

INTRODUCTION

Mechanical ventilation is designed to assist patient with the most fundamental function of the lungs: exchanging oxygen (O₂) and carbon dioxide (CO₂) with the external environment. Several factors influence the ability of the lungs to this function, including airway resistance, mechanical resistance of the chest wall and abdomen, and pulmonary compliance (*Reiff et al., 2008*).

Mechanical ventilation is neither intended to normalize gas exchange nor is a form of cure. The goal of the mechanical ventilator is to ensure tissue viability until the disease process has resolved and to minimize the inevitable complications of the therapeutic intervention itself (*Sarnaik and Mastropietro, 2011*).

In general, there are two main types of ventilators commonly in use in ICU-those that deliver a preset tidal volume (volume-controlled ventilation) and those that deliver a preset inspiratory pressure (pressure-controlled ventilation) during each inspiration (*O'Higgin's, 2003*).

Newer ventilators can be set to modes other than the pressure-control and volume-control modes. These innovations are called "alternative modes" to differentiate them from the basic modes. Driving these innovations is the desire to prevent

ventilator induced lung injury, improve patient comfort, and liberate the patient from mechanical ventilation as soon as possible (*Cabodevila et al., 2009*).

The ventilator discontinuation process is an essential component of overall ventilator management. Undue delay leads to excess stay, iatrogenic lung injury, unnecessary sedation and even higher mortality. On the other hand, premature withdrawal can lead to muscle fatigue, dangerous gas exchange impairment, loss of airway protection, and also a higher mortality. An evidence-based task force has recommended regular assessments focusing on the causes of ventilator dependence, regular assessments for evidence of disease reversal, use of regular spontaneous breathing trial (SBT) as the primary assessment tool for ventilator discontinuation potential, use of separate assessments to evaluate the need for an artificial airway in patients tolerating the SBT, and the use of comfortable interactive ventilator modes in between regular SBTs (*McIntyre, 2013*).

Weaning usually account for approximately 40-50% of the total duration of mechanical ventilation. Approximately two thirds of patients can tolerate withdrawal of ventilation without the need for more gradual weaning, but there are a significant number of patients for whom weaning is difficult (*Walters and Polkey, 2008*).

The pathophysiology of weaning failure is complex and often multifactorial. Accordingly, determining the reason and the subsequently developing treatment strategy require a dedicated clinician with in-depth knowledge of the pathophysiology of weaning failure (*Heunks and Hoeven, 2010*).

AIM OF THE WORK

The aim of the work is to know the definition of difficult weaning, its causes, how to predict this difficulty and the proper strategies to deal with difficulty to wean mechanically ventilated patient.

HISTORY OF MECHANICAL VENTILATION

The first form of mechanical ventilator can probably be credited to **Paracelus**, who used fire- bellows fitted with a tube to pump air into the patient's mouth. In 1653, **Andreas Vesalius** recognized that artificial respiration could be administrated by tracheotoimising a dog (*Hasan, 2010*).

Credit for the first mechanical ventilation in order to sustain life goes to **Robert Hooke**, who used a pair of bellows to ventilate a dog via a tracheostomy during a demonstration to the Royal Society in London in 1664: In prosecution of some enquires into the nature of respiration in several animals, a dog was dissected, and by means of a pair of bellows, and a certain pipe thrust into the wind-pipe of the dog, the heart continued beating for a very long while after all the thorax and heart had been opened (*Mackenzie, 2008*).

The first documented occurrence of effective mouth-to-mouth resuscitation was recorded by **William Tossach** in 1744, later followed by descriptions by **Hooke**-style bellows mechanical ventilation by **John Hunter** in 1776 (*Mackenzie, 2008*).

Later, in that century, a number of investigators developed mechanical devices designed to support respiration. Among these was **Alexander Graham Bell**, who invented a vacuum Jacket powered by a hand-operated bellows in the early 1880s, although

this device was never used clinically. The lack of a non manual power source to propel these devices proved a serious impediment to their use. The inception of electrical energy to continuously drive ventilation machines would be necessary to render them successful (*Chen et al., 1998*).

The use of bellows to resuscitate victims of near drowning was described by Royal Humane Society in the 18th century. The society, also known as the “Society for the Rescue of Drowned Persons” was constituted in 1767, but the development of fatal pneumothoraces produced by vigorous attempts at resuscitation led to subsequent abandonment of such techniques. **John Hunter’s** innovative double-bellows system (one bellow for blowing in fresh air, and another for drawing out the contaminated air) was adapted by the society in 1782, and introduced a new concept into the ventilator care (*Randel-Baker, 1963*).

In 1887, **George Fell of Buffalo** revived the technique, describing an apparatus to maintain artificial respiration via a tracheotomy or facemask (*Mushin et al., 1991*). **Joseph O’Dwyer**, modified this to create the Fell-O’Dwyer apparatus (Fig.1) allowing ventilation with bellows via a tracheal tube, but didn’t use it for anaesthesia.



Fig (1): Fell-O'Dwyer apparatus.

In Europe, thoracic surgeons were drawn to **Sauerbruch's** solution to "pneumothorax problem" because it didn't depend on skilled assistance, but used a negative-pressure chamber similar to a giant "iron lung" to ventilate the patient. The patient was placed in an airtight operating chamber positioned with the head through an opening, which was sealed around the Patient's neck. Outside the chamber an assistant at the patient's head administered the anesthetic, while the surgeon was in the chamber with the rest of the patient (*Mackenzie, 2008*).

A colleague of **Sauerbruch's**, **Brauer** developed apparatus based on the opposite principle. He sealed the patient's head within an air tight, pressurized box with sealed holes for the anesthetist's hands. Anesthesia was administered via a face mask from a **Roth Drager** anaesthetic machine, the bag which was sealed within another pressurized chamber (*Ball, 2012*).

In 1907, **Drager** used the concept of positive airway-pressure in an apparatus named the pulmotor (Fig. 2). This device gained great popularity over the next several decades among the police and fire rescue units in Europe and the US. It was intended to provide artificial ventilation in resuscitation, particularly in fires and mine accidents (*Chen et al., 1998*).

The apparatus forces an air-oxygen mixture into the patient's lungs at a pressure of 20cmH₂O. During expiration, a spring and taggle mechanism altered the machine's valve position so that the lungs were emptied by a negative pressure of 20cmH₂O. **Drinker and Mckhann** judged the effectiveness of the pulmotor to be “disappointing”. They cited the fact that the patients tend to fight any mask over the face and oppose the efforts of the machine. In one patient, in whom life was maintained for two days by the pulmotor, the stomach and esophagus were ruptured and gastric contents were present in large amounts in the mediastinum (*Chen et al., 1998*).

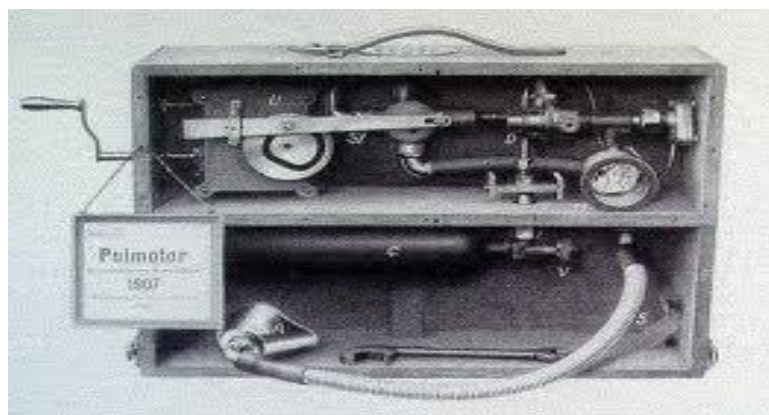


Fig. (2): Draeger pulmotor.

During the first half of the twentieth century, large epidemics of poliomyelitis culminated in the final re-inventions of tank NPV (Negative pressure ventilation) by **Stewart and Rogoff** in 1918 and then by Philip **Drinker and Louis Shaw** in 1927. Evidence confirming the clinical success of long term negative-pressure ventilation was published in 1929 (*Mackenzie, 2008*).

It was the availability of the electrical energy during the poliomyelitis epidemics that rendered Drinker's apparatus at once necessary and practical. The iron lung (Fig. 3) proved durable and was relatively easy to operate. It was, however, a cumbersome device that offered no means for controlling the ventilator flow rate. Moreover, access to the patient was limited to portholes in the tank side and was possible only during the expiratory phase. The negative pressure could produce abdominal vascular pooling resulting in decreased cardiac output and "Tank shock" in polio patients (*Barbarsh, 1990*).

Negative pressure ventilators were extensively used during the polio epidemic that ravaged Los Angeles in 1948 and Scandinavia in 1952. During the Scandinavian epidemic nearly 3 thousand polio affected patients in the community diseases hospitals of Copenhagen over a period of less than six months (*Meyer, 1974*).

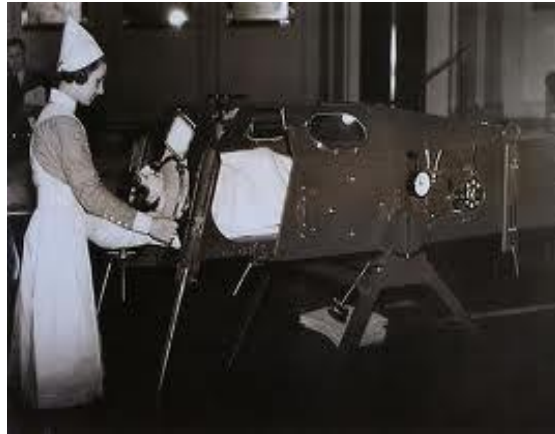


Fig. (3): Drinker's and Shaw's "Iron Lung".

In 1950, responding to a need for better ventilators, **Ray Bennet** and Colleagues developed an accessory attachment with which it became possible to intermittently administer positive pressure breaths in synchrony with negative pressure breaths delivered by a tank ventilator (*Hasan, 2010*).

During an epidemic of poliomyelitis in Stockholm in 1949 and 1950, **Carl-Gunner Engstrom**, an epidemiologist at the Stockholm Hospital for contagious diseases, observed a series of patients with respiratory failure treated with tracheotomy and negative pressure ventilation, a technique recently imported from the US by Sjoberg (*Mackenzie, 2008*).

Engstrom's study of respiratory function in these patients which included blood gases analysis, led him to conclude that these patients were dying of inadequate ventilation rather the poliomyelitis itself, as was believed at that time. He therefore designed and had built a prototype of a mechanical -positive

pressure ventilator which guaranteed the delivery of a set tidal volume rather than inspiratory pressure (*Mackenzie, 2008*).

After polio epidemics, the 1960 became an era of respiratory intensive care. Positive pressure ventilation with use of an artificial airway replaced the bulky and cumbersome negative pressure technology of respiratory support. Two types of ventilators and two modes of mechanical ventilation evolved during this period. First type of ventilators was pressure cycled (PCV). Two ventilators were commonly used for PCV in 1960's and 1970's, Bird Mark 7 and Bennet PR2. Second type of ventilators that got evolved from historical perspective is the volume cycled (VCV). The first fluidic ventilator utilizing moving streams of liquid or gas for sensing, logic amplification and controls was designed for the US army in 1964 by Barila and the first commercial versatile ventilator "Hamilton standard PAD" appeared in 1970 (*Kotur, 2004*).

The Final step into the current generation of ventilators was the development in the 1980s of microprocessor-controlled machines such as the Siemens 900c (1980), Ohmeda CPU-1(1982) and the Puritan –Bennett 7200 (1983) (Fig. 4) (*Mackenzie, 2008*).



Fig. (4): Puritan Bennett 7200.

Of late, resurgence in the popularity of noninvasive positive pressure breathing and the advent of high frequency positive pressure ventilation have further invigorated the area of the mechanical ventilation; it also remains to be seen whether the promise of certain as yet unconventional modes of ventilation will be borne out in the near future (*Hasan, 2010*).

MECHANICS OF BREATHING

Only approximately 10% of the lung is occupied by solid tissue, with the remainder being filled with air and the blood. Supporting structures of the lung must be delicate enough to allow gas exchange, yet strong enough to maintain architectural integrity and sustain alveolar structure. Two interrelated systems exist to perform the functions of the lung: (i) airways for ventilation, divided into the conducting airways (dead-air space) and the gas exchange portions, and the (ii) circulatory system for perfusion. Both operate under low pressure (*Slonim, 2006*).

Breathing involves two actions: *Inspiration* (active process) and *expiration* (a relatively passive process). Both actions rely on respiratory muscle function & the effects of pressure differences in the lungs. During normal respiration, the external intercostal muscles and the diaphragm descends to lengthen the chest cavity while the internal intercostal muscles (located between and along the lower borders of the ribs) contract to expand the anteroposterior diameter. This coordinated action causes a reduction in intrapleural pressure and inspiration occurs. Rising of the diaphragm and relaxation of the intercostal muscles causes increase in the intrapleural pressure and expiration results (*Bilotta et al., 2009*).

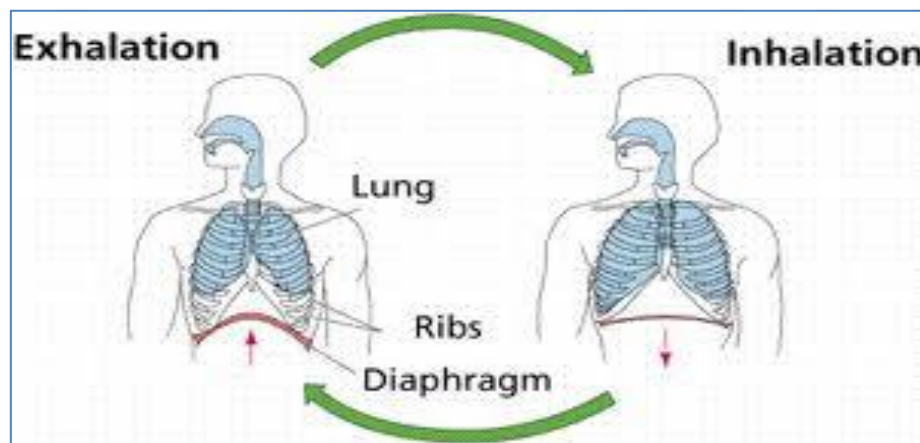


Fig. (5): Muscles of respiration during breathing cycle.

Internal and External respiration:

Effective respiration consists of gas exchange in the lungs, called external respiration and gas exchange in the tissues, called internal respiration. Internal respiration occurs only through diffusion. External respirations occur only through three processes:

- 1- Ventilation: gas distribution into and out of the pulmonary airways.
- 2- Pulmonary perfusion: blood flow from the right side of the heart, through the pulmonary circulation into the left side of the heart.
- 3- Diffusion: gas movement through a semi permeable membrane from an area of greater concentration to one of lesser concentration (*Bilotta et al., 2009*).

Pulmonary perfusion

Blood flow varies in different regions of the lung. In the upper regions (zone 1), alveolar pressure exceeds arterial pressure and units receive no perfusion. In the mid lung (zone 2), arterial pressure exceeds the alveolar and both are greater than venous pressure. Flow will thus depend on the degree of compression of the pulmonary capillaries by alveolar pressure. The greater the arterial pressure, the wider open vessels are held and flow increases. In the lowest parts of the lung (zone 3), both venous and arterial pressure exceed alveolar pressure. The vessel between artery and vein will be held open and flow will relate to the A-V pressure difference (*Mackenzie, 2008*).

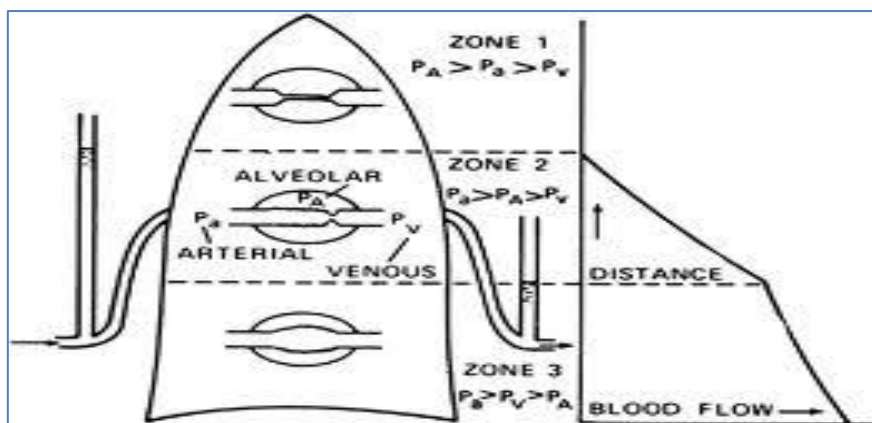


Fig. (6): Three-zone model of the lung.

Ventilation-perfusion match

Gravity can affect O_2 and CO_2 transport in a positive way. Gravity causes more unoxygenated blood to travel to the lower and middle lung lobes than to the upper lobes. This explains why