# Vascular Surgery Board Review

Editors: Adam Johnson, MD, MPH; Matt Smith, MD, PhD; and Audible Bleeding

2022-02-12

# About

The content was developed here by the Audible Bleeding Team to accompany our board review podcast episodes.

## Usage

This is not a comprehensive textbook but instead an outline of the most high yield information to help guide board preparation.

## Comments, Questions or Contributions

Please visit our github page or send us an email.

# Cerebrovascular

**07 Jan 2019:** *Adam Johnson, MD, MPH; Nicole Rich, MD, MPH; Kevin Kniery, MD, MPH*

## Available Guidelines

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

## Presentation and Diagnosis

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

   Frequent repetitive neurological attacks without complete resolution of the deficit between the episodes, producing the same deficit but no progressive deterioration in neurological function If a progressive deterioration then it is a stroke in evolution.

2. **Who needs to be screened?**

   Only 15% of stroke victims have a warning TIA before a stroke so waiting until symptoms occur is not ideal. 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.

   The absence of a neck bruit does not exclude the possibility of a significant carotid bifurcation lesion - 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%).

   Screening of the general population is not indicated. Screening should be considered for patients with:

   - 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 should be expected.
- The benefit of prophylactic treatment of high grade stenosis is estimated at a 1-2% stroke reduction risk per year. (Naylor, 2015)
- Keep in mind that intervention (CEA/CAS) has only demonstrated a benefit in asymptomatic patient with life expectancy greater than 3 years. (Bulbulia & Halliday, 2017; Halliday et al., 2010; Rosenfield et al., 2016)

3. **US findings that confirm disease**
   - 50-69% stenosis of ICA - Low sensitivity for 50-69% stenosis - a negative ultrasound in symptomatic patients necessitates additional imaging
     - PSV 125-229 cm/sec
     - EDV 40-100
     - Internal/Common Carotid PSV Ratio 2-4
   - 70-99% stenosis of ICA
     - PSV >/= 230 cm/sec
     - EDV >100 (EDV > 140 cm/sec most sensitive for stenosis >80%)
     - Internal/Common Carotid PSV Ratio > 4
   - Velocity-based estimation of carotid artery stenosis may need to be adjusted in certain circumstances
     - Higher velocities in women than in men
     - Higher velocities in the presence of contralateral carotid artery occlusion.
   - High carotid bifurcation, severe arterial tortuosity, extensive vascular calcification, and obesity may also reduce the accuracy of DUS imaging
4. **Other Imaging Modalities**
   - CTA
     - Pro - fast, sub-millimeter spatial resolution, visualize surrounding structures

  - Con - cost, contrast exposure

- MRA
  - Pro - no contrast administered; 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 and risk of stroke

# Management

## Optimal medical therapy

### Hypertension

- Lowering blood pressure to a target <140/90 mmHg by lifestyle interventions and anti-hypertensive treatment is recommended in individuals who have hypertension with asymptomatic carotid atherosclerosis or those with TIA or stroke after the hyper-acute period.
- Each 10-mm Hg reduction in blood pressure among hypertensive patients decreases the risk for stroke by 33%.

### Diabetes

- Glucose control to nearly normoglycemic levels (target hemoglobin A1C <7%) is recommended among diabetic patients to reduce microvascular complications and, with lesser certainty, macrovascular complications other than stroke.

### Lipid abnormalities

- Risk of stroke decreased by >15% for every 10% reduction in serum LDL in patients with known coronary or other atherosclerosis
- Statin agents are recommended targeting LDL of 100 mg/dL, for those with coronary heart disease or symptomatic atherosclerotic disease, and LDL of 70 mg/dL for very high-risk persons with multiple risk factors
- High dose statin therapy in patients with TIA/stroke reduce future rates of stroke or cardiovascular events but not overall mortality at 5 years. (Karam et al., 2008)

**Smoking** - Physician counseling is an important and effective intervention that reduces smoking in patients by 10% to 20%

**Antithrombotic therapy** - There is no evidence to suggest that antiplatelet agents other than aspirin have improved benefit in asymptomatic patients with carotid atherosclerosis

## Carotid endarterectomy

### Timing

- Recommendations on when to operate after a stroke
  - Acute stroke with a fixed neurologic deficit of >6h duration - When the patient is medically stable, treatment in less than or equal to 2 weeks after the stroke is preferable. (Meershoek & de Borst, 2018; Rothwell et al., 2004)
  - 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
  - Stroke in evolution (fluctuating / evolving neuro deficit) or crescendo TIA (repetitive transient ischemia w improvement between events) - If neuro status is not stabilized by medical intervention consider urgent CEA
  - CEA is preferred to CAS based on an increased embolic potential of carotid lesions that present in this fashion. (Rantner et al., 2017)
  - Management of acute stroke (Powers et al., 2018)
    - <4.5hrs from onset of symptoms - tPA unless contraindication
      - Age >80 and diabetes are contraindication to tPA after 3hrs.
      - Other contraindications - high BP, intracranial hemorrhage, recent stroke or head trauma, spine/brain surgery within 3mo, GI bleed within 21d
    - <6hr from onset of symptoms - catheter directed therapy
- What is the only emergent indication for CEA?
  - Crescendo TIAs or a stroke in evolution with a surgically correctable lesion that is identified

### Intraoperative Techniques

- General concepts
  - Patch angioplasty or eversion endarterectomy are recommended rather than primary closure to reduce the early and late complications of CEA (GRADE 1, Level of Evidence A).

- Neuromonitoring/Shunting options during a carotid endarterectomy
  - Local anesthesia with direct neuro monitoring - the patient is awake and moving to command throughout the case. Though improved neuromonitoring has not been shown to reduce MI rate with CEA
  - Stump pressure Clamp the inflow and place butterfly attached to a-line tubing into the internal carotid If stump pressure is $> 40$ mmHg can proceed, if $< 40$ place shunt
  - EEG Neuromonitoring - EEG tech places neuromonitoring, monitored by intraop tech and neurologist remotely, generally clamp ICA for 3 minutes before proceeding, if any deficits unclamp, await normalization of EEG then proceed
  - Non-selective shunting - shunt all carotids
- Techniques to reach internal carotid lesions that are high?
  - Nasotracheal intubation will help extend the neck to reach higher lesions
  - Divide posterior belly of digastric to reach high lesions with care to watch for glossopharyngeal
  - Styloidectomy
  - Mandible subluxation with assistance from ENT if previous techniques fail.
- What is the best technique for a patient with a kinked internal carotid artery?
  - Eversion carotid endarterectomy will allow you to reduce the redundancy
  - Otherwise, no advantage has been shown between eversion or patch, both can be shunted
- Discuss nerve injuries – where you would encounter these and what deficit would be seen
  - Hypoglossal Just above the bifurcation of the carotid artery Will see tongue deviation to the side of injury
  - Glossopharyngeal High dissections under digastric Difficulty swallowing, aspiration risk, can be devastating
  - Vagus Adjacent and lateral to carotid, injury occurs with carotid clamping, Hoarseness is noted as RLN is a branch off of vagus
  - Marginal Mandibular (Off of facial nerve) Retraction at the angle of the jaw for high dissections Leads to the corner of lip drooping, can be confused with a neuro deficit following the case

**Postoperative Complications**

- What to do if neuro deficits following your carotid endarterectomy
  - If in OR – perform duplex, if normal open wound and shoot cerebral angiogram
  - If in Recovery or on the floor – many would consider CTA first vs duplex to look for thrombosis
- Risk factors and how to manage hyperperfusion syndrome?
  - Defined as an ipsilateral headache, hypertension, seizures, and focal neurological deficits can present 2-3 days out from surgery
  - Patients with uncontrolled hypertension are at risk for hyperperfusion syndrome, clinical practice guidelines by SVS recommend strict BP control following CEA, maintain a pressure less than 140/80
- High risk groups
  - ESRD patients have higher rates of perioperative stroke, but also have higher rates of stroke if not revascularized. (Klarin et al., 2016)

**Long term complications and follow up**

- Recommend f/u US at </=30 days. >/= 50% stenosis requires further imaging.
- Contralateral stenosis
  - The risk of progression for moderate stenosis at the initial surveillance to severe stenosis can be as high as five times
  - Requires post-operative surveillance.

## Carotid Artery Stenting

- In patients aged >70 undergoing CAS the risk of stroke was the highest, presumably due to calcific 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 can be reduced, but not eliminated, by using flow-reversal embolic protection rather than distal filter protection
- Limited data on CAS in asymptomatic patients - currently is not supported by guidelines or considered reimbursable

- Consider CAS in symptomatic patients with >50% stenosis who are poor candidates for CEA due to severe uncorrectable medical comorbidities and/or anatomic considerations
  - Ipsilateral neck dissection or XRT - equivalent periprocedural stroke rate to CEA, but increased later stroke rate. CEA higher rates of cranial nerve damage (9%). (Giannopoulos et al., 2018)
  - Contralateral vocal cord paralysis
  - Lesions that extend proximally to the clavicle or distal to C2
- Transfemoral Approach vs Transcarotid approach
  - ROADSTER Trial - single arm study with flow reversal for cerebral protection. Suggest lower rates of post-op stroke
- Post-op follow up - Dual-platelet therapy should be continued for 1 month after the procedure, and aspirin should be continued indefinitely
  - In stent restenosis (>50%) - 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)

## Management of uncommon disease presentations

- Occluded Carotid What to do for occluded carotid?
  - Leave it alone
- What if occluded carotid is still causing TIAs?
  - External carotid endarterectomy and ligation of internal
  - The addition of oral anticoagulation is likely to reduce the rate of recurrent CVA
- What if the patient has severe vertebrobasilar insufficiency and carotid artery disease?
  - Should undergo carotid revascularization first to improve flow
  - Vertebrobasilar insufficiency characterized by dizziness, ataxia, nausea, vertigo and bilateral weakness. (Lima Neto et al., 2017)
- 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 first, likely the embolic source
- 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

- Simultaneous coronary and carotid disease
  - Patients with symptomatic carotid stenosis will benefit from CEA before or concomitant with CABG. The timing of the intervention depends on the clinical presentation and institutional experience (GRADE 1, Level of Evidence B).
  - Patients with severe bilateral asymptomatic carotid stenosis, including stenosis and contralateral occlusion, should be considered for CEA before or concomitant with CABG (GRADE 2, Level of Evidence B)
  - Patients undergoing simultaneous CEA/CABG demonstrate highest mortality. (Naylor et al., 2003)

## Prospective Trials - MUST READS

1. Asymptomatic Carotid Atherosclerosis Study (ACAS)
   - Compared medical management with CEA in asymptomatic patients with $> 60\%$ stenosis
   - 5-year stroke and death rate was 5.1% vs 11%
   - In women, the benefit of CEA was not as certain as 5y stroke and death rates were 7.3% vs. 8.7%
   - This was 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.

3. Carotid Revascularization Endarterectomy versus Stenting Trial (CREST)
   - 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)
   - Subanalyses identified that older patients (>70y) had better outcomes after CEA than CAS, the QOL impact of stroke was more significant than that of MI, and anatomic characteristics of carotid lesions (longer, sequential, remote) were predictive of increased stroke and death after CAS
   - Unfortunately, this study provides a benchmark to strive for, but no other large trials have achieved these results.
4. ROADSTER
   - 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.
5. Trials to look out for in the next few years
   - CREST-2 - multicenter, randomized controlled trial is underway that is evaluating revascularization against modern intensive medical management
   - ACT-1 and ACST-2- the role of intervention in asymptomatic patients, designed to compare the early and long-term results of CEA vs CAS and best medical management
   - ROADSTER-2 - TCAR

# Upper Extremity and Thoracic Outlet

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

## Anatomy/ Exposure of Vessels

**What are the zones of the upper extremity?** (Illig, 2019, 2019)

Division of the upper extremity into three zones:

1. Intrathoracic zone including aortic arch, innominate artery, subclavian artery bilaterally, innominate veins, and SVC
2. Thoracic outlet (base of neck to the axilla including the subclavian, proximal vertebral, proximal axillary arteries/veins)
3. Axilla to fingers (the arm)

**What are some common exposures for major upper extremity arteries?**

Right Subclavian Artery: Medial sternotomy (proximal) or right supraclavicular area (mid/distal)

Left Subclavian Artery: Anterolateral thoracotomy in emergent setting for proximal left subclavian artery control. When third space sternotomy, supraclavicular incision with thoracotomy "trap door" exposure

Supraclavicular incision: After division of the platysma and clavicular head of the SCM, fat pat of varying thickness contains the omohyoid muscle. This should be divided and placed superiorly/laterally. At this point, the anterior scalene muscle is exposed medially with phrenic nerve running in lateral to medial direction. Division of anterior scalene for carotid/subclavian bypass should be performed as close to the first rib as possible. After this is performed, the subclavian artery is exposed.

Axillary Artery: Infraclavicular exposure below middle 1/3rd of clavicle. Pec major split and pec minor freed at lateral wound. Axillary vein followed by deep

and superior to get to 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 horizontal skin incision 2 cm below the middle third of the clavicle. Underlying pec major is split by bluntly separating the fibers and followed by exposing the tough clavipectoral fascia. At the lateral wound, the pec minor can be freed and laterally retracted. The axillary vein is first structure encountered in the sheath and the artery lies just superior and deep to the vein. Make sure to avoid nerves of brachial plexus that lie deep to first part of axillary artery and are at risk for injury during blind placement of occluding arterial clamps. (Gary G Wind & R. James Valentine, 2013)

**What steps are involved for brachial artery exposure?**

Brachial artery: incision between biceps/triceps on medial arm (avoid basilic vein damage in subcutaneous and deep to the fasia at medial biceps. Median nerve seen and retracted. Two brachial vein are paired adjacent to artery.

Superficial location makes it vulnerable to injury and accounts for most vascular injuries of 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 in the subcutaneous tissue. Neurovascular bundle exposed by incising the deep fascia at the medial border of the biceps muscle, which is retracted anteriorly. After retracting basilic vein into posterior wound ,brachial sheath is opened and median nerve is most superficial structure and retracted. The artery lies deep to the nerve and surrounded by two brachial veins. Posteriorly, is the presence of the ulnar nerve.

Brachial Artery bifurcates at the radial tuberosity into radial/ulnar branches. After the bifurcation and immediately after its origin, the ulnar artery gives off a short common interosseous branch, which bifurcates at the hiatus in the proximal interosseous membrane. Exposure of brachial artery in the antecubital fossa requires a transverse skin incision 1 cm distal to the midpoint of the antecubital crease. After deepening, avoid injury to subcutaneous veins and mobilize the basilic vein medially. Medial antebrachial cutaneous nerve should be protected. Divide the bicipital aponeurosis and after division, exposure of the brachial artery is present, which is flanked by two deep veins and crossing branches. Isolation of brachial artery requires ligation and division of these crossing vein branches.

Radial artery at the wrist with 2-3 cm longitudinal incision generally between radial artery and cephalic vein. Radial artery was exposed by incising the antebrachial fascia just medial to the radius. Two veins accompany the artery and should be dissected away during arterial isolation. The superficial radial nerve and its medial/lateral branches course between the cephalic vein and radial artery in the area.

Exposure of the ulnar artery is by coursing beneath the superficial flexor muscles in the proximal forearm, emerging near the ulnar border at the point midway between the elbow and the wrist. In the distal forearm, the ulnar artery course just beneath the antebrachial fascia and is easily exposed through a longitudinal incision placed radial to the flexor carbi ulnaris. The palmar branch of the ulnar nerve courses the superficial to the antebrachial fascia and should be preserved during arterial exposure

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

- Bovine arch with left common carotid/left subclavian have common origin
- Vertebral artery directly off the aortic arch
- Aberrant right subclavian where innominate becomes right CCA and right subclavian distal to last branch on left side passing behind esophagus to supply the right arm

## Epidemiology, etiology, and diagnostic evaluation

**How does evaluation of upper extremity ischemia differentiate from lower extremity ischemia?** (Shuja, n.d.)

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

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

Anatomic Location:

- Large vs. Small Vessel

Disease Process:

- Vasospastic or occlusive. Vasospastic disease is more responsive to pharmacologic management while occlusive requiring endovascular/surgical management.

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

Diagnostic Evaluation

1. Detailed H+P evaluation (pulse palpation, auscultation at supraclavicular/infraclavicular fossa may reaveal a bruit concerning for subclavian artery stenosis, upper extremity neurovascular/skin exam)

2. Brachial/forearm blood pressures and if suspected claudication, measured at rest and 2-5 minutes after exercise. Look for a gradient of >20 mmHg is considered significant
3. Some or all of 6 P's of acute limb ischemia with symptoms occurring within 14 days are deemed acute
4. Doppler insonation of radial, ulnar, palmar, and digital arteries
5. Vascular Lab Evaluation
    1. Segmental Pressure Measurements
    2. Duplex Ultrasound (look for large vessel occlusive disease)
6. Other Imaging
    1. CTA/MRA
7. Clinical Lab tests
    1. Inflammatory disorders-CBC, ESR, ANA, RF
    2. Hypercoagulable screening

# Operations/Procedures

**What are some indications for carotid-subclavian bypass?**

1. Atherosclerosis
2. Staged revascularization prior to TEVAR for aneurysmal disease requiring coverage of the LSA

**How does the exposure differentiate in transposition vs bypass?**

Exposure (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?**

Complications (Voigt et al., 2019)

1. Phrenic nerve palsy (most common) - most often managed conservatively.
2. Recurrent laryngeal palsy
3. Lymphatic leak
4. Neck hematoma

**When carotid-subclavian bypass compared to transposition?**

1. Vertebral artery takes origin from the subclavian artery in a very proximal position or is dominant over the contralateral side, then bypass preferred. (Morasch, 2009)
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)

## Vaso-occlusive disease

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

- Etiology: Atherosclerosis is the most common cause of subclavian/axillary occlusive disease. Left SCA > Right involvement. Less common causes include Takayasu disease, giant cell arteritis, or arterial TOS
- Symptoms: Upper extremity arm/hand ischemia or neurologic symptoms due to subclavian-vertebral steal. Because significant collaterals, minimal pain on exertion even with subclavian occlusion

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

- Etiology: MCC of brachial artery occlusion is cardiac origin embolus. Atherosclerosis RARELY affects the brachial artery. Distal axillary/proximal brachial stenosis can be from repetitive trauma from crutch use.
- Forearm occlusive disease can be seen in advanced ESRD/DM where calcific atherosclerosis of radial/ulnar arteries is present. Less common causes include Beurger disease or Raynaud Phenomenon

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

- SCA Occlusive Disease

  - Endovascular with balloon expandable stent via femoral or ipsilateral brachial artery.

    @*chatterjeeAngioplastyAloneAngioplasty*2013; @*bradaricEndovascularTherapyStenoOccl*

    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
    * Ipsilateral CCA to subclavian artery (bypass or transposition)
    * Contralateral CCA (anterior or retropharyngeal)

- Brachial/forearm Occlusive disease

  - Endovascular: PTA evidence is anecdotal with stents for lesions unresponsive to PTA or dissection following angioplasty
  - 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

## Vasospastic Disorders

**What is Raynaud's and what causes it?** (Landry, 2019; Shuja, n.d.)

- Exaggeration of normal physiologic response with episodic pallor or cyanosis of the fingers caused by small digital artery vasoconstriction occurring in response to cold or emotional stress. There is an abnormality with sympathetic nervous system, resulting in a multifactorial problem involving a combination of vascular, neural, and humoral factors.

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

- 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.

- 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. Calcium channel blocker (nifedipine) has been the most effective and losartan has also been beneficial. Fluoxetine (SSRI). Other drugs include alpha blocker, sildenafil, reserpine, cilostazol, captopril. NOT GOOD OUTCOMES IN PATIENTS WITH ARTERIAL OBSTRUCTION
2. Surgical-thoracic sympathectomy (used for treatment of digital artery vasospasm/digital ischemic ulceration). For vasospasm, thoracic sympathetcomy is initially successful, but symptoms return generally within 3-6 months.
3. Immunosuppression/immunomodulation for connective tissue disorders associated with secondary Raynaud phenomenon

## Ergotism

**What is Ergotism?** (Stanley et al., 2014)

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

- Modern day is rare

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

- Ergotamine is chemically like endogenous catecholamines/indolamines and when applied clinically, it behaves as an agonist to alpha-adrenergic, sertoninergic, and dopaminergic receptors. Despite limited bioavailability, vasocontrictive 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)

Treatment:

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

## Buerger's Disease

**How is Buerger's disease categorized?** (Jack L Cronenwett et al., 2020)

- Non-atherosclerotic, segmental, inflammatory disease of small/medium sized arteries in distal extremities of tobacco users distinct from either atherosclerosis of immune arteritis

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

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

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

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

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

- Distal, but not proximal arterial disease (palpable brachial/popliteal but absent/reduced at ankle or wrist)
- DBI<0.6 and flat/reduced digital waveforms
- CTA/MRA/DSA-characteristic corkscrew collateral

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

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

## Large Artery Vasculitis

**What are common characteristics for patients who are suspected to have a large vessel vasculitis?** (Shanmugam, 2019)

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

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

1. Takayasu arteritis
    1. Aorta and primary
    2. Young patients <20 years and female in 80-90% of cases, Asian populations
    3. Criteria (ACR)
        1. Onset <40 years
        2. Claudication of an extremity

3. Decreased brachial pulse
4. >10 mmHg SBP between arms
5. Bruit over subclavian arteries or aorta
6. Arteriographic evidence of narrowing/occlusion in aorta/primary branches/or large upper/lower extremity arteries

2. Giant cell arteritis
    1. Aorta and main branches, but pre-dilection for carotid artery branches
    2. Diagnosis:
        1. Age at disease onset > 50 years
        2. New headache
        3. Temporal artery abnormality
        4. Elevated ESR (>50)
        5. Abnormal 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
    5. Arteriography/MRA/CTA/PET may be used to assess large vessel involvement

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

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

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

- Medical-steroid therapy. In as many as 50% of patients who have a large vessel vasculitis refractory to glucocorticoid therapy alone, patients will trial immunomodulators or cytotoxic truxs (ie methotrexate, azathioprine, mycophenolate, tocilizumab, or leflunomide)
- Intervention-once remission, treatment of symptomatic arterial lesions 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 avoid due to pathology involved)
    * Upper extremity bypass with autogenous vein to the brachial artery
    * Aortic aneurysms should be managed with open surgery

## Aneurysmal Disease

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

Etiology/Pathology:

- Degenerative (atherosclerotic or due to aberrant right subclavian with degenerative changes in proximal subclavian known as "Kommerell diverticulum")
- Traumatic (blunt, penetrating, iatrogenic with attempted catheter placement)
- Thoracic outlet obstruction

Presentation

- 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 thromboembolic phenomenon, brachial plexus compression, hoarseness from right recurrent laryngeal nerve compression
- Dysphagia from esophageal compression in aberrant right subclavian artery

**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

Treatment:

- Open Repair-resection/endoaneurysmorrhaphy with end to end (small aneurysms) or interposition prosthetic graft

  - 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
  - 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.

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

Etiology/Pathology:

- 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?**

Diagnosis:

- Ultrasound
- CTA/MRA of upper extremity

Treatment:

- 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

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

Etiology/Pathology:

- False aneurysms secondary to repetitive trauma
- Iatrogenic complications
- 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 compressions
- Hand/digital ischemia from thrombosis/distal embolization

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

Diagnosis:

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

Treatment:

- 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.

## Occupational Vascular Disease

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

## Hand-Arm Vibration Syndrome

Etiology:

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

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

Diagnosis

- 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

Treatment

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

## Hypothenar hammer syndrome

Etiology:

- Repetitive use of palm of hand in activities that involve pushing, pounding, twisting
- Name comes from reports of mechanics, factory workers, carpenters or laborers who habitually use there hands as a hammer are ad 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

Diagnosis:

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

Treatment

- Conservative-smoking cessation/hand protection/cold avoidance
- Medical-calcium channel blockers/antiplatelet
- Surgical (severe digital ischemia/aneurysm)-ligation if adequate collateral or interposition vein graft

## 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

### Sports Medicine

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

Overview

- 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

## Vascular Trauma-Upper Extremity

**This is discussed in detail here:** @ref(vascular-trauma), **so we will go over some important specifics for upper extremity vascular injury.** (Kauvar & Kraiss, 2020)

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

Mechanism and Pattern

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

Diagnostic Considerations

- 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

Surgical Considerations

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

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

Mechanism and Pattern

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

Diagnostic Considerations

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

Surgical Considerations

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

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

Mechanism and Pattern

- Associated with significant soft tissue pattern

Diagnostic Considerations

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

Surgical Considerations

- 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

## Compression Syndromes

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

## 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

Pathophysiology

- 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

Treatment

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

## Humeral head compression of axillary artery

Anatomy:

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

Etiology/Pathophysiology:

- 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

Diagnosis:

- Provocative maneuvers with impedance of flow through axillary artery on ultrasonography

- Arteriography with rest and provocative position

Treatment:

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

# Thoracic Outlet Syndrome

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

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

Types: Neurogenic, Venous and Arterial

- vTOS – 2-3%
- aTOS – 1%
- nTOS – >95% (Humphries & Freischlag, 2019b)

## Anatomy

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

Anatomy from anterior to posterior

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

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

1. Interscalene Triangle
2. Costoclavicular Passage (Gary G Wind & R. James Valentine, 2013)
3. Subcoracoid Space (Gary G Wind & R. James Valentine, 2013)

**Interscalene Triangle:**

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

**Anterior Scalene:**

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

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

**Middle Scalene:**

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

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

**The First Rib:**

- The broadest and flattest of the ribs and is an 'Atypical Rib.'
- The upper surface of the first rib has the scalene and quadrangular tubercles for attachments of the anterior and middle scalene muscles respectively. There are also three grooves for the Subclavian Vein, artery and the Lower Trunk of the Brachial Plexus.
- The Inferior Surface is smooth and inferior and medially has an attachment for the suprapleural membrane, Sibson's fascia AKA scalenus minimus, which is tethered to the C7 vertebrae.
- This is the passage of the subclavian vein largely as it emerges through the tight space created by the clavicle, the subclavius muscle and the costoclavicular ligament and also more posteriorly this can also compress the artery and nerves as the space can also be narrow in relation with the scapula and subscapularis. (Gary G Wind & R. James Valentine, 2013)

**Subclavius Muscle:**

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

**The Coracoid Process:**

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

**Pectoralis Minor Muscle:**

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

**Clavipectoral Fascia:**

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

**Phrenic Nerve Anomaly:**

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

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

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

nTOS

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

aTOS

- Cervical Rib and Anomalous First Rib
- Scalene Triangle compression

vTOS

- Costoclavicular Passage
- Subcoracoid Space

## Diagnosis and Evaluation

### Patient History

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

### 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.

### Elveys Test

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

**Non-invasive imaging or vascular lab studies**

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

**Paget Schroetter Syndrome**

- First defined by Hughes in 1949 in reference to Sir James Paget who in a hundred years earlier defined acute arm swelling and pain as possibly related to vasospasm and then von Schroetter who in 1884 attributed to the presentation to subclavian and axillary vein thrombosis. (Humphries & Freischlag, 2019a)
- Now vTOS and Paget Schroetter Syndrome are used synonymously.
- Paget Schroetter Syndrome accounts for 10-20% of all upper extremity deep vein thrombosis. (Sekhar, 2018)

## Rib Resection approaches

| | Advantages | Disadvantages |
|---|---|---|
| Tran saxillary | Cosmetically more appealing as it has a limited hidden scar | • Difficult to visualize the anatomy, dependent on good assistance<br>• Risk of injury to T1 nerve root, phrenic nerve, long thoracic, brachial plexus , subclavian vein and arterial with limited exposure to repair<br>• Not able to approach cervical ribs, scalene triangle or patch vein. |

| | Advantages | Disadvantages |
|---|---|---|
| Suprac lavicular | • Good for scalene triangle access and debulking and cervical rib resection<br>• Required for aTOS if arterial reconstruction necessary | • Unable to decompress venous compression or visualize vein adequately<br>• Cosmetically less appealing |
| Infrac lavicular [@ siracuseI nfraclavi cularFirs tRib2015] | • Good access for venous decompression<br>• Allows for excision of subclavius muscle and costoclavicular ligament | • Unable to expose subclavian artery or decompress brachial plexus.<br>• Difficult to access most posterior aspect of rib<br>• Cosmetically less appealing |
| Parac lavicular | • Useful if mixed etiology TOS to adequately decompression all neurovascular structures | • Requires two incisions one above and below the clavicle |

**Post operative complications**

- Post operative patients with hemodynamic instability and ipsilateral effusion on xray should go back to OR for exploration and hemorrhage control. Rinehardt et al. (2017)
- Chyle leak often managed with adequate drainage and medium chain fatty acid diet.

## vTOS

**Demographics**

- Incidence: 2/100,000 persons
- Age: 18 years to 30 years (Illig & Doyle, 2010)
- M>F

**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 & 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 & Freischlag, 2019a)

**History**

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

**Goals of therapy for vTOS**

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

**Initial management strategy**

- As per ACCP Guidelines: Initial management is anticoagulation regardless of etiology. (Kearon et al., 2016)
  - The limitations of anticoagulation alone are that the slow recanalization of the thrombus may lead to eventual valvular damage and intravenous scarring. (Sekhar, 2018)

  - Thrombolysis has been considered superior to anticoagulation alone in minimizing valvular damage due to residual clot. (Urschel & Patel, 2008)
  - Systemic Lysis – non favored due to risk of intracranial hemorrhage. (Grunwald & Hofmann, 2004)
  - Catheter Directed Lysis (CDT) – carries a lower risk of intracranial hemorrhage.
  - Patient should be maintained in a compression sleeve until definitive decompression can be performed.
- Optimal timing of CDT
  - Within 14 days of onset of thrombosis. Excellent results have been reported following CDT if initiated before 14 days. (Wilson et al., 1990)
- Surgical indications for vTOS
  - 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.

    $$@melbyComprehensiveSurgicalManagement2008$$

  - 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)

**Controversy around vTOS**

- 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 & Freischlag, 2019a)

- Decompression during the same admission, as the thrombolysis, with the main benefit being to reduce the risk of re-occlusion. (Humphries & Freischlag, 2019a; Molina et al., 2007)
- Post decompression venography and treatment 2 weeks post rib resection may help to prevent recurrence and long term vein patency. (Chang et al., 2012)

**Landmark papers regarding vTOS and what are the take home messages**

1. Lugo J et al – Acute Paget Schroetter syndrome: does the first rib routinely need to be removed after thrombolysis? Annals of Vascular Surgery 2015 (Lugo et al., 2015)
    1. Systematic literature review analysis. Patients divided into three groups
        1. First Rib resection (FRR) – n=448
        2. First Rib resection and endovenous venoplasty (FRR and PLASTY) n=68
        3. No further intervention after Thrombolysis n=168
    2. Symptom relief after initial follow up more likely in FRR (95%) and FRR and PLASTY (93%) compared to no rib removed (54%) – $p<0.0001$
    3. Results showed superior patency with FRR and PLASTY and FRR compared to anticoagulation alone.
    4. Conclusion was that patients are more likely to experience greater long-term results with FRR compared to no FRR.
2. Sajid MS et al – Upper limb vein thrombosis: a literature review to streamline the protocol for management. Acta Haematology 2007 (Sajid et al., 2007)
    1. a comprehensive review identifying the key papers on this topic and allows for a clear view of the best management strategy.
3. Vemuri, C., Salehi, P., Benarroch-Gampel, J., McLaughlin, L. N., & Thompson, R. W. (2016). Diagnosis and treatment of effort-induced thrombosis of the axillary subclavian vein due to venous thoracic outlet syndrome. *Journal of Vascular Surgery: Venous and Lymphatic Disorders*, *4*(4), 485–500. (Vemuri et al., 2016)
    1. Comprehensive summary of management strategy for effort induced thrombosis.

## aTOS

### Presentation

- Most common: Hand ischemia due to arterial compression or microembolization with subclavian artery aneurysm and pulsatile supraclavicular mass (Boll & Valentine, 2019)
- Less common: Exertional pain, unilateral Raynaud's Phenomena, retrograde embolisation and neurological symptoms
- Clinical Examination
  - Audible Bruit / Palpable thrill over the supraclavicular fossa
  - Pulsatile mass
  - Distal ischemic lesions in the distal hand – Splinter hemorrhages
  - Positive Adson Test
- Differential Diagnosis
  - Trauma
  - Primary and Secondary Raynaud's Phenomena
  - Small Vessel Vasculitis
  - Connective Tissue Disorders
  - Thromboangiitis Obliterans
  - Arterial Embolisation – Aortic or Central Source
  - Radiation Arteritis
  - Atherosclerotic / Dissection causes
- The different anatomical abnormalities causing aTOS (Boll & Valentine, 2019)
  - Cervical Rib (60%)
  - Anomalous First Rib (18%)
  - Fibrocartilaginous band (15%)
  - Clavicular Fracture (6%)
  - Enlarged C7 transverse process (1%)

### Scher Staging of aTOS

- Stage 0: Asymptomatic
- Stage 1: Stenosis of Subclavian Artery with minor post stenotic dilatation with no intimal disruption

- Stage 2: Subclavian artery aneurysm with intimal damage and mural thrombus
- Stage 3: Distal embolisation from subclavian artery disease

### Diagnosis

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

### Management considerations with aTOS

- Symptomatic patients are generally indicated for treatment. Unlike asymptomatic patients in whom it may be appropriate to manage conservatively. (Boll & Valentine, 2019)
- 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.

## nTOS

### 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

### Presentation of nTOS

- Symptoms (Sadeghi-Azandaryani et al., 2009; Sanders et al., 2007)
  - Paraesthesia (98%)
  - Trapezius pain (92%)
  - Neck, shoulder or arm pain (88%)
  - 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

**The role of the Vascular Surgeon with nTOS**

Often these patients have already seen multiple specialists and physiotherapists.

- 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. (Lum et al., 2012; Salhan et al., 2016)
- 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.

# Abdominal/Iliac/Peripheral Aneurysms

**30 Mar 2021:** *Mia Miller, MD and Julie Duke, MD; University of Minnesota*

## Pathogenesis, presentation and risk factors

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

- Defined as a localized dilation of an artery to a diameter greater than 50% (1.5x) of its normal diameter. It is generally accepted that >3cm in adults is considered aneurysmal for the abdominal aorta.
- AAAs can be described as:
  - Infrarenal – distal to the renal arteries with normal aorta between the renal arteries and the aneurysm origin.
  - Juxtarenal – aneurysm extends to the renal arteries but does not involve them
  - Pararenal – aneurysm involving the origin of at least one of the renal arteries
- Estimated 1.1 million Americans have AAAs, which equates to a prevalence of 1.4% in 50-84 year old general population.
- 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)
  - Conversely, if a patient is first found to have a AAA, there is an 11% chance of having associated popliteal artery aneurysms >15mm.

(Tuveson et al., 2016)

- Another study showed a rate of femoral-popliteal aneurysms in AAA patients is approximately 14%. (Diwan et al., 2000)
- This association stresses the importance of a good physical exam when evaluating a patient with a AAA and is commonly tested on exams.

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

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

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

- Risk factors for AAAs are similar to the risk factors for occlusive atherosclerosis and include age, tobacco use, hypertension, male gender and hypercholesterolemia.
- It has been found that diabetes is protective for AAA progression and rupture.
- Cigarette smoking is the single most important modifiable risk factor to prevent occurrence and growth of AAAs. Smoking increases the rate of growth by 35% for abdominal aortic aneurysms.
- Medical therapy has been studied with disappointing results. Beta-blockers and ACE/ARB inhibitors have been studied but have not shown any effect on growth of AAAs.

- Fluoroquinolones
  - In a recent study just published in JAMA Surgery this January, the group at UNC showed an increased short-term risk of developing an aortic aneurysm with fluoroquinolone use. (Newton et al., 2021)
  - They reviewed all prescription fills for fluoroquinolones or comparative antibiotics from 2005-2017.
  - This included >27 million US Adults aged 18-64 years old with no history of aneurysms.
  - 18% of the prescriptions were fluoroquinolones.
  - Fluoroquinolones were associated with increased incidence of aortic aneurysms. Compared to the other antibiotics, fluoroquinolones were associated with a higher 90-day incidence of AAA and iliac aneurysms as well as more likely to undergo aneurysm repair.
  - They recommended that fluoroquinolone use should be pursued with caution in all adults, not just high risk individuals, and they recommended broadening of the warnings from the FDA.
- Fluoroquinolones playing a role in dissections and aneurysm formation is often a highly tested question

**What is the dreaded complication of AAA?** (Moore et al., 2019)

- 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.
- 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):
  - 4 - 5.4cm -> 0.5-1%.
  - 6 - 7cm -> 10%
  - 7 – 8cm -> 19-35%
- Newer data suggests the true rupture risk per year is decreasing with time.
- In a study from the UK published in JVS in 2015, the rupture risks were far lower than previously reported and what is documented in most textbooks. (Parkinson et al., 2015)

  - This systematic review of more recently published data mostly from 1995 to 2014 included a total of 11 studies reviewing 1514 patients. The cumulative yearly rupture risks identified in this study were as follows:
    * 5.5 - 6 cm -> 3.5%
    * 6.1 - 7 cm -> 4.1%
    * >7 cm -> 6.3%
  - Previously published data with meta-analyses from 1970s-1990s reported rupture rates of 3.3%, 9.4% and 24%, respectively, compared to 3.5%, 4.1% and 6.3% in the most recent data.

- 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).

# Evaluation and Diagnosis

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

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

## Imaging

- Ultrasound, when feasible, is the preferred imaging modality for aneurysm screening and surveillance.
  - The Society for Vascular Surgery (SVS) recommends a one-time ultrasound screening in men and women ages 65 to 75 years with either a history of smoking or a family history of AAA, as well as men and women over the age of 75 with a smoking history in otherwise good health who have not previously undergone screening. (Chaikof et al., 2018) Recommended intervals for surveillance imaging:
    * 2.5 – 2.9 cm -> 10 years
    * 3 - 3.9 cm -> 3 years
    * 4 - 4.9 cm -> 1 year
    * 5 - 5.4 cm -> 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.

# Management

**What are the indications for repair?** (Moore et al., 2019)

- The current recommendation to repair a fusiform aneurysm is 5.5cm for men (Level 1, Grade A evidence), 5.0cm for women as they have a higher risk for rupture, and rapid growth (>5mm over 6 months). (Chaikof et al., 2018)
- For saccular aneurysms, the SVS practice guidelines recommend elective repair (Level 2, Grade C evidence). (Chaikof 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?** (Fairman, 2019; Moore et al., 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.
  - 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 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 arrythmia -> 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%). (Greenhalgh, 2004)
    - The DREAM trial, a multicenter randomized trial from 2000-2003, compared open repair with EVAR in 345 patients with a reduction in operative mortality (4.7% vs 9.8%) with the majority of complications accounted for by pulmonary issues. (Prinssen et al., 2004)
- This early survival benefit with EVAR over open repair disappears by the third postoperative year.
    - The Open vs Endovascular Repair (OVER) trial included 881 patients from 42 VA centers randomized to either EVAR or open repair. This demonstrated that perioperative mortality was improved in the EVAR

group (0.5% vs 3.0%), yet no statistically significant difference was seen in mortality at 2 years (7.0% vs 9.8%). (Lederle et al., 2019)

  - Late mortality seems to be higher in EVAR due to ruptures from endoleaks that do not occur in open repair. (Rajendran & May, 2017)

- Reviewing the anatomic criteria for traditional EVAR may rule out EVAR as an option in some patients. These criteria vary slightly depending on the particular device being used.
  - Neck
    * A neck length of at least 10-15mm from the renal arteries to the aneurysm start with a diameter of 18-32mm.
    * It is important that the neck is relatively free of thrombus or calcification to decrease the risk of endoleaks.
    * More complex options like fenestrated EVAR are available for shorter necks but will not be discussed in 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.

## EVAR

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

- EVAR now accounts for approximately 70-80% of elective abdominal aortic aneurysm repairs and 65% of iliac aneurysm repairs in the United States and many other countries.

- Performed in the operating room or IR suite with a fixed or portable C-arm
- Anesthesia
  - Regional block, local anesthesia or general anesthesia depending on surgeon preference and patient risk
- Groin access and short sheath placement
  - Percutaneous - Closure devices are introduced prior to inserting the large sheaths containing the stent-grafts
  - Cut-down
- Pigtail catheter is used to perform an aortogram of the abdominal aorta and iliac arteries
- The renal artery orifices are marked. If there is any concern about good visualization, IVUS (intravascular ultrasound) can be used to assist.
- Systemic heparin is given
- Bilateral femoral sheaths are exchanged over a stiff wire for the necessary sheaths required for the device size chosen.
  - Main trunk and ipsilateral limb sheath on one side
  - Contralateral limb sheath on the other side
- The main body is positioned in the proximal neck and a repeat angiogram is commonly performed to confirm the positioning of the device at the desired level just below the lowest renal artery. It is best to position the main body so that the gate is directed at the simplest angle to cannulate.
- The main body is deployed to the point where the gate is opened
- Contralateral limb gate cannulation is performed using a wire and directional catheter.
- Once in the gate, a pigtail catheter is formed within the main body and must be able to spin freely 360 degrees to confirm placement within the endograft
- The contralateral limb is introduced and deployed taking care to preserve flow to the internal iliac artery.
- The remainder of the main body is deployed and iliac extensions deployed if required.
- The stent graft is ballooned at the neck, within the gate, at the bifurcation, and distal iliac seal zones.
- An aortogram, usually multiple in different views, is performed to exclude any endoleaks.

- The sheaths are removed, and the groin sites are closed using Perclose devices if performed percutaneously, or primary repair if open cutdown performed.
- Check pedal 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.

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

- 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.

**Endoleaks**

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

1. Type I
   - A leak at the graft ends secondary to inadequate seal proximally (1a) or distally (1b)
   - If identified intraoperatively, Type I endoleaks require attention with further balloon angioplasty, proximal or distal extension, or endoanchors.
   - If seen in follow up surveillance, intervention is necessary.
2. Type II
   - Sac filling secondary to retrograde filling via a branch vessel off of the aneurysm sac such as a lumbar artery or the IMA
   - If identified intraoperatively, this typically does not need to be addressed in the OR.
   - Typically, type II endoleaks spontaneously thrombose and therefore can be observed.

- 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.
- It is common to continue monitoring even if there is persistent flow as long as there is no aneurysm sac growth.

3. Type III
   - Separation of graft components
   - Usually identified in follow-up surveillance and necessitates intervention.
4. Type IV
   - Secondary to a porous graft which typically does not occur any longer as endograft material and devices have improved. If seen, no intervention is needed at the time, and they usually thrombose on their own.
5. Type V
   - Increasing aneurysm sac size with no identifiable endoleak. Commonly referred to as endotension.
   - Usually necessitates graft explantation and open repair or re-lining of the graft.

## Open Repair

**Now we can move onto open repair. Describe an open infrarenal aneurysm repair.** (Gary G Wind & R. James Valentine, 2013; Moore et al., 2019)

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

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

- A tube graft or bifurcated graft depending on the patient's anatomy and aortic diameter is chosen. Dacron or PTFE grafts are most common, and the choice depends on physician preference. This is anastomosed proximally in a continuous fashion.
- Once complete, the graft is flushed forward to flush out any thrombus. The graft is then clamped and the aortic clamp removed to test the anastomosis. Repair if needed.
- The distal anastomosis is completed to the aorta or bilateral iliac arteries depending on extent of the aneurysm.
- The graft is flushed forward prior to completion to remove any thrombus within the graft. The anastomosis is completed and clamps removed.

12. Hypotension may occur at this point from re-perfusion of the lower extremities and pelvis. Anesthesia should be notified that unclamping will occur soon prior to completion of the distal anastomosis to allow for fluid resuscitation in preparation.
    - The graft can be slowly unclamped or partially clamped to assist with blood pressure management during this time. You can also place manual pressure on the iliac arteries or femoral arteries to slowly release flow and avoid significant hypotension.
13. Next, the IMA must be addressed. The IMA orifice is identified within the aneurysm sac.
    - Chronically occluded or pulsatile back bleeding -> ligate.
    - Anything between occlusion and strong pulsatile back bleeding requires further evaluation. This should be performed at the end of the case after the internal iliacs have been reperfused. Methods to measure perfusion:
        * Place vessel loops or micro bulldog on IMA and assess the sigmoid colon. If there is a poor doppler signal on the antimesenteric border of the sigmoid colon, the IMA should be reimplanted.
        * Insert blunt-tip needle through the IMA orifice and pull vessel loop around needle to secure and connect to a transducer. Pressure less than 35 mmHg requires reimplantation. (Hoballah, 2021)
    - The Carrel patch technique involves excising a circular button of the aortic wall around the IMA and anastomosing it to the graft wall.
    - Newer studies have shown that IMA reimplantation does not eliminate the risk of ischemic colitis after open AAA repair. In

a study out of George Washington University in DC published in JVS in 2019, there was still significant risk of ischemic colitis rates with IMA reimplantation. (Lee et al., 2019)

* Using NSQIP data collected prospectively and studied retrospectively
* Out of 2397 patients undergoing AAA from 2012-2015, 135 patients (5.6%) had ischemic colitis.
* 672 patients were evaluated further after exclusion criteria applied (suprarenal clamp, emergent or ruptured, occluded mesenteric vessels)
* Of these, 637 patients had IMA ligation, 35 had IMA reimplantation
* Reimplantation was associated with - More frequent return to the OR (20% vs 7.2%), Higher rates of wound complications (17.1% vs 3%), Higher rates of ischemic colitis (8.6% vs 2.4%)

– Difficult to interpret impact of revascularization of IMA on ischemic colitis rates, due to selection bias, but should be noted that patients who require revascularization still may experience colon ischemia.

14. To finish, the aneurysm sac is then closed over the graft to protect the viscera, and the retro-peritoneum is reapproximated. Occasionally, a vascularized omental pedicle flap may be used to separate the graft from the duodenum to prevent an aorto-enteric fistula if the peritoneum cannot be closed securely.

- Steps for the retro-peritoneal approach:
  – Positioned semi-lateral with the left side up with bilateral groins exposed for femoral artery access. This is done in a lazy lateral position where the 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.

### Complications

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

- Myocardial dysfunction which is usually secondary to cardiac ischemia or hemorrhage.
- Abdominal compartment syndrome secondary to coagulopathic bleeding postoperatively or third spacing of fluids can cause abdominal compartment syndrome requiring emergent laparotomy. Unexplained oliguria, difficulty maintaining adequate ventilation, and hypotension with significant abdominal distension is concerning for abdominal compartment syndrome. A sustained bladder pressure $> 20$ 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, therefore, one must keep a heightened suspicion for this in the post-operative period.
    - 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.
- Renal failure can occur due to suprarenal aortic clamping, atheromatous embolization or hypotension causing acute tubular necrosis (ATN).
- Postoperative ileus is common. Duodenal obstruction from dissection of the ligament of Treitz can mimic a gastric outlet obstruction.
- Ischemic colitis of the left colon and rectum is the most serious gastrointestinal complication, and the 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
        - 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.

**Postoperative Surveillance**

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

- That is a great question because it highlights why open repair has continued to be so important, especially for young, healthy patients.
- Post-operative surveillance is necessary in the immediate post-operative period for open repair to evaluate incisions. Follow-up is only needed every 5-10 years, unless the patient becomes symptomatic.
- In contrast, EVAR patients require a strict postoperative surveillance regimen to allow for detection of endoleaks, aneurysm sac expansion, stent fracture, limb kinking and material fatigue.
  - CT scans at 1-, 6- and 12-month intervals initially then annually are recommended which raises concerns related to cost, cumulative radiation exposure, and contrast administration.
  - Some physicians may elect to use ultrasound for surveillance with CTA prompted if an endoleak is identified or the sac is enlarged, particularly in patients with stable aneurysms.
  - The long-term follow-up is often inconsistent and a study of 19,962 Medicare beneficiaries undergoing EVAR from 2001 to 2008 showed that 50% of patients were lost to annual imaging follow-up at 5 years after surgery.(Schanzer et al., 2015)
- Some patients will elect for open repair to avoid frequent surveillance if they are a candidate for both, while other patients will select endovascular management to avoid the short-term effects like longer hospitalizations,

post-operative pain, and longer recovery time to baseline functioning in open surgery.

## 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?** (Lindsay, 2019; Moore et al., 2019)

- Ruptured AAAs have declined secondary to improved medical management, decreased rates of smoking and superior diagnostic imaging and surveillance.
- Traditionally, it has been taught that 50% of ruptured AAAs die in the field and of those remaining, 50% will die in the hospital. With time, the in-hospital mortality rate has decreased.
  - In one study out of Finland, of 712 patients with ruptured AAAs from 2003-2013, 52% died prior to arrival to the hospital. Of those that were offered surgery, 67% of patients were alive at 30 days indicating a mortality rate of 33%. (Laine et al., 2016)
- Diagnostic triad on presentation:
  - Pain, syncope and known or palpable AAA.
- When a ruptured AAA is suspected or diagnosed, permissive hypotension is key in the initial management before surgery.
  - Allowing systolic arterial pressures of 50-70 mmHg as long as the patient is mentating appropriately.
  - Limits internal bleeding which further limits loss of platelets and clotting factors.
- Initial management involves many considerations like patient stability, patient's anatomy and the surgeon's experience with either open or endovascular repair.
- Due to the developments of endovascular techniques, it is ideal to have a CTA prior to the operating room to determine if the patient is a candidate for an EVAR.
- There are two options for expedient aortic control in an unstable patient with a ruptured aneurysm.
  - Open supraceliac aortic clamping
    - Achieved by retracting the stomach caudally, entering and dividing a portion of the gastrohepatic ligament, reaching under and medial to the caudate lobe, dividing the pars flaccida, and identifying the spine. The aorta lies to the patient's left of the

spine and is bluntly dissected anteriorly and laterally for aortic clamp placement.

* Another method of supraceliac exposure and control is to mobilize and reflect the left lobe of the liver, sweep the esophagus to the patient's left, divide the right crus of the diaphragm and bluntly dissect both sides of the aorta then apply the clamp.

* A nasogastric tube can help identify the esophagus when placing this clamp to ensure the esophagus has been swept to the patient's left and protected.

* The clamp should be moved down to the desired position for repair (supra or infrarenal depending on anatomy of the aneurysm neck) to decrease ischemia time to visceral vessels as soon as possible.

– Percutaneous occlusive aortic balloon

* Gain percutaneous access and place an occlusive aortic balloon for stabilization in the distal thoracic aorta. This will require a long support sheath, usually 12fr in size, to prevent distal migration of the occlusive balloon.

- EVAR has been used increasingly to treat ruptured AAAs and offers many theoretical advantages over open repair.
- Less invasive, eliminates risk of damage to periaortic and abdominal structures, decreases bleeding from surgical dissection, minimizes hypothermia and third space losses, and lessens the requirement for deep anesthesia.
- EVAR has been deemed superior to open repair for the treatment of RAAA in many studies.

  – In a study out of UVA published in JVS in August 2020, they looked at ruptures in the VQI database from 2003-2018. This resulted in 724 pairs of open and endovascular pairs after propensity matching. (Wang et al., 2020)

    * There was a clear advantage of endovascular compared to open repair in patient's with suitable anatomy.

    * Length of stay was decreased with 5 vs 10 days in open. 30 day mortality was much lower at 18% vs 32%. Major adverse events like MI, Renal failure, leg ischemia, mesenteric ischemia, respiratory complications were much lower in the EVAR group at 35% vs 68% in the open group.

    * All cause 1 year survival was much higher with EVAR at 73% vs 59% in the open group.

- Despite improved RAAA results with EVAR, conversion from EVAR to open AAA repair appears to have the most unfavorable outcomes in terms

of mortality.

- Conversions can be early or late and are due to access-related problems, errors in endograft deployment, graft migration, persistent endoleak, graft thrombosis, or infection.
- In a study evaluating 32,164 patients from NSQIP with 300 conversions (7,188 standard open repairs and 24,676 EVARs), conversion to open repair was associated with a significantly higher 30-day mortality than standard open repair (10% vs 4.2%) and EVAR (10% vs 1.7%). In addition, conversion patients compared to standard open patients were more likely to undergo new dialysis (6.0% vs. 3.5%), cardiopulmonary resuscitation (5.3% vs. 1.9%), postoperative blood transfusion (42.3% vs. 31.6%), and have a myocardial infarction (5.0% vs. 2.2%). (Ultee et al., 2016)

# Lower Extremity Occlusive Disease

## Pathophysiology

24 Aug 2020: Nedal Katib and Danielle Bajakian

**What is Peripheral Arterial Disease (PAD) and what does it encompass?**

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

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

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

The underlying pathophysiology which results in occlusive arterial disease of the lower limb has somewhat evolved over the last 50 years. While atherosclerosis remains, the main pathological process resulting in occlusive disease, smoking related atherosclerosis has in the last 30 years been confounded with the rising incidence of diabetes and in addition an aging population with progressive arterial disease.

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

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

Modifiable:

1. ***Smoking***

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

2. ***Diabetes , Metabolic Syndrome and Insulin Resistance***
   - Diabetes Mellitus, after smoking, is the most significant modifiable risk factor for developing peripheral arterial disease. Both insulin resistance and hyperinsulinemia are independent risk factors for developing peripheral arterial disease.
   - The Odds Risk for developing PAD in patients with DM ranges from 1.89 to 4.05.
   - An increase in HbA1C by 1% correlates with a 28% increase risk of developing PAD. (Adler et al., 2002)
3. ***Hypertension***
   - The **most common** cardiovascular risk factor worldwide.
   - The Incidence of PAD increases to 2.5-fold in patients with Hypertension. (Kannel & McGee, 1985)
4. ***Dyslipidemia***
   - A strong association has long been identified as a risk factor for cardiovascular disease.
   - 25% cardiovascular event reduction for each 39 mg/dL (1mmol/L) reduction in LDL. (Group, 2007)

Non Modifiable:

1. **Age**
   - Age is identified as a risk factor for PAD regardless of gender.
   - Prevalence of PAD increases with age: 15% $>$ 70 years of age.
2. **Gender**
   - The Framingham Study has found that the risk of developing PAD is doubled in men.
3. **Ethnicity**

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

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

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

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

- Increasing Incidence of Diabetes world-wide. (Boulton et al., 2005)
  - 2.8% in 2000, 4.4% in 2030
  - 25% of patients with diabetes develop a DFU at some stage in their lives
  - Limb Loss every 20 seconds world-wide to Diabetes

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

- Sensory Neuropathy, Motor Neuropathy and Autonomic Neuropathy
- Structural and Gait abnormalities
- Arterial disease
  - Large Vessel
  - Small Vessel

- Both

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

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

## 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 & Fowkes, 1992).

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

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

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

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

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

| Grade/Category | Clinical Description |
|---|---|
| III/6 | Major Tissue Loss |

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

## History / Clinical Examination

SVS Guidelines:

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

*Grade 1 Level of Evidence A*

- ABPI
- Exercise ABPI
- Ultrasound

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

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

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

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

| **Interpreting ABPI** |
|---|
| >1.4 |
| >0.9-1.39 |
| 0.5-0.9 |
| 0.0-0.5 |

**What is Exercise ABPI studies?**

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

Walking distance has been shown to correlate with level and severity of POAD. (Strandness & Sumner, 1975)

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

| Stenosis Category | Peak Systolic Velocity | Velocity Ratio | Distal Artery Spectral Waveform |
|---|---|---|---|
| Normal | <150 | <1.5 | Triphasic, 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 |
| Occlusion | No Flow – B -mode, Terminal Thump | | |

Adapted from (Stone & Hass, 2019)

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

SVS Guidelines (2015)

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

Other Guidelines:

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

**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)
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 (non-invasively) manage Asymptomatic PAOD based on the SVS Guidelines?**

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

  - The Heart Protection Study (Group, 2007) - this study looked at Statins in patients with PAD but not completely asymptomatic, they had other risk factors such as diabetes, IHD, cerebral disease or hypertension. Without these risk factors Statin therapy benefit unsure.
  - However, the AHA from the Framingham Study does recommend using Statins if 10-year risk based on risk calculators >7.5% (which would be positive if PAD present).

- Exercise and Limb Function
  - No clear evidence that physical therapy improves QoL
- Surveillance
  - No benefit from US surveillance, unclear benefit of ABPI surveillance.

## Management

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

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

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

- Cilostozol use – IC without CHF – 3-month Trial (Grade 2 - A)
  - If unable to tolerate Cilostozol – Pentoxifylline (400mg TDS) (Grade 2 – B)
  - Based on Meta-analysis 26 trials (Stevens et al., 2012)

- Exercise Therapy
  - First Line Therapy recommended SEP: minimum three times / week (30-60 min/session) for at least 12 weeks (Grade 1 Level A)
  - Meta-analysis of 32 RCT's: Placebo versus exercise: Walking Time, Walking Ability, Pain Free Walking and maximum walking distance improves. BUT no difference in ABPI, Mortality or amputation. (Lane et al., 2017)
  - Meta- Analysis of 14RCT's: SEP better than Non-Supervised Programs. (Hageman et al., 2018)

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

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

    - Hybrid recommended for Iliac disease involving CFA. (Grade 1 Level B)
    - Direct Surgical reconstruction (bypass, endarterectomy) in patients with reasonable surgical risk and diffuse AIOD not amenable to endovascular approach, after one or more failed attempts at EVT, or combined occlusive and aneurysmal disease. (Grade 1 Evidence B)

- Infrainguinal Disease:
    - When you look at the historical data comparing all EVT together they are less durable than surgical bypass, especially when there's diffuse or long segments of occlusion/multilevel infra inguinal disease.
    - Most recommendations are based on low level evidence when comparing EVT versus Open Surgery
    - Focal + Not involving SFA origin = EVT (Grade 1 Level C)
    - SFA 5-15cm, self-expanding stent (with or without paclitaxel) (Grade 1 Level B) – NB: (This was in 2015 pre Katsanos Paper)
    - Recommend against infrapopliteal treatment for IC (Grade 1 Level C)
    - Initial Surgical Bypass (with vein: Grade 1 Level a): If
        - Diffuse FP disease
        - Small Calibre <5mm
        - Extensive calcification in SFA
        - Average or low operative risk (Grade 1 Level B)

# Mesenteric Disease

# Renal

# Thoracic Aorta

# Venous Disease

# Vascular Trauma

# Angioaccess

# Complications

# Amputations

# Vascular Lab

# Vascular Medicine

# Endovascular

# Applied Science


Aboyans, V., Criqui, M. H., Abraham, P., Allison, M. A., Creager, M. A., Diehm, C., Fowkes, F. G. R., Hiatt, W. R., Jönsson, B., Lacroix, P., Marin, B., McDermott, M. M., Norgren, L., Pande, R. L., Preux, P.-M., Stoffers, H. E. (Jelle)., & Treat-Jacobson, D. (2012). Measurement and Interpretation of the Ankle-Brachial Index: A Scientific Statement From the American Heart Association. *Circulation*, *126*(24), 2890–2909. https://doi.org/10.1161/CIR.0b013e318276fbcb

Aboyans, V., Ricco, J.-B., Bartelink, M.-L. E. L., Björck, M., Brodmann, M., Cohnert, T., Collet, J.-P., Czerny, M., De Carlo, M., Debus, S., Espinola-Klein, C., Kahan, T., Kownator, S., Mazzolai, L., Naylor, A. R., Roffi, M., Röther, J., Sprynger, M., Tendera, M., … Obiekezie, A. (2018). 2017 ESC Guidelines on the Diagnosis and Treatment of Peripheral Arterial Diseases, in collaboration with the European Society for Vascular Surgery (ESVS). *European Heart Journal*, *39*(9), 763–816. https://doi.org/10.1093/eurheartj/ehx095

AbuRahma, A. F., Avgerinos, E. D., Chang, R. W., Darling, R. C., Duncan, A. A., Forbes, T. L., Malas, M. B., Murad, M. H., Perler, B. A., Powell, R. J., Rockman, C. B., & Zhou, W. (2022). Society for Vascular Surgery clinical practice guidelines for management of extracranial cerebrovascular disease. *Journal of Vascular Surgery*, *75*(1), 4S–22S. https://doi.org/10.1016/j.jvs.2021.04.073

Adler, A. I., Stevens, R. J., Neil, A., Stratton, I. M., Boulton, A. J. M., Holman, R. R., & for the U.K. Prospective Diabetes Study Group. (2002). UKPDS 59: Hyperglycemia and Other Potentially Modifiable Risk Factors for Peripheral Vascular Disease in Type 2 Diabetes. *Diabetes Care*, *25*(5), 894–899. https://doi.org/10.2337/diacare.25.5.894

Allison, M. A., Criqui, M. H., McClelland, R. L., Scott, J. M., McDermott, M. M., Liu, K., Folsom, A. R., Bertoni, A. G., Sharrett, A. R., Homma, S., & Kori, S. (2006). The Effect of Novel Cardiovascular Risk Factors on the Ethnic-Specific Odds for Peripheral Arterial Disease in the Multi-Ethnic Study of Atherosclerosis (MESA). *Journal of the American College of Cardiology*, *48*(6), 1190–1197. https://doi.org/10.1016/j.jacc.2006.05.049

Armstrong, D. L., Fisher, T. K., Lepow, B., White, M. L., & Mills, J. L. (2012). 26Pathophysiology and Principles of Management of the Diabetic Foot. In *Mechanisms of Vascular Disease: A Reference Book for Vascular Specialists*. University of Adelaide Press. https://doi.org/10.1017/UPO9781922064004

Baig, m. shadman, & Timaran, C. H. (2019). 84. Upper extremity aneurysms. In *Rutherford's Vascular Surgery and Endovascular Therapy*. Elsevier Inc.

Balderman, J., Abuirqeba, A. A., Eichaker, L., Pate, C., Earley, J. A., Bottros, M. M., Jayarajan, S. N., & Thompson, R. W. (2019). Physical therapy management, surgical treatment, and patient-reported outcomes measures in a prospective observational cohort of patients with neurogenic thoracic outlet syndrome. *Journal of Vascular Surgery*, *70*(3), 832–841. https://doi.org/10.1016/j.jvs.2018.12.027

Bild, D. E. (2002). Multi-Ethnic Study of Atherosclerosis: Objectives and Design. *American Journal of Epidemiology*, *156*(9), 871–881. https://doi.org/10.1093/aje/kwf113

Boll, J. M., & Valentine, R. J. (2019). 122: Thoracic outlet syndrome: Arterial. In *Rutherford's Vascular Surgery and Endovascular Therapy* (Ninth, Vol. 2). Elsevier Health Sciences.

Boulton, A. J., Vileikyte, L., Ragnarson-Tennvall, G., & Apelqvist, J. (2005). The global burden of diabetic foot disease. *The Lancet*, *366*(9498), 1719–1724. https://doi.org/10.1016/S0140-6736(05)67698-2

Brewster, D. C., Franklin, D. P., Cambria, R. P., Darling, R. C., Moncure, A. C., Lamuraglia, G. M., Stone, W. M., & Abbott, W. M. (1991). Intestinal ischemia complicating abdominal aortic surgery. *Surgery*, *109*(4), 447–454.

Brott, T. G., Howard, G., Roubin, G. S., Meschia, J. F., Mackey, A., Brooks, W., Moore, W. S., Hill, M. D., Mantese, V. A., Clark, W. M., Timaran, C. H., Heck, D., Leimgruber, P. P., Sheffet, A. J., Howard, V. J., Chaturvedi, S., Lal, B. K., Voeks, J. H., & Hobson, R. W. (2016). Long-Term Results of Stenting versus Endarterectomy for Carotid-Artery Stenosis. *The New England Journal of Medicine*, *374*(11), 1021–1031. https://doi.org/10.1056/NEJMoa1505215

Bulbulia, R., & Halliday, A. (2017). The Asymptomatic Carotid Surgery Trial-2 (ACST-2): An ongoing randomised controlled trial comparing carotid endarterectomy with carotid artery stenting to prevent stroke. *Health Technology Assessment*, *21*(57), 1–40. https://doi.org/10.3310/hta21570

Chaikof, E. L., Dalman, R. L., Eskandari, M. K., Jackson, B. M., Lee, W. A., Mansour, M. A., Mastracci, T. M., Mell, M., Murad, M. H., Nguyen, L. L., Oderich, G. S., Patel, M. S., Schermerhorn, M. L., & Starnes, B. W. (2018). The Society for Vascular Surgery practice guidelines on the care of patients with an abdominal aortic aneurysm. *Journal of Vascular Surgery*, *67*(1), 2–77.e2. https://doi.org/10.1016/j.jvs.2017.10.044

Chang, K. Z., Likes, K., Demos, J., Black, J. H., & Freischlag, J. A. (2012). Routine Venography Following Transaxillary First Rib Resection and Scalenectomy (FRRS) for Chronic Subclavian Vein Thrombosis Ensures Excellent Outcomes and Vein Patency. *Vascular and Endovascular Surgery*, *46*(1), 15–20. https://doi.org/10.1177/1538574411423982

Chung, J., Valentine, W., Sharath, S. E., Pathak, A., Barshes, N. R., Pisimisis, G., Kougias, P., & Mills, J. L. (2016). Percutaneous intervention for carotid in-stent restenosis does not improve outcomes compared with nonoperative management. *Journal of Vascular Surgery*, *64*(5), 1286–1294.e1. https://doi.org/10.1016/j.jvs.2016.05.086

Cohen, J. E., Gomori, J. M., Itshayek, E., Spektor, S., Shoshan, Y., Rosenthal, G., & Moscovici, S. (2012). Single-center experience on endovascular reconstruction of traumatic internal carotid artery dissections: *The Journal of Trauma and Acute Care Surgery*, *72*(1), 216–221. https://doi.org/10.1097/TA.0b013e31823f630a

Collaboration, A. T. (2002). Collaborative meta-analysis of randomised trials of antiplatelet therapy for prevention of death, myocardial infarction, and stroke in high risk patients. *BMJ : British Medical Journal*, *324*(7329), 71–86.

Committee, C. S. (1996). A randomised, blinded, trial of clopidogrel versus aspirin in patients at risk of ischaemic events (CAPRIE). *The Lancet*, *348*(9038), 1329–1339. https://doi.org/10.1016/S0140-6736(96)09457-3

Conte, M. S., Bradbury, A. W., Kolh, P., White, J. V., Dick, F., Fitridge, R., Mills, J. L., Ricco, J.-B., Suresh, K. R., & Murad, M. H. (2019). Global vascular guidelines on the management of chronic limb-threatening ischemia. *European Journal of Vascular and Endovascular Surgery : The Official Journal of the European Society for Vascular Surgery*, *58*(1 Suppl), S1–S109.e33. https://doi.org/10.1016/j.ejvs.2019.05.006

Conte, M. S., Pomposelli, F. B., Clair, D. G., Geraghty, P. J., McKinsey, J. F., Mills, J. L., Moneta, G. L., Murad, M. H., Powell, R. J., Reed, A. B., Schanzer, A., & Sidawy, A. N. (2015). Society for Vascular Surgery practice guidelines for atherosclerotic occlusive disease of the lower extremities: Management of asymptomatic disease and claudication. *Journal of Vascular Surgery*, *61*(3), 2S–41S.e1. https://doi.org/10.1016/j.jvs.2014.12.009

Criado, E., Berguer, R., & Greenfield, L. (2010). The spectrum of arterial compression at the thoracic outlet. *Journal of Vascular Surgery*, *52*(2), 406–411. https://doi.org/10.1016/j.jvs.2010.03.009

Cua, B., Mamdani, N., Halpin, D., Jhamnani, S., Jayasuriya, S., & Mena-Hurtado, C. (2017). Review of coronary subclavian steal syndrome. *Journal of Cardiology*, *70*(5), 432–437. https://doi.org/10.1016/j.jjcc.2017.02.012

Diwan, A., Sarkar, R., Stanley, J. C., Zelenock, G. B., & Wakefield, T. W. (2000). Incidence of femoral and popliteal artery aneurysms in patients with abdominal aortic aneurysms. *Journal of Vascular Surgery*, *31*(5), 863–869. https://doi.org/10.1067/mva.2000.105955

Duffy, S. T., Colgan, M. P., Sultan, S., Moore, D. J., & Shanik, G. D. (1998). Popliteal Aneurysms: A 10-year Experience. *European Journal of Endovascular Surgery*, *16*, 218–222. https://doi.org/10.1016/s1078-5884(98)80223-2

Elkouri, S., Gloviczki, P., McKusick, M. A., Panneton, J. M., Andrews, J., Bower, T. C., Noel, A. A., Harmsen, W. S., Hoskin, T. L., & Cherry, K. (2004). Perioperative complications and early outcome after endovascular and open surgical repair of abdominal aortic aneurysms. *Journal of Vascular Surgery*, *39*(3), 497–505. https://doi.org/10.1016/j.jvs.2003.10.018

Eskandari, M. K., & Morgan, C. E. (2020). 185: Conditions Arising From Repetitive Trauma and Occupational Vascular Problems. In *Rutherford's Vascular Surgery and Endovascular Therapy, 2-Volume Set*. Elsevier Inc. https://doi.org/10.1016/B978-0-323-42791-3.00185-7

Fairman, R. M. (2019). 72. Aortoiliac Aneurysms: Endovascular Treatment. In *Rutherford's Vascular Surgery and Endovascular Therapy*. Elsevier Inc.

Fontaine, R., Kim, M., & Kieny, R. (1954). [Surgical treatment of peripheral circulation disorders]. *Helvetica Chirurgica Acta*, *21*(5-6), 499–533.

Fowkes, F. G. R. (2010). Aspirin for Prevention of Cardiovascular Events in a General Population Screened for a Low Ankle Brachial IndexA Randomized Controlled Trial. *JAMA*, *303*(9), 841. https://doi.org/10.1001/jama.2010.221

Fowkes, F. G. R., Housley, E., Cawood, E. H. H., Macintyre, C. C. A., Ruckley, C. V., & Prescott, R. J. (1991). Edinburgh Artery Study: Prevalence of Asymptomatic and Symptomatic Peripheral Arterial Disease in the General Population. *International Journal of Epidemiology*, *20*(2), 384–392. https://doi.org/10.1093/ije/20.2.384

Gary G Wind, & R. James Valentine. (2013). *Anatomic Exposures in Vascular Surgery* (Second). Lippincott Williams & Wilkins.

Giannopoulos, S., Texakalidis, P., Jonnalagadda, A. K., Karasavvidis, T., Giannopoulos, S., & Kokkinidis, D. G. (2018). Revascularization of radiation-induced carotid artery stenosis with carotid endarterectomy vs. Carotid artery stenting: A systematic review and meta-analysis. *Cardiovascular Revascularization Medicine*, *19*(5), 638–644. https://doi.org/10.1016/j.carrev.2018.01.014

Gordon, T., & Kannel, W. B. (1972). Predisposition to atherosclerosis in the head, heart, and legs. The Framingham study. *JAMA*, *221*(7), 661–666.

Greenhalgh, R. (2004). Comparison of endovascular aneurysm repair with open repair in patients with abdominal aortic aneurysm (EVAR trial 1), 30-day operative mortality results: Randomised controlled trial. *The Lancet*, *364*(9437), 843–848. https://doi.org/10.1016/S0140-6736(04)16979-1

Group, H. P. S. C. (2007). Randomized trial of the effects of cholesterol-lowering with simvastatin on peripheral vascular and other major vascular outcomes in 20,536 people with peripheral arterial disease and other high-risk conditions. *Journal of Vascular Surgery*, *45*(4), 645–654.e1. https://doi.org/10.1016/j.jvs.2006.12.054

Grunwald, M. R., & Hofmann, L. V. (2004). Catheter-Directed Thrombolysis for the Treatment of Symptomatic Deep Vein Thrombosis. *Circulation*, *109*(2). https://doi.org/10.1161/01.CIR.0000111132.23469.70

Hageman, D., Fokkenrood, H. J., Gommans, L. N., van den Houten, M. M., & Teijink, J. A. (2018). Supervised exercise therapy versus home-based exercise therapy versus walking advice for intermittent claudication. *The Cochrane Database of Systematic Reviews*, *2018*(4), CD005263. https://doi.org/10.1002/14651858.CD005263.pub4

Halliday, A., Harrison, M., Hayter, E., Kong, X., Mansfield, A., Marro, J., Pan, H., Peto, R., Potter, J., Rahimi, K., Rau, A., Robertson, S., Streifler, J.,

& Thomas, D. (2010). 10-year stroke prevention after successful carotid endarterectomy for asymptomatic stenosis (ACST-1): A multicentre randomised trial. *Lancet*, *376*(9746), 1074–1084. https://doi.org/10.1016/S0140-6736(10)61197-X

Hoballah, J. J. (2021). Inferior Mesenteric Artery Reimplantation. In J. J. Hoballah & C. F. Bechara (Eds.), *Vascular Reconstructions* (pp. 373–379). Springer New York. https://doi.org/10.1007/978-1-0716-1089-3_17

Humphries, M. D., & Freischlag, J. A. (2019a). 123: Thoracic outlet syndrome: Venous. In *Rutherford's Vascular Surgery and Endovascular Therapy* (Vol. 2, pp. 1648–1655.e2). Elsevier Inc. https://doi.org/10.1016/j.mpsur.2018.12.010

Humphries, M. D., & Freischlag, J. A. (2019b). 124: Thoracic Outlet Syndrome : Surgical Decompression of the Thoracic Outlet. In *Rutherford's Vascular Surgery and Endovascular Therapy* (Ninth, Vol. 2). Elsevier Inc. https://doi.org/10.1016/B978-0-323-42791-3.00124-9

Illig, K. A. (2019). 57. Upper Extremity Vascular Exposure. In *Rutherford's Vascular Surgery and Endovascular Therapy* (p. 12). Elsevier Inc.

Illig, K. A., & Doyle, A. J. (2010). A comprehensive review of Paget-Schroetter syndrome. *Journal of Vascular Surgery*, *51*(6), 1538–1547. https://doi.org/10.1016/j.jvs.2009.12.022

Jack L Cronenwett, Alik Farber, & Erica L. Mitchell. (2020). *Vascular Decision Making: Medical, Endovascular, Surgical*. Wolters Kluwer.

Kannel, W. B., & McGee, D. L. (1985). Update on some epidemiologic features of intermittent claudication: The Framingham Study. *Journal of the American Geriatrics Society*, *33*(1), 13–18. https://doi.org/10.1111/j.1532-5415.1985.tb02853.x

Karam, J. G., Loney-Hutchinson, L., & McFarlane, S. I. (2008). High-Dose Atorvastatin After Stroke or Transient Ischemic Attack: The Stroke Prevention by Aggressive Reduction in Cholesterol Levels (SPARCL) Investigators. *Journal of the CardioMetabolic Syndrome*, *3*(1), 68–69. https://doi.org/10.1111/j.1559-4572.2008.07967.x

Kauvar, D. S., & Kraiss, L. W. (2020). 184: Vascular Trauma: Extremity. In *Rutherford's Vascular Surgery and Endovascular Therapy, 2-Volume Set*. Elsevier Inc. https://doi.org/10.1016/B978-0-323-42791-3.00184-5

Kearon, C., Akl, E. A., Ornelas, J., Blaivas, A., Jimenez, D., Bounameaux, H., Huisman, M., King, C. S., Morris, T. A., Sood, N., Stevens, S. M., Vintch, J. R. E., Wells, P., Woller, S. C., & Moores, L. (2016). Antithrombotic Therapy for VTE Disease. *Chest*, *149*(2), 315–352. https://doi.org/10.1016/j.chest.2015.11.026

Klarin, D., Lancaster, R. T., Ergul, E., Bertges, D., Goodney, P., Schermerhorn, M. L., Cambria, R. P., & Patel, V. I. (2016). Perioperative and long-term impact of chronic kidney disease on carotid artery interventions. *Journal of Vascular Surgery*, *64*(5), 1295–1302. https://doi.org/10.1016/j.jvs.2016.04.038

Kristmundsson, T., Dias, N., Resch, T., & Sonesson, B. (2016). Morphology of Small Abdominal Aortic Aneurysms Should be Considered before Continued

Ultrasound Surveillance. *Annals of Vascular Surgery*, *31*, 18–22. https://doi.org/10.1016/j.avsg.2015.09.016

Laine, M. T., Laukontaus, S. J., Kantonen, I., & Venermo, M. (2016). Population-based study of ruptured abdominal aortic aneurysm. *British Journal of Surgery*, *103*(12), 1634–1639. https://doi.org/10.1002/bjs.10200

Landry, G. J. (2019). 141. Raynaud Phenomenon. In *Rutherford Vascular Surgery and Endovascular Therapy*. Elsevier Inc.

Lane, R., Harwood, A., Watson, L., & Leng, G. C. (2017). Exercise for intermittent claudication. *The Cochrane Database of Systematic Reviews*, *2017*(12), CD000990. https://doi.org/10.1002/14651858.CD000990.pub4

Lederle, F. A., Kyriakides, T. C., Stroupe, K. T., Freischlag, J. A., Padberg, F. T., Matsumura, J. S., Huo, Z., & Johnson, G. R. (2019). Open versus Endovascular Repair of Abdominal Aortic Aneurysm. *New England Journal of Medicine*, *380*(22), 2126–2135. https://doi.org/10.1056/NEJMoa1715955

Lee, K. B., Lu, J., Macsata, R. A., Patel, D., Yang, A., Ricotta, J. J., Amdur, R. L., Sidawy, A. N., & Nguyen, B.-N. (2019). Inferior mesenteric artery replantation does not decrease the risk of ischemic colitis after open infrarenal abdominal aortic aneurysm repair. *Journal of Vascular Surgery*, *69*(6), 1825–1830. https://doi.org/10.1016/j.jvs.2018.09.046

Lend, G. C., & Fowkes, F. G. R. (1992). The Edinburgh Claudication Questionnaire: An improved version of the WHO/Rose questionnaire for use in epidemiological surveys. *Journal of Clinical Epidemiology*, *45*(10), 1101–1109. https://doi.org/10.1016/0895-4356(92)90150-L

Lima Neto, A. C., Bittar, R., Gattas, G. S., Bor-Seng-Shu, E., Oliveira, M. de L., Monsanto, R. da C., & Bittar, L. F. (2017). Pathophysiology and Diagnosis of Vertebrobasilar Insufficiency: A Review of the Literature. *International Archives of Otorhinolaryngology*, *21*(3), 302–307. https://doi.org/10.1055/s-0036-1593448

Lindsay, T. F. (2019). Chapter 74 - Ruptured Aortoiliac Aneurysms and Their Management. In *Rutherford's Vascular Surgery and Endovascular Therapy* (Ninth, p. 21). Elsevier Inc.

Lugo, J., Tanious, A., Armstrong, P., Back, M., Johnson, B., Shames, M., Moudgill, N., Nelson, P., & Illig, K. A. (2015). Acute Paget–Schroetter Syndrome: Does the First Rib Routinely Need to Be Removed after Thrombolysis? *Annals of Vascular Surgery*, *29*(6), 1073–1077. https://doi.org/10.1016/j.avsg.2015.02.006

Lum, Y. W., Brooke, B. S., Likes, K., Modi, M., Grunebach, H., Christo, P. J., & Freischlag, J. A. (2012). Impact of anterior scalene lidocaine blocks on predicting surgical success in older patients with neurogenic thoracic outlet syndrome. *Journal of Vascular Surgery*, *55*(5), 1370–1375. https://doi.org/10.1016/j.jvs.2011.11.132

Mahmood, S. S., Levy, D., Vasan, R. S., & Wang, T. J. (2014). The Framingham Heart Study and the Epidemiology of Cardiovascular Diseases: A Historical Perspective. *Lancet*, *383*(9921), 999–1008. https://doi.org/10.1016/S0140-6736(13)61752-3

Markus, H. S., Levi, C., King, A., Madigan, J., & Norris, J. (2019). Antiplatelet

Therapy vs Anticoagulation Therapy in Cervical Artery Dissection. *JAMA Neurology*, *76*(6), 657–664. https://doi.org/10.1001/jamaneurol.2019.0072

McMinn, R. H. (2019). *Last's Anatomy: Regional and applied*. Elsevier Inc.

Meershoek, A. J. A., & de Borst, G. J. (2018). Timing of carotid intervention. *The British Journal of Surgery*, *105*(10), 1231–1233. https://doi.org/10.1002/bjs.10950

Mills, J. L., Conte, M. S., Armstrong, D. G., Pomposelli, F. B., Schanzer, A., Sidawy, A. N., & Andros, G. (2014). The Society for Vascular Surgery Lower Extremity Threatened Limb Classification System: Risk stratification based on Wound, Ischemia, and foot Infection (WIfI). *Journal of Vascular Surgery*, *59*(1), 220–234.e2. https://doi.org/10.1016/j.jvs.2013.08.003

Molina, J. E., Hunter, D. W., & Dietz, C. A. (2007). Paget-Schroetter syndrome treated with thrombolytics and immediate surgery. *Journal of Vascular Surgery*, *45*(2), 328–334. https://doi.org/10.1016/j.jvs.2006.09.052

Moore, W. S., Lawrence, P. F., & Oderich, G. S. (Eds.). (2019). *Vascular and endovascular surgery: A comprehensive review* (Ninth edition). Elsevier/Saunders.

Morasch, M. D. (2009). Technique for subclavian to carotid transposition, tips, and tricks. *Journal of Vascular Surgery*, *49*(1), 251–254. https://doi.org/10.1016/j.jvs.2008.10.035

Naylor, A. R. (2015). Why is the management of asymptomatic carotid disease so controversial? *The Surgeon*, *13*(1), 34–43. https://doi.org/10.1016/j.surge.2014.08.004

Naylor, A. R., Cuffe, R. L., Rothwell, P. M., & Bell, P. R. F. (2003). A systematic review of outcomes following staged and synchronous carotid endarterectomy and coronary artery bypass. *European Journal of Vascular and Endovascular Surgery*, *25*(5), 380–389. https://doi.org/10.1053/ejvs.2002.1895

Newton, E. R., Akerman, A. W., Strassle, P. D., & Kibbe, M. R. (2021). Association of Fluoroquinolone Use With Short-term Risk of Development of Aortic Aneurysm. *JAMA Surgery*, *156*(3), 264. https://doi.org/10.1001/jamasurg.2020.6165

North American Symptomatic Carotid Endarterectomy Trial Collaborators. (1991). Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. *New England Journal of Medicine*, *325*(7), 445–453. https://doi.org/10.1056/NEJM199108153250701

Ouriel, K., McDonnell, A. E., Metz, C. E., & Zarins, C. K. (1982). Critical evaluation of stress testing in the diagnosis of peripheral vascular disease. *Surgery*, *91*(6), 686–693.

Parkinson, F., Ferguson, S., Lewis, P., Williams, I. M., & Twine, C. P. (2015). Rupture rates of untreated large abdominal aortic aneurysms in patients unfit for elective repair. *Journal of Vascular Surgery*, *61*(6), 1606–1612. https://doi.org/10.1016/j.jvs.2014.10.023

Pham, M. H., Rahme, R. J., Arnaout, O., Hurley, M. C., Bernstein, R. A., Batjer, H. H., & Bendok, B. R. (2011). Endovascular Stenting of Extracranial Carotid and Vertebral Artery Dissections: A Systematic Review of the Literature. *Neurosurgery*, *68*(4), 856–866. https://doi.org/10.1227/NEU.0b013e318209

ce03

Powers, W. J., Rabinstein, A. A., Ackerson, T., Adeoye, O. M., Bambakidis, N. C., Becker, K., Biller, J., Brown, M., Demaerschalk, B. M., Hoh, B., Jauch, E. C., Kidwell, C. S., Leslie-Mazwi, T. M., Ovbiagele, B., Scott, P. A., Sheth, K. N., Southerland, A. M., Summers, D. V., & Tirschwell, D. L. (2018). 2018 Guidelines for the Early Management of Patients With Acute Ischemic Stroke: A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association. *Stroke*, *49*(3). https://doi.org/10.1161/STR.0000000000000158

Prinssen, M., Cuypers, P. W. M., & Buskens, E. (2004). A Randomized Trial Comparing Conventional and Endovascular Repair of Abdominal Aortic Aneurysms. *The New England Journal of Medicine*, 13.

Rajendran, S., & May, J. (2017). Late rupture of abdominal aortic aneurysm after endovascular repair. *Journal of Vascular Surgery*, *65*(1), 52–57. https://doi.org/10.1016/j.jvs.2016.05.090

Rantner, B., Kollerits, B., Roubin, G. S., Ringleb, P. A., Jansen, O., Howard, G., Hendrikse, J., Halliday, A., Gregson, J., Eckstein, H.-H., Calvet, D., Bulbulia, R., Bonati, L. H., Becquemin, J.-P., Algra, A., Brown, M. M., Mas, J.-L., Brott, T. G., & Fraedrich, G. (2017). Early Endarterectomy Carries a Lower Procedural Risk Than Early Stenting in Patients With Symptomatic Stenosis of the Internal Carotid Artery. *Stroke*, *48*, 1580–1587. https://doi.org/10.1161/STROKEAHA.116.016233

Rinehardt, E. K., Scarborough, J. E., & Bennett, K. M. (2017). Current practice of thoracic outlet decompression surgery in the United States. *Journal of Vascular Surgery*, *66*(3), 858–865. https://doi.org/10.1016/j.jvs.2017.03.436

Rose, G. A. (1962). The diagnosis of ischaemic heart pain and intermittent claudication in field surveys. *Bulletin of the World Health Organization*, *27*(6), 645–658.

Rosenfield, K., Matsumura, J. S., Chaturvedi, S., Riles, T., Ansel, G. M., Metzger, D. C., Wechsler, L., Jaff, M. R., & Gray, W. (2016). Randomized Trial of Stent versus Surgery for Asymptomatic Carotid Stenosis. *New England Journal of Medicine*, *374*(11), 1011–1020. https://doi.org/10.1056/NEJMoa1515706

Rothwell, P., Eliasziw, M., Gutnikov, S., Warlow, C., & Barnett, H. (2004). Endarterectomy for symptomatic carotid stenosis in relation to clinical subgroups and timing of surgery. *The Lancet*, *363*(9413), 915–924. https://doi.org/10.1016/S0140-6736(04)15785-1

Rutherford, R. B., Baker, J. D., Ernst, C., Johnston, K. W., Porter, J. M., Ahn, S., & Jones, D. N. (1997). Recommended standards for reports dealing with lower extremity ischemia: Revised version. *Journal of Vascular Surgery*, *26*(3), 517–538. https://doi.org/10.1016/S0741-5214(97)70045-4

Sadeghi-Azandaryani, M., Bürklein, D., Ozimek, A., Geiger, C., Mendl, N., Steckmeier, B., & Heyn, J. (2009). Thoracic outlet syndrome: Do we have clinical tests as predictors for the outcome after surgery? *European Journal of Medical Research*, *14*(10), 443–446. https://doi.org/10.1186/2047-783X-14-10-443

Sajid, M. S., Ahmed, N., Desai, M., Baker, D., & Hamilton, G. (2007). Upper

Limb Deep Vein Thrombosis: A Literature Review to Streamline the Protocol for Management. *Acta Haematologica*, *118*(1), 10–18. https://doi.org/10.1159/000101700

Salhan, N., Humphries, M., Sheth, S., DeMesa, C., & Freischlag, J. (2016). PC214. Ultrasound-Guided Anesthetic or Botulinum Toxin Injection of the Anterior and Middle Scalene Muscles to Assist With Diagnosis and Treatment of Neurogenic Thoracic Outlet Syndrome. *Journal of Vascular Surgery*, *63*(6), 218S. https://doi.org/10.1016/j.jvs.2016.03.365

Sanders, R. J., Hammond, S. L., & Rao, N. M. (2007). Diagnosis of thoracic outlet syndrome. *Journal of Vascular Surgery*, *46*(3), 601–604. https://doi.org/10.1016/j.jvs.2007.04.050

Schanzer, A., Messina, L. M., Ghosh, K., Simons, J. P., Robinson, W. P., Aiello, F. A., Goldberg, R. J., & Rosen, A. B. (2015). Follow-Up Compliance After Endovascular Abdominal Aortic Aneurysm Repair in Medicare Beneficiaries. *Journal of Vascular Surgery*, *61*(1), 16–22.e1. https://doi.org/10.1016/j.jvs.2014.06.006

Sekhar, R. (2018). *Yearbook of vascular and endovascular surgery-2, 2018*. JAYPEE Brothers MEDICAL P.

Shanmugam, V. K. (2019). 137. Vasculitis and Other Uncommon Arteriopathies. In *Rutherford's Vascular Surgery and Endovascular Therapy* (p. 16).

Shuja, F. (n.d.). *117. Upper Extremity Arterial Disease: Epidemiology, Etiology, and Diagnostic Evaluation* (p. 11).

Stanley, J. C., Veith, F., & Wakefield, T. W. (2014). *Current Therapy in Vascular and Endovascular Surgery*. Elsevier Health Sciences.

Stevens, J. W., Simpson, E., Harnan, S., Squires, H., Meng, Y., Thomas, S., Michaels, J., & Stansby, G. (2012). Systematic review of the efficacy of cilostazol, naftidrofuryl oxalate and pentoxifylline for the treatment of intermittent claudication. *British Journal of Surgery*, *99*(12), 1630–1638. https://doi.org/10.1002/bjs.8895

Stone, P. A., & Hass, S. M. (2019). 21. Vascular Laboratory: Arterial Duplex Scanning. In *Rutherford's Vascular Surgery and Endovascular Therapy* (Ninth). Elsevier Inc. https://doi.org/10.1016/B978-0-323-42791-3.00021-9

Strandness, D. E., & Sumner, D. S. (1975). *Hemodynamics for surgeons*. Grune & Stratton.

Tuveson, V., Löfdahl, H. E., & Hultgren, R. (2016). Patients with abdominal aortic aneurysm have a high prevalence of popliteal artery aneurysms. *Vascular Medicine*, *21*(4), 369–375. https://doi.org/10.1177/1358863X16648404

Ultee, K. H. J., Soden, P. A., Zettervall, S. L., Darling, J., Verhagen, H. J. M., & Schermerhorn, M. L. (2016). Conversion from EVAR to Open Abdominal Aortic Aneurysm Repair. *Journal of Vascular Surgery*, *64*(1), 76–82. https://doi.org/10.1016/j.jvs.2015.12.055

United States Surgeon General. (2014). *The Health Consequences of Smoking – 50 Years of progress: A Report of the Surgeon General: (510072014-001)*. American Psychological Association. https://doi.org/10.1037/e510072014-001

Urschel, H. C., & Patel, A. N. (2008). Surgery Remains the Most Effective

Treatment for Paget-Schroetter Syndrome: 50 Years' Experience. *The Annals of Thoracic Surgery*, *86*(1), 254–260. https://doi.org/10.1016/j.athoracsur.2008.03.021

Vemuri, C., McLaughlin, L. N., Abuirqeba, A. A., & Thompson, R. W. (2017). Clinical presentation and management of arterial thoracic outlet syndrome. *Journal of Vascular Surgery*, *65*(5), 1429–1439. https://doi.org/10.1016/j.jvs.2016.11.039

Vemuri, C., Salehi, P., Benarroch-Gampel, J., McLaughlin, L. N., & Thompson, R. W. (2016). Diagnosis and treatment of effort-induced thrombosis of the axillary subclavian vein due to venous thoracic outlet syndrome. *Journal of Vascular Surgery: Venous and Lymphatic Disorders*, *4*(4), 485–500. https://doi.org/10.1016/j.jvsv.2016.01.004

Voigt, S. L., Bishawi, M., Ranney, D., Yerokun, B., McCann, R. L., & Hughes, G. C. (2019). Outcomes of carotid-subclavian bypass performed in the setting of thoracic endovascular aortic repair. *Journal of Vascular Surgery*, *69*(3), 701–709. https://doi.org/10.1016/j.jvs.2018.07.022

Wang, L. J., Locham, S., Al-Nouri, O., Eagleton, M. J., Clouse, W. D., & Malas, M. B. (2020). Endovascular repair of ruptured abdominal aortic aneurysm is superior to open repair: Propensity-matched analysis in the Vascular Quality Initiative. *Journal of Vascular Surgery*, *72*(2), 498–507. https://doi.org/10.1016/j.jvs.2019.11.063

Wilson, J. J., Zahn, C. A., & Newman, H. (1990). Fibrinolytic therapy for idiopathic subclavian-axillary vein thrombosis. *The American Journal of Surgery*, *159*(2), 208–210. https://doi.org/10.1016/S0002-9610(05)80262-4

Yao, S. T., Hobbs, J. T., & Irivne, W. T. (2005). Ankle systolic pressure measurements in arterial disease affecting the lower extremities. *British Journal of Surgery*, *56*(9), 676–679. https://doi.org/10.1002/bjs.1800560910