Nocturnal Hypoxemia in Sleep Apnea

Since nocturnal hypoxemia plays a key role in the pathogenesis of sleep apnea, it is important to understand the determinants of nocturnal hypoxemia in these patients. These determinants include: (1) the length and number of apneas during sleep; (2) baseline oxyhemoglobin saturation (SaO₂); and (3) the rate of fall of SaO₂ during each apnea (a balance between oxygen stores and oxygen uptake).

Little is known about what determines the number and length of apneas during sleep. Body weight and obesity affects both the number and length of apneas. Drugs such as alcohol may increase the number of apneas during sleep by depressing the CNS or increasing upper airway resistance. Hypoxic ventilatory drive may affect apnea length during sleep, for supplemental oxygen may increase the length of apneas in some patients. In addition, receptors sensitive to inspiratory muscle fatigue may affect the length of apneas. Finally, sleep state affects apneas, for REM sleep is associated with increased number and length of apneas in patients with obstructive sleep apnea.

The baseline SaO₂ of patients determines their position on the oxyhemoglobin dissociation curve and will affect the rate of fall in SaO₂ during apneas. If the baseline SaO₂ is low, the rate of fall in SaO₂ will be increased during apneas. Increasing baseline SaO₂ by supplemental oxygen decreases the rate of fall in SaO₂, although the apneas may become longer.

The rate of fall in SaO₂ during an apnea will depend on a balance between the oxygen stores before and the oxygen uptake during an apnea. Oxygen stores is the amount of alveolar oxygen available for gas exchange during an apnea. These stores are determined by the alveolar gas volume, partial pressure of oxygen of alveolar gas (PAO₂) and the gas exchange efficiency of the lung. The lower the lung volume, the higher the rate at which SaO₂ will fall during apnea. Depressed PAO₂ due to hypercapnia or other causes will increase the rate at which SaO₂ will fall. Finally, a shunt or other lung disorder inhibiting gas exchange will decrease available oxygen stores and increase the rate at which SaO₂ falls. The oxygen uptake from the lung during apnea is determined by mixed venous oxygen (SVO₂) and oxygen consumption. The lower the SVO₂ level, the greater the amount of oxygen required to reoxygenate the venous blood and the greater the fall in SaO₂ during apnea (see Fletcher et al, p 717). A higher oxygen consumption during apnea might rapidly deplete oxygen stores and cause a rapid fall in SaO₂ during apneas. Small increases in oxygen consumption probably have little effect, since vigorous respiratory maneuvers during apnea do not affect the rate of SaO₂ fall during apneas. However, measurements of oxygen consumption have not been related to the rate of fall of SaO₂ during apneas in sleeping humans. The effect of cardiac output on oxygen uptake may be variable. A chronically low cardiac output may lower SVO₂ and increase the rate at which saturation falls during apnea. In contrast, acute declines in right ventricular cardiac output may decrease uptake from the lung oxygen stores and decrease the rate of fall of SaO₂ during apnea.

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REFERENCES