serum HCO_3^- and decreased Cl^- concentrations. In chronic respiratory acidosis, there is a 3.5 mEq/L increase in HCO_3^- for each 10 mm Hg elevation in the $PaCO_2$. Higher or lower plasma HCO_3^- concentrations suggest mixed respiratory and metabolic acid-base disorders.

Treatment

The mainstay of treatment in respiratory acidosis is to recognize and treat the underlying cause when possible. Patients with acute respiratory acidosis are primarily at risk for hypoxemia rather than hypercapnia or acidemia. Immediate therapeutic efforts should focus on establishing and securing a patent airway to provide adequate oxygenation. In patients with status asthmaticus, a lower ventilatory rate and peak inspiratory pressure may be required to minimize barotrauma to the lung, but it is achieved at the expense of a persistently higher PCO_2. Small amounts of $NaHCO_3$ can help to prevent excessive decreases in blood pH in this setting. The downside of this therapy is that infusion of $NaHCO_3$ can result in increased carbon dioxide production, causing a further increase in PCO_2 when ventilation cannot be increased.

Excessive oxygen should be avoided in patients with chronic respiratory acidosis because it may lead to worsening hypoventilation. When mechanical ventilation is required, care should be taken to lower the $PaCO_2$ carefully and slowly because there is the risk of overshoot alkalemia due to a high HCO_3^- (i.e., post-hypercapnic metabolic alkalosis). The kidneys must excrete the HCO_3^- to normalize the acid-base status. This excretion does not occur when the EABV is reduced because of salt depletion due to restricted intake or diuretic therapy or because of a salt-retentive state such as heart failure or cirrhosis. Correction of the superimposed metabolic alkalosis can usually be achieved with saline and discontinuation of loop diuretics. In edematous patients with heart failure, this is not possible, and acetazolamide may be needed to correct the alkalosis.

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