Carotid Endarterectomy

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I. Introduction – Stroke and Transient Ischemic Attacks (TIAs) [1-2]

A. Stroke statistics[1]

- Stroke = third leading cause of death in US (after heart disease and cancer)
- Over 500,000 new cases in US each year (75% in carotid distribution)
- Mortality: nearly 1/4 die.
- Morbidity: often significant and permanent disability

B. Types and causes of stroke[2]

1. Ischemia strokes (75%) – due to occlusion of a blood vessel to the brain
   a. Anterior circulation strokes
      - = carotid territory strokes
      - Usually involve the cerebral hemispheres
   b. Posterior circulation strokes
      - = vertebrobasilar territory strokes
      - Usually affect brain stem or cerebellum
2. Hemorrhagic strokes (25%)
   a. Subarachnoid hemorrhage (SAH)
      - = bleeding on to surface of brain
      - Aneurysm = most common site
   b. Intracerebral hemorrhage
      - = bleeding into brain parenchyma
      - Hypertension = most common cause

C. Treatment of stroke – now includes thrombolytic therapy[2].

- Thrombolytic agents = TPA, urokinase, streptokinase
- IV thrombolytic therapy is best initiated within 3 hours after onset of stroke symptoms.

D. ACLS algorithm for suspected stroke patients[2]

Detection	Early recognition of "brain attack"
Dispatch	Activate EMS (call 911)
Delivery	To receiving hospital
Door	Rapid ER triage
Data	Rapid ER evaluation and CT scan
Decision	About potential therapies
Drug therapy	Including throbolytic agents

1. Detection
   a. Hallmark of stroke = sudden onset of focal brain dysfunction
   b. Early recognition of "brain attack" signs and symptoms

1) Carotid (anterior) circulation
   - Unilateral paralysis
   - Numbness
   - Language disturbance
   - Visual disturbance
   - Monocular blindness
2) Vertebrobasilar (posterior) circulation
   - Vertigo
   - Visual disturbance
   - Diplopia
   - Paralysis
   - Numbness
   - Dysarthria
   - Ataxia

2. **Dispatch** – early activation of EMS
3. **Delivery** – prehospital transport and management
   - Minimize time in the field.
4. **Door**
   a. Immediate general assessment: < 10 min from arrival
      - Assess ABCs, vital signs.
      - Provide oxygen by nasal cannula.
      - Obtain IV access; obtain blood supplies (CBC, electrolytes, coagulation studies).
      - Check blood sugar; treat if indicated.
      - Perform general neurologic screening assessment.
      - Alert stroke team: neurologist, radiologist, CT technician.
   b. Immediate neurologic assessment: < 25 min from arrival
      - Review patient history.
      - Establish onset (< 3 hours required for thrombolytics).
      - Perform physical exam.
      - Perform neurologic exam – determine level of consciousness (Glasgow Coma Scale) and level of stroke severity (NIH Stroke Scale or Hunt-Hess Scale).
      - Order urgent noncontrast CT scan (door-to-CT scan performed: goal < 25 min from arrival).
      - Read CT scan (door-to-CT scan read: goal < 45 min from arrival).
      - Perform lateral cervical spine x-ray (if patient comatose or history of trauma).
   c. Glasgow Coma Scale — Score
      1) Eye opening
         - Spontaneous — 4
         - In response to speech — 3
         - In response to pain — 2
         - None — 1

2) Best verbal response
   - Oriented conversation 5
   - Confused conversation 4
   - Inappropriate words 3
   - Incomprehensible sounds 2
   - None 1
3) Best motor response
   - Obeys 6
   - Localizes 5
   - Withdraws 4
   - Abnormal flexion 3
   - Abnormal extension 2
   - None 1

d. Hunt-Hess Scale for Subarachnoid Hemorrhage

Grade	Neurologic Status
1	Asymptomatic
2	Severe headache or nuchal rigidity; no neurologic deficit
3	Drowsy; minimal neurologic deficit
4	Stuporous; moderate to severe hemiparesis
5	Deep coma; decerebrate posturing

5. **Data** – does the CT scan show intracerebral or subarachnoid hemorrhage?
   a. If yes, consult neurosurgery.
   b. If no, then probable ischemic stroke.
      - Review CT exclusions: are any observed?
      - Repeat neurologic exam: are deficits variable or rapidly improving?
      - Review thrombolytic exclusions: are any observed?
      - Review patient data: is symptom onset now > 3 hours?
6. **Decision** – is the patient a candidate of thrombolytic therapy?
7. **Drug** – begin thrombolytic therapy with IV TPA, streptokinase or urokinase.

E. Transient ischemic attack (TIA)[2]

1. TIA = a reversible episode of focal brain dysfunction
   - Initial signs and symptoms of stroke
   - Lasts only a few minutes to hours before resolving
2. TIA = the most important forecaster of brain infarction
   - 5% will develop cerebral infarction within 1st month.
   - Risk = 12% at one year, and an additional 5% for each year after that
3. Treatments for TIA
   - Carotid endarterectomy – if severe (> 70% narrowing) of internal carotid artery
   - Aspirin or ticlopamide

- Oral anticoagulants – to prevent embolism to brain in patients with cardiac causes of stroke, especially atrial fibrillation

II. History of Carotid Endarterectomy (CEA) Surgery

A. Early history[3]

- 1950s: CEA first used to treat cerebrovascular disease
- 1971: 15,000 operations per year in US
- Mid-1980s: > 100,000 operations per year
- 1984: CEA = 3rd most commonly performed operation in US

B. Late 1980s: the appropriateness of CEA surgery was questioned.[4]

- "Only two prospective randomized trials had been carried out, neither of which demonstrated clear benefit."[3]
- In the patient with TIAs, which is better? Medical management, surgery or both?
- CEA: perioperative mortality and morbidity = 10% (in one large study)[4]

C. Early 1990s

1. Symptomatic carotid stenosis – 3 clinical trials showed benefit.[5-8]
   - > 60-70% stenosis – CEA better than optimal medical care alone
   - 30-69% stenosis – value of surgery unknown, trials in progress
   - 0-29% stenosis – surgery of no value
2. Asymptomatic carotid stenosis[8-11]
   a. Recent Asymptomatic Carotid Atherosclerosis Study (1995)
      - Surgery is also of value for asymptomatic stenosis (≥ 60%).
      - 53% reduction in risk of ipsilateral stroke and any perioperative stroke or death
   b. Asymptomatic carotid bruits are common.[12]
      - Occur in 5% of population > age 45
      - More common in women
      - Risk of stroke = 2% per year

III. Case Presentation and Format for Oral Board Questions

A. Case presentation = 60-year-old male for carotid endarterectomy

- Past medical history – significant for recent TIA, cigarette smoking (1-2 ppd) and hypertension
- Physical exam – 80 kg, BP = 190/110, HR = 95
- Medications = metoprolol, hydrochlorothiazide, aspirin
- Surgeon desires intraoperative EEG monitoring.

B. Format = approach to the case as an oral board question[13]

1. Preoperative concerns (not on short stem)
   a. Problem list?
   b. Further labs and studies?
   c. Optimization? (Is the patient optimal for surgery?)
      1) Consults?
      2) Further medical treatment?
2. Intraoperative concerns
   - Premedication
   - Monitoring
   - Induction
   - Maintenance
   - Intraop complications (2 main complications?)
   - Emergence
3. Postoperative concerns (not on long stem)
   - 2 main complications?
   - Postop pain relief

IV. Preoperative Evaluation

A. Problem list

1. Carotid endarterectomy (recent TIA) – general vs regional technique
2. Cigarette smoking (1-2 ppd) – ("irritable airway," check CXR)
3. HTN – not well controlled (check BUN and Cr)
   - Metorprolol = beta-blocker (? Compliance, HR = 95)
   - Hydrochlorothiazide = thiazide diuretic (check lytes, K^+)
   - Aspirin (check bleeding time)
4. Intraoperative EEG monitoring
5. Incomplete data base (? neurologic sequelae, ? cardiac history, EKG)

B. Further studies

1. Typical diagnostic workup for TIA/stroke[3]
   a. CT scan and/or MRI
   b. Lab studies – CBC, platelet count, PT/PTT
   c. Carotid studies
      - Carotid ultrasound
      - Carotid angiography
   d. Cardiac studies
      - Electrocardiogram
      - Echocardiography
2. Evaluation of carotid artery disease[14]
   a. Many methods to evaluate[15]
   b. Duplex ultrasound = preferred noninvasive diagnostic test
   c. Carotid doppler ultrasound = next best noninvasive test
   d. Carotid angiography = "gold standard"

- Digital and magnetic resonance angiography (MRA) = new refinements
- Serious complications = 1-2%
- Expensive to perform

3. Cardiac evaluation of patients with TIAs or stroke[12]
   - Strong association between carotid and coronary artery disease
   - Cardiac events often determine the fate of the TIA patient.
   - Cardiac events occur 2 times > stroke deaths.
   - Abnormal exercise EKGs = 28% in patients with TIAs and no known cardiac symptoms
   - Abnormal stress or dipyridamole thallium studies = 45% in patients with TIA or mild stroke and no apparent heart disease
4. Combined CEA/CABG procedures
   a. Should CEA and CABG be staged or combined?
   b. Combined CABG/CE procedure was associated with a lower mortality and morbidity than staged reconstruction.[16,17]
      - Staged CABG before carotid: greater risk of cerebrovascular accident (CVA)
      - Staged carotid before CABG: greater risk of myocardial infarction (MI) or death
   c. Management of coexistent carotid and coronary artery disease[16-18]
      1) Staged CEA should rarely be performed to prepare a patient for subsequent CABG.[17]
      2) Staged CABG before CEA or medical therapy if
         - Operable coronary artery disease
         - Asymptomatic unilateral carotid stenosis
      3) Combined CEA and CABG if
         - Active neurologic symptoms or bilateral carotid disease +
         - Diffuse or unstable coronary artery disease

C. Optimization. Is the patient optimal for surgery?

- Consults – neurologist, cardiologist (if warranted by cardiac workup)
- Further medical treatment – better blood pressure control?

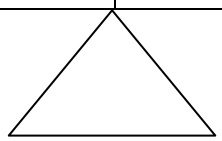
V. Intraoperative Management

A. Three main goals of anesthesia for CEA surgery

1. To protect the heart from ischemia, to protect the brain from ischemia and to have the patient awaken quickly at the end of surgery[19]
   a. **Protect the heart from ischemia.**
      1) Maintain normal hemodynamics, oxygenation and ventilation.
      2) Myocardial oxygen supply and demand balance[20]

- Avoid factors that decrease myocardial O_2 supply.
- Avoid factors that increase myocardial O_2 demand.

Decreased Myocardial Oxygen Supply	Increased Myocardial Oxygen Demand
1. Decreased coronary bloodflow	1. Tachycardia
a. Tachycardia	2. Increased wall tension
b. Dystolic hypertension	a. Increased preload
c. Increased preload	b. Increased afterload
d. Hypocapnia	3. Increased contractility
e. Coronary spasm	
2. Decreased oxygen delivery	
a. Anemia	
b. Hypoxia	
c. Decreased 2.3 DPG	



3) A high percentage of CEA candidates have coexisting severe or advanced CAD, even with no history or EKG evidence.[12]

b. **Protect the brain from ischemia.** Some guidelines to attain this goal[21]

1) Maintain a normal or high normal blood pressure.
   a) Normal or high normal BP preserves collateral flow to the brain during carotid occlusion.[19, 22]
   b) If cerebral autoregulation is impaired in ischemic areas, cerebral blood flow passively follows arterial BP.[23]
   c) Deliberate induction of hypertension with phenylephrine?[24]
      - May increase cerebral blood flow, but
      - May also increase cardiac workload and ischemia
   d) Avoid deliberate hypertension in patients.[25]
      - With known cardiac disease
      - Who develop ST segment depression when hypertension is induced.
2) Maintain normocarbia or slight hypocarbia.
   - Optimal $paCO_2$ for CEA is controversial.
   - Most anesthesiologists opt for normocarbia or slight hypocarbia.[19, 22, 25]
   - Slight hypocarbia may divert cerebral blood flow to potentially ischemic areas of brain by constricting normal nonischemic vessels.
3) Avoid extreme hyperglycemia.

- Hyperglycemia is associated with worse neurologic outcome in some animal models of cerebral ischemia.
- Avoid glucose-containing solutions unless indicated.[30, 31]
- Avoid lactated Ringer's solution; the lactate is metabolized to glucose.[19]

4) Monitor for cerebral perfusion.
5) Treat cerebral ischemia if it occurs.

c. Have the patient awaken quickly after the operation.

2. **Premedication** – "light" premedication is recommended to permit
   - Neurologic assessment during regional anesthesia
   - Fast "wakeup" after general anesthesia
3. **Monitors**. Use routine monitors as reminder for additional monitors.

Routine	Additional
a. Stethoscope	
b. EKG	ST segments
c. Blood pressure	A-line, CVP (+/-), S-G cath (+/-)
d. Temperature	Humidivent, warming methods, maintain normothermia
e. O_2 monitor (volume, PIP)	
f. Pulse oximeter	
g. $ETCO_2$ (mass spect)	Normocapnia or mild hypocapnia (shunt bloodflow to abnormal brain)
h. Restraints	Especially for regional technique, twitch monitor (muscle relaxants)
i. I&O	No D5W, no LR, Foley catheter (+/-)
j. Pressure points	Elbow padding (ulnar nerves)
Extra monitors	EEG (raw vs processed), SSEP's, stump pressures (+/- value) awake neuro assessment

4. Induction and maintenance of anesthesia
   a. Regional vs general anesthesia
      1) Does either have a "better outcome?"
      2) Many uncontrolled studies exist, but no controlled studies
      3) Regional anesthesia
         a) Advantages
            - Awake patient
            - Airway reflexes preserved
            - Intraoperative neurologic exam is possible.
            - Less postop BP problems (?)
            - Shorter ICU stay(?)
            - Shorter hospitalization(?)
         b) Disadvantages[36]
            i) Need for patient cooperation
            ii) Possible loss of patient cooperation, with onset of new neurologic deficit, because of
               - Confusion

- Panic
- Seizures

iii) Inability to secure airway if panic, seizure or oversedation occur

iv) An unexpectant delayed deficit may occur sometime after the test period.

v) Inability to administer drugs such as thiopental that might protect the brain against ischemia.

4) General anesthesia

a) Advantages

- Anesthetized, motionless patient
- Control of airway – endotracheal intubation
- Able to administer drugs such as thiopental to protect brain against ischemia

b) Disadvantages

- Intraop neurologic assessment impossible
- Need sensitive and reliable monitoring for cerebral perfusion in place of awake neurologic assessment.[36]

b. Regional anesthesia

1) Techniques[15]

- Local infiltration by surgeon
- Superficial cervical plexus block
- Deep cervical plexus block
- Combined superficial and deep cervical plexus blocks

2) Superficial cervical plexus block[37]

a) Landmarks? Crossing of...

- External jugular vein and
- Posterior border of sternocleidomastoid muscle

b) Possible complications include[37]

- Accidental injection into internal jugular vein
- Hematoma formation (tear in wall of vein)
- Venous air embolus'

3) Deep cervical plexus block

a) Landmarks?

- Line between mastoid process and suprasternal notch
- Aim perpendicularly for transverse processes of C2-C4.

b) Possible complications?

- Recurrent laryngeal nerve paralysis = most common
- Stellate ganglion block = next most common
- Cervical subarachnoid block with possible phrenic nerve block
- Direct injection of local anesthesia into vertebral artery, resulting in seizures or apnea

c. General anesthesia
   1) Induction. The goal = to avoid extreme changes in blood pressure and heart rate. One suggested regimen
      a) Preoxygenate
      b) Defasciculating or priming dose of non-depolarizing muscle relaxant
      c) Titrate anesthetics slowly. One possible regimen
         - Fentanyl – 3 to 5 mcg/kg
         - Lidocaine 1.5 mg/kg to blunt hyperdynamic response to laryngoscopy and intubation
         - Propofol or thiopental
      d) Institute controlled ventilation with 100% O_2.
      e) Muscle relaxant for intubation
         - Succinylcholine (unless patient has hemiparesis)
         - Vecuronium or rocuronium (altered choices)
      f) Smooth intubation
   2) Maintenance. "Balanced anesthesia" = a reasonable choice
      - Volatile inhalational agent (isoflurane, sevoflurane or desflurane)
      - Nitrous oxide
      - Narcotic (fentanyl)
      - Nondepolarizing muscle relaxant (vecuronium or rocuronium)
   3) Which anesthetic agent or combination of agents is "best"?
      a) Few good outcome studies exist.
      b) Isoflurane, when compared to halothane or enflurane in retrospective studies from 1972 to 1987, had
         - Lower incidence of cerebral ischemia on EEG
         - Lower frequency of fatal myocardial infarction
      c) "Light" general anesthesia is better for the heart than "deeper" general anesthesia plus phenylephrine infusion.
         i) "Light" general anesthesia = isoflurane/N_2O vs halothame/N_2O (1 MAC)
            - No difference in myocardial wall stress
            - No difference in myocardial ischemia
         ii) "Deeper" general anesthesia (1.4-1.5 MAC) plus phenylephrine
            - Increased wall stress
            - 3x increase in myocardial ischemia
      d) Comparison of isoflurane/O_2 vs isoflurane/N_2O/O_2
         - No greater incidence of myocardial ischemia (on EKG or transesophageal ECHO)

- No greater incidence of postoperative myocardial infarction (CK isoenzymes)

5. Two potential intraoperative complications
   a. Bradycardia – during neck dissection
      1) Most likely cause = compression of carotid baroreceptor
      2) Mechanism? Reflex involving
         - CN IX (glossopharyngeal) = afferent
         - CN X (Vagus) = efferent
      3) Lidocaine injection of carotid baroreceptor by surgeon
   b. Evidence of decreased cerebral perfusion – during carotid clamping
      1) Is monitoring for cerebral perfusion even necessary?
         - These monitors have not been shown to improve outcome.
         - Logic dictates that monitoring techniques assuring adequate cerebral function at the lowest myocardial work have a place in CEA surgery.
      2) Types of cerebral perfusion monitors[15]
         a) Raw 16-20 lead encephalogram (EEG)
            i) Considered to be the "gold standard" monitor
            ii) Disadvantages
               - Need specially trained personnel
               - Electrically "noisy" OR environment
               - Bulky equipment
            iii) Significant change =
               - > 50% reduction in EEG amplitude
               - Flattening of EEG
         b) Processed EEG
            i) Types of processed EEGs
               - Cerebral function monitor
               - A periodic analysis (Lifescan monitor)
               - Power spectrum analysis (compressed or density spectral array)
            ii) The processed EEG only monitors 2-4 channels.
               - EEG electrodes placed on "watershed" areas
               - Still may miss ischemia during clamping
         c) Somatosensory evoked potentials
            i) "Subtracts out" background EEG activity, leaving only the evoked potential from
               - Median nerve (wrist)
               - Posterior tibial nerve (ankle)
            ii) Significant change =
               - > 50% reduction in amplitude
               - Flattening of SSEP
            iii) Similar sensitivity and specificity to EEG

iv) Both SSEPs and EEG are associated with a considerable false positive rate.

d) Stump pressures
   i) Surgeon measures pressure in stump of carotid artery above cross-clamp
   ii) Significant reduction = stump pressure < 50 mm Hg
   iii) Problem = poor correlation with EEG findings
      - False positives = stump pressure < 50 mm Hg with normal EEG
      - False negatives = stump pressure > 50 mm Hg with "ischemic" EEG
      - May not adequately assess cerebral perfusion after stroke or RIND

e) Regional cerebral blood flow
   i) ^{133}Xenon washout measurement of CBF
   ii) Significant reduction = rCBF < 20 cc/100 g/min
   iii) Problems
      - Only available in certain centers
      - Indicates global well-being rather than focal cerebral ischemia

f) Jugular venous oxygen saturation: indicates global well-being rather than focal ischemia.

g) Neurologic assessment under regional anesthesia

3) Responses to evidence of decreased cerebral perfusion
   a) Surgeon can unclamp the carotid artery and insert a shunt.
      i) Shunting during carotid endarterectomy – three schools of practice
         - Shunt routinely.
         - Shunt never, or very rarely.
         - Shunt selectively, based on monitoring to detect cerebral ischemia.
      ii) Advantage – preserves carotid flow
      iii) Potential risks[15]
         - Thromboembolism
         - Intimal dissection
         - Thrombus formation
         - Air embolism
         - Obstruction of surgical field
   b) Increase the mean arterial pressure.
      - Increases cerebral perfusion pressure
      - Increases cardiac afterload and may precipitate myocardial ischemia
   c) Pharmacologic cerebral protection with thiopental

- Used more commonly for clipping of intracerebral aneurysms than for carotid endarterectomy
- Thiopental was cerebroprotective in one prospective randomized study in humans.

6. **Emergence** – main concerns
   - Smooth and rapid awakening from general anesthesia, in order to obtain a neurologic assessment
   - Control of blood pressure

VI. Postoperative Consideration

A. Potential postoperative complications occur in 4 locations. [15, 51]

1. "In the arm"
   a. Hypertension = a very common complication
      - Incidence was 19% in one study.
      - More common if patient was hypertensive preoperatively

   b. Hypotension – in approximately 5% of patients
2. In the neck**.** Problems secondary to surgery on the neck
   - Wound hematoma – occurred in 5.5% of NASCET patients
   - Paralysis of cranial nerves (VII, IX, X or XII)
   - Carotid body dysfunction

3. In the head. Cerebrovascular complications include
   a. Carotid artery thrombosis
   b. Emboli
   c. Stroke
   d. Hyperperfusion syndrome
      - Manifests as severe unilateral headache, which is postural
      - Related to preoperative hypoperfusion and loss of autoregulation

   e. Seizures – relatively uncommon
4. In the chest**.** Myocardial infarction occurs 2x more frequently than stroke.

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