Vascular Surgery Exam Prep

Audible Bleeding

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Preface

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

Usage

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

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

We are excited to provide our readers and listeners a free and open opportunity to contribute to this work. However, we do expect that you follow a Contributor Code of Conduct. Public annotations and feedback are considered contributions and by contributing to this ebook, you agree to abide by its terms.

Additional Resources

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

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

- Rutherford's Vascular Surgery and Endovascular Therapy
  - No vascular surgeon bookshelf is complete without a copy of this book sitting on it!
  - Of note, our review book references the 9th edition of Rutherford's, however there is a new edition (10th) has just been released in June 2022.
  - Many institutional libraries carry this book and can be accessed for free. We would recommend checking with that resource prior to purchasing yourself.
  - Some industry representatives offer text book purchasing programs, so check with them prior to purchasing yourself.
- VESAP5
  - This is the premier question bank for preparing for the vascular surgery boards.
  - Some industry representatives offer text book purchasing programs and include this as an option, so check with them prior to purchasing yourself.
  - A new version of VESAP is released every 2-3 years, and once a new one is release, the older versions are no longer accessible. Make sure to check the release schedule so that you can time your purchase to align when you will need to do the majority of your exam preparation.
- SVS Education Website
  - Check this site regularly for free content through the SVS. This is available to all candidate members of the SVS, so sign up!
- VSCORE
  - This is a curated curriculum developed in collaboration with Association for Program Directors in Vascular Surgery (APDVS)
  - Check with your program director to confirm whether this is available at your training program.

Comments, Questions or Contributions

Please visit our github page or send us an email.

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

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Part I

Head and Neck

1 Cerebrovascular

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1.1 Available Guidelines

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

1.2 Presentation and Diagnosis

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

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

   A particularly confusing distinction is between a crescendo TIA and a stroke in evolution. A crescendo TIA is frequent repetitive neurological attacks without complete resolution of the deficit between the episodes, usually over a 24 hour period. The result of which is the same neurological deficit. If the patient exhibits progressive deterioration in neurological function between TIA episodes this is classified as a stroke in evolution not a crescendo TIA.(Ricotta 2019) These are both particularly concerning findings and may be an indication for an emergent intervention.

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

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

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

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

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

3. **Ultrasound Imaging**

- US is considered the best, least invasive, and cost effective method for detecting carotid stenosis on screening.(Shaalan et al. 2008) For more see @ref(extracranial).
- 50-69% stenosis of ICA - US has a low sensitivity for 50-69% stenosis. A negative ultrasound in symptomatic patients necessitates additional imaging. Typically on scanning if 50-69% ICA stenosis is present:
  - PSV 125-229 cm/sec
  - EDV 40-100
  - Internal/Common Carotid PSV Ratio 2-4
- 70-99% stenosis of ICA- Typically on scanning:
  - PSV >/= 230 cm/sec
  - EDV >100 (EDV > 140 cm/sec most sensitive for stenosis >80%)
  - Internal/Common Carotid PSV Ratio > 4
- Velocity-based estimation of carotid artery stenosis may need to be adjusted in certain circumstances:
  - Higher velocities in women than in men.
  - Higher velocities in the presence of contralateral carotid artery occlusion.

- High carotid bifurcation, severe arterial tortuosity, extensive vascular calcification, and obesity may also reduce the accuracy of US imaging.

4. **Other Imaging Modalities**

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

1.3 Management

1.3.1 Medical Management - Asymptomatic Patients

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

Hypertension

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

Diabetes

- In diabetic patients glycaemic control to nearly normoglycemic levels (target hemoglobin A1C <7%) is recommended to reduce microvascular complications and, with a smaller evidence base, macrovascular complications other than stroke.(Ricotta 2019)

Hypercholesterolaemia

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

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

Antiplatelet therapy - Single agent antiplatelet therapy, most often aspirin, is often all that is needed, but this should be weighted with other cardiovascular risk factors. A number of trials have showed limited benefit of adding an additional antiplatelet or routinely testing for clopidogrel resistance.(Ricotta 2019)

1.3.2 Medical Management - Symptomatic Patients

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

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

- < 4.5 hours from onset of symptoms - medical thrombolysis with tPA unless contraindicated.
  - Patient age >80 and diabetes are contraindications to tPA administration after 3hrs.

  - Other contraindications include - hypertension, intracranial hemorrhage, recent stroke or cranial trauma, spine/brain surgery within 3 months, GI bleed within 21 days.
  - BP management post stroke **with thrombolysis** includes treatment of BP to <185/110 prior to administration of thrombolysis, then keeo <180/105 for 24hrs post administration.

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

1.3.3 Surgery - Carotid Endarterectomy

Indications for Surgical Intervention

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

CEA Intraoperative Techniques

- General concepts
  - Patch angioplasty or eversion endarterectomy are recommended over primary arterial closure due to decreased ipsilateral stroke, perioperative carotid occlusion, return to the operating room, restenosis and 1-year stroke rates.(Goodney et al. 2010; Rerkasem and Rothwell 2009) Patch repair is considered the standard of care surgical management for most extracranial carotid lesions.(Arnold and Perler 2019)
- Neuromonitoring/Shunting options during a carotid endarterectomy(Chongruksut, Vaniyapong, and Rerkasem 2014; Wiske et al. 2018)
  - Local/regional anesthesia with direct neurological monitoring - the benefit being that the patient is awake and moving to command throughout the case. Indications for shunting include lateralizing deficits, seizure, lack of consciousness and severe anxiety. Improved neuromonitoring, however, has not been shown to reduce myocardial infarction rate with CEA.
  - Stump pressure- Clamp the inflow and place a butterfly attached to a-line tubing into the internal carotid artery. If the stump pressure is > 50 mmHg the surgeon can chose to proceed without shunt placement, if < 50mmHg then a shunt should be placed prior to proceeding.
  - EEG Neuromonitoring - EEG tech places neuromonitoring, monitored by the technician and neurologist remotely intraoperatively. Generally the surgeon should clamp the ICA for 3 minutes before proceeding, if the patient develops neurological deficit/EEG abnormalities with slowing and decreased alpha and beta waves, then unclamp, await normalization of EEG, then proceed.
  - Cerebral Oximetry - 15% decrease in either hemisphere should be shunted.
  - Transcranial Doppler (TCD) - severe slowing in the middle cerebral artery. An RCT demonstrated that TCD and cerebral oximetry changes most accurately predicted cerebral ischemia. Detection of a greater than 50% drop in middle cerebral artery velocity using transcranial Doppler is 100% sensitive for detecting cerebral ischemia. (Moritz et al. 2007b) For more see @ref(intracranial).
  - Non-selective shunting - the surgeon uses a shunt for all CEAs as standard practice, this is often done for all CEAs performed under general anesthesia
- Techniques to reach high internal carotid lesions?(Beretta et al. 2006)
  - A good knowledge of the available imaging while preparing for the procedure is essential when considering the level of the carotid bifurcation.

  - Nasotracheal intubation will allow for jaw closure and neck extension to facilitate exposure of higher lesions.
  - Division of the posterior belly of the digastric muscle, stylopharyngeal muscle, styloglossus muscle, stylomandibular ligament or the styloid process. Care should be taken to identify and preserve the glossopharyngeal nerve.
  - Division of the occipital artery.
  - Dissection along the posterior parotid gland.
  - ENT surgeon assisted mandible subluxation with assistance if previous techniques fail.

- What is the best technique for a patient with an anatomically kinked internal carotid artery?
  - Eversion carotid endarterectomy (only indication for selection for this technique over patch endarterectomy), transection allows for reduction of redundancy at reanastamosis.
  - Otherwise, no advantage has been shown between eversion or patch, both can be shunted.

- Nerve Injuries – where you would encounter these and, if injured, what deficit would be seen?
  - Hypoglossal: Most commonly injured. Usually passes above the bifurcation of the carotid artery. If injured the tongue will deviate toward the side of injury on protrusion.
  - Glossopharyngeal: Commonly injured during high dissections. Found under the posterior digastric muscle. Injury can be devastating for patients resulting in difficulty swallowing and high aspiration risk.
  - Vagus: Usually found adjacent and lateral to the common carotid. Injury can occur with carotid clamping. Patients develop hoarseness due to innervation of the laryngeal muscles from the recurrent laryngeal nerve which is a branch of the vagus.
  - Marginal Mandibular: Branch of the facial nerve. Runs inferiorly to the angle of the mandible. Commonly a retraction injury during high dissections leading to the corner of the ipsilateral lip drooping, can be confused with a significant neurological deficit following the case.

- Completion duplex for CEA
  - Can identify technical issues with carotid endarterectomy such as clamp site injury, thrombosis, intimal flap or dissection, or missed disease.(Lipski et al. 1996)

        * Acute thrombus may be hypoechoic and difficult to see on B-mode, color flow necessary to determine luminal narrowing or occlusion. For more see @ref(extracranial).

    - Revision required if residual plaque identified with severe velocity elevation.(Weinstein et al. 2015)

        * Elevated velocities can be seen for many reasons that do not require revision, including incorrect technique (compression with probe), tortuosity, size discrepancy, ICA spasm.(Ricco et al. 2013; Weinstein et al. 2015)

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

Postoperative Complications

- What to do if the patient develops neuro deficits following carotid endarterectomy: Two main concerns- an intimal flap causing thrombosis or an embolic event resulting in a stroke. Perform a thorough neurological exam to confirm in operated carotid territory.

    - If in OR – perform duplex, very low threshold to re-explore and confirm patency of ICA with duplex or doppler. If ICA open and concerned for distal embolization, then perform a cerebral angiogram and proceed with thrombolysis or thrombectomy.(Fletcher et al. 2016)

    - If in Recovery or on the floor – many would consider CTA first line vs duplex to look for thrombosis.

- Risk factors and how to manage hyperperfusion syndrome?

    - Defined as an ipsilateral headache, hypertension, neurological deficits likely in distribution of the carotid which can progress to seizures and intracranial hemorrhage (75-100% mortality). Can present 2-3 days post surgery.(Kaku, Yoshimura, and Kokuzawa 2004)

    - Patients with uncontrolled hypertension are at risk for hyperperfusion syndrome, clinical practice guidelines from SVS recommend strict BP control following CEA, with target pressure being less than 140/80mmHg or 20mmHg from preoperative baseline.(Abou-Chebl et al. 2004)

- High risk groups

    - ESRD patients have higher rates of perioperative stroke, but also have higher rates of stroke if not revascularized. (Klarin et al. 2016)

Long term complications and follow up

- Recommend f/u US duplex of bilateral carotids at </=30 days. >/= 50% stenosis further imaging is indicated.
- Ipsilateral restenosis
  - 0-3mo likely due to inadequate endarterectomy or clamp injury.(Kang et al. 2014)
  - 1-3y likely neointimal hyperplasia, smooth and regular. If <80% stenosis and asymptomatic, then duplex surveillance and antiplatelet. If progresses, then often endo treatment first.(Garzon-Muvdi et al. 2016)
  - Risk factors for restenosis include hyperlipidemia(LaMuraglia et al. 2005), female gender, primary closure without patch, younger age, and certain plaque characteristics-abundance of smooth muscle, absence of macrophages/lymphocytes, and lipid core <10%.(Hellings et al. 2008; Pauletto et al. 2000)
- Contralateral stenosis:
  - The risk of progression for moderate stenosis at the initial surveillance to severe stenosis in the contralateral artery can be as high as 5 times.
  - Requires post-operative surveillance.
- Carotid artery patch infections
  - Rare complication (<1%) presenting as phlegmon, pseudoaneurysm, sinus tract or carotid cutaneous fistula.(Patrick A. Stone et al. 2011)
  - Treatment is excision and replacement with autologous tissue.(Fatima et al. 2019)

1.3.4 Endovascular - Carotid Artery Stenting

- In patients aged >70 the risk of intra- or post procedure stroke was the highest, if undergoing CAS, presumably due to calcified disease in the arch.
  - Lesion-specific characteristics are thought to increase the risk of cerebral vascular events after CAS and include a “soft” lipid-rich plaque identified on noninvasive imaging, extensive (15 mm or more) disease, a pre-occlusive lesion, and circumferential heavy calcification.
  - This risk can be reduced, but not eliminated, by using flow-reversal embolic protection rather than distal filter protection.
- Limited data is available on CAS in asymptomatic patients - currently is not supported by guidelines or considered reimbursable.

- Consider CAS in symptomatic patients with >50% stenosis who are poor candidates for CEA due to severe uncorrectable medical comorbidities and/or anatomic considerations:
  - Ipsilateral neck dissection or XRT - equivalent periprocedural stroke rate to CEA, but increased later stroke rate. CEA higher rates of cranial nerve damage (9%). (Giannopoulos et al. 2018)
  - Contralateral vocal cord paralysis.
  - Lesions that extend proximally to the clavicle or distal to C2.
- Transfemoral Approach vs Transcarotid approach
  - ROADSTER Trial - single arm study with flow reversal for cerebral protection. Suggested lower rates of post-op stroke in patients undergoing TCAR.
  - TCAR contraindications include previous CCA intervention, CCA disease at entry site, <5cm CCA (clavicle to bifurcation) for sheath, CCA < 6mm, contralateral recurrent laryngeal or vagus nerve injury.(Kwolek et al. 2015)
  - Patient needs to be preloaded with dual antiplatelet medications for at least 5-7d, should delay procedure if has not been appropriately loaded with antiplatelets.(Brott et al. 2010; Kwolek et al. 2015; Valls et al. 2017)
  - Studies with TCD have shown that there may be reduced embolization rates with TCAR over transfemoral stenting.(Plessers et al. 2016)
- Complications
  - Post stenting ICA thrombosis should first be treated with abciximab and catheter directed thrombolysis, followed by endo-salvage with suction thrombectomy, repeat angioplasty or stenting. May require eventual conversion to CEA.(Coelho et al. 2019; A. R. Naylor et al. 2018)
- Post-CAS follow up - Dual-platelet therapy should be continued for 1 month after the procedure, and aspirin should be continued indefinitely.
  - In stent restenosis (>50%) - 4-times higher rate of stroke than no recurrence of disease, but more benign than de novo disease.(Clavel et al. 2019) Stenosis should be confirmed with CTA. No clear difference between treatments in literature - PBA, Cutting, DBA, stenting, open conversion.(Arhuidese et al. 2017) Repeat angioplasty or stent have low incidence of periprocedural stroke but failed to improve long term stroke/death/MI or patency rates.(Chung et al. 2016)

1.3.5 Summary of Prospective Trials

1. Asymptomatic Carotid Atherosclerosis Study (ACAS)
    - Compared medical management with CEA in asymptomatic patients with $> 60\%$ stenosis.
    - 5-year stroke and death rate was 5.1% vs 11%.
    - In women, the benefit of CEA was not as certain as 5y stroke and death rates were 7.3% vs. 8.7%.
    - This trial was conducted pre-statin and clopidogrel era.
2. North American Symptomatic Carotid Endarterectomy Trial (NASCET) (North American Symptomatic Carotid Endarterectomy Trial Collaborators 1991)
    - Compared medical management vs CEA for symptomatic patients with moderate (50-69%) and severe stenosis (>70%)
    - Only moderate impact for patients with moderate stenosis (50-69%)
    - Symptomatic patients with >70 % stenosis benefited from CEA, at 18 months 7% major stroke in surgical arm, and a 24% stroke rate in medical arm. 29% reduction in 5-year risk of stroke or death.
        - Patients with severe >70% stenosis had such a dramatic effect the trial was stopped early for this subset and all referred for endarterectomy.
    - No benefit is shown in symptomatic patients with $< 50\%$ stenosis
    - European studies have shown similar results
        - ACST = ACAS
        - ECST = NASCET.
    - **Take a listen!** Click hear to listen to Dr. William Jordan discuss this landmark paper!
3. Carotid Revascularization Endarterectomy versus Stenting Trial (CREST).(Silver et al. 2011)
    - Compared CEA vs. CAS in both symptomatic and asymptomatic patients.
    - Composite endpoint of 30-day stroke, MI, death equivalent between CEA and CAS.
    - CAS had a significantly higher incidence of stroke and death than CEA and CEA higher incidence of MI.

  - Follow up at 10 years demonstrated no difference in composite stroke/MI/death but increased rate of stroke/death in stented patients likely attributable to increased periprocedural stroke. (Brott et al. 2016)
  - CAS patients with stroke had a higher impact on QOL than CEA patients with MI.(Brott et al. 2010; Redfern, Rodseth, and Biccard 2011; Mantese et al. 2010)
- Subanalyses identified that older patients (>70y) had better outcomes after CEA than CAS, the QOL impact of stroke was more significant than that of MI, and anatomic characteristics of carotid lesions (longer, sequential, remote) were predictive of increased stroke and death after CAS.
- Unfortunately, this study provides a benchmark to strive for, but no other large trials have achieved these results.

4. ROADSTER(Kwolek et al. 2015)
   - Single arm feasibility trial of transcarotid carotid stenting.
   - The results of the ROADSTER trial demonstrate that the use of the ENROUTE Transcarotid NPS is safe and effective at preventing stroke during CAS. The overall stroke rate of 1.4% is the lowest reported to date for any prospective, multicenter clinical trial of CAS (i.e. CREST). No direct comparison.
5. Trials to look out for in the next few years:
   - CREST-2 - a multicenter, randomized controlled trial underway evaluating revascularization against modern intensive medical management.
   - ACT-1 and ACST-2- the role of intervention in asymptomatic patients, designed to compare the early and long-term results of CEA vs CAS and best medical management.
   - ROADSTER-2 - TCAR.

1.4 Uncommon Carotid Disease

1.4.1 Complicated Extracranial Occlusive Disease

- Occluded Carotid: What is the management of an occluded carotid?
  - Leave it alone.
- What if an occluded carotid on imaging is still causing TIAs?

  - Ongoing symptoms in the setting of carotid occlusion is known as carotid artery stump syndrome. Symptoms are likely due to emboli from friable intima of the internal carotid or from the external carotid through collaterals. Treatment is external carotid endarterectomy and ligation of the internal carotid.(Hrbáč et al. 2012)
  - The addition of oral anticoagulation is likely to reduce the rate of recurrent stroke.

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

- What about tandem lesions in the carotid in a symptomatic patient, carotid bulb and carotid siphon lesion (high ICA)? How should you treat this?
  - Treat carotid bulb and extracranial disease first, likely to be the embolic source.
  - Treatment of intracranial disease has been shown to increase rates of stroke and death.(Chimowitz et al. 2011). Extracranial to intracranial bypass has increased risk of stroke over BMT.(E. B. S. Group 1985)

- What if the patient has severe vertebrobasilar insufficiency and carotid artery disease?
  - Should undergo carotid revascularization first to improve flow. Then reassess for vertebrobasilar insufficiency.
  - Vertebrobasilar insufficiency characterized by dizziness, ataxia, nausea, vertigo and bilateral weakness. (Lima Neto et al. 2017a; Nouh, Remke, and Ruland 2014)

1.4.2 Vertebrobasilar Disease

- How do you treat isolated vertebral disease?
  - Most common etiology is atherosclerosis, but can also be caused by embolism, dissection or migraines.(Lima Neto et al. 2017b)
  - Symptomatic primary vertebral disease with >60% stenosis should be treated. Vertebral divided into segments. V1 preforaminal (lower than C6), V2 foraminal (C6-C2), V3 (C2-dura), V4 (intracranial).(M. Morasch 2019; Berguer, Morasch, and Kline 1998)

  - Disease of V1 should be treated with vertebral transposition of bypass with vein.(Rangel-Castilla et al. 2015)
    * Most common complication is Horner's Syndrome due to disruption of the sympathetic chain (20%), next is TIA (1-3%).(Coleman et al. 2013; C. A. Ramirez et al. 2012)
  - Disease of V2 is most difficult to access and should be treated with bypass from ICA to V3.(Berguer 1985)
  - RCT of BMT vs endo treatment did not show superiority of endo treatment, recurrent symptoms most common, periprocedural stroke was rare.(George A. Antoniou et al. 2012; Coward et al. 2007)

1.4.3 Carotid artery dissection

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

1.4.4 Internal Carotid Artery Aneurysms

- Defined as dilation of the bulb 200% of the internal carotid artery or 150% of the common carotid artery. Like popliteal aneurysms the major risk is thromboembolization.(Bush, Long, and Atkins 2019; Fankhauser et al. 2015; Pourier et al. 2018)
- Treatment is resection with primary repair or interposition. Distorted anatomy increased the risk for cranial nerve injury (12%).(Welleweerd et al. 2015)
- Redundant ICA - should be repaired if patient symptomatic due to kinking. Treated with resection and primary repair with shortening and straightening.(Ballotta et al. 2005)
- Fibromuscular Dysplasia (FMD)(Jeffrey W. Olin and Sealove 2011) - for more, see @ref(fibromuscular-dysplasia-fmd)
  - Asymptomatic disease can often be treated with antiplatelets alone.
  - Duplex ultrasound demonstrates classic "chain of lakes" appearance. For more see @ref(extracranial)

- Associated with intracranial aneurysms (13%), therefore may be reasonable to also pursue brain imaging if identified. However, no formal guidelines exist.(Lather et al. 2017)

1.4.5 Carotid Body Tumors (CBT)

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

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

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

Evaluating for carotid body ligation - When involved with a CBT or other skull base tumors, cerebral perfusion should be thoroughly evaluated prior to surgical resection to determine whether a patient will tolerate carotid ligation. Evaluation includes balloon occlusion testing with xenon-enhanced SPECT imaging. Low risk for ligation if no neurologic deficits (after 30min and hypotensive challenge) and normal diffusion of SPECT (ratio of >0.9). Angiography with contralateral occlusion evaluates collaterals.(Sugawara et al., n.d.; Tansavatdi et al. 2015)

2 Trauma - Cerebrovascular

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2.1 Neck Exposure Techniques

2.1.1 Carotid Artery

Anatomy

- Right common carotid – originates from brachiocephalic artery
  - External landmark: right sternoclavicular joint
- Left common carotid – originates from aortic arch in superior mediastinum
- Common carotid artery (CCA) is within the carotid sheath
  - Contents:
    - Common and Internal carotid (medially)
    - Internal jugular vein (laterally)
    - Vagus nerve (posteriorly)
  - Anterior margin: sternocleidomastoid (SCM) and omohyoid muscle
  - Posterior margin: longus colli and longus capitis muscles
  - Medial margin: esophagus/trachea
- Common carotid bifurcates to external and internal at level of superior border of thyroid cartilage
  - External carotid artery
    - Medial to internal carotid artery (majority of course)
    - First branch is superior thyroid artery (near bifurcation)

        * Terminate in parotid gland, divide into superificial temporal and maxillary arteries
    - Internal carotid artery
        * No extracranial branches
        * Lateral to external carotid artery until level of skull base, where it crosses medially
        * Enters the skull base through the carotid canal behind the styloid process
    - Facial vein is the anatomical landmark that approximates the location of the carotid bifurcation (deep to it).
    - Hypoglossal nerve (CN XII) and posterior belly of digastric muscle superficially cross the internal and external carotid arteries at the angle of the mandible
    - Glossopharyngeal nerve (CN IX) crosses in front of internal carotid artery, superior to hypoglossal nerve (CN XII)

Exposure

- Longitudinal incision along anterior border of SCM, extending from suprasternal notch to mastoid process
    - For proximal common carotid artery control, would do combination of SCM incision with median sternotomy
- Incise platysma and expose anterior border of SCM, retract laterally
    - Identify and avoid Accessory nerve (CN XI) that enters SCM
    - Small branches of external carotid should be ligated to adequately mobilize the SCM and expose carotid sheath
- Carotid sheath is visualized and incised longitudinally
    - Omohyoid muscle may require division if proximal exposure required
- Identify Internal jugular vein, mobilize, and retract laterally
- Identify Vagus Nerve and avoid injury (posterior between internal jugular vein and common carotid)
- Place vessel loops around CCA, internal jugular, and vagus nerve
- Identify facial vein (overlying CCA bifurcation), Hypoglossal nerve (CN XII) and Ansa cervicalis

  - Ligate facial vein to further mobilize the internal jugular vein laterally and expose the underlying carotid bifurcation
  - Identify and protect Hypoglossal nerve (CN XII)
  - Ansa cervicalis may be ligated if necessary for exposure
- Dissect carotid bifurcation
  - Careful to not stimulate carotid body and cause hemodynamic instability
  - If access to distal internal carotid artery is required, may consider subluxation of mandible or mandibular osteotomy to gain appropriate exposure

2.1.2 Vertebral Artery

Anatomy

- First cephalad branch of subclavian artery
- Divided into 3 parts
  - Part I (proximal)
    * Origin at subclavian artery to C6 (enters transverse foramen)
    * External landmarks: found between two heads of SCM (sternal and clavicular) and clavicle
    * Vertebral artery runs between anterior scalene and longus colli muscles
  - Part II - Courses through bony vertebral canal (transverse foramen) from C6 to C1
  - Part III (distal) - Courses outside vertebral canal from C1 to base of skull (enters through foramen magnum) and joins contralateral vertebral artery to form basilar artery (part of circle of Willis)

Exposure

- Will focus on exposure of Proximal Vertebral Artery
- Supraclavicular transverse incision extending between the sternal and clavicular heads of the sternocleidomastoid (SCM) muscle
- Incise platysma and continue dissection into base of triangle
- Carotid sheath is first vascular structure identified
  - Jugular vein – lateral

  - Common carotid – medial
  - Vagus nerve – posterior

- Identify scalene fat pad between two heads of SCM and clavicle, dissect to expose anterior scalene muscle
  - Careful of phrenic nerve as it runs on surface of anterior scalene muscle
- Divide anterior scalene muscle to visualize the subclavian artery and two of its branches (thyrocervical trunk and internal mammary artery). Control each branch.
- Proximal VA is located deep to supraclavicular artery and anterior to groove between C7 vertebral body and transverse process
- Proximal VA is between anterior scalene (laterally) and longus colli (medially)
- Best found through palpation with tip of index finger within groove of C7 vertebral body and transverse process
- Avoid injury to vertebral venous plexus as it is anterior to VA
- Use right angle clamp to dissect out VA

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

2.2 Tenets of Cerebrovascular Injury

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

2.3 Blunt Trauma

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

2.3.1 Evaluation

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

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

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

2.3.2 Managment

Considerations

- Consider the overall injury severity of the trauma patient with any concomitant injuries that may prevent antiplatelet/anticoagulation initiation (ie TBI) or intervention
  - Oftentimes have other blunt vascular injuries, such as aortic injuries, that need to be addressed as well
  - Can consider delay of intervention (approx. 7 days) to decrease risk of neurological events
- If there is a change in the neurological exam after initiation of medical management, considered as failure of medical therapy and surgical intervention is indicated

- If unable to obtain a reliable neurological exam, can consider transcranial doppler or EEG if accessible, but is not mandatory
- Continue supportive measures to prevent secondary injuries, ie optimize cerebral perfusion pressure
- Repeat imaging with change in neurological status
- Serial imaging important in assessing whether injury has evolved
- Endovascular repair usually involves transfemoral approach for access with arch aortogram, selection of defective carotid vessel, and placement of the endovascular treatment (ie: stent graft, bare metal stent, coil embolization of pseudoaneurysm)

Carotid Artery Injury Treatments

- Grade I and II injuries are recommended to be treated medically with antiplatelet/anticoagulation therapy if patient is able to tolerate
  - Repeat CTA imaging in 5-7 days while inpatient to ensure that injury has not evolved in acute phase
  - Follow-up CTA imaging within 1 month of discharge as outpatient to assess healing. Imaging needs to be continued until there is resolution of lesion.
  - Grade I lesions typically improve over time, Grade II lesions or higher have a greater chance of evolving (approximately 60%)
- Accessible Grade III and V carotid injuries should be repaired.
  - Grade III pseudoaneurysms should be repaired endovascularly with a covered stent (ie stent graft that covers the opening of the pseudoaneurysm) or bare metal stent (maintains integrity of the wall). Through these stents, coils can be placed to thrombose the pseudoaneurysm. Flow needs to be maintained.
  - Open approach can be considered for Zone II injuries
- Grade I and IV carotid injuries, and inaccessible Grade II and III carotid injuries should be treated with medical management (antiplatelet/anticoagulation)
  - Grade IV injuries without associated injuries that preclude use of anticoagulation, would heparinize and proceed with anticoagulation (avoid bolus of heparin, goal PTT 50-70 within first 24 hours). Monitor for bleeding. Transition to oral anticoagulant for 30-90 days.
  - Grade IV injuries with contraindications for anticoagulation, would recommend dual antiplatelet therapy (aspirin and clopidogrel)

  - Overall, anticoagulation is preferred if there are no contraindications. If there are, then dual antiplatelet therapy is preferred.

- Grade V and persistent Grade III should be repaired surgically

Vertebral Artery Injury Treatments

- Special Considerations
  - Attempt to determine whether the dominant or diminutive vertebral artery is involved
  - Treatment is again based on severity with grading system (Denver Grading Scale), consider location and extent
  - Consider any associations with a basilar artery defect or posterior circulation stroke
  - Grade I-IV vertebral injuries should be treated with medical management as first line, approximately 90% self-resolve
  - Endovascular interventions can be considered for those patients with severe injury and/or who are symptomatic
    * Usually performed by neuro-interventional as the injury is located within the skull base
  - Serial imaging still needs to be performed to evaluate healing (similar manner to carotid injury)
  - In the setting of trauma, if there is an expanding hematoma due to vertebral artery injury and open approach is performed to control bleeding, vertebral artery is usually ligated in this scenario
    * Vertebral reconstructions are not well-suited for traumas

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

2.4 Penetrating Trauma

Levels

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

Images of the neck zones can be found here

2.4.1 Management

Injury to Zone I and Zone III

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

Injury to Zone II (violation of platysma)

- Direct to OR for exploration, especially with high suspicion of carotid injury and neurovascular deficits (hard sign)
- Repair carotid artery if injured, even if thrombosed
  - The risk of reperfusion injury causing an intracranial hemorrhage was traditionally considered a reason to not repair.
  - However, overall mortality and final neurostatus has shown to be better with intervention, even if patient started with neurodeficits.
  - Requires communication with operative team (ie anesthesia) to mitigate hypoperfusion
- Consider concomitant injury to aerodigestive tract
- Scenario: Patient has large cerebral infarct on same side as carotid injury.
  - Continue to OR to explore
  - Preferred management is to repair and attempt reperfusion of hemisphere, possibly perfusing a previously ischemic penumbra
  - Ligation usually not an option.
    - Unless carotid is completely occluded and there is no retrograde bleeding after attempted thrombectomy » there is potential for ligation, but not readily considered.