Multiple trauma in children: Critical care overview

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Multiple trauma is more than the sum of the injuries. Management not only of the physiologic injury but also of the pathophysiologic responses, along with integration of the child's emotional and developmental needs and the child's family, forms the basis of trauma care. Multiple trauma in children also elicits profound psychological responses from the healthcare providers involved with these children. This overview will address the pathophysiology of multiple trauma in children and the general principles of trauma management by an integrated trauma team.

Trauma is a systemic disease. Multiple trauma stimulates the release of multiple inflammatory mediators. A lethal triad of hypothermia, acidosis, and coagulopathy is the direct result of trauma and secondary injury from the systemic response to trauma. Controlling and responding to the secondary pathophysiologic sequelae of trauma is the cornerstone of trauma management in the multiply injured, critically ill child. Damage control surgery is a new, rational approach to the child with multiple trauma. The selection of children for damage control surgery depends on the severity of injury. Major abdominal vascular injuries and multiple visceral injuries are best considered for this approach.

The effective management of childhood multiple trauma requires a combined team approach, consideration of the child and family, an organized trauma system, and an effective quality assurance and improvement mechanism. (Crit Care Med 2002; 30[Suppl.]:S468–S477)

KEY WORDS: pediatric trauma; acidosis; hypothermia

Multiple trauma is more than the sum of the injuries. A child with a fractured spleen, contused lung, and a fractured femur has more widely disseminated disease than simply the individual trauma to these organs. Multiple trauma, in addition to the structural damage, produces a systemic response that threatens or impairs the function of every organ system in the body and impacts the child's emotional and psychological status. The child's future physical and mental development may be permanently altered. Multiple trauma affects not only the individual child but also her family, friends, and school and has implications for society at large. The profound family impact may alter the function and structure of the family unit. Multiple trauma in children also elicits profound psychological responses from the healthcare providers involved with these children. To optimize the child's physical and mental outcomes requires both timely and expert medical care and attention to family integrity. The child is almost always an innocent victim of someone else's inflicted trauma, either accidental or nonaccidental. Healthcare providers must overcome many barriers to care, including those that arise from their psycho-emotional responses to the acute catastrophe that has befallen the child. This overview will address the pathophysiology of multiple trauma in children and the general principles of trauma management. Trauma's unexpected nature, catastrophic onset, and both physical and mental sequelae, require a highly sophisticated and integrated trauma management system specifically focused on the child.

TRAUMA TEAM

The effective management of pediatric trauma requires the integration of a multidisciplinary team, including surgeons, emergency physicians and nurses, intensivists and nurses, respiratory therapists, radiology, various subspecialties (neurosurgery, orthopedic surgery, etc.), and the ready availability of laboratory and operating room facilities. Nowhere is this more important than in dealing with critically ill children with multiple trauma in which minutes matter. The prepared trauma team improves performance of the resuscitative efforts and the outcome of injured patients (1).

The well-organized trauma team must include a clearly defined trauma leader and assigned roles and responsibilities for each team member. These roles must be understood and respected by the entire trauma team if the high-tension environment of trauma resuscitation is to operate smoothly (2). The trauma team leader should be chosen prospectively by multidisciplinary consensus. In most situations, this person will be the attending traumatologist, attending surgeon, or surgical fellow (3). It is the responsibility of the team leader to assign personnel to the previously defined roles (as outlined in the American College of Surgeons Advanced Trauma Life Support guidelines), to make care decisions, and to direct procedures when necessary. The team leader is also responsible for organizing a care plan to include diagnostic evaluation, radiology, operating room, intensive care unit, hospital ward, discharge home, and appropriate follow-up indicated by the extent of the injuries. In all of these cases, the team leader is responsible for communicating with the appropriate personnel to ensure that the personnel participating in the care of the injured child are well informed and prepared. The findings, plans, and treatments must be well documented as the resuscitation takes place

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to avoid potentially serious errors in patient care. The continuing care of the most severely injured children requires the full cooperation of all subspecialties involved in the child's care, with open communication and cooperation among the caregivers. The final decision making should be directed by the attending service, which may be the trauma team, the critical care team, or in some instances, it may be a subspecialty team. These distinctions must be organized and understood before critical issues arise and inefficiencies have opportunity to manifest and impair the child's care. It is also important to remember that families are also involved in the care of the injured child, and they must also be informed of pertinent findings and plans. In some situations, they also have been included during the resuscitation (4).

PRIORITIES IN ASSESSMENT AND CARE

The initial approach to the child who has suffered multiple trauma is guided by the "ABCDEs" of the primary survey. The well-known need to guarantee the *airway*, ensure *breathing*, restore the *circulation*, and control hemorrhage, followed by the assessment and documentation of neurologic status and other *disabilities*, and finally, *exposure* and *examination* are the tried and true tenets for managing multiple trauma. The assessment of the injured child begins in the field and is communicated to the receiving hospital so that appropriate trauma team notification may occur. Trauma resuscitation conducted in the trauma suite or emergency department follows the protocols outlined by the American College of Surgeons in the Advanced Trauma Life Support course. The primary survey is conducted with attention to the airway with assessment and maintenance of a patent and stable airway. This may require simple supplemental oxygen, endotracheal intubation, or establishing a surgical airway.

Airway Issues. The first priority in managing a child with acute trauma is to ensure a patent airway. This may present some challenges if airway trauma has occurred. Because head injury is the most common form of injury to children due to the fact that they have a relatively large head compared with their torso, facial injuries are common. Soft-tissue, neck, and airway injuries are somewhat less common in children than in adults due to the shorter, better-protected neck and more anterior larynx. Thus, direct, blunt, or penetrating injury to the airway is less common.

Special airway considerations in pediatric trauma include the possibility of cervical spinal injuries, the likelihood of head injury, and the likely presence of full stomach are also important considerations. In addition, if the airway is compromised by direct trauma, special techniques to facilitate endotracheal intubation, such as fiberoptic intubation, may be difficult in smaller children. Finally, the presence of foreign bodies in the airway, either from regurgitation of gastric contents or other foreign objects that may have entered the child's airway, and the presence of burns, which may cause airway edema, also may complicate management of the airway. Spinal injury may be occult (spinal cord injury without obvious radiologic abnormality) (5), and the decreased cervical stability resulting from the increased laxity of cervical supporting ligaments indicates the need to always consider cervical injury (6).

Development of significant respiratory distress and an early suspicion that the airway may require intubation and management underlie the concept of aggressive airway management in multiply traumatized children. All multiple-trauma victims should initially be given supplemental oxygen by face mask. If desaturation occurs, 100% oxygen by nonrebreather should be used and airway intubation considered. A thorough assessment of the child may reveal respiratory distress, airway obstruction, obtundation from head injury with lost airway reflexes, and absent respiration, which may indicate cervical cord trauma or profound central nervous system injury. All indicate the need for urgent airway intervention.

The preferred approach for airway intubation in children with trauma is direct laryngoscopy with oral-endotracheal intubation. The high prevalence of head injury, the possibility of a basal skull fracture, and the difficulty of blind nasotracheal intubation in children make orotracheal intubation the preferred approach. In general, a rapid sequence intubation technique, with the Sellick maneuver, is indicated to secure the airway in all traumatized children requiring endotracheal intubation (7). This may be contraindicated if a difficult airway is expected, as may be suggested with faciomaxillary trauma, mandibular fracture, or laryngeal trauma. In these cases, the ability to endoscopically intubate the airway and the availability of someone who can surgically secure the airway is indicated. In this setting, a laryngeal mask airway may be helpful. Evaluation of the airway and the presence of personnel expert in securing the airway are life saving when direct laryngoscopy and intubation fail.

For routine intubation and rapid-sequence induction, using a nondepolarizing muscle relaxant, such as vecuronium, rocuronium, or cisatracurium, is currently common practice (7). The use of succinylcholine in multiple trauma is best avoided in the presence of multiple long bone fractures and crush injuries. There is also some concern that succinylcholine elevates intracranial pressure in the presence of head trauma. For intubation, sedation and pain management should be provided. The use of a potent narcotic, such as fentanyl, combined with an intense hypnotic, such as propofol, etomidate, or ketamine (in the non–head injured, but hypotensive patient), is adequate. The use of sedation in hypovolemic, hypotensive patients is fraught with risk, and adequate fluid resuscitation is mandatory before consideration of vasopressor agents. In the apneic, obtunded child or in a child with cardiorespiratory arrest, the need for urgency may mandate intubation without sedation or analgesia. In this setting, as the child recovers, pain management will be necessary.

The potential for spine instability and for worsening spinal injury during airway manipulations is an ever-present concern in managing the airway of traumatized children. Cervical injury must always be suspected, and cervical immobilization is a routine first step in pediatric trauma management. It can be expected that the child will arrive from the field with a collar, on a backboard, immobilized, and should be receiving 100% oxygen by nonrebreathing face mask. Intubation should be performed with the head in the midline and immobilized, avoiding flexion and extension of the neck. The neck should remain stabilized after intubation. A normal radiograph does not preclude the need for immobilization and cervical stability during airway manipulation due to the frequency of spinal cord injury without obvious radiologic abnormality (8). Vigorous in-line traction may be deleterious in children and contribute to further dislocation. Avoiding rotation, flexion and extension (i.e., immobility, not traction) is the goal.

In older children, the preferred approach for a tracheostomy is a cricothyroidotomy (9). This is dictated by the child's deeper location of airway and the likelihood of vascular or thyroid injury during an emergency tracheostomy. Although cricothyroidotomy is recommended in older patients for failed intubation, this can be difficult, even in adults, and in small children, it may prove impossible. In younger children <8 yrs of age, a formal cricothyroidotomy may be difficult, and a needle or cannula for puncturing the cricothyroid membrane and use of a Seldinger-type technique for quick cricothyroidotomy is an alternative. Obviously, without prospective attention to airway management and assurance that all critical supplies and equipment are readily available before the child arrives in the hospital, successful resuscitation is unlikely.

Note should be made of the presence of crepitus, indicating subcutaneous emphysema, which may be related to a fractured larynx. The most important signs of laryngeal injury are hoarseness and subcutaneous emphysema (9). This may significantly complicate endotracheal intubation and generally indicates the need for otolaryngological consultation, although it is more commonly related to trauma associated with pneumomediastinum and pneumothorax. Tracheobronchial disruption is less common in children due to the bronchial tree's greater elasticity. If airway (laryngeal, tracheal, or bronchial) disruption is suspected, awake fiberoptic endoscopy, while maintaining bag and mask or spontaneous ventilation, is indicated (7). A CT scan may be helpful. In the case of laryngeal or tracheal disruption, cricothyroidotomy is probably best avoided because it may aggravate laryngeal injury. If possible, endoscopy is best performed in the operating room where full preparations for airway instrumentation are made. Emergency transfer to the operating room for fiberoptic intubation or emergency tracheostomy is indicated. It is worth noting that air leaks in the tracheobronchial tree generally may be managed conservatively with chest tubes.

Assurance of breathing, with assessment of respiratory mechanics, oxygenation status, and maintenance of adequate ventilation, is critical. This may require assistance with bag/valve/mask or mechanical ventilation. In addition, urgent intervention may be needed to correct pneumothorax, hemothorax, open chest wound, or flail chest.

Assessment of the circulation completes the primary survey, with evaluation of pulse, perfusion, and blood pressure. Particular attention is directed to peripheral perfusion. In children, blood pressure is maintained until hypovolemia is quite severe (>25–50% of blood volume loss in children). Vascular access is obtained and resuscitative fluids are given (isotonic crystalloid, without dextrose) in boluses of 20 mL/kg, guided by perfusion, heart rate, urine output, mentation, and blood pressure. If necessary, blood may be administered (10 mL/kg per bolus) in the case of failure to improve with crystalloid resuscitation of >50 mL/kg in an hour. The primary survey should be conducted rapidly and interventions instituted as problems are identified. During this stage of the resuscitation, the trauma team must maintain focused attention on the child's condition because changes may occur very rapidly and at any time. If at any time the child's condition deteriorates, the primary survey must be repeated with evaluation of the ABCs.

The secondary survey is conducted as soon as the injured child is stabilized with respect to the ABCs. During the secondary survey, a thorough head to toe evaluation of the entire child is conducted. Care must always be taken during this (and all) portions of the resuscitation to maintain protection of the spine. Spine precautions include the maintenance of full spine immobilization, even during manipulation and transport of the injured child. The secondary survey may be conducted rapidly but thoroughly, with a cooperative trauma team noting all positive findings to the recorder who will document all positive and pertinent negative findings. During this phase, a neurologic assessment for disability is conducted and must be carefully documented to ensure accurate, longitudinal comparison.

With the completion of the secondary survey, the team leader can develop a plan for further diagnostic studies. In most institutions, radiographs of the chest, pelvis, and lateral cervical spine are conducted routinely. Further evaluation is based on positive findings or suspected injury. The diagnostic studies must be prioritized on the basis of threat to life first, then to limitation of disability. The basic principles of maintaining the ABCs guide the prioritization of diagnostic evaluation. Any injury that poses a threat to the airway is investigated first (this may be facial trauma, blunt or penetrating neck injury, etc.). Injuries jeopardizing breathing are investigated next (chest injury). Next, injuries jeopardizing circulation are investigated. This may include evaluation of the abdomen for major vascular or solid viscus injuries. In the child without a reliable exam (based on age or altered mental status) or with positive findings (abdominal distention or tenderness), evaluation of the abdomen can be completed using computed tomography, or in some institutions, ultrasound has proven to be a quick, reliable screening tool (10).

Evaluation of disability proceeds next and includes assessment of the cause of altered mental status (usually using head computed tomography). This will also include evaluation of any peripheral neurologic findings (additional spine radiographs). Evaluation of extremity injuries must not be overlooked as fractures may result in significant blood loss, either externally or internally, especially with femur fractures. Special consideration must be given to the possibility of fracture, as this can be a source of great blood loss. Our current technology allows these diagnostic procedures to proceed very rapidly. The trauma team must be prospectively aware of the capabilities of their hospital to facilitate the most efficient timing of the necessary studies.

Priorities of care should be established to follow a similar pathway in the resuscitative phase. Interventions needed to preserve the airway take precedence over those that preserve breathing, circulation, and address the disabilities. Although these guidelines are quite obvious and seem simple enough to follow, multiply injured children create confusion, distress, and dilemma on a routine basis. Unfortunately, in the moment, this prioritization all too often breaks down. For instance, in a child with altered mental status, a compromised airway, and shock, a not infrequent situation, the trauma team must simultaneously make interventions (airway) and assessments. They must evaluate the cause of airway compromise, determine the cause of shock and determine the nature of altered mental status in a rapid, organized fashion if mortality and morbidity are to be prevented. During these steps, resuscitation of the injured child is begun, and a management plan is developed and implemented. The continuing challenge lies

ahead in the critical care setting where these children will require constant attention to detail.

PATHOPHYSIOLOGIC RESPONSE TO TRAUMA

Trauma is a systemic disease. In addition to the specific injury, whether fracture, contusion, solid viscus fracture, or head injury, there is a systemic response. The immediate response to trauma includes a broad-spectrum neuroendocrine stress response that has acute effects on cardiovascular function, intravascular volume, cardiovascular performance, and metabolism that may last for several days after the initiating injury. After this immediate response is a more prolonged inflammatory response causing a secondary endotheliopathy and aberrations in intravascular volume control, temperature regulation, hemostasis, and failure of multiple organ systems that is remarkably similar, if not identical, to the systemic inflammatory response syndrome (SIRS) (11).

The stimuli of the acute neuroendocrine stress response, which in multiple trauma often becomes maladaptive, include hypotension, hypovolemia, hypothermia, hypoxemia, and pain (12). Pain, and the psychological sequelae of trauma, are potent initiators of the neuroendocrine stress response (13). Tissue damage, hemorrhage, decreased intravascular volume, hypotension, and hypoxia exaggerate and augment this response. This stress response is characterized by massive catecholamine release, release of adrenocorticotropic hormone, antidiuretic hormone, endorphins, growth hormone, thyroid stimulating hormone, and prolactin. The combined effect is vasoconstriction, increased cardiac output, redistributed blood volume, increased oxygen consumption, increased minute ventilation, and increased catabolic rate with gluconeogenesis and glycogenolysis. The release of cortisol and stimulation of the renin-angiotensin system, along with catechols and vasopressin, support vascular volume and perfusion pressures and defend vital organ function. This response is finite and may be deficient. For example, vasopressin release may become insufficient. This recent understanding of vasopressin's role and the relative vasopressin deficiency in critically ill children suggests a promising new role for the use of vasopressin in refractory hypotension (14).

These adaptive neuro-endocrine responses are directed at maintaining intravascular volume and perfusion pressures while at the same time increasing metabolic substrate. For example, cortisol and catecholamines increase circulating glucose, free fatty acids, glycerol, and amino acids, thus stimulating gluconeogenesis. In addition, catechols cause pancreatic islet cell inhibition, decreased insulin levels, and insulin resistance. This results in the common finding, especially in younger children, of dramatic hyperglycemia until glucose stores are exhausted. Hyperglycemia in head-injured children seems to contribute to morbidity, is a marker of severity of injury, and correlates with outcome (15). Glucose exhaustion follows and occurs more rapidly in neonates, and as glucose stores are depleted, hypoglycemia may occur. Catechols stimulate lipolysis, ketogenesis, and protein catabolism. This triggers an acute metabolic catastrophe characterized by catabolism, negative nitrogen balance, and structural protein loss, and this may continue for days to weeks after trauma and surgery. This can lead to nutritional wasting, which is unresponsive to nutritional intervention for the first several days after multiple trauma (16). If prolonged, the systemic inflammatory response, multiple organ dysfunction syndrome, and ongoing stimulation by tissue trauma and infection continue to trigger this catabolic response. Nutritional catastrophe and immunosuppression can be expected (17). These normally adaptive responses to injury are very responsive, especially in young children, and in response to massive injury, they can be deleterious, impeding recovery and impairing survival.

Multiple trauma stimulates the release of multiple inflammatory mediators (18, 19). These include activation of the complement system, causing increased microvascular permeability, stimulated opsonization, and activation of inflammatory mediator cell types such as polymorphonuclear cells by C5a. The central role of the complement and coagulation cascades in multiple trauma has become increasingly understood in recent years. The initial response to trauma also includes release of both platelet activating factor and arachidonic acid from cell membrane triglycerides. Both are inflammatory mediators and cause a wide spectrum of inflammatory responses. Platelet activating factor causes hypotension, pulmonary vasoconstriction, bronchoconstriction, and increased capillary leak, leading to decreased cardiac output and hypoxemia. Arachidonic acid gives rise to cyclooxygenase products, including the prostaglandins such as prostaglandin E_2 and prostacyclin and the proaggregatory and vasoconstrictor thromboxanes. Arachidonate, via the lipoxygenase pathway, produces leukotrienes, which are potent bronchoconstrictors, vasoconstrictors, modulators of an increased capillary permeability, and chemoattractants, that potentiate leukocyte endothelial interaction, leuko-aggregation, and lysosomal enzyme toxic oxygen radical release and result in endothelial damage.

There are also a broad spectrum of cytokine-mediated responses that have been well delineated. Perhaps the best characterized is the release of tumor necrosis factor, which causes hypotension, tachycardia, acidosis, and fever (20). Tumor necrosis factor is procoagulatory, leading to coagulation abnormalities and increased capillary permeability. In addition, tumor necrosis factor mediates catabolic breakdown and long-range effects, resulting in multiple organ dysfunction syndrome. Tumor necrosis factor is also a potent stimulant of acute phase protein synthesis and endothelial activation. Interleukins also play a key role in the systemic response to multiple trauma. IL-1 stimulates polymorphonuclear release, leuko-adherence, selectin activation, endothelial activation, and lymphokine production. Alpha interleukin 2 leads to T-cell proliferation and further hypotension and enhances capillary leak. IL-6 plays a key role in cellular cytotoxicity and T-cell proliferation (21). IL-8 causes polymorphonuclear chemotaxis and leuko-adhesion and activates the leukocyte endothelial complex interactions. The endothelial dysfunction contributes to multiple organ dysfunction. The role of the immune response, cytokines, endothelial damage, and other systemic responses to multiple trauma that effect cellular signal transduction and crucial oxygen responses are as yet incompletely understood. Nevertheless, it is clear that this complex and chaotic systemic response to trauma critically affects outcome (22).

This very brief overview of the systemic inflammatory response to trauma is necessary to understand the complex systemic results of multiple trauma and underlies the newer approaches, both surgical and medical, to the multiple trauma victim. The presence of systemic inflammatory response, as indicated by a SIRS score, is associated with worsened

outcome, prolonged length of stay, and an increased relative risk of death in multiple trauma patients (22, 23). Of course, whether this reflects more severe injury with a greater response or is in some way causative, remains to be delineated. Nevertheless, a high SIRS score (≥2) is an independent predictor of outcome of multiple or blunt trauma (22, 24). Understanding the impact of the systemic response and the presence of multiple organ system failure in all multiple trauma patients, the catabolic disaster, the coagulation abnormalities, and the ongoing inflammatory response underlie the current medical approach to multiple trauma (24). Recent approaches to minimize the systemic response have altered the surgical approach to the multiple trauma victim and have given rise to the concept of damage-control surgery (25).

TRIAD OF DEATH

All clinicians familiar with critically injured multiple trauma victims recognize that the combination of a cool patient, bleeding from multiple sites, and metabolic acidosis represents an extremely serious state. Cool tissues, hypoperfusion, acidosis, and coagulopathy, even with an adequate blood pressure and heart rate, indicate critical tissue hypoperfusion. This lethal triad of hypothermia, acidosis, and coagulopathy is the direct result of trauma and secondary injury from the systemic response to trauma (26, 27) (Fig. 1).

Clinicians recognize that metabolic acidosis is an ominous sign (28). Acidosis, *per se*, is not harmful; however, it is a reliable indicator of tissue hypoxia. This may be due either to ischemia and necrosis from direct tissue injury or hemorrhage, hypotension, and hypoperfusion or the systemic inflammatory response with hypoperfusion. Impairment of the microcirculation by hypovolemia, direct damage, disseminated coagulopathy, intravascular sludging, and endothelial injury worsens perfusion. Tissue hypoxia leads to anaerobic metabolism and lactic acid synthesis. Lactic acid synthesis is potentiated by the abundant supply of glucose and energy substrates as a result of trauma-induced catabolism and may be aggravated by hepatic impairment as part of the secondary injury of trauma. Continuing lactic acidosis is a grim prognostic indicator and survival rates that are near 100% with clearance of lactic acid within the first several hours, fall to <15% when lactic acid is not cleared after 48 hrs (29). Ongoing acidosis after volume resuscitation and blood pressure restoration is a grim prognostic sign. In children, the probability of mortality increases dramatically with an admission base deficit >8 mEq/L (28). Profound tissue injury, cell death, or as yet untreated hypoperfusion persist and indicate a grim prognosis.

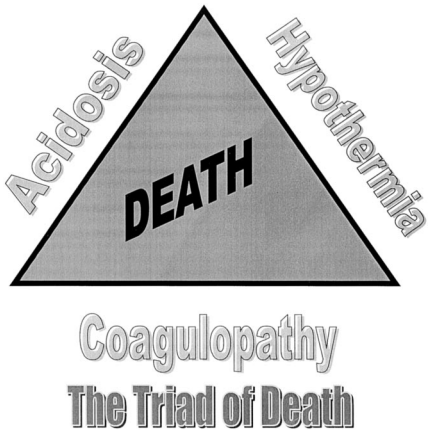

Figure 1. Triad of death.

Hypothermia is a well-known threat to physiologic integrity in children. Every pediatrician knows the multiple derangements initiated by hypothermia in neonates. Over the last several years, it has become clear that hypothermia is also a physiologic homeostatic threat in adults and older children. In addition to the obvious environmental risk factors, the pathophysiologic consequences of severe injury inevitably lead to hypothermia (29). This secondary hypothermia complicates the management and worsens the survival of the multiply injured child. Hypothermia can occur in the field and subsequently in the hospital due to exposure, transfusion with cold fluids, impaired thermogenesis, and additional heat lost to the environment. This is more likely in children due to their high surface area–to–body mass ratio. The consequences of this secondary hypothermia are legion. They include cardiac dysrhythmias, decreased cardiac output, aggravated coagulopathy, and a left shift in the oxyhemoglobin dissociation curve with worsening acidosis. All result in increased morbidity and mortality. Temperatures of <34.5°C are associated with an increased prevalence of multiple organ dysfunction system and increased need for vasopressor and inotropic support. Increased crystalloid infusion is required. Apparent worsening of the endotheliopathy and interstitial fluid leak also occurs with hypothermia. At temperatures below 32°C, mortality approaches 100% in some series (30). Hypothermia is both a marker of profound injury and is itself deleterious.

Several aspects of blood clotting mechanisms are deranged in the cold, acidotic, exsanguinating patient (30). One of the main determining factors of coagulation cascade is body temperature. The temperature-sensitive, serine-dependent esterase reactions are inhibited by hypothermia (31). Thus, it is not surprising that the final part of the lethal triad of acidosis and hypothermia is coagulopathy. In addition, the systemic inflammatory response initiates multiple mediators included in the complement and coagulation cascades and leads to an ongoing coagulopathic situation (32). Hemodilution due to blood loss with lost coagulation factors also aggravates the situation. Platelet function is also depressed by hypothermia. Recently, it has become clear that one of the key factors in SIRS is endothelial injury precipitating aberrant coagulation. This suggests that protein C may have a role in interrupting the SIRS-related coagulopathy cycle (33). The endothelial impairment secondary to trauma, and the interaction with the coagulation cascade and platelet activation, alters the fibrinolytic system, leads to reduction in antithrombin III and fibrinogen levels, and enhanced fibrinolysis. Hypercoagulability is an early stage, with decreased endogenous fibrinolytic activity and decreased antithrombin III. This is rapidly followed by a hyperactive and dysfunctional fibrinolytic system. In the multiple trauma setting, bleeding from puncture wounds and multiple injuries further aggravates hypovolemia and hypotension. The ongoing need for resuscitation and transfusion further aggravates the coagulopathic status and may contribute to hypothermia (30).

This lethal triad causes continuing deterioration and a lethal downward spiral (30, 34, 35). Poor perfusion and thermogenic impairment, coupled with attempts to surgically stop the bleeding and repair the injury, lead to further deterioration in thermoregulation. Hypothermia and ongoing blood loss, coupled with the systemic inflammatory response, worsen the coagulopathy, feeding the cycle of volume loss and hypotension, demanding further transfusion and volume expansion. Tissue perfusion is not restored, worsening the acidosis along with metabolic impairment secondary to the neu-

roendocrine stress response to multiple trauma. This situation can only grow worse. It is hardly surprising that ongoing attempts at resuscitation and operative repair in a child who is hypothermic, acidotic, and coagulopathic is seldom successful. If this vicious cycle is not interrupted, death ensues and is often attributed to irreparable surgical injuries.

DAMAGE CONTROL APPROACH

Recognition of the lethal triad and the futility of continuing to repair massive injuries and perform definitive surgical correction in the presence of hypothermia, acidosis, and coagulopathy has led to the concept of damage control surgery (25, 34). This approach describes a systematic three-stage approach to the multiply injured child. The first stage is a direct surgical approach that is targeted at true damage control. It consists of focused surgery to control hemorrhage and alleviate contamination, such as fecal spillage from damaged bowel. Definitive reconstruction is delayed. Rapid, simple closure is encouraged. In some cases, abdominal packing and closure of the wound with loose retention sutures is indicated. Fractures are immobilized, not definitively reduced or set. The goal of the first stage is to prevent ongoing damage as rapidly as possible (35, 36) (Figure 2).

In the second phase of the damage-control approach, the child is taken to the intensive care unit for further resuscitation and stabilization. The goals of this resuscitation are rewarming, correcting the coagulopathy, and reversing the acidosis (Fig. 2). Complete ventilatory support removes the burden of ventilation and ensures oxygenation by minimizing intrapulmonary shunting and optimizing ventilation-perfusion matching. Hemodynamic support with volume resuscitation and the judicious use of inotropic and vasopressor agents to restore perfusion and decrease the stimuli to the neuro-endocrine stress response is necessary. Although targeting supraphysiologic goals (high cardiac output, the use of vasodilators to enhance perfusion, and aggressive support of oxygen delivery) has been reported occasionally as beneficial in adults with trauma, postoperatively and with sepsis, there are no data supporting this approach in children (37, 38). Maintaining urine output, perfusion pressures, oxygen delivery sufficient to clear acidosis, and clearance of lactate are adequate end points. Targeting high filling pressures, such as a central venous pressure of >8 cm H_2O, makes little sense in children. In addition, children suffer from size and technological limitations in the available physiologic monitoring devices. In the presence of a healthy compliant heart, central venous pressure is a poor indicator of intravascular volume and a central venous pressure of >10 cm H_2O indicates myocardial impairment, high intrathoracic pressure, or pulmonary vascular disease and should suggest, in the setting of trauma, myocardial contusion. Excessive volume expansion to reach an arbitrary central venous pressure will worsen edema and the abdominal compartment syndrome and, in the setting of head injury, worsen cerebral edema and increase intracranial pressure. Volume expansion is best guided by considering perfusion, urine output, blood pressure, and heart rate in conjunction with the filling pressures and, where necessary and possible, measurement of cardiac output using a pulmonary artery catheter. Despite great discussion and study, there remains no clearcut indication for colloids rather than crystalloids in resuscitating children with multiple trauma (9, 39, 40). During this phase, concurrent further tertiary survey and injury identification should be ongoing. This second, critical care stage of damage control may be quite protracted and complicated.

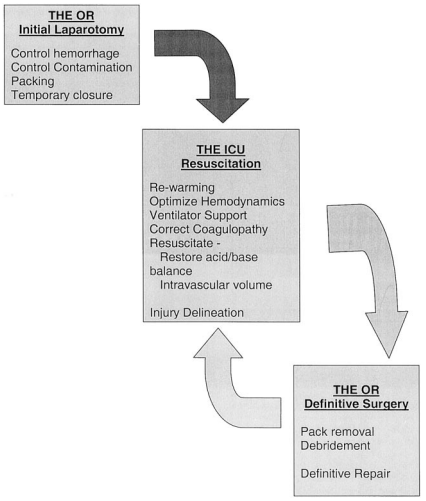

Figure 2. Damage control approach. *OR*, operating room; *ICU*, intensive care unit.

ABDOMINAL COMPARTMENT SYNDROME

One particular challenge during the second stage of the damage control approach is the abdominal compartment syndrome (41). Edema, ascites, tissue swelling, and ongoing bleeding may lead to increased intraabdominal pressure (30, 42). This elevated pressure may have significant cardiorespiratory effects (43). First, infradiaphragmatic pressure with upward displacement of the diaphragm may cause basal atelectasis and a restrictive lung defect. Quite high airway pressures may be required to maintain ventilation and positive end-expiratory pressure may be necessary to maintain lung volumes and oxygenation. Positive end-expiratory pressure titration should focus on both oxygenation and cardiac output to maintain both perfusion pressures and oxygen delivery. Ventilation may be quite difficult, and a permissive hypercapnia strategy may be indicated. Increased intraabdominal pressure can lead to decreased perfusion of the abdominal contents. Most critically, renal perfusion pressure may be impaired with oliguria, anuria, and resulting secondary renal injury resulting. Renal (and other intraabdominal perfusion pressures) may be considered in a similar fashion to lung or cerebral perfusion pressures. When intraabdominal pressure becomes greater than venous pressure, it becomes the downstream pressure for perfusion. Renal perfusion pressure now equals: mean arterial pressure − intraabdominal pressure.

If renal perfusion pressure decreases below about 50 mm Hg, urine output will fall and renal damage may occur. Clearly in a hypotensive, hypovolemic patient, intraabdominal pressures of 20 or 30 mm Hg may be significant. A similar scenario applies to the gut. Splanchnic hypoperfusion due to abdominal compartment syndrome may lead to gut injury and result in gut bacterial translocation and worsening of SIRS and frank bacterial sepsis. Intraabdominal pressures of >25 mm Hg have been associated with elevated intracranial pressure. Aggravating the perfusion pressure issue is the ability of increased abdominal pressure to decrease venous return and thus cardiac output, further worsening both systemic and local hypoperfusion. Intraabdominal pressure can be measured by transducing an indwelling urinary bladder catheter. Alleviation of increased intraabdominal pres-

sure may require abdominal paracentesis with drainage of ascites or opening of the abdominal wound and possibly the use of a prosthetic silo, similar to that used in neonatal omphalocele repair (44, 45).

When the child is stabilized and further stimuli to the inflammatory response are controlled, definitive surgery is indicated (Fig. 2). In this third stage of the damage control approach, packs are removed, tissues are debrided, and repair can proceed in a controlled fashion, avoiding the lethal triad. Definitive repair and fracture reduction can occur.

This relatively new damage-control approach is antithetical to the former approach of definitive surgery at the time of injury with specific control and complete correction of underlying abnormalities (34). This staged approach results in multiple surgeries; however, it is specifically designed to allow the medical resuscitation of the multiply injured patient who is critically ill, thus avoiding the perpetuation of the lethal triad. This surgically conservative but medically aggressive approach places extra strain on the intensivist. During this initial resuscitative phase in the intensive care unit, further ongoing systemic damage, volume impairment, systemic inflammatory response, multiple organ damage, and acute lung injury occurs. Controlling and responding to these secondary pathophysiologic sequelae of trauma becomes the cornerstone of trauma management in the multiply injured, critically ill child. Meticulous ventilatory care, correction of coagulopathy, specific attention directed at rewarming with warm fluids and convective air heating devices, fluid resuscitation, and prevention of secondary injury become a focus in trauma management. This second phase may last from hours to days after the initial injury.

Of course, not all traumatized children require damage control surgery and may benefit from early definitive repair. Experienced surgical judgment is required to determine who would best benefit from this approach. In children, damage control surgery is not so often indicated due to the already well-established conservative surgical management of the fractured abdominal viscus such as a liver or spleen. The selection of children for damage-control surgery depends on the severity of injury. As a general guideline, children who arrive from the field already hypothermic ($<35°C$) or acidemic (pH of <7.15 units) and already with a coagulopathy certainly deserve consideration of this approach. In addition, children who move into the lethal triangle intraoperatively and cannot maintain blood pH above 7.2, prothrombin time/partial thromboplastin time corrected to <1.5 times normal or their core temperature above 35°C, are candidates for damage control surgery (46). Simple control of hemorrhage, decontamination, closure, or packing should proceed with alacrity. Children who are already tachycardic and require airway intubation or who are hypotensive are also with apparent risk. The type of injury is also important. High-energy blunt torso injuries, such as motor vehicle accidents and multiple penetrating injuries, respond well to the damage-control approach. Lap-belt injuries have been reported to benefit from this approach in children (47). Major abdominal vascular injuries and multiple visceral injuries may be considered for this approach. Multiple cavity bleeding with concomitant visceral injuries is also a complex of injuries that benefit from damage-control surgery (48).

Finally, when competing priorities exist, such as in compound limb fractures and head injury, this approach may also be beneficial (49). Critical factors may indicate the need for this approach. When surgery and resuscitation take longer than 90 mins or a coagulopathy develops, or when greater than two blood volumes have been transfused, rapid control of hemorrhage, decontamination, packing and temporary closure, avoiding definitive surgery, and facilitating rapid transfer to the intensive care unit is indicated. Obviously, wise surgical judgment is required for the judicious application of definitive repair and the decision to proceed with the damage-control approach. The decision must frequently be made rapidly but need not be made without consultation among treating physicians. Treatment should not proceed without discussion of the critical care and surgical issues.

RESPIRATORY COMPLICATIONS OF MULTIPLE TRAUMA

Intrinsic to the concept of damage-control surgery is critical care and respiratory management. Clearly, the most significant target organs of secondary damage are the lungs and kidneys. The respiratory complications of multiple trauma arise from both direct chest injury and injury secondary to SIRS (50). Direct chest trauma, with pulmonary contusion, pneumothorax, major vascular disruption, or myocardial contusion, has been discussed elsewhere in this issue. Secondary injury occurs because the lung is the main target organ of SIRS leading to acute lung injury (formerly acute respiratory dysfunction syndrome) in children with multiple trauma. Endothelial injury with impaired pulmonary vascular responses, decreased nitric oxide production, leukotriene release, and pulmonary vasoconstriction, accompanied by enhanced capillary leak and pulmonary interstitial edema, are common in all injured patients. The severity of the generalized systemic response determines the severity of the lung injury. In addition, morbidities such as aspiration pneumonia, atelectasis, and secondary infection complicate the SIRS-induced endothelial injury during the management of the multiply traumatized child. The decreased pulmonary compliance, decreased end-expiratory lung volume below functional residual and closing capacities, increased airway resistance, and increased intrapulmonary shunting all pose a serious threat to ventilation/perfusion matching and require meticulous respiratory management. Although positive end-expiratory pressure improves ventilation/perfusing matching, no amount of airway pressure will prevent the formation of interstitial and alveolar edema. Indeed, the evidence is clear that positive end-expiratory pressure, in and of itself, worsens extravascular lung water. Thus, prophylaxis with positive end-expiratory pressure is futile. Ventilator therapy tailored to the child's specific condition is necessary. Judicious use of oxygen to prevent secondary oxygen toxicity (F_{IO_2} of $<60\%$) and the provision of adequate positive airway pressure to minimize ventilation/perfusion mismatch, although not impairing cardiovascular function, is the goal of respiratory management in multiple trauma. Antibiotics do not decrease complications from aspiration pneumonia or the prevalence of pneumonia in multiply traumatized patients (51). Indeed, polypharmacy with too many antibiotics is associated with impaired outcome.

In general, negative intrathoracic pressure resulting from spontaneous ventilation imposes cardiac afterload. In children with pulmonary and myocardial contusions or impaired cardiac function, imposing further myocardial afterload should be avoided until the child is stable. Thus, spontaneous ventilation is best

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minimized (pressure support breathing) or avoided (controlled ventilation). This will also remove caloric expenditure from the work of breathing with impaired lung function and help optimize the oxygen delivery/demand balance in critically ill children. Despite years of discussion and multiple therapy trials, decreasing pulmonary interstitial and alveolar edema remains a difficult challenge. Fluid restriction can be a hemodynamic catastrophe and worsen the stress response/SIRS cycle. No clear advantage has been demonstrated for albumin, colloids, or hypertonic solutions in this setting. Supportive therapy directed at removing the stimuli to SIRS while supporting oxygen delivery is indicated.

MANAGING THE FAMILY

Injured children have families. The future well-being of the child depends on the family's, and the child's, outcome from the traumatic episode. Even when the injury is not the result of nonaccidental injury, guilt, anxiety, fear, blame, and accusations abound. The caretaker of the child at the time of the injury is particularly at risk. Initiating supportive care for the family at the same time as medical treatment for the child is important. This may be particularly difficult if the mechanism of injury is unclear. Often the suspicion of nonaccidental injury can cloud the family management in the early hours of trauma, and the necessity to report suspicion of nonaccidental injury and involvement of law enforcement agencies early on further complicate family management.

Education and openly answering the family's questions, with an "I do not know" when it is true, are helpful in establishing rapport and confidence. Looking after the family's spiritual needs, contacting clergy and family support, and involving social work support may maintain crucial family support for the critically ill child in the early stages and provide insights that may enable long-range family support to be successful. It is unfortunately true that family breakdown is a common sequelae of the death or severe injury of a child. Family counseling should start early and may be required for years after a significant or fatal injury. Integrating the family into a caring and supportive environment with friends, family, church, or other support structure is important. The goal of early family discussions should be honesty and openness with a hopeful, but realistic, prognosis as appropriate.

CESSATION OF CARE

Resuscitation of the critically injured child to full recovery is the constant goal of the well-organized trauma team. Although traumatic full arrest is not common in children, it is certainly an unavoidable aspect of providing care for severely injured children. It must be recognized that, despite the most heroic efforts, there will be some patients who are so severely injured, or have suffered arrest for a great enough period of time as to render the likelihood of the restoration of any measure of recovery impossible. With this is mind, it is recognized that some severely injured children will die of their injuries.

Although the resuscitation of the most severely injured children is always a highly emotional experience for the entire trauma team, one must consider that when a child is not salvageable, the most beneficial course of action may not always require the most dramatic interventions. The trauma team leader must keep in mind that emotions must be controlled, and the most dignified outcome for the patient, family and caregivers, and indeed, society at large should be pursued. In the case of severe brain injury or brain death, the trauma team should consider the possibility of organ donation, and if possible, the organ procurement personnel may wish to approach the family.

The consideration of cessation of care in the trauma room is only appropriate for the child with no signs of life. All other children should be managed according to institutional policy and in accordance with the American College of Surgeons Advanced Trauma Life Support protocols. In considering policies regarding the cessation of care in the severely injured child with no signs of life, a multidisciplinary institutional committee is necessary to review and clarify the patient group under discussion and when and how these protocols will be implemented. A well-considered and organized approach in developing these policies can alleviate the burden of anxiety that the highly motivated pediatric trauma personnel inevitably feel when faced with the decision to stop, or not to start, a full resuscitation.

At Childrens Hospital Los Angeles, we have organized an institutional policy regarding the resuscitation of children without vital signs (pulse, respiration, blood pressure), absent cardiac electrical activity, and Glasgow Coma Scale score of 3 on arrival to the emergency department. We have further divided these patients into groups based on the mechanism of injury (blunt and penetrating). It is important to note that the final decision about cessation of care rests in the hands of the trauma team leader, preferably with open discussion among the trauma team and with the family when appropriate and in an appropriate and compassionate manner.

Patients without signs of life in the field and without signs of life confirmed in the emergency department represent the most severely injured patients. The possibility of recovery is absent. In these patients, the resuscitation may be discontinued and they may be declared dead in the trauma room. In these patients, it is not appropriate to perform thoracotomy and open cardiac massage, nor is it appropriate to give resuscitative medications (52). In the event that a patient reported to have no signs of life in the field is found to have signs of life in the emergency department, then all resuscitative measures are instituted.

Patients with signs of life in the field but with no signs of life found at arrival in the emergency department should be treated according to the mechanism of injury. Blunt trauma victims are resuscitated according to standard protocols with airway and ventilation management (to include intubation if needed), chest

compressions, intravenous access, and fluid resuscitation. If these patients should fail to show any signs of life, there is no precedent for survival with emergent thoracotomy (53). Penetrating trauma patients who lose signs of life en route to the emergency department should be resuscitated in similar fashion. With this mechanism of injury, emergency department thoracotomy may offer some benefit, however small (54). Children experiencing full arrest while in the emergency department should be resuscitated according to institutional policies and Advanced Trauma Life Support protocols, including the consideration and implementation of all surgical options.

With a multidisciplinary development of an organized approach to this most severely injured group of children, the trauma team can accurately and efficiently make decisions about the appropriate course of action in the management of these children. In addition, prospective discussion about the appropriate course of action in these situations exposes the dismal outcome and allows some alleviation of guilt, anxiety, and the burden of the highly emotional experience of witnessing the death of a child.

QUALITY ASSURANCE AND IMPROVEMENT

The quality of trauma care has been constantly improving since the institution of organized trauma care and regular review of performance. Quality improvement in the care of the trauma patient is an essential part of any successful trauma program. All trauma-related deaths should be reviewed, and all resuscitations that fall outside of normal boundaries should also be reviewed (55). Most regional trauma organizations have developed a trauma registry administered either jointly by cooperative centers or by governmental bodies (56). These registries create filters that review all cases for outcome based on predicted survival. Any deviation from expected outcome or process (e.g., transport time, time spent in emergency department, time waiting for an operating room) must be addressed, understood, and corrected when possible. If a failure is identified, the problem must be identified, and corrective action must be taken and documented. Through these processes, systematic problems and individual failures may be identified and improved or corrected (57, 58, 59). Continuous improvement in the quality of care occurs. It is important to note that all aspects of prehospital and in-hospital care should be evaluated in this process. The intention should not be punitive, but educational. Correction of those system aspects of care that can be improved is crucial to effective and improving care. If trauma care is to reach the highest level of efficiency and to decrease the amount of suffering for the injured child and his or her family, then a regular, objective review of all aspects of the trauma team's performance must be included in the trauma program (60).

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