A Quick Reference on Respiratory Acidosis

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KEYWORDS

• Alveolar hypoventilation • Arterial blood gas • Hypercapnia
• Metabolic compensation • Respiratory acidosis

KEY POINTS

- 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 $Paco_2$, decreases in pH and compensatory increases in blood HCO_3^- concentration are associated with respiratory acidosis.
- Respiratory acidosis can be acute or chronic, with initial metabolic compensation to increase HCO_3^- concentrations by intracellular buffering.
- Chronic respiratory acidosis results in longer lasting increases in renal reabsorption of HCO_3^-.
- Alveolar hypoventilation and resulting respiratory acidosis may also be associated with hypoxemia, especially evident when patients are inspiring room air (20.9% O_2).

Pco_2: RESPIRATORY ACIDOSIS – QUICK REFERENCE

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Analysis

Typical reference range

Normal arterial blood gas values for dogs and cats inspiring room air are presented in Table 1.

Diagnosis

$Paco_2$ should be evaluated in patients suspected of having respiratory failure or increased carbon dioxide production with concurrent alveolar hypoventilation ($Paco_2$ values >45 mm Hg).[1]

Danger values

- Moderately increased $Paco_2$ (60–70 mm Hg) causes sympathetic activation, decreases in myocardial contractility and systemic vascular resistance, increases in cardiac output, and potential tachyarrhythmias.[2–4]
- Extremely high levels of $Paco_2$ (>95 mm Hg) produce disorientation, narcosis, and coma.[5]
- Cerebral blood flow and intracranial pressure increase linearly with increases in $Paco_2$.[6]
- In acute respiratory acidosis, pH can decrease to less than 7.2.
- Extremely acute hypoventilation (eg, cardiopulmonary arrest, airway obstruction) may result in life-threatening hypoxemia within 4 minutes, whereas hypercapnia would not develop for 10 to 15 minutes.[7]

Artifacts

Correct sample handling is imperative to reduce falsely elevated $Paco_2$ readings.

- Pco_2 increases and pH decreases as the sample waits before analysis (20–30 minutes); the rate of increase is quicker at 25°C than at 4°C.[8]
- Failure to obtain an arterial sample is common owing to the close proximity of the arteries and veins, making it easy to obtain a venous sample accidentally; a known paired venous sample should be obtained for comparison if there are questionable results.[9]

Drug effects

Many drugs produce respiratory acidosis by means of respiratory center depression (eg, opioids, barbiturates, inhalant anesthetics) or neuromuscular dysfunction (eg, organophosphates, aminoglycosides used in conjunction with anesthetics).

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)
Po_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.

Respiratory Acidosis

Causes

Most cases result from disturbances in removal of carbon dioxide by the lungs.[1]

- Respiratory acidosis and hypercapnia occur with alveolar hypoventilation resulting from any disruption in the neural control of ventilation, breathing mechanics, or alveolar gas exchange (**Box 1**).
- Although increased production of CO_2 is a possible cause of respiratory acidosis, it is usually associated with impaired alveolar ventilation.
- Acute hypercapnia and respiratory acidosis mainly result from sudden, severe respiratory system (eg, pneumothorax), neurologic system (eg, spinal cord injury), or neuromuscular (eg, botulism) disease.
- Chronic respiratory acidosis has many potential causes that can lead to sustained hypercapnia (see **Box 1**).

Signs

Many clinical signs in patients that have respiratory acidosis are attributable to the underlying disease process itself and not necessarily to the hypercapnia.[1]

- Chronic, compensated patients may exhibit very mild signs; thus, subjective clinical assessment of the patient is not reliable in making a diagnosis of respiratory acidosis.
- In some patients, hypercapnia results in tachyarrhythmias (including ventricular tachycardia), increased cardiac output, and "brick red" mucous membranes associated with vasodilation.

Box 1
Causes of respiratory acidosis

Large airway obstruction[a]
- Physical/mechanical obstruction (eg, aspiration, mass lesion, plug or kink in endotracheal tube), tracheal collapse, brachycephalic syndrome, asthma, chronic obstructive pulmonary disease

Intrinsic pulmonary and small airway disease[a]
- Severe pulmonary edema, pulmonary thromboembolism, pneumonia, asthma, chronic obstructive pulmonary disease

Respiratory center depression[a]
- Drug induced (eg, opioids, barbiturates, inhalant anesthetics), neurologic disease (eg, brainstem or cervical spinal cord lesion)

Restrictive extrapulmonary disorders[a]
- Diaphragmatic hernia, pleural space disease (eg, pneumothorax, pleural effusion)

Neuromuscular disease
- Myasthenia gravis, tetanus, botulism, tick paralysis, electrolyte abnormalities (eg, hypokalemia), drug induced (eg, organophosphates, aminoglycosides used in conjunction with anesthetics)

Increased CO_2 production with impaired alveolar ventilation
- Heatstroke, malignant hyperthermia

Ineffective mechanical ventilation

Marked obesity (Pickwickian syndrome)

[a] Most important causes in small animal practice.

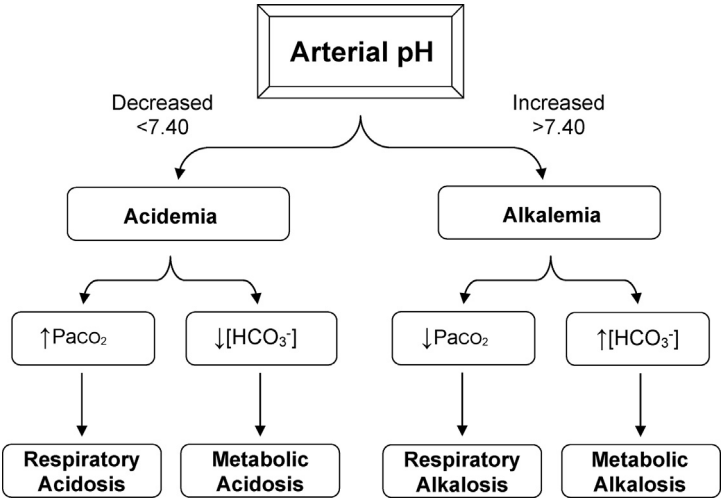

Fig. 1. Algorithm for evaluation of patients with respiratory acid–base disorders.

- Patients may develop a fast shallow breathing pattern with inadequate tidal volumes. Their central nervous function may deteriorate, and they may seem anxious, restless, disoriented, or somnolent.
- Hypercapnia shifts the oxygen–hemoglobin dissociation curve to the right, promoting oxygen unloading at the tissues and enhancing oxygen delivery and carrying capacity.[10]

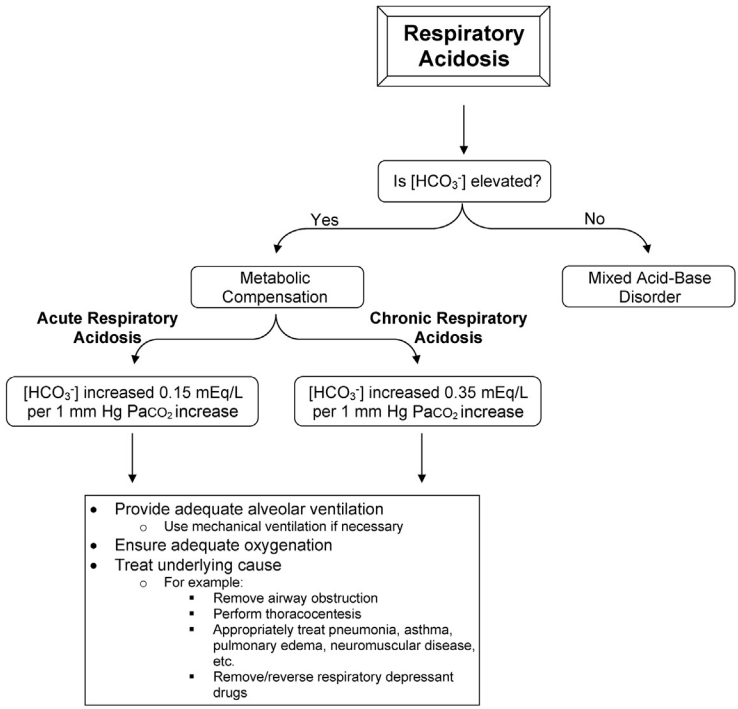

Fig. 2. Algorithm for evaluation of patients with acute versus chronic respiratory acidosis.

Stepwise approach

An algorithm for evaluation of general acid–base disorders, including respiratory acidosis, is shown in **Fig. 1**. A more specific algorithm for the differential diagnosis of acute versus chronic respiratory acidosis is presented in **Fig. 2**. Note that the increases in HCO_3^- seen with acute metabolic compensation are similar in both dogs and cats. However, the degree of metabolic compensation in chronic respiratory acidosis in cats is not known.

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