

Perioperative Implications**Preoperative Preparation**

- Continue antifungal therapy.
- Evaluate for septic shock.
- Rule out infected lines or catheters; change if indicated.

Monitoring

- If septic, A-line, CVP ± PA catheter, along with standard monitoring.

Airway

- Be careful not to aggravate oral lesion at intubation.

Preinduction/Induction

- Choose drugs based on septic signs and symptoms.
- Worry about hypotension and hypoxemia at induction.

Maintenance

- Choose drugs based on hemodynamic status.
- Choose ventilatory modes based on presence of ARDS.

Extubation

- May have to be delayed if ARDS or septic state requires hemodynamic support.

Adjuvants

- In the presence of compromised renal or hepatic function, modify anesthetic drugs accordingly.

Anticipated Problems/Concerns

- Candidemia presents with a diverse clinical picture, from low-grade fever to fulminant septic shock. There is higher periop mortality in this group of pts.

Carbon Monoxide Poisoning

Peter H. Breen

Risk

- CO is the predominant toxic gas in smoke. (COHb can reach 10% in tobacco smokers.)
- CO poisoning is a major cause of death (early symptoms may be only headache and dizziness).
- CO is produced by all internal combustion engines, incomplete oxidative combustion (e.g., house fires, charcoal and gas grills, malfunctioning butane/propane stoves), and endogenous sources (e.g., by the liver from exogenous exposure to paint stripper).
- No odor, taste, or color and causes no irritation.
- Toxicity potentiated by low inspired O₂ concentration (e.g., smoke inhalation).
- To minimize CO in circle circuit carbon dioxide absorbers, use fresh soda lime, use sevoflurane, and minimize drying (lower FGF and stop FGF during use).
- During GA, use semiclosed circuits, especially when machine has not been used for 2–3 d (e.g., Monday morning).

Perioperative Risks

- Main target organs: Heart and brain
- Heart: Effect can resemble ischemia; potentiated by CAD.

- Brain: Acute loss of consciousness; after initial improvement (lucid window), up to 30% risk of secondary syndrome: chronic psychiatric dysfunction and cerebral and cerebellar syndromes.

Worry About

- Seek other smoke inhalation injury.
- Consider concomitant cyanide poisoning, which potentiates CO toxicity.
- Be alert for CO poisoning in donor for organ transplantation.

Overview

- CO, a colorless, nonirritating, odorless gas, is a natural byproduct of combustion.
- CO binds avidly to Hgb (>200 times more than O₂) to form COHb, which carries no O₂ and causes a left shift in the oxyhemoglobin dissociation curve (decreases O₂ off-loading to tissues).
- CO binds to intracellular hemoproteins such as myoglobin and cytochrome *aa*₃ (esp cardiac) to inhibit O₂ uptake and metabolism.
- "Classic" cherry-red complexion rarely observed (need COHb >40%; may be obscured by coexistent hypoxia and cyanosis).
- COHb level correlates poorly with clinical condition (symptoms with "normal" COHb).

- Treatment should be guided by symptoms and signs, not by blood COHb concentration.

Etiology

- CO produced by incomplete oxidative combustion (e.g., house fires, malfunctioning butane/propane stoves, home heaters, all internal combustion engines)
- Suicide attempts

Usual Treatment

- Normobaric O₂: T_{1/2} of COHb decreases from 3.5 hr (air breathing) to 0.75 hr (O₂ breathing).
- Treat clinical symptoms, not just increased COHb.
- General supportive care, especially for other aspects of smoke inhalation injury.
- Hyperbaric O₂ (2.5 atm) decreases COHb T_{1/2} to 20 min, increases dissolved plasma O₂, and has been shown to decrease the likelihood that delayed neurologic complications will develop. For pts with neurologic Sx (including impaired consciousness), evidence of myocardial ischemia, fetal distress (if pregnant), poisoning in pediatric pts, or other Sx of significant exposure (e.g., COHb >25%), hyperbaric O₂ within 6–8 h of exposure if feasible is recommended.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Thermal/toxic upper airway injury	Fire exposure/smoke inhalation	Perioral burns Airway edema	Laryngoscopy/bronchoscopy
RESP	CO diffuses rapidly into blood, leading to COHb Thermal/toxic airway and parenchymal injury	Dyspnea, tachypnea	Bronchoconstriction and pulm edema	Cooximetric COHb: PO ₂ usually normal CXR Bronchoscopy
CV	Lower O ₂ content in blood and lower O ₂ unloading in tissue	Possibly angina or evidence of heart failure, tachycardia	Cardiac failure	ECG: Ischemic ST-T changes CXR
METAB	Tissue hypoxia leading to acidosis			Lactic acidosis
CNS	Coma, cerebral edema Neuropsychiatric syndrome	Temporal headache, N/V, restlessness Cerebral, cerebellar	Muscle weakness, altered mental status	Abnormal neuropsychometric testing Can occur after initial recovery

Key Reference: Breen PH, Isserles SA, Westley J, et al.: Combined carbon monoxide and cyanide poisoning: a place for treatment?. *Anesth Analg* 80(4):671–677, 1995.

Perioperative Implications**Preoperative Preparation**

- Continuous 100% O₂.
- Document CNS status.
- Consider hyperbaric O₂ if mental status altered or pt has myocardial ischemia or is pregnant.

Monitoring

- Routine monitors (if no lung injury and thus no decreased PaO₂, there may be no tachypnea)

- SpO₂ does not distinguish between O₂Hb and COHb. Thus SpO₂ overestimates O₂Hb during CO poisoning.
- Newer SpO₂ monitors (Masimo Corp., Irvine, CA) can discriminate between O₂Hb and COHb (and metHb).
- Mixed venous oximeter catheters overestimate O₂Hb in presence of COHb.
- Arterial cannulation for frequent blood sampling.
- Venous and arterial COHb levels are almost identical.

Airway

- Airway injury and edema often occur during smoke inhalation, which may require emergent airway management.

Induction

- Avoid cardiac depressant agents.

Maintenance

- 100% O₂ (no N₂O)
- Assess muscle weakness to guide dosage of muscle relaxant.

Extubation

- Ensure CNS status permits natural airway maintenance and protection.

Adjuvants

- Consider treatment for concomitant cyanide poisoning.

Postoperative Period

- Maintain 100% O₂.
- Consider hyperbaric O₂.

Anticipated Problems/Concerns

- Heart and brain affected most.
- Follow CNS function carefully.
- Seek concomitant smoke inhalation injury and cyanide toxicity.
- CO toxic in trace quantities (breathing 0.1% inspired CO for 1 h results in significant toxicity, with

COHb ~30%); CO not detectable with conventional gas analysis instruments (e.g., capnographs, mass spectrometers).

- Standard pulse oximeters do not specifically measure COHb, and SpO₂ measurements are only minimally affected, even by severe CO poisoning.

Carcinoid Syndrome

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Risk

- Carcinoid is the most common GI endocrine tumor.
- 15 cases in 1 million population per y.
- Seen in fewer than 20% of pts with carcinoid.

Diagnosis

- Urinalysis for 5-HIAA and serotonin levels
- Platelet serotonin levels
- Serum chromogranin A
- CT scan and MRI
- Octreoscan and MIBG

Perioperative Risks

- Associated with pt's ability to tolerate abrupt hemodynamic change and/or bronchospasm

Worry About

- Abrupt Htn or hypotension with stress
- Right-sided valvular heart disease

- Electrolyte disturbances (due to intestinal secretion of sodium, potassium, and water)
- Bronchospasm

Overview

- Endocrinologically active tumor from GI mucosa
- May release histamine-like substances, leading to hypotension and bronchospasm, or may release serotonin, leading to hypertensive reactions (and hypovolemia)
- Commonly found in ileum or rectum; less so in pancreas and lung
- Systemically active when metastatic to liver, or when released substances avoid metabolism by liver (carcinoid syndrome)
- Left-sided cardiac disease in 10% of pts if there is a pulmonary carcinoid

Etiology

- Acquired disease.

- May be associated with other ectopic humoral tumors, such as MEN 1 syndrome.

Usual Treatment

- Surgery or arterial embolization to reduce tumor burden.
- Histaminic effects blocked only partially by H₁ and H₂ blockers, mainly H₂.
- Somatostatin analogues octreotide and lanreotide block humoral release.
- Interferon α (alpha) and cytotoxic agents may control symptoms.
- Surgical treatment can play a role in metastasis to the liver.
- No specific medical Rx for established valvular heart lesions.
- Catecholamines may increase humoral release and worsen symptoms.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Cutaneous flushing, lacrimation Pellagra-like skin lesions	Episodic flushing induced by stress, eating, alcohol consumption	Hyperkeratosis, hyperpigmentation	
CV	Histamine-induced hypotension Serotonin-induced Htn Endomyocardial fibrosis, especially in right heart	Sx of right-sided CHF	Murmurs of pulmonic stenosis, tricuspid regurgitation, ascites, edema	ECHO Cardiac cath
RESP	Bronchospasm Endobronchial tumor with obstruction	Episodic asthma poorly responsive to medication Focal wheeze at site of obstructing tumor	Wheezing associated with episodes of flushing	
GI	Diarrhea Obstructing tumor	Episodic watery diarrhea		Bowel films, hepatic CT, ultrasound, angiograms
ENDO	Serotonin secretion			Urinary 5-HIAA levels elevated in most pts Occasionally need to measure plasma histamine
RENAL	Dehydration from chronic vasospasm or diarrhea			BUN/Cr, lytes
CNS	Hemodynamic instability, vasodilation	Hypertensive headache Syncope with flushing		
MS	Cutaneous flushing, lacrimation Pellagra-like skin lesions	Episodic flushing, induced by stress, eating, alcohol consumption	Hyperkeratosis, hyperpigmentation	

Key References: Mancuso K, Kaye AD, Boudreaux JP, et al.: Carcinoid syndrome and perioperative anesthetic considerations, *J Clin Anesth* 23(4):329–341, 2011; Poell B, Al Mukhtar A, Mills GH: Carcinoid: the disease and its implications for anesthesia, *Contin Educ Anaesth Crit Pain* 11(1):9–13, 2011.

Perioperative Implications

Preoperative Preparation

- Assess adequacy of electrolyte and fluid balance.
- Assess right-sided valvular status.
- Somatostatin analogue (octreotide) available; its use has dramatically decreased hazards of anesthesia for pts with carcinoid syndrome.

Monitoring

- Expect rapid fluctuation of BP.
- Central venous pressures may not correlate well with fluid volumes.

Airway

- Risk of stress-induced wheezing (Rx: somatostatin analogue)

Induction

- Chronic vasoconstriction and diarrhea may cause hemodynamic instability.

Maintenance

- Volume assessments complicated by changing vascular tone
- Cardiac function limited by right-sided valvular lesions

Extubation

- Possible stress-induced hemodynamic instability (Rx: Somatostatin analogue)

Adjuvants

- Caution: Catecholamines may increase humoral release and worsen symptoms.
- Somatostatin analogue for hypotension or hypertension or bronchospasm has dramatically decreased anesthesia risk for pts with carcinoid syndrome.

Postoperative Period

- Humoral effects of hemodynamically active metastatic carcinoid usually not eliminated by surgery