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Direct Vasodilator Effect of Hyperventilation-Induced Hypocarbia in Autonomic Failure Patients

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DESCRIPTION

Hypocapnia, also known as hypocapnia, is a drop in the level of carbon dioxide (CO_2) in the alveoli and blood below the normal reference range of 35 mmHg. CO_2 is a metabolite of many cellular processes in the body involved in the processing of lipids, carbohydrates and proteins. The major organ systems involved in regulating CO_2 homeostasis are the pulmonary and renal systems. Additionally, CO_2 is regulated by a CO_2/HCO_3 pH buffer system. Abnormalities that lead to hypocapnia usually also lead to respiratory alkalosis. This activity reviews the assessment and management of hypocapnia and highlights the role of professional teams in educating patients with this condition about follow-up care. Hypocapnia, also known as hypocapnia, is a drop in the level of carbon dioxide (CO_2) in the alveoli and blood below the normal reference range of 35 mmHg. CO_2 is a metabolite of many cellular processes in the body involved in the processing of lipids, carbohydrates and proteins. The major organ systems involved in regulating CO_2 homeostasis are the pulmonary and renal systems. Additionally, CO_2 is regulated by a CO_2/HCO_3 pH buffer system. Abnormalities that lead to hypocapnia usually also lead to respiratory alkalosis. Fundamentally, hypocapnia is caused by either decreased CO_2 production or increased CO_2 loss. The primary event is loss of CO_2 due to changes in the pH buffering system or pulmonary system, as metabolic demand usually does not decrease sufficiently to adjust CO_2 levels to meaningful hypocapnic levels. The pulmonary system is very efficient at removing CO_2 from the body by gas diffusion. This requires a diffusion gradient from the high concentration of arteriolar blood to the relatively low concentration of ambient air. This gradient is maintained by continuously flushing CO_2 from the alveolar space regardless of absolute $PaCO_2$ concentration. Thus, a CO_2 gradient is generated and maintained. In this case, arterial $PaCO_2$ is directly proportional to the rate of metabolic CO_2 production and inversely proportional to the rate of CO_2 excretion by the lungs due to increased alveolar ventilation. Alveolar ventilation is the distance from the alveolar air to the environment defined as the expiratory minute volume reaching the alveoli, determined by the ratio of dead space to minute ventilation and tidal volume. The frequency and distribution of disease are variable and depend on the exact etiology of the disease. Similarly, morbidity and mortality are related to the precise etiology of the causative disease. In general, younger patients have better outcomes. The most common acid-base disorder seen in critically ill patients is respiratory alkalosis. Hypocapnia is the result of hyperventilation. Increased ventilation of the alveolar spaces removes gaseous CO_2 rapidly. This increases the diffusion gradient of CO_2 from the blood to the alveoli. CO_2 is then more easily removed from the body. Other than reduced respiratory rate, there is virtually no mechanism to regulate this loss. The partial pressure of carbon dioxide ($PaCO_2$) is maintained between 35 mmHg and 45 mmHg using a feedback controller. Central chemoreceptors (in the brain) and peripheral chemoreceptors (in the carotid artery) sense hydrogen concentration and influence ventilation to regulate pH and $PaCO_2$. For example, when these receptors sense an increased concentration of hydrogen ions, ventilation increases to flush CO_2. Continued hyperventilation eventually leads to hypocapnia as alveolar ventilation exceeds the amount of CO_2 generated.

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CONFLICT OF INTEREST

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