Mechanical Ventilation

A Review for Special Operations Medical Personnel

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ABSTRACT

Mechanical ventilation is machine-delivered flow of gases to both oxygenate and ventilate a patient who is unable to maintain physiological gas exchange, and positive-pressure ventilation (PPV) is the primary means of delivering invasive mechanical ventilation. The authors review invasive mechanical ventilation to give the Special Operations Force (SOF) medic a comprehensive conceptual understanding of a core application of critical care medicine.

KEYWORDS: *mechanical ventilation; invasive ventilation; ventilator; portable ventilator*

Introduction

> *"But that life may be restored to the animal, an opening may be attempted in the trunk of the trachea, into which a tube or reed or can should be put; you will then blow into this, so that the lung may rise again and take air."*
>
> Andrea Vesalius,
> De Humani Corporis Fabrica (1543)

Mechanical ventilation is the means through which machine-delivered flow of gases is used to both oxygenate and ventilate a patient who is unable to maintain physiological gas exchange. PPV is the primary means of delivering invasive mechanical ventilation in modern systems. A thorough understanding of the anatomy and physiology of breathing, gas dynamics, and a basic understanding of the pathophysiology of chest trauma are key to mastering the ventilator. Complicating this topic are the prehospital and often austere conditions in which military medical personnel may be forced to operate. As mechanical ventilation becomes more common in the prehospital setting, combat medical personnel would benefit from understanding fundamentals. Whether as part of medical evacuation (MEDEVAC) or during prolonged field care (PFC), the mechanical ventilator can become a force multiplier by freeing up personnel and preserving the fighting force. This review of invasive mechanical ventilation is designed to give the SOF medic a comprehensive conceptual understanding of a core application of critical care medicine.

Case Study

During routine convoy travel in eastern Afghanistan, members of a Special Forces Operational Detachment – Alpha (SF-ODA) begin receiving random and sporadic small-arms fire from a village as they pass. Before having adequate time to react to contact, an MRZR all-terrain vehicle is hit with a rocket-propelled grenade, which causes the moving vehicle to veer off the road and into a sharp ditch at approximately 30 mph. The convoy elements return fire and take cover while you, a Special Forces Medical Sergeant (18D), tend to the wounded Servicemember (SM).

Your patient is a 30-year-old 5'10", 190-lb male Special Forces Weapons Sergeant (18B) who has mild respiratory distress with a respiratory rate (RR) of 24 and a pulse oxygen saturation of 94%, with otherwise normal vitals. He was not wearing body armor beyond his helmet and hit his chest on the steering wheel during the crash. He has extensive bruising on his right anterior thorax and appears to have semidisplaced broken ribs. He is monitored pending repeat movement. Two hours later he complains of progressive dyspnea. You notice his RR is now 38 and he has a saturation of 83% despite 15 liters per minute supplemental O_2 administration. He appears to be struggling to breathe. The decision is made to intubate pending his MEDEVAC.

1. What equipment will you need to perform this task?
2. What mode and settings on the ventilator will you select and why?
3. How will you monitor the patient's condition in this austere environment?

Anatomy and Physiology of the Respiratory Control Circuit

The respiratory circuit is initiated by positive output from the central pattern generator (CPG) as influenced by central chemoreceptors in the medulla oblongata of the brainstem and peripheral chemoreceptors in the carotid body (Figure 1). Collectively, these receptors are exquisitely sensitive to changes in the partial pressure of CO_2 ($PaCO_2$), pH, and to a lesser extent, the partial pressure of O2 (PaO_2). The neurological signal intensity for the drive to breath is directly proportional to the deviation from normal (baseline) of these values.[1] Neurons from the CNS project action potentials through cranial nerves, which conjoin to form the phrenic nerve, and through the spinal cord, to innervate the diaphragm and other respiratory musculature to maintain subconscious breathing. Higher cortical stimulus can add to respiratory drive through skeletal "accessory" muscle innervation.

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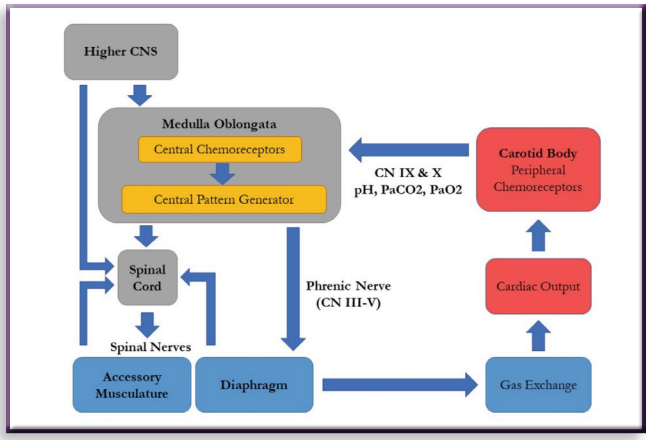
FIGURE 1 *The respiratory circuit.*



During native, nondistressed ventilation, the diaphragm contracts downward, which creates a negative pressure inside the pleural cavity which in turn pulls on the lung surface to expand. This causes air to enter through the nose and mouth, where it travels through the trachea, bronchi, and bronchioles into the alveoli. Alveoli are small air sacs at the terminus of the bronchioles and are the site of gas exchange in the lungs. They are surrounded by a thin capillary network that allows oxygen and carbon dioxide to flow across the alveolar–capillary membrane. Carbon dioxide is diffused out of the blood stream while oxygen enters where it is bound to hemoglobin molecules in red blood cells and then transported to the cells of the body.

Fluid Mechanics

Airway resistance is defined as the impedance that a gas must overcome to move from one location to another within a conduit of the airway – any location from the mouth to the most distal alveoli. Resistance within an airway is modeled on both micro and macroscopic levels by Poiseuille's law:

$$R = \frac{8\eta L}{\pi r^4}$$

With (dynamic viscosity) and established as coefficients, it is apparent that the greatest contribution to airway resistance is the radius of the airway. Starting by the trachea or endotracheal tube, as the airway continues to split into smaller bronchi and then bronchioles, airway resistance begins to dramatically decrease due to the collective surface area of all distal airways. This trend continues until the lowest point of resistance, the terminal bronchioles leading into the alveoli.

High airway resistance on a patient receiving mechanical ventilation may be due to proximal causes (e.g., kinked or plugged endotracheal tube) or distal causes (e.g., obstructed small airways due to "reactive" asthmatic obstruction or lung collapse).

Respiratory system compliance reflects the ability of the lung tissue and chest wall to expand to a given amount of pressure and can be represented by the equation:

$$C = \frac{\Delta V}{\Delta P} = \frac{TV}{(P_{plat} - PEEP)}$$

Low compliance situations can arise from within the lung itself with the acute respiratory distress syndrome (ARDS) or extrinsic to the lung via pneumothorax (PTX).

Indications for Mechanical Ventilation

The need for mechanical ventilation arises frequently in trauma patients. A failure to ventilate is the most apparent clinical form of respiratory failure due to the physical manifestations which hypercapnia effects respiratory drive. Severe tachypnea, accessory muscle use, and the ominous pattern of "belly breathing" are concerning features to consider when determining acute respiratory failure necessitating mechanical ventilation over more conservative measures.[2] A failure to oxygenate may be more subtle and can be determined when a patient is unable to maintain a SpO_2, typically falling below 90%. Significant blood gas abnormalities have also been used as an indication for mechanical ventilation. In pulmonary contusion, a (PaO_2 < 60mmHg and $PaCO_2$ > 60mmHg) despite flow-based oxygen therapy has traditionally indicated intubation and initiation of mechanical ventilation.[3] For the SOF provider, the decision to intubate must take into context both clinical and tactical situations.

The Mechanical Ventilator

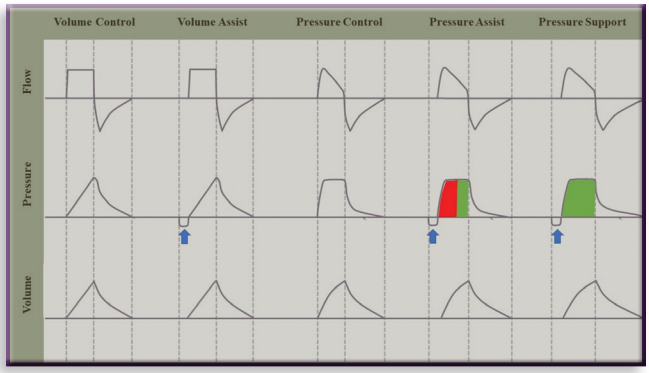
Abstractly, PPV is accomplished by a rapidly actuating gas turbine along a microprocessor-controlled servo which generates flows of a gaseous mixture. The ventilator is first initiated ("triggered") by either patient effort ("assist" or "support" breaths) or a machine-programmed time ("control" breaths). After activating, the turbine generates a *flow* which produces *pressure*, consequently leading to a *volume* of gas delivery. After a flow, pressure, or volume threshold ("target") is met, inspiration is then terminated and switched ("cycled") into the exhalation phase. Exhalation of the lungs occurs due to passive recoil to the natural and resting functional residual capacity of the lung and chest wall. Positive end-expiratory pressure (PEEP) is a fixed, minimum gas pressure can be applied in the circuit at the end of expiration when there is an absence of flow, prior to a cycle to the inspiratory phase. This process occurs in a closed-loop system and relies heavily upon positive and negative feedback loops from the machine.

The mode of the ventilator refers to the pattern by which the device delivers breaths to the patient. Based upon the convention we have now established, modes of mechanical ventilation can be categorized into five groups: volume assist, volume control, pressure assist, pressure control, and pressure support (Figure 2).[4] Most conventional and alternative modes of ventilation are simply variations these five modes, therefore it is paramount to understanding the basis.

The most common mode of mechanical ventilation is assist-control ventilation. As the name implies, breaths can be either patient-triggered (assisted), device-triggered (controlled), or a combination. Targets for assist control include pressure and volume. In volume assist-control (V-AC), a desired volume and rate are set on the ventilator and an independent pressure is then generated to achieve this volume. The patient will be free to take machine-assisted breaths when breathing at above the set rate but will be given backup-controlled breaths when breathing below the set rate (Figure 3). Similarly, in pressure assist-control (P-AC), a desired pressure support target and rate are established. The volume achieved through P-AC (or pressure support) is an independent variable to the pressure applied. Pressure support (PS) is a mode at which a pressure is applied to entirely spontaneous breathing efforts. This mode is often erroneously referred to as "CPAP." Synchronized intermittent mandatory ventilation (SIMV) is a hybrid mode of

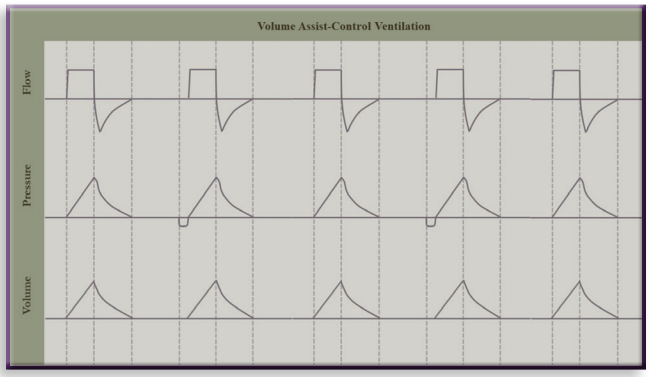
ventilation allowing a set minimum rate of volume- or pressure-controlled breaths in conjunction with the patient's efforts, with the potential for added intermittent assisted breaths to supplement the respiratory requirements of the patient.

FIGURE 2 *Categories of mechanical ventilation.*



Cycle changes from inspiratory to expiratory and vice versa are noted by dotted lines. Blue arrows demonstrate a patient trigger. Note the red area under the curve (AOC) indicating a fixed, machine-programmed inspiratory time while the green AOC reflecting inspiratory effort by the patient.

FIGURE 3 *Scalars of volume assist-control ventilation.*



Note once again the miniscule negative pressure change initiating a patient assisted breath. These triggers are most commonly assigned by a flow or pressure threshold.

Applied Pathophysiology and Trauma

The potential of mechanical ventilation was first appreciated with Alfred Woillez's first workable iron lung "spirophore" to help drowning victims in the Seine River.[5] By the 1950s, Blegdams Infectious Disease Hospital in Copenhagen pioneered tracheostomy and mechanical ventilation during the polio epidemic which led to a mortality reduction from 87% to 40%.[6] In a modern context, as many as 58% of casualties evacuated by critical care air transport from Iraq and Afghanistan received mechanical ventilation.[7]

Trauma often indicates the need for PPV by failure of any element of the respiratory circuit or its control element. Profound neurologic trauma, particularly involving the brainstem, may result in a loss of respiratory drive or the ability to maintain a patent airway. Injury to the thorax, by blunt or penetrating trauma, may result in direct cardiopulmonary injury or indirect effects such as the loss of lung compliance by a pneumothorax.

PPV may independently induce trauma to the lungs.[8] The goal of mechanical ventilation, therefore, is to allow oxygenation and ventilation without incurring ventilator induced lung injury (VILI). VILI can be grossly broken down into three categories: barotrauma, volutrauma, and atelectotrauma.[9] These pathologies collectively result in mechanical damage to the lung and release of inflammatory mediators further exacerbating lung injury. *Barotrauma* occurs with high mean airway *pressures* during ventilation. PPV at relatively high tidal *volumes* may induce *volutrauma*. Overdistension or overpressurization of an individual alveoli unit may certainly result in tissue damage, such as an overinflated balloon that ultimately pops. Lastly, *atelectotrauma* occurs by repetitive opening and closing *alveoli* units resulting in epithelial cell damage – a condition which may be prevented by the application of airway stenting PEEP.[10] Consider two wet (edematous) pieces of tissue paper sticking together and then being pulled apart repetitively and ultimately tearing. VILI may present along a spectrum of illness from as subtle as worsened gas exchange physiology to frank alveolar rupture resulting in pneumothorax, pneumomediastinum, or rarely an acute air embolism.

Blunt chest trauma represents 8% of all trauma admissions in the US.[11] Mortality is typically significantly higher in blunt chest trauma as opposed to penetrating trauma. While fractures of the ribs or axial skeleton may take place, the feared complication of blunt trauma remains pulmonary contusion. Pulmonary contusion leads to leakage of blood and proteinaceous material from increased permeability of the interstitial and alveolar space. Up to 25% of sizeable contusions (> 20% lung volume) will go on to develop ARDS.[12] The mechanical ventilation strategy for pulmonary contusion should closely follow established ARDSnet principles, outlined below.

Acute respiratory distress syndrome is an encompassing term attempting to describe a clinical disease first described in 1967 and characterized by lung tissue permeability, edema, and inflammation resulting from a known insult.[13] ARDS may develop in trauma patients due to pulmonary contusions, inhalational injury, fat emboli, and transfusion reactions.[14] The condition typically develops within 48 hours of insult. While ARDS is common and occurring in 26 to 33% of combat casualties, trauma represents just 4% of overall ARDS cases.[15–17] Intuitively, more severe trauma cases carry higher likelihood of development of ARDS, validated by Injury Severity Scores (ISS) higher than 16 being highly predictive for the development of ARDS.[18] Well-validated guidelines for mechanical ventilation of patients with ARDS have emerged since the late 1990s. These practices include the use of "lung protective" low tidal volume ventilation (LTVV) and the application of incremental PEEP.[19,20] These strategies, particularly the application of LTVV are extended to all patients receiving mechanical ventilation as a current standard of care.[21] Out-of-hospital lung-protective ventilation is associated with a decrease in mechanical ventilation days.[22] Furthermore, ARDSnet compliance is associated with improved mortality in US Air Force data.[23]

High velocity penetrating thoracic trauma causes direct pulmonary parenchymal injury from a missile effect. Secondary consequences include bursting of gas in the lung and the inertial sheering of tissue. In addition to the lung, the tracheobronchial tree, heart, great vessels, esophagus, and diaphragm may also be injured. The resulting clinical situations, including pneumothorax or tracheobronchial injury, may require adaptation of mechanical ventilation such as a minimization of PEEP to decrease the pressure gradient across damaged tissue. To the contrary, a trend towards improved mortality exists in

patients with moderate to severe ARDS utilizing the high PEEP ARDSnet strategy.[20] Adjuncts including tube thoracostomy may be required with mechanical ventilation. Overall, most thoracic injury trauma cases are managed nonoperatively.

Individualization of Ventilator Settings

Establishing initial settings for the mechanical ventilator is straightforward in most patients requiring ventilation. The plurality of patients with profound hypoxic or ventilatory defects can be effectively managed by an algorithmic approach to mechanical ventilation, adjusting to the clinical situation.

1. Establish Ventilator Mode
   - Start with Volume Assist-Control.
2. Set Tidal Volume (TV)
   - Calculate the ideal body weight (IBW) in kilograms. This is not the patient's actual body weight, despite having a fit physique.
     - IBW (male) (kg) = 50 + 2.3 (height in inches – 60)
     - IBW (female) (kg) = 45.5 + 2.3 (height in inches – 60)
   - Select a tidal volume of 7cc/kg multiplied by the IBW.
   - Ultimately, target a 6–8cc/kg IBW goal.
3. Set the Respiratory Rate (RR)
   - Start with a respiratory rate of 18 breaths per minute. Normal ventilation rates run between 10 and 22.
   - Change RR instead of TV to change patient's minute ventilation.
   - Adjust RR to achieve pH > 7.25 to 7.30 or $PaCO_2$ 35–45 or $ETCO_2$ 35–45. As tidal volume is restricted in practicing LTVV, you may require advancing the RR into the 30s in order to achieve respiratory compensation.
4. Oxygenate the Patient
   - Start FiO_2 of 100%.
   - Start PEEP at 5 for patients without significant hypoxia prior to intubation.
   - Alternatively, start PEEP at 10 for hypoxic patients.
   - Decrease FiO_2 by 10% every 2–5 minutes as to keep SpO_2 > 90%.
   - Follow the ARDSnet "high" or "low" titration curve (Table 1).
   - Considerations:
     - Low PEEP strategy may be inadequate for patients with a substantial component of atelectasis and could result in persistent hypoxia.
     - High PEEP may cause a decrease in cardiac output resulting in hypotension in hypovolemic patients.
5. Check Plateau Pressure
   - Look for a manual "inspiratory pause" or "breath hold" setting on your device.
   - Perform a pause during the inspiratory cycle lasting 0.5–1 second beyond the peak pressure. The airway pressure demonstrated during this time is the plateau pressure.
   - Decrease tidal volume by 50 cc at a time to achieve a plateau pressure below 30mmHg.

Adjunctive Equipment

A bag-valve mask (BVM), suction catheter (Yankauer or DuCanto), oral and nasal airways, and oxygen source are the minimum stockage for a provider with a mechanical ventilation capability. The ventilator may allow the medic or provider to perform other functions in a patient with respiratory failure ordinarily requiring BVM treatment. Prehospital or transport ventilators used by SOF medics and providers are traditionally smaller and less feature available than their hospital-based counterparts. Sophisticated turbine-based designs have now largely replaced pneumatic ventilator units. Most of these ventilators have an established ability to maintain adequate SpO_2 in severe acute hypoxic respiratory failure animal models and should be regarded as sufficient to treat even the ARDS patient.[24]

Continuous Monitoring Techniques

Approximately 97% of the capacity of delivered oxygen is bound to the hemoglobin unit. Therefore, pulse oximetry of the peripheral saturation of O_2 (SpO_2) is the mainstay of monitoring for patients receiving mechanical ventilation. More accurate and direct measurement of SpO_2, PaO_2, and $PaCO_2$ can be accomplished by point-of-care oximetry in a prehospital situation but are not essential. Capnography and/or end-tidal CO_2 ($EtCO_2$) can provide capability for monitoring ventilation factors and are considered the gold standard of monitoring tube placement and ventilation status.[25] Independent variables of ventilation should be monitored frequently: peak and plateau airway pressures should be monitored in volume-controlled modes, while TV should be observed when pressure is being controlled. Sedation should be examined by validated tools such as the Richmond Agitation-Sedation Scale (RASS) as undersedation may contribute to patient–ventilator asynchrony

Troubleshooting

There is no compelling evidence to demonstrate superiority of one mode of mechanical ventilation to another. It is up to the provider to determine the most appropriate mode unique to their patient. It is the author's opinion that V-AC is the most intuitive and reliable mode of mechanical ventilation, allowing for the greatest opportunity to practice lung protective ventilation. In the transport environment, volume-SIMV may be a viable option due to the decreased potential for overtriggering and breath-stacking due to vibration. Pressure support or pressure control may be a more comfortable or better tolerated mode for patients who are relatively more conscious.

Mechanical ventilation in the prehospital condition may result in a series of complications that the provider must know how to manage. Ventilator asynchrony is the failure of ventilator to meet the respiratory demands of the ventilator. Asynchronies

TABLE 1 *ARDSnet PEEP Titration Curves*

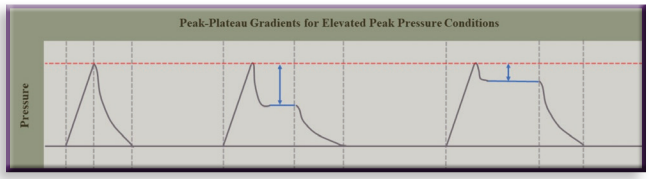
"Low PEEP" FiO_2/PEEP Titration Curve														
FiO_2	0.3	0.4	0.4	0.5	0.5	0.6	0.7	0.7	0.7	0.8	0.9	0.9	0.9	1.0
PEEP	5	5	8	8	10	10	10	12	14	14	14	16	18	18–24
"High PEEP" FiO_2/PEEP Titration Curve														
FiO_2	0.3	0.3	0.3	0.3	0.3	0.4	0.4	0.5	0.5	0.5–0.8	0.8	0.9	1.0	1.0
PEEP	5	8	10	12	14	15	16	16	18	20	22	22	22	24

are grossly categorized into the phases of the mechanical ventilation cycle: triggering, flow, and cycle dyssynchrony.[26] Careful clinical examination of the patient including auscultation takes precedence before attempting ventilator or sedation adjustment. Following this, the entire ventilator circuit should be thoroughly inspected for discontinuity or kinking. The "DOPE" mnemonic may be a helpful reminder in determining the source of potential issues: discontinuity, obstruction, patient problems, equipment failure. Increasing ventilator flow and allowing greater time in the inspiratory limb solve most asynchronies.[27]

Hypoxia may manifest because of the injury or ventilator treatment. Avoiding derecruitment of the lung is critical, particularly when high airway pressures are required to maintain oxygenation. This may involve clamping an endotracheal tube with padded forceps or hemostats when transitioning to and from devices such as a BVM or the ventilator. Short term BVM application using a PEEP valve should be regarded as the default for unexplained hypoxia. Recruitment maneuvers refer to various temporary methods to apply a sustained increase in airway pressure for the purpose of reexpanding heterogeneously atelectatic portions of lung. An example of a common recruitment maneuver protocol is the application of 40cmH_2O of airway pressure for 30 seconds.[28] Recruitment maneuvers are controversial, should not be performed routinely, nor in the absence of expert consultation.

Peak pressure alarms imply either increased airway resistance or a decreased compliance. These two conditions can be distinguished by applying the "inspiratory pause" feature of most ventilators. This causes flow to cease and pressures to equalize across the lung generating the plateau pressure. Large pressure differences from the peak to plateau, typically greater than 5cmH_2O, generally imply that the elevated peak pressure is a result of increased airway resistance. Elevated plateau pressures with a low peak-plateau gradient reflect decreased pulmonary compliance (Figure 4).

FIGURE 4 *Pressure scalars indicating peak* **(RED DOTTED LINE)** *and plateau gradients* **(BLUE ARROWS)** *for normal* **(LEFT)**, *high airway resistance* **(MIDDLE)**, *and low respiratory system compliance* **(RIGHT)** *conditions.*



Note the absence of a plateau pressure (blue line) without the inspiratory pause maneuver being performed.

Expert consultation should be sought in any derangement affecting the patient's clinical status. SOF providers should familiarize themselves with overviews on paralytics, prone positioning, and extracorporeal membrane oxygenation (ECMO) as these treatments are often vital to recovery.

Medical Evacuation and Prolonged Field Care

A critical care air transport team (CCATT) is a modern and specialized aeromedical asset of the United States Air Force. This team employs a physician with expertise in critical care or emergency medicine, a critical care nurse, and a respiratory therapist, and can transport up to three mechanically ventilated patients at one time. Airframes are equipped with portable therapeutic liquid oxygen (PTLOX) systems capable of providing oxygen for extended durations and at altitude.

At lower altitude and without the gas expansion advantages of Boyle's law, mechanical ventilators are limited in hypoxemic patients by O_2 availability. Not all mechanical ventilators utilize the same oxygen flow rates for a given fractional oxygen percentage (FdO_2). Further complicating this matter is a potential lack of electrical power required to operate a device for the extended durations of a prolonged field care situation. Tables are widely available for oxygen tank volumes (and thus run times) are various altitudes. Mechanical ventilators may be powered by ever improving commercial portable batteries.

Case Study Continued

You opt to intubate the patient and begins gathering equipment. A BVM is hooked up to an O_2 tank, Yankeur catheter with suction placed at the head, oral airway inserted, and a portable ventilator with tubing is set up next to you. The patient is successfully intubated using the rapid sequence intubation (RSI) maneuver.

V-AC is set as the mode with a TV of 510cc, RR 18, PEEP 10mmHg, and FiO_2 100% according to our algorithm. Within a minute the SpO_2 recovers to 99% and you begin downtitrating FiO_2 by decrements of 10%. An end-tidal CO_2 monitoring device is placed at the end of the endotracheal tube confirming a level of 38mmHg. The peak airway pressure is noted to be 36mmHg and you perform an inspiratory pause confirming an elevated plateau at 32mmHg. The TV is reduced by 50 cc yielding a relatively unchanged $EtCO_2$ at 40mmHg. The plateau pressure is measured once again and confirmed to now be at 28mmHg. The FiO_2 has been able to be reduced to 50% before a steady-state oxygen saturation of 91% is achieved.

You have a fully charged portable ventilator with a manufacturer's stated capacity of 10 hours of battery life, continuous power from heavy vehicles in convoy, and a portable oxygen concentrator. Additionally, you have immediate access to two D-cylinder tanks with a standard capacity of 425L of O_2 each. Given the relatively high FiO_2 requirements, you reference your O_2 consumption table, and determine you have enough O_2 to last until a relatively quick 30 min MEDEVAC ETA. The patient is evacuated without incident to a higher level of care where he is successfully treated for his wounds and makes a full recovery.

Conclusion

Prehospital mechanical ventilation brings an advanced, force-multiplying life-support capability to the battlefield. Mastery of the concepts, paired with hands on clinical experience, will allow SOF providers to undoubtedly push the envelope of care to service members in their hands. While taking an airway once meant taking at least one member of the fighting force completely out of the fight to provide imprecise, potentially harmful manual breaths, the mechanical ventilator takes their place and does their job with accuracy and precision to the benefit of both provider and patient alike.

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Disclaimer

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