# Portal Hypertension

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## Risk

- Hepatic cirrhosis has a prevalence of about 1–3:1000.
- At the time of diagnosis of cirrhosis, 50% of pts will already demonstrate sequelae of portal hypertension.
- Also, in the setting of schistosomiasis (mainly in developing countries), portal vein thrombosis, Budd-Chiari syndrome, or congestive hepatopathy may be seen in pts with congestive heart failure.

## Perioperative Risks

- Multifactorial increase in risk of coagulopathy:
  - Risk of thrombocytopenia caused by splenic sequestration of platelets due to congestion of the spleen.
  - Splenic sequestration may compound thrombocytopenia caused by decreased synthesis of thrombopoietin in the liver.
  - Underlying hepatic synthetic dysfunction can also lead to decreased synthesis of clotting factors.
- Ascites may increase intra-abdominal pressures, increasing aspiration risk and compromising pulmonary function.
- Risk of total body volume overload in pts with ascites who have been chronically retaining sodium and water.
- Risk of hypotension from both decreased effective circulating volume as well as decreased vascular resistance in chronically vasodilated state.
- Risk of hypoxemia in pts with HPS.
- Risk of development of acute RV dysfunction in pts with portopulmonary hypertension.
- Risk of kidney injury due to reduced renal perfusion, leading to HRS.
- Risk of development of HE in the periop period.

## Worry About

- Aspiration risk from increased intra-abdominal pressure in pts with ascites or from full stomach in pts with active upper GI/variceal bleeding.
- Hypotension from derangements of intravascular volume, peripheral vasodilation, hemorrhage, sepsis, or myocardial dysfunction.
- Sepsis from spontaneous bacterial peritonitis or increased bacterial translocation from intestines (leading to urinary tract infection or pneumonia). Prophylactic antibiotics are recommended for endoscopic procedures for variceal bleeding that would otherwise not have required antibiotics.
- Increased blood loss due to coagulopathy and platelet dysfunction as well as splanchnic congestion in intra-abdominal surgery.
- Potential for worsening of underlying hepatic disease during the periop period.
- Changes in drug pharmacokinetics and pharmacodynamics given increased volume of distribution and impaired liver metabolic function.

## Overview

- Direct cause of many of the complications of hepatic cirrhosis.
- Causes derangement of nearly every organ system.
- Increases periop morbidity and mortality to a variable degree depending on type of surgery. For example, cirrhotic pts undergoing abdominal surgery have an estimated mortality of up to 30%.
- Pts can present for any of a number of elective and emergency surgeries, including cholecystectomy, endoscopy for GI bleeding, hernia repair (abdominal hernias exacerbated by ascites), colorectal surgery for diverticular disease, thoracoscopy for hepatic hydrothorax, liver resection for cancer, and finally (curative) liver transplantation.
- The model for end-stage liver disease (MELD) score has been shown to be useful in predicting surgical risk of morbidity and mortality, with a 14% increase in mortality for every 1-point increase in MELD score above 8. The pt's bilirubin, INR, and serum creatinine are used in calculating the MELD score.

## Etiology

- Although a variety of diseases and disease states can cause portal hypertension, the initial insult is always an increase in pressure within the portal vein caused by an obstruction to flow from the portal vein to the right atrium. This obstruction can be prehepatic (portal vein thrombosis), intrahepatic (hepatic cirrhosis, most common), or posthepatic (Budd-Chiari syndrome or congestive hepatopathy).
- HVPG can be used to diagnose and grade portal hypertension. Portal hypertension is present when HVPG is >5 mmHg. Complications usually begin at HVPG >10 and acute variceal bleeding at HVPG >12.
- In an attempt to alleviate increased pressures, the splanchnic endothelium increases production of vasodilators and vascular endothelial growth factor and decreases sensitivity to vasoconstrictors.
- Vasodilation leads to increased arterial inflow into the splanchnic system, increasing portal vein flow, and further increasing portal pressures. Increased portal flows and pressures leads to increased interstitial tissue hydrostatic pressure and the development of ascites.
- Increased pressure and angiogenesis lead to the formation of portosystemic collateral venous beds, which manifest as esophageal varices, portal-hypertensive gastropathy, and hemorrhoids.
- Collateral flow, bypassing the liver, increases systemic exposure to splanchnic vasodilators, causing HPS as well as the hyperdynamic syndrome of cirrhosis. Collaterals also lead to hyperammonemia and development of HE.

## Usual Treatment

- Currently, most treatments are aimed at specific sequelae of portal hypertension.
- Treatments specifically targeting portal pressures include nonselective beta blockers, placement of TIPS, and liver transplantation.
- Ascites is primarily treated with salt restriction and diuretics. Large-volume paracentesis can be used for tense ascites and to provide symptomatic relief.
- Peritoneal venous shunts (such as Denver shunt) may be used in pts who have ascites refractory to medical therapy.
- Esophageal varices can be treated endoscopically for both prophylaxis and control of acute variceal hemorrhage.
- Pts with type II HRS will sometimes require hemodialysis while waiting for liver transplant.
- Portopulmonary Htn will sometimes require pulm vasodilators such as sildenafil and epoprostenol.
- HPS may require supplemental oxygen while waiting for liver transplant.
- HE is treated with lactulose and oral antibiotics (rifaximin) but may require hospitalization and supportive care while waiting for clinical improvement.

## Assessment Points

| System | Effect | Assessment by Hx | PE | Test |
|---|---|---|---|---|
| CV | Hyperdynamic circulation<br>Hypotension<br>Intravascular volume depletion | Dyspnea on exertion<br>Orthostasis | Vital signs (tachycardia), orthostatic signs | Monitor BP and HR<br>Low SVR |
| RESP | V/Q mismatch in HPS<br>Hepatic hydrothorax | Dyspnea | Vital signs (low $SpO_2$)<br>Decreased breath sounds, dullness to percussion at lung base (most commonly on right) | Monitor $SpO_2$, $PaO_2$ on ABG<br>Increased A-a gradient |
| GI | Ascites<br>Esophageal varices<br>Acute variceal hemorrhage<br>Portal-hypertensive gastropathy<br>Splenomegaly | Abdominal distention<br>Hematemesis<br>Melena<br>BRBPR<br>Abdominal pain | Abdominal distention, positive fluid wave,<br>Palpable spleen<br>Heme-positive stool<br>Caput medusa | Abdominal US, abdominal CT<br>Upper endoscopy,<br>Measurement of HVPG |
| RENAL | Hypervolemic hyponatremia,<br>HRS type I (acute) and type II (chronic) | Oliguria<br>Peripheral edema<br>Abdominal distention | Edema or exam consistent with ascites | Lytes, especially sodium; serum Cr, especially compared with pt's baseline<br>BUN |
| HEME | Thrombocytopenia<br>Platelet dysfunction | Easy bruising/bleeding | Petechiae<br>Bleeding from venipuncture sites | CBC<br>Coagulation studies<br>Thromboelastogram |
| CNS | HE<br>Cerebral edema | Severity ranges from confusion to disorientation to coma | GCS<br>Signs of intracranial Htn or herniation<br>Asterixis | Serum ammonia<br>CT of head if concern for cerebral edema or herniation<br>ICP monitoring (must be weighed against risk of hemorrhage in coagulopathic pts) |

**Key References:** Kiamanesh D, Rumley J, Moitra VK: Monitoring and managing hepatic disease in anaesthesia, *Br J Anaesth* 111(Suppl 1):i50–i61, 2013; Malik SM, Ahmad J: Preoperative risk assessment for patients with liver disease, *Med Clin North Am* 93(4):917–929, 2009.

## 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

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## 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)