Anesthesia for patients with renal failure
JACK L. KOHL, RN, BS
Perrysburg, Ohio

Patients with impaired renal function frequently present the anesthetist with unique problems. The author reviews the kidney's anatomy and physiology as well as techniques for general and regional anesthesia. The effect of renal dysfunction on each major organ system and the pharmacokinetics of currently used drugs also are described.

Review of anatomy and physiology of the kidney

The kidney's function is to excrete urine. Kidneys are the organs of excretion for most of the end products of metabolism including some anesthetic agents. Homeostasis, in large part, depends on the kidney more than any other organ in the body. Fluid, electrolyte, and acid base balance are related directly to the physiology of the kidney. Four processes accomplish the task of urine formation. These are glomerular filtration, tubular reabsorption, tubular secretion, and water conservation and the counter-current mechanism.

Filtration is the first step in the formation of urine. Over 500 ml of blood enter the kidney each minute producing an average of 120 ml of filtrate per minute. The amount of filtrate is greatly affected by glomerular blood pressure, capillary, and capsular wall permeability. Three different pressures determine the effective filtration pressure. These are the glomerular hydrostatic pressure, blood colloid osmotic pressure, and capsular hydrostatic pressure. In addition, capsular colloid osmotic pressure may become a factor when disease has increased glomerular permeability enough to allow protein molecules to diffuse easily back and forth into Bowman's capsule. Glomerular hydrostatic pressure also is affected by changes in systemic blood pressure, which are common under anesthesia. Ninety-nine percent of the filtrate is reabsorbed through the walls of the convoluted tubes, the loop of Henle, and the distal collecting ducts. Water, glucose, amino acids, sodium, chloride, bicarbonate, and other particles are reabsorbed. Osmosis, diffusion, and active transport provide the mechanisms for these movements. Over 80% of the reabsorption occurs in the proximal tubes.1

Regulation of the acid base balance is an important function of the kidneys. The kidney can change the permeability of its tubular cells to secrete varying amounts of hydrogen and ammonia. This change in urine acidity occurs mainly in the distal tubules. Potassium also is regulated in the distal tubule. Excess potassium is excreted and sodium is conserved in this manner. Certain drugs such as penicillin and para-aminohippuric acid (PAH) also are secreted by the tubules. Tubular secretion can be evaluated by measuring the amounts of PAH excreted in the urine.

The volume of urine that is excreted is determined mainly by the hormones ADH and aldosterone. These hormones regulate the amount of
water that is reabsorbed by the distal tubules. The counter-current mechanism is responsible for the dilution or concentration of urine depending upon the body's need. This is made possible by the special anatomic relationship of the loop of Henle and the vasa recta. Acute renal failure (ARF) can be caused by either renal ischemia or nephrotoxic drugs such as antibiotics or radiographic dyes. It is characterized by the body's inability to rid itself of nitrogenous wastes resulting in progressive oliguria and azotemia. Oliguria may be defined as less than 400 ml of urine excreted over 24 hours. Azotemia occurs when the BUN rises by 10 mg/dl, and the serum creatinine increases by 0.5 mg/dl/24 hours. According to one study, 43% of acute renal failure was related to surgery, 26% to medical illness, 13% to pregnancy, 9% to trauma, and 9% to nephrotoxins. The most common cause of ARF is renal ischemia. The duration and severity of hypotension or hypoxia necessary to produce renal failure varies from and according to clinical setting. Brief periods of hypotension may cause failure in one person in a particular situation while another person may survive hours or even days of renal ischemia without change in renal function.

The clinical course of ARF starts with oliguria, which usually begins within a few days to a week after the initial insult. The oliguric phase may last from a few hours to several weeks. Cardiac arrhythmias and congestive heart failure especially are common during this phase and are caused by electrolyte imbalance, fluid overload, or a combination of the two. Salt and water overload also can cause peripheral and pulmonary edema, hyponatremia, and cerebral edema. Urine output may exceed two liters per day during the initial stage of the diuretic phase. Some of the complications of the oliguric phase may persist or first appear during this period. Gastrointestinal bleeding and cardiovascular dysfunction are some of the problems that may be encountered. The recovery phase lasts up to one year after the episode of acute renal failure. However, only a minority of the patients recover normal renal function. About two-thirds of these patients have a glomerular filtration rate (GFR) that is 20 to 40% below normal after one year. The deciding factor in determining survival after ARF is the nature of the causative event. Sixty percent of the patients who develop this syndrome after surgery or trauma die. Thirty percent of the patients whose illness is the result of medical causes, such as nephrotoxins, die. Ten to 15% of obstetric patients fail to recover.

Chronic renal failure (CRF) occurs when a majority of the nephrons are no longer functional and the remaining nephrons are unable to carry the workload. Many times these patients are not aware of the presence of the disease until severe renal failure has developed. CRF can sometimes be differentiated from the acute type by the size of the kidney. The patient with CRF generally has a smaller kidney which can be seen on X-ray. Some of the causes of CRF include chronic glomerulonephritis, traumatic loss of kidney tissue, congenital absence of kidney tissue, urinary tract obstruction from renal stones, pyelonephritis, and diseases affecting the renal vasculature.

Abnormalities in other systems seen in renal failure

Many types of cardiovascular problems are seen in these patients. Hypertension is primarily the result of salt and water retention, although a minority of patients have hypertension because of high renin output. Chronic hypertension can lead to left ventricular hypertrophy and possibly congestive heart failure. The chronic anemia seen in these patients further strains the heart because of a compensatory increase in cardiac output. Other cardiovascular problems are an increased incidence of atherosclerotic changes, coronary artery disease, pericarditis, uremic cardiomyopathy, and autonomic neuropathy.

Uremic lung is an example of the respiratory disorders found in renal failure patients. Radiographically it is characterized by perihilar pulmonary congestion. Pulmonary infections are common with patients who are immunosuppressed. General anesthesia and instrumentation of the airway may exacerbate the infection.

Nervous system symptoms of renal failure include neuromuscular irritability, fatigue, lethargy, and behavioral changes. Patients with terminal uremia are likely candidates for seizure activity. Two types of seizure disorders may occur. In the patient with malignant hypertension, the increased cerebral edema may precipitate the seizure. Patients on hemodialysis may develop a dialysis dementia or disequilibrium syndrome, both of which may cause seizures.

Gastrointestinal symptoms associated with renal failure can complicate the anesthetic induction. Nausea, vomiting, and frequent episodes of hiccoughs can make aspiration of stomach contents a real danger. Gastric emptying time is delayed and may be almost doubled if the patient is on hemo-

432

Journal of the American Association of Nurse Anesthetists
dialysis. These patients should be treated as a full stomach case, since both gastric volume and acidity are increased. The incidence of hepatitis and hepatic venous congestion also is increased.

Anemia is a common finding in the CRF patient. Hemoglobin ranges from 3.9 grams per 100 ml with hematocrit levels averaging 15-25%.

The greatest danger of this anemia is the reduced oxygen carrying capacity. As mentioned before, it is compensated for by an increased cardiac output. The metabolic acidosis seen in these patients causes a shift to the right of the oxyhemoglobin dissociation curve, resulting in an increased release of oxygen from the hemoglobin. The increase in 2,3 DPG further shifts the curve to the right. It should be pointed out that 2,3 DPG is an enzyme system that enhances the dissociation of oxygen from hemoglobin by competing with oxygen for the iron-binding site.\(^6\) Bleeding tendencies are the result of platelet dysfunction. Therefore, a full coagulation screening should be done before any major surgery.

A mild metabolic acidosis is observed because of a decrease in serum bicarbonate levels. There is a compensatory respiratory alkalosis with the pH remaining slightly below normal. The increased body water causes a hyponatremia that is worsened by diuretics.

Hyperkalemia is the most serious electrolyte abnormality, and surgery should probably be avoided if the serum potassium level is greater than 5.5.\(^6\)

Premedication

These patients are sensitive to all psychotropic drugs. Benzodiazepines can be used because they are administered orally and are metabolized by the liver. The effect of diazepam may be prolonged because of a reduction in protein binding. Anti-cholinergies may be given in their usual doses but should not be given intramuscularly because of the bleeding problems. Narcotics may be given in the usual dosage range since urinary excretion is not a significant mode of elimination. Some authors recommend cimetidine (Tagamet\(^\text{®}\)) as a pre-medication because of the increased gastric volume and acidity.\(^7\)

General anesthesia

Special attention should be taken with these patients during the induction of general anesthesia. Patients should be pre-oxygenated because of the reduced oxygen-carrying capacity resulting from anemia. A rapid sequence induction is preferred, as stated earlier, since aspiration is a possibility. These patients are more sensitive to the hypnotic effects of barbiturates. This is the result of the decreased protein binding. Distribution and metabolism are not changed in renal failure. Therefore, barbiturate doses should not be decreased, but should be given more slowly.

Narcotics may be used since their route of elimination is mainly through liver metabolism and not through urinary excretion. Morphine appears 15% unchanged in the urine while 10% of fentanyl is similarly unchanged. In the past, it was thought that meperidine and morphine caused a decrease in urinary output by stimulation of the antidiuretic hormone (ADH). It is now believed that surgical stimulation is the cause of the increased ADH.

Succinylcholine commonly is used for induction and endotracheal intubation. This causes a rise in the serum potassium level of 0.5 to 0.7 mEq/L. Pretreatment with d-tubocurarine does not prevent this increase. Prolonged apnea following the use of succinylcholine apparently does not occur, indicating that serum cholinesterase activity is not affected either by renal failure or dialysis treatment.

The nondepolarizing muscle relaxants have been studied extensively in patients with renal dysfunction. There usually is a decreased muscle mass in patients with chronic renal failure and, therefore, doses of nondepolarizing agents should be reduced. The dependence on renal excretion of these drugs varies from complete to none.

Gallamine is contraindicated in patients with renal failure because it is excreted mainly unchanged by the kidneys.

The effects of pancuronium bromide may be increased by up to 80% in these patients. Its effect may be prolonged by respiratory and metabolic acidosis, hypokalemia, hypocalcemia, hypermagnesemia, aminoglycoside antibiotics, furosemide, or mannitol. These potentiating factors also affect the other nondepolarizing agents. The advantage of pancuronium is its cardiovascular stability.

D-tubocurarine was, until recently, the preferred drug for these patients. The biliary pathway of excretion is utilized more readily by this drug. This method of elimination can increase four times in the anephric patient.

Vecuronium is a monoquaternary drug that is very similar to pancuronium. It has a more stable cardiovascular effect than pancuronium and uses the biliary route of excretion to a greater extent.

Atracurium is broken down by Hoffman elimination and hydrolysis. The kidney and liver do...
not play a significant role in its elimination. Therefore, it may be the ideal drug for the patient with renal problems.

Inhalational agents have the advantages of rapid elimination and independence from renal function.

Enflurane is partially metabolized, but serum inorganic fluoride levels usually do not exceed 20 μm/L. Levels above 50 μm/L are considered nephrotoxic, but this may be lower in diseased or transplanted kidneys.

Halothane, like the other agents, lowers the RBF, GFR, and urine output secondary to reduced blood pressure and increased ADH levels. Serum fluoride levels are not increased significantly following halothane anesthesia.

Isoflurane undergoes less biotransformation than its isomer enflurane and has a more stable cardiovascular profile than halothane. These properties appear to make it superior to the other volatile inhalational agents.

Intraoperative management of fluids is very important. The following is an example of a fluid replacement schedule:

1. 500 ml/day insensible water loss should be replaced with dextrose 5% in water.
2. Replace any urine output with half normal saline.
3. Replace blood loss with packed red cells.
4. Replace third space loss with normal saline alternating with plasma protein solutions at 2.5 ml/kg/hour, depending on the location and type of surgery.

These amounts can be varied when other monitors such as central venous pressure or pulmonary capillary pressure are available.

The postoperative management goals are the maintenance of normovolemia, a stable cardiovascular system, supplemental oxygen to offset the lower hemoglobin level, and appropriate analgesia. Adequate reversal of muscle relaxants is mandatory. Metabolic disturbances and the use of certain antibiotics may potentiate the neuromuscular blockade resulting in hypoventilation. This will lead to hypoxia, hypercapnia, and respiratory acidosi. Continued ventilation for a short time after surgery, until adequate muscle power has returned, is sometimes necessary.

In most patients, the kidney plays an important role in homeostasis. The dysfunction of this organ system can cause a multitude of problems. It is up to the anesthetist to anticipate these problems and to plan the appropriate measures necessary to insure the safety of the patient in the operating room.

REFERENCES


AUTHOR

Jack L. Kohl, RN, BS, received an associate degree in nursing from Michael J. Owens Technical College and a BS degree in health from the University of Toledo. At the time this paper was written, he was a senior nurse anesthesia student at St. Vincent Medical Center School of Anesthesia for Nurses in Toledo. He graduated from that program in September, 1985.