**Diseases of the Respiratory System**

**PRINCIPLES OF RESPIRATORY INSUFFICIENCY**

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**PRINCIPLES OF RESPIRATORY INSUFFICIENCY**

**-The principal function of the respiratory system is gas exchange in which oxygen**

**transferred from the environment to the blood and carbon dioxide is moved in the opposite direction.**

**-Other important functions include a role in thermoregulation in most species, acid-base regulation in concert with the kidney, as an endocrine organ.**

**-The most readily apparent failure of the respiratory system is failure of gas exchange with resultant hypoxemia and hypercapnia. However, failure of other functions of the respiratory system can also result in clinically apparent disease.  
-Failure of gas exchange, and the resultant hypoxia and hypercapnia, is responsible for most of the clinical signs of respiratory disease and for respiratory failure, the terminal event of fatal cases.  
-Death due to respiratory failure is due to hypoxia.   
-An understanding of hypoxia, hypercapnia and respiratory failure is essential to the study of clinical respiratory disease.**

**DEFINITIONS:**

**A number of terms are used to describe the function of the respiratory tract, or abnormalities that arise because of a variety of diseases.   
Hypoxia: is a broad term meaning diminished availability of oxygen to tissues  
Hypoxemia: is deficient oxygenation of blood, usually assessed by measurement of blood oxygen tension, or by measurement of blood hemoglobin saturation and hemoglobin concentration, and subsequent calculation of blood oxygen content.  
Hypercapnia: is an abnormally high carbon dioxide tension in blood.  
Po02: is the oxygen tension (partial pressure) in arterial blood.  
 PA02: is the oxygen partial pressure in alveolar gas.**

**Dyspnea: refers to signs of respiratory distress in animals (in humans it describes the sensation of air hunger, which is a symptom and not a sign).  
Polypnea: is an excessively high rate of breathing.  
Tachypnea: is an excessively high rate of breathing, with the implication that the breathing is shallow.  
Hyperpnea: is an increased minute ventilation.  
 HYPOXIA: Failure of the tissues to receive an adequate supply of oxygen occurs in a number of ways and the differences are clinically relevant, in that they are associated with failure of different organ systems, different diseases, and have fundamentally different pathophysiological mechanisms.**

**Hypoxic (or hypoxemic) hypoxia: occurs when there is inadequate oxygenation of blood (hypoxemia) and is usually associated with disease of the respiratory tract or other causes of hypoventilation. Situations in which there is inadequate oxygenation of blood in the lungs include, hypoventilation, ventilation/perfusion mismatches, diffusion impairment, low inspired oxygen tension and extra pulmonary right-to-left shunting.  
 Hypoventilation: occurs in animals with depressed consciousness, such as occurs with general anesthesia and heavy sedation, or in newborns, in which the central respiratory drive is suppressed.**

**Airway obstruction: caused by the presence of foreign bodies in the airway, luminal obstruction by masses, such as retropharyngeal abscesses in horses with strangles, laryngeal spasm or bronchoconstriction can cause inadequate alveolar ventilation and hypoxemia.  
 Low inspired oxygen tension: occurs naturally only in animals at high altitude. It can also occur during anesthesia if there are defects in the ventilator causing low oxygen tension in the gases delivered to the animal.  
 Anemic hypoxia: occurs when there is a deficiency of hemoglobin per unit volume of blood (anemia) .**

**The percentage saturation of the available hemoglobin and the oxygen tension of arterial blood are normal but as a result of the low hemoglobin concentration the oxygen carrying capacity of the blood is reduced.**

**Circulatory hypoxia: occurs as a result of inadequate delivery of oxygen to tissue because of inadequate perfusion of tissues by blood.   
Histotoxic anoxia: occurs when oxygen delivery to tissue is adequate because both oxygen content of arterial blood and blood flow are appropriate, but the tissue is unable to utilize oxygen . Cyanide poisoning is the only common cause of this form of anoxia.  
Consequences of hypoxia: Consequences of inadequate delivery of oxygen include changes in almost all body systems. The central nervous system and heart are most susceptible to the immediate and acute effects of hypoxia, whereas clinical signs related to hypoxic damage to the gastrointestinal tract and kidneys are somewhat delayed. CNS hypoxia is evident as mild changes in denotation, such as depression, progressing through decreased alertness to coma and death.**

**COMPENSATORY MECHANISMS  
Compensation o f respiratory insufficiency occurs as both short-term and long-term events. Short-term compensatory mechanisms for low arterial oxygen tension or oxygen delivery to tissues occur within seconds to minutes and include respiratory, cardiovascular and behavioral responses. Stimulation of respiratory centers in the medulla oblongata by low arterial oxygen tension, and high arterial carbon dioxide tension causes an increase in respiratory minute volume mediated by an increase in tidal volume and respiratory frequency. Both low oxygen tension and high carbon dioxide tension in arterial blood, together or separately, are potent stimulators of these events. Inadequate tissue oxygenation also stimulates an increase in cardiac output, mainly as a result of increased heart rate and to a lesser extent by an increase in stroke volume.**

**Longer-term compensatory mechanisms include an increase in erythropoietin secretion by the kidney with subsequent increases in bone marrow production of red blood cells and an increase the in hemoglobin concentration in blood. This polycythemia increases oxygen­carrying capacity of blood. Severe polycythemia, such as occurs with congenital cardiac anomalies causing chronic right­to-left shunting, increases the viscosity of blood and impairs tissue perfusion, increases the workload of the heart and the risk of thromboembolism. Longer­term compensatory mechanisms also heaves, and include changes in ventilatory pattern, such as in horses with heaves and behavior.**

**CARBON DIOXIDE RETENTION (HYPERCAPNIA)  
-Respiratory insufficiency results in decreased elimination of carbon dioxide and its accumulation in blood and tissues. Animals breathing room air that are hypercapnic are always hypoxemic. Increasing the oxygen tension of inspired air can alleviate the hypoxemia but, by reducing hypoxic stimulation of the respiratory center, can cause further increments in arterial Pco2.  
-Acute hypercapnia causes a respiratory acidosis that reduces both blood and cerebrospinal fluid pH.2 The clinical signs of acute hypercapnia are initial anxiety followed by central nervous system depression and eventual coma and death.**

**-These clinical abnormalities are attributable to declines in the pH of cerebro­spinal fluid (CSF), a consequence of the ease with which carbon dioxide crosses the blood-brain barrier. Decreases in CSF pH are greater for respiratory acidosis than for a similar degree of metabolic acidosis. Severe hypercapnia also causes peripheral vasodilatation, which can contribute to arterial hypotension, and cardiac arrhythmia. The acid-base effects of chronic hypercapnia are compensated by renal mechanisms that return the arterial and CSF pH to almost normal and therefore do not cause more than mild clinical disease in most instances.**

**-So as long as oxygen delivery to tissue is maintained, animals can tolerate quite high arterial carbon dioxide tensions for a number of days or longer - this is referred to as 'permissive hypercapnia' and is sometimes an alternative to artificial or mechanical venilation of animals with respiratory insufficiency.  
 RESPIRATORY FAILURE  
Respiratory movements are involuntary and are stimulated and modified by the respiratory centers in the medulla.  
 Respiratory failure is the terminal stage of respiratory insufficiency in which the activity of the respiratory centers diminishes to the point where movements of respiratory muscles cease.**

**-Respiratory failure can be paralytic, dyspneic or asphyxial, or tachypneic, depending on the primary disease.  
The respiratory failure that occurs in animals with pneumonia, pulmonary edema and upper respiratory tract obstruction is caused by combinations of hypoventilation, mismatch and ventilation/perfusion diffusion impairment, which leads to hypercapnia and hypoxemia. Hypercapnia and hypoxia stimulate the respiratory center and there is a potent respiratory drive evident as markedly increased respiratory rate and effort. As the disease progresses these changes become more marked until death occurs as a result of central nervous system or cardiac failure.**

**Animals that die of the central nervous system effects of respiratory failure typically have dyspnea followed by periods of gasping and apnea just before death.**

**-Paralytic respiratory failure is caused by depression of the respiratory centers or paralysis of the muscles of respiration. Depression of the respiratory center occurs with poisoning by respiratory center depressants, such as general anesthetics, or damage to the respiratory center, such as might occur with brainstem injury.  
- Paralysis of respiratory muscles occurs in disease such as tetanus, botulism, strychnine poisoning, white muscle disease, severe hypocalcemia and tick paralysis. The signs of paralytic respiratory failure are a gradual or abrupt cessation of respiratory movements without preceding signs of increased respiratory effort or dyspnea. The animal is often unconscious, or unable to move, during the later stages of the disease.**

**References**

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