

Chapter 27

RECEIVING THE CRITICAL CARE PATIENT

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INTRODUCTION

This chapter provides the deploying intensivist guidance to care for patients admitted to the intensive care unit (ICU) within the first 24 hours of admission. Care in this setting must be delivered through teamwork involving surgeons, anesthesiologists, nurses, physiotherapists, and other paramedical practitioners. It is paramount that this team operates holistically to stabilize the many physiologic challenges encountered in the deployed military setting.

Casualties present with a wide variety of pathology in addition to the expected polytrauma. Trauma patterns will differ from those encountered in civilian practice. Combat injuries will consist of primarily penetrating and blast injuries, and there may also be a requirement to care for local civilians, including children. Unlike fit and healthy soldiers, civilians may have significant comorbidity.

Patients may be admitted to the ICU following surgery or prior to surgery if the patient workload

exceeds operating room (OR) capacity. These latter patients may therefore require ongoing resuscitation and stabilization. The concept of damage control (originally a US Navy term), denoting the resuscitative and surgical procedures, performed swiftly, that are required to save life and limb—as opposed to longer and more definitive surgical techniques—has been widely adopted in recent conflicts and is practiced throughout the whole care pathway from the emergency department (ED), through radiology, OR, and into the ICU. Never was the concept of an ICU “without walls” more appropriate than in the deployed military setting.

This chapter will focus on the military population. Primary topics include resolution of hemorrhagic shock and the potential for respiratory failure due to trauma. Management strategies are focused on oxygen delivery, avoidance of tissue hypoperfusion, and prevention of the systemic inflammatory response progressing to multiorgan dysfunction.

ADMISSION HISTORY AND PHYSICAL EXAMINATION

History

Critically ill patients admitted to the Role 2 and 3 deployed hospitals may arrive either by land or air transport. Initial history consists of “MIST-AT”: mechanism of injury, injuries sustained, symptoms and signs, treatment given—age (adult/child), and time of injury. In patients who are awake, an “AMPLE” (allergies, medication, pregnancy, last eaten) history may have been taken prior to intubation. Once the situation allows, further attempts should be made to gather a more detailed history, which may necessitate the use of interpreters and family members.

Examination

Depending on the patient’s consciousness level, however, further history may be limited. Irrespective of the origin of the patient (ie, Role 1 vs point of wounding), a full primary survey must be carried out. This typically takes place in the ED. However, if the injuries dictate immediate admission to the OR, bypassing the ED, then primary and secondary surveys may need to be undertaken in parallel with surgery. Occasionally, if the capacity of the ED and OR is overwhelmed, patients may be admitted directly to the ICU. It is therefore important to understand the degree to which history and examination have been undertaken and to perform repeated triaging and assessment, using the <C>ABCDE (catastrophic hemorrhage, airway,

breathing, circulation, disability, exposure) approach. Repeated assessments are particularly important because patients’ physiology will change rapidly in the initial hours postadmission.

Thus a “head-to-toe” and “back-to-front” examination is required. This examination should include review of all radiology films and laboratory data in consultation with the trauma team surgeons to ascertain all initial injuries. The initial physical examination, which serves as the baseline reference point for further therapy, should follow Battlefield Advanced Trauma Life Support guidelines of <C>ABCDE (Figure 27-1). The examination must include a basic neurologic assessment covering reflexes, motor power, and mental status, as well as an otoscopic ear examination and fundoscopic eye examination. Critically ill patients in the ICU are at risk of developing serious neurologic complications including ICU psychosis, septic encephalopathy, critical illness polyneuropathy, entrapment neuropathies, compartment syndromes, cerebral edema, intracerebral hemorrhages (related to coagulopathies), cerebral ischemia (related to hemodynamic instability), and cerebral embolism. These conditions are principally detected and diagnosed by clinical examination (computed tomography [CT] scanners are not always available). Furthermore, these conditions are frequently masked in sedated patients. If the patient does not respond to a noxious stimulus, the sedation must immediately be stopped to facilitate further neurologic evaluation. It should be policy

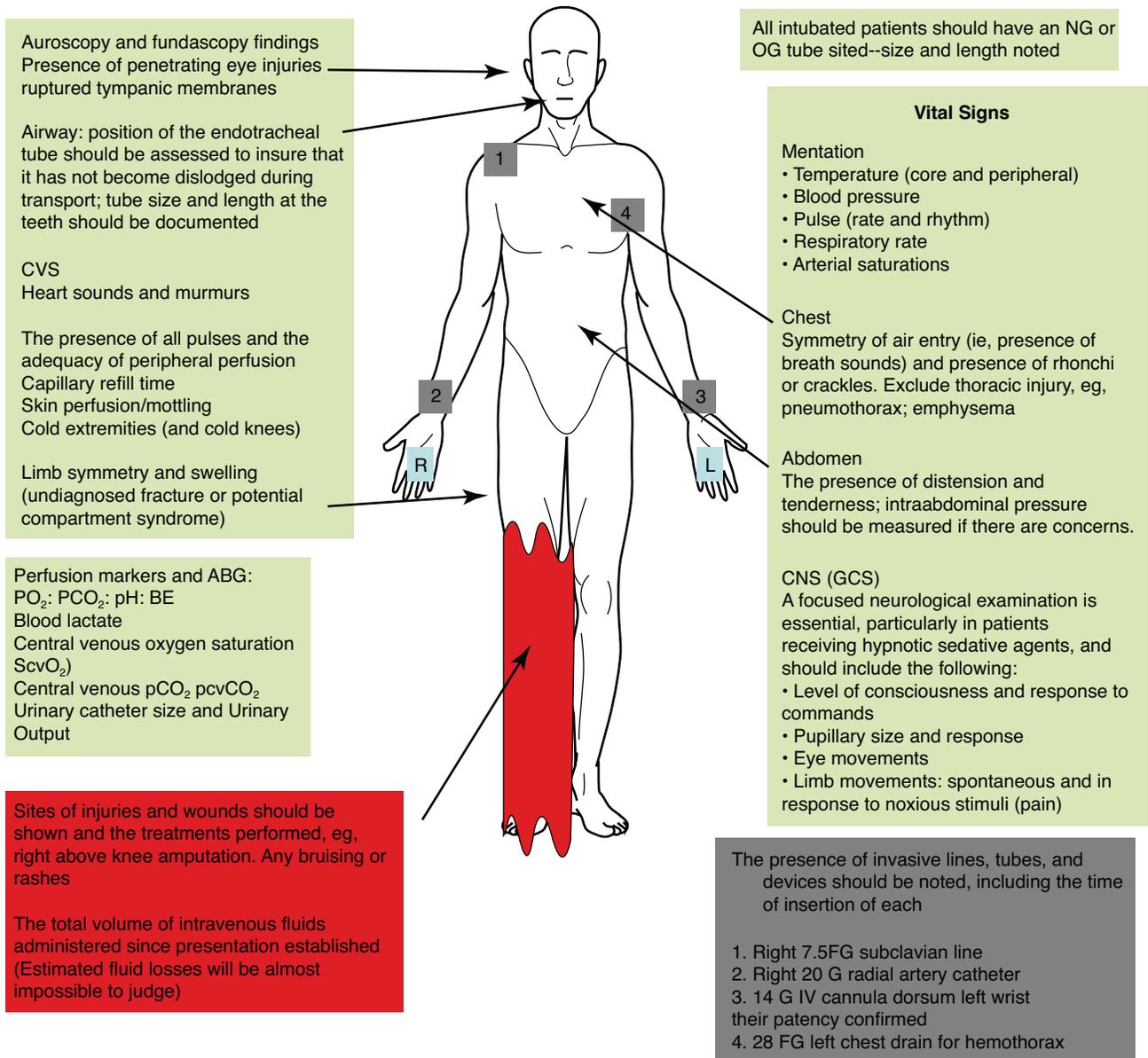


Figure 27-1. The initial physical examination and documentation, following Battlefield Advanced Trauma Life Support guidelines of <C>ABCDE (catastrophic hemorrhage, airway, breathing, circulation, disability, exposure). ABG: arterial blood gases; BE: base excess; CNS: central nervous system; CVS: cardiovascular system; FG: French gauge; G: gauge; GCS: Glasgow coma score; IV: intravenous; NGT: nasogastric tube; OGT: orogastric tube

that patients require daily awakenings to determine neurologic status and allow reevaluation of the need for sedation.

All findings must be documented. Initial and ongoing reassessment is critical to appropriate care of trauma patients. This is especially true in patients who have undergone damage control surgery or whose injuries are being managed nonoperatively. Patients who undergo damage control surgery will often have more complicated management plans and require more

resources. Nonoperative management of hemodynamically stable victims of both penetrating and blunt abdominal trauma has become more common. These approaches require a greater commitment to on-going patient reassessment and monitoring. Depending on the maturity of the operation and size of the hospital facility, access to radiological investigations such as ultrasound and CT will be invaluable in assisting the clinical team in the decision to follow a conservative treatment plan so that OR resources may be more efficiently utilized.

INVESTIGATIONS

Hematology

Hematology investigations for critically ill patients consist of a full blood count, including platelet number, coagulation screen or a thromboelastogram, urea, electrolytes, and regular blood glucose estimations. In massively transfused patients, K^+ , Ca^{2+} , and Mg^{2+} should be checked to ensure they are within normal range. In patients with substantial tissue trauma, a creatine kinase check should be requested due to the risk of rhabdomyolysis and subsequent renal failure. In these patients ensuring normovolemia and an adequate urinary output is essential. Other investigations should be guided by the injury mechanism, eg, an electrocardiogram and troponin in those patients with chest injuries. Oxygenation and oxygenation

index should be assessed in all patients (usually by pulse oximetry and blood gases when appropriate).

Imaging

A chest radiograph to ensure correct placement of lines and tubes is warranted on admission but should otherwise be performed only as clinical circumstances dictate. A CT traumagram is an invaluable tool but may not be available in all deployed environments. Where it is, close liaison with a radiologist is essential. Ultrasound is increasingly used in the ICU¹ and may rapidly provide information on hemodynamic status. Thus it is increasingly important that clinicians become familiar with the various ultrasonic techniques prior to deployment.

MANAGEMENT PLAN

Following the history and physical examination, and review of the available laboratory data and all radiology, a management plan should be formulated. This plan should involve input from all staff involved in the patient's care. Specific questions that should be addressed are:

1. What is the status of this patient's intravascular volume? Has hemodynamic stability been achieved or is continued resuscitation required? Does this patient have evidence of impaired tissue or organ perfusion?
2. Does this patient have an acute lung injury (ALI)? Does this patient require ongoing ventilation?
3. Does this patient display evidence of systemic

inflammatory response, sepsis, or multiple organ dysfunction?

4. Have all the presenting injuries been identified (more likely if the patient has been admitted directly to the ICU without a trauma scan due to logistical reasons)? Is the patient at risk of developing other life-threatening conditions associated with the presenting injury pattern that may present later in the clinical course (blast lung, compartment syndromes, and renal failure)?
5. What is the discharge plan? This will depend upon the nationality of the patient and the tactical situation. The management plan may differ depending on the length of time that the patient remains in the deployed environment.

HEMODYNAMIC CONSIDERATIONS

Adequacy of Resuscitation

Early posttraumatic mortality is determined by the initial traumatic impact and success of early resuscitation. The single goal is to prevent, detect, and treat tissue hypoperfusion. Ideally, markers of resuscitation adequacy should be obtainable without specialized equipment and easy to interpret.² Metabolic parameters show the most usefulness in assessing resuscitation from shock.³ The arterial base deficit (negative base excess), serum lactate, and central venous oxygen saturation [$ScvO_2$] are all readily available measures using standard blood-gas analyzers. A subtle marker of tissue hypoperfusion that is also readily obtainable

is the central venous to arterial CO_2 difference [$P(cv-a)CO_2$].⁴ In the presence of a $ScvO_2$ greater than 71%, a $P(cv-a)CO_2$ greater than 0.7 kPa suggests ongoing tissue hypoperfusion. Although these markers assess oxygen delivery and tissue hypoxia globally and not regionally, they provide the most consistent measures of shock severity and resuscitation response. Serum pH is a useful measure but is too easily influenced by respiratory factors and renal function to provide a reliable pure marker of tissue perfusion.

The goal of fluid replacement is best achieved by determination of fluid responsiveness. High blood to fresh frozen plasma (FFP) ratios are the norm in the early stages of resuscitation in trauma. In the later

stages of management in the ICU, the use of appropriate volumes of crystalloid solutions to avoid tissue hypoperfusion is the goal. Currently no single monitor of cardiac output has been shown to affect outcome in the trauma patient population, and therefore such monitors are not used in deployment (although this may change in the future). In the meantime, determination of fluid balance is clinically led, and care must be taken to avoid under replacement (risking acidosis and pre-renal failure) or over replacement (risking tissue edema, exacerbation of reperfusion injury, and dysfunctional leukocyte adhesion). Clinical measures include monitoring trends in hemodynamic variables and urine output in response to fluid challenges. However, these measurements have drawbacks. The endocrine response posttrauma includes fluid retention, so the intensivist should not pursue an unrealistic urinary output.

Measurements of urinary osmolality and Na^+ are simple and can be effective as a guide to the need for additional fluid. It is essential to avoid “dry-land salt-water drowning” syndrome.⁵ Excess fluid and hypervolemia may induce an increase in natriuretic hormone levels; this causes destruction of the endothelial glycocalyx layer, altering the oncotic gradient

across capillary endothelium and leading to increased loss of fluid into the interstitium. Compounded with the immobility from sedation and associated decreased lymphatic flow, the result is worsening tissue edema. Current practice, while supporting an aggressive fluid strategy in the management of shock, requires a change to a conservative restrictive fluid policy once resuscitation has been achieved. It is thus important for the intensivist to recognize this phase change.

Volume Status of the Cardiovascular System

Evidence supports the use of hemodynamic parameters for assessing fluid responsiveness, based on cardiopulmonary interaction during positive pressure ventilation.⁶ The resulting transpulmonary pressure changes cause variations in venous return, blood pressure, and stroke volume. The amplitude of this variation is inversely proportional to volume status. Thus, hypovolemia is characterized by larger swings in blood pressure and stroke volume. Clinicians have subjectively used this method for a long time by observing swings in the arterial or plethysmographic waveform (Figure 27-2). Response to simple tests such as passive leg raising⁶ (Figure 27-3)

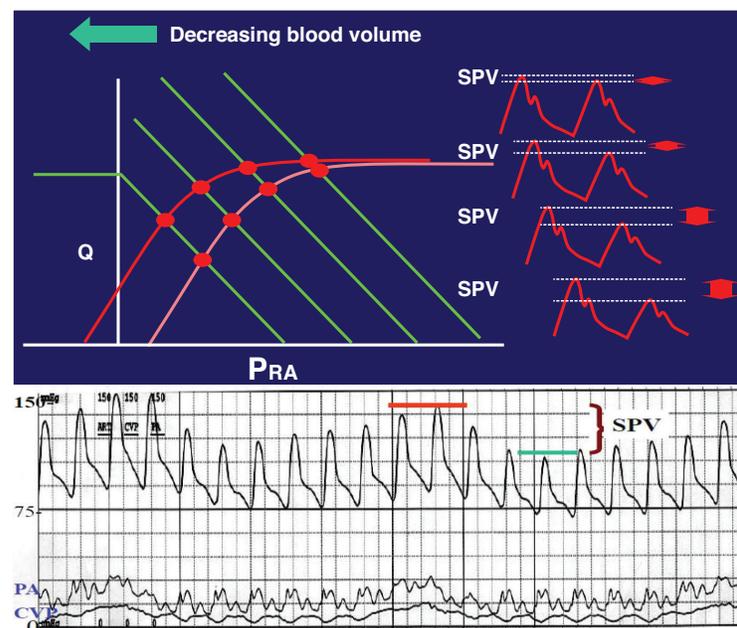


Figure 27-2. Effect of hypovolemia on systolic pressure variation (SPV). The red line indicates normovolemia; the green line indicates hypovolemia. With increasing hypovolemia there is a greater variation in systolic blood pressure between mechanical inspiration and mechanical expiration. The bottom chart shows pulmonary artery pressure (PA) and central venous pressure (CVP). $\Delta\text{SPV} > 10\%–15\% \Rightarrow$ fluid responsive.

Δ : difference; PRA: pressure of right atrium; Q: cardiac output

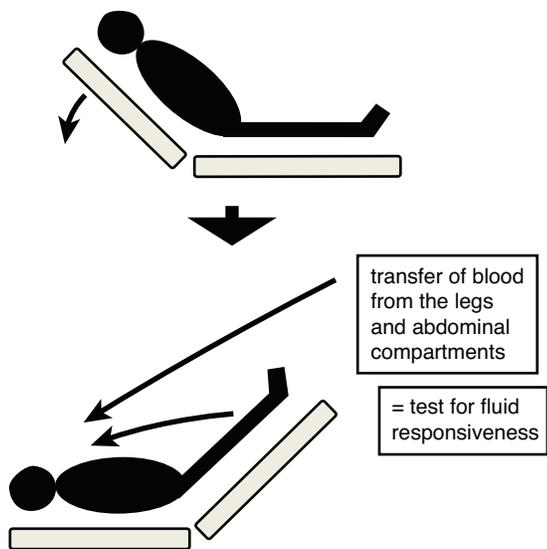


Figure 27-3. Passive leg raising test for fluid responsiveness. Approximately 150 to 200 mL in blood volume is autotransfused from the peripheral to central blood compartment. A systolic pressure variation greater than 15% implies the patient is fluid responsive.

may be useful in assessing fluid responsiveness, but this test is not possible in patients with traumatic lower limb amputations. In such patients the end-occlusion test⁷ may be employed. Systolic pressure variation provides useful information on filling status when a patient is mechanically ventilated with a tidal volume greater than 7 mL/kg and in sinus rhythm (Exhibit 27-1).

Targets for blood pressure in adults should not be set arbitrarily; rather, they should be guided by adequacy of end-organ perfusion.⁸ In the head-injured patient, in the absence of intracranial pressure, monitoring mean arterial pressure should be set at a higher level (> 80 mm Hg is recommended). Focused transthoracic echocardiography may be employed if available. Fluid replacement should be targeted to ensure that inferior vena caval diameter is over 1.5 cm and diameter variation with respiration is less than 20%.

In the first 24 hours of management in the deployed ICU, fluid should be administered as required, typically as boluses, and the response assessed. Maintenance fluids are not required and may serve only to cause overload and worsen tissue edema. A considerable volume of fluid is given in administering drugs (sedation, analgesia, antibiotics), and for every unit of blood and FFP, 150 mL of crystalloid is administered. Blood product replacement should be guided by the presence of coagulopathy.

The stress response to the injury or surgery causes

antidiuresis and oliguria, mediated by vasopressin (antidiuretic hormone), catecholamines, and the rennin-angiotensin-aldosterone system (RAAS). Water and salt are therefore retained in the extracellular space even in the presence of overload. The excretion of excess sodium takes days and appears to be dependent on the passive and permissive suppression of the RAAS rather than any positive action of atrial natriuretic peptide. Salt and water retention is exacerbated by resuscitation with saline-rich fluids and results in peripheral and visceral edema. Following surgery, even when the serum osmolarity is reduced by administration of hypotonic fluid, the ability to excrete free water is limited because the capacity of the kidney to dilute, as well as to concentrate, the urine is impaired. Hyperchloremia from chloride ion retention causes renal vasoconstriction and reduced glomerular filtration rate, further compromising the ability of the kidney to excrete sodium and water.⁹ In more seriously ill surgical patients who are catabolic with increased urea production, sodium and chloride excretion competes with excretion of nitrogen; again, much of administered sodium, chloride, and water is retained as interstitial edema. This is exacerbated by endogenous water production from fat and protein breakdown, which is increased (1 kg of either will yield approx 1 L water). Sodium reabsorption is linked to increased H⁺ and K⁺ excretion caused by RAAS activation and metabolic acidemia related to

EXHIBIT 27-1

MARKERS OF ADEQUATE RESUSCITATION

- Stable hemodynamics without the need for vasoactive or inotropic support
- Serum lactate ≤ 2 mmol/L
- Normal renal function (urinary output > 1 mL/kg/h)
- Core to periphery temperature gradient < 3°C
- P(cv-a)CO₂ < 0.7 kpa (appears to be a useful tool to identify persistent hypoperfusion when goal-directed therapy is associated with a ScvO₂ ≥ 71%)
- Normal coagulation (Hb > 100 g/L)
- Normothermia
- No hypoxemia or hypercapnia (not applicable when lung-protective ventilation is in use)

hypoperfusion and tissue trauma. Potassium depletion, due both to RAAS activity and the cellular loss of potassium that accompanies protein catabolism, further reduces the ability to excrete a sodium load. A sustained increase in systemic capillary permeability (as occurs during severe inflammatory responses of trauma and sepsis) allows albumin and its attendant fluid (18 mL for every gram of albumin) to leak into the interstitial space, also worsening interstitial edema. This increase in capillary permeability results in intravascular hypovolemia with further sodium and water

retention by continuing activation of the RAAS and secretion of vasopressin. This “relative” hypovolemia is often associated with hypotension and is worsened by further fluid administration. Vasoactive drug support is required.¹⁰

Fluid balance and urine output are vitally important in the daily and ongoing evaluation of the ICU patient. Even more important is the cumulative fluid balance. Major efforts should be expended in ensuring a neutral or slightly negative fluid balance, especially once the patient is out of the resuscitation phase.

ACUTE LUNG INJURY

Acute Lung Injury, Blast Lung, and Pulmonary Contusion

Many patients develop an ALI following massive transfusion (26%), multiple trauma (16%), and pulmonary contusion (50%). ALI is thought to be the uniform expression of a diffuse and overwhelming inflammatory reaction of the pulmonary parenchyma to either a direct injury to the lung or an indirect lung injury (eg, sepsis). Blast lung (and pulmonary contusion) is a direct injury with initial diffuse bleeding within the lung (causing hypoxemia), which then progresses to an inflammatory state within the lung. ALI has the potential to progress to acute respiratory distress syndrome (ARDS).

Blast lung rarely presents alone; it is often associated with other blast-related traumatic injuries. In current conflicts blast lung results primarily from improvised explosive devices (IED). Any thoracic abnormalities on plain radiographs or CT scan should heighten the clinical suspicion of blast lung if associated with a history of blast exposure. Examples include focal or diffuse opacifications that are clearly not penetrating fragments, pneumothoraces, pneumopericardium, or pneumomediastinum. Clinical symptoms of blast lung include shortness of breath, hemoptysis, and cough associated with hypoxemia (although this may be delayed). Other injuries tend to obscure the lung injury, which may not manifest for several hours.

Management of blast lung is exactly the same as for any patient with ALI/ARDS. General management considerations for all patients include ventilation, sedation, nutrition, and infection control. For patients at high risk of ALI, smaller tidal volumes are more appropriate provided atelectasis and excessive acidemia (due to hypercapnia) are avoided or minimized. The patient with damaged lungs is at an increased risk of morbidity and mortality due to fluid mismanagement and misapplied ventilation.

Ventilation Strategies

Patients with traumatic limb amputation will invariably have received a massive transfusion. They require a lung-protective ventilatory strategy both in the OR and the ICU. It is essential that these fragile lungs do not suffer further (iatrogenic) damage. However, this strategy is not without drawbacks (Exhibit 27-2).

The goal of ventilation for patients with ALI or ARDS is to oxygenate the vital organs but minimize further lung damage (caused by stretching or shearing forces induced by the ventilator) by using a lung-protective ventilatory strategy. Below are suggestions for ventilation; however, ventilator settings must be guided on an individual patient basis.

EXHIBIT 27-2

PROS AND CONS OF LOW-VOLUME* VENTILATION IN PATIENTS AT RISK OF ACUTE LUNG INJURY

Pros

- Proven mortality benefit in those with ARDS
- Decreased cytokine production
- High V_T associated with ALI/ARDS

Cons

- Hypercapnia may cause raised ICP, myocardial depression, and pulmonary hypertension
- Atelectasis if inadequate PEEP is used
- Patients may require more sedation and use of muscle relaxants

* $V_T < 6$ mL/kg

ALI: acute lung injury; ARDS: acute respiratory distress syndrome; ICP: intracranial pressure; PEEP: positive end-expiratory pressure; V_T : tidal volume

- Plateau airway pressure < 30 cm H₂O, and set positive end-expiratory pressure (PEEP) at a level to maintain an SpO₂ > 90%.
- Tidal volume no more than 6 mL/kg (lean body weight); lower if possible.
- Permissive hypoxemia may represent a reasonable strategy in the presence of severe lung injury, but minimizing oxygen consumption rate will be required. Target PaO₂: 7 kPa (pO₂ = 50 mm Hg).
- Pursue a strategy of permissive hypercarbia. A target pH taking into account any metabolic acidemia of as low as 7.1 may be acceptable. To counter a low pH the respiratory rate should be altered (up to 30 breaths/minute) in preference to increases in tidal volume.
- Advanced ventilatory techniques such as airway pressure release ventilation may be appropriate to enable alveolar recruitment maneuvers, but may not be available.
- Aim for an FiO₂ < 0.5 or the lowest FiO₂ consistent with an acceptable blood oxygen level.
- Prone positioning may be beneficial if the appropriate use of FiO₂, PEEP, recruitment maneuvers, and neuromuscular blockade has failed to achieve adequate oxygenation.
- Endobronchial toilet in the presence of pulmonary hemorrhage may be required if clot obstruction is contributing to hypoxemia. Segmental obstruction on chest radiograph or CT should prompt careful consideration of the potential risks and benefits of performing endobronchial suction/bronchoscopy.
- United Kingdom Clinical Guidelines for Operations recommends considering recombinant factor VIIa (rFVIIa) in pulmonary hemorrhage related to blast lung.¹¹

Sedation

Currently combination treatment with propofol and fentanyl (or remifentanyl) is the mainstay of sedation. Benzodiazepines should be used with caution (because of the high incidence of delirium) and barbiturates reserved for treatment of intractable seizures. Sedation should be titrated to the Richmond Agitation-Sedation Scale (RASS) score, unless there are indications for deeply sedating the patient, eg, severe closed head injury with evidence of raised intracranial pressure. The RASS has been shown to be a reproducible and reliable instrument with high inter-rater reliability. Sedation breaks should be considered. The daily interruption of sedative administration allows accumulated sedative agent to

dissipate, permits the patient to recover consciousness for assessment purposes, and facilitates recovery from the sedated state. There is a lower incidence of post-traumatic stress disorder in the civilian population with this approach.

Reduction in sedation and a spontaneous breathing trial should be performed as early as possible. The question of awakening patients who are to be transferred may arise. This decision must be discussed with the aeromedical team. If the tactical tempo and patient pathology permit, it may be preferable to extubate patients prior to aeromedical evacuation, provided adequate analgesia is established. Transferring ventilated patients to a Role 4 hospital carries risks that can be minimized if the patient is awake and stable; however, these patients must be truly stable with no risk of deterioration in-flight.

Nutrition

The maturity of the military operation will dictate the sophistication of caloric replacement therapies available. In general, the mainstay of treatment is to start enteral nutrition once resuscitation has been completed. Close liaison with the surgical team will facilitate the placement of feeding tubes at initial surgery and the subsequent timing of nutrition initiation. Parenteral feeding remains controversial (even in the civilian setting) and is outside the parameters of the first 24 hours of care.

Although most service personnel are pre-morbidly physically fit, adequate, balanced nutrition in austere environments can present challenges. Additionally, the nutritional state of any local nationals presenting to the field hospital may vary widely. Following injury, polytrauma patients are catabolic, and providing sufficient protein, fat, and carbohydrate often remains a problem into Role 4. For this reason, if feeding can be commenced in the first 24 hours, it should be.

Infection Prevention and Control

In the military setting, traumatic wounds are often penetrating and heavily contaminated. Dirt and debris must be subject to extensive debridement as soon as possible. Coverage with empirical antimicrobials should be provided as early as possible, following locally generated clinical guidelines for operations. Within the ICU setting, signs of sepsis should be treated aggressively in a multidisciplinary manner; further debridement is often more successful than stronger antibiotics. If the tactical tempo allows, changing invasive lines placed before the ICU admission should be the rule.

MANAGEMENT PROBLEMS FOLLOWING DAMAGE CONTROL SURGERY

The goals of therapy in the ICU after damage control surgery and hemorrhagic shock are well-defined:

1. correct metabolic acidosis;
2. restore normothermia;
3. reverse coagulopathy; and
4. ensure adequate oxygen delivery and consumption.

Correction of the physiologic dysfunction is necessary prior to returning to the OR. The components of the “lethal triad” (items 1–3) act synergistically. Of the three, coagulopathy is most affected by the presence and severity of the other two. Preventing or attenuating the severity of coagulopathy depends on limiting ongoing tissue injuries and hypoxia, and preemptively transfusing clotting factors and platelets. In injured patients arriving from the OR following damage control resuscitation, disturbances in coagulation may be observed at a temperature

of 35°C. The contributing factors of coagulopathy should therefore be aggressively addressed, in an effort to minimize their additive effects, until normothermia is restored. To treat any hypothermia, all intravenous fluids (especially blood products) should be warmed and forced-air warming blankets at 42°C for the patient should be used. Additionally, the efficiency and overall activity of most clotting factors are substantially reduced in an acidic environment (pH < 7.40). In patients with ongoing hemorrhage, resuscitation with blood, FFP, and platelets, as well as tranexamic acid, Ca²⁺, and rFVIIa, may be required. There is no place for colloid or crystalloid use. Similarly, there is no place for hypertonic saline except in the management of raised intracranial pressure. Use of thromboelastogram (ROTEM [TEM International GmbH, Munich, Germany]) and blood counts will assist in treatment guidance. Repeated clinical and lab assessment, especially with respect to volemic status, is essential.

PATIENT DISCHARGE

The maturity and tempo of the operation will dictate the options for patient discharge. United Kingdom, US, and coalition patients whose level of care cannot be swiftly reduced will need to be returned to their host nation via a critical care team. This is covered in more detail elsewhere (see Chapter 38, Air Transport of the Critical

Care Patient). Patients who can be transferred to the ward will need detailed documentation of their present and future care and appropriate handover to ward staff. Wherever possible, the ICU staff can continue providing outreach care and support to the wards, especially when epidural analgesic techniques are employed.

SUMMARY

Military traumatic injuries have multiple presentations; however, their ICU management is relatively straightforward and is directed toward reversing the

lethal triad. The importance of serial reevaluation cannot be overemphasized. Teamwork among all providers is also essential.

REFERENCES

1. Sarti A, Lorini FL, eds. *Echocardiography for Intensivists*. Milan, Italy: Springer-Verlag; 2012.
2. Blow O, Magliore L, Claridge JA, Butler K, Young JS. The golden hour and the silver day: detection and correction of occult hypoperfusion within 24 hours improves outcomes from major trauma. *J Trauma*. 1999;47:964–969.
3. Vallet B, Futier E, Robin E. Tissue oxygenation parameters to guide fluid therapy. *Transfusion Alternatives Transfusion Med*. 2010;11(3):113–117.
4. van Beest PA, Lont MC, Holman ND, Loef B, Kuiper MA, Boerma EC. Central venous-arterial pCO₂ difference as a tool in resuscitation of septic patients. *Intensive Care Med*. 2013;39:1034–1039.
5. Gosling P. Salt of the earth or a drop in the ocean? A pathophysiological approach to fluid resuscitation. *Emerg Med J*. 2003;20:306–315.
6. Marik PE. Hemodynamic parameters to guide fluid therapy. *Transfusion Alternatives Transfusion Med*. 2010;11(3):102–112.

7. Monnet X, Osman D, Ridet C, Lamia B, Richard C, Teboul JL. Predicting volume responsiveness by using the end-expiratory occlusion in mechanically ventilated intensive care unit patients. *Crit Care Med.* 2009;37:951–956.
8. Chowdhury AH, Cox EF, Francis ST, et al. A randomized, controlled, double-blind crossover study on the effects of 2-liter infusions 0.9% saline and Plasma-Lyte® on renal blood flow velocity and renal cortical tissue perfusion in healthy volunteers. *Ann Surg.* 2012;256:18-24.
9. Morrison CA, Carrick MM, Norman MA, et al. Hypotensive resuscitation strategy reduces transfusion requirements and severe postoperative coagulopathy in trauma patients with hemorrhagic shock: preliminary results of a randomized controlled trial. *J Trauma.* 2011;70:652–663.
10. Voelckel WG, Convertino VA, Lurie KG, et al. Vasopressin for hemorrhagic shock management: revisiting the potential value in civilian and combat casualty care. *J Trauma.* 2010;69(1 Suppl):S69–74.
11. UK Ministry of Defence. Clinical Guidelines for Operations website. <https://www.gov.uk/government/publications/jsp-999-clinical-guidelines-for-operations>. Accessed March 7, 2014.