Vascular Surgery Board Review

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Chapter 1

About

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

1.1 Usage

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

1.2 Comments, Questions or Contributions

Please visit our github page or send us an email.

Chapter 2

Cerebrovascular

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

2.1 Available Guidelines

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

2.2 Presentation and Diagnosis

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

   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 and 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

2.3 Management

2.3.1 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

2.3.2 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. [Rothwell et al., 2004, Meershoek and de Borst, 2018]
  - Consider urgent intervention in a medically stable patient with mild-moderate neurologic deficit, if there is a significant area of ischemic penumbra at risk for progression
  - 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.

2.3.3 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]

2.3.4 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]

2.4 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

Chapter 3

Upper Extremity and Thoracic Outlet

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

3.1 Anatomy/ Exposure of Vessels

What are the zones of the upper extremity? [Illig, 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 and 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

3.2 Epidemiology, etiology, and diagnostic evaluation

How does evaluation of upper extremity ischemia differentiate from lower extremity ischemia? [Shuja]

- 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

3.3 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]

3.4 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

3.5 Vasospastic Disorders

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

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

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

- 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

3.5.1 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

3.5.2 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

3.5.3 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

3.6 Aneurysmal Disease

How are subclavian aneurysms caused and how can they present? [shadman Baig and 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.

3.7 Occupational Vascular Disease

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

3.7.1 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

3.7.2 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

3.7.3 Environmental Exposures

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

Acrosteolysis

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

Electrical burns

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

Extreme thermal injuries

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

3.7.4 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

3.8 Vascular Trauma-Upper Extremity

This is discussed in detail here: 10, **so we will go over some important specifics for upper extremity vascular injury.** [Kauvar and 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

3.9 Compression Syndromes

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

3.9.1 Quadrilateral space syndrome

Anatomy:

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

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

3.9.2 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

3.10 Thoracic Outlet Syndrome

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

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

Types: Neurogenic, Venous and Arterial

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

3.10.1 Anatomy

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

Anatomy from anterior to posterior

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

- The middle scalene muscle.

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

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

Interscalene Triangle:

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

Anterior Scalene:

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

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

Middle Scalene:

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

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

The First Rib:

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

Subclavius Muscle:

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

The Coracoid Process:

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

Pectoralis Minor Muscle:

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

Clavipectoral Fascia:

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

Phrenic Nerve Anomaly:

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

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

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

nTOS

- Scalene Triangle compression – most common cause of brachial plexus and neurogenic TOS

- Cervical Rib and Anomalous First Rib

aTOS

- Cervical Rib and Anomalous First Rib
- Scalene Triangle compression

vTOS

- Costoclavicular Passage
- Subcoracoid Space

3.10.2 Diagnosis and Evaluation

3.10.2.1 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.

3.10.2.2 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 and Freischlag, 2019b]

3.10.2.3 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 and Freischlag, 2019a]
- Now vTOS and Paget Schroetter Syndrome are used synonymously.
- Paget Schroetter Syndrome accounts for 10-20% of all upper extremity deep vein thrombosis. [Sekhar, 2018]

3.10.3 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 • Risk of injury to T1 nerve root, phrenic nerve, long thoracic, brachial plexus , subclavian vein and arterial with limited exposure to repair • Not able to approach cervical ribs, scalene triangle or patch vein.
Suprac lavicular	• Good for scalene triangle access and debulking and cervical rib resection • Required for aTOS if arterial reconstruction necessary	• Unable to decompress venous compression or visualize vein adequately • Cosmetically less appealing
Infrac lavicular [@ siracuseI nfraclavi cularFirs tRib2015]	• Good access for venous decompression • Allows for excision of subclavius muscle and costoclavicular ligament	• Unable to expose subclavian artery or decompress brachial plexus. • Difficult to access most posterior aspect of rib • 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

3.10.3.1 Post operative complications

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

3.10.4 vTOS

3.10.4.1 Demographics

- Incidence: 2/100,000 persons
- Age: 18 years to 30 years [Illig and Doyle, 2010]
- M>F

3.10.4.2 Presentation

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

3.10.4.3 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

3.10.4.4 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

3.10.4.5 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 and Patel, 2008]
  - Systemic Lysis – non favored due to risk of intracranial hemorrhage. [Grunwald and Hofmann, 2004]
  - Catheter Directed Lysis (CDT) – carries a lower risk of intracranial hemorrhage.
  - Patient should be maintained in a compression sleeve until definitive decompression can be performed.
- Optimal timing of CDT
  - Within 14 days of onset of thrombosis. Excellent results have been reported following CDT if initiated before 14 days. [Wilson et al., 1990]
- 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.

    @*melbyComprehensiveSurgicalManagement*2008

  - Venous occlusion in vTOS may be treated with jugular turn down or venous bypass to IJ of SVC if patients remain symptomatic. [Vemuri et al., 2016]

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

3.10.4.7 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.

3.10.5 aTOS

3.10.5.1 Presentation

- Most common: Hand ischemia due to arterial compression or microembolization with subclavian artery aneurysm and pulsatile supraclavicular mass [Boll and Valentine, 2019]
- Less common: Exertional pain, unilateral Raynaud's Phenomena, retrograde embolisation and neurological symptoms
- 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 and Valentine, 2019]
    - Cervical Rib (60%)
    - Anomalous First Rib (18%)
    - Fibrocartilaginous band (15%)

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

3.10.5.2 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

3.10.5.3 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. [Vemuri et al., 2017, Criado et al., 2010]

3.10.5.4 Management considerations with aTOS

- Symptomatic patients are generally indicated for treatment. Unlike asymptomatic patients in whom it may be appropriate to manage conservatively. [Boll and 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.

3.10.6 nTOS

3.10.6.1 Demographics

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

- F>M – 70% Female
- Ages 20-40
- Occupational Exposure
- Trauma history

3.10.6.2 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

3.10.6.3 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. [Salhan et al., 2016, Lum et al., 2012]
- Botulinum injection may give an average of 6 weeks of relief. [Salhan et al., 2016]

- Be selective in patients who may require surgery

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

Chapter 4

Abdominal/Iliac/Peripheral Aneurysms

4.1

Chapter 5

Lower Extremity Occlusive Disease

5.1

Chapter 6

Mesenteric Disease

6.1

Chapter 7

Renal

7.1

Chapter 8

Thoracic Aorta

8.1

Chapter 9

Venous Disease

9.1

Chapter 10

Vascular Trauma

10.1

Chapter 11

Angioaccess

11.1

Chapter 12

Complications

12.1

Chapter 13

Amputations

13.1

Chapter 14

Vascular Lab

14.1

Chapter 15

Vascular Medicine

15.1

Chapter 16

Endovascular

16.1

Chapter 17

Applied Science

17.1

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