RESPIRATORY FAILURE IN ADULTS

Introduction

Respiratory failure is an important and frequently encountered problem in intensive care units all over the world. Respiratory failure is defined as the inability of the respiratory system to maintain the normal homeostasis of arterial blood gases, so that the oxygen tension in arterial blood (PaO_2) is < 60mm Hg, and / or carbon dioxide tension in arterial blood ($PaCO_2$) is 50 mm Hg or greater. Respiratory failure may be acute or chronic depending on the onset and duration of the failure.

Epidemiology, prevalence, and economic burden

Because so many underlying causes contribute to it, respiratory failure is a common and major cause of illness and death. It is the main cause of death from pneumonia and chronic obstructive pulmonary disease (COPD), which together comprise the third-leading cause of death. It is also the main cause of death in many neuromuscular diseases, as these diseases weaken the respiratory muscles, rendering them incapable of sustaining breathing. Epidemiologic studies suggest that respiratory failure will become more common as the population ages, increasing by as much as 80 percent in the next 20 years.

Because respiratory failure is such a common cause of illness and death, the cost to society in terms of lost productivity and shortened lives is enormous. However, it is hard to quantify because the cause of death is more likely to be listed as pneumonia, COPD, or another underlying condition, rather than respiratory failure.

Physiology of respiration

Gaseous exchange between the environment and the pulmonary capillary blood constitutes external respiration. The functioning unit of the lung is alveolus with its capillary network. Various factors govern transport of air from the environment to the alveoli (ventilation) and supply of blood to the pulmonary capillaries (perfusion).

Delivery of O_2 to the alveolus is directly related to the sweep rate of air (ventilation), and composition of the sweeping gas (partial pressure of O_2 in the inspiratory air; FIO_2). In general, alveolar O_2 tension (PAO_2) increases with increase in inspiratory O_2 tension and increase in ventilation. Extraction of O2 from the alveolus is determined by the saturation, quality and quantity of the haemoglobin of the blood perfusing the alveoli. The O_2 saturation of the haemoglobin in the pulmonary capillary blood is affected by the supply of O_2 to the tissues (cardiac output) and the extraction of the O2 by the tissues (metabolism). In general, lower the haemoglobin saturation in the blood perfusing the pulmonary capillaries, a result of low cardiac output (increased tissue extraction) and/or increased tissue metabolism, higher the extraction of O_2 in the alveoli and lower the equilibration partial pressure of O_2.

The partial pressure of O2 in the alveolus is further affected by the partial pressure of CO_2 in the pulmonary capillary blood. As mentioned earlier partial pressure of CO_2 in the alveolus is

because of dynamic equilibrium between CO_2 transported to the alveolus and CO_2 removed from the alveolus. Amount and the partial pressure of CO_2 in the alveolus increases with the increase in tissue metabolism and in presence of low cardiac output (CO_2 produced in the tissues is transported in less amount of the venous blood).

In general, perfusion is more at the lung bases as compared to the apex and this difference increases with decrease in cardiac output, hypotension and with the application of positive pressure ventilation. Distribution of ventilation is influenced by regional transpulmonary pressure (TPP) gradient and changes in the TPP during inspiration. In general alveolar volume is bigger in the apical regions as compared to the alveolar volume at the base and ventilation is more at the base as compared to the apex.

Theoretically, the most efficient gaseous exchange would occur if a perfect match exists between ventilation and perfusion in each of the functioning unit of the lung. The partial pressure of O_2 and CO_2 contained in each alveolus, and therefore of the capillary blood leaving it, are primarily determined by the ventilation perfusion ratio of that alveolus. The functioning unit can exist in one of the four absolute relationships:

1. The normal unit in which both ventilation and perfusion are matched
2. The dead space unit in which the alveolus is normally ventilated but there is no blood flow through the capillary
3. The shunt unit in which the alveolus is not ventilated but there is normal blood flow through the capillary
4. The silent unit in which the alveolus is unventilated and the capillary has no perfusion.

The complexities of the ventilation-perfusion (VA/Q) relationship are caused primarily by the spectrum between the two extremes of dead space and shunt units. The lung consists of millions of alveoli with its network of capillaries. In health and disease states ventilation and perfusion relationship can exist in various combinations.

Classification of respiratory failure

Type 1 or hypoxic respiratory failure is due to a failure of oxygenation with a PaO_2 < 60mm Hg.

Causes of Type I (Oxygenation) respiratory failure

1. Adult respiratory distress syndrome (ARDS)
2. Asthma
3. Pulmonary oedema
4. Chronic obstructive pulmonary disease (COPD)
5. Interstitial fibrosis
6. Pneumonia
7. Pneumothorax
8. Pulmonary embolism
9. Pulmonary hypertension

Type 2 or hypercapnic respiratory failure (ventilator failure) is due to hypoventilation and is characterized by a $PaCO_2$ > 50 mm Hg.

Common causes of Type II Respiratory Failure:

A. Disorders affecting central ventilatory drive

1. Brain stem infarction or haemorrhage
2. Brain stem compression from supratentorial mass
3. Drug overdose, Narcotics, Benzodiazepines, Anaesthetic agents etc.

B. Disorders affecting signal transmission to the respiratory muscles

1. Myasthenia Gravis
2. Amyotrophic lateral sclerosis
3. Gullain-Barre syndrome
4. Spinal-Cord injury
5. Multiple sclerosis
6. Residual paralysis (Muscle relaxants)

C. Disorders of respiratory muscles or chest-wall

1. Muscular dystrophy
2. Polymyositis
3. Flail Chest

Type 3 respiratory failure is that occurring peri – operatively and is largely due to basal atelectasis. Cardiothoracic surgery and / or major upper abdominal surgery splint the diaphragm and induce an abnormal mechanics of the abdominal muscles. These factors cause a fall in the functional residual capacity and an increase in the closing volume of the lungs. The end result is increasing atelectasis of the dependent alveoli, "small lungs" with a diaphragm, respiratory distress and hypoxemia.

Common causes of Type III respiratory Failure:

1. Adult respiratory distress syndrome (ARDS)
2. Asthma
3. Chronic obstructive pulmonary disease

Type 4 respiratory failure is that associated with shock – a poorly functioning circulatory system with a low cardiac output is the main cause of hypoxaemic failure in this situation.

Compensatory Mechanisms in presence of respiratory failure:

The response to hypoxaemia depends on the ability of the patient to recognize the hypoxemic state and then to increase cardiac output and minute ventilation to improve the situation. Peripheral chemoreceptors located in the arch of aorta and at the bifurcation of carotid artery send afferent signals to the brain.

Diagnosis of respiratory failure

The diagnosis of respiratory failure is based on analysis of arterial blood gas. However, it is important to suspect its presence on clinical grounds. The patients with respiratory failure will have clinical features of underlying disease; in addition they may have signs of hypoxaemia and hypercapnia. Hypoxaemia may be accompanied by the presence of tachypnoea, tachycardia, dyspnea, hypertension, intercostals retraction, and use of accessory muscles of ventilation.

Cerebral hypoxia produces changes in mentation that can range from mental confusion and restlessness to delirium. Cyanosis of the nail beds may be evident. Hypercapnia exerts its major effects on central nervous system. As the $PaCO_2$ increases, patients typically progress through the stages of lethargy, stupor and finally coma (CO_2 narcosis).

Other symptoms are secondary to catecholamine release and simultaneous hypoxaemia. The patient is often described as appearing "fatigued" or "tired out." However, the clinical manifestations described are non-specific and may occur in the absence of respiratory failure. Therefore, the diagnosis of respiratory failure must be confirmed by arterial blood gas analysis.

Management of respiratory failure

1. Maintenance of clear airways

   Clear secretions

   - Liquefy secretions
   - Promote cough – good physiotherapy
   - Suctioning of secretions
   - Use of an airway – oropharyngeal airway, other airways, endotracheal intubation / tracheostomy

2. Maintenance of adequate ventilation
   - Artificial ventilation with AMBU bag in emergency, till mechanical ventilator support is organized
   - Use of respiratory stimulants (in rare situations)
3. Use of oxygen
4. Treat cause of acute respiratory failure whenever possible
5. Use of mechanical ventilator support if cause cannot be treated or if patient hypoxic or hypercapnic despite above measures

Conclusion

Respiratory failure in critical care unit is a medical emergency and a real threat to the life of the patient.

Though many diseases of diverse etiology are associated with respiratory failure the initial management of respiratory failure is same.

Reference
1. Indian J. Anaesth 2003; 47: 360 – 366
2. Principles of Respiratory Medicine 2010: 575 - 583