

## **PART ONE CHAPTER ONE**

### **PRIMARY CONSIDERATIONS RELATING TO THE PHYSIOLOGICAL AND PHYSICAL ASPECTS OF THE MECHANICAL VENTILATION OF THE LUNG**

#### **POSSIBLE ORIGIN OF THE MECHANICAL VENTILATION OF THE LUNG-**

The first means of the mechanical ventilation of the lung most likely employed a rudimentary bellows device on the order of today's ornamental "fireplace bellows". If the proximal pulmonary airway entrances (nose and mouth) were isolated by some sort of a mechanical airway sealing device enabling a direct connection to the single "spout" of the bellows, a mechanical physical/physiological airway would have been created.

When the bellows were expanded a "sub ambient (suction) proximal airway pressure could be created" in the process of attempting to "suck" air out of the lungs. When the inflated bellows were squeezed, a positive air pressure could be created at the proximal airway. Rudimentally, this form of mechanical ventilation of the lung could be called a "suck and blow" respiration. In this case the neutral pressure (REFERENCE PRESSURE) around the proximal airway (head) could be called Ambient.

Over time, the provisions for the mechanical ventilation of the lung became increasingly sophisticated. The body (tank) respirator encapsulated the patient's body with a seal around the neck venting the patient's proximal airway to ambient. As the (iron lung) bellows expanded the pressure around the entire body became Sub Ambient creating a "negative" pressure at the proximal airway.

This was called the "inspiratory phase" causing ambient air at a higher pressure than within the lungs to flow down the tracheo-bronchial tree to inflate the pulmonary alveoli. During the "expiratory phase" the tank respirator bellows were compressed, causing the pressure around the body to become positive in reference to the ambient proximal airway pressure. This increased the air pressure within the lungs, causing air to flow out of the lungs to ambient through the mouth and nose. In this case the REFERENCE PRESSURE around the body could be called ambient (neutral pressure being neither positive nor negative).

The inflationary pressure difference between the Proximal (mouth-nose) and the Distal (pulmonary alveoli) lung is called a "pressure gradient" when measured from ambient. When the pressure is higher at the Proximal airway (mouth and nose), air-flows into the lungs. Then, when the Distal pressures within the pulmonary alveoli are higher than the proximal airway, air flows back out of the lung to ambient.

When the pressure is higher at the Proximal airway than in the Distal lungs and air flows into the lungs, the pressure difference (PRESSURE GRADIENT) between the Proximal airway and Distal lungs will determine how fast the air will flow into the lungs. This is called the "INSPIRATORY FLOWRATE".

After the lungs are mechanically inflated with a scheduled positive inspiratory inflow gradient, the inspiratory air must be exhaled. During the “Expiratory Phase” the inspired air within the pulmonary alveoli must be exhaled before the lungs can be mechanically re-inflated during the next breath.

It is important to understand that the lungs can be inflated by mechanically increasing the pressure at the Proximal airway (TRACH POSITIVE VENTILATION) above ambient, thus creating an inspiratory flow gradient into the lungs. The “Proximal airway inspiratory pressure gradient” is regulated, creating an inspiratory flowrate (pressure gradient) sufficient to inflate the lungs against their internal resistance to expansion. Even more important is to not use too much “sustained inspiratory positive pressure” to create a sustained peak positive pressure (PIP) sufficient to over expand the pulmonary structures causing pulmonary barotrauma.

When the pulmonary structures are inspiratorily inflated, there are elastic components (and surface tensions) that are stretched beyond their normal resting positions. After inspiration, during either spontaneous (normal) or mechanical ventilation, the lungs are then said to “PASSIVELY RECOIL” toward their normal “END EXPIRATORY resting position”. It is this normal expiratory passive recoil (after inspiration) which serves to partially empty the lungs. The lungs do not completely empty during exhalation. Air is only exhaled from the lungs until all the emptying forces within the pulmonary structures reach their “end exhalation resting position”. This end resting position determines how much air is left in the partially deflated lungs, and is called the “FUNCTIONAL RESIDUAL CAPACITY (FRC)”

The amount of air exchanged during each breath is called the Tidal Exchange. The “breath by breath Tidal Exchange” changes only part of the air within the lungs. The pulmonary alveoli and their immediate peripheral airways (respiratory bronchioles) have imbedded micro capillaries which form a tight network having a pulsatile blood flow propelled through them by the beating (pumping action) of the right ventricle of the heart.

The blood flowing through the capillaries lining the semi porous membranous walls of the peripheral pulmonary gas exchange structures (blood gas interface) allow oxygen molecules to pass through them into the red cells (hemoglobin), to then be transported (pumped by the left ventricle) out of the lungs to the tissue cells of the entire body.

Each repetitive tidal volume creating intrapulmonary gas exchange serves to mix the intrapulmonary gases mainly consisting of nitrogen, oxygen and carbon dioxide. Carbon dioxide is continually released from the pulmonary capillaries entering the alveoli to be mixed with intrapulmonary gases for exhalational “wash out”.

The upper bronchial airways serve only as transporting channels to deliver fresh oxygen to the peripheral pulmonary airways with pulmonary capillaries, as well as to “flush” carbon dioxide out of the lungs to ambient.

The volume of the non perfused airways (without pulmonary capillaries) is termed “Anatomical Dead Space” which can be less than 5cc in a very small neonate to over 175 cc in a large adult.

During “spontaneous basal (normal) ventilation” the Tidal Volumes are generally less than the Anatomical Dead Space. From 10 cc in a baby, to about 500 cc in a large adult. Respiratory rates in a very small baby can be over 25 breaths per minute, up to 20 breaths per minute in an adult with an adult average of about 13-15.

Sub Tidal gas exchange (less than the anatomical dead space) is referred to as a “Sub Tidal Volume”. Tidal exchanges greater than the Anatomical Dead Space are called Convective Tidal Volumes or sometimes Sighs.

Gas mixing within the lungs during spontaneous breathing is caused primarily by diffusion (the kinetic {Brownian} randomized movement of molecules at a given temperature within an atmosphere) within the pulmonary airways. CO<sub>2</sub> diffusion is some 20 times greater than oxygen within the lungs, greatly enhancing its upward travel in a head ward (cephalad) direction out of the lungs.

Convection is defined as a mass tidal air movement (such as large volumes of warm air rising from the earth’s surface upward into cooler air). When a tidal volume greater than the Anatomical Dead Space is mechanically delivered into the lungs it is said to be a “CONVECTIVE TIDAL EXCHANGE”.

During the mechanical ventilation of the lungs, Tidal Deliveries less than the Anatomical Dead Space are considered to be SUB TIDAL VOLUMES, more related to enhancing diffusive OXYGEN delivery than CO<sub>2</sub> “wash out”. When Tidal Volumes in excess of the “existing physiological Anatomical Dead Space” are programmed with a Mechanical Respirator/Ventilator they produce a CONVECTIVE PULMONARY VENTILATION favoring CO<sub>2</sub> “wash out”.

The pulmonary airways and their alveoli are physiologically maintained in a partially expanded position by the semi rigidity of the thoracic cage and diaphragmatic limits, with a potential Sub Ambient pressure maintained within the pulmonary pleural membranes. The (non Neonatal) patency of the uppermost large tracheo-bronchial airways is maintained in part by C shaped cartilaginous rings.

The scheduling of a potential proximal airway SUB AMBIENT PRESSURE during the mechanical ventilation of the lung can serve to collapse the pulmonary airways (especially in neonates or in patients with obstructive airways) if allowed to substantially cross the mechanical airway into the physiological airway structures. Therefore, the only known practical use of a Sub Ambient phase during the mechanical ventilation of the lung is in Neonates with small indwelling airway catheters (2.5 mm), to overcome the physiological expiratory flow resistance imposed by the indwelling mechanical airway.

The level of maximal inspiratory resistance (bottleneck) will almost always be the caliber of the indwelling airway catheter. Therefore, there may be a considerable proximal airway pressure drop across the endotracheal tube (R4/L).

Supplemental Oxygen during the mechanical ventilation of the lung is referred to as the “FINAL INSPIRATORY OXYGEN CONCENTRATION (FIO<sub>2</sub>) to enhance the existing PaO<sub>2</sub> (arterial oxygen) during the mechanical ventilation of the lung.

The blood gas interface can be enhanced during the mechanical ventilation of the lung by increasing the Dynamic Functional Residual Capacity (D/FRC) of the lung by using a PEEP or CPAP to maintain a positive expiratory pressure at the proximal airway.

Any increase in mean Intrathoracic Pressures are reflected upon the Pulmonary Arterial Pressures, which can be increased to the point of right heart strain or failure.

The longer the sustained inspiratory positive pressure (PIP) “during the mechanical ventilation of the lung”, the greater the tendency toward preferential airway and secondary barotrauma within the most dependent pulmonary structures. This is secondary to alveolar hyperinflation and Penduluft (post inspiratory apneustic back filling).

Mechanical ventilators can become challenged to effectively ventilate the lung through diffusive partial airway obstruction or interstitial congestion without increasing the mean intrathoracic pressures potentially to the point of right heart strain or failure. Therefore, lung protective strategies such as Intrapulmonary Percussive Ventilation (IPV®), which provides for ventilation associated with secretion mobilization and raising as well as the enhancement of physiological Vesicular Peristalsis to enhance the intrathoracic Lymph Pump.

All mechanical ventilators are pressure limited. However, it is not the pressure rise but the sustenance of a potentially barotraumatic pressure at the Proximal Airway which creates the intrapulmonary over-expansion potential.

Each type of mechanical ventilator has advantages and disadvantages. They are:

1. The body (iron lung) respirator favors cardiac output being most physiological of all forms of mechanical ventilation of the lung. However, it will not ventilate lungs with low compliance.
2. The Trach Positive mechanical ventilator with a cuffed endotracheal tube will ventilate lungs with very low compliance. However, it can encroach upon cardiac output especially with low circulatory blood volumes.
3. The Pressure limited Respirator with a “moderate inspiratory Flowrate” will have the least potential for barotrauma. However, it may hypo or hyperventilate in the presence of changing gross pulmonary compliances.

4. **The Volume oriented ventilator, delivering a scheduled Tidal Volume under a selected peak inspiratory pressure (PIP), using a constant flow with a timed interruption to a volume (flowxtime=volume), will effectively ventilate patients with fairly normal lungs, such as non complicated post operative patients.**

However, patients with combined major diffuse airway obstruction, atelectasis, oxygen toxicity and interstitial congestion can be advanced toward “Ventilator Induced Respiratory Distressed Syndromes” with Volume Limiting Techniques.

5. **High Frequency Diffusive Ventilators (Jet Ventilators) can facilitate PaO<sub>2</sub>, however they may not have “sufficient Convective Component” to blow off CO<sub>2</sub> leading to Hypercapnia. Thus the patient can die nice and pink with a CO<sub>2</sub> narcosis. Potential Carinal (Airway Bifurcation) trauma “from Jet Ventilation (technique) mandating uncuffed indwelling airway catheters”, limit this modality to short term procedures.**
6. **High Frequency Vibratory Ventilators generating high impedance “Shock Waves” directed into the proximal airway produce a pure diffusive ventilation with equalized directional molecular diffusion at the proximal airway. High Oxygen partial pressures can be diffused into the airway while “diffusing respiratory gases with lower oxygen concentrations” out of the airway to Ambient, while maintaining Ambient airway pressures. If the patient is apneic the lack of an adequate Convective Ventilation can lead to a CO<sub>2</sub> narcosis.**
7. **High Frequency Oscillatory (push-pull) Ventilators for (HFOV), enhance endobronchial diffusion with a limited Tidal Exchange by modulating a continuous positive airway pressure (CPAP). Continuous CPAP values must be sustained at levels approaching 20 cm H<sub>2</sub>O to potentially control CO<sub>2</sub>. HFOV basically provides for a (high lung volume) Neonatal Ventilator for selected patients with systemic hyper perfusion without cardiac complications. The lack of Convective Ventilatory “wash out” ability to control CO<sub>2</sub>, increases with patient size.**
8. **Intra Pulmonary Percussive Ventilation (IPV®) provides for a unique high frequency “Percussive endobronchial delivery” of Sub Tidal Volumes to a scheduled limit of lung inflation, at which point the lung is Percussively Pulsed to produce intrapulmonary mechanical gas mixing. The Percussive Mechanical Gas Mixing serves to produce intense endobronchial diffusion. An automatic intrapulmonary wedge pressure is maintained between sub tidal deliveries by an automatic 1:2.5 i/e ratio to prevent the collapse of recruited peripheral airways. Concomitant with pulsed Tidal Deliveries, selected aerosols are topically delivered to provide for vasoconstriction and bronchodilation. Spontaneous breathe through is unobstructed.**

**IPV® can produce selected levels of Endobronchial Percussion to provide for the initial mobilization of retained endobronchial secretions followed by the generation of a secondary level of Percussion for the Cephalad raising of mobilized secretions. Additionally, the Percussive amplitude can be further increased to provide for a Vesicular Peristalsis, to the three intrathoracic circulations, namely Bronchial, Pulmonary and Lymph.**

**Traditional volume oriented critical care ventilators can be provided with a “Percussive Ventilatory component” to enhance diffusion by means of an available IPV® interfacing circuit.**

- 9. Volumetric Diffusive Respiration (VDR®) is the critical care version of Intrapulmonary Percussive Ventilation (IPV®). VDR® is designed to provide a combination of Diffusive Intrapulmonary gas mixing with an intermittent scheduled Convective Tidal Exchange to “wash out” CO<sub>2</sub>. One ventilator is capable of being programmed to ventilate the smallest Neonate through Pediatrics to the largest Adult. VDR® programmed for High Frequency Percussive Ventilation (HFPV) continues to prevail when all other means of mechanical ventilation have failed.**
- 10. High Frequency Percussive Oscillatory Ventilation (HFPOV™) provides for a Percussive (push-pull) Oscillation for endobronchial gas mixing. Unique to the Oscillatron® Percussionator® Ventilator is the 7 millisecond Transition penalty compared to about a 22 millisecond Transition penalty mandated by design on Piston and Magnetically servoed (push-pull) Oscillators.**

**The major reduction in the Transition penalty enables the “Oscillatron® fluidic oscillator” to obtain major increases in Sub Tidal exchanges at the same cyclic frequency. Therefore, the Continuous Positive Airway Pressure (CPAP) serving as a carrier for Percussive Oscillatory modulation can be reduced from about 20 cm H<sub>2</sub>O during HFOV to less than 10 cm H<sub>2</sub>O, with HFPOV, greatly reducing the mean intrathoracic pressures.**

**By reducing the mean intrathoracic pressures, the impact upon pulmonary arterial pressures lessens the impact upon right heart strain. A major advantage of the Oscillatron® with Percussive (push-pull) oscillation is that of ventilating larger patients without CO<sub>2</sub> limitations.**

**The Oscillatron® is available as a self contained High Frequency Oscillatory Ventilator with Demand CPAP or as an Oscillatron® Servo to interface with most Convective CMV critical care ventilators.**

**OSCILLATION IS DEFINED AS A TO AND FRO MOVEMENT**

**A POSITIVE PHASE OSCILLATOR HAS A PASSIVE EXHALATION**

**A DYNAMIC (PUSH-PULL) OSCILLATOR IS A POSITIVE PHASE OSCILLATOR WITH A POTENTIAL SUB AMBIENT EXPIRATORY PHASE**

**A PERCUSSIVE OSCILLATOR CAN BE EITHER A POSITIVE OR A DYNAMIC PHASE OSCILLATOR PROVIDING FOR**

**THE INJECTION OF PHASIC HIGH FREQUENCY SUB-SONIC SHOCK WAVES INTO THE ENDOBRONCHIAL STRUCTURES**

**CREATING A FOLLOW ON CAVITATION AS THE HIGH VELOCITY SHOCK WAVES TRAVEL DOWN THROUGH THE DESCENDING AIRWAYS**

**THE ACCELERATED HIGH ENERGY SUB TIDAL VOLUME, WHICH SERVED TO CREATE THE DETACHED SHOCK WAVE (BOUNDARY LAYER) IS GUIDED PROXIMALLY INTO THE PERIPHERAL AIRWAYS BY THE CAVITATIONS BEHIND THE DESCENDING SHOCK WAVES**

**THE AMPLITUDE AND SUSTENANCE OF THE GENERATED SUB TIDAL VOLUME, IS REGULATED TO PRODUCE A PERIPHERAL INSPIRATORY PENETRATION OF THE PULMONARY ALVEOLI**

**NEAR INSTANTANEOUSLY THE PROXIMAL AIRWAY IS VENTED TO AMBIENT CREATING A DISTAL/PROXIMAL EXPIRATORY FLOW GRADIENT, WHICH IS ENHANCED BY THE PULMONARY AIRWAY COUNTER FLOW CREATED BY THE NEWTONIAN AND BERNOULLIAN INFLOW/OUTFLOW EFFECTS**

**THE PROGRAMMED INSPIRATORY/EXPIRATORY "i/e RATIO" 1.5 to 2:2.5 MEASURED IN MILLISECONDS, CAUSES THE EXPIRATORY FLOW FROM THE PERIPHERAL LUNG STRUCTURES TO CEASE, BEFORE TERMINAL AIRWAYS COLLAPSE, BY INITIATING A FOLLOW ON SUB- TIDAL DELIVERY**

**THE NEAR INSTANTANEOUS DYNAMIC EXPIRATORY SUB AMBIENT FLOW GRADIENT REVERSAL IS CREATED WITHIN THE MECHANICAL AIRWAYS REDUCING THEIR EXPIRATORY RESISTANCES THUS ENHANCING PHYSIOLOGICAL INTRAPULMONARY EXPIRATORY OUTFLOW**

**THE CREATION OF A SUB AMBIENT PRESSURE WITHIN THE PHYSIOLOGICAL AIRWAYS CAN CAUSE THEIR COLLAPSE**