

Newer Modes Of Ventilation: An Overview

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Citation

L Weavind, O Wenker. *Newer Modes Of Ventilation: An Overview*. The Internet Journal of Anesthesiology. 1999 Volume 4 Number 4.

Abstract

PATHOPHYSIOLOGY

Before visiting new modes of ventilation lets shortly review some reasons why patients have to be ventilated and why we are looking for new and more physiological ways to ventilate patients.

Patients are intubated and ventilated in order to treat acute respiratory failure. Acute respiratory failure has been defined as

- Type 1: Hypoxemia without CO₂ retention (e.g. asthma, pneumonia, pulmonary edema and pulmonary embolism.)
- Type 2: Hypoxemia with CO₂ retention (e.g. chronic bronchitis, chest injuries, unconscious drug overdose, postoperative hypoxemia and neuromuscular disease-hypoventilatory causes)

Hypoxemia is defined as a PaO₂ < 60 mmHg on > 50% FiO₂.

Reasons for Acute Respiratory Failure (ARF) in our ICU population (most of our surgical patients have underlying COPD secondary to smoking):

- Acute bronchitis and pneumonia from viruses, bacteria and fungi
- Mucous plugs may precipitate ARF in our tenuous patient population
- Left ventricular failure secondary to ischemia or postoperative fluid volume
- Pulmonary embolism is not commonly seen in our ICU, but is an important cause of both ARF and acute cardiac failure.

- Pulmonary hemorrhage seen as diffuse alveolar hemorrhage in our immunosuppressed patients
- Pneumothoraces whether iatrogenic or spontaneous
- Sedation and aspiration
- Bronchospasm

METHODS OF VENTILATION

We all know the standard Assist/Control (A/C) and Synchronized Intermittent Mandatory Ventilation (SIMV), these two methods are flow triggered/limited and volume-controlled, they may also be pressure limited to prevent peak airway pressures going up over 50 mmHg. An alternative to this strategy is Pressure Controlled Ventilation (PCV), which while limiting peak airway pressures to acceptable levels does not guarantee acceptable minute ventilation as the tidal volume achieved at these limited pressures may be inadequate to ventilate and oxygenate a patient.

ADVERSE EFFECTS OF VENTILATION

- Hemodynamic compromise as ventilatory pressures are transmitted to the heart and cause cardiac embarrassment.
- Barotrauma (interstitial emphysema, pneumothorax and pneumomediastinum) as high airway pressures and levels of PEEP are used causing alveolar rupture.
- Epithelial leaks from increased shear forces can lead to increased microvascular permeability and alveolar edema
- Oxygen Toxicity (substernal distress, absorption atelectasis and pulmonary edema, also cns toxicity-seizures-divers) and V/Q mismatch

- Need for sedation/neuro-muscular blockers because patients are dyssynchronous with the ventilator.
- Lung injury with surfactant deficiency.

Our goals in limiting ARDS and ventilator induced lung injury can be realized by preventing lung overdistention and repeated collapse and reopening of alveoli. Oxygen delivery and not ventilation is now our primary goal as we try to optimize alveolar ventilation with the application of the least possible positive airway pressure. Permissive hypercapnea is now an accepted practice as we sacrifice CO₂ removal if it conflicts with low-pressure goals.

VENTILATION MODES

All these above reasons have spurred a search for newer more physiological methods to ventilate patients. Technical development has evolved and upgraded the ventilator to allow new ventilatory strategies which now allow us to use pressure constant breaths in Pressure Control-Inverse Ratio Ventilation (PC-IRV), Airway Pressure Release Ventilation (APRV) or BiLevel to attempt to limit lung damage and preserve spontaneous breathing.

PRESSURE CONTROL-INVERSE RATIO VENTILATION (PC-IRV)

In PC-IRV we set an inflating pressure, inspiratory time (usually the I:E ratio is > than 1:1) and frequency. There is a rapid achievement of inflation pressure with high initial gas flows and full inflating pressure is reached early in the inspiratory cycle. The selected inflation pressure is held throughout the inspiratory cycle and then followed by a rapid deceleration in flow. The advantage of PC-IRV to lungs with V/Q mismatch are: 1) the prolonged inspiration ensures a more homogeneous ventilation and keeps collapsible alveoli open for a longer period of time (alveolar recruitment); and 2) during the short expiration slower compartments will not exhale completely, they remain distended by auto-peep (improved compliance and FRC).

PC-IRV is also associated with a higher mean airway pressure (MAP) than standard modes of ventilation and MAP is a key determinant of gas exchange. Inspiratory pressure also remains constant throughout the whole inspiratory cycle and this may be reason for alveolar recruitment as closed airways often require moderately high and sustained pressures to open.

Disadvantages of using PC-IRV include the need for deep

sedation and neuro-muscular blockade, because it is uncomfortable and to prevent dyssynchrony between patient and ventilator. Prolonged sedation and neuro-muscular blockade contributes to patient confusion, inability to clear secretions, increased muscle catabolism and inability to assess patient's neurologic status. Ultimately it may lead to a significantly longer ICU and Hospital stay.

AIRWAY PRESSURE RELEASE VENTILATION (APRV)

Airway Pressure Release Ventilation (APRV) is basically a set level of CPAP that intermittently releases to a lower level using a time-controlled release valve. High CPAP and lung volumes are re-established when the release valve closes. The principle of reducing lung volume distinguishes this technique from other modes of ventilation. APRV always implies a reverse I:E ratio as it utilizes a very short expiratory time for PRESSURE RELEASE. Tidal ventilation occurs between the increased lung volume established by the application of high CPAP and the relaxation volume of the respiratory system i.e. the return to FRC. The mechanical tidal volume is determined by the release pressure, release time, and airway and breathing circuit impedance.

The clinical benefits of APRV include the preservation of spontaneous breathing and comfort with most spontaneous breathing occurring at high CPAP but it can occur throughout the ventilatory cycle with little work of breathing being imposed by the ventilator. This results in less barotrauma; reduction in circulatory compromise; and a better matching of pulmonary ventilation and perfusion.

Certain patients are poor candidates for APRV secondary to their underlying pulmonary states namely patient's with increased airway resistance or those unable to empty their lungs in 2 seconds (Asthma or severe COPD).

BIPHASIC POSITIVE AIRWAY PRESSURE (BIPAP) AND BILEVEL VENTILATION

Biphasic positive airway pressure (BIPAP) is pressure-controlled ventilation, which allows unrestricted spontaneous breathing throughout the respiratory cycle. In BiPAP the circuit switches between a high and low airway pressure in an adjustable time sequence. If the patient is not breathing spontaneously, this mechanical volume displacement is taken as a pressure controlled mechanical ventilation. The I:E ratio and the ventilatory frequency can be adjusted to optimize ventilation and oxygenation. If you reverse the I:E ratio then you have APRV. BiLevel is a

combination of APRV and BIPAP, it mixes spontaneous and mandatory breath types. The mandatory breaths are pressure controlled and the spontaneous breaths can be pressure supported. In BiLevel the ventilator cycling between the two pressure levels can be synchronized with the patient to prevent a cycle to low pressure just as a patient takes a breath. Spontaneous breaths can be pressure supported at the high and low pressure levels. The advantages of this method of ventilation are the decreased level of sedation required to facilitate patient acceptance of the ventilator and the concept of a single modality to ventilate the patient. BiLevel may initially be used as pressure controlled ventilation, weaned to BIPAP and then weaned to CPAP prior to extubation without a mode change on the ventilator. BiLevel can offer full ventilatory support and can then be weaned off, as the patient ventilatory needs resolve.

HIGH FREQUENCY VENTILATION

High Frequency Ventilation can be either jet ventilation or oscillation. In jet ventilation high-pressure gas (30-300kPa) is delivered into the airway via a small-bore catheter at high frequencies (60-300/min). Air or supplementary O₂ is entrained with the jetted ventilatory breath and expiration is purely passive. This form of ventilation is mostly used in the operating room where a surgeon is working in the airway (laser Rx of papilloma on vocal cords) and you can not place an ETT or as an emergency airway. Another situation where it may come in handy is when you are treating a disrupted airway or massive bronchopleural fistula as the non bulk flow of gas decreases the amount of gas escaping out of the fistula. Always insure that you monitor expiration as it is passive and if you do not allow enough time for full exhalation you may cause iatrogenic barotrauma and

pneumothoracies.

High frequency oscillation is a difficult concept to explain. Oscillators operate with a to-and-fro application of pressure on the airway opening at high frequency (100-1000/min). Oscillator tidal breaths are usually smaller than anatomic dead space. Five mechanisms to explain gas transport under these unphysiologic conditions are: (1) bulk flow can still provide conventional gas delivery to proximal alveoli with low regional dead space volumes. (2) Coaxial flow (i.e., gas in the center flows inward, while gas on the periphery flows outward) can develop because of the asymmetric low profile of high-velocity gases. (3) Taylor dispersion can produce a mixing of fresh and residual gas along the front of a flow of gas through a tube. (4) Pendelluft can mix gases between lung regions having different impedences. (5) Augmented molecular diffusion can occur at the alveolar level secondary to the added kinetic energy from the oscillations.

The two conceptual advantages to HFO are lower peak airway pressures and the fact that nonbulk-flow mechanisms may improve V/Q matching. Inadequate humidification is a well-known complication when using high gas flows and delivered minute volumes and may result in necrotizing tracheobronchitis. HFO may also cause direct physical airway damage.

LIQUID VENTILATION

Tracheal Gas Inflation

Extra Corporeal Membrane Oxygenation (ECMO)

ExtraCorporal CO₂ Removal (ECCO₂R)

References

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