Respiratory Acid–Base Disorders in the Critical Care Unit

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KEYWORDS

• Oxygenation • Ventilation • Blood gas • Potassium • Brain injury

KEY POINTS

- Changes in carbon dioxide tension (P_{CO_2}) can have a variety of physiologic effects; some may be beneficial, while others can cause harm.
- Respiratory acid–base disorders can have particular relevance to specific patient populations, such as those with brain injury.
- Increased P_{CO_2} on venous blood gas analysis can be due to low cardiac output.
- Treatment of most respiratory acid–base abnormalities is based on resolution of the underlying disease.
- Mechanical ventilation is indicated in the management of severe or progressive respiratory acidosis.

INTRODUCTION

There are a variety of causes of respiratory acid–base disorders in critically ill and injured animals, although the incidence of these abnormalities is unknown in veterinary patients and not well described in people. Given the underlying causes for respiratory acid–base disorders, it is likely that they are common in the critical care patient population. The recognition of respiratory acid–base disorders is important from both a diagnostic and therapeutic perspective, emphasizing the role of blood gas evaluation in critical care.

Respiratory acid–base disorders are marked by changes in carbon dioxide tension (P_{CO_2}). The terminology associated with changes in P_{CO_2} can be confusing and lacks standardization. Respiratory acidosis is characterized by an increase in P_{CO_2} above the reference range for that species. The terms hypoventilation, hypercapnia, and hypercarbia are synonymous with respiratory acidosis and can be used

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interchangeably. Similarly, a decrease in P_{CO_2} can be labeled respiratory alkalosis, hyperventilation, hypocapnia, or hypocarbia. For the purpose of this discussion, the terms respiratory acidosis or hypercapnia and respiratory alkalosis or hypocapnia will be used.

CONTROL OF CARBON DIOXIDE TENSION

Arterial P_{CO_2} is proportional to carbon dioxide production ($\dot{V}CO_2$) and inversely proportional to alveolar minute ventilation ($\dot{V}_A$), as outlined by the following formula:

$$Pa_{CO_2} \propto \dot{V}CO_2/\dot{V}_A$$

From this formula, it can be appreciated that changes in P_{CO_2} can be divided into those that change $\dot{V}CO_2$ and those that change $\dot{V}_A$. Carbon dioxide is a product of cellular metabolism, and steady-state $\dot{V}CO_2$ is related to metabolic rate. In the normal state, the respiratory center of the brain regulates $\dot{V}_A$ in response to changes in $\dot{V}CO_2$ to target a preset P_{CO_2}.[1] Metabolic acid–base abnormalities will override the P_{CO_2} set point, and the respiratory center will alter $\dot{V}_A$ to change P_{CO_2} in a manner that will minimize the overall change in extracellular pH. This is known as respiratory compensation for a metabolic acid–base disorder.

Titration of bicarbonate by metabolic acids will also increase the production of CO_2. Hence bicarbonate administration can be a cause of respiratory acidosis if the animal is unable to maintain appropriate increases in $\dot{V}_A$. Carbon dioxide is transported to the alveoli via the pulmonary capillaries, where it is eliminated by $\dot{V}_A$. Alveolar minute ventilation is the product of respiratory rate and the portion of tidal volume that reaches perfused gas exchange units. This can be written as:

$$\dot{V}_A = f \times (V_T - V_D)$$

where f = respiratory frequency; V_T = tidal volume, and V_D = dead space volume.[2] For patients connected to a breathing circuit, such as ventilator patients, increases in the fraction of inspired CO_2 can also be a cause of respiratory acidosis.

Respiratory acid–base disorders are classically identified on arterial blood gas analysis. The reader is reminded that on venous blood gas analysis, decreased cardiac output can cause an increase in venous P_{CO_2} that does not reflect decreased $\dot{V}_A$.[3,4] As a result, perfusion abnormalities need to be considered as a potential cause of respiratory acidosis when evaluating venous blood gas results.

CLINICAL EFFECTS OF RESPIRATORY ACID–BASE ABNORMALITIES

The clinical effects of respiratory acid–base abnormalities may be due to either the change in P_{CO_2} or the change in the pH, or both. Investigations into the impact of respiratory acid–base disorders often struggle to separate these 2 effects.

Respiratory Effects

Respiratory acidosis can have fatal consequences as a result of hypoxemia rather than the associated acidemia. Increases in Pa_{CO_2} reflect an increased alveolar P_{CO_2}, which in turn causes a decrease in alveolar P_{O_2}. If breathing room air, Pa_{CO_2} greater than 80 mm Hg can cause life-threatening hypoxemia.[5] Oxygen supplementation is recommended in all patients with severe hypercapnia (Pa_{CO_2} >60 mm Hg). It should be noted that increased venous P_{CO_2} due to decreased cardiac output will not be a cause of hypoxemia, if $\dot{V}_A$ is adequate. Hypercapnia also has pulmonary effects including bronchodilatation and enhancement of hypoxic pulmonary vasoconstriction.