

Field Intensive Care – Weaning and Extubation

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Abstract

Injury following ballistic trauma is the most prevalent indication for providing organ system support within an ICU in the field. Following damage control surgery, postoperative ventilatory support may be required, but multiple factors may influence the indications for and duration of invasive mechanical ventilation. Ballistic trauma and surgery may trigger the Systemic Inflammatory Response Syndrome (SIRS) and are important causative factors in the development of Acute Lung Injury (ALI) and Acute Respiratory Distress Syndrome (ARDS). However, their pathophysiological effect on the respiratory system is unpredictable and variable. Invasive mechanical ventilation is associated with numerous complications and the return to spontaneous ventilation has many physiological benefits. Following trauma, shorter periods of ICU sedation-amnesia and a protocol for early weaning and extubation, may minimize complications and have a beneficial effect on their psychological recovery. In the presence of stable respiratory function, appropriate analgesia and favourable operational and transfer criteria, we believe that the prompt restoration of spontaneous ventilation and early tracheal extubation should be a clinical objective for casualties within the field ICU.

Introduction

Injury following ballistic trauma is the most prevalent indication (50%) for providing organ system support to UK personnel within an intensive care unit (ICU) in the field [1]. In general the role of the field ICU is to continue the post-injury resuscitation process and provide organ system support, until the casualty is discharged to the ward or evacuated to the Role 4 facility.

Trauma casualties may require admission to an ICU for mechanical ventilation, which may be indicated for a variety of reasons, which include: impaired ventilatory effort, impaired respiratory motor function, increased mechanical load and impaired alveolar gas exchange [2]. Postoperative respiratory support is also indicated in those casualties who require surgery for further haemorrhage control or pack removal, bowel anastomosis and abdominal wall closure. Tracheal intubation is usually performed to facilitate invasive mechanical ventilation, but may also be instituted for endobronchial suctioning or maintenance of upper airway patency and protection.

Mechanical ventilation is associated with multiple complications (Table 1) and prolonged periods of ventilation and intubation should therefore be avoided. In some studies, greater than 40% of the total duration of mechanical ventilation involves the weaning process [3, 4] and adopting an early weaning and extubation policy, may minimize the risk of complications associated with mechanical ventilation.

Following mechanical ventilation, up to 20% of patients fail their first attempt at weaning [3, 4]. Whilst data on the incidence of weaning failure in a military population remains unreported, premature extubation and re-intubation should be avoided, as this is associated with an increased morbidity and mortality [5]. The purpose of this manuscript is to review

the evidence for a 'prompt weaning protocol' leading to the restoration of spontaneous ventilation and tracheal extubation, within a field ICU.

Causative Factor	Complication
Endo-tracheal intubation	Sinusitis
	Ventilator-associated pneumonia (VAP)
	Tracheal stenosis
	Vocal cord trauma
Mechanical ventilation	Formation of fistulae (tracheal-oesophageal or tracheal-vascular)
	Pneumothorax
	Oxygen toxicity
	Hypotension
	Ventilator associated lung injury (VALI)
Immobility	Venous thrombo-embolism
	Loss of skin integrity and pressure necrosis
	Muscle wasting and weakness
	Atelectasis

Table 1: Complications of mechanical ventilation.

Effect of Anaesthesia & Sedation on normal respiratory function

Anaesthesia causes an impairment of pulmonary function, irrespective of whether the patient is breathing spontaneously, or is being mechanically ventilated. Impaired oxygenation of blood occurs in most subjects who are anaesthetised [6]. Lung function remains impaired postoperatively, and clinically significant pulmonary complications can be seen in 1% to 2% after minor surgery, and in up to 20% after upper abdominal and thoracic surgery [7].

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The effects of anaesthesia and mechanical ventilation on lung function are described below. These effects are causative - in that they relate to a sequence of events the end result of which is a decrease in the lungs ability to oxygenate the blood and remove Carbon Dioxide. The first phenomenon that is seen following induction of anaesthesia is loss of muscle tone with a subsequent reduction in Functional Residual Capacity (FRC) due to a change in the balance between the outward forces (respiratory muscles) and inward forces (elastic tissue of the lung). This reduction in FRC is paralleled by an increase in the elastic behaviour of the lung (reducing compliance) and an increased airways resistance. The decreased FRC also predisposes to airway closure and the development of atelectasis - which is made worse by the application of high concentrations of inspired oxygen. These changes in lung patency alter the distribution of ventilation, and the matching of ventilation and perfusion, and impede efficient gas exchange.

Lung Volume

Resting lung volume (or FRC) is reduced by 0.8 to 1.0 L by changing body position from upright to supine, and there is another 0.4 to 0.5L decrease when anaesthesia has been induced [8]. End-expiratory lung volume is thus reduced from approximately 3.5 to 2L (this being close or equal to the Residual Volume of the lung (RV)). Anaesthesia itself causes a fall in FRC despite maintenance of spontaneous breathing [9, 10]; the average reduction corresponds to around 20% of awake FRC and may contribute to an altered distribution of ventilation. The decrease in FRC occurs regardless of whether the anaesthetic is inhaled or administered intravenously [11]. Muscle paralysis and mechanical ventilation cause no further decrease in FRC.

This decrease in FRC seems to be related to loss of respiratory muscle tone, which shifts the balance between the elastic recoil force of the lung and the outward force of the chest wall to a lower chest and lung volume.

Compliance and Resistance

Static compliance of the total respiratory system (lungs and chest wall) is reduced on average from 95 to 60 mL/cm H₂O during anaesthesia [12]. Resistance of the total respiratory system and the lungs during anaesthesia increases during both spontaneous breathing and mechanical ventilation [12].

Atelectasis

Atelectasis (lung tissue collapse) appears in approximately 90% of all patients who are anaesthetised [13]. It is seen during spontaneous breathing and after muscle paralysis and whether intravenous or inhaled anaesthetics are used [11]. Up to 15% to 20% of the lung is regularly collapsed at the base of the lung during uneventful anaesthesia as a result of anaesthesia alone. Abdominal surgery does not add much to the atelectasis, but it can remain for several days in the postoperative period [14]. After thoracic surgery and cardiopulmonary bypass, more than 50% of the lung can be collapsed even several hours after surgery is complete [15]. It is likely that it is a focus of infection and can contribute to pulmonary complications [16].

The amount of atelectasis decreases toward the apex, which is mostly spared and thus fully aerated. There is a weak correlation between the size of the atelectasis and body weight or body mass index (BMI), with obese patients showing larger atelectatic areas [11,17]. Atelectasis is independent of age, with children and young people showing as much atelectasis as elderly patients [18].

There is a good correlation between the amount of atelectasis and the subsequent pulmonary shunt.

Distribution of Ventilation and Perfusion

Redistribution of inspired gas away from dependent to nondependent lung regions has been observed in anesthetized supine humans; ventilation was shown to be distributed mainly to the upper lung regions, and there was a successive decrease down the lower half of the lung [19]. Positive end expiratory pressure (PEEP) increases dependent lung ventilation in anaesthetised subjects, so the distribution of ventilation is more similar to that in the awake state [20]. Thus, restoration of overall FRC toward or beyond the awake level returns gas distribution toward the awake pattern. This is an effect of the recruitment of collapsed, dependent lung regions (atelectasis), of reopening of closed airways in the lower lung regions, and possibly of increased expansion of the upper lung regions so that they become less compliant and less ventilated.

PEEP will impede venous return to the right heart and therefore reduce cardiac output. It may also affect pulmonary vascular resistance, although this may have less of an effect on cardiac output. In addition, PEEP causes a redistribution of blood flow toward dependent lung regions [21]. By this means, upper lung regions may be poorly perfused, thereby causing a dead space-like effect.

Respiratory Drive

Spontaneous ventilation is frequently reduced during anaesthesia. Inhaled anaesthetics [22], as well as intravenous agents [23] reduce sensitivity to CO₂. The response is dose dependent, with decreasing ventilation with deepening anaesthesia. Anaesthesia also reduces the response to hypoxia. Attenuation of the hypoxic response may be attributed to an effect on the carotid body chemoreceptors [24].

Mechanical ventilation and acute lung injury

Following major trauma, an acute lung injury (ALI) and the risk of Acute Respiratory Distress Syndrome (ARDS) is a significant clinical concern [25, 26]. The aetiology of ALI / ARDS following trauma is multifactorial and the risk factors are shown in (Table 2). Recent studies have shown that inappropriate ventilator strategies can also induce ALI [27].

Old age
Preexisting physiological impairment (diabetes, etc)
Direct pulmonary or chest wall injury
Aspiration of blood or stomach contents
Prolonged mechanical ventilation
Severe traumatic brain injury
Spinal cord injury with quadriplegia
Massive transfusion
Haemorrhagic shock
Occult hypoperfusion
Sepsis

Table 2: Risk factors for development of ALI/ARDS after trauma.

Following injury, casualties are immobilized in the supine position. This posture is responsible for the development of early dependent atelectasis, which may be further exacerbated by opioid analgesia and sedation, due to cephalad movement of the diaphragm into the thorax and compression of dependent lung parenchyma. General anaesthesia and the use of muscle relaxants may also exacerbate this problem [28]. Pulmonary contusions, if severe, can be life-threatening early in the hospital course and the disruption in pulmonary integrity due to “leaky” capillaries and lung parenchyma, may be further exacerbated by volume resuscitation with blood and crystalloid solutions. Rib fractures can cause splinting of the diaphragm leading to atelectasis and hypoxaemia.

Mechanical ventilation can initiate lung injury. High peak inspiratory pressure and low PEEP, may lead to inflammatory processes in the lung that can progress to multi-organ dysfunction. Animal studies that induce pulmonary damage to simulate ALI or ARDS cannot be distinguished from the lung damage occurring after studies that use mechanical ventilation strategies to induce injury [29]. ALI causes an alteration in lung mechanics that leads to an uneven distribution of ventilation, resulting in a disordered pattern of pressure / volume changes as the lung inflates and deflates [30]. This non-uniform ventilation results in alveolar stress which effects alveolar gas exchange. Alveolar shear forces may also develop in dependent regions. The early stages of ventilator-associated lung injury develop at commonly used airway pressures (transalveolar pressure >35 cmH₂O) in animals with normal lungs, but the threshold for lung injury may occur at lower pressure in injured lungs.

Several studies [31-33] have shown that modern ventilatory strategies using low tidal volumes (6 to 8 ml/kg), limiting alveolar distending pressures (plateau pressure <30 - 35 cmH₂O), and using optimum PEEP; may decrease mortality, reduce ICU length of stay and decrease days of mechanical ventilation. The combination of traumatic lung injury, fluid resuscitation and supine positioning, results in alveolar damage, collapse, deteriorating alveolar gas exchange and poor compliance. As ARDS develops, less of the lung is recruitable and compliance decreases, so in order to maintain oxygenation other measures, e.g. nitric oxide, nebulised prostacyclin and the prone position, may be instituted.

Newer ventilator modalities, e.g. Airway Pressure Release Ventilation (APRV), help obviate the need for deep sedation or muscle relaxation by allowing spontaneous breathing throughout the respiratory cycle. This significantly improves ventilation-perfusion matching, cardiac output, CO₂ clearance, and renal blood flow [34]. APRV has been used in trauma patients with ARDS (adopting lower peak inspiratory pressures and higher airway pressures), without derangement in alveolar gas exchange [35]. Ventilator modalities currently available in UK field ICUs include: Assist Control (A/C), Synchronised Intermittent Mandatory Ventilation (SIMV), Pressure Control Ventilation (PCV) and Pressure Support/Continuous Positive Airway Pressure (PS/CPAP). As a ventilatory mode that reduces total ventilatory requirements, APRV may represent the standard which could facilitate the optimum conditions for the prompt discontinuation of mechanical ventilation.

Blast lung is the most important primary blast injury and is the most prevalent cause of fatal injury amongst initial survivors following an explosion [36]. The blast wave propagates through lung parenchyma, as two different wave types: stress waves and shear waves. Stress waves are longitudinal pressure forces that

create a “spalling” effect at air–tissue interfaces; shear waves are transverse waves that cause asynchronous movement of tissue and possible disruption of attachments. Complex interactions of kinetic energy transfer cause tissue disruption at the capillary–alveolar interface [36]. This may result in minor or massive parenchymal haemorrhage, pulmonary oedema, pneumothorax, or air embolism. The reflection of the blast wave off mediastinal structures causes peri-hilar or “batwing” infiltrates seen on chest x-ray or computed tomography scan. The optimal management of a blast lung injury involves avoiding positive pressure ventilation, minimizing PEEP and a judicious fluid resuscitation strategy. However, limited peak inspiratory pressures (<40 cmH₂O), volume-controlled ventilation, permissive hypercapnia and other innovative ventilatory techniques may be necessary when ventilation is difficult [36]. Blast lung should be differentiated from lacerated lung early in the clinical course, as the former is treated with careful ventilation to limit further alveolar damage and systemic air embolism, whilst the latter may require surgery.

Early predictors for prolonged mechanical ventilation

When studying mechanical ventilation in trauma casualties, a confounding factor has been the presence of concomitant traumatic brain injury. The overriding determinant of outcome in this group is the severity of the traumatic brain injury (primary or secondary) and early management is divergent from casualties without brain injury [37]. In contrast, the epidemiology of trauma without brain injury is very different [38]. In these casualties, clinical data or predictors (Table 3) can be used to determine the need for prolonged mechanical ventilation (>14 days) and can be accurately predicted within 24 hours of admission to the ICU [39]. In this population, the severity of shock and its resuscitation are the overriding determinants of outcome of mechanical ventilation [40].

Once resuscitation is complete (typically within 24 hours) these patients usually remain stable and subsequent nosocomial infections (mainly ventilator associated pneumonia) serve to increase morbidity and lengthen ICU stay.

Independent Predictor

Total initial 24 hr fluids (L)
(increased risk as total initial fluid increased)

Facial Fractures

Age (increased risk as age increases)

Admission PEEP >10

PO₂/FiO₂ ratio <300 at 24hrs

Chest AIS score (the greater the score the greater the risk)

Table 3: Independent predictors of prolonged (>14 days) mechanical ventilation. (PEEP = positive end expiratory pressure; PO₂/FiO₂ = arterial partial pressure of oxygen divided by the fraction of inspired oxygen ratio; AIS score = abbreviated injury scale).

The weaning process – failure versus success

Prior to discontinuing ventilatory support, the primary aim of treatment is to correct the pathophysiological processes which created the need for mechanical ventilation. However, it is not essential to achieve complete resolution of the causative processes prior to attempting to wean and discontinue ventilation. Partial resolution of the underlying pathophysiology may be sufficient.

Studies have shown that 40% of the period undergoing mechanical ventilation may be devoted to the weaning process [41]. Current evidence supports a protocol driven approach to weaning, as extubation performed over the shortest possible period is associated with improved patient outcomes [4, 42, 43]. Extubation is 70 - 80% successful once the precipitating process has been corrected and the 20 - 30% who require re-intubation largely includes those who have been ventilated in excess of 48 hrs.

Failure to wean can be viewed as an imbalance between the capacity of the respiratory system and the load placed on that system. This imbalance may be due to: inadequate resolution of the primary problem, the occurrence of a new problem, a ventilator associated complication, or combinations of these. The predominant pathophysiological characteristic of failure to wean is high levels of load relative to the strength of the respiratory muscles, which should be optimised to increase the success of weaning (Table 4).

Haemodynamic instability	Avoiding myocardial ischaemia or new arrhythmias, which decrease cardiac function (or require high inotropic support).
Acid-Base imbalance	A normal pH is desirable but not essential. Avoid an acidaemia as this may increase the minute ventilation.
Electrolyte abnormalities	Hypophosphataemia, hypocalcaemia, hypomagnesaemia and hypokalaemia reduce muscle contractility and adversely effect weaning [25].
Intravascular volume	Avoid intravascular volume overloading, which increases the interstitial volume and decreases the functional residual capacity leading to alveolar collapse – leading to ventilation- perfusion mismatch and poor oxygenation. Mobilisation of the extra fluid usually occurs during resolution of the inflammatory processes.
Mental status	The aetiology of mental status alteration is multifactorial, but over-sedation may prolong mechanical ventilation [26]. Sedation holidays and a sedation scoring system is advised [27].
Nutrition	Malnutrition decreases muscle mass, strength and reduces immunity. Nutritional supplementation improves respiratory capacity and weaning [5, 25].

Table 4. Factors which effect weaning, by decreasing the capacity of or the demand on the respiratory system.

A protocol driven approach to weaning

Physiological parameters have been evaluated, which allow casualties who may benefit from prompt weaning and extubation to be identified early [4, 42, 43]. Unfortunately specialised measuring equipment is required which is not available in a field ICU. However, the Rapid Shallow Breathing Index (RSBI) has been shown to accurately predict weaning outcome [48]. This is the ratio of frequency (f) to tidal volume (VT) measured after 1 minute of spontaneous breathing. In general, casualties who fail to wean have low tidal volumes and a high respiratory rate. The advantage of f/VT is that it is easy to measure and is independent of patient cooperation and effort. The predictive value of RSBI is highest when measured during spontaneous ventilation through a tracheal tube.

Casualties who pass the initial RSBI assessment should proceed to a formal Spontaneous Breathing Trial (SBT). The optimal mode of ventilation for this weaning trial has not been established, but it is generally accepted that SIMV weaning prolongs the duration of mechanical ventilation. Daily T-Piece trials have consistently been shown to be superior to SIMV, but there is little evidence of an outcome difference between T-Piece based weaning and use of Pressure Support/Assist ventilation [44, 49]. An American task force has published evidence based guidelines [50] for discontinuing mechanical ventilation and the first seven of these guidelines which relate to weaning individual casualties are shown in Table 5.

Evacuation to Role 4

In a field hospital the general wards are usually staffed and equipped for basic care, observations and monitoring, which ensures that the ICU has a high bed occupancy rate. During a casualty surge, it is standard practice to review extubated casualties to determine their suitability for being moved to the ward, thus freeing beds and equipment for those who require ICU care. Adopting a prompt weaning and extubation protocol has obvious tactical and strategic implications for both current and future operations and may influence the decision process regarding whether to deploy the Critical Care Air Support Team (CCAST) or an aeromedical evacuation team.

The CCAST provides an intercontinental critical care evacuation service. The well equipped team provides expert care in a flexible working environment aboard a fixed-wing airframe that can be adapted for transferring critically ill and ventilated casualties. Whilst vigilance should minimise many of the risks associated with transferring ventilated casualties, inadvertent extubation, disconnection and migration of tubes, may still occur. Some casualties require significant doses of sedation to allow optimal mechanical ventilation, which may mask the pain associated with a compartment syndrome. As a general rule if the casualty is unstable or is at risk of requiring re-intubation during flight, extubation should not be attempted at the field ICU. As a CCAST consultant is collocated at the field hospital in Camp Bastion, the decision to extubate can be quickly confirmed. In flight, the cabin pressure is lower than at ground level; so oxygen requirements are increased and gas filled spaces expand. The cabin may be pressurized to ground or sea level, but this negatively impacts on aircraft range and flight duration.

Whilst enteral feeding is important following trauma e.g. burns, at the time of writing, the current CCAST policy is that intubated casualties are not fed during their evacuation flight to the Role 4. Until there is evidence to prove that in flight vibrations

Recommendation 1	A search for all contributing causes to ventilator dependence should be undertaken when mechanical ventilation is > 24 hours. All possible ventilatory and non-ventilatory issues should be corrected and are integral to the ventilator-discontinuation process.
Recommendation 2	Casualties receiving mechanical ventilation for respiratory failure should undergo an individualised assessment of discontinuation potential if there is: <ul style="list-style-type: none"> a. Evidence for some reversal of the underlying cause for respiratory failure. b. Adequate oxygenation (e.g. P_{aO_2}/F_{iO_2} ratio > 150 to 200, requiring positive end-expiratory pressure [PEEP] < 5 to 8 cmH₂O and F_{iO_2} < 0.4 to 0.5) and pH (e.g., pH > 7.25) c. Haemodynamic stability, as defined by the absence of active myocardial ischemia and conditions requiring no vasopressor therapy or therapy with only low-dose vasopressors. d. The capability to initiate an inspiratory effort. <p><i>Note: Those not satisfying these criteria (e.g. casualties with chronic hypoxaemia values below the thresholds cited) may still be ready to wean from mechanical ventilation.</i></p>
Recommendation 3	Formal discontinuation assessments for casualties receiving mechanical ventilation should be performed during spontaneous breathing rather than while the casualty is still receiving substantial ventilatory support. An initial brief period of spontaneous breathing can be used to assess the capability of continuing onto a formal spontaneous breathing trial (SBT). The criteria with which to assess patient tolerance during SBTs are the respiratory pattern, the adequacy of gas exchange, haemodynamic stability, and subjective comfort. The tolerance of SBTs lasting 30 to 120 minutes should prompt consideration for permanent ventilator discontinuation.
Recommendation 4	The removal of the artificial airway from a casualty who has successfully been discontinued from ventilatory support should be based on assessments of airway patency and the ability of the casualty to protect their airway.
Recommendation 5	In casualties receiving mechanical ventilation for respiratory failure who fail an SBT, the cause of the failure should be determined. After reversible causes are corrected subsequent SBTs should be performed every 24 hours.
Recommendation 6	Casualties receiving mechanical ventilation for respiratory failure, who fail an SBT, require a stable and comfortable form of ventilatory support, to lower work of breathing and to allow for rest and recovery.
Recommendation 7	Anaesthesia or sedation strategies and ventilator management aimed at early extubation should be used in postoperative patients.

Table 5. Guidelines for discontinuing mechanical ventilation [50].

do not exacerbate micro aspiration around the tracheal tube cuff, this policy is unlikely to change.

Pain management

In the polytrauma casualty, pain management issues may also influence the decision to prolong mechanical ventilation and sedation. Whilst regional anaesthesia and continuous peripheral nerve blockade has become an established method of providing analgesia for a large proportion of casualties with limb injuries [51], it may not be sufficient in every polytrauma casualty. Pain associated with thoraco-abdominal injuries can be difficult to manage and some casualties may require large supplementary doses of intravenous opioid. For this group, maintaining mechanical ventilation, analgesia and sedation until arrival at the Role 4 would be more appropriate and is an exception to rapid weaning and extubation at the field ICU.

Psychological Stress

At present we are uncertain if prolonging the duration of mechanical ventilation and sedation following traumatic injury and damage control surgery may affect the levels of post-extubation psychological stress or post traumatic stress disorder (PTSD).

In theory, pharmacologically prolonging the period of amnesia following injury, may potentially deny the casualty the opportunity to: begin to come to terms with the severity of their injuries, to be debriefed by their own unit personnel and supported by their colleagues. Clearly this theory requires further clinical investigation using, e.g. the PTSD checklist (PCL-C), which is a self-report instrument designed to assess symptoms of post traumatic stress disorder [52, 53]. Until reliable evidence is available that supports early psychological debriefing and extubation following a traumatic injury, maintaining ventilation and sedation should be determined on an individual patient basis.

Conclusion

All injured or ill service personnel should, whenever possible be offered medical care to a standard equal to that which they receive in the UK. Therefore, applying an early weaning and extubation protocol to appropriate military casualties does reflect best practice in the UK. Restoring a patent airway and spontaneous ventilation early in the postoperative period has significant clinical and logistical advantages, but conversely also requires that adequate analgesia be provided.

Maintaining mechanical ventilation and sedation during the transfer to the Role 4 should be determined on an individual

basis. Ensuring early consultation with the deployed CCAST anaesthetist should significantly reduce the risk of both reintubation being performed prior to evacuation from the field ICU, or conversely missing the opportunity to waken and extubate casualties early.

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