1. Carotid body: Hypoxic drive

The carotid bodies are chemosensitive cells at the bifurcation of the common carotid that respond to changes in oxygen tension and, to a lesser extent, pH. in contrast to central chemoreceptors, which primarily respond to $PaCO_2$ and the aortic bodies [output via cn X], which have primarily circulatory effects (bradycardia, hypertension, adrenal stimulation, and also bronchoconstriction), carotid bodies are most sensitive to PaO_2. At a PaO_2 of approximately 55-60 mmHg, they send their impulses via CN IX to the medulla, increasing ventilatory drive (increased respiratory rate, tidal volume, and minute ventilation). Thus, patients who rely on hypoxic respiratory drive will typically have resting PaO_2 around 60 mm Hg.

These cells are thought to be primarily dopaminergic, therefore, antidopaminergic drugs may decrease peripheral response to hypoxemia.

Attenuation of the hypoxic response during anesthesia with volatile agents as well as both opioids and benzodiazepines and may be attributed to an effect on the carotid bodies. Carotid body denervation may occur after carotid endarterectomy as a result of surgical disruption. Unilateral loss of function may result in an impaired ventilatory response to mild hypoxemia. Bilateral carotid endarterectomy is associated with loss of the normal ventilatory response to acute hypoxia and an increased resting partial pressure of arterial carbon dioxide. In this situation, the central chemoreceptors are the primary sensors for maintaining ventilation, and serious respiratory depression may result from opioid administration.

Carotid Bodies

- Location: bifurcation of internal and external carotid
- Threshold for Increased Ventilation: paO_2 of 55-60 mmHg
- Inhibition: volatile agents; benzodiazepines, opioids

Note that a bilateral carotid endarterectomy would result in significant impairment of the hypoxic drive.

Carotid endarterectomy: Complications

Carotid endarterectomy is an procedure performed to remove plaque from the endothelium of the common carotid artery in order to improve flow through the internal carotid and thus perfusion of intracranial structures including the brain.

Several randomized controlled trials have proven benefit of this procedure in symptomatic patients with 70-99% stenosis. Different sources quote risk of stroke or death associated with CEA as anywhere from 1 to 7%.

Anesthetic complications related to hypoperfusion during or after the procedure (i.e. stroke) may be mitigated by maintaining adequate CPP. Invasive blood pressure monitoring is indicated (arterial-line), though other monitoring devices (EEG, stump pressures) have not been shown to improve outcomes. During clamping, perfusion is solely from the Circle of Willis and most sources suggest that you must maintain MAP within 20% of baseline. Also, hypocarbia should be avoided as it that may lead to increased cerebrovascular resistance, vasoconstriction and cerebral ischemia.

The most important intraoperative complications include **embolic stroke** secondary to dislodgement of a carotid plaque or piece thereof, and **myocardial ischemia or infarction**.

Postoperative complications include stroke and MI, but also may include neck hematoma and airway compromise. Patients should be monitored for extreme changes in blood pressure during extubation and awakening, and should avoid coughing or straining to the extent possible.

According to the NASCET trial, approximately 1 in 10 CEA patients experienced some medical complication not directly related to the surgery, and "[e]ndarterectomy was ≈1.5 times more likely to trigger medical complications in patients with a history of myocardial infarction, angina, or hypertension ($p < 0.05$)."

Proper preoperative investigation, assessment, and optimization may mitigate some of these risks.

Bilateral carotid endarterectomy: Physiology

Definition

According to Miller, "bilateral recurrent laryngeal nerve injury and resultant bilateral vocal cord paralysis can result in **life-threatening upper airway obstruction.** This situation must be anticipated in patients who have previously undergone contralateral carotid endarterectomy or neck surgery… **Bilateral carotid endarterectomy is associated with loss of the normal ventilatory and arterial pressure responses to acute hypoxia and an increased resting partial pressure of arterial carbon dioxide**. In this situation, the central chemoreceptors are the primary sensors for maintaining ventilation, and **serious respiratory depression may result from opioid administration"**

Bilateral Carotid Endarterectomy: Risks

Bilateral recurrent nerve injury **(complete upper airway obstruction)**

Loss of normal response to hypoxia and hypercarbia **(exquisite opioid sensitivity)**

Loss of normal blood pressure responsiveness

2. Complex regional pain syndrome (CRPS)

Complex regional pain syndrome (CRPS), is a **regional, posttraumatic, neuropathic pain problem** that most often affects one or more limbs. Like most medical conditions, early diagnosis and treatment increase the likelihood of a successful outcome. Accordingly, patients with clinical signs and symptoms of CRPS after an injury should be referred immediately to a physician with expertise in evaluating and treating this condition. **Physical therapy is the cornerstone and first-line treatment for CRPS.** Mild cases respond to physical therapy and physical modalities. **Mild to moderate cases may require adjuvant analgesics, such as anticonvulsants and/or antidepressants** . An opioid should be added to the treatment regimen if these medications do not provide sufficient analgesia to allow the patient to participate in physical therapy. **Patients with moderate to severe pain and/or sympathetic dysfunction require regional anesthetic blockade to participate in physical therapy.** A small percentage of patients develop refractory, chronic pain and require long-term multidisciplinary treatment, including physical therapy, psychological support, and pain-relieving measures. Pain-relieving measures include medications, sympathetic/somatic blockade, spinal cord stimulation, and spinal analgesia.

Diagnosis

CRPS-1 is a syndrome where chronic pain (normally in an extremity) appears to be **associated with sympathetic nervous system dysfunction after trauma** . No single theory has explained sympathetic nervous system dysfunction in CRPS-1 and the pathophysiology is poorly understood. In general, the patient must meet the following criteria for diagnosis:

1. There must be some initial noxious event (contusion, crush, or laceration, surgery, sprain, fracture, or dislocation). This initial event normally involves any kind of discrete nerve injury and may indeed be occult.
2. There must be involvement distal to the site of injury.
3. Pain, allodynia, and/or hyperalgesia must be present.
4. Pain is always disproportionate to injury.
5. Pain is never limited to a single nerve.
6. The patient must have symptoms associated with sympathetic nervous system dysfunction (i.e. edema, changes in skin blood flow, sudomotor activity, etc…)

Patients complaining of pain in an area of decreased sensation, pain without cutaneous hyperalgesia or allodynia, pain only in a specific nerve distribution, or only proximal symptoms do NOT have CRPS-1. Finally, **three phases of CRPS (acute, dystrophic, and atrophic) can often be identified: (see Table)**

Treatment

CRPS Treatment Algorithm (Adapted from Rho et al. Mayo Clin Proc 77: 174, 2002)

- Step 1: physical therapy
- Step 2: TCA, gabapentin, mild opioid if needed for physical therapy
- Step 3: diagnostic sympathetic block
- Step 4: somatic block of sympathetic block fails
- Step 5: spinal cord stimulator / intrathecal medications

CRPS is essentially a result of autonomic nervous system dysfunction. It can be categorized as CRPS Type 1 (formally Reflex sympathetic dystrophy) or CRPS Type 2 (formally Causalgia). It may or may not involve the sympathetic nervous system, hence the phrase sympathetically independent pain. Only one thing is certain about CRPS and that is the unpredictability of the condition. The cause is obscure and elusive, although we may identify sympathetic nervous system involvement in a subset of this population, hence the phrase sympathetically maintained pain.

Multimodal treatment is often necessary (and what I typically employ in my practice): Physical Therapy, anticonvulsants, antidepressants, occasional opioids, sympathetic nerve blocks, epidural infusions, bier blocks, intravenous infusions, radiofrequency ablation of sympathetic nervous system, and spinal cord stimulation. Also, CBT (Cognitive Behavioral Therapy) may be necessary.

The category of phases may be considered as an older way to view CRPS, however, currently CRPS may or may not have some or many of those features listed above.

Three stages may occur during the course of CRPS:

Stage 1 Either following an event or without apparent cause, the patient develops pain in a limb. The essential features include burning and sometimes throbbing pain, diffuse uncomfortable aching, sensitivity to touch or cold, and localized edema. The distribution of the pain is not compatible with a single peripheral nerve, trunk, or root lesion. Vasomotor disturbances occur with variable intensity, producing altered color and temperature. The radiograph is usually normal but may show patchy demineralization.

Stage 2 The second stage is marked by progression of the soft tissue edema, thickening of the skin and articular soft tissues, muscle wasting, and the development of brawny skin. This may last for three to six months.

Stage 3 The third stage is most severe. It is characterized by limitation of movement, the shoulder-hand syndrome (capsular retraction producing a frozen shoulder), contractures of the digits, waxy trophic skin changes, and brittle ridged nails. Bone radiography reveals severe demineralization.

Autonomic features including cyanosis, mottling, increased sweating, abnormal growth of hair, diffuse swelling in nonarticular tissue, and coldness may occur in the

later stages. Urologic manifestations include detrusor hyperreflexia or areflexia, producing urgency, frequency, incontinence, or urinary retention.