International Federation for Emergency Medicine

Best Practices in Trauma Resuscitation

Trauma Special Interest Group

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Best Practices in Trauma Resuscitation

The Trauma Special Interest Group, IFEM

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Foreword

This is a position statement paper created by the International Federation for Emergency Medicine (IFEM) Trauma Special Interest Group (TSIG) to provide contemporary advice on key areas for the assessment and management of patients with severe trauma. This paper is relevant to patients of all ages, including a chapter highlighting unique considerations for children.

Members of the TSIG authored this paper using an extensive literature review methodology. All the relevant literature is cited should the reader wish to understand the current evidence. At the end of each chapter, essential and desirable recommendations are stated based on the evidence and approved by the Trauma Special Interest Group. Essential recommendations are for all locations who may have severe trauma presentations. Desirable recommendations are for locations where increased health resources are available to provide them, such as major trauma centres.

IFEM member organisations are encouraged to utilize this document when discussions with their national health ministries happen to advocate for the creation of trauma systems in their country to deliver optimal trauma resuscitation; this is very important in regions where Emergency Medicine is still developing and major trauma care systems may not yet be fully developed.

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Introduction - Defining the Burden of Major Trauma in Society

Trauma such as road traffic accidents, falls, burns, self-harm and interpersonal violence are a major global public health challenge, causing the deaths of 4.4 million persons each year. These injuries cause approximately 8% of all deaths worldwide.[1] The burden of trauma is not equal, with low- and middle-income countries (LMIC) accounting for 90% of the deaths from injuries. Morbidity and mortality (M+M) for a given injury is often higher in LMIC compared to countries with more developed trauma systems of care. [2] Often a leading cause of morbidity and mortality in children and working-age adults, the societal and economic impact is greater than for many other diseases. Reducing trauma M+M globally has been projected to save 2 million lives annually and 49-52 million disability adjusted live years (DALYs). This would provide an estimated economic benefit of USD 758-786 billion per year. [3]

Epidemiology of trauma varies across countries and across age, gender, socioeconomic strata and rural-urban gradient within countries, making broad generalizations difficult. Even defining major trauma is challenging, with many studies from countries with developed trauma systems using standardized scoring systems, which can only be

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applied retrospectively. The American College of Surgeons Committee on Trauma defines major trauma as “a traumatic injury that results in death, prolonged disability, or requires significant intervention, such as Intensive Care Unit admission or surgical intervention”. Trauma registries often use the Injury Severity Score (ISS) to describe the severity of injuries, with an ISS >15 being a common threshold for sustaining major trauma.

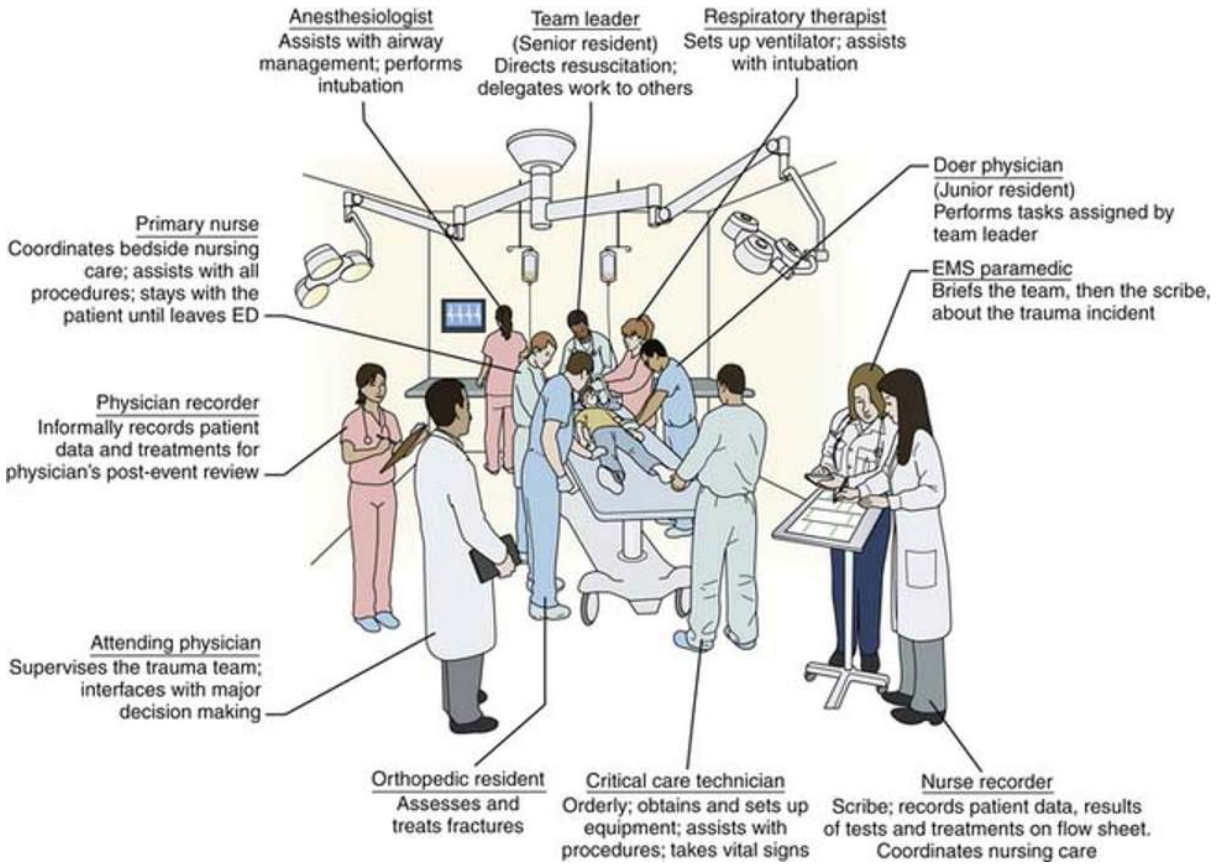
A desire to help prehospital personnel predict which patients may benefit from triage to a trauma center has resulted in further attempts to operationalize a definition of major trauma. Mechanism of injury (MOI) such as falls from height or proximal penetrating trauma has been used to define major trauma but remain controversial as they can lead to both over- and under- triage.[4] Patients with altered level of consciousness, perceived limb-threatening injuries, multiple injuries, abnormal vital signs, and special circumstances such as extremes of age, pregnancy, and bleeding diathesis can be identified early in their care and these factors serve as important indicators of major trauma. [5,6]

Emergency Departments (EDs) serve as the primary point of entry for injured patients into the healthcare system and timely, appropriate care can reduce morbidity and mortality in severely injured patients even in resource-limited settings. [7] ED-based registries can provide crucial information to local and national authorities to direct prevention efforts and funding to mitigate the most important causes of trauma for their patient population like frequent alcohol consumption in adults. [8] Emergency practitioners regularly counsel patients on prevention of injuries and can advocate for policy changes benefiting their population.[9, 10] Trauma prevention and care are chronically underfunded compared to the impact on the population. [11, 12] Improving trauma care globally should be a vital health equity issue. [13]

Essential Take-Home Points

1. Trauma has a major global burden for morbidity and mortality
2. The burden is influenced by high inequity regarding economic resources and healthcare resources, resulting in unequal outcomes and system gaps when comparing countries.
3. ED based registries provide crucial epidemiological information to direct prevention and mitigation efforts.
4. Early recognition of the need for an integrated trauma health care system is critical before improvement in patient outcomes can occur, and Emergency Departments are central to this system.

Chapter 1: Trauma Assessment and Occult Shock



It is important to understand that trauma care goes beyond any given guidelines. Guidelines are the minimum framework one should know regarding trauma management. Emergency medicine and trauma care are like the rails running parallel to the "sleepers" or guidelines. We know any resuscitation in the emergency departments begins with the primary survey.

Zero Point Survey

Recently there has been focus on the "pre-primary" survey also known as the "Zero point Survey" (ZPS). A structured approach to optimising preparedness for the individual, the resuscitation team, and the environment, before the primary survey is what is proposed by the ZPS.[14] We recommend all trauma teams incorporate this best practice into their daily trauma care procedures, especially in situations where prehospital information is routinely received.

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The mnemonic STEPUP can be remembered for all trauma scenarios which includes processes to be followed pre-resuscitation and during resuscitation.

S elf Check – Physical and mental readiness of the team, I'M SAFE (Illness, medications or drugs, stress, alcohol, fatigue, eating)
T eam Readiness – Team Leader chosen and roles/responsibilities assigned
E nvironmental scan – Space, light, crowd control, dangers
Resuscitation begins
P atient – Primary survey (ABCDE – Airway, Breathing, Circulation, Disability, Exposure)
U pdate – shared mental model of patient status
P riorities – Identify goals and set trajectory

Trauma resuscitation is a complex process involving multiple team members with various roles who perform simultaneous evaluations, examinations, management and disposition in a coordinated manner. A rehearsed, coherent trauma team with camaraderie is like a well-oiled machine and can reduce the time from injury diagnosis to critical interventions. It is advisable to have a horizontal approach to the trauma assessment as each team member carries out their tasks simultaneously.[15, 16] The Shared Mental Model (SMM) theory optimises team performance through a common understanding of the team and task requirements amongst the team members. The term 'mental model' described by Holyoak in 1984 refers to a symbolic representation of a system and its expected behaviour.[17] Psychological Ownership, Continuous learning and Heedful interrelating are core mental models that predict team effectiveness. In Trauma Teams, regular team training/simulation, and pre-briefings (STEPUP) can be used to establish a workable shared mental model. This includes determining team-based and task-based processes. The successful integration of SMM in a functional team are influenced by each team member's prior positive experience in teams, clarity in team tasks, and a supportive organizational culture and environment [18]

Airway

Moving on to actual resuscitation. Airway assessment is always the first priority in trauma resuscitation. One should consider four key elements of a physiological difficult airway, being hypoxemia, hypotension, severe metabolic acidosis and right ventricular failure. The Emergency physician should account for these physiological derangements first with airway management regardless of predicted anatomic difficulty of intubation. Definitive management of the airway can be held back for a few moments while physiological issues of breathing and circulation are addressed such as obstructive or haemorrhagic shock. Intubation can have the following effects of increased intrathoracic pressure and decreased right atrial pressure. These can worsen the already low venous return states in haemorrhagic shock and obstructive shock like tension pneumothorax and cardiac tamponade.[19] Specific interventions like needle or tube thoracostomy, pericardiocentesis, transfusion protocols should be performed before intubating the massive trauma shock patient. In the meantime the airway can be supported with other non-invasive maneuvers.

Basic airway maneuvers like head tilt–chin lift and jaw thrust along with adjuncts like Oropharyngeal airway (OPA) and Nasopharyngeal airway (NPA) should be considered while resuscitating patients before going for the definitive management. Definitive airway management can be performed immediately in the following conditions –

1. Critical hypoxia not responding to high flow oxygen or other maneuvers.
2. Dynamic airway like blunt, penetrating or burn injuries to the head, neck, oropharynx.

Even if definitive airway management cannot be held back, the team should ensure that simultaneous management with focused interventions for breathing and circulation issues be carried out. Resuscitation sequence intubation is the ideal technique for such patients.

Shock and Occult Shock

Shock in trauma is usually one of three types – obstructive, hemorrhagic and neurogenic. It is imperative for the trauma team to identify early the cause of shock to prevent deterioration and refractory shock states, and to initiate critical, timely interventions to address the shock.

During initial resuscitation, the parameters that can be utilized to rapidly identify any shock are broadly divided into the following categories –

1. Clinical parameters – at the bedside, including
    a. Vital Signs (VS) – Prehospital vitals should be communicated to the Emergency team before arrival. Zero point survey should be conducted by the ED team. Communicate concern for shock if systolic blood pressure (SBP) <110 mmHg which is associated with increased mortality in penetrating major trauma patients as depicted in multicenter cohort study. [20] Request blood products and activate Massive transfusion protocol if hemorrhage confirmed.[21]
    b. Shock Index (SI) – Heart rate/SBP ≥1.0 – raises concern for shock. If hemorrhage is confirmed – administer tranexamic acid and blood products. The Shock Index (SI) is useful in trauma owing to its strong inter-rater reliability, ease of use and accuracy as a predictor of blood loss. Note – the SI can be unreliable in the presence of altered physiologies (geriatrics), underlying conditions (eg. Hypertension) and use of medications.[22]
    c. Ultrasonography – the eFAST should be promptly used in all trauma patients. Rule out pericardial fluid, pneumothorax or obvious hemorrhage in the abdomen before deciding on intubating the patient. If the scan is positive for any of the above, proceed to perform appropriate interventions like needle decompression, pericardiocentesis, blood transfusion before definitive airway management (unless critical hypoxia or dynamic airway are present).[23]

2. Laboratory parameters – including:
    a. Base deficit (BD) – BD >6 requires aggressive resuscitation. Can become hypotensive during/after intubation. Look for sources of shock aggressively and perform appropriate interventions. BD is strongly associated with need for transfusions, injury severity and death.[24]
    b. Lactate – Complements BD. Lactate >4 indicative for an occult shock. It is a surrogate marker for blood loss. Alcohol intoxication should be ruled out in trauma patients with high lactate levels.
    c. Viscoelastic assays – Rotational Thromboelastometry (ROTEM) and Thromboelastography (TEG) are two point of care assays which can rapidly identify the coagulation status of the patient. Acute Coagulopathy of Trauma Shock (ACoTS) patients have high rates of mortality and organ failure. It is imperative to diagnose ACoTS early and begin damage control resuscitation at the outset. There is still low level of evidence for the use of these bedside assays but they are gaining traction.[25]

As a trauma team treats the patient, all the clinical and laboratory parameters need to be integrated to guide resuscitation strategies and goals. If obstructive and hemorrhagic shock are ruled out, one should have a high level of suspicion of neurogenic shock. In these cases, vasopressor support with noradrenaline or adrenaline can be commenced to maintain perfusion of the spinal cord.[26]

The final decision to intubate the massive trauma shock patient relies on the following factors –

- Hemodynamic status – would the patient be able to tolerate optimization of hemodynamics without intubation?
- Facilitation of resuscitation – would the patient be able to tolerate procedures without intubation?
- Expected clinical deterioration – Is the patient likely to collapse before intubation?
- Clinical gestalt – The value of the clinicians' experience should not be forgotten.
- Of course, critical hypoxia and dynamic airways as mentioned previously would warrant intubation before resuscitation.

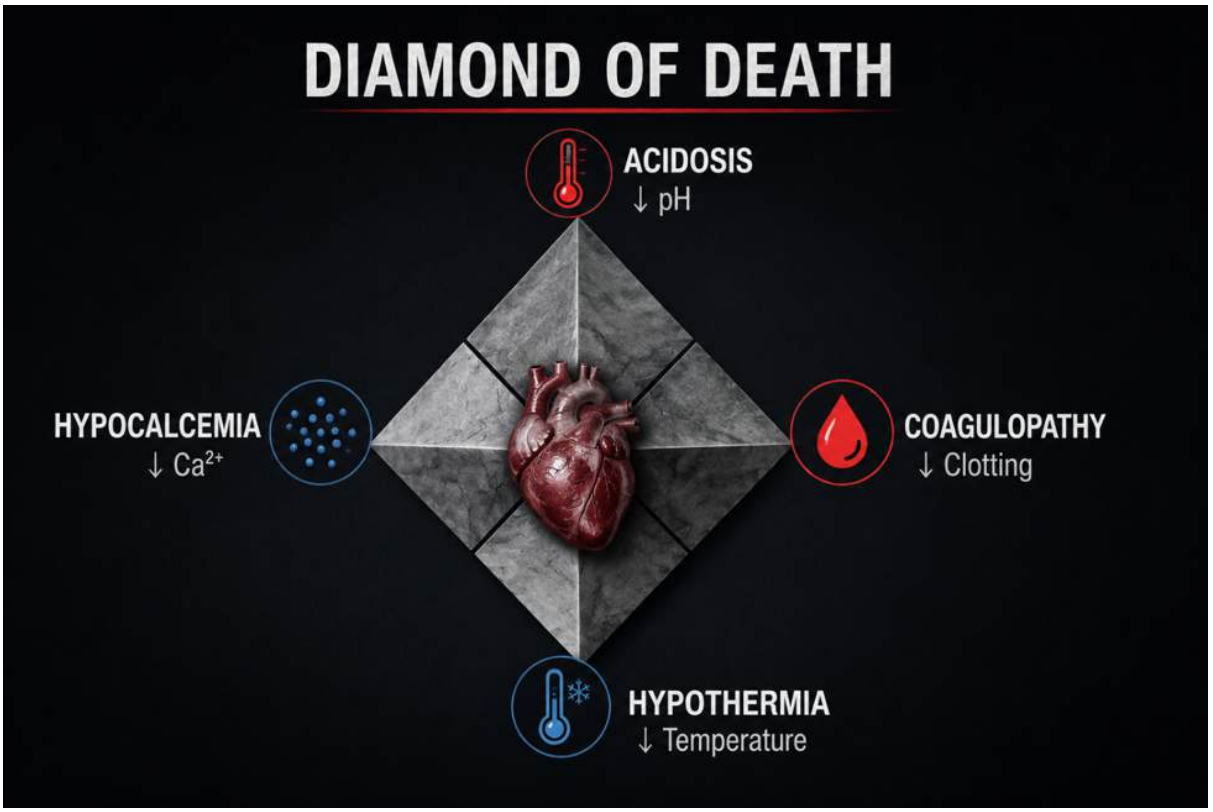
Essential Recommendations

- Zero point survey should be integrated into standard major trauma assessment systems where a prenotification process for patients arriving to emergency departments exists.
- Practicing a Shared Mental Model within a trauma resuscitation team will optimise patient outcomes, so that simultaneous assessment and initiation of treatment can occur in the patient.
- Resuscitate before intubate / Resuscitation Sequence Intubation are terms that should now be used to prevent mortality/morbidity from a physiologically difficult airway.

Desirable Recommendations

- Diagnose Shock/occult shock with more modern indicators such as Clinical (VS, SI, e-FAST) and Laboratory (BD, lactate, ROTEM, TEG).

Chapter 2: Diamond of Death



Trauma is a primary cause of death amongst young adults, with hemorrhage taking precedence as a preventable cause of death [27, 28]. In trauma, the primary aim for patients is to prevent the lethal triad (acidosis, coagulopathy, hypothermia) from setting in, which has shown to worsen prognosis and increases 24-hour morbidity and mortality amongst patients.[29, 30] This cascade peaks into a vicious cycle where the combination of acidosis and hypothermia synergistically worsens coagulopathy. Currently, another aspect has been added to the lethal triad, creating a diamond of death, hypocalcemia.[31, 32, 33] Calcium, has been shown to be an essential cofactor for numerous physiological and enzymatic reactions at a cellular level, such as coagulation, platelet adhesion, myocardial activity as well as vasomotor tone.[34]

It is imperative to understand that only ionized calcium is bioactive, which is tightly regulated to produce a plasma concentration between 1.1 -1.3 mmol/L.[35] Severe trauma patients can present with hypocalcemia even before the initiation of blood transfusion, which can be exacerbated by the transfusion of blood and blood products.[36, 37] This is caused primarily by the presence of citrate in the blood products. Transfusion of blood and blood products exacerbates hypocalcemia due to binding of calcium by citrate in the blood products. In addition to this, hemorrhagic shock and hypothermia also reduce citrate clearance from the liver, which further

aggravates the ionized hypocalcemia.[38, 39] In trauma patients, hypocalcemia exacerbates the blood product requirements and worsens outcomes.[40] It is after much diligence and studies, that the lethal triad has now become a diamond with hypocalcemia taking precedence in urgent emergency treatment.

Recently many studies have been done to legitimize the theory and pathogenesis behind the diamond of death. The lethal diamond concept in some cases falls short in capturing the multifaceted nature of the post traumatic patho-physiology [41]. Studies on trauma induced coagulopathy emphasize the role of fibrinolytic imbalance and endothelial dysfunctions[42], whereas mitochondrial research undermines the effects of trauma shock on global energy metabolism, driving cellular failure across multi-organ systems[43]. This study associated hypocalcemia with mortality in a 24 hour framework. Although substantial data existed on the lethal triad leading to 24 hour mortality, there is still limited data of hypocalcemia worsening prognosis. This study identified 23.7% patients in the lethal diamond with a 24 hour mortality of 32.6% which was comparable to that of the lethal triad.[41] These findings were also in accordance with other studies, done by Mackay et al [31] and Chanthima et al .[44] As opposed to this, a recent study in 2024, in Srikakulam, India, 100 trauma patients were taken for comparison of serum calcium and its relation to hypotension, amongst them 80 patients were seen to have hypocalcemia thus worsening the lethal triad. Amongst the 80, 59 landed in the lethal triad, amongst the 18 with normal calcium, 5 landed in the lethal triad[45].

These findings highlight the urgent need for perhaps a more comprehensive framework that integrates metabolic, vascular and coagulation dimensions to create ideal therapeutic strategies. It is evident that calcium is a key component of the coagulation cascade, which is integral in trauma. Recent studies have started creating reverberations about the importance of calcium in trauma patients but it is evident that further research is required.

Essential Recommendations

- Hemorrhage-driven trauma mortality should be recognised as fundamentally a failure of physiology.
- Hypocalcemia is an early, prevalent, and biologically plausible contributor to trauma-induced coagulopathy, and thus should be vigorously screened.
- Ionized calcium, as a critical, yet under-recognized, determinant of coagulation efficiency, cardiovascular stability, and cellular function, should be checked during initial and ongoing assessment.
- Early calcium assessment and correction represents a low-cost, high-yield intervention.

Desirable Recommendations

- More research is required as current evidence suggests the lethal diamond does not independently outperform the lethal triad in predicting early mortality.

Chapter 3: Damage Control Resuscitation in Trauma: An Overview

Trauma remains one of the leading causes of morbidity and mortality worldwide.[46] In the context of traumatic injuries, hemorrhagic shock is a significant contributor to early death. Traditional resuscitation methods often involve aggressive fluid replacement and blood component therapy, which can lead to coagulopathy and other complications.[47] Damage Control Resuscitation (DCR) offers a paradigm shift, focusing on early hemostatic resuscitation, minimizing crystalloid infusion, and rapidly addressing coagulopathy.

Damage control resuscitation (DCR) is a critical strategy in the management of trauma patients, particularly those suffering from severe hemorrhage. DCR seeks to combat metabolic decompensation by battling three major areas: Permissive Hypotension, Hemostatic Resuscitation, and Damage Control Surgery. The primary goal of DCR is to rapidly restore physiological stability while preventing the lethal triad of acidosis, hypothermia, and coagulopathy.[48] This paper explores the principles, strategies, and outcomes associated with DCR.

Principles of Damage Control Resuscitation (DCR)

DCR is based on several key principles aimed at mitigating the effects of severe hemorrhage. These include rapid control of bleeding, avoidance of hemodilution, and early empiric balanced transfusions with red blood cells, plasma, and platelets, or whole blood when available. Additionally, the use of intravenous or mechanical hemostatic adjuncts is recommended when indicated.[49, 50, 51] As mentioned in chapter three, the triad of death is well known and describes a combination of hypothermia, acidosis and coagulopathy.[52] A fourth component, hypocalcemia, also plays a key role in the outcomes of trauma patients with massive hemorrhage. Packed red blood cells, whole blood, fresh frozen plasma (FFP) and platelets are stored with citrate, which binds calcium.[40, 53] In massive hemorrhage requiring rapid transfusions, the liver's ability to metabolize citrate is surpassed and exacerbated by hypothermia.[40] Hypocalcemia leads to impaired coagulation by two methods, increasing acidosis and also impacting platelet function. [32]

Strategies in Damage Control Resuscitation

The strategies employed in DCR are multifaceted and include:

- Permissive Hypotension is maintaining a lower blood pressure to reduce bleeding until definitive hemorrhage control can be achieved.[50, 51, 54] Permissive hypotension may offer survival benefit for patients with massive trauma. Studies have also shown that permissive hypotension may also reduce volumes of crystalloid administration, packed red blood cell utilization and overall blood loss.[55] The European Guidelines for Management of Major Bleeding recommend "a target systolic blood pressure of 80 mm Hg to 90 mm Hg (mean arterial pressure 50–60 mmHg) until major bleeding has been stopped in the initial phase following trauma without brain injury. In patients with severe traumatic brain injury (TBI) (GCS ≤ 8), we recommend that a mean arterial pressure ≥ 80 mmHg be maintained. (Grade 1C)".[56]

- Balanced Blood Product Administration. Excessive crystalloid resuscitation is linked to increased ICU days, ventilator days, and mortality, thus a judicious approach is recommended.[54, 57] Early use of blood products in a balanced ratio (1:1:1 of plasma, platelets, and red blood cells) to address coagulopathy and improve outcomes.[48, 57, 58] This strategy has shown decreased mortality from about 65% to about 19% along with reduced incidence of complications including sepsis and rebleeding.[59, 60] The goals of blood loss replacement are the following: Hemoglobin of >8g/dL, Platelet count >100,000/dL, Normalization of coagulation times (thromboelastography / Rotational Thromboelastometry),

and Fibrinogen >100 mg/dL. Whole blood, while difficult to obtain in some trauma centers, contains the essential components including red blood cells, clotting factors, and platelets, which may in turn decrease total blood usage and complications related to massive blood transfusions.[61, 62] Over half of all trauma patients that are brought for treatment have hypocalcemia prior to receiving blood products.[63] Ditzel and Anderson have included hypocalcemia as a new component of the lethal triad, creating the "Lethal Diamond".[64] Correction of hypocalcemia is imperative to avoid worsening coagulopathy.[32]

- Damage Control Surgery (DCS) helps to safeguard the trauma patients physiologic reserve but can be harmful in patients that do not meet the indications for its use.[65, 66, 67] Damage control surgery is divided into four stages.

Stage 0: which requires accurate prehospital triage, rapid transport to an appropriate facility, stopping bleeding and prevention of hypothermia.[68] Pre-hospital bleeding control using the principles of "Stop the bleed®" include use of direct pressure, wound packing, use of a tourniquet on extremities and use of a pelvic binder for possible pelvic fractures. Tranexamic acid (TXA) has shown strong evidence for use in adult trauma and some evidence in pediatric trauma and can be considered in patients with hemorrhage.[48, 57] Once the patient arrives at the trauma facility, the multidisciplinary team established the clinical management algorithm to be followed and determines if the patient is a candidate for DCS.[68] Balanced blood product resuscitation is initiated, and the patient is moved quickly to the operating room (OR).

Stage 1: The process of containment of the injury begins in the OR. The goal of the first surgical intervention is to control the sources of surgical bleeding and to carry out brief interventions which include packing, vessel ligation, temporary shunts and balloon tamponading among others to achieve this goal.[69, 70, 71] Hollow viscus injuries can be controlled by simple procedures such as transection and ligation of ends. The goal is to limit the initial operative procedure to <120 minutes.[72, 73] A temporary closure of the cavity should follow with a negative pressure system and the patient is then transferred to the intensive care unit (ICU) for rewarming, correction of coagulopathy, hypocalcemia, and acidosis.[74]

Stage 2: Hemodynamic correction requires several measures which begin in the ED and are continued in the OR and ICU. These include continuing the 1:1:1 transfusion ratio of blood products, plus prevention of hypothermia and close hemodynamic monitoring.[75] A review of additional non-life threatening injuries that need to be addressed is completed and additional imaging is ordered as warranted. Continuous

monitoring of intra-abdominal pressure is essential to avoid abdominal compartment syndrome, which can occur even with an open abdomen.[76]

Stage 3: When the initial ICU goals of correcting coagulopathy, hypocalcemia, and acidosis have been addressed, the patient is taken back to the OR for definitive surgical management. Ideally this is a planned return to the OR, however, an unplanned scenario can occur if there is ongoing hemodynamic instability, which is typically due to uncontrolled surgical bleeding, missed hollow viscus injury or developing compartment syndrome. Typically, a planned return to the OR occurs within 24-48 hours once the resuscitation efforts have restored hemostasis.[72, 77]

Additional Considerations

REBOA: A bridge to pre-hospital management and before surgery that can be considered is Resuscitative Balloon Occlusion of the Aorta (REBOA). REBOA aids in hemodynamic resuscitation by allowing the intravascular content to remain directed towards essential organs.[78, 79] Placement and ongoing management should be performed by a well-trained physician in the emergency department.[80, 81] REBOA allows for early proximal control of the bleeding source in both penetrating and blunt trauma. It allows for real time blood pressure monitoring and is indicated in patients with a systolic blood pressure of <70 mmHg. The balloon can be inflated in the descending thoracic aorta (zone 1) to reduce blood flow below the diaphragm or in the abdominal aorta below the renal arteries (zone 3) to stop bleeding from the pelvis or lower extremities.[82, 83] Quality studies on the effectiveness of REBOA are limited. REBOA has shown a positive effect on overall mortality when compared to resuscitative thoracotomy, but no valid conclusion can be made due to selection bias.[84]

Pediatric Considerations

While the principles of DCR are similar for both adult and paediatric patients, there are important physiological differences that necessitate slight variations in approach. Early recognition of shock in pediatric patients is critical, as their physiological signs may differ from adults. Establishing rapid vascular access and correcting hypothermia and acidosis are essential steps in pediatric DCR.[49, 57]

Implementation, Outcomes, and Future Challenges

The implementation of DCR strategies has been shown to significantly improve outcomes in trauma patients. The use of massive transfusion protocols (MTP) and high ratios of plasma and platelets to red blood cells (1:1:1) have been associated with

decreased mortality.[48, 58] Additionally, the integration of damage control surgery (DCS) with DCR allows for a structured intervention that begins immediately after initial assessment and continues through to the ICU.[54, 58]

Despite its benefits, DCR faces several challenges:

1. Resource Availability: Access to blood products and rapid testing can be limited in some settings.
2. Standardization of Protocols: Variability in DCR implementation across institutions can lead to inconsistent outcomes.
3. Ongoing Research: Continued investigation into optimal resuscitation strategies, including the role of novel anticoagulants and blood substitutes, is necessary.

Conclusion

Damage control resuscitation is a vital component in the management of trauma patients with severe hemorrhage. By focusing on rapid hemorrhage control, balanced transfusion strategies, and the prevention of acidosis, hypothermia, hypocalcemia and coagulopathy, DCR improves the chances of survival and recovery. Continued research and refinement of these strategies will further enhance the care of both adult and pediatric trauma patients.

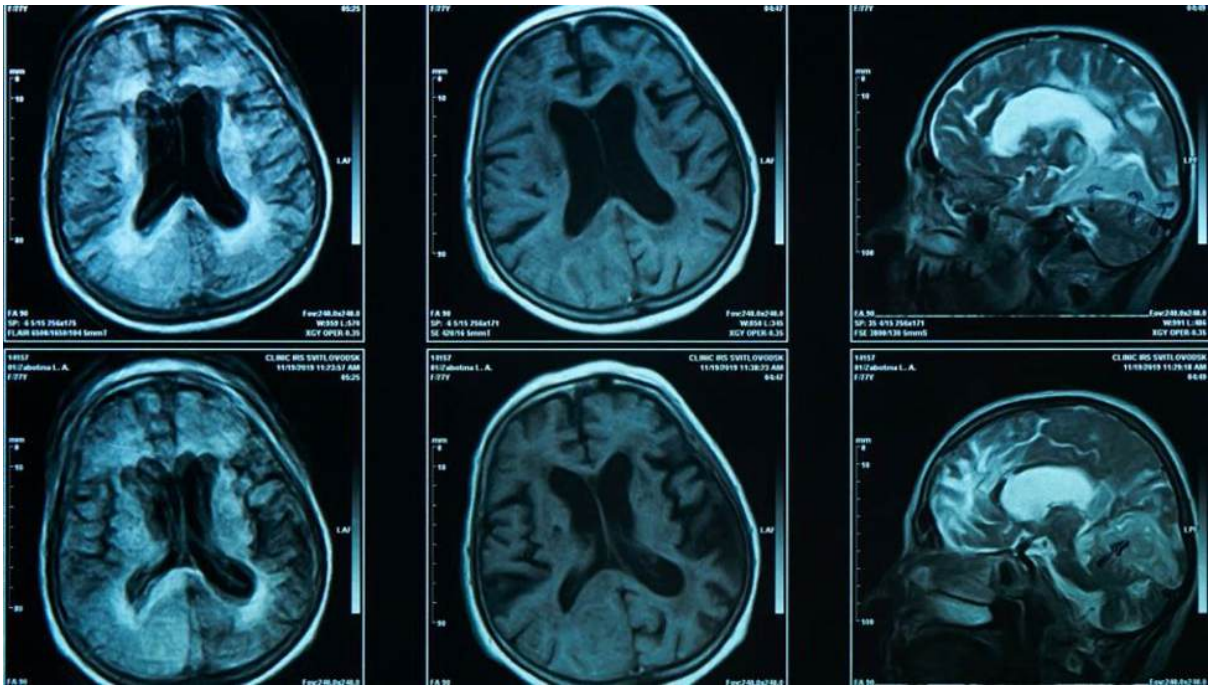
Essential Recommendations

- Use blood products early to avoid large crystalloid resuscitation.
- Always correct calcium in massive transfusion.
- Avoid permissive hypotension in traumatic brain injury.

Desirable Recommendations

- Damage Control Resuscitation and Damage Control Surgery is an integrated strategy and should be used where facilities allow DCS.
- Use Resuscitative Balloon Occlusion of the Aorta (REBOA) if available to decrease use of blood products for resuscitation.

Chapter 4: Traumatic Brain Injury (TBI)



Traumatic Brain Injury (TBI) is a non-degenerative, non-congenital insult to the brain due to an external mechanical force. It can lead to a possible permanent or temporary impairment of cognitive, physical or psychosocial functions, with a dissociated mentation .[85] TBI is the leading cause of morbidity and mortality worldwide.[86] In the USA alone, TBI accounts for 1.4% of all Emergency department visits and 4.8% of injuries presenting to the ED .[87] The National Hospital Discharge survey deduced, 10.6% increase in TBI related admissions from 1995-2010, likely due to increase in falls amongst the elderly and the use of anticoagulant therapy.[88, 89] Falls now supersede Road Traffic Accidents (RTA) with 61% of TBI above the age of 60 years being due to fall. Moreover, patients above the age of 75 years have the highest rates of TBI related hospitalizations and death.[87] Men are twice as likely as women to sustain a TBI. The Male:Female is 3.4:1 for TBI, 6:1 for firearm related injuries and 2.4:1 for RTA's.

Definitions

TBI is an umbrella term for a group of injuries with several classification schemes, depending on mechanism, primary pathology or clinical severity. For instance, Glasgow Coma Scale (GCS), defines the severity of TBI within 48 hours of injury with severe TBI – 3-8, moderate TBI – 9 – 12, mild TBI – 13 – 15. Ranchos Los amigos scale of cognitive functioning depicts the severity of deficit in cognitive functioning and is defined as follows –

Level I = No response
Level II = Generalized response
Level III = Localized response
Level IV = Confused-agitated
Level V = Confused-inappropriate
Level VI = Confused-appropriate
Level VII = Automatic-appropriate
Level VIII = Purposeful-appropriate

TBI defined by the Head Injury Interdisciplinary Special Interest group of American Congress of Rehabilitation Medicine, defines mild head injury as a "traumatically induced physiologic disruption of brain function as manifested by one of the following –

- Any period of Loss of Consciousness
- Any loss of memory for events immediately before or after the accident
- Any alteration in mental state at the time of accident
- Focal neurological deficits which may or may not be transient "(85, 87)

Other criteria used –

Mild TBI	Moderate TBI
GCS >12	GCS 9-12
No abnormalities on CT scan	Abnormal CT scan findings (89)
No operative lesions	Operative intracranial lesions
Length of hospital stay <48 hours	Length of stay in hospital at least 48 hours

Mechanic categories define TBI into closed head / penetrating or blast injuries. Closed or blunt injuries result due to direct impact outside the skull and can result in

deceleration / acceleration or rotational forces. Penetrating injuries as the name suggests, result from a direct penetration of any object directly into the brain parenchyma most commonly gun shots / shrapnel. Blast injuries are a slightly newer commodity to TBI most commonly incurred by military personnel due to high grade explosives causing thermal, mechanical and electromagnetic energy transfer. These often cause early brain edema, Subarachnoid hemorrhage and vasospasm (91)

Classification of TBI based on structural pathological conditions – (images in appendix)

INJURIES	**PATHOPHYSIOLOGY**	**IMAGING CHARACTERISTICS**
CEREBRAL CONTUSION (Image 1)	COUP / CONTRE COUP	INFERIOR FRONTAL AND ANTERIOR INFERIOR TEMPORAL LOBES
SUB DURAL HEMORRHAGE (Image 2)	BRIDGING VEINS	CONCAVE / CROSSES SUTURE LINES
EPIDURAL HEMORRHAGE (Image 3)	MIDDLE MENINGEAL ARTERY / VEIN, DIPLOIC VEIN, DURAL VENOUS SINUS INJURY	CONVEX / DOES NOT CROSS SUTURE LINES / ADJACENT TO SKULL FRACTURES
SUBARACHNOID HEMORRHAGE (Image 4)	PIAL VESSEL INJURY	CEREBRAL CONVEXITIES / BASAL CISTERNS
INTRAVENTRICULAR HEMORRHAGE (Image 5)	SUBEPENDYMAL VEIN INJURY / EXTENSION FROM INTRAPARENCHYMAL HEMORRHAGE/ REDISTRIBUTION FROM SUBARACHNOID SPACE	LAYERING WITHIN VENTRICULAR SYSTEMS

DIFFUSE AXONAL INJURIES (Image 6)	AXONAL SHEAR TEAR	CORTICAL WHITE MATTER JUNCTION MIDLINE WHITE MATTER STRUCTURES
CEREBRAL EDEMA (Image 7)	CEREBRAL AUTOREGULATION/ BBB DISRUPTION	CYTOTOXIC OR VASOGENIC EDEMA

Diffuse Axonal Injury patients generally present to the ED in coma, do not have a very high Intracranial pressure, and have unfavorable outcomes. (91)

CT venography and CT arteriography are also utilized to look for isolated vascular damage. Aortic dissections and luminal sac emboli can also occur leading to infarcts in the brain, in a delayed manner. The Monroe – Kellie doctrine states that, there is a fixed volume in the Intracranial vault, consisting of three compartments – blood, Cerebrospinal Fluid (CSF) and brain. If there is an increase in the volume of one compartment, the other two compartments must reduce their volume to compensate and maintain normal Intracranial pressure (ICP). An autoregulatory mechanism sets in to regulate brain perfusion through a varied Mean Arterial Pressure. However, in case any of the compensatory mechanisms fail, elevated ICP can cause herniation syndromes and cerebral ischemia. In TBI, this can most significantly be due to hemorrhage or cerebral edema. (85, 86, 91)

TBI in children also needs to be kept in mind. Since children may not be able to express to their caregiver after a TBI. It is imperative for adults to pay careful attention in children and look for the following symptoms or signs (92) –

- Changes in eating or nursing habits
- Persistent crying, irritability and inability to be consoled
- Changes in ability to pay attention
- Lack of interest in a favourite sport / play
- Changes in sleep patterns
- Seizures
- Sadness
- Loss of established skill
- Loss of balance
- Vomiting

Serial Examinations

Clinical exam is paramount in TBI cases and should be tracked over time, to check for improvement or decline. Pupillary size, reaction to light and motor examination is paramount to exam findings. Anisocoria is defined as a difference of more than 1mm between pupil size. Trans tentorial herniation compressing the ipsilateral oculomotor nerve produces a unilateral dilated and non-reactive pupil. Traumatic cord dissection can result in Horner's syndrome leading to ipsilateral miosis, anhidrosis and ptosis. (85, 89)

As discussed earlier, GCS and grading can be used to evaluate the individual. Increase in Intra Cranial Pressure, such as asymmetric, dilated pupils, GCS decline of more than 2 points and Cushing's Reflex (Hypertension/ Bradycardia). Any sudden change in condition or appearance of new symptoms require prompt treatment, such as head end elevation, hyperventilation, osmotic therapy. (85, 93)

Primary vs Secondary Injuries

TBI can manifest clinically from concussion to coma to death. Primary injury occurs at the moment of trauma and secondary injury occurs immediately after trauma that produces lingering effects. (85, 88)

The physical mechanisms of brain injury are classified according to the following category –

- Impact loading – collision of head with solid object at a tangible speed
- Impulsive loading – sudden motion without significant impact
- Static or quasistatic loading – loading in which the effect of speed of occurrence may not be significant

The three basic types of tissue deformation are as follows –

- Compressive – tissue compression
- Tensile – tissue stretching
- Shear – tissue distortion with tissue sliding

Types of primary injuries

- Skull fractures – vault or basilar skull fractures. Hematomas, cranial nerve damage and increased brain injury are often associated. Vault fractures tend to

be linear in nature and extend into sinuses. Injuries can be open or closed, where open fractures allow communication with the outside compartment. A simple fracture is defined as having one bone fragment, whereas a compound exists with two or more bone fractures. Basal skull fractures are often associated with dissipated forces and may be associated with nerve or auditory-vestibular damage.

- Auditory / vestibular dysfunction – BPPV can occur when trauma causes dislodgement of calcium oxalate crystals. Tympanic perforation, hemotympanum, ossicular disruption can occur and cause conductive deafness. Sensorineural hearing loss maybe secondary to inner ear defects.

- Intracranial hemorrhages – epidural hematomas occur often with associated fractures bones. Disruption of middle meningeal artery can lead to rapid neurological deterioration. Subdural hematomas have a mortality of approx. 60-80%. (94) Intravascular hemorrhage tends to occur with severe TBI and intracranial hemorrhages occur with disruption of deep veins. Subarachnoid hemorrhages occur with aneurysmal ruptures due to laceration of superficial microvasculature. It may also lead to communicating hydrocephalus incase of clots disrupting fourth ventricle flow.

- Coup and contrecoup contusions – combination of vascular and tissue damage. Coup contusions occur at the site of impact created due to negative pressure impact on the skull. Contrecoup contusions occur in a similar fashion, but on the exact opposite side of the injury. (95)

- Concussion – caused by the deformity of the deep structure of the brain leading to widespread neurologic dysfunction that may result in impaired consciousness. (96)

- Diffuse axonal injury – extensive generalized damage to white matter in the brain. Strains on the falx and tentorium due to high-speed acceleration – deceleration produced by lateral motions of the head may cause injury. Also, could occur due to ischemia. Gennarelli graded the injuries based on – grade I – axonal injury in parasagittal white matter or cerebral hemispheres. Grade II – grade I plus lesions in corpus callosum. Grade III – grade II plus focal lesion in cerebral peduncle. (97)

- Penetrating head injuries – the energy dissipated on entry, is equal to ½ the mass X velocity squared, therefore high velocity bullets tend to cause the most extensive damage. (85, 86)

Secondary injuries

These injuries develop from further cellular level damage once the primary head injury has occurred, and may develop over a period of hours or days. (98)

- Excitatory amino acids – glutamate and aspartate are significantly elevated. EAA's can cause cell swelling, vacuolization and cell death. Also cause influx of sodium and chloride can cause acute neuronal swelling. (99)
- Endogenous Opioid Peptides – they worsen neurologic damage by modulating the presynaptic release of EAA neurotransmitters. Activation of muscarinic cholinergic systems in the rostral pons mediates behavioural suppression. Increases extracellular potassium leading to edema. (100)
- Increased ICP – the severity of the TBI tends to increase due to heightened ICP, especially if the pressure increases above 40mm Hg. Cerebral hypoxia, ischemia, edema, hydrocephalus and brain herniation can also occur. (101)
- Cerebral edema – can be caused due to the above mentioned neurotransmitters. Disruption of blood brain barriers with impairment of vasomotor autoregulation, causing dilatation of blood vessels.
- Hydrocephalus – the communicating type is more common. Mostly occurs due to the presence of blood products that causes obstruction of the flow of CSF in the subarachnoid space and the absorption of CSF. The non communicating type, is caused by a blood clot obstructing the interventricular foramen, third ventricle, cerebral aqueduct or fourth ventricle. (102)
- Chronic traumatic Encephalopathy – occurs mostly in persons with repetitive brain trauma, like boxers and football players, leading to a progressively degenerative disease. Degenerative changes occur months after the initial damage has occurred. Atrophy of the medial temporal lobe, thalamus, mammillary bodies. The condition is also characterized by dilated ventricles and fenestration of the cavum septum pellucidum. Symptoms include memory loss, confusion, impaired judgment, reduced impulse control, aggression, explosive anger, depression and progressive dementia. (103, 104)

Imaging Modalities

- A non-contrast Computed Tomography (CT) scan should be obtained once the patient's ABC's have been stabilized i.e. Airway, Breathing and Circulation. This is in adjunct to a comprehensive neurological exam, as discussed. CT scans are preferred due to their rapidity and detection of early bleeds and fractures. (91) The primary CT identifies early lesions that may require urgent surgical interventions. Hematomas are common in moderate to severe TBI occurring in 25-30% cases. (91, 95, 97) They also occur approx. 15% in patients with a GCS score of 13-14. (105)
- Patients who experience head trauma, also are at a significant risk of spinal injury, with cervical spine injuries occurring at approx. 6.5% in one recent systematic review, (106) thus making C-spine CT scans a must with traumatic head injuries.
- Hematoma expansion is another concern in TBI and 16% cases demonstrate deterioration on later scan, in patients with diffuse injury on CT scans. (107) Patients with severe (GCS 3-8) and moderate (GCS 9-12) TBI have a 43% and 25% progression. (108) Lesion growth of more than 5cm, effacement of basal cisterns on initial CT and worsening GCS before repeat scan are predictors of late surgical evacuation. (109)
- Magnetic Resonance Imaging (MRI) is not really recommended in an acute setting due to its clearance requirements for metallics and lengthiness of procedure. Its usage is mostly in the subacute or chronic phases in order to deduce DAI and other complications of TBI. CT or MR venography / angiography should be considered to evaluate vascular injury when there is penetrating head trauma / fracture over a venous sinus, selected C-spine injuries or unexplained neurological deficits. (110)

Resuscitation and Management

Appropriate triaging of TBI patients is critical. As expected, the ABCs of primary survey should be followed along with stabilizing the C-spine. Intubation should be considered for any comatose patient with a severe TBI (GCS<9) or in moderate TBI (GCS 9-12) with

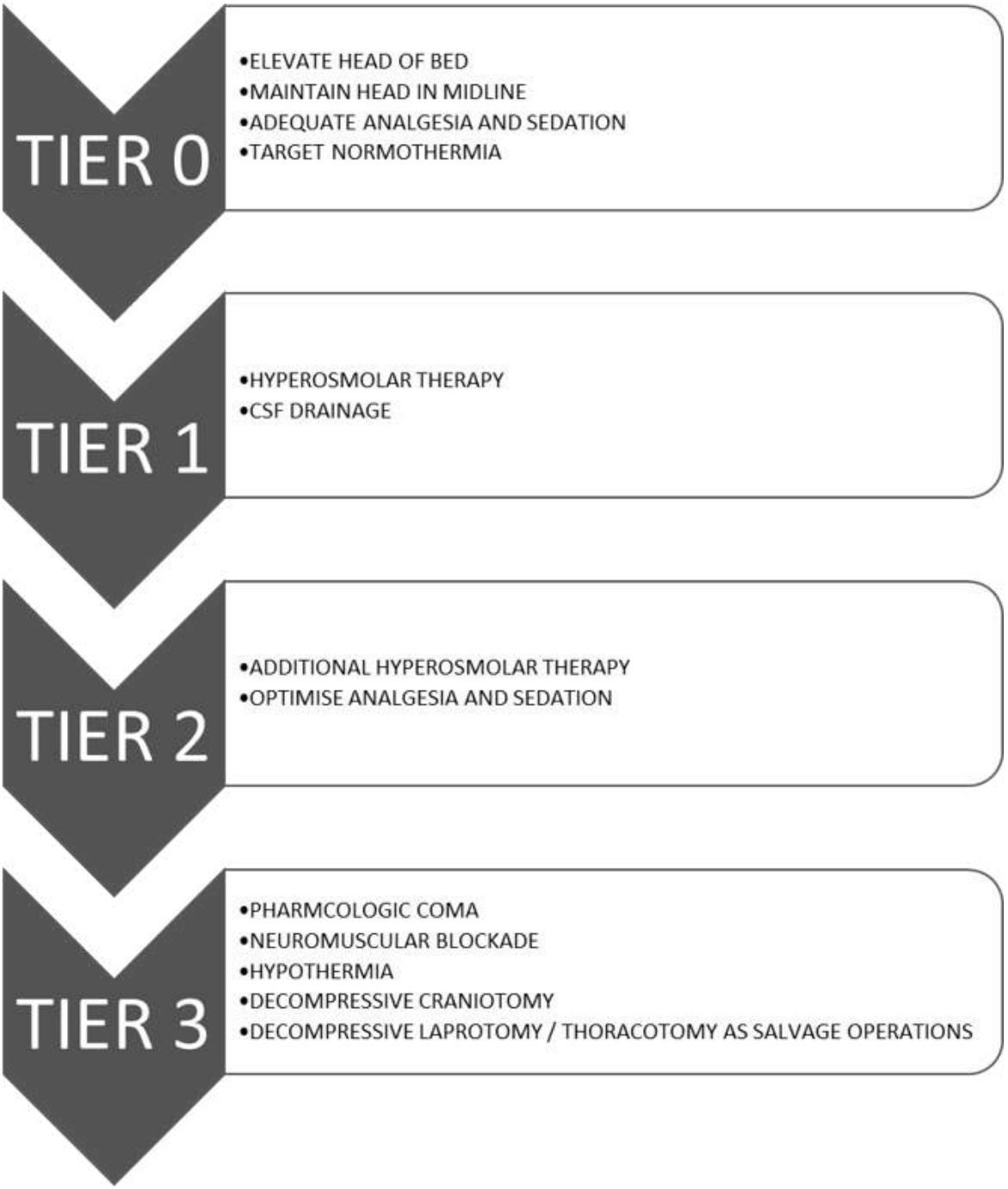
significant extracranial injuries, agitation and declining mentation (26,27). Brain injured patients have a decreased respiratory drive and plummeting mental status hence requiring placement of a secure airway. Rapid Sequence Intubation is generally used with proper pre-oxygenation of 100% FiO2 in order to reduce secondary hypoxic damage to the brain. Maintaining In Line Stabilisation (MILS) should also be properly established while intubating. The reflex sympathetic response from intubation is of concern as it can increase the ICP. Pretreatment with Fentanyl can be used but with caution as to not cause shock in patients especially in multimodal trauma. Ketamine and Etomidate are hemodynamically neutral agents and are thus preferred. (111). All previous myths about ketamine causing increased ICP have been debunked. (112)

Both hypotension and hypoxia are associated with worse outcomes in the emergency. A single episode of hypotension Systolic Blood Pressure (SBP) <90 mmHg occurring at the time of injury through resuscitation has been associated with doubled mortality and patients whose hypotension wasn't corrected have worsened outcomes(113, 114). Hypotonic fluids like D5NS and half Normal Saline should be avoided as they can worsen the cerebral edema(115). Current evidence now shows a target SBP ≥110 mmHg in 15–49-year-olds and SBP ≥100 mmHg in 50–69-year-olds. It should be noted that future studies are needed on whether accepting Mean Arterial Pressure (MAP) goals are a more appropriate end point (116).

Coagulopathy is common in TBI with an overall prevalence of 33% and is widely recognized as a risk factor of morbidity and mortality (117). In a retrospective study of 3000 patients, with isolated TBI, coagulopathy was associated with higher rates of craniotomies, single and multiple organ failures, increased length of stay and an adjusted odds ratio for hospital mortality of 2.97 (118). The pathophysiology is slightly complex due to the release of tissue factor into the systemic circulation and the activation of Protein C leading to Disseminated Intravascular Coagulopathy (DIC) and platelet dysfunction (117, 118) . The CRASH -3 trials showed the efficacy of Tranexamic Acid as an antifibrinolytic agent, to be safe and efficacious , (91, 110, 117, 118) but unfortunately the current guidelines have not incorporated this into practice for TBI.

Management of TBI has become more integrated and standardized over time to national and international guidelines. The central goal of TBI management is maintaining normal ICP and Cerebral Perfusion Pressure (CPP). CPP is defined as MAP – ICP, with a normal value lying between 60-80 mm Hg. Cerebral autoregulation is impaired or absent in 49-87% of patients with severe TBI, such that a fall in SBP causes a decrease in CPP, decreased Cerebral Blood Flow and ultimately ischemia (119). Ischemia can also occur in settings of increased ICP and brain herniation. Current recommendations for CPP are 60-70 mm Hg (120).

The figure below shows the tiered approach to management of Intracranial Hypertension.



Another question that is frequently asked is, do all patients of TBI need seizure prophylaxis? The Brain Trauma Foundation estimates that in severe TBI, clinical seizures occur in up to 12% of patients and subclinical seizures occur in 20-25% patients. (109, 110, 116, 117) Post traumatic seizures (PTS) are defined as either early – occurring within 1 week of injury, or late – occurring after 1 week of injury. Early PTS can contribute to secondary brain injury by causing increased metabolic demand, increased ICP, release of neurotransmitters and eventual development of hippocampal atrophy .(99 - 102, 116). Treatment with antiepileptics sadly comes with

its own side effects. A landmark RCT assigned patients to phenytoin vs placebo group, found a significant reduction in early but not late PTS (121). This concern for negative effects of phenytoin have led to the interest in evaluating newer antiepileptics. A 2013 RCT of 813 patients found no clinical difference in PTS between phenytoin and levetiracetam in TBI (122) . The American Academy of Neurology (AAN) and BTF provide level IIA recommendation for prophylactic phenytoin or levetiracetam for 7 days to decrease the incidence of early PTS (121, 122). Prophylaxis should be stopped if no seizures have occurred during this time. Given the high rate of Non convulsive status epilepticus and subclinical seizures, continuous electroencephalographic monitoring should go on for comatose patients.

Prognosis

Accurately predicting the outcomes of TBI is very difficult. The CRASH and IMPACT models are most widely used for prognostication and are based on data of two large clinical trials (123). Each model predicts clinical mortality in the next 6 months along with functional outcomes, based on clinical factors – age, pupil response, GCS motor score, CT findings and lab values.

The Transforming Research and Clinical Knowledge in Traumatic Brain Injury (TRACK-TBI) is a large prospective cohort study that enrolled patients presenting to the ED within 24 hours of injury at 18 level 1 trauma centres in the USA and followed them for 12 months post hospitalisation. It showed 484 (18%) out of 2679 individuals with either moderate to severe TBI. Amongst those patients, 19.3% with severe TBI, 32% with moderate TBI reported no disability at 12 months period. Amongst participants in vegetative states, at 2 weeks, 62/79 (78%) regained consciousness and 25% of them gained orientation at 12 months. This data suggests that even patients with severe TBI may experience a favourable outcome in the long term and clinicians should be wary of prognosticating poor outcomes at the two weeks point (124).

Conclusion

As seen by data, TBI seems to be a leading cause of morbidity and mortality worldwide with higher rates of hospitalizations in the higher age groups, and a higher chance of death. Management in the acute phase is maintaining the ABC's of the patient in the ED and stabilizing C-spine along with head injuries. A special concern to prevent secondary brain injury is also raised thus preventing hypotension, hypoxia and increased ICP. Unfortunately, Tranexamic acid and hyperosmolar agent usage has not been

incorporated into the day-to-day practice. Care should be taken that the hyperventilation initiated on ventilator patients, should be left on for a long time as it can lead to cerebral ischemia, due to chronic vasoconstriction. Sudden decline in GCS by 2 points or more, pupillary dilatation or becoming non-reactive and worsening motor posturing should be immediately acted upon and brain herniation should be at the top of the differential. Another aspect, that is more psycho-social than medical is for clinicians to not have early prognostications as it has been shown that patients with severe TBI even can have a favorable outcome at the 12-month period.

Essential Recommendations

- Severity stratification (GCS + imaging + LOS) is essential to decide appropriate treatment for TBI, the leading global cause of death and disability.
- Whilst primary injury is irreversible; the main aim of resuscitation is to prevent secondary injury. Hypotension (SBP <90 mmHg) or hypoxia doubles mortality.
- Coagulopathy affects about 33% of TBI patients and should be routinely screened.
- Aggressively treat raised intracranial pressure. Monro–Kellie doctrine governs TBI physiology: hemorrhage or edema overwhelms compensatory mechanisms → ICP rise, ischemia, herniation
- CT scan is the acute imaging modality of choice and should be ordered as soon as possible.
- ICP and CPP-directed management is central and transfer to a critical care unit able to measure these parameters will improve prognosis.
- Diffuse Axonal Injury often presents with coma and normal ICP.
- Seizure prophylaxis for a total of 7 days should be started as this reduces early post-traumatic seizures.

Appendix – Images for Classification of TBI based on structural pathological conditions:

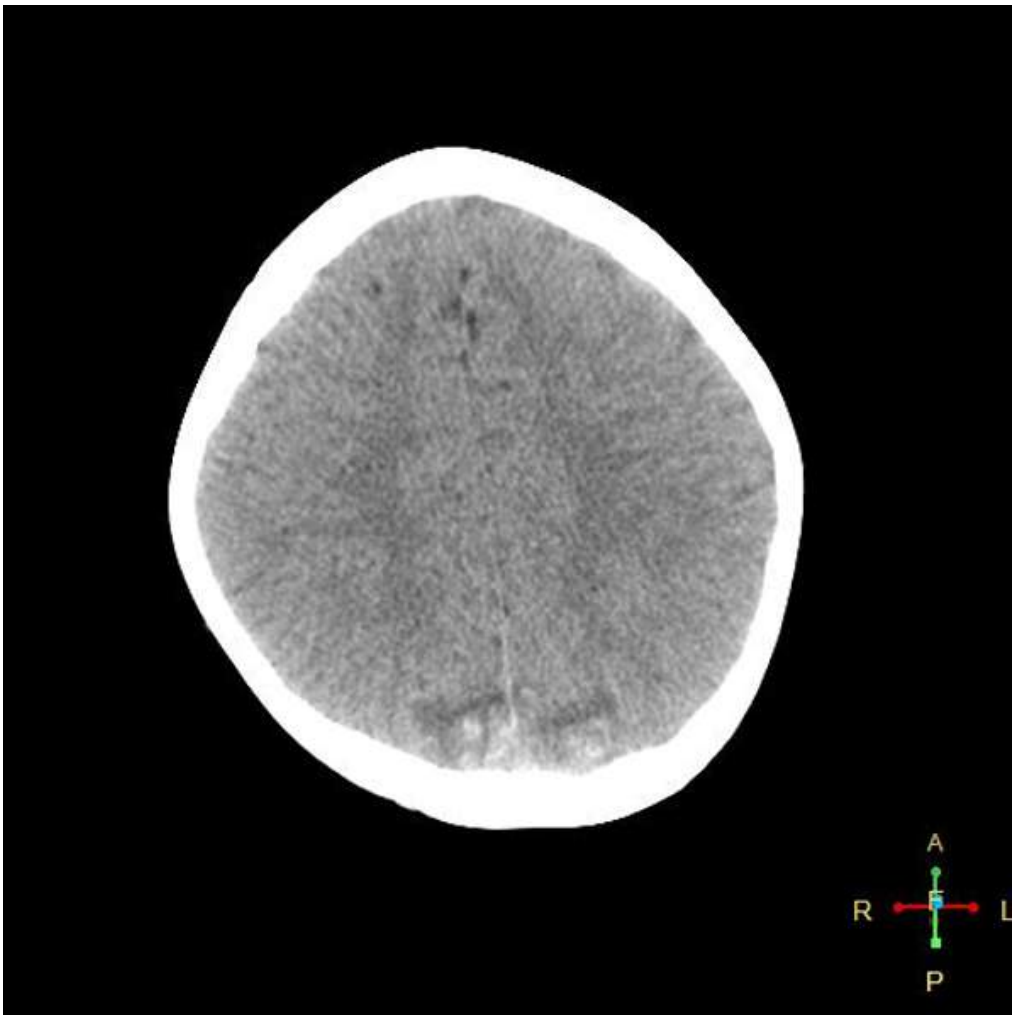


Image 1: CT demonstrating bilateral hemorrhagic cerebral contusions in the posterior parietal lobes opposite to the site of trauma. **Case courtesy of Heba Abdelmonem, Radiopaedia.org, rID: 48869**

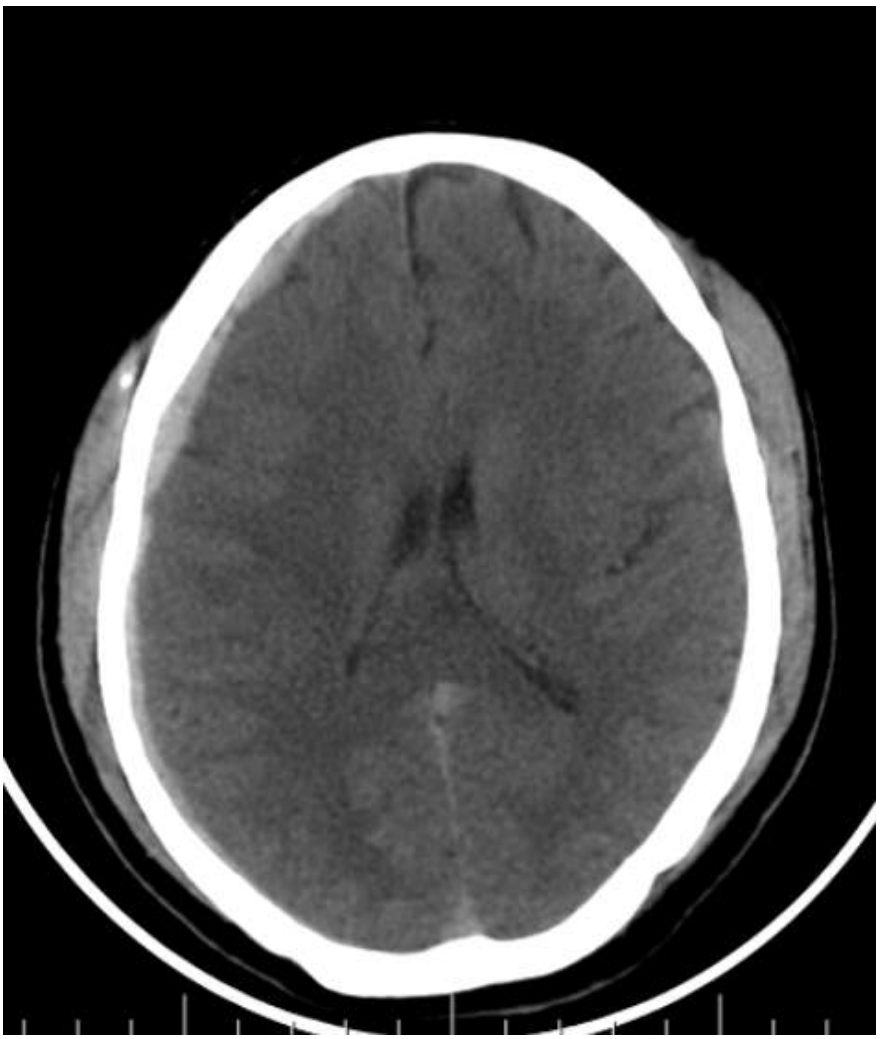


Image 2: CT demonstrating right front-temporo-parietal convex hyperdense subdural hemorrhage. **Case courtesy of Aditya Shetty, Radiopaedia.org, rID: 27889**

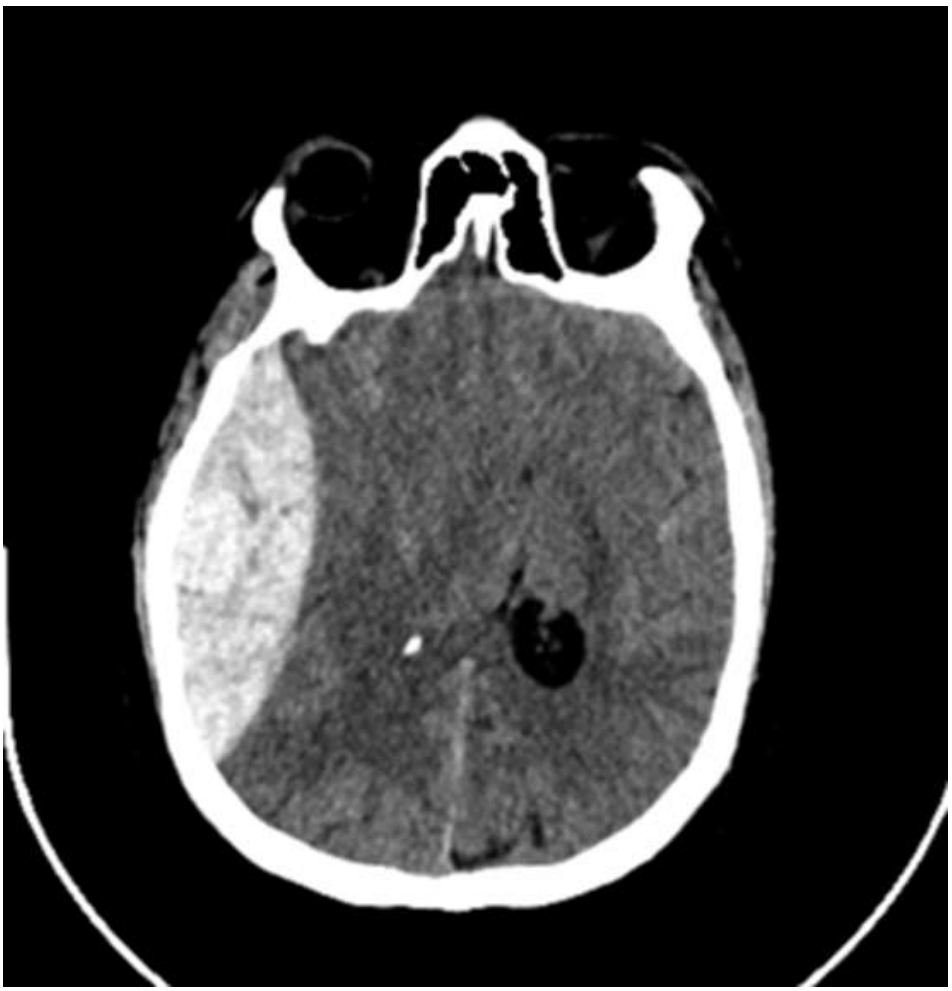


Image 3: CT demonstrating biconvex shaped hyperdense epidural hemorrhage with midline shift. **Case courtesy of Keshaw Kumar, Radiopaedia.org, rID: 165693**

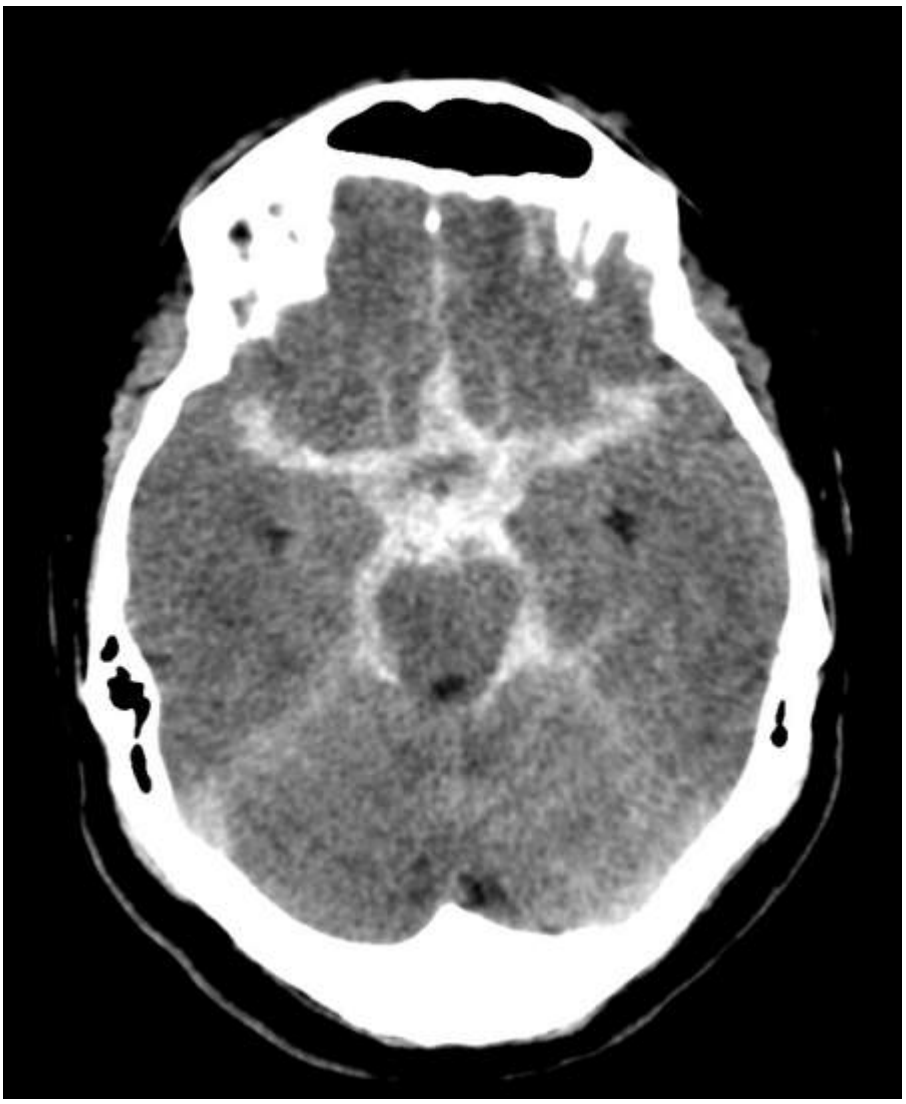


Image 4: CT demonstrating diffuse Subarachnoid hemorrhage in all basal cisterns, bilateral sylvian fissure and interhemispheric fissure. **Case courtesy of David Puyó, Radiopaedia.org, rID: 22377**

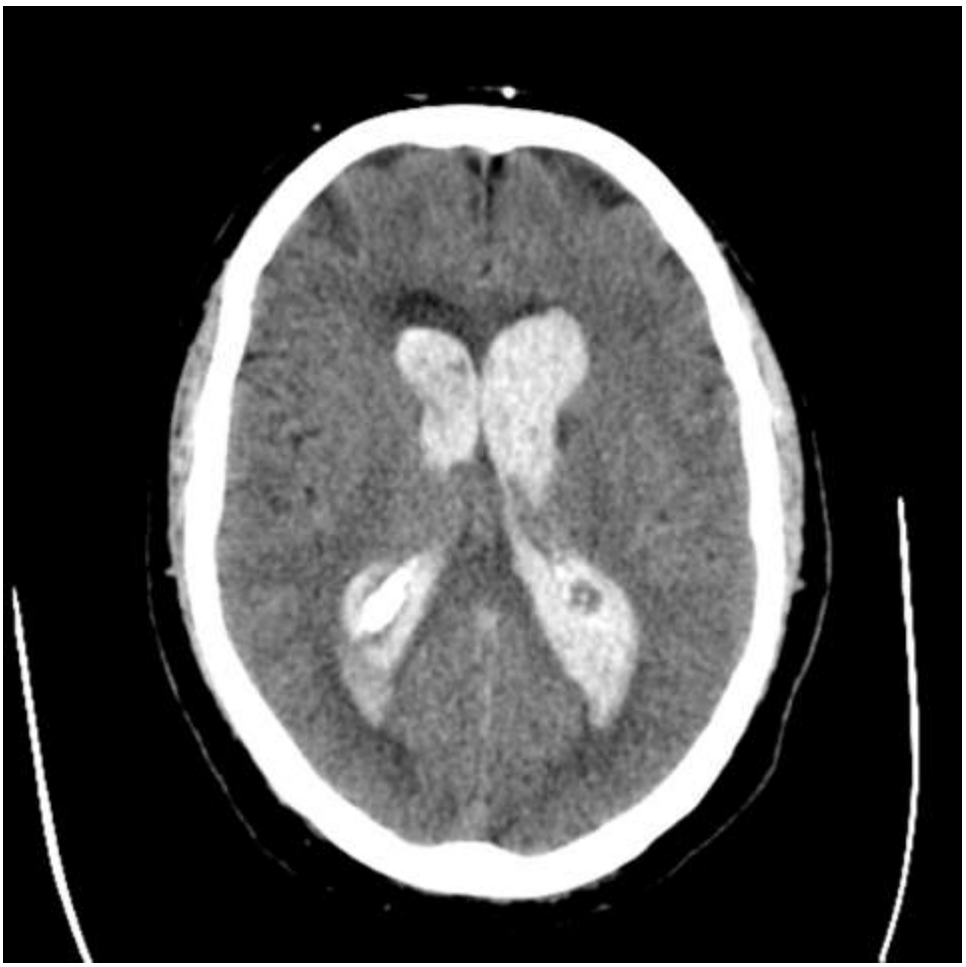


Image 5: CT demonstrating hyperdense lateral ventricles. **Case courtesy of David Puyó Vera, Radiopaedia.org, rID: 36523**

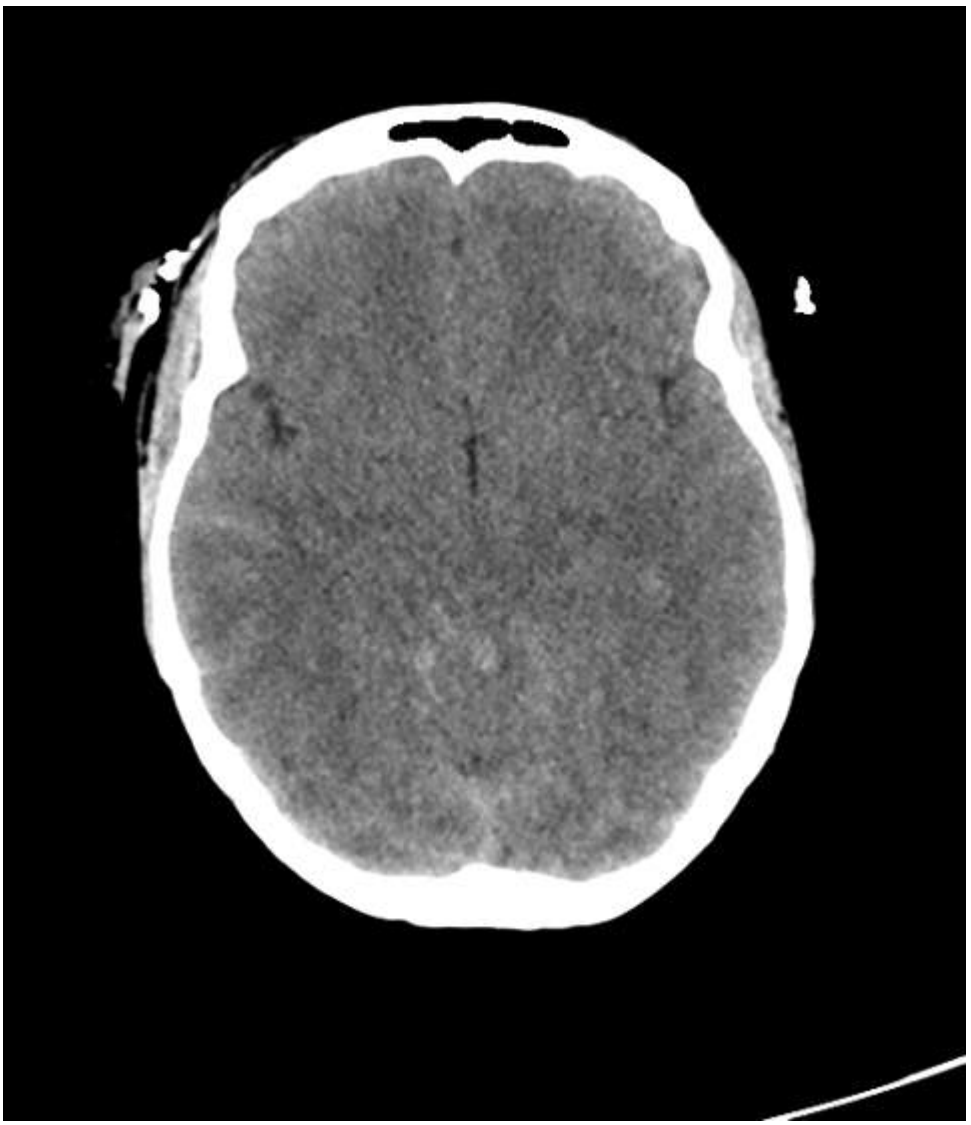


Image 6: CT demonstrating Multiple small foci of intraparenchymal hemorrhage scattered throughout both hemispheres consistent with severe traumatic brain injuries related to shearing forces. **Case courtesy of Bruno Di Muzio, Radiopaedia.org, rID: 56466**

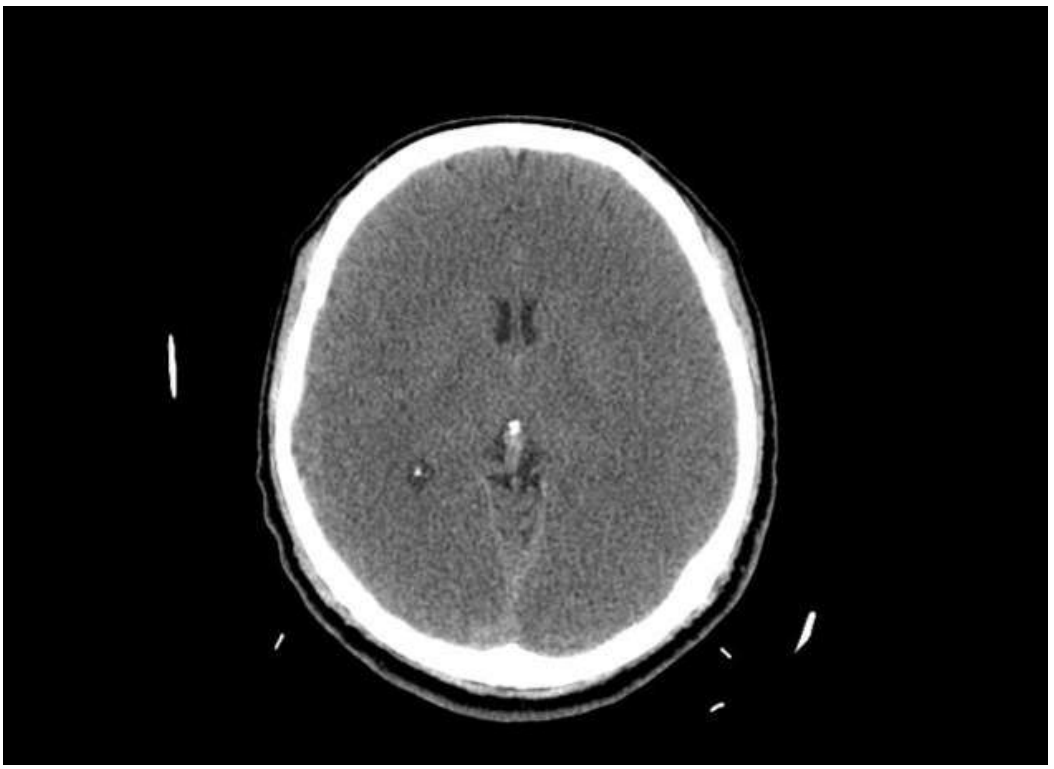
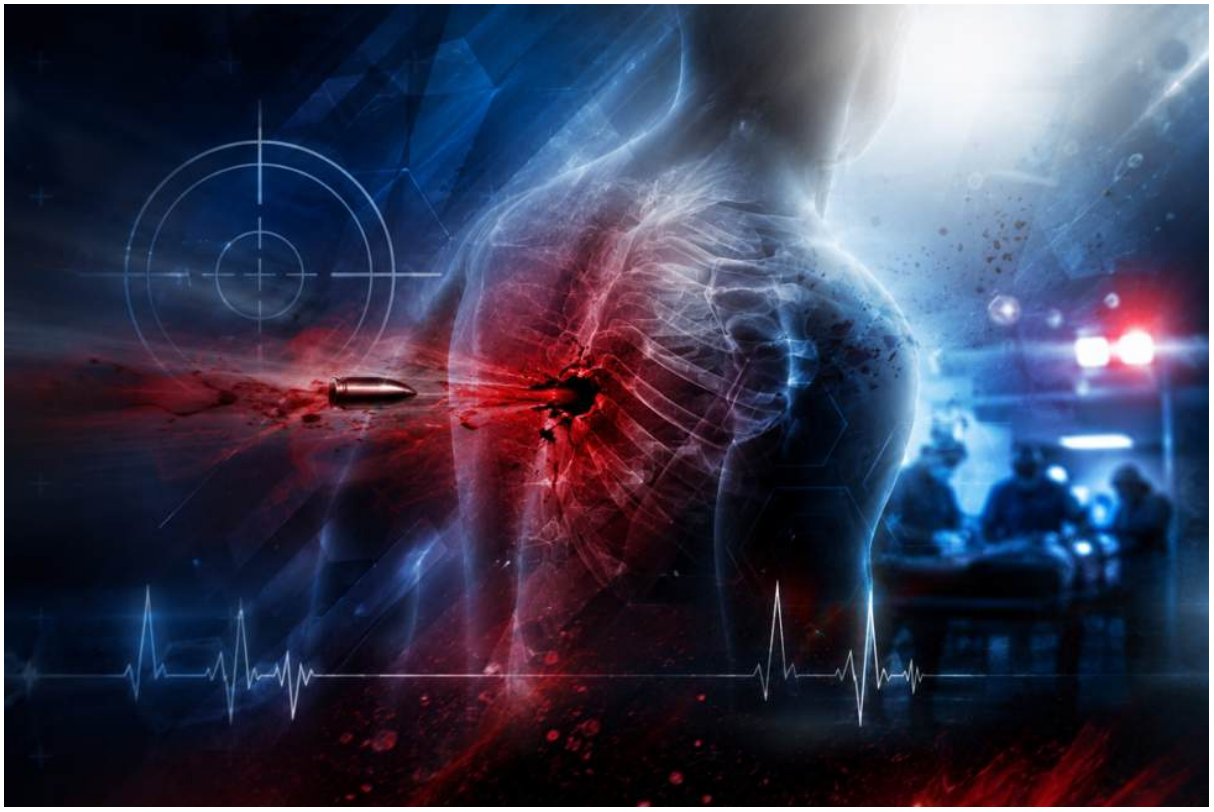


Image 7: CT demonstrating Compressed sulci, grey and white matter differentiation is lost. **Thin and symmetric ventricles. Case courtesy of Orsolya Lajos, Radiopaedia.org, rID: 8251**

Chapter 5: Resuscitation in Penetrating Trauma



Penetrating trauma, a wound that pierces the skin and enters the underlying tissue, is one of the two primary categories of trauma.(125). Depending on the region of the world, penetrating trauma comprises 4-16% of total trauma.(126). Although less common than blunt trauma, penetrating trauma is associated with higher prehospital and ED mortality. (127, 128). It is more likely to require operative intervention and procedures, including laparotomy and tube thoracostomy. (129,130). Increasingly, the work up of penetrating trauma patients in high resource settings includes axial imaging, to better diagnose and manage occult injuries caused by the unpredictable path of bullets and other projectiles. (131). Given the above, it is vital for all emergency care providers to understand the differences to consider in the resuscitation of patients with penetrating trauma, and to move quickly to provide the specialized care for these time-critical patients. The care of patients with a penetrating brain injury will be covered in another chapter.

The primary, and likely most time-sensitive cause of mortality in penetrating trauma is hemorrhagic shock. Trauma induced coagulopathy, though a later complication, is also

a significant issue faced by emergency physicians. In settings where available, the administration of prehospital transfusion in patients with penetrating trauma and hypotension appears to provide a mortality benefit, but only if it does not delay transport times (132). A recent South African study found that guiding resuscitation in penetrating trauma with the use of thromboelastography (TEG) led to shorter ICU stays and fewer complications (133) . This lab test, however, is generally not available in LMIC. In such settings, it is reasonable to consider a 1:2 RBC: plasma ratio for massive transfusion, as this may be beneficial in penetrating trauma (134, 135). Early and balanced damage control resuscitation is supported by the current literature on penetrating trauma resuscitation (125, 135, 136).

Increasingly, the use of group O whole blood in the resuscitation of penetrating trauma patients has gained favor (137). If blood products of any kind are not available, judicious use of crystalloid is a reasonable alternative for volume expansion. The literature does not currently support the use of colloid volume expanders or hypertonic saline (138, 139) . There is some literature to support using a different threshold for hypotension in penetrating trauma in contrast to other forms of trauma, with one study suggesting an SBP <110 mmHg be triaged to higher level trauma care. (140). Damage control resuscitation is covered in detail in a previous chapter. In terms of priorities and timing of the airway in exsanguinating patients, it appears that prioritizing circulation over airway yields mortality and morbidity benefits, especially given the worse outcomes associated with post-intubation hypotension in trauma patients (141), and is discussed additionally in the Airway chapter. In terms of prehospital care, there is evidence that suggests increased time on scene and prehospital procedures in lieu of a “scoop and run” approach has negative impacts on mortality in penetrating trauma. (142- 144)

Although the use of ultrasound in trauma resuscitation has generally focused on blunt trauma, especially the FAST exam, more recent research has shown its utility in penetrating trauma, especially in diagnosing hemopericardium, pneumothorax, hemothorax, and diaphragmatic injuries. (145)

Resuscitation by Location of Penetrating Trauma

Neck

Early securement of a definitive airway, hemorrhage control, and DCR continue to be vital in penetrating neck injury with hard signs of injury (including airway compromise, bubbling, expanding hematoma, ongoing bleeding, shock, hematemesis, or neuro deficit) (146 - 148). Current guidelines do not recommend routine spinal immobilization in penetrating trauma. (149)

Chest

Although the rates of ED thoracotomy have risen and fallen in some regions for penetrating, traumatic cardiac arrest, there is not good evidence that they have a significant impact on mortality. They are likely only indicated in a high resource setting, and even there, it is unclear if they provide a benefit (150). The World Trauma Association only recommends them for patients with penetrating torso trauma and less than 15 minutes of CPR, penetrating trauma to the neck or extremity with less than 5 minutes of prehospital CPR, and patients in profound refractory shock (151). There is ongoing controversy about whether signs of life need to be present for benefit. (152, 153).

Renal/Genitourinary System

The Urology literature has broadened substantially in the last ten years, instituting more multi-center studies, and even some randomized clinical trials. (154) The tendency to try to avoid nephrectomy, even in high grade trauma, and prefer non-operative management of renal and GU trauma continues to grow in popularity and evidentiary support (155).

Abdomen

The current guidelines from the Eastern Association for the Surgery of Trauma on penetrating abdominal wounds do not recommend surgery for patients with stab wounds who are hemodynamically stable and without signs of peritonitis or diffuse abdominal pain. They similarly do not recommend surgery for patients with abdominal gunshot wounds if they are tangential and there are no signs of peritonitis. They recommend 24 hours of observation and serial abdominal exams in these cases (129). Current guidelines also recommend 24 hours of prophylactic antibiotics in penetrating hollow viscus injury undergoing laparotomy, not all penetrating abdominal injury (156). In the last half of pregnancy, anterior abdominal penetrating trauma nearly always injures the uterus and fetus. Emergent cesarean or resuscitative hysterotomy should be considered as part of the trauma resuscitation. (157, 158)

Limbs

Current guidelines encourage the use of detailed physical exams and the use of Ankle Brachial Indices or Wrist Brachial Indices to assess for possible arterial injury. (159). These can guide management and in centers with access to CT angiography determine

if the study is warranted.(160). It is important to realize that initial physical exam can be normal in 5-15% of patients with extremity vascular injury. (161)

Challenges and Future Directions

Additional research is needed to clarify the utility and selection criteria for the use of whole blood in resuscitation, indications for thoracotomy, and the uses of damage control resuscitation by trauma mechanism and location. (162, 163). The use of TEE during penetrating trauma resuscitation is one new avenue for improving care of these critically ill patients. There are international efforts underway, from the WHO Global Alliance for the Care of the Injured, and other groups, to reduce the highly heterogeneous care of trauma patients, especially penetrating trauma patients, and in the realm of prehospital care (128, 164, 165). Establishing best practices is in order.

Essential Recommendations

- Prioritise circulation over airway in patients who are externally exsanguinating.
- Employ a rapid "scoop and run" approach in prehospital care.
- Initiate early and balanced damage control resuscitation using blood products in specific ratios.
- Utilize point-of-care ultrasound–It's not just for blunt trauma.

Desirable Recommendations

- Consider a lower blood pressure threshold for triage to a higher level of care given the often operative nature of penetrating trauma.
- Create a local working group to stay up to date with recent developments regarding penetrating trauma. Care of penetrating trauma by location is evolving rapidly.

Chapter 6 – CT vs Point of Care (POC) Investigations

Ultrasonography (USG) has considerable benefit in managing trauma patients at the bedside. It is easy to use, having no radiation exposure. It has demonstrated a sensitivity between 85% to 96% and specificity of 98% (166, 167). An experienced provider can be able to perform a FAST scan within 5 min. (168)

While FAST is easy to perform but it also has its limitations. Clinicians should know how to interpret and needs to be experienced in performing ultrasound evaluation. Sometimes bowel gas shadow interfere with the interpretation. Even presence of pneumomediastinum and pneumoperitoneum evaluation needs expertise. An advance imaging and serial e - Fast needs to be carried out in case to case basis. Radiologist help may require but he/she may not be available always in the night time . (169, 170)

When a patient is presenting with suspected blunt thoracoabdominal trauma and a positive POCUS findings is helpful for guiding the treatment decisions. This may not hold true in case of abdominal trauma. Even a negative POCUS assessment does not completely rules out injuries and must be verified by a reference test such as CT specially when the suspicion is high. Particularly in paediatric trauma, the sensitivity of POCUS is poor. A Cochrane review of point of care ultrasonography for diagnosing

thoracoabdominal injuries in patient with blunt injury by dirk stengel et al. concluded "Based on a small number of studies in a mixed population, POCS may have a higher sensitivity in chest injuries. This warrants larger, confirmatory trials to affirm the accuracy of POCS for diagnosing thoracic trauma". (171)

The European guideline on management of major bleeding and coagulopathy following trauma, in their 6th edition recommends the following:

- We suggest the use of pre-hospital ultrasonography (PHUS) for the detection of haemo-/pneumothorax, haemopericardium and/or free abdominal fluid in patients with thoracoabdominal injuries, if feasible without delaying transport (Grade 2B).
- We recommend the use of point-of-care ultrasonography (POCUS), including FAST, in patients with thoracoabdominal injuries (Grade 1C).
- We recommend early imaging using contrast-enhanced whole-body CT (WBCT) for the detection and identification of the type of injury and the potential source of bleeding (Grade 1B).

NIRS (Near-Infrared Spectroscopy) is a non-invasive, real-time point-of-care tool increasingly used in trauma and critical care settings. NIRS continuously measures tissue oxygen saturation (StO_2) using light in the near-infrared spectrum (700–1000 nm), most commonly in the brain (cerebral NIRS) and skeletal muscle (commonly use the thenar eminence or forearm). Maria et. al in "Near Infrared Spectroscopy in Traumatic Brain Injury" has pointed its utilisation in Traumatic brain Injury and adjustment of treatment by measuring brain tissue oxygen pressure (172). Hans J. Hennes Rt.al in their paper "Non-invasive detection of intra cerebral Haemorrhage using near infrared spectroscopy" has found early detection of intracerebral haemorrhage with this device. In their study they have identified Sensitivity 0.96, specificity 0.29. For sub dural haemorrhage sensitivity 0.96 and specificity 0.79. (173) A study published in Journal of Neurotrauma in 2012 regarding detection of intracranial haemorrhage in children has strongly recommended its benefit in evaluation of a child with possible ich. Sensitivity 1.0, Specificity 0.8. Positive predictive value 0.8 and negative predictive value 1.0. (174) A Systemic review and meta-analysis published recently highlighted the usefulness of NIRS in detecting traumatic intracerebral haematomas in paediatric as well as in adults in pre hospital triage setting. It has emphasised the high sensitivity , specificity makes it a broader tool to use by physician as well as paramedic in trauma scenario. (175)

Limitations of NIRS

- Skin pigmentation, oedema, or probe placement may affect accuracy
- Limited specificity in some conditions (e.g., sepsis vs trauma)
- Operator experience still matters (for placement and interpretation)
- Cannot localise bleeding or structural damage (unlike CT)

Utility of Point of Care (POC) Testing:

In the Emergency Department (ED), especially for trauma patients, point-of-care tests (POCTs) can provide rapid bedside diagnostic insights to guide immediate management before or alongside imaging like CT scans. In trauma settings, POC testing, especially ultrasonography, lactate, and coagulation parameters, offers rapid bedside insights that complement and sometimes outperform CT scan in the unstable patient. Whilst acknowledging CT scanning is the gold standard for diagnosing many traumatic injuries (e.g. Intra-abdominal bleeding, diaphragm rupture, pneumothorax, solid organ lacerations) due to its high accuracy.

- Point of care tests which usually can be done in Emergency settings includes-POCS-FAST/EFAST, ABG/VBG, TEG, LACTATE, NIRS-For brain and skeletal muscle.
- POCUS-such as the FAST or eFAST is a rapid bedside tool, radiation-free and useful in unstable patients or during resuscitation.

Benefits of POCTs without CT Scan in Trauma

- Immediate diagnosis of life-threatening injuries
- Guiding resuscitation without delay
- Safe for hemodynamically unstable patients
- Detects coagulopathy and guides transfusion
- Useful in resource-limited or prehospital settings
- Reduces time to definitive treatment

FAST/eFAST ultrasound can identify free fluid or injury with sensitivity ≈ 0.74, specificity ≈ 0.96 compared to CT scan.

- Hemoperitoneum → sintra-abdominal bleeding
- Pericardial effusion → tamponade
- Pneumothorax/haemothorax → guides chest tube placement
- No recruitment of moving unstable patients to radiology
- Can be repeated serially at bedside

Safe for Hemodynamically Unstable Patients

- Unstable patients may deteriorate during transport to CT.
- POCT allows early surgical decision-making or transfer to OR/ICU.

- Time-sensitive interventions (e.g., thoracotomy, laparotomy) should not wait for CT.

Useful in Resource-Limited or Prehospital Settings

- Rural hospitals, ambulances, and disaster zones may lack CT.
- POCUS and lactate meters allow early triage and referral decisions.
- Improves outcomes even before a patient reaches tertiary care.

NIRS Devices (700–1000 nm wavelength) as Point-of-Care Tools in Trauma Patients

It calculates a tissue saturation index (StO_2 or rSO_2) based on this light absorption, reflecting the balance between oxygen delivery and consumption at the microcirculatory level. Unlike CT scans, which require patient transport and expose patients to radiation, NIRS devices are**:**

- Portable
- Radiation-free
- Repeatable
- Effective even in prehospital or resuscitation bay settings

Devices available named as following and their use

- InSpectra StO_2 Monitor - commonly used in trauma and shock resuscitation studies
- INVOS™ System - cerebral and somatic oxygenation; used in TBI
- FORE-SIGHT™ - crain-specific rSO_2 monitoring
- Equanox™ - portable; used in prehospital care

Applications of NIRS in Trauma

- It cannot replace CT for diagnosis of structural injuries (bleeding, fracture, organ trauma), but can supplement it, especially when imaging is delayed or the patient is unstable.
- Best combined with clinical exam, POCUS, lactate, and CT for full trauma evaluation.
- Haemorrhagic shock- Early detection of impaired perfusion
- Traumatic brain injury (TBI)- Cerebral NIRS for monitoring rSO_2
- Compartment syndrome- Muscle StO_2 monitoring
- Field triage / Mass casualty- Prehospital triage and monitoring

Performance Metrics for NIRS

- Shock Prediction
  - Sensitivity: 84%, Specificity: 85%
  - NIRS detected shock earlier than lactate or SBP
- Compartment Syndrome
  - Sensitivity: 94%, Specificity: 98% for impending compartment syndrome
- TBI Monitoring
  - rSO_2 <50% predicted cerebral desaturation events
  - Sensitivity: 82%, Specificity: 79% vs jugular bulb oximetry
- Limitations of NIRS
  - Skin pigmentation, edema, or probe placement may affect accuracy
  - Limited specificity in some conditions (e.g., sepsis vs trauma)
  - Operator experience still matters (for placement and interpretation)
  - Cannot localize bleeding or structural damage (unlike CT)

Essential Recommendations:

- CT is the gold standard for structural injury but should only be used in stable patients.
- POCUS (FAST/eFAST) enables rapid detection of lethal pathology with high specificity (~96%) and should be used early in unstable patients.
- POCTs enable safe, repeatable, radiation-free monitoring.
- Best trauma care integrates POCTs + clinical judgment + CT

Desirable Recommendations:

- NIRS detects shock and tissue hypoxia earlier than vital signs or lactate, offering continuous, non-invasive monitoring of cerebral and peripheral perfusion.

Chapter 7 – Paediatric Considerations

Every 9 minutes a person dies of trauma, children being 20-25% of these deaths. (176, 177, 178) The majority of these happen in low-to-middle income countries [LMIC] where trauma is the leading cause of death in children. (179, 180, 181) For each child dying, there are probably ten with severe prolonged disability requiring ongoing care, which impacts the child, their caregivers and peers, and the community. (176, 182) There are many more children who have less severe injuries but need medical attention. Importantly, childhood injury numbers will likely rise with increased urbanization and motorization in LMIC. (183)

The major reasons for childhood injury are – in order of burden- road traffic injury, falls, burns, drowning, and poisoning. Not included in this list but, sadly enough, equally creating pediatric victims are war, disasters and more individually inflicted injury due to violence (knives, firearms, etc) or abuse. (184, 185, 186, 187)

While certain dimensions of trauma care are very similar across all ages and causes, many things are specific to children and those involved should be aware of these differences (IFEM PEMSIG white paper). Children (0-18 years of age, as per WHO definition) are more prone to specific trauma mechanisms and incur specific injury patterns and presentations, which is caused by age-dependent anatomic and physiological characteristics, and pathophysiological responses. There is an increased risk of traumatic brain injury [TBI] and brain injury responses differ from those in adults.

Importantly, critically injured or ill children in general are far less frequently encountered than adult victims and individual healthcare workers thus have less exposure, often making them less prepared if and when. (188, 189, 185, 190) Specific equipment and procedures are needed to accommodate all different age groups and pathologies. However, these procedures and suggested treatments have rarely been studied in children and are often extrapolated from adult literature. Direct communication with young children is more difficult and parents are still very much involved (emotionally and as surrogate decision makers).

A Trauma Chain of Survival

A trauma system encompasses the full range of care to all injured within a defined geographic region in a coordinated way. It is strongly integrated within the local public health system and essentially demands a well-developed emergency care system. (191, 192, 193) Such an integrated approach is also described in cardiac arrest, where it is called 'the chain of survival.' (194) Important in that chain of survival is not only each of its individual steps, but also the transitions between them. Optimal outcome can only be achieved when each link is strong – as in a chain- and where possible, further strengthened. As such this chain of survival guides both treatment and (secondary) prevention. The essential components of a pediatric trauma chain of survival and likewise of a trauma system adapted to children are:

1. Early adequate support by bystanders or first aid providers
2. Adequate and timely prehospital care by well-trained EMS teams
3. Adequate and timely emergency department [ED] – operation room [OR] acute treatment by well-trained ED-OR teams
4. Adequate intensive care unit [ICU] treatment
5. Adequate and timely rehabilitation – long-term follow-up

Concerning children, optimising these steps is not easy. Low resources provide extra barriers. Management options and implementation strategies that make sense or prove to work in high resource settings might fail when resources are low. The cost-efficiency of such strategies might largely differ but is rarely researched. Transport and handover provide the links between these 5 steps and are thus key. Primary prevention is obviously crucial as it makes all other steps obsolete and should be seen as the obligatory step 0 (183).

Very early mortality of children after severe trauma is caused by either cardiac, aorta or massive spinal or brain injury. About half of all pediatric trauma deaths occur in the first 6 hours (195, 187). Overall, the highest impact on mortality and morbidity is seen – in order of importance - as a result of traumatic brain injury, massive bleeding, obstructive shock (pneumothorax – tamponade), infection and multi-organ failure. Timely adequate treatment of these problems, adapted to the specific needs of the individual child, will significantly improve outcomes. As described in the chain of survival this care starts as early as the first moments after the incident, lasting beyond intensive care.

1. Early adequate support by bystanders or first aid providers

   While it is not likely that children presenting in cardiac arrest after trauma will survive nor that those severely injured not-in-cardiac-arrest will significantly improve from any action by a bystander or (trained) first responder, we do not see any reason to not promote certain bystander interventions (196). Depending on the possibility of training or (EMS dispatcher) support such actions could include safely opening the airway, external bleeding control (including temporary tourniquet placement), proper immobilization of selected victims, as well as creating as far as possible a safe environment for both the victim and bystanders.

2. Adequate and timely prehospital care by well-trained EMS teams

   In essence, adequate timely prehospital care incorporates three key parts. The first one is the provision of emergency prehospital care for problems that cannot really wait. Second, the decision about which hospital is appropriate for the injured child (by age-adjusted triage, adjusted to the local context) and third, the transportation of the injured children to that appropriate hospital facility. Strategic placement of ambulances with a trained crew, mostly in places where there is a higher likelihood of trauma such as urban centers, might accommodate for this (179). It would clearly not be cost-effective to separately accommodate pediatric and adult victims; thus, these teams will need to get both adult and pediatric trauma training at regular intervals. In contexts where this is not feasible, (community) first responders might provide a valuable alternative. These need proper (ongoing) training and support as well. Prehospital care should be limited to those interventions that are live- or organ saving and have sufficient cost-benefit considering the regional healthcare system and resources available. Proper airway management and spinal immobilization (when indicated) is a crucial part of prehospital care. Early brain protection is imperative for a good outcome and measures to do so should be implemented as soon as possible, including prehospitally (see below under ‘the big five of pediatric trauma care’).

The risk for c-spine injury, even in children after severe trauma, is exceedingly rare. Nevertheless, spinal immobilization should be considered prehospitally in each of them depending on clinical assessment and the use of existing clinical decision rules such as Nexus or the PECARN rules (197, 198, 199, 200, 201, 202, 203, 204). Considering the lack of evidence for and the potential of harm from rigid collars and spine boards, we advise against their use in children (apart from during extraction). If immobilization is considered necessary, this should be done by manual in-line stabilization and subsequent use of headblocks. The rest of the body then should be equally immobilized by means of either a scoop stretcher with appropriate strapping or a vacuum mattress..

The algorithms for advanced life support in those in cardiac arrest should be adjusted to the context of trauma, as in adults (204, 205). Cardiopulmonary resuscitation should focus on reversible causes. Bilateral thoracostomy might be performed to rule out tension pneumothorax. Advanced therapeutic interventions such as thoracotomy and REBOA have been reported in children but the indications to do so should be strict and even then, the outcome is most often dismal (206, 207, 208, 209, 210).

3. Adequate and timely emergency department [ED] - operation room [OR] treatment

   While ABCDE serves as the common language for the whole team taking care of the severely injured child in the ED, care should be provided as a team where team members each have specific tasks to perform in parallel and a team leader coordinates with the overall treatment goals in mind, keeping situational oversight and leading interdisciplinary communication (204, 190). Before the child arrives (from prehospital), as far as possible, teams should prepare themselves in turns of collecting the necessary materials, medications and fluids (blood products). The team leader should run through the case specifics, highlight expected handling and the choreography of the initial resuscitation. The general team approach to the polytraumatized child does not differ much from the adult one. We wanted to highlight the so-called 'BIG FIVE'; topics leading to the most morbidity and mortality in children - each with distinct pediatric considerations and insights.

   Airway: Due to its specific anatomy and physiology, the airway is at increased risk of obstruction in the polytraumatized child (204,190). Maintaining an open airway is therefore a crucial part of the trauma resuscitation, considering also the necessary in-line stabilization of the cervical spine. To do this, the head needs to be a neutral (young child) position or slightly in extension (older child). In some children this demands putting something under the shoulders to accommodate for the large occiput. In children with decreased consciousness, the relatively

large tongue might rapidly obstruct the airway. An oropharyngeal (or rarely nasopharyngeal) airway, appropriately sized, is of great value in these circumstances. An oropharyngeal airway will only be tolerated by children who are deeply unconscious; a nasopharyngeal is more easily tolerated but relatively contra-indicated when there is a risk of neurotrauma. A tracheal tube might provide a more definite and secure airway, but the act of intubation is a high-risk procedure demanding competent providers, strict procedures and good preparation (211, 212, 213, 214). Unless in a situation of 'can't ventilate, can't oxygenate' it is probably advisable to perform bag-mask ventilation with an airway adjunct in place or, if properly trained, use a supraglottic airway (iGel, laryngeal mask) to do so. Intubation can then be performed by the most competent team under the best circumstances possible. Backup plans should be available. The place of videolaryngoscopy in children is yet to be defined and providers should use the technique they are most comfortable with. Once a tracheal tube is in situ, capnography should be attached and monitored. Be aware that children have less functional residual capacity and will quickly desaturate when there is apnea. Extra oxygenation before and during the intubation procedure is advisable. To protect the brain and facilitate the procedure, the use of induction medication is mandatory. However, even with so-called hemodynamic stable medications (e.g ketamine), the team should be prepared for sudden deterioration and hypotension.

Tension Pneumothorax: although children in general have a more compliant thorax and thus need higher force to break, tension pneumothorax – if and when- will rapidly lead to hypoxemia, obstructive shock and death if not treated. The diagnosis is primary clinical, but ultrasound might be a rapid and reliable bedside way of confirmation, also in children. Tension pneumothorax demands immediate treatment. One could do a needle decompression either at the intercostal 2 midclavicular point or -as we advise, in line with the adult guideline- at the intercostal 4-5 anterior midaxillary line. A mini thoracostomy at that position is at least as efficient. A chest drain needs to be put in place afterwards at the earliest convenience.

Haemorrhage: major haemorrhage is an important reason for haemodynamic instability in children. To avoid mortality and morbidity, proper fluid resuscitation is crucial. The historical way of trauma resuscitation resulted too often in hypothermia, coagulation disorders and acidosis ('the triad of death') in adults but definitely also in children who by nature have a less efficient temperature control system, a low absolute circulating volume (approx. 80 ml/kg) and less efficient circulatory compensation mechanisms. Most signs of haemodynamic instability are aspecific and thus it is not easy to identify early shock in these

children. The actual evidence is often of very low certainty to non-existing. We largely based the subsequent suggestions on expert opinion:

- Haemostatic resuscitation demands early blood products (limiting the amount of crystalloids to 10-20ml/kg), including packed red cells, early fresh frozen plasma and timely platelet transfusion. Whole blood (low O titer) can be used as an alternative in specific settings. Products should be delivered warm, at fast rate (via rapid infuser systems) through -as far as possible- large bore catheters or if that is impossible intraosseous access (preferably humeral, to avoid leakage if the bleeding comes from an abdominal large vein). After every 10 ml /kg the clinical condition (and possible improvement) should be reevaluated for set goals (vital signs). Further support of coagulation can be done by giving IV tranexamic acid 20mg/kg bolus and 2mg/kg/h for the next 8hours (antifibrinolysis). According to results of TEG/ROTEM testing, additional suppletion of fibrinogen or clotting factors might be considered. Hypocalcemia – induced by the fluid resuscitation- should be avoided.
- Permissive hypotension: as in adults, some children might benefit from not aiming the blood pressure too high and by doing so allow for better clot formation and less use of fluids and drugs. However, blood pressure should be sufficiently high to allow for organ and specifically brain perfusion, especially when there is concomitant brain injury presumed. While there is no clear evidence to identify an optimal cut-off, based on expert opinion and indirect evidence we advise for isolated penetrating injury to aim for a mean blood pressure at p5 for age (204). When there is a potential of brain injury or in any stump trauma, we advise aiming for mean blood pressure of p50 for age. Be aware that hypotension can also be obstructive, neurogenic, spinal or cardiogenic in origin. Ultrasound might help in differential diagnosis but often this is not easy. Inhibition or loss of sympathetic drive might further worsen circulatory failure. Therefore, even if the initial approach to shock in trauma is fluid resuscitation, at least some patients will (also) need continuous (or limited bolus) vasoactive drugs to keep the mean arterial pressure above set goals.
- Damage control surgery: certain sources of bleeding will need surgery to control. However, with appropriate haemostatic resuscitation, most can be managed conservatively, regardless of the extent of the initial lesion (215, 216). For those hemodynamically stabilized with an arterial blush on imaging, stopping the bleed via interventional radiology might be an option also in older children. If surgery is needed for bleeding control or for e.g. orthopaedic emergencies then these procedures should be

limited to life-, organ or extremity-saving interventions, postponing all other to a later moment.

Brain Protection: in as much as two-thirds of all severe trauma, the brain is injured as well. While little can be done for the primary injuries sustained, most of the damage is actually secondary, meaning that it is still amenable to recuperation if properly managed. As such the actual outcome of traumatic brain injury largely depends on appropriate brain-protective measures and this from prehospital up till during intensive care. There are no specific drugs or treatments to do so but overall, it comes down to -

- Avoiding increased oxygen consumption due to agitation, catecholamine stress, increased temperature, convulsions (incl. non-convulsive status epilepticus)
- Avoiding increased intracranial pressure due to mass effects, oedema, decreased venous outflow (iugular), hypercapnia…
- Avoiding decreased substrate/oxygen delivery to tissue due to hypotension, hypoxaemia, hypocapnia…
- Avoiding direct toxicity to neurons of hyperglycaemia, major sodium shift…

As such, key therapeutic interventions are proper analgosedation, tailoring a normal blood pressure (MAP above p50, if necessary, by means of vasoactive drugs), aiming for normal oxygenation and ventilation (avoiding hyperventilation), adequate positioning of the head (30 degrees, unobstructed iugulars), osmotic drugs (hypertonic sodium) in case of oedema and urgent neurosurgical intervention for epidural or subdural hematoma (with mass effect).

Iatrogenic Effects: while it is important to treat major pediatric trauma in a rapid and aggressive way, some of the things we do might generate more harm than benefit. Given the lack of evidence, many of our actions are either extrapolated from protocols and research in adult patients or are expert-driven or even done out of habit without knowing how and why it became part of our protocol. Examples of such practices are:

- Early intubation without adequate preparation for non-absolute indications (such as decreased Glasgow Coma Score) with concomitant hypoxemia and hypotension
- Spinal immobilisation in children who are agitated and/or by means of spinal boards and stiff collars.

- Massive fluid resuscitation, especially if also crystalloids but even if appropriate blood products, in non-bleeding children with hypotension for other reasons. The obligatory avoidance of vasoactive drugs in hypotensive children.
- Early surgical intervention in abdominal bleeding based on the severity of the lesion on imaging regardless of the possibility to stabilize conservatively, especially for what concerns early splenectomy.
- Inappropriate analgosedation due to fear of side-effects, misinterpretation of clinical signs, presumed need to allow for clinical neurological follow-up with impact on airway, breathing, circulation and brain protection
- Increased radiation exposure due to the liberal use of CT scanning, often similar to adult protocols (whole- body, 3 phase). Imaging by definition, not by decision. While certain diagnoses are difficult to make early on without advanced imaging, sometimes prolonged observation (combined with lab testing) might be an acceptable alternative. Not wanting to miss any injury early on means significantly increasing the number of children that will get a CT without actually needing it. However, this CT scan is not without risk in the long-term. It is therefore imperative that we try to better identify the population of children actually benefiting from CT scan and decide actively for each child which scans are actually needed (according to the ALARA concept: as low as reasonably acceptable). In recent literature several decision rules to guide imaging practices have been researched (217, 218, 219). These rules generally showed good test performance and allowed for significantly less CT scans in clinical settings where there was a high incidence of CT scanning before implementation of such a rule. However, the existing rules tend to have low specificity (especially in young children) and might actually increase imaging in settings that were conservative with imaging at start. In selected populations of stable children, the combination of clinical examination, lab testing, ultrasound, and subsequent observation is a good / the preferred alternative to early CT scan (220).

4. Adequate intensive care treatment & Adequate timely rehabilitation

   The eventual outcome of any individual child after severe injury equally depends on the (intensive) care provided after the initial ED/OR resuscitation. Systems that only provide early resuscitation, even if appropriate, but do not have the capacity for further life- and organ-supporting treatment, whether locally or at a (tertiary) referral hospital (including the means to safely transport interhospital),

are bound to fail. The proven impact on outcomes of adequate and timely rehabilitation makes it a crucial part of any overall 'trauma system'.

Essential Recommendations

- Prehospital care should be optimised. The care of a critically injured child is a continuum, starting prehospitally and ending with rehabilitation: a 'trauma chain of survival'.
- Appropriate airway management is life-saving. Use oropharyngeal airway adjuncts when indicated. Consider early intubation only if competent providers can perform it in well-prepared conditions unless there is a situation of 'can't oxygenate, can't ventilate by any other means'.
- Identify and treat tension pneumothorax immediately
- Do not allow hypotension. Actively search for bleeding and provide appropriate fluid resuscitation if. Consider vasoactive drugs for persistent hypotension or if shock is not due to hemorrhage.
- Provide brain-protective care from early on
- Avoid iatrogenic effects of your diagnostic and therapeutic interventions

Chapter 8 – Summary of Recommendations

Essential Recommendations

- Zero point survey should be integrated into standard major trauma assessment systems where a prenotification process for patients arriving to emergency departments exists.
- Practicing a Shared Mental Model within a trauma resuscitation team will optimise patient outcomes, so that simultaneous assessment and initiation of treatment can occur in the patient
- Resuscitate before intubate / Resuscitation Sequence Intubation are terms that should now be used to prevent mortality/morbidity from a physiologically difficult airway
- Hemorrhage-driven trauma mortality should be recognised as fundamentally a failure of physiology
- Hypocalcemia is an early, prevalent, and biologically plausible contributor to trauma-induced coagulopathy, and thus should be vigorously screened.

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- Ionized calcium, as a critical, yet under-recognized, determinant of coagulation efficiency, cardiovascular stability, and cellular function, should be checked during initial and ongoing assessment.
- Early calcium assessment and correction represents a low-cost, high-yield intervention
- Use blood products early to avoid large crystalloid resuscitation
- Always correct calcium in massive transfusion
- Avoid permissive hypotension in traumatic brain injury
- Severity stratification (GCS + imaging + LOS) is essential to decide appropriate treatment for TBI, the leading global cause of death and disability.
- Whilst primary injury is irreversible; the main aim of resuscitation is to prevent secondary injury. Hypotension (SBP <90 mmHg) or hypoxia doubles mortality.
- Coagulopathy affects about 33% of TBI patients and should be routinely screened.
- Aggressively treat raised intracranial pressure. Monro–Kellie doctrine governs TBI physiology: hemorrhage or edema overwhelms compensatory mechanisms → ICP rise, ischemia, herniation
- CT scan is the acute imaging modality of choice and should be ordered as soon as possible
- ICP and CPP-directed management is central and transfer to a critical care unit able to measure these parameters will improve prognosis.
- Diffuse Axonal Injury often presents with coma and normal ICP
- Seizure prophylaxis for a total of 7 days should be started as this reduces early post-traumatic seizures
- Prioritise circulation over airway in patients who are externally exsanguinating.
- Employ a rapid "scoop and run" approach in prehospital care.
- Initiate early and balanced damage control resuscitation using blood products in specific ratios.
- Utilize point-of-care ultrasound–It's not just for blunt trauma.
- CT is the gold standard for structural injury but should only be used in stable patients
- POCUS (FAST/eFAST) enables rapid detection of lethal pathology with high specificity (~96%) and should be used early in unstable patients
- POCTs enable safe, repeatable, radiation-free monitoring
- Best trauma care integrates POCTs + clinical judgment + CT
- Prehospital care should be optimised. The care of a critically injured child is a continuum, starting prehospitally and ending with rehabilitation: a 'trauma chain of survival'.
- Appropriate airway management is life-saving. Use oropharyngeal airway adjuncts when indicated. Consider early intubation only if competent providers

can perform it in well-prepared conditions unless there is a situation of 'can't oxygenate, can't ventilate by any other means'.
- Identify and treat tension pneumothorax immediately
- Do not allow hypotension. Actively search for bleeding and provide appropriate fluid resuscitation if. Consider vasoactive drugs for persistent hypotension or if shock is not due to hemorrhage.
- Provide brain-protective care from early on
- Avoid iatrogenic effects of your diagnostic and therapeutic interventions

Desirable Recommendations

- Diagnose Shock/occult shock with more modern indicators such as Clinical (VS,SI,e-FAST) and Laboratory (BD, lactate, ROTEM, TEG)
- More research is required as current evidence suggests the lethal diamond does not independently outperform the lethal triad in predicting early mortality
- Damage Control Resuscitation and Damage Control Surgery is an integrated strategy and should be used where facilities allow DCS.
- Use Resuscitative Balloon Occlusion of the Aorta (REBOA) if available to decrease use of blood products for resuscitation.
- Consider a lower blood pressure threshold for triage to a higher level of care given the often operative nature of penetrating trauma.
- Create a local working group to stay up to date with recent developments regarding penetrating trauma. Care of penetrating trauma by location is evolving rapidly.
- NIRS detects shock and tissue hypoxia earlier than vital signs or lactate, offering continuous, non-invasive monitoring of cerebral and peripheral perfusion

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