

Intensive care unit management of the trauma patient

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LEARNING OBJECTIVES

On completion of this article, the reader should be able to:

1. Describe successful resuscitation of the patient with multiple injuries.
2. Explain the management of the patient with traumatic brain injury.
3. Use this information in the clinical setting.

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Objective: The goal of this concise review is to provide an overview of some of the most important intensive care unit issues and approaches that are unique to trauma patients as compared with the general intensive care unit population.

Study Selection: Clinical trials in trauma patients focusing on hemorrhage control, issues in resuscitation, staged operative repair of multiple injuries, the diagnosis and therapy of the abdominal compartment syndrome, and the treatment of traumatic brain injury were identified on PubMed.

Conclusions: The intensive care unit care of the trauma patient differs from that of other intensive care unit patients in many ways, one of the most important being the need to continuously integrate

operative and nonoperative therapy. Although progress in the care of the injured has been made, death due to uncontrolled bleeding, severe head injury, or the development of multiple organ dysfunction syndrome remains all too common in this patient population. Furthermore, due to the potential nature of the injuries, the conundrum not infrequently arises that the optimal treatment for one injury or organ system, such as preoperative permissive hypotension in actively bleeding patients, may result in suboptimal or even deleterious therapy in the presence of another injury, such as traumatic brain injury. (Crit Care Med 2006; 34:2294–2301)

KEY WORDS: trauma; brain injury; hemorrhagic shock; resuscitation; organ failure; abdominal compartment syndrome

Injuries are the leading cause of death in patients <45 yrs of age and the third leading cause of death overall in the United States, claiming >100,000 lives in 2002 (1). In fact, each year, >1.5 million people are hospitalized as a result of acute injury, and injury-related disability is a major public health problem (2), with head injury, hemorrhage, and sepsis/multiple organ failure (MOF) being the three major causes of death and disability in this patient population (3). Because the majority of the

unique aspects of intensive care unit (ICU) care of the trauma patient are focused on head injury, hemorrhage control, resuscitation, and the staged treatment of multiple injuries, we will primarily focus on these topics rather than on issues that are common to all ICU patients, such as ventilatory strategies, infection, and nutrition. As in other areas of ICU care, management of patients in a trauma-specific ICU or a closed unit is associated with improved clinical outcome (4).

Generally speaking, the ICU care of the trauma patient has overlapping treatment phases, which include the resuscitative and operative phases. However, for clarity, each of these phases will be discussed separately.

Resuscitative Phase

The resuscitative phase has as its central goals the restoration of an effective blood volume, optimization of tissue per-

fusion, and the prevention/limitation of ischemia-reperfusion injury. Recognition that shock causes a global ischemia-reperfusion injury, which directly and indirectly leads to cellular and hence organ injury, has led to an increasing emphasis on the adequacy of volume resuscitation and a search for more effective resuscitation fluids. Although the colloid-crystalloid controversy continues and remains to be resolved (5), there is increasing data suggesting that Ringer lactate is proinflammatory and thus may exacerbate the inflammatory response and contribute to the development of organ injury in shock states (6–9). Given these concerns, plus the recent recognition that large-volume resuscitation with crystalloid solutions contributes to the development of the abdominal compartment syndrome (10), attention has refocused on the early re-

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suscitation of trauma patients with hypertonic (7.5%) saline. Hypertonic saline is a promising *initial* resuscitation option, and multiple investigators have shown experimentally that at low volumes (4 mL/kg), it is more effective at restoring the extravascular volume, cardiac output, and organ perfusion than large-volume resuscitation with Ringer lactate (11–14). Another important aspect of hypertonic saline is its ability to limit hemorrhagic shock-induced immunosuppression and organ injury in clinically relevant animal models (15–17), although there is some evidence that the administration of hypertonic saline after the initial resuscitation period may be deleterious due to its ability to stimulate neutrophil function (18). The largest clinical trial of hypertonic saline vs. Ringer lactate tested hypertonic saline's ability to improve survival and limit organ injury when administered in the field (19). Overall survival was similar between the two groups, although survival was increased in the subgroup of trauma patients requiring emergency surgery. In addition, the hypertonic group had a decreased prevalence of acute respiratory distress syndrome, renal failure, and coagulopathy. Nonetheless, at the current time, due to a paucity of clinical trials, there are not enough data to determine whether initial hypertonic resuscitation is superior to standard crystalloid resuscitation of the trauma patient (20).

Another encouraging approach is the use of resuscitation fluids containing antioxidants. Three clinical trials have been published showing that splanchnic-directed antioxidant therapy helps prevent MOF in trauma patients (21–23). These trials were based on experimental studies indicating that splanchnic blood flow is disproportionately decreased after injury, stress, or shock in trauma and ICU patients and in clinical gastric tonometry studies indicating that gut ischemia is a better predictor of the development of acute respiratory distress syndrome and multiple organ dysfunction syndrome than global indices of oxygen delivery (24). In addition, based on an enlarging investigative effort, other novel resuscitative fluids, such as ethyl pyruvate (25), that have pharmacologic and volume restorative effects are being actively tested in preclinical studies.

The role of blood transfusions in trauma patients has also undergone an intense reevaluation based on clinical studies showing that blood transfusions are an independent predictor of the development of MOF,

especially when blood older than 2 wks is administered (26, 27). This observation, plus the fact that blood is immune suppressive (28, 29) and that ICU patients can be safely managed with hemoglobin levels in the range of 7g/dL (30, 31), has led to the emergence of a selective transfusion policy, in which prophylactic transfusions are no longer routinely administered. The mechanisms by which "old" blood potentiates organ failure has been investigated in animal models and seems to be related to the proinflammatory nature of plasma and lipid factors released/produced during the storage period (32) and to the inability of stored red blood cells to traverse the microcirculation, resulting in microcirculatory dysfunction (33). Because large-volume blood transfusions are necessary in many trauma patients with hemorrhagic shock, one potential solution is the use of blood substitutes (34). However, until recently, enthusiasm for stroma-free hemoglobin solutions has been low based on the results of a phase III clinical trial, in which the administration of diaspirin cross-linked hemoglobin to trauma patients was associated with almost a three-fold increase in mortality (46% vs. 17%) (35). In contrast to this earlier study, recent clinical trials with human polymerized hemoglobin (PolyHeme) are encouraging (36). This difference in clinical outcome seems to be related to the fact that this newer polymerized hemoglobin-based blood substitute, in contrast to the older products, does not avidly bind nitric oxide and hence does not act as a vasoconstrictive agent. Thus, although hemoglobin solutions can potentially limit blood transfusion, caution must be exercised because other polymerized hemoglobin solutions seem to manifest some degree of vasoconstrictive activity (34), and the administration of a resuscitative fluid that has vasoconstrictive properties to a hypovolemic patient could be catastrophic.

A second issue related to volume resuscitation of the trauma patient is the development of abdominal compartment syndrome (ACS). Because the treatment of ACS is surgical, this topic will be discussed below in the operative phase section of the review. However, it is important to stress that there is a correlation between the magnitude of crystalloid fluid resuscitation and the development of ACS, and patients receiving supranormal resuscitative regimens were found to have twice the rate of ACS as normally

resuscitated patients (10). A third issue also related to volume resuscitation is the end point of resuscitation. In the severely injured trauma patient, blood pressure may be restored by vasoconstriction; thus, restoration of blood pressure or even urine output does not ensure that organ blood flow and tissue perfusion have been adequately restored. In this setting, arterial base deficit or serum lactate have been found to be better indicators of the adequacy of tissue perfusion and hence volume resuscitation than blood pressure or urine output (37). A worsening base deficit or serum lactate has been shown to correlate with ongoing blood loss or inadequate volume resuscitation, whereas improvements in these variables are indicative of adequate volume resuscitation. In severely injured patients, because the period of volume resuscitation may last 24–48 hrs, serial measurements are important. The resuscitative goal should be to reduce and keep the base deficit at less than -2 mmol/L and serum lactate at <1.5 mEq/L. In fact, prospective studies have documented both a correlation between the magnitude of the initial base deficit and survival and between survival and clearance of the base deficit; those patients who cleared their base deficits within 2 days had a high survival rate, whereas only 13% of those who had not cleared their deficit by 2 days survived (38, 39). As an aside, a persistently elevated base deficit, despite what should be adequate volume resuscitation, indicates ongoing hemorrhage in the trauma patient population and may be an indication for urgent surgery (40).

Lastly, identifying all significant injuries is a key part of the resuscitative phase of trauma care. Missed injuries are not uncommon in trauma patients (41–43), and the consequences of missed injuries may be significant because many of these missed injuries require prompt operative treatment, and deaths from missed injuries are not rare (43). The highest prevalence of missed significant injuries occurs in trauma patients with a decreased sensorium, hemodynamic instability, or substance abuse.

Operative Phase

Although the specifics of the operative care of the trauma patient are beyond the scope of this review, certain aspects are important for ICU management. An example is when the initial operation must be terminated before the definitive repair

of all injuries because of hemodynamic instability or because of the need to limit the development of coagulopathy, acidosis, and hypothermia—the so called damage-control laparotomy. The concept of damage control is to control hemorrhage by repair or packing and to control feculent contamination of the peritoneal cavity. This concept was learned from the care of trauma patients with multiple injuries, in which utilization of a damage-control approach was shown to improve survival (44–46). The rationale behind a damage-control laparotomy is the clinical observation that *prolonged* attempts at definitive control of intraabdominal injuries can result in hemodynamic instability, acidosis, and surgically uncontrollable coagulopathic bleeding. If the patient survives the operation, the prevalence of postoperative MOF is high. The damage-control approach allows for the rapid transport of the patient to the ICU for continued resuscitation and correction of metabolic abnormalities. A planned reoperation is safer and easier in patients who have been warmed, fully resuscitated, and who have had their coagulopathy and acidosis corrected. In patients with ongoing surgical bleeding after damage-control laparotomy (patients with major liver injuries or pelvic fractures), angiographic control of bleeding has been shown to be successful (47–50). Although the data are limited, in patients with medical (coagulopathic) bleeding who do not respond to standard therapy, there is an emerging role for factor VIIa therapy (51).

A second aspect to be cognizant of in the ICU management of the trauma patient is ACS. ACS can be viewed as a reversible mechanical cause of MOF that is related to increased intraabdominal pressure (52, 53). As the intraabdominal pressure increases, abdominal visceral perfusion decreases, ventilation is impaired, and cardiac output declines (53–55). Clinically, this manifests as a decreasing urine output, inadequate ventilation associated with elevated peak airway pressures, and hypotension. Patients sustaining multiple trauma, massive hemorrhage, prolonged operations with massive volume resuscitation, and those requiring intraabdominal packing to control bleeding or who have massively distended bowel are at the highest risk of developing the ACS. ACS can also develop in patients in severe hemorrhagic shock without an abdominal or retroperitoneal injury. This phenomenon is known as secondary ACS and is due to progressive abdominal visceral

and retroperitoneal edema associated with massive fluid resuscitation in patients with hemorrhagic shock (56). The diagnosis of ACS is made by measuring the abdominal pressure through a Foley catheter placed in the bladder. ACS is defined as the combination of 1) a urinary bladder pressure of >25 mmHg, 2) progressive organ dysfunction (urinary output of <0.5 mL·kg⁻¹·hr⁻¹ or PaO₂/FiO₂ of <150 or peak airway pressure of >45 cm H₂O or cardiac index of <3 L·min⁻¹·m⁻² despite resuscitation), and 3) improved organ function after decompression. Untreated, ACS is lethal, with a mortality rate of 100% documented in subsets of patients with ACS who did not undergo abdominal decompression (54). Thus, a high index of suspicion leading to an early diagnosis is critical because the treatment of ACS with a decompressive laparotomy improves organ dysfunction. In fact, surgical treatment of increased intraabdominal pressure leads in most instances to a rapid and profound correction of the physiologic abnormalities. Return of urine output is almost immediate, as is an increase in cardiac output and a decrease in peak airway pressures (55). The abdomen may be opened in the ICU or in the operating room, depending on the patient's condition. Once a patient has an open abdomen, whether due to ACS or as a consequence of a damage-control laparotomy, the exposed abdominal viscera must be managed. Several techniques are available, including the use of vacuum-assisted closure (57). Regardless of the method used to manage the exposed abdominal viscera, the goals of management include prevention of evaporative water loss, limitation of bacterial contamination, drainage of abdominal fluid, prevention of abdominal visceral desiccation and fistula formation, and optimization of the abdominal cavity for secondary abdominal wall closure.

In addition to the other surgical issues, early fixation of long-bone fractures in severely injured patients seems to have resulted in a dramatic reduction in the prevalence of acute respiratory distress syndrome and MOF as compared with those patients treated with prolonged traction (58–60). However, there is some evidence that patients with significant thoracic injuries or head trauma may be harmed by early operative fracture fixation due to a higher prevalence of acute respiratory distress syndrome and secondary brain injury, respectively (61, 62). Despite these subgroups, most evidence supports early fracture fixation as an effective method of reducing organ failure

in patients with long-bone fractures. Thus, an important aspect of ICU care is the integration and timing of secondary operations in the therapy of the multiply injured patient.

Lastly, the ICU team must recognize the potential need for prompt operations or reoperations on properly selected patients who are showing continued signs of organ dysfunction before MOF becomes established. This concept is especially important in patients who have undergone previous abdominal surgery, in whom the abdomen may be the source of infection or have retained necrotic tissues, large hematomas, or fluid collections that are exacerbating the inflammatory response.

Traumatic Brain Injury

Management of the patient with traumatic brain injury (TBI) is a major portion of the ICU care of the multiple-trauma patient because approximately 500,000 patients in the United States sustain a TBI, of whom about 10% (50,000) die, making the brain one of the most frequently and important parts of the body injured in polytrauma patients (63, 64). Mortality due to TBI occurs throughout the postinjury period (3), with the most severely injured patients (Glasgow Coma Score of ≤ 8) requiring intubation at the scene of the accident or in the emergency department (65). It is now well established that episodes of hypoxia or hypotension exacerbate the extent of the brain injury and are associated with a worsened long-term outcome (66, 67). In this context, failure to maintain a systolic blood pressure of >90 mm Hg and a PaO₂ of >60 mm Hg is associated with increased morbidity and mortality (68, 69). Thus, avoidance of hypoxia and hypotension are critical during the resuscitative and ICU phases of care. Because cerebral edema is a contributing factor to the progression of TBI and hypertonic saline has been shown to reduce intracranial pressure (ICP) in experimental brain injury (70), several studies have investigated the prehospital use of small-volume hypertonic saline in these patients (19, 71). In fact, a meta-analysis published in 1997 of eight prospective trials indicated that hypertonic saline–dextran resuscitation increased survival from 27% to 38% in patients with very severe TBI (72). However, a recent, large, controlled trial in hypotensive TBI patients did not show any early or long-term benefits of hypertonic

saline administration (73). Thus, the use of hypertonic saline in these patients remains controversial, although studies in pediatric patients indicate that refractory intracranial hypertension can be successfully treated with hypertonic saline (74, 75). Although the role of hypotensive resuscitation in patients with surgical sites of bleeding to limit blood loss before operative or angiographic control of the bleeding remains controversial (76, 77), it is clear that hypotensive resuscitation is absolutely contraindicated in severe TBI patients because it is associated with a poor neurologic outcome (77). In addition, because patients with TBI may have surgically treatable lesions (i.e., subdural or epidural hematomas), it is critical to obtain a head computed tomographic scan as early as feasible to guide therapy and serve as a baseline for further scans. In fact, daily head computed tomographic scans are frequently required in the patient with severe TBI.

In addition to avoiding hypoxia and hypotension, a major therapeutic goal in the care of the patient with TBI is the maintenance of adequate cerebral perfusion. Because increased ICP is associated with impaired cerebral perfusion, the placement of an invasive pressure monitor to measure ICP is a key element of care (78, 79). Because patients with severe TBI (i.e., Glasgow Coma Score of ≤ 8) have a 60% chance of having an increased ICP (80), it has been recommended that ICP monitoring be routinely employed in this patient population (63, 65). The two key hemodynamic factors associated with cerebral perfusion are the ICP and the cerebral perfusion pressure (cerebral perfusion pressure = mean arterial pressure minus the ICP). Based on class II data showing that functional outcome is inversely related to the longer the percentage of time the ICP is >20 mm Hg, it seems that attempts to lower the ICP should be initiated when it is >20 mm Hg (63, 68, 81). Likewise, there are excellent class II data showing that maintaining a cerebral perfusion pressure of >60 mm Hg is associated with a reduction in morbidity and mortality (82, 83) and reduced long-term neurologic disability (84). Thus, once an ICP monitor is in place, therapy is directed at maintaining an adequate cerebral perfusion pressure, either by reducing the ICP, increasing the mean arterial pressure, or both.

As a rule of thumb, whenever possible, the bed should be kept elevated at 30

degrees to reduce the ICP and fluid should be administered to have a mean arterial pressure of 90 mm Hg. In addition, control of fever, analgesia, and sedation and ventilatory management to ensure adequate oxygenation and ventilation are important adjuvant therapies. Although, in the past, patients with severe TBI were treated with hyperventilation to improve cerebral blood flow, prospective randomized trials have shown that hyperventilation ($Paco_2$, ≤ 25 – 30 mm Hg) worsens the neurologic outcome (85). This has resulted in the recommendation that the $Paco_2$ of ventilated patients be kept in the range of 30–35 mm Hg (63). Interestingly, although evidence-based guidelines exist to direct the management of severe TBI, such as the use of ICP monitoring, limiting hyperventilation, maintaining cerebral perfusion pressure, and so on (63), there remains considerable institutional variation in following these guidelines (86).

If the ICP remains at >20 mm Hg, despite adequate sedation, then additional therapies are utilized. If hyperthermia ($>38.5^\circ\text{C}$) is present, especially when the ICP is elevated, then it should be aggressively lowered because hyperthermia in this situation has been associated with an increased mortality rate and a worsened neurologic outcome (87). In fact, several groups have proposed the use of deliberate mild hypothermia (33– 35°C) to limit cerebral oxygen demand, although its effectiveness remains controversial (88). In the normothermic patient with an increased ICP and a ventriculostomy, cerebrospinal fluid drainage is an effective strategy to decrease the ICP (89). If the patient does not have a ventriculostomy or if cerebrospinal fluid drainage is ineffective, mannitol should be administered because of its diuretic effect and its ability to decrease the ICP, increase the cerebral perfusion pressure, and thereby improve cerebral blood flow (90). Although prospective trials have shown that mannitol is more effective than barbiturate therapy in improving mortality and neurologic outcome (91), high-dose barbiturate therapy needs to be considered for intracranial hypertension that is refractory to maximal medical and surgical therapy, although it is associated with substantial complications (92). Likewise, in desperate situations of refractory intracranial hypertension, decompressive craniotomy should be considered as a last resort (93). Although it was hoped that steroids would be effective in reducing

brain edema, multiple clinical trials have clearly shown that steroids are not helpful (94, 95). On the other hand, there is abundant class I data documenting the beneficial effects of a 7-day course of seizure prophylaxis in patients with severe TBI (96).

Maintenance of Organ Function and Additional ICU Therapies

Strategies directed at the protection and maintenance of pulmonary, cardiac, intestinal, and renal function are part of the ICU care of the trauma patient. In fact, many of the ICU therapies shown in prospective, randomized, clinical trials to reduce mortality in other populations are commonly used in the treatment of trauma patients, although they remain to be fully validated in the trauma population. These include: 1) glycemic control (97, 98), 2) early goal-directed hemodynamic therapy beginning in the emergency department (99), 3) the use of early adequate empirical antibiotic use for patients with suspected pneumonia (100), 4) the use of lower tidal volumes and limitation of plateau pressures to reduce pulmonary injury in ventilated patients (101), 5) use of low-dose steroids in patients with septic shock associated with adrenal insufficiency (102), and 6) the creation of a closed ICU (103). A number of other therapeutic organ-directed strategies are important in trauma as in other patient populations. Some of these strategies support several systems, such as early enteral alimentation of the trauma patient, which helps preserve intestinal function and limit infectious complications (104). Other strategies, such as elevation of the head of the bed of ventilated patients, seems to both reduce the prevalence of pneumonia and to better preserve pulmonary function (105). Methods of supporting renal function, such as low-dose dopamine, have not been found to be effective (106), and thus, currently, the best way to limit renal failure is to avoid under-resuscitation and to promptly diagnose and treat infectious complications. Once renal failure has occurred, continuous venovenous hemodialysis seems to be superior to hemodialysis because it avoids the need for systemic anticoagulation and is less likely to cause hypotensive episodes (107).

Cardiac issues can complicate the recovery of trauma and other ICU patients and range from cardiac contusion to the consequences of preexistent coronary artery disease. Many trauma patients pre-

senting to the ICU present with tachyarrhythmias, hypotension, or both. Before treatment is initiated, it is important to differentiate their pathogenesis. The differential diagnosis includes, but is not limited to, preexisting cardiac dysfunction, sepsis, under-resuscitation, anxiety, and pain. A specific issue related to trauma patients is blunt cardiac injury (BCI) (108, 109). Trauma victims who have had direct precordial impact or crush injuries are known to be at increased risk for BCI, formerly known as myocardial contusion. There is no diagnostic gold standard for the diagnosis of BCI. The presentation may vary from minor electrocardiographic or cardiac enzyme changes to cardiovascular collapse. The right heart is the most common site of BCI secondary to its anatomic position under the sternum. Most patients with BCI recover without any sequelae, and due to its frequent benign nature, the diagnosis of BCI may never even be suspected. However, BCI should be suspected in patients with chest trauma who present with a poor cardiovascular response to therapy, and in this subgroup of patients, inotropic support may be required. An incompletely resolved issue is the use of beta-blockade in the trauma patient because its use can be beneficial or detrimental, depending on the situation. For example, beta-blockers should not be instituted in patients who are actively being resuscitated unless the patient has previously been on a beta-blocker for cardiac dysfunction. On the other hand, if there is clinical and laboratory evidence of myocardial injury, beta-blocker use should be entertained.

In addition to these topics, the notion of pharmacologic manipulation of the metabolic response to injury has been an area of intense study because it is well recognized that trauma- or sepsis-induced hypercatabolism and muscle wasting is a major cause of morbidity and mortality. Based on research in this field, two major approaches have emerged. The first is nutrient pharmacology, in which specific nutrients and growth factors are administered for their ability to support the physiologic function and repair of certain organs and tissues and to augment the immune system and promote wound healing (110). A second approach involves the use of hormones or anabolic agents to improve wound healing, improve immune function, or support organ function in addition to limiting the hypermetabolic response. Anabolic agents used to improve the metabolic response

have included, insulin-like growth factor-1, insulin, beta-blockade, and the use of anabolic androgens with minimal virilizing activity, such as oxandrolone. Although encouraging results from prospective randomized trials in burn patients (110–112) have resulted in the Food and Drug Administration approving oxandrolone for use as an anabolic agent in severely stressed patients, there are no class I or II data in nonburn trauma patients to support its use as of yet.

In addition to organ-directed therapies, the prompt and efficient diagnosis of infectious complications is important because, after major trauma, infectious complications are relatively common and are a major cause of morbidity and mortality (113). Because many of the noninfectious stimuli associated with trauma, such as soft-tissue and bony injuries, traumatic pancreatitis, shock, or brain injury, can generate a septic-like state and even lead to acute respiratory distress syndrome or multiple organ dysfunction syndrome, it can be clinically difficult to accurately differentiate the infected from the noninfected, seemingly septic trauma patient without an apparent source of infection (i.e., abdomen, wound, or injury site). The approach to this conundrum remains to be fully resolved, and little if any prospective data are available in this area. Nonetheless, after doing a “fever workup,” most trauma intensivists would consider starting empirical antibiotics, especially in high-risk patients, and because of the risk of the emergence of antibiotic-resistant organisms, plan on stopping these antibiotics in 3–5 days if no site of infection is documented during this time period. In addition to pneumonia, which is common in trauma ICU patients, as in all mechanically ventilated ICU patients, trauma patients are at increased risk of developing wound and soft-tissue infections. Because trauma injuries can even devitalize tissues, trauma patients are uniquely susceptible to the development of soft-tissue and wound sepsis at both operative and nonoperative injury sites, and such infections occur in about 10% of trauma ICU patients (113). For this reason, daily examination of all incisions and sites of injury must be performed, looking for signs of infection, and in patients who are septic and deteriorating, all closed wounds, no matter how seemingly innocuous, must be considered a potential septic source and thoroughly investigated. In essentially all cases, it is better to locally explore a wound or injury site

unnecessarily than to miss or delay the diagnosis of a deep infection or necrotizing fasciitis. Once diagnosed, the principles of therapy for soft-tissue infections are relatively straight forward and include adequate drainage of the site, evacuation of hematomas if present, removal of devitalized tissues, and the appropriate institution of antibiotics followed by aggressive wound care.

Conclusion

In this article, we have mainly focused on areas of ICU management that are unique to trauma patients. Although the principles of the critical care of the trauma patient are similar to those of the care of other severely ill patients, the presence of injuries to a wide variety of organ systems and the risk and consequences of severe hemorrhage can complicate diagnosis and the delivery of necessary care. In addition, the optimal treatment for one injury, such as maintaining an elevated mean arterial pressure in patients with head injuries, can confound the treatment of other injuries, such as aortic transections. Likewise, the optimal timing of the definitive operative treatment of secondary injuries, such as fractures, may be influenced by the treatment needed for the patient's other injuries and the patient's hemodynamic status. Thus, there is an intricate balance between the operative and nonoperative care of the trauma patient that is observed in few other patient populations.

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