A Quick Reference on Respiratory Alkalosis

Rebecca A. Johnson, MS, DVM, PhD

KEYWORDS

• Alveolar hyperventilation • Arterial blood gas • Hypocapnia
• Metabolic compensation • Respiratory alkalosis

KEY POINTS

- Respiratory alkalosis, or primary hypocapnia, occurs when alveolar ventilation exceeds that required to eliminate the carbon dioxide produced by tissues.
- Concurrent decreases in $Paco_2$, increases in pH, and compensatory decreases in blood HCO_3^- levels are associated with respiratory alkalosis.
- Respiratory alkalosis can be acute or chronic, with metabolic compensation initially consisting of cellular uptake of HCO_3^- and buffering by intracellular phosphates and proteins.
- Chronic respiratory alkalosis results in longer-lasting decreases in renal reabsorption of HCO_3^-; the arterial pH can approach near-normal values.

Pco_2: RESPIRATORY ALKALOSIS: QUICK REFERENCE

- Respiratory alkalosis, or primary hypocapnia, occurs when alveolar ventilation exceeds that required to eliminate the carbon dioxide produced by tissues.
- Concurrent decreases in $Paco_2$, increases in pH, and compensatory decreases in blood HCO_3^- concentration are associated with respiratory alkalosis.
- Respiratory alkalosis can be acute or chronic. Metabolic compensation initially consists of cellular uptake of HCO_3^- and buffering by intracellular phosphates and proteins.
- Chronic respiratory alkalosis results in longer-lasting decreases in renal reabsorption of HCO_3^-; the arterial pH can approach near-normal values.

ANALYSIS

- Typical reference range: Normal arterial blood gas values for dogs and cats inspiring room air are presented in **Table 1**.
- Diagnosis: Respiratory alkalosis is a common finding in compromised patients, but clinical signs specifically attributed to respiratory alkalosis are uncommon.

The author has nothing to disclose.
Department of Surgical Sciences, University of Wisconsin – Madison, 2015 Linden Drive, Madison, WI 53706, USA
E-mail address: rebecca.johnson@wisc.edu

Vet Clin Small Anim 47 (2017) 181–184
http://dx.doi.org/10.1016/j.cvsm.2016.10.005 **vetsmall.theclinics.com**

Table 1
Typical reference ranges for normal arterial blood gas values for dogs and cats inspiring room air

	Dog	Cat
pH	7.407 (7.351–7.463)	7.386 (7.310–7.462)
$Paco_2$, mm Hg	36.8 (30.8–42.8)	31.0 (25.2–36.8)
HCO_3^-, mEq/L	22.2 (18.8–25.6)	18.0 (14.4–21.6)
Pao_2, mm Hg	92.1 (80.9–103.3)	106.8 (95.4–118.2)

Data from Haskins SC. Blood gases and acid-base balance: clinical interpretation and therapeutic implications. In: Kirk RW, editor. Current veterinary therapy VIII. Philadelphia: WB Saunders; 1983. p. 201.

$Paco_2$ should be evaluated in patients with apparent hyperventilation to diagnose respiratory alkalosis ($Paco_2$ values <35 mm Hg).[1]

- Danger values
  - Acute respiratory alkalosis presents more danger than chronic respiratory alkalosis because metabolic compensation is very efficient in chronic respiratory alkalosis.
  - When arterial pH approaches approximately 7.6 (possible only in acute situations) or $Paco_2$ decreases to less than 20 to 25 mm Hg, arteriolar vasoconstriction results, potentially reducing cerebral and myocardial blood flow.[2]
  - Acute alkalemia shifts the oxygen-hemoglobin dissociation curve to the left, increasing the affinity of hemoglobin for oxygen and reducing the release of oxygen in the tissues.[3]
  - For each 10-mm Hg decrease in $Paco_2$ seen in *acute* respiratory alkalosis, serum potassium levels decrease 0.4 to 0.6 mEq/L and patients may exhibit signs of hypokalemia (eg, neuromuscular weakness, arrhythmias).[4]
- Artifacts: Correct sample handling is imperative to reduce falsely low $Paco_2$ readings.[5]
  - Pco_2 of room air is very low, and the presence of air bubbles within the sample reduces the Pco_2 and increases blood pH.
  - Sample dilution by large amounts of heparin erroneously decreases Pco_2.
- Drug effects: Pharmacologic agents, such as salicylates, corticosteroids, and xanthines (eg, aminophylline), may produce respiratory alkalosis through activation of the respiratory centers.[1]

RESPIRATORY ALKALOSIS

- Causes: Respiratory alkalosis and hypocapnia occur with alveolar hyperventilation resulting from the following (**Box 1**)[1]:
  - Stimulation of peripheral chemoreceptors by hypoxemia
  - Activation of pulmonary stretch receptors or nociceptors independent of hypoxemia
  - Direct activation of central respiratory centers
  - Overzealous mechanical ventilation
  - Fear, excitement, pain, fever, or sepsis
  - After treatment of metabolic acidosis, because hyperventilation may still be present for 24 to 48 hours after therapy
- Signs: Clinical signs in patients that have respiratory alkalosis are mainly attributable to the underlying disease process and are uncommon because of the efficient metabolic compensation that occurs.[1]

Box 1
Causes of respiratory alkalosis

Hypoxemia and Stimulation of Peripheral Chemoreceptors[a]
- Right-to-left shunting, decreased Fio_2, congestive heart failure, severe anemia, severe hypotension, decreased cardiac output, ventilation-perfusion mismatch (eg, pneumonia, pulmonary thromboembolism, pulmonary fibrosis, pulmonary edema)

Activation of Stretch/Nociceptors Independent of Hypoxemia[a]
- Pneumonia, pulmonary thromboembolism, interstitial lung disease, pulmonary edema

Centrally Mediated Hyperventilation
- Liver disease, hyperadrenocorticism, sepsis, pharmacologic agents (eg, salicylates, corticosteroids, xanthines), progesterone, recovery from metabolic acidosis, central nervous system disease, exercise, heatstroke

Overzealous Mechanical Ventilation

Situations Causing Pain, Fear, or Anxiety[a]

Fever

Sepsis

[a] Most important causes in small animal medicine.

  - Tachypnea may be the only clinical sign, especially in patients that have chronic hypocapnia.
  - In some patients that have acute alkalemia, cardiac arrhythmias, confusion, and seizures from arteriolar vasoconstriction with decreased cerebral or myocardial perfusion may be seen.
  - Alkalemia-induced translocation of potassium into cells with additional renal and extrarenal losses may produce signs attributable to hypokalemia (eg, neuromuscular weakness, arrhythmias, polyuria) in acute respiratory alkalosis.
- Treatment: Treatment is directed toward the underlying cause of the hyperventilation; no other treatment is effective.[1]
- Stepwise approach: An algorithm for evaluation of general acid-base disorders, including respiratory alkalosis, is shown in **Fig. 1**. A more specific algorithm for the differential diagnosis of acute versus chronic respiratory alkalosis is presented in **Fig. 2**. Note that the decreases in HCO_3^- seen with acute metabolic compensation are similar in both dogs and cats. The degree of metabolic

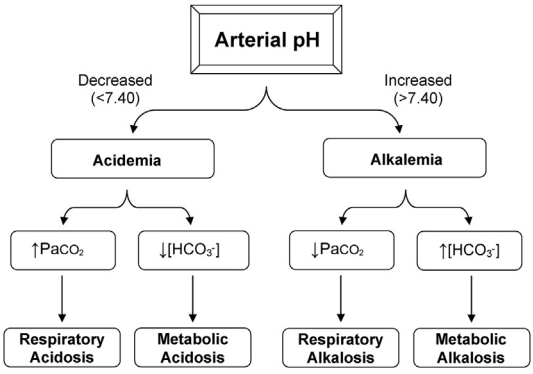

Fig. 1. Algorithm for evaluation of patients with respiratory acid-base disorders.

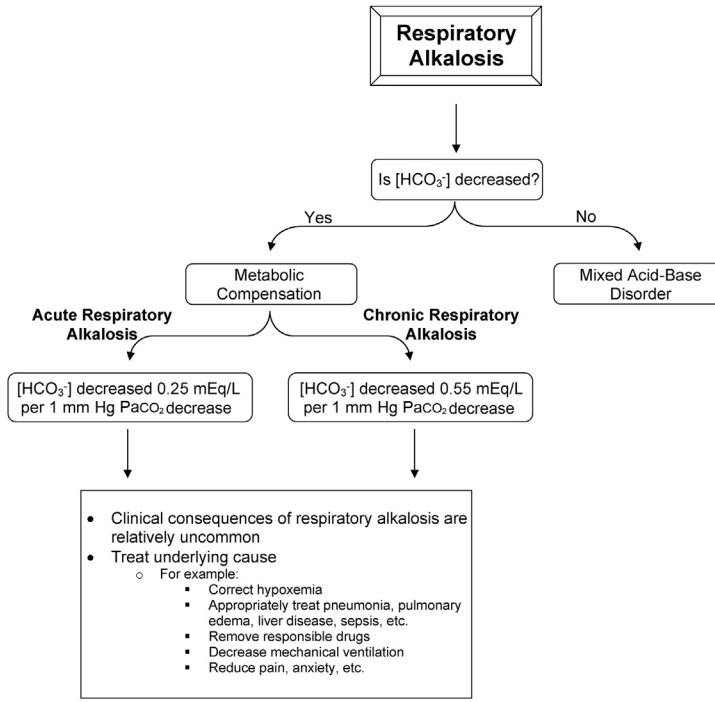

Fig. 2. Algorithm for evaluation of patients with acute versus chronic respiratory alkalosis.

compensation in chronic respiratory alkalosis in cats is not known; however, the pH is frequently normal or just slightly alkalemic.

REFERENCES

1. Johnson RA, de Morais HA. Respiratory acid-base disorders. In: DiBartola SP, editor. Fluid, electrolyte, and acid-base disorders in small animal practice. 4th edition. St Louis (MO): Elsevier; 2012. p. 292–8.
2. Adrogué HJ, Madias NE. Management of life-threatening acid-base disorders. N Engl J Med 1998;338:26–34.
3. Hodgkin JE, Soeprono FF, Chan DM. Incidence of metabolic alkalemia in hospitalized patients. Crit Care Med 1980;8:725–8.
4. Muir WW 3rd, Wagner AE, Buchanan C. Effects of acute hyperventilation on serum potassium in the dog. Vet Surg 1990;19:83–7.
5. Rieser TM. Arterial and venous blood gas analyses. Top Companion Anim Med 2013;28:86–90.