Summary

Respiratory failure is failure to maintain the normal delivery of oxygen to the tissues or the normal removal of carbon dioxide from tissues. There are several mechanisms involved in acute respiratory failure; including hypoventilation, ventilation-perfusion mismatching, shunt mechanism, abnormal diffusion, low inspired oxygen fraction and venous admixture. From the pathophysiologic point of view, respiratory failure can be classified into four main types; each has a predominant mechanism including acute hypoxemic (Type I), hypercapnic (hypoventilation or Type II), perioperative (Type III) and hypoperfusion (Type IV) respiratory failure.

Chronic obstructive pulmonary disease (COPD) is characterized by airflow limitation that is not fully reversible, usually progressive in course and associated with an abnormal inflammatory response of the lungs to naxious particles. Diagnosis of COPD should be considered in any patients who suffer from cough, sputum production, dyspnea, FEV_1/ FVC ≤ 0.7 asscociated with the presence of history of α-1 antitrypsin deficiency, tobacco smoking and chemical exposure.

COPD patients have several structural changes; affecting all body systems. The lung shows irreversible airflow obstruction, airway remodling associated with hyperinflation causing impending of respiratory muscle functions, rapid development of respiratory muscles fatigue and increased incidence of bullae formation with associated risk of pneumothorax. Malnutrition, electrolyte abnormalities and critical illness polyneuropathy are potentially reversible causes of muscle dysfunction during critical illness that may prolong the need for ventilatory support. Therapeutic interventions for COPD may also contribute to muscle dysfunction as using corticosteroids, neuromuscular

blocking agents. Most COPD patients have an increased pulmonary blood volume and short pulmonary circulation time. They have high serum level of liver enzymes reflectin the effect of hypoxemia on liver cells. There is a left ventricular dysfunction and right ventricular restrictive diastolic filling caused by ventricular interdependence and associated hypoxia, hypercapnia and acidosis increasing the incidence of atrial and ventricular arrhythmia. Those patients suffer both sensory and motor neuropathies and evidence of autonomic nervous system disturbance. During exacerbations of COPD, alveolar-arterial oxygen differences often widen, due to worsening ventilation-perfusion mismatch and increases in shunt caused by retained secretions or additional contributing factors such as superimposed pneumonia, congestive heart failure, pulmonary embolism, or pneumothorax. There is a concern that uncontrolled administration of oxygen to patients with chronic CO_2 retention will blunt the hypoxic drive and worsening CO_2 retention. Carful administration of oxygen is mandatory aiming to maintain PaO_2 between 55 to 65 mmHg. Secondary to hypoxemia, polycythemia can be developed increasing the blood viscosity which may contribute to the development of pulmonary arterial hypertension. There are several factors contributing to the development of auto-PEEP including increased airway resistance, extrathoracic obstruction, high tidal volume or reduced exhalation time due to inappropriately high respiratory rate, tachypneic, prolonged inspiration. Also auto-PEEP has a deterious effects on the hamodynamic state of the patient. Static auto-PEEP refers to the measured intrinsic PEEP in paralysed patients while dynamic auto-PEEP refers to the measured intrinsic PEEP in spontaneously breathing patients. The addition of extrinsic PEEP may reduce the effort required to trigger the inspiratory phase of the ventilator cycle by counterbalancing auto-PEEP and raising

the pressure threshold for initiating inspiration; values of 75–85% of the static measured auto-PEEP are the values most often recommended.

Acute exacerbation of COPD defined as a sustained worsening of the patient's condition that is acute in onset and necessitates a change in regular medication in a patient with underlying COPD. These patients have a PaO_2 less than 60 mmHg and/ or $PaCO_2$ greater than 50 mmHg or above their previous stable hypercapnic state and consequent respiratory acidosis (pH<7.35) and worsen symptoms compared with the stable clinical state. Etiology of acute exacerbation of COPD includes respiratory tract infections, central nervous system (CNS) depression, impaired neuromuscular competence; extensive respiratory load and minute ventilation loads.Managment of AECOPD include both pharmacological treatment and ventilator support.

Pharmacological treatment of AECOPD includes bronchodilator (Beta- agonist, Ipratropium bromide, Tiotropium, Phosphodiesterase Inhibitors), corticosteroids (Methylprednisolone 0.5-1 mg/ Kg every 6 hours for 72 hours), antibiotics if there is a documented infections; and dealing with associated complications as arryhtmias, increased in airway secretions, pulmonary embolism, gastric distention, intestinal ileus, stress ulcers.

Ventilatory support in COPD patients aiming to avoid hyperinflation, managing auto-PEEP and can be applied using either noninvasive or invasive mechanical ventilation.

Noninvasive ventilation avoids intubation and its complications and associated with a decrease in the mortality and morbidity. The most important factor for its success is the appropriate choice of the interface and the ventilator mode and setting. The noninvasive interfaces include the mouthpiece, oronasal mask, nasal pillow and the helmet system. Modes of noninvasive ventilation include CPAP, PSV, BiPAP and

recently PAV. Complication of NIV includes claustrophobia, pressure sore, and rebreathing and air leak. However, when there is worsening of blood gases or there is a hemodynamic instability or the patient is tachypneic or has a decreased level of consciousness, mask intolerance or unable to clear secretions; the use of invasive ventilation is mandatory.

Invasive ventilation implies the use of endotracheal tube or tracheostomy tube as an acess for ventilation. On intiation of MV, there is a risk of hypotension which is corrected by intravenous fluid and risk of post hypercapnic metabolic alkalosis. Adjusting the ventilatory setting is an important issue including the FiO_2, triggering level, inspiratory flow rate, PS level, extrinsic PEEP, ventilatory rate, tidal volume, maintaining plateau pressure ≤ 30 mmHg to avoid barotrauma.

Traditional modes of invasive ventilation include controlled mode ventilation, assist control ventilation, synchronized intermittent mechanical ventilation and mandatory minute ventilation and pressure controlled ventilation.

New modes of mechanical ventilation generated to improve patient- ventilator synchronization as pressure-controlled inverse ratio ventilation which is not used in COPD patients, proportional assist ventilation, airway pressure release ventilation, dual control ventilation whether it is within a breath as in volume assured pressure support or between breath to breath as in volume support, automode, adaptive support ventilation, neurally adjusted ventilatory assist.

Successful, timely weaning and extubation of critically ill patients has a considerable bearing an ultimate outcome. Thus, both prolonged duration of mechanical ventilation and untimely premature extubation resulting in extubation failure and the need for re-intubation are associated with increased mortality and morbidity. ICU physicians must weigh the benefit of rapid liberation form mechanical ventilation against

risk of premature trials of spontaneous breathing. Premature weaning and its hazards can be decreased by accurate prediction of weaning outcome.

The weaning predictors of COPD patients are numerous. Some of them are commonly used traditional indices e.g. VT, RR, and MV. Among recently proposed predictors, breathing pattern (RSBI) has been shown to occupy a major role. The airway occlusion pressure (P0.1) measured at 100 milliseconds from the onset of inspiration, estimating the central drive to breath, vital capacity, arterial oxygenation, maximal inspiratory pressure, work of breathing. Also assessment of some lung mechanics is very helpful in avoiding ventilator induced complications as well as determining the patient's readiness for the discontinuation of ventilator support. Of these lung mechanics, intrinsic positive end expiratory pressure (PEEPi), compliance and airway resistance, pressure time index. Gastric tonometry also has been used to predict weaning.

Weaning can be abrupt process or gradual process using spontaneous beathing trial performed by T-tube trial, PSV, SIMV, NPPV and recently Automated tube compensation and the SmartCare.