Post-op Carotid Complications
A Nursing Perspective of What to Watch Out for

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Post-op Carotid Management

Objectives

- Review the potential complications of carotid surgery: CEA and CAS
  - Cranial nerve palsies
  - Hemodynamic instability
  - Hyperperfusion syndrome
  - Stroke, MI
  - Post op neck hematoma
- Discuss the clinical management of these complications when applicable

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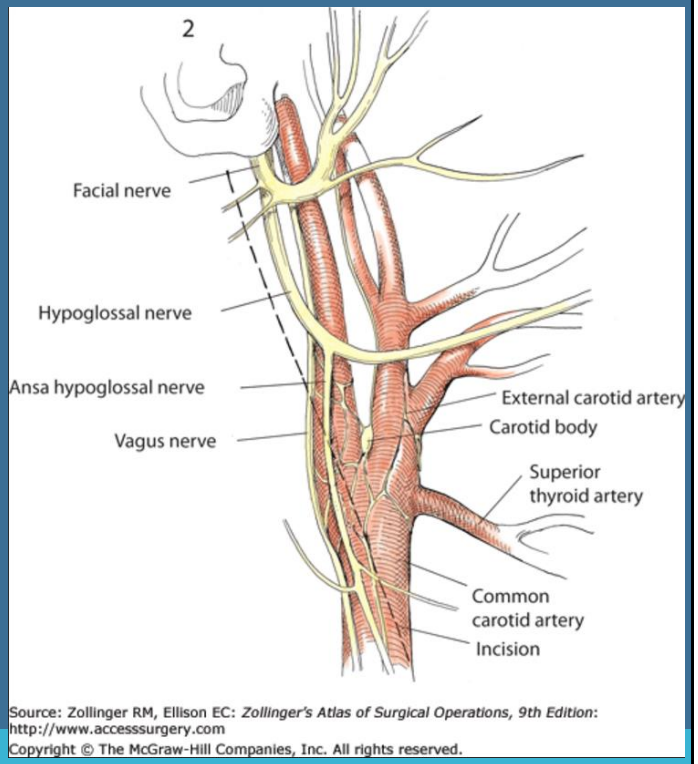
Cranial Neuropathies Post Carotid Endarterectomy (CEA)

- Cranial neuropathies are more commonly associated with CEA & rarely carotid artery stenting (CAS)
  - CAVATAS Study. Lancet 2001; 357: 1729
- Reported incidence is ~ 5% of post op CEA patients will have cranial nerve injury at discharge
- Types of cranial nerve injuries include:
  - Hypoglossal nerve
  - Facial nerve
  - Vagus nerve/laryngeal nerve (branch of vagus)
  - Glossopharyngeal nerve
- Factors associated with cranial n. injuries per Vascular Surgery Group New England (VSGNE)
  - Urgent surgery, immediate re-exploration/unintended return to the OR



Source: Zollinger RM, Ellison EC: *Zollinger's Atlas of Surgical Operations, 9th Edition*: http://www.accesssurgery.com

European J Endov Surg 2014; 47(1): 2-7

Cranial Neuropathies Post Carotid Endarterectomy (CEA)

- Types of cranial nerve injuries include
  - Hypoglossal nerve injury is the most common
    - Symptoms: tongue deviation
  - Facial nerve, marginal mandibular branch injury:
    - Symptomas: lower facial weakness causing asymmetric smile
  - Vagus nerve/laryngeal nerve injury:
    - Symptoms: hoarse voice, vocal cord paralysis
  - Glossopharyngeal nerve injury
    - Symptoms: difficulty swallowing

Treatment

- No specific treatment
- Important to distinguish from stroke

Prognosis

- 88% of the cranial nerve injuries had resolved at year

European J Endov Surg 2014; 47(1): 2-7

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Post-op Hemodynamic Instability

Review of the functions of a Normal Carotid sinus:

Within the adventitia of the internal carotid artery are baroreceptors that can sense change in the blood pressure. They are sensitive to stretch

These barorecptors receive innervation from the glossopharyngeal nerve

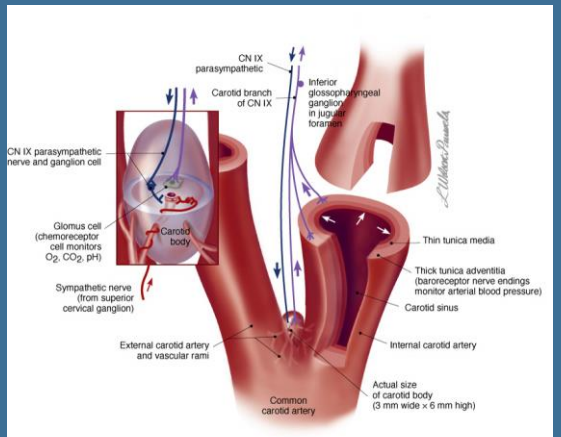

Figure IX–6 The bifurcation of the common carotid artery demonstrating baroreceptors in the wall of the carotid sinus and chemoreceptors within the carotid body.

From "Cranial Nerves in Health and Disease" 2002, © Wilson-Pauwels, Akesson, Stewart, Spacey, B C Decker Inc.

High blood pressure: baroreceptors are stretched, leading to **decreased** sympathetic stimulation
Low blood pressure: baroreceptors are not stretched, leading to **increased** sympathetic stimulation

Hemodynamic Instability Post CEA & CAS

During CEA or CAS, these baroreceptors are disrupted, which leads to hemodynamic instability

- Hypotension
- Hypertension
- Bradycardia

Incidence: more common in CAS but present in both, reported between 20-80% of patients post CAS

Timing: symptoms start peri-operatively and are usually short lived from hours up to 2 days.

Hemodynamic Instability Post CEA & CAS

Treatment

Bradycardia: symptomatic and/or severe bradycardia (< 40) may be treated with atropine or glycopyrrolate

- For severe cases external pacing may need to be considered

Hypotension: symptomatic and/or severe hypotension (<90 systolic) may be treated with IV fluid boluses and/or vasopressor therapy (such phenylephrine)

***caution should be used to make sure that the patient does not become hypertensive which could cause hyperperfusion syndrome and/or intracranial hemorrhage**

Hypertension (> 140/90): may be treated with IV labetolol depending on the heart rate or Nicardipine infusion

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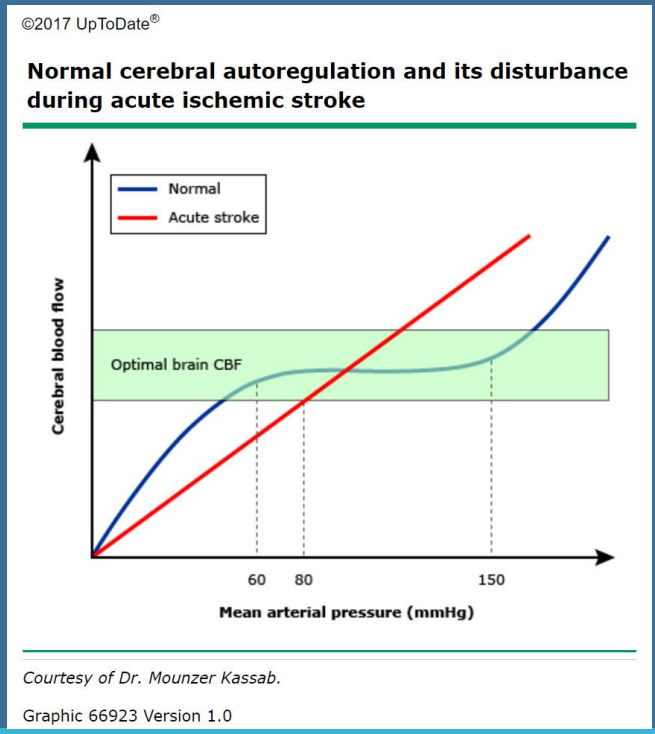
Cerebral Hyperperfusion Syndrome Post CEA & CAS

Before reviewing what happens in cerebral hyperperfusion syndrome, quick review of cerebral autoregulation

In cerebral autoregulation, cerebral blood flow is maintained at a constant level despite a change in mean arterial pressure

- There is dilation and constriction of cerebral vessels in response to changes in mean arterial pressure
- So if the MAP increases, cerebral vessels constrict to maintain stable cerebral blood flow
- Cerebral vessels also dilate and constrict in response to other factors such as hypoxemia and increased CO2



Up to Date April 2017 "Pathophysiology of Ischemic Stroke"

Cerebral Hyperperfusion Syndrome Post CEA

What is it?

- In a chronically hypoperfused cerebral hemisphere as is the case with severe carotid stenosis, the small vessels in the brain ipsilateral to the carotid stenosis are chronically maximally dilated
- Post CEA or CAS, normal blood flow is restored, but the small vessels that are maximally dilated are unable to vasoconstrict due to impaired cerebral autoregulation. Thus cerebral blood flow and cerebral perfusion are too high
- Too high cerebral blood flow leads to hemorrhage and/or cerebral edema

Up to Date April 2017 "Complications of Carotid Enderacterctomy"

Cerebral Hyperperfusion Syndrome Post CEA & CAS

Incidence: most studies have reported this syndrome in 0-3% of patients. Less common with CAS than CEA

- Recent VQI (vascular quality initiative) Registry data 2003-2013 showed 0.18% risk in 51K CEA cases
- Mortality with the syndrome was 38%

J Vasc surgery 2017; 65(2): 381-9

Timing: within 2 weeks of the procedure

Factors associated with hyperperfusion syndrome:

- < 1 month ipsilateral stroke
- ≥ 70% stenosis or occlusion of contralateral carotid
- Female gender
- Coronary artery disease
- Post operative blood pressure lability

J Vasc Surgery 2017; 65(2): 381-9

Cerebral Hyperperfusion Syndrome Post CEA & CAS

Clinical signs and symptoms:

- Elevated blood pressure
- Headache
- Change in mentation and/or focal neurological deficit
- Seizures
- **Intracranial hemorrhage: most serious complication**
- Brain imaging: ICH and/or cerebral edema without cerebral infarction

CT & MRI Brain show vasogenic edema in L hemisphere in a patient with cerebral hyperperfusion syndrome, but diffusion weighted images are negative for cytoxic edema

MRI 2 months later shows resolution of the vasogenic edema

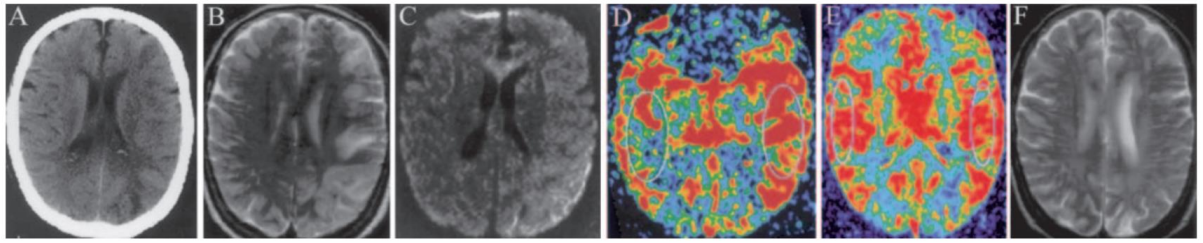


Figure 3. Patient 2. Nonenhanced axial CT, 7 hours after the onset of symptoms, shows left frontal, parietal, and occipital hypodensities involving predominantly the white matter, and some involvement of the overlying cortex with sulcal effacement (A). A few hours later, axial T2-weighted MR images demonstrate diffuse edema of the left hemisphere (B). On axial DW images at the same level, these regions are isointense to slightly hyperintense, consistent with the absence of acute brain ischemia. The slight diffuse DWI hyperintensity is most probably caused by the T2 component of DW images, known as the T2 shine-through effect. (C). PWI documents a relative hyperperfusion (RID=44%) of the left hemisphere (D). A repeat PWI 6 days later demonstrates complete resolution of the previous relative interhemispheric difference (E). Two months later, the extensive T2 hyperintensities present in B have resolved (F).

Stroke 2005; 36: 21-26

Cerebral Hyperperfusion Syndrome: Post CEA & CAS

Prevention & Treatment

- **Strict blood pressure control post operatively**
  - < 140/90mmHg vs. 150/90
  - Nitrates could cause cerebral vasodilation so these should not be used
- Seizure management with anticonvulsant (AED)
  - Prophylactic AED is not recommended
  - AED is reserved for patients with seizures
- ICH management
  - Patient should be transferred to the ICU and placed on an ICH protocol
  - Blood pressure control

Outcome

- Patients with no ICH can recover fully
- Patients with ICH have reported mortality up to 50%

Cardiology in Review 2012; 20(2): 84-89

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Symptomatic Stenosis

Trials including Symptomatic Carotid Stenosis	Perioperative 30 day risk of stroke and/or death	Perioperative risk of MI
CEA		
NASCET *NEJM* 1991; 325(7): 445	5.8%	0.9%
ECST *Lancet* 1991; 337 (8752)): 1235	7.5% (for severe stenosis)	---
CREST (CEA arm) NEJM 2010: 363 (1): 11	3.2%	2.3%
ICSS Lancet 2010; 375: 985	4%	0.6%
CAS		
CREST (CAS arm) NEJM 2010: 363 (1): 11	6%	1%
ICSS Lancet 2010; 375: 985	7.4%	0.4%

Asymptomatic Stenosis

Trials including Asympatomatic Carotid Stenosis	Perioperative 30 day risk of stroke and/or death	Perioperative risk of MI
CEA		
VA Trial NEJM 1993; 328: 221	4.7%	3.8%
ACAS JAMA 1995; 273: 1421-8	3.4%	
ACST Lancet 2004; 363: 1491	3.1%	---
CREST NEJM 2010: 363 (1): 11	1.4%	2.2%
CAS		
CREST NEJM 2010: 363 (1): 11	2.5%	1.2%

New Sudden onset neurological symptoms

- Notify Provider immediately
  - Possibilities include cranial neuropathy, hyperperfusion syndrome and/or ICH, or acute ischemic stroke
  - At Swedish nurses are empowered to activate our stroke alert, "Code Bart" even prior to calling the attending of record
  - Expedites rapid neurological evaluation of the patient
- Ischemic Stroke, as opposed to potentially reversible cerebral hyperperfusion syndrome, is associated with cytotoxic edema, i.e. brain cell death
  - Stroke can lead to permanent neurological disability and/or death

Acute Ischemic Stroke post CEA or CAS

- Causes: thrombosis at CEA site or stent site, plaque emboli, or hypoperfusion
- Treatment options
  - All patients should have a CT head to exclude ICH
  - Concern for thrombosis vs. emboli:
    - Surgeon may choose return to the OR immediately for exploration of the surgical site **vs.** obtain STAT duplex , CTA, or angiography
    - Endovascular treatment of intracranial large vessel occlusion (i.e. terminal ICA or MCA occlusion)

Myocardial Infarction (MI) Post CEA and CAS

- Reported incidence is low after CEA or CAS
- If patients have chest pain post procedure, they should have work up for MI including EKG, cardiac enzymes, and cardiology consultation

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  - **Post op neck hematoma vs. groin hematoma**
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How Common are Neck Hematomas vs. Groin Hematomas?

CEA

CEA Trials	Neck Hematoma
NASCET NEJM 1991; 325(7): 445	4.3% "wound complications"
CREST NEJM 2010: 363 (1): 11	3.1%
ICCS Lancet 2010; 375: 985	5.8%
CAVATAS Lancet 2001; 357: 1729	6.7%

CAS

CAS Trials	Groin Hematoma and/or pseudoaneurysm
French EVA-3S NEJM 2006; 355: 1660-71	1.9%
ICSS Lancet 2010; 375: 985	3.6%
CAVATAS Lancet 2001; 357:1729	1.2%

Post-op Neck Hematoma after CEA

- Neck hematoma is associated with
  - Potential life threatening airway compromise

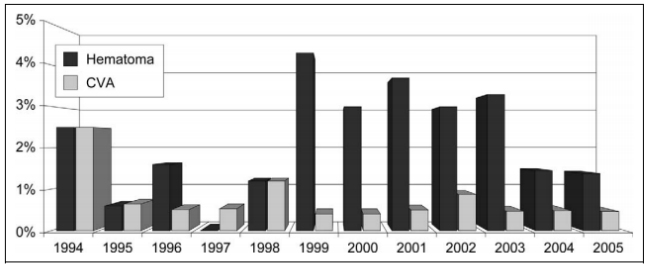

Figure 1. Percentage occurrence of cervical hematoma requiring operative evacuation and operative stroke over a 12-year period.

swelling, hypoxia, dysphagia, dyspnea, stridor, agitation

- Risks
  - **Uncontrolled post operative HTN**
  - Post-operative use of ASA + either clopidogrel vs. dextran
    - Increased incidence of neck hematomas was published in Ann Vasc Surg 2012; 46(8): 610-6
  - Mixed results regarding pre-operative clopidogrel.
    - **Recent study of 188 patients in Italy showed no increased risk in patients with CAD on dual antiplatelet therapy** (ASA + Plavix) in the peri-operative period of CEA. No post op dextran

      Ann of Vasc Surg 2017; 40: 39-43

Post-op Groin Hematoma and/or pseudoaneurysm after CAS

- Risk factors:
  - Obesity, HTN, hemodialysis, inadequate post procedure compression of the groin site, age >65, post procedural anticoagulation, PVD
- Timing of pseudoaneurysms:
  - most within 3 days,
  - maybe delayed until 7 days post –op
- Treatment:
  - Smaller ones – compression +/- thrombin injection
  - Larger ones/more complex ones – may require surgical repair

Nursing Check List
Post-op Carotid Management

✓Frequent vital signs with close monitoring of the blood pressure

PRN anti-hypertensives. Strict goal BP of LESS than 140/90 or < 150/90

Patient needs to be on a floor where PRN IV meds may be given: ICU vs. prolonged post anesthesia recovery unit then step down unit

✓Vascular access monitoring

Patients must be monitored clo atomas

✓F uro checks

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- Cranial neuropathy
- Cerebral hyperperfusion syndrome
- ICH
- Ischemic stroke