Mechanical ventilation in neurosurgical patients

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ABSTRACT

Mechanical ventilation significantly affects cerebral oxygenation and cerebral blood flow through changes in arterial carbon dioxide levels. Neurosurgical patients might require mechanical ventilation for correction and maintenance of changes in the pulmonary system that occur either due to neurosurgical pathology or following surgery during the acute phase. This review discusses the basics of mechanical ventilation relevant to the neurosurgeon in the day-to-day management of neurosurgical patient requiring artificial support of the respiration.

Key words: Acute respiratory distress syndrome, hyperventilation, mechanical ventilation, neurosurgery, pulmonary mechanics

INTRODUCTION

Neurosurgical intensive care unit (ICU) is a place where the patient care is a shared obligation between neurosurgeon and neuroanesthesiologist and hence the former also needs to have a good understanding of the dynamics of mechanical ventilation. This article intends to simplify the essence of mechanical ventilation.

For scientific use of mechanical ventilation we need to conceptualize:

1. Basic of pulmonary mechanics and ventilator settings
2. Its effect on intracranial pressure (ICP), cerebral perfusion pressure (CPP) and brain oxygenation
3. Varied ventilatory strategies that help in different neurological scenarios, which may not conform to the set protocols in medical and surgical ICU
4. Weaning from the ventilator.

The important goals of mechanical ventilation are adequacy of ventilation and oxygenation, a reduction of work of breathing and the assurance of patient comfort and synchrony with the ventilator.

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INDICATIONS OF MECHANICAL VENTILATION

- Respiratory failure
- Severe hypoxemia
- Excessive work of breathing
- Hypoventilation
- Respiratory muscle dysfunction
- Respiratory muscle fatigue
- Chest wall abnormalities
- Neuromuscular disease
- Increased airway resistance or airway obstruction
- Cardiopulmonary arrest
- Trauma (especially head, neck and chest)
- Pulmonary impairment (infections, tumors, pneumothorax, chronic obstructive pulmonary disease, trauma, pneumonia, poisons)
- Refractory shock
- Use of hyperventilation to reduce ICP
- Sedation and/or neuromuscular blockade
- Prevention of atelectasis.

BASICS

Pulmonary mechanics

Compliance - work required to inflate the lungs

$$= \frac{\Delta \text{volume}}{\Delta \text{pressure}} (\text{L/cm of } H_2O) \quad (1)$$

Normal compliance is 0.2 L/cm of H_2O. Lesser the compliance harder it is to inflate the lungs.

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Elastance - work required to exhale

$$= \frac{\Delta \text{pressure}}{\Delta \text{volume}} (\text{cm of } H_2O/L) \quad (2)$$

Normal Elastance is 5 cm of H_2O/L.

Resistance – it is the work required for moving air through lungs $= \frac{\text{pressure}}{\text{flow}} (\text{cm of } H_2O/L/s)$ (3)

It is affected by airway diameter. Normal resistance is 0.6-2.4 cm of H_2O/L/s.

Basics of ventilation

A ventilator works on 3 sets of parameters:

1. Triggering/starting of inspiration: How a breath is initiated, i.e. transition from expiration to inspiration-it is of 4 types – manual trigger, patient trigger (assist), time trigger (control), patient/time trigger. Breath can be triggered either by ventilator (time as a variable) or by the patient (pressure, flow and volume as variables)
2. Limiting of inspiration/control of gas delivery: It is based on attaining desired pressure or flow
3. Cycling/end of inspiration: How a breath is terminated, i.e. transition from inspiration to expiration-based on set volume, set inspiratory time or set flow.

Basic breath types

1. Volume-cycled breath: It ensures the delivery of a preset tidal volume (Tv) (unless the peak pressure limit is exceeded). With volume-cycled breath, worsening airway resistance or lung compliance results in increases in peak inspiratory pressure with continued delivery of the set Tv (unless the peak pressure limit is exceeded)
2. Time cycled breath: This breath applies a constant pressure for a preset time. With this type of breath, changes in airway resistance or lung/chest-wall compliance will alter Tv (i.e. worsening of airway resistance or lung compliance results in decrease in tidal volume). E.g., pressure control ventilation
3. Flow cycled breath: These breaths are similar to time cycled but the inspiratory flow waveform is decelerating and they are terminated when the flow rate decreases to a predetermined percentage of initial flow rate (typically 25%). E.g., pressure support ventilation (PSV).

MODES OF MECHANICAL VENTILATION

Commonly used modes of mechanical ventilation include:

a. Assist Control Ventilation (ACV):
   1. Volume control ventilation
   2. Pressure Control Ventilation
b. Synchronized intermittent mandatory ventilation (SIMV)
c. Pressure support ventilation (PSV)
d. Controlled mechanical ventilation (CMV).

ACV

A preset Tv or a preset applied pressure and inspiratory time (pressure controlled ventilation) is delivered at a preset minimum rate. This mode is commonly used at the initiation of mechanical ventilation in patients of acute respiratory failure. With proper use of assist control, the work of breathing may be significantly decreased. Ventilator does not prevent patient's spontaneous effort for breathing. In some ventilators, this spontaneous effort can be supported by a pressure support augmenting patient synchrony and minute ventilation.

SIMV

This delivers either volume-cycled or time-cycled breaths at a preset mandatory rate. SIMV is initially set to deliver full ventilator support and support is then decreased as the patient tolerates spontaneous breathing. Synchronization allows for enhanced patient-ventilator interaction by delivering the preset machine breaths in conjunction with the patients inspiratory effort. When no effort is sensed, the ventilator delivers the set Tv at the set rate. SIMV is almost always combined with PSV to augment the spontaneous breaths.

PSV

This mode provides a preset level of inspiratory pressure assist with each spontaneous breath. PSV augments the patient's own efforts and is best adjusted by observing changes in the patient's respiratory rate (RR), Tv and comfort.

CMV

This delivers unassisted ventilator breaths at a preset RR. With current ventilators, there is no direct setting for this mode but it is achieved only in patients who are not capable of spontaneous respiratory efforts.

EFFECTS OF VENTILATION

On ICP

Positive end expiratory pressure (PEEP): PEEP has been known to increase intrathoracic pressure leading to a decrease in cerebral venous drainage and consequently to an increase in cerebral blood volume and ICP. Pulmonary compliance is one of the main factors affecting the transmission of PEEP to intracranial system. The increase in central venous pressure, and reduction

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in mean arterial pressure, CPP after PEEP have been found in patients with normal pulmonary compliance. Whereas in patients with low pulmonary compliance, where generally PEEP is used, it is shown that PEEP up to 12 mm Hg has no significant effect on cerebral and systemic hemodynamics.[1-3] One should be watchful of ICP, when applying PEEP in a patient with normal lung compliance and reduced intracranial compliance.

$PaCO_2$: High $PaCO_2$ > 45 mm Hg is associated with risk of cerebral vasodilatation, increased cerebral blood flow and ICP.

High frequency ventilation (HFV): HFV causes a decrease in ICP (especially traumatic brain injury (TBI) patients with both severe lung and brain injury). Here a RR is >150/min with a low T_V of about 1-5 ml/kg. This allows efficient ventilation and oxygenation with minimal ventilator induced lung injury and causes a decrease of intrathoracic pressure.[4]

Transition from controlled to spontaneous ventilation can be done only if ICP is within normal range since with a poor intracranial compliance, spontaneous breathing trial (SBT) will cause a rise of ICP.[5]

Inspiratory: Expiratory (I:E) ratio: Inversion of ratio have no direct effect on ICP.[6]

Fiberoptic bronchoscopy: It causes an increase in ICP (occurs even if patient is paralyzed and with the use of cough suppressant).[7]

On $PaCO_2$

Hyperventilation is used to reduce $PaCO_2$ from the normal range of 35-45 mm of Hg to a range of 30-35 mm of Hg to cause an acute decrease of ICP.[8]

This is a temporary phenomenon as it reverts back due to adaptive intracerebral shifts of bicarbonate ion within 6-12 hrs of initiation of hyperventilation. Studies have shown an increasing trend toward mortality with aggressive hyperventilation in TBI (Brain Trauma Foundation (BTF) guidelines do not recommend pre-emptive hyperventilation).[9]

On brain oxygenation ($pbtO_2$)

BTF guidelines suggest a target $PbtO_2$ > 15 mm of Hg.[10]

Brain cannot tolerate a level of <10 mm of Hg for 30 min or a lowest level of 6 mm of Hg regardless of duration.

Hyperventilation can lead to a decrease in global brain oxygenation but $PbtO_2$ monitoring routinely cannot be recommended.[11]

VENTILATOR SETTINGS FOR THE INITIATION OF MECHANICAL VENTILATION

1. Choose a familiar ventilator mode to provide adequate oxygenation/ventilation, reduced work of breathing, synchrony between patient and ventilator and avoidance of high airway pressures.
2. The FiO_2 can be titrated to maintain SpO_2 at >92%.
3. Initial Tv = 8-10 ml/kg in patients with normal lung compliance. In patients with poor lung compliance (e.g. acute respiratory distress syndrome (ARDS) a target V_T = 6 ml/kg is recommended to avoid over distension and to maintain an inspiratory plateau pressure <30 cm H_2O.
4. Choose a RR and minute ventilation appropriate for particular clinical requirements. Optimal pH, PaO_2 and $PaCO_2$ should be targeted according to the requirement of clinical condition.
5. I:E (time): It is generally kept 1:2. A decrease in I:E ratio will prevent formation of auto PEEP.
6. Peak inflation pressure (PIP): More important than PIP is P_{plat} since it causes alveolar over distension hence a P_{plat} < 30 cm of H_2O is preferred (use of sedation helps).
7. PEEP: It distends airways down to alveoli to allow for complete exhalation and CO_2 removal, which decreases air trapping and also improves V/Q mismatch Range is 0-20 cm of H_2O and adjusted up and down in 2.5-5 cm of H_2O increments and always the lowest possible PEEP is used. Use PEEP in diffuse lung injury to maintain open alveoli at end expiration.
8. Sensitivity: It detects the amount of drop in airway pressure that is required before ventilator senses patient's effort and assist during PS and AC mode. A sensitivity around 0.5-1 cm of H_2O allows weaker patient to initiate breath while higher values makes it more difficult to initiate a breath and used for patients with high respiratory drive.
9. Call the critical care physician for assistance.

SEDATION, ANALGESIA AND NEUROMUSCULAR BLOCKADE

To improve patient comfort, anxiolytics, sedatives and analgesics should be administered. Neuromuscular blocking agents should be avoided as they are associated with complications. If at all they need to be used, they should be used with caution.

MONITORING MECHANICAL VENTILATORY SUPPORT

Observe the patient in relation to patient-ventilator interaction and measure inspiratory plateau pressure as

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required. Chest radiograph, arterial blood gases, pulse oximetry and other vital signs should be monitored after starting mechanical ventilation.

Use ventilator alarms to monitor key physiological and ventilator parameters.

COMMON ALARMS

1. Low pressure alarm:
   a. Leak in the circuit
   b. Disconnection
2. High pressure alarm:
   a. Airway obstruction, mucous plug
   b. Tube kinking
   c. Bronchospasm
   d. Increased resistance
   e. Decreased compliance.

SPECIFIC SITUATIONS

Cervical cord injury

It causes type-II respiratory failure and its presentation and management depends on the cervical level involved.[12,13]

The goal in management of mechanical ventilation of cervical spinal injury patients is to prevent respiratory complications and optimize patients for early transfer to a rehabilitation facility. To minimize atelectasis, current guidelines recommend the use of very high Tv (15 mL/kg) or setting high Tv (10 mL/kg) in addition to using positive end-expiratory pressure.[14] Low Tv (<10 ml/kg) has been associated with atelectasis, mucous plugging and less surfactant production. High Tv (>10 ml/kg) has the risk of barotrauma. High Tv patients have been shown to have less atelectasis, better dynamic compliance and earlier weaning.[15]

ARDS

ARDS is common in neurosurgical ICU especially in patients of head injury and other post-operative neurosurgical patients with associated sepsis.[16]

The ARDS network protocol provides for ventilator strategy to reduce mortality by using 2 components-permissive hypercapnia and increased PEEP. Permissive hypercapnia should be avoided in patients with TBI due to the risk of cerebral vasodilatation, increased cerebral blood flow and ICP.[17] Increased PEEP can cause an increase of ICP, but this effect is offset by the concomitant reduction in V_T and P_{plat}.

Neurogenic pulmonary edema (NPE)

It occurs in patients with acute neurologic injuries (40% of the times in head injuries and 90% in intracranial hemorrhage) due to surge of catecholamine.[18] Patients with NPE are responsive to PEEP and prone positioning.[19] It is a diagnosis of exclusion and some pointers in this direction are: Occurs rapidly, with onset at time of neurologic injury in patients with no prior history of cardiac pathology, often involves one lung field, temporary in duration and exclusively PEEP responsive.

WEANING

The society of critical care medicine advocates SBT for a patient to pass before weaning is initiated.[20] In neurosurgical patients even a low glasgow coma scale (GCS) (<4) have been extubated with no difference in outcome.[21] Besides GCS, Intact cough and gag reflex is important during weaning.

To extubate a patient following factors should be considered:

1. Duration and reversibility of primary injury
2. Reason for intubation
   a. Airway protection
      Patients who do not have respiratory failure and have recovered with an intact cough and gag reflex maybe extubated
   b. Poor respiratory drive
      These patients cannot be extubated until there is return of spontaneous drive and generally need a tracheostomy
   c. Mechanical failure due to neurologic injury
      Pump failure can occur in NPE, stunned myocardium and aspiration pneumonia and these patients cannot be extubated until there is an intact arousal and reversibility of the mechanical failure.
   d. Peripheral mechanism of respiratory failure (e.g. type-II respiratory failure)

Ventilation mode	Trigger	Limit/target	Cycling
VA (volume assist)	Patient	Insp. flow	Set T_v
VC (volume control)	Ventilator	Insp. flow	Set T_v
PA (pressure assist)	Patient	Insp. pressure	Insp. time
PC (pressure control)	Ventilator	Insp. pressure	Insp. time
PS (pressure support)	Patient	Insp. pressure	% decrease in Insp. flow

Level of injury	Presentation	Strategy
C_1-C_3	Apnea	Early tracheostomy
C_3-C_5	Mixed presentation with ability to initiate ventilation but not enough stamina to maintain it	Common to both (b) and (c) Avoid atelectasis Aggressive pulmonary Toileting Patient education and motivation to use voluntary muscles of respiration
Below C_5	Can have ventilator independence	

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These patients cannot be extubated until they have tolerance to prolonged CPAP and T-piece trial.

e. Weaning criteria used here are: Vital capacity >15-20 ml/kg, mean Inspiratory pressure < -20 to -50 cm of H_2O, $FiO_2 < 40\%$ and PEEP ≤ 5 cm of H_2O with no other medical complications.

CONCLUSION

Intubation and extubation in neurosurgical patients depends on type of injury, site of lesion, neurological status and neuromuscular strength. Ventilatory parameters should be set keeping in mind the effects on ICP, CPP and cerebral oxygenation and repeated assessment through clinical examination and arterial blood gases.

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