Perioperative Implications

Preoperative Preparation

- Optimize bronchodilation.
- Eradicate any underlying bacterial infection.
- Encourage smoking cessation if this can occur >6 wk before surgery.
- Consider regional anesthesia where appropriate; associated with lower incidences of pneumonia, prolonged ventilator dependence, and unplanned postop intubation.

Monitoring

- Be cognizant of potential for increased gradient between $PETCO_2$ and $PaCO_2$.

Airway

- None, unless tumor present in airway

Preinduction/Induction

- If pt has airway reactivity, consider issues related to asthma/chronic bronchitis.
- Avoid N_2O when expansion of bullae is a risk.
- May avoid high concentrations of desflurane if airway reactivity is of concern.

Maintenance

- Recumbent positions impair chest wall muscle function, and abdominal muscle function usually needed for spontaneous ventilation.
- Ventilator settings: Avoid dynamic hyperinflation and development of intrinsic PEEP. Long expiratory times may be required; try to avoid high positive pressures (consider pressure controlled ventilation), especially if bullae are present.

Extubation

- Residual anesthetics may blunt the ventilatory response to CO_2, increasing the risk of postop respiratory failure.
- Pre-extubation bronchodilators.
- Unrelieved incisional pain, especially after abdominal or thoracic surgery, will impair breathing; consider postop epidural analgesia.
- Consider regional block and/or NSAIDs to lessen risk of respiratory depression.
- Pts may be semiconscious and combative owing to hypoxia and hypercarbia on emergence.
- Evaluate whether postop ventilation may be the safest approach until the residual anesthetic effects have dissipated. Extubation to NIPPV may be useful in such cases.

Adjuvants

- β-adrenergic agonists and anticholinergic agents for airway reactivity (may consider theophylline)
- Oral or inhaled steroids in selected pts

Anticipated Problems/Concerns

- Postop respiratory failure; consider NIPPV rather than reintubation in selected pts.
- Tension pneumothorax from ventilator-induced barotrauma.
- Airway plugging from secretions.

Encephalitis

Mary J. Njoku | David L. Schreibman

Risk

- Age; animal contact and occupational exposure to animals; ingestion of raw, partially cooked meat, fish, reptiles, or unpasteurized milk; insect contact; laboratory workers; healthcare workers; person-person transmission; recent vaccination and unvaccinated status; season (late summer/early fall, winter); travel and geographic exposure; immunocompromised state; transfusion and transplantation

Perioperative Risks

- Mental status alteration: Delirium, altered level of consciousness, clinical and subclinical seizures, increased ICP, and SIADH
- Unpredictable sedative and amnestic effects of anesthetics and adjunct drugs

Worry About

- Delayed awakening, postop delirium, clinical and subclinical seizures
- Hyperkalemic response to succinylcholine
- Transient myocardial dysfunction
- Paroxysmal sympathetic hyperactivity: Hyperthermia, tachycardia, hypotension, bradycardia
- Electrolyte abnormality secondary to SIADH and CPM with rapid correction of Na^+ abnormality

Overview

- Inflammation of brain parenchyma associated with clinical evidence of neurologic dysfunction.
- Manifestation of disease process or a component of another CNS or systemic illness.
- Organisms enter CNS via bloodstream or peripheral nerves.
- Symptoms: Altered mental status, altered consciousness, with or without focal neurologic abnormality, behavioral and personality changes in the presence of fever, irritability, changes in speech, changes in hearing, headache, photophobia, nuchal rigidity, vomiting, disorientation, lethargy, confusion, hallucinations, memory loss, clinical or subclinical seizures, myoclonus, coma.
- Dx is established by symptoms, epidemiologic Hx (exposure, season, geographic location), culture of blood/sputum/nasopharynx/stool, biopsy skin lesion/lymph node, serologic testing, CSF cells/protein/culture, CSF bacterial and viral antigens, CSF viral PCR, virus specific DNA sequencing, MRI, EEG, and CT scan (if MRI unavailable). Brain biopsy is rarely performed but should be early.

Etiology

- Unknown in most pts; manifestation of illness outside CNS
- Infectious:
  - Viral (most common): Herpes simplex, varicella zoster, CMV, EBV, influenza, RSV, enteroviruses, arboviruses, HIV, JC virus, rabies
  - Nonviral: Bacteria, prion, parasitic, fungal
- Noninfectious:
  - Postinfectious/immune mediated: ADEM immunologic response to antecedent antigenic stimulus
  - Paraneoplastic: Anti-NMDA receptor, which induces glutamatergic transmission impairment

Usual Treatment

- Empiric antibiotics: Acyclovir (important as viral etiology is most common infectious cause), ampicillin, ceftriaxone, vancomycin
- Doxycycline if rickettsial or ehrlichial disease suspected
- ADEM: Steroids, plasma exchange, chemotherapeutic agents
- Human rabies immunoglobulin infiltration of inoculation site immediately after bite
- Specific antimicrobial therapy according to culture and sensitivity
- Supportive care:
  - Intubate, ventilate, if dictated by mental status, airway reflexes
  - Hemodynamic support
  - Nutrition
  - DVT prophylaxis
  - GI prophylaxis
  - Physical therapy
  - Dx and treatment of extracranial infection
- Management of complications: Seizure, increasing ICP, SIADH, resp failure

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Virus access to CNS from nasal mucosa to olfactory bulb and olfactory tracts	Preceding URI		Nasopharyngeal swab Throat culture
CV	Autonomic dysfunction Neurogenic stunned Myocardium	Transient myocardial dysfunction	Labile BP, HR	ECG, troponin, CK, ECHO, left ventricular angio
HEME	Increased or normal WBC			CBC, WBC differential, serum antibody titers
RENAL	SIADH	Water intoxication Anorexia N/V Personality disorders Neurologic abnormality	No evidence of volume depletion Normal skin turgor Normal BP Mental status changes from lethargy to coma	Serum Na^+ and osmolality Urine Na^+ and osmolality BUN, Cr
CNS	Focal, global neurologic disturbances	Fever Headache Seizure Personality change Memory loss Confusion Weakness Sleep/awake abnormality Hearing, speech, visual changes	Focal neurologic deficits, altered mentation, papilledema, anisocoria; if spinal cord involvement: flaccid paraplegia, increased DTRs	CSF: Cell count (increased WBC, lymphocyte predominance), protein (increased), Gram stain, viral and bacterial culture, antibodies, antigens, viral PCR, viral DNA sequencing, MRI (temporal lobe involvement, hemorrhagic lesions, ±mass effect), EEG, CT

Key References: Tunkel AR, Glaser CA, Bloch KC, et al.: The management of encephalitis: clinical practice guidelines by the Infectious Diseases Society of America, *Clin Infect Dis* 47(3):303–327, 2008; Pryzbylkowski PG, Dunkman WJ, Liu R, et al.: Case report: anti-*N*-methyl-D-aspartate receptor encephalitis and its anesthetic implications, *Anesth Analg* 113(5):1188–1191, 2011.

Preoperative Implications

Preoperative Preparation

- Document neurologic exam.
- Elicit Hx of increased ICP or seizure.
- If suspicion of anti-NMDA receptor encephalitis:
  - Concern for anesthetic interaction with a dysregulated NMDA receptor (i.e., propofol, midazolam, methadone, N_2O).
- If SIADH present, correct electrolyte and free water abnormality.
  - Sodium administration or fluid restriction depending on severity of hyponatremia.
  - Beware of central pontine myelinolysis with rapid correction of hyponatremia.

Monitoring

- Standard ASA monitors
- Invasive monitors when indicated; arterial line if ICP is an issue
- If EVD in use, continue monitoring ICP in the OR
- Lytes

Airway

- None

Induction

- Potential for hyperkalemic response to depolarizing NMBs if myopathy, paralysis, or prolonged immobilization; prefer use of nondepolarizing NMBs
- Autonomic instability and labile hemodynamics

Maintenance

- If pt is receiving seizure prophylaxis (e.g., phenytoin [Dilantin], carbamazepine [Tegretol], phenobarbital [Luminal], primodone [Mysoline], valproic acid [Depakote]) be aware of potentiation of sedative effects and alteration of hepatic metabolism of anesthetics and muscle relaxants.

Extubation

- Delayed awakening
- Seizures on emergence

Postoperative Period

- Delirium
- Other neurologic deterioration, including clinical or subclinical seizures

Anticipated Problems/Concerns

- Delayed emergence.
- SIADH, careful selection of replacement fluid.
- Hyperkalemic response to succinylcholine.
- Universal precautions for contact with infected materials; sterilization of reusable instruments.
- Use disposable instruments, specifically with JC virus disease.

Encephalopathy, Hypertensive

Shane V. Cherry | Christian Diez

Risk

- Chronic Htn, renal disease (particularly end-stage renal disease), malignancy, sympathomimetic drugs, and a history of transplantation and immunosuppressive therapies

Perioperative Risks

- Increased risk of myocardial ischemia, ventricular dysrhythmias, HF, aortic dissection, cerebral hemorrhage, coma, long-term neurologic disability, renal failure, or sudden death

Worry About

- Myocardial ischemia or infarction
- Aortic dissection
- HF
- Pulm edema
- Cerebral infarction (ischemic or hemorrhagic) or intracranial hemorrhage
- Acute renal failure
- Eclampsia in at-risk parturients
- Microangiopathic hemolytic anemia

Overview

- The most common clinical presentations of hypertensive emergencies are cerebral infarction (24.5%), pulm edema (22.5%), hypertensive encephalopathy (16.3%), and HF (12%).
- Hypertensive encephalopathy is by definition a hypertensive emergency and has recently come to fall under the umbrella term PRES.
- Hypertensive encephalopathy is a relatively rapidly evolving syndrome of severe Htn in association with (most commonly) seizures, headache, visual disturbances, altered mental status, vomiting, ataxia, and focal neurologic deficits that may become rapidly fatal.
- Occurs when the systemic BP is elevated beyond the cerebral autoregulatory threshold of MAP, typically greater than 160 mm Hg ("autoregulation breakthrough").
- Differential Dx: Ischemic or hemorrhagic stroke (particularly posterior circulation stroke), toxicology syndrome from drugs of abuse (e.g., cocaine), encephalitis, and venous sinus thrombosis.
- It is critically important to distinguish between ischemic stroke and hypertensive encephalopathy because the treatment for hypertensive encephalopathy is lowering of BP, whereas outcomes are improved with higher BPs after acute ischemic stroke and therefore antihypertensives are generally not recommended.
- Hypertensive encephalopathy can develop in pts with or without chronic Htn. However, because the cerebral autoregulation curve is shifted to the right in chronically hypertensive pts, it may take significantly higher BPs for these pts to develop signs of encephalopathy.
- As the name implies, PRES is usually reversible if diagnosed early and treated appropriately but can quickly become irreversible and fatal.
- Diagnostic test of choice is MRI, which will reveal symmetric reversible T2 high signal intensities located in the occipital and parietal lobes as a result of subcortical vasogenic edema. CT is not sensitive for the lesions of PRES and will often be normal.