Ascites is a frequently encountered sequela of severe hepatic disease. The LeVeen shunt, a peritoneovenous conduit, is a surgical means of overcoming this serious problem. In order to plan and carry out anesthetic management of these patients, it is necessary to understand the major physiological derangements of hepatic disease and their impact on anesthetic technique.

Ascites is a frequent sequela of severe hepatic disease. Recently, surgical intervention in the form of a peritoneovenous (P-V) LeVeen shunt has been introduced in the treatment of ascites. Anesthetic management of the patient with ascites undergoing placement of a LeVeen Shunt requires an understanding of: (1) the physiology of hepatic circulation; (2) the systemic consequences of ascites; (3) surgical technique; and (4) anesthetic considerations.

The liver receives approximately 25% of the cardiac output via the hepatic artery and 75% from the portal vein. The portal vein is at the junction of the superior mesenteric vein and the splenic vein; it supplies the liver with blood from capillaries in the gastrointestinal (GI) tract. Blood from the heart is transported to the liver from the hepatic artery. It flows through the sinusoids of the liver from the hepatic arterioles and portal venules. The sinusoids are the vessels of exchange for the liver and function through active transport, diffusion and pinocytosis. The sinusoids empty into an intralobular vein which drains into a hepatic vein and, finally, into the inferior vena cava.

**Ascites formation**

Normal pressure in the portal vein is approximately 10 mmHg and in the hepatic vein, is 0 mmHg. Portal hypertension is a persistent increase in portal vein pressure. The exact etiology is not fully understood but may be generated by a primary increase in vascular resistance in the portal system.

Degeneration of the parenchymal cells of the liver leads to impairment of circulation through the liver, which interferes with the low-pressure portal vein system. The resulting portal hypertension favors the exudation and accumulation of fluid in the abdomen, both from the capillaries within the liver and also from the capillaries in the intestine.

Portal hypertension is not the only factor involved in ascites formation and collection. Impaired liver function results in hypoalbuminemia, lowered colloid osmotic pressure, and an effective outward gradient for filtration of fluid. There may also be an increased level of antidiuretic hormone (ADH) in the urine, probably due to failure of the liver to inactivate this hormone. Aldosterone activity may also be enhanced, thus favoring salt and water retention.
The liver, the bowel and the mesentery surfaces appear to be the source of the ascitic fluid. However, the actual etiological mechanisms are extremely complex and multiple factors are often associated with the formation of ascites.4

The movement of fluids transperitoneally have been compared to Starling forces that control the movement of fluids across capillary beds. Fluid is pushed out of the capillaries into tissue spaces by intravascular hydrostatic (portal) pressure and is pulled out by interstitial (intraperitoneal) protein oncotic pressure.

Opposing these forces is intravascular protein oncotic pressure, which pulls fluid out of the tissues. Interstitial oncotic and hydrostatic pressures, both difficult to measure, also contribute. Ascitic fluid is in equilibrium with the other body fluids, with 40-80% of the total ascitic fluid turned over every hour.4

If obstruction occurs between sinusoids and the hepatic vein, protein-rich hepatic fluid forced by hydrostatic pressure crosses the sinusoidal wall and enters the peritoneal cavity. Although thoracic duct lymph flow increases, ascses accumulates if lymph removal cannot keep pace with the increased production. This theory is supported by the observation that recently synthesized albumin may enter ascitic fluid directly from the liver rather than through the bloodstream.5 In cirrhotic patients, low serum protein levels occur because of decreased production and loss of albumin.3 4

When hypoalbuminemia, which results in decreased intravascular protein oncotic pressure, is combined with portal hypertension (due to hepatic vein blockage) and subsequently increasing portal hydrostatic pressure, an alteration in Starling forces occurs. Whereas neither hypoalbuminemia nor portal hypertension alone may be sufficient to produce ascites in the cirrhotic patient, their combination may be critical in ascites formation.4 5 6

Another factor is the buildup of aldosterone which occurs in these patients. Urinary and blood aldosterone levels may be increased up to 25 times the normal level.6 The primary action of aldosterone in man is the regulation of sodium homeostasis and maintenance of a constant volume of extracellular fluid despite variations in sodium intake.

**Effects of ascites**

When ascites accumulates, the circulating arterial blood volume is reduced. This diminished circulatory flow is interpreted as a "blood-loss" situation. The adrenals, through the renin-angiotensin-aldosterone system, produce more aldosterone to compensate for this blood loss via fluid retention. Aldosterone acts in the distal renal tubule to cause additional fluid to be retained, simply providing yet more water for additional ascites formation.4

Liver disease produces many other alterations of physiological function. Since the liver is the major site of detoxification of many drugs, the pharmacological action of these drugs may be prolonged. Endocrine changes may occur due to altered hepatic metabolism or conjugation and excretion of hormones by the liver. Magnesium deficiency and hypokalemia may aggravate muscle weakness. Hypoglycemia can occur as a result of liver necrosis or impaired glycolysis and gluconeogenesis. Malabsorption of vitamin K can cause hypoprothrombinemia.

Other systemic disturbances the patient with ascites can exhibit include:

**Neurological disturbances.** Drowsiness, confusion and euphoria, hallucinations, delirium tremens (DTs) and convulsions have been reported.7 Hepatic encephalopathy may be present. Apnea, cardiac arrest and hypothermia may also occur.

**Acid-base disturbances.** Hyperventilation with accompanying hypocapnia and respiratory alkalosis is possible. Respiratory alkalosis can cause impaired oxygen dissociation, reduced cerebral and peripheral perfusion and reduce cerebral O2 consumption. Hypoxia is a common finding and may be due to infection or elevation of the diaphragm by the ascites. Transient pulmonary atelectasis may also be present. The presence of intra-pulmonary shunts may further lower arterial O2 tensions.

**Cardiac disturbances.** Cardiac output is high and may reflect a low peripheral resistance with increased arterial-venous shunt. Transient hypotension and bradycardia can occur. Baroreceptor-mediated sympathetic reactivity which can alter relationships in plasma volume, PCO2, and P02 is frequently seen. Changes in potassium, PO2, PCO2 and intra-cranial pressure can cause arrhythmias.8

**Renal disturbances.** There may be increased ADH and aldosterone activity. Renal impairment can lead to renal failure. Uremia may be due to dehydration or absorption of nitrogen compounds from the gastrointestinal (GI) tract, especially if there has been a GI hemorrhage. Renal blood flow is diverted from the cortex to the medulla which causes a reduction in glomerular filtration rate (GFR) and increases sodium retention. Hypotension can lead to acute tubular necrosis.7

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Journal of the American Association of Nurse Anesthetists
sion of hypertonic glucose solutions can cause glycosuria and polyuria.

Reduced renal blood flow secondary to decreased blood volume and cardiac output may also alter renal function. There may be kidney damage caused by the agents producing the liver disease. The hepatorenal syndrome is not fully understood at present. The patient with liver disease who develops hepatorenal syndrome has histologically normal kidneys, oliguria, azotemia, and high urine osmolality with low sodium content.\(^9\)

The underlying abnormality in these patients is not only an excess of fluid but also a maldistribution of extracellular fluid. Diuretics are commonly used to treat severe ascites with limited success. A diuresis that exceeds the ability for mobilization of ascitic fluid or edema is done at the expense of the plasma volume, which thus aggravates the underlying already diminished volume.

**Metabolic disturbances.** Hypoglycemia can develop and lead to a deterioration in mental status progressing to irreversible brain damage. Oral feedings are usually not possible in these patients due to GI bleeding, coma or placement of nasogastric (NG) tubes. Amino acid solutions are contraindicated as are fat emulsions and alcohols, which may damage the liver further. Solutions containing sodium should be avoided because these patients lack the ability to excrete the ion. Glucose solutions stronger than 10\% may induce an osmotic diuresis and may lead to lactic acidosis.\(^9\) The serum protein is frequently at abnormally low levels.

**Hematological disturbances.** Impaired coagulation and a decline in clotting factors are constant threats, specifically, a prolonged prothrombin time, decreased levels of fibrinogen, factors II, VII, IX and X. (Many coagulation factors require only 30\% of normal liver activity for adequate clotting to occur.)\(^10\) Anemia, disseminated intravascular coagulation (DIC) and thrombocytopenia may also occur. Plasma cholinesterase levels are decreased. There may be bleeding from the pharynx, esophagus, stomach, GI tract, retroperitoneal space and the bronchial tree. Localized areas in the stomach, esophagus and duodenum are the most common sites of bleeding.\(^9\)

**Surgical intervention**

Early attempts to reinfuse the ascitic fluid were not very practical. Establishment of permanent P-V shunts with flow-activated valves were attempted but were unsuccessful due to technical problems. Shunt patency was difficult to maintain.

In 1974, the LeVeen shunt-ascites valve was developed. This device is a pressure activated valve and tubing assembly consisting of a valve body, venous tube unit and intraperitoneal tube. Some authors estimate that more than 5,000 LeVeen shunts have been inserted in patients worldwide since 1974.\(^11\)

In our institution, shunt insertion is performed under general anesthesia, although local may sometimes be used. A liver biopsy is frequently done at the time of insertion. The amount of ascites removed during the insertion procedure has been as little as 100 ml to as much as 15,000 ml.

Insertion involves a small (7-8 cm) incision below the liver edge and anterior to the mid-axillary line. The peritoneum is exposed and a small stab incision is made. The perforated collecting tube is then inserted up to the valve stem and closed with sutures. An incision is then made in the neck for exposure of the internal jugular vein for insertion of the venous end of the tubing. Alligator forceps are used to create a subcutaneous tunnel to carry the tubing to the site of insertion in the internal jugular vein. The superior vena cava may be used as an alternate site of access. Closure and functioning of the shunt is then done with the application of gentle pressure to check for fluid leaks and air.\(^12\)

**Anesthetic management**

Anesthetic management of these patients must include a multisystem approach. Preoperative assessment of the patient may be difficult due to patient cooperation and reliability. Laboratory data may not present a complete picture of the physiological changes involved.

Specifically, these patients are poor anesthetic risks. The degree of monitoring, both invasive and non-invasive, will depend on the severity of the disease. Monitoring adjuncts may include intrarterial lines for blood pressure and blood gases, CVP or Swan-Ganz\(^*\) lines for assessment of cardiac output and fluid replacement/status. Ultimately, the level of monitoring will depend on: (1) the severity of the disease; (2) the extent of the procedure; (3) available equipment in the institution; and (4) whether general or local anesthesia is to be used.

Metabolism of anesthetic agents is altered in liver failure from cirrhosis (e.g., the half-life of phenobarbital increases 50\% whereas the half-life of lidocaine increases 300\%). There is also a great degree of individual variability in the extent to which a drug's metabolism will be affected by a given amount of liver dysfunction.\(^18\)
If local anesthesia is used, patient cooperation is essential. Judicious use of sedation and the amount of local anesthetics, due to prolongation of their half-lives, is essential. Monitoring will depend on the previously mentioned criteria.

If general anesthesia is selected, a rapid sequence induction or awake intubation is essential to avoid aspiration. A combination of a "balanced" technique supplemented with inhalational agents works best in our experience. Stoelting states: "In addition to altered responses to thiobarbituates and neuromuscular blockers, it is a clinical impression that inhalational anesthetic requirements are increased in patients who chronically abuse ethanol. This impression is supported by the observation that mice exposed to alcohol manifest increased isoflurane requirements. In man, halothane MAC in alcoholic patients was 1.0% compared with .75% in normal patients."10

These patients can have dangerously low levels of serum albumin. Albumin is largely responsible for the protein binding of many drugs. By reducing its concentration in hepatic failure, the effect of a given dose of protein-bound drugs may be increased.13

Intraoperative management may include starting these patients on 2.5-5cm H₂O of positive end expiratory pressure (PEEP). Since sodium retention and a low serum albumin are usually present, intravenous albumin in a 5% solution, in addition to glucose, is recommended. Whole blood or packed cells are used to combat anemia.

Because too rapid removal of ascitic fluid can cause hypotension, slow removal of ascitic fluid is recommended. Low dose dopamine (5mcg/kg) will not only improve blood pressure but will also assist in maintaining renal perfusion and urinary output. Excess bleeding from the site of the liver biopsy, if also performed, can usually be controlled by direct pressure for 5 min by the surgeon.

Controversy exists as to the best anesthetic technique for these patients. Postoperative liver dysfunction is likely to be exaggerated in any patient with chronic liver dysfunction regardless of the anesthetic agent administered.

Conclusion

Ascites is a frequently encountered sequela of severe hepatic disease. The LeVeen shunt, a peritoneovenous conduit, is a surgical means of overcoming this serious problem. In order to plan and carry out anesthetic management of these patients, it is necessary to understand the major physiological derangements of hepatic disease and their impact on anesthetic technique.

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