New trends of mechanical ventilation and weaning of chronic obstructive lung disease patients

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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, FEV1/ 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