RESPIRATORY ALKALOSIS (PRIMARY CARBONIC ACID DEFICIT)

Respiratory alkalosis is a loss of carbon dioxide ($\text{P}_{\text{CO}_2} < 35 \text{ mm Hg}$) with a resultant decrease of carbonic acid ($\text{H}_2\text{CO}_3$) due to a marked increase in the rate of respirations. The two primary mechanisms that trigger hyperventilation are (1) hypoxemia and (2) direct stimulation of the central respiratory center of the brain (such as occur with high fever, head trauma/CNS lesions, early salicylate intoxication).

Compensatory mechanisms include decreased respiratory rate (if the body is able to respond to the drop in $\text{Pa}_{\text{CO}_2}$), increased renal excretion of bicarbonate, and retention of hydrogen. It is the most frequently occurring acid-base imbalance in hospitalized patients, with the elderly at increased risk because of high incidence of pulmonary disorders and alterations in neurological status.

CARE SETTING

This condition does not occur in isolation, but rather is a complication of a broader problem and usually requires inpatient care in a medical/surgical or subacute unit.

RELATED CONCERNS

Plans of care specific to predisposing factors, e.g.:
- Anemias (iron deficiency, pernicious, aplastic, hemolytic)
- Cirrhosis of the liver
- Craniocerebral trauma
- Hyperthyroidism
- Fluid and electrolyte imbalances
- Heart failure: chronic
- Pneumonia: microbial
- Sepsis/septicemia
- Ventilatory assistance (mechanical)

OTHER CONCERNS

- Metabolic acidosis
- Metabolic alkalosis

Patient Assessment Database

Dependent on underlying cause.

CIRCULATION

**May report:**
- History/presence of anemia
- Palpitations

**May exhibit:**
- Hypotension
- Tachycardia, irregular pulse/dysrhythmias

EGO INTEGRITY

**May exhibit:**
- Extreme anxiety (most common cause of hyperventilation)

FOOD/FLUID

**May report:**
- Dry mouth
- Nausea/vomiting

**May exhibit:**
- Abdominal distension (elevating diaphragm as with ascites, pregnancy)
- Vomiting

NEUROSENSORY

**May report:**
- Headache, tinnitus
- Numbness/tingling of face, hands, and toes; circumoral and generalized paresthesia
- Lightheadedness, syncope, vertigo, blurred vision
May exhibit: Confusion, restlessness, obtunded responses, coma
Hyperactive reflexes, positive Chvostek’s sign, tetany, seizures
Heightened sensitivity to environmental noise and activity
Muscle weakness, unsteady gait

PAIN/DISCOMFORT
May report: Muscle spasms/cramps, epigastric pain, precordial pain (tightness)

RESPIRATION
May report: Dyspnea
History of asthma, pulmonary fibrosis
Recent move/visit to location at high altitude
May exhibit: Tachypnea; rapid, shallow breathing; hyperventilation (often 40 or more respirations/minute)
Intermittent periods of apnea

SAFETY
May exhibit: Fever

TEACHING/LEARNING
May report: Use of salicylates/salicylate overdose, catecholamines, theophylline
Discharge plan considerations: DRG projected mean length of inpatient stay: 5.4 days
May require change in treatment/therapy of underlying disease process/condition
Refer to section at end of plan for postdischarge considerations.

DIAGNOSTIC STUDIES
Arterial pH: Greater than 7.45 (may be near normal in chronic stage).
Bicarbonate (HCO₃⁻): Normal or decreased; less than 25 mEq/L (compensatory mechanism).
Paco₂: Decreased, less than 35 mm Hg (primary).
Serum potassium: Decreased.
Serum chloride: Increased.
Serum calcium: Decreased.
Urine pH: Increased, greater than 7.0.
Screening tests as indicated to determine underlying cause, e.g.:
CBC: May reveal severe anemia (decreasing oxygen-carrying capacity).
Blood cultures: May identify sepsis (usually Gram-negative).
Blood alcohol: Marked elevation (acute alcoholic intoxication).
Toxicology screen: May reveal early salicylate poisoning.

NURSING PRIORITIES
1. Achieve homeostasis.
2. Prevent/minimize complications.
3. Provide information about condition/prognosis and treatment needs as appropriate.

DISCHARGE GOALS
1. Physiological balance restored.
2. Free of complications.
3. Condition, prognosis, and treatment needs understood.
4. Plan in place to meet needs after discharge.
NURSING DIAGNOSIS: Gas Exchange, impaired
May be related to
Ventilation perfusion imbalance (e.g., altered oxygen supply, altered blood flow, altered oxygen-carrying capacity of blood, alveolar-capillary membrane changes)

Possibly evidenced by
Dyspnea, tachypnea
Changes in mentation
Hypocapnia, tachycardia
Hypoxia

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:
Electrolyte and Acid-Base Balance (NOC)
Demonstrate improved ventilation and adequate oxygenation of tissue as evidenced by ABGs within patient’s acceptable limits and absence of symptoms of respiratory distress.
Verbalize understanding of causative factors and appropriate interventions.
Participate in treatment regimen within level of ability/situation.

ACTIONS/INTERVENTIONS

Acid-Base Management: Respiratory Alkalosis (NIC)

Independent
Monitor respiratory rate, depth, and effort; ascertain cause of hyperventilation if possible, e.g., anxiety, pain, improper ventilator settings.

Encourage patient to breathe slowly and deeply. Speak in a low, calm tone of voice; provide safe environment.

Assess level of awareness/cognition and note neuromuscular status, e.g., strength, tone, reflexes, and sensation.

Demonstrate appropriate breathing patterns, if appropriate, and assist with respiratory aids, e.g., rebreathing mask/bag.

Provide comfort measures; encourage use of meditation and visualization. Use tepid sponge bath/cool cloths.

Provide safety/seizure precautions, e.g., bed in low position, padded side rails, frequent observation.

RATIONALE

Identifies alterations from usual breathing pattern and influences choice of intervention.

May help reassure and calm the agitated patient, thereby aiding the reduction of respiratory rate. Assists patient to regain control.

Decreased mentation (mild to severe) and tetany or seizures may occur when alkalosis is severe.

Decreasing the rate of respirations can halt the “blowing off” of CO$_2$ elevating Pco$_2$ level and normalizing pH.

Promotes relaxation and reduces stress. Control and reduction of fever reduces potential for seizures and helps reduce respiration rate.

Changes in mentation/CNS and neuromuscular hyperirritability may result in patient harm, especially if tetany/convulsions occur.
### ACTIONS/INTERVENTIONS

**Acid-Base Management: Respiratory Alkalosis (NIC)**

#### Independent

- Discuss cause of condition (if known) and appropriate interventions/self-care activities.

#### Collaborative

- Assist with identification/treatment of underlying cause.

#### Independent

- Monitor/graph serial ABGs, and pulse oximetry.
- Monitor serum potassium. Replace as indicated.
- Provide sedation/pain medication, as indicated.
- Administer CO₂, or use rebreathing mask as indicated. Reduce respiratory rate/tidal volume, or add additional dead space (tubing) to mechanical ventilator.

### RATIONALE

**Promotes participation in therapeutic regimen and may reduce recurrence of disorder.**

- Respiratory alkalosis is a complication, not an isolated occurrence; addressing the primary condition (e.g., hyperventilation of panic attack, organ failure, severe anemia; drug effect, such as with paraldehyde or epinephrine) promotes correction of the disorder and reduces likelihood of recurrence.

**Identifies therapy needs/effectiveness.**

- Hypokalemia may occur as potassium is lost (urine) or shifted into the cell in exchange for hydrogen in an attempt to correct alkalosis.
- May be required to reduce psychogenic cause of hyperventilation.
- Increasing CO₂ retention may correct carbonic acid deficit.

### POTENTIAL CONSIDERATIONS following acute hospitalization (dependent on patient’s age, physical condition/presence of complications, personal resources, and life responsibilities)

Refer to Potential Considerations relative to underlying cause of acid-base disorder.