

Vascular Surgery Exam Prep

Audible Bleeding

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Preface

This content was developed to accompany our Audible Bleeding Exam Prep 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 embedded into the text.

Editors: Adam P Johnson; Matt Smith; and *Audible Bleeding Team*

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Usage

This e-book is intended to be a high level, easily accessible review for exam preparation and paired with our podcast content to help streamline your studying.

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Additional Resources

We hope you use this e-book as a high level review for your exam preparation. This resource is by no means comprehensive. We have done our best to reference and link to more comprehensive resources throughout the text so that you can dive deeper into topics that are beyond

the scope of this review. We try to prioritize open access publications and free, high quality, unbiased materials. A few highly recommended additional resources include:

- [Rutherford's Vascular Surgery and Endovascular Therapy](#)
 - No vascular surgeon bookshelf is complete without a copy of this book sitting on it!
 - Of note, our review book references the 9th edition of Rutherford's, however there is a new edition (10th) has just been released in June 2022.
 - Many institutional libraries carry this book and can be accessed for free. We would recommend checking with that resource prior to purchasing yourself.
 - Some industry representatives offer text book purchasing programs, so check with them prior to purchasing yourself.
- [VESAP6](#)
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 - Check this site regularly for free content through the SVS. This is available to all candidate members of the SVS, so sign up!
- [VSCORE](#)
 - This is a curated curriculum developed in collaboration with Association for Program Directors in Vascular Surgery (APDVS)
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This book is built on [Quarto](#).

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This book is the result of a broad community of vascular surgeons and trainees. They have all freely donated their time to contribute to this work and we owe them a debt of gratitude. Follow them on twitter or other social media platforms to keep up to date with what they have going on.

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Part I

Head and Neck

1 Cerebrovascular

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1.1 Available Guidelines

Society for Vascular Surgery clinical practice guidelines for management of extracranial cerebrovascular disease (AbuRahma et al. 2022)

1.2 Presentation

1. What is the definition of crescendo TIAs?

The presenting symptoms of extracranial carotid disease is of the utmost importance to understand because it is a major factor in determining treatment and prognosis. Focal neurologic symptoms that occur suddenly and then resolve within 24 hours are called a transient ischemic attack (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 with complete resolution of the deficit between the episodes, usually over a 24 hour period. The result of which is the same neurological deficit. (R. Naylor et al. 2023) 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 2019) 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, there is merit in screening high risk patients as only 15% of patients experience a warning TIA prior to a stroke. (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%).(Sauve 1994) 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)

1.3 Evaluation

1. 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 Section [20.2](#)
- 70-99% stenosis of ICA - Duplex diagnostic criteria:sec-extracranial
 - PSV $>/= 230$ cm/sec
 - EDV >100 (EDV > 140 cm/sec most sensitive for stenosis $>80\%$)
 - Internal/Common Carotid PSV Ratio > 4
- 50-69% stenosis of ICA - Duplex diagnostic criteria:
 - PSV 125-229 cm/sec
 - EDV 40-100
 - Internal/Common Carotid PSV Ratio 2-4
- US has a low sensitivity for 50-69% stenosis. A negative ultrasound in symptomatic patients necessitates additional imaging.
- Velocity-based estimation of carotid artery stenosis may need to be adjusted in certain circumstances:

- Higher velocities often seen in women than in men.
- Higher velocities often seen 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.

2. 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.

1.4 Management

1.4.1 Medical - 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. (A. Ross 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)

💡 Take a Listen

Check out [our conversation with Prof Ross Naylor](#) where he discusses in depth his decision making for asymptomatic carotid disease and other controversies in carotid disease

management.

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 hyperacute 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 2019)

Hyperlipidemia

- 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, Loney-Hutchinson, and McFarlane 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; P. W. Wilson et al. 1997) For more on smoking cessation, see Section [15.2.2.1](#)

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 2019)

1.4.2 Medical - 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.
 - BP management post stroke **with thrombolysis** includes treatment of BP to <185/110 prior to administration of thrombolysis, then keep <180/105 for 24hrs post administration.
- <6 hours from onset of symptoms - catheter directed therapy
- After 6 hours, there is limited benefit to thrombolytic therapy.
 - BP management post stroke **without thrombolysis** includes an initial period of permissive hypertension, treat only if >220/120. Slowly lower BP to target of 140/90 over 7-14d - particularly important to go slow in patients with intra- or extra-cranial large vessel stenosis.
 - Contraindications to permissive hypertension include active CAD, heart failure, aortic dissection, hypertensive encephalopathy.

1.4.3 Surgical - Carotid Endarterectomy

1.4.3.1 Indications

- 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.

1.4.3.2 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; K. 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, Vaniyapong, and Rerkasem 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.(A. Rerkasem et al. 2021)
 - **Stump pressure-** Clamp the inflow and place a butterfly attached to a-line tubing into the internal carotid artery. If the stump pressure is >40mmHg the surgeon can chose to proceed without shunt placement, if <40mmHg then a shunt should be placed prior to proceeding. There remains some controversy with some literature advocating cutoff as high as 50mmHg or as low as 30mmHg.(Arnold and Perler 2019; Chia et al. 2020)
 - **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. 2007b) For more see Section 20.3.
- **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 inferior 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 Section 20.2.
 - 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)

1.4.3.3 Complications

- Post-operative stroke - 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.
- ESRD patients have higher rates of perioperative stroke, but also have higher rates of stroke if not revascularized. (Klarin et al. 2016)
- 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, Yoshimura, and Kokuzawa 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; Aburahma and Perler 2022)
- Carotid artery patch infections
 - Rare complication (<1%) presenting as phlegmon, pseudoaneurysm, sinus tract or carotid cutaneous fistula.(Patrick A. Stone et al. 2011)
 - Treatment is excision and replacement with autologous tissue.(Fatima et al. 2019)

1.4.3.4 Outcomes and Surveillance

- Recommend follow up US duplex of bilateral carotids at 30 days. If stenosis >50% is identified, then further imaging is indicated.
- Ipsilateral restenosis
 - 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)
 - Risk factors for restenosis include hyperlipidemia(LaMuraglia et al. 2005), female gender, primary closure without patch, younger age, and certain plaque characteristics-abundance of smooth muscle, absence of macrophages/lymphocytes, and lipid core <10%.(Hellings et al. 2008; Pauletto et al. 2000)
- 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.

1.4.4 Endovascular - Carotid Artery Stenting

1.4.4.1 Indications

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.

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.

1.4.4.2 Technique

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.(Kwollek 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; Kwollek 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; A. R. Naylor et al. 2018)

Take a Look

Check out [our How I Do It Presentation](#) where Dr Christopher Henry reviews their approach to TCAR at Baylor Scott & White.

1.4.4.3 Outcomes and Surveillance

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)

1.4.5 Summary of Prospective Trials

1. Asymptomatic Carotid Atherosclerosis Study (ACAS)(Walker 1995)
 - 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

Check out [our conversation with Dr. William Jordan](#) where he discusses this landmark NASCET 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, Rodseth, and Biccard 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.(Voeks et al. 2011)
 - 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.
- Recent multi-institutional post market surveillance study demonstrated found TCAR outcomes to be “broadly similar” to carotid endarterectomy.(Kashyap et al. 2019)

Take a Listen

Check out [our conversation with Dr Jeffrey Jim](#) where he discusses details of comparative effectiveness research and how it has been applied to TCAR.

1.5 Uncommon Carotid Disease

1.5.1 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 (50-99%) will benefit from CEA before or concomitant with CABG. The timing of the intervention depends on the clinical presentation and institutional experience (*grade 2C*). (Aburahma and Perler 2022)
- Patients with severe bilateral asymptomatic carotid stenosis, including stenosis and contralateral occlusion, should be considered for CEA before or concomitant with CABG (*grade 2C*). (Aburahma and Perler 2022)

- Patients undergoing simultaneous CEA/CABG demonstrate highest mortality. (A. R. 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.(E. B. S. 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. 2017a; Nouh, Remke, and Ruland 2014)

1.5.2 Vertebrobasilar Disease

Most common etiology is atherosclerosis, but can also be caused by embolism, dissection or migraines.(Lima Neto et al. 2017b)

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).(M. Morasch 2019; Berguer, Morasch, and Kline 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; C. A. 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.(George A. Antoniou et al. 2012; Coward et al. 2007)

1.5.3 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

1.5.4 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, Long, and Atkins 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)(Jeffrey W. Olin and Sealove 2011) - for more, see Section [12.2](#)

- Asymptomatic disease can often be treated with antiplatelets alone.
- Duplex ultrasound demonstrates classic “chain of lakes” appearance. For more see Section [20.2](#)
- 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)

1.5.5 Carotid Body Tumors (CBT)

1.5.5.1 Demographics

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)

Blood supply primarily from the external carotid artery.(Robertson et al. 2019; Davila et al. 2016)

Shamblin criteria utilized to determine difficulty of resection and is associated with rates of carotid reconstruction or cranial nerve injury.(Chow, Moore, and Lamuraglia 2019; Shamblin et al. 1971)

1. Type 1 - abut but do not involve internal or external carotid arteries or cranial nerves can be easily dissected.
2. Type 2 - partially surround external carotid artery or cranial nerves and may be dissected free but risk injury to these structures.
3. Type 3 - fully surround either internal or external carotid artery or cranial nerves and pose a high risk of injury and need for reconstruction.

1.5.5.2 Management

Risk of injury or revascularization during resection determined by Shamblin criteria as discussed above. In patients with Shamblin Type 3 or with involvement of the internal carotid, these patients should be evaluated for carotid artery 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., n.d.; Tansavatdi et al. 2015)

Embolization if tumor arterial inflow may help reduce risk of perioperative blood loss but is not associated with reduced cranial nerve injury. Resection should take place 24-36hrs after embolization.(Power et al. 2012)

2 Trauma - Cerebrovascular

Authors: *Kevin Kniery, Nicole Rich, Nakia Sarad, and Todd Rasmussen*

These trauma episodes were developed in collaboration with [Behind the Knife: The Premier Surgery Podcast](#).

Vascular trauma requires close collaboration between vascular surgeons and trauma surgeons. The decision of which specialty should manage which injuries vary across different centers. Through these trauma chapters we will discuss what management decisions the majority of vascular surgeons should be comfortable managing as a part of a multidisciplinary trauma team.

💡 Take a Listen

Check our [debate between leading trauma and vascular surgeons](#) about how to best develop a collaborative team to manage vascular trauma.

For relevant images and a more in-depth discussion of this topic, please review **Chapter 20: Neck and Thoracic Outlet**(Magee and Weaver 2022) in Dr. Rasmussen's 4th Edition of *Rich's Vascular Trauma*.

2.1 Neck Exposure Techniques

2.1.1 Carotid Artery

Anatomy

- Right common carotid – originates from brachiocephalic artery
 - External landmark: right sternoclavicular joint
- Left common carotid – originates from aortic arch in superior mediastinum
- Common carotid artery (CCA) is within the carotid sheath
 - Contents:
 - Common and Internal carotid (medially)

- Internal jugular vein (laterally)
- Vagus nerve (posteriorly)
- Anterior margin: sternocleidomastoid (SCM) and omohyoid muscle
- Posterior margin: longus colli and longus capitis muscles
- Medial margin: esophagus/trachea
- Common carotid bifurcates to external and internal at level of superior border of thyroid cartilage
 - External carotid artery
 - Medial to internal carotid artery (majority of course)
 - First branch is superior thyroid artery (near bifurcation)
 - Terminate in parotid gland, divide into superficial temporal and maxillary arteries
 - Internal carotid artery
 - No extracranial branches
 - Lateral to external carotid artery until level of skull base, where it crosses medially
 - Enters the skull base through the carotid canal behind the styloid process
 - Facial vein is the anatomical landmark that approximates the location of the carotid bifurcation (deep to it).
 - Hypoglossal nerve (CN XII) and posterior belly of digastric muscle superficially cross the internal and external carotid arteries at the angle of the mandible
 - Glossopharyngeal nerve (CN IX) crosses in front of internal carotid artery, superior to hypoglossal nerve (CN XII)

Exposure

- Longitudinal incision along anterior border of SCM, extending from suprasternal notch to mastoid process
 - For proximal common carotid artery control, would do combination of SCM incision with median sternotomy
- Incise platysma and expose anterior border of SCM, retract laterally
 - Identify and avoid Accessory nerve (CN XI) that enters SCM

- Small branches of external carotid should be ligated to adequately mobilize the SCM and expose carotid sheath
- Carotid sheath is visualized and incised longitudinally
 - Omohyoid muscle may require division if proximal exposure required
- Identify Internal jugular vein, mobilize, and retract laterally
- Identify Vagus Nerve and avoid injury (posterior between internal jugular vein and common carotid)
- Place vessel loops around CCA, internal jugular, and vagus nerve
- Identify facial vein (overlying CCA bifurcation), Hypoglossal nerve (CN XII) and Ansa cervicalis
 - Ligate facial vein to further mobilize the internal jugular vein laterally and expose the underlying carotid bifurcation
 - Identify and protect Hypoglossal nerve (CN XII)
 - Ansa cervicalis may be ligated if necessary for exposure
- Dissect carotid bifurcation
 - Careful to not stimulate carotid body and cause hemodynamic instability
 - If access to distal internal carotid artery is required, may consider subluxation of mandible or mandibular osteotomy to gain appropriate exposure

2.1.2 Vertebral Artery

Anatomy

- First cephalad branch of subclavian artery
- Divided into 3 parts
 - Part I (proximal)
 - Origin at subclavian artery to C6 (enters transverse foramen)
 - External landmarks: found between two heads of SCM (sternal and clavicular) and clavicle
 - Vertebral artery runs between anterior scalene and longus colli muscles
 - Part II - Courses through bony vertebral canal (transverse foramen) from C6 to C1

- Part III (distal) - Courses outside vertebral canal from C1 to base of skull (enters through foramen magnum) and joins contralateral vertebral artery to form basilar artery (part of circle of Willis)

Exposure

- Will focus on exposure of Proximal Vertebral Artery
- Supraclavicular transverse incision extending between the sternal and clavicular heads of the sternocleidomastoid (SCM) muscle
- Incise platysma and continue dissection into base of triangle
- Carotid sheath is first vascular structure identified
 - Jugular vein – lateral
 - Common carotid – medial
 - Vagus nerve – posterior
- Identify scalene fat pad between two heads of SCM and clavicle, dissect to expose anterior scalene muscle
 - Careful of phrenic nerve as it runs on surface of anterior scalene muscle
- Divide anterior scalene muscle to visualize the subclavian artery and two of its branches (thyrocervical trunk and internal mammary artery). Control each branch.
- Proximal VA is located deep to supraclavicular artery and anterior to groove between C7 vertebral body and transverse process
- Proximal VA is between anterior scalene (laterally) and longus colli (medially)
- Best found through palpation with tip of index finger within groove of C7 vertebral body and transverse process
- Avoid injury to vertebral venous plexus as it is anterior to VA
- Use right angle clamp to dissect out VA

Detailed exposure techniques can be found here(E. Kwon, Grabo, and Velmahos 2019)

2.2 Tenets of Cerebrovascular Injury

- Control of catastrophic bleeding — with manual compression and resuscitation
- Ensure that injury is not causing airway compromise
- Evaluate the neurological status of patient — have a thorough baseline neuro exam and look for signs of ischemia, hemispheric stroke, arousability/mental status
- Prevent secondary injury — maintain blood pressure to maintain cerebral perfusion pressure and prevent hypoxia
- After appropriate exposure and control, recommend that patient be heparinized during carotid artery repair. Be prepared for bleeding, especially in patients with polytrauma. Resuscitate appropriately until repair completed.

2.3 Blunt Trauma

- Mechanism of Injury: history of a motor vehicle collision, blunt force to neck, severe hyperextension/rotation/flexion injuries, etc.
- External signs of injury: marks on neck or around the thoracic inlet (i.e. “seatbelt sign”), unilateral neurodeficits (suggestive of hemispheric ischemia or stroke)
- Accompanying injuries: concomitant closed head injuries (TBIs), complex facial fractures, cervical vertebral body and transverse foramen fractures

2.3.1 Evaluation

- CTA is gold standard. Limitation, image is static.
- US duplex combines B mode ultrasound with pulse doppler. Limitation in visualization of injury above the angle of the mandible (distal internal carotid and proximal thoracic outlet)
- These imaging modalities are used to assess visibility of defect (i.e. dissection, thrombus, intimal flap) and degree of flow limitation by measured velocity.

Denver Grading Scale for Blunt Vascular Carotid Injury (BCVI)(Biffl et al. 2001)

- Grade I: Luminal irregularity or dissection with < 25% luminal narrowing
- Grade II: Dissection or intraluminal hematoma with > 25% luminal narrowing, intraluminal thrombus, or raised intimal flap
- Grade III: Pseudoaneurysm
- Grade IV: Occlusion
- Grade V: Transection with free extravasation

2.3.2 Management

Considerations

- Consider the overall injury severity of the trauma patient with any concomitant injuries that may prevent antiplatelet/anticoagulation initiation (i.e. TBI) or intervention(Bromberg et al. 2010)
 - Oftentimes have other blunt vascular injuries, such as aortic injuries, that need to be addressed as well
 - Can consider delay of intervention (approx. 7 days) to decrease risk of neurological events
- If there is a change in the neurological exam after initiation of medical management, considered as failure of medical therapy and surgical intervention is indicated
- If unable to obtain a reliable neurological exam, can consider transcranial doppler or EEG if accessible, but is not mandatory
- Continue supportive measures to prevent secondary injuries, i.e. optimize cerebral perfusion pressure
- Repeat imaging with change in neurological status
- Serial imaging important in assessing whether injury has evolved
- Endovascular repair usually involves transfemoral approach for access with arch aortogram, selection of defective carotid vessel, and placement of the endovascular treatment (ie: stent graft, bare metal stent, coil embolization of pseudoaneurysm)

Carotid Artery Injury Treatments

- Grade I and II injuries are recommended to be treated medically with antiplatelet/anticoagulation therapy if patient is able to tolerate
 - Repeat CTA imaging in 5-7 days while inpatient to ensure that injury has not evolved in acute phase
 - Follow-up CTA imaging within 1 month of discharge as outpatient to assess healing. Imaging needs to be continued until there is resolution of lesion.
 - Grade I lesions typically improve over time, Grade II lesions or higher have a greater chance of evolving (approximately 60%)
- Accessible Grade III and V carotid injuries should be repaired.

- Grade III pseudoaneurysms should be repaired endovascularly with a covered stent (ie stent graft that covers the opening of the pseudoaneurysm) or bare metal stent (maintains integrity of the wall). Through these stents, coils can be placed to thrombose the pseudoaneurysm. Flow needs to be maintained.
- Open approach can be considered for Zone II injuries
- Grade I and IV carotid injuries, and inaccessible Grade II and III carotid injuries should be treated with medical management (antiplatelet/anticoagulation)
 - Grade IV injuries without associated injuries that preclude use of anticoagulation, would heparinize and proceed with anticoagulation (avoid bolus of heparin, goal PTT 50-70 within first 24 hours). Monitor for bleeding. Transition to oral anticoagulant for 30-90 days.
 - Grade IV injuries with contraindications for anticoagulation, would recommend dual antiplatelet therapy (aspirin and clopidogrel)
 - Overall, anticoagulation is preferred if there are no contraindications. If there are, then dual antiplatelet therapy is preferred.
- Grade V and persistent Grade III should be repaired surgically

Vertebral Artery Injury Treatments

- Special Considerations
 - Attempt to determine whether the dominant or diminutive vertebral artery is involved
 - Treatment is again based on severity with grading system (Denver Grading Scale), consider location and extent
 - Consider any associations with a basilar artery defect or posterior circulation stroke
 - Grade I-IV vertebral injuries should be treated with medical management as first line, approximately 90% self-resolve
 - Endovascular interventions can be considered for those patients with severe injury and/or who are symptomatic
 - Usually performed by neuro-interventionalist as the injury is located within the skull base
 - Serial imaging still needs to be performed to evaluate healing (similar manner to carotid injury)

- In the setting of trauma, if there is an expanding hematoma due to vertebral artery injury and open approach is performed to control bleeding, vertebral artery is usually ligated in this scenario
 - Vertebral reconstructions are not well-suited for traumas

Guidelines and treatment algorithm can be found here(Geddes et al. 2016)

2.4 Penetrating Trauma

Levels(Roon and Christensen 1979)

- Zone I: thoracic inlet (sternal notch) to level of cricothyroid cartilage
- Zone II: cricothyroid cartilage to angle of mandible
- Zone III: angle of the mandible to the base of the skull

[Images of the neck zones can be found here](#)

2.4.1 Management

Injury to Zone I and Zone III

- If hemodynamically stable, requires imaging prior to consideration of intervention/repair
 - Imaging: CTA and/or angiogram
 - CTA positive » Endovascular intervention vs. OR
 - Consider concomitant injury to aerodigestive tract as well
- If hemodynamically unstable with/without hard signs (aerodigestive or neurovascular injuries)
 - Tamponade » Secure Airway for Air leak/hematoma » OR

Injury to Zone II (violation of platysma)

- Direct to OR for exploration, especially with high suspicion of carotid injury and neurovascular deficits (hard sign)
- Repair carotid artery if injured, even if thrombosed.
 - The risk of reperfusion injury causing an intracranial hemorrhage was traditionally considered a reason to not repair.

- However, overall mortality and final neurostatus has shown to be better with intervention, even if patient started with neurodeficits.
- Requires communication with operative team (i.e. anesthesia) to mitigate hypoperfusion
- Consider concomitant injury to aerodigestive tract
- Scenario: Patient has large cerebral infarct on same side as carotid injury.
 - Continue to OR to explore
 - Preferred management is to repair and attempt reperfusion of hemisphere, possibly perfusing a previously ischemic penumbra
 - Ligation usually not an option.
 - Unless carotid is completely occluded and there is no retrograde bleeding after attempted thrombectomy » there is potential for ligation, but not readily considered.
 - Observation without repair can risk evolution of the injury into pseudoaneurysm with need to repair and reoperate days/weeks later - Outcome of this repair is likely a stroke

Repair Techniques

1. Appropriate exposure of carotid artery (see above for details)
2. Gain proximal and distal control
3. Identify injury
4. Remove burden of thrombus (if present)
 - Thrombus burden must be removed prior to repair
 - Via flushing from back-bleeding and forward-bleeding of internal carotid artery - Via Fogarty or thrombectomy catheter, carefully
 - Recommend 2 French or 3 French size
 - Pass only 1-2 cm up into distal internal carotid
5. Debride any devitalized tissue prior to repair
 - May cause defect to be larger, but repair requires healthy tissue planes

6. Repair carotid - Heparinization required when repairing carotid artery and/or clamping. Be efficient, as these patients usually have significant polytrauma and other areas that are susceptible to bleeding which will need to be controlled when heparin administered. Repair options, include:

- Primary Repair
- Patch Repair - Used for short-segment injuries. Conduits include:
 - Dacron or bovine pericardium
 - Autologous vein - Not first-line due to extra time required for harvest. Used if there is concern for contamination (i.e. shot gun wound with large soft tissue injury, aerodigestive injury, etc)
- Interposition Graft - Used for long-segment injuries. Conduits include:
 - Autologous vein (great saphenous vein) - Good option when there is concomitant aerodigestive injury. Consider shunting while waiting for harvest
 - Synthetic (i.e. Dacron/PTFE)
- Transposition of Internal Carotid Artery to External Carotid artery - Can avoid need for shunting

Consideration of Shunting

- In most cases, shunting not required because adds extra level of complexity to trauma case that may not have the luxury of time
- Cases where shunting can be used:
 - Interposition graft repair for long-segment carotid injury that requires vein harvest > shunting can be performed to reperfuse the affected brain hemisphere while waiting for conduit preparation
- Typical shunts used: Argyle or Sundt

Assessment of Repair

- Assess repair with removal of thrombus and once back-bleeding restored
- Duplex US
 - Best option when time is limited to assess flow
 - Usually also used most often with elective carotid repairs

- If ultrasound is not available, then a continuous wave doppler is at least necessary to demonstrate monophasic signal with diastolic flow in the revascularized internal carotid artery.
- Intraoperative arteriograms
 - If have luxury of time to perform and set up is appropriate, used for completion studies
 - Documents complete evacuation of distal thrombus
 - Performed by placing 18-gauge butterfly needle in CCA (remove air) and inject contrast to view repair and distal internal carotid to ensure lack of thrombus

Consideration of Drains

- If there is contamination (aerodigestive injury), then can place a small JP drain (7mm) to protect the repair
- Buttressing repair in contaminated cases usually not used as the neck is highly vascular
 - Can consider a muscle flap to separate arterial injury from esophageal injury (commonly not seen) with gross contamination

Guideline Algorithm for Penetrating Neck Trauma(Sperry et al. 2013)

2.5 Internal Jugular Vein Injuries

- If patient has significant TBI, consider repairing Internal Jugular venous injuries to maintain venous outflow to that side of the brain — especially if there is a transduced venous pressure gradient. Cases include patients who have intracranial hypertension and require a decompressive craniectomy.
- Otherwise, able to ligate without much consequential effects in most patients due to appropriate amounts of collateral outflow.

Part II

Upper Extremity

3 Upper Extremity

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3.1 Overview

3.1.1 Anatomy

What are the zones of the upper extremity?(Karl A. 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?(Karl A. 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 which is a combined 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 opening 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 expose 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?

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 and anterior to the spine.

Of note, 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)

3.1.2 Etiology

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

Upper extremity ischemia <5% of patients with limb ischemia are a result of underlying atherosclerosis, which is in contrast to lower extremity.(Shuja 2019)

Vast majority of cases caused by embolic, autoimmune, or 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, CRP, ANA, RF
 2. Hypercoagulable screening

3.2 Vaso-occlusive Disease

3.2.1 Demographics

What are causes and symptoms associated with subclavian/axillary occlusive disease? (Jack L Cronenwett, Alik Farber, and Erica L. Mitchell 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. Forearm occlusive disease can be seen in advanced ESRD or DM where calcific atherosclerosis of radial/ulnar arteries is present.

- 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)
- Less common causes include Buerger's disease or Raynaud Phenomenon

3.2.2 Management

3.2.2.1 Chronic Limb Threatening Ischemia

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)

Innominates 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, Morasch, and Kline 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; 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, Robinson, and Jennings 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, seen more on the L with thoracic duct injury
 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. (M. D. Morasch 2009a)

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 bypass with anterior or retropharyngeal tunneling.

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, Smith, and Arko 2007)

Surgery: GSV vein bypass remains standard for revascularization with bypasses to superficial or deep palmar arch have good patency rates. Tunneling is subcutaneous if to distal ulnar or superficial palmar arch whereas anatomical to distal radial artery over the anatomic snuffbox.(B. B. Chang et al. 2003; Masden, Seruya, and Higgins 2012; Spinelli et al. 2010)

3.2.2.2 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, Goldstone, and Olofsson 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.(D. D. 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, Ilyas, and Thoder 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 pollicus longus.(Ronel, Mtui, and Nolan 2004)
- Volar/Henry approach decompresses lateral and volar compartments with a single incision, includes carpal tunnel release.

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). (Gregory J. 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, Modrall, and Clagett 2005)

3.3.2 Raynaud's Phenomenon

What is Raynaud's and what causes it?

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. (Shuja 2019; Gregory J. Landry 2019)

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.(Gregory J. 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 sympathectomy 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.(Gregory J. Landry 2013)

3.3.3 Ergotism

What is Ergotism?

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.**(Stanley, Veith, and Wakefield 2014)

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. Can be seen with ergot alkaloid migraine medications. 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. Avoid or delay amputations as much as possible to maximize amount of tissue recovery.

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 (upper and lower) of tobacco users distinct from either atherosclerosis or immune arteritis(Jack L Cronenwett, Alik Farber, and Erica L. Mitchell 2020; Le Joncour et al. 2018)

What clinical criteria can help diagnose Buerger's?

Diagnosis of Buerger's disease requires 5 criteria(Akar 2019)

1. Smoking history
2. Onset before age 45 or 50
3. Distal extremity ischemia with infrapopliteal arterial occlusions and often upper limb involvement
4. Classic angiographic findings of segmental occlusions of distal arteries with corkscrew collaterals
5. Absence of atherosclerotic risk factors (besides smoking, of course), autoimmune disease, diabetes or proximal embolic source.

Although rarely are biopsy sent, pathology will show panvasculitis within small and medium-sized arteries, highly cellular intraluminal thrombus (contains CD68, Cd3, Cd8 inflammatory cells), preserved elastic lamina structure, unlike atherosclerotic lesions.(Akar 2019; M. Kobayashi et al. 1999)

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)
- Digital brachial index (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.(K. 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. May even require inpatient admission for intensive smoking cessation therapy.(Hooten, Bruns, and Hays 1998) It is important to remember following treatments will likely fail without smoking cessation.(J. W. Olin 2000)
2. If smoking cessation does not improve, medical management with antiplatelet agents, immunomodulators, vasodilators (Calcium channel blockers and cilostazol(Dean and Satianni 2001)), anticoagulants(Kubota et al. 1997), and IPC.(Montori et al. 2002)
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(Jeffrey W. Olin 2018)

3.3.5 Large Artery Vasculitis

What are common characteristics for patients who are suspected to have a large vessel vasculitis? (Shanmugam 2019; 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 (ESR/CRP), 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. Involves aorta and major arch branches(Ehlert and Abularrage 2019a)
2. Young patients (20-40 years) and female in 80-90% of cases, Asian populations
3. American College of Rheumatology (ACR) Criteria(Maz et al. 2021)
 1. Onset <40 years
 2. Claudication of an upper 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

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 (temporal) artery biopsy (gold standard test)
3. Other symptoms include jaw pain with mastication or visual changes
4. Associated with Polymyalgia rheumatic, characterized by morning stiffness in shoulders/hips occurring in 40-50% of patients.(Weyand and Goronzy 2014)
5. Arteriography/MRA/CTA/PET may be used to assess large vessel involvement - classic finding is smooth, tapering stenosis.

3.3.5.3 Management

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 - Avoid intervention in the acute setting. Once in 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)
 - Mid aortic syndrome likely needs open repair from uninvolving aorta-usually thoracic aorta-to bifurcation bypass with jump graft to involving visceral vessels. (Ehlert and Abularage 2019a)

3.3.5.4 Surveillance

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)

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 2019; Weyand and Goronzy 2003)

Behçet Disease - recurrent oral pathos ulcers, genital ulcers and uveitis.(Shanmugam 2019; Weyand and Goronzy 2003)

3.4 Aneurysmal Disease

3.4.1 Subclavian Artery Aneurysms

How are subclavian aneurysms caused and how can they present? (Baig and Timaran 2019)

Etiology

- Degenerative - atherosclerotic or due to aberrant right subclavian with degenerative changes in proximal subclavian known as “Kommerell diverticulum”(Tanaka, Milner, and Ota 2015)
- 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.

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.(Tanaka, Milner, and Ota 2015)
- Rarely rupture

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

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.(Tanaka, Milner, and Ota 2015)

3.4.2 Axillary Artery Aneurysms

How are axillary aneurysms caused and how can they present?

Etiology

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

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?

Evaluation

- Ultrasound
- CTA/MRA of upper extremity

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?

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 Chapter 22
- IV drug abuse - infected pseudoaneurysms in antecubital fossa.
- Connective tissue disorders (ex. type IV Ehlers Danlos)

Presentation

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

What are diagnostic studies and treatment modalities for brachial aneurysms?

Evaluation

- Duplex Ultrasound

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

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.
- Neurological symptoms from median nerve compression require an 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 on year ago?

Small pseudoaneurysms <3cm have a high rate of spontaneous thrombosis. However, larger psuedoaneurysms 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, Sayit, and Tanrivermis Sayit 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.(Eskandari and Morgan 2020)

3.5.1 Hand-Arm Vibration Syndrome

Etiology

- Vibrating handheld machines (i.e. 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

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

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

Management

- Avoidance of vibratory tools
- Nifedipine (Ca²⁺ channel blocker) in advanced cases
- IV prostacyclin (i.e. prostacyclin) for digital gangrene
- Surgery-cervical sympathectomy or digital sympathectomy rarely needed

3.5.2 Hypothenar hammer syndrome

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 there hands as a hammer are at risk for disease
- Repetitive trauma leads to thrombotic occlusion, aneurysm formation or both

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

Evaluation

- Duplex ultrasound

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

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?

Acrosteolysis

- 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

3.6 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.

3.6.1 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

3.6.2 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

Management

- 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 pectoralis minor muscle

3.6.3 Thoracic Outlet Syndrome

Thoracic outlet syndrome and cervical rib abnormalities are covered extensively in another chapter. For more information see Chapter 4

3.7 Vascular Trauma-Upper Extremity

This is discussed in detail in Chapter 19, so we will go over some important specifics for upper extremity vascular injury. (David S. Kauvar and Kraiss 2020)

3.7.1 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; V. 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.

3.7.2 Axillary Artery Trauma

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

Etiology

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

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

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. Discussed in more detail in Section 9.3

3.7.3 Brachial Artery Trauma

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

Etiology

- Frequently associated with humerus fractures/elbow dislocation. Seen often in children.
- Penetrating trauma

Evaluation

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

Management

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

3.7.4 Radial/ulnar artery trauma

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

Etiology

- Associated with significant soft tissue pattern

Evaluation

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

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

3.8 Upper Extremity Lymphedema

3.8.1 Etiology

Often due to breast cancer therapy and axillary lymph node dissection.(Morrell et al. 2005)

3.8.2 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, Riffenburgh, and Johnstone 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)

4 Thoracic Outlet

Author: *Nedal Katib and Matt Smith*

4.1 Overview

4.1.1 Demographics

Thoracic Outlet Syndrome (TOS) is 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)

4.1.1.1 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

1. Subclavian vein
2. Phrenic nerve
3. Anterior scalene muscle attachment to the first rib
4. The subclavian artery
5. The brachial plexus
6. The middle scalene muscle

Three spaces where the neurovascular structures are at risk of compression

1. 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’ because it only one articular facet.
 - 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 muscle, which is tethered to the C7 vertebrae.

2. Costoclavicular Passage - 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)

3. Subcoracoid Space - This space is best appreciated by intimate knowledge of three things(Gary G Wind and R. James Valentine 2013)

1. 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.

2. Pectoralis Minor Muscle:

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

3. 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.

Anomalous Anatomy

- **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.
- **Cervical Rib** - 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)

4.1.1.2 Etiology

nTOS

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

aTOS

1. Cervical Rib and Anomalous First Rib - most common
2. Scalene Triangle compression

vTOS

1. Costoclavicular Passage - most common
2. Subcoracoid Space

4.1.1.3 Presentation

- Identify symptoms and thoroughly interrogate timing
- Exclude history of trauma - clavicle fracture and malunion
- 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.

4.1.2 Evaluation

4.1.2.1 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 maneuver 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)

4.1.2.2 Imaging

Vascular Lab

- Digital Brachial Index (DBI)
- Arterial Duplex - can identify subclavian artery aneurysms.
 - Can also be performed with provocative maneuvers
- Venous Duplex

Axial Imaging

- 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

Angiography

- In the setting of vTOS, some advocate for upper extremity venogram with provocative maneuvers to evaluate upper arm collaterals and compression

Other studies

- Electromyography and nerve conduction studies for nTOS - performed after evaluation with neurologist and physiotherapist.
- In the setting of nTOS, scalene block can be diagnostic if the patient has temporary relief.

4.1.3 Management

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

Transaxillary

- *Advantages:*

- Cosmetically more appealing as it has a limited hidden scar
- Allows for removal of a significant portion of the rib anteriorly and posteriorly

- *Disadvantages:*

- Difficult to visualize the anatomy, dependent on good assistance
- Risk of injury to T1 nerve root, phrenic nerve, long thoracic, brachial plexus , subclavian vein and arterial with limited exposure to repair
- Not able to approach cervical ribs, scalene triangle or patch vein.

💡 Take a Look

Check out [Dr. Freischlag's operative video](#) where she reviews her approach to a transaxillary rib resection.

Supraclavicular

- *Advantages*

- Good for scalene triangle access and debulking and cervical rib resection
- Required for aTOS if arterial reconstruction necessary

- *Disadvantages*

- Unable to decompress venous compression or visualize vein adequately
- Cosmetically less appealing

💡 Take a Look

Check out [our How I Do It Presentation](#) where Dr Westley Ohman and Dr. Robert Thompson review their approach to a supraclavicular rib resection.

Infraclavicular (Siracuse et al. 2015)

- *Advantages*

- Good access for venous decompression
- Allows for excision of subclavius muscle and costoclavicular ligament
- *Disadvantages*
 - Unable to expose subclavian artery or decompress brachial plexus.
 - Difficult to access most posterior aspect of rib
 - Cosmetically less appealing

Paraclavicular

- *Advantages*
 - Useful if mixed etiology TOS to adequately decompression all neurovascular structures
- *Disadvantages*
 - Requires two incisions one above and below the clavicle

4.1.3.1 Complications

- Post operative patients with hemodynamic instability and ipsilateral effusion on xray should go back to OR for exploration and hemorrhage control. (Rinehardt, Scarborough, and Bennett 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, Salem, and Kaiser 1999)

4.2 Venous (vTOS)

4.2.1 Demographics

- Incidence: 2/100,000 persons
- Age: 18 years to 30 years (Karl A. Illig and Doyle 2010)
- M>F
- **Paget Schröetter 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 synonymous.
- Paget Schroetter Syndrome accounts for 10-20% of all upper extremity deep vein thrombosis. (Sekhar 2018)

4.2.1.1 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)

4.2.2 Evaluation

Upper extremity venogram is a very useful modality for diagnosis. Compression likely will not be seen in normal orientation, however can be demonstrated with provocative maneuvers including 90 degree arm abduction and external rotation.(Moriarty et al. 2015)

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

4.2.3 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

4.2.3.1 Medical Management

As per ACCP Guidelines: Initial management is anticoagulation regardless of etiology. (Kearon, Akl, Ornelas, Blaivas, Jimenez, Bounameaux, Huisman, King, Morris, Sood, Stevens, 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)

Catheter Directed Thrombolysis (CDT) has been considered superior to anticoagulation alone in minimizing valvular damage due to residual clot and carries a lower risk of intracranial hemorrhage.(Urschel and Patel 2008)

- Systemic Lysis – non favored due to risk of intracranial hemorrhage. (Grunwald and Hofmann 2004)
- Optimal timing of CDT - Within 14 days of onset of thrombosis. Excellent results have been reported following CDT if initiated before 14 days. (J. J. Wilson, Zahn, and Newman 1990)

Patient should be maintained in a compression sleeve until definitive decompression can be performed.

4.2.3.2 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)

4.2.4 Summary

4.2.4.1 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, Hunter, and Dietz 2007)
- Post decompression venography and treatment 2 weeks post rib resection may help to prevent recurrence and long term vein patency. (K. Z. Chang et al. 2012)

4.2.4.2 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)
 - 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
 - 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
 - Results showed superior patency with FRR and PLASTY and FRR compared to anticoagulation alone.
 - 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. Freischlag J - The art of caring in the treatment of thoracic outlet syndrome. *Diagnostics* 2018.(Freischlag 2018)
 - Review of key publications guiding her Dr. Freischlag's care of these patients.

 Take a Listen

Check our interview with Dr Julie Freischlag where she discusses her career and management of patients with thoracic outlet syndrome.

4.3 Arterial (aTOS)

4.3.1 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. For more see Section [3.3.4](#)
- Arterial Embolization – 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%)

4.3.2 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; E. Criado, Berguer, and Greenfield 2010)

4.3.3 Management

Symptomatic patients are generally indicated for treatment. Unlike asymptomatic patients in whom it may be appropriate to manage conservatively. (Boll and Valentine 2019) Symptoms are classified according the the Scher staging system.

- **Scher Staging of aTOS**(Scher et al. 1984)
 - 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.

4.4 Neurogenic (nTOS)

4.4.1 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.

4.4.1.1 Presentation

- Symptoms (Sadeghi-Azandaryani et al. 2009; Sanders, Hammond, and Rao 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

4.4.2 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, Brooke, Likes, et al. 2012)
- 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.

5 Hemodialysis

Authors: *Young Lee and Matthew Smeds*

5.1 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 healthcare 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 $<25\text{mL/min}$ 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 before 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 non-dominant 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. Placement of AV access in a patient with undiagnosed SVC stenosis or obstruction can cause SVC syndrome.(Kalra, Bjarnason, and Gловички 2019)

Beyond signs of central venous stenosis, when examining a patient, an Allen's test should always be performed to evaluate palmar 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 particularly in smaller patients.(Huber et al. 2002; Smith, Gohil, and Chetter 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 difference 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-Appell (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)

Take a Listen

Check out [our episode on the history of AV Access](#) where we interview Dr. Kenneth Appell, one of the surgeons who pioneered this surgical technique.

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, Seo, and Ryu 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, 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 option, however, studies have shown no improvement in graft patency with HB-PTFE compared to standard PTFE grafts.(Shemesh et al. 2015; Davies, Anaya-Ayala, and El-Sayed 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.(Julien Al Shakarchi and Inston 2019; J. K. 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/grafs. Patency of femoral vein transposition is better than hybrid catheter/graft 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, Niyyar, and Vachharajani 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.(J. 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, n.d.)

5.2 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.

 Take a Listen

Check out [our episode on the basics of AV Access placement](#).

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.(G. A. 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.(G. A. Antoniou et al. 2009; Bourquelot et al. 2012; Gradman, Laub, and Cohen 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.

5.2.1 Anesthetic Considerations

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)

Due to underlying comorbidities, it is often best to avoid general anesthesia in these patients. Many access procedures can be performed under regional or straight local anesthetic. There, it is very important to understand the pharmacokinetics and maximum doses of common local anesthetics.(Neal et al. 2018) Historically, it has been taught that the inclusion of epinephrine allows for higher dosages, but more recent reports do not include this adjustment.(Sztajnkrycer 2019) Common local anesthetics include:

- Bupivacaine - Long acting. Max SubQ Dose is 2mg/kg.
- Lidocaine - Medium acting. Max SubQ Dose is 4.5 mg/kg.
- Mepivacaine - Medium acting. Max SubQ Dose is 4.4 mg/kg.
- Ropivacaine - Long acting. Max SubQ Dose is 3 mg/kg.

 Check out this Calculator

MedCalc has a useful calculator for estimating maximum allowable volume of anesthetic based on patient weight and dose percentage.

5.2.2 Endovascular AV Fistula

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)

5.3 Maintenance and complications

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)

5.3.1 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?

First a duplex ultrasound can help to identify whether this is an inflow, outflow, or conduit issue. Once the likely source of the issue is identified, then there are multiple open or endovascular techniques to assist maturation.

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) Open procedures include vein patches, interposition vein grafts, vein transposition to proximal arteries, branch ligations, and vein superficialization.

5.3.2 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, Calligaro, and Sidawy 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, Calligaro, and Sidawy 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, Seo, and Ryu 2017; X. 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. Thrombosed fistulas are difficult to salvage, but thrombosed AV grafts have a high likelihood of successful recannulation and should be managed aggressively.

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, Ram, and Zibari 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, Son, and Salehi 2020)

5.3.3 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 effort induced ischemic pain.
- 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 immediately after AVF creation should first have arterial injury/thrombosis ruled out, but then after diagnosed with steal syndrome should undergo fistula ligation.(H. Schanzer and Eisenberg 2004; Yevzlin, Chan, and Asif 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.(Julien 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. One technique describes using intra-operative finger pressures and plication to result in intraoperative increase in finger pressure by 20% or a minimum of 90mmHg. 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, Kruger, and Scholz 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.

5.3.4 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 (IMN) 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.

5.3.5 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, Calligaro, and Sidawy 2008)

Prosthetic grafts can results in seroma from ultrafiltration of the graft and most resolve without intervention.

5.3.6 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, Remuzzi, and Boccardo 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.(Hossny 2014; M. S. Patel et al. 2015)

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)

5.4 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 patients requiring abdominal or inguinal surgery.

Which site is the most ideal site for a hemodialysis catheter?

The right internal jugular vein with a tip in at the cavo-atrial junction is preferred because it has the best patency. Subclavian veins are avoided due to high risk of stenosis and femoral veins are avoided due to infection risk.

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(V. A. 2006. W. Group 2006)

5.5 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.(S. Li et al. 2017)

Part III

Thorax

6 Aortic Dissection

Authors: *Matt Spreadbury, Adham Elmously, Einar Brevik, and Joseph Lombardi*

6.1 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

6.1.1 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

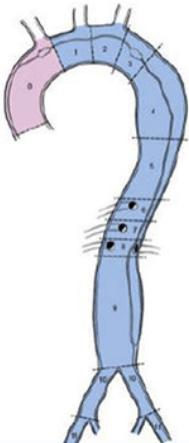
Debakey

- 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, Hughes, et al. 2020)

**Society for Vascular Surgery and Society of Thoracic Surgery
Reporting Standards for Type B Aortic Dissections**



Type	Proximal Extent	Distal Extent
A_D Entry tear: Zone 0	0	0
	1	1
	2	2
	3	3
	4	4
B_{PD} Entry tear: ≥Zone 1	5	5
	6	6
	7	7
	8	8
I_D Unidentified entry tear involving Zone 0	9	9
	10	10
	11	11
	12	12

**Anatomic Reporting of Aortic Dissections
are based on:**

- ✓ Location of Entry Tear (A vs B)
- ✓ Proximal & Distal Extent

EXAMPLES

- Type A₉:** Entry tear identified in zone 0 (A),
Distal extent in zone 9.
- Type B_{4,10}:** Entry tear is identified > zone 0 (B)
Proximal extent in zone 4,
Distal extent in zone 10.

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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.

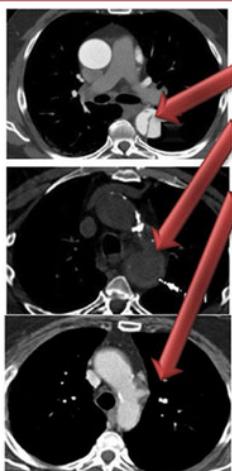
6.1.1.1 Penetrating Aortic Ulcer and Intramural Hematoma

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 Society of Thoracic Surgery
Reporting Standards for Type B Aortic Dissections**



DEFINITIONS		
Aortic Dissection	Tear in the intima that results in separation of layers of the media and allows blood to flow through the false lumen	
Intramural Hematoma	There is no identifiable direct communication between true and false lumen. Characterized by hyperdense, crescent-shaped hemorrhage within aortic wall	
Penetrating Aortic Ulcer	Atherosclerotic lesion that penetrates the internal elastic lamina of the aortic wall. Often diagnosed in presence of intramural hematoma	
Aortic Dissection Acuity		
High Risk	Refractory pain or HTN Bloody pleural effusion Aortic diameter > 40 mm Radiographic only malperfusion Readmission Entry tear: Lesser curve location False lumen diameter > 22mm	CHRONICITY
		Hyperacute < 24 hours
		Acute 1-14 days
		Subacute 15-90 days
		Chronic > 90 days
Complicated	Rupture 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 / malperfusion
 - Rupture or impending rupture
- High risk for early complication or continued growth (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 down stream 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) vs cocaine or Meth (younger patients)

Marfans, Loeys-Dietz, Vascular 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.

For more on the management of blunt aortic trauma, see Chapter [9](#)

6.1.2 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

6.2 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.

6.3 Management

What are the details of Type A treatment?

Operative treatment with a 30% operative mortality. Let cardiothoracic 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 aimed to reduce aortic wall stress.(G. C. Hughes, Andersen, and McCann 2013; Nienaber and Clough 2015) That means reducing the force of transmitted impulse down the aorta with blood pressure goals of 100-120mmHg and 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.(Trimarchi et al. 2006)

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 equalize the pressure in the true and false lumen. (Lombardi, Gleason, et al. 2020)

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.

Take a Listen

Check out [Dr Arko's conversation on the BackTable podcast](#) to learn more about the complications of TEVAR management in dissections.

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, Meyermann, and Lombardi 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.(M. D. Morasch 2009b; Matsumura et al. 2009)

7 TAAA

Authors: *Rachael Forsythe, Mohamed Barkat, Nicholas Greaves, and Michael Jenkins*

Contributors: *Sharif Ellozy, Leanna Erete*

This episode was developed as a collaboration between Audible Bleeding and [The Rouleaux Club](#) in the United Kingdom.

Treatment for thoracoabdominal aneurysms has become a bit of sub-specialization within vascular surgery, with the best outcomes achieved at high-volume centers with close partnerships with cardiothoracic surgery.(Cowan et al. 2003) While the majority of vascular surgeons may not manage these patients in their day to day practice, and many trainees have limited exposure, this topic is still fair game for examinations and should be reviewed. However, endovascular techniques are still evolving and practice varies widely between centers and so covering this topic may not be particularly high yield.

💡 Take a Listen

Check out [our conversation with Dr. Tom Forbes](#) where we discuss some challenges and opportunities in specialized aortic training and centralization of services.

This chapter was developed by surgeons in the United Kingdom, where manufactured custom devices have broader regulatory approval and are more widely available. The availability of these devices in the United States is more limited and so management decisions are different depending on what devices are available. However, the basic principles of open management and overall clinical decision making are shared no matter what side of the Atlantic ocean you practice.

7.1 Demographics

7.1.1 Anatomy

Can you take us through the Crawford classification to start off?

The Crawford classification is relatively recent (1986) and is a very practical classification, depending on body cavity and how to get to an aneurysm.(Crawford et al. 1986) This classification doesn't follow an expected pattern as getting either more extensive or less extensive.

1. **Type I** - These extend from the left subclavian down to just below the diaphragm, which distinguishes it from an isolated thoracic aneurysm, which you can get to just from the chest. Going into a second body cavity is a very important differentiating marker.
2. **Type II** - These are the biggies, they extend 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 lumbers—so big impact for cord supply, et cetera.
3. **Type III** - These extend from the mid chest down to and involving the viscera, renals, and bifurcation. These differentiate from Type 2 because you may not need to utilize full support and may risk a clamp and go approach.
4. **Type IV** - These are characterized by being accessible from the abdomen, in most patients, although it often does require division of the crus. 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.
5. **Type V** - This was an additional classification that was added by Dr. Hazim Safi's group later, which is a bit like a type III at the top of the type I at the bottom effectively.(Safi and Miller 1999)

 Take a Listen

Check out [our conversation with Dr. Hazim Safi](#) where he discusses the history of TAAA repair and much, much more.

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?

The overall incidence of infrarenal atherosclerotic abdominal aneurysms is going down every year in the national UK vascular registry, likely related to smoking reduction 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.

So that's an artificial increase in incidence, but also the rate of aortic dissection is on the rise and chronic post dissection aneurysms are increasing. In addition, I think it's a small group, but there is more knowledge about connective tissue disease, genetic studies and family screening, which perhaps is also a small part of annual increase. So over the next 10-20 years, aneurysms are not going to go away, and there is a huge number now within the national UK screening program under surveillance for small aneurysms.

7.1.2 Etiology

Are there major differences in presentation and etiology between thoracoabdominal aneurysms and abdominal aortic aneurysms from your experience?

On presentation, the majority of aneurysms are asymptomatic so they are most often found incidentally. Rarely, they get symptomatic and 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 but may have some atypical symptomatic presentations.

- Some that extend into the arch may present with hoarseness from recurrent laryngeal tension or bronchial compression.
- There is a group that cause dilatation of the crus. The crus of the diaphragm acts like an extrinsic wrap and can be extremely tight. These patients present with excruciating pain, radiating around their costal margin, which is a presentation not often thought of as a sign of an aneurysm.
- The larger extent and more chronic aneurysms may even present with things like weight loss and general poor health.

The other group that is a little different than infrarenal aneurysms, is the post dissection aneurysms. The majority of these are known and followed closely, however there was a time when patients with a type A dissections that extended all the way down were only followed with echo of the ascending and the rest was forgotten about. Many of them 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.

Do you think patients with thoracic aneurysms as a result of dissection or connective tissue disease 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—Marfans, Loeys Dietz, vascular Ehlers Danlos—are probably the tip of the iceberg. There are likely a number of other cases that we don't have genetic sequencing for, but behave differently than classic atherosclerotic aneurysms. 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.

For more about connective tissue related aortopathies, see Chapter 8

Also, be aware if it's something odd anatomically—a saccular bulge, an eccentric penetrating aortic ulcer (PAU), or 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 repair at an earlier threshold because they do not behave like a conventional fusiform aneurysm.

Can you talk us through the Ishimaru zones of the aorta?

Ishimaru is a useful classifications. There are a lot classifications in medicine, but it's nice to find one that's actually useful. This classification made sure that everyone was on the same page when reporting proximal seals zones for thoracic devices. That is important for two reasons:

1. The extent of coverage
2. The complications increased, the more proximally you go. The main complication for that is stroke.

So historically, Ishimaru decided to classify these zones as(Ishimaru 2004):

- **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 TEVAR 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 type II thoracoabdominal aneurysms, you're going to have a very different

outcomes from someone who's got mainly type IV aneurysms. Both classifications allow you to look at and compare data between groups.

For a discussion of the new SVS/STS classification system for aortic dissections, please see Chapter 6

7.2 Management

7.2.1 Ascending aorta and arch

The cardiac surgeons use many different techniques for managing an ascending aortic aneurysm or dissections, which is beyond the scope of this review. However, in preparation for management of aneurysms or dissections that extend beyond the arch, they often utilize an elephant trunk down 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 left problems for later.

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. 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, 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 then inverting it down the descending thoracic aorta. The benefit of that was when returning to repair descending thoracic segment via left thoracotomy, you could quickly open the aorta and clamp that Dacron for a ready, made proximal anastomosis. This was much easier, because it meant you didn't have to dissect all the way up to the left subclavian with scar tissue and a previous anastomosis.

Now, what industry realized is that they could help with this procedure by developing a Dacron device sized to be an arch replacement which had three or four ready-sewn 10 millimeter branches with an extra pipe for rewarming. These branches are used as bypasses to the innominate, carotid and subclavian and attached to that piece of Dacron was a stent graft, which could 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.

7.2.2 Isolated Thoracic Aneurysms

For isolated thoracic aneurysm treated with TEVAR, what is the optimal landing zone?

So I think the isolated, thoracic aneurysm is a perfect application for TEVAR because if you can get a good seal zone, proximally and distally, it provides an endoluminal approach and saves a thoracotomy, which is a massive difference for these 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 a 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.(Marrocco-Trischitta et al. 2019)

Modern day outcomes are very good, because I think modern devices have become much more conformable. We've learned many lessons to avoid too much oversizing and 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. Gothic arches, torturous and large sacks, are a problem and can 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.

For more on the endovascular management of traumatic aneurysms, see Chapter [9](#)

Do you utilize rapid ventricular pacing when deploying these stents?

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 tortuous 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.

As we have described TEVAR is a great option for the majority of isolated, thoracic aneurysms, but is there still a place for open repair?

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.

7.2.3 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 water to a greater or lesser extent as well as the abdominal aorta. We'll come back to type IV later on in this podcast.

So type II 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 II 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 atheromatous 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 TEVAR above. However, there wasn't the option for what we've now got from an endovascular perspective.

Take a Listen

The availability of fenestrated and branched devices is very different between the United Kingdom, Europe and the United States. In the UK, custom fenestrated and branched devices have received CE mark approval, which is similar to FDA approval in the United States. These devices do not have FDA approval in the United States, so access to them is far more limited.

Check out [our conversation with Dr. Gustavo Oderich](#) where we discuss the advances in endovascular techniques for branched and fenestrated devices in the United States.

Check out [our conversation with Dr. Benjamin Starnes](#) where we discuss the process of an investigation device exemption (IDE), which is how the majority of custom devices are performed in the United States.

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, atheromatous 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 III and type IV, where there really is a different option and these patients are in their sixties and seventies with classic atheromatous disease. The really, young fit non-connective tissue patient with a type II 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 were you 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?

In terms of the patient, for anything juxtarenal or type IV, 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. By breaking the table at that level, you can increase the exposure to that segment of the aorta.

However, there are some patients even with a type IV 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 harder 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 IV 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 II, 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 medially 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 lumbars or intercostals because you really don't want to cause a problem there because you can't 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 II 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 II 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 cannulating 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.

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 III 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 III 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 Carrel 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.

7.2.3.1 Complications

As you've mentioned, open repair is a high tariff, high risk operation, but apart from death, what are the 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 increase your risk of 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 lumbers. 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. Important aspects to reduce ischemia include:

- Getting the legs back in circuit early, whether that's removing the large sheaths, during a endovascular repair or getting the leg supply earlier in an open approach.
- Keeping the left subclavian in circuit.
- Maintain the mean arterial pressure in theater
- Place a spinal drain and ensure it is working.

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, can you explain in more detail the blood supply for the spinal cord and how this plays into the occurrence of spinal cord ischemia?

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 the level of the 9th-12th intercostal, 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 II 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 the differential position of that blood supply is why patients then get a particularly prominent 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, which 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 100 (which is equal to 10 g/dL in the USA), 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 artery and internal iliac artery supply. This is probably akin 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, drops in blood pressure, with hypotensive episodes really don't help.

When do you remiplant intercostal arteries?

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.

Several spinal protection 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 mad 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 that 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 perfusing the vital organs retrograde.

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.(M. P. Jenkins et al. 2011) 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. If the left renal is beyond 3:30 and posterior on the sidewall of the aorta or if the left renal is more distal compared to the right renal, then it's a long way away from the right renal artery and 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 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 camp zone. You don't need much, just 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 III repair, because with a type III 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 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 IV, 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 IV 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 data 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 III 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 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 Aortopathies

Authors: *Anna Ohlsson and Sherene Shalhub*

Contributor: *Wen Kawaji*

8.1 General Considerations

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.(Black 2019)

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.

8.2 Demographics

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 excavatum), and club feet. They can also have a history of spontaneous pneumothorax 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 pneumothorax, 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 Ehlers-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 Ehlers-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.

8.3 Evaluation

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 criteria is used to clinically diagnose Marfan 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 iridodonesis (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.

8.4 Management

8.4.1 Medical Management

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 Marfans 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 Marfans 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.(Ong et al. 2010) 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.

8.4.2 Surgery

What about surgical treatment for those who need it?

For patients with Marfan’s, prophylactic surgery is recommended for aortic root dilation >5cm or thoracic aorta >5.5cm. 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, Brooke, Arnaoutakis, et al. 2012) 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 pledged 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. Some have described a technique of open exposure and buttressed repair, when endovascular approaches are required.(Black 2019)

In situations of extremis, like a rupture, these patients' tissues have been known to completely breakdown. This is often their first presentation of their aortopathy so there is no time for previously discussed work up or considerations. 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.

Some specific considerations for TEVAR in these patients is that it may be important to have cardiac surgery readily available because they are high risk for retrograde dissection. Also, avoid aggressive oversizing, consider not ballooning if no leak is identified and avoid excessive wire manipulation.

8.5 Summary

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 feature 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.

8.6 Additional Resources

VEDS Research Collaborative study:

- <https://www.vedscollaborative.org/get-involved>
- <https://depts.washington.edu/vedscoll/>

9 Trauma - Endo

Authors: *Kevin Kniery, Marlin "Wayne" Causey, Nakia Sarad, and Todd Rasmussen*

These trauma episodes were developed in collaboration with [Behind the Knife: The Premier Surgery Podcast](#). In addition, Dr. Causey is a co-host for [WarDocs: Military Medicine Podcast](#).

Vascular trauma requires close collaboration between vascular surgeons and trauma surgeons. The decision of which specialty should manage which injuries vary across different centers. Through these trauma chapters we will discuss what management decisions the majority of vascular surgeons should be comfortable managing as a part of a multidisciplinary trauma team.

💡 Take a Listen

Check our [debate between leading trauma and vascular surgeons](#) about how to best develop a collaborative team to manage vascular trauma.

For relevant images and a more in-depth discussion of this topic, please review **Chapter 11: Resuscitative Endovascular Balloon Occlusion of the Aorta**(Yi, Fox, and Moore 2022)and **Chapter 17: Blunt Thoracic Aortic Injury**(Demetriades, Talving, and Inaba 2022) in Dr. Rasmussen's 4th Edition of *Rich's Vascular Trauma*.

Although open surgical treatment is the mainstay of trauma management, there are some unique injuries where endovascular management has become the standard of care in centers with the resources and expertise to perform it.(Katsanos et al. 2009)

9.1 Blunt Thoracic Aortic Injury (BTAI)

9.1.1 Demographics

9.1.1.1 Guidelines

[SVS Guidelines for Blunt Thoracic Aortic Injury](#)(W. A. Lee et al. 2011)

9.1.1.2 Epidemiology

- Rare, but lethal. Less than 1% of all blunt traumas but second leading cause of death in blunt trauma.
- Due to high velocity mechanism of injury, usually are polytrauma patients with other severe injuries (i.e. intracranial injury)
- Common mechanisms
 - High-speed motor vehicle collision
 - Motorcycle collisions
 - Pedestrian vs. motor vehicle
 - Falls from heights
- Most patients have unsurvivable devastating injuries (75%) and pass prior to arrival to hospital for definitive care
- Rates of paraplegia, rates of blood utilization, and mortality rates have decreased with prominence of endovascular repair of injuries

Further Reading

Seminal Papers in Blunt Thoracic Aortic Injury (BTAI) outlining the evolution of management from open to endovascular treatment.

- Fabian et al. (1997). Prospective study of blunt aortic injury: Multicenter Trial of the American Association for the Surgery of Trauma. *The Journal of Trauma*, 42(3), 374–380(T. C. Fabian et al. 1997)
- Demetriades et al. (2008). Diagnosis and treatment of blunt thoracic aortic injuries: Changing perspectives. *The Journal of Trauma*, 64(6), 1415–1418(Demetriades et al. 2008)
- Alarhayem et al. (2021). Timing of repair of blunt thoracic aortic injuries in the thoracic endovascular aortic repair era. *Journal of Vascular Surgery*, 73(3), 896–902(Alarhayem et al. 2021)

9.1.1.3 Anatomy

- Aortic arch » relatively mobile
 - Not common location for blunt injury
- Descending Thoracic aorta
 - Tethered
 - Junction just distal to left subclavian artery is where most injuries occur (within 0.5-2cm)
 - Tear injury due to deceleration

9.1.1.4 Grading Systems

SVS BTAI Grading System(W. A. Lee et al. 2011)

- Grade I: Intimal Tear
- Grade II: Intramural Hematoma
- Grade III: Pseudoaneurysm
- Grade IV: Rupture

Harborview Grading System(Heneghan et al. 2016; Starnes et al. 2012; Quiroga et al. 2019) - Classification scheme helps dictate which patients can be managed operatively vs. non-operatively

- Minimal Injury
 - Absence of aortic external contour abnormality
 - Intimal tear and/or thrombus of <10mm in length or width
 - Treatment: No intervention and surveillance with follow-up imaging
 - Antiplatelet therapy
 - CTA at least within 30 days of injury, progression is rare (5-7%) and often identified early(Osgood et al. 2014)
- Moderate Injury
 - External contour abnormality, includes pseudoaneurysms
 - Intimal tear >10mm in length or width

- Treatment: Delayed repair, semi-elective
 - Stabilization of concomitant injuries(Azizzadeh et al. 2009)
 - Impulse control
 - Short-acting beta blocker (i.e. esmolol)
 - SBP < 120 (coordinate with neurosurgery for TBI patients)
 - Repair within 1 week (TEVAR)
 - For those patients with severe TBI who are unable to undergo anti-impulse therapy due to maintenance of cerebral perfusion pressure, will recommend repair sooner
 - Repeat CTA chest within 5-7 days for follow-up
- Severe Injury
 - Active extravasation (free contrast extravasation or hemothorax at thoracotomy)
 - Hemodynamic instability
 - Left subclavian artery (LSA) hematoma > 15 mm
 - Treatment: Emergent repair
 - BTAI takes first priority

 Take a Listen

Check the [BTK episode](#) with Dr. Benjamin Starnes discussing blunt thoracic aortic trauma and this classification scheme.

9.1.2 Evaluation

- Be aware that patients likely have other injuries
- Recommend CTA chest, abdomen, pelvis
- Perform CTA follow-through to upper/lower extremities if those areas are of concern for injury

9.1.3 Management

Overview

- Endovascular repair of BTAI has better outcomes than Open repair
- Need to consider that most TEVAR devices are designed for aneurysmal disease
 - Generally require a larger diameter and their compliance is not ideal for these smaller/normal caliber aortic arch sizes
 - Average size of aorta proximal to injury site is 19mm (smallest devices are at 22mm)
 - Previously, iliac devices used to treat aortic injuries
- There are new advanced TEVAR devices that are available for non-aneurysmal disease with normal caliber aortic sizes.
 - ex: GORE TAG Conformable Thoracic Stent Graft with ACTIVE CONTROL System
- Recommend heparinization if not contraindicated
 - i.e. TBI, femoral shaft fracture with hematoma, solid organ injury, etc.
 - TEVAR can still be performed safely without heparin
 - Higher threshold for heparinization in COVID+ patients due to their hypercoagulable state
 - Goal Activated Clotting Time (ACT) > 250 - Ensure that ACT machine is available prior to start of case

Methods of Endograft Size Measurement

- CTA imaging
 - Used to help measure aortic size
 - Need to consider that patient is usually hypovolemic, constricted, and young (so aorta more compliant/elastic) — true aortic size may be under-represented, 40% discrepancy between pre- and post-resuscitation, on imaging when choosing the appropriate device for repair
 - Recommend over-sizing stent graft by 20-25% from original measurement
- Intravascular Ultrasound (IVUS)
 - During angiogram, can confirm or redirect sizing in real-time during systole(Azizzadeh et al. 2011; Shi et al. 2015)

Access

- If iliac size is >7-8 mm, can use transfemoral access
- Small iliacs (< 6mm) may need conduits
 - Recommend retroperitoneal approach for exposing common femoral and/or external iliac and using a Dacron conduit(F. J. Criado 2007)
- If iliacs are diseased (i.e. calcium burden), use of conduits would be difficult
 - Can consider putting mineral oil over sheath to help pass and deliver the endograft
- Femoral cutdowns considered to ensure safe delivery and removal of device

Positioning of Device

- Most important aspect is to cover the proximal defect and seal the entry point
- Average distance between the injury and the LSA is approximately 5.8mm (located at aortic isthmus)
- Recommend 2cm coverage proximally of healthy vessel and 2cm coverage distally
 - With these recommendations, approximately 40% of TEVAR repairs cover the LSA
 - Preemptive revascularization of LSA is not necessarily required, most patients do fine without arm claudication
 - Complications to consider when covering LSA
 - Stroke
 - If there is concern for vertebral perfusion, LSA must be revascularized prior to covering
 - Left vertebral artery may be dominant or significant disease in right vertebral artery
 - Arm claudication and ischemia
 - Spinal cord ischemia
 - Rare
 - Collateral pathways between LSA and IMA (internal mammary artery) can be compromised
 - Spinal cord ischemia can present at end of case or delayed 24-72 hours post-operatively

- Spinal cord drainage not routinely done to prevent paraplegia unless there is concern if abnormal neuro-exam.
- If abnormal neuro exam at the end of the case, elevate blood pressure to improve perfusion and consider placing a drain at that time
- There is high risk of placing and monitoring drains if not needed
- Relative contraindications to covering LSA without revascularization
 - Dominant Left Vertebral Artery perfusion to brain
 - Right Vertebral Artery atherosclerotic disease
 - Hemodialysis access on ipsilateral arm
 - Prior CABG with LIMA (left internal mammary) to LAD (left anterior descending artery)
- Some devices (i.e. GORE conformable as above) allow control of first stent ring to better conform/lay down the stent within the arch

Dealing with Malpositioned Devices

- Can deploy too proximal or too distal
- “Bird Beaking” - Where the lesser curve of the graft is not well approximated to the lesser curve of the aorta (i.e. the lifting of the inferior aspect of the most proximal endograft with the first covered stent ring now lifted off into the inner curve of the aortic arch)(Frohlich et al. 2020) This can cause endoleaks. Methods to avoid “bird beaking”
 - Use stiff wire to get into the aortic root (i.e. Amplatz or Lunderquist wires) with forward pressure to have a constant tension
 - Wire should be snug against the greater curvature of the aorta
 - Can consider taking the graft over the origin of the LSA
 - Allows more room in the flatter inner arch to lay the graft down

Timing of Repair

- SVS Grade 3 or Harborview Classification of Moderate Injury
 - Delayed repair 24-48 hours after injury is acceptable and there is a survivable benefit(Alarhayem et al. 2021)
 - Some delays can be up to 5-7 days with medical management
 - Allows time for operative planning and optimization with anti-impulse therapy/resuscitation

- Also enables evaluation of any underlying infectious process that may compromise and infect the endograft
- SVS Grade 4 or Evolving SVS Grade 3/Harborview Moderate injuries
 - More urgent repair (within 24 hours)

9.2 Blunt Abdominal Aortic Injuries

9.2.1 Demographics

9.2.1.1 Epidemiology

- Very Rare
- Commonly associated with polytrauma patients, especially with concomitant blunt thoracic aortic injuries
- Similar mechanisms of injury as BTAI

9.2.1.2 Anatomy

- Zone 1: Diaphragm to SMA (superior mesenteric artery)
- Zone 2: SMA to renal arteries
- Zone 3: Inferior renal artery to aortic bifurcation

9.2.2 Evaluation

- CTA
- IVUS - Also used for operative planning and evaluating extent of defects into branches of the mesentery

9.2.3 Management

Overview

- Zone 1 and 3 can be repaired endovascularly (above the SMA and below the renal arteries)
- Can cover the celiac artery safely if there is no obvious compromise of the other mesenteric arteries (i.e. inferior mesenteric artery, internal iliac arteries, prior gastrectomy or other complex intra-abdominal operation)(Banno et al. 2020)
- Zone 2 injuries typically repaired with open approach
- Endovascular approach can minimize risk of contamination if there is bowel spillage etc.
- Want to use minimum amount of endograft that will appropriately cover injury 2cm coverage proximally and 2 cm coverage distally
- Injury can sometimes result in occlusion, which can also be treated endovascularly.

Types of Endografts Used - Can use combination of all grafts types to get the appropriate coverage

- TEVARs
- Covered stents
- Aortic cuffs

9.3 Blunt Axillo-Subclavian Injuries

9.3.1 Demographics

Epidemiology

- Very rare, 9% of all vascular trauma
- Low incidence due to protection of vessels from surrounding bony structures and high degree of exsanguination with devastating injuries
- Endovascular repair preferred due to location of these injuries and highly morbid open repair due to central hemorrhage and risk of brachial plexus injury.(Branco et al. 2016; DuBose et al. 2012)
- Difficult to access via open methods with proximal/distal control due to protective anatomic nature of overlying skeleton

Anatomy

- Thoracic Outlet
 - Axillo-subclavian vein located anterior to anterior scalene and runs between the first rib and clavicle through the subclavius muscle/tendon
 - Axillo-subclavian artery is located in the arterial triangle
 - More lateral than the axillo-subclavian vein
 - Courses posteriorly to anterior scalene

9.3.2 Management

Access

- Transbrachial or transradial access preferred
- Can combine transbrachial and transfemoral
 - Provides more options for delivering stent grafts with difficult aortic arches (anterograde and retrograde approach) via a through wire
- With transbrachial access, brachial cutdown can be more reliable in trauma setting
- Can also consider HYBRID approach
 - Gain proximal and distal control via through wires from transbrachial/transradial and then perform the open repair
 - Can perform coil embolization of subclavian arteries to gain control
 - Caution to vertebral perfusion
 - Stent graft to temporize injury and maintain in-line flow, then have interval axillary artery repair with a bypass (within a week to a month later)

Types of Endografts used

- Self-expanding stents (i.e. Viabahn)
 - Traditional approach
 - May provide greater flexibility - important for mid-subclavian penetrating trauma due to mobility of this region. Stents in the axilla have particularly poor patency for this reason.(Chopra et al. 2016; Gray et al. 2017; Shalhub et al. 2011)
 - Helpful for more distal injuries
- Balloon-expandable stents

- Newer devices, also flexible
- Has more precise landing (especially if injury is near branch points)

Part IV

Abdomen

10 AAA

Authors: *Mia Miller and Julie Duke*

10.1 Demographics

What is an abdominal aortic aneurysm (AAA)? (Moore, Lawrence, and Oderich 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, Dalman, Eskandari, Jackson, Lee, Mansour, Mastracci, Mell, Murad, Nguyen, and al. 2018)
 - 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. 2000a)
- Conversely, if a patient is first found to have a AAA, there is an 11% chance of having associated popliteal artery aneurysms >15mm. (Tuveson, Löfdahl, and Hultgren 2016)
- Another study showed a rate of femoral-popliteal aneurysms in AAA patients is approximately 14%. (Diwan et al. 2000a)
- This association stresses the importance of a good physical exam when evaluating a patient with a AAA and is commonly tested on exams.(Chaikof, Dalman, Eskandari, Jackson, Lee, Mansour, Mastracci, Mell, Murad, Nguyen, and al. 2018; Diwan et al. 2000b)

10.1.1 Etiology

What is the pathogenesis of an abdominal aortic aneurysm? (Moore, Lawrence, and Oderich 2019)

- More than 90% of AAAs are associated with atherosclerosis.
- Other causes include cystic medial necrosis, dissection, Marfan's syndrome, Ehlers-Danlos syndrome, Loeys-Dietz, 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 upregulation 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.
- Nicotine increases MMP expression in vascular smooth muscle cells.(Z.-Z. Li and Dai 2012; Rabkin 2016)
- 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 anti-coagulation.(Singh et al. 2019; Cameron, Russell, and Owens 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; T. E. Rasmussen and Hallett 1997)
- For more about aortitis, see Section [3.3.5](#)

What are the risk factors for AAA occurrence and growth? (Moore, Lawrence, and Oderich 2019)

- Risk factors for AAAs are similar to the risk factors for occlusive atherosclerosis and include age, tobacco use, hypertension, male gender and hyperlipidemia.
- 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.
 - Smoking has a dose dependent relationship with AAA, smoking 1/2 ppd has one third the risk of smoking 1 ppd.(Kent et al. 2010)
- It has been found that diabetes is protective for AAA progression and rupture.
- Men more likely to have AAA, but women develop at a later age with more aggressive growth and risk for rupture. (Lo and Schermerhorn 2016a)
 - 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(C.-C. Lee et al. 2015, 2018; LeMaire et al. 2018; Pasternak, Inghammar, and Svanström 2018) - In a recent study just published in JAMA Surgery in January 2021, 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
- 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.

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, Roy, and Upchurch, n.d.)
- 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 10.1: Historical reporting of AAA rupture risk(Tracci, Roy, and Upchurch, n.d.)

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 10.2: 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).

10.2 Evaluation

What is the work up for a AAA? (Moore, Lawrence, and Oderich 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.

10.2.1 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, Dalman, Eskandari, Jackson, Lee, Mansour, Mastracci, Mell, Murad, Nguyen, Oderich, et al. 2018)
 - For more technical details on duplex screening see Section [20.6.1](#).

Table 10.3: Recommended Intervals for AAA Surveillance(Thompson et al. 2013; Chaikof, Dalman, Eskandari, Jackson, Lee, Mansour, Mastracci, Mell, Murad, Nguyen, Oderich, et al. 2018)

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.

10.3 Management

What are the indications for repair? (Moore, Lawrence, and Oderich 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, Dalman, Eskandari, Jackson, Lee, Mansour, Mastracci, Mell, Murad, Nguyen, Oderich, et al. 2018)
- For saccular aneurysms, the SVS practice guidelines recommend elective repair (*Level 2, Grade C evidence*). (Chaikof, Dalman, Eskandari, Jackson, Lee, Mansour, Mastracci, Mell, Murad, Nguyen, Oderich, et al. 2018)
 - 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, Lawrence, and Oderich 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, Dalman, Eskandari, Jackson, Lee, Mansour, Mastracci, Mell, Murad, Nguyen, and al. 2018; 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 PaO₂ < 50 mmHg, elevated PCO₂, or both.
- To help delineate a patient's risk, a preoperative workup is necessary. The SVS practice guidelines recommend the following: (Chaikof, Dalman, Eskandari, Jackson, Lee, Mansour, Mastracci, Mell, Murad, Nguyen, Oderich, et al. 2018)
 - 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 ≥ 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%). (R. Greenhalgh 2004)
- The DREAM trial, a multicenter randomized trial from 2000-2003, compared EVAR vs Open repair 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, Cuypers, and Buskens 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)

Take a Listen

Check out [our episode with Dr. Julie Freischlag](#) where we discuss her involvement with the OVER trial.

- 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 infrarenal 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.(Gustavo S. Oderich et al. 2014)

- 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. (Gustavo S. Oderich, Correa, and Mendes 2014)
- Parallel grafts for short necks (i.e. snorkel or chimney) require 20mm seal for good outcomes. (Donas et al. 2015)

Take a Look

Check out [our “How I Do It” episode](#) where Dr. Wes Jones and Murray Shames review their technique for performing a Zfen endovascular aortic aneurysm repair. This technique is beyond the scope of this review.

- 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, Pegna, and Boyne 2013; R. Schmidt et al. 1994) High risk of persistent endoleak due to ongoing flow into IVC. Open repair often best.(Orion, Beaulieu, and Black 2016)

10.3.1 EVAR

Can you briefly go over the steps of an EVAR? (Moore, Lawrence, and Oderich 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.

1. Performed in the operating room or IR suite with a fixed or portable C-arm
2. Anesthesia
 - Regional block, local anesthesia or general anesthesia depending on surgeon preference and patient risk
3. Groin access and short sheath placement
 - Percutaneous - Closure devices are introduced prior to inserting the large sheaths containing the stent-grafts
 - Cut-down
4. Pigtail catheter is used to perform an aortogram of the abdominal aorta and iliac arteries
5. The renal artery orifices are marked. If there is any concern about good visualization, IVUS (intravascular ultrasound) can be used to assist.
6. Systemic heparin is given
7. 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
8. 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.
9. The main body is deployed to the point where the gate is opened
10. Contralateral limb gate cannulation is performed using a wire and directional catheter.
11. 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. There are multiple techniques to confirm that the wire is in the endograft, but this MUST be confirmed before proceeding to deploy the contralateral limb.

12. The contralateral limb is introduced and deployed taking care to preserve flow to the internal iliac artery.
13. The remainder of the main body is deployed and iliac extensions deployed if required.
14. The stent graft is ballooned at the neck, within the gate, at the bifurcation, and distal iliac seal zones.
15. An aortogram, usually multiple in different views, is performed to exclude any endoleaks.
16. The sheaths are removed, and the groin sites are closed using Perclose devices if performed percutaneously, or primary repair if open cutdown performed.
17. Check pedal signals 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.

 Take a Listen

Check out [our episode where we review the basics of the EVAR procedure in more detail.](#)

You mentioned endoleaks, can you discuss the complications specific to EVAR and the management? (Moore, Lawrence, and Oderich 2019)

- 15 year follow up from EVAR-1 shows inferior late survival and higher re-intervention in EVAR patients when compared with open repair. (R. 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.(R. M. 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)

10.3.1.1 Endoleak

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

- 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.
- 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(T. J. 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 2016b; Abularrage et al. 2010; Timaran et al. 2004; Shalaby et al. 2016; Sarac et al. 2012; J. E. 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)

Take a Listen

Check out [our conversation with Drs Gregorio Sicard and Frank Caputo](#) where we discuss management of endograft failures and some techniques for open conversion that are a little beyond the details of this review.

- Type III
 - Separation of graft components. Treated by relining the EVAR graft.
 - Usually identified in follow-up surveillance and necessitates intervention.
- 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.
- 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.

10.3.2 Open Repair

Now we can move onto open repair. Describe an open infrarenal aneurysm repair. (Moore, Lawrence, and Oderich 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 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. (K. B. 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.

💡 Take a Look

Check out [our “How I Do It” episode](#) where Dr. Ashlee Vinyard and Dr. John Eidt review their technique for performing a open AAA repair.

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 patients 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. Patients can also develop denervation of the lateral abdominal wall and develop a bulge there.

Take a Listen

Check out [our Origin Stories episode reviewing the history of aortic aneurysm repair.](#)

10.3.2.1 Complications

What are some of the complications with open aortic aneurysm repair? (Moore, Lawrence, and Oderich 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.
 - Acute conditions associated with poor post-operative cardiac outcomes include recent MI (30d), unstable angina, decompensated heart failure, high-grade arrhythmias, severe valvular disease.(Devereaux and Sessler 2015; Fleisher et al. 2014; Kristensen et al. 2014; Tashiro et al. 2014)
- 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; T. E. 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).
 - A suprarenal clamp time of less than 30 minutes is ideal to reduce the risk of renal failure.

- 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; Gustavo S. Oderich et al. 2011; Smeds et al. 2016)

 Take a Listen

Check out [our conversation with Miss Rachel Bell](#) where we discuss management of infected aortic grafts and mycotic aneurysms that is a little beyond the scope of this review.

10.3.2.2 Postoperative Surveillance

What is the post-operative surveillance required for open and endovascular approach, and how do they differ? (Moore, Lawrence, and Oderich 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.
 - Perianastomotic degeneration occurs in 0.5-10% of cases. Can be diagnosed with CT scan or ultrasound.(Curl et al. 1992)
- 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.(A. 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.

10.3.3 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, Lawrence, and Oderich 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%. (M. T. 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. (L. J. 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.

Take a Listen

Check out [our conversation with Professor Janet Powell](#) where we discuss her landmark IMPROVE trial comparing open and endovascular management of ruptured aortic aneurysms.

- 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). (Goudeketting et al. 2019)

10.4 Iliac Artery Aneurysms

10.4.1 Demographics

CIAA aneurysm defined as >1.5cm.

10.4.2 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, Inderbitzi, and Bär 1991)

10.4.3 Management

Treatment threshold for CIAA is often accepted at 3.5cm.(Dix, Titi, and Al-Khaffaf 2005; Matti T. 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)

Take a Look

Check out [our “How I Do It” episode](#) where Dr. Lily Johnston and Dr. Gustavo Oderich review their technique for performing an iliac branch device repair with is a little beyond the scope of this review.

- 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.(Angiletta et al. 2011; Karch et al. 2000)

11 Mesenteric Disease

Authors: *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. (A. Chandra and Quinones-Baldrich 2010; van Gulik and Schoots 2005)

11.1 Acute Mesenteric Ischemia

11.1.1 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 pneumatosis.

CT angio:

Probably represents the most common diagnostic modality to diagnose acute mesenteric ischemia. CTA has the advantages of speed, availability, and noninvasiveness 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?!

11.1.2 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 systematically 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 benefited 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.(Scali et al. 2019)

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 mid-line 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 mid-line is easily lengthened into a traditional

vertical laparotomy incision.(Milner, Woo, and Carpenter 2004; Blauw et al. 2014; Gustavo S. Oderich et al. 2018)

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 very high rates. Probably not your first answer for oral boards.(Lim et al. 2019)

11.1.3 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, pneumatosis 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)

11.2 Chronic Mesenteric Ischemia

11.2.1 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: 50 in the celiac or 50 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)

11.2.2 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. (Gustavo S. Oderich and Ribeiro 2019a; Foley et al. 2000)

Any other technical tips for endo intervention on the mesenteric arteries?

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.(Gustavo S. Oderich et al. 2013; Gustavo S. 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)

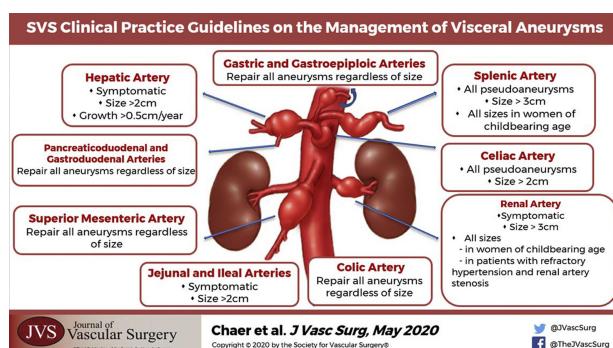
How about open revascularization?

In most centers, up to 80% of CMI is treated endovascularly. Open is often reserved for those that fail endovascular therapy.(Gustavo S. Oderich, Gloviczki, and Bower 2010; Gustavo S. 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.

11.3 Visceral Aneurysms

Aneurysms can occur in a diverse range of vascular beds in the visceral vessels. Size criteria for repair is dependent on the location and the etiology, with some aneurysms mandating repair at any size (gastric, gastroepiploic, gastroduodenal, pancreaticoduodenal, SMA, colic), and others considered appropriate to observe in asymptomatic patients until certain size thresholds are met. The SVS clinical practice guidelines are the optimal resource to review these criteria.(Chaer et al. 2020)



11.3.1 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.(W.-K. Lee et al. 2008 Nov-Dec)

11.3.2 Splenic Aneurysm

Repair all pseudoaneurysms, or if size > 3cm, or at any size if of childbearing age. Consider splenectomy vaccines pre-treatment (covers encapsulated organisms Shigella, Haemophilus influenzae, and Neisseria).

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, Rockman, and Kashyap, n.d.; Piffaretti et al. 2007; C. 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)

11.4 Median Arcuate Ligament Syndrome (MALS)

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

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 of 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.

11.5 SMA Syndrome

What is SMA syndrome?

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, loss of 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, Büchler, and Kienle 2007; Merrett et al. 2009)

11.6 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)

12 Renal

Authors: *Cullen McCarthy and Matthew Edwards*

12.1 Renovascular Ischemic Disease

12.1.1 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 2019a)

Who is at risk for ischemic nephropathy?

Renal injury can develop in anyone with a kidney or kidney region beyond a critically stenotic artery. Any of the following flow limiting lesions can cause ischemic neuropathy OR renovascular hypertension.

- Atherosclerotic disease of the aorta or renal arteries (most common) - 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.
- Fibromuscular dyplasia - 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.
- Coarctation of the aorta or mid-aortic syndrome

- Large vessel arteritis (rare) - 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 Abularbage 2019b; F. A. Weaver et al. 2004; F. P. Zhu et al. 2012)
- Iatrogenic causes - Rarely iatrogenic from malposition or migration of endovascular aortic stent grafts over the renal orifices.

The prevalence of renovascular hypertension in patients with mild hypertension is probably less than 1%. ***However in patients with acute progression, severe or refractory hypertension the prevalence may be as high as 10 to 40%.*** However, disease may not entirely be attributable to a flow limiting lesion. There may be a component of cholesterol embolization or small vessel atherosclerotic disease that may contribute to progression of renovascular disease.

The effect of the stenosis may not be clinically apparent due to compensatory function of the unaffected contralateral kidney. We know 5-22 % of patients 50 years or older who have advanced CKD have some degree of renal artery stenosis and 23-54% of those patients with stenosis have bilateral renal artery disease.

- 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 2019a)
- 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.
- Bilateral renal arterial stenosis is associated with more widespread atherosclerotic disease, higher serum creatinine levels, and higher mortality than unilateral disease

However, flow limiting lesions may be an “incidental” finding in patients who have CKD or hypertension that is caused by a separate disorder (i.e. 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.

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.

The clinical findings that suggest that ischemic renovascular disease is an underlying cause of hypertension, include:

- Recent or rapid development of severe hypertension.
 - Relatively specific for renovascular hypertension and is the strongest predictor of anti-hypertensive 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
- Recent reduction in kidney function (over last 6-12 months) with or without 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. 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 2019a; Schoolwerth et al. 2001)
 - 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)

12.1.2 Evaluation

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?

- Doppler ultrasound criteria (Hoffmann et al. 1991; Zierler and Dawson 2016)
 - PSV >200 cm/sec or renal aortic ratio >3.5 and post stenotic turbulence, suggests >60% stenosis, but some labs have proposed higher velocities (250-285 cm/s). (Schäberle et al. 2016; Rickey and Geary 2019b)
 - Elevated velocities can be seen with tortuosity, but this should be able to be confirmed with B-mode. For more see Section 20.6.4
- Generally, luminal occlusion of at least 60 to 75 % is required to limit blood flow and reduce perfusion pressure. CTA or angiogram with CO₂ are helpful to delineate the anatomy. 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 (Galan, Cowper, and Bucala 2006) and degrades with motion and respiration (Nelson et al. 1999)
- This degree of stenosis is usually associated with a measurable translesional “pull-back” pressure gradient of 10 to 15 mmHg, which can be measured during conventional angiography.

Can we tell who has cortical hypoxia through diagnostic tests?

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. Blood Oxygen Level Dependent Magnetic Resonance (BOLD-MR) can identify levels of cortical hypoxia which may be associated PSV > 385 cm/sec or reduced GFR (20 to 25 ml/min).

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?

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. 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.*

Here are some additional criteria that can be useful adjuncts to duplex velocities and cross sectional imaging.

- **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. Initially, many were hopeful that this would be a very useful indicator, but not many have been able to reproduce the initial studies describing this association.(Radermacher et al. 2001)
- **Kidney size** - Very small kidneys (less than 8 cm in longest diameter) are usually considered unlikely to recover after revascularization.
- **Kidney biopsy** (not usually performed) - Previous studies suggest that biopsy demonstrating preexisting atheroembolic changes and interstitial fibrosis indicate a limited potential for recovery.
- **Comparison of kidney morphology with kidney function** - Some investigators have recommended assessing morphological parameters, such as renal parenchymal volume and cortical thickness with MRI, and comparing these parameters with kidney function measured by radionuclide scanning. A kidney with normal morphology in the

setting of renal artery stenosis and reduced function may indicate a “hibernating kidney” which may respond to 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.

12.1.3 Management

12.1.3.1 Medical 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... can patients with renovascular hypertension just be treated with anti-hypertensives, such as ACE inhibitors?

Many of these patients can be treated with medical therapy alone without loss of function or irreversible fibrosis, sometimes for many years.

Some in-human studies demonstrate that even with moderate reduction in renal perfusion pressure (40%) and in renal blood flow (30%), GFR may be reduced but renal cortex and medulla tissue oxygenation may be able to adapt and avoid developing severe hypoxia. However, there are limits to this adaptation.

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 limits our ability to control the hypertension medically without causing further damage to the kidney.

Further reduction in renal function 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.

In addition, all of these patients should receive secondary prevention of cardiovascular morbidity with aspirin, statins, smoking cessation, and, in patients with diabetes, glycemic control. We're addressing or prevention progression of stenosis in those with atherosclerotic disease.

For more on the medical management of vascular disease, see Section [15.2.2.1](#)

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. ***Again, the factors most closely associated with recoverability include:***

- A recent onset of hypertension prior to the diagnosis of renal artery stenosis (strongest predictor of response to treatment)
- Failure of optimal medical therapy to control the blood pressure or intolerance to optimal medical therapy (i.e. deterioration of renal function during antihypertensive drug therapy, such as ACE)
- 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, Misra, and Oderich 2013)

12.1.3.2 Endovascular

Before undergoing diagnostic angiography or potential intervention, the patient's ACE inhibitor and metformin should be stopped. Continuing or even initiating anti-platelet therapy and statins are important to prevent atheroembolization during wire manipulation of the renal arteries and stent placement.(Hiramoto et al. 2005)

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

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.

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.(Cooper et al. 2014) Tell me about that.

- 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 m² 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 (A. Investigators 2009)

Well, that sounds pretty convincing, however there are 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 (i.e. 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 (A. 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

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 are 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.

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.

- 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
- 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 PTRA 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 and intervention (Edwards and Cooper 2019)

1. Supine with arms overhead or straight out to the sides
2. LAO 15-20deg
3. Femoral access
4. Flush catheter placed just above the renals. Often start with CO2 and selective contrast runs.
5. Breath hold, high frame rate and non-DSA due unavoidable patient movement
6. Often utilize an 0.014 to cross the lesion, and advance a guide catheter to secure access.

7. Many advocate for primary stenting in atherosclerotic disease with uncovered stents to minimize manipulation. Some believe that covered stents carry benefit, but they are more expensive, and the evidence does not directly support this added cost.

- 2005 ACC/AHA guidelines on peripheral artery disease recommends that a stent be placed in patients undergoing percutaneous transrenal angioplasty (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 (i.e. atheroemboli)

12.1.3.2.1 Outcomes

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
- In-stent restenosis rates are closer to ~11-17%, with 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

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

12.1.3.2.2 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

12.1.3.2.3 Surveillance

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 at 2-4 weeks 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
- Re-treatment 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.

12.1.3.3 Open Surgery

What about an open operation?

Surgical revascularization used in addition to medical therapy is less common since the widespread application of effective antihypertensive drug therapy and endovascular stents in the mid 1990s.

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 concomitant 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 nonfunctional, 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 is reserved primarily for patients with:

- Multiple small renal arteries
- Early primary branching of the main renal artery
- Require aortic reconstruction near the renal arteries for other indications (i.e. aneurysm repair or severe aortoiliac occlusive disease)
- In order to avoid manipulation of a highly diseased aorta or failed endovascular stents, using extra-anatomic bypass (i.e. splenorenal, ileorenal, or hepatorenal bypass).

12.1.3.3.1 Complications

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).

12.2 Fibromuscular Dysplasia (FMD)

12.2.1 Demographics

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

Fibromuscular dysplasia (FMD) is a non-atherosclerotic, non-inflammatory disorder that results in progressive arterial stenosis, occlusion, aneurysm, dissection, or tortuosity.(Jeffrey W. Olin and Seale 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. If FMD is, in fact, a single disease?

Where does it occur?

FMD has been observed in nearly every arterial bed. Two thirds of patients have multiple arteries involved.

- Involvement of the renal arteries ~75-80%
- Involvement of the extracranial cerebrovascular arteries (i.e. carotid and vertebral arteries) ~75%

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)

12.2.1.1 Etiology

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. (Jeffrey W. 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.

12.2.1.2 Presentation

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 (i.e. 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.

12.2.2 Evaluation

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.
- Multi-row detector CT scanners may provide additional advantages in diagnosis and surveillance because they offer more rapid image acquisition, variable section thickness, three-dimensional rendering, diminished helical artifacts, and smaller contrast requirements.

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 help identify dissection or intramural hematoma or determine response to angioplasty.
 - Negative DSA excludes a diagnosis of FMD in the vascular bed that was imaged.

Any place for pathological 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.

12.2.3 Management

12.2.3.1 Medical Management

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

12.2.3.2 Endovascular

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 and do we have good results treating FMD with endovascular or open interventions?

- Patients most often treated with angioplasty alone with good success. (Davies et al. 2008; T. L. 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?

- Angioplasty is often performed through a transfemoral approach, but concomitant stenting is rare, 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?

Two situations that may require stenting include dissection or perforation (rare) after 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 because there is an 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 (IVUS) 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

12.2.3.3 Surgery

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 does 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.

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).

12.3 Renal Artery Aneurysms

12.3.1 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 atherosclerotic 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.

12.3.2 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

12.3.3 Presentation

- Majority are associated with hypertension (70%) (Coleman and Stanley 2015)
- 10% mortality
- 90% risk of kidney loss
- Less than 3% rupture.

12.3.4 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, however anecdotally calcification on imaging does not correlate to appearance of intraoperatively

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.

12.4 Renal Artery Dissection

12.4.1 Demographics

12.4.1.1 Presentation

Renal ischemia from renal artery dissection should be considered if a patient presents with acute onset of hypertension, flank pain, hematuria and proteinuria. (B. T. Müller et al. 2003)

Renal artery dissection can sometimes be found incidentally on imaging performed for other reasons.

12.4.2 Evaluation

CTA is the best modality of imaging because it is rapid and accurate at diagnosis.

12.4.3 Management

If the patient has normal renal function, normal blood pressure or stable hypertension, is asymptomatic or the dissection appears chronic, then there likely is no role for intervention.

However, if emergent therapy is required if there is concern for renal perfusion and viability.(Calligaro and Dougherty 2019) There is a role for endovascular stenting, however endovascular therapy should be avoided in the setting branch involvement.

Bypass, in situ repair, auto-transplant if renal branch involvement and possibility of renal salvage. Nephrectomy may be required in uncontrolled hypertension, extensive dissection and irreversible ischemia.

12.5 Nutcracker Syndrome

12.5.1 Demographics

12.5.1.1 Etiology

Renal vein compression between the SMA and the aorta, often seen when the SMA angle is <40 deg.(Yun et al. 2016)

12.5.1.2 Presentation

Classically presents with left flank pain and hematuria (microscopic and macroscopic), and sometimes pelvic or vulvar varicosities. Symptoms are often non-specific and other abdominal causes of pain should be evaluated for simultaneously.

12.5.2 Evaluation

Often first identified on axial imaging with a narrowed renal vein and a prominent ovarian vein. Definitive diagnosis often requires venogram and absolute pressures.(K. W. Kim et al. 2011; Yun et al. 2016)

12.5.3 Management

Open surgery has been the mainstay of treatment, which usually requires reconstruction of the renal vein to avoid compression. This can be done with a transposition of the renal vein with or without a vein cuff or transposition of the ovarian vein over the IVC.(Erben et al. 2015; Kurklinsky and Rooke 2010; N. R. Reed et al. 2009)

Endovascular therapy with renal vein stenting has shown some promising results but can be complicated by stent migration and fracture. Antiplatelet therapy is recommended and there has been good patency out to one year.(Erben et al. 2015; Ananthan, Onida, and Davies 2017; Quevedo, Arain, and Abi Rafeh 2014; X. Wang et al. 2012)

12.6 Renal Vein Thrombosis

12.6.1 Evaluation

CT scan is best. Difficult to visualize native renal vein on duplex imaging. (Asghar et al. 2007; Velazquez-ramirez and Corriere 2019)

12.6.2 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.

12.7 Renal Arteriovenous Fistula

Relatively common complication of renal biopsy (9-18%). (Schwarz et al. 2008)

12.7.1 Presentation

Bruit over kidney, renal impairment, varicocele, hematuria and abdominal pain. (Hunter, Berman, and Walser 2019)

12.7.2 Evaluation

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)

12.7.3 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, Saad, and Turba 2009; Saliou et al. 1998)

13 Trauma - Arterial

Authors: *Kevin Kniery, Adham Elmously, Nakia Sarad, and Todd Rasmussen*

These trauma episodes were developed in collaboration with [Behind the Knife: The Premier Surgery Podcast](#).

Vascular trauma requires close collaboration between vascular surgeons and trauma surgeons. The decision of which specialty should manage which injuries varies across different centers. Through these trauma chapters we will discuss what management decisions the majority of vascular surgeons should be comfortable managing as a part of a multidisciplinary trauma team.

💡 Take a Listen

Check our [debate between leading trauma and vascular surgeons](#) about how to best develop a collaborative team to manage vascular trauma.

For relevant images and a more in depth discussion of this topic, please review **Chapter 18: Abdominal Aortic Trauma, Iliac and Visceral Vessel Injuries**(Aylwin and Jenkins 2022) in Dr. Rasmussen's 4th Edition of *Rich's Vascular Trauma*.

13.1 General Principles

As in all traumatic injuries, ensure that the patient is appropriately resuscitated with supportive care.

It is important to consider the broad principles and approach:

1. Urgency of situation
2. Resuscitation Efforts
3. Communication with Team
4. Optimal Exposure
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.

Angiography can be utilized in conjunction to open operative explorations in areas of injury that are difficult to access/control (i.e. Zone 3 retroperitoneal hematomas)

💡 Take a Look

Here is an [image of the zones of the retroperitoneum](#), hosted by basicmedicalkey.com.

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.

13.2 Basic Exposure Techniques

Procedural Sequence in Operative Abdominal Trauma

- Expose
- Achieve Proximal Control
- Explore and Evaluate Injury
- Restore Flow

Supraceliac aortic and thoracic cross-clamping are important surgical maneuvers to control massive intra-abdominal bleeding in hemodynamically unstable patients.

Tips and Tricks

- In order to get umbilical tape or vessel loop around aorta, use combination of Metzenbaum scissors and controlled pressure.
 - As you try to free the aorta from the connective tissue around it to get back to the spine, can use a metallic pediatric yankauer (small profile and blunt tip) as a dissection tool and push with the Metzenbaum scissors along with direct feel to get the aorta freed.

- Can then use a right angle to get underneath the aorta.
- Ensure that lumbar vessels are identified when trying to get around the aorta
 - “find the outside before you find the inside”
 - Can clip lumbar vessels with medium/large Weck clips
- Use blue operative towels to cover and bring up the viscera to be held with the large retractors (i.e. Omni retractor) 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 viscera either right or left and exposing the retroperitoneum

 Take a Listen

Check out [our conversation with spine surgeons at HSS in New York City](#) on collaborative work between vascular and spine surgeons to improve the safety of spine surgery.

13.2.1 Supraceliac Control

Supraceliac control is recommended prior to any medial visceral rotations if it is a Zone I hematoma.

Exposure

- Basics
 - Exposure starts with midline laparotomy incision from xiphoid to pubis that may be extended to the first 2-3 cm of a sternotomy.
 - This releases the inferior costal margins of the thoracic area under the diaphragm to pull the costal margins laterally and up – which suspends the patient’s rib cage up and away from the aorta and the abdominal contents
 - Recommend use of Omni retractor so that retractors can be placed under costal margin
 - Ensure that there is nasogastric tube placed by anesthesia
 - Allows circumferential control of esophagus with either umbilical tape or penrose drain for appropriate left-sided retraction of the esophagus, away from the crus that is surrounding the aorta
- Steps

- Midline laparotomy incision supraviphioid to pubis with release of costal margin (suspended away from abdominal contents)
- Clear left lateral segment of liver by mobilizing left lateral segment either cephalad or tucked inferiorly to the right side.
- For better exposure, place patient in reverse Trendelenburg to let the stomach down and pull it inferiorly to create a better window
 - Esophagus is retracted to the patient's left and the stomach pulled down (further retraction can be performed by a resident or operative assistant by putting their hand on the stomach and retracting it towards the pelvis)
- Next, divide the crus overlying the aorta, using a large right angle and a bovie extender
 - Maintain adequate control during muscle dissection by feeling the aorta and its pulse (even a weak pulse)
 - Divide the crus over the aorta with electrocautery and with blunt dissection on either side.
 - Note: do not need to spend time to get circumferential control of the aorta like in an elective aneurysm repair.
- Supraceliac aorta should now be exposed enough on either side to have a clamp appropriately placed to gain control

For more on exposure of the visceral aorta, check out Chapter [7](#)

13.2.2 Visceral Rotation

Left medial visceral rotation (Mattox Maneuver)

- Maneuver performed when aortic injury or a close branch off the aorta is suspected
- 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
- Steps
 - Divide the white line of Toldt along the sigmoid colon and the left colon.
 - Establish plane in the retroperitoneum
 - Achieved through steady blunt dissection facilitated with Bovie electrocautery

- The 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 mid-line and then ultimately to the patient's right.
- 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

Take a Look

Check out [this video for an example of a left medial visceral rotation](#) by Dr. Alan Lumsden at Houston Methodist.

Right medial visceral rotation (Cattell-Braasch Maneuver)

- Important when concerned about an IVC injury
- Steps
 - Divide the white line of Toldt starting at the cecum and coming along the right colon.
 - Bring the cecum and right colon cephalad and then to the patient's left.
 - Note: 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 plane is similar to the left medial visceral rotation as it is largely developed with blunt dissection in combination with Bovie electrocautery to open the retroperitoneum.
 - At this point, you will almost immediately see the left iliac vein and then the vena cava.
 - 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.

Take a Look

Check out [this video for an example of a right medial visceral rotation](#) by Dr. Alan Lumsden at Houston Methodist.

13.2.3 Exposure of Infrarenal Aorta

The exposure of the infrarenal aorta is also called the transperitoneal infrramesocolic exposure

Inframesocolic means that the transverse colon is reflected cephalad.

Steps

- 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).
- 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.
 - Note: 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
 - Note: 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

13.3 Management

13.3.1 Celiac Artery

Exposure

- Origin of the celiac artery can be seen with left medial visceral rotation.
 - This maneuver will expose the entire paravisceral segment of the aorta
- Can also use anterior approach through the lesser sac (very similar to exposure needed for the supraceliac aortic exposure)
 - Stomach is retracted caudally towards the pelvis, lesser sac is opened, and then the stomach is pulled down further.

- Pancreas is pulled down as well and the origin of the first centimeter of the celiac artery is now exposed.

Repair

- Small and proximal injuries to celiac - Primary repair may be an option use 4-0 or 5-0 prolene sutures.

Ligation

- Large injuries with destruction of vessel wall - Primary repair may not be an option
- Considered as damage-control situation to control bleeding and ligation considered as an initial step to save a patient's life
- Ligation may be tolerated if there is robust GDA collateral circulation. However, once bleeding is controlled, this should be assessed with doppler.
- If the patient is stable or improving and particularly if there is clear signs of liver, pancreas or duodenal ischemia, then this should be reconstructed.

13.3.2 Common Hepatic Artery (CHA)

Exposure

- 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
- Recommend positioning 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.

Repair

- Primary repair is ideal
- Ligation may be only option in damage control surgery
 - 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.

13.3.3 Gastroduodenal Artery (GDA)

Exposure

The gastroduodenal artery is the first branch off of the common hepatic artery and defines the junction between the common and proper hepatic arteries.

Repair

Primary repair should be sought if possible. If it is a damage control situation, then the GDA must be ligated to control hemorrhage.

Ligation

- Assess patient's condition, resuscitate, utilize the doppler, and assess the area distal to ligation to evaluate the consequences
- If ligation showed appropriate collaterals, then the vessel can remain ligated
- If there is clear ischemia, then there is a difficult decision between repair vs. shunt vs. permanent ligation
- Consider metabolic consequences of ligation as well.
- Decision is situationally dependent - the principles of damage control resuscitation and damage control surgery should always be maintained.

13.3.4 Splenic Artery

One of the key tenets of splenic injury is exposure. Ensure good retraction with incision up to xiphoid and alongside the left costal margin.

Low threshold for splenectomy for control of hemorrhage from the splenic artery or splenic parenchyma.

13.3.5 Superior Mesenteric Artery (SMA)

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.

SMA is divided into 3 segments based on its course

- Segment 1: Aorta to Pancreas
- Segment 2: Pancreas to Root of Mesentery

- Segment 3: Mesentery to Small Bowel

Exposure

- Visualization is dependent on the segment that needs to be exposed
- Segment 1 (most proximal) - left medial visceral rotation.
- Segment 2 (near head of pancreas)
 - Head of pancreas may need to be divided to get appropriate exposure
 - If distal to head of pancreas, can use inframesocolic approach
- Segment 3 (most distal) - anterior transperitoneal approach

Repair

- All efforts should be made to maintain flow through the SMA
- Primary repair - performed on small injuries with 4-0 or 5-0 prolene suturing
- Patchy angioplasty of interposition graft
 - For larger injuries
 - Conduit types: 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.
- Again, repair in a strictly traumatic setting is rare, because injuries that include the proximal mesenteric arteries are unlikely to be survivable. However, these techniques can be applied to other situations when assisting other surgeons who have to resect the SMA as a part of an oncologic resection or have induced an iatrogenic injury.

Take a Listen

Check our episode where we discuss the role of a vascular surgeon in assisting with oncologic resections.

Ligation

- Distal SMA segments may be more tolerant of ligation.

13.3.6 Inferior Mesenteric Artery (IMA)

IMA can be ligated in majority of cases.

Prior to ligation, assess the patency of the iliac vessels — specifically the internal iliac vessels.

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.

For more discussion of evaluation and re-implantation of the IMA, see Section [10.3.2](#)

13.3.7 Renal Arteries

Renal artery is unlikely to be reconstructed in a trauma setting (similar to SMA)

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.

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 thrombosed, salvageability is unlikely due to prolonged warm ischemic time of the kidney.

Repair

- Occurs if source of hemorrhage is from an injured renal artery that still has flow
 - Seen during exploration of expanding hematoma in Zone II
- Most commonly, if renal artery is injured it is ligated for damage-control
- If patient hemodynamically stable, primary repair recommended
 - 4-0, 5-0, or even 6-0 monofilament prolene suture
 - If a primary repair would compromise the lumen, then consider patch angioplasty
- Distal branch artery injuries can be managed with endovascular coil embolization.
 - Again, the decision to take the time to coil embolize is dependent on the patient's current hemodynamics

Incidental CT findings

- Types: intimal flap, fistula, and/or possible pseudoaneurysm of the renal artery

- Management
 - 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.

14 Trauma - Venous

Authors: *Kevin Kniery, Adham Elmously, Nakia Sarad, and Todd Rasmussen*

These trauma episodes were developed in collaboration with [Behind the Knife: The Premier Surgery Podcast](#).

For relevant images and a more in depth discussion of this topic, please review **Chapter 19: Inferior Vena Cava, Portal and Mesenteric Venous System** in Dr. Rasmussen's 4th Edition of *Rich's Vascular Trauma*. (T. Fabian and Savage 2021)

14.1 General Principles

14.1.1 Anatomy

For the discussion today we will divide the IVC into four segments:

- Infrarenal
- Renal/suprarenal
- Retrohepatic
- Suprahepatic

We will then discuss management of portal vein and SMV separately.

14.1.2 Non-operative management

- Generally well-tolerated due to low pressure system and difficulty of exposure increasing risk of more bleeding
 - Especially for suprarenal and retrohepatic segments
- Option for blunt mechanism of injuries
- If patient is hemodynamically stable and there are no other associated injuries, can consider non-operative management with serial imaging (CT with contrast)

14.1.3 Hemorrhage Control

- Pre-operative planning with appropriate resuscitation and blood products available
- Keep in mind coordination with other surgical teams and anesthesia
- Patients with abdominal venous injuries likely to be polytrauma patients, approach to assess and repair injuries should be time efficient
- IVC injuries considered Zone I retroperitoneal hematomas
- Follow general principles of gaining proximal and distal control
 - Can use low profile sponge sticks (Kittner sponge stick) or manual pressure
 - Be prepared to refine where you control as the dissection progresses
- Immediate control of hemorrhage may require ligation. However, once the field is cleared, coordinate with anesthesia to confirm they are making progress with resuscitation and then evaluate whether the patient can tolerate reconstruction at that time or whether you need to continue to follow damage control principles.
- 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. (J. T. Lee and Bongard 2002)

14.1.4 Primary repair

- Option for partial tears without risk of narrowing lumen
 - Narrowing lumen increases risk of thrombosis and thromboembolism
- Recommend figure of 8 or 16 to repair with 4-0 prolene suture on SH (small half circle) needle
 - Larger needles are easier to visualize in cases with high amounts of bleeding
 - When first suture is placed, recommend lifting on either side of suture with both strands to elevate the defect and maintain tension » improves visualization and placement of subsequent sutures to achieve full thickness bites

14.1.5 Ligation

- Well-tolerated really only for infrarenal segment IVC
- Not a viable option renal/suprarenal, retrohepatic, and suprahepatic IVC due to compromised outflow of vital organ structures (i.e. kidneys and liver)
- Interposition grafts are not readily performed due to inefficiency of time.

14.2 Infrarenal IVC

14.2.1 Anatomy

IVC from inferior portions of renal veins to iliac bifurcation

14.2.2 Non-operative Management

This is a low pressure system and therefore non-operative management can be observed in patients with blunt injuries and hemodynamically stable. However if there is penetrating trauma, or a laparotomy is performed for other reasons, the infrarenal IVC should be explored and repaired.

14.2.3 Exposure

- Right medial visceral rotation (Cattell-Braasch maneuver)
- Exposes in the proximal portion the IVC, inferior renal veins, 4th portion of duodenum, and pancreatic head
- Be aware of lumbar vessels posteriorly and control with vessel loop or clamp

14.2.4 Hemorrhage Control

- Proximal and distal control may not be sufficient as there can be significant bleeding from the many lumbar veins that feed into this area of the vena cava.

14.2.5 Primary Repair

- SH needle on 4-0 prolene
- Avoid luminal narrowing of IVC to prevent thrombosis/thromboembolism

14.2.6 Ligation

- Ligation can be tolerated and is appropriate when primary repair narrows lumen or if primary repair cannot be performed due to damage-control nature of operation
- Empirical 4-compartment fasciotomies of lower extremities is dependent on patient's resuscitation status
- Recommend fasciotomy if patient received large volumes of fluids/products, continues to be hemodynamically tenuous, anticipate long recovery

14.3 Renal/Suprarenal IVC

14.3.1 Anatomy

- 2-3 cm of IVC, from renal vein to inferior portion of hepatic veins
- Includes renal veins and gonadal veins

14.3.2 Non-operative Management

- Preferred due to location of injury » similar to retrohepatic and suprahepatic segments
- Exposure of segment may increase risk of bleeding, increase size of defect, or unroof hemostasis formed

14.3.3 Exposure

- Right medial visceral rotation (Cattell-Braasch) and mobilizing liver cephalad
- Liver mobilization may be required to adequately expose this portion of the IVC as it is below the hepatic veins but posterior to the liver parenchyma.
 - Divide the attachments of the liver to the diaphragm to be able to move liver cephalad
 - Caution as there are posterior lumbar veins and other branches that can cause more significant bleeding

14.3.4 Primary Repair

- Option if liver mobilization does not increase size of defect/tear
- Follow principles as described above

14.3.5 Ligation

- Not as well tolerated as infrarenal segment due to compromised of outflow to renal veins

14.4 Retrohepatic IVC

14.4.1 Anatomy

This region is very difficult to access and includes the IVC at the confluence of the hepatic veins.

14.4.2 Management

Non-operative management is preferred due to the location of injury. **The liver provides hemostasis due to overlying pressure and if liver mobilized, can cause more significant bleeding.**

14.4.3 Exposure

- Right medial visceral rotation (Cattell-Braasch) and mobilizing liver cephalad. Mobilizing the liver completely off the diaphragm can allow for better visualization.

14.4.4 Hemorrhage Control

- As in the more inferior segments of the IVC, it is important to control venous inflow from the more inferior IVC and lumbar veins.
- Direct control of the hepatic veins is difficult, so ongoing bleeding may require Total Hepatic Isolation to locate bleeding behind the liver
 - Total Hepatic Isolation is accomplished with vascular control of the porta hepatis with a Rommel tourniquet, also known as the Pringle maneuver.
 - Portal Vein - Accounts for 70-80% of hepatic inflow

- Hepatic Artery
- Finally, you need to control the suprahepatic segment of the IVC under the diaphragm to prevent back bleeding.
- Other options for bleeding control if total hepatic isolation not successful
 - Supraceliac aortic clamp
 - Can be considered if bleeding continues to help identify injury
 - Last option, usually unsurvivable
- If vascular control is required for an extended period, then an Atriocaval Shunt (AKA Schrock shunt) may be required to maintain venous return to the right atrium and prevent cardiovascular collapse.(Schrock 1968)
 - Can be accomplished with large chest tube with extra hole cut near base
 - Chest tube clamped at base and inserted via purse string suture at the right atrial appendage > advanced to infrarenal IVC
 - Place Rommel tourniquet below area of suprarenal IVC (above the renal veins and last hole in tube)
 - Second Rommel tourniquet placed above hepatic veins in intrapericardial IVC
 - [Click here for an image](#)

14.4.5 Primary Repair

- Careful liver mobilization due to multiple branch points and high risk of tear > will likely require total hepatic isolation for bleeding control
- Be careful of timing and patient status. May require packing and resuscitation or atrio-caval shunt during repair.

14.4.6 Ligation

Not as well tolerated due to compromised outflow of both renal and hepatic veins, can cause both kidney and liver ischemia

14.5 Suprahepatic IVC

14.5.1 Anatomy

Includes phrenic veins and pericardium to right atrium

14.5.2 Non-operative Management

- Preferred due to location of injury, difficult to access
- Liver provides hemostasis due to overlying pressure
- If liver mobilized, can cause more significant bleeding

14.5.3 Exposure

Divide xiphoid to open costal margins (approximately 2-3 cm cephalad). Wiley vein retractor may be used to fully mobilize the diaphragm and access the IVC in the chest through the diaphragm from the abdomen.

14.5.4 Hemorrhage Control

Likely will require total hepatic isolation and atriocaval shunt to fully control bleeding and maintain venous return to the heart.

14.5.5 Primary Repair

Can consider supraceliac aortic clamp and/or atriocaval shunt if extensive repair is required.

14.5.6 Ligation

Not as well tolerated due to compromised outflow of both renal and hepatic veins, can cause both kidney and liver ischemia.

14.6 Portal Vein and Superior Mesenteric Vein Injuries

Injuries to portal vein and SMV can be very catastrophic. Non-isolated injury — usually associated with other injuries (ie pancreatic + celiac vessels).

14.6.1 Exposure

- Pringle maneuver to gain control of porta hepatis
- Division of pancreatic head for better visualization

14.6.2 Management

- Resuscitation is key
 - 50% of blood volume is sequestered by splanchnic circulation
 - Coordination with surgical team and anesthesia to resuscitate appropriately
 - Consider insensible losses with open abdomen during OR time
- Repair these injuries if possible
 - Primary repair
 - Interposition graft conduits
 - Great saphenous vein
 - PTFE
 - Bovine pericardium
 - Ligation an option in damage-control (high mortality)
 - Temporizing measure to prepare for reconstruction once bleeding is controlled

Part V

Lower Extremity

15 Medical Management and Claudication

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15.1 Pathogenesis of peripheral arterial disease

What is Peripheral Artery Occlusive Disease (PAD)?

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

The disease has a step-wise 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 PAD?

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 with the rising incidence of diabetes a second population of patients with a significant PAD burden has developed.

Atherosclerosis begins with an injury to the intimal lining of the arterial wall, which, for example, can result from smoking, hypertension, hyperglycemia, or advanced age. Repeated injury leads to a chronic inflammatory process resulting in the uptake and oxidation of LDL, resulting in foam cells.(Hiltunen et al. 1998; Tsimikas et al. 2005; Witztum 1994) Foam cells result when unregulated scavenger receptors promote uptake of oxidized LDL by macrophages and smooth muscle cells. These foam cells become necrotic and then create the central core of the atheroma.(Owens 2019) This oxidized LDL form into intimal plaque, build up and calcification that may result in progressive vessel stenosis and subsequent occlusion, or plaque rupture with acute occlusion.

Atherosclerosis is particularly prone to developing at sites of arterial bifurcation (i.e. carotid bifurcation, femoral bifurcation) because turbulent flow and low laminar shear stress results

in the down regulation of eNOS and NO production. This in turn increases V-CAM-1 and reduces inhibition of KF-kB (resulting in its elevation). (Owens 2019)

💡 Take a Listen

Check out [our episode with Dr. Melina Kibbe, who is actively researching the role of nitric oxide in vascular pathology.](#)

Table 15.1: **AHA lesion classification** - defines plaque risk for rupture/thrombosis. Extrapolated from the coronary artery disease literature. (McGill et al. 2000)

Classification	Description
0	Normal artery
1	Initial atherosclerotic lesion
2	Fatty streak
3	Fatty plaque/raised fatty streak
4	Atheroma
5	Fibroatheroma - development of fibrotic cap, vascularization or calcium
6	Complicated lesion

Since the publication of WIfI in 2014 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 (see Chapter 16). (Mills et al. 2014a; Conte et al. 2019)

15.1.1 Risk Factors

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, platelet adhesion, and permeability of endothelial surface to fibrinogen. (United States Surgeon General 2014; Hackam and Anand 2003).
- Causes a prothrombotic environment by causing an increase in thromboxane A2 and decreasing prostacyclin thus overall resulting in 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. Increasing Incidence of Diabetes world-wide.(Boulton et al. 2005)
 - 2.8% of the global population affected by diabetes in 2000, estimated to be 4.4% by 2030.
 - 25% of patients with diabetes develop a diabetic foot ulcer (DFU), at some stage in their lives.
 - 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. (D. G. Armstrong, Boulton, and Bus 2017)
 - Limb Loss occurs every 20 seconds world-wide due to Diabetes related PAD or infection.
 - The Odds Risk for developing PAD in patients with DM ranges from 1.89 to 4.05.
 - Factors associated with developing PAD in diabetics include female gender, chronicity/severity of DM, insulin dependence, African American race or Hispanic ethnicity.(Regensteiner et al. 2015; Selvin et al. 2006; Wattanakit et al. 2005) An increase in HbA1C by 1% correlates with a 28% increase risk of developing PAD. (Amanda I. Adler et al. 2002)
 - The following mechanisms may lead to PAD in diabetics
 - Hyperglycemia reduces bioavailability of NO, which is a vasodilator, enhances endothelial function, and inhibits both platelet activation and smooth muscle migration.(Higashi et al. 2009)
 - Hyperglycemia activates protein kinase C and thus accumulation of mitochondrial ROS and prostanooids.(Geraldes and King 2010)
 - Endothelial dysfunction leads to increased endothelin-1 and vasoconstriction.(Ahlborg et al. 2007)
 - Given the pathological effect of diabetes upon multiple structures and systems the normal progressive history of diabetes related PAD is somewhat different to smoking related PAD. Clinically due to the combined effect of peripheral neuropathy and microvascular arterial disease, foot ulceration for many patients tends to be the initial presentation. 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, leading to poor outcomes and greater morbidity and mortality. (D. L. Armstrong et al. 2012)

- Neuropathy. The loss of the basic nociceptive mechanisms in the foot among diabetics, presents as a loss of protective sensation (LOPS). Neuropathy can be divided into three types:
 - Sensory: “stocking-glove” distribution
 - Motor: Intrinsic muscle wasting – resulting in deformities
 - Autonomic: Sympathetic nervous system pathology
- Structural deformities and gait disturbances
- Arterial disease of large and small vessels.
 - Large Vessel
 - Small Vessel
 - Both
- Foot infection(Dennis F. Bandyk 2018; KAYSSI, ROGERS, and NEVILLE 2019; Lepäntalo et al. 2011)

3. Hypertension

- The **most common** cardiovascular risk factor worldwide.
- USPSTF Grade A recommendation for HTN screening confirm readings outside clinical setting or at home before initiating treatment.(Piper et al. 2014)
- The Incidence of PAD increases to 2.5-fold in patients with Hypertension. (Kannel and McGee 1985)
- Each 20mg increase in BP, results in a 35-63% increase risk in PAD. Recommend keeping BP <140/90 or <130/80 in diabetics or CKD.(A. I. Adler et al. 2000; Bavry et al. 2010; Emdin et al. 2015; Itoga et al. 2018)

4. Dyslipidemia

- A strong association has long been identified as a risk factor for cardiovascular disease.(Langlois and Nordestgaard 2018; Diehm et al. 2004)
- 25% cardiovascular event reduction for each 39 mg/dL (1mmol/L) reduction in LDL. (H. P. S. C. Group 2007)

- JUPITER Study - Randomized rosuvastatin 20mg to placebo in healthy patients. Found significant decrease in unstable angina, myocardial infarction and stroke. However, patients had a higher incidence of physician-reported diabetes, but no difference in myopathy.(Ridker et al. 2008)
- Fish oil is also indicated for secondary prevention among patients with known cardiovascular disease.(Grenon et al. 2012; Kris-Etherton et al. 2002; Siscovick et al. 2017)

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. *Race and Ethnicity*

- The MultiEthnic Study on Atherosclerosis (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)

4. Family History

- Family history of atherosclerotic disease increases an individual's risk of atherosclerotic disease themselves.

15.1.2 Landmark Studies

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

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.(Ikram et al. 2017)
3. **CVHS:** 1989-1999 Longitudinal Study : n>5000 Multicenter 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. (F. G. R. Fowkes et al. 1991)

15.2 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 of intermittent claudication was historically graded by Fontaine (1954) (Fontaine, Kim, and Kiely 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	A symptomatic - no hemodynamic significant occlusive disease
I	1	Mild Claudication

Grade	Category	Clinical Description
I	2	Moderate Claudication
I	3	Severe Claudication
II	4	Ischemic Rest Pain
III	5	Minor Tissue Loss
III	6	Major Tissue Loss

Leriche Syndrome - Is a clinical triad of: claudication (buttock, thigh, and calf), impotence, and decreased lower limb pulses, which signifies aortoiliac occlusive disease. (Frederick, Newman, and Kohlwes 2010; Leriche and Morel 1948; Setacci et al. 2012)

Neurogenic Claudication - 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)

15.2.1 Evaluation

What is involved in the work up of patients with PAD/Intermittent Claudication?

SVS Guidelines:

"We recommend using ABPI 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 ABPI."(Conte, Pomposelli, Clair, Geraghty, McKinsey, Mills, Moneta, Murad, Powell, Reed, and al. 2015)

Grade 1 Level of Evidence A

- ABPI
- Exercise ABPI
- Ultrasound

What is an ABPI and how is it measured?

The AHA released guidelines on how to perform an ABPI in an attempt to standardize 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, Hobbs, and Irivne 2005; K. Ouriel et al. 1982)

Table 15.3: Interpretation of ABPIs.

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

Patients with diabetes may have non-compressible tibial vessels and therefore toe pressures can be particularly useful in this population.

Segmental pulse volume recordings (PVR) can provide information about the quality of perfusion at the level of the thigh or calf helping to localize the likely level of occlusive disease.

What are Exercise ABPI studies?

Constant Load Testing – (unlike the Graded Test – Bruce Protocol used in testing for coronary artery disease).

The physiological changes in ABPI related to exercise are due to the fact that exercise increases SBP and decreases SVR and, therefore, will affect flow through a stenosis making it more physiologically relevant during exercise and result in a decreased pressure in the lower extremity. (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)

Furthermore patients with single level iliac disease may present with buttock/thigh claudication with preserved pulses. Exercise treadmill ABPI is particularly useful in determining the severity of disease in these patients. (Aboyans et al. 2010) For more on exercise testing see Section 20.5

What is the ultrasound duplex criteria for defining PAD?

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

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

Adapted from Stone and Hass. Vascular Laboratory: Arterial Duplex Scanning. Rutherford's Vascular Surgery and Endovascular Therapy. 2019.(Patrick A. 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, Pomposelli, Clair, Geraghty, McKinsey, Mills, Moneta, Murad, Powell, Reed, Schanzer, et al. 2015)

Additional Guidelines

- TASC 1 and 2(Norgren et al. 2007)
- European Society of Vascular Surgery(Aboyans et al. 2018)
- AHA/ACC Guidelines(Gerhard-Herman et al. 2017) - multidisciplinary, involving vascular surgeons

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Check out [our episode with Dr. Leila Mureebe](#), where we briefly discuss her role in the multidisciplinary development of the AHA/ACC guidelines.

15.2.2 Management

15.2.2.1 Medical Management

What is the initial management of asymptomatic patients with PAD?

1. Smoking Cessation – Multidisciplinary comprehensive smoking cessation interventions – repeatedly until tobacco use has stopped (*Grade A – 1*) First line therapy is behavioral therapy + varenicline/bupropion or nicotine replacement therapy (NRT) per EAGLES trial.(Anthenelli et al. 2016; Vogeler, McClain, and Evoy 2016)
 - Preloading with varenicline (4 weeks) or bupropion (5-7d) prior to quitting has demonstrated higher abstinence at 12w.(Ratchford and Black 2011)

- Varenicline is a partial nicotine receptor agonist.(Cahill et al. 2016)
- Bupropion is a selective dopamine and norepinephrine uptake inhibitor.(J. R. Hughes et al. 2014)
- NRT is preferred if rapid onset of action is needed.(Thomsen, Villebro, and Møller 2014) However, contraindicated in setting of recent MI, severe angina, or life threatening arrhythmias.

2. Intervention is not only not recommended, but invasive treatment is recommended against, in the absence of symptoms (Grade A -1)

How can we medically manage asymptomatic PAD based on the SVS and AHA Guidelines?

- ***Anti-Platelet Therapy***

- The Aspirin for Asymptomatic Atherosclerosis Trial – n=3350, aspirin versus placebo. 8 years follow up no difference in events (F. Gerald R. Fowkes 2010) – therefore benefit unknown

- ***Statin Therapy***

- The Heart Protection Study (H. P. S. C. Group 2007) - this study looked at statins in patients with PAD but not completely asymptomatic, they had other risk factors such as diabetes, IHD, cerebrovascular disease, or hypertension. Without these risk factors the benefit of Statin therapy remains unclear.
- 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).
- Statins are particularly useful for risk reduction of cardiovascular events in diabetics.(Collaborators et al. 2008; Colhoun et al. 2004; Stamler et al. 1993)
- Statin-associated muscle symptoms (SAMS) is the most common side effect and occurrence has been reported as high as 30% in some cohorts.(Keen et al. 2014) Other side effects are rare (1.5-5% of patients) and include DM2 new onset, neurocognitive effects, hepatotoxicity, renal toxicity and pancreatitis.(Bitzur et al. 2013; Saxon and Eckel 2016; N. C. Ward, Watts, and Eckel 2019)

- ***Exercise and Limb Function***

- No clear evidence that physical therapy improves the patient outcomes or quality of life.

- ***Surveillance***

- No benefit from US surveillance, unclear benefit of ABPI surveillance.

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

- **Smoking Cessation:** As in with asymptomatic disease above, 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*)
- **Anti-Platelet Therapy:** Aspirin therapy (75-325mg daily) is recommended to reduce cardiovascular events in patients with PAD (*Grade 1-Level A*) (Antithrombotic Trialists'Collaboration 2002; Antithrombotic Trialists'(ATT) Collaboration et al. 2009)
 - Literature reviews find benefits in secondary prevention with antiplatelet monotherapy (aspirin or clopidogrel) in symptomatic PAD.(Banerjee et al. 2015) Some initial studies did demonstrate benefit from vorapaxar as well.(Rooke et al. 2013)
 - There is evidence that Clopidogrel 75mg compared to Aspirin is better in event reduction. (CAPRIE)(Committee 1996a)– replacing Aspirin with Clopidogrel (*Grade 1-Level B*)
 - Recent evidence from the COMPASS trial has suggested that the addition of low dose rivaroxaban (2.5mg BID) to aspirin (100mg daily) has lower MALE, composite endpoint of cardiovascular death, stroke and MI. Subgroup analysis suggested clopidogrel may be better with reduced risk of GI bleed and MACE.(Anand et al. 2018; Eikelboom et al. 2017)
- **Diabetes Mellitus:** Optimization of HbA1C < 7% (*Grade 1-Level B*)(Lajoie 2019)
- **Hypertension:** Indicated B-Blockers for hypertension. (*Grade 1-Level B*)
 - There's no evidence that beta blockers worsen the symptoms of intermittent claudication, but the patients should be monitored for episodes of hypotension
 - Initiation of beta-blockers in the immediate preoperative period is associated with worse outcomes.(Blessberger et al. 2018; P. S. Group et al. 2008)
 - ACE Inhibitors are first line in patients with heart failure, diabetes or kidney disease but should be avoided in patients with renal artery stenosis (RAS).(Barrons and Woods 2016)
- **Homocysteine:** While higher homocysteine levels are associated with PAD and risk of venous and arterial thrombosis.(D'Angelo and Selhub 1997; Khandanpour et al. 2009; Liebman 2019) Recommendation against Folic Acid and Vit B12(*Grade 2 – C*)

- Patients are at higher risk of depression (almost 20%), therefore PHQ-9 and -2 are good screening tools and should be used in these patients.(Jha et al. 2019; McDermott et al. 2016; J. L. Ramirez, Drudi, and Grenon 2018)

To improve Limb Function in patients with Claudication:

- **Cilostozol** use – Claudication 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)
- **Supervised Exercise Therapy**
 - First Line Therapy recommended SEP: minimum three times per week (30-60 min/session) for at least 12 weeks (*Grade 1 Level A*)
 - More durable functional improvements than angioplasty alone, however no current data demonstrating impact on need for revascularization or amputation rate.(F. Fakhry et al. 2013; Farzin Fakhry et al. 2015; T. P. Murphy et al. 2012, 2015)
 - Meta-analysis of 32 RCTs: 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 14 RCTs: Supervised better than Non-Supervised Programs. (Hageman et al. 2018)

15.2.2.2 Open and Endovascular Management

What is the surgical 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, Pomposelli, Clair, Geraghty, McKinsey, Mills, Moneta, Murad, Powell, Reed, and al. 2015; Norgren et al. 2007)
- Patient Selection for Intervention:
 - As above, 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 hemodynamic (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 categorize 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 is preferred as primary therapy over open surgery for focal aortoiliac, common Iliac and external iliac disease causing Claudication. (*Grade 1 Evidence B*)
 - Hybrid approaches are recommended for Iliac disease involving CFA with the addition of an open femoral endarterectomy. (*Grade 1 Level B*)
 - Direct Surgical reconstruction (bypass, endarterectomy) in patients with reasonable surgical risk and diffuse aortoiliac occlusive disease not amenable to endovascular approach, after one or more failed attempts at EVT, or combined occlusive and aneurysmal disease. (*Grade 1 Evidence B*)
 - Bilateral external iliac occlusion may be best treated with end to side aortobifemoral bypass to allow for continued perfusion of the pelvis. (Jquinandi et al. 2008; Akker et al. 1992; Brewster and Darling 1978)
- ***Infrainguinal Disease:*** Reviewing historical data comparing all EVT compared with surgical bypass EVTs are less durable, especially when there's diffuse or long segments of occlusion/multilevel infrainguinal disease. **Most recommendations are based on low level evidence when comparing EVT versus Open Surgery**
 - Endovascular therapy has become first line therapy for focal SFA disease not involving the vessel origin. (*Grade 1 Level C*)
 - Endovascular therapy with self-expanding stent (with or without paclitaxel) is recommended for SFA disease 5-15cm (*Grade 1 Level B*)
 - After SFA stenting, SVS and ACC recommend at least 30d dual anti-platelet, then single anti-platelet or anti-platelet and rivaroxaban.(Hussain et al. 2018; Vascular Surgery Lower Extremity Guidelines Writing Group et al. 2015; Strobl et al. 2013; Tepe et al. 2012)

- This was in 2015 prior to the Katsanos meta-analysis suggesting there may be a mortality risk with paclitaxel drug delivery(Katsanos et al. 2018)
- Recommend against treatment of isolated infrapopliteal disease for claudication (*Grade 1 Level C*)
- Initial Surgical Bypass (*with vein: Grade 1 Level A*) is recommended in the following clinical scenarios.
 - Diffuse femoropopliteal disease
 - Small caliber vessels <5mm
 - Extensive diffuse calcification in SFA
 - Average or Low Operative Risk (*Grade 1 Level B*)

 Take a Listen

Check out [our episode](#) where we discuss overuse of early peripheral interventions in patients with claudication with Dr. Caitlin Hicks.

16 CLTI

Authors: *Nedal Katib and Danielle Bajakian*

Contributor: *Eilidh Gunn*

16.1 Guidelines

Much of the content here is adapted from the most recent **Global vascular guidelines on the management of chronic limb-threatening ischemia**(Conte et al. 2019).

In 2019 the SVS, the ESVS and the World federation of Vascular Societies (WFVS) joined forces to put together the structure and funding of the ***Global Vascular Guidelines Initiative (G VG)***. 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.”
- Chronic Limb Threatening Ischemia (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 utilized the structure of the ***GRADE*** certainty of evidence system.

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

It is important to note that compared to most guidelines, unfortunately in the management of CLTI, particularly when it comes to revascularisation, the level of evidence is generally **LOW**. Again, it is important to highlight that the significance of these guidelines in developing a standard approach and appropriately stratifying patients in not only management but ongoing research.

16.2 Demographics

16.2.1 Etiology and Presentation

How does PAD pathologically progress clinically into Chronic Limb Threatening Ischemia (CLTI)?

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 spreading 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 hemodynamic significant occlusive disease
I	1	Mild Claudication
I	2	Moderate Claudication
I	3	Severe Claudication
II	4	Ischemic Rest Pain
III	5	Minor Tissue Loss
III	6	Major Tissue Loss

What is Chronic Limb Threatening Ischemia (CLTI), sometimes previously known as Critical Limb Ischemia (CLI)?

In the last decade leading up to the 2021 Global Vascular Guidelines (GVG), the term (Chronic Limb threatening Ischemia) 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 (no gravitational assistance) and systolic BP drop during sleep.

16.2.2 Natural History

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).

Patients with CLTI have a high mortality risk (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)

What has changed in the last few decades?

Etiologically the prevalence of smokers(Ex and Current) in the population has decreased and the prevalence of diabetes has increased. For more details on the relationship between diabetes and pad, see Section [15.1.1](#)

16.2.3 WiFi Classification

What is the WiFi Classification?

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 behavior 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.(Mills et al. 2014a)

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.”

💡 Get the App!

Check out the [SVS iPG App](#) for help using the WiFi classification during your day-to-day care of CLTI patients. The best way to understand the WiFi score is to use it on a few patients and see what recommendations are provided.

16.2.4 Differential Diagnosis

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

Acute lower limb ischemia has a different clinical presentation, but there may be some overlap with Acute on 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 Section [3.3.4](#)
- Scleroderma
- Fibromuscular Dysplasia - for more see Section [12.2](#)
- Popliteal Artery Entrapment - for more see Section [17.4.1](#)
- Cystic Adventitial Disease - for more see Section [17.4.2](#)
- Persistent Sciatic Artery Disease
- Neurogenic pain

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 Chapter [17](#) for more.

16.3 Evaluation

What aspects of the clinical assessment is important?

Clinical Assessment involves a full history (the differential mentioned in Intermittent Claudication) and examination.

- **Clinical Examination:**
 - *Buerger's Test (Buerger 1908) / AKA Ratschows Test* (Max-Ratschow-Klinik) identifies when there is critical ischemia without necrosis yet or gangrene, and is characterized 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 ischemia because of the autoregulation being paralyzed a significantly higher portion of the capillaries open up.

- ***The ischemic 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 etiologies) – important not only to identify extent of disease but also to exclude other etiologies:
 - 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)

16.3.1 Physiologic Testing

What other clinical evaluation is necessary for the patient with CLTI?

In addition to history, examination, and 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.

In the PAD and intermittent claudication sub chapters the non-invasive methods of assessment for these patients have been discussed. In addition it is important to emphasize the role of Toe Pressures (TP) and Toe Pressure Index (TPI) in this cohort of patients.

CLTI measurements most predictive of non-healing are ankle pressure <50mmHg, ABI <0.4, TcPO₂ <20mmHg, and TP <20mmHg.(Gerhard-Herman et al. 2017; Wickström et al. 2017). 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 ABPI (>1.3) - which is associated with an elevated risk of cardiovascular mortality. Outcomes in patients with ESRD are worse in relation to amputation free survival and amputation rates, regardless of revascularization strategy.(Meyer et al. 2018) Toe pressures are particularly important in this scenario. (Resnick et al. 2004; Vitti et al. 1994)

Regarding non-invasive assessment for wound healing, a TcPO₂ greater than 40mmHg has the greatest correlation with amputation stump healing.(Malone et al. 1987)

16.3.2 Imaging

What imaging assessment is required?

The CLTI Guidelines outline an algorithm of attaining Arterial Anatomical 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.(Giurato et al. 2017)

The most recently published RCT in CLTI, BEST-CLI, highlights the good outcomes of surgical bypass with saphenous vein, suggesting that vein mapping also be considered a routine imaging study during the workup of patients. This will help to clearly understand revascularization options before attempting endovascular diagnostic or therapeutic interventions.(Farber et al. 2022)

💡 Take a Listen

Check out [our episode with BEST-CLI Principle Investigators Dr. Alik Farber and Dr. Matthew Menard](#) as they discuss the development of this trial. This episode was created as they were completing enrollment and prior to the release of the results of their study.

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

Because the existing arterial anatomical staging of disease is vague, “lesion focused”, and not all encompassing (beyond the concept of ‘in-line pulsatile flow to the foot’), **GLASS** focuses on infrainguinal disease, and attempts to incorporate all aspects in its staging to improve vascular care and evidence-based revascularisation (EBR) outcomes.

GLASS is a grading system based on anatomical and subjective assessment of calcification and incorporates two novel and important concepts:

1. The Target Arterial Path (TAP)
2. Estimated Limb-Based Patency (LBP)

As GLASS focuses on Infrainguinal disease, with the aortoiliac (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 CTOs. 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 endoVascular 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.”

💡 Again, Get the App!

Check out the [SVS iPG App](#) for help with also the GLASS Criteria during your day-to-day care of CLTI patients. The best way to understand the GLASS Criteria is to use it on a few patients and see what recommendations are provided.

16.4 Management

16.4.1 Medical 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 1996b)

- **High-intensity statin therapy** to reduce all-cause and cardiovascular mortality - Atorvastatin 80mg or Rosuvastatin 40mg. These can be titrated down to atorvastatin 40mg or rosuvastatin 20mg if unable to tolerate. (*Grade 1 Level A*) (Arya et al. 2018; Grundy et al. 2019)
- 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 investigated with plain radiography, ESR, CRP, cultures and, managed with iv antibiotics. Signs of systemic sepsis, such as fevers, tachycardia or shock, such as hypotension, should warrant urgent debridement and drainage, regardless of vascular status.
- For more details, see Section [15.2.2.1](#)

 Take a Listen

Check out [our episode with Dr. Venita Chandra](#), where we discuss the best practices for multidisciplinary limb salvage care.

16.4.2 Endovascular and Surgical Management

Which revascularisation management strategies exist for CLTI?

The mainstay of management for patients with CLTI have always been based on the fundamental principle of limb salvage. Given the high risk of limb loss in these patients there is a low threshold to revascularize these patients if they have occlusive disease that is treatable. But strategy has varied significantly.(Conte et al. 2019)

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 groups include African Americans, lowest median income, medicaid insurance, uninsured, or those from regions with less access to vascular surgeons.(K. Hughes et al. 2019; Ho et al. 2005; Eslami, Zayaruzny, and Fitzgerald 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?

Patients with rest pain 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.”

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)

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”

- 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)
- If the target is below the knee, then all autogenous conduits—contralateral GSV, SSV, and spliced arm vein—are preferred over prosthetic grafts.(Brochado Neto et al. 2014; Faries et al. 2000; Taylor et al. 1987)

2. Patient overall risk (as mentioned above) and Limb Staging

- Planning distal bypasses should take into account the angiosome of the wound and the most distal healthy inflow vessel to result in the shortest bypass possible.(Hingorani et al. 2016; Jongsma et al. 2017)
- Intensified anti-thrombotic therapy may be needed in patients with “high risk” infrainguinal bypasses—prosthetic conduit, below the knee target, suboptimal conduit, poor arterial runoff, extensive lesions or tissue loss. Single anti-platelet may not be sufficient, and should be intensified to asa/rivaroxaban, dual antiplatelet, or anti-coagulation with Vit K antagonist.(Conte, Pomposelli, Clair, Geraghty, McKinsey, Mills, Moneta, Murad, Powell, Reed, and al. 2015; Hussain et al. 2018; Strobl et al. 2013; Tepe et al. 2012)

3. The Target EndoVascular Intervention (TVI) outcomes

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

In November of 2022, the first results of the **BEST-CLI** were published in the NEJM. BEST-CLI is the first international, open-label, multicenter, superiority trial of its scope and scale. The key results found that in patients with adequate great saphenous vein conduit, the surgical group demonstrated a lower incidence of major adverse limb events or death (the study’s

primary endpoint) than the endovascular group. They found relatively equivalent outcomes in patients with inadequate autogenous conduit.(Farber et al. 2022) The full impact of the results of this study is yet to be determined, and there will likely be several additional analyses published from this study's data. It is unlikely that this data will impact what is tested on this year's exams. However, you should keep an eye out for future results!

Take a Listen

Check out [our episode with BEST-CLI Principle Investigators Dr. Alik Farber and Dr. Matthew Menard](#) as they discuss the development of this trial. This episode was created as they were completing enrollment and prior to the release of the results of their study.

Other than this most recent study, the evidence is largely of poor quality, and is retrospective, non-controlled, or industry sponsored. **BASIL** (Bypass versus Angioplasty in Severe Ischemia of the Leg) previously had been the only multicenter RCT (BASIL -2 and 3 underway) directly comparing an endo versus open management strategy for CLTI and Infra-inguinal occlusive disease.

BASIL compared POBA and Bypass across multiple centers (27 centers, n=452, 1999-2004) in the UK. Primary endpoint was Amputation-Free Survival (AFS). (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:
 - Prosthetic Bypass Patients did very poorly (even compared to POBA).
 - Patients who had bypass after failed POBA had significantly worse AFS compared to those treated with a bypass as initial treatment.

Criticism of BASIL:

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 transluminal, or subintimal angioplasty. Atherectomy, stenting, and drug coated balloons are often used, but should currently not be considered first line, 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 follow up for endovascular interventions of the lower extremity has not yet been established, but should at least include a pulse exam, ABPI and duplex to establish a new baseline after intervention.(Zierler et al. 2018; Mohler et al. 2012)

What are the **BASIL 2, **BASIL 3**, and **BEST-CLI** Trials?**

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).

16.4.3 Complications

What are some complications of lower extremity revascularization procedures?

For a comprehensive list of access complications after endovascular therapy, see Section [22.1](#)

Femoral exploration carries high risk of infection or lymphatic leak. Lymphatic leaks often resolve spontaneously. Infected lymphatic leaks, particularly in the setting of prosthetic bypass require exploration. Should a lymph leak not resolve with conservative management, 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.(A. Obara et al. 2014; M. V. Weaver et al. 2014)

17 Acute Limb Ischemia

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Contributors: *Eilidh Gunn*

17.1 Overview

17.1.1 Demographics

17.1.1.1 Etiology

What is Acute Limb Ischemia and what does it encompass?

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

- **Embolism**
 - *Cardiac*
 - Typically due to atrial fibrillation.
 - Acute upper limb 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.
 - *Atherosclerosis* – 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** - Small aneurysms (such as popliteal) are less likely to rupture, but more likely to thrombose or embolize distally, causing an acute limb ischemic event.
- **Acute on Chronic Progression of Atherosclerosis** - Acute on Chronic Progression of Atherosclerosis: 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.
- **Bypass Graft**
- **Dissection** - for more, see Chapter 6
- **Thoracic Outlet Syndrome** in the upper extremity - for more, see Section 4.3
- **Vasospasm** (severe) - for more, see Section 3.3

17.1.1.2 Presentation

What is the patient presentation of ALI? Are there any differences between upper and 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 ischemic time increases until neuroischemic sensory loss occurs.
- **Pallor:** the limb appears pale compared to the non ischemic limb. There is delayed or absent capillary refill.
- **Poikilothermia:** (just a way to make “cold” into a “P” – really means cold limb) Literally the inability to regulate one’s body temperature, or dependent on ambient temperature as cold blooded animals are. If there is no limb perfusion, it acclimates to the ambient temperature and feels cold to the touch.
- **Pulseless:** Self explanatory. It is worth examining the contralateral limb, if it has normal pulses, it suggests the absence of PAOD or CLTI making thrombosis in situ less likely to be the cause of ALI.
- **Paresthesia and Paralysis** are the last two Ps. Paresthesia is an earlier sign of ischemic nerve dysfunction, and paralysis is a later sign. In the lower leg, ischemic changes often affects the anterior compartment first, and sensory loss over the dorsum of the foot is, therefore, one of the earlier neurologic deficits in ALI.

This is why a thorough physical exam is key; comparing both limbs and including handheld doppler exam. It can be difficult 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 it is also essential to assess sensory and motor function in addition to the pulse exam. This helps gauge the urgency of intervention.

Acute paralysis, mottling of bilateral lower extremities and absent femoral pulses should raise concern for aortic occlusion.(J. C. Wang, Kim, and Kashyap 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)

1. Viable - Not immediately threatened
 - Sensory Loss: None; Motor Weakness: None
 - Arterial Signal: Present; Venous Signal: Present
2. Threatened
 - a. Marginally - Salvageable if promptly treated (<24hrs)
 - Sensory Loss: Minimal (toes) or none; Motor Weakness: None
 - Arterial Signal: Weak/absent; Venous Signal: Present
 - b. Immediately - Salvageable if immediately treated (<6hrs)
 - Sensory Loss: More than toes; Motor Weakness: Mild/Moderate
 - Arterial Signal: Absent; Venous Signal: Absent
3. Irreversible - Major tissue loss or permanent nerve damage inevitable; Minimal benefit from revasc
 - Sensory Loss: Profound anesthesia; Motor Weakness: Dense paralysis (rigor)
 - Arterial Signal: Absent; Venous Signal: Absent

17.1.2 Evaluation

What does the work up for ALI entail? How is the diagnosis made?

- The diagnosis can often be made on history, physical exam, and bilateral ABPIs. 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 at your institution, but generally 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 management.
- Bedside ultrasound can also be very helpful, especially color flow doppler US.

The decision to perform cross-sectional imaging before angiography varies depending on how severe the presentation is and how quickly you can obtain imaging. The patient's renal function may also influence the decision to administer two contrast loads (CT followed by endovascular intervention). If a femoral pulse is present it could be feasible to proceed with on-table angiogram without a CT scan. If femoral pulses are absent there is increased suspicion of aortoiliac disease and therefore CT angiogram will be beneficial for both diagnosis and operative planning.

In patients with severe renal insufficiency, MRA or MR time-of-flight can be helpful, but these studies usually take longer to obtain and may not be quickly available in an acute threatened limb situation. Therefore going straight to angiography should be considered, as to get the most immediate and best imaging of the tibial vessels. (Creager, Kaufman, and Conte 2012; Earnshaw 2019)

17.1.3 Management

What is normally done in the initial management of ALI?

- ***Anticoagulation:*** IV unfractionated heparin should be 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). 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 – or follow local protocol.
- ***Supportive Care:*** Analgesia, iv fluids.
- ***Labs:*** A full panel including serum chemistry with BUN, Cr, CBC, and coagulation studies should be obtained. Baseline plasma CPK can be helpful to monitor for evidence of rhabdomyolysis after reperfusion.

17.1.3.1 Medical Management

Primarily with anticoagulation using heparin or a direct Xa inhibitor.

17.1.3.2 Surgical Management

- ***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, multiple life limiting comorbidities, and significant aortoiliac baseline disease may benefit from primary axillo-bifemoral bypass. (Mohanapatra et al. 2018)
- ***Endarterectomy*** – not necessarily required but might be used for common femoral exposure and distal access depending on vessel disease burden.

17.1.3.3 Endovascular Management

- ***Pharmacological 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 CT head. (Kenneth Ouriel, Veith, and Sasahara 1998)
 - If fibrinogen drops below 100mg/dl, recommend immediate cessation of lytic agent and consider cyroprecipitate (includes fibrinogen, factor VIII, von Willebrand's factor, factor XIII). (Mann 2019)
 - Pharmacological thrombolysis may be delivered over a short duration in the operation room, or a catheter may be left and tPA administered over a longer period. Two catheters for delivery include an Cragg-McNamara catheter with multiple side holes or an EKOS catheter that uses ultrasonic waves to aid with thrombus lysis.

- **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.

17.1.3.4 Relevant Literature

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:
 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.
 2. Patients with very long duration of ischemia, up to 6 months, were also included in the study.
- When outcomes for patients who had been symptomatic for less than 2 weeks were analysed, the thrombolysis patients actually did better. (T. S. 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 between the open surgery and CDT groups but higher major bleeding in the CDT group. (Kenneth Ouriel, Veith, and Sasahara 1998)

Meta Analysis: 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)

- Evidence considered, it is very reasonable to think about a catheter-directed therapy especially if the presentation is acute, less than 2 weeks or so. There are, however, certain anatomical locations, such as common femoral or brachial arteries, that most surgeons would favor a simple open procedure (i.e. balloon thrombectomy).

17.1.3.5 Decision Making

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

- **Class I** patients 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.
- **Class III** ALI is usually treated with primary amputation because revascularization is unlikely to restore function to the limb and restoring blood flow 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 liquefaction 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. (H. Obara, Matsubara, and Kitagawa 2018) Treatment of this is largely supportive with fluids.

17.2 Compartment Syndrome

17.2.1 Etiology

What is the pathophysiology and presentation 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 6 hours of reperfusion. (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, reducing arterial pressure and increasing venous pressure.(Frink et al. 2010; McQueen and Court-Brown 1996; Elliott and Johnstone 2003; Papalambros et al. 1989)
- Clinically patients can have neurological dysfunction with sensory motor deficits, but the most common presentation is a tense extremity with severe pain on passive movement of the muscles in the compartment. If the lower leg is affected this is often on dorsiflexion/plantar flexion of the ankle. A sensitive indicator is loss of two point discrimination (proprioception).

17.2.2 Evaluation

How do you diagnose compartment syndrome?

- **Physical Exam:** Tenderness, (especially over anterior compartment) and paresthesias (especially between first and second toes).
 - ***Anterior Compartment:*** Sensory distribution and muscular innervation from the deep peroneal nerve (this is a VSITE favorite).
 - ***Deep Posterior Compartment:*** Sensory distribution and muscular innervation from the tibial nerve(Velmahos and Toutouzas 2002).
- **Compartment Pressures:**
 - A needle is required to access the compartment and a pressure monitoring system (this 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. 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 the clinical suspicion of compartment syndrome is high, do not be reassured by “normal” compartment pressures. It is relatively low risk to do fasciotomies, but the risk of limb loss is high for a missed compartment syndrome.

17.2.3 Management

Management of compartment syndrome is a fasciotomy, what are the types of fasciotomies?

- **Forearm and Upper Arm Fasciotomies:** 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.
- **Thigh:** Performed through medial and lateral incisions to release the lateral, medial and posterior compartment.
- **Lower Leg:** The most common type is a lower leg 4 compartment fasciotomy and is explained in more detail below.

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 should be made generously. Sometimes the skin incision can be a little short of the fascial incision, but they should be long in order to fully release the compartments.
4. After hemostasis loose dressings should be applied. 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.

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.

17.3 Blue Toe Syndrome

17.3.1 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)

17.3.2 Evaluation

Patients should have a work up for investigation of an embolic source. Patients without a clear recent source of embolization on history or physical examination, should undergo:

- CTA of chest/abdomen/pelvis to look for an arterial lesion.
- Echocardiogram to identify a cardiac source.
- Holter monitor to evaluate for an underlying arrhythmias.

If no obvious source of embolism can be identified, further investigation for underlying cancer may be required due to malignancy leading to prothrombotic state.

17.3.3 Management

Cholesterol Embolization: Medical management with a single antiplatelet agent and statin. Anticoagulation may precipitate further embolization. (Ghahramani, Seline, and Wanat 2016; Quinones and Saric 2013)

Specific Thrombogenic Arterial Lesion: Medical management with anticoagulation. Recurrent symptoms on anticoagulation may require stent coverage or, in a young patient, thrombectomy. (Reyes Valdivia et al. 2017; Jeyabalan et al. 2014; Verma et al. 2014)

17.4 Non-atheromatous Popliteal Artery Disease

17.4.1 Popliteal Artery Entrapment Syndrome (PAES)

17.4.1.1 Etiology and Presentation

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

Etiologically due to embryologically abnormal lateral attachment of the medial head of the gastrocnemius muscle.(Gokkus et al. 2014; Lejay et al. 2014) Type IV PAES caused by compression with popliteus muscle.

17.4.1.2 Evaluation

Physical examination findings will be loss of pedal pulse on active plantar flexion 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)

17.4.1.3 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)

17.4.2 Cystic Adventitial Disease

17.4.2.1 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.

17.4.2.2 Evaluation

Often diagnosed on duplex ultrasound or CTA. For a representative image, see Section [20.5.1.4](#)

17.4.2.3 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 an interposition graft can be effective but should be considered as a final management strategy.(S. Li et al. 2017)

17.5 Amputations

The majority of questions regarding amputations stem from decision making around peripheral arterial disease. You may not see many questions asking uniquely about amputation technique or management, however this topic is VERY important for the successful management of peripheral arterial disease. Rutherford's has two fantastic chapters on decision making(Bianchi and Jr 2018) and techniques(Eidt and Kalapatapu 2019).

The most commonly performed lower extremity amputations include:

17.5.1 Above-knee, trans-femoral amputation (AKA)

Above the knee amputations are most often performed with a fish mouth incision. Suture ligation of the neurovascular bundle at the level of the mid SFA and transection of the femur.

17.5.2 Below-knee, trans-tibial amputation (BKA)

Below the knee amputation often performed with a long posterior, or Burgess, flap. Each tibial neuro vascular bundle is ligated individually. The fibula is transected 2-3cm more proximally than the tibia to avoid weight bearing from the prosthetic.

Although hotly debated, a Cochrane review demonstrated no difference in outcomes based on flap type. However, the review does recommend performing amputations in two stages in the setting of wet gangrene.(Tisi and Than 2014)

17.5.3 Transmetatarsal amputation (TMA)

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)

18 Venous Disease

Authors: *Leanna Erete, Andrew Nickinson, Aminder Singh and Manj Gohel*

18.1 Chronic Venous Insufficiency

18.1.1 Terminology and Presentation

What is chronic venous insufficiency and how common is it?

I thought the first question was going to be nice, easy and uncontroversial, but you've started with CVI. What you've highlighted 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 venous space and everywhere else. A really important documents to guide people 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 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 one of the terms that was described was chronic venous insufficiency. The official definition is it is a venous disease between C3 and C6 on the CEAP 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.(Eklöf et al. 2004; Eklof et al. 2009)

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 a very common problem, with lots of people that need treatment.

The Edinburgh Vein Study found that half of the general population with chronic venous disease deteriorated over the 13 year study. Factors associated with disease progression included family/personal history of DVT. Of note, female gender, obesity, number of pregnancies, and smoking status were not associated with progression.(C.-C. Lee et al. 2015)

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 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 status 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 part is the clinical bit, which is C1 to C6. CEAP 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 this was a useful revision of CEAP.(Lurie et al. 2020)

CEAP Classification System and Reporting Standard Revision 2020	
C (Clinical Manifestations), E (Etiology), A (Anatomic Distribution), P (Pathophysiology)	
C0	No visible or palpable signs of venous disease
C1	Telangiectasias or reticular veins
C2	Varicose veins
C2r	Recurrent varicose veins
C3	Edema
C4	Changes in skin and subcutaneous tissue secondary to chronic venous disease
C4a	Pigmentation or eczema
C4b	Lipodermatosclerosis or atrophie blanche
C4c	Corona phlebectatica
C5	Healed
C6	Active venous ulcer
C6r	Recurrent active venous ulcer

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Now in response to the limitations of CEAP, 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 CEAP.(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 (CEAP and VCSS) are the most relevant and important ones to know about. Many of the QOL tools are too cumbersome to use in day to day practice. We are in dire need of an 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 CEAP 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.

18.1.2 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 are really 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 venous flow, antegrade or 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 any 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 Section [20.7](#).

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. But clinical assessments and duplex assessment are the bedrock of every assessment of every venous patient. So that's the foundation.

18.1.3 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 ESCCHAR(Manjit S. Gohel et al. 2007) and the EVRA(Manjit S. 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 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, which may be addressed. Decision making also takes into account patient presentation. For example a patient with asymptomatic varicose veins, even with proximal obstruction and superficial reflux, should still only be treated with compression.(Boezem et al. 2011; Wittens et al. 2015)

But there are 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 reducing the venous pressure in 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 superficial reflux. So what is the role of treating the superficial reflux? That's the simple question that was asked by these trials.

The ESCHAR trial recruited between 1999 and 2002, and included 500 patients with healed and open leg ulcers. They were 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.(Manjit S. 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 deal with the underlying problem (usually superficial reflux), the venous ulceration healed significantly quicker.(Manjit S. Gohel et al. 2018)

So putting them together, there is a clear, unequivocal argument for treating the superficial reflux in these patients as quickly as possible.

18.1.3.1 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 destroying a saphenous vein. 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 reduced effectiveness in closing the vein, doesn't really seem to translate into a significant drop in clinical effectiveness after foam sclerotherapy.

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. However, there are a few relative contraindications to GSV ablation.(Atasoy 2015; Gloviczki et al. 2011; Lowell S. Kabinick and Sadek 2019) These include:

- GSV >12mm width raises concern for incomplete obliteration
- Superficial GSV unlikely to be pushed down with tumescence - could cause staining or thermal injury to the skin
- GSV tortuosity may limit the ability to pass a catheter and sufficiently ablate the vein
- Acute superficial vein thrombosis.

Take a Listen

Check out [our episode with Dr. Andrew Meltzer](#) as he discusses some controversies in variations in practice regarding the use of endovenous ablation procedures in the United States.

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, as 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. *Extension of proximal thrombus into the deep system is termed endothermal heat-induced thrombosis (EHIT) and is evaluated and treated aggressively in the United States.*(Sadek et al. 2013; Lowell S. Kabinick et al. 2021) EHIT is defined as:

EHIT Grade	Description	Treatment
1	Extension to SFJ	None
2	Extension into deep system <50% of lumen diameter	Close observation vs anticoagulation
3	Extension into the deep system >50% of lumen diameter	Anticoagulation
4	Occlusion of the femoral or popliteal vein	Anticoagulation

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. Recurrence of varicose veins in the lateral thigh may be related to untreated anterior accessory greater saphenous vein disease.(Laredo, Lee, and Neville 2010)

Foam sclerotherapy has a different mechanism of action. It's a detergent that's injected into the vein that destroys 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.

There are a number of complications related to sclerotherapy that should be understood. Hyperpigmentation is probably the most common complication (11-80%), but only a small percentage persist up to a year (1-2%). Incision and drainage of the thrombus 2-4 weeks after treatment may reduce hyperpigmentation. New appearance of fine red telangiectasias (AKA telangiectatic matting) is related to underlying vessel injury in 5-57% of patients and can persist up to 1 year (1%). Cutaneous necrosis is rare (<1%) and results from extravasation of sclerosing agent, injection into dermal arteriole, reactive vasospasm or excessive cutaneous pressure. DVT and cutaneous nerve injury are also rare.(Bergan, Pasarella, and Mekenas 2006; Munavalli and Weiss 2007)

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. It's a very rare event after superficial venous interventions, but it's potentially catastrophic, with sometimes national press coverage for bad outcomes. What we've got to realize is that the rate of VTE events is very low, there are almost certainly some people that have 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.

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 or HRT a full month before and after the procedure.

For preventing EHIT specifically, the radiofrequency ablation (RFA) catheter should be placed 2cm from the SFJ. EHIT is a primarily technical complication.(Joh et al. 2014)

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 incompetent perforator veins in these patients?

In general, the more advanced the venous disease, the more likely it is that the disease is recurrent, the more likely there is to be deep venous disease or posts thrombotic disease, and the more likely you are to finding incompetent 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.

The clinical criteria most often used to as an indication for treatment is >350ms of deep to superficial reflux, diameter greater than 3.5mm (associated with reflux

in 90% of patients)(Sandri et al. 1999) **and near a healed or active ulcer.**(O'Donnell et al. 2014; Min, Khilnani, and Golia 2003; Rueda et al. 2013)

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 Cocker's perforator lower down on the medial calf. So, I do treat perforators, but almost always it's in recurrent disease with recurrence or deteriorating symptoms.

18.1.3.2 Open Surgery

So we've discussed quite a bit about endovenous intervention. Is there a role 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 treatment if you do it well and if you use modern approaches, such as tumescent anesthesia and 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 move 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. For trainees, it is important to recognize that if you want to change practice, the single biggest driver for change is reimbursement. You can change whatever you like. So, 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.

For deep venous reflux, surgical management follows a relatively complex algorithm.(O'Donnell et al. 2014) Valve prolapse due to vein wall dilation may be treated with external banding. Isolated valve prolapse may be treated with external valvuloplasty, and in severe cases an internal valvuloplasty. When no adequate valve exists, then valve transplant from a distant

source, such as the axilla, may be necessary.(Kabbani et al. 2011) These are not commonly performed procedures and should be referred to centers with significant experience.

18.1.3.3 Medical Management

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'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 processes. 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 role for some venoactive medications such as pentoxifylline —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.

Wound management in patients with venous insufficiency however has many medical adjuncts.Topical agents include excimer (cadexomer) iodine, silver sulfadiazine, silver-containing dressings, growth factors or cell-based therapy.

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.

18.1.4 Other Complications of Superficial Venous Disease

18.1.4.1 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, which can lead to useless courses of antibiotics. Of course, there's no infection, it's a thrombus in a superficial vein. Traditionally, it's just 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 can be a really sinister pathology and associated with VTE. If you 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 going on here. These people likely have an thrombogenic innate tendency that leads to this. That's shown out in the studies, some really good work in some French studies, such as the POST study. 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 remote to the superficial vein thrombosis.(Decousus et al. 2010) 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. A patient with superficial vein thrombus and a previous history of DVT could suggest a subsequent risk of VTE up to 20%.

So in the latest ESVS guidelines, there is quite a lot of content about SVT treatment with algorithms. I would urge people to have a look at that, and it's important to risk stratify people. Limited disease can be followed with ultrasound at 1-2 weeks, but 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.(Kakkos et al. 2021; Di Nisio, Wichers, and Middeldorp 2018; Scovell, Ergul, and Conrad 2018)

NSAIDs and Warm Compresses	Anticoagulation
Low risk Patients	High risk patients
Segment less than 5cm	Segment greater than 5cm
Remote from SFJ	Less than 5cm from SFJ

CALISTO is the largest RCT and used treatment with 2.5mg SC Fondaparinux.(C. S. Group et al. 2010; Blondon et al. 2012) Rivaroxaban did subsequently demonstrate non-inferiority.(Werth et al. 2016) 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 or excised, otherwise they'll recur. So a real change in mindset and level of aggression.(Kakkos et al. 2021; Di Nisio, Wickers, and Middeldorp 2018; Scovell, Ergul, and Conrad 2018)

Pregnant patients are often high risk for lower extremity venous complications and pose difficult clinical challenges. Extensive superficial vein thrombus in pregnant patients should be treated with LMWH and paused at delivery.(Kupelian and Huda 2007; Litzendorf and Satiani 2011) If symptoms persist 3-6mo after delivery, the further workup for pelvic congestive syndrome may be required with a transvaginal ultrasound or cross sectional imaging.(Labropoulos et al. 2017)

18.1.4.2 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, with limited mobility , 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 have 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 for 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.

18.2 Acute 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.

18.2.1 Demographics

18.2.1.1 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.

18.2.1.2 Prevalence

Incidence of DVT increases with age by a factor of 1.9 per 10 years. Of patients who develop DVT, 1% will have phlegmasia and 10% may develop a pulmonary embolism.

For post-thrombotic syndrome, 17% will develop at 1 year and 29% by 8 years after the initial DVT. For recurrence, 30% will develop over 10 years but the highest risk for recurrence is in the first year.(Beckman et al. 2010; C. M. Bulger, Jacobs, and Patel 2004; Meissner et al. 2007)

18.2.1.3 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—a common major, transient, provoking factor is major 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 anticoagulation. 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 this review we are discussing mostly lower extremity DVTs, however catheter-associated DVTs are sometimes encountered on examinations. In general the DVT is considered to be a result of the line. However, management is treatment of the DVT with or without line removal. If the line is functioning well and the patient requires the line for other therapy, then simply treating the DVT with anticoagulation should be sufficient.(Kearon, Akl, Ornelas, Blaivas, Jimenez, Bounameaux, Huisman, King, Morris, Sood, and al. 2016; Kovacs et al. 2007) Treatment is similar in patients with cancer.(Debourdeau et al. 2009)

Prevention is a key component to mitigating the impacts of DVT. One of the more common risk stratification tools in the United States is the Caprini risk assessment model.(Bahl et al. 2010) Patients identified to be high risk according to the Caprini risk index require mechanical prophylaxis, pharmacological prophylaxis and early ambulation.(Gould et al. 2012; Laryea and Champagne 2013)

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, symptom driven age and sex specific screening—PSA, breast assessments, colonoscopy, etc.

One unique situation is recurrent or migratory superficial vein thrombosis, particularly in older patients, can be associated with an underlying malignancy.(Litzendorf and Satiani 2011)

For patients with established diagnosis of thrombophilia (Factor V Leiden homozygous mutation, antithrombin deficiency, prothrombin G20201A homozygous) who become pregnant, recommendations state they should be on LMWH prophylaxis during pregnancy and 6w post-partum regardless of personal history of DVT.(Heit et al. 2005)

18.2.2 Evaluation

What is the algorithm for investigation?

The most important part 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. (Tritschler et al. 2018)

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 effectively 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.

On ultrasound, acute DVT is characterized by an enlarged vein with a non-compressible lumen and hypoechoic thrombus. Chronic post-thrombotic scarring is often contracted with echogenic appearance.(Yusof et al. 2019) For more details, see Section [20.7](#).

18.2.3 Management

18.2.3.1 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 limitations, 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.(Kearon, Akl, Ornelas, Blaivas, Jimenez, Bounameaux, Huisman, King, Morris, Sood, and al. 2016) 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 addition, a recent meta-analysis found that recurrence rates were lower with DOACs, but bleeding rates were no different.(Dong et al. 2019; Rossel et al. 2019)

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)

To step back a little, trainees should make sure they understand some basics behind the coagulation pathway and anticoagulants. Warfarin is one of the most studied and longest used anticoagulants and is a Vitamin K antagonist. Vit K is essential for activation of factors IX, X, VII, prothrombin (II), C and S, which are integral to the extrinsic pathway of coagulation.(Fair, Marlar, and Levin 1986; Wu, Morris, and Stafford 1991) The extrinsic pathway is activated by vessel wall damage and exposure of tissue thromboplastin or tissue factor (TF), which binds Factor VIIa activating Factor X and Xa.(Morrissey 2001)

Warfarin induced skin necrosis is important to understand, although very rare (0.01-0.1%). This occurs shortly after initiating warfarin and presents with purple, cool, painful toes and punctate areas of necrosis and petechiae. Treatment includes immediate cessation of warfarin and transition to LMWH, normalization of INR with Vit K and FFP.(Pourdeyhimi and Bullard 2014; H. A. Tran et al. 2013)

Reversal agents are also another tested topic in the medical management of vascular disease. Other anticoagulants and their reversal agents include:

- Dabigitran - Idarucizumab.(Glund et al. 2019)
- Edoxaban, apixaban and rivaroxaban - andexanet alfa.(Connolly et al. 2019)
- Nonspecific with some effect on other DOACs - PPC, Factor 7, and activated PPC.(Cuker et al. 2019; Kearon, Akl, Ornelas, Blaivas, Jimenez, Bounameaux, Huisman, King, Morris, Sood, and al. 2016)

Finally it is also good to be aware of a few additional coagulation factors and their function:

- Integrin glycoprotein GPIa/IIa - platelet aggregation
- Protease-activated receptors (PARs) - platelet aggregation
- Thrombomodulin - activation of protein C
- Plasminogen activator inhibitor-1 - suppression of plasmin formation/fibrinolysis.

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. A very important study was the IDEAL DVT study(Ten Cate-Hoek et al. 2018), run by 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 mmHg, so that in practice is a class two stocking with a bandage on top. The benefits are that this reduces the early pain and swelling of the DVT, but also interestingly, it reduces the risk of post-thrombotic syndrome and post-thrombotic venous obstruction. 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)

18.2.3.1.1 Complications - Pulmonary Embolism

More and more vascular surgeons are becoming involved with **Pulmonary Embolism Response Teams (PERT)** and so surgeons should be familiar with the acute management of DVTs that progress to pulmonary embolism. The majority of pulmonary embolisms simply require anticoagulation.

However, if a patient becomes hemodynamically unstable, such as develops hypotension, then the patient should undergo systemic thrombolysis.(Kearon, Akl, Ornelas, Blaivas, Jimenez, Bounameaux, Huisman, King, Morris, Sood, and al. 2016) Contraindications to systemic thrombolysis include:

- Age >75
- Intracranial neoplasm
- Intracranial/spinal surgery or trauma with the last 2 months
- History of hemorrhagic stroke
- Active bleeding or known bleeding disorder
- Non-hemorrhagic stroke within the past 3 months.

Patients who do not immediately respond to systemic thrombolysis, are a high risk of bleeding, or in shock that may cause death before thrombolytics take effect, then catheter based thrombus removal may be considered. This complex decision making is best made through the previously mentioned PERT.(Eleftherios S. Xenos et al. 2019) Endovascular techniques include:

- Thrombus fragmentation
- Suction thrombectomy
- Rotational embolectomy
- Ultrasound assisted thrombolysis

There are very high risk procedures and complications include pulmonary artery perforation, pericardial tamponade, cardiogenic shock, hemoglobinuria or other hemorrhagic complications.

18.2.3.1.2 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 summery of symptoms for PTS, seen in up to 50% but probably closer to 25% of proximal DVT.

18.2.3.1.3 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. The most important factor in successful IVC filter retrieval is dwell time less than 7 months. Type of filter, tilting or protruding struts are not associated with failure. Adverse events have found to be as high as 2%. (Desai et al. 2017) 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.

A rather challenging and unique patient would be a pregnant patient with a DVT and placenta previa. Anticoagulation is contraindicated in placenta previa and in addition, an infrarenal IVC filter is also contraindicated, so she would require a suprarenal IVC filter.(Bates et al. 2018; DeYoung and Minocha 2016; Harris, Velineni, and Davies 2016)

In the setting of a thrombosed filter, a patient may require urgent intervention if they develop phlegmasia. Suprarenal filter placement may be useful during the procedure to prevent embolization. Removal of the IVC filter is contraindicated unless all clot is cleared.(Sheth et al. 2015; Teter et al. 2019)

18.2.3.2 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 weren't candidates for thrombolysis. However, if 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)

18.2.3.3 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 and long-term surveillance, et cetera. So I think the jury is very much out. 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 its severely symptomatic, the studies that actually you 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 initial conservative management, compression with 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 patients 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 rethrombose. 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.(W. Wang et al. 2018; Meissner et al. 2012; Rollo et al. 2017) 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.

18.3 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 of 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. Presentation often includes iliofemoral DVT with non-palpable pulses and

loss of sensation with out improvement on anticoagulation and elevation. Endovascular early thrombus removal is the first line with fasciotomies, with open thrombectomy as back up for salvage.(Erdoes et al. 2011; Thomas, Hollingsworth, and Mofidi 2019)

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.

18.4 Deep Venous Obstruction

18.4.1 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 issue causing the consequences. As opposed to occlusion, which is an anatomical term. So venous outflow obstruction, 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 proportional to 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 venous compliance. It's not the same compliant vein that can respond to demands and physiological need that you have, in a primary non-scarred 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 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.(Nazzal et al. 2015) 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.(Liddell and Evans 2018; Rollo et al. 2017)

The other big group of patients of course, is malignant lymph node or retroperitoneal fibrosis related venous outflow obstruction. For example, a patient with a large retroperitoneal leiomyosarcoma may be involving the vena cava. In this case, these patients may require en bloc resection of the IVC and reconstruction with ringed PTFE, sized off the preoperative imaging.(Quinones-Baldrich et al. 2012; Fiore et al. 2012) But in the end, post-thrombotic by far was the most commonly diagnosed pathology.

18.4.2 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 Section [20.7](#).

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 reflux.(Manjit S. Gohel et al. 2007; Manjit S. Gohel et al. 2018) So again, the key message is treat the superficial reflux if it's there. However, if the patient has isolated deep reflux then management is limited to wound care and compression, often as high as 40-50mmHg. Some series have found that 70-80% will heal.(O'Donnell et al. 2014; Maleti and Perrin 2011)

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 clear signs and symptoms of major venous outflow obstruction, particularly venous claudication, history of previous DVT, really disproportionate venous changes to the superficial and the infrainguinal imaging. Proximal obstruction can present with atypical proximal varicose veins in the mons pubis, perineum, buttock, high posterior/lateral thigh, abdominal wall. These findings are 93% predictive of deep venous obstruction proximal to the inguinal ligament.(Kurstjens et al. 2016) 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 is a 9F 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. ***Recommended oversizing for vein stenting is 10-15% of largest diameter of adjacent vein.***(Gloviczki et al. 2011; O'Donnell et al. 2014; Raju, Owen, and Neglen 2002; Raju, Darcey, and Neglén 2010)

In terms of when you use it, in some parts of the world it's commonly used for diagnostic purposes, but beware of the risk of over-diagnosis. More commonly, almost routinely, IVUS

is used for therapeutic, particularly when we're stenting deep veins to plan our landing zones. When CTA/MRA shows compression, it must be confirmed with IVUS prior to treatment. IVUS has far greater sensitivity and specificity and must show a cross-sectional surface area reduction of greater than 50%.(Gagne et al. 2018; Forauer et al. 2002)

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.

18.4.3 Management

18.4.3.1 Endovascular

There is variability in the availability of stents for venous obstruction. However, closed cell braided stents (i.e. the Wallstent) have been around for many years and has the most data related to long term durability.(Gagne et al. 2019) These stents have the least radial force at the ends, so it is recommended to extent 1-2cm beyond the venous obstruction. In cases of proximal iliac vein obstruction, this may require extending into the IVC 1-2cm which may cross, or "jail" the contralateral iliac vein. When jailed, the rate of DVT on that side has been reported as high as 10%.(E. H. Murphy et al. 2017; Le et al. 2018)

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 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, Ten Cate, and Ten Cate-Hoek 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 in 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.

 Take a Listen

Check out [our episode with Drs. David Gordon and David Williams](#) on a recent publication looking at the evolution of in stent restenosis and the classification of thrombus forming in venous stents.

18.4.3.2 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, Porembeskaya, and Geroulakos 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.

18.5 Pelvic Congestive Syndrome

18.5.1 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. Knowing the proximal anatomy of the saphenous vein tributaries is important, as the pudendal vein tributary connects vulvar veins and inferior gluteal vein connects to the posterior buttock.(Iafrati and Donnell 2019) 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.

18.5.2 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 similar to that of 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 as 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.

18.6 Superior Vena Cava (SVC) Syndrome

SVC syndrome is an acute presentation of head and neck venous congestion often due to SVC obstruction. This can be intrinsic due to indwelling devices or extrinsic due to malignancy or mediastinal fibrosis (half of benign cases).(Kalra, Bjarnason, and Gloviczki 2019; Rice, Rodriguez, and Light 2006; Parish et al. 1981)

18.6.1 Management

Angioplasty and stenting are first line treatment for benign etiology.(Rizvi et al. 2008; Sheikh et al. 2005) Angioplasty alone may be sufficient, but stenosis refractory to angioplasty may require stenting. SVC syndrome with failed endovascular management in reasonable operative candidates can undergo open reconstruction with large diameter prosthetic graft or spiral vein graft.(Doty, Flores, and Doty 1999) However, restenosis in open repair can be as high as 15%.(Kalra, Sen, and Gloviczki 2018; Sfyroeras et al. 2017)

SVC syndrome secondary to malignancy, bronchogenic or lymphoma, is often most effectively treated with palliative radiotherapy with good response.(Talapatra et al. 2016) Endovascular or open recannalization is often reserved only for those with severe symptoms refractory to radiation or chemotherapy.(Higdon and Higdon 2006) Lymphedema pumps can help with symptom management as well.

18.7 Lymphedema

Here we will discuss lymphedema of the lower extremity. This is a broad topic, but the management decisions are relatively straightforward and knowledge is required for examinations. For upper extremity lymphedema, see Section [3.8](#)

18.7.1 Demographics

18.7.1.1 Etiology

Lymphedema can be thought of as a primary dysfunction of the lymphatic channels or the secondary result of a disease that results in the destruction of lymphatic flow. Causes of secondary lymphedema include filariasis, which is endemic to parts of South America.(Pfarr et al. 2009; Shenoy 2008)

18.7.1.2 Presentation

The management of lymphedema is determined primarily on its clinical stage at presentation.(Grada and Phillips 2017) The staging for lymphedema is as follows:

Lymphedema Stage	Description	Symptoms
0	Sub-clinical	No swelling, heaviness or discomfort
1	Spontaneous Reversible	Swelling improved with limb elevation

Lymphedema Stage	Description	Symptoms
2	Spontaneous Irreversible	Pitting and swelling, not improved with elevation
3	Lymphostatic elephantiasis	Skin hardening, non-pitting edema, verrucas changes, recurrent infection

18.7.2 Evaluation

On physical exam, a classic finding is Stemmer's sign, which is a thickened skin fold at the base of the second toe/finger. The sign is positive when the tissue is hardened and cannot be lifted.(S. G. Rockson et al. 1998; Stanley G. Rockson 2019)

Lymphedema is most often diagnosed based on clinical presentation and physical exam. Lymphoscintigraphy can be used to confirm the diagnosis in complex presentations and would demonstrate dermal back flow, absent or delayed transport, crossover filling, absent or delayed visualization of lymph beds.(Stanley G. Rockson 2019; O'Donnell, Rasmussen, and Sevick-Muraca 2017)

18.7.3 Management

Treatment is best initially managed with combined decongestive therapy (CDT) which includes an intensive reductive phase (4-8wks), followed by life long maintenance, often with specific compression garments.(Grada and Phillips 2017) CDT in the setting of heart failure has been reported to lead to volume overload.(Lawenda, Mondry, and Johnstone 2009)

In extreme cases, after CDT therapy has been instituted for at least 6 months and the patient has persistent refractory symptoms, surgery may be used for reconstruction or debulking.(Grada and Phillips 2017; Baumeister et al. 2016)

19 Trauma - Peripheral

Authors: *Kevin Kniery, Jason Bingham, Nakia Sarad, and Todd Rasmussen*

These trauma episodes were developed in collaboration with [Behind the Knife: The Premier Surgery Podcast](#).

DO NOT GET DISTRACTED

- These patients are often poly-trauma patients with likely other injuries
- Follow ABCDE 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

Vascular trauma requires close collaboration between vascular surgeons and trauma surgeons. The decision of which specialty should manage which injuries varies across different centers. Through these trauma chapters we will discuss what management decisions the majority of vascular surgeons should be comfortable managing as a part of a multidisciplinary trauma team.

Take a Listen

Check our [debate between leading trauma and vascular surgeons](#) about how to best develop a collaborative team to manage vascular trauma.

For relevant images and a more in depth discussion of this topic, please review **Chapter 21: Upper Extremity and Junctional Zone Injuries**(Vuoncino, White, and Clouse 2022) and **Chapter 22: Lower Extremity Injuries**(David S. Kauvar and Propper 2022) in Dr. Rasmussen's 4th Edition of *Rich's Vascular Trauma*.

19.1 Demographics

19.1.1 Epidemiology

Penetrating extremity trauma is leading cause of peripheral vascular injuries (75-80%)

Most common arterial injury found in femoral or popliteal arteries (50-60%), then brachial artery (30%)

19.1.2 Types of Injuries

Can occur with either blunt or penetrating trauma

1. Complete Wall Defect (hemorrhage or pseudoaneurysm)
2. Intimal Injury (subintimal/intramural hematomas, flaps, disruptions)
3. Arteriovenous fistulas
4. Complete Transection (with hemorrhage or occlusion)
5. Spasm

19.1.3 Presentation

Hard Signs of Peripheral Vascular Injury

- Active Hemorrhage
 - Obvious bleeding (arterial or venous)
 - Can be pulsatile or history of bleeding in field
- Profound Ischemia of the extremity
 - Determined by absence of palpable pulse or dopplerable signal beyond the area of injury
- Expanding hematoma
 - Can occur underneath a closed wound/injury
- Audible Bruit or Palpable thrill near site of injury
 - Concern for an arterial-venous fistula, often high flow

Soft Signs of Peripheral Vascular Injury

- Subjective reduced or unequal pulses

- Diminished blood flow to extremity
- Weak, but audible arterial doppler signal
- Large non-pulsatile hematoma
- Orthopedic injury patterns carrying a high index of suspicion of vascular injury
 - Examples - posterior knee dislocation, displaced long bone fractures
- 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 (E. M. Bulger et al. 2014)

19.2 Evaluation

Continuous Wave Doppler

- Handheld device that can amplify arterial signal to measure quality of pulse
 - Triphasic, biphasic, monophasic, or absent
- **Injured Extremity Index**(Vuoncino, White, and Clouse 2022)
 - Utilizes continuous wave doppler with manual blood pressure cuff to obtain objective measurements of perfusion

- On injured extremity, inflate cuff proximal to arterial signal and measure pressure at which arterial signal diminishes. Compare to a normal/non-injured extremity. Injury extremity index is the ratio between these two values.
 - **Injured Extremity Index > 0.9**, no flow limiting vascular injury - Recommend observation
 - **Injured Extremity Index <0.9**, flow limiting vascular injury likely - Recommend further imaging with CTA
- If patient in shock and cold, repeat injury extremity index as the patient is resuscitated and warmed

CT angiography (CTA)

- Gold standard for evaluation of extremity vascular injury
- Ensure appropriate timing of contrast to follow-through to distal extremity
 - Can occur in conjunction with CT imaging of head/chest/abdomen/other extremities to evaluate for traumatic injury

Invasive angiography

- Has been readily replaced by CTA for evaluation of extremity vascular injury

19.3 Management

If patient has hard sign of vascular injury, OR for exploration and vascular repair/bypass

If patient has suspicion of vascular injury (soft signs), recommend bedside injury extremity index.

If injured extremity index > 0.9 , recommend observation.

If injured extremity index < 0.9 , recommend further imaging with CTA or angiography (if patient stable). If CTA positive for occlusion or extravasation, OR for operative repair.

19.3.1 General Considerations

Exposure Basics

- Prep patient widely
 - Any lower extremity injury, prep patient from umbilicus down to both legs
 - Anticipate need for proximal control, intraoperative angiograms at the femoral level, and potential harvesting of saphenous vein for conduit
- Recommend leaving tourniquet on and prep into field
 - Removing tourniquet prematurely can result in arterial bleeding, leading to hypotension, hemorrhagic shock, and multi-organ system failure
 - Communicate with anesthesia and OR team when tourniquet will be put down and have blood ready for transfusion
- Incision
 - Recommend making incision twice as long as what you would initially anticipate
 - Important to have long incision to get appropriate control and visualization of injury
- Junctional Hemorrhages
 - Junctional area between the torso and extremity, most difficult to control
 - Lower extremity vessels include distal external iliac and common femoral
 - Commonly presents as a groin hematoma
 - Upper extremity vessels include subclavian and axillary

Take a Look

You can review surgical exposure of lower extremity arteries [here](#).

You can review surgical exposure of upper extremity arteries [here](#).

Repair

- Gain proximal and distal control
- Debride injury back to healthy tissue
- Perform thrombectomy
 - Both proximal and distal to area of injury using Fogarty catheter to completely clear the inflow/outflow thrombus

- Once thrombus burden removed, apply 40-60cc of regional heparinized saline down the outflow before applying the clamp. Perform again on the inflow.
- Types
 - Primary Repair
 - Good for focal injuries (grazing wounds or stab wounds from a knife) where there is minimal tension and arterial lumen maintains its integrity after debridement to healthy tissue
 - Can consider with venous injuries due to higher compliance or when vessels are redundant (ie brachial artery)
 - Patch Repair
 - Also good for focal injuries not amenable to primary repair
 - Again, can perform if there is no concern for tension or compromise of arterial lumen
 - Interposition graft
 - Most common repair type, avoids issue of tension and compromise of arterial lumen
 - Conduit types
 - Autologous vein (great saphenous vein)
 - Preferred choice with concern for contamination and for more distal extremity injuries (i.e. brachial, radial, SFA, and popliteal)
 - Saphenous vein from contralateral extremity (non-injured) routinely recommended
 - Ipsilateral saphenous vein CAN be used if there is no deep vein injury and it is part of the incision/exposure
 - Prosthetic - ePTFE or Dacron
 - Suitable if vein is not available, wound with minimal contamination or injury is more proximal (axillary, subclavian, proximal femoral, iliac)
 - Ligation
 - Dependent on location of injury and overall hemodynamic status of patient
 - If patient unstable, ligate as damage control maneuver (Liang et al. 2016; Fox et al. 2012)

Take a Listen

Check out our [Vascular Origins Story outlining the history of arterial injury management in military medicine](#) and the transition from ligation to arterial repair during the Korean war.

Heparinization

- Recommend selective heparinization
- If patient has isolated extremity injury, recommend systemic heparin during revascularization
- If patient has concomitant injuries (i.e. head trauma, torso bleeding, or extensive soft tissue injury), recommend regional heparin flushed up and down injured vessel (Liang et al. 2016; Fox et al. 2012)

Shunts

- Can be utilized in damage control maneuvers to prevent need for ligation and achieve temporary perfusion
- Larger caliber vessels with high flow can be shunted without need for heparinization for 4-6 hours
- Smaller caliber vessels (in distal extremities) usually clot off but do not cause harm when clot occurs intra-operatively as the vessel needs to be re-assessed for repair
 - However, recommend that shunts are used in patients that do not have contraindications to systemic heparin use
- Types
 - Javid
 - Sundt
 - Argyle
 - Has advantage of multiple sizes
 - Pruitt-Inahara
 - Makeshift shunts – small caliber chest tubes
 - Can place in injured vessel proximally and controlled with either ligature around vessel to lock shunt in place or rubber vessel loop
- Placement

- Insert in injured vessel proximally first, then allow some pulsatile bleeding to allow any thrombus to exit before inserting it distally
- Shunts can be secured either with heavy silk tie or plastic vessel loops
- Can use doppler with water or acoustic gel over shunt to verify arterial flow
- [More information about the types and use of intravascular shunts can be found here.](#)

Fasciotomies

- Avoid hesitation to perform 4-compartment fasciotomies in threatened limb ischemia
- Recommend prophylactic fasciotomy to avoid compartment syndrome
- Especially important when transporting patients to higher level of care facilities
- Two-incision Four Compartment Lower Extremity Fasciotomy can reviewed here (Bowyer 2015)
 - [Reference points for lateral incision here.](#)
 - [Reference points for medial incision here.](#)
- [Comprehensive review of fasciotomy techniques in both upper and lower extremities can be reviewed here.](#)

19.3.2 Iliac Vessels

Exposure

- Retroperitoneal exposure preferred - oblique incision in lower quadrant of abdomen (i.e. transplant incision)
- Midline laparotomy can also be considered
- Groin hematomas can either be iliac or femoral etiology, recommend extensive prep with either transplant incision or midline laparotomy incision to be able to gain common iliac artery control if needed

Repair

- Internal iliac injury
 - Prefer ligation
 - Difficult access for exposure and repair

- Patients usually in hemorrhagic shock and control is time-consuming and difficult to achieve
- Can be safely achieved, pelvic ischemia rare due to collateral blood supply from contralateral iliac vessel
- If repair can be performed, recommend larger needle (SH blunt tip needle on 3-0 or 4-0) for repair due to low visibility in pelvis
- External iliac injury
 - Repair at all costs
 - Gain control first then decide on method of repair depending on patient stability (immediate repair or temporary repair with vascular shunt)
 - 14 French Argyle vascular shunts can be used on iliac vessels
 - May need to use prosthetic interposition graft due to larger caliber of vessels
- Ligation has high morbidity and mortality, should be avoided
 - If external iliac artery is ligated, patient will experience proximal limb ischemia and will either need an above knee amputation (AKA) or hip disarticulation
 - In life or death situations, ligation and subsequent amputation is inevitable to save a patient's life
 - Can consider temporary ligation with return to OR in 2-3 hours to re-establish inline flow (consider cross femoral graft) when patient is more resuscitated, however, not common in trauma situation

19.3.3 Femoral Vessels

Includes the Common Femoral Artery (CFA), Superficial Femoral Artery (SFA), and the Deep Femoral (Profunda femoris)

Exposure

- Proximal CFA exposure similar to exposure of external iliac (midline laparotomy or transplant incision)
- Distal CFA, SFA, and Deep Femoral can be exposed with vertical groin incision (below inguinal ligament midway between pubis and ASIS over anterior proximal thigh) or oblique groin incision (directly beneath inguinal ligament or few centimeters distal)

Repair

- Gain control

- May be difficult to only use vessel clamps for more proximal injuries
- At common femoral junction, can use Fogarty catheter into orifice of deep femoral with balloon inflated to control back bleeding
- If SFA injured, can clamp distal CFA for control
- Recommend shunt placement
- Repair with primary, patch, or interposition graft (autologous vein preferred)
- Ligation also has high morbidity and mortality, should be avoided (see iliac section for consequences of ligation and options of shunting) (Abou Ali et al. 2017; Subramanian et al. 2008)

19.3.4 Popliteal Artery

Exposure

- Need to gain control of popliteal artery above and below knee
 - Above knee popliteal artery is a continuation of the distal SFA through the adductor magnus (Hunter's canal)
- Position patient in "frog-leg" with bump of rolled towels placed underneath calf
- Make medial incision from above the knee to mid-thigh
 - Locate the distal SFA just at or beyond Hunter's canal in order to locate the above knee popliteal artery
- Move bump to above the knee and make a medial incision from below knee to mid calf
 - Expose the gastrocnemius and soleus muscles and bring down to the inferior edge of the tibia to open the below the knee popliteal space
- Retraction and good lighting is key
 - Can use Weitlaner retractor, Pilling retractor, or Henly popliteal retractor to retract muscles and better visualize space
 - Recommend narrow handheld retractor (appendiceal or Wylie renal vein retractor) on either the superior or inferior aspect of below or above knee incision, respectively, to gain more exposure of the popliteal space

Repair

- Recommend bypass repair (autologous vein preferred if available)

- Inflow from distal SFA to distal below knee popliteal target
- Always check repair with intraoperative doppler and check distal flow to foot
 - If signal goes down and you are concerned about the technical repair, be prepared to re-open and pass thrombectomy catheters proximally and distally to evaluate issue
- Even with good technical repair, spasm may occur
 - Common among young patients
 - Signal usually improves after re-warming and resuscitation
 - Ensure that there is audible flow in the bypass and patient well resuscitated prior to re-opening injury to assess repair
 - On-table angiogram also an option, however, spasm may appear as occlusion and repair is unnecessarily re-opened
 - **Important to note: Water hammer signal is not appropriate audible flow as it indicates diastolic flow in the arterial signal**

 Take a Listen

Check out [our discussion with Mr. Paul Blair](#) where he discusses management of peripheral arterial trauma during the troubles in Northern Ireland. This episode was developed in collaboration with the [Rouleaux Club](#).

19.3.5 Tibial Vessels

Repair is selective on extent of injury

Recommend repair if all three tibial vessels are injured and there is no flow to the foot (anterior tibial, posterior tibial, and peroneal arteries) (Burkhardt et al. 2010)

- Saphenous vein graft preferred

Recommend ligation if one or two tibial vessels injured, as there is redundant arterial flow to the lower extremity

 Take a Look

[Find additional resources for lower artery exposure here.](#))

19.3.6 Subclavian and Proximal Axillary Arteries

Important to keep in mind, upper extremity disability can have a significant impact on quality of life.

Long term functional impairment is measured by the Disabilities of Arm, Shoulder, Hand (DASH) questionnaire. Impairment most closely associated with duration and severity of ischemia and concomitant nerve injury (36%). Open reconstruction is preferred and patency rates are high.(Frech et al. 2016)

Due to protected anatomic location of subclavian vessels, most subclavian artery trauma is usually caused by penetrating mechanism.

Considered a junctional region for hemorrhage with difficult access for exposure

Endovascular approach with covered stents may be used in traumatic axillo-subclavian injuries that are stable enough for intervention (i.e. do not require a resuscitative thoracotomy) (Eleftherios S. Xenos et al. 2003; Branco et al. 2016)

Exposure

- Requires thoracotomy or sternotomy associated with clavicular incision for access
- Right subclavian/axillary arteries
 - Median sternotomy with extension to supraclavicular incision
 - Divide sternocleidomastoid muscle 1 cm above clavicle to expose subclavian vein
 - Retract vein and divide anterior scalene muscle to expose subclavian artery
 - Careful to locate and avoid phrenic nerve (traverse laterally over anterior scalene)
 - Aberrant right subclavian artery directly arises from thoracic aorta (0.5-2%) of population
- Left subclavian/axillary arteries
 - Left posterolateral thoracotomy is most optimal, however, requires lateral recumbent positioning
 - If patient is requiring midline laparotomy for operative management of abdominal trauma, recommend anterolateral thoracotomy in 3rd or 4th intercostal space
 - Extension with supraclavicular incision or median sternotomy to better expose proximal subclavian
 - Extension with infraclavicular incision to deltopectoral groove to gain access to proximal axillary artery

Repair

- Primary, patch repair, interposition graft, or bypass dependent on extent of injury after debridement to healthy tissue and follows tenets of avoiding tension and preserving arterial lumen integrity

19.3.7 Distal Axillary and Brachial Arteries

Brachial artery is continuation of distal axillary artery

Repair

- Primary Repair can be considered in brachial artery if there is redundancy, lack of tension, injury is focal, and no compromise to arterial lumen.
- Great saphenous vein preferred over prosthetic for conduit.
- Ligation should be avoided as upper extremity limb ischemia is highly disabling and morbid

19.3.8 Radial and Ulnar Artery

Repair

- Ligation can be considered if there is appropriate collateralization from either the radial or ulnar artery into the distal palmar arch
- If either radial or ulnar injury causes distal hand ischemia, should be repaired

19.4 Venous Injury

Venous injuries can be selectively repaired. More proximal venous injuries should be repaired, while distal injuries can be ligated.

Primary repair with lateral venorrhaphy can be performed due to higher intrinsic compliance of veins.

19.4.1 Lower Extremity Venous Injury

Proximal Vessels (popliteal and femoral veins)

- Ligate if patient unstable, acceptable as damage-control maneuver
- Repair if patient is stable and not in profound shock
 - Can consider venous shunts as well
- Studies confirm that venous repair improves arterial flow in the arterial repair since venous outflow is not impeded (Quan et al. 2008; Clouse et al. 2007; Todd E. Rasmussen and Tai 2022)
- Even if repair of injured venous vessels thrombose over time, thrombosis occurs slowly and does not lead to pulmonary emboli

19.4.2 Upper Extremity Venous Injury

All EXCEPT proximal axillary and subclavian vein injuries can be ligated in the upper extremity (ie distal axillary, basilic, cephalic, brachial, veins).

Repair can be performed primarily or with graft (autologous or synthetic).

Part VI

General

20 Vascular Lab

Authors: Alaska Pendleton and Anahita Dua

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.

20.1 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.

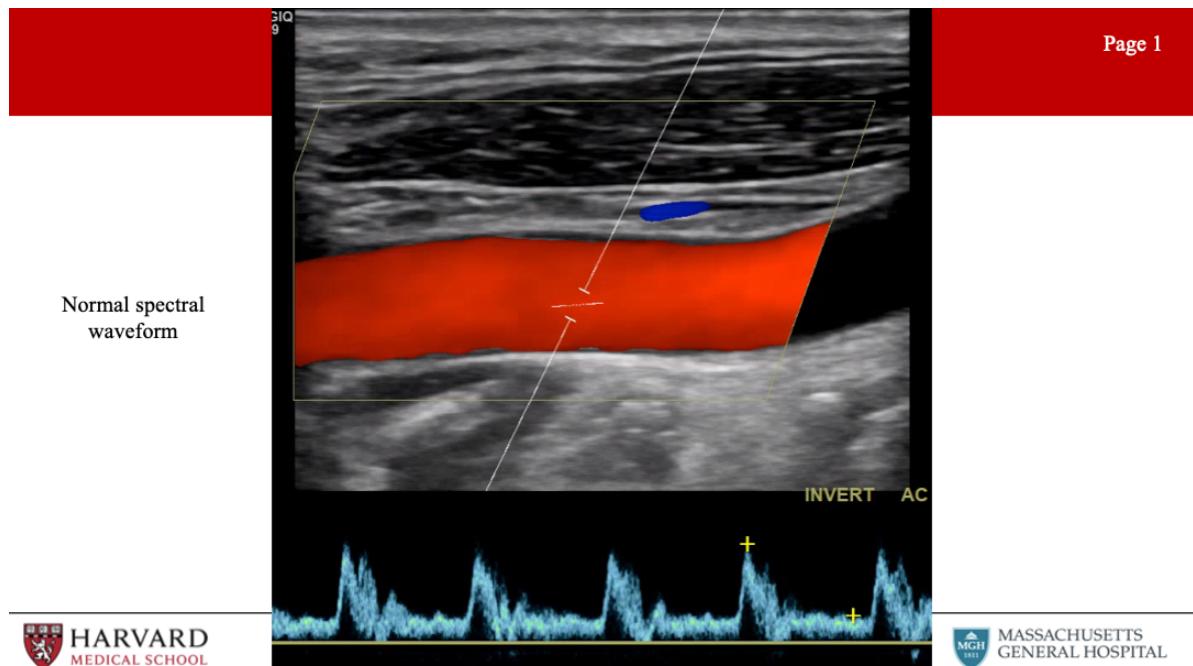
Color Doppler presents velocity information as a color-coded overlay on top of a B-mode image. Duplex ultrasonography is a term commonly used for the simultaneous presentation of B-mode and doppler data. Of note, when looking at a still image of a color flow duplex, the color may be misleading for direction because it may not be noted at which part of the cardiac cycle the picture was taken. It is far more reliable to look at the spectral doppler waveform.

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)

20.1.1 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)



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.



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.

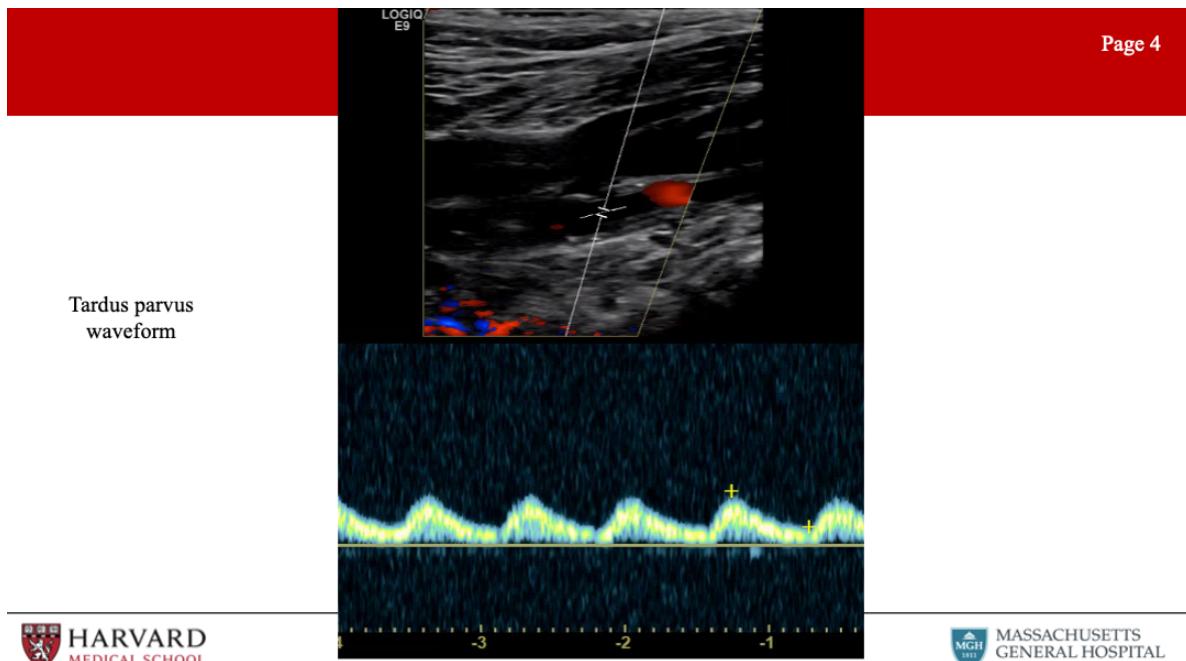


How does flow-limiting stenosis change the waveform?

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)



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)



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 - Vr >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 - Vr > 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.(Muella 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.

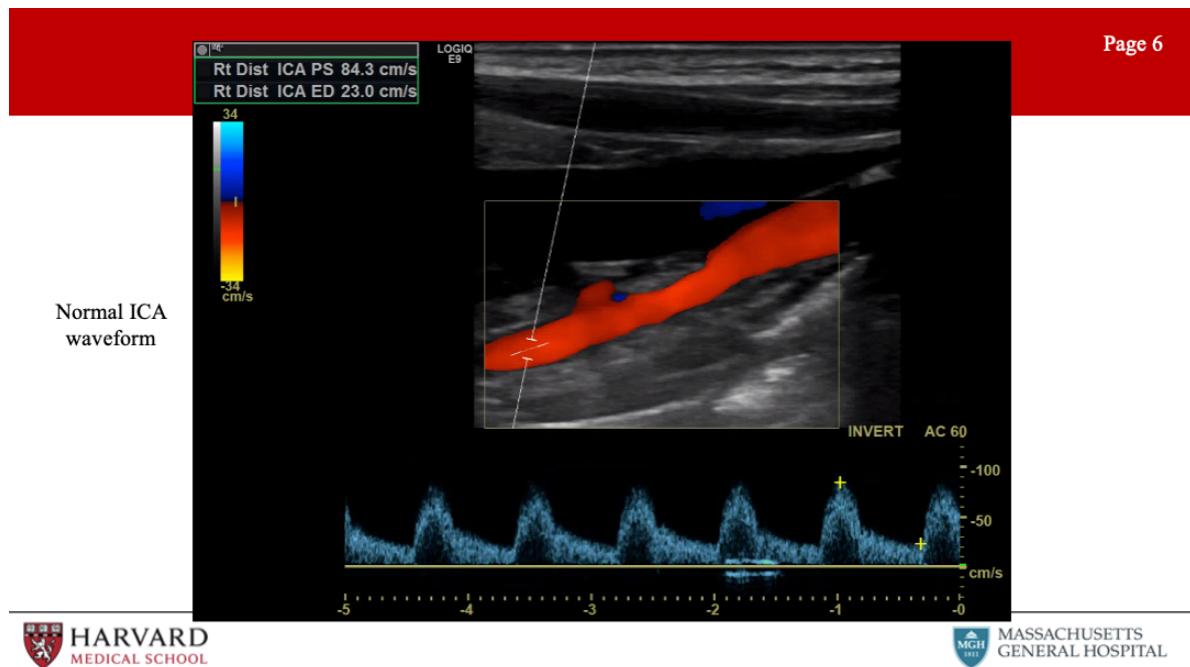
20.2 Extracranial

What does a typical extracranial evaluation involve?

Examine CCA (2 views), ICA (2 views), ECA, vertebral arteries

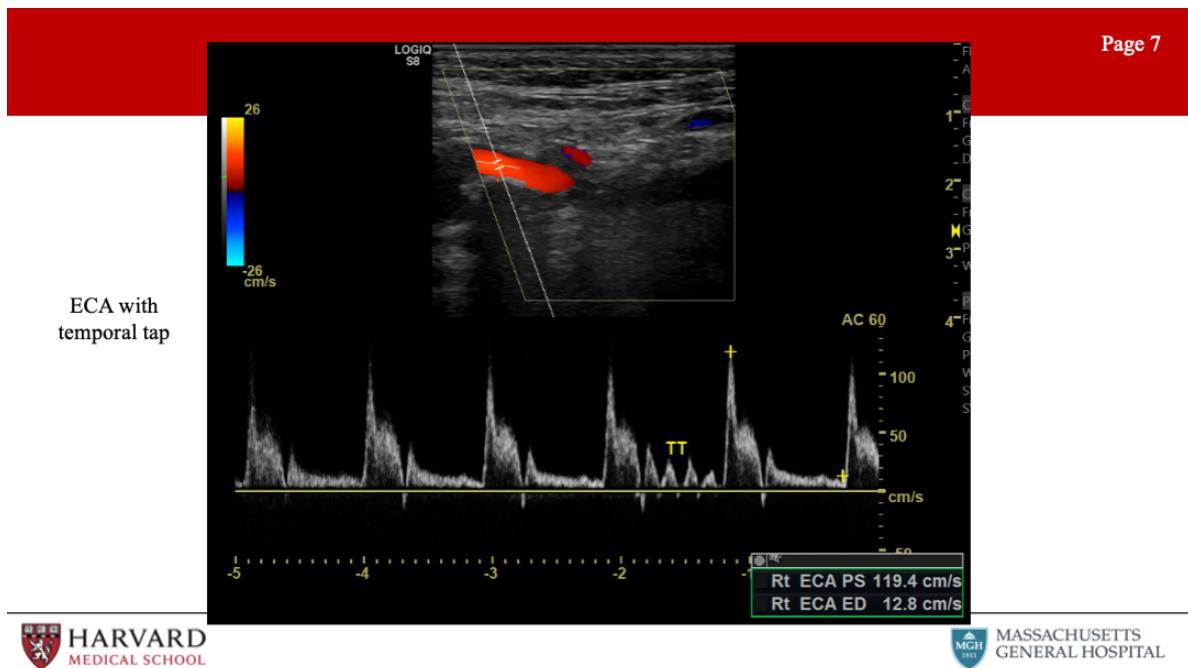
What are normal ICA and ECA waveforms?

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.



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.

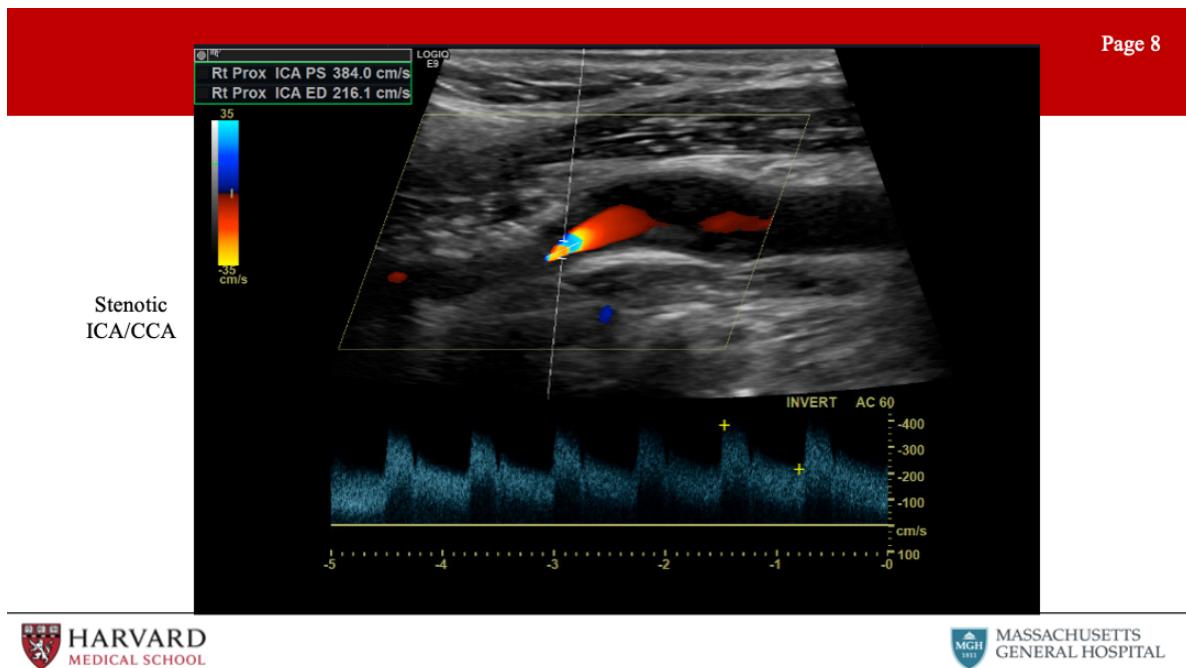
Another way of differentiating the external and internal carotid arteries is the “temporal tap”. Tapping on the superficial temporal artery (a branch of the ECA) will be transmitted as small pulsations in the diastolic component of the external carotid artery.(Pellerito and Polak 2019; Size et al. 2013)



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 ≥ 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.



When is surgery indicated for ICA stenosis?

Parameters for when surgery indicated:

Asymptomatic Carotid Atherosclerosis Study (ACAS) >60% stenosis asymptomatic, NASCET >50% stenosis symptomatic

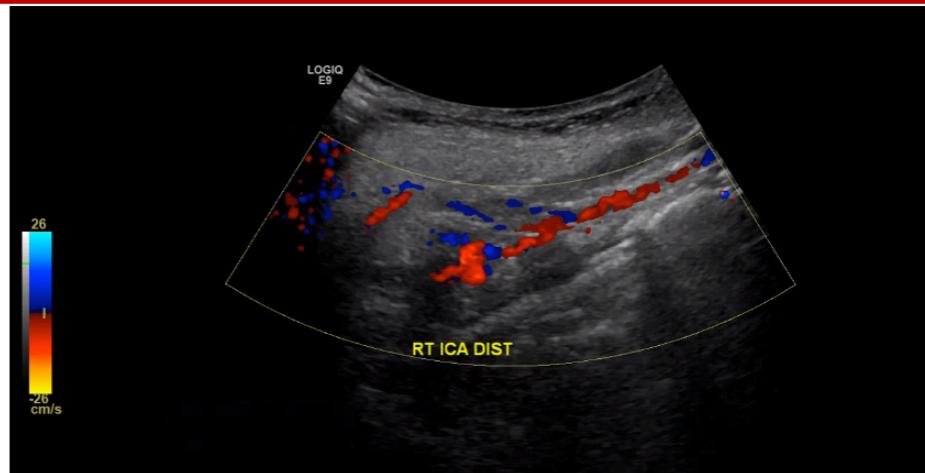
For more see Chapter 1.

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.(Jeffrey W. 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?

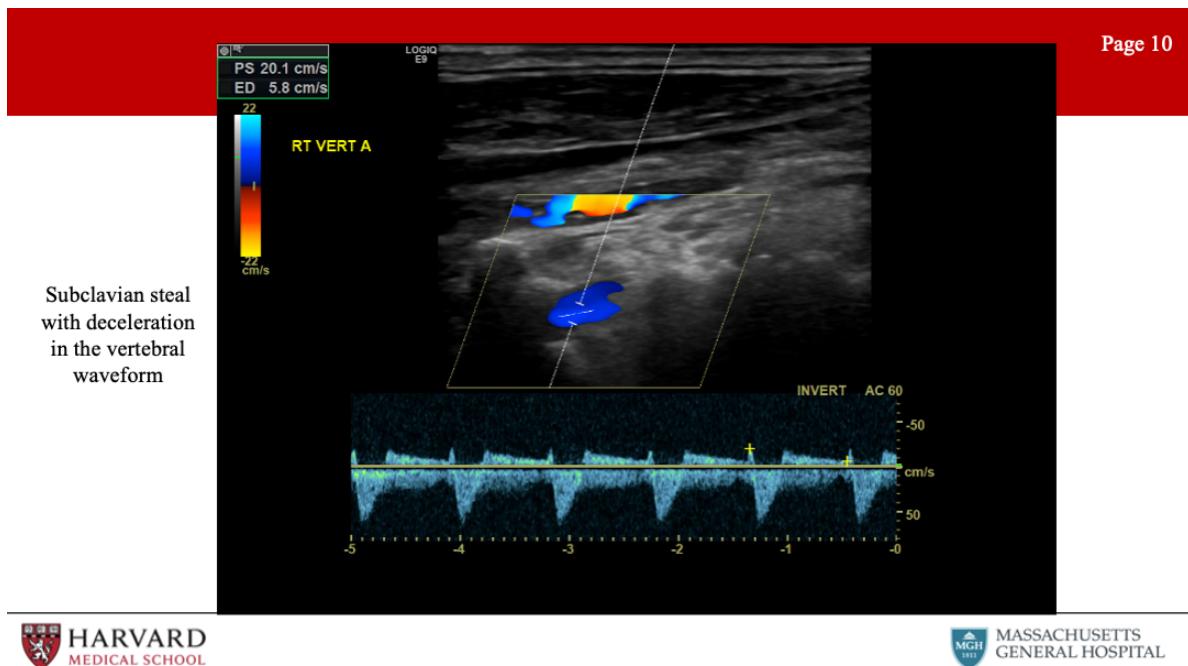
Aspirin if asymptomatic, POBA if symptomatic.

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)



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; W. A. Schmidt 2014)

20.3 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 - Arteries differentiated by depth. MCA 3-6 cm, everything else deeper. Three primary 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 (Foraminal) view:** Basilar and vertebral arteries (both away).
- **Orbital view:** Ophthalmic and ICA

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. 2007a)
 - 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.(M. 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.

20.4 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 <70 mmHg is abnormal and <60 mmHg 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)

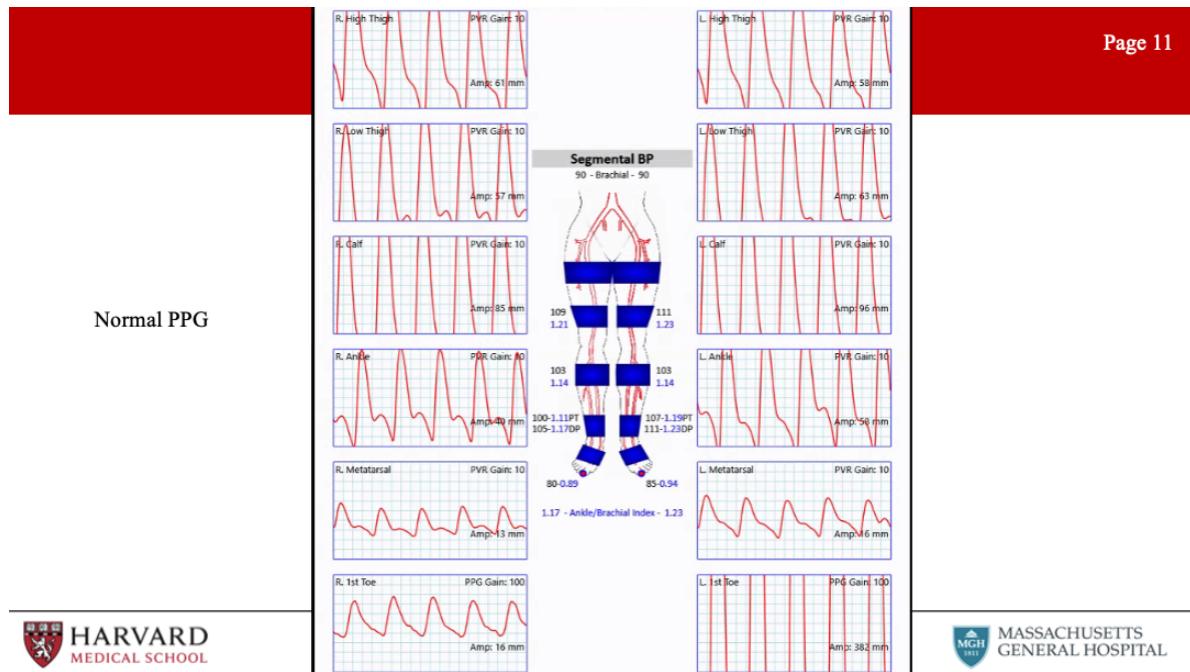
Let's talk about some of the most frequently tested pathologies, starting with arterial TOS.

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)



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 (Buerger's Disease): 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.

20.4.1 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 non-invasive 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 venous outflow stenosis or arterial inflow stenosis.

Loss of phasic variation in the upper extremity veins may indicate central stenosis and that extremity may not be appropriate for access creation.(Y. C. Kim et al. 2009; Lok et al. 2020) 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) To determine the volume flow (VF) the sample volume (SV) should be opened wall to wall in a segment of fistula that is straight with a constant diameter and include all velocities present within the fistula. Angle correction should be less than 60 degrees.(Gerrickens et al. 2018; 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, Gustavson, and Schanzer 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.(Dennis F. 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 Section [5.3.3](#)

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 represented 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.(Dennis F. Bandyk 2013)

20.5 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.

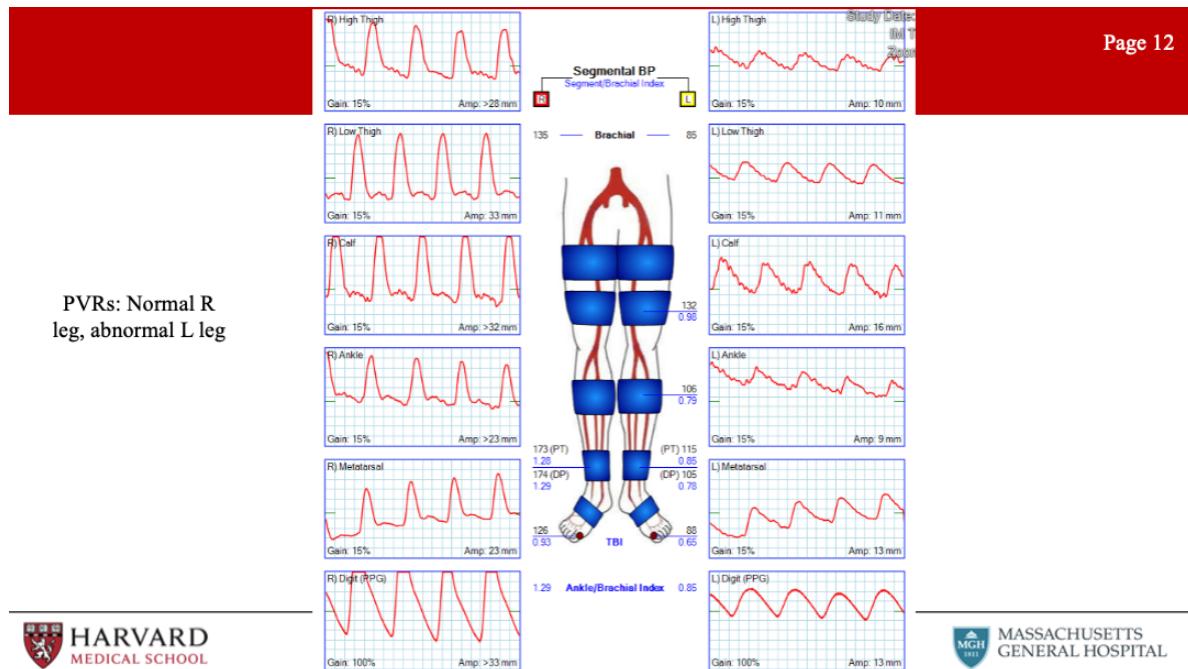
The ankle-brachial index in diabetic patients is frequently unreliable due to incompressibility of the tibial vessels at the level of the cuff secondary to calcification. Consequently, toe pressures are mandatory in all patients with diabetes mellitus. Normal TBI>0.7.

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 representative of central blood pressure.(A. Chen, Prout, and Jain 2014)

TcPO₂: When there is significant tissue loss, preventing TBI measurement, another option is transcutaneous oximetry (TcPO₂). (Mills et al. 2014b) Transcutaneous oximetry is a non-invasive method of measuring the tissue partial pressure of oxygen through a heated sensor on the skin. A TcPO₂ 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.



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, TcPO₂ <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. (Dennis F. 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, Hykes, and Starchman 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)

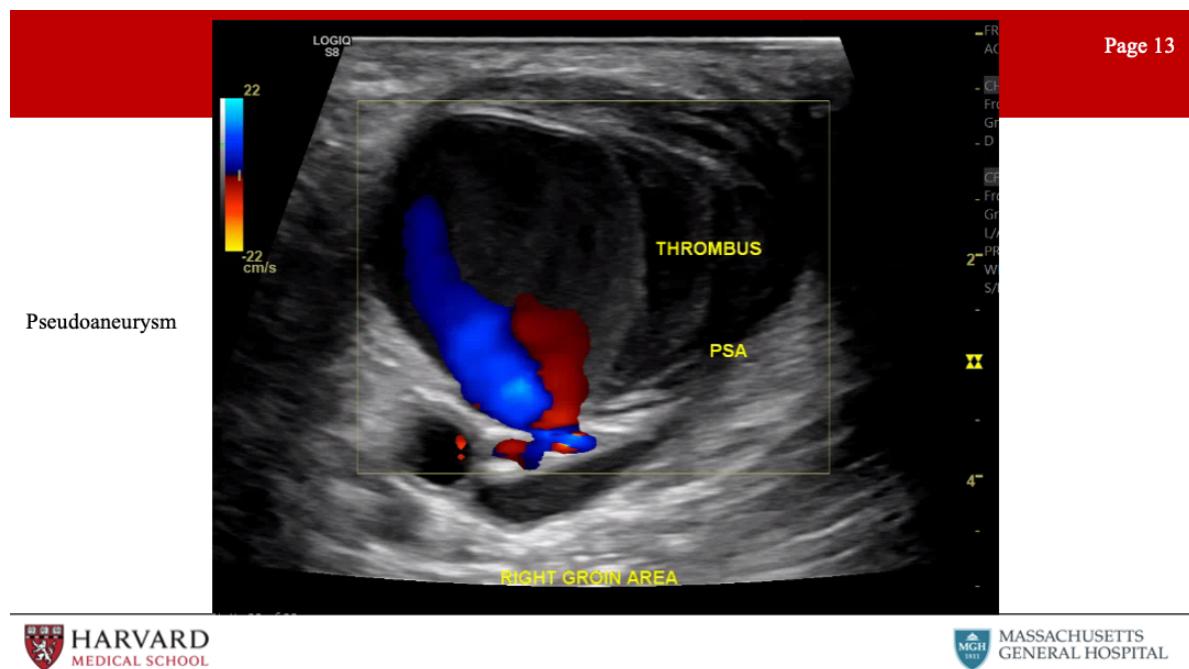
Presenting with new swelling, pain and fluid around a prosthetic graft on duplex ultrasound may be a sign of underlying graft infection. PET and WBC scans may be positive up to 4 months after implantation, so do not provide much utility in the early post-operative period.(Puges et al. 2019)

20.5.1 Pathologies

Let's talk about pathologies frequently encountered in the lower extremities:

20.5.1.1 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.



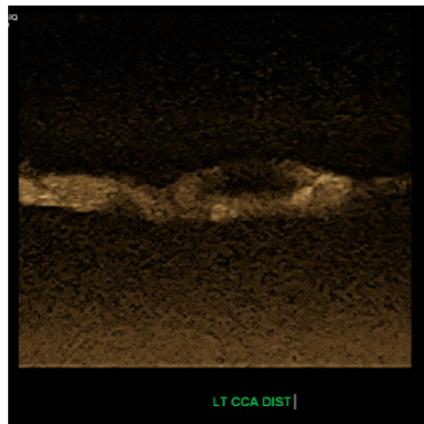
Anastomotic pseudoaneurysms develop in around 3% of femoral anastomoses and can be associated with arterial wall weakness, endarterectomy, mismatch between artery/graft, dilation/deterioration of graft material, increased/uneven tension of the anastomosis, or underlying infection.(Pellerito and Polak 2019)

20.5.1.2 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; N. 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 non-calcified plaque.

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CCA Dissection



20.5.1.3 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.(K. Busch 2011; Fujii et al. 2011)

For more see Section [3.3.4](#)

There are several disease pathologies that are frequently tested relating to the popliteal fossa. Can you talk briefly about these?

20.5.1.4 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.(M. Shaw et al. 2007; Winn et al. 2015)



For more on adventitial cystic disease, see Section 17.4.2. This is not to be confused with:

20.5.1.5 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.



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.

20.6 Abdominal

Lowest frequency transducers (2-4MHz) are used for imaging abdominal vessels.

20.6.1 Aorta

Abdominal Aortic Aneurysms (AAA): Ultrasound screening of a AAA is non-invasive, accurate, and cost-effective.(Chaikof, Dalman, Eskandari, Jackson, Lee, Mansour, Mastracci, Mell, Murad, Nguyen, and al. 2018)

- 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, see Chapter 10

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; Marrewijk et al. 2004)

Type II endoleaks with stable sac size can continue surveillance with color duplex.(Chaikof, Dalman, Eskandari, Jackson, Lee, Mansour, Mastracci, Mell, Murad, Nguyen, and al. 2018; 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.(K. J. Busch et al. 2009)

Velocities within stents are elevated from baseline due to stent rigidity, but a limb velocity $>300\text{cm/s}$ with a $V_r >3.5$ is associated with a high risk of limb occlusion from stenosis or kinking and should undergo further investigation or intervention.(Chaikof, Dalman, Eskandari, Jackson, Lee, Mansour, Mastracci, Mell, Murad, Nguyen, and al. 2018; 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.

20.6.2 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; Gustavo S. 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)

20.6.3 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/hyper-echoic 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.

20.6.4 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 > 200 cm/s and Renal-aortic ratio (RAR) >3.5, suggest >60% stenosis, however some labs use higher PSV criteria (250-285 cm/s). (Schäberle et al. 2016; Rickey and Geary 2019b) Of note, for RAR, the 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. (Radermacher et al. 2001) 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)

20.7 Venous

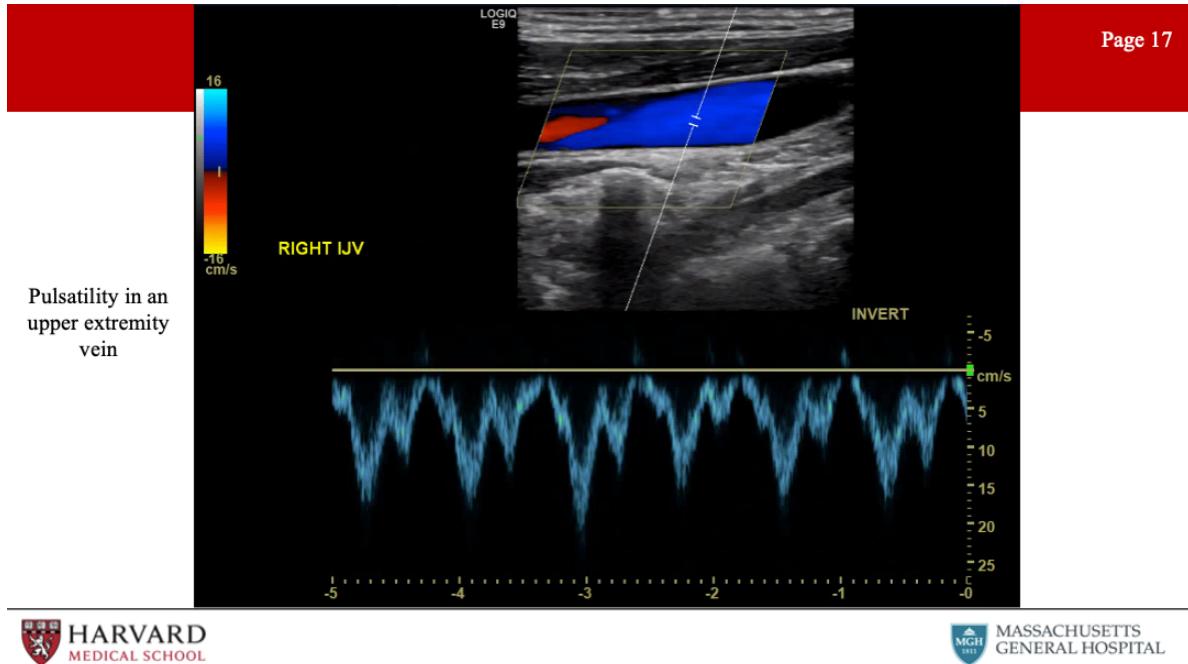
Venous Exam: Uses a linear transducer. Complete deep venous reflux duplex examination includes color and pulsed wave spectral doppler imaging. Spontaneous Doppler waveforms as well as provocative maneuvers are recorded in the common femoral, femoral, popliteal, and tibial deep veins. Superficial veins (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.

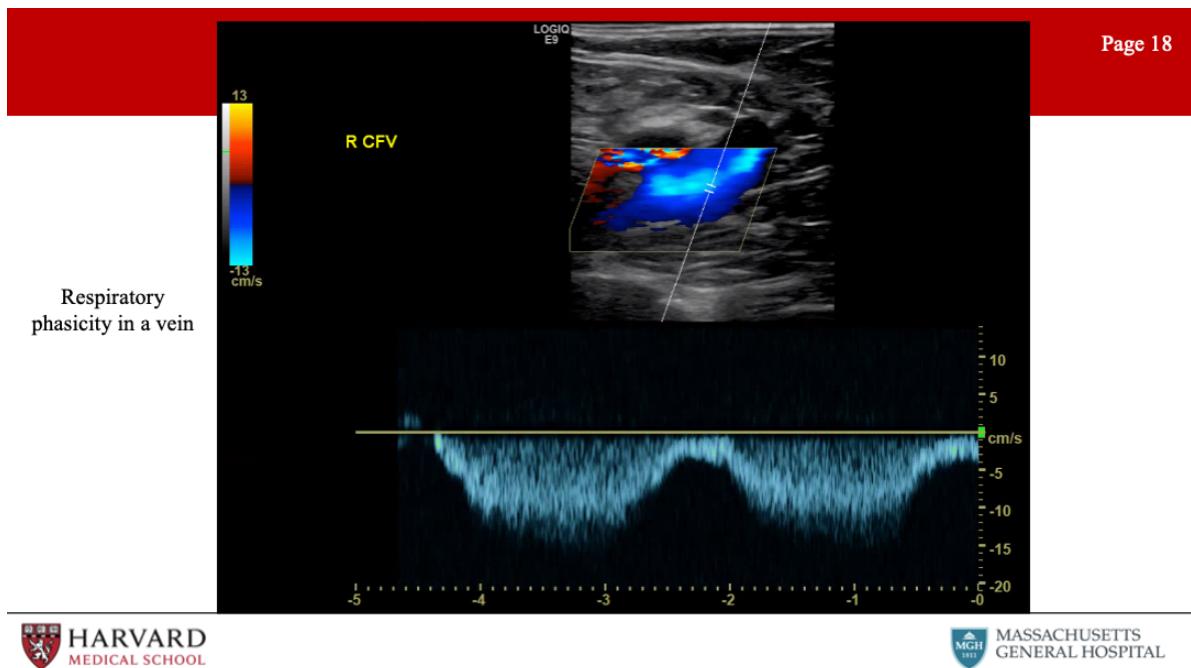
What does this mean? Pulsatility, phasicity, and augmentation?

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.

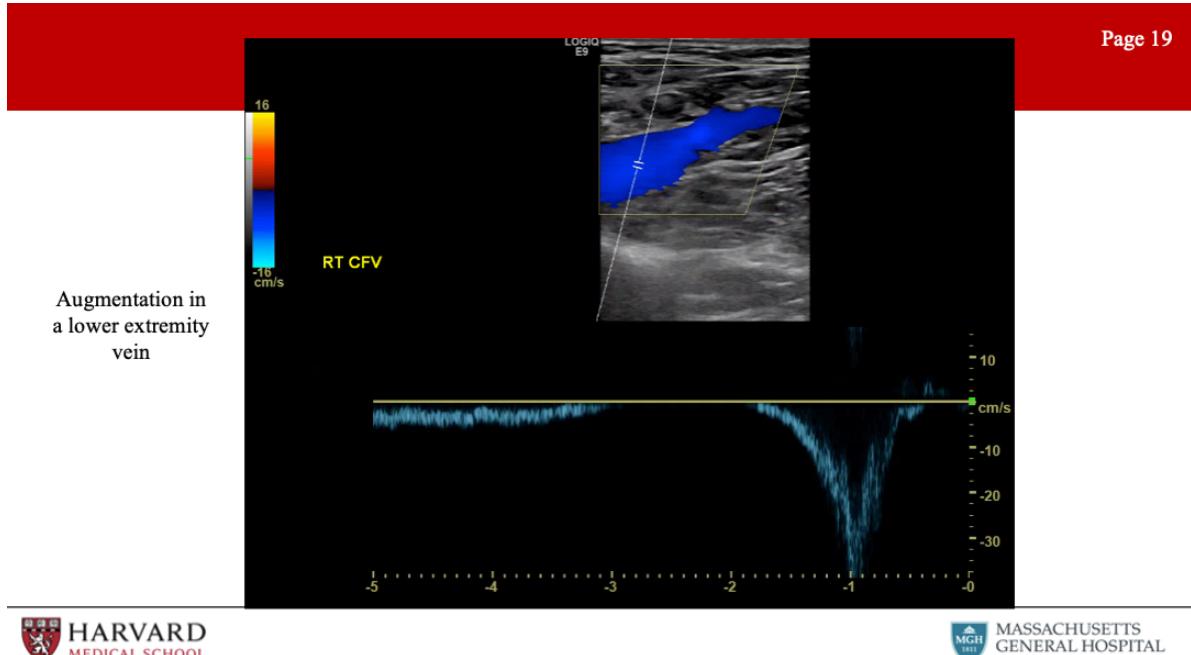


This is an abnormal finding in the lower extremity veins, and may be a sign of pulmonary hypertension, right heart failure, or tricuspid regurgitation.(Kakish et al. 1996; McClure et al. 2000)

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.



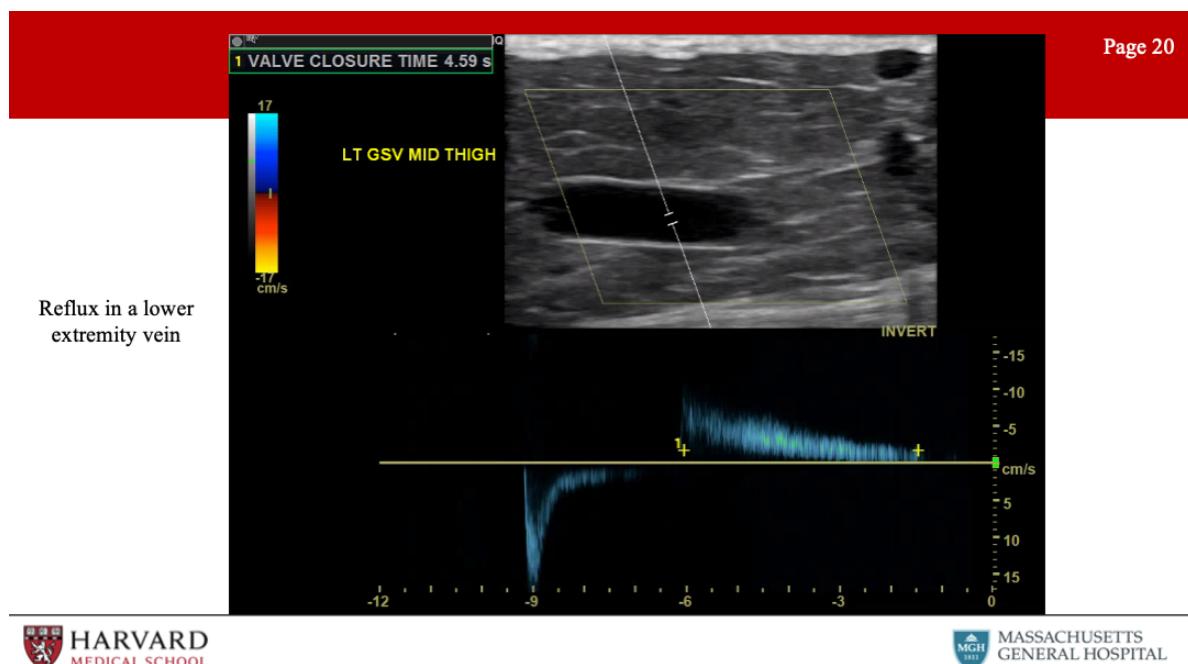
Augmentation: Distal compression that augments forward flow. For example, if we are measuring flow at the femoral vein and we squeeze the calf and we see augmentation in the waveform, this indicates lack of occlusion in the venous system from the knee to the probe.



What are venous pathologies that are commonly tested/encountered?

20.7.1 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.



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.(Gloviczki et al. 2011; Lurie et al. 2012; Labropoulos et al. 2003)

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.(Min, Khilnani, and Golia 2003; O'Donnell et al. 2014; Sandri et al. 1999) 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 a fasting patient 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.

20.7.2 May Thurner Syndrome

May Thurner syndrome is 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.

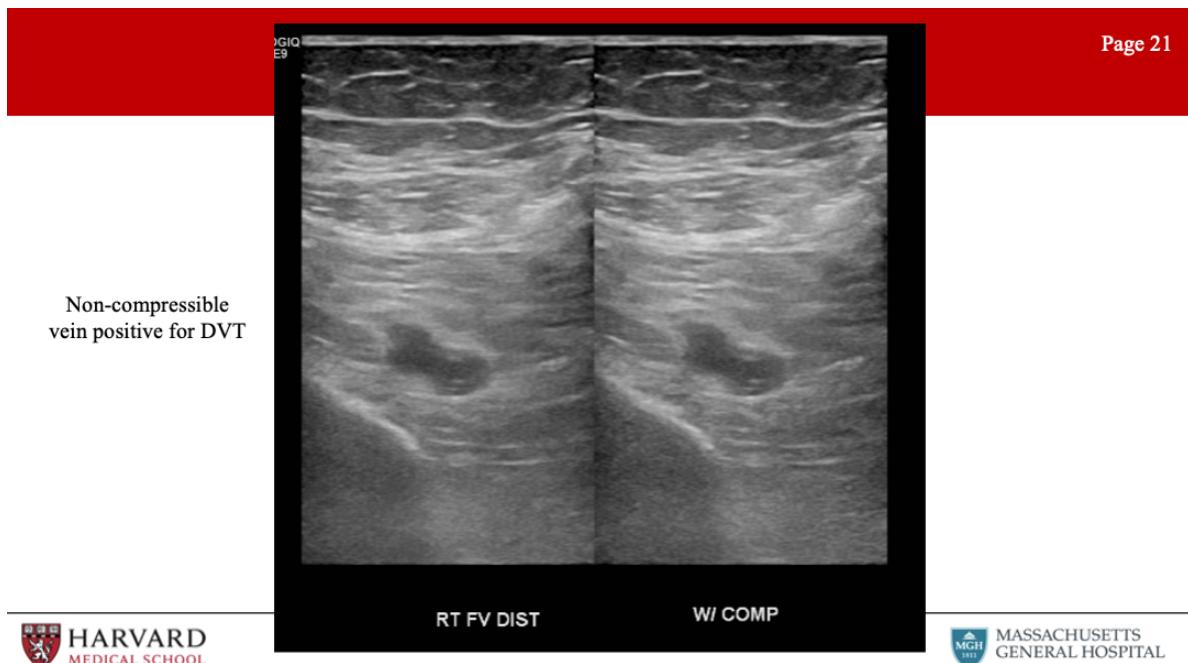
Iliac obstruction, whether a result so May Thurner or not, would demonstrate continuous flow with a loss of phasicity and reduced augmentation with distal compression or valsalva. Additional indirect ultrasound findings include collaterals and reversal of flow. Direct ultrasound findings include intraluminal changes.(Metzger et al. 2016)

20.7.3 Nutcracker Syndrome

Nutcracker syndrome is most often diagnosed based on IVUS, venogram or renal vein pressures, but there are also diagnostic criteria for duplex ultrasonography. The left renal vein velocity ratio is the velocity at the aortomesenteric angle divided by the velocity near the left renal hilum. A ratio greater than 5 has a sensitivity of 80% and specificity of 94% for Nutcracker syndrome.(S. H. Kim et al. 1996; Seung Hyup Kim 2019)

20.7.4 Thrombosis

Thrombosis: Characteristics of acute thrombus are an echolucent and incompressible thrombus in a thin-walled vein with significant distension.



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 collaterals are found in chronic thrombosis.

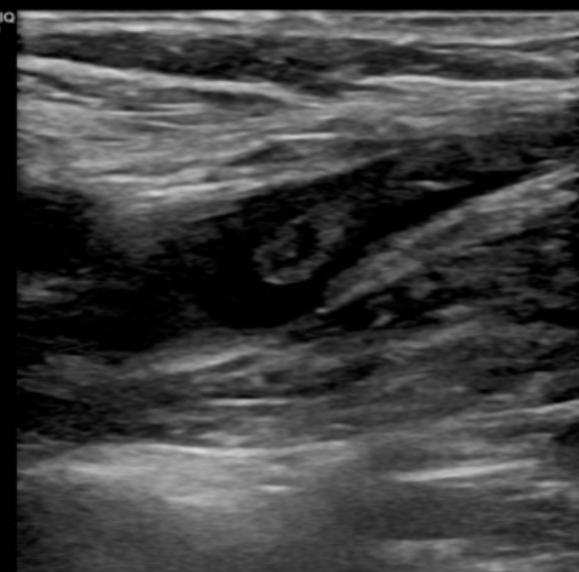
Components of a lower extremity DVT study include doppler waveforms in response to provocative maneuvers in the common femoral, femoral and popliteal veins. When performing a DVT scan, the technician should evaluate the contralateral femoral vein flow to compare flow patterns.(Needleman et al. 2018; Vascular Ultrasound 2019) Transverse views are used for compression and compressibility testing every 3-5cm is the most reliable evaluation for thrombus.(Malgor and Labropoulos 2013)

Let's discuss some examples of venous thrombosis

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. Duplex ultrasound is 81-100% sensitive for diagnosing upper extremity DVT.(Mustafa et al. 2002)

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



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.

Superficial vein thrombosis: when scanning for superficial vein thrombosis, sonographers should scan superficial and deep veins of the ipsilateral leg and the contralateral central veins with doppler wave form, because the incidence of concomitant thrombosis is not insignificant.(Decousus et al. 2010; IAC 2021; Vascular Ultrasound 2019) This is discussed more in our venous chapter: Section [18.1.4.1](#)

20.7.5 Vein Mapping

One additional venous study often performed in the lower extremity is mapping of the GSV for planning lower extremity bypasses. Mapping should identify the general course of the GSV and any major tributaries, particularly if the anterior or posterior accessory veins are of a size that might be a reasonable additional conduit.(Cohn, Caggiati, and Korver 2006) Sonographers should also make note of any significant areas of tortuosity or aneurysm that might compromise the quality of the bypass conduit. Size measurements are usually made along the GSV.

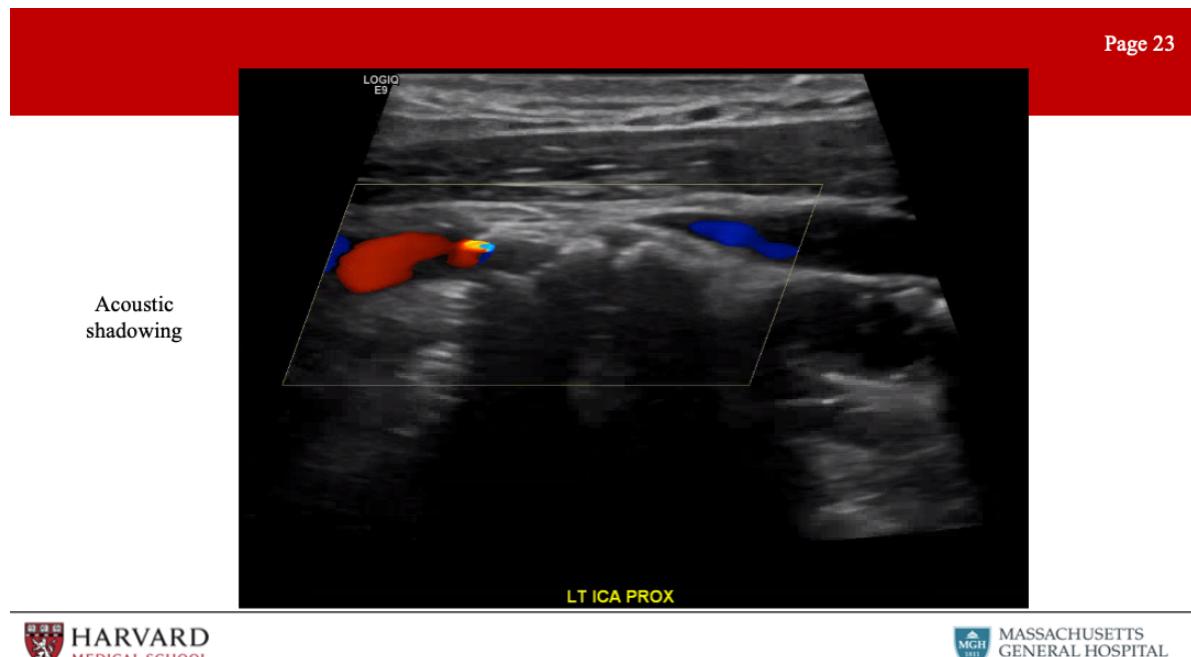
In general the ideal size for conduit is 3-4mm throughout it's length, with a size of greater than 2mm at the ankle.(Leopold et al. 1989) These size assessments are usually the same regardless of whether the vein will be fully harvested and reversed or utilized in situ. The small saphenous

vein can be utilized for a conduit if the GSV is not adequate and the diameter is 3-4mm.(B. B. Chang et al. 1992; Nierlich et al. 2019)

20.8 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.

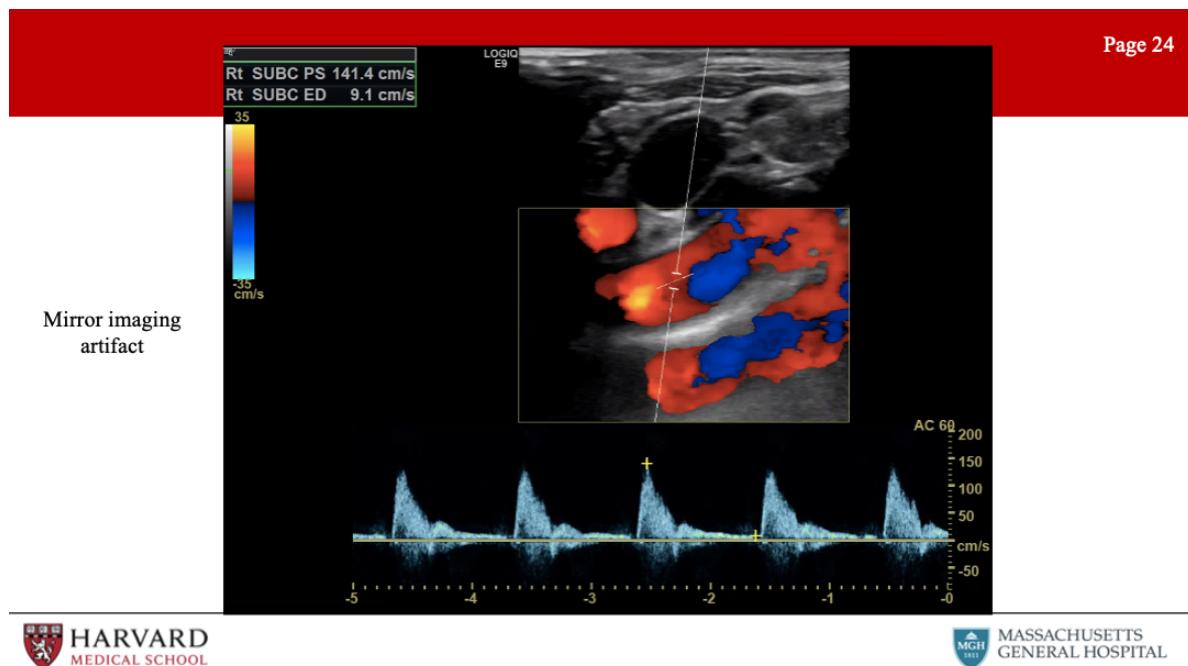
Acoustic shadowing: Shadowing on an ultrasound image is characterized by a signal void behind structures that strongly absorb, reflect, or refract ultrasonic waves.



Practically speaking, this most typically occurs deep to strongly reflective surfaces such as calcified plaques, and appears as a “dark area” beneath the plaque. Subcutaneous air can result in shadowing and loss of imaging information, which can be seen in some soft tissue necrotising infections.(Thom and Warlaumont 2017)

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.

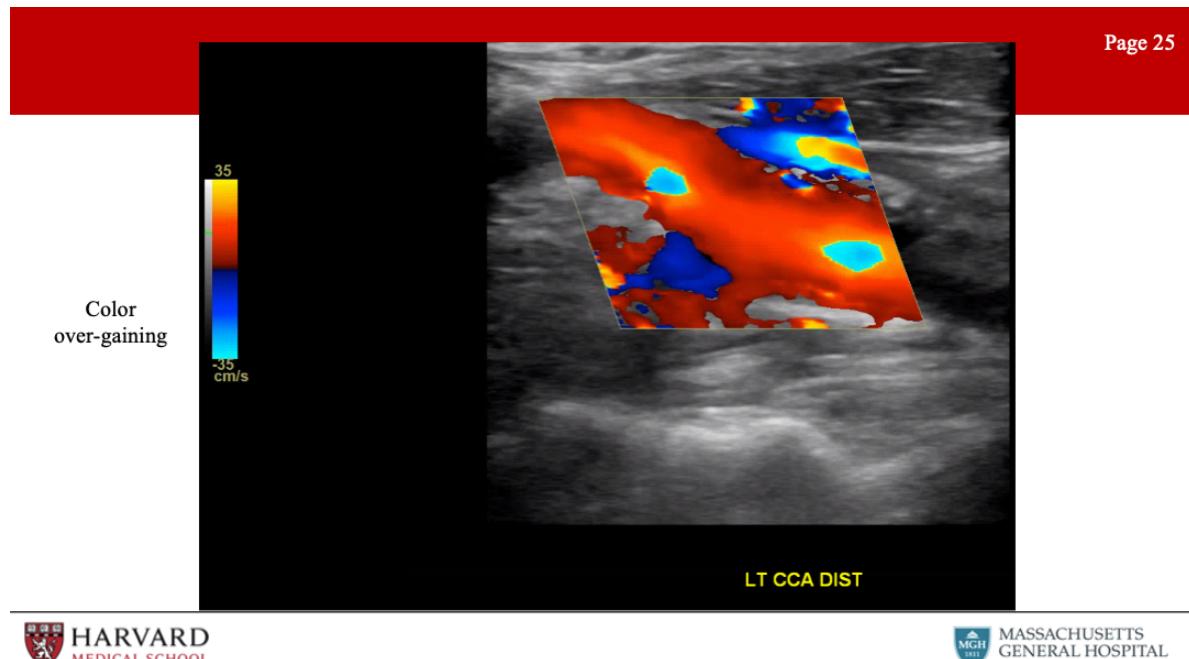


This is often seen around the pleura and the diaphragm, due to the strong reflection of ultrasound from the air-filled lung. These artifacts can occur in both B-mode imaging where you see the mirrored image and Doppler, in which you see the mirrored waveform. The clavicle is also a strong spectral reflector, and thus can result in a mirror effect and a duplicate subclavian artery.(Rubin et al. 2010)

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.

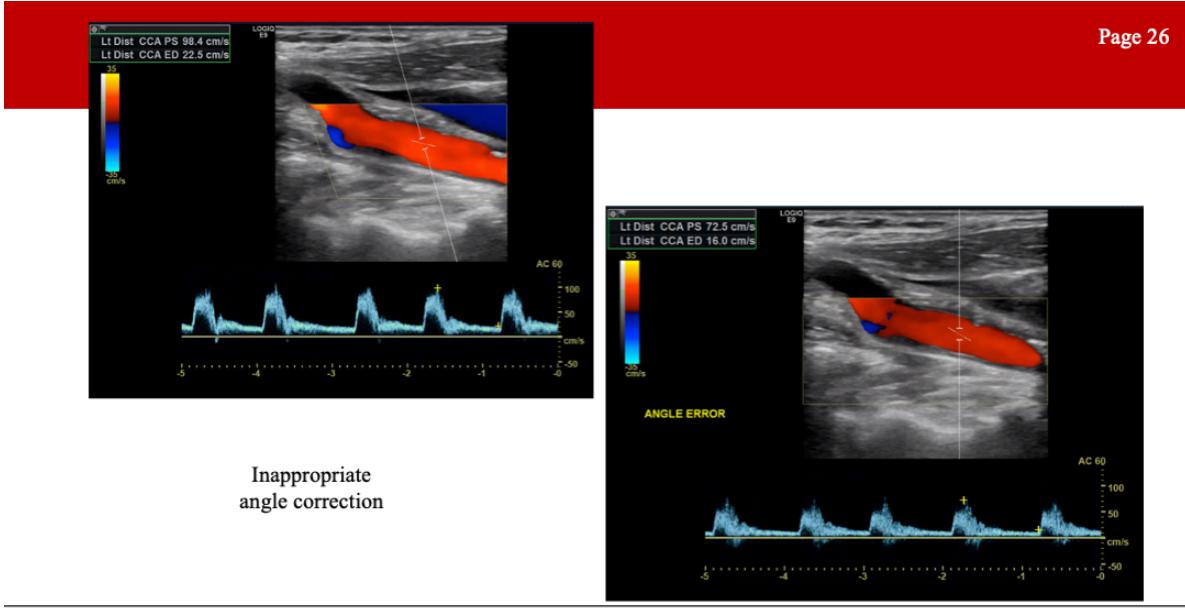
Speed artifact: Depth determination by an ultrasound machine is based on calculations using an average propagation velocity of sound in soft tissue of 1540 m/s. If the ultrasound wave passes through a medium at a different speed than predicted by the machine, an inaccurate image depth will be displayed. 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) The noise added to the spectral waveform can result in PSV overestimation.



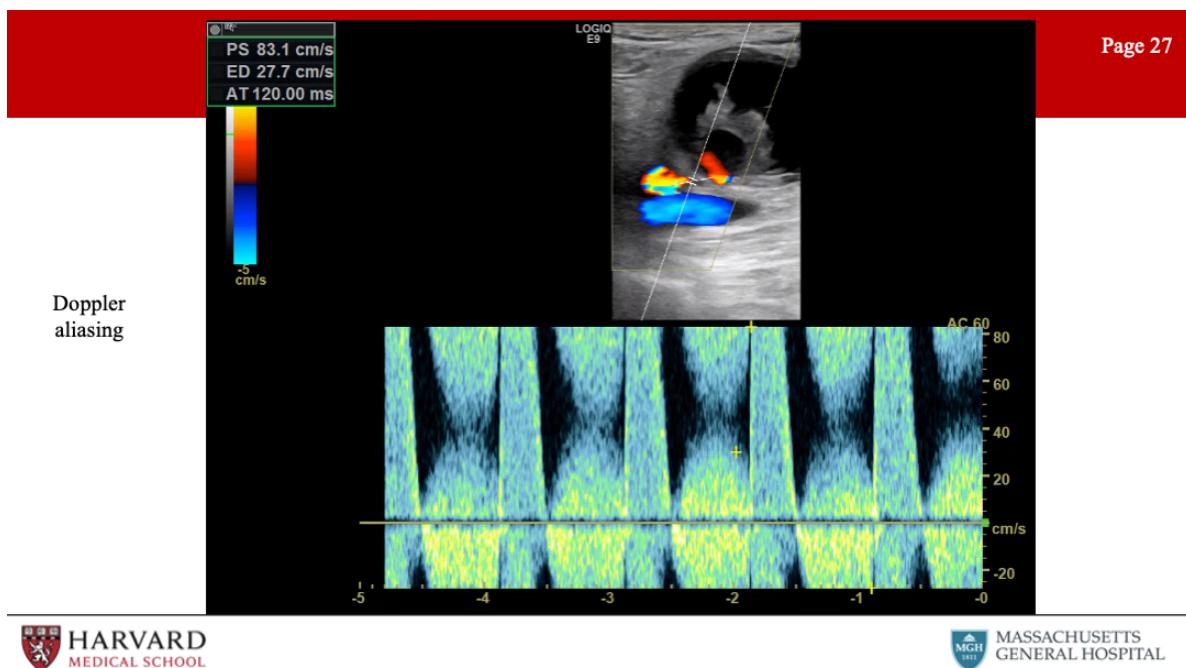
Under-gaining will result in reduced sensitivity to low velocity flow.

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. Increasing the doppler angle will increase the velocity measurement (if no change to angle correction is made).



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.



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.

20.9 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 re-credentialing every three years.(IAC 2021)

- Physician accreditation with RPVI requires re-certification 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.(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.(Diagnostic Medical Sonography 2015)

21 Radiation Safety

Authors: Adam P Johnson and Melissa Kirkwood

21.1 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.(A. B. 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.(A. B. 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).(D. Kwon, Little, and Miller 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 RAK and should trigger specific patient follow up. Of note, 5Gy refers to the RAK that is reported at the end of the procedure, which is an approximation of the peak skin dose (PSD).(Baiter et al. 2011; Hirshfeld et al. 2005; Kirkwood et al. 2015; Stecker et al. 2009)

21.2 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 21.1: 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; L. K. Wagner et al. 1999)

Dose	Effect
0-2Gy	No observable effects
2-5Gy	Transient skin erythema and dermatitis, full recovery 6w-1yr(Guesnier-Dopagne et al. 2019; Kirkwood et al. 2014)
5-10Gy	Erythema and epilation (hair loss), prolonged erythema up to 1y
10-15Gy	Permanent epilation, atrophy or induration up to 1y
>15Gy	Dermal Necrosis

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 occupational exposure include 20mSv per year and total threshold of 0.5Gy, which was recently lowered due to risk of cataract development from ocular exposure.(Hamada et al. 2017)

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)

21.3 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

21.4 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.
- Preoperative planning is paramount to efficient use of radiation during a procedure. Recent EVS guidelines recommend utilization of a 3D pre-operative planning software and image fusion (when available) for complex endovascular procedures.(Modarai et al. 2022)
- Use last image hold to allow for procedural planning
- Decrease the frame rate to reduce the frequency of radiation exposure per second.

Take a Look

Check out [Dr. Ellozy's operative planning video](#) where he reviews how to plan for an EVAR procedure utilizing Tera-Recon 3D imaging software.

- 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

- Recommendations are that clinicians wear lead aprons covering their torso and legs with a thyroid shield and leaded glasses.
- Collimation can be helpful to reduce patient and operator dose, scatter and improve image quality.(Haqqani et al. 2012)
- The location with the highest exposure to scatter is below the table.(Gonzales, Moran, and Silberzweig 2014; Miller et al. 2010) Therefore, leaded 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.(Healthcare Inspections 2014; University, Stanford, and Complaints, n.d.)

Take a Look

Check out our [episode with Dr. Jasmine Bhinder](#), discussing a recently published quality improvement project for institutional level changes that lead to reductions in radiation exposure for trainees.

21.4.1 Pregnancy considerations

Pregnancy of both patients and clinicians need to be considered in relation to radiation exposure and safety.(V. Chandra et al. 2013; Mitchell and Furey 2011; P. 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.(P. 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 5 mSv (500mrem) or 0.5 mSv (50mrem) per month.(Dauer et al. 2015; V. 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.
- 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.(P. Shaw et al. 2011)
 - Original studies demonstrated reduction in fetal exposure by 80% and is currently common practice.(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.(V. Chandra et al. 2013)

21.4.2 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, n.d.) Sentinel events include:

1. The patient has a permanent cutaneous injury and the proper dose saving techniques were not used during the procedure
2. Cumulative dose of 15 Gy for a single field over 6mo - 1yr.
3. Delivery of radiotherapy to the wrong body region.
4. Actual dose more than 25% above planned radiotherapy dose.(A. K. 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 0.1 mSv (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)

21.5 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)

21.5.1 CO₂ Angiography

CO₂ angiography is often utilized in place of iodinated contrast during fluoroscopy. A bolus of CO₂ is injected, which then absorbs less ionizing radiation than surrounding tissue and provides a map of the arterial tree. CO₂ can be used in a wide range of endovascular procedures, even a ruptured AAA.(Knipp et al. 2010) However, there are specific limitations and complications that should be understood.

21.5.1.1 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; Kyung Jae Cho 2015; E. Criado et al. 2012; Sharafuddin and Marjan 2017)

21.5.1.2 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.(Kyung J. Cho and Hawkins 2011; Kyung Jae 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 management 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)

Take a Listen

Check out our [previous episode with a panel with Drs. Kirkwood, Wohlauer, and Chandra discussing occupation hazards for the vascular surgeon](#), of which an important hazard includes radiation safety.

22 Endovascular Access

Authors: *Sammy Siada and Rafael Demarchi Malgor*

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 post-operatively 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.(Sorrentino et al. 2020)

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 contraindicated 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.

22.1 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; Patrick A. Stone, Campbell, and AbuRahma 2014) For a representative image, see Section [20.5.1.1](#)

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.

23 Rapid Review

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In this chapter we discuss a number of disparate topics that are often seen on the VSITE examination in the US, and often just require rote memorization. We have included links to the relevant chapters for you to see the information in more context if needed. This chapter is not meant as a stand alone review.

23.1 Venous Disease

Memorization CEAP classification:

- C0: no visible or palpable signs of venous disease.
- C1: telangiectasias or reticular veins.
- C2: varicose veins.
- C3: edema.
- C4a: pigmentation and eczema.
- C4b: lipodermatosclerosis and atrophie blanche.
- C5: healed venous ulcer.
- C6: active venous ulcer.

EHIT (DVT after EVLA), endothermal heat-induced thrombosis

1. Thrombus without propagation into the deep vein: no treatment
 - a. Peripheral to superficial epigastric vein
 - b. Central to superficial epigastric vein, up to and including the deep vein junction: can consider antiplatelets
2. Thrombus propagation into the adjacent deep vein but comprising <50% of the deep vein lumen: weekly surveillance

3. Thrombus propagation into the adjacent deep vein but comprising >50% of the deep vein lumen: therapeutic anticoagulation until resolution to the SFJ
4. Occlusive deep vein thrombus contiguous with the treated superficial vein: treat like DVT

Reasons to treat superficial venous thrombosis (fondaparinux x 45 days)

- Within 3-5 cm of deep system (e.g., SFJ)
- > 5cm long
- Propagates with conservative management

For more see Chapter 18

23.2 Vascular Lab

23.2.1 TCD

TCD temporal window:

- Toward probe is up on waveform (MCA, ICA)
- Away from probe is down on waveform (ACA)
- PCA is bidirectional (P1 before PCommA is up, P2 after PCommA is down)

TCD orbital window:

- Ophthalmic is toward probe
- ICA in siphon is bidirectional

TCD occipital window:

- vertebral and basilar away from probe (think direction on carotid duplex)

Consider shunt during CEA if MCA velocity drops by 50% or more A doubling of MCA mean velocity suggests cerebral hyperperfusion syndrome

Lindegaard ratio 3-6 indicates spasm, > 6 indicates severe spasm. PSV in MCA relative to extracranial ICA.

For more see Section 20.3

23.2.2 Basics

Power doppler is like amplitude, good for low flow evaluation but you lose directionality (think absolute values). Good for detecting string sign, low flow in renal parenchyma, or torsion. Not useful for directionality of flow (e.g., TCD or vertebral direction), and will not alias.

Depth resolution (synonymous with axial, longitudinal range) in PW mode is limited by half the spatial pulse length (SPL). As long as the resolution between objects is less than half the SPL, objects can be discriminated (because the total distance doubles going round). Shorter SPL means you can distinguish smaller things, which means better (smaller) resolution. Shorter SPL theoretically is achieved by shorter wavelength/higher frequency, more damping, shorter pulse duration. It is not technically affected by PRF/PRP.

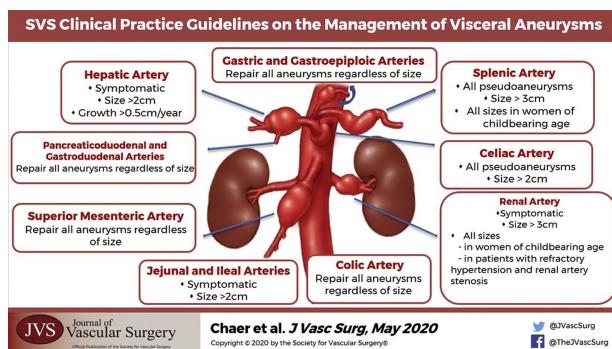
PRP = time between pulses = imaging depth (ID) * 13 microseconds/cm note that PRF = 1/PRP, and that PRFs are generally 5-40 kHz

Maximum Doppler shift detectable = Nyquist limit = PRF/2. Remember this only applies to PW Doppler. CW Doppler has no practical limit beyond which aliasing occurs. Use caution between depth resolution (2x SPL) and Nyquist limit (1/2 PRF)

For more, see Section 20.1

23.2.3 Sizes and Velocities

Intracranial aneurysm and carotid stenosis: no need to treat if < 10mm



(Chaer et al. 2020)

AAA screening

- In any pt with suspected or confirmed AAA, physical exam of femoral + popliteal pulses
- Any pt with femoral or popliteal aneurysm should undergo AAA screening

- Men or women 65-75 years w/ h/o tobacco use (SVS recommendation)

AAA surveillance

- 2.5-3cm: 10 years
- 3-3.9cm: 3 years
- 4-4.9cm: 1 year
- 5-5.4cm: 6 months

Carotid velocities

- > 70%
 - PSV > 230
 - EDV > 100
 - ICA/CCA > 4.0
- > 50%
 - PSV > 125
 - EDV > 40
 - ICA /CCA > 2.0

CMI velocities

- CA PSV > 200
- SMA PSV > 275

Renal stenosis criteria

- PSV > 200 (some advocate for PSV > 285)
- RAR > 3.5, but aortic velocity should be 40-100 cm/s
- Parenchymal disease is RI > 0.7
 - $(PSV-EDV)/PSV$

23.3 Zebras

Indications for surgical management of vertebral disease

- At least 60% stenosis
 - In both arteries if patent
 - In one artery if contra is hypoplastic, occluded, or terminates in PICA
- If low flow: other etiologies have been ruled out (arrhythmia, emboli, inner ear dysfunction, electrolyte imbalance, intracranial tumor, hypotension)
- Posterior embolism should be considered for treatment
- Location
 - V1 can be treated by transposition onto CCA or vein bypass
 - Proximal vertebral artery reconstruction is associated with Horner's syndrome
 - V2 not usually accessible, if uncontrolled traumatic hemorrhage, ligate at V1+V3
 - V3 disease described treatments include bypass, transposition of ECA or occipital artery, or transposition onto ICA

For more, see Section 1.5.2

Surgical exposure of supra-aortic vessels

- Median sternotomy for: innominate, proximal R SCA, R CCA, and L CCA
- L trapdoor (L anterolateral thoracotomy and supraclavicular incision) for proximal L SCA
 - Watch for L vagus n. and thoracic duct in chest, and watch for phrenic n. on anterior scalene

MALS: compression with EXPiration, normalization with INSpiration

Electrolyte abnormality with re-feeding

- Phos down (ATP)
- K, Mg down (taken intracellular for anabolism)
 - Mg down can lead to Ca down
- Torsades can be caused by low K, Mg, or Ca > treat with Mg

Popliteal artery disease

- Baker's cyst vs cystic adventitial disease (knee flexion)
 - Baker's cyst is contiguous with joint space
 - Cystic adventitial disease is within the arterial wall
- Popliteal entrapment (passive dorsiflexion or active plantar flexion)
- Popliteal artery aneurysms
 - If patients present with PAA, need screening for AAA
 - Above 2 cm should undergo repair, unless high clinical risk may wait until 3 cm
 - Can also consider repair at smaller sizes if high embolic risk
 - If life expectancy > 5 years, should undergo open repair
 - If acutely thrombosed
 - I + IIa should undergo lysis if tibial outflow bad
 - IIb should undergo thrombectomy of some fashion
 - III undergo primary amputation

For more, see Section 17.4

HITT

- 4 Ts
 - Thrombocytopenia - Platelets fall > 50% and are less than 20k
 - Timing - Clear onset between 5-10 days or > 1 day w/ previous heparin exposure
 - New Thrombosis
 - No other cause for Thrombocytopenia
- Bivalirudin if hepatic failure
- Argatroban for renal failure (OK for hepatic fail, but needs titration)

Obturator foramen

- Valentine/Wind
 - Nerve and vessels traverse the foramen superolateral corner
 - Tunnel should be created “centrally” by incising the medial portion of fascia
- Chaikof/Cambria

- Nerve and vessels traverse posterolaterally
 - Tunnel should be created anteromedially
- Rutherford
 - Nerve and vessels are posterolateral
 - Tunnel should be created anteromedially

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