Guidelines

For the Management of

Respiratory Alkalosis

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Respiratory alkalosis is a disturbance in acid and base balance due to alveolar hyperventilation. Alveolar hyperventilation leads to a decreased partial pressure of arterial carbon dioxide ($PaCO_2$). In turn, the decrease in $PaCO_2$ increases the ratio of bicarbonate concentration to $PaCO_2$ and, thereby, increases the pH level, thus the descriptive term of respiratory alkalosis.
In acute respiratory alkalosis, the $PaCO_2$ level is below the lower limit of normal and the serum pH is alkalemic.
In chronic respiratory alkalosis, the $PaCO_2$ level is below the lower limit of normal, but the pH level is relatively normal or near normal.

Acute hypocapnia causes a reduction of serum levels of potassium and phosphate secondary to increased intracellular shifts of these ions. A reduction in free serum calcium also occurs. Calcium reduction is secondary to increased binding of calcium to serum albumin due to the change in pH. Many of the symptoms present in persons with respiratory alkalosis are related to hypocalcemia. Hyponatremia and hypochloremia may also be present.

Compensation for respiratory alkalosis is by increased renal excretion of bicarbonate.
The relationship of $PaCO_2$ to arterial hydrogen and bicarbonate is 0.7 mmol/L per mm Hg and 0.2 mmol/L per mm Hg, respectively.
After 2-6 hours, respiratory alkalosis is renal compensation begins by decreasing bicarbonate reabsorption. The kidneys respond more to the decreased $PaCO_2$ rather than the increased pH. Complete kidney compensation may take several days.
The expected change in serum bicarbonate concentration can be estimated as follows:
<u>In acute respiratory acidosis</u> - Bicarbonate ($HCO3^-$) falls 2 mEq/L for each decrease of 10 mm Hg in the PCO_2; that is, Δ HCO 3 = 0.2(ΔPCO2);
maximum compensation: $HCO3^-$ = 12-20 mEq/L
<u>In chronic respiratory acidosis</u> - Bicarbonate (: $HCO3^-$) falls 5 mEq/L for each decrease of 10 mm Hg in the PCO_2; that is, $\Delta HCO3 = 0.5(\Delta PCO_2)$;
maximum compensation: HCO 3^- = 12-20 mEq/L
A plasma bicarbonate concentration of less than 12 mmol/L is unusual in pure respiratory alkalosis alone and should prompt the consideration of a metabolic acidosis as well

Clinical Presentation:
Symptoms may include paresthesias, circumoral numbness, chest pain or tightness, dyspnea, and tetany.
Acute onset of hypocapnia can cause cerebral vasoconstriction, reduces cerebral blood flow and can cause neurologic symptoms, including dizziness, mental confusion, syncope, and seizures. Hypoxemia need not be present for the patient to experience these symptoms

Physical examination
Findings in patients with respiratory alkalosis are nonspecific and are typically related to the underlying illness or cause of the respiratory alkalosis.
Positive Chvostek and Trousseau signs may be elicited.
Patients with underlying pulmonary disease may have signs suggestive of pulmonary disease,

such as crackles, wheezes, or rhonchi. Cyanosis may be present if the patient is hypoxic.
If the underlying pathology is neurologic, the patient may have focal neurologic signs or a depressed level of consciousness

Work up

- Arterial blood gas determination: which reveal an increased pH level (>7.45). The presence of a decreased $PaCO_2$ level (< 35 mm Hg) indicates a respiratory etiology of the alkalemia.
- Complete blood cell count: An elevation of the WBC count may indicate early sepsis as a possible etiology of respiratory alkalosis. A reduced hematocrit value may indicate severe anemia as the potential cause of respiratory alkalosis.
- Serum chemistries: Acute respiratory alkalosis causes small changes in electrolyte balances. Minor intracellular shifts of sodium, potassium, and phosphate levels occur.
A minor reduction in free calcium occurs due to an increased protein-bound fraction.
- Liver function test to exclude hepatic failure as the etiology of the respiratory alkalosis.
- Cultures of blood, sputum, urine, and other sites should be considered, depending on information obtained from the history and physical examination and if sepsis or bacteremia are thought to be the cause of the respiratory alkalosis.
–Thyroid function testing to rule out hyperthyroidism.
- Beta-human chorionic hormone levels to rule out pregnancy.
- Drug screens especially theophylline and salicylate levels.
- Chest radiography to rule out pulmonary disease as a cause of hypocapnia and respiratory alkalosis (pneumonia, pulmonary edema, aspiration pneumonitis, pneumothorax, and interstitial lung disease).
- CT scanning of the chest to reveal abnormalities not seen on the chest radiograph.
CT scanning of the brain if a central cause of hyperventilation and respiratory alkalosis is suspected (cerebrovascular accident tumor and trauma).
- Brain magnetic resonance imaging (MRI): If a central cause of hyperventilation and respiratory alkalosis is suggested and the initial brain CT scan findings are negative or inconclusive. MRIs may reveal abnormalities not seen on CT scans, particularly lesions of the brain stem.
-Ventilation perfusion scanning to exclude pulmonary embolism in patients who are unable to undergo an intravenous contrast injection associated with CT scanning.
-Echocardiography can be preformed to assess myocardial and valvular function assessing patients for unexplained hypoxemia and right-to-left shunting of blood.
- Lumbar puncture if a CNS infectious process or meningeal metastasis suspected.

Management

The treatment of respiratory alkalosis is primarily directed at correcting the underlying disorder as respiratory alkalosis itself is rarely life threatening therefore; emergent treatment is usually not indicated unless the pH level is greater than 7.5.
If the $PaCO_2$ is corrected rapidly in patients with chronic respiratory alkalosis, metabolic acidosis may develop due to the renal compensatory drop in serum bicarbonate.
In mechanically ventilated patients who have respiratory alkalosis, the tidal volume and/or

respiratory rate may need to be decreased. Inadequate sedation and pain control may contribute to respiratory alkalosis in patients breathing over the set ventilator rate.

In hyperventilation syndrome, patients benefit from reassurance, re-breathing into a paper bag during acute episodes, and treatment for underlying psychological stress.
Sedatives and/or antidepressants should be reserved for patients who have not responded to conservative treatment.
Beta-adrenergic blockers may help control the manifestations of the hyperadrenergic state that can lead to hyperventilation syndrome in some patients.

Differential diagnosis

Central nervous system causes:

Pain
Hyperventilation syndrome
Anxiety
Panic disorders
Psychosis
Fever
Cerebrovascular accident
Meningitis
Encephalitis
Tumor
Trauma

Hypoxia-related causes:

High altitude
Right-to-left shunts

Drug-related causes:

. Progesterone
Methylxanthine toxicity
Salicylate toxicity
Catecholamines
Nicotine

Endocrine-related causes:

. Pregnancy
Hyperthyroidism

Pulmonary causes:

Pneumothorax/hemothorax
Pneumonia
Pulmonary edema
Pulmonary embolism
Aspiration
Interstitial lung disease
Asthma
Emphysema
Chronic bronchitis

Miscellaneous causes:

Sepsis
Severe anemia
Hepatic failure
Mechanical ventilation
Heat exhaustion
Recovery phase of metabolic acidosis
Congestive heart failure

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