**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 \( \text{PacO}_2 \), decreases in pH and compensatory increases in blood \( \text{HCO}_3^- \) concentration are associated with respiratory acidosis.
- Respiratory acidosis can be acute or chronic, with initial metabolic compensation to increase \( \text{HCO}_3^- \) concentrations by intracellular buffering.
- Chronic respiratory acidosis results in longer lasting increases in renal reabsorption of \( \text{HCO}_3^- \).
- Alveolar hypoventilation and resulting respiratory acidosis may also be associated with hypoxemia, especially evident when patients are inspiring room air (20.9% \( \text{O}_2 \)).

**\( \text{PCO}_2 \): RESPIRATORY ACIDOSIS – QUICK REFERENCE**
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The author has nothing to disclose.
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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

Paco₂ should be evaluated in patients suspected of having respiratory failure or increased carbon dioxide production with concurrent alveolar hypoventilation (Paco₂ values >45 mm Hg).¹

Danger values

- Moderately increased Paco₂ (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 Paco₂ (>95 mm Hg) produce disorientation, narcosis, and coma.⁵
- Cerebral blood flow and intracranial pressure increase linearly with increases in Paco₂.⁶
- 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 Paco₂ readings.

- PCO₂ 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>
<thead>
<tr>
<th>Table 1</th>
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<tbody>
<tr>
<td>Typical reference ranges for normal arterial blood gas values for dogs and cats inspiring room air</td>
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<tr>
<td>Paco₂ (mm Hg)</td>
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<tr>
<td>[HCO₃⁻] (mEq/L)</td>
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<tr>
<td>Po₂ (mm Hg)</td>
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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.

<table>
<thead>
<tr>
<th>Box 1</th>
<th>Causes of respiratory acidosis</th>
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<tbody>
<tr>
<td>Large airway obstruction(^a)</td>
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<tr>
<td>Physical/mechanical obstruction (eg, aspiration, mass lesion, plug or kink in endotracheal tube), tracheal collapse, brachycephalic syndrome, asthma, chronic obstructive pulmonary disease</td>
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<tr>
<td>Intrinsic pulmonary and small airway disease(^a)</td>
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<tr>
<td>Severe pulmonary edema, pulmonary thromboembolism, pneumonia, asthma, chronic obstructive pulmonary disease</td>
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<tr>
<td>Respiratory center depression(^a)</td>
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</tr>
<tr>
<td>Drug induced (eg, opioids, barbiturates, inhalant anesthetics), neurologic disease (eg, brainstem or cervical spinal cord lesion)</td>
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<tr>
<td>Restrictive extrapulmonary disorders(^a)</td>
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<tr>
<td>Diaphragmatic hernia, pleural space disease (eg, pneumothorax, pleural effusion)</td>
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<tr>
<td>Neuromuscular disease</td>
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<td>Myasthenia gravis, tetanus, botulism, tick paralysis, electrolyte abnormalities (eg, hypokalemia), drug induced (eg, organophosphates, aminoglycosides used in conjunction with anesthetics)</td>
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<tr>
<td>Increased CO(_2) production with impaired alveolar ventilation</td>
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<td>Heatstroke, malignant hyperthermia</td>
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<tr>
<td>Ineffective mechanical ventilation</td>
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<td>Marked obesity (Pickwickian syndrome)</td>
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\(^a\) Most important causes in small animal practice.
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.\textsuperscript{10}

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

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- Hypercapnia shifts the oxygen–hemoglobin dissociation curve to the right, promoting oxygen unloading at the tissues and enhancing oxygen delivery and carrying capacity.\textsuperscript{10}

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

- Provide adequate alveolar ventilation
  - Use mechanical ventilation if necessary
- Ensure adequate oxygenation
- Treat underlying cause
  - For example:
    - Remove airway obstruction
    - Perform thoracocentesis
    - Appropriately treat pneumonia, asthma, pulmonary edema, neuromuscular disease, etc.
    - Remove/reverse respiratory depressant drugs
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 HCO₃⁻ 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.

REFERENCES