The Multiply Injured Child

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Abstract

Multiple trauma (MT) is an injury to more than one body system or at least two serious injuries to one body system. In the developed world, trauma is the leading cause of death and acquired disability in the 1–45 years age groups with staggering burden of medical and societal costs. Moreover, more than 95 % of pediatric injury deaths occur in the developing world, where the magnitude of trauma toll is increasing with the trends of expanding urbanization and motorization.

MT is a "systemic" disease, and is best approached according to the "two-hits hypothesis": The initial injury causes organ and tissue damage (first hits), that activate the neuroendocrine and metabolic stress response and the systemic inflammatory response (SIRS), causing 'second hits' such as respiratory distress syndrome, reperfusion injury, compartment syndromes and infections. Exogenous 'second hits' include surgical interventions, hypothermia, massive transfusions, inadequate or delayed surgical or intensive care interventions and line infections. Thus, MT increases the probability of secondary damage – especially to the brain. MT complicates the clinical course and the patient's management, makes clinical decision making far more complicated and requires different priority setting.

Management issues discussed in this chapter include pertinent aspects of pre-hospital, emergency room and intensive care evaluation and treatment, imaging of the multiply injured child, the pivotal role of the intensivist in the ICU care, approach to the bleeding patient with hypothermia and acidosis ("Triad of Death"), the damage control paradigm, and management of the multiply injured child with abdominal and chest trauma.

Keywords

Multiple trauma • Polytrauma • Children • Triad of death • Damage control surgery • Abdominal trauma • Chest trauma

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Introduction

Trauma is defined as a body injury or wound produced by physical force or energy, whether mechanical, chemical, thermal or electrical. It can be caused by accident (unintentional) or by violence (intentional) – including, in children, child abuse. Definition of Multiple trauma (MT) or Polytrauma is rather controversial [1]. Still, MT may be defined as injury to more than one body system or at least two serious injuries to one body system – such as multiple lower extremity fractures [1].

More children and young adults die from trauma than from all other diseases combined [2]. Moreover, the magnitude of this so called "neglected disease", in terms of acute morbidity and chronic disability, societal costs of direct medical and rehabilitation expenses, lost productivity by care providers and loss of years of potential productive life, let alone the immeasurable psychological burden, is staggering.

According to the trimodal model of the temporal course of trauma-related deaths, very early (immediate) deaths occur within minutes of the event and are practically unavoidable. The second, highest mortality peak occurs within the first 24 h, often within the first "platinum half hour" or the "golden hour", and these patients may benefit from aggressive, efficient and organized emergency medical services (EMSs) and hospital emergency departments (EDs) [3]. The third mortality peak occurs beyond the first 24 h as a consequence of the combination of the primary injury, secondary damage and the pathophysiologic processes initiated by them. These children usually die in the pediatric intensive care unit (PICU), and will be the focus of this chapter.

Trauma Related Mortality and Morbidity

In the developed world, trauma continues to be the leading cause of death and acquired disability among children (beyond their first year of life) and adults up to the age of 45 [2, 4, 5]. In the US, more children and adolescents die from injuries (including suicide) than from all other diseases combined [2]. Back in 1966, a special report by the American National Academy of Sciences defined trauma as the "neglected disease of modern society" [6]. Since then, significant advances in prevention, pre-hospital care and transport systems, emergency and hospital care – including intensive care – and subsequent rehabilitation, have resulted in substantial reductions in mortality, residual morbidity and disability. According to the Center for Disease Control (CDC) data, between 1981 and 1998, mortality rates due to unintentional trauma decreased by about 41 % in the 1–19 years age groups [5]. Between 1981 and 2007 crude mortality due to unintentional trauma decreased in the 0–14 years age groups by 43.2 % [5].

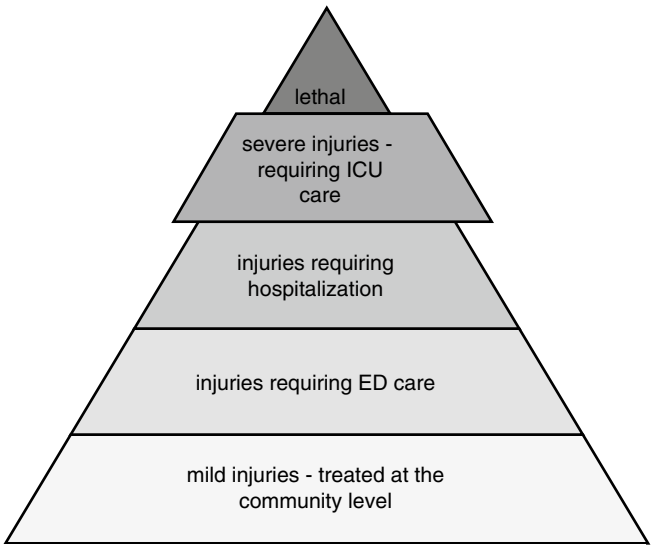

Fig. 32.1 Pyramid of injuries with added level for severe injuries requiring admission to an ICU

These numbers, however, grossly under-represent the true world-wide magnitude of trauma related toll: According to a recent World Health Organization (WHO) report [7, 8], more than 95 % of injury deaths occur in children and adolescents in the developing world, accounting for nearly one million deaths annually. Despite a full order of magnitude lower vehicle ownership rates, 95 % of road traffic crash deaths of children and adolescents occur in the low- and middle-income countries [7]. The magnitude of road traffic injuries is expected to further increase with the trends of increasing urbanization and motorization in the developing world: India and China alone are expected to see by 2020 an increase in the number of road-traffic deaths by 147 and 97 %, respectively [7].

Parallel to continuous decreases in the magnitude of other "traditional" causes of death among the young age groups, the relative importance of trauma as a worldwide health problem is therefore increasing. Based on the WHO database, Viner et al. analyzed global mortality trends for people aged 1–24 years across the past 50 years in low-, middle- and high-income countries [9]. Mortality in children aged 1–9 years declined by 80–93 % in this 50 years period, largely due to steep declines in mortality from communicable diseases. However, improvements in mortality in young people aged 15–24 years were only half those seen in children, largely due to static or rising injury-related deaths. In the UK, mortality in the 15–24 years age group has exceeded that of children aged 1–4 years since the 1970s due to increasing mortality from transport injuries, suicide and homicide [10].

Most statistics relate to trauma associated mortality, as it is the most convenient parameter to record. However, mortality represents only the tip of the "pyramid of injuries" (Fig. 32.1) that stratifies injuries according to severity and the level of medical attention they require. The basis of the pyramid consists of mild injuries treated at the community level; above it

are injuries requiring ED care, then injuries requiring hospitalization and the tip consists of lethal injuries. Clearly, a new level should be added to this traditional pyramid, namely severe injuries requiring admission to an ICU, as these obviously differ in every aspect from injuries occupying the "admission to hospital" level. In general, the available epidemiological data relate to the overall magnitude of injuries. No 'PICU specific' epidemiological and clinical data are available.

Age Distribution

Road accidents are the most frequent cause of mortality among children older than 1 year, and falls from height are the most frequent cause of injuries requiring hospitalization [11]. There are no major differences in the overall incidence of childhood injuries by age, though in the developed world the incidence increases with age, with the highest incidence in the teen-age group [12–14]. Overall age distributions by specific mechanisms of injury show distinct patterns: Injuries due to falls occur predominantly in the 1–4 years age group [12, 15, 16], pedestrian and bicyclists injuries peak in the 6–14 years age group [12, 13] and car occupant and sport and leisure-associated injuries peak in the 15–19 years age group [12, 13].

Injury Patterns and Mechanisms of Injury

The effects of injury on a child are related to the amount of delivered kinetic energy (1/2 mV^2, where m = mass, V = relative velocity). With increased body surface area to volume ratio, the delivered kinetic energy is compacted to a smaller volume and multiple organ involvement is common. Hence, injured small children are at high risk for mortality and morbidity because of their small body size and because of their limited physiologic reserves. Moreover, EMS and ED teams are relatively less trained in their acute care – especially in airway management and in obtaining vascular access. Therefore, a small body weight, especially <10 kg, receives a distinct unfavorable grade in the Pediatric Trauma Score (PTS) [17].

Similar mechanisms of trauma result in different injuries in adults and children. Because of their relatively large and heavy heads, falling children tend to land on their heads – accounting for the very high incidence of traumatic brain injury (TBI) among children admitted to hospital and PICU following falls. Among 188 children admitted to our PICU following falls from height, 92 % suffered TBI, 20 % had facial injuries, 18 % chest injuries and only ten had skeletal injuries.

The increased elasticity of the immature bones results in fewer fractures but in more soft tissue injuries. Thoracic trauma is generally associated with a high transmitted kinetic energy and therefore with high mortality rates [18, 19]. In children it results in injuries to the mediastinum and lungs but only rarely in rib fractures due to their elasticity.

The clinician taking care of the severely injured child should pay careful attention to the injury mechanism, as this determines to a great extent the "quality" and "quantity" of the resultant injuries. Unfortunately, information regarding the actual circumstances or mechanism of injury is often unknown or inaccurate – especially in the early management phase. For example, the relative speed in motor vehicle crashes determines crash severity and influences injury severity [20], yet it is unknown to the clinician taking care of the injured child in the ED or later in the ICU. As a "rule of thumb", an automobile crash in which other occupants have suffered lethal injuries carries high risk for very severe injuries to other occupants as it usually involves very high impact energy. Children who are thrown out of a crashed vehicle often suffer very severe injuries because of the absorbed energy as their body impacts with a solid surface. As mentioned, rib fractures in children are a "red flag" as they often signify severe mechanism of trauma.

Several distinct injury patterns are associated with specific injury mechanisms. Pedestrian – motor vehicle crash often results in the Waddell's triad of injuries to the lower extremities and/or pelvis, torso and head [21] – although this association has been questioned [22]. Unrestrained car occupants often suffer head, face and neck injuries as their head hits the dashboard or the windshield. Restrained children – especially when using lap belt – may present with the "seatbelt syndrome", consisting of intraabdominal injuries (gastric or bowel contusions or ruptures and/or injuries to solid organs) and of Chance fractures of the lumbar or cervical vertebra [23, 24]. With the widespread use of helmets, resulting in reduced incidence of TBI, the major severe injuries among bicyclists has become abdominal trauma – including deep organs such as pancreas or duodenum, as a result of impact by the handlebar [25].

Incidence of Multiple Trauma and Trauma Scoring

Multiple injuries – as opposed to a single injury – have far reaching clinical implications: Their presence impacts on the patient's physiologic status, on the intensity and complexity of his management, on his chances of survival and of residual disability and on the decision making processes. MT require a coordinated teamwork of multiple subspecialties (a major factor in the development of dedicated trauma centers), longer ICU and hospital stays and therefore much higher resource utilization and costs.

TBI is the most frequent type of severe injury and the major cause of mortality and residual disability. Injuries to more than one organ system were diagnosed in 52.5–67 % of children with severe TBI [26, 27]. In fact, when whole-body computed tomography was utilized, MT was diagnosed in even 79 % of severe TBI cases [27]. The most frequently associated injuries

were lung contusion and pleural effusion (62 %); bones fractures – mainly in the upper limbs, femoral shafts or pelvic ring (32 %); facial fractures and lacerations (29 %); abdominal solid organ lesions (20 %) and spinal cord injuries (5 %) [27].

Injury scoring systems are discussed elsewhere in this textbook. Basically, injury severity scores attempt to quantify the complexity of multiple injuries, and were shown to correlate with all of the above mentioned outcome variables. They are used mainly for epidemiological and research purposes, and, with the exception of the Revised Trauma Score (RTS) that is used for triage purposes, are of limited usefulness for clinical decision making or patient's management. In fact, it has been shown in both adults [28] and children [29], that the single worst injury actually predicts mortality more accurately than the complex cumulative scores. Furthermore, it seems that simpler, more readily available variables are as reliable as the combined scoring systems in predicting severity of trauma: For example, in severely injured children, with or without severe TBI, admission base deficit reflected injury severity and predicted mortality [30–32]. Base deficit less than −8 mEq/L should alert the clinician to the presence of potentially lethal injuries or uncompensated shock [31]. Recently, Borgman et al. [33] have proposed a simple pediatric trauma mortality prediction score developed in military hospitals in Iraq and Afghanistan and validated in civilian patients. This BIG score takes into account only three, early available variables (base deficit, international normalized ratio – INR – and the Glasgow Coma Scale score) and showed a higher sensitivity compared to other commonly used pediatric trauma scores.

Pathophysiology of Major Trauma and Mechanisms of Secondary Damage

TBI and hemorrhagic shock are responsible for the great majority of immediate and early traumatic deaths [34]. Late mortality and the complex clinical course that dominates the care of the multiply injured child in the PICU are caused mostly by secondary TBI and by the systemic inflammatory effects of the host defense responses [3].

The "two-hits hypothesis" states that this complex cascade of host defense responses is stimulated by both primary (first hits) and secondary (second hits) insults [35, 36]. The initial trauma causes primary organ and tissue damage (trauma load, first hits), and it activates the systemic inflammatory response (SIRS) that is then involved in causing secondary complications (second hits) such as respiratory distress syndrome, repeated cardiovascular instability, ischemia and reperfusion injury, metabolic acidosis, compartment syndromes and infections. Other "second hits" are exogenous, including surgical interventions with severe tissue damage, hypothermia or blood loss, massive transfusions, inadequate or delayed surgical or intensive care interventions and line infections (interventional Load) [35]. These second hits often further stimulate the SIRS in a vicious cycle pattern.

The pathophysiologic consequences of major injury can be grossly categorized into a) impaired oxygen delivery due to hypoxemia or shock, resulting in cellular hypoxia, dysfunction and cell death; b) the cascades of biochemical processes following reperfusion after "successful" resuscitation and c) the multiple mechanisms aiming at restoration of homeostasis, clearance of necrotic cells and repair of damaged tissues that are needed to ensure survival and recovery.

All of these processes are intertwined, and although they are discussed separately for didactic purposes, their combined effect creates an extremely complicated pathophysiologic picture. Even after successful resuscitation that ensures immediate survival, the effects of these complex processes turn major trauma into a systemic disease, cause secondary damage and multisystem organ failure that often dominate the clinical picture and impact heavily on the ICU course, residual disability and survival.

Hypoperfusion, Hypoxemia and Tissue Hypoxia

Oxygen delivery to the tissues (VO_2) is the product of arterial blood oxygen content (CaO_2) multiplied by cardiac output (CO); CaO_2 depends mostly on the hemoglobin concentration and oxygen saturation (O_2 Sat), that depends on PaO_2 and the affinity of hemoglobin for oxygen:

$$\begin{aligned} &VO_2\left(\text{ml}\,O_2/\text{minute}\right) = CaO_2\left(\text{ml}O_2/\text{dL}\right)\times CO/100 \\ &= \left[\text{Hb}\right]\left(\text{gr/dL}\right)\times 1.36\times O_2\text{Sat}/100\times CO\left(\text{ml/minute}\right)/100 \end{aligned}$$

Multiple injuries – rather than injury to a single organ – frequently expose the trauma victim to profound compromise of each or several of these physiologic variables. Unfortunately, we still can not routinely monitor cellular PO_2 and therefore do not have direct measures of cellular hypoxia.

When trauma causes significant bleeding, both hemoglobin concentration and blood volume decrease. Decreased circulating blood volume decreases cardiac preload and stroke volume, resulting in hemorrhagic shock and tissue ischemia. Direct injury to the heart or chest can cause, among others, hemopericardium with cardiac tamponade or tension pneumothorax – both reduce cardiac output precipitously by interfering with blood return to the heart and cause traumatic cardiogenic shock.

PaO_2 and O_2 Sat can be critically reduced by multiple mechanisms. In the setting of trauma, the most frequent are central hypoventilation and inability to maintain patent airway as a result of TBI. Facial injuries, aspiration, chest trauma with lung contusion, rib fractures or tension pneumothorax, abdominal distention due to intra-abdominal bleeding and cervical spine injury make up only a partial list of other pathologies causing hypoxemia. Finally, the use of analgesic and sedative drugs during resuscitation and failure to secure patent airway and provide effective ventilation and

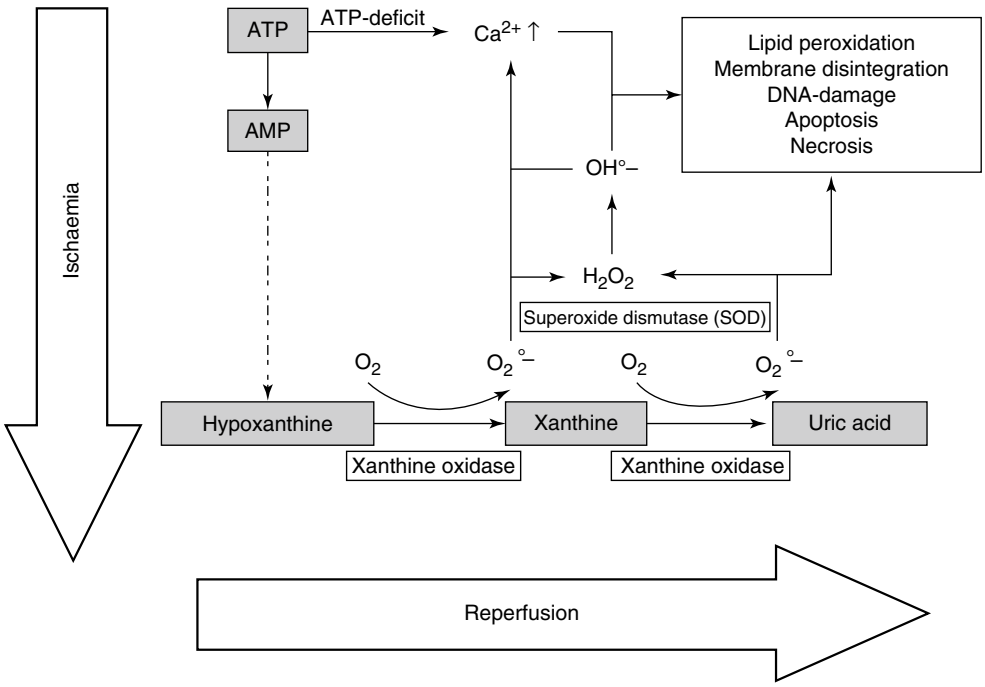

Fig. 32.2 Ischemia – reperfusion injury (Reprinted from Keel and Trentz [35]. With permission from Elsevier)

oxygenation are a rather frequent cause of hypoventilation and hypoxemia in the injured child.

The injured brain is extremely sensitive to secondary insults, and hypotension and hypoxemia in the early stages following TBI were identified as the most deleterious factors contributing to secondary brain damage in both adults [37, 38] and children [39–41].

Ducrocq et al. [26] have analyzed a large cohort of children with severe TBI treated at the scene by the SAMU emergency teams in Paris, and found that hypotension at hospital's arrival was an independent predictor of death and poor neurological outcome. Zebrack et al. [42] found that untreated hypotension – but not untreated hypoxemia – during the early care of children with TBI, was associated with much higher odds for death and residual disability when compared with treated hypotension. In both studies the untreated hypoxemic groups were too small to draw any conclusions, and it may be a grave mistake to conclude that hypoxemia should not be corrected promptly.

Reperfusion Injury

Systemic hypoxemia and hypoperfusion (shock), local hypoperfusion due to contusions, lacerations, vascular injuries or compartment syndromes lead to cellular hypoxia and energy depletion [35] (see Fig. 32.2). Hypoxia leads to decreased production and increased consumption of adenosine triphosphate (ATP), that is degraded to ADP and AMP, which are further degraded to hypoxantine [35, 43]. Cellular energy depletion result in intracellular accumulation of Na^{+} and Ca^{++} that may lead to structural cell damage and death.

Following effective resuscitation and organ reperfusion, hypoxantine is degraded to xantine and finally to uric acid, with the generation of superoxide anions (O_2^{-}) that are further reduced to hydrogen peroxide (H_2O_2) and hydroxyl ions (OH^{-}) by superoxide dismutase [35, 43]. These free oxygen radicals enhance disturbances in intracellular Ca^{++} homeostasis and induce lipid peroxidation, membrane disintegration and DNA damage, resulting in cell apoptosis and necrosis [35] (Fig. 32.2).

Pathophysiologic Responses to Major Trauma

Regardless of the specific mechanism and organ injured, MT trauma is a systemic disease, involving complex, predictable systemic changes. This systemic reaction encompasses a wide range of responses, including activation of the sympathetic nervous system, neuro-endocrinological "stress response" and complex immunological-hematological effects [35, 44, 45]. Following injury, these measures aim at restoration of homeostasis, clearance of necrotic cells and repair of damaged tissues to ensure survival and recovery.

Neuroendocrine and Metabolic Stress Response

The metabolic neuroendocrine response to stress, including multiple injuries, can be triggered by pain, stress, fear and other stimuli that occur in trauma and is augmented by tissue damage, hemorrhage, decreased intravascular volume,

hypotension and hypoxia [35, 44, 45]. The Immediate stress response following injury is characterized by activation of the sympathetic nervous system and by increased pituitary hormone secretion that affect hormone secretion from target organs. These result in a massive release of catecholamines, glucagon, glucocorticoids (cortisol) and mineralocorticoids (aldosterone), anti-diuretic hormone, endorphins, growth hormone, TSH and prolactin. These increased hormone levels can be detected in the serum within minutes after the injury [35].

These concerted responses aim at achieving cardiovascular homeostasis, retention of salt and water to maintain fluid volume and at mobilizing substrates to provide energy sources. The combined effects of the stress response include vasoconstriction, redistribution of blood volume, increased cardiac output, increased oxygen consumption, increased minute ventilation, and increased catabolic rate with gluconeogenesis and glycogenolysis. During this initial phase, the injured patient is therefore relatively oliguric, catabolic and hyperglycemic [35, 45, 46].

Following initial stabilization, increased energy expenditure and catabolism are the hallmarks of the further adjustment of the body to injury. Catabolic metabolism includes fat, muscle and serum protein breakdown with enhanced amino acids mobilization towards the circulation. These amino acids are used by the liver to produce glucose for energy in the gluconeogenesis pathway.

Elevated levels of the stress hormones – cathecholamines, cortisol and glucagon – not only stimulate gluconeogenesis but also inhibit insulin secretion by the pancreas and cause insulin resistance, resulting in hyperglycemia. Early hyperglycemia was shown to be an independent predictor of mortality in both adult [47] and pediatric [27] trauma patients. It should be stressed that glucose stores are limited in young children and neonates, and once they are exhausted, dangerous hypoglycemia may occur.

During this phase, liver metabolic processes are shifted toward production of acute-phase proteins, resulting in a marked rise in the circulatory levels of C-reactive protein, fibrinogen, haptoglobin, alpha-1 antitrypsin and more. Concomitantly, production of nutrient transporters such as albumin is markedly decreased [48, 49]. Decreased production, increased breakdown by the catabolic processes and enhanced vascular permeability are the major causes of the marked hypoalbuminemia, typically seen early following severe multiple injury [50]. Admission serum albumin was predictive of outcome in critically ill adult trauma patients [51].

This phase of increased energy expenditure and enhanced catabolism peaks after 5–10 days and can last for 2 weeks. This adaptive mechanism, generating amino acids for wound healing and glucose for energy usage, is a very pronounced, short term compensatory mechanism in children. In the long run, if the metabolic and nutritional needs are not met by appropriate caloric support, these compensatory mechanisms become insufficient as they exhaust the body proteins stores. Progressive loss of muscle mass leads, among others, to respiratory compromise and cardiac dysfunction. Therefore, early and appropriate nutrition – either enteral or parenteral – is crucial. Following abatement of the catabolic phase, the final recovery anabolic phase gradually takes place, aiming at wound healing, buildup of new tissues and renewal of energy stores [35, 46].

The Systemic Inflammatory Response to Trauma

The delayed responses to trauma aim at clearance and repair of damaged tissues and incite a complex inflammatory response, which basically involves a twofold dysregulation of the immune system (Fig. 32.3). Initially, hyperinflammation dominates – clinically expressed as the systemic inflammatory response syndrome (SIRS). Subsequently the compensatory anti-inflammatory response syndrome (CARS) sets in, resulting in immune suppression and predisposing the patient to sepsis [35, 52]. If severe enough, these processes can result in multi-organ dysfunction syndrome (MODS), multiple organ failure (MOF) and death.

The innate immune system, the first line of defense against infection, recognizes pathogen-associated molecular patterns through pathogen recognition receptors (PRRs). PRRs also recognize products of tissue damage, known as ‘alarmins’ [53, 54]. These are intracellular components that, when released into the extracellular space, signal danger to surrounding tissue. Of the PRRs, the most extensively studied are the toll-like receptors (TLR) [54, 55]. PRRs are located on or in cells that act as sentinels of infection and tissue damage and initiate the complex inflammatory response – both locally and systematically, remote from the site of injury [53–55].

Binding of ‘alarmins’ to the PRRs induces production of proinflammatory cytokines like TNF-ά, IL-6, IL-8, IL-10, chemokines and type I interferon [55, 56]. The degree of cytokine production correlates with the severity of injury and with outcome [57–59]. This binding also initiates the priming of neutrophils for increased release of toxic oxidants and enzymes, resulting – among others – in endothelial damage – again predisposing the patient to subsequent SIRS and MODS [57, 60–62].

MT further activates the complement system, the coagulation cascades, platelet activating factors and the arachidonic acid pathway producing various prostanoids [35, 52, 63]. These basically distinct systems are interconnected and usually exert mutual positive feedbacks, so that the activation of one system augments the activity of other systems.

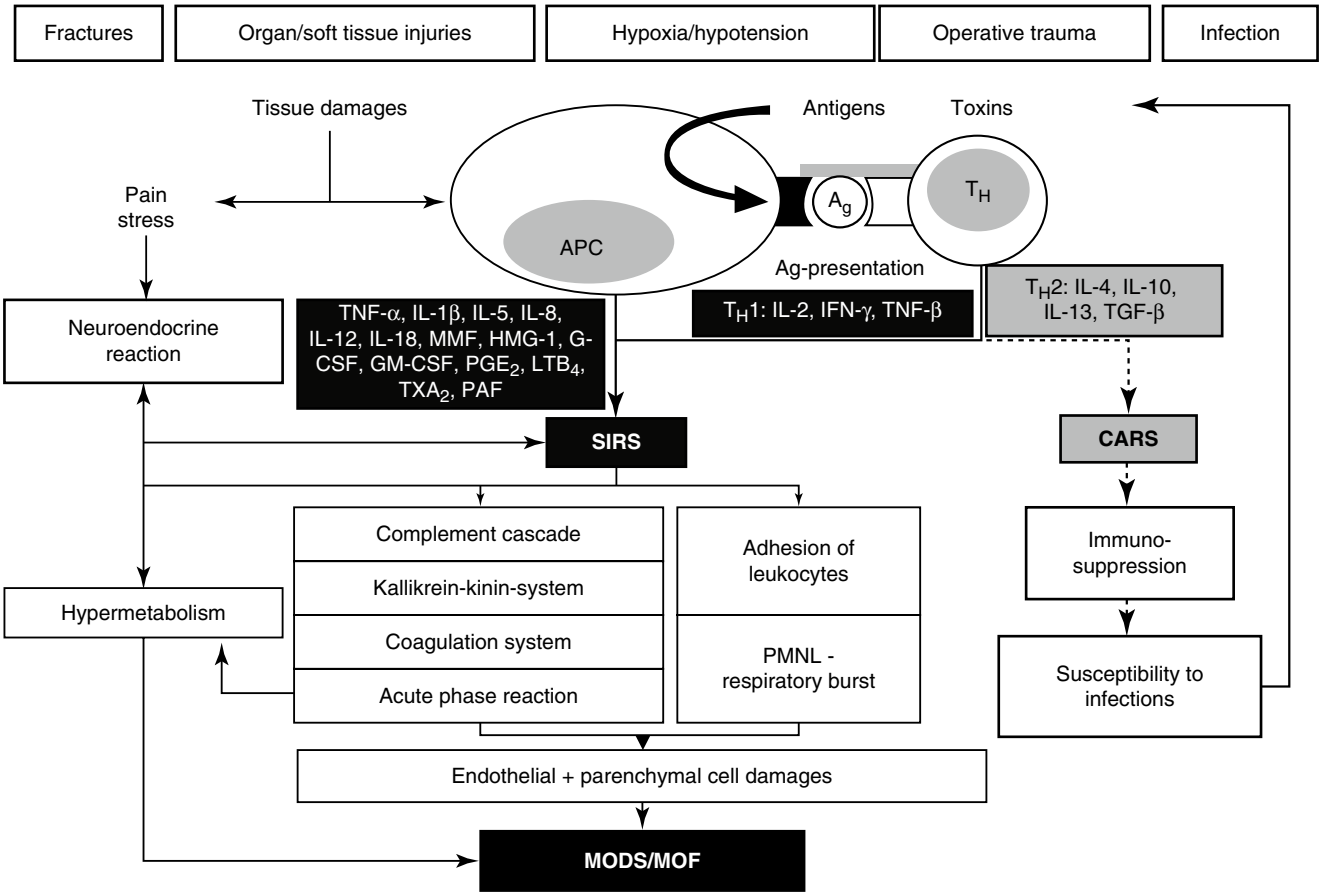

Fig. 32.3 Host defence response after trauma. *APC* antigen presenting cells, *TH* T-helper cells, *SIRS* systemic inflammatory response syndrome, *CARS* compensatory anti-inflammatory response syndrome, *PMNL* polimorphonuclear leukocytes, *MODS* multiorgan dysfunction syndrome, *MOF* multiple organ failure (Reprinted from Keel and Trentz [35] With permission from Elsevier)

The immune response to trauma has been modeled by Bone et al. to consist of the pro-inflammatory SIRS arm followed by a compensatory anti-inflammatory response arm (CARS), aiming at deactivation of the "hyperactive" immune system and restoration of homeostasis [64] (Fig. 32.3). The resultant immune suppression predisposes the trauma patient to local infection and to sepsis, which are associated with late mortality. More recently, it has been hypothesized that following trauma, these two divergent response arms are in fact concurrent, and that their timing and relative magnitude have a profound impact on patient outcome [65, 66].

The activation of all of these basically protective mechanisms may result in multiple, variable, often unpredictable, potentially deleterious physiologic responses, including increased microvascular permeability and edema, vasodilation and decreased cardiac output that may progress to irreversible shock, vasoconstriction that can cause thrombosis and local ischemia, pulmonary vasoconstriction, coagulopathies including DIC, intense catabolic state causing hypoalbuminemia, hyperglycemia and insulin-resistance, direct endothelial damage, acidosis, fever and more. These processes are augmented by hemorrhage, shock and hypoxia and also by antecedent therapeutic measures like fluid resuscitation and the administration of vasopressors. These intense responses may evolve into dysfunctions and failure of various body systems such as the respiratory, cardiovascular, gastrointestinal, hepatic, renal, coagulation and immune systems, that often dominate the clinical course following MT, require complex therapeutic measures and are associated with prolonged ICU stay, high ICU costs, worsened outcome and increased risk of mortality.

Multi-Organ Dysfunction Syndrome (MODS) and Multiple Organ Failure (MOF) Following Trauma in Children

With the advancement of life-support care of major trauma, resulting in impressive decreases in early mortality, multiple organ failure has emerged as a major pathway to delayed death in intensive care units [67]. It has been further recognized that regardless of age, organ dysfunction represents a

continuum of physiologic derangements rather than a dichotomous state of "normal" vs. "failure"- hence the more appropriate terminology of multi-organ dysfunction syndrome (MODS) rather than multi-organ failure (MOF) [67].

Calkins et al. [68] reported no MODS in 334 children admitted to the PICU with isolated brain injury. Only 3 % of multiply injured children developed MOF – defined as moderate to severe MODS – with a low (17 %) mortality. Compared with adults, seriously injured children had a four- to eightfold lower incidence of MODS and of MOF related mortality [52, 68, 69]. It is unclear whether this low rate of MOF is due to a different inflammatory response, as speculated by Calkins [68], or whether it is due to other factors, including comorbidities [70]. Wood et al., suggested that differences in the innate immune response of children may go beyond simple intensity of responsiveness and that children have a fundamentally unique inflammatory system with a relatively protective response to traumatic injury [52].

Clinical Implications of Multiple Trauma

The presence of MT, especially if associated with severe TBI, has several important clinical implications: First, it increases the probability of secondary damage – especially to the brain. Second, it complicates the clinical course, impacts on patient's management, worsens the outcome of each single injury and is associated with a higher case-fatality rate. This basically obvious fact is quantified by the various injury severity scoring systems (ISS, NISS etc.). Lastly, MT requires different priority setting and makes clinical decision taking in the PICU far more complicated.

Pre-hospital Care of the Multiply Injured Child

The crucial importance of prompt and effective resuscitation of the multiply injured child at the scene, during transport and in the emergency department (ED) cannot be overemphasized. The paradigm of preventing early hypoxemia and hypotension to prevent secondary brain damage has become a firm cornerstone of all adult and pediatric guidelines for the care of severe injuries [41, 71, 72]. Efficient emergency medical services (EMS's) and well trained medical and paramedical teams, capable of rapidly providing professional primary treatment at the scene, followed by safe and expedite evacuation – preferably to a designated trauma center – have reduced the frequency of critical complications and were repeatedly shown to improve the outcome of severely traumatized patients [42, 73–76].

Discussion of basic and advanced life support is provided in specific chapters in this textbook. The following paragraphs discuss aspects that are specific to the multiply injured child.

Pre-hospital Airway Management

Inadequate airway management in the field and on the way to hospital is the major cause of secondary damage. While endotracheal intubation (ETI) is considered a "gold standard" in the hospital setting, it was not shown to provide unequivocal outcome benefit over bag-valve mask (BVM) ventilation in the field. Retrospective studies comparing pre-hospital ETI and BVM in both adult and pediatric trauma and in urban and rural settings reached contradicting results: Some studies found better survival with ETI [76–79], while others found no benefit [80] or even worse outcome [81–85]. Gauche et al. conducted a large, prospective trial on children requiring airway intervention in the pre-hospital setting in the Los Angeles County [86]. The results indicated no difference in survival or neurologic outcome between paramedic ETI versus BVM ventilation. It should be noted that ETI skills were added to the paramedic scope of practice for the purpose of this study, resulting in paucity of practical experience, poor ETI success rate (57 %) and relatively high complications rate. Moreover, the mean transport time to the nearby ED was only 6 min. These results, therefore, may be relevant to trauma occurring in a densely populated, inner-city environment with abundance of medical facilities but not to trauma occurring in different circumstances, such as rural environments.

Importantly, most of the studies reported high rates of ETI failures and complications, and in many of them ETI's were performed without adequate sedation [83, 85–89] – both factors contributing to adverse outcome. Hence, the lack of a proven outcome benefit of pre-hospital ETI possibly stems from deficient operator's skills combined with the difficult nature of performing ETI in the field [90, 91].

Active gag and cough reflexes are maintained even in the comatose patient with severe TBI, and when ETI with inadequate sedation is attempted, he often becomes combative. This may result in laryngospasm, vomiting, coughing, aspiration, hypoxemia, aggravated cervical spine damage, elevated intracranial pressure, hypertension and enhanced bleeding – all contributing to intubation failure and potentially to secondary damage [85, 88, 92, 93]. Experienced emergency teams using sedation protocols that included neuromuscular blockage reported high ETI success rates and very low complication rates [77, 94, 95].

Rapid sequence intubation (RSI) with muscle paralysis is the recommended approach to ETI in emergency situations. Etomidate and midazolam are the most commonly used sedatives, while opiates and thiopental are hardly ever used in the pre-hospital setting. Midazolam, opiates and thiopental may decrease blood pressure – more so in the unstable trauma patient, and a significant number of children respond paradoxically to benzodiazepines and become more agitated and combative [96].

Ketamine is a safe and effective sedative-hypnotic and may be optimal for short interventions in emergency situations,

including RSI [97–101]. Within its therapeutic range it does not depress spontaneous ventilation nor lowers blood pressure. Its use in trauma situations and especially in patients with TBI was very limited due to its alleged effect of ICP elevation [100, 102, 103]. This notion has been recently refuted in a prospective controlled trial demonstrating that ketamine was in fact effective in decreasing elevated ICP and in preventing untoward ICP elevations during distressing activities in ventilated children [98]. It is currently used successfully and extensively not only in the ED but also by civilian and military emergency services [104–106].

Emergency Department Management of the Multiply Injured Child

The complicated, very demanding rapid sequence of actions undertaken in the admitting ED is organized in a standardized scheme, aiming at detecting and treating immediate life-endangering conditions (primary survey) and subsequently at diagnosing all other injuries (secondary survey) and constructing the treatment plan. This standardized approach is crucial in face of the wide variability of major trauma, the very short time frame available to prevent secondary damage, loss of organs or death (the "platinum half hour" or the "golden hour") and the dramatic nature of caring for a severely injured child in the ED. Without a systematic approach and a concerted teamwork under one team leader overseeing the entire scene, the intricate situation may become chaotic, more subtle injuries may be missed and wrong decisions may be taken – leading to potentially catastrophic results.

Role of the PICU Physician

The role of the pediatric intensivist in the pre-PICU management of the severely injured child varies according to local organizational structures and policies. Pediatric intensivists often lead ground or air medical transport teams. In the ED, the trauma team leader is most frequently a surgeon or an emergency physician, and the pediatric intensivist is often responsible for airway management, ventilation or intravenous line placement. Subsequently, again according to local policies and the patient's condition, the pediatric intensivist may be in charge of attending the ventilated child through imaging and during transport to the operating room or PICU.

Primary Survey, Resuscitation and Initial Stabilization

The primary survey follows the Airway, Breathing, Circulation, Disability and Exposure sequence (ABCDE), though in reality the evaluation of the neurological status (part of "Disability") is performed as a first step concomitant with airway evaluation. Treatment of life threatening conditions often takes place concurrent with the primary survey.

Airway

Airway compromise, the most urgent medical problem in the severely injured child, is caused first and foremost by altered sensorium. In the comatose child the relatively large tongue, the floppy epiglottis, secretions, blood or foreign body in the oral cavity and loss of effective coughing are the main mechanisms of upper airway obstruction. In the neurologically intact child, maxillo-facial trauma, facial burns or direct laryngeal injury can obstruct the airway. All ETI's in the ED should be performed under the same RSI approach outlined above.

Clear cut indications for securing the airway through ETI in the ED include clinical signs of upper airway obstruction, inability to cough or clear secretions and inadequate respiratory effort or a GCS $\leq$8 [41]. Of note: children with GCS $\leq$8 may temporarily be able to maintain airway and ventilate effectively, and this should not be mistakenly interpreted as if they do not need ETI, as their sensorium should be expected to deteriorate further. When this happens, they may be out of the closely observed surrounding of the trauma bay, on intra- or inter-hospital transport or in the imaging department, under far less favorable conditions for emergency ETI.

There are some "relative" indications for ETI in the ED: The multiply injured child with no major TBI is typically painful, frightened and combative. To reduce pain and anxiety and to enable effective, thorough evaluation and rapid initiation of treatment, he will require generous doses of analgesics and sedatives. Uncompensated shock presents another relative indication for early assisted ventilation. The injured child will be much safer and his management much smoother if he will undergo "semi-elective" ETI in the ED. Similarly, if the child is planned for surgery under general anesthesia, or even imaging procedures that require heavy sedation, ETI should not be postponed. The disadvantage of this approach is the loss of the ability to clinically monitor the child, but in reality most management decisions are made on the basis of the initial evaluation in the pre-hospital phase or upon ED admission and according to imaging findings.

Breathing and Mechanical Ventilation

Once the airway is secured, adequacy of breathing should be evaluated clinically, by oxygen saturation and by blood gases analysis. In the injured child, ventilation may be compromised mainly due to decreased central ventilatory drive or to thoracic injury. Very frequently, however, hypoventilation or apnea are iatrogenic, caused by sedative-anesthetic medications, and obviously by muscle relaxants, used during RSI.

Every intubated child must be mechanically ventilated. No child, certainly not the injured child, should ever be expected to breathe effectively through a narrow pipe that

typically reduces the diameter of the trachea by half and therefore increase resistance to airflow by 16.

The injured child should initially be ventilated with $FiO_2 = 1.0$. Although there is insufficient evidence to recommend any specific FiO_2, it is reasonable to titrate it to maintain $SatO_2$ >94 % [107]. In many ventilators available in ambulances and in the ED, FiO_2 is not adjustable, but can be set to FiO_2 of 0.4 or 1.0. As long as there is no clear cut evidence that short-term hyperoxemia is detrimental, it seems safer to avoid hypoxemia and use the higher FiO_2.

Circulation

Shock in the multiply injured child is primarily hemorrhagic. The classical assessment of end-organ perfusion may be of limited value in this situation: CNS function is often depressed due to TBI or the use of sedatives, tachycardia may also result from pain and anxiety, skin temperature and capillary refill are affected by exposure to environmental temperature and urinary output is not indicative during the primary survey. The more "reliable" signs of shock include tachycardia in the sedated or comatose child, thready or absent pulses and hypotension.

Shock is conveniently categorized into compensated and uncompensated: In compensated shock blood pressure is maintained above the lower limit (5th percentile) of age-adjusted values. In uncompensated shock, blood pressure drops below these values, sensorium is usually markedly depressed and peripheral pulses are not palpable. It should be stressed that hypotension is a relatively late sign, developing following loss of at least 30 % of blood volume. The response to resuscitation measures and fluid boluses is extremely helpful in the evaluation of the hemodynamic status.

Uncontrolled hemorrhage occurs usually in hidden body cavities and compartments – the abdominal cavity, retroperitoneum, pleural space and thighs. Scalp lacerations can cause fatal hemorrhage and may be easily missed as the child is lying supine, covered with a blanket and the blood accumulates posteriorly, under his back on the stretcher – hence the utmost importance of examining the child's "back". Less frequent causes of shock in the traumatized child are tension pneumothorax and pericardial tamponade, and more rarely spinal cord or severe traumatic brain injuries ('neurogenic' shock).

Vascular Access

Vascular access may be challenging in the injured child and should be obtained as early as possible, preferably in the prehospital phase before shock develops. Two large bore peripheral catheters should be inserted, at least one of them in an upper extremity, ensuring effective fluid resuscitation in case of intra-abdominal injury.

Intraosseous (IO) needle is an excellent alternative whenever peripheral line insertion fails, especially in infants and young children and during shock [107, 108]. Several IO insertion devices are currently marketed: The traditional Jamshidi needle is available in 15G and 18G diameters. We prefer the larger, 15G needle for all ages beyond the neonatal period as the smaller 18G needles tend to bend during insertion. Significant force should be applied for successful insertion and care should be given to correct placement and fixation to avoid needle displacement.

Other mechanical IO insertion devices are available for use in children. The Bone Injection Gun (BIG) is a spring-loaded single-use device that "shoots" the needle into the bone, and is available in pediatric and adult sizes. The EZ-IO is a hand-held drill with detachable IO needle. Although clinical data in pediatric patients are very limited, these mechanical devices seem to be easier for use and to have a higher insertion success rates compared with the Jamshidi needle [109]. Tested on turkey bone model, the EZ-IO had a higher insertion success rate compared to BIG and was the preferred device by the users [110].

In the ED setup, central venous catheters are used infrequently. They may be indicated when no other vascular access can be established or when a very large bore catheter is needed for massive blood and coagulation factors transfusion. The pediatric intensivist is relatively more trained in central line placement and may undertake this task.

Fluid Resuscitation

After over 30 years of controversy, the issue of colloids versus crystalloids is still debated. A recent comprehensive meta-analysis of 56 randomized controlled clinical trials that compared colloids and crystalloids in patients requiring volume replacement, concluded that there is no evidence that resuscitation with colloids reduces the risk of death compared to resuscitation with crystalloids in patients with trauma, burns or following surgery [111].

Clinical trials of fluid resuscitation in children with trauma were not published. Rather small size randomized trials in children with septic shock or dengue shock syndrome found no clinical benefit of colloids over isotonic crystalloid resuscitation [112–114].

Another unsettled issue is that of hypertonic (7.5 % saline or 7.5 % saline + 6 % dextran 70) vs. isotonic (normal saline or lactated Ringer's) solutions. Potential benefits of hypertonic solutions include restoration of intravascular volume and tissue perfusion with smaller fluid volumes, and attenuation of the inflammatory response and secondary ischemia-reperfusion injury. Thus, they may reduce the development of MOF and ARDS, and prevent brain edema and intracranial hypertension in patients with both TBI and shock [115–118].

Though earlier clinical trials demonstrated some overall survival benefit for patients resuscitated with hypertonic solutions [119], two recent large multicenter clinical trials failed to demonstrate any advantage of hypertonic solutions over normal saline in adult patients with either traumatic shock [120] or severe TBI [121].

Hydroxyethyl starch (HES), an artificial colloid, has been used extensively in adults. Recent controlled clinical trials documented increased incidence of acute kidney injury and acute renal failure requiring dialysis in adult ICU patients [122] and in adults with severe sepsis [123], assigned to fluid resuscitation with HES as compared with those receiving normal saline or Ringer's acetate.

A recent meta-analysis [124] concluded that the use of HES – compared with other resuscitation solutions – in critically ill patients requiring acute volume resuscitation, was associated with a significant increased risk of mortality and acute kidney injury. The authors concluded that the use of HES for acute volume resuscitation is not warranted due to serious safety concerns.

Blood and Coagulation Factors Transfusion

If signs of shock persist after three fluid boluses (60 ml/Kg), red blood cells (RBC's) transfusion should be initiated at 15 ml/Kg or one adult unit. Although the great majority of children stabilize after the initial fluid resuscitation and a single RBC's transfusion, bleeding in the mutiply injured child may be massive and coagulopaties may develop due to dilution or consumption of platelets and coagulation factors and due to hypothermia. Recent evidence in adult trauma patients indicates that earlier and increased plasma and platelet to RBC's ratio improve outcome following massive transfusion [125–127]. This has led to the development of massive transfusion protocols that include empiric early transfusion of RBC's, FFP, platelets and cryoprecipitate in balanced ratios for adults [128, 129] and children [130–132] (see Fig. 32.4). A recent study in children [131] showed that the introduction of massive transfusion protocol to a pediatric trauma center resulted in earlier FFP transfusion but was of a far too small scale to detect any potential effects on outcome.

Imaging of the Multiply Injured Child

Patients who are referred to a Trauma Center usually have undergone imaging studies in the referring hospital. It is very helpful to obtain the original images as they serve as 'reference point' and help avoiding repetition of examinations and radiation exposure.

Chest x-ray is the first and usually the only x-ray needed in the ED. Multiply injured patients and those with suspected isolated abdominal trauma should undergo focused abdominal sonography for trauma (FAST) scan in the ED, preferably by a radiologist. To detect free fluid – that will usually be due to internal bleeding – FAST is directed to four locations: the hepato renal fossa (Morison's pouch), subxyphoid view (pericardium), perisplenic space and the pelvis [133].

Ultrasound is a sensitive tool for diagnosing small amounts of intra-peritoneal fluid (200 ml), but is an operator dependent examination. In adults, FAST was shown to have a high sensitivity (73–88 %), very high specificity (98–100 %) and accuracy of 96–98 % [134]. In a recent prospective study sensitivities and specificities of FAST scans for blunt and penetrating trauma were even higher – 93.1 and 100 %, and 90.0 and 100 %, respectively [135]. In the pediatric population, FAST has not gained the same enthusiasm by clinicians, as the indices were lower (sensitivity around 75 %). These examinations, however, were performed by surgeons: When performed by a radiologist, FAST was shown to have sensitivity of 92.5 %, specificity of 97.2 % and accuracy of 95.5 % [136].

In children with less severe trauma, FAST may be used alongside the clinical assessment as a discriminative tool, and can safely exclude the need for computed tomography (CT) scan [137]. The multiply injured child will usually proceed to CT regardless of the FAST findings, which are mainly used for initial assessment of unstable patients who may require immediate surgery.

Multiply injured children should have a 'whole body' CT scan, including a non- contrast head and neck scan, CT angiography (CTA) of the chest, and arterial and venous phase scans of the abdomen and pelvis with no enteral contrast media. With reconstructions in the coronal and sagittal planes, CT scans allow excellent characterization of multiple injuries and CTA has virtually supplanted interventional angiography. Reconstruction images of bone fractures waive the need for additional x-rays of the bony trunk.

CT provides a quick overview of the extent of injuries and additional studies may be required as judged by the radiologist and the case manager. As with all pediatric CT, efforts should be made to minimize radiation dose through technical settings [138, 139]: Effective radiation dose from radiologic studies in pediatric trauma patients admitted to trauma centers was calculated as 14.9 mSv [140] and 12 mSv [141]. CT accounted for 97.5 % of total effective dose [140]. However, when weighing immediate benefits versus potential radiation 'costs' to the multiply injured child, whole body CT scan is 'logically' justified.

In the acute setting Magnetic Resonance Imaging (MRI) is used mainly in cases of complicated cervical injuries. Compared to CT, MRI has superior contrast resolution with higher sensitivity for soft tissue injuries, and is the imaging modality of choice in assessing soft tissue injuries, spinal cord injury, and intervertebral discs and ligaments.

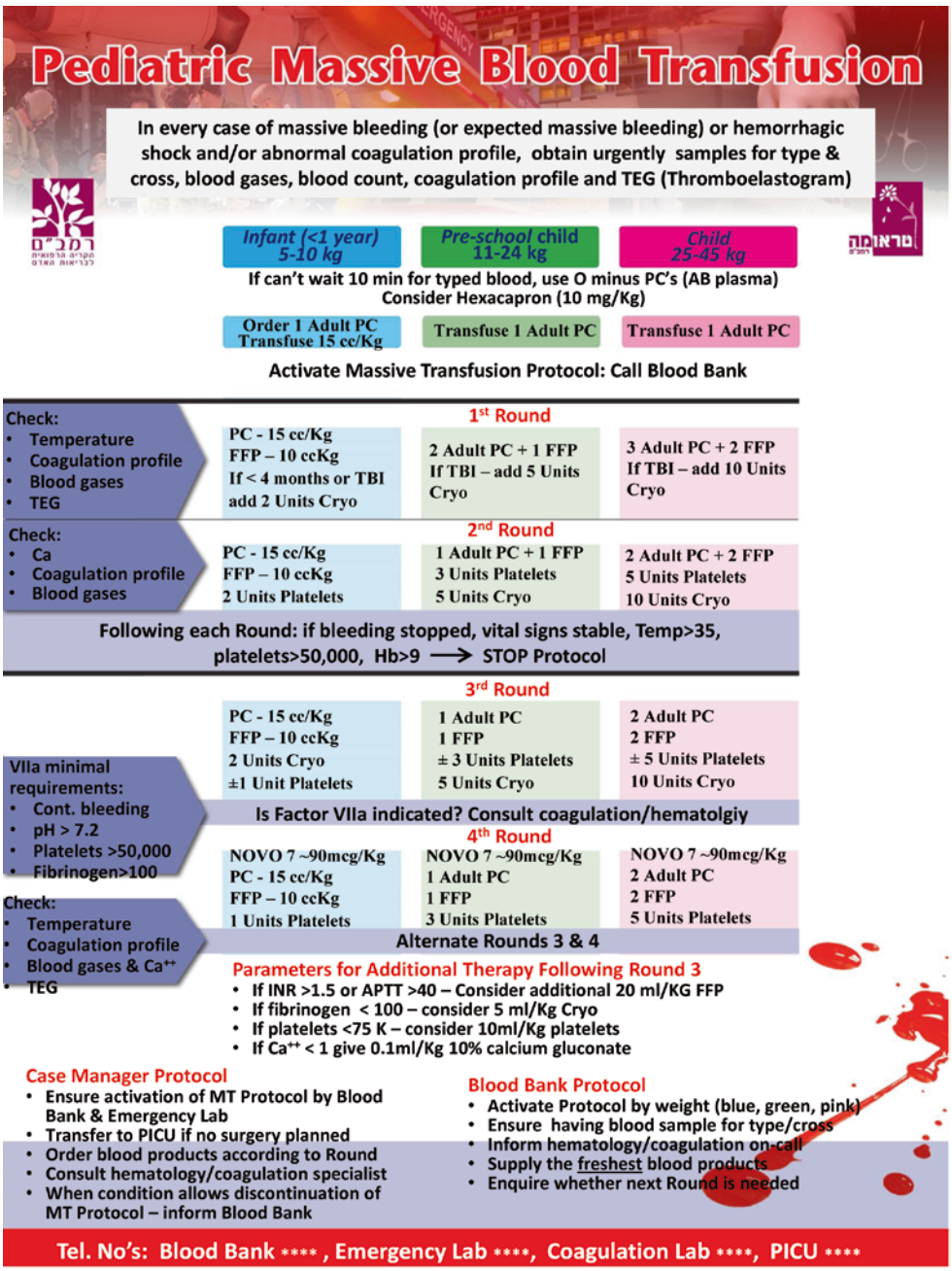

Fig. 32.4 Rambam Medical Center's pediatric massive transfusion protocol (Courtesy of E. Dann MD, Rambam Medical Center, Haifa, Israel)

Management of the Multiply Injured Child in the Intensive Care Unit

The child with multiple, severe injuries, represents a difficult and complex management case. Although the involved surgeon will perform necessary surgeries and will make decisions regarding management of the specific organ system under his 'jurisdiction', it is the intensivist's responsibility to overview the entire clinical picture and to be deeply involved in the decision making processes and in leading all clinical activities. In the PICU, the intensivist should assume the role of "case manager": He is at the patient's bedside 24/7, and is basically the only person able to integrate the complexity of the patient's clinical problems, physiologic parameters, laboratory and imaging results and the various consultants' opinions and recommendations. We have adopted this approach for many years and time and time again realize that its cardinal value cannot be overemphasized.

As the pediatric intensivist oversees the entire clinical picture, one of his crucial roles is to set priorities: He should determine what are the most critical, life or organ endangering problems and prevent or postpone the performance of interventions with potential to cause secondary damage. For example, as the child with low intracranial compliance is

exquisitely sensitive to any external stimulus, it is worthwhile to minimize interventions and delay those considered postponable if they can potentially increase ICP. In general, activities in- and outside the PICU (imaging, surgeries) should be coordinated to minimize patient's risk, pain and discomfort, to avoid excessive use of analgesics and sedatives, and to reduce both nursing and medical staff workload.

Multiply injured children may present some of the most difficult to manage clinical problems, such as MOD and MSOF. One of the more complicated, life-endangering conditions, encountered early in the clinical course, is that of the bleeding patient with hypothermia and acidosis – the so called "Triad of Death". These patients may be admitted to the PICU following surgery or for stabilization prior to surgery.

Triad of Death

Injured patients presenting with hypothermia, acidosis and coagulopathy have been identified at very high risk of death, hence the term 'triad of death'. The triad can develop rapidly in the exsanguinating trauma patient, and is the result of the primary trauma and the secondary systemic response. Once established, it forms a vicious circle that may be impossible to overcome [45, 142–144].

Hypothermia is both a marker of profound injury and is by itself deleterious, promoting this vicious cycle in the bleeding patient. Hypothermia can occur in the field and subsequently in the various hospital environments, due to exposure and heat loss, transfusion of cold fluids and impaired thermogenesis (use of muscle relaxants, for example). It develops more rapidly in infants and children due to their small body mass and large surface area to volume ratio. Hypothermia induces multiple adverse effects: It causes cardiac dysfunction (arrhythmias, decreased contractility) and increased inotropic requirement. It shifts the oxyhaemoglobin dissociation curve to the left and impairs oxygen delivery, thereby aggravating tissue hypoxia in the trauma patient with a preexisting 'oxygen debt'. Hypothermia suppresses enzymatic activity, induces endotheliopathy and increases fluid leak, and promotes platelet dysfunction and coagulopathy [45, 145–149].

Although metabolic acidosis has been considered not harmful per se in this setting, but rather a marker of tissue hypoxia [45, 150], recent studies indicate that low pH by itself severely impairs thrombin generation and accelerates fibrinogen degradation [151]. Tissue hypoxia may be due to direct tissue damage, hypotension or impairment of the microcirculation by hypovolemia, disseminated coagulation, intravascular sludging and endothelial damage. Tissue hypoxia leads to anaerobic metabolism and lactic acid production that may be aggravated by abundant glucose supply and by hepatic function impairment. In pediatric trauma, the probability of mortality increases precipitously in children with a base deficit less than -8 mEq/L [31].

Coagulopathy after major trauma is a multifactorial, global failure of the coagulation system to sustain adequate hemostasis [146]. Derangements in coagulation are detectable already in the hyperacute phase following severe trauma, driven by the combination of tissue trauma and systemic hypoperfusion, and are characterized by global anticoagulation and hyperfibrinolysis [146, 152]. Subsequently, coagulopathy proceeds due to continued blood loss, hemodilution and consumption of platelets and clotting factors, and is exacerbated by hypothermia and acidemia [147, 153].

Studies analyzing the effects of the 'triad of death' on mortality in pediatric trauma patients are lacking. Mortality in adult trauma patients presenting with this triad approximates 50 % [143, 154]. Hypothermia contributes to mortality over and above the mortality associated with multiple severe injuries, independent of hypotension, fluid requirements, age, or duration of surgery [143, 145, 148]. Coagulopathy on presentation has been associated with a fourfold increase in overall mortality [152].

The presence of all three conditions not only adds to mortality, but they further potentiate each other, forming a "vicious cycle resulting in death" [155]. Continuing attempts to stop hemorrhage and repair the injury in the hypoperfused patient result in deepening hypothermia and coagulopathy. SIRS is evolving, further lowers blood pressure, worsens tissue acidosis and enhances capillary leak. The intravascular hypovolemia requires further transfusion and crystalloids volume infusion, resulting in dilution of coagulation factors and enhanced SIRS. The patient is now "oozing" extensively and the vicious cycle spirals down, often resulting in the patient's death in the OR.

Even if the bleeding can be eventually stopped, the patient will arrive at the ICU with massive fluid overload and ongoing capillary leak and is at high risk of developing MODS, MOF and abdominal compartment syndrome. If he also has a significant TBI, the previously incurred hypoperfusion and generalized edema will negatively impact on the brain's ability to recover and on the development of cerebral edema.

The recognition of the poor outcome of these multiply injured, bleeding patients, had led in the early 1990s to a paradigm shift – to the 'damage control surgery' and subsequently to the 'damage control resuscitation' paradigms.

Damage Control in the Unstable Injured Child

"Damage control (DC) surgery" was coined in 1993 by Rotondo et al. [156], though the concept of 'staged laparotomy' has been pioneered by several groups since the mid-1970s [155]. The 'traditional' approach up to this time has been adopted from the elective surgery paradigm, calling for definitive repair of all injuries with abdominal wall closure in a

Table 32.1 The three stages of damage control surgery

1. Theatrer – damage control surgery
Rapid haemostasis
Control contamination
Temporary abdominal closure
2. Intensive care – resuscitation
Re-warming
Correct shock – optimize oxygen delivery
Correct coagulopathy
Correct acidosis
Detect abdominal compartment syndrome
3. Theatre – second look laparotomy
Definitive repair
Abdominal closure
Primary
Prosthetic

Reprinted from Hamill [150]. With permission from Elsevier

single operative session. The paradigm shift to staged laparotomy was driven by the recognition of the 'triad of death' and the realization of the disastrous results of continuing attempts to perform definitive repair of massive injuries in these unstable patients. The focus has been directed to the injured patient's physiology rather than to the completeness of anatomy.

The accepted indications for damage control surgery include hypothermia (temperature <35°), acidosis (pH <7.2 or base deficit >8 mEq/L), clinical coagulopathy or massive transfusion (whole blood volume replacement or – in adults – ≥10 units of pRBC's), hemodynamic instability or profound hypoperfusion and prohibitive operative time needed for definitive repair (>90 min).

Although DC surgery was conceptualized for hepatic and other major abdominal trauma, it was later adopted by other specialties, especially orthopedic [157] and vascular surgery. DC in children follows the same principles as in adults [45, 150, 158] (Table 32.1).

The first stage is targeted at rapid damage control. It includes surgery to control hemorrhage in the abdominal cavity, chest, neck or extremities, and at alleviating contamination, such as fecal spillage from damaged bowel. This is achieved by packing of organs or spaces to control nonsurgical bleeding and by resection of damaged bowel without performing anastomoses or stomas. Because of the high probability of developing abdominal compartment syndrome (ACS), the abdomen is often closed temporarily with loose retention sutures or left open, covered with a prosthetic material. Fractures are immobilized, not definitively reduced.

The second stage is resuscitation and stabilization in the ICU. It includes re-warming of hypothermic patients, correcting coagulopathy and acidosis and restoring adequate cardiovascular state. Resuscitation in this setting calls for several specific considerations. The patient should remain mechanically ventilated to ensure adequate oxygenation, sedation and pain control. He is at a significant danger of developing ARDS and is planned for repeated surgery in a few days; hence attempts at weaning are unadvisable. Hemodynamic resuscitation should be guided by few crucial endpoints – there is no need to 'normalize' all values. Blood pressure should be targeted to values sufficient to achieve urinary output of 1 ml/Kg/h and lactate clearance (to correct acidosis) and to maintain minimal cerebral perfusion pressure in case of coexisting TBI. Normal – and certainly elevated – blood pressure should be avoided in light of the bleeding tendency.

Fluid resuscitation should be judicious as the child is already fluid overloaded and is at a high risk of developing brain edema, respiratory insufficiency and ACS. Blood products, needed for correcting the coagulopathy, are excellent volume expanders. When appropriate, we prefer the use of judicious dosages of cathecholamines rather than 'fill the patient up' with crystalloids. Central venous pressure monitoring is of limited value and should serve as an adjunct guide – not as a targeted endpoint. If the hemodynamic status cannot be stabilized, cardiac echocardiography is indicated to exclude myocardial contusion and to provide useful information regarding myocardial function and volume status. High index of suspicion towards the possibility of ACS is mandatory, even if the abdomen was left partly or entirely open, as discussed elsewhere in this Textbook.

The third stage, reoperation, should be undertaken after the patient's condition has stabilized. This 'second look' aims at searching for missed injuries, definitive repair and, if possible, formal closure of the abdominal wall. In the adult patient population with massive injuries and the catastrophic 'triad of death', the DC approach has reduced mortality to around 50 % [159]. Data regarding outcome of DC surgery in children is minimal, probably because the numbers are much smaller. Stylianos et al. [160] reported on DC in 22 children aged 6 days to 20 years, 13 of whom were trauma patients and 90 % had the 'triad of death': packing controlled hemorrhage in 95 % and survival rate was 82 %. Porras-Ramirez [161] reported on four pediatric MT patients – three of them with multiple penetrating abdominal injuries – managed with DC surgery. Yin et al. [162] compared 32 children who have undergone DC surgery with 17 children who have undergone conventional surgery for serious abdominal diseases. Recovery rates were 84.4 % in the DC group compared to 52.9 % in the conventional group.

Blunt Abdominal Trauma in the Multiply Injured Child

The spleen and the liver are the most frequently injured organs in blunt abdominal injury, each accounting for a third of abdominal injuries. In the hemodynamically stable child, non-operative management has become standard practice. Should these patients be taken care of in the PICU? Guidelines

based on a retrospective review of 832 children with isolated liver or spleen injury proposed no ICU admission for isolated injuries with CT grades I, II, and III, and only one ICU day observation for grade IV injury [163]. A follow-up study, however, found that in 19 % of the cases admitted to 'committed' pediatric surgical centers, actual practice deviated from these recommendations – assumingly towards a more cautious approach towards ICU admission [164]. Jim et al. [165] concluded that nonoperative management of splenic injury must include close monitoring, because 16 % required delayed operation. For high grade liver or spleen injuries it seems justified to prefer the safer approach over the "limitation of resource utilization" approach.

Until the late 1990s, the non-operative approach was limited to isolated liver or spleen injuries. The approach to these injuries in multiply injured children, especially those with associated TBI, remained controversial as their clinical evaluation is unreliable. Based on analysis of children with combined spleen and/or liver and head injuries, registered in the National Pediatric Trauma Registry, Keller et al. [166] found that when stratified for type of injury and severity, both mortality and abdominal and neurologic morbidity were improved in children managed non-operatively. Similarly, Coburn et al. [167] concluded that non-operative management of splenic and hepatic injury in multiply injured pediatric patients, including those with head injury and injury remote from the abdomen that requires surgical intervention, is successful and is not associated with a prohibitive morbidity.

In our PICU we routinely apply the non-operative approach to children with combined abdominal and brain trauma, as long as they are hemodynamically stable. Obviously, clinical evaluation of the abdomen is impaired in comatose or heavily sedated patients, but they can be reliably monitored through hemodynamic parameters (all must have an arterial line) and hematocrit, abdominal girth measurement and abdominal ultrasound when needed. For high grade liver or spleen lacerations, elective mechanical ventilation with complete rest under sedation and minimal handling for at least 24–48 h is indicated.

ICP monitoring should always be applied in patients with MT involving severe TBI. In a child with "borderline" severe TBI (GCS 8–9), one may consider clinical follow up with early cessation of sedatives and early extubation. However, if this child has a significant abdominal or chest trauma and requires mechanical ventilation and sedation, ICP monitoring is indicated as no other follow up is available.

Thoracic Injury in the Multiply Injured Child

Thoracic injury is common among multiply injured children and is an important marker of the severity of injury. Pedestrian and car occupant injuries are the major cause of blunt chest trauma, while penetrating injuries are caused mainly by gunshot or stab wounds. Direct blunt trauma to the chest results in lung contusions or lacerations, rib fractures, pneumo- and/or hemothorax, major vascular disruption or myocardial contusion [18, 45, 168–170].

Severe thoracic injury (AIS ≥3) was detected in 38 % of multiply injured children in the German trauma registry [171]. In two French studies of severe TBI, the most frequently associated injury was chest trauma [26, 27]. Several other studies reported far lower incidence of chest trauma among hospitalized pediatric trauma patients [18, 172–174].

Lung contusions are detected very frequently with advanced CT technology, though their clinical significance is incompletely understood [175, 176]. While lung contusions occupying >20 % of total lung volume were shown to be highly predictive of the need for assisted ventilation in adults [175], similarly large lung contusions did not carry the same morbidity in children [176].

In children, chest trauma as a single injury only rarely results in death [19, 168, 172]. On the other hand, mortality rates in multiply injured children with chest trauma are very high [18, 19, 169, 170, 172, 173, 177].

Peclet et al. [18] analyzed thoracic injuries in children admitted to a level 1 pediatric trauma center. Although thoracic injury was detected in only 4.4 % of this patient population, it was strongly associated with severity of injury: 71 % of the children with thoracic injury were admitted to the PICU, and MT was present in 81.7 % of the children with thoracic injury. Mortality rates were 20 times higher for children with thoracic injury compared to those with no thoracic injury. Mortality was 28.6 % for children with injuries to the chest and another body region, compared to 5.3 % for children with thoracic injury alone. Multiple rib fractures and contusions to several pulmonary lobes were strongly correlated with the risk of death [18].

Chest injuries in multiple trauma victims resulted in a mortality of 19 % in children and 9 % in adults ($P<0.05$) [19]. Mortality was highest in combined head, chest and abdominal trauma: 25 % percent in children and 28 % in adults. For comparison, mortality of multiply injured children with TBI but with no chest injury was only 3 % [19].

Injuries to head, abdomen and chest were associated with the highest overall mortality in the study by Meier et al. [169]. Thoracic injury was associated with the highest odds ratios for death compared to all other injured regions.

The grave consequences of thoracic injury as part of a MT, relate to several factors. First and foremost, thoracic injuries are caused by high energy impacts absorbed by relatively small bodies. In children, bony thoracic structures are more elastic, and if an impacting force is of sufficient energy to result in rib fracture, major intrathoracic injury and trauma to other regions can be expected [18, 19, 169, 170]. Moreover, chest injuries contribute to the unfavorable outcome in

multiply injured children as they may interfere with pulmonary gas exchange and cause hypoxemia and secondary brain damage. Children with chest injury had significantly greater physiological derangement compared to those with no chest injury, reflected by their lower Trauma Scores [18]. Thus, a significant, treatable chest injury, such as pnemo/hemothorax represents true emergency in the initial care of the multiply injured child.

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