Perioperative Acid-Base and Electrolyte Disturbances

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KEYWORDS

• Acidosis • Alkalosis • Electrolytes • Oxygenation • Ventilation

KEY POINTS

- Acid-base and electrolyte abnormalities are common in perioperative patients, and appropriate recognition and treatment is essential to optimize outcome.
- Fluid therapy provides treatment of most metabolic acid-base disturbances.
- Respiratory support, including supplemental oxygen and occasionally mechanical ventilation, may be necessary to correct respiratory disturbances.
- Electrolyte disturbances may be corrected by fluid therapy or a variety of pharmacologic agents.
- Correction of these disorders preoperatively and intraoperatively results in a more stable anesthetic candidate.

INTRODUCTION

Obtaining and interpreting values for blood gases and electrolytes is essential in the management of many perioperative veterinary patients. Metabolic and electrolyte alterations are common in critically ill surgical patients, and can lead to alterations in cardiovascular function, neurologic status, respiratory function, and even response to various drug therapies. Several common preoperative and postoperative conditions are discussed in this article. **Box 1** contains a 6 step method for the interpretation of blood gases, a skill that is needed to diagnose some of the derangements that are discussed in this article. Normal arterial and venous blood gas values for dogs and cats are listed in **Table 1**, and the expected compensatory changes are listed in **Table 2**.

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Box 1
Interpreting blood gas results

There are 6 steps required to interpret blood gas results:

1. Determine whether sample is venous or arterial. Either sample type can be used to evaluate overall acid-base status, with the exception of severe shock and postarrest situations, which may result in large discrepancies between arterial and venous samples. Poor tissue perfusion can result in sizable increases in CO_2 and secondary decreases in pH on the venous side despite low to normal CO_2 on the arterial side.

   Although information can be gained about ventilation from a venous sample, only an arterial sample can assess oxygenation.

   If unable to obtain an arterial sample, use pulse oximetry to measure oxygen saturation and a venous sample to evaluate acid-base status and estimate ventilation.

   If the patient is intubated, end-tidal CO_2 can also be used to estimate ventilation, but, with severe pulmonary disease, end-tidal CO_2 can be much lower than $Paco_2$.

2. Assess the patient for acidemia (pH <7.35) or alkalemia (pH >7.45).

   If pH is within normal limits, the patient's body may have compensated for an underlying disturbance or a mixed disturbance may be present. See steps 3 and 4 to evaluate whether metabolic or respiratory disturbances are present despite normal pH.

3. Assess for acidosis.

   Respiratory acidosis is present if $Paco_2$ is greater than 45 mm Hg.

   Metabolic acidosis is present if base excess (BE) is less than −4 mmol/L (or HCO_3^- <19 mmol/L).

4. Assess for alkalosis.

   Respiratory alkalosis is present if $Paco_2$ is less than 35 mm Hg.

   Metabolic alkalosis is present if BE >2 mmol/L (or HCO_3^- >25 mmol/L).

5. Assess oxygenation.

   Normal Pao_2 is 90 to 100 mm Hg. If the patient is on supplemental oxygen, Pao_2 should equal approximately 5 times the fraction of inspired oxygen (Fio_2); the Fio_2 of room air is 21%.

   These rules apply to the normal values listed in **Table 1** for dogs. For cats, substitute the reported normal values for $Paco_2$ and BE from **Table 1** into steps 3 and 4.

6. Determine whether compensatory changes have occurred.

   For example, if a primary metabolic acidosis is present, a compensatory respiratory alkalosis may also exist. Remember the rules of compensation:

   A change in the respiratory or metabolic component of the acid-base status normally induces an opposite compensatory response in an effort to normalize the pH.

   The lungs can compensate quickly by adjusting minute ventilation in a matter of minutes.

   The kidneys compensate more slowly, with compensation beginning within a few hours and maximum compensation taking 4 to 5 days.

   The absence or presence and degree of compensation provide some information about the chronicity of the disturbance (see **Table 2**).

   Overcompensation does not occur.

Table 1
Normal values for canine and feline arterial and venous blood gases

	Arterial Values	Venous Values
Dogs[1]		
pH	7.395 ± 0.03	7.352 ± 0.02
P_{O_2} (mm Hg)	102.1 ± 6.8	55 ± 9.6
P_{CO_2} (mm Hg)	36 ± 2.7	42.1 ± 4.4
HCO_3^- (mmol/L)	21.4 ± 1.6	22.1 ± 2
BE (mmol/L)	−1.8 ± 1.6	−2.1 ± 1.7
Cats[2]		
pH	7.34 ± 0.1	7.30 ± 0.08
P_{O_2} (mm Hg)	102.9 ± 15	38.6 ± 11
P_{CO_2} (mm Hg)	33.6 ± 7	41.8 ± 9
HCO_3^- (mmol/L)	17.5 ± 3	19.4 ± 4
BE (mmol/L)	−6.4 ± 5	−5.7 ± 5

Abbreviation: BE, base excess.

PREOPERATIVE DERANGEMENTS

Preoperative patients, especially those undergoing emergency procedures, commonly have several acid-base and electrolyte derangements secondary to hypovolemia and underlying systemic illness. The most common abnormalities are discussed here.

Metabolic Acidosis

Metabolic acidosis occurs when endogenous or exogenous acids exceed the body's buffering capacity, resulting in a decrease in pH to less than 7.35. Systemic acidosis is important to recognize and treat because prolonged acidosis results in denaturation of proteins within the body, leading to impaired cellular function; organ dysfunction, including decreased myocardial contractility, ventricular arrhythmias, and fibrillation;

Table 2
Expected compensatory responses

Disorder	Changes	Compensatory Response
Metabolic acidosis	↓ HCO_3^-	0.7 mm Hg decrease in P_{aCO_2} for each 1 mEq/L decrease in HCO_3^-
Metabolic alkalosis	↑ HCO_3^-	0.7 mm Hg increase in P_{aCO_2} for each 1 mEq/L increase in HCO_3^-
Acute respiratory acidosis	↑ P_{aCO_2}	1.5 mEq/L increase in HCO_3^- for each 10 mm Hg increase in P_{aCO_2}
Chronic respiratory acidosis	↑ P_{aCO_2}	3.5 mEq/L increase in HCO_3^- for each 10 mm Hg increase in P_{aCO_2}
Acute respiratory alkalosis	↓ P_{aCO_2}	2.5 mEq/L decrease in HCO_3^- for each 10 mm Hg decrease in P_{aCO_2}
Chronic respiratory alkalosis	↓ P_{aCO_2}	5.5 mEq/L decrease in HCO_3^- for each 10 mm Hg decrease in P_{aCO_2}

Data from DiBartola SP. Introduction to acid–base disorders. In: DiBartola SP, editor. Fluid, electrolyte, and acid–base disorders in small animal practice, 4th ed. St. Louis: Elsevier Saunders; 2012. p. 231–52.

vasodilation with decreased response to catecholamines; insulin resistance; increased work of breathing (to compensate for a metabolic acidosis); and potentially death. The major causes of metabolic acidosis include lactic acidosis, exogenous acids, ketoacidosis, uremia, and loss of bicarbonate.

Lactic Acidosis

The most common cause of metabolic acidosis is lactate accumulation. Lactate is the end product of anaerobic metabolism; lactic acidosis occurs when plasma lactate is increased in conjunction with a decrease in systemic blood pH.[3,4] Lactic acidosis is common in patients with hypoperfusion or hypoxia, and has also been reported in association with other conditions (sepsis, neoplasia, drugs/toxins, mitochondrial dysfunction, inborn errors of metabolism).[5,6] In addition, an increased lactate level can be seen with end-stage liver failure (because it normally metabolizes most of the lactate produced) or with a focal area of tissue ischemia or necrosis (eg, a loop of infarcted bowel). Two types of lactic acidosis exist: type A and type B. Type A lactic acidosis is more common and occurs with tissue hypoxia and increased lactate production caused by anaerobic glycolysis. Type B lactic acidosis occurs when oxygen delivery is adequate but use of oxygen is impaired, as with altered mitochondrial function or carbohydrate metabolism. There are 3 subtypes of B lactic acidosis: B1 includes diseases that cause decreased lactate clearance, B2 includes drugs or toxins that interfere with oxidative phosphorylation, and B3 includes mitochondrial defects.[7] Although type A lactic acidosis is the likely cause of metabolic acidosis in many perioperative patients, both types likely exist concurrently in many patients, especially those with sepsis.[3,7,8] Diagnosis of lactic acidosis is made by measuring serum L-lactate concentration. Two forms of lactate exist: L-lactate is the most commonly measured and is produced by cellular metabolism in healthy monogastric animals; D-lactate is produced during bacterial glucose metabolism or alternate metabolic pathways in some intoxications or diseases.[8] L-Lactate can be quickly measured on many of the newer blood gas analyzers or with a hand-held point-of-care lactate analyzer. Several factors, including stress, seizures, recent exercise, excitement, food intake, and prolonged venous stasis during collection, can potentially increase lactate concentrations from 2.5 to 10 mmol/L.[3,9] In cats, one study showed a 10-fold increase in lactate levels in healthy cats that were stressed before sample collection, although a more recent study showed no statistical differences in lactate levels with struggling during sampling.[10,11] If measurement of plasma lactate is not possible, it can be suspected when a metabolic acidosis is present on blood gas analysis that is not secondary to diabetic ketoacidosis, renal failure, renal tubular acidosis, or exogenous acids such as ethylene glycol.

In human patients, lactate monitoring is commonly used to guide resuscitation and for prognostication purposes, and numerous studies have shown that as blood lactate concentrations increase, probability of survival decreases.[12–15] More recently in human medicine, emphasis has been placed on monitoring serial lactate concentrations, because studies have shown that patients who clear increased lactate levels have improved outcomes compared with those who do not.[15–18] Several veterinary studies, mostly in dogs, have also shown that lactate can be used to identify hypoperfusion and assess response to therapy.[5,19–24] A notable area of lactate research in canine patients has focused on lactic acidosis with gastric dilatation-volvulus, which can develop secondary to either regional or systemic hypoperfusion.[21,24] Studies have shown correlations between initial plasma lactate concentrations and the presence of gastric necrosis as well as outcome, and more recent studies have shown correlations between lactate clearance and resuscitation and survival.[19–21,24]

Many surgical patients present with a lactic acidosis secondary to hypovolemia, whether they are patients with trauma; acute abdomens, including gastric dilatation-volvulus; septic peritonitis; hemoabdomen, or other causes. Treatment of lactic acidosis is designed to improving oxygen delivery to the tissues. Most commonly, this is corrected by volume resuscitation (assessment of perfusion and fluid balance is discussed by Boller elsewhere in this issue), but, in some cases, providing supplemental oxygen or a hemoglobin source such as packed red blood cells is necessary. Although aggressive fluid therapy may be warranted in some hypovolemic patients, caution should be exercised, especially in septic surgical patients, because recent human studies have shown worse outcomes in septic patients with fluid overload.[25] Bicarbonate administration should never be necessary when a metabolic acidosis is caused by lactate. Without treatment, severe acidosis can lead to vasodilation and hypotension, arrhythmias, decreased cardiac contractility, increased respiratory effort, mental dullness, insulin resistance, and death.[7,8]

Uremia can also result in a severe metabolic acidosis. This condition can be a concern in patients with uroabdomen, ureteral obstructions, or urethral obstruction that cannot be relieved and requires anesthesia and surgery. Treatment consists of appropriate fluid therapy; treatment of concurrent hyperkalemia, if present; as well as sodium bicarbonate therapy, if indicated. In cases of oliguric or anuric acute kidney injury, advanced modalities such as peritoneal dialysis, intermittent hemodialysis, or continuous renal replacement therapy may be recommended.

Hyperkalemia

Severe hyperkalemia can quickly become a life-threatening emergency because of its effects on the cardiovascular system. High potassium levels decrease the transmembrane potassium gradient and depolarize the cell membrane, impairing excitation and conduction. These changes can result in cardiac effects, including bradycardia, atrial standstill, and cardiac arrest. Typical electrocardiogram (ECG) changes include a peaked, narrow T wave (serum potassium concentration >5.5 mEq/L); prolonged QRS complex and PR interval; depressed R wave amplitude and ST segment (>6.5 mEq/L); depressed P wave amplitude (>7 mEq/L); atrial standstill or sinoventricular rhythm (>8.5 mEq/L); and biphasic QRS complexes, ventricular flutter, ventricular fibrillation, or asystole (>10 mEq/L).[26,27] Although experimental studies of hyperkalemia have been shown to induce these specific electrocardiographic changes, the same levels of hyperkalemia may not show parallels in clinical patients, and potassium should always be measured in clinical patients.[27] Patients with just a moderate hyperkalemia may be more at risk for anesthetic complications even if they have not been clinical before anesthesia.[28] In addition, systemic effects, including weakness, flaccid paralysis, respiratory failure, gastrointestinal hypomotility, and hyporeflexia can occur.

Causes of hyperkalemia include decreased excretion of potassium caused by urethral obstruction, rupture of the urinary tract, acute renal failure, hypoadrenocorticism, severe metabolic acidosis, and crush injury. However, many of these causes are surgical, and stabilization of the patient, including normalization of potassium concentration, is necessary before anesthesia and surgery are advisable. Treatment may include intravenous (IV) fluid therapy if the patient is hypovolemic or dehydrated, calcium gluconate, insulin and dextrose, and sodium bicarbonate administration.

Treatment of hyperkalemia involves 3 phases: immediate cardioprotection from life-threatening hyperkalemia, redistribution of serum potassium, and excretion of potassium from the body. Immediate cardioprotection can be achieved with IV calcium gluconate while monitoring the ECG. Calcium gluconate does not decrease the serum K^+ concentration, but does decrease the threshold potential for the cardiac cells,

reestablishing the normal difference between resting and threshold potentials. This process helps to normalize membrane excitability within minutes of administration. In dogs and cats, the recommended dose of 10% calcium gluconate is 50 to 150 mg/kg as a slow (5–10 minutes) IV bolus. Administering calcium gluconate too quickly can result in worsening bradycardia and severe ventricular arrhythmias, so an ECG should be monitored while administering. The onset of action is rapid (within 5 minutes) but short acting, so additional steps must be taken to reduce serum potassium levels. Redistribution of serum potassium can be achieved with dextrose and/or regular insulin. Dextrose can be given as an IV bolus (0.5 g/kg) to promote endogenous insulin release and movement of potassium intracellularly, as it is cotransported with glucose. Regular crystalline insulin can be given at the same time at a dose of 0.25 to 0.5 U/kg IV. Exogenous insulin may have a quicker onset of action and a more dramatic reduction in potassium concentration, but hypoglycemia can result. When using insulin and dextrose, it is imperative that dextrose be supplemented in the fluids at 2.5% to 5% in addition to giving a dextrose bolus at the dose mentioned earlier to prevent secondary hypoglycemia. Sodium bicarbonate can also be used to treat hyperkalemia. It promotes movement of potassium intracellularly as hydrogen ions move extracellularly to buffer the bicarbonate. This treatment may be especially helpful in cases with hyperkalemia and a concurrent metabolic acidosis. An IV dose of 1 to 2 mEq/kg of sodium bicarbonate is given slowly over 15 minutes, or the base excess (BE) can be used to calculate the base deficit (base deficit = $0.3 \times BE \times$ weight in kilograms) and one-quarter to one-third of that calculated value is given intravenously. Terbutaline can also be used to stimulate Na^+/K^+-ATPase to cause translocation of potassium into the cell.[29] It can be given as a dose of 0.01 mg/kg subcutaneously or intravenously.

The underlying cause of hyperkalemia should be addressed as soon as possible. Preoperatively, this may include relieving a urethral obstruction, providing a method of drainage for a uroabdomen, or improving renal perfusion and increasing urine output in patients with oliguric or anuric acute kidney injury. Although 0.9% NaCl is the hypothetical fluid of choice for hyperkalemia because it contains no additional potassium, administration of any balanced electrolyte solution (Normosol-R, Plasmalyte-A, or lactated Ringer solution [LRS]) helps to dilute the serum potassium concentration. One study in cats with urethral obstruction comparing the use of 0.9% NaCl and Normosol-R showed that the use of a balanced electrolyte solution such as Normosol-R may allow more rapid normalization of acid-base abnormalities and does not affect the rate of normalization of serum potassium.[30]

Once the patient's potassium has been stabilized, anesthesia and surgery to correct the underlying problem can be considered. During anesthesia, the patient's ventilation should be monitored carefully because hypercapnia may decrease pH and result in release of potassium from cells.[28] In severe cases, the treatments mentioned earlier may not be sufficient treatment of the hyperkalemia. In these cases, peritoneal dialysis or, ideally, hemodialysis is indicated. If surgery is attempted in patients with hyperkalemia that has not/cannot be corrected medically, it should be kept in mind that these cases are poor anesthetic candidates and are at high risk of cardiac arrest. Every attempt should be made to stabilize the potassium and its cardiovascular effects before anesthesia.

Hypochloremic Metabolic Alkalosis

This preoperative derangement is classically seen with high gastrointestinal obstruction, although a recent study reported that hypochloremic, hypokalemic metabolic alkalosis is seen with proximal and distal foreign body obstructions in dogs.[31] Other

causes of metabolic alkalosis identified in dogs and cats in a recent retrospective study include respiratory disease, furosemide administration, and renal disease.[32] With a gastrointestinal obstruction, vomiting results in a loss of hydrogen, sodium, potassium, chloride, and water, resulting in extracellular fluid and intravascular volume depletion, and development of a hypochloremic metabolic alkalosis. Gastric fluid has a very high concentration of chloride (150 mEq/L). Continued loss of hydrogen in the vomitus creates a metabolic alkalosis, because 1 bicarbonate molecule is produced for every hydrogen ion. The concurrent volume depletion stimulates the kidneys to reabsorb sodium. Because of continued loss of hydrogen and chloride and lack of intake of dietary salt, a deficit of chloride occurs. These patients are in a volume-depleted state that activates the renin angiotensin system. As the kidneys reabsorb sodium, they are unable to reabsorb chloride because of chloride depletion. Instead, they rely on reabsorption of sodium in place of potassium and hydrogen. This process results in hypokalemia and a paradoxic aciduria.[1]

Treatment should be designed to correct the underlying cause after correcting intravascular volume and chloride concentrations. The fluid therapy of choice for volume replacement is 0.9% NaCl because of its high chloride concentration. Potassium should be supplemented, if indicated, once any boluses have been completed. The metabolic alkalosis may take several days to resolve, even with appropriate fluid therapy. Once the patient has been stabilized cardiovascularly, the underlying cause can be addressed. Surgery for removal of a pyloric foreign body or for treatment of pyloric hypertrophy is often indicated. Care should be taken while the animal is under anesthesia to avoid hyperventilation. A compensatory respiratory acidosis will be present because of the metabolic alkalosis, and hyperventilation abolishes this compensatory response and results in severe alkalemia.[1]

INTRAOPERATIVE DERANGEMENTS

Hypoxemia/Hypercapnia

Hypoxemia is defined as a partial pressure of oxygen (Pa_{O_2}) less than 80 mm Hg or arterial hemoglobin saturation less than 95%. The 3 major causes of hypoxemia are low inspired oxygen, hypoventilation, and venous admixture.[33] In the perioperative period, hypoxemia commonly occurs secondary to all of these causes. Low inspired oxygen can occur with anesthetic equipment complications (eg, neglecting to turn on an oxygen source or running out of oxygen from a tank). Hypoventilation is defined as an increased Pa_{CO_2} (usually >45 mm Hg) and can rapidly cause a life-threatening respiratory acidosis. Causes can include impairment of the normal respiratory pathway from the respiratory center in the brain stem, the cervical spinal cord, and neuromuscular disease as well as restrictive disorders preventing lung expansion, such as pleural space disease; or, rarely, severe primary pulmonary disease such as pneumonia. The most common cause of hypoventilation in perioperative patients is depression of the respiratory center from anesthetics and/or analgesics, but it can also be caused by upper airway obstruction, especially in brachycephalic breeds, as well as neurologic impairment in postoperative cervical intervertebral disc disease, cervical spinal fracture repairs, or craniotomies. Treatment is designed to correct the underlying problem. If respiratory center depression is the cause and the patient is under anesthesia, positive pressure ventilation is indicated. In the postoperative period, partial reversal of the anesthetic drugs may be sufficient to correct the hypoventilation. Neurologic causes may also require positive pressure ventilation (oxygenation and ventilation are discussed by Rozanski elsewhere in this issue). Venous admixture, the third cause of hypoxemia, occurs when venous blood moves from the right to

the left side of the circulation without being appropriately oxygenated. In perioperative patients, this commonly occurs secondary to atelectasis from small airway and alveolar collapse. Supplemental oxygen should be provided to these patients, and postoperative patients should be turned regularly and encouraged to stand and walk to attempt to recruit collapsed alveoli. Also, during prolonged anesthesia times, medical air can be used instead of 100% oxygen to reduce the risk of absorption atelectasis.[34]

POSTOPERATIVE DERANGEMENTS

Hypernatremia

Central diabetes insipidus (CDI) results from partial or complete lack of vasopressin production from the neurohypophysis.[1] Although CDI is most common in veterinary patients with intracranial disease, it can also been seen secondary to hypoxic or ischemic encephalopathy following trauma, severe shock, or after cardiopulmonary arrest causing decreased antidiuretic hormone (ADH) release.[35–38] This cause of CDI is usually transient, but it can result in significant hypernatremia in postoperative patients.[39] These patients have increased serum sodium concentrations and osmolality, and usually have hyposthenuric urine (unless severely dehydrated, in which case they may approach isosthenuria). Clinical experience indicates that this is most common in postoperative gastric dilatation-volvulus cases, but other surgical cases, including gastrointestinal resection and anastomosis, septic peritonitis, hemoabdomen, and occasionally thoracotomy, may also be affected. These patients are often administered high rates of sodium-rich fluids, such as Normosol-R, Plasmalyte-A, or LRS, often with hetastarch, which is most commonly administered in 0.9% NaCl. Most patients with normal renal function are able to handle the high sodium load that is provided by this type of fluid administration, and as the serum sodium concentration and osmolality increase, the patients have increased thirst. The increased intake of free water corrects the serum sodium concentration and osmolality. However, in postoperative patients that may be unable or unwilling to drink right away, severe hypernatremia may result.

Before a treatment plan for hypernatremia is instituted, the patient's volume status must be assessed. If the patient is hypovolemic and hypernatremic, then volume expansion should occur with isotonic fluids. The degree of hypernatremia helps determine which fluid is most appropriate. With mild hypernatremia, Normosol-R or Plasmalyte-A (sodium concentration of approximately 145 mEq/L) may be reasonable choices. If the patient is moderately hypernatremic, then 0.9% NaCl is a better choice because of its higher sodium concentration (154 mEq/L). More commonly, these postoperative patients are euvolemic, because they have already had adequate fluid resuscitation. Although the lack of ADH causes water loss, it is usually not to the degree that they become hypovolemic because they are on concurrent IV fluid therapy. The patient's free water deficit can be calculated using the equation[40]:

$$\text{Free water deficit (L)} = (\text{current } [Na^+]/\text{normal } [Na^+] - 1) \times [0.6 \times \text{body weight (kg)}]$$

This equation gives the total volume of free water that should be replaced, and can be given over the number of hours that have been calculated to be needed to safely reduce the sodium concentration (typically 0.5 mEq/L/h or 10–12 mEq/L/d) if the hypernatremia is chronic, although this is rarely a concern in postoperative patients. Too-rapid correction of hypernatremia can result in cellular swelling and neuronal edema and can lead to life-threatening neurologic complications. Treatment of these patients is designed to increase free water to reduce the sodium concentration. If the patient is willing and able to drink, providing access to small amounts of water divided

incrementally may be sufficient therapy. In more severe cases in which serum sodium concentration is rapidly increasing (>160 mmol/L) and the patient is not drinking, water loss can be replaced with 5% dextrose, which is given along with a maintenance rate of isotonic fluids (2 mL/kg/h). Alternatively, 5% dextrose at a rate of 3.7 mL/kg/h can be given along with maintenance isotonic fluids, and this usually reduces the sodium concentration at the desired rate in dogs.[28] This protocol is contraindicated in patients that cannot tolerate high fluid rates, and should only be used in patients in which frequent measurement (every 2–4 hours) of sodium concentration can be performed.

The most common complication of hypernatremia therapy is neuronal and cerebral edema. If a patient shows neurologic signs such as a change in mental status, seizures, head-pressing or other disorders of behavior or movement, fluid therapy should be stopped immediately and serum sodium should be rechecked. A decrease in measured sodium level in combination with neurologic signs should prompt treatment of cerebral edema, which may include mannitol (0.5–1 g/kg IV over 20–30 minutes) or hypertonic saline (7.2% solution at 3–5 mL/kg over 20 minutes).[40]

An additional treatment that may be indicated in severe cases of hypernatremia secondary to CDI is the administration of exogenous ADH. If the patient has CDI, vasopressin or desmopressin can be used to control the sodium. Desmopressin acetate can be given at a dose of 1 to 2 drops in both eyes every 12 to 24 hours.[1] Careful monitoring of both the patient's sodium concentration and body weight should occur to prevent rapid changes. Fluid therapy may need to be adjusted as well, particularly if large amounts of free water were being administered. Exogenous ADH and fluids containing free water can cause rapid and dangerous decreases in sodium concentration and plasma osmolality.

Renal Tubular Acidosis

Renal tubular acidosis (RTA) is associated with a metabolic acidosis caused by either decreased sodium bicarbonate reabsorption (proximal RTA or type 2) or a lack of hydrogen ion excretion (distal RTA or type 1).[41] Type 1 RTA can occur secondary to marked volume depletion or urinary tract obstruction, and so can become evident in postoperative patients.[42] The diagnosis of type 1 RTA can be made when there is an increased urine pH (>6.0) with a concurrent metabolic acidosis, providing that a urinary tract infection with a urease-positive organism has been ruled out. RTA should be suspected in cases that have a persistent metabolic acidosis that is not secondary to more common causes such as lactic acidosis, uremia, ketoacidosis, severe diarrhea, or exogenous acids (ethylene glycol). Evaluation of the anion gap is helpful, because the anion gap is normal in cases with RTA and diarrhea, and increased in all other causes of metabolic acidosis.

Treatment of RTA depends on the severity of the metabolic acidosis. Mild cases with minimal effect on the blood pH may be self-limiting and not require any therapy. More severe cases may benefit from the administration of sodium bicarbonate. There are several potential risks of sodium bicarbonate therapy, and administration should only be considered when necessary. Risks include hypercapnia and paradoxic cerebral acidosis, hypokalemia and ionized hypocalcemia, hypernatremia, hypervolemia, and hyperosmolality. Dosing is based on the following equation: mEq of sodium bicarbonate = 0.3 $\times$ body weight (kg) $\times$ base deficit. Usually one-quarter to one-third of the dose is given over 4 to 6 hours. Because sodium bicarbonate is hypertonic, is must be diluted to an osmolality of less than 600 mOsm/L before peripheral administration. For the commercially available sodium bicarbonate (8.4%), its osmolality is roughly 2000 mOsm/L, and hence it should be diluted at least 1:3 with sterile water for administration through a peripheral catheter.[43] This dilution is particularly important in patients

that are hypernatremic concurrently, because the sodium load provided by the bicarbonate is significant. However, RTA secondary to severe hypovolemia is a transient disease process, so repeat dosing of sodium bicarbonate is not usually needed while hospitalized, and no long-term therapy is necessary.

SUMMARY

Correction of acid-base and electrolyte disturbances is crucial for optimal outcome in perioperative patients. A thorough physical examination and close monitoring are key in recognition and treatment of many of the most common disorders discussed earlier, and the importance of serial examinations cannot be overemphasized in these dynamic patients. Cardiovascular and respiratory failure and death may result if acid-base and electrolyte disturbances are not promptly recognized and treated appropriately.

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