Chapter

28 RENAL, LIVER, AND BILIARY TRACT DISEASE

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RENAL DISEASE

Normal renal function is important for the excretion of anesthetics and medications, maintaining fluid and acid-base balance, and regulating hemoglobin levels in the perioperative period.

Renal disease is quite prevalent in patients presenting for surgery and is associated with increased likelihood of poor postoperative outcomes. Even mild renal dysfunction is associated with a more likely risk of postoperative complications.[1]

Multiple preoperative risk factors have also been identified that predict renal dysfunction in the postoperative period (Box 28.1).[2,3]

Renal Blood Flow

Although the kidneys represent only 0.5% of total body weight, their blood flow is equivalent to about 20% of cardiac output. Approximately two thirds of renal blood flow is distributed to the renal cortex. Renal blood flow and the glomerular filtration rate (GFR) remain relatively constant at renal arterial blood pressures in the range of 80 to 180 mm Hg (Fig. 28.1). Being able to maintain renal blood flow at a constant rate independent of changes in perfusion pressure is known as autoregulation. It is achieved by adjustment of afferent arteriolar tone, which alters the resistance to blood flow. Autoregulation protects the glomerular capillaries from hypertension during acute hypertensive episodes and maintains GFR and renal tubule function during modest decreases in arterial blood pressure. When mean arterial blood pressure is outside the autoregulatory range, renal blood flow becomes pressure dependent. Autoregulation is reset by chronic hypertension and may be abolished in the diabetic kidney.

Renal blood flow is also strongly influenced by the activity of the sympathetic nervous system and by release of renin and other hormones. Sympathetic nervous

Box 28.1 Predictors of Postoperative Acute Kidney Injury

- Preexisting chronic kidney disease
- Advanced age
- Emergent surgery
- Liver disease
- High-risk surgery
- Body mass index > 32
- Peripheral vascular occlusive disease
- Chronic obstructive pulmonary disease

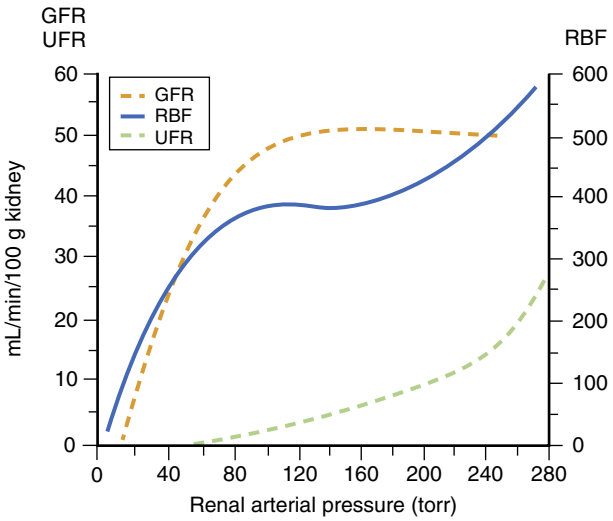

Fig. 28.1 Autoregulation of renal blood flow (RBF) and the glomerular filtration rate (GFR). The relationships between RBF, GFR, and urine flow rate (UFR) and mean renal arterial pressure in dogs are shown as renal arterial pressure is varied from 20 to 280 mm Hg. Autoregulation of RBF and GFR is observed between about 80 mm Hg and 180 mm Hg. (Redrawn from Hemmings HC. Anesthetics, adjuvants and drugs and the kidney. In Malhotra V, ed. *Anesthesia for Renal and Genitourinary Surgery.* New York: McGraw-Hill; 1996:18.)

system stimulation can produce renal vasoconstriction and a marked decrease in renal blood flow even if systemic blood pressure is within the autoregulatory range. Any decrease in renal blood flow will initiate the release of renin, which can further decrease renal blood flow.

Glomerular Filtration Rate

GFR reflects glomerular function and is a measure of the ability of the glomerular membrane to allow filtration. About 90% of the fluid filtered at the glomeruli is reabsorbed from renal tubules into peritubular capillaries and thus returned to the circulation (Fig. 28.2). Normal GFR is approximately 125 mL/min and is very dependent on glomerular filtration pressure (GFP). GFP, in turn, is a function of renal artery pressure, afferent and efferent arteriolar tone, and glomerular oncotic pressure. Hydrostatic pressure within the glomerular capillaries is about 50 mm Hg. This pressure forces water and other low-molecular-weight substances such as electrolytes through the glomerular capillaries into Bowman space. Plasma oncotic pressure is about 25 mm Hg at the afferent arteriole and with filtration increases to about 35 mm Hg at the efferent arteriole. Despite a relatively low net filtration pressure, the glomerular capillaries are able to filter plasma at a rate equivalent to about 125 mL/min. GFR is reduced by significantly decreased mean arterial pressure or renal blood flow. Afferent arteriolar constriction decreases GFR by decreasing glomerular flow. Conversely, afferent arteriolar dilation and mild efferent vasoconstriction increase GFP and GFR.

Humoral Mediators of Renal Function

Renin-Angiotensin-Aldosterone System

Renin is a proteolytic enzyme secreted by the juxtaglomerular apparatus of the kidneys in response to (1) sympathetic nervous system stimulation, (2) decreased renal perfusion pressure, and (3) decreases in the delivery of sodium to the distal convoluted renal tubules. Renin acts on angiotensinogen (a circulating globulin in plasma) to form angiotensin I. Angiotensin I is converted in the lungs by angiotensin-converting enzyme to angiotensin II. Angiotensin II, a potent vasoconstrictor, stimulates the release of aldosterone from the adrenal cortex. It selectively increases efferent renal arteriolar tone at low levels and causes afferent arteriolar constriction at higher levels. Aldosterone, in turn, stimulates reabsorption of sodium and water in the distal tubule and collecting ducts.

Prostaglandins

Prostaglandins are produced in the renal medulla via the enzymes phospholipase A_2 and cyclooxygenase and released in response to sympathetic nervous system stimulation, hypotension, and increased levels of angiotensin II. During periods of hemodynamic instability, prostaglandins act to modulate the effects of arginine vasopressin (AVP), the renin-angiotensin system, and norepinephrine by vasodilating juxtamedullary vessels and maintaining cortical blood flow.

Arginine Vasopressin

Previously known as antidiuretic hormone, AVP regulates osmolality and diuresis. Although secreted in the supraoptic and paraventricular nuclei in the hypothalamus, it exerts significant effects on the renal collecting system. AVP actions are concentrated on collecting duct V_2 receptors to increase membrane permeability and facilitate water reabsorption. The overall effect of AVP is to decrease serum osmolality and increase urine osmolality.

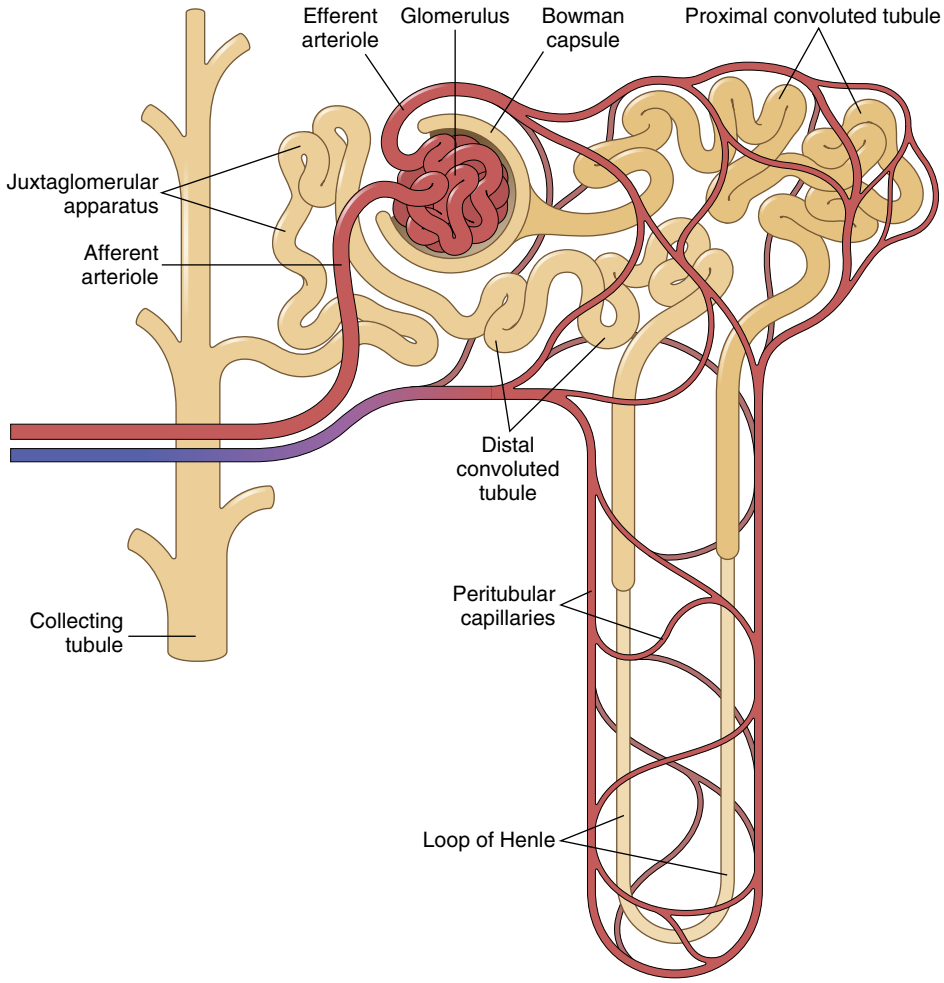

Fig. 28.2 Anatomy of a nephron. The glomerulus is formed by the invaginated and blind end of the nephron known as *Bowman capsule*. Hydrostatic pressure in these capillaries causes water and low-molecular-weight substances to filter through the glomerulus. Glomerular filtrate travels along the renal tubule (proximal convoluted tubule, loop of Henle, distal convoluted tubule), during which most of its water and various amounts of solutes are reabsorbed from the renal tubular lumen into peritubular capillaries. The remaining glomerular filtrate becomes urine.

Atrial Natriuretic Peptide

Atrial natriuretic peptide (ANP) is secreted when stretch receptors in the atria of the heart, and other organs, are stimulated by increased intravascular volume. ANP acts by relaxing vascular smooth muscle to cause vasodilation, inhibiting the renin-angiotensin system, and stimulating diuresis and natriuresis. The net effect of ANP is to decrease systemic blood pressure and intravascular volume.

Drug Clearance

Excretion of drugs or their metabolites into urine depends on three mechanisms: (1) glomerular filtration, (2) active secretion by the renal tubules, and (3) passive reabsorption by the tubules. The glomerular filtration of small molecules characteristic of anesthetic drugs depends on the GFR and the fractional plasma protein binding. Drugs that are highly protein bound will be inefficiently filtered at the glomerulus. Nonionized acidic and basic compounds undergo passive reabsorption by backdiffusion in the proximal and distal renal tubules. Ionized forms of these weak acids and bases, on the other hand, are trapped within renal tubules, accounting for increased renal elimination by either alkalinization or acidification of urine. Conjugation of drugs in the liver to water-soluble metabolites is another mechanism by which renal excretion of substances is achieved.

Renal Function Tests

Renal function can be evaluated preoperatively by using several laboratory tests (Table 28.1). These tests are not sensitive measurements, and significant renal disease (more than a 50% decrease in renal function) can exist while laboratory values remain normal. Furthermore, the normal values established in healthy individuals may not be adjusted for age or applicable during anesthesia. Trends are more useful for evaluating renal function than a single laboratory measurement.

Serum Creatinine

Serum creatinine concentration, which reflects the balance between creatinine production by muscle and its renal excretion, is often used as a marker of GFR. In contrast to blood urea nitrogen (BUN) concentration, serum creatinine level is not influenced by protein metabolism or the rate of fluid flow through renal tubules. It is, however, influenced by skeletal muscle mass. Furthermore, increases in serum creatinine are not typically noted until GFR has declined by at least 50%. Thus, increased creatinine level may serve as a late marker of renal injury. For example, elderly patients, with known decreases in GFR, frequently display normal serum creatinine concentrations because of decreased creatinine production as a consequence of the decrease in skeletal muscle mass. Indeed, mild increases in the serum creatinine concentration in elderly patients may suggest significant renal disease. Likewise, in patients with chronic renal failure, serum creatinine concentrations may not accurately reflect the GFR because of (1) decreased creatinine production, (2) the presence of decreased skeletal muscle mass, or (3) nonrenal (gastrointestinal tract) excretion of creatinine. GFR can be estimated from serum creatinine by a variety of methods including the following formula:

$$\text{GFR} = (140 - \text{age}) \times \text{weight (in kg)} / (\text{serum creatinine} \times 72)$$

Table 28.1 Tests Used for Evaluation of Renal Function

Test	Normal Value	Factors That Influence Interpretation
Test of Glomerular Filtration		
Blood urea nitrogen	8-20 mg/dL	Dehydration Variable protein intake Gastrointestinal bleeding Catabolism
Serum creatinine	0.5-1.2 mg/dL	Age Skeletal muscle mass Catabolism
Creatinine clearance	120 mL/min	Accurate urine volume measurement
Tests of Tubular Function		
Urine specific gravity	1.003-1.030	All are affected by dehydration, solutes, filtrates, proteins, diuretics, dehydration, drugs, and extremes of age
Urine osmolality	350-500 mOsm	
Urine sodium	20-40 mEq	

Blood Urea Nitrogen

BUN concentrations, which are normally 10 to 20 mg/dL, vary with changes in GFR. The relationship between serum creatinine and BUN levels is particularly useful in diagnosing the cause of renal failure. Like serum creatinine, increases in BUN level are frequently a late sign of renal injury and are affected by dietary intake, coexisting illnesses, and intravascular fluid volume. For example, high-protein diets or gastrointestinal bleeding can increase the production of urea and thereby result in increased BUN concentrations (azotemia) despite a normal GFR. Other causes of increased BUN concentrations in the presence of normal GFR are increased catabolism during febrile illnesses and dehydration. Conversely, BUN concentrations can remain normal in the presence of low-protein diets despite decreases in GFR.

Increased BUN concentrations relative to serum creatinine in the presence of dehydration likely reflect increased urea absorption due to decreased urinary flow through the renal tubules, which results in a BUN-creatinine ratio more than 20. Although BUN concentration is susceptible to multiple extraneous influences, values more than 50 mg/dL inevitably reflect a decreased GFR.

Creatinine Clearance

Creatinine clearance (normal, 110 to 150 mL/min) is a measurement of the ability of the glomeruli to excrete creatinine into urine for a given serum creatinine concentration. Because clearance does not depend on corrections for age or the presence of a steady state, measurement of GFR is more reliable than the serum BUN or creatinine values. The principal disadvantage of this test, however, is the need for timed (2 hours may be as acceptable as 24 hours) urine collections. Creatinine clearance (CrCl) and by proxy GFR can be calculated from the formula

$$\text{GFR} = \text{CrCl} = \text{Ucr} \times \text{V}/\text{Pcr}$$

where Ucr is urine creatinine, Pcr is plasma creatinine drawn at midpoint of the timed collection, and V is urinary flow rate.

Proteinuria

Small amounts of protein are normally filtered through glomerular capillaries and then reabsorbed in the proximal convoluted tubules. Proteinuria (excretion of more than 150 mg of protein per day) is most likely due to

abnormally high filtration rather than impaired reabsorption by the renal tubules. Intermittent proteinuria occasionally occurs in healthy individuals when standing and disappears when supine. Other nonrenal causes of proteinuria include exercise, fever, and congestive heart failure.

Urine Indices

Measurement of urine osmolality and urinary sodium and calculation of the fractional excretion of sodium can help differentiate between prerenal and renal tubular causes of azotemia.

Newer Tests of Renal Function

Several new markers of renal function have recently been identified. Serum cystatin C, a ubiquitous protein that is exclusively excreted by glomerular filtration, is less influenced by variations in muscle mass and nutrition than creatinine. It may better predict risk of death and end-stage renal disease (ESRD) across diverse populations.[4]

Other biomarkers such as *N*-acetyl-β-D-glucosaminidase, kidney injury molecule-1, and interleukin 18 are promising in the early detection of kidney injury. These biomarkers may have a role in the future in reducing morbidity and mortality rates associated with kidney injury in the perioperative setting.[5]

Pharmacology of Diuretics

Thiazide Diuretics

Thiazide diuretics (hydrochlorothiazide, chlorthalidone) are generally administered for the treatment of essential hypertension and for mobilization of the edema fluid that is associated with renal, hepatic, or cardiac dysfunction. Diuresis occurs as a result of the inhibition of reabsorption of sodium and chloride ions from the early distal renal tubules. Side effects associated with diuretic-induced hypokalemia may include (1) skeletal muscle weakness, (2) increased risk for digitalis toxicity, and (3) enhancement of nondepolarizing neuromuscular blocking drugs (Table 28.2).

Table 28.2 Side Effects of Diuretics

Diuretic Class	Hypokalemic, Hypochloremic Metabolic Alkalosis	Hyperkalemia	Hyperglycemia
Thiazide diuretics	Yes	No	Yes
Loop diuretics	Yes	No	Minimal
Osmotic diuretics	No	No	No
Aldosterone antagonists	No	Yes	No

Loop Diuretics

Loop diuretics (ethacrynic acid, furosemide, bumetanide) inhibit the reabsorption of sodium and chloride and augment the secretion of potassium, primarily in the loop of Henle. Intravenous administration of these drugs produces a diuretic response within minutes. Chronic administration of loop diuretics may result in hypochloremic, hypokalemic metabolic alkalosis and, in rare instances, deafness.[6]

Osmotic Diuretics

The most frequently administered osmotic diuretic is the six-carbon sugar mannitol. Mannitol produces diuresis because it is filtered by the glomeruli and not reabsorbed within the renal tubules. This leads to increased osmolarity of the renal tubule fluid and associated excretion of water.

Mannitol increases fluid movement from intracellular spaces into extracellular spaces such that intravascular fluid volume expands acutely. This redistribution of fluid from intracellular to extracellular compartments decreases brain size and intracranial pressure (also see Chapter 30). Mannitol may further diminish intracranial pressure by decreasing the rate of cerebrospinal fluid formation.

Aldosterone Antagonists

Spironolactone blocks the renal tubular effects of aldosterone and offsets the loss of potassium from administration of thiazide diuretics. Ascites and peripheral edema secondary to cirrhosis of the liver is often treated with spironolactone. The most serious toxic effect of spironolactone is hyperkalemia. Serum potassium concentration should be monitored closely in patients taking spironolactone.

Dopamine and Fenoldopam

Dopamine dilates renal arterioles via its agonist action at the DA1 receptor, leading to increased renal blood flow and GFR. Treatment with low-dose dopamine (0.5 to 3 μg/kg/min) may increase urine output but yet not alter the course of renal failure. In addition, dose-dependent side effects of dopamine include tachydysrhythmias, pulmonary shunting, and tissue ischemia (gastrointestinal tract, digits).[7,8]

Fenoldopam, a dopamine analog, also possesses DA1 agonist activity but lacks the adrenergic activity of dopamine. It also increases renal blood flow and GFR, which may help the treatment of acute kidney injury. Yet its role in the treatment of renal failure is unclear. It is currently approved for short-term parenteral treatment of severe hypertension.[9]

Pathophysiology of End-Stage Renal Disease

ESRD causes profound physiologic changes that affect several organs (Box 28.2 and Table 28.3).

Box 28.2 Changes Characteristic of Chronic Renal Disease

- Anemia
- Decreased ejection fraction
- Decreased platelet adhesiveness
- Hyperkalemia
- Unpredictable intravascular fluid volume
- Metabolic acidosis
- Systemic hypertension
- Pericardial effusion
- Decreased sympathetic nervous system activity

Table 28.3 Stages of Chronic Renal Failure

Stage	Glomerular Filtration Rate (mL/min/1.73 m^2)
1	>90
2	60-89
3	30-59
4	15-29
5	<15

Cardiovascular Disease

Cardiovascular disease is the predominant cause of death in patients with ESRD. Acute myocardial infarction, cardiac arrest of unknown cause, cardiac dysrhythmias, and cardiomyopathy account for more than 50% of deaths in patients receiving dialysis. Hypertension commonly exists in patients with ESRD. This systemic hypertension can be severe and refractory to antihypertensive therapy. Hypervolemia and excess activation of the renin-angiotensin-aldosterone system are the most common causes.

Additionally, the accumulation of uremic toxins and metabolic acids may contribute to poor myocardial performance. Yet, the presence of ESRD with significantly depressed cardiac function does not necessarily contraindicate renal transplantation because cardiac ventricular function often improves after transplantation.

Uremia causes changes in lipid metabolism that lead to increased concentrations of serum triglycerides and reduced levels of protective high-density lipoproteins. Thus, ESRD accelerates the progression of atherosclerosis. Pericardial disease and cardiac dysrhythmias can also be encountered in patients with ESRD. Pericardial effusions typically resolve when patients are adequately dialyzed.

Metabolic Disease

Many patients with ESRD also have diabetes mellitus. Kidney failure as a result of diabetes develops in nearly 30% to 40% of patients with ESRD and accounts for 30% of those on the waiting list for kidney transplantation. In fact, nephropathy develops in nearly 60% of insulin-dependent diabetic patients. Patients with ESRD and diabetes have a more likely risk of cardiovascular problems than do patients with renal failure alone.[10]

Once patients are unable to excrete their dietary fluid and electrolyte loads, abnormalities in plasma electrolyte concentrations (sodium, potassium, calcium, magnesium, and phosphate) can develop. The most life-threatening electrolyte abnormality is hyperkalemia.

Anemia and Abnormal Coagulation

Patients with renal failure generally display a normochromic, normocytic anemia because of decreased erythropoiesis and retained toxins that are secondary to renal failure. Treatment with recombinant erythropoietin can frequently increase hemoglobin concentrations. Symptoms of fatigue are reduced and both cerebral and cardiac function are improved. Occasionally, recombinant erythropoietin therapy may exacerbate preexisting essential hypertension. Patients with renal failure may also display uremia-induced defects in platelet function.

Management of Anesthesia in Patients With End-Stage Renal Disease

General anesthesia with tracheal intubation provides acceptable hemodynamics, excellent skeletal muscle relaxation, and a predictable depth of anesthesia in patients with ESRD who are undergoing major operations. Patients with advanced stages of comorbid conditions may require more extensive monitoring, such as continuous monitoring of systemic blood pressure and perhaps central venous pressure. Large variations in arterial blood pressure may occur with hypotension being more likely than hypertension during maintenance of anesthesia. This is especially the case if the patient has recently been hemodialyzed in preparation for the surgical procedure. Those with the most severe comorbid conditions, such as symptomatic coronary artery disease or a history of congestive heart failure, may benefit from monitoring with a pulmonary artery catheter or transesophageal echocardiography (TEE).

The status of hemodialysis shunts or fistulas should be monitored and documented (e.g., presence of a palpable thrill) during positioning and intraoperatively to confirm continued patency. Peripheral lines and arterial blood pressure monitoring cuffs should not be placed in proximity to such implanted vascular access devices.

Normal saline (NS) is often given instead of lactated Ringer solution for intravascular fluid resuscitation in patients with ESRD. The rationale is the hypothesized risk of hyperkalemia from potassium contained in lactated Ringer solution. Yet this conclusion has not been proved to be likely. For example, a prospective randomized double-blind clinical trial comparing the two intraoperative fluid therapies in ESRD patients undergoing renal transplantation has shown more hyperkalemia and greater degree of acidosis with NS than lactated Ringer solution.[11]

Patients with uremia and other comorbid conditions (e.g., diabetes mellitus) are at an increased risk for aspiration of gastric contents during induction of anesthesia. The use of a rapid-sequence induction of anesthesia technique may be indicated in such patients. Succinylcholine is not contraindicated in patients with ESRD. The increase in serum potassium concentration after a large dose of succinylcholine is approximately 0.6 mEq/L for patients both with and without ESRD. This increase can be tolerated without imposing a significant cardiac risk, even if initial (i.e., preanesthetic) serum potassium concentration is more than 5 mEq/L.

Several strategies have achieved adequate heart rate and arterial blood pressure control during induction of anesthesia. Moderate to large doses of opioids, such as fentanyl, can blunt the response to laryngoscopy. However, systemic blood pressure is frequently more difficult to maintain after induction of anesthesia, and hypotension may require treatment with vasoconstrictors. The short-acting β-adrenergic blocker esmolol can blunt the hemodynamic response to tracheal intubation and is ideally suited for patients with an adequate ejection fraction.

Drugs or their metabolites that depend on renal elimination (pancuronium, vecuronium, morphine, meperidine) should be used cautiously or avoided. Cisatracurium is a good choice as most of it is metabolized by spontaneous Hoffman degradation, which makes its duration of action independent of liver or kidney function. The elimination half-life of rocuronium is increased because of increased volume of distribution with no change in clearance. Mivacurium is metabolized by plasma cholinesterase but its action may be prolonged by 10 to 15 minutes as a result of reduced cholinesterase activity in these patients (also see Chapter 11). Because morphine has long-acting renally excreted metabolites such as morphine-6-glucuronide, alternative opioid choices are preferred (e.g., fentanyl, sufentanil, alfentanil, remifentanil).

Appropriate choices of inhaled anesthetics include desflurane, isoflurane, and sevoflurane. The metabolism of sevoflurane to inorganic fluoride has been implicated in experimental studies of renal toxicity, although no controlled human studies are available to indicate either safety concerns or danger when using sevoflurane in the setting of ESRD.

Differential Diagnosis and Management of Perioperative Oliguria

Prerenal Oliguria

Prerenal oliguria is characterized by the excretion of concentrated urine that contains minimal amounts of sodium (Table 28.4). Excretion of highly concentrated and sodium-poor urine confirms that renal tubular function is intact and reflects an attempt by the kidneys to conserve sodium and restore intravascular fluid volume in response to decreased renal blood flow. The decreased renal blood flow most likely reflects an acute decrease in intravascular fluid volume or decreased cardiac output. Other causes of decreased renal blood flow are sepsis, liver failure, and congestive heart failure.[11]

Table 28.4 Oliguria Versus Acute Tubular Necrosis: Preoperative Differential Diagnosis

Diagnostic Feature	Prerenal Oliguria	Acute Tubular Necrosis
Fractional excretion of sodium	<1%	>3%
Urine specific gravity	>1.015	1.01-1.015
Urine sodium (mEq/L)	<40	>40
Urine osmolality (mOsm/L)	>400	<400
Causes	Decreased renal blood flow (hypotension, hypovolemia, decreased cardiac output)	Renal ischemia, nephrotoxins, free hemoglobin or myoglobin

The initial management of patients with perioperative oliguria is influenced by their risk for the development of acute renal failure. A brisk diuresis in response to an intravascular fluid challenge suggests that an acute decrease in intravascular fluid volume is the cause of the prerenal oliguria. When intravascular fluid replacement does not result in increased urine output, intrinsic renal disease or hemodynamic causes should be considered. Prompt recognition and treatment of prerenal oliguria is critical as prolonged severe ischemia can lead to necrosis of renal tubules and convert reversible injury to irreversible intrarenal disease.

Administration of diuretics to maintain or stimulate urine flow in the perioperative period is controversial. One theory is that prevention of renal tubule urine stasis with diuretics can prevent prerenal oliguria from progressing to acute tubular necrosis. Nevertheless, urine output that is enhanced by the administration of a diuretic does not necessarily predict postoperative renal function. There is no evidence that drug-induced diuresis (dopamine, furosemide, mannitol) in the presence of reduced cardiac output or hypovolemia, or both, protects renal function. In fact, a recent meta-analysis of clinical trials did not find any interventions (e.g., diuretics, dopamine and its analogs, calcium channel blockers, angiotensin-converting enzyme inhibitors, specific hydration fluids, *N*-acetylcysteine, ANP, or erythropoietin) that can reduce the risk of perioperative renal failure.[12]

Table 28.5 Liver Function Tests With Normal Values

Test	Normal Values[a]
Albumin	3.5-5.5 g/dL
Bilirubin	0.3-1.1 mg/dL
Unconjugated bilirubin (indirect reacting)	0.2-0.7 mg/dL
Conjugated bilirubin (direct reacting)	0.1-0.4 mg/dL
Aspartate aminotransferase (i.e., SGOT)	10-40 U/mL
Alanine aminotransferase (i.e., SGPT)	5-35 U/mL
Alkaline phosphatase	10-30 U/mL
Prothrombin time	12-14 s

[a]Normal values for each individual laboratory should be considered in interpreting liver function test results.
SGOT, Serum glutamic-oxaloacetic (acid) transaminase; *SGPT,* serum glumate-pyruvate transaminase.

Intrinsic Renal Disease

Acute tubular necrosis, glomerulonephritis, and acute interstitial nephritis are intrinsic renal causes of oliguria. In contrast to oliguria secondary to hypovolemia, the urine of patients with acute tubular necrosis is poorly concentrated and contains excessive amounts of sodium (Table 28.5). Intrinsic renal disease is the most severe of the different forms of oliguria and is typically the hardest to reverse.

Postrenal Oliguria

An obstruction that is distal to the renal collecting system usually involves a mechanical problem such as a blood clot in the ureter, bladder, or urethra. Surgical ligation, renal calculi, and edema are other postrenal causes of low urine output. Another common postrenal cause is bladder catheter obstruction. Postrenal oliguria is frequently reversible once the source of the obstruction is removed.[13]

LIVER DISEASE

The liver is responsible for the production of essential plasma proteins, the metabolism and detoxification of drugs and deleterious xenobiotics, the absorption of critical nutrients, and carbohydrate metabolism. Impaired liver function affects nearly every organ system in the body.

Hepatic Blood Flow

The liver is unique in that it receives a dual afferent blood supply that is equal to about 25% of cardiac output (Fig. 28.3). Approximately 70% of hepatic blood flow is supplied by the portal vein with the remainder supplied by the hepatic artery. Under normal conditions, each blood vessel contributes roughly 50% to the liver's oxygen supply. Portal vein flow is not regulated and is susceptible to systemic hypotension and decreases in cardiac output.

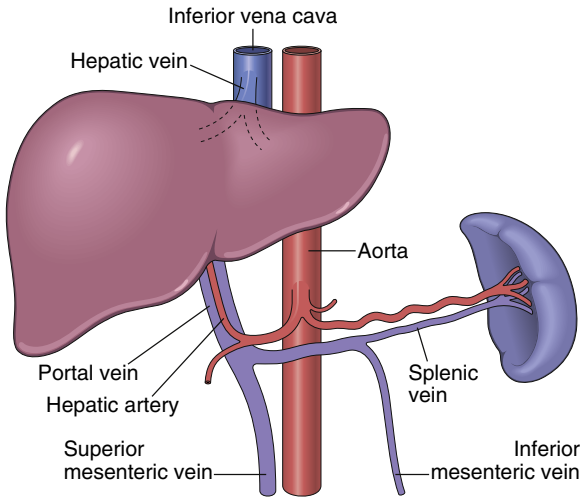

Fig. 28.3 Schematic depiction of the dual afferent blood supply to the liver provided by the portal vein and hepatic artery. About 70% of hepatic blood flow is via the portal vein, with the remainder via the hepatic artery. Total hepatic blood flow is directly proportional to perfusion pressure across the liver and inversely related to splanchnic vascular resistance. Cirrhosis of the liver increases resistance to blood flow through the portal vein and decreases hepatic blood flow.

Intrinsic Determinants of Hepatic Blood Flow

Reduction in portal flow (up to a 50% reduction) is compensated by modulating hepatic artery tone to maintain perfusion to the liver. This is primarily mediated via the hepatic arterial buffer response, which reciprocally varies hepatic arterial blood flow to changes in portal flow mediated by adenosine. The response is stimulated by low pH and O_2 content and increased P_{CO_2}. Volatile anesthetics and cirrhosis of the liver attenuate this reciprocal relationship and render the liver vulnerable to ischemia.

Extrinsic Determinants of Hepatic Blood Flow

Hepatic perfusion pressure (mean arterial or portal vein pressure minus hepatic vein pressure) and splanchnic vascular resistance determine hepatic blood flow. The splanchnic vessels receive vasomotor innervation from the sympathetic nervous system. Splanchnic nerve stimulation (pain, arterial hypoxemia, surgical stress) increases splanchnic vascular resistance and decreases hepatic blood flow.

Surgical stimulation and the proximity of the operative site to the liver are important determinants of the magnitude of the decrease in hepatic blood flow seen

during general anesthesia. β-Adrenergic receptor blockers such as propranolol are associated with decreases in hepatic blood flow. Positive-pressure ventilation of the lungs, congestive heart failure, and administration of excessive intravascular fluid cause increased central venous pressure, resulting in increased hepatic venous pressure, which effectively decreases hepatic perfusion pressure and blood flow.

Glucose Homeostasis

The liver is the main organ for the storage and release of glucose. Hepatocytes extract glucose via an insulin-mediated mechanism, where it can be stored as glycogen. Glucagon-mediated catabolism of glycogen (glycogenolysis) releases glucose back into the systemic circulation for maintenance of euglycemia. Surgical stress, starvation, and sympathetic nervous system activation stimulate glycogen depolymerization to glucose. When glycogen stores are depleted hepatic gluconeogenesis from substrates such as lactate, glycerol, and certain amino acids restores blood glucose levels.

Coagulation

Hepatocytes are responsible for the synthesis of the majority of procoagulant proteins as well as regulators such as proteins C and S and antithrombin III. An important exception to this is factor VIII, which is partially produced in endothelial cells. Vitamin K, which is absorbed by bile secretion into the gastrointestinal tract, plays an important role in catalysis of some of the procoagulant proteins to produce factors II, VII, IX, and X. Laboratory studies such as prothrombin time (international normalized ratio [INR]), partial thromboplastin time (PTT), and fibrinogen levels can be used to evaluate impaired coagulation and hepatic function. Impaired laboratory studies reflect significant hepatic dysfunction because most coagulation factors maintain function at up to 20% to 30% of their normal levels.

Drug Metabolism

Hepatic drug metabolism is characterized by the conversion of lipid-soluble drugs to more water-soluble forms to facilitate renal excretion, transformation to pharmacologically less active substances, and excretion in bile.

Three major pathways are utilized to accomplish these goals. Phase 1 metabolism involves an increase in polarity of drugs via cytochrome P and mixed function oxidases. Phase 2 metabolism involves conjugation of metabolites to water-soluble substrates. Phase 3 elimination relies on energy-dependent excretion of drugs into bile. Chronic liver disease may interfere with the metabolism of drugs because of the decreased number of enzyme-containing hepatocytes or the decreased hepatic blood flow that typically accompanies cirrhosis of the liver. Prolonged elimination half-times for morphine, alfentanil, diazepam, lidocaine, pancuronium, and vecuronium occur in patients with cirrhosis of the liver. Likewise, chronic drug therapy can inhibit hepatic enzymes and inhibit metabolism of anesthetic drugs leading to higher circulating blood levels. Conversely, enzyme induction, particularly of cytochrome P isoforms, can also occur as a response to chronic therapy with drugs such as phenytoin, isoniazid, and rifampin or as a result of alcohol abuse. Induction of hepatic enzymes can increase metabolism of administered anesthetic and therapeutic drugs, thereby reducing plasma levels.

Heme Metabolism

Although fetal erythrocyte production occurs exclusively in the liver, hepatic hematopoiesis accounts for only 20% of adult heme synthesis with the remainder produced in the bone marrow. Heme synthesis occurs from glycine and succinyl coenzyme A (CoA) through a reaction catalyzed by aminolevulinic acid (ALA) synthase. ALA synthase is the rate-limiting step in the heme synthesis pathway and is regulated by feedback inhibition by its end product heme. Porphyrias are rare genetic diseases characterized by interruption of feedback inhibition of ALA synthase.

Heme degradation, primarily by the reticuloendothelial system, results in formation of bilirubin as an end product. Formed bilirubin is then bound to plasma albumin for transport to the liver, where it is extracted and conjugated for secretion into canalicular bile. The majority of bilirubin excretion occurs in the gut, although a small portion is recirculated to the liver via the enterohepatic circulation. This accounts for the small amount of bilirubin conjugates present in blood. Conjugated bilirubin is water soluble and about 10% is excreted in the urine.

Cholesterol and Lipid Metabolism

The liver stores dietary fat as triglycerides, cholesterol, and phospholipids and releases free fatty acids via triglyceride hydrolysis. In addition, the liver synthesizes free fatty acids from glucose, lipids, and protein. The liver also plays an important role in regulation of cholesterol uptake, metabolism, and transport. Bile salts, the end product of cholesterol synthesis, serve as regulators of lipid metabolism. Elimination of cholesterol is achieved by biliary secretion and by excretion of bile acids.

Protein Metabolism

The liver plays a vital role in protein metabolism. Numerous biologically active proteins including albumin, cytokines, hormones, and coagulation factors are manufactured in the liver. In addition, nonessential amino acid synthesis can also occur in hepatocytes when necessary.

Protein degradation is another important function of the liver. The urea (Krebs) cycle is utilized by hepatocytes to convert the end products of amino acid degradation, such as ammonia and other nitrogenous waste products, to urea, which is readily excreted by the kidney. Severe hepatic dysfunction, such as that which occurs in end-stage liver disease (ESLD), leads to accumulation of ammonia in the serum resulting in hepatic encephalopathy (HE).

Pathophysiology of End-Stage Liver Disease

Cardiovascular Complications

Severe parenchymal disease that has advanced to the point of cirrhosis usually results in a hyperdynamic circulation. Hemodynamic measurements generally reveal normal to low systemic blood pressure, increased cardiac output, and decreased systemic vascular resistance. Decreased systemic vascular resistance is a result of vasodilation and abnormal anatomic and physiologic shunting. Physiologic shunting is the passage of blood from the arterial to the venous side of the circulation without effectively traversing a capillary bed. Abnormal blood vessels, such as those seen in the skin as spider angiomas, represent an anatomic shunt.[14,15]

Portal Hypertension

High resistance to blood flow through the liver, a hallmark of ESLD, causes an accumulation of blood in the vascular beds that are immediately upstream of the liver. Vessels draining the esophagus, stomach, spleen, and intestines dilate and hypertrophy, which leads to the development of splenomegaly and esophageal, gastric, and intra-abdominal varices. Symptoms of portal hypertension include anorexia, nausea, ascites, esophageal varices, spider nevi, and HE. It is central to the pathogenesis of a variety of complications associated with ESLD including massive hemorrhage, increased susceptibility to infection, renal failure, and mental status changes.

Pulmonary Complications

ESLD is associated with the hepatopulmonary syndrome and portopulmonary hypertension. Hepatopulmonary syndrome develops as a result of intrapulmonary arteriovenous communications that are not ventilated, impairment of hypoxic pulmonary vasoconstriction, atelectasis, and restrictive pulmonary disease secondary to ascites and pleural effusion. Arterial hypoxemia, secondary to the hepatopulmonary syndrome, may improve somewhat with supplemental oxygen in the early stages of the disease, but oxygen may not be effective with disease progression.

Portopulmonary hypertension is an increase in intrapulmonary vascular pressure in patients with portal hypertension. The cause is not well established. This syndrome occurs in less than 5% of patients, including the liver transplant population. Nevertheless, these patients are at increased risk for acute right-sided heart failure if physiologic conditions that increase pulmonary vascular resistance (acidosis, arterial hypoxemia, hypercapnia) occur during anesthesia. Hepatic hydrothorax, defined as pleural effusions occurring in the absence of cardiopulmonary disease, can also occur in up to 10% of cirrhotic patients. In some patients, the pleural effusions from hepatic hydrothorax are large enough to impair oxygenation.

Hepatic Encephalopathy

Altered mental state is a frequent complication of both acute and chronic liver failure with a clinically variable presentation ranging from minor changes in brain function to deep coma. The cause of this complex neuropsychiatric syndrome is multifactorial. The serum concentrations of many chemicals, which are normally filtered by the healthy liver and are present in higher concentrations with hepatic dysfunction, likely play an important role. Ammonia is heavily implicated as a precipitating factor of episodes of HE. Other etiologic factors include disruption of the blood-brain barrier, increased central nervous system inhibitory neurotransmission, and altered cerebral energy metabolism. The reversibility of symptoms of HE with administration of flumazenil supports an important role for the GABA (γ-aminobutyric acid) receptor activation in HE pathogenesis. It is also important to rule out other causes of altered mental status in the patient with liver disease, such as intracranial bleeding or masses, hypoglycemia, or a postictal state. As effective treatments for many of the putative etiologic factors in HE do not yet exist, current treatment still revolves around reducing the production and absorption of the ammonia. Typically, neomycin (to reduce ammonia production by urease-producing bacteria) and the administration of lactulose (to reduce ammonia absorption) are employed.[16]

Impaired Drug Binding

When liver disease is so severe that albumin production is decreased, fewer sites are available for drug binding. This limited availability can increase levels of the unbound, pharmacologically active fraction of drugs, such as thiopental and alfentanil. Increased drug sensitivity as a result of decreased protein binding is most likely to be manifested when plasma albumin concentrations are lower than 2.5 g/dL.

Ascites

Ascites is a common complication of cirrhosis affecting up to 50% of cirrhotic patients. The development of ascites is associated with significant morbidity and heralds the end stages of cirrhosis. Complications associated with ascites include marked abdominal distention (leading to atelectasis and restrictive pulmonary disease),

spontaneous bacterial peritonitis, and circulatory instability due to compression of the inferior vena cava and right atrium. Although the exact mechanism of ascites is unclear, excess sodium retention by the kidney, decreased oncotic pressure due to hypoalbuminemia, and portal hypertension appear to play a central role. Initial therapy includes restriction of fluid administration, reduction of sodium intake, and administration of diuretics. In severe cases, abdominal paracentesis can be effective at transiently reducing abdominal distention and restoring hemodynamic stability.[17,18] Some patients with refractory ascites are candidates for transjugular intrahepatic portosystemic shunt (TIPS), an interventional radiologic procedure to place a stent between a branch of the hepatic vein and portal vein (also see Chapter 38).

Renal Dysfunction and Hepatorenal Syndrome

Renal dysfunction can develop in a significant portion of patients with cirrhosis. A variety of etiologic factors including diuretic therapy, reduced intravascular volume secondary to ascites or gastrointestinal hemorrhage, nephrotoxic drugs, and sepsis can provoke acute renal failure and ultimately acute tubular necrosis in cirrhotic patients.

In the absence of obvious factors precipitating renal failure, the hepatorenal syndrome (HRS) can be diagnosed. HRS is characterized by intense renal vasoconstriction as an end-stage response to decreased effective arterial blood volume. Type 1 HRS, typically presenting as rapidly progressing prerenal failure, is associated with a poor prognosis in the absence of therapeutic intervention. Conversely, type 2 HRS presents with a milder degree of renal dysfunction. Treatment with octreotide, glucagon, and midodrine have shown some promise at reversing type 1 HRS.[19,20]

Effects of Anesthesia and Surgery on the Liver

Impact of Anesthetics on Hepatic Blood Flow

Inhaled anesthetics and regional anesthesia both typically decrease hepatic blood flow 20% to 30% in the absence of surgical stimulation. These changes reflect drug- or technique-induced effects on hepatic perfusion pressure or splanchnic vascular resistance, or both. For example, reduced hepatic blood flow from volatile anesthetics, as well as regional anesthesia (T5 sensory level), is likely due to decreased hepatic perfusion pressure. Autoregulation (increased hepatic artery blood flow offsetting decreases in portal vein blood flow) of hepatic blood flow may be best maintained with isoflurane. However, hepatic blood flow during the administration of desflurane and sevoflurane is maintained by a similar mechanism.

Volatile Anesthetic–Induced Hepatic Dysfunction

A rare, but life-threatening form of hepatic dysfunction may reflect an immune-mediated hepatotoxicity caused by halothane. Two patterns of hepatic injury have occurred with use of halothane. A mild form occurs in up to 20% of patients and is associated with minimal sequelae. A rare fulminant form is associated with a fatality rate of 50% to 70%. Risk factors for development of this condition include prior exposure to halothane, age older than 40 years, obesity, and female gender. Isoflurane and desflurane are also capable of causing hepatic dysfunction, but the incidence of hepatitis after exposure to these volatile anesthetics is extremely rare, mainly because of the decreased magnitude of metabolism in comparison to halothane. Given its rare incidence and the disappearance of halothane in modern clinical practice in North America, volatile anesthetic–induced hepatic dysfunction remains a diagnosis of exclusion in the patient presenting with hepatitis in the perioperative period.[20,21]

Management of Anesthesia in Patients With End-Stage Liver Disease

Preoperative Evaluation of Liver Disease

Liver function tests (Table 28.6) detect the presence of liver disease preoperatively and establish the diagnosis when postoperative liver dysfunction occurs. The Child-Pugh and Model for End-Stage Liver Disease (MELD) scores are two methods of evaluating severity of liver dysfunction (Table 28.7). Patients with Child-Pugh class C liver dysfunction or MELD score greater than 14 have an increased risk for perioperative morbidity and death. Morbidity and mortality rates after elective operations are more frequent in patients with preexisting cirrhosis of the liver than in patients

Table 28.6 Child-Pugh Classification System and MELD Score Formula for Liver Disease

	Child-Pugh Score		
Finding	**A**	**B**	**C**
Serum bilirubin (mg/dL)	<2.0	2.0-3.0	>3.0
Serum albumin (g/dL)	>3.5	2.8-3.5	<2.8
Prothrombin time (seconds prolonged)	1-4 s	4-6 s	>6 s
Ascites	None	Slight	Moderate
Encephalopathy	None	Minimal	Advanced

MELD (Model for End-Stage Liver Disease) Score Formula

MELD score = $(0.957 \times \log_e$ [serum creatinine (mg/dL)] + $0.378 \times \log_e$ [total serum bilirubin (mg/dL)] + $1.120 \times \log_e$ [INR]) $\times 10$
Minimum for all values is 1.
Maximum value for creatinine is 4.

Table 28.7 Classification and Causes of Postoperative Liver Dysfunction

Diagnostic Feature	Prehepatic	Intrahepatic	Posthepatic
Bilirubin	Increased (unconjugated fraction)	Increased (conjugated fraction)	Increased (conjugated fraction)
Aminotransferase enzymes	No change	Markedly increased	Normal to slightly increased
Alkaline phosphatase	No change	No change to slightly increased	Markedly increased
Prothrombin time	No change	Prolonged	No change to prolonged
Albumin	No change	Decreased	No change to decreased
Causes	Hemolysis Hematoma reabsorption Bilirubin overload from whole blood	Viruses Drugs Sepsis Arterial hypoxemia Congestive heart failure Cirrhosis	Stones Cancer Sepsis

undergoing similar operations but in the absence of liver disease.[20,21]

Unfortunately, liver function tests are rarely specific. Postoperative liver dysfunction is more likely in the presence of coexisting liver disease. Furthermore, the large reserve of the liver means that considerable hepatic damage can be present before liver function test results become altered. Indeed, cirrhosis of the liver may cause little alteration in liver function. It may take additional stressors, such as anesthesia and surgery, to reveal the underlying liver disease. Inadequate hepatocyte function during anesthesia and surgery can be manifested as metabolic acidosis intraoperatively.

Intraoperative Management

Most major operations in patients with significant liver disease involve the use of general anesthesia. Regional techniques can be considered in selected patients who have normal coagulation values.

The magnitude of the operation determines the extent of invasive monitoring that is required. Major operations during which blood loss is likely require continuous means of monitoring arterial blood pressure (arterial line) and filling pressure (central venous line). Patients with significant comorbid conditions (including cardiac diseases) undergoing procedures involving large anticipated blood loss may require placement of a pulmonary artery catheter.

Correction of severe coagulopathy before vascular line placement should be considered. Ultrasound guidance may minimize the risk of complications related to vascular access. Communication with the blood bank (also see Chapter 24) before surgery is crucial to ensure adequate availability of red blood cells, platelets, and clotting factors, including fresh frozen plasma and cryoprecipitate. In patients with esophageal varices, the risk of bleeding from insertion of a TEE probe is increased.

Induction and Maintenance of Anesthesia

Most patients have well-preserved cardiac function and no significant systemic or pulmonary hypertension. Induction of anesthesia can be achieved with an intravenous anesthetic such as propofol, thiopental, or etomidate, along with opioids and short- or intermediate-acting neuromuscular blocking drugs. Intravenous anesthetics have minimal impact on hepatic blood flow provided arterial blood pressure is adequately maintained. Thus, arterial blood pressure should be preserved and sympathetic stimulation avoided, which also has an adverse effect on hepatic blood flow. A rapid-sequence or modified rapid-sequence induction of anesthesia is warranted if patients have significant ascites or delayed gastric emptying. Hypotension after induction of anesthesia occurs commonly as a result of the low systemic vascular resistance and relative hypovolemia. This can usually be treated with small doses of vasoconstrictors such as phenylephrine. With the exception of halothane, all volatile anesthetics are suitable for patients with severe liver disease. No optimal anesthetic technique has been established for the maintenance of anesthesia.

Management of Coagulopathy

Traditionally, surgical blood loss and coagulopathy have been managed by administering blood products either by clinical judgment alone, if bleeding is rapid, or guided by conventional laboratory tests (e.g., PTT, INR, platelet count), if bleeding is controlled (also see Chapter 24). Standard laboratory testing, however, can be slow to yield results and does not provide information about the qualitative aspects of clot formation.

Advances in point-of-care coagulation technology, such as rotational thromboelastometry and platelet function analysis, however, allow the clinician to rapidly

diagnose and manage coagulopathy associated with ESLD in the perioperative setting. Additional information, unavailable through conventional laboratory tests, such as clot strength, platelet function, and hyperfibrinolysis, can be assessed rapidly at the bedside with these newer techniques.[22]

The introduction of various factor concentrate therapies, such as prothrombin complex concentrate and fibrinogen concentrate, into clinical practice may also play a significant role in blood product management in patients with ESLD. Hemostatic algorithms that utilize point-of-care coagulation testing and factor concentrate-based therapy are showing considerable promise in the management of patients with coagulopathy related to liver disease.[23]

Postoperative Jaundice

Halothane or other volatile anesthetics are often implicated as the cause of postoperative jaundice, but there are many other and probably more likely causes (see Table 28.7). A surgical cause of postoperative jaundice is likely if the operation involved the liver or biliary tract. Similarly, multiple blood transfusions and resorption of surgical hematoma can lead to jaundice in the perioperative period. Drugs, including antibiotics, and other metabolic or infectious causes such as sepsis, must also be considered in the differential diagnosis of postoperative jaundice.

Management of Anesthesia in Intoxicated Patients

Acutely intoxicated patients require less anesthetic because there is an additive depressant effect between alcohol and anesthetics. Lower minimum alveolar concentration (MAC) levels in the acutely intoxicated patient may also reduce the amount of volatile anesthetic needed to maintain anesthesia. Intoxicated patients are more vulnerable to regurgitation of gastric contents and aspiration pneumonia because alcohol slows gastric emptying and decreases the tone of the lower esophageal sphincter.

Alcohol Withdrawal Syndrome

Initial symptoms of alcohol withdrawal, including agitation, tachycardia, and signs of increased sympathetic stimulation, may be subtle and mistaken for other common perioperative complications such as pain and delirium. However, a history of chronic alcohol use should always prompt consideration of this entity in the differential diagnosis and prophylactic benzodiazepine treatment may be promptly initiated. Manifestations of severe alcohol withdrawal syndrome (delirium tremens) usually appear 48 to 72 hours after cessation of drinking. This syndrome represents a medical emergency. Such patients may manifest tremulousness and hallucinations. There is significantly increased activity of the sympathetic nervous system with subsequent catecholamine release, leading to diaphoresis, hyperpyrexia, cardiac dysrhythmias, and hemodynamic instability. In some patients, grand mal seizures may be the first indication of alcohol withdrawal syndrome. When seizures occur, hypoglycemia and other possible causes, including brain injury, should also be ruled out.

Treatment

Treatment of delirium tremens must be aggressive and typically consists of benzodiazepine administration at regular intervals. A β-antagonist (propranolol or esmolol) can be used to control the heart rate. If mental status declines significantly, airway protection may be achieved by endotracheal intubation. Correction of fluid, electrolyte (magnesium, potassium), and metabolic (thiamine) derangements is important. Despite aggressive treatment, mortality rate from delirium tremens is about 10%. Death is often due to hemodynamic instability, cardiac dysrhythmias, or seizures.[24]

DISEASES OF THE BILIARY TRACT

Gallstones are reported to be present in 10% of men and 20% of women between 55 and 65 years of age. These patients usually have normal liver function test results, except for increased serum bilirubin or alkaline phosphatase concentrations due to choledocholithiasis (common bile duct stone) or chronic cholangitis. Gilbert syndrome, a benign disorder causing elevation in unconjugated bilirubin, is one of the most common causes of jaundice and may occasionally be mistaken for postoperative hepatobiliary dysfunction. Conversely, Dubin-Johnson and Rotor syndromes are congenital disorders leading to elevated conjugated bilirubin levels that can be exacerbated by surgery.

Management of Anesthesia

Anesthesia for cholecystectomy or exploration of the common bile duct, or both, is influenced by the effect of the drugs used for anesthesia on intraluminal pressure in the biliary tract. Specifically, opioids can produce spasm of the choledochoduodenal sphincter, which increases common bile duct pressure. Such spasm may impair the passage of contrast medium into the duodenum and erroneously suggest the need for sphincteroplasty or the presence of common bile duct stones. However, opioids have been used in many instances without adverse effect, which emphasizes the fact that not all patients respond to opioids with choledochoduodenal sphincter spasm. Treatment of biliary spasm includes naloxone, glucagon, and nitroglycerin.

Laparoscopic Cholecystectomy

Anesthetic considerations for laparoscopic cholecystectomy are similar to those for other laparoscopic

procedures.[24] For example, insufflation of the abdominal cavity (pneumoperitoneum) with carbon dioxide introduced through a needle placed via a supraumbilical incision results in increased intra-abdominal pressure that may interfere with ventilation of the lungs and venous return. During laparoscopic cholecystectomy, placement of the patient in the reverse Trendelenburg position favors movement of the abdominal contents away from the operative site and may facilitate mechanical ventilation of the lungs. This position, however, may further interfere with venous return. Generous intravascular fluid replacement during laparoscopic cholecystectomy may facilitate recovery from this type of surgery.[25]

Monitoring end-tidal carbon dioxide concentrations during laparoscopic abdominal surgical procedures is useful because of the unpredictability of systemic absorption of the carbon dioxide used to create the pneumoperitoneum. Intraoperative decompression of the stomach with a nasogastric or orogastric tube may decrease the risk for visceral puncture at the time of needle insertion and may subsequently improve laparoscopic visualization. Administration of nitrous oxide during laparoscopic cholecystectomy has typically not been recommended because of the possibility that it could expand bowel gas volume, causing interference with surgical working conditions and the theoretical possibility that diffusion into the abdominal cavity could support combustion.[26] Loss of hemostasis or injury to the hepatic artery or liver may require prompt intervention via a conventional laparotomy incision.

QUESTIONS OF THE DAY

1. What are the humoral mediators of renal function? What are their effects on the cardiovascular system?
2. What are the complications of administering thiazide, loop, and osmotic diuretics?
3. What is the differential diagnosis of prerenal and postrenal oliguria?
4. What are the physiologic changes associated with end-stage liver disease (ESLD)?
5. What is the differential diagnosis of postoperative jaundice?
6. What are the effects of carbon dioxide insufflation in the abdominal cavity during laparoscopic biliary surgery?

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