

Anesthetic Concerns for the Patient with Liver Disease

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Advanced hepatic disease should really be considered a systemic disease process, affecting multiple organ systems. Like kidney failure, it reflects a fundamental defect in protein metabolism, i.e., nitrogen elimination after deamination of amino acids. In renal failure, ammonia is converted to urea, which accumulates as BUN. In liver failure, the arginine cycle fails to convert ammonia to urea, so that ammonia accumulates and blood urea nitrogen (BUN) remains very low. In fact, hyperammonemia is a marker for other circulating byproducts of protein metabolism that cause defective ion transport across cell membranes, resulting in intracellular sodium and water accumulation. Every organ system is affected.

Patients with advanced hepatic disease present a challenge to anesthesiologists because liver failure implies abnormal handling of anesthetic agents, as well as multiorgan system dysfunction, general debility and specific problems associated with replacement therapy and transplantation. Moreover, when hepatic insufficiency is severe, anesthesia and surgery may themselves precipitate acute failure. This outline will address an approach to the patient with severe hepatic disease undergoing non-liver transplant surgery. It will focus on the manifestations of organ dysfunction, pharmacology of anesthetic agents and selected aspects of anesthetic preparation and perioperative management.

THE HEPATIC CIRCULATION

Anatomy

The hepatic circulation is intimately associated with that of the rest of the gastrointestinal tract. The hepatic artery is derived from the celiac trunk, the first major branch of the abdominal aorta, and provides only about a third of the total blood flow of the liver. The celiac trunk provides the arterial supply to the foregut (stomach, spleen, duodenum); the superior mesenteric artery supplies the midgut (jejunum and ileum) and the inferior mesenteric artery the hindgut (colon and rectum). These organs all drain into the portal vein, which bathes the liver and provides two thirds of its circulation.

Hepatic Blood Flow Regulation

Hepatic blood flow is intrinsically regulated by a phenomenon known as "reciprocity of flow." The oxygen delivery (DO_2) from the hepatic artery (high saturation, lower flow) normally balances that of the portal vein (low saturation, higher flow). A decrease

in hepatic artery flow is balanced by an increase in portal vein flow, to maintain DO_2 from each. Reciprocity of flow is impaired by anesthesia and lost in cirrhosis.

Autoregulation – so important at maintaining blood flow over a wide range of perfusion pressure in the brain, heart and kidney – does not exist in the portal circulation, which is perfusion pressure dependent. Vascular adrenergic receptors do play an important role in mediating hepatic and portal blood flow. Alpha receptors are distributed throughout the hepatic and portal systems, so sympathetic activation causes both hepatic artery and portal vein constriction. However, the portal circulation is devoid of β_2 receptors, so the potential benefit of β_2 receptor-induced arterial vasodilation (e.g., with dobutamine) is realized in the hepatic artery only. Dopaminergic receptors are distributed throughout the hepatic artery and portal vein, so dopaminergic agonists promote both hepatic and portal blood flow. This may or may not be beneficial (see below).

Portal constriction is induced by sympathetic stimulation, hypoxemia and hypo- and hypercarbia. In patients with severe liver disease it is prudent to ensure adequate anesthesia, intravascular volume and cardiac output, and to maintain adequate oxygenation and normocarbica.

Pharmacologic Protection

An increasing number of pharmacologic agents have been studied as agents that potentially provide liver protection during ischemia-reperfusion injury. These include vasoactive agents that promote portal flow (β -adrenergic agents, dopaminergic agents, prostaglandins); enhance liver regeneration (pentoxifylline, ciprofloxacin) and anti-oxidants (N-acetyl cysteine, NAC). Animal data have been encouraging, and in some centers NAC is added to the preservative solution or infused after high-risk liver transplantation. However, as yet there are no prospective human trials that have confirmed a benefit.

Promotion of portal vein flow is not always beneficial. In a model of hemorrhage-induced splanchnic ischemia in dogs, infusion of fenoldopam, a selective dopaminergic-1 agonist, attenuated the sympathetic splanchnic vasoconstrictor response and restored portal blood flow to near baseline. However, in a human study in patients with alcoholic cirrhosis and ascites, infusion of fenoldopam resulted in mild hypotension,

increased plasma norepinephrine and renin, and increased portal pressure, presumably due to increased mesenteric blood flow.

SYSTEMIC MANIFESTATIONS OF LIVER DISEASE

Ascites, Fluid, and Electrolyte Imbalance

Hypoalbuminemia and portal hypertension combine to induce ascites and intravascular hypovolemia. This triggers secondary hyperaldosteronism, with sodium and water retention and potassium excretion. The result is hypokalemic metabolic alkalosis, generalized edema (anasarca) and worsening ascites.

Ascites elevates the diaphragms and decreases functional residual capacity (FRC), resulting in basal atelectasis and hypoxemia. Tense ascites may increase intra-abdominal pressure to the extent that venous return and renal blood flow are decreased. Spontaneous bacterial peritonitis occurs in about 10% of patients. It is important to distinguish this from surgical peritonitis and avoid unnecessary (and potentially devastating) exploratory laparotomy.

The administration of loop diuretics to treat edema and ascites may simply exacerbate intravascular hypovolemia and hypokalemia and worsen hepatic perfusion. The specific aldosterone antagonist spironolactone is most effective in maintaining a modest potassium-sparing diuresis. However, it acts through intracellular protein induction so that its onset and offset are slow (2–3 d), and its potassium-sparing effect in acute renal insufficiency can provoke acute hyperkalemia.

Metabolic alkalosis worsens hepatic encephalopathy by nonionic diffusion trapping. With a decrease in extracellular hydrogen ion concentration, ammonium (NH_4^+), which is polarized and lipid insoluble, is converted to ammonia (NH_3) which is nonionic and crosses lipid membranes. Treatment consists of administration of potassium chloride with careful volume repletion. Refractory alkalosis has been corrected by the central venous administration of dilute (0.1N) hydrochloric acid.

Gastrointestinal Dysfunction

All patients have the potential for active viral hepatitis (A, C, D). Hepatic encephalopathy is associated with anorexia, hiccups, nausea and vomiting. As in uremia, gastric emptying is delayed and increases the risk of regurgitation and aspiration during anesthetic induction. This risk is exacerbated by severe ascites with increased abdominal pressure.

Patients with portal hypertension are at constant risk of massive hemorrhage from esophageal and/or gastric varices. However, there is also an increased risk of peptic ulcer disease, which must be considered as a potential source when gastrointestinal bleeding occurs.

Hepatorenal Syndrome

The term hepatorenal syndrome is often used to refer to any degree of renal insufficiency that occurs in the presence of liver failure. It is in fact an advanced, resistant prerenal syndrome, a form of vasomotor nephropathy, characterized by severe prerenal oliguria, low urine sodium (≤ 10 mEq/L) and progressive azotemia.

The syndrome is seen with severe obstructive jaundice (total bilirubin > 8 mg/dL) or hepatic failure. Bile salts bind endotoxin in the gut, and their absence allows access of endotoxin into the portal circulation. Because of portasystemic shunting and hepatic Kupffer cell dysfunction, endotoxin readily enters the systemic circulation and reaches the kidney. There it induces renal vasoconstriction and intense activation of renal tubular salt and water retention.

Acute tubular necrosis (ATN) may complicate liver failure independently of, or concomitant to, the hepatorenal syndrome. Endotoxin also has direct nephrotoxic effects. Tense ascites exacerbates renal dysfunction by increasing renal vein pressure, which impairs glomerular filtration. Variceal bleeding with hemorrhagic shock is one of several insults that may induce ischemic ATN.

As previously stated, in advanced liver failure the BUN remains low (< 10 mg/dL) even in the presence of gastrointestinal bleeding or acute renal failure. There is impairment of the hepatic arginine cycle that converts urea to ammonia. Creatinine production is low in cachectic liver failure patients and serum creatinine often underestimates the severity of decrease of GFR. Accurate estimation of GFR and renal reserve may require measurement of creatinine clearance.

Hyperdynamic Circulation

Severe liver disease is characterized by a hyperdynamic circulation with a fixed low SVR. The vascular resistance is lowered by countless tiny arteriovenous shunts in the skin (spider nevi, palmar erythema), gastrointestinal tract and lung. Patients tend to have chronic low systemic arterial pressure. Circulatory reserve is impaired and decompensation and shock occurs rapidly with hypovolemia, sepsis or myocardial ischemia.

Alcohol-induced cirrhosis may be accompanied by alcoholic cardiomyopathy, with a predilection to cardiac arrhythmias, in which thiamine deficiency may play a contributory role.

Respiratory Failure

The hepatopulmonary syndrome describes the phenomenon of hypoxemia refractory to increased inspired oxygen fraction found in some patients with advanced liver failure. It is caused by intrapulmonary shunting through arteriovenous anastomoses, and may be associated with reactive or fixed pulmonary hypertension.

All patients with severe liver disease are at high risk of perioperative pulmonary complications, especially pneumonia. The combination of ascites, elevated diaphragms, and hypoalbuminemia predisposes to pleural effusions, atelectasis and pulmonary edema. Aspiration risk increases with worsening hepatic encephalopathy.

Hematologic Abnormalities

Liver failure patients become coagulopathic for many reasons. However the most consistent is Factor VII deficiency as a consequence of impaired hepatic synthesis and impaired gastrointestinal vitamin K absorption. Prolongation of the prothrombin time (PT) with increased International Normalized Ratio (INR) is an important marker of hepatic synthetic dysfunction, and an independent predictor of perioperative risk. Thrombocytopenia (platelet count 50–75 k) is commonly found, chronically with hypersplenism in portal hypertension, and acutely with gastrointestinal bleeding or DIC.

Factor V deficiency is a sensitive marker of acute liver dysfunction, and has been used as such after orthotopic liver transplantation.

Dysfibrinogenemia (production of an abnormal fibrinogen) occurs in advanced liver failure, and implies that fibrinogen function is abnormal even though plasma levels may be in the normal range.

Anemia is common, via several mechanisms: acute or chronic blood loss, malnutrition, and bone marrow suppression. Chronic alcoholism may be associated with macrocytic anemia.

Nutritional-Metabolic Problems

Loss of glycogenesis (hepatic glycogen synthesis) removes the ability to regulate blood glucose and patients become “poikiloglycemic” – that is, blood glucose becomes dependent on exogenous administration. Hypoglycemia (blood glucose <100 mg/dL) is almost pathognomonic of acute hepatic failure or end-stage liver disease.

Loss of hepatic albumin synthesis, protein malnutrition and the catabolic effects of hepatic failure lead to depleted lean body mass, hypoalbuminemia, and low colloid oncotic pressure (COP). This exacerbates ascites, anasarca and pulmonary edema. Loss of lean body mass also impairs normal immune and healing mechanisms. As a consequence, patients are at high risk of nosocomial and opportunistic infections, wound dehiscence, fistulas and bedsores.

Neurologic Complications

Hepatic encephalopathy is the most important neurologic complication of liver failure. Although elevated arterial ammonia (normal upper limit: 35 mg/dL) is usually associated with abnormal CNS function, it is generally accepted that it is merely a marker of disordered protein metabolism. Encephalopathy is probably caused by a variety of peptides,

mercaptans and false or depressive neurotransmitters. Examples of the latter include octopamine, a catecholamine formed from phenylalanine as a consequence of a block in the synthetic pathway of the normal neurotransmitter, norepinephrine. An aromatic amino acid, tryptophan, also accumulates and is the precursor of 5-hydroxy-tryptamine (serotonin), a potent neurodepressor transmitter.

Hepatic encephalopathy may be graded as follows:

Grade 1: confabulation, constructional apraxia (loss of graphic ability)

Grade 2: drowsiness, asterexis, confusion

Grade 3: stupor

Grade 4: coma

Fulminant hepatic failure rapidly leads to hepatic coma. Breakdown of the blood-brain barrier results in acute cerebral edema, the most important determinant of outcome.

In patients with chronic liver disease, acute encephalopathy may be precipitated by a number of factors, including hypovolemia (e.g., excessive loop diuresis), gastrointestinal bleeding, surgery or infection. Another important precipitant is hypokalemic metabolic alkalosis. In an alkalotic milieu, ionized hydrophilic ammonium (NH_4^+) converts to non-ionized lipophilic ammonia (NH_3), which crosses the blood-brain barrier (nonionic diffusion trapping).

Alcoholic cirrhosis may be associated with alcohol-induced encephalopathy (thiamine deficiency), Wernicke’s encephalopathy (oculomotor palsy, cerebellar ataxia), and/or Korsakoff’s psychosis (amnesia, confabulation).

Pharmacologic Impact of Liver Disease

Most IV anesthetic agents are lipid-soluble and non-ionized, and undergo hepatic biotransformation to active or inactive water-soluble metabolites, which are then excreted in the bile or the urine. Lipid insoluble, highly ionized drugs (e.g., some neuromuscular blocking agents) are directly excreted by the kidney. Hepatic disease alters anesthetic and parenteral drug clearance by several mechanisms. They include decreased organ blood flow (i.e., decreased drug delivery), increased unbound free fraction of highly protein-bound drugs (hypoalbuminemia or acidosis) and decreased enzymes and transport processes that irretrievably remove the drug from the blood.

The duration of action of many drugs administered by bolus or short-lived infusion is dependent on redistribution, not elimination. Their loading doses may not require to be decreased unless unbound free fraction is increased or there is known to be a greater pharmacodynamic effect. However, maintenance doses can accumulate and should be reduced accordingly.

Liver disease alters drug pharmacodynamics even if pharmacokinetics are not changed. Patients are often debilitated, with depleted lean body mass.

Table 1. Drugs Independent of Hepatic Function for Elimination. These Drugs Undergo Enzymatic or Spontaneous Breakdown in the Blood

Drug	Mode of breakdown
Succinylcholine	Pseudocholinesterase
Esmolol	Red cell esterase
Remifentanyl	Nonspecific esterases
Cisatracurium	Hofmann elimination

Respiratory depression is much more likely with opioid or volatile anesthetic agents. Therefore consideration should be given to reducing all drug dosages by 25–50%.

Drugs Independent of Liver and Renal Function for Elimination

Examples. succinylcholine, esmolol, cisatracurium, remifentanyl.

These drugs undergo enzymatic or spontaneous breakdown in the blood (Table 1). Accumulation is unlikely, but altered pharmacodynamic responses should be anticipated.

Drugs with Increased Unbound Fraction in Hypoalbuminemia

Examples. thiopental, methohexital, diazepam.

In the presence of hypoalbuminemia associated with chronic renal or liver failure, these drugs have increased free or active fraction. Doses should be decreased 20–50%, depending on the degree of hypoalbuminemia.

Drugs Predominantly Dependent on Hepatic Biotransformation

Examples. lidocaine, all benzodiazepines, all opioids, dexmedetomidine, most nondepolarizing muscle relaxants (except cisatracurium).

These drugs should be restricted or used with care in hepatic failure. Drugs whose metabolism is dependent on the cytochrome oxidase (CP₄₅₀) system (e.g., diazepam, midazolam) are much more sensitive to liver dysfunction than those that undergo simple glucuronide conjugation (e.g., lorazepam, propofol). Lidocaine is so dependent on hepatic biotransformation that its metabolism to its primary metabolite, methylglycinexylydide (MEGX), is used as a sensitive indicator of liver function and reserve. Cumulative lidocaine toxicity with local or regional anesthesia, or continuous infusion, presents a special risk in patients with end-stage liver disease.

Volatile Anesthetic Agents

All volatile anesthetic agents have the potential to decrease hepatic blood flow, depending on their effect on the central circulation. Agents with potent negative inotropic effects such as halothane or enflurane, may decrease blood flow by 30–50%.

Table 2a. Child-Turcotte-Pugh Score for Cirrhosis

Parameter	1	2	3
Serum bilirubin (mg/dL)	<2	2–3	>3
Serum albumin (g/dL)	>3.5	3–3.5	<3
PT (sec > control)	1–4	4–6	>6
CNS (coma grade)	Normal	Confused (1–2)	Coma (3–4)
Ascites	None	Easily controlled	Poorly controlled

Table 2b. Child-Turcotte-Pugh Class and Preoperative Risk Assignment

Class	A	B	C
Score	5–6	7–9	10–15
Risk	Minimal	Moderate	Severe
Operative Mortality	0–10%	4–31%	19–76%

Hepatotoxic Effects

The potential for hepatotoxicity appears to be somewhat related to the extent of hepatic metabolism of the volatile agent. About 20% of halothane is eliminated by hepatic biotransformation, whereas only 2% of enflurane and sevoflurane, and 0.2% of isoflurane are metabolized by the liver.

Mild hepatotoxicity (transient modest elevation of liver enzymes) probably occurs in about 1 in 700 cases, and is related to injury caused by reductive metabolites, which are more likely to be formed in an hypoxic milieu.

Fulminant hepatic necrosis (“halothane hepatitis”) is a devastating injury with a high mortality. It appears to be induced by immune sensitization to the trifluoroacetylated products of oxidative metabolism (CP₄₅₀ 2E1). The true incidence is difficult to assess. The 1966 National Halothane Study concluded that it occurred in 1: 35,000 exposures to halothane. Subsequent large scale studies have suggested an equivalent risk of hepatic injury with enflurane and isoflurane, but that the actual incidence is considerably lower. Unfortunately, any occurrence of postoperative jaundice or elevation in liver enzymes tends to be labeled “halothane hepatitis” by our surgical and medical colleagues. Patients appear to be genetically predisposed, and the risk may be enhanced by the concomitant use of agents that induce mixed function oxidases, such as acetaminophen. The most important risk factor is re-exposure to halothane within two weeks. There appears to be cross-reactivity with other agents, e.g., a patient with a history of halothane-induced hepatotoxicity may develop recurrent injury on exposure to isoflurane.

PREOPERATIVE EVALUATION

Child-Turcotte-Pugh Score

The most widely used tool for assessment of risk in patients with cirrhosis is the Child-Turcotte-Pugh Classification (Table 2a and 2b), which is reasonably

Table 3. High Risk Procedures in Cirrhotic Patients Independent of Child-Turcotte-Pugh Classification

Procedure	Risks and complications
Emergency surgery (laparotomy)	Liver failure, 25% mortality rate
Prior abdominal surgery	Neovascularization: bleeding
Cardiopulmonary bypass	Severe coagulopathy and bleeding, high mortality
Ileostomy, colostomy	High incidence of ascitic leaks
Cholecystectomy	Portal hypertension, coagulopathy: bleeding from gall bladder bed
Hepatic tumor resection	Bleeding, liver failure

Table 4. Contraindications to Elective Surgery in Liver Disease

1. Acute viral hepatitis
2. Acute alcoholic hepatitis
3. Fulminant liver failure
4. Chronic active hepatitis (symptomatic)
5. Child's Class C cirrhosis
6. Severe coagulopathy
 - a) Prothrombin time >3 sec above control, not correctable
 - b) Platelet count <50 k/mm3
7. Comorbidity:
 - a) Congestive heart failure
 - b) Acute renal failure
 - c) Hypoxemia

predictive of perioperative mortality. A score of 1–3 is ascribed based on the degree of abnormality of five parameters, including bilirubin, albumin, PT, grade of encephalopathy and ascites. Thus, the minimal score is 5 (Child's A) and the maximum score is 15 (Child's C). In general, patients with a Child's A score present minimal risk for elective surgery, which should proceed; with Child's C it is contraindicated. Patients with Child's B criteria fall into an intermediate category and must be evaluated on an individual basis. However, regardless of the Child's classification, a prothrombin time prolonged >3 sec above control that does not correct with Vitamin K is an important predictor of poor outcome. Other independent risk factors are listed in Table 3.

Meld Score

More recently the MELD (Model for End-stage Liver Disease) Score has emerged as an important predictor of mortality that is used predominantly to prioritize patients for orthotopic liver transplantation. It is based on a complex nomogram that incorporates exponentials of the bilirubin, serum creatinine and INR.

Contraindications to elective surgery in liver disease are detailed in Table 4.

PREOPERATIVE PREPARATION

Medical Management

It may be helpful to drain tense ascites preoperatively – this will decrease diaphragmatic pressure and allow more easy positioning of the patient. It must be done with caution because of the risk of inducing acute intravascular hypovolemia, hypotension and further liver injury.

Many patients are on the aldosterone antagonist, spironolactone, which promotes sodium excretion and potassium retention. It is long acting and could exacerbate hyperkalemia in the presence of acute renal insufficiency or failure. If possible, spironolactone therapy should be discontinued 3 to 4 days before surgery.

An attempt to correct factor VII deficiency and prolonged prothrombin time should be made with parenteral Vitamin K and/or fresh frozen plasma (FFP). However, these may be largely ineffective in patients with severe liver damage, and administration of several units represents a substantial volume load.

Precipitating Factors of Encephalopathy Should Be Treated or Removed by Protein Restriction, Lactulose and/or Neomycin

Patients with end-stage renal disease have a very high incidence of hepatorenal syndrome and are exquisitely sensitive to small decreases in intravascular volume. Steps should be taken to ensure adequate preoperative hydration in these patients, i.e., maintenance saline infusion during preoperative fasting. Pharmacologic renal protection (low dose dopamine, furosemide infusion, fenoldopam) is frequently used during orthotopic liver transplantation. Although these agents are effective at inducing diuresis, there are few if any prospective data that suggest that they decrease the risk of perioperative renal injury.

Transjugular Intrahepatic Portasystemic Shunt (TIPS)

The TIPS procedure is being used with increasing frequency especially in patients who are candidates for orthotopic liver transplantation. It decompresses the portal system, relieves severe ascites, decreases the risk of variceal bleeding, and in some patients improves renal perfusion and hepatorenal syndrome. The procedure is performed in the invasive radiology suite. A metallic shunt is passed via the internal jugular route into the hepatic vein, and thence driven through the liver until the portal vein is reached and pressure gradient drops. Acute risks include bleeding, acute heart failure from sudden increase in right atrial filling, and endotoxemia from portasystemic shunting. There is also an increased risk and susceptibility to encephalopathy.

Immediate Preoperative Preparation

Omit oral premedication except for aspiration prophylaxis. If necessary, give small doses of IV sedation

in induction room or operating room, but always under direct observation.

Use universal precautions and asepsis throughout; all staff should have been vaccinated against hepatitis B whether or not the patient is known to be a carrier.

There should be a low threshold for using invasive monitoring for any surgical procedure liable to involve fluid shifts. Patients with hepatorenal syndrome are at very high risk of perioperative acute renal failure and intravascular volume status is difficult to assess because of ascites and anasarca.

Considerations for positioning and avoidance of hypothermia are as for chronic renal failure. Tense ascites adds an additional degree of difficulty.

ANESTHETIC PLANNING AND MANAGEMENT

Anesthetic Plan

Regional anesthesia may help to preserve hepatic blood flow if blood pressure and cardiac output are maintained. However, the common presence of coagulopathy, ascites, and encephalopathy limit its application.

Drug handling is extremely variable. Altered pharmacokinetics are a consequence of a large volume of distribution but markedly impaired hepatic elimination. Thus, the loading dose requirement for certain drugs may be high, but emergence is substantially delayed. This applies for example to rocuronium, whose onset of action is delayed by an enlarged volume of distribution in patients with severe liver disease. Moreover, even though its elimination kinetics are unaltered, the time to recovery is prolonged.

Doses of all sedative agents should be substantially decreased in severe liver disease.

Anesthetic Induction

Management of anesthetic induction is similar to chronic renal failure, and should incorporate preoxygenation, adequate fluid loading and aspiration precautions.

Succinylcholine apnea has been rarely reported in patients with severe liver dysfunction and is related to very low levels of plasma cholinesterase. Metabolism of cisatracurium is independent of liver function and it is the neuromuscular blocker of choice. A metabolite, laudanosine, may accumulate in liver disease. In dogs, high laudanosine levels are associated with electrical seizure activity, but these have never been encountered in humans nor reported in patients.

Anesthetic Maintenance

All volatile anesthetic agents decrease hepatic blood flow based on their effects on the central circulation, but this can be overcome by appropriate hemodynamic management. Hypercarbia and hypocarbia decrease portal flow and should be avoided. Opioids, with the notable exception of remifentanyl, may accumulate and delayed emergence should be anticipated if they are used. Remifentanyl pharmacokinetics are

unchanged even in the presence of severe liver disease, but patients are more sensitive to its pharmacodynamic effect in suppressing ventilatory drive.

The short duration of propofol effect is related to its high lipid solubility and rapid distribution out of the CNS. Thus, it remains a relatively short-acting drug even in patients with advanced cirrhosis. However this advantage is offset by its effects on the circulation, which include myocardial depression, inhibition of reflex tachycardia and vasodilation, keeping in mind that these patients are already hypotensive at baseline.

The anesthesiologist should anticipate intraoperative hypoxemia (ascites, hepatopulmonary syndrome), bleeding (coagulopathy) and oliguria (hepatorenal syndrome).

An important intraoperative consideration in the anesthetic management of partial hepatectomy or liver transplantation is the avoidance of excessive volume loading. Hepatic venous congestion increases venous oozing and markedly increases intraoperative blood loss, perhaps the most important determinant of outcome after hepatic resection. A fluid restrictive approach during hepatic resection has been shown to decrease intraoperative blood loss. Hepatic swelling can also irreparably injure the newly transplanted liver. Although it is essential to maintain intravascular volume and hepatic perfusion, efforts should be made to keep the CVP ≤ 10 mm Hg in patients with normal cardiac function.

Emergence and Postoperative Care

Anesthetic emergence may be delayed and complicated by vomiting and aspiration, hypotension, respiratory depression and acute respiratory failure. Patients should have their trachea extubated only when they are fully awake to reduce the risk of aspiration. Similarly, a short period of postoperative mechanical ventilation allows controlled emergence, avoids reversal agents, and facilitates evaluation of neurologic and ventilatory function prior to extubation.

Potential postoperative problems include bleeding, oliguria, encephalopathy, acute respiratory failure, sepsis, wound dehiscence and acute hepatic failure.

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