

**Guidelines
For the Management of
Respiratory Acidosis**

By

**Dr. Sinan Butrus
F.I.C.M.S**

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Respiratory acidosis is an acid-base balance disturbance due to alveolar hypoventilation, leading to increased partial pressure of arterial carbon dioxide (PaCO₂) above the normal range (>45 mm Hg) leading to a decrease in bicarbonate (HCO₃⁻)/PaCO₂ ratio, thereby decreasing the pH lower than 7.35.

Hypercapnia also occurs in metabolic alkalosis, which is a response to the high arterial pH, which distinguishes the two.

Acute respiratory acidosis- occurs when there is abrupt failure of ventilation leading to sudden and sharp increase in PaCO₂ (>45 mm Hg) usually associated with severe hypoxemia and acidemia (pH < 7.35).

Chronic respiratory acidosis (>24 h) - occurs with gradual and sustained increase in PaCO₂ above the upper limit of the reference range, with a normal or near-normal pH secondary to renal compensation and an elevated serum bicarbonate levels (>30 mEq/L).

The clinical manifestations of respiratory acidosis are often those of the underlying disorder & depending on the severity and on the rate of development of hypercapnia.

Respiratory acidosis does not have a great effect on serum electrolyte levels.

Some small effects might occur in calcium and potassium levels. Acidosis decreases binding of calcium to albumin and tends to increase serum ionized calcium levels.

In addition, acidemia causes an extracellular shift of potassium. Respiratory acidosis, however, rarely causes clinically significant hyperkalemia

History

The following questions should be asked in the course of the history:

- * Does the patient have a history of headaches? (With chronic hypercapnia, headaches typically occur at night time or when the patient awakens in the morning)
- * Does the patient have disturbed sleep patterns? (Chronic hypercapnia can disturb sleep patterns, leading to a reversed sleep-wake cycle)
- * Is the patient irritable or anxious, or is he or she having trouble concentrating?
- * Does the patient have an acute change in mental status (e.g., signs of stroke, postictal state)? If so, is the change in mental status associated with a fever, which may suggest encephalitis or meningitis? Does the patient have signs of increased intracranial pressure (e.g., headaches, visual changes, or emesis)?
- * Does the patient have a long-standing pulmonary disease, such as broncho-pulmonary dysplasia, cystic fibrosis, asthma, or emphysema?
- * Does the patient have a possible or known exposure to sedatives (e.g., narcotics, benzodiazepines, tricyclic antidepressants) or recovering from a procedure in which general anesthesia was used?
- * Does the patient have symptoms of neuromuscular weakness or paralysis? (Such symptoms might include bulbar dysfunction suggesting myasthenia gravis, proximal or distal weakness suggesting a myopathy or Guillain-Barré syndrome, and apnea associated with a traumatic injury suggesting an injury to the cervical spinal cord)
- * Does the patient have a potential for an anaphylactic reaction?
- * Does the patient have a potential traumatic mechanism leading to brain injury?

Physical Examination

Neurologic findings:

- Early signs include anxiety, disturbed consciousness. Coma occurs when the arterial partial pressure of carbon dioxide (PaCO_2) exceeds 70 mm Hg
- Tremor, myoclonus, or asterixis (flapping tremor) are occasionally seen
- Brisk deep tendon reflexes are seen in mild-to-moderate respiratory acidosis while depressed deep tendon reflexes are seen in severe respiratory acidosis
- Papilledema or blurring of the optic disc may be present

Cardiovascular findings:

- Tachycardia & bounding arterial pulses
- Hypotension (severe respiratory acidosis or acidemia and hypoxemia)
- Cutaneous findings like warm, flushed, or mottled skin & diaphoresis

Respiratory findings:

- Tachypnea, dyspnea, or deep labored breaths may be observed with accessory muscle use and nasal flaring however, in central or peripheral nervous system disease, respiratory distress may not be present
- Decreased aeration, crackles, wheezes, or other signs of airway disease
- Clubbing is a sign of chronic respiratory disease

Work up

1. Arterial blood gas (ABG) analysis & bicarbonate level:

Acidemia is documented by the presence of a decreased pH (< 7.35) on ABG analysis. High partial pressure of arterial carbon dioxide (PaCO_2) (>45 mm Hg) indicates a respiratory etiology of the acidemia whereas if pH is ≥ 7.45 , the elevated PaCO_2 indicate compensation for metabolic alkalosis and is not a primary process.

The serum HCO_3^- level and pH can be helpful in distinguishing acute hypercapnia from chronic hypercapnia.

In acute respiratory acidosis; for every 10-mm Hg increase in PaCO_2 , pH decreases by 0.08 & HCO_3^- concentration increases by 1 mEq/L. i.e. If PaCO_2 increases acutely to 80 mm Hg, the pH is 7.12, and HCO_3^- is 28 mEq/L. The acute change in bicarbonate is relatively modest and is generated by the blood, extracellular fluid, and cellular buffering system

In chronic respiratory acidosis; for every 10-mm Hg increase in PaCO_2 ; pH decreases by 0.03, while HCO_3^- concentration increases 4 mEq/L for every 10-mm Hg increase in PaCO_2 greater than 40 mm Hg. i.e. if the PaCO_2 is 80 mm Hg, the pH is 7.28, and the HCO_3^- value is 40 mEq/L. The greater change in bicarbonate in chronic respiratory acidosis is accomplished by the kidneys. The response begins in 6-12 hours, but 3-5 days pass before maximal compensation occurs.

The HCO_3^- resorption process is very efficient. If a patient with chronic hypercapnia has a pH less than 7.20, a superimposed acute-on-chronic respiratory acidosis or a concomitant metabolic acidosis is most likely occurring as well.

2. Send for serum electrolytes and biochemistries for abnormalities associated with muscle weakness (e.g., hypophosphatemia, hypokalemia, hypomagnesemia, and hypocalcemia), thyroid studies, a complete blood count (CBC), and drug and toxicology screens.

3. Pulmonary function test measurements are required for the diagnosis of obstructive lung

disease and for assessment of the severity of disease.

4. Electromyography (EMG) and measurement of nerve conduction velocity (NCV) are useful in diagnosing neuromuscular disorders

5. Radiography, computed tomography (CT), and fluoroscopy of the chest to determine causes of respiratory acidosis

Radiologic studies of the brain (CT and MRI) should be considered if a central cause of hypoventilation and respiratory acidosis is suspected.

Treatment

- primarily directed to the underlying disorder or pathophysiologic process of respiratory acidosis. Mechanical ventilator is indicated if ventilation fails.

- The criteria for admission to the intensive care unit (ICU) include patient confusion, lethargy, respiratory muscle fatigue, and a low pH (< 7.25).

- Oxygen therapy should be used with caution because it may worsen hypercapnia which is best avoided by titrating oxygen delivery to maintain oxygen saturation in the low 90% range and partial arterial pressure of oxygen (PaO₂) in the range of 60-65 mm Hg.

Rapid correction of chronic hypercapnia as can result in metabolic alkalemia; alkalization of the cerebrospinal fluid (CSF) can result in seizures.

Infusion of sodium bicarbonate is rarely indicated. This measure may be considered after cardiopulmonary arrest with an extremely low pH (< 7.0-7.1)

- Bronchodilators such as beta agonists (e.g., albuterol and salmeterol), anticholinergic agents (e.g., ipratropium bromide and tiotropium), and methylxanthines (e.g. theophylline) & steroids are helpful in treating patients with obstructive airway disease

- Medroxyprogesterone increases central respiratory drive and may be effective in treating obesity-hypoventilation syndrome (OHS)

- Naloxone may be used to reverse the effects of narcotics. Flumazenil may be used to reverse the effects of benzodiazepines

Metabolic Acidosis vs. Respiratory Acidosis

• Both conditions are increases of acidity of blood, but metabolic acidosis has more causes & is more severe than respiratory acidosis.

• Bicarbonate concentration can be either normal or increased in respiratory acidosis, whereas metabolic acidosis is associated with low level of bicarbonates

Further reading

Acute respiratory acidosis is present when an abrupt failure of ventilation due to depression of the central respiratory center by one or another of the following:

* Central nervous system disease or drug-induced respiratory depression

* Inability to ventilate adequately, due to a neuromuscular disease or paralysis

(e.g., myasthenia gravis, amyotrophic lateral sclerosis ALS, Guillain-Barré syndrome, muscular dystrophy)

* Airway obstruction, usually related to asthma or chronic obstructive pulmonary disease (COPD)

Chronic respiratory acidosis may be secondary to many disorders, including COPD, obesity hypoventilation syndrome OHS (Pickwickian syndrome), neuromuscular disorders such as ALS, and severe restrictive ventilatory defects such as are observed in interstitial fibrosis and thoracic skeletal deformities. Hypoventilation in COPD involves multiple mechanisms, including the following:

- * Decreased responsiveness to hypoxia and hypercapnia
- * Increased ventilation-perfusion mismatch leading to increased dead space ventilation
- * Decreased diaphragmatic function due to fatigue and hyperinflation

Chronic respiratory acidosis also may be secondary to

Causes of respiratory acidosis

- 1) CNS depression
 1. Opioids
 2. Oxygen in patient with chronic hypercapnia
 3. Central sleep apnea
 4. CNS lesion
 5. Extreme obesity (Pickwickian syndrome)
- 2) Neuromuscular disorders
 1. Myasthenia gravis
 2. Guillain-Barre
 3. ALS
 4. Poliomyelitis
 5. Muscular dystrophy
 6. Multiple Sclerosis
- 3) Chest wall or Thoracic Cage Abnormality
 1. Kyphoscoliosis
 2. Flail Chest
 3. Myxedema
 4. Rib Fracture
 5. Scleroderma
- 4) Disorders affecting gas exchange
 1. COPD
 2. Severe asthma or pneumonia
 3. Pneumothorax or Hemothorax
 4. Acute pulmonary edema
- 5) Airway obstruction
 1. Aspiration of foreign body
 2. Obstructive sleep apnea
 3. Laryngospasm

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