Chapter 40

Hepatic Disease

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Hepatic Blood Supply

About 20% of cardiac output is continually being delivered to the liver. Of this, the hepatic artery supplies 30% of the blood flow and 90% of the oxygen, and the remainder is supplied by blood flowing through the portal vein. Anesthetics may affect hepatic blood flow by altering vascular tone in the hepatic artery, the portal vein, or both (Table 40.1). Methoxyflurane decreases both portal vein and hepatic arterial blood flow. Halothane decreases portal vein blood flow but has only a slight effect, if any, on hepatic arterial blood flow. Isoflurane decreases portal vein blood flow but increases hepatic artery blood flow with a net overall increase in flow. Nitrous oxide has no direct effect on liver perfusion. Sevoflurane and desflurane maintain hepatic artery and portal vein resistance and are associated with decreased portal vein and total hepatic blood flow.[1] Hypercarbia has also been associated with decreased hepatic perfusion during anesthesia. Decreased blood flow may occur in cirrhotic livers or patients with portocaval shunt syndrome. Detrimental effects of decreased blood flow are likely to be more significant when an animal is hypotensive. Significant decreases in blood flow may be associated with decreased hepatic extraction and, ultimately, elimination of drugs.[2]

Hepatic Insufficiency

Clinical signs of hepatic insufficiency include ascites, depression, seizures, jaundice, hepatic encephalopathy, anorexia, and weight loss. Horses with colic have an increased incidence of concurrent hepatic disease.[3] Tests of substrate metabolism may give an indication of hepatic function. Low values for albumin, urea nitrogen, glucose, and cholesterol are associated with poor hepatic function. Coagulation defects such as prolonged prothrombin time, prolonged partial thromboplastin time, and increased fibrinogen values may also indicate decreased hepatic function. Bilirubin is formed by metabolism of degraded hemoglobin by macrophages and carried by albumin to the liver for conjugation to a diglucuronide. In the horse, fasting for more than 24 h causes fatty acids to compete with bilirubin for hepatic metabolism. In this way, hyperbilirubinemia frequently causes icterus in anorexic horses. In dogs and cats, hyperbilirubinemia may be due to prehepatic, hepatic, or posthepatic causes.

Blood ammonia concentration and retention of dyes such as sulfobromophthalein (BSP) or indocyanine green (ICG) indicate liver dysfunction as evidenced by the liver's inability to eliminate these substances in a normal manner. The dyes, like bile acids, are more sensitive indicators of liver dysfunction than is a change in bilirubin concentration.[4] Bile acids are produced by the liver and excreted in the bile. They undergo a process termed *enterohepatic circulation.* Following biliary excretion, bile acids are reabsorbed by the ileum and enter the portal circulation. Bile acids are then removed by the hepatocytes, leaving only a low concentration to enter the systemic circulation during normal circumstances. Fasting does not affect bile acid concentration. Normal fasting bile acid concentrations are below 5 mmol/L for cats, below 10 mmol/L for dogs, and below 15 mmol/L for horses.

Postprandial blood samples will demonstrate an elevation in bile acid concentration caused by gallbladder contraction. Within 2 h, as a result of gallbladder emptying, the liver can remove most of the bile acids presented to it. Thus, a marked increase in postprandial bile acid concentration indicates decreased hepatic function or the presence of a portocaval vascular shunt. Normal 2-h postprandial concentrations of bile acids are below 15 mmol/L in cats and below 25 mmol/L in dogs. Because horses lack a gallbladder, postprandial elevations in bile acid concentrations are not expected. Use of bile acid concentrations to aid in assessment of hepatic disease in horses is best when combined with other tests of hepatobiliary function.[5] Tests of cell membrane integrity (i.e., alanine aminotransferase, γ-glutamyltransferase, sorbitol dehydrogenase, and lactate dehydrogenase) and serum alkaline phosphatase indicate hepatocellular damage but may not reflect altered hepatic function.

Pharmacological Considerations for Patients with Hepatic Disease

Tranquilizers/Sedatives

The use of acepromazine, droperidol, and α_2-adrenergic agonists should be avoided in patients with moderate to severe liver dis-

Table 40.1. Effect of inhaled anesthetics on hepatic blood flow

Agent	Portal Vein	Hepatic Artery
Desflurane	Decrease	No change
Halothane	Decrease	Decrease or no change
Isoflurane	Decrease	Increase
Nitrous oxide	No direct effects	
Sevoflurane	Decrease	No change
Methoxyflurane	Decrease	Decrease

ease. Hypotension may occur after administration of phenothiazine (acepromazine) or butyrophenone (droperidol) tranquilizers because of peripheral vasodilation mediated by α-adrenergic blockade. Phenothiazine administration has also been associated with thrombocytopathy and may exacerbate coagulopathy associated with hepatopathy. Dysrhythmias such as bradycardia or atrioventricular block and alterations of plasma glucose concentration may occur after administration of xylazine or detomidine. Diazepam is generally considered safe when used in intravenous doses of less than 0.2 mg/kg because it causes minimal changes in cardiovascular function. However, in animals with severe liver dysfunction, the duration of action of benzodiazepines may be significantly prolonged because of decreased hepatic biotransformation. Diazepam may not consistently tranquilize healthy young animals but frequently produces tranquilization in animals with liver disease. When diazepam is administered intravenously, it should be injected slowly to decrease vessel irritation and prevent hypotension associated with the propylene-glycol carrier. Use of benzodiazepines in animals exhibiting signs of hepatic encephalopathy is controversial.

Opioids

These can be used in patients with liver disease. Morphine and meperidine can cause release of histamine, which may cause hypotension and a decrease in total hepatic blood flow. Morphine also constricts the sphincter of Oddi in people, yet the significance in obstructive biliary disease in animals is unknown. Opioid side effects should be treated if they appear to affect cardiac or respiratory function. Opioid-induced bradycardia may decrease cardiac output and should be prevented by administration of an anticholinergic agent. Respiratory depression from opioids may lead to decreased oxygen delivery to all body tissues, including the liver. Careful dose titration of the opioid antagonist naloxone can relieve depression of respiration while maintaining some of the analgesic and sedative effects of the opioid agonist. Butorphanol and buprenorphine are associated with less respiratory depression than other opioid agonists and may be reasonable choices for patients with hepatic disease when maximal opioid analgesia is not required. Fentanyl, delivered by continuous-rate intravenous infusion, enables rapid individual patient dose titration and is an effective and reasonably safe method for providing opioid analgesia to patients when liver function is unknown.

Thiobarbiturates

These should be used in low doses or their use avoided in patients with liver disease. A single, intubating dose of thiobarbiturate is not necessarily contraindicated because it will be redistributed from the brain to less well perfused tissues, terminating the anesthetic effect. However, liver disease may affect the duration and depth of thiobarbiturate-induced anesthesia because of increased sensitivity of the central nervous system or hypoalbuminemia and decreased protein binding of the anesthetic. Anesthesia should not be maintained by redosing thiobarbiturates in patients with liver disease.

Methohexital is a methylated oxybarbiturate, and although it is more rapidly metabolized than thiobarbiturates, it is associated with excitation and possible seizures during the recovery period. For this reason, methohexital should be avoided in patients with hepatic encephalopathy.

Propofol and Etomidate

Propofol is an alkylphenolic compound used for induction of anesthesia that is supplied in a lecithin emulsion, giving it a milky white appearance. Redistribution and metabolism of propofol after a single injection are extremely rapid. The total body clearance of propofol exceeds hepatic blood flow, indicating sites other than the liver (e.g., lung) may play a role in its elimination. Propofol is about 90% excreted by the kidneys as glucuronate-conjugated metabolites.[6] Because of its dependence on glucuronidation for biotransformation, cats appear to have a more prolonged duration of propofol-induced sedation and hypnosis compared with dogs, especially following large or repeated doses. Indications for use of propofol are similar to those for the thiobarbiturates. The major advantage of propofol over a thiobarbiturate is the rapid rate of propofol elimination. In dogs, the incidence of apnea immediately after injection seems to be greater than that associated with thiobarbiturates.

Etomidate is an imidazole compound used for induction of anesthesia. It is supplied as a weak base in a propylene-glycol vehicle. As with other drugs formulated with propylene glycol, it can be irritating when injected intravenously.[7] Etomidate does not decrease hepatic perfusion. It has a short duration of action, primarily because of rapid redistribution from the brain to muscle tissue. Etomidate is metabolized by the hepatic microsomal enzyme system, as well as by plasma esterases. The total body clearance rate for etomidate is five times as fast as for thiopental, although clinically the duration of action is similar after a single dose.[4] Etomidate has been shown to cause adrenocortical suppression with repeated administration.[8] The importance of etomidate-associated inhibition of steroidogenesis after single-bolus administration in patients with hepatic disease is unknown. In both humans and dogs, etomidate continuous-rate intravenous infusion has been associated with hemolysis.[9,10] Hemolysis appears to be caused by the propylene-glycol vehicle in which etomidate is formulated. In dogs, red blood cell counts during and 10 min after termination of a 60-min infusion of etomidate were not significantly different from preinfusion values.[10] All dogs in the aforementioned study appeared normal after recovery from anesthesia maintained with etomidate. Thus, the clinical signifi-

cance of etomidate-induced or propylene-glycol-induced hemolysis in dogs is not clear. Etomidate appears to be a reasonable drug choice for induction of anesthesia in patients with cardiac and/or hepatic disease.

Dissociative Anesthetics

Dissociative anesthetics such as tiletamine (Telazol) or ketamine are generally acceptable for induction of anesthesia in patients with hepatic disease. Intravenous administration is preferred in order to minimize the dose required for tracheal intubation. In dogs, these drugs are largely metabolized by the liver, so maintenance of anesthesia should be with an inhalant. Dissociative anesthetics may induce seizures in dogs or cats. In cats, ketamine is metabolized to a lesser extent by the liver to form norketamine, which has about 10% of the activity of ketamine.

Zolazepam, the benzodiazepine tranquilizer in Telazol, has been suspected of causing the prolonged recovery after intramuscular injection in cats. Flumazenil has been used intravenously (0.1 mg/kg) to antagonize midazolam in cats anesthetized with a combination of ketamine and midazolam.[11] Zolazepam's effect in cats is similarly antagonized by flumazenil, providing an alternative to the apparently slow hepatic metabolism of the benzodiazepines in this species.

Inhalation Anesthetics

Inhalation anesthetics are the best choices for maintenance of anesthesia in patients with severe liver disease. Halothane decreases hepatic blood flow and is metabolized up to 20% by the liver.[12] Halothane anesthesia in ponies is associated with decreased bile acid excretion and increased conjugated bilirubin excretion.[13] It is recommended that halothane be avoided, if possible, in patients with liver disease. However, the presence of hepatic disease does not necessarily result in increased hepatotoxicity when a patient is subsequently exposed to an unpredictable hepatotoxin such as halothane.[14] Halothane has been implicated in causing liver disease in people and possibly in animals. This is a problem in about one of every 6000 to 10,000 people and is probably genetically related.[15,16] Halothane is metabolized to trichloroacetic acid, which may undergo reductive metabolism to produce hepatotoxins during hypoxic conditions. Studies indicate that the trifluoroacetate metabolite of halothane combines with a hepatic protein, resulting in the formation of a hapten.[17–19] The trifluoroacetate hapten is subsequently attacked by serum antibodies, causing hepatitis. A clinical test for this hapten is not yet available for animals. In a study of goats hyperimmunized for production of anti–human-lymphocyte serum that were subsequently exposed to 3, 6, or 9 h of halothane anesthesia, seven (24%) of 29 showed evidence of hepatic necrosis.[20] A rat model of halothane-induced hepatopathy showed that hepatic hypoxia was required to produce symptoms.[21] To ensure adequate blood pressure, flow, and oxygen delivery during anesthesia, precautions should be taken when using halothane (or any other anesthetic!). In preventing the occurrence of postanesthetic hepatopathy, prevention of hepatic hypoxia during inhalation anesthesia is probably more important than choice of anesthetic.

Methoxyflurane decreases hepatic blood flow, and up to 50% of an inhaled dose is metabolized by the liver.[22] In addition, oxalate and free fluoride-ion metabolites of methoxyflurane are potential renal toxins. Of the volatile anesthetics, methoxyflurane is associated with the highest metabolite production of fluoride ion.

Isoflurane increases hepatic artery blood flow in humans, whereas halothane preserves hepatic arterial flow at 1 MAC but decreases it at 2 MAC.[23] Less than 1% of inhaled isoflurane is metabolized. It has not been associated with hepatic or renal toxicity.[24] Cardiovascular function during isoflurane anesthesia is less depressed than with halothane or methoxyflurane. The higher cardiac output associated with isoflurane anesthesia is likely to maintain better hepatic perfusion and oxygen delivery than is halothane. Thus, isoflurane appears to be a good choice for maintenance of anesthesia in patients with hepatic disease.

Biotransformation of sevoflurane (3% to 5%) is less than halothane but more than isoflurane or desflurane.[25] Production of the trifluoroacetic acid hapten is extremely low and unlikely to induce an immune response such as the well-documented halothane hepatopathy. Free fluoride–ion production after exposure to sevoflurane is comparable to that from desflurane.[25] Sevoflurane reduces portal vein flow and oxygen delivery more than isoflurane. Hepatic artery flow increases in pigs concurrently receiving nitrous oxide and sevoflurane. Other studies have shown no change in hepatic artery flow associated with clinically useful concentrations of sevoflurane. Animal models therefore demonstrate that sevoflurane maintains good hepatic blood flow and liver oxygenation when administered at concentrations less than 2 MAC.[25]

The effects of desflurane on hepatic blood flow are similar to those of halothane (Table 40.1). Desflurane differs from isoflurane structurally only by substitution of a fluorine atom for a chlorine atom. The substitution of a fluorine atom for other halogens generally makes a molecule more stable in terms of biotransformation. Indeed, desflurane metabolism to free fluoride ion is less than that of isoflurane.[26] Because of its low metabolism, low blood solubility (enabling rapid induction and recovery from anesthesia), and cardiovascular stability (maintaining hepatic perfusion), desflurane would appear to be a good anesthetic for patients with hepatic disease.

Nitrous oxide has been used in patients with hepatic disease in an effort to decrease the amount of volatile anesthetic required for anesthesia. There are no reports of direct hepatic injury caused by exposure to nitrous oxide as used in clinical practice. There is evidence of hepatic injury (centrilobular necrosis) caused by nitrous oxide in the hypoxic rat model.[27] It is unknown whether this mechanism of hepatic injury in hypoxic rats is the same as for halothane.

Muscle Relaxants

Nondepolarizing muscle relaxants such as pancuronium and vecuronium are metabolized by the liver. Effects of these relaxants may be prolonged in patients with hepatic disease. Concurrent administration of aminoglycoside antibiotics with nondepolarizing muscle relaxants may also prolong neuromuscular blockade.[28] This is likely to be a problem in large animals such as horses, for which prolonged recumbency may adversely affect

recovery to standing. Atracurium is a nondepolarizing muscle relaxant whose metabolism is independent of hepatic function. Atracurium undergoes plasma degradation via a metabolic pathway termed *Hofmann elimination* that depends primarily on plasma pH and temperature.[29] For patients with hepatic disease requiring neuromuscular blockade, atracurium seems to be a good choice.

Succinylcholine is a depolarizing muscle relaxant that is degraded by plasma cholinesterase. Cholinesterases are produced by the liver. Organophosphate compounds have cholinesterase-inhibiting activity and may potentiate the action of succinylcholine.[30] The phenothiazine tranquilizers (e.g., acepromazine) may inhibit cholinesterase activity, so succinylcholine should be used cautiously in patients medicated with acepromazine. The use of both acepromazine and succinylcholine should be avoided in patients with hepatic disease.

Centrally acting muscle relaxants such as guaifenesin (glyceryl guaiacolate) or mephenesin are metabolized by the liver.[31,32] Although the margin of safety for use of guaifenesin is wide, side effects following administration of large doses include moderate hypotension, decreased tidal volume, and concentration-dependent hemolysis. Toxic effects may be caused by the metabolite catechol, which causes seizures, opisthotonos, prolonged incoordination, and respiratory paralysis. Increased incidence in side effects or occurrence of toxic effects may be observed when guaifenesin is administered to animals with hepatic disease.

General Guidelines for Patients with Hepatic Disease

Hepatic disease affects several vital body functions, with significant impact on anesthetic considerations (Table 40.2). Most injectable anesthetics are metabolized by the liver, so elimination may be significantly slowed in animals that are hypothermic. Use of heated circulating water blankets and administration of warm intravenous or irrigation fluids is advised. The advent of force air-warming systems (e.g., Bair Hugger) has dramatically reduced intraoperative and postoperative hypothermia.

Glucose metabolism is frequently affected by hepatic disease. Homeostasis of glucose can be maintained with loss of up to 80% of functional liver mass. Nevertheless, patients with severe hepatic disease that are stressed by anesthesia and surgery may become hypoglycemic. Hypoglycemia is present in 35% of dogs with portocaval shunts.[33] For this reason, blood glucose concentration is routinely determined and glucose administered in combination with isotonic crystalloids when indicated. If not corrected, hypoglycemia may delay recovery from anesthesia.

Excessive weight of ascitic fluid may impede lung expansion and pulmonary function. Ascitic fluid should be removed prior to anesthesia. Rapid removal of a large quantity of fluid may cause a fluid shift from the vascular space to the abdominal cavity as the formation of ascites continues. This can cause serious hypovolemia and cardiovascular compromise. Hypoalbuminemia is often present in patients with liver disease. When the albumin concentration is less than 1.5 g/dL, the plasma oncotic pressure is decreased such that pulmonary edema may occur following intravenous fluid administration with crystalloids. Arterial blood pressure may be difficult to maintain when plasma oncotic pressure is significantly decreased. Replacement of albumin and plasma proteins is indicated in these cases. If matched plasma from a donor animal is not available, dextran 70 (up to 20 mL/kg) or Hetastarch (10 to 20 mL/kg) may be administered. Although dextran will aid in reestablishing plasma oncotic pressure, its duration of effect is not as great as that of plasma. Human albumin solution (2.5 to 5.0 mL/kg intravenously) has been used in dogs and cats, as well.

Table 40.2. General guidelines for patients with hepatic disease

Evaluate coagulation profile prior to surgery
Use rapidly eliminated agents when appropriate (e.g., inhalant anesthetics and propofol)
Monitor hydration status
Monitor blood glucose, plasma albumin, and total protein concentrations
Monitor and maintain arterial blood pressure greater than 60 mmHg
Maintain eucapnia using appropriate ventilatory support
Be prepared to treat seizures with diazepam
Be aware of potential thromboembolic complications

Seizures caused by hepatic encephalopathy may require treatment. Diazepam or phenobarbital is commonly used to control seizures, but both drugs are metabolized by the liver, and significantly altered pharmacokinetics may result in prolonged duration of action. Avoid the use of anesthetics that may induce seizure activity, such as enflurane, ketamine, tiletamine (Telazol), or methohexital. There may be increased cerebral sensitivity to γ-aminobutyric acid (GABA) in some patients with hepatic disease. This increased sensitivity to GABAergic inhibition within the central nervous system may enhance the depressant effects of the barbiturates and benzodiazepine tranquilizers.

When there is concern about an animal's ability to metabolize anesthetics because of hepatic disease, use of local anesthesia may be considered as an adjunct to general anesthesia or as a substitute when appropriate. Although animals that are not depressed usually require tranquilization for effective use of local anesthesia, those with hepatic disease are likely to be depressed and therefore may require less tranquilization or sedation. Local anesthetics of the ester class (e.g., procaine and tetracaine) are metabolized by plasma cholinesterases produced by the liver, whereas the amide class (lidocaine, mepivacaine, and bupivacaine) is directly metabolized by the liver. For this reason, the generalized effects of local anesthetics may be prolonged and the toxic effects more apparent in patients with severe hepatic disease. This is unlikely to be a major deterrent for their clinical use, however.

Because of the liver's role in production of coagulation factors, the coagulation profile (one-stage prothrombin time and activated partial thromboplastin time) should be evaluated before surgery in patients with hepatic disease. If coagulopathy exists, fresh-frozen plasma or whole blood should be available and administered. Additionally, some hepatic neoplasms are associated

with significant abdominal hemorrhage and thrombocytopenia. A complete blood count with an estimate of the platelet concentration should be performed as part of the diagnostic workup.

Little is known about the influence of specific anesthetic techniques on coagulation in animals. In dogs, platelet aggregation was significantly decreased after administration of acepromazine and atropine but returned to normal during subsequent halothane anesthesia and surgery.[34] Coagulation deficiencies such as thrombocytopenia, thrombocytopathy, increased prothrombin time, increased activated partial thromboplastin time, hypofibrinogenemia, increased fibrin degradation products, and decreased antithrombin have been observed in dogs with gastric dilation–volvulus.[35] In dogs undergoing colonic anastomosis, there was less intraoperative bleeding and more advanced healing (detected histologically) 1 and 7 days after surgery following administration of epidural bupivacaine and general anesthesia when compared with general anesthesia alone.[36] Much work remains to be done to identify anesthetic techniques having the least effect on normal coagulation and healing during the convalescent period.

Portosystemic Shunt

Dogs with this congenital vascular anomaly are usually gaunt and small for their breed and age. Anesthetic management of dogs with portocaval shunt should be based on presenting clinical signs and physical status (Table 40.3). Signs of hepatic encephalopathy and hypokalemia may be present. Hypokalemia in animals with portocaval shunt may be caused by gastrointestinal (vomiting and diarrhea) or urinary loss (diuresis). Hypokalemia ($[K+] < 3.5$ mEq/L) should be corrected gradually prior to anesthesia. Intravenous potassium administration should not exceed the rate of 0.5 $mEq \cdot kg^{-1} \cdot h^{-1}$.

Chronic hepatic dysfunction may be associated with increased GABAergic sensitivity and permeability of the blood-brain barrier. The effect of anesthetics and anesthetic adjuncts may be greater than expected and unpredictable. Diazepam has been used as a preanesthetic medication in dogs with portocaval shunt. However, reduced dosage requirements for benzodiazepines in cases of hepatic disease have been reported.[37,38] Further, cases of fatal hepatic toxicosis associated with benzodiazepine administration have been observed in cats. Antagonists of the benzodiazepine receptor, such as flumazenil, have ameliorated signs of encephalopathy in a woman and in animal models of portocaval shunt.[37–39] The value of flumazenil in the perioperative management of dogs with portocaval shunt is controversial and remains to be determined.

Animals with portocaval shunt may have hepatic insufficiency. When termination of action is highly dependent on drug hepatic metabolism (e.g., ketamine, acepromazine, xylazine, and diazepam), the use of such drugs should be avoided. Drugs that are highly protein bound (e.g., barbiturates) will be more active in animals with hypoalbuminemia. Drugs such as methohexital and ketamine that may induce seizure activity are contraindicated in animals with hepatic encephalopathy. Of the inhaled anesthetics, methoxyflurane undergoes the greatest metabolism by the liver. Thus, elimination of methoxyflurane may be prolonged in animals with portocaval shunt. The use of halothane may cause hepatopathy in animals with poor hepatic perfusion and oxygenation, and therefore should be avoided in patients with portocaval shunt. Isoflurane, sevoflurane, or desflurane are suitable anesthetics for patients with this condition. Mask induction and maintenance with an inhalant are reasonable for animals with portosystemic shunts displaying advanced signs of hepatic dysfunction. Opioids may be used as analgesic supplements prior to or following anesthesia for surgical correction of the shunt.

Table 40.3. Problems associated with portocaval shunt

Problem	Significance
Weight loss	May affect drug dose or disposition
Hypoalbuminemia	May affect drug dose, disposition, or plasma oncotic pressure
Hepatic shunt	Loss of hepatic metabolism of drugs
Low bile salts	Increase in absorption of intestinal endotoxin
Portal hypertension	May require second surgery and central venous pressure monitoring

Arterial blood pressure should be monitored during portocaval shunt surgery. Hypoalbuminemia and hypothermia predispose patients to hypotension that may be exacerbated by isoflurane-induced peripheral vasodilation. Judicious use of plasma, dextran 70 (10 to 20 mL/kg given over 1 h) or hetastarch to aid in maintenance of plasma oncotic pressure may be indicated. At high concentrations of isoflurane, myocardial contractility and cardiac output are also decreased. Hypotensive patients with portocaval shunt may fail to respond to catecholamine infusions such as dobutamine or dopamine. Surgical retraction of the liver or compression of the caudal vena cava may further decrease venous return, cardiac output, and arterial blood pressure.

Intraoperative management of patients for correction of portocaval shunt is often uneventful until the shunt is ligated. In normal dogs, portal pressure is reported to be between 8 and 13 cm H_2O, whereas portal pressure in dogs with portocaval shunt is usually lower.[40] Following surgical correction of the shunt, portal venous pressure should not be more than 9 to 10 cm H_2O above baseline measurement, or a maximum of 20 to 23 cm H_2O. Intraoperative measurement of central venous pressure is useful for estimating portal resistance and for predicting the development of postoperative portal hypertension.[41] A decrease in central venous pressure after ligation of a single portocaval shunt indicates decreased blood transit from the intestine to the vena cava. Central venous pressure should not decrease by more than 1 cm H_2O from baseline measurement at 3 min after ligation to avoid postoperative portal hypertension.[42]

Animals with portocaval shunts or intestinal ischemia may have increased absorption of endotoxin. Medical management of these patients includes fluid administration (e.g., 0.9% or 0.45% sodium chloride combined with 2.5% dextrose), potassium supplementation, dietary management, and appropriate antibiotic therapy to reduce toxin production from enteric bacteria.[43]

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