Perioperative Implications

Preoperative Preparation

- Control underlying disease as well as possible (i.e., sobriety for alcoholic cirrhosis, stress-dose steroids for autoimmune hepatitis).
- Risk stratify pt based on MELD score; use platelet count as an indicator of severity of portal hypertension.
- Correction of hyponatremia preop to avoid rapid increases intraop.
- Optimize diuretic regimen for pts with ascites to control hypervolemia.
- Assess and correct coagulopathy, with appropriate additional product available to the OR for intraop administration.

Monitoring

- Standard monitors.
- Urinary catheter for UOP monitoring.
- Frequently will require arterial catheter for continuous BP monitoring as well as stroke volume variation to determine volume status and ventricular loading conditions.
- Although CVP is known to be a poor indicator of volume status, central access can be helpful for transfusion as well as administration of vasoactive and inotropic medications.

Airway

- With ascites or acute variceal hemorrhage, aspiration precautions and RSI indicated.

Induction

- May see hemodynamic instability in pts with recent hemorrhage or in sepsis.
- May need to adjust choice and dosage of anesthetic drugs to account for renal dysfunction and underlying hepatic dysfunction.
- If considering regional anesthesia, attention should be paid to pt's coagulation status.

Maintenance

- Must be cognizant of dosing adjustment for renal and hepatic dysfunction.
- Active warming to avoid hypothermia and potentiation of coagulopathy.
- In abdominal surgery that drains a large volume of ascites, rapid fluid shifts can require the administration of albumin to maintain intravascular volume.
- Pts with HPS may require high FiO_2 and high PEEP to maintain oxygenation; however, this may need to be balanced with PEEP compromising venous blood return.

Extubation

- Requires full reversal of neuromuscular blockade, as duration of action of neuromuscular blockers may be altered.
- Ensure that pt fully awake and protecting airway before extubation without new or worsened encephalopathy.

Postoperative Period

- Pain control with PCA or oral opioids, again acknowledging altered metabolism.
- Regional anesthesia can be helpful as long as not contraindicated by coagulopathy.
- Watch for acute decompensation of either hepatic or renal function caused by decreased hepatic or renal blood flow while under general anesthesia.
- Risk for development of HE, especially if administered benzodiazepines.

Anticipated Problems/Concerns

- Aspiration risk in presence of ascites or acute variceal hemorrhage.
- Hemodynamic instability due to derangement of volume status, sepsis, or myocardial dysfunction.
- Pts susceptible to acute decompensation of renal function.
- Multifactorial risk for increased blood loss intraop.

Postoperative Encephalopathy, Metabolic

Steven Roth | Lee A. Fleisher

Risk

- Pts undergoing any surgical procedure are at risk. It is especially of concern following brain or cardiac surgery or interventional neuroradiology procedures and in pts with COPD, cancer, renal or hepatic failure, and those with lyte abnormalities.
- Post–liver transplant.
- No gender predominance.

Perioperative Risks

- Aspiration, fluid and lyte imbalances, circulatory failure, hypoxia, insulin use

Worry About

- Suspect in any pt who fails to awaken or awakens more slowly than expected following GA.
- Evaluate for the presence currently or earlier in the periop period of severe hypotension, hypoxemia, fluid and lyte disorders, cancer, renal or liver dysfunction, and thyroid abnormalities.
- Seizures, increasing intracranial pressure; persistent coma may result.

Overview

- Altered state of consciousness that becomes apparent in the perioperative period.
- Pts may fail to awaken after GA for these reasons: Anesthesia-associated narcotics, inhalational anesthetics, benzodiazepines, hypnotics (may impair consciousness), brain injury. Direct surgical intervention (e.g., occlusion of major intracranial vessel, intracranial hemorrhage, edema) may result in impaired consciousness, or embolization to a major artery may occur (e.g., during or after cardiac surgery, interventional neuroradiology procedures).
- Metabolic abnormalities: Circulatory failure, hypoxia, insulin use, hepatic and renal insufficiency. Lyte abnormalities can result in failure or slowness to awaken. In all cases, Dx should proceed quickly in order to treat underlying cause before severe brain injury results.
- Could be confused with delirium.

Etiology

- Anoxic-ischemic encephalopathy.
- Hypercapnic encephalopathy ($PaCO_2$ >70 mm Hg).
- Hypoglycemic encephalopathy (glucose ≤30 mg/dL).
- Hyperglycemic coma (glucose ≥450 mg/dL; Osm >319 $mOsm/mm^3$).
- Acute hepatic encephalopathy: Liver failure.
- Uremic encephalopathy: Renal failure.
- Other brain injuries: SIADH, seizures.
- Electrolyte imbalance: Hypokalemia or hyponatremia, hypercalcemia.
- Endocrine: Thyrotoxicosis, hypothyroidism.
- Drug and/or toxin exposure; use a drug and/or toxicology screen.

Usual Treatment

- Depends on the etiology (see Assessment Points)

Assessment Points

Etiology	Examples	Diagnosis	Treatment
ENDO	Hyperthyroid Hypothyroid	Thyrotoxicosis Myxedema	PTU Thyroid hormone replacement
ANOXIC-ISCHEMIC	Cardiac arrest Prolonged shock Hypoxemia	Obvious from clinical course	Reverse acute event Decrease cerebral edema, maintain BP, decrease temperature?, prevent seizures
HYPERCAPNIC	Narcotic-induced Severe COPD, sleep apnea	Increased heart rate and BP Increased end-tidal or arterial Pco_2	Reverse narcotic Mechanical vent to decrease Pco_2
HYPOGLYCEMIC	Insulin overdose Ethanol ingestion Neonatal (idiopathic)	No IVF and PO ingestion From Hx and alcohol level Decreased blood glucose	IV glucose (D50)
HYPERGLYCEMIC	Hyperosmolar nonketotic coma Ketoacidosis	Suspect in known diabetic Ketones in blood, urine Acidosis	Insulin, correct acidosis and fluid volume deficit
ION DISTURBANCES	Decreased Na^+ Decreased K^+	Serum Na^+ <125 mmol/L (e.g., SIADH) Serum K^+ <2.5 mEq/L Severe muscle weakness	Hypertonic saline (caution) NaCl and diuretics K^+ replacement
RENAL	Renal failure		
HEPAT	Hepatic encephalopathy		

Key References: Bozbora A, Coskun H, Erbil Y, et al.: A rare complication of adjustable gastric banding: Wernicke's encephalopathy, *Obes Surg* 10(3):274–275, 2000; Brown EG, Douglas VC: Moving beyond metabolic encephalopathy: an update on delirium prevention, workup, and management, *Semin Neurol* 35(6):646–655, 2015.

Perioperative Implications

- Correct ion and fluid disturbances.
- Normalize blood glucose.
- Optimize organ function (e.g., renal, hepatic).
- Adequate hormone replacement.
- Search for drug/toxin exposure (sedative/hypnotics; ethanol and its street substitutes, such as ethylene).

Posttransplant Lymphoproliferative Disorder

Tamas Seres

Risk

- Cumulative incidence over 5 y: 1–2% in liver, 1–3% in kidneys, 2–6% in heart, 2–9% in lung, and 11–33% in intestinal or multiorgan transplants
- Major risk factors:
  - EBV positive serology in the recipient (multisystem PTLD)
  - EBV negative recipient and EBV-positive donor (PTLD limited to allograft tissue)
  - The degree of T-cell immunosuppression (induction with OKT3, ATGAM, thymoglobulin, and maintenance with tacrolimus)
- Additional risk factors:
  - Time after transplant (highest incidence during the first y)
  - Recipient age (<25 y)
  - Ethnicity (Caucasians)
- Overall survival rates ranging between 25–35%

Perioperative Risks

- Increased risk of airway or bowel obstruction and hemodynamic compromise
- Increased risk of dysfunction of the transplanted organs
- Increased risk for infection and CNS involvement

Worry About

- Enlarged tonsils and cervical adenopathy increasing difficulty of airway
- Thoracic adenopathy complicating intubation, ventilation, and cardiac output
- Pulm involvement causing decreased oxygenation and/or ventilation
- Dysfunction of the transplanted kidneys, liver, or heart
- GI involvement manifesting as N/V or bowel obstruction
- CNS involvement manifesting as mental status change or increased ICP
- Immunosuppression causing an increased rate of infection

Overview

- Lymphoproliferative disorders are among the most serious and potentially fatal complications of chronic immunosuppression in organ transplant recipients.
- These tumors are mostly B-cell-type large-cell lymphomas. Extranodal involvement occurs in 30–70% of these cases as a localized tumor in either the transplanted organ or another site, such as the GI system, lungs, skin, liver, and CNS.

Etiology

- B lymphocytes, infected by EBV, proliferate in the setting of immunosuppression, where T-cell immune surveillance is significantly decreased.

Usual Treatment

- Reduction of immunosuppression, rituximab, cytotoxic T-cell infusions, and radiation (CNS).
- Surgery may be necessary to debulk large masses and relieve bowel obstructions.
- Chemotherapy for disseminated unresponsive disease.