most extent of the access.

The CFA is most often accessed in a retrograde fashion in between the inguinal ligament and femoral bifurcation. This allows for a multitude of potential diagnostic and therapeutic procedures in most parts of the body.

#### **Can the CFA be accessed antegrade?**

Yes. Sometimes antegrade CFA access is used when performing an intervention distal on the ipsilateral leg. The advantage of antegrade access is better pushability and torquability of wires, catheters, and sheaths when performing complex peripheral intervention where no other proximal procedures are needed.

Antegrade access is more challenging than retrograde access, however. This is particularly true in patients with a very short CFA, short distance between the inguinal ligament and the femoral bifurcation because the needle requires a steeper angle of entry to allow for cannulation well above the femoral bifurcation.

Obtaining antegrade access is especially difficult in obese patients and will usually require an assistant to retract the pannus to allow proper needle placement. I would say antegrade access is relatively contra-indicated in morbidly obese patients with large pannus. Ultrasound guidance remains key here as well.

**What are some other commonly accessed arteries for endovascular procedures?**

The tibial vessels can be accessed percutaneously for retrograde recanalization for severe LE PAD. It is usually performed using micropuncture kits which we will discuss a little later. It is usually done with ultrasound guidance and uses small sheaths and wires. The PT and AT are more commonly used because they are easier to access.

The radial artery is commonly used in coronary interventions and is increasingly being used by vascular surgeons. It is easily palpable over the distal radius and can be cannulated with ease. Hemostasis is straightforward using compression. In the rare setting of radial occlusion, the hand rarely becomes ischemic because most people are ulnar dominant. It can accommodate sheaths up to 6 French.

- The best way to reduce vasospasm and injury to the radial artery is utilizing a radial artery cocktail - calcium channel blocker, vasodilator, and anticoagulation.[Mason et al., 2018, Cauley et al., 2019]

The brachial artery can be accessed percutaneously over the olecranon process with the arm supinated.[Alvarez-Tostado et al., 2009] Ultrasound guidance allows for visualization of the brachial bifurcation. It can accommodate 6-7 Fr sheaths. Hemostasis is critically important as bleeding can result in a hematoma that results in median nerve compression, which is a surgical emergency. Cut down after brachial access has been shown to reduce complications.[Kret et al., 2016]

**Let's not forget about venous access. What are some of the most commonly accessed veins?**

The CFV is commonly accessed for procedures involving the IVC and iliac vessels and their branches for conditions such as May-Thurner, pelvic congestion syndrome, and IVC filter placement. Treatment of PE can also be performed through the CFV. The CFV can easily be compressed over the femoral head and is located medial to the CFA. Ultrasound guidance should be used to prevent arterial injury and backwalling.

The internal jugular vein can be accessed using US guidance (to prevent carotid injury; IJ is lateral to the carotid). IJ access is used most commonly for central venous catheters as well as IVC placement and filter retrieval. It is also an excellent access to treat pulmonary embolism via thrombolysis or thrombectomy. The IJ can be utilized to perform ovarian and internal iliac vein embolization. IJ is also the preferred access to perform TIPS, which is often of less interest to vascular surgeon.

The popliteal vein can be accessed with the patient in the prone position or the distal femoral vein in the supine position to diagnose and treat DVTs of the extremity veins. Ultrasound is also helpful to avoid arterial access and especially if the vein is thrombosed.

Arm veins (cephalic/basilic) can be also readily accessed for vein mapping or fistula interventions.

**Let's move on to access technique. Historically, there are two types of puncture needles: single-wall and double-wall. Can you talk about the differences?**

Double wall needles were commonly used back in the day for femoral access. They have an outer hollow blunt-tipped needle and an inner sharp stylet. The needle was inserted through and through the artery and the stylet removed and the blunt hollow needle pulled back until blood is returned. These aren't favored anymore because they cause unnecessary backwalling of the artery. Double wall access kits are used in treating endoleaks from both a trans caval and translumbar routes to allow access into the aneurysm sack and needle removal to avoid puncturing the endograft.

Single wall needles are typically the choice for diagnostic procedures. 18-gauge needles accommodate an 0.035 in wire and 21 gauge accommodates a 0.018 in wire.

**Can you describe the micropuncture technique for percutaneous access?**

Micropuncture technique is the most commonly used method for percutaneous access nowadays. The advantage of the micropuncture technique is the use of a small needle which can be removed and repositioned with a negligible risk of bleeding and minimal amount of manual compression needed.

Ultrasound is used to cannulate the artery with a 21-gauge needle. It is best to visualize the needle entering the artery and to be intraluminal without being against the wall. Blood return is then seen and a floppy tip micropuncture (0.018) wire is inserted under fluoroscopic guidance to make sure the wire passes into the vessel easily. A 4 Fr introducer sheath is placed over the wire gently to avoid kinking the wire. The inner cannula of the sheath is removed, and a 0.035 guidewire is placed under fluoroscopic guidance. It is important to remember that there are two types of introducer sheath depending on amount of subcutaneous scar tissue containing either a soft or a stiffened cannula. The 4 Fr is removed over the wire while holding manual pressure and desired sheath (usually 5 or 6 Fr) is placed for definitive access. The side port is then aspirated for arterial blood and flushed with heparinized saline.

## 13.2 Complications

**With any invasive procedure, there are risks of complications. What are some of the complications of percutaneous vascular access?**

Hematomas are the most common complication and have an incidence of about 3%. Most of these hematomas are clinically insignificant but retroperitoneal hemorrhage from a high puncture above the inguinal ligament can be life-threatening. These may require conversion to open and direct repair of the vessel or covered stent placement (especially if the puncture is above the inguinal ligament). Proximal balloon occlusion can be helpful to control hemorrhage while the vessel is being repaired.

Groin hematomas are not uncommon and are usually self-limiting. An expanding hematoma that is seen early can be treated with simple manual pressure at the bedside. If the hematoma is large and compressing surrounding structures or threatening skin integrity or if the patient is hemodynamically unstable, then surgical evacuation may be necessary

Pseudoaneurysms are an uncommon complication with an incidence of about 0.6%. Most pseudoaneurysms are treated with ultrasound-guided compression or thrombin injection. Thrombin injection requires a narrow neck into the pseudoaneurysm. If the pseudoaneurysm is >2cm, compresses surrounding structures, threatens skin integrity, or has failed thrombin injection, then surgical repair is required.[Morgan and Belli, 2003, Stone et al., 2014] For a representative image, see 12.5.1.1

Thrombosis of the CFA is a known complication but fortunately is rare with an incidence of 0.2%. This can result from manual compression of the CFA that has severe atherosclerotic disease or prior groin reconstruction. This generally requires a cutdown, endarterectomy, thrombectomy, and patch angioplasty.

Lastly, AV fistula can form and are usually between the femoral artery and vein with an incidence of 0.5-0.9%). These usually occur from a low puncture of the CFA bifurcation or profunda. They are usually asymptomatic and detected on exam (palpable thrill) and confirmed with duplex imaging. If the fistula is small, it generally can be observed with close duplex surveillance. If it enlarges or becomes symptomatic, then repair is indicated. Covered stent grafts can be placed with minimal morbidity, making this optimal for high risk patients. Open surgery also is highly successful. Deciding between non-operative, endovascular, or open treatment is up for debate and is up to the clinical judgement of the surgeon.

## 13.3 Radiation Safety

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### 13.3.1 Important Terms

**Absorbed Dose** - energy deposited by ionizing radiation in a medium per unit mass. Dosage measured and communicated most commonly in Gray (Gy) equal to 100 rad.[Reed, 2019]

**Effective Dose** - energy deposited by ionizing radiation in a medium, taking into account the sensitivity of the specific tissue, time and duration of exposure. These factors compose a weighting factor (W) and thus this is calculated by multiplying absorbed dose (Gy) by the weighting factor (W). Dosage measured and communicated most commonly in Sievert (Sv) equal to 100 rem.[Reed, 2019]

**Reference Air Karma (RAK)** - Radiation output at a specific reference point along a fluoroscopic axis.[Kirkwood et al., 2013]

- Located 15cm along the beam axis toward the focal spot from the isocenter.
- Best approximation of cumulative patient dose or peak skin dose (PSD).[Kwon et al., 2011]
- Includes dose from fluoroscopy and fluorography acquisitions, however does not account for gantry angulation or changes in height.

**Substantial Radiation Dose Level (SRDL)** - defined as 5Gy exposure, which translates to a skin dose of 3Gy and should trigger specific patient follow up.[Baiter et al., 2011, Hirshfeld et al., 2005, Kirkwood et al., 2015, Stecker et al., 2009]

### 13.3.2 Radiation effects

The effects can impact both patients and clinicians. Patients often receive a higher dose during a single procedure, whereas clinician doses, even with proper shielding, accumulate over multiple procedures throughout their careers. The effects of radiation are defined as either deterministic or stochastic.

Table 13.1: Skin related deterministic effects that can be seen after a single exposure over a certain threshold [Balter et al., 2010, Hirshfeld et al., 2005, Kirkwood et al., 2014, Stecker et al., 2009, Wagner et al., 1999]

| Dose  | Effect  |
|-------|---|
| 0-2Gy | No observable effects   |
| 2-5Gy | Transient skin erythema and dermatitis, full recovery<br>6w-1yr[Guesnier-Dopagne et al., 2019, Kirkwood et al., 2014] |
| 5-    | Erythema and epilation (hair loss), prolonged erythema up to 1y   |
| 10Gy  |   |
| 10-   | Permanent epilation, atrophy or induration up to 1y   |
| 15Gy  |   |

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| Dose  | Effect          |
|-------|-----------------|
| >15Gy | Dermal Necrosis |

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The ocular lens is the most radio-sensitive tissue and cataracts is an unfortunately common deterministic effect.[Machan, 2018, Brown and Rzucidlo, 2011] Annual limits for ocular exposure include 20mSv per year and total threshold of 0.5Gy.

Stochastic effects can occur after exposure of any dose, but are seen more frequently in populations with higher radiation exposure. These are based on population studies and exact dose dependent relationships have not been established. These include:

- Brain cancer[Rajaraman et al., 2016, Kirkwood et al., 2018]
- Breast and thyroid cancer[Johnson et al., 2001, Einstein, 2012]

### 13.3.3 Risk factors

Certain patient, procedure and clinician factors put patients at increased risk for radiation exposure.[Killewich et al., 2011, Mitchell and Furey, 2011] These should be considered when planning and executing endovascular procedures to ensure that dosages are as low as reasonably achievable (ALARA)

- Patient Factors
  - Obesity
- Procedure Factors
  - Case complexity
  - Need for magnification
  - Case orientation and angulation
- Clinician Factors
  - Use of multiple subtraction runs

### 13.3.4 Reduction strategies

The main source of radiation to the clinicians is scatter from the patient. There are a number of techniques that can be used to reduce exposure of clinicians to this radiation.[Heidbuchel et al., 2014, Kirkwood et al., 2013]

- Reduce the time of exposure, keeping a close eye on fluoroscopy time and dosages as a procedure progresses.

- Position the source as far away from the operator as possible to still achieve optimal imaging. Radiation dose changes according to the inverse square of distance. Therefore twice the distance results in one quarter the dose.
  - For example, left anterior oblique (LAO) will bring the source to the patient's right side, causing higher doses to clinicians on that side of the patient.[Kirkwood et al., 2013, Sailer et al., 2019]
- Appropriate shielding
  - The location with the highest exposure to scatter is below the table.[Gonzales et al., 2014, Miller et al., 2010] Therefore, lead skirt and extended lower body shields reduce radiation to the operator's legs.[Kirkwood et al., 2015]
  - Of note: leaded caps DO NOT reduce radiation exposure to the brain because the majority of radiation is received as scatter from the patient up through the face and neck.[Kirkwood et al., 2018]
  - Lead shielding should be regularly inspected and discarded if damaged. Particularly if defect is  $>15\text{mm}^2$  on a critical organ area,  $>670\text{mm}^2$  along a seem/overlapping area,  $>11\text{mm}^2$  on a thyroid shield.[of Healthcare Inspections, 2014, University et al.]
- Collimation can be helpful to reduce patient and operator dose, scatter and improve image quality.[Haqqani et al., 2012]
- Use last image hold to allow for procedural planning

### 13.3.5 Pregnancy considerations

Pregnancy of both patients and clinicians need to be considered in relation to radiation exposure and safety.[Chandra et al., 2013, Mitchell and Furey, 2011, Shaw et al., 2011] This is a highly tested subject on vascular surgery examinations. Some important take aways include:

- CDC has released guidance for potential prenatal effects on radiation exposure[CDC, 2011] The majority of effects on fetuses is extrapolated from studies of the fall out from Hiroshima, Nagasaki and Chernobyl.
  - $<0.05\text{Gy}$  represents no measurable risk to embryo or fetus at any gestational age.
  - $0.05\text{-}0.5\text{Gy}$  can be dangerous in the first trimester, but has not been associated with defects later in pregnancy.[Shaw et al., 2011]
  - $>0.5\text{Gy}$  can be dangerous at any point during pregnancy.
- Dose limit recommendations during the 9 months of pregnancy is 500mrem or 50mrem per month.[Dauer et al., 2015, Chandra et al., 2013]

Strategies to reduce exposure include:

- Avoid direct fluoroscopy to the fetus, high-gantry angulation, and femoral access
  - Use collimation to ensure fetus is excluded from imaging field.
- Use adjuncts of intravascular ultrasound and lead shielding when able.
- Reduce machine kilo-voltage and milliampere/second.
- Remove the anti-scatter grid to reduce dosage to patient.[Tomà et al., 2019]
- Limit fluoro time.
- Some recommendations state that operators who intend to get pregnant should start wearing maternity aprons (lead equivalent to 1mm) even prior to knowing they are pregnant.[Shaw et al., 2011]
  - Original studies demonstrated reduction in fetal exposure by 80%. [Wittrak and Sprawls, 1984]
  - However, a recent multi-institutional review showed that fetal exposure is minimal even in regular lead, therefore the additional weight of maternal lead may be unnecessary.[Chandra et al., 2013]

### 13.3.6 Regulation

Joint Commission Oversight - sentinel radiation reporting is aimed to promote awareness of preventable events perform root cause analyses to understand the reasons for events.[Commission, 2019] It is important to escalate events early, as effects may not occur until much later.[Arbique] Sentinel events include:

1. Cumulative dose of 15 Gy for a single field over 6mo - 1yr.
2. Delivery of radiotherapy to the wrong body region.
3. Actual dose more than 25% above planned radiotherapy dose.[Jones and Pasciak, 2011]

Institutional Oversight - many institutions develop their own guidelines for employee exposure and mitigation strategies. Some common policies regarding dose limits include:

- Monthly limit for dosimeter reading of 100mrem per month.
- Recommendations for annual occupational dose is <20mSv per year averaged over 5y and no more than 50mSv in any one year. Occupational dose <100mSv per year is not thought to increase cancer risk
  - Recent reduction of occupational dose limit to 50mSv is due to increasing data connecting cataracts to radiation exposure.[Hamada et al., 2017]

## 13.4 Contrast Reduction

The most common complications related to iodinated contrast during endovascular procedures are hypersensitivities and acute kidney injury. There are a number of mitigation strategies to limit the effect of contrast on patients undergoing endovascular procedures.

In general patients with food allergies do have an increased incidence of contrast media allergies, however no specific common allergen has been identified. A new seafood allergy should not postpone or require pre-medication if a patient has previously tolerated IV contrast.[Schabelman and Witting, 2010]

### 13.4.1 CO<sub>2</sub> Angiography

CO<sub>2</sub> angiography is often utilized in place of iodinated contrast during fluoroscopy. A bolus of CO<sub>2</sub> is injected, which then absorbs less ionizing radiation than surrounding tissue and provides a map of the arterial tree. CO<sub>2</sub> can be used in a wide range of endovascular procedures, even a ruptured AAA.[@knipp2010] However, there are specific limitations and complications that should be understood.

#### 13.4.1.1 Limitations

- Contraindicated in imaging above the diaphragm.[Caridi and Hawkins, 1997, Sharafuddin and Marjan, 2017]
- Susceptible to bolus fragmentation and often requires stacking to fully visualize the target arterial bed.[Caridi and Hawkins, 1997, Sharafuddin and Marjan, 2017]
- Bowel gas can limit imaging of the abdomen. Glucagon can be administered to reduce bowel gas motion artifact and improve the image.[Caridi and Hawkins, 1997, Cho, 2015, Criado et al., 2012, Sharafuddin and Marjan, 2017]

#### 13.4.1.2 Complications

- Vapor lock - can occur with high volume, serial injections where contaminated air accumulates. Strategies to reduce incidence include waiting 1-3min between angiography runs. Operators should use a one way valve to reduce risk of air contamination.[Cho and Hawkins, 2011, Cho, 2015]
  - Cardiac/pulmonary vapor lock can occur with venography. Mimics a PE with hypoxia and hypotension. Initial management is to place patient in the left lateral decubitus/trendelenberg position.[Caridi and Hawkins, 1997, Sharafuddin and Marjan, 2017]
  - Mesenteric vapor lock presents with significant unrelenting abdominal pain. Fluoroscopy can confirm a retained bubble. Initial manage-

ment includes ongoing heparinization to prevent down stream thrombosis and maneuvers to break up the bubble by rotating the patient side-to-side or deep abdominal massage. Catheter aspiration may be needed.[Caridi and Hawkins, 1997, Sharafuddin and Marjan, 2017]



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