Chapter 9

PERIOPERATIVE AND INTER-OPERATIVE CRITICAL CARE

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INTRODUCTION

Decision-making and treatment for physiological correction following major combat-related injury can have effects beyond the initial surgical and anesthetic interventions. It is vital that the entire clinical team (including the intensive care physician) is involved as early as possible in the treatment pathway. The treatment of these patients can be protracted (commonly referred to as the prolonged-care phase), and the initial injury may become of secondary importance to the effects of systemic inflammatory response syndrome (SIRS), acute lung injury (ALI), nosocomial infection, and intercurrent multiorgan dysfunction syndrome (MODS). Patients with multiple injuries often require lengthy periods of mechanical ventilation. The surgical approach to the most injured patients has changed in recent years with the adoption of damage control surgery and resuscitation.[1,2] The triad of hypothermia, acidosis, and coagulopathy, as well as complications such as abdominal compartment syndrome, require surveillance and management prior to and after admission to the intensive care unit. Recent research and clinical work have clearly shown that the early phase of trauma is characterized by a marked inflammatory response. Modifying this response in the initial treatment and early intensive care phase has the potential to mitigate its progression and impact on end organs.[3,4]

Trauma patients requiring intensive care interventions generally fall into two groups: (1) those immediately requiring organ support upon admission to the hospital, and (2) those experiencing the later complications of trauma, such as SIRS, sepsis, ALI, or MODS. The first group includes patients with thoracic injuries, major head injuries, or circulatory shock, as well as those who require an extended recovery period following resuscitative surgery. For service personnel, extended care and rehabilitation are usually delivered back in their home nations. Local civilians may pose particular challenges because their medical requirements may exceed the capacity of facilities within the theater of operations to manage their care in the medium to long term (see Chapter 42, Ethical Challenges of Deployed Military Critical Care).[5–7]

INITIAL MANAGEMENT

Treatment of trauma patients begins with the standard <C>ABC (catastrophic hemorrhage, airway, breathing, circulation) assessment by the trauma team. Intensive care specialists should be represented within the trauma team; this is often in the role of an anesthesiologist, whose responsibility goes beyond assessment of the airway (Exhibits 9-1 and 9-2, and Figure 9-1). Knowledge of the injury mechanism allows potential injuries to be identified or ruled out; specifically, the team must know whether the injury was blunt or penetrating and if it involved blast and penetrating fragments. The team should be aware that although most cases of shock in trauma will be hypovolemic, shock may have other causes (eg, cardiogenic from a myocardial injury, sepsis if the presentation is delayed, tension pneumothorax); the injury mechanism can assist with this diagnosis.

The concept of damage control resuscitation is now well established and over the last decade has become the accepted method of managing unstable trauma patients. Damage control surgery is a component of damage control resuscitation, and resuscitation occurs both before and after the surgical intervention. Major hemorrhage from amputated limbs or internal hemorrhage requires immediate surgical intervention to gain proximal vascular control. Hemorrhaging patients require endotracheal intubation concurrently with insertion of large-bore venous access and hemostatic resuscitation. The patient may be resuscitated in the emergency department first or taken immediately to the operating room.[8]

IDEAL ASSESSMENT REGIME

History

Attempted verbal communication gives a good indication of whether the patient has a patent airway (is able to speak clearly), has sufficient respiratory drive (is able to speak in full sentences), has sufficient blood pressure to provide adequate cerebral perfusion, and can facilitate determination of the Glasgow coma score and level of pain. Asking the patient to cough gives valuable quick information about respiratory capacity. An AMPLE history (Exhibit 9-3) can be taken in less than 30 seconds.

Airway

Airway can be assessed within the first few seconds. A patent airway without requirement for airway adjuncts or jaw thrust, in a conscious patient, can be considered to be stable. A rapid sequence induction of anesthesia (RSI) should be considered urgently in

EXHIBIT 9-1

IMMEDIATE REQUIREMENTS AND DECISION POINTS IN TREATING THE SEVERELY INJURED PATIENT

1. Consider the injury mechanisms and potential structures at risk in blunt and penetrating trauma.
2. Initiate damage control resuscitation and massive transfusion protocol.
3. Administer appropriate and timely airway and ventilatory interventions.
4. Accurately assess volume status, particularly in patients in compensated shock.
5. Achieve appropriate endpoints of preoperative and intraoperative resuscitation.
6. Use advanced monitoring to aid resuscitation.
7. Assess requirement for intensive care and communicate with nurse in charge.

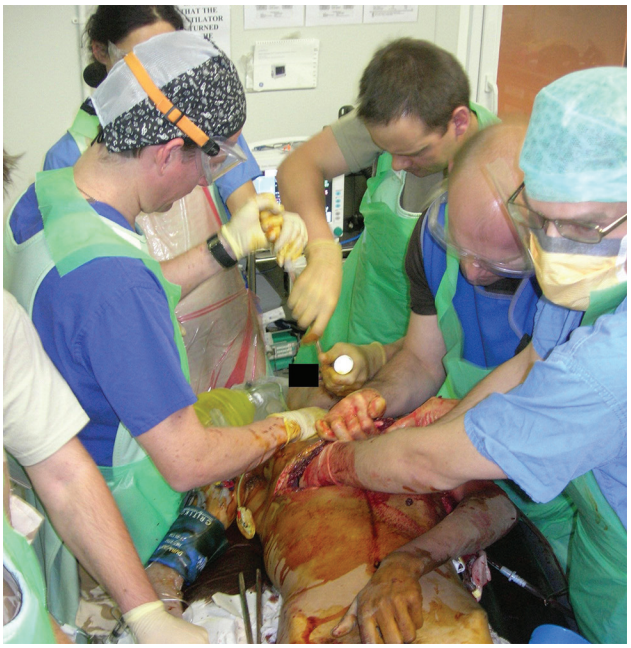

Figure 9-1. The trauma team in action: simultaneous resuscitation and damage control surgery.

patients with conditions listed in Exhibit 9-4. The last two indications listed are subject to debate because of concerns about physiological decompensation. RSI requires an appropriate patient assessment and appropriate drug dosages for the patient's condition, as described below. There is usually no good reason to excessively delay the provision of anesthesia and airway support to severely injured patients. Most trauma patients will have a hard collar in place for C-spine protection. This collar should be removed for intubation, with manual in-line stabilization maintained throughout the RSI. The need for C-spine control implies that all trauma intubations should be considered more challenging than elective intubations, and a bougie should be routinely used. The importance of trained assistants in trauma airway management cannot be overstated.

Although no induction agent is ideal, most can be used appropriately if titrated according to the hemodynamic status of the patient. Most anesthetic agents are direct vasodilators and negative inotropes. Even

EXHIBIT 9-2

EARLY INTENSIVE CARE REQUIREMENTS FOR THE SEVERELY INJURED PATIENT

1. Ensure safe transfer to the CT scanner, the operating theater, or the intensive care unit.
2. Clear the cervical spine in a sedated and ventilated trauma patient.
3. Determine timing of fracture fixation and further surgery.
4. Achieve medium and longer-term resuscitation endpoints.
5. Follow aseptic strategies for mitigation of central venous catheter bloodstream infections.
6. Instigate enteral nutrition.
7. Perform an early rehabilitation assessment.

CT: computed tomography

EXHIBIT 9-3

THE "AMPLE" HISTORY

Allergies

Medications

Past medical history

Last food or drink

Everything else: history of incident including mechanism, time, etc

EXHIBIT 9-4

CONDITIONS REQUIRING RAPID SEQUENCE INDUCTION OF ANESTHESIA

1. Actual or impending loss of airway.
2. Reduced consciousness level; patient is unable to maintain own airway.
3. Head injury, especially if patient is combative or has a low GCS.
4. Injuries or decreased level of consciousness causing ventilatory failure.
5. Likelihood of immediate surgical intervention being required on arrival at surgical facility due to injury pattern.
6. Humanitarian reasons (distress and pain relief).

GCS: Glasgow coma score

agents, recommended for their cardiovascular stability, such as etomidate and ketamine, have indirect negative effects mediated through reduction of circulating catecholamine levels in patients who are in pain or otherwise physiologically stressed.[9] Induction doses should be reduced by up to 50% to 90% in hemodynamically compromised patients, and maintenance doses should be started at low levels and titrated upward only as tolerated. An induction with propofol reduced to as little as one tenth of normal induction doses has been shown to produce equivalent reductions in cerebral electrical activity when administered to an animal in shock, even after adjustment for a reduced blood circulating volume.[10]

Although hemorrhage control should not be delayed to pursue resuscitation, bolus administration of fluid at the time of anesthetic induction may help to prevent catastrophic vascular collapse. In the hemorrhaging patient, this bolus therapy should be carefully monitored and balanced against the administration of anesthetics to preserve a state of controlled hypotension.[11] Practice varies depending on the provider's training and background.

Clearance of the Cervical Spine

Cervical spine injury occurs in 5% to 10% of blunt polytrauma patients. Despite several published clinical guidelines, treatment remains controversial. The application of these guidelines to the obtunded trauma patient is limited. The presence of a severe head injury increases the relative risk of a cervical spine injury by as much as 8.5 times, and the risk of focal neurological deficit by 58 times.[12] When the patient is unlikely to be fully evaluated within 24 hours, complications associated with prolonged immobilization shifts the risk–benefit analysis: rather than waiting for an opportunity to do a full clinical evaluation, providers should opt for a nonclinical clearance, given that the vast majority (90% –95%) will not have a cervical injury. A protocol using computed tomography (CT) scanning for blunt trauma patients who were obtunded has shown the risk of missing a cervical spine injury to be 0.04%.[13] In any of the following circumstances casualties should undergo CT imaging of the cervical spine as the primary imaging modality:

- Glasgow coma score below 13 on initial assessment;
- the patient is intubated;
- plain film series is technically inadequate (for example, desired view unavailable), suspicious, or definitely abnormal;
- continued clinical suspicion of injury despite a normal x-ray; or
- the patient is being scanned for multiregion trauma.

Many combat trauma patients have one or more of these risk factors after complex blast injuries, so the early involvement of a radiologist is essential.[12–14]

Ventilation

Pulmonary dysfunction in trauma patients is multifactorial and may result from direct contusion of the lung tissue, lung injury by fractured ribs, loss of chest wall function, fat embolism to the lung from long bone fractures, aspiration of blood or gastric contents, the activation of SIRS, shock, reperfusion, or transfusion therapy.[15,16] Ventilatory failure can have a profound effect on physiology.

The lungs are especially susceptible to injury from blast shock waves because of the contrast between tissue density and the gas inside. A specific syndrome of lung damage can result from blast shock waves: "blast lung" is a progressive condition characterized by the development of pulmonary inflammation and edema following initial intrapulmonary hemorrhage. These conditions can cause a decrease in pulmonary gas transfer, initially leading to hypoxia and hypercarbia. The resulting lung damage is exacerbated by the other mechanisms of traumatic lung injury described.[17–19]

Pneumothoraces are frequent in trauma patients and should be actively excluded in the initial examination. They may expand to produce tension physiology:

impaired ventilation due to decreased air entry and hypotension from vena caval compression. Mediastinal shift is common in the absence of tension physiology and cannot guarantee the diagnosis of tension pneumothorax.[20] Small pneumothoraces (visible on chest CT but not chest radiograph) may not require treatment.[21] However, pneumothoraces behave very differently in spontaneously versus mechanically ventilated patients. Rapid development of tension physiology is much more likely in the ventilated patient, so vigilance should be maintained. If in doubt, immediate decompression of the thoracic cavity either with a simple thoracostomy or chest drain is required.

It is important to reduce the additional iatrogenic effects of mechanical ventilation, which can cause barotrauma, volutrauma, and atelecttrauma[22–24] and potentiates circulating inflammatory markers (biotrauma).[25] Physiological tidal volumes and limiting the inspiratory plateau have been widely accepted in patients with ALI.[26,27] Because all trauma patients are at risk of developing ALI, strategies to protect the lung should be applied immediately following intubation, aiming for tidal volumes of 6 to 8 mL/kg and plateau airway pressures below 30 cm H_2O. This reduction in alveolar volume may result in alveolar derecruitment if insufficient positive end-expiratory pressure (PEEP) is applied to prevent alveolar collapse. The use of high PEEP to compensate for this de-recruitment may be associated with excessive lung parenchyma stress and strain, which may have the most impact on a severely injured lung after traumatic injury. However, stepwise titration of PEEP to higher levels has been shown to have positive effects in trauma patients.[28,29] A minimum PEEP of 5 cm H_2O should be applied and adjusted upward to ensure adequate oxygenation, with awareness that increasing the PEEP may precipitate hypotension in hypovolemic patients. In patients with head injuries, there may be a need to control P_{CO_2}, causing potential conflicts with the ventilation strategy employed to protect the lungs.[30–33]

In most cases conventional lung-protective ventilation has provided adequate respiratory support of blast casualties. A small number of these patients have required high frequency oscillation ventilation, but only after the first 24 hours of management. This type of ventilation is currently unavailable in the operational theater. [34]

Pump-less interventional lung assist (iLA) can be used in patients with acute respiratory distress syndrome (ARDS) to improve extracorporeal gas exchange by means of a membrane integrated into a passive arteriovenous shunt. Effective carbon dioxide removal through iLA has been demonstrated in early studies, but only a moderate improvement in oxygenation, with no survival benefit, in life-threatening hypoxemia and hypercapnia was shown.[35] More recently iLA has been used for extracorporeal carbon dioxide removal to enable lung-protective ventilation in patients with ARDS. In life-threatening gas exchange limitation, iLA has been used to facilitate lung-protective ventilation by enabling low tidal volume and reduced inspiratory plateau pressure.[36] Insertion of the arteriovenous iLA is not without risk, including critical limb ischemia, and high bilateral amputees may have difficult groin access. In the deployed intensive care setting it may be appropriate to consider iLA early to aid lung-protective ventilation and transfer to Role 4 in some multiply injured patients. The procedure has been used successfully in US soldiers being transferred from Iraq to higher echelons of medical care.[37]

Circulation

The decompensated, bleeding patient requiring massive transfusion should be immediately resuscitated as described in the massive transfusion chapter. The subsequent management of these patients becomes more challenging. End points in resuscitation (below) should be used to guide continuing volume resuscitation and thromboelastography (as described in other chapters), as well as the use of clotting product to correct the coagulopathy of trauma that will inevitably be present. Both the injury process and treatment can have a profound effect on the patient's physiology.

Recognition of a hemodynamically compensated trauma patient who is not obviously exsanguinating can be difficult; the use of pulse, respiratory rate, and blood pressure is neither sensitive nor specific for hemorrhagic shock.[38,39] Blood pressure is determined by the ratio between the functional capacity of the vascular system and the volume of fluid that fills it, along with the pumping power of the heart. Young adults may lose 30% of their circulating volume with little change in their vital signs, and up to 40% of the normal circulating volume can be lost before the limits of compensation are reached and catastrophic vascular collapse occurs. Again, knowledge of the injury mechanism is important.

Vascular Volume Status

The traditional approach to measuring intravascular fluid volume is changing. Routine clinical use of "gold standard" methods, such as central venous pressure (CVP) monitoring and pulmonary artery catheter monitoring, are declining. CVP correlates poorly with total blood volume, and does not always reliably predict fluid responsiveness, so its use in guid-

ing fluid management should be limited.[40,41] Like CVP, pulmonary artery occlusion pressure may fail to reflect changes in preload and may not always be suitable for predicting the response to further fluid administration. However, the effect of volume therapy can be detected in combination with other measures derived from the pulmonary artery catheter, such as cardiac output and mixed venous oxygen saturation. Decreased venous saturation is an indirect indicator of poor tissue perfusion and the need for resuscitation. Newer technologies are available to guide fluid resuscitation with a more dynamic approach, which works better than using historical static parameters. Determining where patients lie on their individual Starling's curve during the resuscitation process may be more important than the type of fluid being administered.[42]

Arterial pressure waveform systems function on the relationship between pulse pressure and stroke volume. Systolic pressure variation, the difference between maximum and minimum systolic pressure during one mechanical breath, has been shown to predict fluid responsiveness to volume loading. Concepts such as pulse pressure variation and stroke volume variation in ventilated patients have been extensively reviewed in the literature and found to be reliable predictors of volume responsiveness.[43] Arterial-based systems in clinical use today include the PiCCO (Phillips; Andover, MA); PulseCO (LiDCO Ltd; Lake Villa, IL); and the FloTrac/Vigileo (Edwards Lifesciences; Irvine, CA). These systems are all minimally invasive.[43]

Stroke volume variation and pulse pressure variation are more reliable indicators of volume responsiveness than CVP, artery occlusion pressure, left ventricular end-diastolic volume index, and global end-diastolic volume index. However, stroke volume variation and pulse pressure variation do have limitations in clinical use. They can be affected by alterations in ventilator settings, chest wall compliance, and dysrythmias, as well as by pharmacologically induced changes in ventricular and aortic compliance.[44]

Esophageal Doppler has been shown to be a clinically useful alternative to thermo-dilution in determination of cardiac output,[45–48] but the process shares a common problem of noninvasive monitors in that the interpretive algorithms have been developed and much of the clinical validation studies performed in relatively healthy and normal patients. However, in multiple trauma patients with at least 2 liters of blood loss, optimization of intravascular volume using esophageal Doppler was associated with decreased blood lactate levels, a lower incidence of infectious complications, and reduced duration of time in intensive care.[48]

Ultrasound assessment of the inferior vena cava to evaluate intravascular volume during resuscitation in trauma patients might be useful in the diagnosis of hypovolemia and is more sensitive than blood pressure alone.[49] A focused transthoracic echocardiographic assessment to evaluate cardiac function and volume status in trauma and critical care patients correlates well with data obtained from a pulmonary artery catheter.[50]

One of the most recent developments in technology is the development of tissue hemoglobin oxygen saturation (StO_2) monitoring. StO_2 has been shown to accurately correlate with peripheral oxygen delivery and to be predictive for those patients who are likely to decompensate and die early from exsanguinating hemorrhage. StO_2 employs near-infrared spectroscopy to permit continuous, noninvasive measurement of StO_2 in muscle. StO_2 is a parameter of tissue perfusion and oxygenation and performs similarly to base deficit in predicting the development of MODS or death after severe torso trauma.[49] It can be used to monitor resuscitation status and guide therapeutic end points in severely injured trauma patients.[50,51]

The splanchnic bed is very sensitive to hypoperfusion and is thought to play a major role in postinjury multiorgan failure.[52] Tissue oxygen tension measurements of the deltoid muscle reflect liver oxygen tension and may serve as a surrogate marker of splanchnic perfusion.[53] As such, these measurements may be used as an index of adequate resuscitation and a predictor of infection and multiorgan failure.[54]

Sublingual and buccal capnometry may be useful in identifying patients in a state of occult circulatory failure and in predicting survival in the trauma patient.[55,56] Although these techniques appear promising, large clinical studies are required to confirm their usefulness.

Correction of Base Deficit and Lactate

Lactate and base deficit have been used as parameters of tissue hypoperfusion and predictors of outcome in hemorrhagic shock. Elevated lactate levels on hospital admission and delayed normalization are associated with increased mortality.[57,58] The lactate level and base deficit should be used to identify patients who need more fluid resuscitation.[59]

The inability to normalize lactate is a predictor of death after trauma, but means to measure lactate may not be immediately available in every facility. It has commonly been thought that, in a normal acid-base environment, lactate would correlate with the anion gap and the base excess of an arterial blood gas. Neither anion gap nor base excess can be used to predict lactate levels; therefore, lactate must be directly measured. The lack of correlation between anion gap and base excess or lactate suggests the presence of unmeasured anions, an impairment in acid-base regulation after injury and resuscitation, or both.[60]

END POINTS IN RESUSCITATION

The goals of resuscitation include restoring circulatory volume via fluid resuscitation, restoring microcirculation, preventing clot disruption (thereby preventing re-bleeding), and maintaining adequate perfusion pressure to the brain and other vital organs. An adjunct to these goals is modification of the inflammatory process.

It is important to correct acidosis by resuscitating the patient to appropriate end points. Current monitoring technology now allows the physician to more rapidly identify the relationship between oxygen delivery and consumption (Exhibit 9-5). End points allow uniformity in gauging the adequacy of resuscitation: preventing under- and over-resuscitation and serving as a basis to compare outcome measures in resuscitation trials. Recent technological advances in patient monitoring allow a wider scope of clinical data to be obtained via less invasive means.

The expedient detection and correction of tissue hypoperfusion associated with compensated shock may limit organ dysfunction, reduce complications, and improve patient outcome. It seems logical that the earlier tissue hypoperfusion is detected and corrected, the greater the likelihood that outcome (ie, lactate and base deficit) will be improved to maximize oxygen delivery and correct tissue dysoxia. The use of StO_2 monitoring is undergoing evaluation in an operational setting. It is difficult to understand why esophageal Doppler technology in particular has not been evaluated in the same way. In the absence of specific cardiac output monitoring in current deployed operations, a combination of transthoracic echo assessment of both the heart and the inferior vena cava caliber on admission,[61] followed by CVP, pulse pressure observations, and regular measurements of base excess and lactate should be used to guide resuscitation.

HYPOTENSIVE RESUSCITATION

Uncertainty exists as to whether blood pressure and heart rate should be restored to normal before definitive hemorrhage control has been established. Work in 1994 showed better outcomes from patients with penetrating torso trauma if fluid resuscitation was delayed until surgical control was achieved.[62] The study patients had reached the hospital quickly, in less than 75 minutes (which happens with some but not all military casualties), and it is unclear whether the finding is applicable in cases of blunt trauma.[63]

Evidence from investigations into combined hemorrhage and blast injury showed that hypotensive resuscitation exacerbated a profound acidosis, possibly due to a compromise in tissue oxygen delivery, which is not compatible with survival after primary blast exposure.[64] Other studies have looked at intraoperative hypotensive resuscitation, initially in both blunt and penetrating trauma patients, then subsequently in only penetrating trauma patients. The researchers found that patients in the lower mean arterial pressure (LMAP) group, who were resuscitated to a mean arterial pressure (MAP) of 50 mmHg, received significantly less blood products and total intravenous fluids than those in the control high MAP (HMAP) group, who were resuscitated to a target MAP of 65 mmHg. The LMAP group also had a significantly lower mortality in the early postoperative period, and a significantly lower international normalized ratio in the postoperative period, indicating less severe coagulopathy. The study has limitations, however, and the results encompass only patients with penetrating injuries, but its findings support hypotensive resuscitation, particularly in penetrating injuries.[65]

As with ventilation, a dilemma exists in treatment of bleeding patients with traumatic brain injury.[66,67] Under-resuscitation, hypotension, and decreased cerebral perfusion can result in devastating secondary brain injury. In contrast, over-resuscitation in the face of ongoing hemorrhage increases blood pressure, reverses vasoconstriction, dislodges early thrombus, and causes dilution coagulopathy. These effects promote further bleeding and accelerate hypothermia, acidosis, and coagulopathy.[63] Fluid overload causes or exacerbates tissue edema, manifested by worsening ALI and brain edema in patients with head injury. Uncontrolled resuscitation is an independent predictor of secondary abdominal compartment syndrome, which is associated with MODS and a poor outcome.[68–70] Even after normalization of blood pressure and heart

EXHIBIT 9-5

IDEAL GOALS OF RESUSCITATION

1. Systolic blood pressure greater than 100 mm Hg.
2. Hematocrit greater than 30%.
3. Urine output at least 1 mL/kg/hour.
4. Base deficit on the arterial blood gases less than -3.
5. Cardiac index at least 4.5 L/min/m2.

rate, up to 85% of severely injured patients still have evidence of inadequate tissue oxygenation (ongoing metabolic acidosis), ie, they are still in compensated shock.[59]

ADJUNCTS AND FLUIDS IN TRAUMA RESUSCITATION

Fluids

Patients resuscitated with crystalloids require a larger amount of fluid to achieve the same end points of resuscitation as compared to plasma expanders, which results in more edema formation.[71] Colloids are generally considered to be a more effective volume expander than crystalloids. However, in the case of an impaired endothelial barrier and increased capillary permeability (as in trauma patients), this volume expansion effect is grossly reduced and is potentially counterproductive.[72,73] Several adverse effects (renal failure, bleeding complications, and anaphylaxis) have been reported with the use of artificial colloids. Colloids are not superior to crystalloids in treating hypovolemia in critically ill patients and show no survival benefit.[74,75] As a result, the use of crystalloids is currently recommended in trauma resuscitation.[76]

Hypertonic saline solutions are effective and well tolerated in the treatment of hypovolemic shock. Potential benefits are reduced requirements for fluids as well as less edema formation and immune modulation, thereby decreasing the risk of ARDS and MODS. Hypertonic saline is effective in reducing intracranial pressure in traumatic brain injury patients, but its use in trauma resuscitation is not associated with improved survival.[77]

Inotropic and Vasoactive Agents

Comparing the early use of vasopressors with the aggressive early use of crystalloid resuscitation in severely injured patients has revealed that early use of vasopressors almost doubles mortality.[78] Aggressive crystalloid resuscitation was independently associated with a survival benefit in the younger population (age < 55 years). The study concluded that early hemodynamic support in the trauma patient should rely primarily on aggressive fluid administration, and vasopressors should not be used in the early resuscitation period.[78] Beneficial effects of both arginine vasopressin and phenylephrine, as compared with crystalloid alone, have been found with traumatic brain injury, pulmonary contusion, and hemorrhagic shock. The Vasopressin in Refractory Traumatic Hemorrhagic Shock (VITRIS) study[79] is an international randomized controlled trial, recently initiated to assess the effects of arginine vasopressin in traumatic hemorrhagic shock patients who don't respond to standard shock treatment in the prehospital setting. Vasopressin may have three different beneficial effects: increasing blood pressure in refractory shock, shifting blood from a subdiaphragmatic bleeding site towards the heart and brain, and decreasing fluid resuscitation requirements.[79]

FRACTURE FIXATION

The optimal timing of fracture fixation in multiply injured patients is still widely debated. However, there is no doubt that early fixation of fractures reduces inflammation at the site of injury and decreases pain and opiate requirements. Evidence also shows that this approach reduces overall pulmonary complications and promotes early mobilization. Larger studies tend to indicate that the early stabilization of femoral fractures with definitive intramedullary nailing appears to be the treatment of choice, even for patients with combined head and chest injuries.[80–82] Practically, in the field hospital, external fixation will be the norm due to damage control resuscitation and infection control principles. It is vital that the timing of definitive fixation is discussed as part of the onward movement of the patient.

ADMISSION TO THE INTENSIVE CARE UNIT

When the patient first arrives in the intensive care unit, physiological correction and further evaluation of injuries must continue. The patient needs to be fully evaluated as quickly as possible so that all injuries and concurrent medical conditions are recognized.[83] The more severely injured patients, particularly those with traumatic brain injury, are at the greatest risk for occult lesions. A common pitfall is to focus only on the immediately life-threatening wounds, while inadvertently ignoring less obvious but potentially debilitating injuries. Repeated limb compartment checks and continued presence of distal pulses must be recorded in all patients with limb injuries. Clinical vigilance (and rising serum creatinine kinase levels) at this stage can prevent limb loss later. A thorough examination of the eyes

and ears is also indicated (an often overlooked part of the examination).

Infection Care Bundles

In addition to mechanical and thrombotic complications of central venous catheter (CVC) insertion, which can be life threatening, CVC-related blood stream infections are a significant source of morbidity and mortality. CVC insertion guidelines should be adhered to in the intensive care unit at all times, and should be the gold standard in the trauma resuscitation room as well. In sick patients where central venous access is a a time-critical intervention, strict adherence to an aseptic technique is not always possible, in which case any deviations from local guidelines should be documented. Some units advocate changing the CVC line within 24 hours of admission to intensive care. Clear documentation is the key to avoiding potential contamination from catheters left in place too long.

In addition to particular attention to CVC care, all staff involved in the care of critically ill trauma patients must pay close attention to simple precautions to prevent the spread of infections. Hand-washing and use of alcohol gel after contact with any patient or bed space, and again before the next patient contact, must be rigidly enforced. Gloves and aprons should be worn for any direct patient contact. Ward rounds should be limited to essential team members only around the patient bed space. Senior staff (medical and nursing) have a significant leadership role in gaining compliance with these simple measures.[84–86]

Early Enteral Nutrition

Clinical practice guidelines have been published that recommend initiating enteral nutrition (EN) in the trauma patient "as early as feasible."[87,88] Laboratory studies have established the physiological benefits attributable to the provision of EN in trauma patients within 24 hours of injury. Early EN (within 24 h) is associated with a significant reduction in mortality Trials have reported significant reductions in infectious complications in patients receiving early EN, and one reported a trend toward a decrease in the severity of MODS in patients receiving early EN.[89] The provision of early EN in the critically ill trauma patient is standard practice to preserve gut barrier function and maintain gut-associated lymphoid tissue mass and function.[85,86]

By maintaining the host defense functions of the intestine, translocation of bacteria from the gut into the bloodstream and consequent systemic infectious complications are reduced.[90] The provision of early EN also down-regulates the systemic immune response to bacterial translocation, which reduces overall oxidative stresses and moderates the expression of SIRS[89] and subsequent progression to MODS. The appropriate provision of EN may also decrease aspiration-related complications such as pneumonia.[90,91]

SUMMARY

Field anesthesia and intensive care are inextricably linked.[92] However, the progress of severely injured patients through the medical chain has a tendency to be compartmentalized. Recent experience has shown that austerity is no barrier to high standards of care and successful outcomes.[93] The role of the intensive care team in general and the physician in particular should extend beyond the boundaries of the intensive care unit. The expectation should be that care in the field will strive to follow recognized good practices established at home, including simple preventive strategies such as sepsis bundles and other quality improvement measures. Rather than being associated with particular equipment and technology, high-quality intensive care should imply the vigilant attention of a skilled multidisciplinary team. Operations in Iraq and Afghanistan have allowed development of military trauma systems resulting in an increasing number of multiply injured patents surviving.[94–97]

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