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Helio Autran de Morais and Stephen P. DiBartola

Quick Reference

A Quick Reference on Hypoxemia 175
Jonathan Bach

This article describes the calculation of the alveolar-arterial gas gradient and its clinical application in determining the cause of hypoxemia. It also outlines the analysis of arterial blood gases and the clinical approach toward diagnosis and treatment of respiratory disease.

A Quick Reference on Respiratory Alkalosis 181
Rebecca A. Johnson

Respiratory alkalosis, or primary hypocapnia, occurs when alveolar ventilation exceeds that required to eliminate the carbon dioxide produced by tissues. Concurrent decreases in PaCO₂, increases in pH, and compensatory decreases in blood HCO₃⁻ levels are associated with respiratory alkalosis. Respiratory alkalosis can be acute or chronic, with metabolic compensation initially consisting of cellular uptake of HCO₃⁻ and buffering by intracellular phosphates and proteins. Chronic respiratory alkalosis results in longer-lasting decreases in renal reabsorption of HCO₃⁻; the arterial pH can approach near-normal values.

A Quick Reference on Respiratory Acidosis 185
Rebecca A. Johnson

Respiratory acidosis, or primary hypercapnia, occurs when carbon dioxide production exceeds elimination via the lung and is mainly owing to alveolar hypoventilation. Concurrent increases in PaCO₂, decreases in pH and compensatory increases in blood HCO₃⁻ concentration are associated with respiratory acidosis. Respiratory acidosis can be acute or chronic, with initial metabolic compensation to increase HCO₃⁻ concentrations by intracellular buffering. Chronic respiratory acidosis results in longer lasting increases in renal reabsorption of HCO₃⁻. Alveolar hypoventilation and resulting respiratory acidosis may also be associated with hypoxemia, especially evident when patients are inspiring room air (20.9% O₂).

A Quick Reference on Anion Gap and Strong Ion Gap 191
Carlos Torrente Artero

Metabolic acid-base disorders are common in emergency and critically ill patients. Clinicians may have difficulty recognizing their presence when multiple acid-base derangements are present in a single patient simultaneously. The anion gap and the strong ion gap concepts are useful calculations to identify the components of complex metabolic acid-base
associated to the presence of unmeasured anions. This article presents their definition, normal values, indications, limitations, and guidelines for interpretation of changes in the clinical setting.

A Quick Reference on Metabolic Alkalosis

Daniel S. Foy and Helio Autran de Morais

Metabolic alkalemia is characterized by an increase in bicarbonate concentration and base excess, an increase in pH, and a compensatory increase in carbon dioxide pressure. This article outlines indications for analysis, reference ranges, causes, and clinical signs of metabolic alkalosis. Algorithms for evaluation of patients with acid-base disorders and metabolic alkalosis are included.

A Quick Reference on Hyperchloremic Metabolic Acidosis

Silvia Funes and Helio Autran de Morais

Metabolic acidosis results from an increase in the concentration of a strong anion. Metabolic acidosis is divided into hyperchloremic metabolic acidosis and high anion gap acidosis based on the changes in the anion gap. Hyperchloremic metabolic acidosis is the result of chloride retention, excessive loss of sodium relative to chloride, or excessive gain of chloride relative to sodium. Clinical signs are related to the underlying disease that accompanies the metabolic acidosis. Treatment of hyperchloremic acidosis is based on addressing the underlying disease process.

A Quick Reference on High Anion Gap Metabolic Acidosis

Silvia Funes and Helio Autran de Morais

High anion gap (AG) metabolic acidoses can be identified by a decrease in pH, decrease in HCO$_3^-$ or base excess, and an increased AG. The AG represents the difference between unmeasured cations and unmeasured anions; it increases secondary to the accumulation of anions other than bicarbonate and chloride. The most common causes of high AG acidosis are renal failure, diabetic ketoacidosis, and lactic acidosis. Severe increases in concentration of phosphorus can cause hyperphosphatemic acidosis.

A Quick Reference on Hypernatremia

Julien Guillaumin and Stephen P. DiBartola

Hypernatremia most commonly is associated with water loss in excess of sodium or salt gain (typically associated with restriction of access to water). Most of the signs of hypernatremia arise from the central nervous system; the more rapid the development of hypernatremia, the more severe the neurologic signs are likely to be. Anorexia, lethargy, vomiting, muscular weakness, behavioral changes, disorientation, ataxia, seizures, coma, and death have been identified in dogs and cats with hypernatremia and hypertonicity.

A Quick Reference on Hyponatremia

Julien Guillaumin and Stephen P. DiBartola

The article focuses on causes of hyponatremia, including hypovolemia, diabetes mellitus and others. Hypovolemia is a major cause of
hyponatremia in veterinary medicine. Hypovolemia causes a decrease in effective circulating volume, triggering antidiuretic hormone (ADH) secretion and free water retention, and develops after gastrointestinal losses, renal losses, hemorrhagic shock, hypoadrenocorticism, and other causes of hypovolemia. The article reviews the clinical approach to diagnosing the cause of hyponatremia in critically ill patients, including recognition of the syndrome of inappropriate ADH secretion (SIADH).

A Quick Reference on Chloride
Andrea A. Bohn and Helio Autran de Morais

Chloride is an essential element, playing important roles in digestion, muscular activity, regulation of body fluids, and acid-base balance. As the most abundant anion in extracellular fluid, chloride plays a major role in maintaining electroneutrality. Chloride is intrinsically linked to sodium in maintaining osmolality and fluid balance and has an inverse relationship with bicarbonate in maintaining acid-base balance. It is likely because of these close ties that chloride does not get the individual attention it deserves; we can use these facts to simplify and interpret changes in serum chloride concentrations.

A Quick Reference on Hyperkalemia
Márcia Mery Kogika and Helio Autran de Morais

The clinical signs of hyperkalemia usually are less evident than hypokalemia. Arrhythmia and bradycardia could be the first changes noticed. Most cases of persistent hyperkalemia are associated with renal retention of potassium. Common causes for hyperkalemia include hypoadrenocorticism, ruptured bladder, and urethral or bilateral ureteral obstruction. Drugs such as angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, potassium-sparing diuretics, and nonsteroidal antiinflammatory drugs can also lead to hyperkalemia.

A Quick Reference on Hypokalemia
Márcia Mery Kogika and Helio Autran de Morais

Hypokalemia is more common than hyperkalemia and usually is caused by excessive losses of potassium from the kidneys or gastrointestinal tract. Serum potassium should be investigated in patients developing chronic or frequent vomiting or diarrhea, marked polyuria, muscle weakness, or unexpected cardiac arrhythmias, as well as in those undergoing therapy with insulin, diuretics, or total parenteral nutrition. Clinical signs develop when serum potassium deficit is moderate or severe.

A Quick Reference on Magnesium
Shane W. Bateman

This article serves as a quick reference on the distribution, handling, and supplementation of magnesium. It also lists the manifestations and causes of magnesium deficit and provides criteria for the diagnosis of a magnesium deficit.
In dogs, neoplasia is the most common cause of hypercalcemia, followed by primary hyperparathyroidism, chronic kidney disease, and hypoadrenocorticism. In cats, idiopathic hypercalcemia is the most common cause, followed by chronic kidney disease and then neoplasia. Prognosis and treatment ultimately depend on the cause of the hypercalcemia.

Primary hypoparathyroidism should be considered in dogs with vague signs, including tremors, facial rubbing, and seizures. Ionized hypocalcemia should be considered in dogs with protein-losing enteropathy, especially lymphangiectasia caused by hypovitaminosis D. Ionized hypocalcemia typically occurs only in advanced chronic kidney disease.

Phosphorus, or phosphate, is the body’s major intracellular anion involved in numerous biological processes. Most phosphate is intracellular, with the remaining amount contained within soft tissues and the extracellular space. Parathyroid hormone, calcitriol, calcitonin, and phosphatoninins regulate normal phosphate homeostasis by adjusting renal and/or gastrointestinal absorption and/or excretion. Hypophosphatemia occurs secondary to decreased gastrointestinal absorption, transcellular shifts, increased renal excretion, or some combination of these general mechanisms. Hyperphosphatemia results from decreased renal excretion, increased intake or iatrogenic administration, transcellular shifts, or some combination of these.

Assessment of health status and the course of treatment of patients are often determined by results obtained from analysis of blood parameters. Errors in results can occur and cause inappropriate interpretation of laboratory data and, therefore, disease states. Electrolytes and minerals are particularly prone to spurious results; therefore, it is critical that factors influencing inappropriate resulting be recognized, and steps taken to minimize them.

Hypercalcemia in cats is recognized with increased frequency, especially idiopathic hypercalcemia, which is the most common cause. Idiopathic hypercalcemia seems to be unique to the cat, not occurring in the dog as a specific syndrome. There are many causes of hypercalcemia, and
diagnosis relies on evaluation of clinical signs, physical examination, diagnostic imaging, serum biochemistry, urinalysis, and evaluation of calcium metabolic hormones. With an accurate diagnosis, treatment options can be tailored to the individual.

**Disorders of Sodium and Water Homeostasis**

Julien Guillaumin and Stephen P. DiBartola

This review article discusses normal and abnormal sodium balance in small animals. The terms and concepts central to understanding normal sodium and water balance are presented as well as of the physiology of body fluid compartments and the movement of fluid between those compartments. As dysnatremia is a very common disorder across the spectrum of critically ill patients, the main focus of the article is to present several clinical examples of both acute and chronic hypernatremia and hyponatremia and their practical, clinical management.

**Compensation for Acid-Base Disorders**

Chiara De Caro Carella and Helio Autran de Morais

Hydrogen concentration is a critical determinant of many physiologic functions and is tightly regulated. Any alteration in acid-base equilibrium sets into motion a compensatory response by either the lungs or the kidneys. The compensatory response attempts to return the ratio between $\text{P}_{\text{CO}_2}$ and $[\text{HCO}_3^-]$ to normal and thereby minimize the pH change. A primary increase or decrease in one component is associated with a predictable compensatory change in the same direction in the other component, and the expected compensation can be estimated clinically in dogs and cats.

**Update: Clinical Use of Plasma Lactate**

Íde Gillespie, Patricia G. Rosenstein, and Dez Hughes

Lactate is an essential, versatile metabolic fuel in cellular bioenergetics. In human emergency and critical care, lactate is used as a biomarker and therapeutic endpoint and evidence is growing in veterinary medicine supporting its clinical utility. Lactate production is a protective response providing ongoing cellular energy during tissue hypoperfusion or hypoxia and mitigating acidosis. Hence, hyperlactatemia is closely associated with disease severity but it is an epiphenomenon as the body attempts to protect itself. This article reviews lactate biochemistry, kinetics, pathophysiology, some practical aspects of measuring lactate, as well as its use in diagnosis, prognosis, and monitoring.

**Is Bicarbonate Therapy Useful?**

Kate Hopper

Despite concerns about the negative effects of metabolic acidosis, there is minimal evidence that sodium bicarbonate administration is an effective treatment. In addition, sodium bicarbonate therapy is associated with many adverse effects, including paradoxic intracellular acidosis, hypokalemia, hypocalcemia, hypernatremia, and hyperosmolality. Definitive recommendations regarding bicarbonate therapy are challenging as there is
little high-quality evidence available. In most clinical scenarios of metabolic acidosis, treatment efforts should focus on resolution of the underlying cause, and sodium bicarbonate therapy should be used with caution, if at all. An exception to this is kidney disease, wherein sodium bicarbonate therapy may have a valuable role.

Respiratory Acid–Base Disorders in the Critical Care Unit 351

Kate Hopper

The incidence of respiratory acid–base abnormalities in the critical care unit (CCU) is unknown, although respiratory alkalosis is suspected to be common in this population. Abnormal carbon dioxide tension can have many physiologic effects, and changes in P\textsubscript{CO\textsubscript{2}} may have a significant impact on outcome. Monitoring P\textsubscript{CO\textsubscript{2}} in CCU patients is an important aspect of critical patient assessment, and identification of respiratory acid–base abnormalities can be valuable as a diagnostic tool. Treatment of respiratory acid–base disorders is largely focused on resolution of the primary disease, although mechanical ventilation may be indicated in cases with severe respiratory acidosis.

Fluid Therapy

Fluid Therapy: Options and Rational Selection 359

Christopher G. Byers

Administration of appropriate types and volumes of parenteral fluids is of paramount importance when treating sick and debilitated patients, especially those fighting critical illness. Fluid selection and accurate calculations must be performed logically and accurately to maximize positive outcomes. Knowledge of fluid types, as well as the complex relationship of the body’s fluid compartments, helps clinicians develop rational fluid therapy plans for their patients.

Fluid Therapy for Pediatric Patients 373

Justine A. Lee and Leah A. Cohn

Young puppies and kittens have unique physiologic needs in regards to fluid therapy, which must address hydration, vascular fluid volume, electrolyte disturbances, or hypoglycemia. Pediatric patients have a higher fluid requirement compared with adults and can rapidly progress from mild dehydration to hypovolemia. Simultaneously, their small size makes overhydration a real possibility. Patient size complicates fluid administration because catheters used in larger pets may be difficult to place. Routes of fluid administration used in the neonate or pediatric patient include oral, subcutaneous, intraperitoneal, intraosseous, and intravenous. Clinicians should be aware of the pros and cons of each route.

Maintenance Fluid Therapy: Isotonic Versus Hypotonic Solutions 383

Bernie Hansen and Alessio Vigani

The goal of maintenance fluid therapy in small animals is to replace normal ongoing losses of water and salts when oral intake is withheld.
Hospitalized dogs and cats may have multiple stimuli for antidiuretic hormone release that disrupt normal osmoregulation and predispose to water retention. Severe illness promotes retention of both sodium and water as edema. Commercially available fluids have electrolyte concentrations that are very different from dietary maintenance requirements, and potential consequences include development of hypoosmolality, edema, or both when excesses of water or sodium are administered. Suggestions for tailoring fluid administration toward specific goals are provided.

Fluid Management in Patients with Trauma: Restrictive Versus Liberal Approach
Lee Palmer

Massive hemorrhage remains a major cause of traumatic deaths. The ideal fluid resuscitative strategy is much debated. Research has provided inconsistent results regarding which fluid strategy is ideal; the optimum fluid type, timing, and volume remains elusive. Aggressive large-volume resuscitation has been the mainstay based on controlled hemorrhage animal models. For uncontrolled hemorrhagic shock, liberal fluid resuscitative strategies exacerbate the lethal triad, invoke resuscitative injury, and increase mortality while more restrictive fluid strategies tend to ameliorate trauma-induced coagulopathy and favor a greater chance of survival. This article discusses the current evidence regarding liberal and restrictive fluid strategies for trauma.

The Colloid Controversy: Are Colloids Bad and What Are the Options?
Christine Wong and Amie Koenig

Biologic and synthetic colloid solutions are frequently used to increase oncotic pressure and to treat shock. Research has shown that each product has both risks and benefits. Hydroxyethyl starches have gained a reputation for increasing risk of death, acute kidney injury, and coagulation abnormalities in people, but additional studies are needed to see whether these concerns hold true in veterinary patients. This article reviews the risks and benefits of currently available products.

Perioperative Fluid Therapy
Denise Fantoni and Andre C. Shih

Anesthesia can lead to pathophysiologic changes that dramatically alter the fluid balance of the body compartments and the intravascular space. Fluid administration can be monitored and evaluated using static and dynamic indexes. Guidelines for fluid rates during anesthesia begin with 3 mL/kg/h in cats and 5 mL/kg/h in dogs. If at all possible, patients should be stabilized and electrolyte disturbances should be corrected before general anesthesia.

Lipid Therapy for Intoxications
Joris Henricus Robben and Marieke Annet Dijkman

This review discusses the use of intravenous lipid emulsion (ILE) in the treatment of intoxications with lipophilic agents in veterinary medicine. Despite growing scientific evidence that ILE has merit in the treatment of
certain poisonings, there is still uncertainty on the optimal composition of the lipid emulsion, the dosing, the mechanism of action, and the efficacy. Therefore, a critical view of the clinician on the applicability of this modality in intoxications is still warranted. The use of ILE therapy is advocated as an antidote in cases of intoxications with some lipophilic substances.

**Treating Hypoalbuminemia**  
Bobbi J. Conner

Video content accompanies this article at http://www.vetsmall.theclinics.com.

Hypoalbuminemia is common and associated with a variety of disease processes, including those leading to systemic inflammatory response syndrome, gastrointestinal disorders, hepatic disorders, and glomerular diseases. Some animals develop clinical signs directly caused by low serum albumin concentration. There is strong evidence that hypoalbuminemia is associated with worse outcomes; however, evidence justifying albumin supplementation is lacking. Severe adverse events are frequently reported with administration of human serum albumin and there is little evidence of benefit from other products. Most patients will not require administration of albumin-containing products. Clinicians should consider early enteral nutritional supplementation in critically ill patients.

**Fluid Therapy in Lung Disease**  
Elizabeth Rozanski and Alex Lynch

Fluid therapy is the cornerstone of supportive care in veterinary medicine. In dogs and cats with preexisting confirmed or suspected pulmonary disease, concerns may exist that the fluid therapy may impair gas exchange, either through increases in hydrostatic pressures or extravasation. Colloidal therapy is more likely to magnify lung injury compared with isotonic crystalloids. Radiographic evidence of fluid overload is a late-stage finding, whereas point-of-care ultrasound may provide earlier information that can also be assessed periodically at the patient side. Cases should be evaluated individually, but generally a conservative fluid therapy plan is preferred with close monitoring of its tolerance.

**Managing Fluid and Electrolyte Disorders in Kidney Disease**  
Cathy Langston

Because of the role of the kidneys in maintaining homeostasis in the body, kidney disease leads to derangements of fluid, electrolyte, and acid-base balance. The most effective therapy of a uremic crisis is careful management of fluid balance, which involves thoughtful assessment of hydration, a fluid treatment plan personalized for the specific patient, and repeated and frequent reassessment of fluid and electrolyte balance. Disorders of sodium, chloride, potassium, calcium, and phosphorus are commonly encountered in kidney disease and some may be life-threatening. Treatment of metabolic acidosis and nutritional support is frequently needed.
Fluid and Electrolyte Therapy in Diabetic Ketoacidosis 491
Elizabeth Thomovsky

Diabetic ketoacidosis is a dynamic disease that requires regular reassessment of an affected patient. Typical treatment regimens include crystalloid fluid therapy, insulin, and supplementation of dextrose, phosphorus, and potassium. This article presents an approach to and considerations for treatment of a diabetic ketoacidotic dog or cat.

Fluid and Electrolyte Therapy During Vomiting and Diarrhea 505
Luis Tello and Rossana Perez-Freytes

Fluid therapy is generally the most life saving and important therapeutic measure in a critical pet suffering from dehydration due to gastrointestinal losses (vomiting and/or diarrhea). Fluid therapy should be personalized to the patient’s history, complaint, physical examination and laboratory findings. It is directed to the patients needs and modified based of the physical and laboratory findings until fluid therapy resuscitation end points are achieved.

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